Surgery of the TRACHEA and Bronchi

Hermes C. Grillo

Illustrations by Edith Tagrin

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Dedication

To my teachers, my colleagues, and my students—whose roles so often coincided.

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Contents

PART 2 THERAPEUTIC TECHNIQUES AND MANAGEMENT

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Preface

This may seem a rather large book to devote to an anatomic structure that measures only 10 to 12 cm in length and that is often considered a passive conduit. In addition, lesions affecting the trachea are hardly common. Their very rarity, however, is a principal reason for this book. I have tried to distill here 40 years of experience, ranging over a period in which contemporary airway surgery essentially developed. I hope that surgeons or physicians facing a clinical airway problem can amplify their knowledge here. Contributors to this book are all practitioners, who write from mature experience.

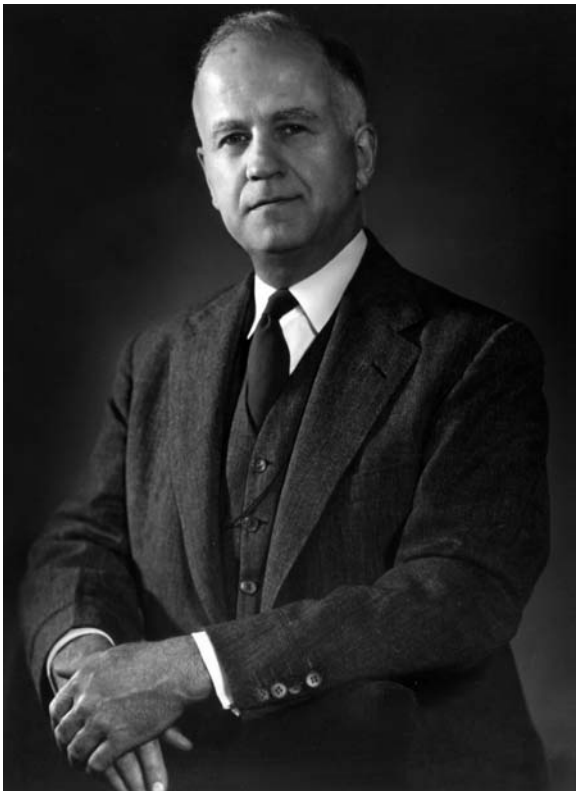
Part 1 presents basic information on the trachea, its diseases, diagnosis, and results of treatment. Part 2 provides a surgical manual plus descriptions of special problems and their management. The organization of the "manual" is based on the fact that surgical strategy often depends as much on the location and extent of a lesion as it does on its etiology. Edith Tagrin has worked long and hard to produce elegant drawings that are detailed and precise.

An experienced thoracic surgeon who observed our tracheal surgery for half a year commented that one of the most important things he had learned was how different each case could be—often in subtle ways. Such differences influence operative decisions and strategies, and affect outcome in major ways. If the reader finds useful guidance in these pages, then the book will have met its goals.

Hermes C. Grillo
September 2003

"A distinction can…be drawn between *specialization* in a technical field and *concentration* in a circumscribed area of learning. The difference lies in the tendency of a technical specialist to exclude all other subjects from his interest and study. The concentrator seeks to maintain an active curiosity and interest concerning all techniques that might be useful in his area of concentration, and views his work in proper perspective with science as a whole."

Edward D. Churchill, 1946



"…the relative success or failure of any surgical procedure lies in attention to what may on first thought appear to be unimportant small details."

Richard H. Sweet, 1950

Acknowledgments

I am thankful to a host of teachers, colleagues, students, friends — and let me not forget patients — who have participated in weaving the fabric of my 55 years of surgery at Massachusetts General Hospital (MGH). I know and regret that the following list of acknowledgments will inevitably be incomplete.

Edward D. Churchill was a thoughtful teacher, guide, and friend. Richard H. Sweet provided impeccable standards in the craft of thoracic surgery. Both of these individuals, giants in their field, stimulated my interest in thoracic surgery. J. Gordon Scannell was a valued teacher, friend, and colleague. Afield from MGH, I learned much about thoracic surgery from Clifford Storey at US Naval Hospital, St. Albans, a billet which I thought of as a valued reward after a year of combat surgery with the First Marine Division in Korea in 1951.

Earle W. Wilkins Jr, Douglas J. Mathisen, Ashby C. Moncure, John C. Wain, Cameron D. Wright, and more recent colleagues, all worked together to establish and develop the General Thoracic Surgical Unit at MGH. Drs. Mathisen and Wright have labored particularly assiduously in the vineyard of tracheal surgery. Paul S. Russell was supportive of this project in tracheal surgery from its beginning. W. Gerald Austen initiated the founding of a thoracic surgical unit in 1969, where this work progressed. David Crockett helped to obtain support for fundamental early laboratory efforts. Sue Robinson tied the thoracic efforts together as Unit Coordinator.

I think fondly and with appreciation of the following research fellows and their productive efforts: Drs. Tsuyoshi Miura, Ellen Dignan, Masazumi Maeda, Yahiro Kotake, Joel D. Cooper, John Mulliken. As well, of many clinical fellows, to name but a few: Piero Zannini, Luciano Landa, Salvino Saita, Joo Hyun Kim. Further, of many thoracic surgical residents, some of whom continue to work with special interest in tracheal surgery, notably Douglas Wood, Richard Heitmiller, Joseph Newton, Mark Allen. This is an admittedly incomplete list.

Colleagues in several disciplines have collaborated helpfully and productively over many years. These include Alexander MacMillan, Reginald Greene, Alfred Weber, Theresa McLoud, and Jo-Anne Shepard in Radiology; Eugene Mark in Pathology; so many able, patient, and innovative anesthetists (I cannot list them all) including Henrik Bendixen, Henning Pontoppidan, Bennie Geffin, John Bland, Roger Wilson, Paul Alfille, William Hurford, and Warren Zapol; Noah Choi in Radiotherapy; and Robert Lofgren and William Montgomery in Otolaryngology.

I cannot begin to express my appreciation for the care given to patients by the devoted and skilled nurses in the operating rooms, respiratory intensive care unit, and surgical nursing unit of MGH. I must mention Ruth Dempsey, RN, who struggled so hard and effectively to get the original Thoracic Surgical Unit up and running, and who guided it so well for many years.

One of my greatest pleasures has been to meet, exchange ideas, and to work with thoughtful and innovative thoracic surgeons around the world, all of whom share a keen interest in tracheal surgery. I mention only the foremost: F. Griffith Pearson of Toronto, Mikhail Perelman of Moscow, Henry Eschapasse of Toulouse, Louis Couraud of Bordeaux, and Masazumi Maeda of Shikoku, Japan. They all contributed generously to my thinking.

I appreciate the valuable efforts of contributors to this book, who offer their special knowledge based on profound experience. Tracheal surgery, perhaps as much as any subdivision of surgery, crosses anatomic boundaries and conventional specialty jurisdictions. Solutions to its problems have arisen from a *concentration* of knowledge and techniques from several areas of *specialization*, rather than from a narrow technical specialization itself. This repeats a distinction made by Edward D. Churchill, who clearly saw the wisdom of general education in surgery as distinct from education in general surgery. He cautioned us to avoid "myopic" specialization.

Edith Tagrin, friend and colleague for many years, has provided a wealth of illuminating, meticulous, and beautiful illustrations. She richly deserves the many awards and praises that she has received in the field of medical illustration. I am grateful for her contributions. The Photography Laboratory of MGH and that of the Department of Pathology deserve special thanks.

This book would not exist without the indefatigable, unfailingly cheerful, and intelligent labors of my colleague and secretary of many years, Patricia Guerriero. The publisher, Brian C. Decker, has patiently and with quiet enthusiasm supported and encouraged this work for more years than either of us wishes to recall.

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And I must express special thanks to two generous friends in Italy, both of the University of Naples, who over the years had provided me with Elysian retreats in which to work — the late, eminent Professor of Surgery Giuseppe Zannini, at his beloved villa, *La Casupola*, on Capri, the most beautiful of isles, and Professor of Architecture Camillo Gubitosi, at *San Gismondo*, the ancient monastery he so attractively restored on a hilltop in Montefollonico, Toscana.

My wife, Sue Robinson, has been most patient and encouraging, although the last thing we need in our home is one more book.

Hermes C. Grillo

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Development of Tracheal Surgery:
A Historical Review

Hermes C. Grillo, MD

Despite the antiquity of tracheostomy, tracheal surgery was the last anatomic subdivision of cardiothoracic surgery to develop. In 1950, Belsey observed, "The intrathoracic portion of the trachea is the last unpaired organ in the body to fall to the surgeon, and the successful solution of the problem of its reconstruction may mark the end of the 'expansionist' epoch in the development of surgery."[1] Following the introduction of intratracheal anesthesia, enormous strides were taken in pulmonary surgery in the 1930s, in esophageal surgery in the 1940s, and in cardiac surgery in the 1950s after cardiopulmonary bypass became a reality.[2,3] In 1961, Richard Meade noted, "Carcinoma of the trachea is a rather rare lesion, and when it is found, it is usually found to be entirely inoperable. In rare instances, the lesion is so localized that the involved trachea can be resected, and with mobilization, the ends can be brought together. This is seldom true and one is faced with the problem of what to do after resection of the trachea."[4] The 1960s proved to be a decade when advances in tracheal surgery quickened.[5] Thus, by 1990, resection rates for tracheal tumors reached 63% for squamous carcinoma, 75% for adenoid cystic carcinomas, and 90% for other tumors.[6]

The following is not a comprehensive review of the literature on tracheal surgery. Rather, it is a selective account of tracheal surgical development. Current references are not necessarily included unless they report progress in the fundamentals or significant evolution of the techniques. For historical reasons, I have therefore often referred to an author's earlier paper rather than to more complete later reports. Emphasis is on the beginnings and early development of important concepts and procedures. I am certain that there are omissions in this account, for which I express regret. The review is divided into two parts. The first part traces the evolution of techniques of tracheal surgery. The second part records the acquisition of information about characteristics and treatment of specific diseases of the trachea. There is, of course, considerable overlap.

Techniques of Tracheal Surgery

Tracheostomy

Even a brief history must note the ancient use of tracheostomy for a variety of indications. The story has been traced by a number of authors.[7–10] Although Aretaeus and Galen remarked on the use of tracheostomy in the

second and third centuries, the *arteria aspera*, the "rough artery," as the trachea was known for generations, only slowly entered the surgical theater. The specific technique of Antyllus in the fourth century CE is recorded.[7] Fabricius of Aquapendente, who introduced the idea of a tracheostomy tube, warned of the danger of this intervention. Tracheostomy was regarded with fear and considered inappropriate by most. In 1546, Antonio Musa Brasavola of Ferrara treated a pharyngeal abscess by tracheostomy after the patient had been refused by barber surgeons. In his thorough and excellent review, Goodall identified this as the first recorded successful tracheostomy, despite many ancient references to the trachea and possibly to its opening.[7] Marco Aurelio Severino used tracheostomy during an epidemic of diphtheria in Napoli in 1610, performing it through a vertical incision recommended by Fabricius (Gerolamo Fabrizio d'Aquapendente).[11] In Paris, 1620, Nicholas Habicot performed tracheostomies, which he termed "bronchotomy," for one patient who had blood clots in the trachea, and for another who attempted to foil a highwayman by swallowing a bag of gold coins which then stuck in his esophagus and compressed the airway. Tracheostomy relieved the obstruction. We have no record of what happened to the bag of gold. Surprisingly contemporary tracheostomy devices are illustrated in seventeenth-century texts, including Habicot's *Question Chirurgicale* (Figure 1), Sanctorius' (Santorio Santorio) *Commentaria* in 1625, and Julius Casserius' *Tabulae Anatomicae* in 1627. Thomas Fienus of Louvain used the word "tracheotomy" in 1649, although it was not often so called for another century.

Over the centuries, a few reports of successful tracheostomies have been made.[7] A drowning victim was treated with tracheostomy by Georges Détharding in 1714. In 1720, René-Jacques Croissant de Garengeot described the division of the thyroid isthmus in order to accomplish a tracheostomy, using a long vertical incision that went almost from chin to sternum. He further argued that failure of the tracheostomy was often due to its being performed too late. Lorenz Heister, in 1718, is said to have been the first to use the word "tracheostomy." In 1730, George Martin described an inner cannula for the tracheostomy tube, a device suggested to him by a layman. Chovell, in 1732, performed a tracheostomy at the request of a patient who faced death by hanging. Unfortunately, this did not save the accused. Goodall found reports of 28 successful tracheostomies done prior to 1825, when Pierre Bretonneau in Tours used tracheostomy with success in treating "croup."[7] In Paris, 1831, Bretonneau's pupil, Armand Trousseau, applied the technique in management of diphtheria, saving about a quarter of 200 children who were dying from the disease.

Tracheostomy changed little technically, although controversy continued about its indications, locations, and hazards.[9] Chevalier Jackson largely cast it in its modern form, cautioning against tracheostomy high in the trachea.[12] He believed that "tracheotomy is the worst done of all operations."[13] Tracheostomy found application in general anesthesia, but was soon displaced by endotracheal intubation.[14] As diphtheria waned, tracheostomy was used in poliomyelitis, to prevent infection, in head and chest injuries, after major surgery, and to reduce dead space. The endotracheal tube largely replaced tracheostomy as a preferred method to establish an emergency airway. Later, tracheostomy vied with endotracheal intubation for management of secretions, and, subsequently, as a route for mechanical positive pressure ventilation. A high incidence of complications was recognized even prior to the frequent appearance of postintubation injuries.[8,15,16] Plastic surgical closure of a persistent tracheostomy by a cutaneous inversion technique was described by Lawson and Grillo in 1970.[17]

Repair and Healing of the Airway

An ancient concern that cast a shadow on tracheal surgery into the twentieth century was that cartilage healed poorly. Hippocrates had cautioned, "The most difficult fistulae are those which occur in the cartilaginous areas…."[18] In the second century CE, Aretaeus pronounced, "The lips of the wound do not coalesce, for they are both cartilaginous and not of a nature to unite."[7] As late as 1990, Naef repeated that "tracheobronchial tissue, as compared to the stomach, intestine, or even skin, does not heal well… both the rigidity and the poor blood supply of the cartilaginous structure are definitely major handicaps."[19]

FIGURE 1 *Tracheostomy pictured by Nicolas Habicot in* Question Chirurgicale. *Par laquelle il est demonstré que le chirurgien doit assurément pratiquer l'operation de la bronchotomie. J. Corrozet, Paris, l620.* A, *The patient.* B, *The larynx.* C, *The wound or bronchotomy.* D, *The instrument for bronchotomy.* E, *The hollow cannula.* F, *The straps for fastening it on the neck.* G, *Plain smooth band to apply over the cannula to scatter the air stream.* H, *Needle to suture the wound when one removes the dressing to make the wound heal.*

Nonetheless, examples of early attempts and sometimes success in bronchial and tracheal repairs after trauma are recorded. Indeed, *The Rigveda*, a book of Hindu medicine dating from between 2000 and 1000 BCE, noted that the trachea can reunite "when the cervical cartilages are cut across, provided they are not entirely severed."[9] Ambroise Paré described suture of tracheal lacerations in the mid-1500s in three patients, the first from a sword wound, and the latter two from knife wounds.[20] The first patient survived despite a concomitant injury to the internal jugular vein. The second patient suffered division of both the trachea and esophagus and died. We do not know the outcome of the third patient. Brasavola observed recovery after a suicide attempt severed five tracheal rings.[7]

Eventually, cumulative clinical experience in the twentieth century established that the trachea healed firmly with suture repair after laceration or rupture.[21–27] Jackson and colleagues demonstrated firm healing of experimental bronchial anastomosis in 1949.[28] In 1950, Daniel and colleagues confirmed fibrous tissue repair of linear tracheal incisions in the laboratory, as did Rob and Bateman clinically in 1949.[29,30] Quinby and Morse pointed out experimentally, for the first time in 1911, the importance of peribronchial tissue in bronchial closure.[31] In 1942, Rienhoff and colleagues made fundamental observations that bronchial healing after pneumonectomy was accomplished by new connective tissue, which grew over the ends of the stump, rather than by mucosal healing alone.[32]

End-To-End Tracheal and Bronchial Anastomosis

Glück and Zeller, in 1881, demonstrated healing after end-to-end tracheal anastomosis in dogs and believed the technique could be applied in man.[33] Colley, in 1895, in order to avoid stenosis, tried elliptical and bayonet anastomoses in dogs after resecting five rings.[34] Primary anastomosis of the cervical trachea, after limited resection for post-traumatic stenosis, followed in 1886 by Küster, apparently the first in man.[35] In 1898, Bruns performed an extended lateral excision of a papillary tumor in the cervical trachea, but managed the tracheal defect by packing and with a cannula.[36] Complex methods for repair of cervical tracheal defects, with skin or fascia lata, were also explored in the early twentieth century by Nowakowski in 1909 and by Levit in 1912, among others.[37,38] Eiselsberg successfully performed a second resection of 1.5 cm of trachea in one patient.[39] Mathey and colleagues commented in 1966, "This type of radical tracheal surgery was then forgotten for half a century."[40]

The era of open thoracic surgery had arrived. By 1936, Churchill had refined the technique of lobectomy to achieve a 2.6% mortality rate.[41] As interest in bronchial and tracheal surgery grew by the mid-twentieth century, laboratory experiments confirmed that healing followed end-to-end anastomosis of both bronchi and trachea, although sometimes with stenosis.[28–30,42–45]

Bronchial repair after trauma proved the feasibility of airway reconstruction. Sanger described bronchial repair in patients during World War II.[46] In 1949, Griffith resected a stricture and anastomosed the bronchus 3 months after rupture.[47] Other late repairs of ruptured bronchi followed.[48] Scannell first performed immediate repair of a bronchus ruptured during closed injury in 1951.[49] Belcher in 1950 and Mathey and Oustrieres in 1951 reported reanastomosis of main bronchi after accidental division during surgery.[50,51]

Earlier cautious enlargement of bronchial stenosis by wire-supported dermal grafts were replaced by resection and reconstruction.[52,53] The technique was applied to low-grade tumors and to carcinomas as sleeve lobectomy evolved.[54–58] The evolution of sleeve lobectomy is described in more detail in Chapter 16, "Bronchial Sleeve Resection." Concurrent vascular sleeve resection was also pursued by Johnston and Jones.[59] Main bronchial resection without removal of lung tissue was extensively described by Newton and colleagues.[60]

Inhibitions to Tracheal Reconstruction

With retrospective wisdom, we may ask, "What were the barriers to application of the bronchoplastic and tracheal anastomotic techniques, just noted, to clinical tracheal resection and reconstruction?" I have men-

tioned a persistent suspicion that tracheal cartilage healed poorly. A second, more insistent concern was that only a very limited segment of trachea could be removed and reanastomosis accomplished. In 1909, Nowakowski placed the limit of resection at 3 to 4 cm, from cadaver studies.[37] Colley and Küster respectively reported resections of three rings and 2 to 4 cm.[34,35] Rob and Bateman, on the basis of cadaver dissection, placed the limit at 2 cm.[30] Belsey believed that three or four rings, about 2 cm, was the limit in man.[1] Cantrell and Folse placed the limit at two rings if over 80 years of age.[61] Nicks cited "one inch or more" as a limit in the cervical trachea.[62] These presumed limits led to devising complex methods of cervical tracheal reconstruction with available tissue flaps and transfers, and, further, to a century-long search for a means of tracheal replacement. This search ranged over foreign material in many forms, autogenous tissue constructions, tissue and foreign material complexes, fixed or "tanned" tissues, transplantation, and, recently, tissue engineering. Success has eluded investigators to date. The story of this frustrating pursuit and the reasons for its overall failure thus far are detailed in Chapter 45, "Tracheal Replacement."

An additional difficulty for reconstruction was maintenance of safe, continuous, and stable ventilation throughout the procedure, especially for intrathoracic tracheal operations. The evolution of anesthetic techniques is discussed later. Finally, primary tumors of the trachea remained rare, as can be seen from earlier chronicles of their occurrence.[63,64] Stenoses from traumatic, iatrogenic, or inflammatory causes were not seen frequently before 1960. Thus, any single thoracic surgeon was not often challenged. Each case was largely dealt with in ad hoc fashion.

Primary Resection and Reanastomosis: Initial Experiences

In the mid-twentieth century, the recrudescence of interest in tracheal surgery was marked by experiments in tracheal healing and replacement, and by renewed clinical efforts. Earliest attempts at reconstruction of the cervical trachea were still most often by staged, complex repairs, typified by Crafoord and Eindgren's cutaneous reconstruction after tumor removal in 1945.[65] Belsey appears to have been the first to have dared to remove intrathoracic tracheal tumors, but his repair was with wire-supported fascia, leaving a residual strip of mucosa for continuity and for epithelial regeneration.[1] Clagett and colleagues and others followed, using polyethylene tubes or patches to repair the defects.[66] The story of these efforts to replace the trachea partially or circumferentially is related in Chapter 45, "Tracheal Replacement."

Despite continued concerns about the feasible length of tracheal resection and lingering doubts about cartilaginous healing, a number of successful resections and reconstructions with primary anastomosis were described in the 1950s and early 1960s, most often for shorter, benign lesions such as stricture.[66] Conley successfully resected the second and third rings for scar in 1953, with end-to-end anastomosis.[67] Kay removed four rings of proximal trachea for leiomyoma, without event, in 1951.[68] Sweet, in 1952, resected a cervical "cylindroma" with end-to-end anastomosis and questioned whether this might be possible intrathoracically.[69] Macmanus and McCormick, in 1954, excised a three-ring segment for the same tumor, which lay about 2 cm above the carina, with end-to-end repair.[70] An anastomotic leak was patched with fascia lata and a protective tracheostomy added. Forster and colleagues reported in 1957 and 1958 a series of three successful cervical and cervicomediastinal tracheal resections with primary anastomosis of 1.5, 4, and 3 cm for tumor, post-traumatic stenosis, and postintubation stenosis, respectively.[71,72] Other similar resections were reported separately by Binet and Miscall and their colleagues.[73,74] In 1959, Flavell had successfully corrected a postintubation stricture at the thoracic outlet by resection, but did this from a difficult, transthoracic approach—an error that was to be repeated later by other surgeons.[75] Mattes performed a 4 cm transthoracic lower tracheal resection for cylindroma in 1958, wrapping the anastomosis with pleura.[76]

Indicative of the revived interest in tracheal surgery was the extensive report in 1960 by Baumann and Forster of worldwide experiences in tracheal surgery.[77] They pointed out that improvements in diagnosis (endoscopy) and technical and ventilatory methods had served to widen the field beyond tra-

cheostomy and endoscopic treatment alone. At the same time, the potential for surgery of the *thoracic* trachea was of exciting interest.

Anatomic Mobilization of Trachea

These mid-century experiences in tracheal reconstruction, chiefly in the upper trachea, and most often of limited extent, made it clear that the basic techniques of tracheal anastomosis could achieve sound healing. The "2 cm rule," which had served to inhibit advances in tracheal surgery, was now challenged by experimental studies reinvestigating the extent of trachea that could be removed, and approximation achieved by *anatomic tracheal mobilization*, *without use of prosthetic replacement*. Clinical experiences, especially with intrathoracic and carinal lesions, contributed to widening the possibilities for more extended resection.

Ferguson and colleagues determined the extensibility of human trachea from cadavers to be 35% at 29 years and 17% at 76 years, with the most stretch reached with 200 g of tension.[43] In living dogs, the majority of resectable length was obtained at 450 g of tension at the anastomosis, which is about 30 to 35% of the tracheal length. Michelson and colleagues, in an effort to increase the length of resectable trachea, freed the right main bronchus in dogs by incising the right pulmonary ligament and resecting the left main bronchus at the carina, and then reanastomosing it to the bronchus intermedius.[78] This permitted resection of twelve rings in the dog. They found that the human trachea could be stretched 4 to 6 cm by mobilization, and that an added 2.5 to 5 cm could be obtained by the maneuver described in dogs. Tracheal elongation in fresh human cadavers, with the same dissection and 450 g of upward pull, allowed 2.5 to 3 cm elevation after division of the pulmonary ligament, and 5 to 6 cm after freeing the left main bronchus in four cadaver subjects under 50 years of age. Respectively, 1 to 1.5 and 2 to 3 cm were measured in four subjects at 50 to 75 years of age. Cantrell and Folse sought to determine the limits of feasibility of primary anastomosis in repair of circumferential defects.[61] In resection of 20 to 58% of dog trachea, the suture line tension ranged from 400 to 2,750 g. The tension required for anastomosis varied markedly between flexed and extended neck positions. Disruption of anastomosis occurred between 1,700 and 3,100 g, at resection lengths of 46 to 63% of the trachea. However, they noted in human trachea obtained at autopsy that resection of more than 2 cm over the age of 80 produced unacceptable anastomotic tension, based on experimentally derived standards.

In 1959, Harris showed radiologically that neck extension elongated the trachea by 2.6 cm.[79] Som and Klein extended the length of human cadaver trachea by only 1.6 cm by circumferential incision of the intercartilaginous annular ligaments.[80]

Grillo and colleagues reported in 1964, from autopsy studies in man, that over half of the adult trachea could be resected and continuity reestablished by full mobilization of limiting structures (Figure 2).[81] Steps in mobilization were 1) right hilar dissection and division of right pulmonary ligament, 2) division of left main bronchus, and 3) freeing pulmonary vessels from the pericardium. With the subject's neck in neutral position, these steps permitted tracheal excisions averaging 3 cm (3 to 8 rings), 2.7 cm (3 to 12 rings), and 0.9 cm (0.5 to 3 rings), for a total of 6.4 cm (11 to 18 rings). Anastomotic tension rose exponentially with resection of successive 1 cm segments, from 25 g at 1 cm to 675 g at 7 cm. Age did not prove to be seriously limiting. This was considerably below the biologically dangerous limit of 1,700 g determined by Cantrell and Folse.[61] Division of the left main bronchus allowed the advancement of the distal tracheal stump and right main bronchus.

In addition, if an even more extended resection was to be necessary, division of the cervical trachea two to three rings below the cricoid allowed this segment of cervical trachea to be devolved into the mediastinum with intact lateral vascular supply.[81] This maneuver proposed to allow reconstruction of the intrathoracic trachea by simple anastomosis, while permitting later staged reconstruction of the cervical trachea, which would be more safely possible. Because of the complexity of this last approach, division and reimplantation of the left main bronchus was later applied clinically, only in the case of carinal reconstruction, and then, rarely.

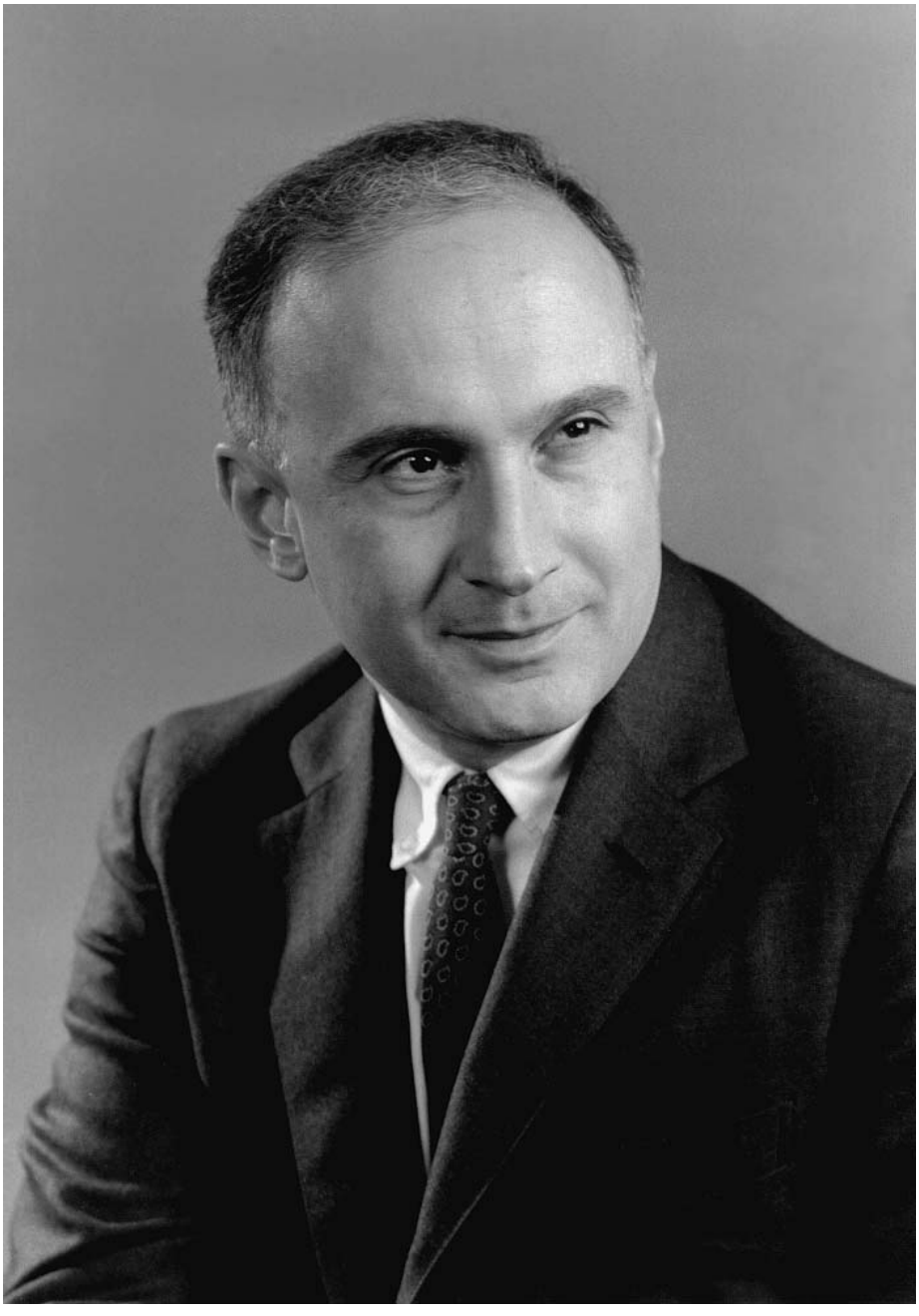

FIGURE 2 *Hermes C. Grillo, MD, in the 1960s, when work on tracheal surgery was underway at Massachusetts General Hospital (MGH). He became the first Chief of General Thoracic Surgery at MGH when a specialized unit was founded in 1969.*

Stimulated by Grillo's clinical experiences with cervical tracheal resection for postintubation stenosis, Mulliken and Grillo reported in 1968 an investigation of the amount of trachea that might be resected by cervical and mediastinal mobilization and still permit anastomosis, leaving the pleural cavity intact.[82] Pretracheal mobilization was done down to the carina, with division of the thyroid isthmus, in cadavers. With the neck in 15 to 35 degrees of flexion, 1,000 to 1,200 g of tension applied to the divided tracheal ends

permitted an average resection and reapproximation of 4.5 cm (7.2 rings). Right hilar mobilization with the pleura open allowed an increment of resection of 1.4 cm (2.5 rings), giving a total of 5.9 cm. The average tracheal length was 11 cm. Cervical flexion permitted a gain of 1.3 cm (2.5 rings) over the neutral position. Thus, cervical flexion and pretracheal mobilization alone appeared to allow significant cervical or cervicomediastinal resection and anastomosis, especially important for the postintubation lesions, which were increasing in frequency, and that often occurred in patients who could not tolerate thoracotomy.

Appreciation of the possible degree of tracheal mobilization, based upon anatomic principles(ie, pretracheal mobilization, cervical flexion, hilar dissection, including intrapericardial freeing, and mobility of detached main bronchi), made possible a systematic and aggressive approach to tracheal resection and reconstruction not previously conceived. The episodic ad hoc approach, which produced single case reports, at times almost expressing a surgeon's surprise at what he was able to accomplish, yielded to more confident and planned approaches. Using such principles, significant series of resections and reconstructions of cervical and thoracic tracheae for stenosis and tumor were reported by Grillo, Deverall, Perelman, Naef, Couraud, Pearson, Dor, Levasseur, and Harley and their colleagues.[83–93]

Laryngeal Release

An additional dividend for extended upper tracheal resection came from otolaryngology. Ogura and colleagues had suggested dividing hyoid muscles to help close the gap produced by resection of a subglottic laryngeal stenosis.[94,95] In 1969, Dedo and Fishman offered thyrohyoid laryngeal release as a necessary adjunct to tracheal resection for stenosis.[96] Division of the thyrohyoid muscles, the superior cornua of the thyroid cartilage, and of the thyrohyoid membrane, with care to preserve superior laryngeal nerves, allowed the larynx to drop about 2.5 cm. Montgomery described an alternative method for laryngeal release—suprahyoid release.[97] Muscle attachments to the superior surface of the hyoid bone, the stylohyoid muscles, and the hyoid bone anterior to the digastric slings were all divided, allowing the larynx to drop. It was the opinion of both Grillo and Pearson (unpublished, c. 1979) that less severe deglutitional disorders of shorter duration followed suprahyoid release than thyrohyoid release. Release is not routinely necessary for upper tracheal resection.[98] Grillo observed clinically that laryngeal release did not transfer effective relaxation for lower tracheal or carinal resections.[99] Valesky and colleagues confirmed this in cadaver studies.[100]

Tracheal Blood Supply

The detailed arterial supply of the trachea was described as a necessary corollary to tracheal surgery. Grillo emphasized the entry of small segmental arteries via "lateral pedicles" of tissue attached to either side of the trachea.[84] Miura and Grillo precisely defined the blood supply to the upper trachea in 1966, usually from three principal branches of the inferior thyroid artery, with the first (or lowest) branch most often predominant.[101] Salassa and colleagues completed a definitive study of the tracheal blood supply in 1977, confirming Miura and Grillo's description of the cervical tracheal supply, and mapping the arterial supply of the thoracic trachea from bronchial, supreme intercostal, subclavian, right internal thoracic, and innominate arteries.[102] Segmental tracheoesophageal arteries connected often to lateral longitudinal arteries and then to transverse intercartilaginous arteries. Three to 7 tracheal arteries were found in the "lateral pedicles."

Unlike the intramural collateral of tracheal blood supply in the dog, which maintains tracheal viability despite complete circumferential dissection, and subsequent division and anastomosis, the same procedure in man has led to necrosis.[61]

Carinal Resection and Reconstruction

At the lower end of the trachea, the special problems (anatomic, technical, and anesthesiologic) of carinal reconstruction loomed. Lesions, most often neoplastic, were centered in the carina, extended to the

carina from low in the trachea, or to the carina from main bronchi or lungs. *Experimentally*, Grindlay and colleagues resected right lung and carina in dogs in 1949, with end-to-end anastomosis of trachea to left main bronchus.[103] Ferguson and colleagues also performed right and left pneumonectomies in dogs in 1950, with resection of carina and end-to-end anastomosis.[43] In 1951, Juvenelle and Citret, working at the University of Buffalo, showed experimentally the feasibility of lateral implantation of bronchus into trachea, without loss of blood supply and without interference in ventilation.[104] They further described experiments in which they resected the carina with a three to four ring segment of trachea, and then anastomosed the trachea to the right or left main bronchus and implanted the other main bronchus into the side of the trachea. They found it necessary to free the trachea to reduce otherwise excessive tension. Additionally, they remarked that freeing the trachea permitted anastomosis of the trachea directly to right and left main bronchi without excessive tension, after short segment resection.[105] Meyer and colleagues experimentally implanted the right upper lobe and right main bronchus into the trachea in 1951.[106] Ehrlich and colleagues, in 1952, transposed a right main bronchus to the lateral tracheal wall, and later resected the left lung and carina in dogs.[107] Kiriluk and Merendino, in 1953, described a variety of experimental tracheal, bronchial, and carinal reconstructions, including reapproximation of both main bronchi to the carina and tracheobronchial anastomosis after carinal pneumonectomy.[45] Nicks similarly reconstructed the carina after resection in pigs in 1956, but under hypothermia.[62] In 1958, Björk and Rodriguez described experiments in reconstruction by direct anastomosis after resection of the carina and twelve tracheal rings in dogs.[108] The right main bronchus was sutured end-to-end to the trachea and the left main bronchus end-to-side to the intermediate bronchus. This followed the similarly successful clinical procedure by Barclay and colleagues, described below.[109] The same anastomoses after carinal resection were performed in dogs in 1969 in confirmatory studies.[110]

Clinically, Abbott repaired large oval defects created by right pneumonectomy and right carinal lateral excision for bronchogenic carcinoma in 5 patients in 1950, by transverse closure.[111] Two of the patients died. Other patching techniques were used to repair such lateral defects, including dermal grafts, synthetic materials, and patches or flaps of retained bronchial wall.[112,113] These complex and frequently unsuccessful patch techniques are reviewed in Chapter 45, "Tracheal Replacement."

In 1951, Mathey locally resected a "cylindroma" of the back wall of the trachea at the carina, including posterior walls of both proximal and main bronchi.[114] Repair was effected by longitudinal suture of the medial bronchial margins and transverse suture of the remaining defect. In these years, surgeons struggled with the problem of tracheobronchial anastomosis at the carina. In 1954, Crafoord and colleagues reported anastomosis of the bronchus intermedius to the trachea at the site of main bronchial origin, after upper lobectomy and bronchial excision.[115] The next year, Björk obtained access to the carina from the left chest, mobilizing the aorta after division of four pairs of intercostal arteries, in order to successfully resect the left main bronchus and anastomose its lobar bifurcation to the prior origin of the bronchus at the trachea.[116] In 1959, he presented follow-up of 16 patients who had undergone bronchotracheal anastomosis, with four stenoses.[117] Abbey-Smith and Nigan described a similar left-sided approach in 1979, for amputation of the left main bronchus at the carina, for pneumonectomy in a case of proximal lung tumor.[118]

Barclay and colleagues, in 1957, resected about 5 cm of trachea and carina to remove a recurrent adenoid cystic carcinoma.[109] Division of the pulmonary ligament allowed anastomosis of the trachea to the right main bronchus. The left main bronchus was anastomosed end-to-side to the bronchus intermedius. Intermittent ventilation sufficed for the second anastomosis. A second patient was handled identically. Both patients recovered. The authors reported in the same paper that dissection in fresh cadavers prior to operation permitted resection of 6 cm of trachea, using this technique. They also proposed, where carinal resection was not required, to close the left main bronchial stump. Eschapasse and colleagues used this technique in 1961.[119] Archer and colleagues similarly excised a granular cell myoblastoma at the carina in 1963.[120] The

procedure was a major step in carinal surgery. Grillo and colleagues described resection of the carina and trachea for a length of 4 cm to remove adenoid cystic carcinoma, in 1963.[121] The right hilum was mobilized and the trachea anastomosed to the right main bronchus. The left main bronchus reached the trachea easily enough to be anastomosed there end-to-side. The patient did well. Cross-field intubation and ventilation were used. Temporary occlusion of the pulmonary artery to the nonventilated lung eliminated shunting, but later rarely proved to be necessary. In 1966, Mathey and colleagues reported results in 7 patients, who underwent carinal excision with or without bronchial resection, using thoracotomy.[40] They believed, however, that sternotomy might be preferable. Three patients had pneumonectomy, and 2 had partial lung resection. The following anastomoses were done: trachea to main bronchus; side-to-side left main and intermediate bronchus and both end-to-end to trachea; 2 patients had dermal graft patches. There were 2 postoperative deaths. Eschapasse and colleagues, in 1967, cited 3 patients who had circumferential resection of the entire carina with primary reconstruction.[122] Anastomoses were of right main bronchus to trachea with left main end-to-side to bronchus intermedius in 2; left main to trachea with intermediate bronchus to left main. One patient died postoperatively. Eschapasse favored right thoracotomy, cross-table ventilation, avoidance of prostheses, and primary reconstruction (Figure 3). Anesthesia for carinal resection, which had initially seemed formidable, was managed easily enough in patients by cross-table ventilation of the trachea and bronchi as the procedure progressed, so that ventilation was not interrupted or uncontrolled at any point.

Nonetheless, the anesthetic challenge of carinal resection suggested the use of extracorporeal circulation to some. Nissen removed a "malignant adenoma" in this way.[123] Under bypass, Woods and colleagues excised recurrent "cylindroma" from the carina with very limited margin.[124] Reconstruction was by suture and a patch of skin supported by wire mesh. Adkins and Izawa performed lateral resection of the carinal wall for cylindroma, patching the defect with Marlex and mediastinal tissues.[125] As might be expected, granulation tissue formed at the patch site. The considerable potential for hemorrhage due to the need for heparinization during bypass was not encountered in these technically limited cases.

Experience with carinal resection and reconstruction grew slowly. In 1974, Eschapasse collected 19 cases from several French teams, Perelman and Koroleva recorded 29 carinal resections with reconstruction in 1980, and Grillo had performed 36 carinal reconstructions by 1982.[99,126,127] Twenty-three of Grillo's group were primary tracheal neoplasms, 5 were bronchogenic carcinomas, and 8 were inflammatory lesions. Eleven were reconstructed without loss of lung tissue. On the basis of this experience, Grillo presented a comprehensive schema for carinal reconstruction.[99] For short resections, carinal restoration was by side-to-side main bronchial anastomosis, which was then joined end-to-end to the trachea; for longer lesions, the trachea was placed end-to-end to the left main bronchus (if the gap was less than 4 cm) and the right main bronchus end-to-side to the trachea; for still more extensive tracheocarinal removal, the "Barclay" anastomosis of the right main bronchus to the trachea and the left main bronchus end-to-side to the intermediate bronchus was used. Other special problems were also presented, including the problem of lesions involving a long length of trachea and also of the left main bronchus. Recent exploration of problems with carinal reconstruction has updated this experience in 143 resections.[128]

Approach for carinal resection via right thoracotomy has been preferred by most surgeons.[99,121,126,129,130] Left thoracotomy with subaortic dissection was employed for specific lesions, principally those involving the left main bronchus and the carina, but little of the tracheal length.[40,99,126,131,132] Left thoracotomy with retroaortic dissection was also explored early, but failed to gain acceptance.[116,118] Median sternotomy for carinal access was described in 1907 by Goeltz for foreign body removal, in 1960 by Padhi and Lynn for bronchopleural fistula, in 1961 by Abruzzini for treatment of postpneumonectomy tuberculous fistulae, and was reintroduced with anterior and posterior pericardial opening by Perelman (Figure 4).[130,133–135] Pearson and colleagues favored this approach for cari-

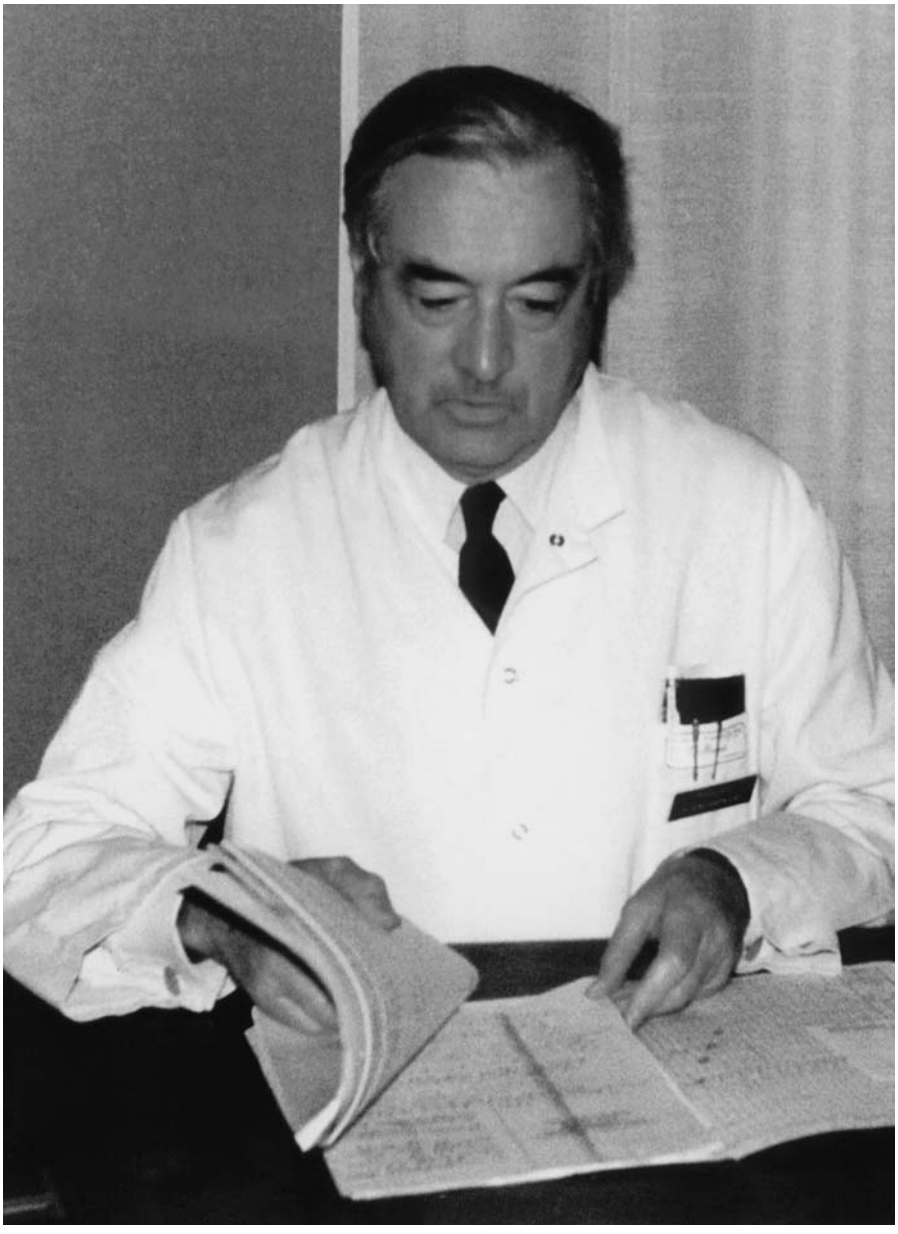

FIGURE 3 *Henry Eschapasse, MD, Chief of Thoracic and Cardiovascular Service Emeritus, Regional Hospital Center of Toulouse, and Professor Emeritus, University of Toulouse. In the decades post World War II, there was great interest and activity in tracheal surgery and pathology in France. Dr. Eschapasse was a leader in this field, and especially interested in the study of primary tracheal neoplasms and carinal reconstruction. Toulouse became a center for tracheal surgery.*

nal resection.[136] Maeda and colleagues added left anterior thoracotomy to sternotomy to improve access.[132] Grillo employed bilateral thoracotomy ("clamshell" incision) for free access to the carina and to both thoraces for treatment of complex lesions, especially those involving the left main bronchus, carina, and a long extent of the lower trachea.[99]

FIGURE 4 *Mikhail I. Perelman, MD, Consulting Surgeon, National Research Center of Surgery, Moscow, and Professor of Surgery and Physiopneumonology, Moscow Medical Institute. Professor Perelman had an early interest in airway surgery, acquired a large clinical experience, and published the first comprehensive books in the field. Tracheal tumors were a special interest of his.*

Carinal Pneumonectomy

Surgeons early conceived of extension of pneumonectomy for bronchogenic carcinoma to include tumors that also involved the carina.[111,113,137] Carinal pneumonectomy with anastomosis of the terminal end of the trachea to the left main bronchus was reported in 1958 by Mattes and in 1959 by Gibbon.[76,138] Hardin and Fitzpatrick, in 1959, excised the carina for bronchogenic carcinoma, reconstructing the carina by direct suture with the aid of free cartilage graft, using ventilatory anesthesia delivered by a tube placed in a distal left main bronchial aperture.[139] The graft was taken from an uninvolved portion of the right bronchus.

MacHale did the same in 1972.[140] In 1966, Thompson described anastomosis of the left main bronchus to a tailored trachea after right pneumonectomy for squamous cell carcinoma, which included a sleeve of carina.[141] Also in 1966, Mathey and colleagues expressed a preference for circumferential carinal resection over lateral resection and patching.[40] Grillo included carinal pneumonectomy with circumferential resection in the spectrum of techniques of carinal resection and reconstruction.[99]

Carinal pneumonectomy for bronchogenic carcinoma became further established with significant series reported by Jensik in 1972, Ishihara in 1977, Deslauriers in 1979, Dartevelle in 1988, Tsuchiya in 1990 and their colleagues, and Mathisen and Grillo in 1991.[142–147] The initially high operative mortality of nearly 30% proved to be largely due to a form of acute respiratory distress syndrome labelled postpneumonectomy pulmonary edema, of noncardiogenic origin. Mathisen and colleagues showed favorable response to prompt treatment with nitric oxide.[148] Believed to be the result of barotrauma, this dread complication has reduced in incidence with close attention to ventilatory volumes and pressures, so that mortality has fallen to about 10% or lower.[145,149,150]

Anesthesia for Tracheal Surgery

McClish and colleagues noted that concern about anesthesia for major airway reconstruction "stems from the complexity of simultaneously controlling the airways, maintaining satisfactory gas exchange, and ensuring good surgical exposure to the trachea."[151] The technique of ventilation across the operative field, with direct insertion of endotracheal tubes into the trachea and bronchi during phases of surgery when the airway is open, developed early and is described with variations in reports of tracheal reconstructions cited earlier. Tubes across the operative field were used in experimental work and Ravitch early mentioned clinical usage.[42,103,104,152] In 1951, Friedmann and Emma described a catheter insufflation technique for carinal resection in one patient.[153] Grigor and Shaw, working with Barclay and his colleagues, used cross-field ventilation in combination with endotracheal intubation, depending on intermittent ventilation during the implantation of the left main bronchus into the bronchus intermedius.[154] They recognized that the preceding development of one-lung anesthesia provided the groundwork for carinal anesthesia. Baumann and Forster, in 1960, described systematic approaches to anesthesia for cervical, intrathoracic, and carinal tracheal surgery, including intubation via distal tracheostomy and also across the operative field.[155] Grillo and colleagues, in 1963, detailed similar technique for carinal resection and reconstruction.[121] Cross-table anesthetic techniques were fully described by Grillo in 1965 and expanded in 1970.[83,129] The potential use of two anesthesia machines for complex carinal reconstruction was also noted. Geffin and colleagues summarized cross-table anesthetic techniques for tracheal and carinal reconstruction in 1969 on the basis of their accumulated experience by then with 31 operations.[156] Theman and colleagues confirmed these techniques.[157]

Lee and English, in 1974, described the use of a Saunders-type bronchoscopic injector through a catheter passed beyond a tracheal stenosis.[158] El Baz and colleagues favored high-frequency positive pressure ventilation for tracheoplasty to permit better visualization and access.[159] McClish and colleagues expressed similar convictions in a series of 18 patients.[151] Wilson thoroughly updated these anesthesiologic approaches.[160] Although his preference was for cross-table ventilation, high-frequency ventilation was valued as a useful adjunctive technique in special circumstances, such as complex carinal reconstruction. These choices remained a matter of preference, with both techniques proving satisfactory in experienced hands. All agree that close communication and cooperation between surgeon and anesthesiologist are uniquely demanded for this type of surgery, preoperatively, intraoperatively, and, optimally, postoperatively.

The use of cardiopulmonary bypass for tracheal and especially carinal resection has been mentioned earlier. In their extensive experiences with tracheal surgery, Eschapasse, Grillo, Pearson, and Perelman found bypass to be unnecessary. Its use in very complicated cases, where it might be theoretically desirable,

but where extensive dissection and manipulation of the lung was required, led to fatal pulmonary parenchymal hemorrhage in 2 patients due to necessary anticoagulation (Grillo, Pearson, unpublished).

Laryngotracheal Resection and Reconstruction

Just as reconstruction of the carina presented unusual difficulties, so did the proximal end of the airway. When tracheal lesions also affect the subglottic larynx, the anatomic and functional characteristics of that structure offer special problems. Many otolaryngologic procedures were developed to manage inflammatory stenosis at this level, when conservative measures failed. The latter measures included dilation, stents, intubation, steroid injection, cryotherapy, and laser surgery. Surgical procedures that were devised included anterior and posterior cricoid splits, placement of stents, mucosal and cutaneous grafts, free grafts of cartilage and hyoid, pedicled hyoid, cutaneous flaps variously supported with cartilage, and multistage "trough" procedures. These many operations will not be reviewed here, but, in general, success was limited.[161]

A one-stage approach to subglottic stenosis characterized by cricoid involvement developed slowly. The initial work was done by otolaryngologists, but full development of the techniques was accomplished by thoracic surgeons who faced the problem of subglottic stenosis as it presented in the spectrum of post intubation tracheal stenosis. Conley removed the entire cricoid in 1953 for a chondroma, preserving the mucoperichondrium, which was held in place by a foam rubber stent.[67] Great care was taken to avoid injury to the recurrent laryngeal nerves. Shaw and colleagues resected damaged or stenotic cricoids in 2 patients with anastomosis to the thyroid cartilage.[27] Existing vocal cord paralysis simplified the problem in these patients. Ogura and Roper apposed the second tracheal ring to thyroid cartilage after subtotal excision of traumatically scarred and stenotic cricoid in 2 patients.[94] The recurrent nerves were paralyzed, arytenoidectomy was done, and a stent was used postoperatively. The distal trachea was mobilized and the thyrohyoid muscles and constrictors, which are attached to the thyroid cartilage, were divided to assist in approximation. Subperichondrial cricoid resection avoided injury to the recurrent nerves.[95] Six of 7 patients with chronic subglottic stenosis were helped by this procedure.

In 1974, Gerwat and Bryce placed the upper line of resection for stenosis at the lower border of the thyroid cartilage anteriorly, and through the posterior cricoid lamina below the cricothyroid joints posteriorly.[162] Thyrohyoid release was added and believed to be important. Four patients were so treated. In 1975, Pearson and colleagues followed the same line of cricoid resection, but rongeured all but a thin shell of posterior lower cricoid plate, sutured the ends of the first intact cartilaginous ring of trachea together, and inset this into the rongeured groove to form the laryngotracheal anastomosis (Figure 5).[163] Recurrent nerves were preserved. Superior laryngeal release was done, and a splinting T tube was added postoperatively. Six patients were successfully treated. Couraud and colleagues, in 1979, added 4 patients, all but one successful (Figure 6).[164] They pointed out that there was no use in disturbing the recurrent nerves, that sometimes the posterior cricoid cartilage did not need to be tailored, and that tracheostomy was not regularly necessary. Grillo, in 1982, described 18 patients with subglottic stenosis treated with a somewhat modified procedure.[161] In patients with anterolateral stenosis, a simple bevelled cricoid resection was sufficient, and the tracheal cartilage to be anastomosed was obliquely tailored to fit easily. For circumferential stenosis, scar over the posterior cricoid plate was excised and the raw area resurfaced with a broad-based flap of posterior membranous tracheal wall shaped for this purpose. Neither laryngeal release nor tracheostomy was routinely needed.

In 1992, Grillo and colleagues reviewed 80 patients who underwent one-stage laryngotracheal resection and reconstruction for subglottic stenosis by these techniques: 50 with postintubation lesions, 7 from trauma, 19 idiopathic, and 4 others.[165] Thirty-one patients required circumferential resection with posterior flap resurfacing. There were 2 failures. If glottic correction was also needed, it was done initially as a separate procedure. Maddaus, with Pearson's group, proposed synchronous glottic reconstruction where that was

FIGURE 5 *F. Griffith Pearson, MD, Chief of Thoracic Surgery and Surgeon-in-Chief Emeritus, Toronto General Hospital, and Professor of Surgery Emeritus, University of Toronto. Dr. Pearson, who founded and led the Thoracic Surgical Division in the Toronto General Hospital, early became interested in tracheal surgery. He contributed richly to the understanding and treatment of postintubation stenosis, to the development of one-stage laryngotracheal reconstruction, and to our knowledge of adenoid cystic carcinoma.*

also required, reporting 15 such cases of 53 subglottic repairs.[166] They also adopted the posterior tracheal membranous wall flap described by Grillo and his colleagues.[161,165] Monnier and colleagues proved this type of repair to be useful in children.[167]

Cervicomediastinal Exenteration and Mediastinal Tracheostomy

Rarely, following extensive resection of the larynx and upper trachea for neoplasms, such as thyroid carcinoma, adenoid cystic carcinoma, or recurrent laryngeal carcinoma after laryngectomy, there is need for mediastinal tracheostomy, well below the sternal notch. Watson, in 1942, devised a procedure to treat a patient with squamous carcinoma 4 cm above the carina.[168] The patient had undergone laryngectomy for cancer 15 years earlier, followed by radium treatment. A "V" of sternum was resected and skin flaps mobi-

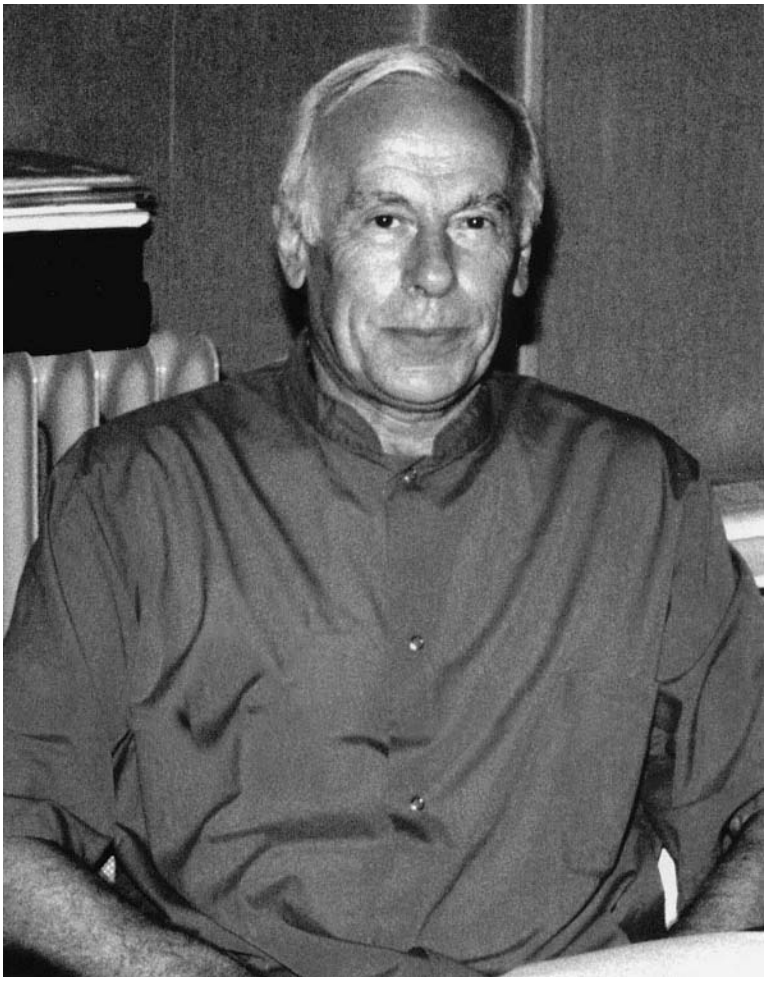

FIGURE 6 *Louis Couraud, MD, Chief of Thoracic Surgery, Emeritus, Xavier Arnozan Hospital, Pessac, and Professor of Surgery Emeritus, University of Bordeaux II. Professor Couraud made Bordeaux renowned for airway surgery, producing excellent surgical results and adding to our knowledge of many aspects of tracheal disease: postintubation stenosis, laryngotracheal stenosis, juvenile tracheal growth, tracheoesophageal fistula, primary tumors, postsurgical complications, and the airway in transplantation.*

lized to allow closure of the margins of the tracheal stoma. In 1951, Sloan and Cowley managed the problem of tracheal compression by an aortic aneurysm by establishing a side tracheostomy, the tube of which emerged from the back medial to the right upper scapula, after removal of proximal rib segments.[169] After wrapping the aneurysm, it was possible to remove the tube. The authors discussed earlier proposals and even attempts to establish transpleural bronchial fistulae for this purpose, and a proposal, not acted upon, by Gluck in 1907, to perform posterior bronchotomy.

In 1952, for mediastinal tracheostomy, Kleitsch removed the upper sternum and inserted a polythene tube.[170] A sequence of irradiation necrosis and recurrent tumor frustrated plans to line the opening with skin grafts. In the same year, Minor, after removal of recurrent carcinoma of the tracheal stoma, brought skin flaps as a tube through a sternal opening to connect with the trachea.[171] Healing failed, and the patient bled to death 4 months later. Waddell and Cannon, in 1959, pulled a short tracheal stump to the right of

the ascending aorta and created a skin tube from crossed anterior chest skin flaps which passed through a hole rongeured in the sternum and was anastomosed to the tracheal end.[172] Two of 4 patients, all with squamous cell carcinoma, died of massive hemorrhage.

In 1962, Sisson and colleagues, operating for recurrent laryngeal carcinoma at the tracheal stoma, excised a large portion of surrounding skin with the specimen and removed the manubrium and the heads of the clavicles.[173] Skin flaps were turned up to effect closure about the stoma, and an inferior defect was skin grafted. After 2 patients died from innominate artery hemorrhage postoperatively, the pectoralis muscles were undermined and rotated between the innominate and left carotid arteries and the trachea. Also in 1962, Ellis and colleagues used a tube of heavy Marlex mesh to reach the surface after low transection of the trachea.[174] Granulation tissue formation and the possibility of infection, erosion, and hemorrhage make tubes of foreign material undesirable in this setting. In an effort to eliminate tension at the tracheal cutaneous anastomosis, which carried the threat of subsequent nonhealing and fatal innominate hemorrhage, Grillo in 1966 proposed fashioning a broad full-thickness bipedicled flap of anterior chest wall skin and subcutaneous tissue formed with two long, horizontal incisions (Figure 7).[175] This flap was depressed to meet the end of the trachea in the mediastinum, made accessible by resection of manubrium, heads of clavicles, and upper two costal cartilages. The stoma emerged in midflap, resulting in a simple suture line more likely to heal well. Two end stomas and one in-continuity stoma were reported. Grillo and Mathisen subsequently offered further guard against vessel erosion in the event of deficits in peristomal healing by 1) advancing omentum routinely to separate trachea and great vessels, and 2) electively dividing the brachiocephalic artery under electroencephalographic monitoring, where the tracheal stump was very short, following preoperative angiography.[176] One operative death occurred in 18 patients. Additional experiences have been recorded in this area by Stell, Krespi, Gomes, and Orringer and their colleagues.[177–180] Withers and colleagues suggested use of a pectoralis musculocutaneous flap, which has particular application to cases where a wide resection is necessary around an existing stoma for reason of peristomal carcinoma or irradiation damage.[181]

Complications of Tracheal Surgery

As tracheal surgery became more common, a pattern of complications inevitably appeared. These were analyzed by Levasseur in 1971, Couraud in 1982, and by Grillo in 1986, along with their colleagues.[92,182,183] In 11 patients, Levasseur and colleagues observed 1 restenosis and 4 lethal erosions of brachiocephalic artery.[92] This led the authors to recommend cervical muscle or thymic interposition for prophylaxis against hemorrhage. Couraud and colleagues, reporting on 122 cases of resection with only 4 deaths and 1 failure, emphasized anti-infectious and anti-inflammatory precautions.[182] Grillo and colleagues reviewed incidence, causes, treatment, and prevention of complications in 416 primary reconstructions, 279 for postintubation lesions.[183] Suture line granulations occurred in 28 of 209 cases, when various nonabsorbable sutures were used for anastomosis, but in none of 113 after the adoption of absorbable Vicryl sutures. Brachiocephalic artery injuries were best avoided by avoiding any dissection of the artery, and where that was not possible, interposing a strap muscle. From the first to the second halves of the series, deaths fell from 4 to 1, failures from 13 to 7, and complications from 42 to 30.

The Unreconstructible Trachea: T Tubes and Stents

T tubes had been devised and variously employed in the past, as by Bond in 1891 and Falbe-Hansen in 1955, citing Laurens' earlier use of a T tube.[184,185] Aboulker and colleagues treated postintubation injuries with a T tube in 1960.[186] The silicone rubber T tube developed by Montgomery in 1965 proved widely useful in tracheal surgery, although it was developed initially in the false hope that prolonged stenting would resolve

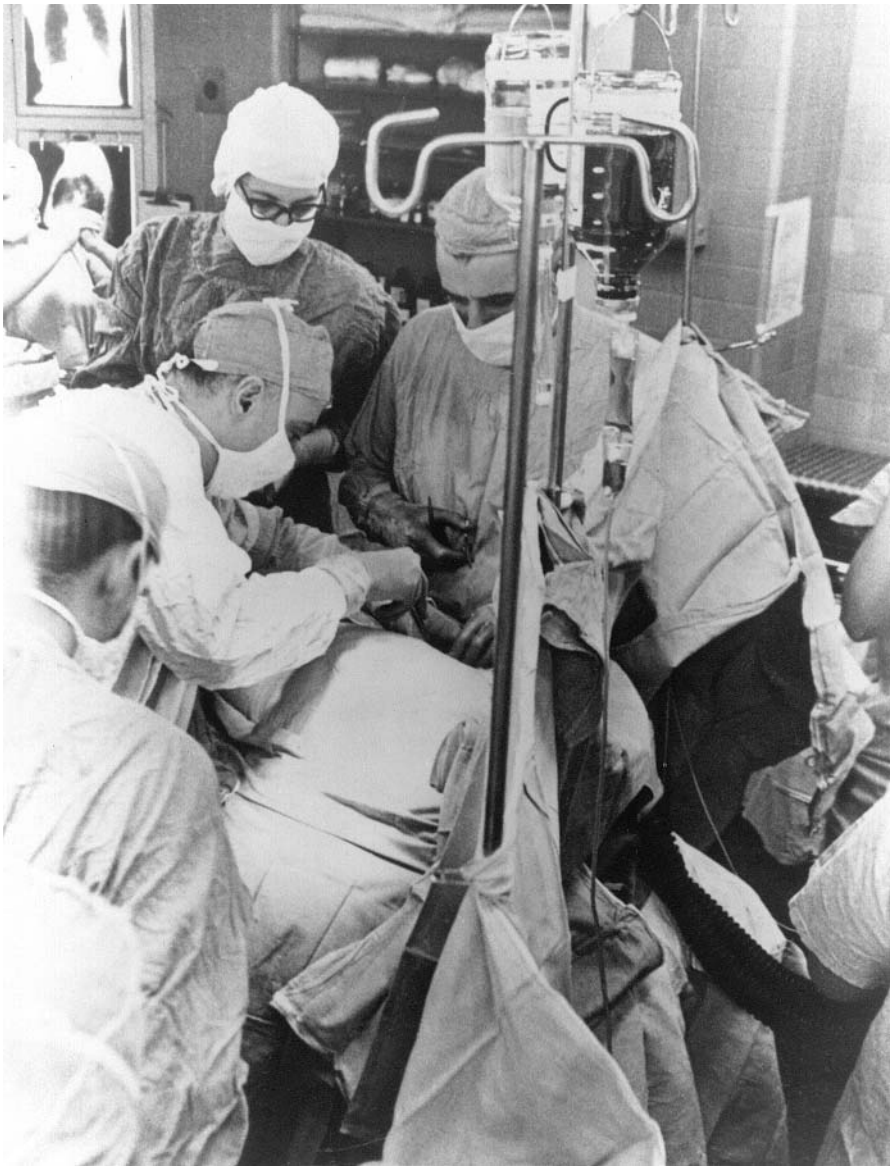

FIGURE 7 *Hermes C. Grillo, MD, Chief of General Thoracic Surgery, Emeritus and Senior Surgeon, Massachusetts General Hospital (MGH), and Professor of Surgery Emeritus, Harvard Medical School. The picture shows Dr. Grillo commencing a cervicomediastinal exenteration in 1966. His first assistant is Mortimer J. Buckley, then Chief Resident in Surgery at MGH, later to become Chief of Cardiac Surgery.*

tracheal stenosis.[187] Cooper and colleagues in 1981 and Gaissert and colleagues in 1994 used it for permanent and temporary restorations of airway continuity when the trachea was not reconstructible, a lesion was not removable, or a temporary airway was needed.[188,189] Westaby and colleagues added a bifurcated T tube for help in carinal problems.[190]

The development and deployment of *stents* will not be reviewed here. However, caution needs to be raised against tendencies to use essentially permanent expandable stents where lesions might otherwise be readily and definitively corrected by surgery. The result too often is doubly negative: correction of the lesion is permanently prevented and severe complications may develop from the stent.[191] Removable silicone

stents also hinder curative treatment and may quite readily cause granulations, especially in the subglottic region. These are sometimes reversible, however, in contrast to problems caused by permanent stents.

Treatment of Tracheal Diseases

Primary Tracheal Tumors

Thus far, this review has focused on the evolution of techniques of tracheal surgery. Application of these and other additionally developed techniques to specific diseases of the airways will now be considered. The challenge of treating the rare tracheal tumors which were seen provided the initial stimulus for tracheal resection.[1,5] The very rarity of primary tracheal neoplasms, on the other hand, provided limited incentive to attack this problem systematically. In 1938, Culp collected 433 reported cases of primary tracheal tumors, beginning with Lieutaud's discovery of fibroma at autopsy in 1767.[63] From prior cumulative series, Culp noted the slow increment from 147 cases in 1898, to 201 in 1908, to 252 in 1914, and 351 in 1929. He provided an exhaustive bibliography, but personally found only 1 carcinoma in 9,000 autopsies at McGill University, and 1 in 12,700 autopsies at Montreal General Hospital. Ellman and Whittaker raised the total to 507 in 1947.[64] "Cylindroma" was often classified as adenocarcinoma, and tracheopathia osteoplastica was included as a tumor. Houston and colleagues collected 53 primary cancers of the trachea in over 30 years at Mayo Clinic, showing a distribution now recognized as expected: 45% squamous, 36% "cylindroma" (adenoid cystic carcinoma), and the balance of other origins, including mesenchymal tumors.[192] Reporting a 30-year experience in 1969, only 2 squamous cancers had been removed, 1 by lateral excision, 6 adenoid cystic (none by circumferential resection and anastomosis), and 1 mucoepidermoid by end-to-end repair. The next year, Hajdu and colleagues described 41 patients with primary tracheal carcinoma from Memorial Hospital over 33 years: 30 squamous and 7 adenoid cystic.[193] Few were treated by resection.

Times were changing, however, as techniques of resection based on anatomic mobilization were increasingly applied to tracheal neoplasms. Forster and colleagues resected a cervical tracheal epithelioma in 1957 with end-to-end suture.[71] Forster and Holderbach published a voluminous report in 1960 on the pathology and clinical presentation of tracheal tumors, and of experimental and a few clinical trials at that early date.[194] Non-neoplastic lesions were also included. Grillo recounted treatment of three primary tumors by circumferential resection in 1965, using cross-table ventilation through the open trachea.[83] Mathey and colleagues reported resecting 5 patients with primary tracheal neoplasms in 1966, with 1 early and 1 late postoperative death.[40] Perelman and Korolyova successfully treated 5 patients with primary tracheal intrathoracic cancer by circular resection and anastomosis in 1968.[87] They introduced an anesthesia tube into the left main bronchus via an incision in the membranous wall of the right main bronchus. Dor and colleagues reported in 1971 on resections of tracheal tumors in 6 patients, with 3 postoperative deaths.[91] By 1973, Grillo had excised 11 primary tumors and 5 secondary tumors in a series of 100 tracheal resections with reconstruction.[195] Nine of the 11 patients were alive without disease; 1 patient died postoperatively.

Experience with surgical management began to grow. In 1974, Eschapasse reported on 152 patients with primary tracheal tumors treated by 12 French and 2 Russian groups, which included 32 circumferential resections and 18 carinal reconstructions.[126] The poorest long-term results were with squamous carcinoma, which also gave the highest postoperative mortality. Adenoid cystic carcinoma showed prolonged survival, but late recurrence. Also in 1974, Pearson and colleagues accomplished 5 resections of adenoid cystic carcinoma with primary anastomosis, without postoperative death.[196] In 6 others, prosthetic replacement was made using Marlex. Grillo reported seeing 63 patients with tumors by 1978.[85] Nineteen patients with primary tumors (and 5 more with secondary) were treated by cylindrical resection and anastomosis and 10 additional patients by carinal resection and reconstruction. Ten others underwent staged reconstruction, and 10 had laryngotracheal resection, or were treated by other means. Two died after cylindrical resection and 3 after carinal resection and reconstruction.

Subsequent major series began to define the long-term oncologic expectation. In 1990, Grillo and Mathisen reported the largest single institutional series of 198 primary tumors treated at Massachusetts General Hospital (MGH) over 26 years.[6] Resection rates were 63% for squamous carcinoma and 75% for adenoid cystic carcinoma. Pearson and colleagues updated their experience with 44 tracheal tumors in 1984 and a subsequent report by Maziak and colleagues did the same for 38 adenoid cystic carcinomas in 1996.[136,197] Regnard and colleagues collected 208 patients in a multicenter series in France in 1996.[198] In that same year, Perelman and colleagues summarized an experience with 144 primary tumors.[199] Squamous carcinoma and adenoid cystic carcinoma together compose about 75% of all primary tracheal tumors, in comparable numbers. The etiology, curability, and associated aerodigestive carcinomas of squamous cancer were much like squamous lung cancer. Surgery for adenoid cystic carcinoma, combined with radiotherapy, produced high 5-year survival rates, but a continued fall in survival at 10 years and thereafter occurred, due to local recurrence and the appearance of metastases. The rarity and idiosyncratic and prolonged course of adenoid cystic carcinoma clearly requires very prolonged observation for complete clinical definition. An enormously wide variety of tumors of other histology, most often benign or of low-grade malignancy, composed the remaining 25% of cases.[6] Operative mortality in all patients ranged from 5.3 to 10.5% in various series. Mortality and morbidity fell with surgical experience, but remained highest in carinal reconstruction.[183]

Secondary Tracheal Tumors

Resection of the carina for bronchogenic carcinoma has been discussed under the section, "Carinal Pneumonectomy," above. The proximity of the thyroid gland makes the trachea and lower larynx targets susceptible to invasion by cancer in this gland.[200] Localized tracheal invasion by thyroid neoplasms was resected episodically as tracheal surgery evolved. Rob and Bateman, in 1949, resected six rings of trachea and a portion of cricoid for recurrence of thyroid cancer "of low malignancy," 7 years after initial excision and radiotherapy.[30] Tantalum gauze-fascia lata reconstruction was done, leaving a strip of posterior mucosa. After a checkered course, the patient survived. Conley did a staged repair with tantalum mesh and fascia plus skin flaps after resection of anterior tracheal wall invaded by "adenocarcinoma of the thyroid."[67] Lazo resected the anterior wall of the cervical trachea for thyroid cancer in 1957, using a prosthesis for speech.[201] In 1965, Grillo resected a six-ring segment of trachea, including a portion of cricoid invaded by papillary carcinoma that had paralyzed the left cord and obstructed the tracheal lumen.[83] Tracheal reconstruction was staged with a cutaneous tube supported by inlying polypropylene rings. The result was satisfactory. In 1966, Mathey and colleagues resected 3.5 cm of trachea for papillary carcinoma and performed an end-to-end anastomosis, but placed a tracheostomy in the suture line postoperatively.[40]

An aggressive approach was accepted early in Japan, but only slowly in the west. Ishihara and colleagues reported operation on 11 patients in 1978, 8 of whom had recurrent papillary adenocarcinoma after prior surgery.[202] Sleeve resections were done with resection of the anterior cricoid in 3 of the patients. Two died from operation and 3 developed laryngeal stenosis. Five were long-term survivors. This same group reported on 60 patients by 1991.[203] In 1985, Tsumori and colleagues reported 18 resections with anastomosis.[204] In 1986, Fujimoto and colleagues performed sleeve resection in 6 patients and window resection in 3.[205] A survey of tracheobronchial surgery in Japan, reported in 1989 by Maeda and colleagues, revealed 151 cases of tracheoplasty for thyroid cancer against 147 tracheobronchial tumors over a period of 30 years.[206]

In the west, Grillo listed 3 patients in 1978, resected for thyroid carcinoma, and recommended that this treatment be applied more widely.[85] In 1986, Grillo and Zannini cited 16 patients treated by resection and reconstruction, and by 1992, Grillo and colleagues reported 27 cases.[207,208] Rationale for this approach is adherence to the oncologic principle of thyroid surgery, that local disease be removed totally. The surgery is not high-risk surgery or radical surgery in competent hands. Given the proclivity of papillary tumors to become more

aggressive in time, plus the observation that many of these patients had undergone "shave" procedures, often years before, anything less than complete removal (including airway, if necessary) seems inappropriate. Nonetheless, "shave" procedures in the case of superficial invasion, and "window" resection in the case of deep invasion, are still being recommended by surgeons without extensive experience in tracheal reconstruction.[209]

Radical extirpation of invasive undifferentiated thyroid carcinoma and also of massive recurrences of papillary carcinoma, to include laryngectomy and extended tracheal resection, was described by Hendrick, in 1963, in 11 patients with 5 long-term survivors and 5 alive without disease from 4 to 16 years.[210] In 1958, Frazell and Foote noted 3 of 4 patients, who had laryngeal and tracheal resection for thyroid cancer, lived 4½ to 5 years.[211] Grillo and colleagues reported in 1992 on radical extirpation of tumor in 7 patients, by cervicomediastinal exenteration including esophagectomy.[208] Palliation is a principal goal of these procedures.

Postintubation Lesions

The poliomyelitis epidemics of the mid-twentieth century introduced and led to an ever-widening use of mechanical ventilators to treat respiratory failure. The iatrogenic lesions that resulted provided a whole new field of endeavor for the tracheal surgeon. Gradually, a spectrum of lesions was recognized, attributable to ventilatory apparatus: endotracheal and tracheostomy tubes and the cuffs necessary to seal the trachea.[84,90,186,212] Principal among these were 1) circumferential stenosis that appeared at the level of the sealing cuff, and 2) anteriorly pointed, arrow-shaped stenosis, which occurred at the stomal level. Additionally, granulomas occurred at the point where a tube tip impinged on the tracheal wall. Areas of malacia were seen less often at the level of the cuff and sometimes in the segment between a tracheal stoma and a cuff stenosis. Tracheoesophageal fistulae occurred principally between the trachea and the esophagus at the cuff level, usually with accompanying circumferential tracheal damage. Rare, but disastrous when they occurred, were tracheal innominate artery fistulae. These lesions proved to be of two types: one where a tracheostomy tube rested immediately on the innominate artery near the stoma, and another, where the cuff, or, even less often, the tube tip, eroded through the trachea anteriorly into the innominate artery.

In the 1960s, numerous papers, often single case reports, appeared in Europe and North America, describing surgical resection of *postintubation strictures*. Included among these together with their colleagues were Forster in 1957, Flavell in 1959, Witz in 1960, Binet and Aboulker in 1961, Van Wien in 1961, Mathey in 1966, Byrn as well as Fraser in 1967, and Jewsbury, Dor, Dolton, Schaudig, Lindholm, and Naef in 1969.[40,71,73,75,88,213–221] Series of cases also were reported by the following authors together with their colleagues: Deverall reported 6 patients in 1967, Pearson reported 15 in 1968, Grillo reported 14 whereas Couraud reported 9 in 1969, and Dor reported 9, Levasseur reported 10, and Harley reported 11 in 1971.[84,86,89,91–93,212] These last authors, especially Pearson, Grillo, and Harley, with somewhat broader experience, defined the anatomic and pathologic differences between stomal and cuff stenoses and other postintubation injuries, and discussed their pathogenesis. Malacia instead of stenosis was also described, although a rare finding, by Grillo.[222] Deverall, Pearson, Grillo, and Couraud and their colleagues stressed the importance of allowing florid inflammation to subside prior to surgical correction.[84,86,89,212] Their generally good results showed the superiority of definitive surgical resection and anastomosis over prior alternative methods of treatment, such as repetitive dilation, steroid injection, or cryotherapy. Unfortunately, the lesson is being relearned today, with uncritical use of laser surgery for these lesions,[223] and, more lately, with much more disastrous results, the attempted use of stents to treat postintubation stenosis.[191]

Postintubation lesions became, and remain, the most common indication for tracheal resection and reconstruction. Generally very good results have been obtained in major cumulative series of patients with iatrogenic tracheal and subglottic laryngotracheal stenosis: Bisson and colleagues achieved an 87.5% "cure" in 200 patients in 1992, Couraud and colleagues reported 96% success in 217 patients in 1994, and Grillo and colleagues cited 94% success in 503 patients in 1995.[98,224,225]

Correction of *postintubation stenosis involving the subglottic larynx* remains more difficult than lesions confined to the trachea. The evolution of procedures for laryngotracheal resection and reconstruction by partial cricoid resection has been outlined and their application to iatrogenic stenosis noted. Monnier and colleagues applied this approach in infants and children, also with encouraging success.[167]

Reoperative tracheal resection and reconstruction for unsuccessful repair of postintubation stenosis proved to be surprisingly manageable. In 1997, Donahue and colleagues tallied 92% good or satisfactory results in 75 patients who had failed prior surgical repairs, 59 of whom were referred.[226]

Tracheoesophageal fistulae (TEF) due to erosion by tracheal cuffs and often of "giant" size were noted early, in 1966 by Le Brigand and Roy and several other French surgeons in the same period, by Flege in 1967, and by Hedden and colleagues in 1969.[227–229] Scattered attempts of repair by sometimes multistaged techniques, including Braithwaite's successful use of a cutaneous flap to seal the tracheal side of a large fistula in 1961, did not often meet with success.[227,230,231] Grillo and colleagues, in 1976, described a definitive one-stage technique for esophageal closure, tracheal resection (where a circumferential cuff lesion was present), and strap muscle interposition, with good results in 7 patients.[232]

Postintubation injury, however infrequent, has become the most frequent cause of acquired TEF. It is now effectively managed by the type of procedure noted, and has been further described by Mathisen, Couraud, and Macchiarini and their colleagues.[233–235] When the fistula is small and the tracheal lesion is not circumferential, tracheal closure is performed. The techniques developed have been applied effectively to closure of TEF from a variety of causes, including trauma and inflammation.

Tracheal innominate artery fistula (TIF), described by Lunding in 1964, Silen and Spieker and Stiles in 1965, Couraud and colleagues in 1966, and Foley and colleagues in 1968, as a consequence of tracheostomy and ventilation, was effectively approached surgically by Grillo in 1976, Cooper in 1977, and Couraud and colleagues in 1984.[236–243] The mechanism of fistulization was either erosion of the tracheal wall by a high-pressure cuff, angulation of a tracheostomy tube tip, or most commonly, erosion by the tube in a low-lying tracheostomy where the elbow of the tube essentially rests on the artery. Jones and colleagues reviewed the topic extensively in 1976, including delineation of types of erosion, emergency management, safety and desirability of arterial resection, and success rates.[244]

The *etiology of postintubation stenosis* and other injuries was initially unclear. Among the factors thought to be implicated were irritation from materials of which tube and cuff were made, elution of chemicals by gas sterilization, age, debility, steroids, bacterial infection, and direct irritation by the tube's presence. Although some of these likely contributed to the injuries seen, pressure and necrosis from tubes and cuffs, whether endotracheal or by tracheostomy, with subsequent efforts at tissue repair, and, finally, cicatrization, proved to be the fundamental explanation.

Post-tracheostomy stenosis had been pointed out as early as 1886, when Colles found four strictures in 57 patients treated for diphtheria.[245] However, only with the growing use of ventilation, during and after the 1952 poliomyelitis epidemic, did postintubation injuries become more frequent.

In 1960, Aboulker and colleagues identified inflammation as a major factor in the spectrum of post-tracheostomy stenosis.[186] On the basis of 12 autopsy studies in patients who were ventilated via tracheostomy for differing time periods, Bignon and Chrétien in 1962 described inflammation, metaplasia, and stenosis at the tracheostomy site; pseudopolyps, ulceration, and stenosis in the trachea at cuff level; and, sometimes, softening of the tracheal wall.[246] They attributed these changes principally to trauma from the cannula above and to ischemic compression by the cuff or erosion by the tip of the tube below. The severity of lesions did not correlate with the length of ventilation.

Yanagisawa and Kirchner as well as Atkins, in 1964, described severe damage to the trachea and stenosis from use of cuffed tracheostomy tubes.[247,248] In 1965, after careful autopsy studies of tracheostomized and ventilated subjects, Florange and colleagues reconstructed the evolution of tracheal necrosis from

mucosal inflammation to erosion of the mucosa, loss of cartilage, and localized mediastinitis.[249] They concluded that this damage could result in stenosis. In 1965, Stiles described severe changes at the stomal, cuff, and tube tip levels in 23 patients in 37 consecutive tracheostomies, all of whom died after ventilation.[238] He was inclined to relate the damage to the materials from which the tubes were manufactured. Gibson concluded in 1967 that the "main factors" in producing stenosis were cuff trauma plus infection at the stoma.[250] Most tracheae of patients who died while being ventilated via tracheostomy showed necrosis. Murphy and colleagues, in 1966, could only produce stenosis in dogs with cuff tracheostomy when infection was also present.[251] In 1968, Foley and colleagues described the tracheal changes due to abutment of tubes and cuffs in patients with fatal burns.[240]

In 1969, Grillo showed similar changes as a result of ventilation.[84] Cooper and Grillo presented a detailed pathologic study of autopsy specimens from patients dying on respirators.[252] A spectrum of changes was described similar to that noted by Florange. Lesions appeared within 48 hours and progressed from tracheitis to ulceration of the mucosa, to fragmentation of cartilage, to replacement of the tracheal wall with scar tissue. The location and nature of the lesions also correlated with surgically removed stenotic lesions. Lindholm presented a detailed study in 1969 of lesions developed in the larynx and also in the trachea from ventilation.[221] The severity of histologic changes was vastly greater than those described after tracheostomy alone.[253] Andrews and Pearson prospectively examined the trachea of 103 patients receiving ventilator support in 1971.[254] Twelve stomal and 6 cuff stenoses developed. Bronchoscopic examination was of little value in predicting which patients would go on to stomal stenosis, but circumferential mucosal ulceration at the cuff level dependably predicted stenosis at that level. Additional statistically significant factors observed in this study were large tracheostomy tubes and high-dose steroids. The same erosive processes were observed to cause tracheoesophageal fistulae and tracheoinnominate artery fistulae.

Prevention of postintubation injury quickly became a priority once the origin of these lesions was evident. In 1957, Adriani and Phillips found that most of the intracuff pressure necessary to inflate the then conventional cuffs (90 to 220 mm Hg) was expended on distending the cuff, and the pressure on the tracheal wall was low (10 to 15 mm Hg) in order to develop ventilatory pressures of 10 to 20 mm Hg.[255] Cooper and Grillo later pointed out that excessive pressures were necessary to seal the irregularly-shaped trachea by distending the relatively rigid small volume cuffs that were then in use.[256] Knowlson and Bassett also noted that small increments over the minimal occlusive volume necessary for the seal of conventional cuffs at 20 cm H_2O caused a rapid rise in the pressure exerted on the tracheal mucosa.[257] In 1943, Grimm and Knight had proposed that the ideal cuff "should have sufficient volume when inflated, without stretching, to fill the diameter of the trachea."[258] Lomholt offered a cuff of thin and elastic Teflon in 1967, lying in folds so that intracuff pressure would be identical with pressure on the mucosa.[259] Carroll and colleagues, in 1969, recommended a cuff with large residual volume, a large sealing area, a centered tube, and the development of only small increases in tracheal wall pressure with overinflation.[260]

Cooper and Grillo reproduced severe stenosing cuff lesions in dogs in 1969, which were entirely parallel with lesions seen in man (Figure 8).[256] They used standard balloon cuffs and inflation necessary for ventilation at 20 to 25 cm H_2O. Intraluminal pressures were 180 to 250 mm Hg. Experimental large-volume, thin-walled latex cuffs produced seals at 20 to 40 mm Hg intraluminal pressure, and no mucosal damage followed. Since this conclusively proved that tracheal lesions were due to cuff pressure, a large-volume, compliant cuff was designed for clinical use by Grillo and colleagues.[261] Forty-five patients were randomly selected for ventilation with a then standard Rusch cuff or the experimental large-volume, compliant latex cuff, and the resulting tracheal injuries were evaluated and compared. Any degree of injury severe enough to evolve into stenosis was produced by the standard (high pressure) cuff. The average intracuff pressure in the new cuff was 33 mm Hg compared with 270 mm Hg in the standard cuff. In extensive clinical use, no tracheal lesions resulted from use of this large-volume, compliant cuff.

FIGURE **8** *Joel D. Cooper, MD, in about 1970, when, as surgical resident at Massachusetts General Hospital, he worked with Dr. Grillo on the etiology and prevention of postintubation cuff tracheal stenosis. Dr. Cooper went on to perform successful lung transplantation at Toronto General Hospital and developed lung volume reduction surgery for emphysema when Chief of Thoracic Surgery at Barnes-Jewish Hospital and Professor of Surgery at Washington University in St. Louis.*

For economic reasons, manufacturers later abandoned latex in favor of plastic cuffs, which lack extensibility. When overinflated just a bit, the present day large-volume cuffs invariably produce steep rises in intracuff pressure, with the potential for tracheal injury severe enough to result in stenosis.[257] Careful attention to cuff inflation and pressures, however, have avoided any incidents of cuff stenosis since 1970 at MGH. A variety of other seals, including prestretched cuffs, flanges, and alternating cuffs, were also proposed as solutions, but they lacked the simplicity and effectiveness of properly used large-volume cuffs.

After adopting the lightweight swivel trachea connectors used at MGH, Andrews and Pearson observed a drop in the incidence of stomal stenosis from 17.5% to 6.9%.[254] The addition, since then, of a suspension of connecting tubing to avoid leverage of the tube against the tracheal stoma, has essentially eliminated stomal stenosis at the MGH.

Elimination of TEF has followed proper use of large-volume cuffs for ventilation, along with avoidance of inlying rigid nasogastric tubes. TIF has all but disappeared with attention to accurate placement of tracheostomy tubes at the level of the second and third tracheal rings and not below, and also, by appropriate use of large-volume, low-pressure cuffs.

Management of Tracheal Trauma

Early experiences with tracheal and bronchial laceration and rupture have been described. In 1959, Hood and Sloan listed their 18 experiences with tracheal injuries in a series of 91 tracheobronchial cases from the literature, and these were more commonly of linear lacerations.[26] Shaw and colleagues, in 1961, added 9 cervical and 4 intrathoracic tracheal ruptures, recommending primary repair of acute injuries and resection of scar with accurate anastomosis for post-traumatic stenosis.[27] Baumann reviewed the limited knowledge about tracheal trauma in 1960, recommending tracheal bronchoscopy in all serious thoracic trauma.[262] Ogura and Powers approached traumatic stenosis of the subglottic larynx aggressively in 1964.[95] Chodosh as well as Ashbaugh and Gordon and others described laryngotracheal avulsion injuries.[263,264]

Beall and colleagues presented 23 tracheal injuries in 1967 and favored immediate treatment, advising airway maintenance and reanastomosis where possible.[265] Ecker and colleagues described 21 tracheal injuries in 1971, with 18 successfully treated.[266] Bertelson and Howitz reported cervical tracheal rupture and perforating wounds in 1972, recommending tracheostomy alone for small wounds.[267] Symbas and colleagues by 1976 progressed from tracheostomy alone to repairs of penetrating wounds in a series of 20 patients.[268] Grover and colleagues reported experience with a variety of tracheobronchial injuries in 1979.[269] Angood and colleagues added to experience in extrinsic trauma to larynx and cervical trachea in 1986.[270] In 1987, Mathisen and Grillo reported good results with immediate repair of acute tracheal injuries and also of concurrent esophageal transection in 1 patient, and good results in 16 of 17 chronic patients, 14 with vocal cord paralysis and 4 with esophageal injury.[271] They emphasized the importance of airway control acutely, assessment of glottic competence where recurrent nerves may be defunctioned, subsidence of inflammation before repair of old injuries, conservation of tracheal tissue, separation of tracheal and esophageal suture lines, and also that a paralyzed larynx can be made functional by adjustment of the glottic aperture. Couraud and colleagues addressed the especially difficult problem of traumatic disruption of the laryngotracheal junction in 1989, describing 19 patients, with restoration of airway and voice in all.[272]

In general, results of treatment of both acute and late tracheal injuries are very satisfactory, in accord with these established principles. Many additional studies have expanded since then on blunt and penetrating injuries and on iatrogenic lacerations or ruptures due to intubation.

Gaissert and colleagues described principles of management of *inhalation burns* of the trachea in 1993, recommending a conservative approach and very patient use of T tubes.[273] Any surgery required is performed very late, after subsidence of the cicatricial response.

The *irradiated trachea* heals poorly when transected and reanastomosed, particularly with rising doses of irradiation and increased intervals between radiation and surgery. Muehrcke and colleagues showed that improved results may be obtained in these difficult problems by wrapping an anastomosis with pedicled omentum.[274] Such anastomoses, however, remain at greater risk for serious complications.

Congenital and Pediatric Lesions

Concern as to whether *growth* would occur following resection and anastomosis of the trachea in infants and small children was early allayed by experiment, although occasional success had also been noted clinically. Kiriluk and Merendino had observed growth of main bronchi after anastomosis experimentally.[45] Borrie had found stenosis to occur after excision of more than three tracheal segments in lambs.[275] Sorensen and colleagues, in 1971, noted somewhat limited growth in anastomotic sites in puppies, after resection of zero to five

rings.[276] Maeda and Grillo, in 1972, noted only mild narrowing of the anastomotic site in puppies without resection, at full growth (Figure 9).[277] They found that, after resection, growth also occurred, but the safe anastomotic tension permitting predictable healing was 58% of that acceptable in adult dogs (1,000 g versus 1,750 g).[278] Kotake and Grillo observed in puppies that tracheal "stay sutures" reduced anastomotic tension.[279] In 1973, Murphy and colleagues noted unpredictably variable growth at anastomosis in piglets, after resection of only two rings.[280] Mendez-Picon and colleagues confirmed anastomotic growth in puppies, in 1974.[281] In 1978, Burrington found that cartilage grew continuously by proliferation on the convex surface without specific growth centers.[282] Vertical incisions, hence, do not interrupt growth.

FIGURE 9 *Mazazumi Maeda, MD, pictured in his research laboratory. In 1970 and 1971, he worked as a Research Fellow in Surgery at Massachusetts General Hospital with Dr. Grillo, precisely describing healing of the juvenile trachea after resection. From the University of Osaka, he went to Shikoku, as Professor of Surgery and Chief of Surgery at Kagawa University. He was a leader in introducing tracheal and bronchial surgery in Japan.*

Cantrell and Guild classified *congenital tracheal stenosis* in 1964 and reported a case of resection of what later was termed a "bridge bronchus," with side-to-side anastomosis.[283] Tracheal resection and primary anastomosis in children were explored by Carcassonne and colleagues in 1973, Mansfield in 1980, Nakayama and colleagues in 1982, and Grillo and Zannini, and Alstrup and Sorensen, in 1984.[284–288] Couraud and colleagues demonstrated long-term growth of anastomotic scars in 1990, particularly after resection of stenosis and anastomosis.[289] Monnier and colleagues showed that single-stage laryngotracheal resection and anastomosis was also applicable in small children.[167] This procedure appeared likely to largely replace cartilage graft procedures developed earlier.[290] However, the length of many congenital tracheal stenoses prohibited resectional treatment.

Kimura and colleagues provided a solution in 1982, by inserting a cartilage patch longitudinally the length of the stenosis.[291] In 1984, Idriss and colleagues used pericardium for the same purpose.[292] Heimansohn and Jaquiss and their colleagues confirmed the use of pericardium and cartilage insets, respectively.[293,294] Although successful in most cases, a considerable incidence of repetitive granulations formed on the mesenchymal patch until epithelization eventually occurred, and in some patients, necrosis of the patch required reoperation or tracheostomy.[295,296]

Tsang and colleagues, working with Goldstraw, solved the problem with slide tracheoplasty, described in 1989.[297] Grillo's report in 1994, describing 4 successful cases so treated, established the procedure.[298] A subsequent publication by Grillo and colleagues, reporting a total of 8 successful patients, 1 of whom was 10 days old, confirmed that satisfactory long-term growth occurred after slide tracheoplasty.[299] The procedure corrected a long stenosis by providing a firm reconstruction with tracheal tissue, lined with ciliated epithelium and hence with little tendency to form granulomas, which did not require postoperative intubation for support and (absent left pulmonary artery sling or other cardiac anomaly) did not require cardiopulmonary bypass for surgery.

Complete laryngotracheoesophageal cleft was successfully repaired in 1984 by Donahoe and Gee.[300]

Infectious and Inflammatory Lesions

The techniques of tracheal and bronchial reconstruction have been applied successfully to infections such as tuberculosis, histoplasmosis, and mucormycosis, and also to a miscellaneous group of lesions including sarcoid and Wegener's granulomatosis. These are not individually referenced since no new principles were necessary for their treatment. The "new" techniques replaced the wire-supported dermal grafts pioneered by Gebauer for tuberculous airway strictures.[52]

Idiopathic laryngotracheal stenosis had been identified in scattered case reports in the 1970s.[301–303] Grillo and colleagues presented a series of 49 such patients in 1993, 39 of whom were treated by one-stage tracheal or laryngotracheal resection, with 32 good or excellent results.[304] Twenty-six patients had been followed from 1 to 15 years, with extension of fibrosis occurring in only 1 patient. The pathology showed dense collagenous fibrosis with little inflammation. No new surgical principles were involved, but definitive delineation of the condition, its pathology, and surgical treatment were provided for the first time. It is, therefore, discouraging to see a recent report of repetitive laser treatment used in 30 patients, who suffered recurrent progressive stenosis, and of failure in 7 patients who did undergo open operation.[305] By 2002, 75 patients have been treated surgically at MGH and all but one successfully decannulated.[306]

Tracheopathia osteoplastica, a very rare condition characterized by submucosal cartilaginous nodules with calcification, most often involving the entire trachea, but also the main bronchi, is sometimes severely obstructive. It was treated by Mark and Grillo and their colleagues, by tracheoplasty over a T tube, which was later removed.[307,308] The operation is based on the fact that all pathologic changes are in relation to the cartilaginous wall, allowing outward hinging of the walls to enlarge the lumen.

Tracheomalacia appears in many forms. Short segments related to postintubation lesions have been resected, whereas longer segments have sometimes been splinted with external polypropylene rings, or internally with stents or T tubes.[222] Expiratory collapse associated with chronic obstructive pulmonary disease was treated by Herzog and by Nissen with posterior membranous wall splinting and quilting, pulling the ends of the splayed, softened cartilaginous rings into a more normal C shape.[309–311] Thin bone slabs, fascia lata, and later Goretex were used as splint materials. Rainer and colleagues reported results with perforated plastic splints of several designs in 1968.[312] Wright and colleagues found posterior splinting of intrathoracic trachea and both main bronchi with strips of Marlex to be effective.[313] Importantly, Marlex becomes permanently incorporated by scar tissue, preventing recurrence. The principal clinical benefit is the improved ease in raising secretions.

Postpneumonectomy Syndrome

Severely symptomatic airway compression caused by extreme mediastinal shift and rotation after right pneumonectomy was especially noted in children, but has since been identified quite commonly in adults. Its occurrence remains unpredictable. The same effect was observed by Maier and Gould in 1953, in patients with agenesis of the lung.[314] The phenomenon was also recognized to occur following left pneumonectomy with a right aortic arch, and, rarely, even after left pneumonectomy with a normal aortic arch.[315–318] In 1949, Johnson and colleagues suggested filling a hemithorax with lucite balls to prevent "overdistension" of a remaining lung.[319] Adams and colleagues used this technique in a symptomatic child.[320] In 1966, Kaunitz and Fisher proposed continued refills of air to maintain normal mediastinal position following pneumonectomy.[321] Powell and colleagues used a prosthesis preventively in 1979.[322]

In 1978, Szarnicki and colleagues divided the aortic arch after placing a graft between the ascending and descending aorta in order to relieve compression.[323] Wasserman and colleagues successfully used Silastic breast implants to correct the problem in 1979.[324]

Rasch and colleagues placed an expandable prosthesis in an infant therapeutically in 1990.[325] Grillo and colleagues produced the first report of a series of any size, involving 11 adult patients, in 1992.[316] Ten underwent repositioning with implants. Good results were obtained generally, but not in 4 who showed severe residual tracheobronchial malacia after mediastinal repositioning. Malacia was more likely to be present in patients with a very long interval between pneumonectomy and operation. Interestingly and encouragingly, since that report, further severe malacia has not been encountered. Saline-filled breast prostheses are presently successfully employed.

Conclusion

Tracheal surgery has largely developed and matured in the last 40 years. In summary, using mobilization procedures, present surgical techniques permit resection of approximately half of the adult trachea with reconstruction by primary anastomosis. Proven methods are also available for laryngotracheal as well as carinal resection and reconstruction. The daunting problem of long congenital tracheal stenosis seems largely solved. Much has also been learned in these decades about the etiology, natural history, pathology, and, in some cases, prevention of various tracheal diseases.

The principles of tracheal repair differ little from those of all surgery: accurate diagnosis, thoughtfully designed procedures, refined anesthesia, meticulous and gentle dissection, preservation of blood supply, precise reconstructive technique, scrupulous avoidance of excessive anastomotic tension, protection of suture lines and major vessels by tissue interposition, and avoidance of trauma to fresh anastomoses. In 1960, Baumann and Forster wisely counselled that the simplest solution was likely to be the best, and that the unnecessary sacrifice of even half a centimeter of trachea might force a change in surgical plan.[77]

References

1. Belsey R. Resection and reconstruction of the intrathoracic trachea. Br J Surg 1950;38:200–5.
2. Dobell ARC. The origins of endotracheal ventilation. Ann Thorac Surg 1994;58:578–84.
3. Mushin WW, Rendell-Baker L. The origins of thoracic anaesthesia. Park Ridge (IL): Wood Library-Museum of Anesthesiology; 1991.
4. Meade RH. A history of thoracic surgery. Springfield (IL): CC Thomas; 1961.
5. Grillo HC. Notes on the windpipe [Presidential address]. Ann Thorac Surg 1989;47:9–26.
6. Grillo HC, Mathisen DJ. Primary tracheal tumors: treatment and results. Ann Thorac Surg 1990;49:69–77.
7. Goodall EW. The story of tracheostomy. Br J Child Dis 1934;31:167–76;253–72.
8. Nelson TG. Tracheotomy: a clinical and experimental study. Am Surg 1957;23:660–94;750–84;841–81.
9. Frost EAM. Tracing the tracheostomy. Ann Otol 1976;85:618–24.
10. Eavey RD. The evolution of tracheostomy. In: Myers EN, Stool SE, Johnson JT, editors. Tracheostomy. New York (NY): Churchill Livingstone; 1985. p. 1–11.
11. Castiglione A. A history of medicine. New York (NY): AA Knopf; 1947.
12. Jackson C. High tracheotomy and other errors the chief causes of chronic laryngeal stenosis. Surg Gynecol Obstet 1921;32:392–8.
13. Jackson C. Tracheotomy. Laryngoscope 1909;19:285–90.
14. Macewen W. Clinical observations on the introduction of tracheal tubes by the mouth instead of performing tracheotomy or laryngotomy. BMJ 1880;2:122–4;163–5.
15. Head JM. Tracheostomy in the management of respiratory problems. N Engl J Med 1961;264:587–91.
16. Watts JM. Tracheostomy in modern practice. Br J Surg 1963;50:954–75.
17. Lawson DW, Grillo HC. Closure of a persistent tracheal stoma. Surg Gynecol Obstet 1970;130:995–6.
18. Chadwick J, Mann WN. The medical works of Hippocrates [translation]. Oxford: Blackwell; 1950. p. 264.
19. Naef AP. The story of thoracic surgery. Milestones and pioneers. Toronto: Hogrefe and Huber; 1990.
20. Paré A. The case reports and autopsy records of Ambrose Paré. Hamby WB, editor. Springfield (IL): CC Thomas; 1960. p. 47–9.
21. Scott GD. Extensive cut throat with complete laceration of trachea and esophagus. JAMA 1928;90:689–90.
22. Nach RL, Rotyhman M. Injuries to the larynx and trachea. Surg Gynec Obstet 1943;76:614–22.
23. Thompson JV, Eaton ER. Intrathoracic rupture of the trachea and major bronchi due to crushing injury. Thorac Surg 1955;29:260–70.
24. Baumann J, Verdoux P, Millner T. Rupture subtotale de la trachée cervicale avec pneumothorax bilatéral. Mém Acad Chir 1957;83:180–6.
25. Dubost C, Dubois F, Duranteau A, Rollin G. Rupture complète de la trachée cervicale. Mém Acad Chir 1957;83:180–6.
26. Hood RM, Sloan HE. Injuries of the trachea and major bronchi. J Thorac Cardiovasc Surg 1959;38:458–80.
27. Shaw RR, Paulson DL, Kee JL Jr. Traumatic tracheal rupture. J Thorac Cardiovasc Surg 1961;42:281–97.
28. Jackson TL, Lefkin P, Tuttle W, Hampton F. An experimental study in bronchial anastomosis. J Thorac Surg 1949;18:630–42.
29. Daniel RA Jr, Taliaferro RM, Schaffarzick WR. Experimental studies on the repair of wounds and defects of the trachea and bronchi. Dis Chest 1950;17: 426–41.
30. Rob CG, Bateman GH. Reconstruction of the trachea and cervical esophagus. Br J Surg 1949;37:202–5.
31. Quinby WC, Morse GW. Experimental pneumonectomy; the application of data so obtained to the surgery of the human thorax. Boston Med Surg J 1911;165:121–4.
32. Rienhoff WF Jr, Gannon J Jr, Sherman I. Closure of the bronchus following total pneumonectomy. Ann Surg 1942;116:481–531
33. Glück T, Zeller A. Die prophylactische Resektion der trachea. Arch Klin Chir 1881;26:427–36.
34. Colley F. Die Resection der Trachea. Eine experimentelle Studie. Deutsche Ztschr Chir 1895;40:150–62.
35. Küster E. Über narbige Stenosen der Trachea. Zentralbl Chir 1886;13:759–60.
36. Bruns P. Resektion der Trachea bei primärem Tracheal krebs. Beitr Klin Chir 1898;21:284–8.
37. Nowakowski K. Beitrag zur tracheoplastik. Arch Klin Chir 1909;90:847–61.
38. Levit H. Deckung von Trachealdefecten durch eine freie Plastik aus der Fascia lata femoris. Arch Klin Chir 1912;97:686–99.
39. Eiselsberg A. Zur Resektion und Naht der Trachea. Dtsch Med Wochenschr 1896;22:343–4.
40. Mathey J, Binet JP, Galey JJ, et al. Tracheal and tracheobronchial resections: technique and results in 20 cases. J Thorac Cardiovasc Surg 1966;51:1–13.
41. Churchill ED. Lobectomy and pneumonectomy in bronchiectasis and cystic disease. J Thorac Surg 1936;6:286–311.
42. Carter MG, Strieder JW. Resection of the trachea and bronchi: an experimental study. J Thorac Surg 1950;20:613–27.
43. Ferguson DJ, Wild JJ, Wangensteen OH. Experimental resection of the trachea. Surgery 1950;28:597–619.
44. Maisel B, Dingwall JA. Primary suture of the divided cervical trachea. Surgery 1950;27:726–9.
45. Kiriluk LB, Merendino KA. An experimental evaluation in the dog of bronchial transplantation, bronchial, tracheal and tracheobronchial resection with reconstruction. Ann Surg 1953;137:490–503.
46. Sanger PW. Evacuation hospital experience with war wounds and injuries of the chest. Ann Surg 1945; 122:147–62.
47. Griffith JL. Fracture of the bronchus. Thorax 1949; 4:105–9.
48. Weisel W, Jake RJ. Anastomosis of right bronchus to trachea forty-six days following complete bronchial rupture from external injury. Ann Surg 1953;137:220–7.
49. Scannell JG. Rupture of the bronchus following closed injury to the chest. Report of a case treated by immediate thoracotomy and repair. Ann Surg 1951; 133:127–30.
50. Belcher JR. Accidental section and successful resuture of a bronchus. Br J Surg 1950;38:121–2.
51. Mathey J, Oustrieres G. End to end bronchial anastomosis after an accidental division of the main stem bronchus. Thorax 1951;6:71.
52. Gebauer PW. Plastic reconstruction of tuberculous bronchostenosis with dermal grafts. J Thorac Surg 1950; 19:604–28.
53. Métras H, Grégorie M, Longepait H. Reconstruction plastique de deux cas de sténose bronchiques. Rev Tuberc Paris 1952;16:54–9.
54. D'Abreu AL. Bronchial "adenoma" treated by local resection and reconstruction of the left main bronchus. Br J Surg 1952;39:355–7.
55. Price-Thomas C. Conservative resection of the bronchial tree. J R Coll Surg Edin 1955;1:169–86.

56. Bikfalvi A, Dubecz S. Resection and anastomosis of the bronchus carried out in a case of benign tumor. J Thorac Surg 1955;29:489–96.
57. Tuttle W. Discussion of Jackson TL, Lefkin P, Tuttle W, Hampton F. J Thorac Surg 1949;18:641.
58. Paulson DL, Shaw RR. Bronchial anastomosis and bronchoplastic procedures in the interest of preservation of lung tissue. J Thorac Surg 1955;29:238–59.
59. Johnston JB, Jones PH. The treatment of bronchial carcinoma by lobectomy and sleeve resection of the main bronchus. Thorax 1959;14:48–54.
60. Newton JR, Grillo HC, Mathisen DJ. Main bronchial sleeve resection with pulmonary conservation. Ann Thorac Surg 1991;52:1272–80.
61. Cantrell JR, Folse JR. The repair of circumferential defects of the trachea by direct anastomosis; experimental evaluation. J Thorac Cardiovasc Surg 1961;42:589–98.
62. Nicks R. Restoration and reconstruction of the trachea and main bronchi. J Thorac Surg 1956;32:226–45.
63. Culp OS. Primary carcinoma of trachea. J Thorac Surg 1938;7:471–87.
64. Ellman P, Whittaker H. Primary carcinoma of the trachea. Thorax 1947;2:151–61.
65. Crafoord C, Eindgren AGG. Mucous and salivary gland tumors in bronchi and trachea formerly generally called bronchial adenoma. Acta Chir Scand 1945;92:481–506.
66. Clagett OT, Moersch HJ, Grindlay JH. Intrathoracic tracheal tumors: development of surgical technics for their removal. Ann Surg 1952;136:520–32.
67. Conley JJ. Reconstruction of the subglottic air passage. Ann Otol Rhinol Laryngol 1953;62:477–95.
68. Kay EB. Tracheal resection and primary anastomosis. Ann Otol Rhinol Laryngol 1951;60:864–70.
69. Sweet RH. [Discussion]. Ann Surg 1952;136:530.
70. Macmanus JE, McCormick R. Resection and anastomosis of the intrathoracic trachea for primary neoplasm. Ann Surg 1954;139:350–4.
71. Forster E, Viville C, Moeller R, et al. Résection de la trachée. Suture bout a bout. Apropos de deux observations. Poumon Coeur 1957;13:367–7.
72. Forster E, Molé L, Fromes R. Sténose trachéale annulaire serrée après trachéotomie. Résection du segment sténosé. Anastomose bout a bout. Guérison. Mém Acad Chir 1958;84:188–93.
73. Binet J, Aboulker P. Un cas de sténose trachéal apres trachéotomie. Résection-suture de la trachée. Guérison. Mém Acad Chir 1961;87:39–42.
74. Miscall L, McKittrick JB, Giordano RP, Nolan RB. Stenosis of trachea. Resection and end-to-end anastomosis. Arch Surg 1963;87:726–31.
75. Flavell G. Resection of tracheal stricture following tracheostomy with primary anastomosis. Proc R Soc Med 1959;52:143–5.
76. Mattes TH. Resektionsmöglichkeiten bei Geschwûlsten des intrathorakalen Abschnittes der Trachea und ihre Kontinuitätswiederberstellung. Der Chirurg 1958;20:32–6.
77. Baumann J, Forster E. Chirurgie de la trachee. Poumon Coeur 1960;16-5:5–119.
78. Michelson E, Solomon R, Maun L, Ramirez J. Experiments in tracheal reconstruction. J Thorac Cardiovasc Surg 1961;41:748–59.
79. Harris RS. The effect of extension of the head and neck upon the infrahyoid respiratory passage and the supraclavicular portion of the human trachea. Thorax 1959;14:176–80.
80. Som ML, Klein SH. Primary anastomosis of the trachea after resection of a wide segment. J Mt Sinai Hosp 1958;25:211–20.
81. Grillo HC, Dignan EF, Miura T. Extensive resection and reconstruction of mediastinal trachea without prosthesis or graft: an anatomical study in man. J Thorac Cardiovasc Surg 1964;48:741–9.
82. Mulliken JB, Grillo HC. The limits of tracheal resection with primary anastomosis: further anatomical studies in man. J Thorac Cardiovasc Surg 1968;55:418–21.
83. Grillo HC. Circumferential resection and reconstruction of mediastinal and cervical trachea. Ann Surg 1965;162:374–88.
84. Grillo HC. The management of tracheal stenosis following assisted respiration. J Thorac Cardiovasc Surg 1969;57:52–71.
85. Grillo HC. Tracheal tumors: surgical management. Ann Thorac Surg 1978;26:112–25.
86. Deverall PB. Tracheal stricture following tracheostomy. Thorax 1967;22:572–6.
87. Perelman M, Korolyova M. Surgery of tumors in the thoracic portion of the trachea. Thorax 1968;23:307–10.
88. Naef AP. Extensive tracheal resection and tracheobronchial reconstruction. Ann Thorac Surg 1969;8:391–401.
89. Couraud L, Chevalier P, Bruneteau A, DuPont P. Le traitement des sténoses trachéales après trachéotomie. Ann Chir Thorac Cardiovasc 1969;8:351–7.
90. Pearson FG, Andrews MJ. Detection and management of tracheal stenosis following cuffed tube tracheostomy. Ann Thorac Surg 1971;12:359–74.
91. Dor V, Kreitmann P, Arnaud A, et al. Résection étendue de la trachée pour tumeur et sténose. Presse Med 1971;79:1843–6.
92. Levasseur P, Rojas-Miranda A, Kulski M, et al. Les complications de la chirurgie des sténoses trachéales non tumorales. Ann Chir Thorac Cardiovasc 1971; 10:393–8.
93. Harley HRS. Laryngotracheal obstruction complicating tracheostomy or endotracheal intubation with assisted respiration. A critical review. Thorax 1971;26:493–533.
94. Ogura JH, Roper CL. Surgical correction of traumatic stenosis of larynx and pharynx. Laryngoscope 1962;72:468–80.
95. Ogura JH, Powers WE. Functional restitution of traumatic stenosis of the larynx and pharynx. Laryngoscope 1964;74:1081–110.
96. Dedo HH, Fishman NH. Laryngeal release and sleeve resection for tracheal stenosis. Ann Otol Rhinol Laryngol 1969;78:285–96.
97. Montgomery WW. Suprahyoid release for tracheal anastomosis. Arch Otolaryngol 1974;99:255–60.
98. Grillo HC, Donahue DM, Mathisen DJ, et al. Postintubation tracheal stenosis: treatment and results. J Thorac Cardiovasc Surg 1995;109:486–93.
99. Grillo HC. Carinal reconstruction. Ann Thorac Surg 1982;34:356–73.
100. Valesky A, Hohlbach G, Schildberg FW. Wertigkeit unterschiedlicher Mafsnahmen zur Minderung der Anastomosen-spannung nach Kontinuitätsresektion der Trachea. Langenbecks Arch Chir 1983;360:59–69.
101. Miura T, Grillo HC. The contribution of the inferior thyroid artery to the blood supply of the human trachea. Surg Gynecol Obstet 1966;123:99–102.
102. Salassa JR, Pearson BW, Payne WS. Gross and microscopic blood supply of the trachea. Ann Thorac Surg 1977;24:100–7.
103. Grindlay JH, Clagett OT, Moersch HJ. Experimental surgery of the thoracic trachea and its bifurcation: preliminary report. Proc Staff Meet Mayo Clin 1949;24:555–6.
104. Juvenelle AA, Citret C. Transplantation de la bronche souche et résection de la bifurcation trachéale. Une étude expérimentale sur le chien. J Chir 1951;67:666–88.

105. Juvenelle A, Citret C. [Discussion]. J Thorac Surg 1950;20:647–8.
106. Meyer RP, Ehrlich RP, Taylor CB, et al. Experimental bronchotracheal anastomosis. Arch Surg 1951;62:753–66.
107. Ehrlich RW, Meyer RP, Taylor C, et al. Reconstruction of the tracheobronchial tree with resection of the lung, carina and lower trachea. Surg Gynecol Obstet 1952;94:570–6.
108. Björk VO, Rodriguez LE. Reconstruction of the trachea and its bifurcation: an experimental study. J Thorac Surg 1958;35:596–603.
109. Barclay RS, McSwan N, Welsh TM. Tracheal reconstruction without the use of grafts. Thorax 1957;12:177–80.
110. Eijgelaar A, Leezenberg JA. Technical aspects of wide resection of trachea and carina. An experimental study in dogs. Arch Chir Neerl 1969;21:303–11.
111. Abbott OA. Experiences with the surgical resection of the human carina, tracheal wall and contralateral bronchial wall in cases of right total pneumonectomy. J Thorac Surg 1950;19:906–22.
112. Bérard M, Juttin P, Maret G. Blessures opératoires des grosses bronches ou de la trachée basse lors de lobectomies et pneumonectomies difficiles. Poumon Coeur 1954;10:149–72.
113. Kergin FG. Carcinoma of the trachea. J Thorac Surg 1952;23:164–8.
114. Mathey J. Tumeur bénigne de l'éperon trachéal. Résection et réfection du carrefour trachéo-bronchique. La Sem des Hopitaux, Paris 1951;27:2699–703.
115. Crafoord C, Björk VO, Hilty H. Zur Bronchialresektion und broncho-trachealen Anastomose bei tuberkulösen Bronchial-stenose. Thoraxchir 1954;2:1–7.
116. Björk VO. Left-sided bronchotracheal anastomosis. J Thorac Surg 1955;30:492–8.
117. Björk VO. Bronchotracheal anastomosis. A follow-up of 9 cases on the left side and 7 on the right. J Thorac Surg 1959;37:800–9.
118. Abbey-Smith R, Nigan BK. Resection of proximal left main bronchus cancer. Thorax 1979;34:616–20.
119. Eschapasse H, Validat F, Mathe J. A propos d'une observation de résection trachéo-bronchique pour un cylindrome. Mém Acad Chir 1961;87:43–7.
120. Archer FL, Harrison RW, Moulder PV. Granular cell myoblastoma of the trachea and carina treated by resection and reconstruction. J Thorac Cardiovasc Surg 1963;45:539–47.
121. Grillo HC, Bendixen HH, Gephart T. Resection of the carina and lower trachea. Ann Surg 1963;158:889–93.
122. Eschapasse H, Vahdat F, Gaillard J, Besso JC. Réflexions sur le résection de la trachée inférieure et de la bifurcation bronchique. Ann Chir Thorac Cardiovasc 1967;6:63–70.
123. Nissen R. Extrakorporelle Zirculation für langdauernde (30 Minuten) Atemunterbrechung zur Operation bifurkationsnaher Trachealgeschwülste. Schw Med Wschr 1961;91:957–60.
124. Woods FM, Neptune WB, Palatchi A. Resection of the carina and main-stem bronchi with the use of extracorporeal circulation. N Engl J Med 1961;l264:492–4.
125. Adkins PC, Izawa EM. Resection of tracheal cylindroma using cardiopulmonary bypass. Surgery 1964; 88:405–9.
126. Eschapasse H. Les tumeurs trachéales primitives: traitement chirurgical. Rev Fr Mal Respir 1974;2:425–46.
127. Perelman M, Koroleva N. Surgery of the trachea. World J Surg 1980;4:583–93.
128. Mitchell JD, Mathisen DJ, Donahue DM, et al. Clinical experience with carinal resection. J Thorac Cardiovasc Surg 1999;117:39–53.
129. Grillo HC. Surgery of the trachea. Curr Probl Surg 1970; July:1–59.
130. Perelman MI. Surgery of the trachea. Moscow: Mir; 1976.
131. Salzer GM, Muller LC. Resection of the tracheal bifurcation through a left thoracotomy. Eur J Cardiothorac Surg 1987;1:125–8.
132. Maeda M, Nakamoto MD, Tsubata N, et al. Operative approaches for left-sided carinoplasty. Ann Thorac Surg 1993;56:441–6.
133. Goeltz FA. Successful anterior thoraco-bronchotomy for a foreign body impacted in the bronchus. Ann Surg 1907;43:369–72.
134. Padhi RK, Lynn FB. The management of bronchopleural fistulas. J Thorac Cardiovasc Surg 1960;39:385–93.
135. Abruzzini P. Trattamento chirurgico delle fistule del bronco principale consecutive a pneumonectomia per tuberculosi. Chir Thorac 1961;14:165–71.
136. Pearson FG, Todd TRJ, Cooper JD. Experience with primary neoplasms of the trachea and carina. J Thorac Cardiovasc Surg 1984;88:511–8.
137. Chamberlain JM, McNeill TM, Parnassa P, Edsall JR. Bronchogenic carcinoma. An aggressive surgical attitude. J Thorac Cardiovasc Surg 1959;38:727–45.
138. Gibbon JH Jr. [Discussion]. J Thorac Cardiovasc Surg 1959;38:741.
139. Hardin CA, Fitzpatrick MJ. Primary carcinoma of the carina and bifurcation successfully treated by resection and reconstruction. Surgery 1959;46:534–8.
140. MacHale SJ. A new technique for repairing the major air passages. J Thorac Cardiovasc Surg 1972;64:6–10.
141. Thompson DT. Tracheal resection with left lung anastomosis following right pneumonectomy. Thorax 1966;21:560–3.
142. Jensik RJ, Faber LP, Milloy FJ, Goldin MD. Tracheal sleeve pneumonectomy for advanced carcinoma of the lung. Surg Gynecol Obstet 1972;134:231–6.
143. Ishihara T, Ikeda T, Inoue H, Fukai S. Resection of cancer of lung and carina. J Thorac Cardiovasc Surg 1977;73:936–43.
144. Deslauriers J, Beaulieu M, Bénazéra A, McClish A. Sleeve pneumonectomy for bronchogenic carcinoma. Ann Thorac Surg 1979;28:465–74.
145. Dartevelle PG, Khalife J, Chapelier A, et al. Tracheal sleeve pneumonectomy for bronchogenic carcinoma: report of 55 cases. Ann Thorac Surg 1988;46:68–72.
146. Tsuchiya R, Goya T, Naruke T, Suemasu K. Resection of tracheal carina for lung cancer. Procedure, complications and mortality. J Thorac Cardiovasc Surg 1990; 99:779–87.
147. Mathisen DJ, Grillo HC. Carinal resection for bronchogenic carcinoma. J Thorac Cardiovasc Surg 1991; 102:16–23.
148. Mathisen DJ, Kuo EY, Hahn C, et al. Inhaled nitric oxide for adult respiratory distress syndrome after pulmonary resection. Ann Thorac Surg 1998;66: 1894–902.
149. Roviaro GC, Varoli F, Rebuffat C, et al. Tracheal sleeve pneumonectomy for bronchogenic carcinoma. J Thorac Cardiovasc Surg 1994;107:13–8.
150. Mitchell JD, Mathisen DJ, Wright CD, et al. Resection for bronchogenic carcinoma involving the carina: long-term results and effects of nodal status on outcome. J Thorac Cardiovasc Surg 2001;121:465–71.
151. McClish A, Deslauriers J, Beaulieu M, et al. High flow catheter ventilation during major tracheobronchial reconstruction. J Thorac Cardiovasc Surg 1985; 89:508–12.
152. Ravitch MM. [Discussion]. J Thorac Surg 1951;22:583.
153. Friedmann JB, Emma E. A consideration of anesthesia during carinal resection. Anesthesiology 1951; 12:740–4.
154. Grigor KC, Shaw M. Anaesthesia for tracheo-bronchial reconstruction. Anaesthesia 1958;13:299–305.

155. Baumann J, Forster J. L'anesthésia en chirurgie trachéale. Poumon Coeur 1960;16:19–32.
156. Geffin B, Bland J, Grillo HC. Anesthetic management of tracheal resection and reconstruction. Anesth Analg 1969;48:884–94.
157. Theman TE, Kerr JH, Nelems JM, Pearson FG. Carinal resection. A report of two cases and a description of the anesthetic technique. J Thorac Cardiovasc Surg 1975;71:314–420.
158. Lee P, English ICW. Management of anaesthesia during tracheal resection. Anaesthesia 1974;29:305–6.
159. El Baz HN, Jensik R, Faber LP, Faro RS. One-lung high-frequency ventilation for tracheoplasty and bronchoplasty. A new technique. Ann Thorac Surg 1982;34:564–70.
160. Wilson RS. Anesthetic management for tracheal reconstruction. In: Grillo HC, Eschapasse H, editors. Major challenges. International trends in general thoracic surgery. Vol 2. Philadelphia: WB Saunders; 1987. p. 3–12.
161. Grillo HC. Primary reconstruction of airway after resection of subglottic laryngeal and upper tracheal stenosis. Ann Thorac Surg 1982;33:3–18.
162. Gerwat J, Bryce DP. The management of subglottic laryngeal stenosis by resection and direct anastomosis. Laryngoscope 1974;84:940–57.
163. Pearson FG, Cooper JD, Nelems JM, Van Nostrand AWP. Primary tracheal anastomosis after resection of the cricoid cartilage with preservation of recurrent laryngeal nerves. J Thorac Cardiovasc Surg 1975;70:806–16.
164. Couraud L, Martigne C, Houdelette P, et al. Intérêt de la résection cricoidienne dans le traitement de sténoses crico-trachéales après intubation. Ann Chir 1979; 33:242–6.
165. Grillo HC, Mathisen DJ, Wain JC. Laryngotracheal resection and reconstruction for subglottic stenosis. Ann Thorac Surg 1992;53:54–63.
166. Maddaus MA, Toth JLR, Gullane PJ, Pearson FG. Subglottic tracheal resection and synchronous laryngeal reconstruction. J Thorac Cardiovasc Surg 1992;104:1443–50.
167. Monnier P, Savary M, Chapuis G. Partial cricoid resection with primary tracheal anastomosis for subglottic stenosis in infants and children. Laryngoscope 1993;103:1273–83.
168. Watson WL. Cancer of the trachea fourteen years after treatment for cancer of larynx. J Thorac Surg 1942; 12:142–50.
169. Sloan H, Cowley RA. Posterior mediastinal tracheotomy. J Thorac Surg 1951;21:602–7.
170. Kleitsch WP. Anterior mediastinal tracheostomy. J Thorac Surg 1952;24:38–42.
171. Minor GR. Trans-sternal tracheal excision for carcinoma. J Thorac Surg 1952;24:88–92.
172. Waddell WR, Cannon B. A technic for subtotal excision of the trachea and establishment of a sternal tracheostomy. Ann Surg 1959;149:1–8.
173. Sisson GA, Straehley CJ Jr, Johnson NE. Mediastinal dissection for recurrent cancer after laryngectomy. Laryngoscope 1962;72:1064–77.
174. Ellis PR Jr, Harrington DB, Beall AC Jr, DeBakey ME. The use of heavy Marlex mesh for tracheal reconstruction following resection for malignancy. J Thorac Cardiovasc Surg 1962; 44:520–7.
175. Grillo HC. Terminal or mural tracheostomy in the anterior mediastinum. J Thorac Cardiovasc Surg 1966; 51:422–7.
176. Grillo HC, Mathisen DJ. Cervical exenteration. Ann Thorac Surg 1990;49:401–9.
177. Stell PM, Bickford BJ, Brown GA. Thoraco-tracheostomy after resection of the larynx and cervical trachea for cancer. J Laryngol Otol 1970;84:1097–102.
178. Krespi YP, Wurster CF, Sisson CA. Immediate reconstruction after total laryngopharyngo-esophagectomy and mediastinal dissection. Laryngoscope 1985;95:156–61.
179. Gomes MN, Kroll S, Spear SL. Mediastinal tracheostomy. Ann Thorac Surg 1987;34:539–43.
180. Orringer MB, Sloan H. Anterior mediastinal tracheostomy. Indications, techniques and clinical experience. J Thorac Cardiovasc Surg 1979;78:850–9.
181. Withers EH, Davis JL, Lynch JB. Anterior mediastinal tracheostomy with a pectoralis major musculocutaneous flap. Plast Reconst Surg 1981;67:381–4.
182. Couraud L, Bruneteau A, Martigne C, Meriot S. Prevention and treatment of complications and sequelae of tracheal resection anastomosis. Int Surg 1982; 67:235–9.
183. Grillo HC, Zannini P, Michelassi F. Complications of tracheal reconstruction. J Thorac Cardiovasc Surg 1986; 91:322–8.
184. Bond CJ. Treatment of tracheal stenosis by a new T-shaped tracheotomy tube. Lancet 1891;1:539.
185. Falbe-Hansen J. Tracheal stenosis in polio patients. Acta Oto-Laryngol 1955;45:498–508.
186. Aboulker P, Lissac J, Saint-Paul O. De quelques accidents respiratoires dus au rétrécissement du calibre laryngo-trachéal asprès trachéotomie. Acta Chir Belg 1960;59:553–61.
187. Montgomery WW. T-tube tracheal stent. Arch Otolaryngol 1965;82:320–1.
188. Cooper JD, Todd TRJ, Ilves R, Pearson FG. Use of the silicone tracheal T-tube for the management of complex tracheal injuries. J Thorac Cardiovasc Surg 1981; 82:559–68.
189. Gaissert HA, Grillo HC, Mathisen DJ, Wain JC. Temporary and permanent restoration of airway continuity with the tracheal T-tube. J Thorac Cardiovasc 1994; 107:600–6.
190. Westaby S, Jackson JW, Pearson FG. A bifurcated silicone rubber stent for relief of tracheobronchial obstruction. J Thorac Cardiovasc Surg 1982;83:414–7.
191. Grillo HC. Stents and sense [editorial]. Ann Thorac Surg 2000;70:1139.
192. Houston HE, Payne WS, Harrison EG Jr, Olsen AM. Primary cancers of the trachea. Arch Surg 1969;99:132–40.
193. Hajdu SI, Huvos AG, Goodner JT, et al. Carcinoma of the trachea: clinicopathologic study of 41 cases. Cancer 1970;25:1448–56.
194. Forster E, Holderbach L. Rapport sur la chirurgie des tumeurs et des rétrécissements de la trachée. Poumon Coeur 1960;16:33–119.
195. Grillo HC. Reconstruction of the trachea. Experience in 100 consecutive cases. Thorax 1973;28:667–79.
196. Pearson FG, Thompson DW, Weissberg D, et al. Adenoid cystic carcinoma of the trachea. Experience with 16 patients managed by tracheal resection. Ann Thorac Surg 1974;18:16–29.
197. Maziak DE, Todd TRJ, Keshavjee SH, et al. Adenoid cystic carcinoma of the airway: thirty-two-year experience. J Thorac Cardiovasc Surg 1996;112:1522–32.
198. Regnard JF, Fourquier P, Levasseur P. Results and prognostic factors in resections of primary tracheal tumors: a multicenter retrospective study. J Thorac Cardiovasc Surg 1996;111:808–14.
199. Perelman MI, Koivoleva N, Birjukov J, Goudovsky L. Primary tracheal tumors. Sem Thorac Cardiovasc Surg 1996;8:400–2.
200. Shin DH, Mark EJ, Suen HC, Grillo HC. Pathological staging of papillary carcinoma of the thyroid with airway invasion based upon the anatomic manner of extension to the trachea: a clinicopathologic study based on 22 patients who underwent thyroidectomy and airway resection. Hum Pathol 1993;24:866–70.

201. Lazo VV. Resection of the cervical part of the trachea in cancer of the thyroid gland. Vopr Onkol 1957; 3:635–6.
202. Ishihara T, Kikuchi K, Ikeda T, et al. Resection of thyroid carcinoma infiltrating the trachea. Thorax 1978;33:378–86.
203. Ishihara T, Kobayashi K, Kikuchi J, et al. Surgical treatment of advanced thyroid carcinoma invading the trachea. J Thorac Cardiovasc Surg 1991;102:717–20.
204. Tsumori T, Nakae K, Miyata M, et al. Clinicopathologic study of thyroid carcinoma infiltrating the trachea. Cancer 1985;56:2843–8.
205. Fujimoto Y, Obara T, Ito Y, et al. Aggressive surgical approach for locally invasive papillary carcinoma of the thyroid in patients over forty-five years of age. Surgery 1986;100:1098–107.
206. Maeda M, Nakamoto K, Ohta M, et al. Statistical survey of tracheobronchoplasty in Japan. J Thorac Cardiovasc Surg 1989;97:402–14.
207. Grillo HC, Zannini P. Resectional management of airway invasion by thyroid carcinoma. Ann Thorac Surg 1986;42:287–98.
208. Grillo HC, Suen HC, Mathisen DJ, Wain JC. Resectional management of thyroid carcinoma invading the airway. Ann Thorac Surg 1992;54:3–10.
209. Czaja JM, McCaffrey TV. The surgical management of laryngotracheal invasion of well-differentiated papillary thyroid carcinoma. Arch Otol Head Neck Surg 1997;123:484–90.
210. Hendrick JW. An extended operation for thyroid carcinoma. Surg Gynecol Obstet 1963;116:183–8.
211. Frazell EL, Foote FW Jr. Papillary cancer of the thyroid: a review of 25 years of experience. Cancer 1958; 11:895–922.
212. Pearson FG, Goldberg M, daSilva AJ. Tracheal stenosis complicating tracheostomy with cuffed tubes: clinical experience and observations from a prospective study. Arch Surg 1968;97:380–94.
213. Witz JP, Greiner G, Reys P, Lang S. Complication rare de la trachéotomie. Sténose basse de la trachée thoracique. Mém Acad Chir 1960;86:123–8.
214. Van Wien A, DeClercq F, Van Eych M, Reinhold H. Tracheal stenosis after tracheostomy. Acta Chir Belg 1961;60:85–93.
215. Byrn FM, Davies CK, Harrison GK. Tracheal stenosis following tracheostomy: a case report. Br J Anaesth 1967;39:171–3.
216. Fraser K, Beli PRF. Distal tracheal stenosis following tracheostomy. Br J Surg 1967;54:302–3.
217. Jewsbury P. Resection of tracheal stricture following tracheostomy. Lancet 1969;2:411–3.
218. Dor J, Dor V, Malmejac C, et al. Résection - suture de la trachée cervico-thoracique pour sténose post-trachéotomie. Controle éloigné de trois observations. Ann Chir 1969;23:177–82.
219. Dolton EG. Resection of tracheal strictures. Lancet 1969;2:544.
220. Schaudig A. Tracheal resektion bei benigner und maligner Stenose. Thoraxchir 1969;17:508–11.
221. Lindholm C-E. Prolonged endotracheal intubation. Acta Anaesth Scand 1969;Suppl 33:1–131.
222. Grillo HC. Surgical treatment of postintubation tracheal injuries. J Thorac Cardiovasc Surg 1979;78:860–75.
223. Shapshay SM, Beamis JF, Hybels RI, et al. Endoscopic treatment of subglottic and tracheal stenosis by radial laser incision and dilation. Ann Otol Rhinol Laryngol 1987;96:661–4.
224. Bisson A, Bonnette P, El Kadi B, et al. Tracheal sleeve resection for iatrogenic stenosis (subglottic laryngeal and tracheal). J Thorac Cardiovasc Surg 1992; 104:882–7.
225. Couraud L, Jougon J, Velly JF, Klein C. Sténoses iatrogênes de la voie respiratoire. Evolution des indications thérapentiques. Ann Chir Thorac Cardiovasc 1994;48:277–83.
226. Donahue DM, Grillo HC, Wain JC, et al. Reoperative tracheal resection and reconstruction for unsuccessful repair of postintubation stenosis. J Thorac Cardiovasc Surg 1997;114:934–9.
227. Le Brigand H, Roy B. Fistules trachéo-oesophagiennes après trachéotomies: à propos de 4 observations. Mém Acad Chir 1966;92:405–16.
228. Flege JB Jr. Tracheoesophageal fistula caused by cuffed tracheostomy tube. Ann Surg 1967;166:153–6.
229. Hedden M, Ersoz CJ, Safar P. Tracheo-esophageal fistulas following prolonged artificial ventilation via cuffed tracheostomy tubes. Anesthesiol 1969;31:281–9.
230. Braithwaite F. Closure of a tracheo-oesophageal fistula. Br J Plast Surg 1961;14:138–43.
231. Thomas AN. The diagnosis and treatment of tracheoesophageal fistula caused by cuffed tracheal tubes. J Thorac Cardiovasc Surg 1973;65:612–9.
232. Grillo HC, Moncure AC, McEnany MT. Repair of inflammatory tracheoesophageal fistula. Ann Thorac Surg 1976;22:112–9.
233. Mathisen DJ, Grillo HC, Wain JC, Hilgenberg AD. Management of acquired nonmalignant tracheoesophageal fistula. Ann Thorac Surg 1991;52:759–65.
234. Couraud L, Bercovici D, Zanotti L, et al. Treatment of oesophago-tracheal fistula secondary to respiratory intensive care. Report of seventy cases. Ann Chir 1989;43:677–81.
235. Macchiarini P, Verhoye JP, Chapelier A, et al. Evaluation and outcome of different surgical techniques for post-intubation tracheoesophageal fistulas. J Thorac Cardiovasc Surg 2000;119:268–76.
236. Lunding M. The tracheotomy tube and postoperative tracheostomy complications. Acta Anaesth Scand 1964; 8:181–9.
237. Silen W, Spieker D. Fatal hemorrhage from the innominate artery after tracheostomy. Ann Surg 1965; 162:1005–12.
238. Stiles PJ. Tracheal lesions after tracheostomy. Thorax 1965;20:517–22.
239. Couraud L, Favarrel-Garrigue JC, Chevais R. Les hémorragies cataclysmiques secondaires des trachéotomies. Considérations anatomiques et thérapeutiques. Ann Chir Thorac Cardiovasc 1966;5:772–83.
240. Foley FD, Moncrief JA, Mason AD Jr. Pathology of the lung in fatally burned patients. Ann Surg 1968; 167:251–64.
241. Grillo HC. Congenital lesions, neoplasms and injuries of the trachea. In: Sabiston DC, Spencer FC, editors. Gibbon's surgery of the chest. 3rd ed. Philadelphia: WB Saunders; 1976. p. 256–93.
242. Cooper JD. Tracheo-innominate artery fistula. Successful management of three consecutive patients. Ann Thorac Surg 1977;24:439–46.
243. Couraud L, Hafez A, Velly JF. Hémorragies endotrachéales par fissuration du tronc artériel brachiocéphalique au cours des canulations prolongées. Cure chirurgicale. Presse Med 1984;13:2577–9.
244. Jones JW, Reynolds M, Hewitt RL, Drapanas T. Tracheoinnominate artery erosion: successful surgical management of a devastating complication. Ann Surg 1976;184:194–204.
245. Colles CJ. On stenosis of the trachea after tracheotomy for croup and diphtheria. Ann Surg 1886;3:499–507.
246. Bignon J, Chrétien J. Étude post-mortem des altérations laryngo-trachéo-bronchiques au cours de la trachéotomie avec respiration assistée. J Franc Méd Chir Thorac 1962;16:125–55.

247. Yanagisawa E, Kirchner JA. The cuffed tracheotomy tube. Arch Otolaryngol 1964;79:80–7.
248. Atkins JP. Tracheal resections following the use of cuffed tracheotomy tubes. Ann Otol Rhinol Laryngol 1964; 73:L1124–9.
249. Florange W, Muller J, Forster E. Morphologies de la nécrose trachéale après trachéotomie et utilisation d'une prosthèse respiratoire. Anesth Analg 1965; 22:693–703.
250. Gibson P. Aetiology and repair of tracheal stenosis following tracheostomy and intermittent positive pressure respiration. Thorax 1967;22:1–6.
251. Murphy DA, MacLean LD, Dobell ARC. Tracheal stenosis as a complication of tracheostomy. Ann Thorac Surg 1966;2:44–51.
252. Cooper JD, Grillo HC. The evolution of tracheal injury due to ventilatory assistance through cuffed tubes: a pathologic study. Ann Surg 1969;169:334–48.
253. Friedberg SA, Griffith TE, Hass GM. Histologic changes in the trachea following tracheostomy. Ann Otol 1965;74:785–98.
254. Andrews MJ, Pearson FG. The incidence and pathogenesis of tracheal injury following cuffed tube tracheostomy with assisted ventilation: an analysis of a two-year prospective study. Ann Surg 1971;173:249–63.
255. Adriani J, Phillips M. Use of the endotracheal cuff; some data pro and con. Anesthesiol 1957;18:1–14.
256. Cooper JD, Grillo HC. Experimental production and prevention of injury due to cuffed tracheal tubes. Surg Gynecol Obstet 1969;129:1235–41.
257. Knowlson GTG, Bassett HFM. The pressures exerted on the trachea by endotracheal inflatable cuffs. Br J Anesth 1970;42:834–7.
258. Grimm JE, Knight RT. An improved intratracheal technic. Anesthesiol 1943;4:6–11.
259. Lomholt N. A new tracheostomy tube. Acta Anaesth Scand 1967;11:311–8.
260. Carroll R, Hedden M, Safar P. Intratracheal cuffs: performance characteristics. Anesthesiology 1969;31:275–81.
261. Grillo HC, Cooper JD, Geffin B, Pontoppidan H. A low-pressure cuff for tracheostomy tubes to minimize tracheal injury: a comparative clinical trial. J Thorac Cardiovasc Surg 1971;62:898–907.
262. Baumann J. Rapport sur les traumatismes de la trachée. Poumon Coeur 1960;16:7–18.
263. Chodosh PL. Cricoid fracture with tracheal avulsion. Arch Otolaryngol 1968;87:461–7.
264. Ashbaugh DG, Gordon JH. Traumatic avulsion of the trachea associated with cricoid fracture. J Thorac Cardiovasc Surg 1975;69:800–3.
265. Beall AC, Noon GP, Harris H. Surgical management of tracheal trauma. J Trauma 1967;7:248–56.
266. Ecker RR, Libertini RV, Rea WJ, et al. Injuries of the trachea and bronchi. Ann Thorac Surg 1971;11:289–98.
267. Bertelsen S, Howitz P. Injuries of the trachea and bronchi. Thorax 1972;27:188–94.
268. Symbas PN, Hatcher CR Jr, Boehn GAW. Acute penetrating tracheal trauma. Ann Thorac Surg 1976;22:473–7.
269. Grover FL, Ellestad C, Aron KV, et al. Diagnosis and management of major tracheobronchial injuries. Ann Thorac Surg 1979;28:384–91.
270. Angood PB, Attia EL, Brown RA, Mulder DS. Extrinsic civilian trauma to the larynx and cervical trachea. Important predictors of long term morbidity. J Trauma 1986;26:869–73.
271. Mathisen DJ, Grillo HC. Laryngotracheal trauma. Ann Thorac Surg 1987;43:254–62.
272. Couraud L, Velly JF, Martigne C, N'Diaye M. Post-traumatic disruption of the laryngotracheal junction. Eur J Cardiothorac Surg 1989;3:441–4.
273. Gaissert HA, Lofgren RH, Grillo HC. Upper airway compromise after inhalation injury: complex strictures of larynx and trachea and their management. Ann Surg 1993;218:672–8.
274. Muehrcke DD, Grillo HC, Mathisen DJ. Reconstructive airway surgery after irradiation. Ann Thorac Surg 1995;59:14–8.
275. Borrie J. Tracheal stenosis in infancy. Thorax 1960; 15:64–9.
276. Sorensen HR, Moesner J, Nielsen PA. Influence of growth upon the development of stenosis following experimental resection of the trachea in puppies. Scand J Thorac Cardiovasc Surg 1971;5:202–5.
277. Maeda M, Grillo HC. Tracheal growth following anastomosis in puppies. J Thorac Cardiovasc Surg 1972; 64:304–13.
278. Maeda M, Grillo HC. Effect of tension on tracheal growth after resection and anastomosis in puppies. J Thorac Cardiovasc Surg 1973;65:658–68.
279. Kotake Y, Grillo HC. Reduction of tension at the anastomosis following tracheal resection in puppies. J Thorac Cardiovasc Surg 1976;71:600–4.
280. Murphy DA, Dunn GL, Poirier N, Martin M. Growth of tracheal anastomoses: an experimental study in weanling pigs. Ann Thorac Surg 1973;16:158–62.
281. Mendez-Picon G, Hutcher NE, Neifeld V, Salzberg AM. Long term study of tracheal growth after segmental resection in puppies. J Pediatr Surg 1974;9:615–9.
282. Burrington JD. Tracheal growth and healing. J Thorac Cardiovasc Surg 1978;76:453–8.
283. Cantrell JR, Guild HG. Congenital stenosis of the trachea. Am J Surg 1964;108:297–305.
284. Carcassonne M, Dor V, Aubert J, Kreitman P. Tracheal resection with primary anastomosis in children. J Pediatr Surg 1973;8:1–8.
285. Mansfield PB. Tracheal resection in infancy. J Pediatr Surg 1980;15:79–81.
286. Nakayama DK, Harrison MR, deLorimier AA, et al. Reconstructive surgery for obstructing lesions of the intrathoracic trachea in infants and small children. J Pediatr Surg 1982;17:854–68.
287. Grillo HC, Zannini P. Management of obstructive tracheal disease in children. J Pediatr Surg 1984; 19:414–6.
288. Alstrup P, Sorensen HR. Resection of acquired tracheal stenosis in childhood. J Thorac Cardiovasc Surg 1984; 87:547–9.
289. Couraud L, Moreau JM, Velly JF. The growth of circumferential scars of the major airways from infancy to adulthood. Eur J Cardiothorac Surg 1990;4:521–6.
290. Fearon B, Cotton R. Surgical correction of subglottic stenosis of the larynx in infants and children. Ann Otol Rhinol Laryngol 1974;83:428–31.
291. Kimura K, Mukohara N, Tsugawa C, et al. Tracheoplasty for congenital stenosis of the entire trachea. J Pediatr Surg 1982;17:869–71.
292. Idriss FS, DeLeon SY, Ilbani MN, et al. Tracheoplasty with pericardial patch for extensive tracheal stenosis in infants and children. J Thorac Cardiovasc Surg 1984;88:527–36.
293. Heimansohn DA, Kesler KA, Turrentine MW, et al. Anterior pericardial tracheoplasty for congenital tracheal stenosis. J Thorac Cardiovasc Surg 1991;102:710–6.
294. Jaquiss RDB, Lusk PR, Spray TL, et al. Repair of long-segment tracheal stenosis in infancy. J Thorac Cardiovasc Surg 1995;110:1504–12.
295. Dunham ME, Holinger LD, Backer CL, Mavroudis C. Management of severe congenital tracheal stenosis. Ann Otol Rhinol Laryngol 1994;103:351–6.
296. Backer CL, Mavroudis C, Dunham CE, Holinger LD. Reoperation after pericardial patch tracheoplasty. J Pediatr Surg 1997;32:1108–12.

297. Tsang V, Munday A, Gilbe C, Goldstraw P. Slide tracheoplasty for congenital funnel-shaped tracheal stenosis. Ann Thorac Surg 1989;48:632–5.
298. Grillo HC. Slide tracheoplasty for long segment congenital tracheal stenosis. Ann Thorac Surg 1994;58:613–21.
299. Grillo HC, Wright CD, Vlahakes GJ, MacGillivray TE. Management of congenital tracheal stenosis by means of slide tracheoplasty or resection and reconstruction with long term follow-up of growth after slide tracheoplasty. J Thorac Cardiovasc Surg 2002;123:145–52.
300. Donahoe PK, Gee PE. Complete laryngotracheoesophageal cleft: management and repair. J Pediatr Surg 1984;19:143–8.
301. Brandenberg JH. Idiopathic subglottic stenosis. Trans Am Acad Opthalmol Otolaryngol 1972;76:1402–6.
302. Mikaelian DO. Idiopathic subglottic stenosis in an adult. J Laryngol Otol 1974;88:467–72.
303. Jazbi B, Goodwin C, Tackett D, Faulkner S. Idiopathic subglottic stenosis. Ann Otol Rhinol Laryngol 1972;86:644–8.
304. Grillo HC, Mark EJ, Mathisen DJ, Wain JC. Idiopathic laryngotracheal stenosis and its management. Ann Thorac Surg 1993;56:80–7.
305. Dedo HH, Catten MD. Idiopathic progressive subglottic stenosis: findings and treatment in 52 patients. Ann Otol Rhinol Laryngol 2001;110:305–11.
306. Ashiku SK, Kuzucu A, Grillo HC, et al. Laryngotracheal resection as an effective definitive surgical treatment for idiopathic laryngotracheal stenosis. J Thorac Cardiovasc Surg. [In press]
307. Mark JE, Patterson GA, Grillo HC. Case records of the Massachusetts General Hospital 46 – 1992. N Engl J Med 1992;327:1512–8.
308. Mark EJ, Braman SS, Grillo HC. Case records of Massachusetts General Hospital 32 – 1999. N Engl J Med 1999;341:1292–9.
309. Herzog H, Nissen R. Erschlaffung und expiratorische Invagination des membranösen Teils der intrathorakalen Luftröhre und der Haupt bronchien als Ursache der asphyktischen Anfälle beim Asthma bronchiale und bei der chronischen asthmoiden Bronchitis des Lungenemphysems. Schweiz Med Wochenschr 1954;84:217–9.
310. Herzog H. Exspiratorische Stenosis der Trachea und der grossen Bronchien durch die erschlaffte Pars membranacea. Operative Korrektur durch Spanplastik. Thoraxchir 1958;5:281–319.
311. Nissen R. Tracheoplastik zur Beseitigung der Erschlaffung des membranösen Teils der Intrathorakalen Luftröhre. Schweiz Med Wochenschr 1954;84:219–21.
312. Rainer WG, Newby JP, Kelble DL. Long term results of tracheal support surgery for emphysema. Dis Chest 1968;53:765–72.
313. Wright CD, Hahmoud Z, Grillo HC, Mathisen DJ. Results of membranous wall tracheoplasty for large airway expiratory collapse. [In preparation]
314. Maier HC, Gould WJ. Agenesis of the lung with vascular compression of the tracheobronchial tree. J Pediatr 1953;43:38–42.
315. Quillin SP, Shackleford GD. Postpneumonectomy syndrome after left lung resection. Radiology 1991;179:100–2.
316. Grillo HC, Shepard JO, Mathisen DJ, Kanarek DJ. Postpneumonectomy syndrome: diagnosis, management and results. Ann Thorac Surg 1992;54:638–51.
317. Shamji FM, Deslauriers J, Daniel M, et al. Postpneumonectomy syndrome with an ipsilateral aortic arch after left pneumonectomy. Ann Thorac Surg 1996;62:1627–31.
318. Boiselle PM, Shepard JO, McLoud TC, et al. Postpneumonectomy syndrome: another twist. J Thorac Imaging 1997;12:209–11.
319. Johnson J, Kirby CK, Lazatin CS, Cocke JA. The clinical use of a prosthesis to prevent overdistention of the remaining lung following pneumonectomy. J Thorac Surg 1949;18:164–72.
320. Adams HD, Junod FL, Aberdeen E, Johnson J. Severe airway obstruction caused by mediastinal displacement after right pneumonectomy in a child. J Thorac Cardiovasc Surg 1972;63:534–9.
321. Kaunitz VH, Fisher FC. Continued pneumothorax in the management of the postpneumonectomy pleural space. Ann Thorac Surg 1966;2:455–63.
322. Powell RW, Luck SR, Raffensperger JG. Pneumonectomy in infants and children: the use of a prosthesis to prevent mediastinal shift and its complications. J Pediatr Surg 1979;14:231–7.
323. Szarnicki R, Maurseth K, Defeval M, Stark J. Tracheal compression by the aortic arch following right pneumonectomy in infancy. Ann Thorac Surg 1978;25:231–5.
324. Wasserman K, Jamplis RW, Lash H, et al. Postpneumonectomy syndrome: surgical correction using Silastic implants. Chest 1979;75:60–78
325. Rasch DK, Grover FL, Schnapf BM, et al. Right pneumonectomy syndrome in infancy treated with an expandable prosthesis. Ann Thorac Surg 1990;50:125–6.

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Part 1

Diseases, Diagnosis, Results of Treatment

ANATOMY, PHYSIOLOGY, PATHOLOGY, DIAGNOSTIC METHODS

DISEASES AND RESULTS OF TREATMENT

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Anatomy of the Trachea

Hermes C. Grillo, MD

The trachea functions as a conduit for ventilation and also for clearance of tracheal and bronchial secretions. In disease, purulent matter and blood are evacuated via the trachea. Viewed as a simple tube, the trachea appears to be a structure ideally suited for surgical reconstruction or even for replacement following removal of a diseased segment. However, the trachea exhibits a number of anatomical features that may cause great difficulties for surgical reconstruction or replacement. These include its unpaired nature, its unique lateral semirigidity but longitudinal flexibility, its short length, its limited longitudinal elasticity, its proximity to major vascular structures, and its largely segmental blood supply. Barriers to tracheal replacement with an epithelial lined structure are discussed in Chapter 45, "Tracheal Replacement."

Tracheal Structure

The adult male trachea averages 11.8 cm in length (range 10 to 13 cm) from the lower border of the cricoid cartilage to the top of the carinal spur, varying with the patient's height. There are usually from 18 to 22 cartilages within this length, approximating almost two rings per cm.[1] Cartilaginous rings may be incomplete or bifid. The lateral tracheobronchial angles are located slightly higher than the carinal spur so that the length of the trachea proper along its lateral wall is slightly shorter than that measured anteriorly in the midline to the carinal spur (Figure 1-1). The carina in the adult projects quite consistently on the body surface at the level of the sternal angle since it is held in place by the aortic arch. The right main bronchus continues more vertically, whereas the left is always more horizontal with respect to the trachea. The angles between the bronchi and trachea vary quite widely. In infants, the subcarinal angle between the bronchi is much wider and the bronchi lie more transversely.

The level of the trachea and of its lesions is often described by reference to specific vertebrae. Since levels vary in an individual with cervical flexion and extension as well as with respiration and deglutition, and among individuals by age, spinal curvature, anteroposterior diameter of the thorax, and body build, denomination by vertebral level is of little use. More to the point is the length of airway from the vocal cords to carina, or better, of the trachea from cricoid to carina, using the carinal spur as the lower point of mea-

surement. Also important are measures of length of uninvolved trachea above and below a lesion, plus the longitudinal extent of the lesion. These distances are easily determined, although without absolute precision, by imaging (see Chapter 4, "Imaging the Larynx and Trachea") and bronchoscopy (see Chapter 5, "Diagnostic Endoscopy"). As a practical rule, I consider about three-fifths of the juvenile trachea to reside above the sternal notch, about one-half in the young adult, and one-third or less in older adults. In the first two groups especially, the proportion of cervical trachea varies with cervical extension and flexion.

In the adult, the tracheal lumen is often roughly ovoid, flattened anteroposteriorly (Figure 1-2). The rings are normally C-shaped, with the posterior membranous wall connecting the arms of the "C" in an essentially straight line measuring generally less than one-third of the circumference of the trachea (see Figure 1-2*A*–*C*). The proportion of cross-sectional cartilaginous length to length of membranous wall does not change from infancy during growth.[2] An adult tracheal ring is about 4 mm high. Therefore, there are about two rings per cm of trachea. The length and diameter of the trachea is roughly proportional to the size of the individual. Men generally have a trachea of larger diameter than women (see Figures 1-2*B*,*C*). In the adult male, the external diameters of the trachea measure about 2.3 cm coronally and 1.8 cm sagitally. Corresponding figures in the female are 2.0 and 1.4 cm. The tracheal wall is about 3 mm thick. The trachea narrows somewhat as it progresses distally to the carina, more notably in children. These are important points in selecting an endotracheal tube, especially for ventilation, where the tube may be in place for a long time. A small man or woman, even if obese, will nonetheless have a trachea of shorter length and narrower diameter. An excessively wide tube can produce subglottic erosion and consequent stenosis.

Griscom and Wohl together with Fenton measured the length, diameters, cross-sectional area, and volume of the trachea in children under the age of 6 years, asleep or resting quietly, and of older children and adolescents at total lung capacity using computed tomography (CT) scans (Table 1-1).[3–5] All parameters correlated with body height with little variance for gender in young children. Considerable variation in measurements of length and diameter are recorded in the literature depending upon the artifacts of handling specimens, the methods of observation, and whether the observations were made in the living or postmortem. The techniques involved measurements of fixed and unfixed autopsy specimens, the use of x-ray or CT images, and bronchoscopy. In small children, body weight may actually correlate better with tracheal growth than height or age. Increase in length outstrips growth of cross-sectional area in the first year of life.[6] Thereafter, the rate of lengthening falls below the rate of area growth until puberty.[2] Initially, the anteroposterior diameter is slightly greater than the transverse, producing a nearly circular lumen (see Figure 1-2*A*). Gradually, as the child grows, the adult configuration emerges. At first, the trachea is somewhat funnel-shaped, but the discrepancy between the area at the subcricoid level and the carina gradually diminishes, first to a cylindrical form and later to the more ovoid adult shape (see Figure 1-2*B*).[2,6] After age 14 years, female tracheae generally stop growing whereas male tracheae continue to enlarge in cross section but not in length.[4] Great care must be taken not to use excessively large endotracheal tubes for ventilation in infants and children. Formulas for selecting tube size with relation to age are not of great value because of individual variation.

The shape of the adult trachea varies even without disease. Some remain nearly circular rather than becoming ovoid (see Figure 1-2*C*). In others, the sagittal diameter may be greater than the coronal. A slightly triangular configuration occurs less often. Unique and unexplained distortions also occur. The cross-sectional area as well as shape changes dynamically with intraluminal pressure alterations due to cough (see Figure 1-2*B*), respiration, and ventilation; tracheal length and volume vary similarly. The aorta may displace and deform the lower trachea (see Figure 1-2*D*).

The cross-sectional configuration of the trachea may be markedly altered with increasing age, particularly in the presence of chronic obstructive lung disease. The lower two-thirds of the trachea may gradually become flattened from side to side with a consequent decrease in lateral diameter and increase in anteroposterior diameter (see Figure 1-2*E*). This deformation is called, "saber sheath trachea."[7] In this

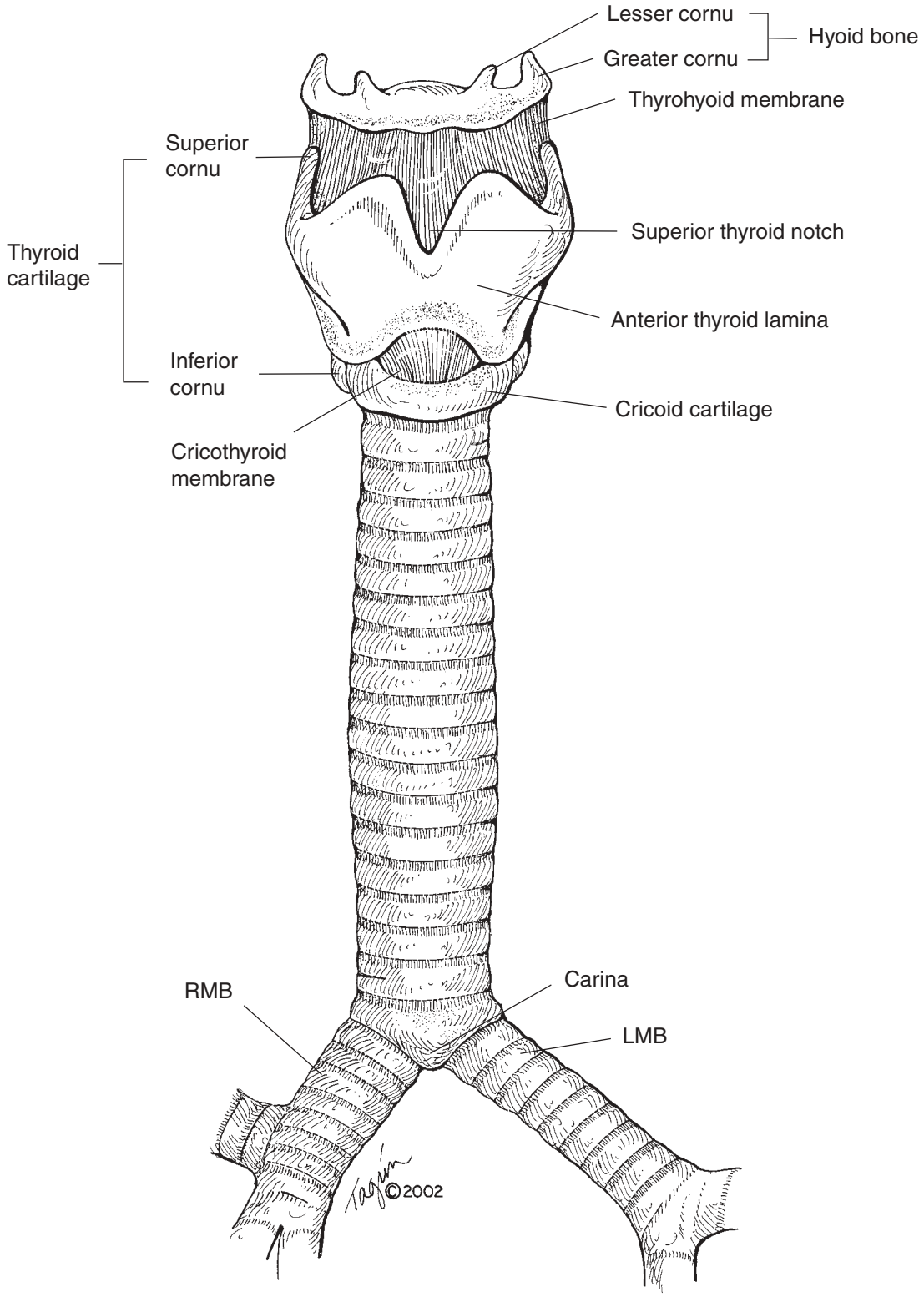


FIGURE 1-1 *Principal features of the larynx and trachea. Anterior view. Tracheobronchial angles vary widely. LMB, RMB = left, right main bronchus.*

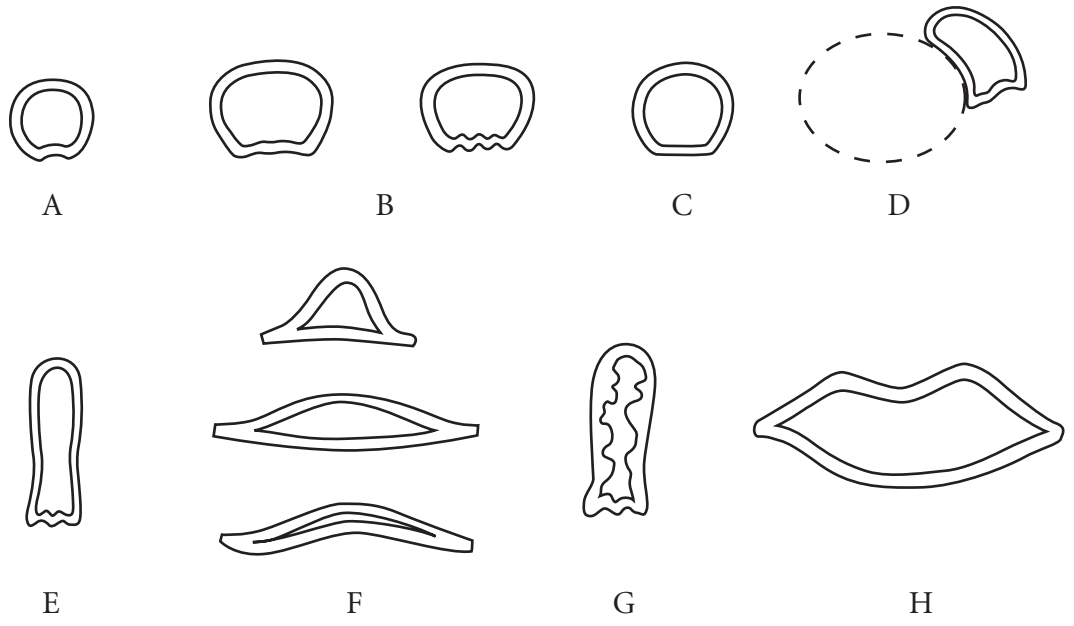


FIGURE 1-2 *Cross-sectional configuration of the trachea in health and disease.* A, *Juvenile trachea appears quite circular.* B, *Adult male trachea at rest on the left. The shape of the lumen can also be more circular. With cough, the membranous wall "accordions," pulling cartilage together (at the right).* C, *Female trachea is smaller in diameter. In this case, the lumen is circular.* D, *Common deformity related to proximity to the aorta just above the left main bronchus.* E, *Saber-sheath trachea. The walls are not malacic.* F, Above. *Triangular deformation which occurs in some patients with chronic obstructive pulmonary disease, especially in proximal trachea.* Middle. *In the thoracic trachea, flattening and softening of cartilage occurs, with elongation of membranous wall.* Below. *Obstructive approximation of anterior and posterior walls with expiratory effort or cough.* G, *Tracheopathia osteoplastica. Saber-sheath configuration with characteristic submucosal osseocartilaginous nodules.* H, *Deformity occurring in tracheobronchomegaly, Mounier-Kuhn disease. The initially huge circular lumen may become distorted, as shown. The cartilage remains firm even when angulated.*

case, the rings are not malacic and indeed may even calcify. Rarely, obstruction follows if the deformity narrows the airway markedly. Another deformation in chronic obstructive pulmonary disease is anteroposterior flattening of the thoracic trachea, which is accompanied by softening of the rings (see Figure 1-2*F*). The cartilages may assume the configuration of an archer's bow. The membranous wall widens and becomes redundant. These changes in configuration and accompanying malacia result in various degrees of tracheal obstruction, notably in expiration and on cough (see Figure 1-2*F*). The upper trachea may become triangular in these patients (see Figure 1-2*F*). Unusual tracheal configurations occur in unique tracheal diseases such as tracheopathia osteoplastica (see Figure 1-2*G*) and tracheobronchomegaly (see Figure 1-2*H*). With advancing age or as a result of local trauma or disease, the tracheal rings and laryngeal cartilages may calcify.

The only complete cartilaginous ring in the normal airway is the cricoid cartilage. Congenital tracheal stenosis (see Chapter 6, "Congenital and Acquired Tracheal Lesions in Children") is characterized by segments of variable length composed of completely circular rings of cartilage or, rarely, by irregular circular plates. The cricoid level is the narrowest point in the upper airway below the glottis both in children and in adults. The cricoid has a broad posterior plate and is shaped much like a reversed signet ring (Figures 1-3*A*,*B*). The first ring of the trachea may be wider and partly recessed into the lower margin of the cricoid. The endoscopist must appreciate that the vocal cords lie approximately in midlarynx, just above the level of the anterior inferior margin of the thyroid cartilage, and that there is about 1.5 cm of subglottic larynx present between the vocal cords and the lower margin of cricoid (see Figures 1-3*A*–*D*). Only there does

the trachea actually begin. Endoscopists sometimes underestimate the involvement of subglottic larynx by a pathologic process, believing they are examining the trachea once they pass the vocal cords. The conus elasticus is a dome-shaped configuration just below the vocal cords (see Figure 1-3*D*). Laryngeal anatomy is briefly described in Chapter 35, "Laryngologic Problems Related to Tracheal Surgery."

Particularly in youth, the tracheal wall is quite elastic laterally. The normally thin cartilages in childhood are more easily compressed than those of the young adult, and these more easily than cartilages in the older adult. With normal cough, the membranous wall accordions, with the trachealis muscle pulling the lateral cartilaginous walls of the trachea medially (see Figure 1-2*B*). The intercartilaginous muscles between the rings and muscles in the membranous wall contract simultaneously. The tracheal walls may also be deformed by lateral pressure of the aortic arch (see Figure 1-2*D*), the brachiocephalic artery, and by extrinsic masses (see Chapter 15, "Tracheobronchial Malacia and Compression"). The trachea also bends flexibly but there is limited longitudinal extensibility, somewhat greater in the young. It is estimated to amount to about 10%. Flexibility and elasticity become more limited with advancing age, particularly when calcification occurs in cartilages.

The mucosa of the trachea is tightly applied to the inner surface of the cartilage. The two are not easily dissected apart. Mucosa covers the posterior muscular membranous wall as well. The mucosa is normally respiratory in character, being ciliated pseudostratified columnar epithelium. Goblet cells are liberally present. Submucosal mucous glands are numerous and connect to the surface by ducts. In habitual smokers and others with chronic irritation, squamous metaplasia may occur and the cilia are destroyed. Secretions may be successfully cleared by cough despite metaplasia and even when the mucosa has been totally replaced by either a cutaneous tube reconstruction or by interposition of synthetic materials.

Anatomic Relationships

The normal trachea viewed anteroposteriorly lies in the midline, connecting the larynx with the carina. In the lateral view, however, the trachea is tilted, slanting from an anterior, nearly subcutaneous position just below the larynx to a posterior one at the carinal level, where it lies against the esophagus close to the vertebrae. Although there is great individual variation, this angle from the vertical gradually increases with age

Table 1-1 Tracheal Dimensions in Children and Young Adults

					Internal Diameters		*Cross-sectional*			
Age (Years)	*Height Percentile*		*Tracheal Length (cm)*		*Anteroposterior (cm)*		*Transverse (cm)*	*Area (cm^2)*		*Volume (cm^3)*	
0–2	40		5.4		0.53		0.64	0.28		1.57	
2–4	47		6.4		0.74		0.81	0.48		3.11	
4–6	57		7.2		0.80		0.90	0.58		4.16	
6–8	54		8.2		0.92		0.93	0.69		5.67	
8–10	54		8.8		1.03		1.07	0.89		7.87	
10–12	49		10.0		1.16		1.18	1.10		11.1	
12–14	58		10.8		1.30		1.33	1.39		15.4	
	F	M	F	M	F	M	F	M	F	M	F	M
14–16	67	53	11.2	12.4	1.39	1.45	1.46	1.43	1.62	1.62	18.2	20.2
16–18	47	47	12.2	12.4	1.37	1.57	1.40	1.59	1.54	2.01	18.8	25.1
18–20	60	62	11.8	13.1	1.42	1.75	1.39	1.66	1.59	2.30	18.9	30.3

Data from computed tomography measurements in life, near total lung capacity if over 6 years.[4] "Tracheal length" is from vocal cords to carina. F = female; M = male.

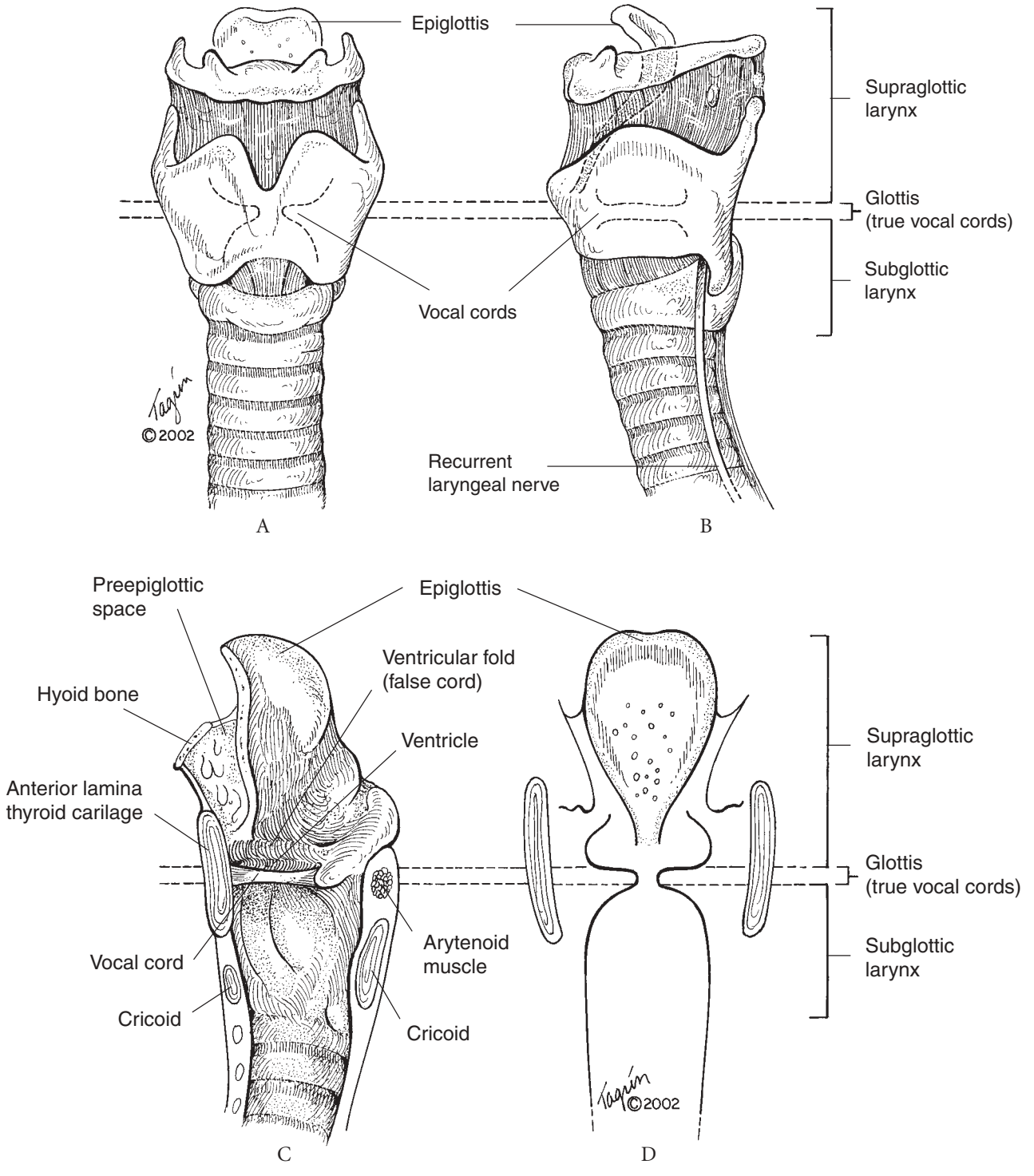


FIGURE **1-3** *External laryngeal relationships. Anterior* (A) *and lateral* (B) *views. Note the position of the true vocal cords (vocal folds) in the midlarynx. The cricoid cartilage shows the configuration of a reversed signet ring. The inferior cornu of the thyroid cartilage is close to the entry point of the inferior laryngeal nerve.* C, *Lateral view of the interior of the larynx. Anterior surface to the left. Note the relationships of the ventricular fold (false vocal cord), ventricle and vocal fold (true vocal cord), and their locations. The subglottic larynx lies between the glottis and inferior cricoid border.* D, *Diagram of interior configuration of larynx (anterior view). Note the dome-shaped airway beneath the glottis. This is the conus elasticus, shaped by intrinsic muscles. See Chapter 35, "Laryngologic Problems Related to Tracheal Surgery," for description of intrinsic laryngeal musculature.*

(Figure 1-4). In many old people, the trachea becomes increasingly horizontal in its course from the larynx to the carina and may approach a nearly transverse position. This worsens with severe kyphosis. The sternum also tends to flare out with aging. The larynx lies closer to the sternal notch with increasing age and the trachea loses mobility upon attempted cervical extension. This explains how subglottic damage may be occasioned by upward and backward erosion of a tracheostomy tube, even though the stoma was placed at a correct level in the trachea. In youth, a large proportion of the trachea presents in the neck above the level of the sternal notch even when the neck is in neutral position. With extension, more than half of the trachea rises into the neck, and sometimes, by as much as two-thirds (see Figure 1-4*A*). In contrast, attempted cervical extension in old age may bring very little, if any, trachea into the neck (see Figure 1-4*B*). The surgical implications are clear. The amount of trachea that can be brought into the neck on hyperextension of the cervical spine determines the percentage of trachea that may be resected and approximation obtained by cervical flexion alone.[8]

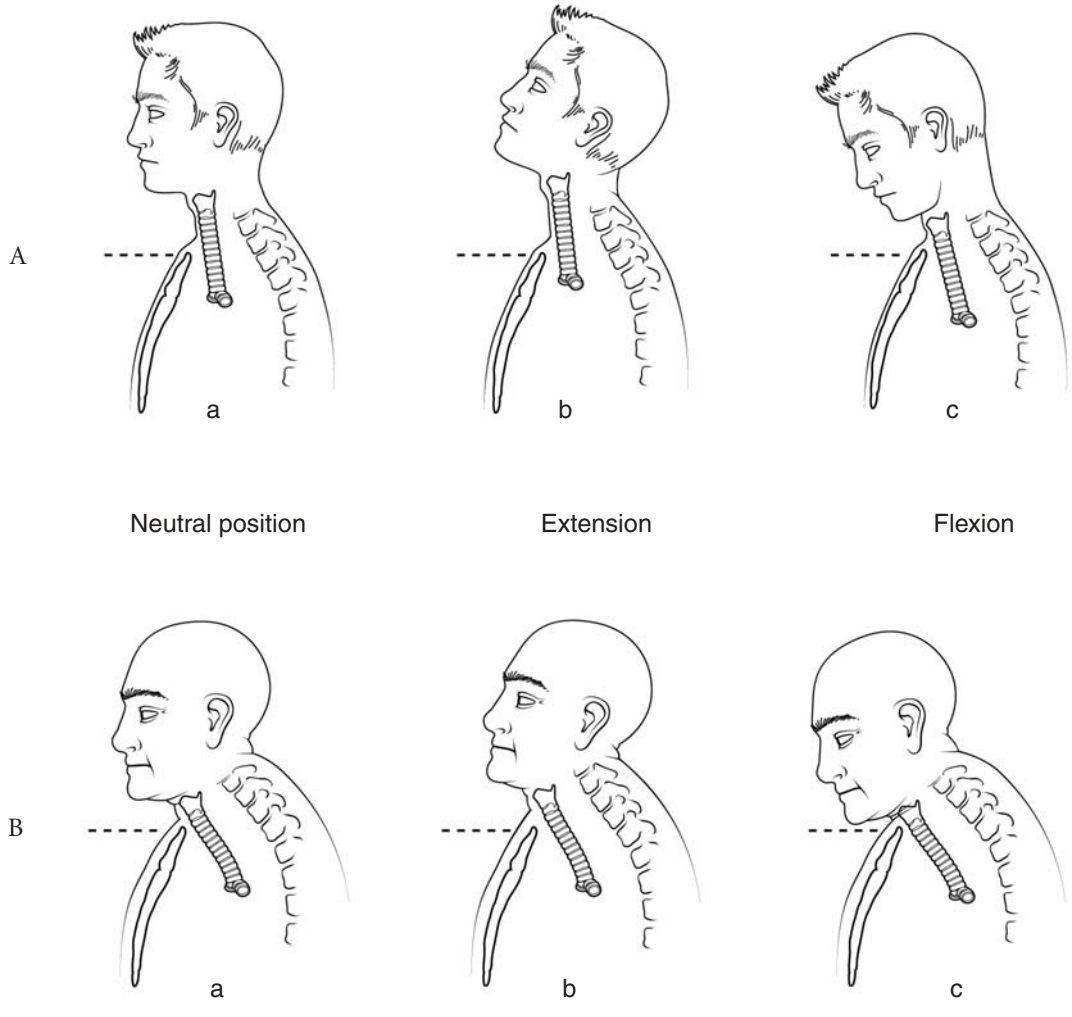


FIGURE **1-4** *Tracheal position in youth and old age with cervical extension and flexion. The trachea is much more vertical on lateral projection in youth* (A) *than in old age* (B). A, *(a) In youth, approximately one-third of the trachea is in the neck above the sternum* (dashed line) *in neutral position. (b) With cervical extension, one-half or more rises into the neck. (c) Most of the trachea devolves into the thorax on full flexion.* B, *In the aged, the level of the larynx (a) changes little with attempted cervical (b) extension and (c) flexion. Surgical implications are obvious.*

The *esophagus* lies in close relation to the trachea throughout its course (Figure 1-5). The esophagus commences at the level of the posterior cricoid, attached to it by the sling of cricopharyngeus muscle. Since the esophagus is a little to the left, the right posterior margin of the trachea is immediately in front of the vertebral bodies. In inflammatory disease, this portion of the posterior tracheal wall can adhere to the vertebral bodies. A layer of areolar tissue lies between the membranous wall of the trachea and the esophagus. This close juxtaposition of the walls of these two organs has been termed the "party wall." Normally, the plane is easily separable. A common blood supply, as noted below, is shared by these two tubular organs.

Anteriorly, the *thyroid isthmus* usually crosses and is closely applied to the trachea at the level of the second and third rings (see Figure 1-5*A*). The isthmus is sometimes very broad, but in a very few patients is absent. The pyramidal lobe commonly arises from the isthmus, often slightly to the left. The lateral lobes of the thyroid gland are also closely applied to the anterolateral and lateral walls of the trachea. Multiple small blood vessels, lymphatic channels, and fibrous attachments bind the isthmus and adjacent portions of the thyroid lobes to the tracheal wall.[9] The inferior thyroid artery supplies the lower portion of the thyroid gland and contributes importantly to the blood supply of the upper trachea. Details are provided below.

The *superior laryngeal nerves* concern the tracheal surgeon in connection with laryngeal release procedures and thyroidectomy. An *external* branch lies deep and parallel to the superior laryngeal artery and innervates the cricothyroid muscle. It gives a branch to the inferior pharyngeal constrictor. The *internal* branch passes into the thyrohyoid membrane with the superior laryngeal artery. It provides sensation to laryngeal mucosa and hence reflex protection to the larynx.[10]

The *recurrent laryngeal nerves* follow different courses right and left (see Figure 1-5). The left nerve originates from the vagus beneath the arch of the aorta and lies close to the tracheoesophageal groove along its entire course. The right nerve loops around the subclavian artery and therefore approaches the tracheoesophageal groove from a more lateral position. The right recurrent laryngeal nerve often passes between branches of the right inferior thyroid artery whereas the left often is posterior to the left inferior thyroid artery.[10] They enter the larynx between the cricoid and thyroid cartilages deep to the inferior cornua of the thyroid cartilage, behind the articulation of the thyroid and cricoid cartilages, to innervate the intrinsic laryngeal muscles.[10–12] Small branches travel to the trachea, trachealis muscle, esophagus, and inferior constrictors, including the cricopharyngeus muscle. Proximal branches near the recurrent nerve loops lying beneath the right subclavian artery and aorta on the left contribute to the cardiac plexus intrathoracically.[10]

Rarely, the right inferior laryngeal nerve is not recurrent but crosses the neck transversely from the vagus in one or more branches to enter the larynx. This occurs in conjunction with an anomalous right subclavian artery arising from a left aortic arch and passing posterior to the esophagus. The nonrecurrent nerve passes from the vagus beneath the carotid artery, may have two terminal branches, and may also give off branches to the trachea, esophagus, and thyroid. Even more rarely, the left inferior laryngeal nerve may be nonrecurrent in conjunction with the right aortic arch and left retroesophageal aberrant subclavian artery. Estimated incidence is 0.63% on the right and 0.04% on the left.[13]

The left *brachiocephalic vein* is well anterior to the pretracheal plane. The *brachiocephalic artery*, however, crosses over the midtrachea obliquely from its point of origin from the aortic arch to reach the right side of the neck (see Figures 1-5 through 1-7). In children, the artery rises higher and is encountered in the lower part of the extended neck. In young adults also, this artery crosses the trachea at the base of the neck with even moderate cervical extension. Thus in the young, a large proportion of the trachea and the brachiocephalic artery regularly rise into the neck on extension (see Figure 1-4). If a tracheostomy is placed in a child or young adult with reference to the sternal notch rather than the cricoid cartilage, it is easy to see how tracheal arterial fistula can occur (see Chapter 13, "Tracheal Fistula to Brachiocephalic Artery"). The brachiocephalic artery branches into the right common carotid and subclavian arteries a short distance to the right of the trachea and behind the origin of the internal jugular vein. The left common carotid nor-

mally arises a short distance from the origin of the brachiocephalic artery from the aorta forming a "V" slightly to the left of the tracheal midline. The brachiocephalic artery and the left common carotid artery may arise from a common arterial trunk, which overlies the trachea (see Figure 1-7).[14] Anatomic variation can be important if the trachea is adherent to the back of a common trunk (see Figure 1-7) as a result of inflammation or prior tracheal surgery. Occasionally (less than 10%), a small thyroidea ima artery arises from the back of the brachiocephalic artery and travels superiorly to the thyroid gland.

At the *carinal level*, the left main bronchus passes beneath the aortic arch and the right main bronchus beneath the azygos vein. The superior vena cava lies just anterior and to the right of the trachea. The pulmonary artery lies inferiorly in front of the carina (see Figures 1-5, 1-6). Thus, in anterior approach to the carina, a deep quadrilateral space is developed transpericardially in front of the carina, bordered by the

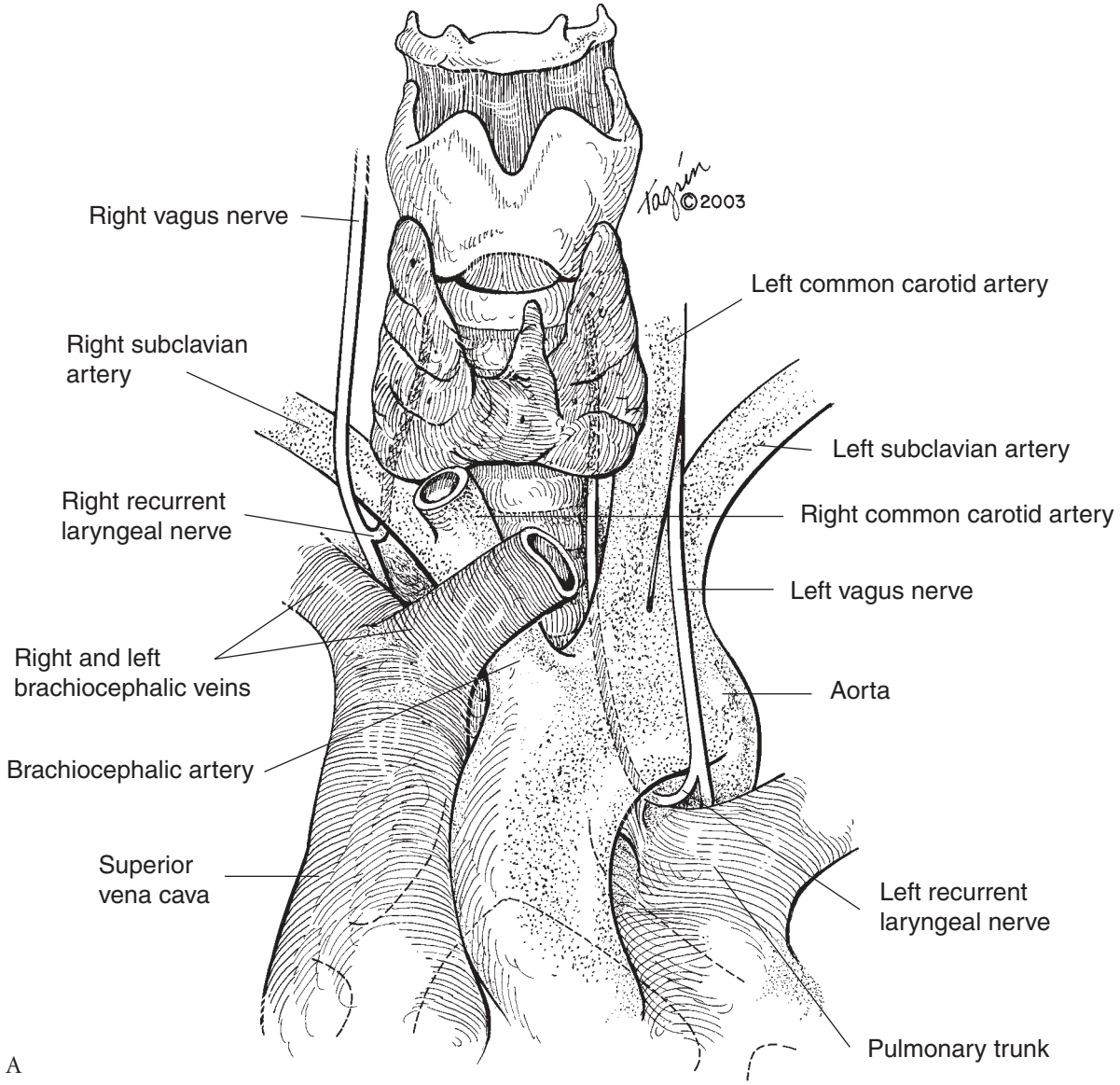
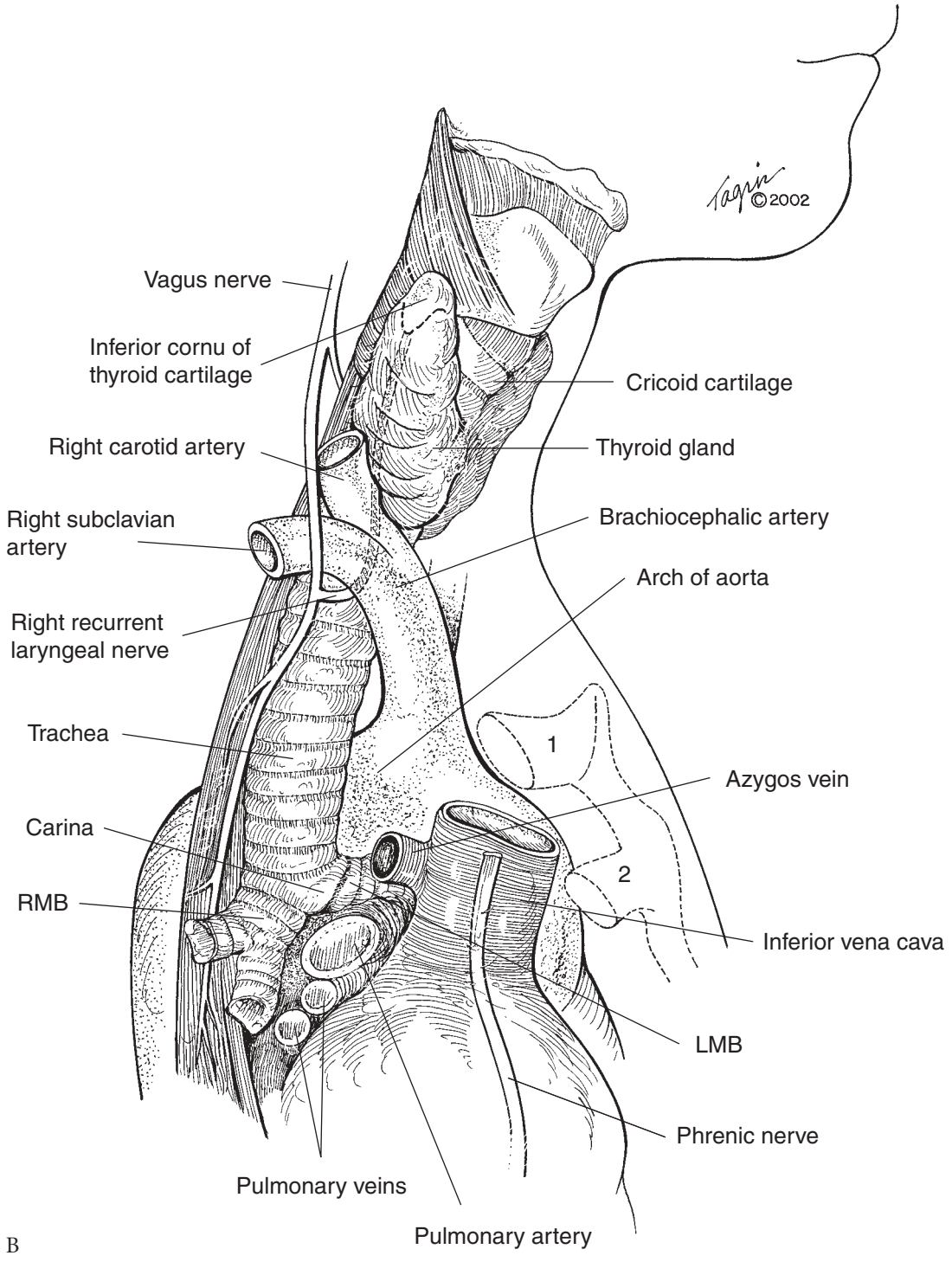


FIGURE 1-5 *Relationships of trachea to surrounding structures.* A, *Anterior view. Note the tight packing of major mediastinal vessels adjacent to the trachea.*



B

FIGURE 1-5 (CONTINUED) B, *Right-sided oblique view shows the complete access to the intrathoracic trachea, crossed only by the vagus nerve and azygos vein. LMB, RMB = left, right main bronchus.*

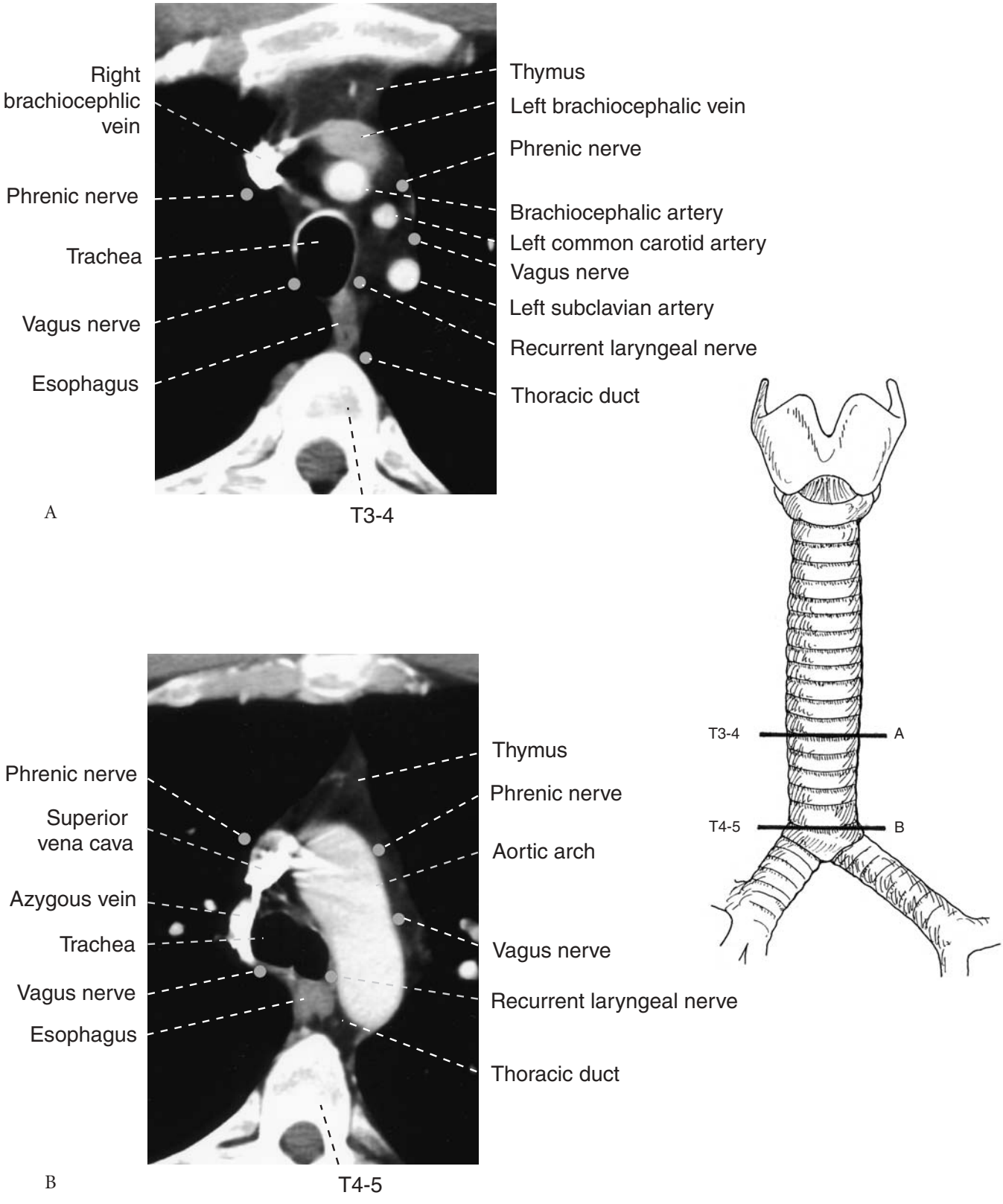


FIGURE **1-6** *Cross-sectional computed tomography views of tracheal anatomic relationships in the mediastinum. Diagram shows level of sections A at T3-4, and B at T4-5.* A, *Thoracic trachea.* B, *Supracarinal trachea. Mediastinal structures are labelled.*

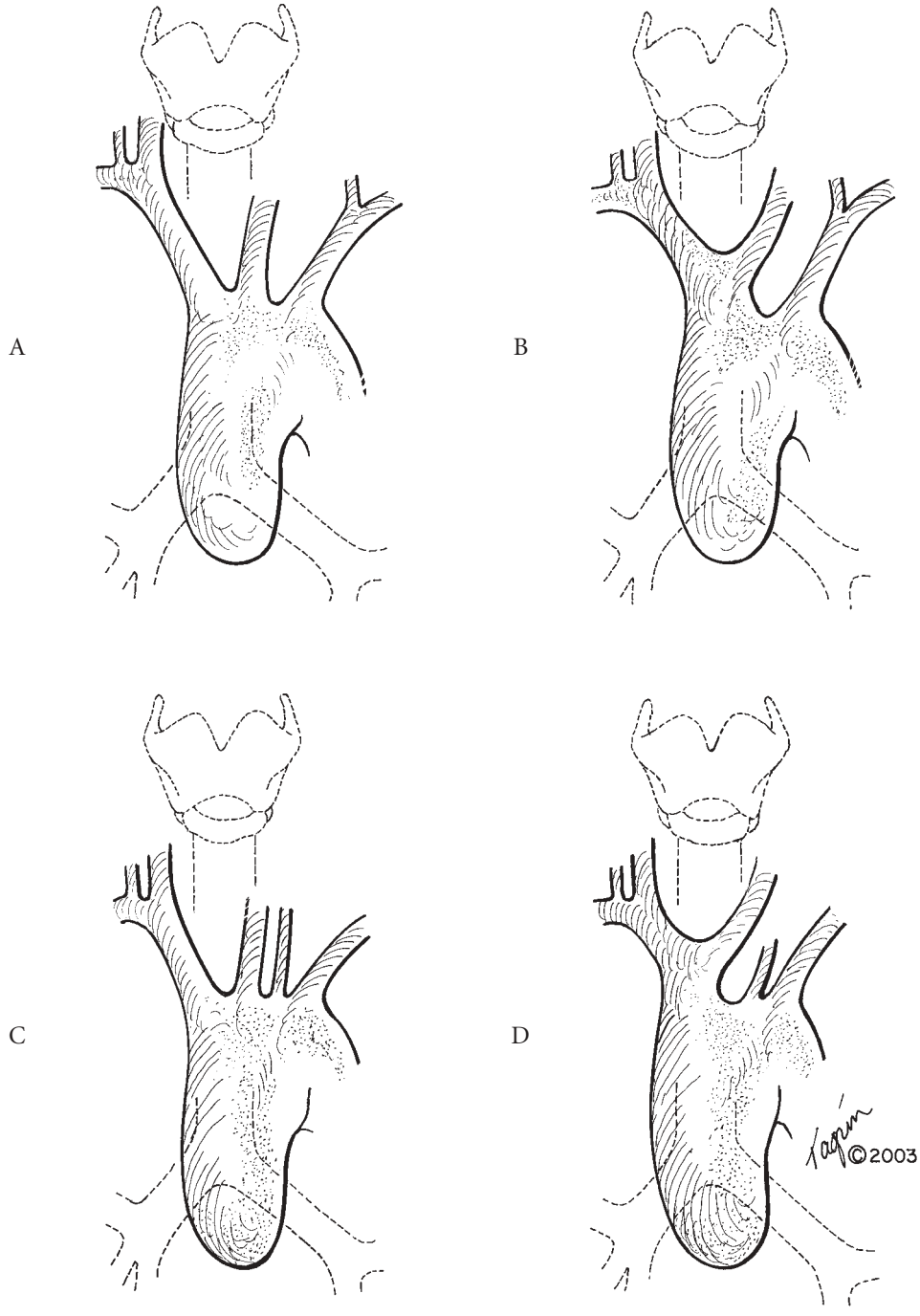


FIGURE 1-7 *Many variations occur in the arrangement of the branches arising from the aortic arch. The two most common patterns are* (A) *with separate origins of brachiocephalic and left carotid arteries, and* (B) *with a common origin. In both, a separate left vertebral artery may arise from the arch distal to the left carotid* (C,D) *and confuse the tracheal surgeon. The common origin may complicate resection of tracheal lesions that are adherent to the trunk or the treatment of tracheoarterial fistula. Adapted from Williams GD and Edmonds HW.*[14]

superior vena cava on the right, the aortic arch on the left, the pulmonary artery inferiorly, and the brachiocephalic vessels superiorly (see Figure 23-7 in Chapter 23, "Surgical Approaches"). The presence of major vascular structures in close proximity to the trachea makes exposure of its full extent difficult through any single incision. These anatomic facts must be considered in planning surgical approach to a tracheal lesion (see Chapter 23 "Surgical Approaches").

The previously undisturbed *pretracheal plane*, except for the point of attachment of the thyroid isthmus, may be easily developed bluntly because it consists of areolar tissue with few blood vessels. Normally, it is essentially avascular except for the rare thyroidea ima artery or an even rarer small posterior branch from the brachiocephalic artery to the trachea. A few inferior thyroid veins overlie the upper trachea immediately below the thyroid isthmus. These drain into the left brachiocephalic vein most commonly. The attachments of connective tissue to the trachea are loose enough so that vertical movement is easily possible to a considerable degree both functionally and surgically. The trachea is, however, fixed by the sling of the aortic arch over the left main bronchus where relatively little sliding motion occurs. With the increasing anteroposterior diameter of the thorax with age, related to vertebral kyphosis, this point of fixation draws the carina further posteriorly and the trachea falls into a more horizontal position when viewed laterally (see Figure 1-4*B*). Mobility of the trachea with cervical extension becomes limited, as previously noted.

Blood Supply

Prior to the development of tracheal surgery, detailed description of the arterial blood supply of the trachea was unknown. Using radiographs of injected specimens of the human trachea, Miura and Grillo showed that blood supply of the cervical trachea originates from the inferior thyroid artery in a variable pattern (Figure 1-8).[15] The blood supply enters the trachea through lateral tissue pedicles in segmental fashion throughout the trachea. Complete description of the entire tracheal blood supply was made by Salassa and colleagues (Figure 1-9).[16]

The upper half of the trachea is supplied in most cases by three tracheoesophageal branches of the *inferior thyroid artery* (see Figure 1-8*A*). The first branch supplies the lower cervical trachea with no or minor contributions to the esophagus. The second supplies the middle section of the cervical trachea, and the third the upper section. Both of these branches contribute to esophageal blood supply. The tracheal branches pass either anterior or posterior to the recurrent laryngeal nerves or both. The pattern varies, and there may be only one or two arteries. One or other artery may predominate (see Figure 1-8*B*). In 2 of 17 specimens studied, the lower cervical trachea was supplied instead by a branch originating from the *subclavian artery* (see Figures 1-8*A*d,*A*e). The superior thyroid artery does not give direct branches to the trachea, but it does anastomose with the inferior thyroid artery and also contributes with fine branches running from the thyroid isthmus to the adjacent tracheal wall.

The *bronchial arteries* provide consistent blood supply to the carina and lowermost trachea (see Figure 1-9).[16] An anterior branch of the superior bronchial artery originates from the right side of the aorta posteriorly. This branch usually travels over to the proximal left main bronchus to the anterior carina. The principal and posterior branches of this vessel pass behind the esophagus to the right main bronchus. One of these branches may arise from a supreme intercostal artery. The middle bronchial artery courses around the medial aspect of the left bronchus and anastomoses at the carina with the superior bronchial artery or higher tracheal vessels. The inferior bronchial artery appears to supply chiefly the left bronchial tree. The left main bronchus is most often supplied by two left-sided aortic branches. Bronchial artery patterns are very varied (Figure 1-10).[17] Flow in bronchial arteries after main bronchial artery division is given detailed consideration in Chapter 44, "Airway Management in Lung Transplantation." These considerations apply in carinal resection and reconstruction, although not as critically as in lung transplantation.

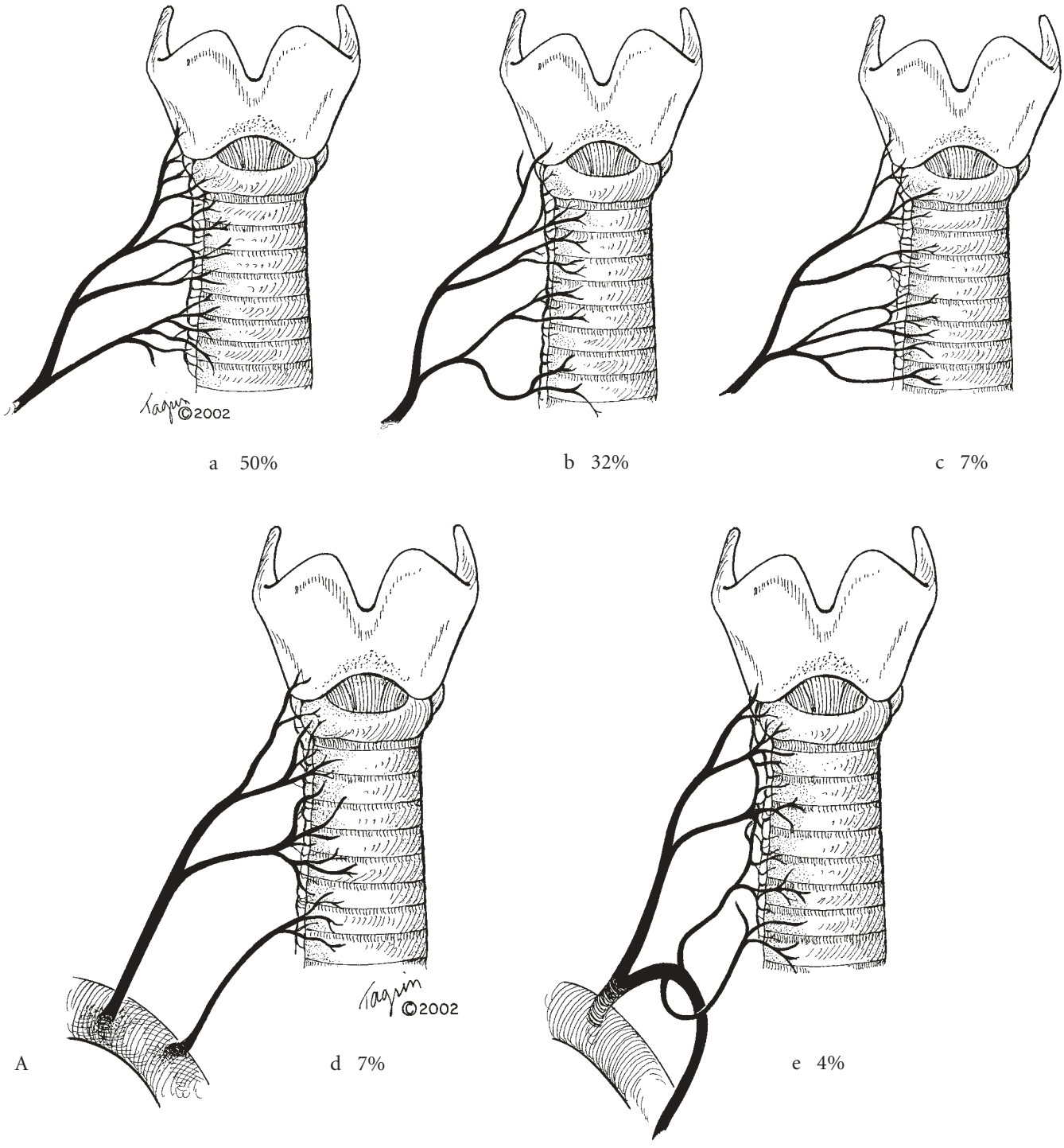
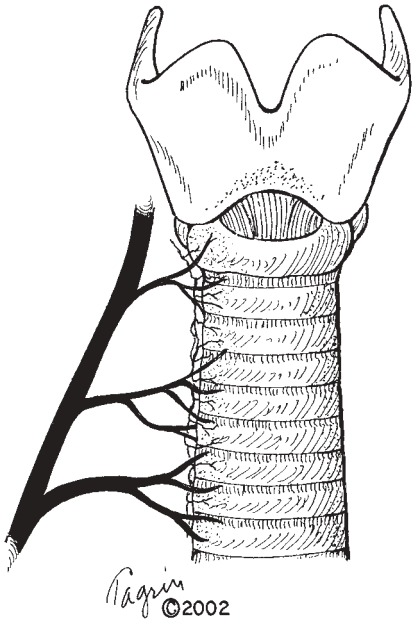


FIGURE **1-8** A, *Variants in distribution of inferior thyroid artery to cervical trachea. Distribution in 28 specimens. The frequency of each pattern is indicated. In (d) and (e), the major vessel is the subclavian artery.*



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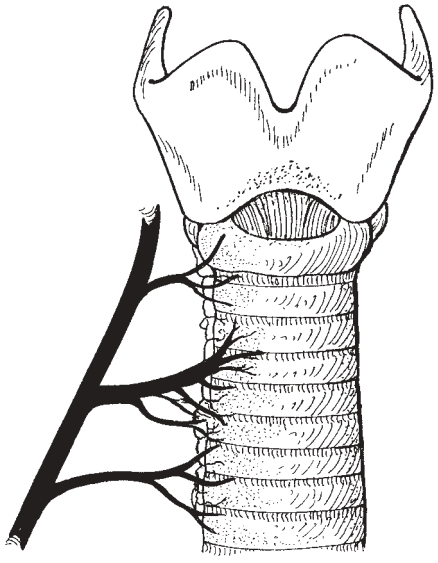

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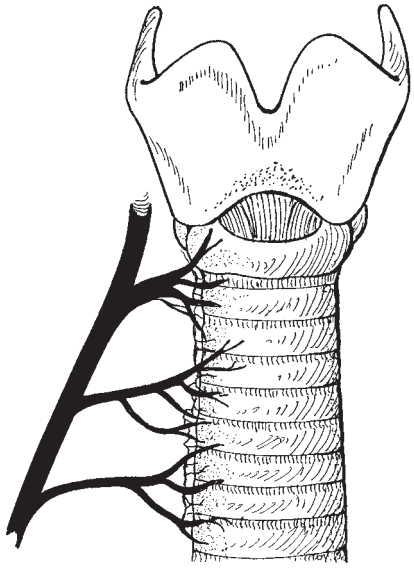

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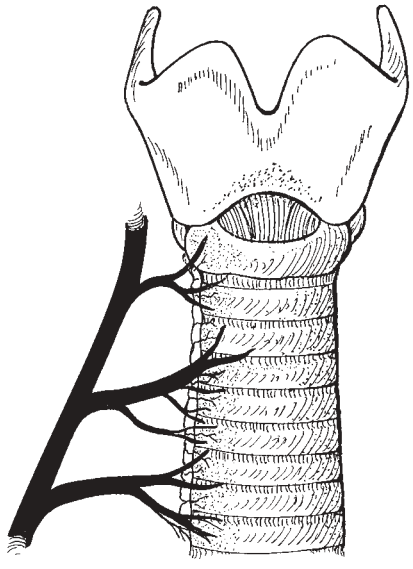

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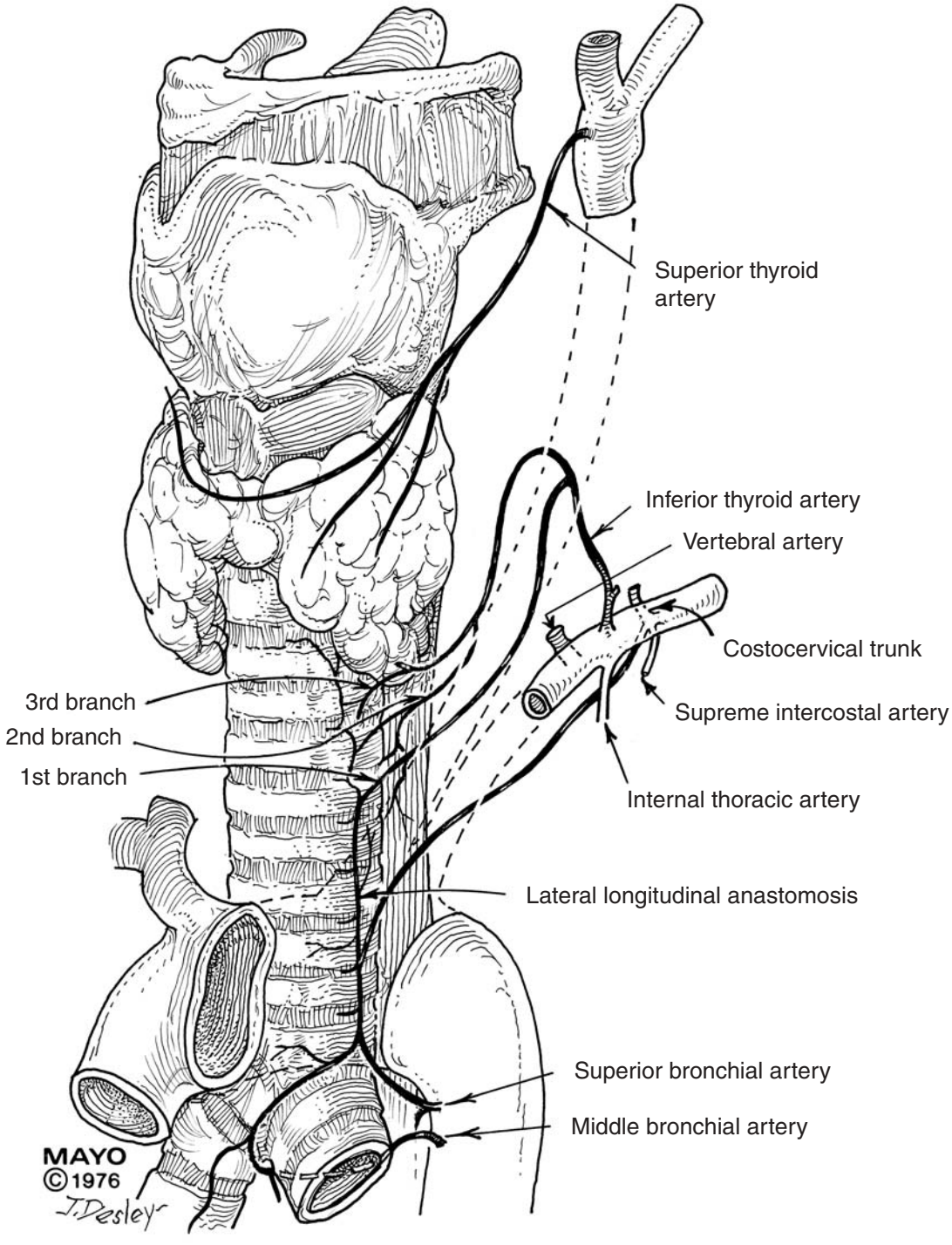
B

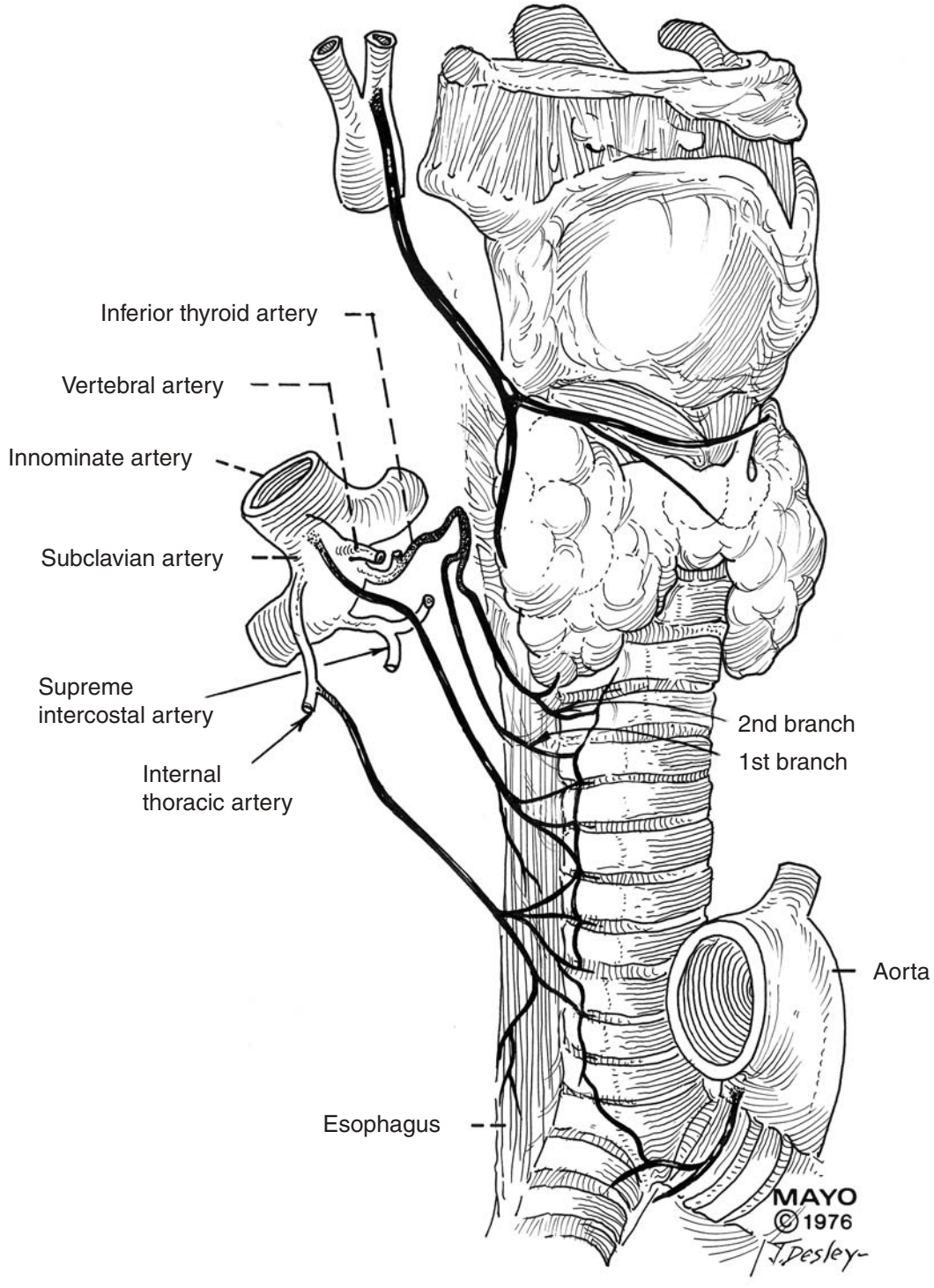
FIGURE 1-8 (CONTINUED) B, *Frequency of predominance of inferior thyroid artery branches. Adapted from Miura T and Grillo HC.*[15]



A

FIGURE 1-9 *Tracheal blood supply.* A, *Left anterior view. Reproduced, by permission of Mayo Foundation for Medical Education and Research, from Salassa JR et al.*[16]

The balance of the middle and lower tracheal blood supply is derived in variable fashion from a *brachiocephalic-subclavian system*: from the supreme intercostal artery, the subclavian artery, the right internal thoracic artery, and the brachiocephalic artery (see Figure 1-9).



B

FIGURE 1-9 (CONTINUED) B, *Right anterior view. Note the basically segmental nature of distribution. Reproduced, by permission of Mayo Foundation for Medical Education and Research, from Salassa JR et al.*[16]

Salassa and colleagues identified from three to seven principal tracheal arteries along the entire length of the lateral tissue pedicles.[16] Just lateral to the tracheoesophageal groove, the primary vessels divide into tracheal and esophageal branches (Figure 1-11). The tracheal branches pass directly to the tracheal wall,

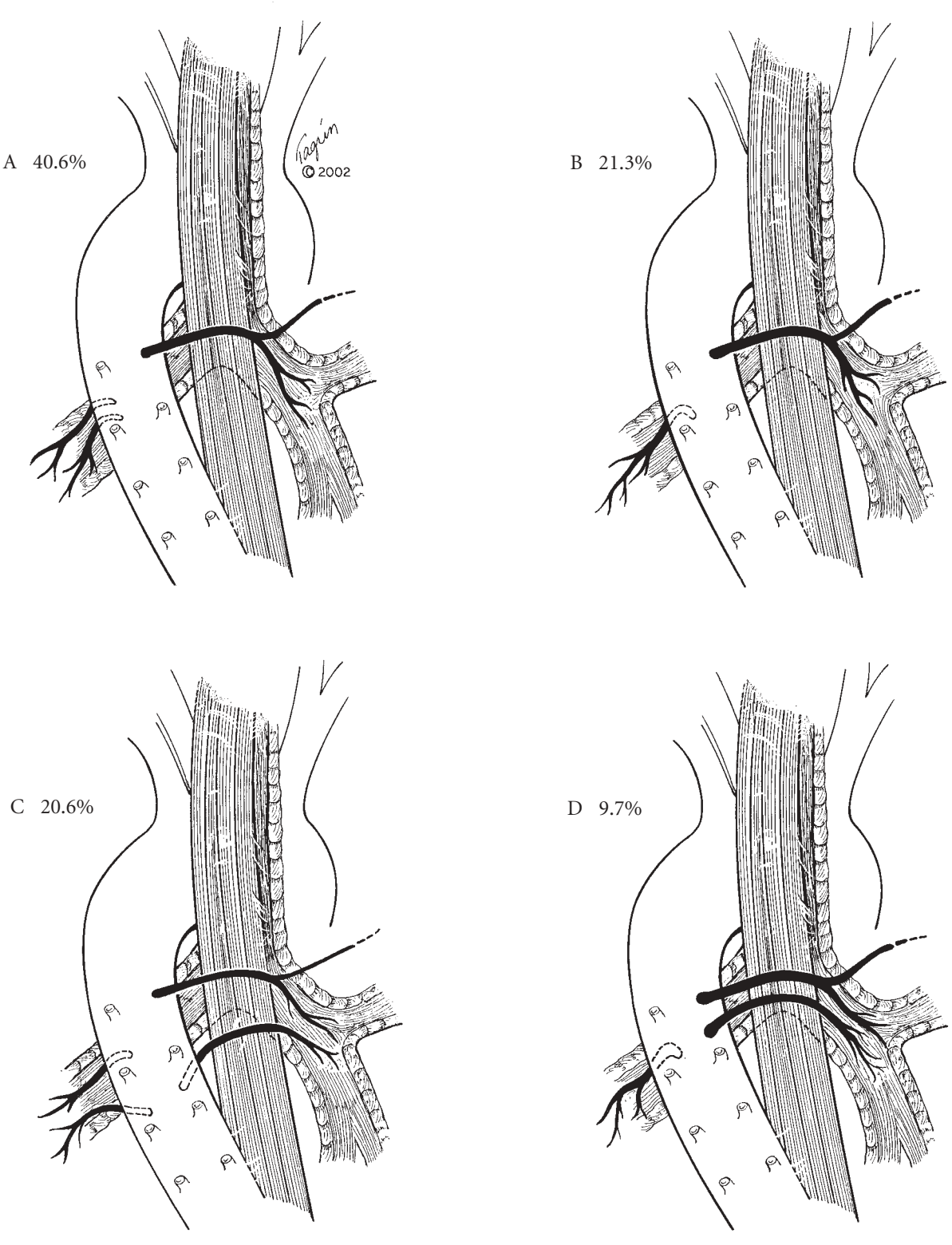


FIGURE 1-10 *Principal patterns of bronchial artery supply to the trachea and bronchi. Frequency of occurrence is noted. These patterns account for over 92% of variations. Not shown in these diagrams are the proximal branch of the superior bronchial artery which courses anteriorly over the left main bronchus to carina (see Figure 1-9A) and the middle bronchial branch passing beneath the left main bronchus to carinal anastomosis (see Figure 1-9B). Adapted from Cauldwell EW et al.*[17]

branching up and down over the width of several rings. These fine branches in turn connect with the branches of the next segmental vessels above and below. These vessels form a somewhat irregular but generally complete series of fine longitudinal anastomoses on the wall of the trachea.

From the vessels that reach the trachea, transverse intercartilaginous arteries extend deeply into the tracheal wall and anastomose with those from the opposite side at the midline (see Figure 1-11). These vessels branch into the submucosa. Smaller intercartilaginous branches point posteriorly and terminate in the membranous tracheal wall. The posterior membranous wall of the trachea is also supplied by secondary small branches from the primary esophageal vessels branching from the tracheoesophageal arteries. Well-developed longitudinal anastomoses are also present. The tracheal cartilages receive nourishment from the submucosal plexus only. The submucosal plexus of both the mucosa overlying the cartilages and that overlying the membranous wall interconnect and are important in supplying the membranous wall.

Although a large part of the length of the trachea can usually be circumferentially dissected without necrosis if the trachea remains intact (and with it the vertical longitudinal vessels), circumferential dissec-

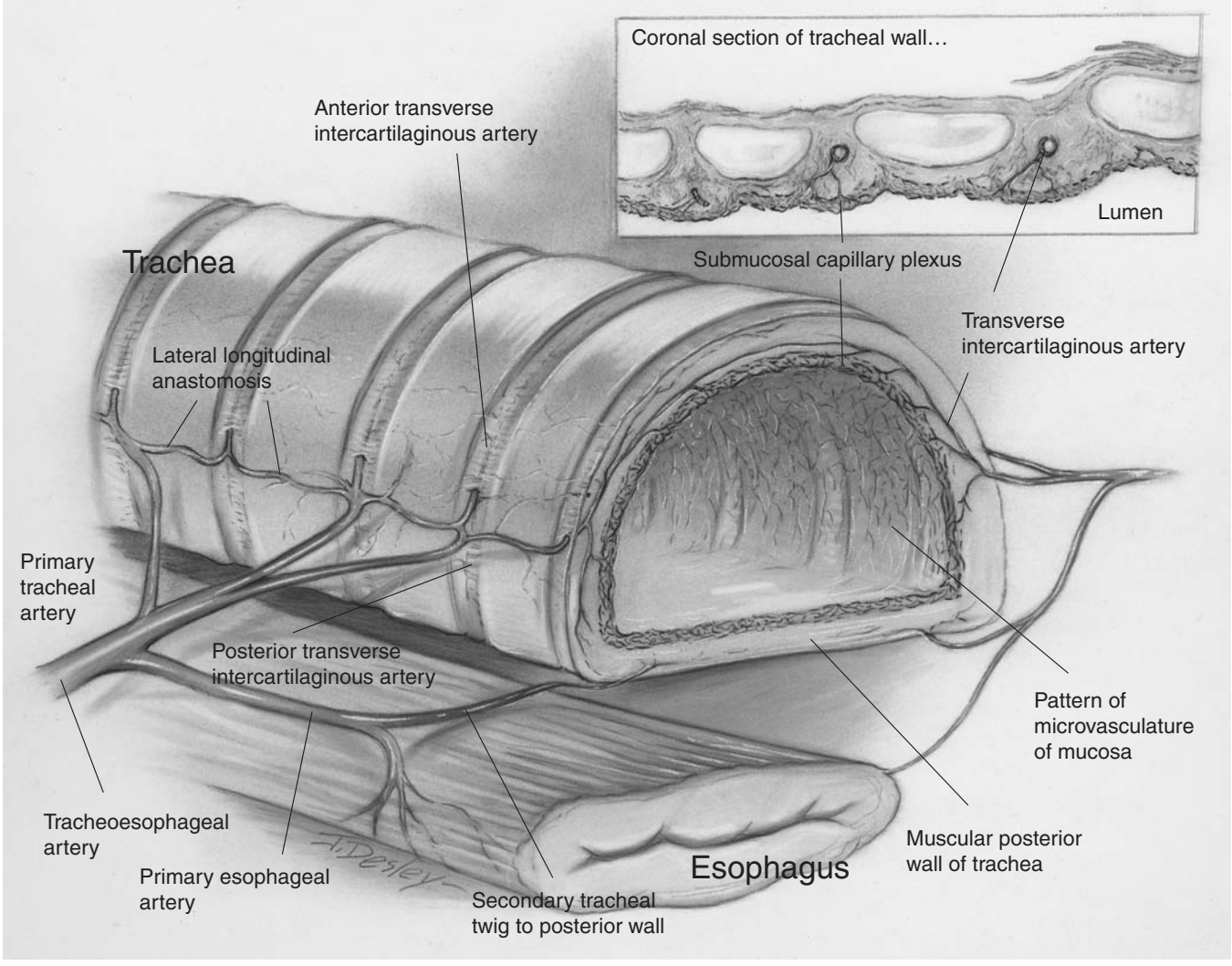


FIGURE **1-11** *Microscopic blood supply of the trachea. See text. Reproduced, by permission of Mayo Foundation for Medical Education and Research, from Salassa JR et al.*[16]

tion of an excessively long segment of trachea above or below a point of *tracheal division* can lead to devascularization. Necrosis may follow. No absolute distances acceptable for toleration of circumferential dissection have been experimentally determined since the intimate blood supply of the trachea varies with species. Clinical experience, however, dictates the wisdom of minimizing circumferential dissection of trachea that is to remain in the patient, with a goal of dissecting free no more than 1 or 2 cm of trachea above or below an anastomotic line. Mediastinal lymph node dissection for tracheal tumors should be limited to lymph nodes immediately adjacent to the segment to be resected, in order to avoid contributing to devascularization. Surgical caution applies to the very rare situation where circumferential tracheal resection may seem to be desirable during concomitant esophagectomy. Tracheal necrosis may well follow because the tracheoesophageal arteries are interrupted by esophagectomy.[18]

Lymphatics

Detailed studies of tracheal lymphatic drainage are few. Following submucosal injections of India ink and dye into the canine trachea, Strauss observed that

1) tracheal lymphatic vessels were present as fine intercellular spaces beneath the mucous membrane over the cartilages,
2) the lymph flowed up or down the trachea to the nearest interspace between cartilages,
3) one to three trunks flowed horizontally in the interspaces between rings,
4) flow from the anterior wall went to either side,
5) flow from the lateral wall passed to the membranous wall,
6) the membranous wall contained larger vessels in greater numbers and particles traveled up and down the wall,
7) there were more horizontal collecting vessels in the lower interspaces, especially near the carina, and
8) lymph vessels left the tracheal wall, especially at the lower end of the trachea, and passed to perivascular lymphatics and then to lymph nodes along the trachea.[19]

Primary tracheal lymph nodes are pretracheal, paratracheal, and subcarinal.[20] The anatomy of the mediastinal lymph nodes has been well described in connection with lung cancer in many places.[21]

Pathways of lymph drainage from nodes along the trachea have been elucidated somewhat by Ricquet and colleagues.[22] The right lower paratracheal lymph nodes drain into thoracic duct tributaries which travel along the course of the azygos vein. Left superior bronchial nodes below the trachea drain directly to the mediastinal thoracic duct or to the arch of the duct via the left recurrent chain. An alternative pathway is to the aortic arch node and up along the arch. Tracheal bifurcation nodes drain through accessory ducts on either side of the esophagus to the mediastinal thoracic duct.

Clinical observations of peritracheal, pretracheal, and subcarinal nodal metastases, principally from primary squamous and adenoid cystic carcinomas of the trachea, not surprisingly show that cells reach the nodes most often nearest to the tumor. I have not often seen obvious "skip" nodal involvement. Although limited node dissection has been practiced in order to preserve tracheal blood supply, recurrence in most cases has been distant rather than local. It must, however, be noted that mediastinal irradiation has been given quite routinely postoperatively. These are admittedly anecdotal observations. A limited number of intratracheal dye injections in vivo by Miura and Grillo (unpublished, 1965) confirmed tracking to the nearest paratracheal mediastinal lymph nodes. I believe that primary tracheal tumors with mediastinal lymph node involvement should be thought of as N1 disease.

References

1. Grillo HC, Dignan EF, Miura T. Extensive resection and reconstruction of mediastinal trachea without prosthesis or graft: an anatomical study in man. J Thorac Cardiovasc Surg 1964;48:741–9.
2. Wailoo MP, Emery JL. Normal growth and development of the trachea. Thorax 1982;37:584–7.
3. Griscom NT, Wohl MEB. Dimensions of the growing trachea related to body height. Am Rev Respir Dis 1985;131:840–4.
4. Griscom NT, Wohl MEB. Dimensions of the growing trachea related to age and gender. Am J Roentgenol 1986;146:233–7.
5. Griscom NT, Wohl MEB, Fenton T. Dimensions of the trachea to age 6 years related to height. Pediatr Pulmonol 1989;6:186–90.
6. Butz RO. Length and cross section growth pattern in the human trachea. Pediatrics 1968;42:336–41.
7. Greene RE, Lechner GL. "Saber-sheath" trachea. A clinical and functional study of marked coronal narrowing of the intrathoracic trachea. Radiologe 1975;115:265–8.
8. Mulliken J, Grillo HC. The limits of tracheal resection with primary anastomosis: further anatomical studies in man. J Thorac Cardiovasc Surg 1968;55:418–21.
9. Shin DH, Mark EJ, Suen HC, Grillo HC. Pathological staging of papillary carcinoma of the thyroid with airway invasion based upon the anatomic manner of extension to the trachea. Hum Pathol 1993; 24:866–70.
10. Monfared A, Corti G, Kim D. Microsurgical anatomy of the laryngeal nerves as related to thyroid surgery. Laryngoscope 2002;112:386–92.
11. Wang CA. The use of inferior cornu of the thyroid cartilage in identifying the recurrent laryngeal nerve. Surg Gynecol Obstet 1975;140:91–4.
12. Pearson FG, Cooper JD, Nelems JM, Van Nostrand AWP. Primary tracheal anastomosis after resection of the cricoid cartilage with preservation of recurrent laryngeal nerves. J Thorac Cardiovasc Surg 1975;70:806–16.
13. Henry J-F, Audiffret J, Denizot A, Plan M. The nonrecurrent inferior laryngeal nerve: review of 33 cases, including two on the left side. Surgery 1988; 104:977–84.
14. Williams GD, Edmonds HW. Variations in the arrangement of the branches arising from the aortic arch in American whites and negroes (a second study). Anat Rec 1935;62:139–46.
15. Miura T, Grillo HC. The contribution of the inferior thyroid artery to the blood supply of the human trachea. Surg Gynecol Obstet 1966;123:99–102.
16. Salassa JR, Pearson BW, Payne WS. Gross and microscopical blood supply of the trachea. Ann Thorac Surg 1977;24:100–7.
17. Cauldwell EW, Sickert RG, Lininger RS, Anson BJ. The bronchial arteries. An anatomic study of 150 human cadavers. Surg Gynecol Obstet 1948; 86:395–412.
18. Fujita H, Kawahara H, Hidaka M, et al. An experimental study on viability of the devascularized trachea. Jpn J Surg 1988;18:77–83.
19. Strauss JF. The intimate lymphatics of the trachea. Ann Otol Rhinol Laryngol 1922;31:715–26.
20. Rouvière H. Anatomy of the human lymphatic system. Tobian MJ, translator. Ann Arbor (MI): Edwards Bros; 1938.
21. Shields TW. Lymphatics of the lungs. In: Shields TW, LoCicero J, Ponn RB, editors. General thoracic surgery. 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2000. p. 77–90.
22. Ricquet M, Le Pimpec Barthes F, Souilamas R, Hidden G. Thoracic duct tributaries from intrathoracic organs. Ann Thorac Surg 2002;73:892–9.

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Physiology of the Trachea

David J. Kanarek, MD

Anatomy

Some knowledge of tracheal anatomy is relevant to appreciating the physiological characteristics of the trachea.

The trachea is a tube of approximately 11 cm in length and 1.6 to 2.4 cm in width. It is composed of C-shaped cartilage rings, spanned by the trachealis muscle, which provides support anteriorly and laterally, and a posterior membrane that stretches and can become partially redundant when exposed to high extrapleural pressures. The distal two-thirds of the trachea is intrathoracic, whereas the proximal third is extrathoracic when the neck is extended and intrathoracic with the neck flexed. Thus, there is a zone of the trachea that can be exposed to either intrapleural or extrapleural pressure.

Flow Volume Loop

The flow volume loop (FVL) has become the standard method for assessing the volume and flow characteristics of the lung, as well as for physiologic evaluation of the upper airway (Figure 2-1). The total volume exhaled, the forced vital capacity (FVC), is plotted against the simultaneously obtained flow rate (see Figure 2-1*A*). Flow is obtained either directly from a pneumotachograph or by the derivative of volume against time (flow). Spirometry displays volume against time, and although it yields similar information on the expiratory curve, it has less of a pictorial element. The inspiratory portion of the FVL is important in diagnosing nonfixed or variable forms of upper airway obstruction, and it is not represented in spirometric tracings.

Understanding the influence of pressure changes with phases of respiration is essential in following the methods of diagnosis of upper airway obstruction. The key factor is the transluminal pressure, which is the relationship between intraluminal and extraluminal pressures. The extraluminal pressure is atmospheric pressure in the extrathoracic region and intrapleural pressure in the intrathoracic area. With inspiration, the diaphragm and chest wall muscles expand the thoracic cavity, decompressing the intrathoracic gases. This causes a negative pressure within the pleura that is greater than the negative intraluminal pressure, thus dilating the intrathoracic airways. The positive extrathoracic extraluminal pressure does not col-

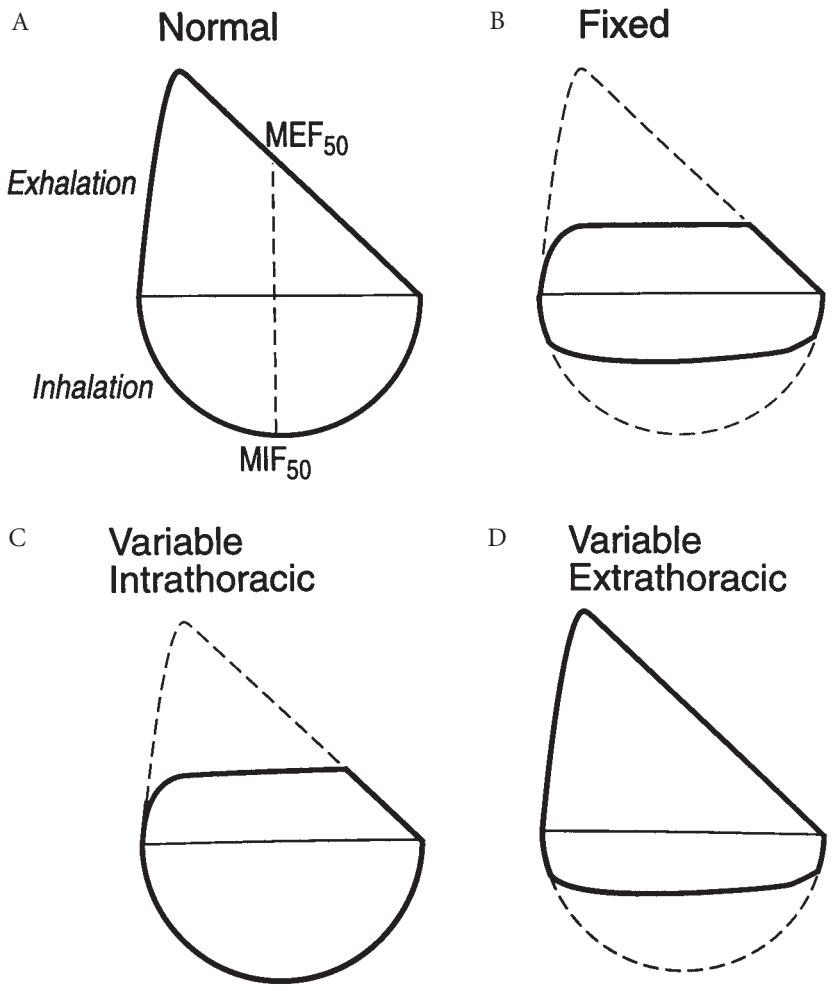

FIGURE 2-1 *Diagrammatic representation of a normal flow volume loop* (A) *and different types of upper airway obstruction* (B–D). *Flow is the vertical axis and volume is the horizontal axis.* MEF_{50} *and* MIF_{50} *are the maximum expiratory and inspiratory flows, respectively, at 50% vital capacity.*

lapse the extrathoracic trachea because of the rigidity of the trachea and stiffening of pharyngeal muscles by neurological influences. During unforced exhalation, the intraluminal airway pressure becomes positive relative to both the intrapleural and extrathoracic atmospheric pressures. With forced exhalation, however, the intrapleural pressure may exceed the intraluminal pressure and cause dynamic compression of the airway. This becomes important in interpreting the FVL, and in diagnosing the site and nature of an obstruction.

The normal expiratory FVL has two components (see Figure 2-1*A*). The first part is approximately the initial 20% of the FVC, comprised of the rise and the initial descent of flow rate, and is effort dependent. The remainder is effort independent, meaning that, above a moderate effort, no increase in flow is obtained by increasing muscular effort. This is related to the phenomenon of dynamic compression, in which high intrapleural pressure, caused by contraction of the diaphragm and intercostal muscles, is added to the elastic recoil of the distended alveoli to compose the intra-alveolar driving pressure. However, the same intrapleural pressure also compresses the airways, leaving alveolar elastic recoil as the sole driving pressure, irrespective of the degree of muscular effort. At high lung volume, airflow increases with effort because high intrapleural pressures cannot be developed and because elastic recoil is high. Furthermore,

the so-called choke point, the point at which intraluminal pressures equal extraluminal pressures, is in the trachea, and the more proximal trachea with its cartilaginous support is resistant to collapse. This choke point moves more distally with decreasing volume to a point where the airways are susceptible to pressure and collapse.

The expiratory portion of the FVL of emphysema (Figure 2-2) has a recognizably different shape from a normal FVL. The resistance of the small airways is increased by intrinsic disease and by the loss of the tethering effect of alveoli on the airways. In addition, there is loss of alveolar elastic recoil as a result of alveolar destruction. Thus, at every lung volume, flow rates are lower than normal. In addition, the expiratory loop becomes concave downwards. The maximum flow of the inspiratory loop may be almost normal because the laxity of the airways allows them to be easily opened by negative intrapleural pressure, or the flow may be reduced when intrinsic inflammatory disease stiffens airways, producing increased resistance to flow.

Proper performance of the FVL is extremely important in interpretation (Figure 2-3). A poor initial expiratory effort may produce an apparent plateau on the expiratory loop and simulate a variable intrathoracic obstruction such as tracheomalacia. This often presents as a rounding rather than a plateau-like flat-

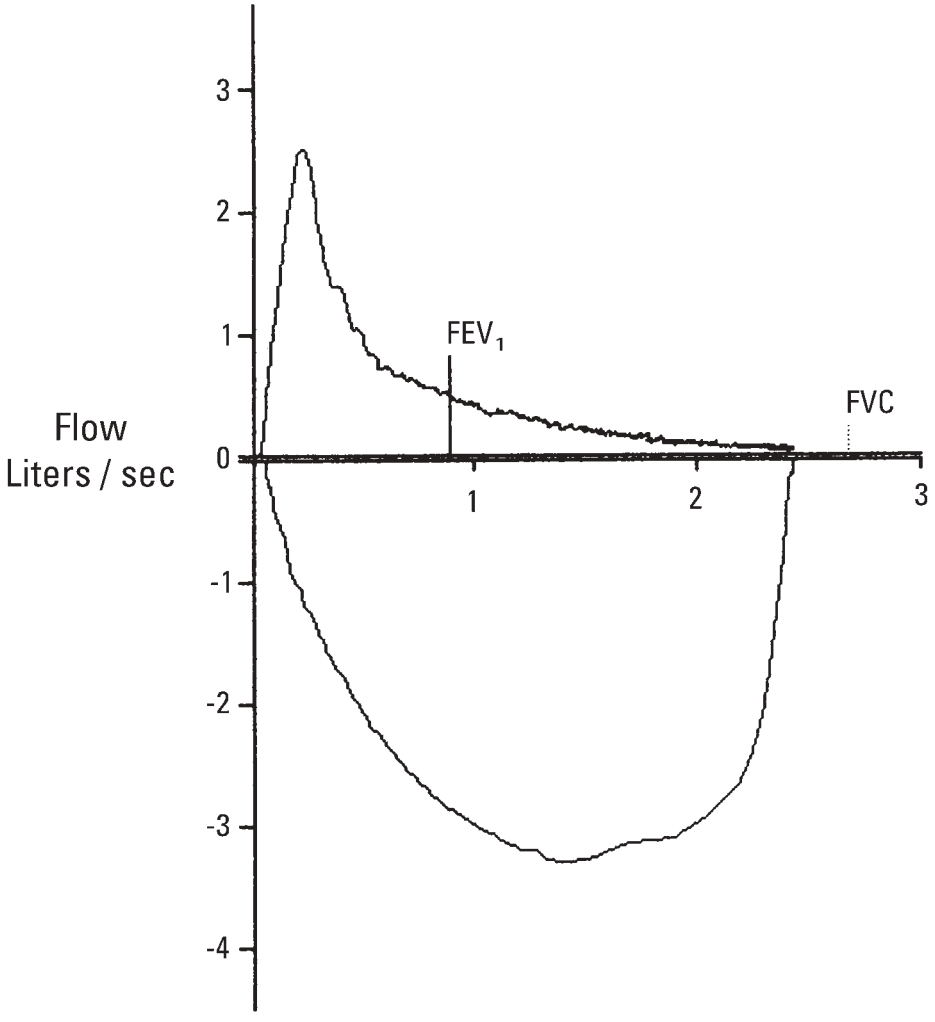

FIGURE 2-2 *The flow volume loop in chronic obstructive pulmonary disease (emphysema). Flow in the vertical axis and volume is the horizontal axis. FEV_1 is the forced expiratory volume in the first second of time. FVC is the forced vital capacity.*

tening of the expiratory curve, but it can be distinguished at times by a lack of reproducibility of successive curves. The report of the technician administering the test is perhaps the most helpful factor in distinguishing poor effort from disease, in many cases.

Another frequent error in interpretation of expiratory curves is the presence of a knee (see Figure 2-3) high up on the curve, close to total lung capacity (TLC). This can be corrected by repeating the study while hyperextending the neck. The explanation is thought to be due to stiffening of the trachea and movement of the equal pressure point. In some younger patients, a "shoulder" can be seen further down the expiratory curve, where flows that were being maintained during the early part of the curve decrease suddenly and more steeply. In both these variants, however, a peak is visible. In healthy subjects, where these variants are usually seen, the peak flow rate is in the normal range, whereas with true tracheal disease, the rate is reduced.

The inspiratory portion, in contradistinction to the expiratory portion of the FVL, is entirely effort dependent. Whereas the effort independent portion of the FVL is a function of elastic recoil of the lung as the driving force, the inspiratory portion is entirely driven by the force generated by the inspiratory muscles. Interpretation of this portion of the loop can be very difficult because many subjects without upper airway obstruction have difficulty in making a sustained forceful inhalation and technicians may not compel full efforts. For example, variable extrathoracic airway obstruction may be overdiagnosed, based solely on interpretation of the inspiratory portions of the loop.

Upper Airway Obstruction

The presence of obstruction to airflow in the trachea can be evaluated by inspection of the FVL. Traditionally, this has been divided into fixed obstruction and mobile obstruction, and the latter subdivided into extrathoracic or intrathoracic obstruction. The term mobile is related to fluctuation of the diameter of the affected region under the influence of intraluminal or extraluminal pressure. In turn, these pressures are dependent on whether flow is occurring during inspiration or expiration.

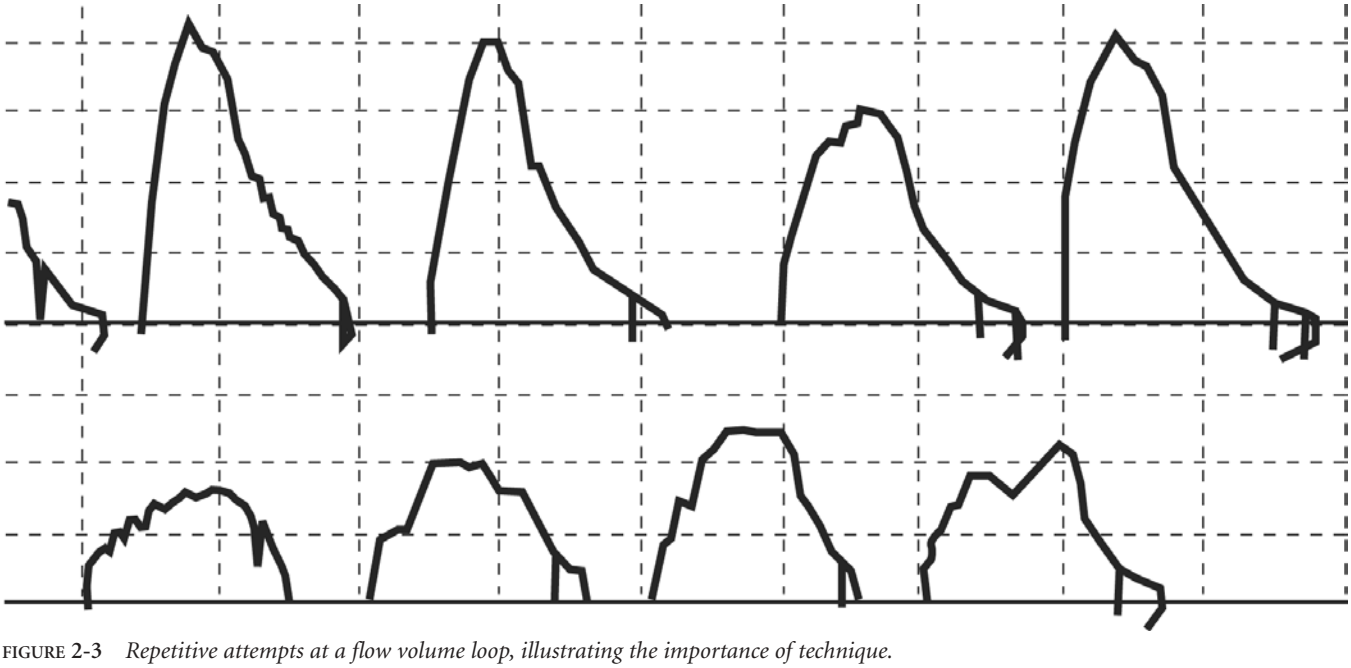

FIGURE 2-3 *Repetitive attempts at a flow volume loop, illustrating the importance of technique.*

Fixed Upper Airway Obstruction

In fixed upper airway obstruction, both the inspiratory and expiratory loops demonstrate a plateau (see Figure 2-1*B*). Miller and Hyatt carried out experiments, in which forced exhalation and inspiration was performed through a series of progressively narrower tubes (Figure 2-4).[1,2] These studies demonstrated that flattening of both inspiratory and expiratory curves occurred to an increasing degree with narrowing of the orifices. The features on the expiratory curve are first visible at about a 1-cm tracheal diameter, implying that there is a lack of sensitivity with mild narrowing of the trachea. However, once flow limitation begins, the reduction in flow rate is very rapid, with the peak flow rate falling from about 90% predicted at a 1-cm tracheal diameter to 25% at a 5-mm diameter. Both the length of the plateau and the degree of peak flow reduction are proportional to the degree of obstruction.

Miller and Hyatt also evaluated the sensitivity of other standard pulmonary function tests in the diagnosis of tracheal obstruction (Figure 2-5).[1,2] The peak expiratory flow rate was the most sensitive test, followed by the maximum voluntary ventilation. The forced expiratory volume in the first second (FEV_1) does not show a recognizable fall outside of the normal range until at an approximately 6-mm tracheal diameter. The peak inspiratory flow rate is the most sensitive test for detecting inspiratory flow limitations. The ratio of maximum inspiratory flow (MIF) to maximum expiratory flow (MEF) at 50% vital capacity (MIF_{50}/MEF_{50}) remains about 1.0, since both parts of the FVL are altered to about the same degree.

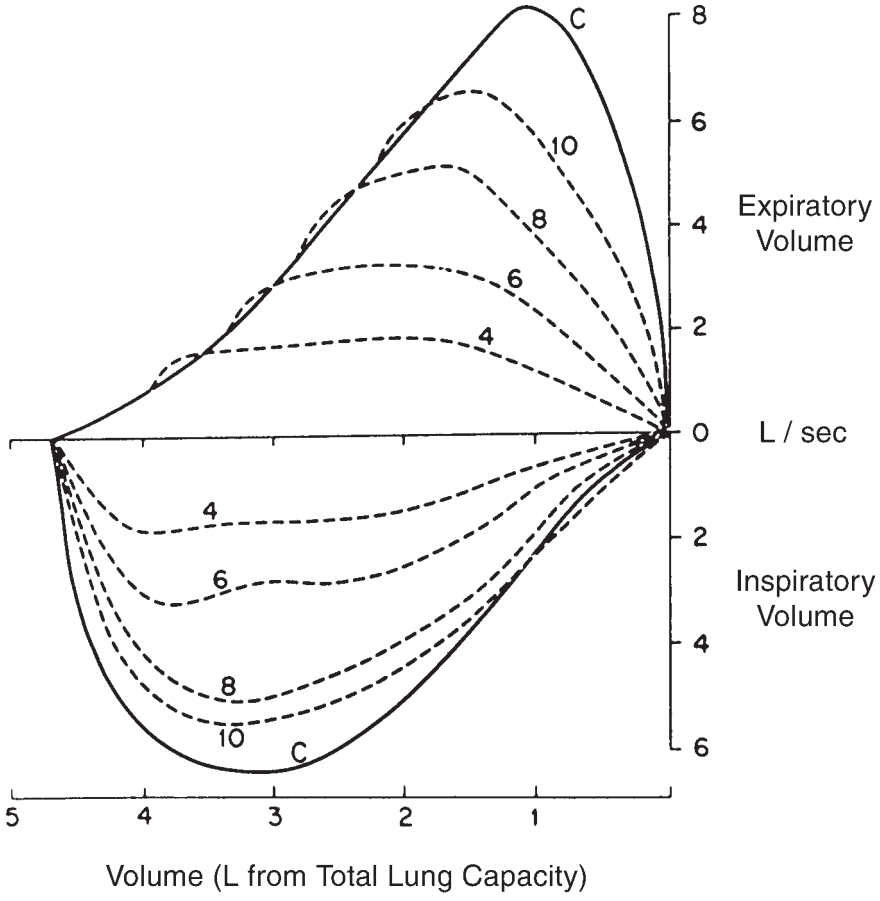

FIGURE **2-4** *Flow volume loops obtained from breathing through progressively smaller orifices. The solid line is the unobstructed loop. C = control. Reproduced with permission from Miller RD and Hyatt RE.*[2]

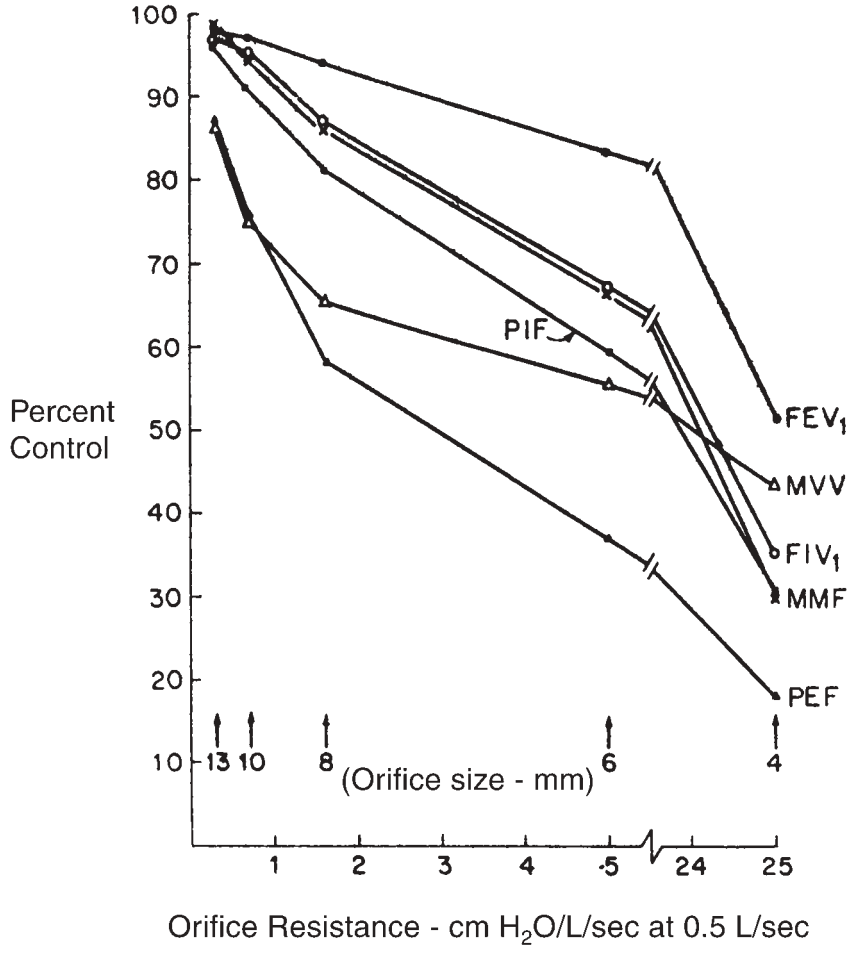

FIGURE 2-5 *Inspiratory and expiratory flows obtained through progressively narrower external tubes. FEV_1 = forced expiratory volume; FIV_1 = forced inspiratory volume; MMF = maximum mid-expiratory flow; MVV = maximum voluntary ventilation; PEF = peak expiratory volume; PIF = peak inspiratory volume. Reproduced with permission from Miller RD and Hyatt RE.*[2]

The most common cause of fixed stenosis is postintubation stenosis. The lesion is circumferential and usually related to mucosal and cartilage damage at the site of the cuff. Other causes are neoplasms, goiters, and stenosis of both main bronchi. Flow volume loops performed though a tracheostomy tube also show the same pattern. Gamsu and colleagues (Figure 2-6) derived a graphic representation of flow related to driving pressure through a series of fixed tubes of varying diameters.[3] From this, and assuming a driving pressure of 100 cm H_2O, the diameter of a stenotic segment may be estimated by measuring the MIF_{50} and MEF_{50}. The method is reasonably accurate, within about a 1.5-mm tracheal diameter for rigid lesions, but it is less accurate when individuals have emphysema or marked malacic segments.

Variable Upper Airway Obstruction

The term variable obstruction describes the situation where intraluminal and extraluminal pressures affect the anatomy of the lesion. This may occur because the involved area is malacic, or because the lesion, although firm, is not circumferential, and normal tracheal wall movement, usually the posterior membrane, alters the shape of the lumen. The alteration may also be postural, as in the case of a hemangioma.

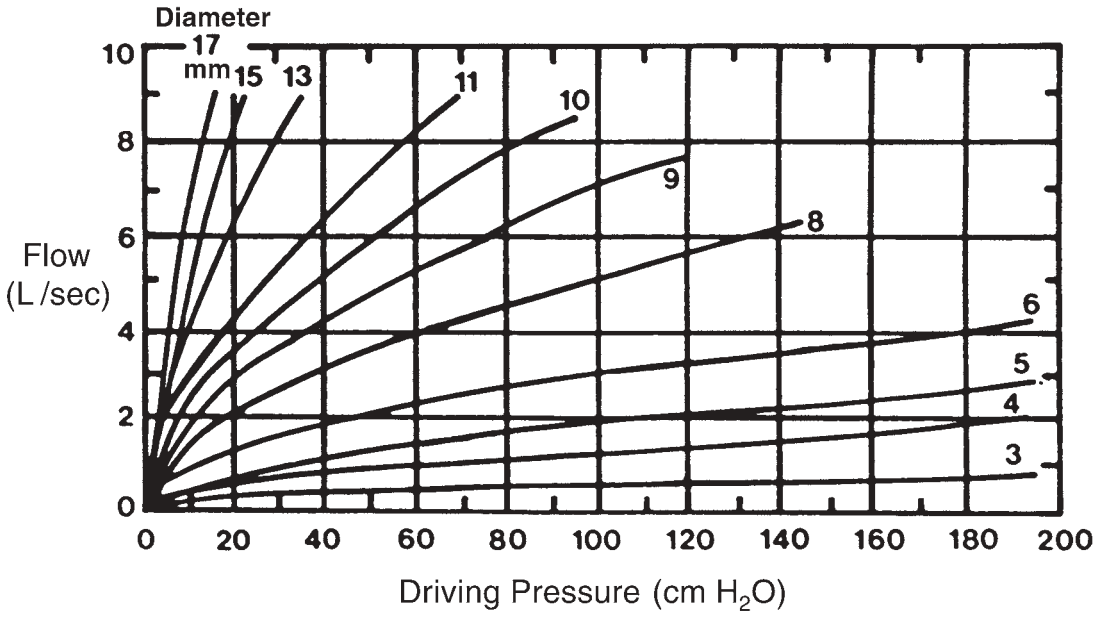

FIGURE 2-6 *Graphic representation of airway diameter and flow (forced expiratory flow$_{50}$ or forced inspiratory flow$_{50}$) with airway pressure between 50 and 150 cm H_2O, derived from progressively increasing external resistances in a normal subject. Reproduced with permission from Gamsu G et al.*[3]

In variable extrathoracic obstruction (see Figures 2-1*D*, 2-7), the negative intraluminal pressure relative to the atmospheric pressure produced during inspiration causes narrowing of, for example, a malacic segment, resulting in limited inspiratory flow.[4] During exhalation, a positive intraluminal pressure is generated, which results in maintenance or expansion of the diameter of the airway and a normal expiratory flow rate. Thus, a plateau is observed on the inspiratory loop alone, and the MEF_{50}/MIF_{50} is greater than 1. Vocal cord paralysis, usually bilateral but also unilateral, is one of the most common causes of this pattern.[5] The features are also seen in individuals with severe burns and vocal cord dysfunction.

Variable intrathoracic obstructions (see Figures 2-1*C*, 2-8), such as tracheomalacia, demonstrate a reduction in the peak flow rate and a flattening of the expiratory loop, in response to the high positive intrapleural and, therefore, extraluminal pressures generated.[4] The inspiratory loop is normal, responding to negative inspiratory pressures. The MEF_{50}/MIF_{50} is low and the FEV_1 less than the forced inspiratory volume in first second (FIV_1). The major causes are malacic segments or noncircumferential tumors.

Upper Airway Obstruction and Chronic Obstructive Pulmonary Disease

Tracheal abnormalities frequently develop in the presence of chronic obstructive pulmonary disease (COPD). This may be due to injuries, such as cicatrization at the site of an endotracheal cuff producing fixed stenosis, tracheomalacia at the site of an endotracheal cuff leading to a variable intrathoracic stenosis, or variable extrathoracic stenosis due to vocal cord dysfunction or malacia at the site of a tracheostomy. The differentiation between the effect of COPD and tracheal injury is important because (a) recognition can lead to repair and (b) it is important to understand the relative contribution of the stenosis and COPD to symptoms such as dyspnea. For example, the presence of a minor degree of radiographically and bronchoscopically recognizable stenosis may have little significance in the presence of very severe COPD, where the flow limitation is mainly in the distal airways, hence the stenosis does not provide any further impediment to flow nor to expectoration of secretions.

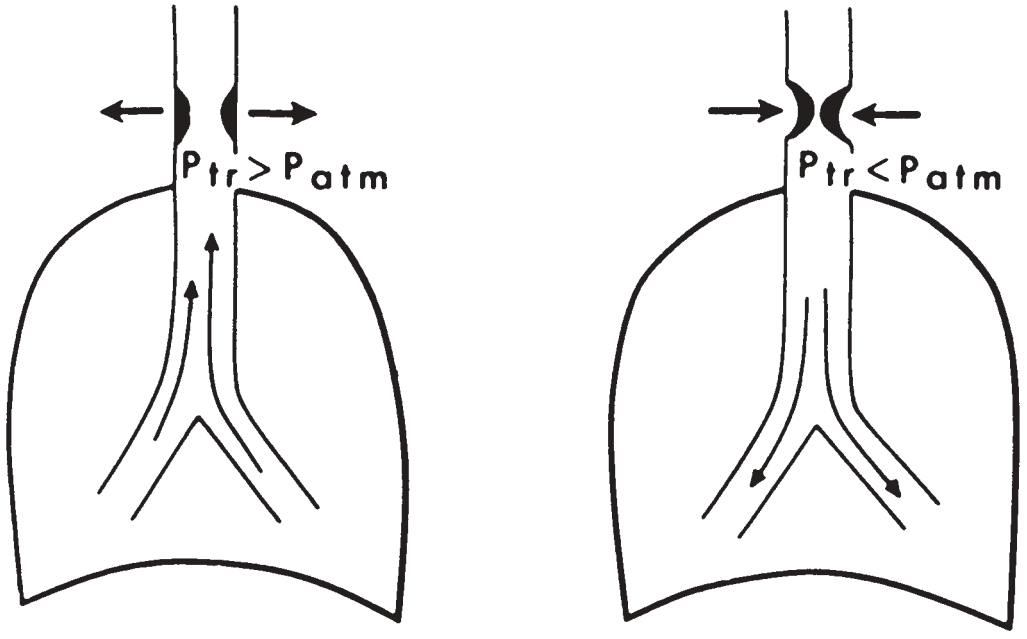

FIGURE 2-7 *Inspiration and expiration influencing variable extrathoracic obstruction. P_{atm} is the atmospheric pressure; P_{tr} is the intratracheal pressure. Reproduced with permission from Kryger M et al.*[4]

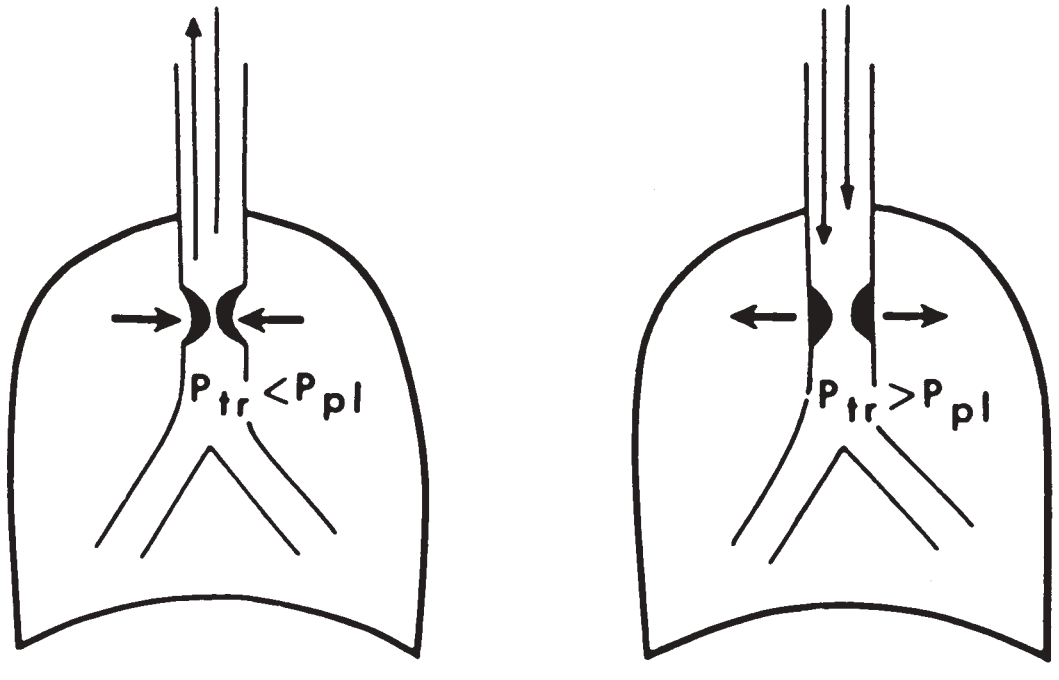

FIGURE 2-8 *Inspiration and expiration influencing variable intrathoracic obstruction. P_{pl} is the intrapleural pressure; P_{tr} is the intratracheal pressure. Reproduced with permission from Kryger M et al.*[4]

Fixed tracheal stenosis, variable intrathoracic stenosis, and COPD all reduce the peak flow rate. However, the tracheal lesions produce a plateau, whereas a sharp peak is still seen in COPD, together with concave expiratory flow patterns. The length of the plateau and the reduction of peak flow by the plateau, as described earlier, are approximately proportional to the severity of the stenosis. As exhalation proceeds, the site of flow limitation moves peripherally until the collapsing small airways inhibit flow. At that point, the concave pattern of COPD will become the visible feature (Figure 2-9). Thus, the ratio of plateau to concavity gives an approximation of the relative significance of the lesions. If no plateau is visible, the stenosis is not contributing to flow limitation, nor is it likely to contribute to dyspnea. However, the reverse is not necessarily true; that is, where stenosis is contributing to flow limitation, release of stenosis may not relieve dyspnea much, since other factors such as hyperinflation related to the residual COPD or the presence of cor pulmonale may be very important in the mechanism of dyspnea. However, improvement in the ability to cough up secretions may have a significant effect. Thus, COPD may conceal stenosis, and stenosis may cause one to underestimate the severity of COPD. In addition, the presence of signs or symptoms also depends on the relationship between the flow required for a specific activity and the degree of obstruction. Geffin and colleagues demonstrated that, at rest, stridor was only heard when the airway was reduced to a 5 mm stenosis.[6] However, with exercise, a less severe stenosis may also cause stridor.[7]

Another useful technique is the superimposition of the tidal volume loop on the FVL, with tidal volume expressed both at rest and during hyperventilation. The point of contact of the tidal volume with inspiratory or expiratory plateaus or with the concavity of COPD provides very useful information about the limiting factors and the contribution of the plateau to limiting flow and to the symptoms. Accurate positioning of the tidal volume on the axis can however be difficult.

Helium-oxygen inhalation, which reduces the density of the gas, has also been used to differentiate COPD from upper airway obstruction (UAO).[8] Since flow in the upper airway is density dependent, helium-oxygen inhalation can increase expiratory flow in the UAO, but not in COPD when laminar flow is much less dependent on density.[9] Nitrogen washout using 100% oxygen is normal in UAO, but abnormal in COPD[10,11]; however, this is mainly useful when pure forms of these processes are present, whereupon an FVL should suffice.

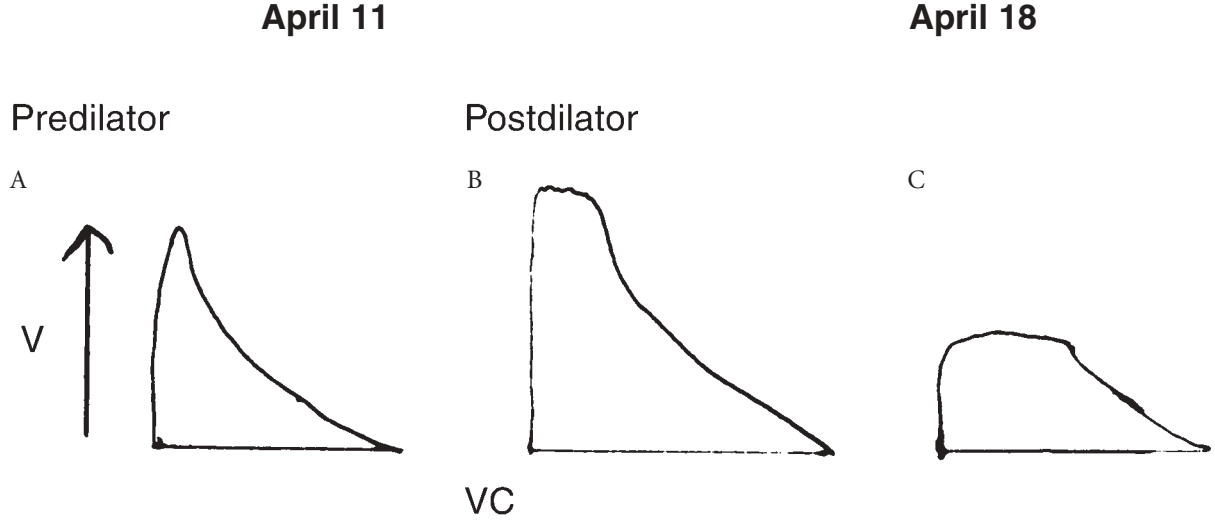

FIGURE **2-9** *Flow volume loops of an individual with chronic obstructive pulmonary disease (COPD), taken prior to tracheal stenosis and a bronchodilator* (A), *with bronchodilator, where flow increases sufficiently to demonstrate a plateau due to concomitant tracheal stenosis* (B), *and progression of the tracheal stenosis, concealing much of the COPD* (C). *V= flow; VC = vital capacity.*

Miller and Hyatt presented data suggesting that the ratio of the mid-expiratory flow rate (MEF_{50}) to the mid-inspiratory flow rate (MIF_{50}) would enhance the visual evaluation of the FVL.[2] A normal ratio would be from 0.9 to 1.0. A fixed obstruction, with both loops limited, would remain at about 0.9, whereas a variable extrathoracic obstruction would be greater than 1.0, and a variable intrathoracic obstruction would be 0.2 or less. These values for variable obstruction relate mainly to severe disease.

The dorsal membrane of the trachea is invaginated to some degree in healthy individuals when exposed to high intraluminal pressures, such as may occur with a cough or during exercise. In some individuals with severe obstructive lung disease, this invagination as visualized at bronchoscopy may be so severe as to almost completely obliterate the lumen. The effect is to markedly limit the ability of individuals to clear secretions via coughing. This may be particularly important in patients with bronchitis extending to their distal airways. Herzog and colleagues investigated a group of these patients using a technique of intrabronchial pressure measurements, slowly withdrawing a catheter from distal to central airways and measuring simultaneous alveolar pressure and flow rate in a plethysmograph.[12] They calculated the flow resistance over bronchial segments and found patients with collapse of the central airways, as well as those with collapse of both peripheral and central airways, when exposed to high intrapleural pressure. These findings were correlated with bronchoscopic inspection, under local anesthesia, of the posterior membrane being invaginated by high intrapleural pressure induced by coughing and hyperventilation. Herzog and colleagues described a typical appearance of a spirometric tracing, in which there is a sudden fall in the flow rate, known as the "check valve" phenomenon. In an individual with severe COPD, this can also be seen in an FVL as a sudden fall in expiratory flow, followed by a slow further decline in flow (Figure 2-10). The posterior membrane was stabilized by grafting fascia or plastic material. Herzog and colleagues showed a marked increase in FEV_1 in some cases, although not all. There was a general improvement in the partial pressure of oxygen (PO_2) and a fall in the partial pressure of carbon dioxide (PCO_2) where this was initially elevated. The authors were careful to point out that these results did not mean that the collapse of airways in COPD was solely in the central airways; the small airways were still the major site of collapse. However, the ability to clear secretions and limit the number of episodes of bronchitis and reactive airway constriction was probably the most important factor in the improvement. They noted that this process was of most assistance in individuals with a clinical syndrome of chronic bronchitis and severe attacks of coughing even up to the point of cough syncope. This is a concept that probably needs to be re-addressed. Certainly, the results in the small number of cases described appear to at least equal the effect of volume reduction surgery on the FEV_1.

The effect of exercise in patients with tracheal stenosis has been sparsely studied. This is a difficult task because many patients with tracheal stenosis also have parenchymal disease, due to coexistent problems such as chronic obstructive lung disease, asthma, or bronchiectasis, which influences the results. Seven patients without diffuse lung disease were studied using mild exercise,[7] since patients with tracheal stenosis are limited in their ability to exercise by dyspnea. In all patients, the PO_2 decreased with exercise, with the mean being 11 mm Hg. In general, the magnitude of PO_2 decrease correlated with the degree of obstruction. PCO_2 only increased an average of 2 mm Hg. In the three subjects who had surgical correction, the PO_2 rose by a mean of 9 mm Hg and the PCO_2 fell slightly with exercise. The vital capacity was not changed by corrective surgery, but the FEV_1, maximum breathing capacity, and peak expiratory flow rate all increased markedly.

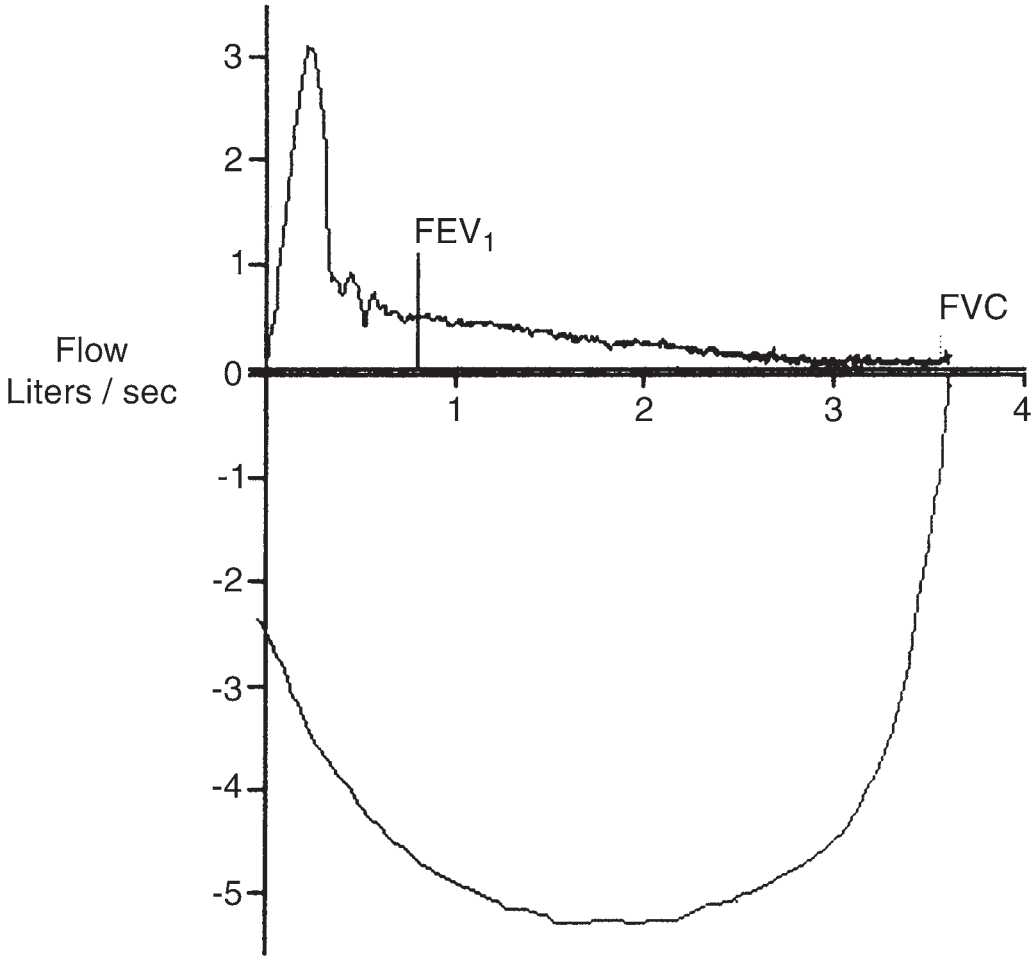

FIGURE **2-10** *Flow volume loop showing a sudden dramatic decline in expiratory flow followed by a long plateau, normal inspiratory loop because of the effect of a negative inspiratory intrapleural pressure on the floppy airways. Flow is the vertical axis and volume is the horizontal axis. FEV, is the forced expiratory volume at first second. FVC is the forced vital capacity.*

References

1. Miller RD, Hyatt RE. Evaluation of obstructing lesions of the larynx and trachea by flow-volume loops. Am Rev Respir Dis 1973;108:475–81.
2. Miller RD, Hyatt RE. Obstructing lesions of the larynx and trachea: clinical and physiological characteristics. Mayo Clinic Proc 1969;44:145–61.
3. Gamsu G, Borson DB, Webb WR, et al. Structure and function in tracheal stenosis. Am Rev Resp Dis 1980;121:519–31.
4. Kryger M, Bode F, Antic R, et al. Diagnosis of obstruction of the upper and central airways. Am J Med 1976;61:85–93.
5. Cormier Y, Kashima H, Summer W, et al. Airflow in unilateral vocal cord paralysis before and after Teflon injection. Thorax 1978;33:57–61.
6. Geffin B, Grillo HC, Cooper JD, Pontoppidan H. Stenosis following tracheostomy for respiratory care. JAMA 1971;216:1–8.
7. Al-Bazzaz F, Grillo H, Kazemi H. Response to exercise in upper airway obstruction. Am Rev Respir Dis 1975;111:631–40.
8. Lavelle TF Jr, Rotman HH, Weg JG. Isoflow-volume curves in the diagnosis of upper airway obstruction. Am Rev Respir Dis 1978;117:845–52.
9. Gold M, Marks A, Bocles JS. Effects of reduction in air density on dynamic function in obstructive airways disease. Am Rev Respir Dis 1964;90:316–20.
10. Sackner MA. Physiologic features of upper airway obstruction. Chest 1972;62:414–7.
11. Simonsson BG, Malmberg R. Differentiation between localized and generalized airway obstruction. Thorax 1964;19:416–9.
12. Herzog H, Keller R, Allogower M. Special methods of diagnosing and treating obstructive diseases of the central airway. Chest 1971;60:49–67.

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Pathology of Tracheal Tumors

3A Epithelial Tumors of the Trachea

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Epithelial proliferations of the trachea, like the bronchus and lung and many other organs, comprise the common tumors obstructing the trachea. As with the bronchus and lung, the majority of epithelial neoplasms in the trachea are malignant, although the frequency of carcinomas in the trachea is much less. This lowered frequency may be due to the limited surface area of the epithelium, greater mucociliary stream, and more laminar airflow compared to the bronchial tree.

Normally, the trachea is lined by columnar ciliated respiratory epithelium, which has both mucinous and nonmucinous cells. Many neoplasms of the trachea, both benign and malignant, are squamous rather than mucinous or glandular, reflecting the conversion of respiratory epithelium to squamous epithelium under noxious stimuli, particularly cigarette smoke. The squamous proliferations resemble those in the lung in many regards. The glandular neoplasms can resemble either tumors of the lung or salivary gland, the latter because the mucus glands in the trachea are similar to mucinous glands and ducts in the oropharynx and salivary glands. Because tumors of the trachea obstruct this single vital structure, detection occurs when the tumor is at a smaller size compared to the average for a pulmonary tumor, and metastasis even to regional lymph nodes is not common. Nevertheless, difficulties in resecting tracheal lesions adversely affect the prognosis in some patients, where recurrent tumor develops after incomplete resection or, in the rare case, where local metastasis has already occurred.

Squamous Epithelial Tumors

Squamous Papilloma and Papillomatosis

Squamous papillomas of the trachea are rare benign tumors composed of stratified squamous epithelium with acanthosis and papillomatosis, supported by a fibrovascular core. They are either multiple and recurrent (papillomatosis) or solitary exophytic growths into the tracheal lumen.

Biology. Squamous papillomatosis occurs as multiple and recurrent squamous cell papillomas of the trachea, often associated with upper (mostly laryngeal) and/or lower (bronchial) involvement. Children and adolescents are most commonly affected, hence the term "juvenile papillomatosis"; occasional adults with disease have been reported.[1]

The patient may present with stridor, wheezing, dyspnea, chest pain, or hemoptysis. The lung parenchyma is affected in about 1% of cases, most of them complicated by necrosis, cavitation, or pneumonia.

The disease sometimes regresses in adulthood, but the course is usually protracted in extensive papillomatosis with complications of obstruction in the trachea, larynx, or bronchi, with atelectasis, bronchiectasis, and pneumonia. Surgical resection, laser fulguration, and interferon therapy are usually attempted to secure patent airways in these patients.

Human papilloma virus (HPV) is a known cause of these tumors, and different types are detected. HPV types 6 and 11 are commonly found in benign lesions, whereas types 16 and 18 are mostly associated with malignant transformation.[2] Other types including 31, 33, and 35 are also occasionally found in malignantly transformed cases.[2]

Solitary papillomas often occur in adults, but children may also have solitary tumors.

Squamous cell carcinomas may develop in about one-third of solitary squamous papillomas without other risk factors.[3] In contrast, malignant transformation is less common in multiple papillomas (papillomatosis), occurs on average about 15 years after initial diagnosis, and is often associated with risk factors such as radiation, cytotoxic drug therapy, and smoking.[4]

Pathology. Grossly, the papillomas appear as cauliflower-like excrescences, protruding into the tracheal lumen (Figures 3-1, 3-2 [Color Plate 1]).

Microscopically, papillomas are composed of loose fibrovascular cores covered by hyperplastic stratified squamous epithelium with papillomatosis. Keratinization may be present sometimes with small parakeratotic foci. Koilocytosis (perinuclear halo and nuclear wrinkling) is seen in all papillomatosis cases and in solitary papillomas associated with HPV. The epithelium is usually cytologically bland; mitoses, atypicality, or dysplastic cells are infrequent, but are occasionally seen in solitary papillomas (Figure 3-3 [Color Plate 1]).

A case of a cytologically benign papilloma was reported in a 27-year-old man, where the tumor invaded the tracheal wall and adjacent soft tissues, without nodal or distant metastasis in 4 years of follow-up. The term "invasive tracheal papillomatosis" was suggested in this case.[5]

Papillomas must be differentiated grossly and microscopically from papillary squamous cell carcinomas, which they resemble because of papillary configuration and layers of neoplastic squamous cells, but lack similar atypical cellular and architectural features. Squamous cell carcinomas may also arise in papillomas, showing focal cellular pleomorphism, loss of maturation, dyskeratosis, and increased hyperkeratosis. It then extends through the epithelium and ultimately invades the underlying connective tissue. It may go through the tracheal wall into adjacent soft tissues and lymphatics. These can be well or moderately differentiated, with or without keratinization. Small endoscopic biopsies can result in improper interpretation, since malignant transformation can be well differentiated or focal. Conversely, some focal atypia have been observed in benign papillomas.[6] Extension of squamous epithelium to bronchial glands should not be interpreted as invasion.

Squamous Cell Carcinoma

In many published series, squamous cell carcinoma is the most common primary tracheal tumor, whereas in others, it equals in frequency or comes after adenoid cystic carcinoma as the second-most common neoplasm in adults.[7–9]

Biology. Although one of the two most common tracheal malignancies, squamous cell carcinoma is far less common than its laryngeal or bronchogenic counterparts, which are about 75 and 180 times more frequent, respectively.[10] Age distribution is between 20 and 80 years with peak incidence in the sixth and seventh decades, similar to bronchogenic squamous cell carcinoma. An infant with tracheal squamous cell carcinoma has been reported.[8] Men are significantly more affected than women, with a ratio of 2:1 to 4:1 in large series.[8,9] Smoking history is present in most patients, accounting for almost all cases in some series, suggesting a strong association as in bronchogenic squamous cell carcinoma.[7]

The lower incidence of tracheal squamous cell carcinomas compared with those of the bronchus is attributed by some to laminar airflow in the trachea (because of large diameter) and effective mucociliary clearance (because of evenness and absence of bifurcation). These may prevent accumulation of carcinogens in the mucosa which can promote a malignant transformation sequence.[11] Six patients have been reported with tracheal squamous cell carcinoma arisen from tracheostomy scars, probably the result of carcinogenesis in active repair, similar to scar carcinoma elsewhere.[12] One case was reported in a plumber exposed to asbestos.[13]

Most cases of primary squamous cell carcinoma occur as solitary lesions, but synchronous and metachronous tumors, mostly with bronchogenic, laryngeal, or esophageal squamous cell carcinomas, have been reported.[14,15]

Pathology. Grossly, squamous cell carcinomas usually arise from the posterior tracheal wall as polypoid growths, most commonly in the lower third, followed by the upper third (Figures 3-4, 3-5 [Color Plate 1]).[8] Surface ulceration is often present.

Microscopically, the tumor is composed of nests and sheets of squamous cells with various degrees of differentiation (Figures 3-6, 3-7, 3-8 [Color Plate 1]). Squamous differentiation, as in the lung, includes intercellular bridges and keratin pearl formation. Two cases of basaloid variants have been reported, wherein like the cutaneous basal cell carcinoma, the cells are smaller, with scant cytoplasms and high nuclear to cytoplasmic ratios.[16] Combined squamous cell and small cell carcinomas have also been reported.[17]

The differential diagnosis includes papilloma, squamous cell carcinoma arising in papilloma, mucoepidermoid carcinoma, and necrotizing sialometaplasia. It must also be distinguished from squamous cell carcinomas invading from adjacent structures such as the esophagus, lung, thymus, and even thyroid gland. These primaries must always be considered when squamous cell carcinoma is diagnosed by endoscopic biopsy.

Adenocarcinoma

Although the most common lung malignancy, adenocarcinomas are usually located in the lung periphery, away from the central airways; they are infrequent in central bronchi and much rarer in the trachea.

Biology. Adenocarcinoma has been reported in several recent series of primary tracheal tumors as a rare entity.[9,18] However, in older literature, its occurrence was much higher, and exceeded squamous cell carcinoma as the most common tracheal malignancy in some series.[19] It is not clear whether some cases of other primary tumors, such as mucoepidermoid carcinoma or adenoid cystic carcinoma, adenocarcinomas invading from adjacent structures, and metastatic adenocarcinomas, had been misclassified as primary adenocarcinomas in those series, and hence partly responsible for the high incidence of this tumor in older publications. Adenocarcinoma affects both men and women, and occurs mostly in the fifth to eighth decades of life. Presentation is often with obstructive symptoms. One patient presented with a thyroid mass, later found to be invasion from tracheal adenocarcinoma.[20]

Pathology. Grossly, adenocarcinomas are bulky tumors that may bulge into the lumen, but they also invade through the tracheal wall into adjacent structures.

Microscopically, most of the reported cases have been of the mucin-producing category. They form glands, with the epithelial cells containing large vesicular nuclei and prominent nucleoli (Figures 3-9, 3-10 [Color Plate 2]). Differential diagnosis includes adenoid cystic carcinoma, mucoepidermoid carcinoma, and secondary involvement of trachea from other primaries. The latter includes direct invasion from thyroid gland carcinomas and adenocarcinomas of proximal bronchi extending up to the trachea. Metastatic adenocarcinomas must be distinguished from tracheal primaries, as cases have been reported from colorectal adenocarcinomas and endometrial adenoacanthomas.[21,22]

A case of adenocarcinoma ex pleomorphic adenoma (arising in pleomorphic adenoma) of the trachea has been reported.[23] Also reported are cases of thyroid carcinoma arising from ectopic thyroid tissue in the trachea.[24] Various degrees of histologic differentiation in tracheal adenocarcinomas remain to be clarified, since this description is not given in most of the published series. Tracheal adenocarcinomas seem to follow an invasive and rapidly fatal course; however, valid survival analyses have not been performed so far.

Large Cell Undifferentiated Carcinoma

Also called "large cell carcinoma" or "anaplastic carcinoma," this malignant epithelial tumor consists of sheets of cells with large nuclei, prominent nucleoli, abundant cytoplasm, and usually well-defined cell borders, without the characteristic features of squamous cells, small cells, or adenocarcinomas.[25]

Biology. Large cell carcinoma of the lung almost always occurs in smokers with a median age of 60 years.[26] Its incidence in the trachea ranges from none in some series to as high as 21% in others.[11,27]

Pathology. Grossly, these tumors are usually large tan or white necrotic masses, sometimes with hemorrhage.

Microscopically, sheets of large polygonal cells with large vesicular nuclei and prominent nucleoli are observed. Cytoplasm is abundant and the cell membrane is usually prominent. Squamoid features may be seen, but keratin pearls and intercellular bridges are lacking. Gland formation and mucin production, which may be identified by mucin stains, are not present. Therefore, the diagnosis is made by exclusion of squamous cell carcinoma and adenocarcinoma. Small endoscopic samples may lack foci of differentiation that are evident later in a resected specimen. When neuroendocrine differentiation is suggested by immunohistochemical or electron microscopic examinations, the tumor corresponds to "large cell carcinoma with neuroendocrine differentiation" as occurs in the bronchi. It is distinguished from "large cell neuroendocrine carcinoma," which shows an organoid pattern, rosetting, and peripheral palisades. Differentiation from small cell carcinoma is evidenced by large vesicular nuclei, prominent nucleoli, abundant cytoplasm, and absence of nuclear molding. Smearing (crushing) artifact is more usual for small cell carcinoma.

Histological variants include giant cell carcinoma, spindle cell carcinoma, pleomorphic (giant cell/spindle cell) carcinoma, clear cell carcinoma, and lymphoepithelioma-like carcinoma. Only giant cell carcinoma and lymphoepithelioma-like carcinoma have been reported in the trachea.[28,29]

Giant cell carcinoma has large cells (ie, two or three times the size of classic large cell carcinomas) and huge, bizarre, and pleomorphic giant cells. Giant cells may be seen focally in many lung carcinomas and may also occur after radiation therapy of tumors, and a prominent component of giant cell change (ie, above 10% of tumor cells) is necessary to make the diagnosis.[25,28] It is not certain whether the reported giant cell carcinomas in the trachea meet the given criteria. The only reported case of lymphoepithelioma-like carcinoma had similarity to the so-called nasopharyngeal lymphoepithelioma.[29]

Mixed small cell/large cell carcinoma has been seen in the lung, and a case of mixed small cell/squamous cell/giant cell carcinoma in the trachea has been reported.[30] Anaplastic or undifferentiated carcinoma invading the trachea from adjacent structures must always be considered in the differential diagnosis of primary tracheal large cell carcinoma. In a series of anaplastic thyroid carcinoma, tracheal invasion was reported in some cases.[31]

Neuroendocrine Tumors

This group of malignant neoplasms comprises a spectrum from low-grade typical carcinoid to high-grade neuroendocrine tumors. These tumors are much less frequent in tracheae than in bronchi, probably due to a scarcity of Kulchitsky cells (neuroendocrine cells of origin) in the trachea. Bronchopulmonary neuroendocrine tumors are classified into low-grade typical carcinoid, intermediate-grade atypical carcinoid, and high-grade carcinoid categories of large cell neuroendocrine carcinomas and small cell carcinomas. Because of their rarity in the trachea and lack of large studies, some of the data given here are based on bronchial counterparts, which probably do not differ much from tracheal ones.

Typical and Atypical Carcinoid Tumors

Carcinoid tumors are low-grade malignant neoplasms of neuroendocrine cells. They are divided into typical and atypical subtypes, with the latter possessing more malignant histologic and clinical features.[32]

Biology. Carcinoid tumors are reported from childhood to old-age.[33] The median age for patients with bronchial carcinoid tumor is 55 years, and it is the most common bronchial tumor of childhood.[34] Males and females are equally affected.

Pathology. Grossly, carcinoid tumors usually have a main polypoid intraluminal component, with a smooth and tan-yellow to pink cut surface (Figure 3-11 [Color Plate 2]). Occasionally, they can be virtually confined to the polyp and have only minor growth in the lamina propria of the tracheal wall. Atypical carcinoids may be more infiltrative through the wall, sometimes with areas of necrosis or hemorrhage. Local lymph node metastasis is frequently seen in atypical carcinoid tumors.

Microscopically, both typical and atypical carcinoids show the so-called "neuroendocrine look," which is an organoid pattern of uniform epithelial cells with a finely granular chromatin pattern, inconspicuous nucleoli, and moderate eosinophilic cytoplasm (Figures 3-12, 3-13, 3-14 [Color Plate 2]). Atypical carcinoids have coarser chromatin with more prominent nucleoli. Other histologic patterns are those that are trabecular, glandular, paraganglioma-like, rosette-like, and palisaded. Usually, more than one pattern is seen in a given tumor. Uncommon patterns seen in tracheal carcinoids are those that are oncocytic and melanin-producing.[35]

It is well known that atypical carcinoids are cytologically more atypical with a higher mitotic rate and necrosis, but the criteria of separation from typical carcinoids have been challenging over the years. The most recent criteria were proposed by Travis and colleagues for bronchopulmonary carcinoids, and seem to be reproducible and well correlated with clinical behavior and survival.[36] Based on these criteria, atypical carcinoids are characterized by a mitotic rate of 2 to 10 per 10 high-power fields or foci of coagulative necrosis; that is, either punctate or large and infarct-like.

Typical carcinoids are often well delimited or have a broad front of invasion. Atypical carcinoids are more likely to have tongues of invasion into the tracheal wall. Vascular and lymphatic invasions are common in atypical carcinoids.

Differential diagnosis includes small cell carcinoma and undifferentiated large cell carcinoma, which can be very difficult on interpretation of small or crushed endoscopic biopsies. Depending on

the various patterns present in carcinoid tumors, other tumors may be considered in the differential diagnosis, but it is not clear how often uncommon patterns may be seen in tracheal carcinoids.[37] An oncocytic pattern must be separated from other forms of oncocytic tumors. A glandular pattern may be difficult to differentiate from adenoid cystic carcinoma and mucoepidermoid carcinoma. Important is the absence of hyaline cores and mucin, respectively. Immunohistochemical studies for neuroendocrine markers are often necessary on a small biopsy. Melanocytic carcinoid tumors must be separated from melanomas.

Metastatic carcinoma of the trachea is another group in the differential diagnosis. The 5- and 10-year survival rates for typical bronchial carcinoid tumors are both at 87%, and for atypical carcinoid tumors, are at 56% and 35%, respectively.[36] The survival for atypical carcinoid tumor of the trachea is probably less than its bronchial counterpart, because local lymph node metastasis for this midline organ can be bilateral and difficult to encompass surgically at the outset.

Large Cell Neuroendocrine Carcinoma

Large cell neuroendocrine carcinoma (LCNEC) is a high-grade neuroendocrine tumor, which may be mistaken pathologically for a carcinoid or atypical carcinoid tumor, but has a worse prognosis. This tumor shows a light microscopic, immunohistochemical, and ultrastructural neuroendocrine differentiation.

Biology. There is only one published study on these tumors in the trachea.[38] This could be due to its rarity, but it may also be due to misclassification as an atypical carcinoid or large cell carcinoma. The data and descriptions that follow are mainly from pulmonary LCNECs. The patient ages range from 35 to 75 years, and the tumor occurs mostly in cigarette smokers.[39]

Pathology. Grossly, the cut surface is usually yellow, tan, or white, with necrosis. Hemorrhagic foci may be present.

Microscopically, the tumor shows neuroendocrine features with organoid, palisading, trabecular, or rosette-like growth patterns. It usually has nests of tumor cells with central necrosis. The cells are large with vesicular nuclei and prominent nucleoli. The mitotic rate is more than 10 per 10 high-power fields. Endocrine differentiation may be shown on immunohistochemical or ultrastructural studies.

The main differential diagnosis is of large cell carcinoma with neuroendocrine features. This distinction is made by light microscopic evidence of a "neuroendocrine look," such as an organoid pattern, peripheral palisades, or rosetting in LCNEC. Immunohistochemistry and electron microscopy are not helpful since both have neuroendocrine granules. Atypical carcinoid is separated based on mitotic activity (less than 5 per 10 high-power fields). Small cell carcinoma is sometimes difficult to differentiate, particularly when the cells are larger in small cell carcinomas or when the biopsy is small and crushed. Overall, smaller cells in small cell carcinomas, as well as nuclear molding, a fine chromatin pattern, absence of inconspicuous nucleoli, a smearing effect, and basophilic staining of vessels (Azzopardi effect) are helpful distinguishing features.

The prognosis is worse than for atypical carcinoids, and approaches that for small cell carcinoma.[38] The response to chemotherapy seems to be better than that of nonsmall cell carcinomas.

Small Cell Carcinoma

Small cell carcinoma is the other high-grade neuroendocrine carcinoma of the tracheobronchial tree.

Biology. Relatively common among bronchial malignancies, small cell carcinoma has been regarded as a very rare tumor in the trachea. In most studies, it is either not present or is reported as one of the least

common tracheal malignancies. However, in two of the largest series with 321 and 43 cases, it comprised 6% and 7% of tracheal tumors, respectively.[18,40] Hormonal activity was reported in a case with both oncogenic hypophosphatemia and ectopic corticotropin production causing Cushing's syndrome.[41]

Pathology. In bronchi, small cell carcinoma typically infiltrates the wall, with irregular thickening of mucosa, and it less commonly produces a tapered narrowing or intraluminal mass. When there is extensive paratracheal small cell carcinoma, it may be difficult to know whether the tumor arose in the hilum of the lung, esophagus, thymus, or trachea.

Microscopically, the tumor consists of irregular islands of small cells, about two or three times the size of lymphocytes, with a high nuclear to cytoplasmic ratio. Nuclei are round to oval with hyperchromatic finely-granular chromatin, nucleoli are absent or inconspicuous, and cytoplasm is scant (Figures 3-15, 3-16 [Color Plate 2]). The cells usually lie close to each other, sometimes with nuclear molding. Extensive or punctate necrosis is common and mitotic figures are frequent. Less common morphologic features include slightly larger polygonal cells with more cytoplasm, formation of tubules and rosettes, and peripheral palisading, all of which are seen in bronchial small cell carcinomas.

The neuroendocrine nature of the tumor cells is occasionally demonstrable by immunohistochemical studies with neuroendocrine markers, such as chromogranin, synaptophysin, neuron-specific enolase (NSE), and neuron cell adhesion molecule (N-CAM), or as dense core granules on electron microscopy. In practice, however, these studies may be difficult to interpret in a small crushed biopsy.

A case of mixed squamous and small cell carcinoma and another of mixed squamous cell, small cell, and giant cell carcinoma have been reported in the trachea.[17,30]

Differential diagnosis includes other neuroendocrine tumors (carcinoid, atypical carcinoid, large cell neuroendocrine carcinoma), nonsmall cell carcinoma (adenocarcinoma, squamous cell carcinoma, large cell carcinoma, whose cells are sometimes relatively small), small round cell tumors (such as lymphoma, embryonal rhabdomyosarcoma), and adenoid cystic carcinoma.

The diagnosis may be particularly difficult in small endoscopic biopsies due to crushing artifact of cells, which can also occur in lymphocytic infiltration and neuroendocrine and other tumors. The exclusion of malignant lymphoma by cytology or immunohistochemical markers is particularly important because of the different therapy and prognosis for tracheal lymphoma.

Similar to bronchial small cell carcinoma, this tumor has the worst prognosis among the tracheal malignancies.

Salivary Gland-Type Tumors

The minor salivary glands existing in the mucous membranes of the head and neck extend downward as the submucosal glands in the trachea. Therefore, they are subject to most of the so-called "salivary gland-type tumors" that involve the salivary glands of the head and neck, with only few exceptions. However, the frequency of such tumors is much lower in the trachea than in the head and neck region.

Pleomorphic Adenoma (Mixed Tumor)

Pleomorphic adenoma is primarily a benign tumor of the major salivary glands. Rarely, it can arise from seromucous glands of the tracheobronchial tree.

Biology. Approximately 30 cases of tracheal pleomorphic adenoma have been reported in the literature. The male to female ratio is about 2:1 with an age range of 15 to 80 years. Over one-half of patients were in the fifth and sixth decades of life.

Pathology. The upper third of the trachea is the most common site of origin, followed by the middle and lower thirds.[42] The tumor typically grows as a polypoid intraluminal mass, causing various degrees of obstruction (Figure 3-17 [Color Plate 3]).

Microscopically, pleomorphic adenoma is relatively well circumscribed, but without a capsule. It consists of a mixture of epithelial cells, myoepithelial cells, and stroma (Figures 3-18, 3-19 [Color Plate 3]). The epithelial component forms sheets, ducts, trabeculae, and small nests of cells with vesicular nuclei and small to moderate amounts of cytoplasm. Foci of squamous differentiation may be seen. These areas merge with alternating areas of spindled and stellate cells in myxoid, hyaline, or chondromyxoid stroma. The terms "pleomorphic" and "mixed" both describe the contribution of epithelial and mesenchymal elements, but one or the other may predominate, almost to the exclusion of the other.

Differential diagnoses include other salivary gland-type tumors like adenoid cystic carcinoma and mucoepidermoid carcinoma. Polymorphous low-grade adenocarcinoma, which sometimes is a difficult tumor to distinguish from pleomorphic adenoma in salivary glands, has been reported in the bronchi but not in the trachea.

The prognosis is excellent, with only one recurrence reported. One patient died of tracheal obstruction. A case of carcinoma arising in pleomorphic adenoma (carcinoma ex pleomorphic adenoma) has also been reported, and we have seen a case where the majority of the tumor was pleomorphic adenoma, but the invasive tumor in the adventitia was carcinoma.[23]

Mucous Gland Adenoma

Mucous gland adenoma is a benign tumor of salivary gland-type, which can rarely arise in seromucous glands of the tracheobronchial tree. A small number of cases has been reported in bronchi and two cases in the trachea.[43,44] This tumor is characterized by cystically dilated mucous-filled glands, hence the synonymous term "mucous gland cystadenoma." The cystic glands are lined by monomorphic cuboidal to columnar cells. Papillary proliferation can be present in the luminal surface of the tumor in both bronchial and tracheal mucous gland adenomas.

The major differential diagnosis is mucoepidermoid carcinoma, which in low-grade cases could be predominantly glandular with a banal cytology. The presence of intermediate cells in mucoepidermoid carcinoma helps in this distinction.

Adenoid Cystic Carcinoma

Called "cylindroma" in the past because of the production of cylindrical structures by the tumor cells, adenoid cystic carcinoma is one of the two most common tracheal malignancies, and together with squamous cell carcinoma, accounts for more than two-thirds of all tracheal tumors.[9,18,45] Although it is less common than its head and neck counterpart, its occurrence is higher than bronchial adenoid cystic carcinomas.

Biology. The age distribution for patients with this tumor is generally between 15 and 80 years, with the peak incidence occurring in the fifth decade, and the mean age being 10 years less than patients with squamous cell carcinoma.[9,45,46] The sex distribution is almost equal.[9,45,46] No association with smoking has been reported. The slow-growing nature of this malignancy causes late symptoms, which adds to the general diagnostic delay of tracheal tumors due to anatomic characteristics of the trachea (mainly its large luminal diameter) and often unhelpful chest x-rays.

Pathology. Grossly, the tumor grows as a polypoid intraluminal mass or infiltrates the wall to cause thickening which can be vertical, horizontal, or circumferential, with eventual narrowing of the lumen (Figures 3-20 through 3-23 [Color Plate 3]).[47]

Histologically, the tumor consists of small basaloid cells with a relatively high nuclear to cytoplasmic ratio and scant cytoplasm (Figure 3-24 [Color Plate 3]). The nuclei are round to oval, and dark and monotonous. Three histologic subtypes are identified: cribriform, tubular, and solid (Figures 3-25, 3-26, 3-27 [Color Plate 4]). The most common is the cribriform type, in which the neoplastic cells are arranged in nests and sheets fenestrated by round to oval spaces. These spaces contain eosinophilic periodic acid-Schiff (PAS)-positive basement membrane-like material. The tubular subtype has single-lumen tubular units with smaller nests than the cribriform subtype. In the solid pattern, the cells are packed together to form nests and sheets with few lumina. Commonly, more than one pattern is present in a given tumor. The overlying epithelium is usually intact, with the tumor infiltrating submucosal tissue, going through the wall to adjacent structures, particularly from the posterior wall, where there is no cartilage. The neoplastic cells have a marked tendency for perineural invasion (Figure 3-28 [Color Plate 4]). Because of this infiltrating nature, microscopic examination often shows tissue involvement beyond grossly visible or palpable tumors.

Histological grading is based on the presence and percentage of solid pattern, which inversely affects prognosis.[48] Grade 1 is a tumor composed of tubular and cribriform subtypes, without any solid areas. Grade 2 consists of tubular and cribriform subtypes, with less than 20% solid subtype. In Grade 3, the solid subtype comprises more than 20% of the area of the section. This grading system has been shown to correlate with tumor behavior and patient prognosis.[48,49]

Differential diagnosis includes other carcinomas, mostly adenocarcinomas. Solid areas with few cystic spaces may be confused with adenocarcinomas, particularly since the nuclei can be larger and vesicular in poorly differentiated adenoid cystic carcinomas. In cases in which the morphology is close to adenocarcinoma with large sheets and few cystic or glandular spaces, the behavior is likely to be that of an adenocarcinoma.

Small cell carcinoma must also be differentiated from the solid subtype. The more even chromatin pattern, nuclear crowding, and molding in small cell carcinoma are of help in this distinction.

Frozen section examination of proximal, distal, and circumferential resection margins are essential at the time of definitive resection because of the surreptitious manner in which this tumor spreads. Two potential oversights are noteworthy. First, nests of tumor the size of lobules of normal tracheal mucous glands may partially replace the normal lobules without altering the overall mucosal architecture, thus escaping detection on casual examination. Second, perineural infiltration by individual cells may be the only tumor present in peritracheal adventitia. The paratracheal nerves deserve identification.

Adenoid cystic carcinomas tend to be locally recurrent, particularly with inadequate safe margins, but nodal paratracheal metastases develop late in the course. Distant hematogenous metastases are infrequent in our experience, and fatal complications are more commonly due to recurrent tumor. When they occur, they are usually in the lung, liver, or bone.

The prognosis is much better than squamous cell carcinoma, with 5- and 10-year survivals being reported to be about 75% and 50%, respectively.

Mucoepidermoid Carcinoma

Mucoepidermoid carcinoma is a malignant salivary gland-type neoplasm arising from submucosal glands of the trachea. Until its histological description and definition as a separate entity in the tracheobronchial tree by Smetana and colleagues in 1952, and by Liebow 1975, and even years thereafter, it was classified as a "bronchial adenoma" together with carcinoid tumor and adenoid cystic carcinoma.[50,51]

Biology. Mucoepidermoid carcinoma is a rare tracheobronchial tumor, which affects the trachea even less often than bronchi. Because of its rarity, no large series has ever been published exclusively on tracheal mucoepidermoid tumors, and precise demographic data are lacking. Although it occurs in a wide age range, from childhood to the elderly, many cases are reported in children, and a predilection for the teenage

and young adult population seems to be present, as is the case in its bronchial counterpart. In a large series of bronchial mucoepidermoid carcinomas, low-grade tumors comprised 90% of cases, and over half of them occurred in individuals younger than 30 years of age.[51,52] High-grade tumors occurred in 10% of cases, 70% of these in individuals older than 30 years.

Pathology. Grossly, this tumor typically grows as a polypoid tan-gray to pink endotracheal mass (Figures 3-29, 3-30 [Color Plate 4]). Invasion through the trachea into adventitia is seen in some high-grade tumors.

Microscopically, mucoepidermoid carcinomas are characterized by a variable admixture of mucus-secreting cells, squamous cells, and cells of intermediate type (Figures 3-31, 3-32 [Color Plate 4]).[53] Mucin-producing cells are scattered individually, in clusters, or more commonly, arranged in glandular or cystic structures, which are intimately intermixed with sheets of intermediate or squamous cells. Intermediate cells are taken to be smaller than squamous cells, with scant cytoplasm and without any differentiation toward squamous or mucinous cells. Obvious features of squamous differentiation as squamous pearls, eddies, or dyskeratotic cells are often absent. Oncocytic and clear cell changes are described in bronchial counterparts. One case of oncocytic mucoepidermoid carcinoma has been reported in the trachea, in which sheets and nests of oncocytes comprised 75% of the tumor.[54] The cells stained positive for phosphotungstic acid-hematoxylin (PTAH) and negative for PAS and neuroendocrine markers. Anaplastic features were absent and the tumor was classified as low grade. Regional lymph node metastasis was found. Clear cell change is not reported in tracheal tumors but could possibly occur as it does in bronchial tumors.

Differential diagnosis includes squamous cell carcinoma, adenocarcinoma, other salivary gland-type tumors (like pleomorphic adenoma), carcinoid tumor, and mucous gland adenoma. The oncocytic variant must be differentiated from oncocytoma and carcinoid tumor. Adenosquamous carcinoma is a major differential diagnosis in the lungs (bronchial tree), but no case has been reported in the trachea.

A grading system is not as well established as is in salivary gland mucoepidermoid carcinomas since there are no large follow-up studies. The grading system that follows is used for bronchial mucoepidermoid carcinomas and seems to correlate with tumor behavior and disease outcome in tracheal tumors as well.[52]

In low-grade mucoepidermoid carcinomas that comprise the majority, glands or cysts of mucinous cells predominate. The minor components are the solid sheets of intermediate and squamous cells, which show little mitotic activity, nuclear pleomorphism, or necrosis.

High-grade tumors have sheets of intermediate and squamous cells with high mitotic rate (4 per 10 high-power fields on average), nuclear atypia, or cellular necrosis. Glandular structures are the minor component.

Older literature is conflicting with respect to disease outcome and patient survival, partly due to the misclassification of this tumor with others as a "bronchial adenoma," and partly due to the lack of a grading system. Hence, both excellent and very poor prognoses are reported, now attributable to low and high grades of tumors. The strong association between tumor grade and clinical course is now well established, with an excellent course in low-grade and a fatal course in most high-grade tumors.[55,56]

Malignant Melanoma

Malignant melanoma of the skin can rarely metastasize to the trachea. Even less frequent is a malignant melanoma primary in the trachea, with only about 4 cases reported in the literature so far.[57] An additional case of multiple tracheobronchial melanoma has also been published, in which the primary site could have been either the trachea or bronchi.[58]

Pathology. Grossly, most of these tumors have been polypoid, one of them with a long, narrow stalk (Figure 3-33 [Color Plate 5]).[59] Another of the tumors had a flat subepithelial location.[60] This flat melanoma had metastasized to the adjacent lymph node. Pulmonary metastasis was present in one case.[59]

Microscopically, the tumor classically consists of large cells with atypical vesicular nuclei and prominent nucleoli. The cells are arranged in nests and sheets (Figure 3-34 [Color Plate 5]) and contain melanin. If no melanin is seen, one can prove melanocytic differentiation by immunohistochemical or ultrastructural analyses. Intraepithelial location of malignant melanoma suggests that the tumor has arisen at that site. Differential diagnosis is broad, and includes various tumor categories such as carcinomas, sarcomas, and lymphomas. The most important ones to differentiate in this location are large cell carcinoma and large cell neuroendocrine carcinoma.

Metastatic Tumors to the Trachea

Secondary involvement of the trachea mostly occurs as direct invasion from adjacent organs, such as the larynx, thyroid (Figures 3-35, 3-36, 3-37 [Color Plate 5]), esophagus, or mediastinal structures, and is rarely due to metastasis (Figure 3-38 [Color Plate 5]). This is in contrast to the bronchial tree, which more commonly receives metastases from distant primaries. All published tracheal metastases consist of carcinomas and melanomas, and no sarcoma has been reported, although the latter has been recognized as a form of bronchial metastasis.

Table 3-1 shows the demographic data and types of the 14 published cases of tracheal metastasis.[21,22,61–71,] Carcinomas of the breast and colon and cutaneous melanoma were the most common

Table 3-1 Reported Cases of Metastases to the Trachea

Reference Number	*Date*	*Age*	*Sex*	*Primary Tumor Site*	*Primary Tumor Type*	*Time Interval to Metastasis*	*Metastases to Other Sites*
61	1954	41	M	Colon	Adenocarcinoma	6 years	None
62	1965	35	F	Colon	Adenocarcinoma	3 years	?Lung, ?Liver
63	1974	35	F	Breast	Medullary carcinoma	5 years	Lung
22	1975	62	F	Endometrium	Adenoacanthoma	11 years	Lung
64	1978	61	M	Kidney	Renal cell carcinoma	6 years	Femur, supraclavicular and mediastinal lymph nodes
65	1980	61	F	Ovaries	Bilateral papillary cystadenocarcinoma	7 years	Pleura, axillary lymph nodes
66	1980	57	F	Breast	Invasive ductal carcinoma	6 years	Lung
66	1980	50	F	Breast	Invasive ductal carcinoma	12 years	None
67	1981	28	M	Skin (shoulder)	Melanoma	2 years	None
68	1982	40	F	Skin (upper arm)	Melanoma	2 years	Ribs, vertebral column
69	1987	41	M	Nose	Esthesioneuroblastoma	1 year	None
70	1991	44	F	Skin (breast)	Melanoma	2 months	Pharynx, axillary lymph nodes
21	1994	73	M	Colon	Adenocarcinoma	13 months	Liver
71	2001	56	M	Left lung	Squamous cell carcinoma	2 years	Pulmonary hilar lymph nodes

metastatic tumors to the trachea. All patients had a history of primary cancer. Melanoma had the least time interval from diagnosis of primary cancer to recognition of tracheal metastases, which was 2 months in 1 of the cases and 2 years in 2 others.[67,68,70] Concurrent or prior metastasis to the lung, lymph nodes, or bone had been present in most patients.

Common symptoms at presentation were dyspnea, wheezing, cough, and hemoptysis. On bronchoscopy, most of the tumors showed a polypoid growth into the lumen, which was pedunculated in a minority of cases.[69]

Histologically, most of the tumors had a subepithelial location, some extending deep in the tracheal wall and even surrounding soft tissue. The overlying epithelium was hyperplastic or ulcerated. No melanocytic junctional activity was found in the overlying tracheal epithelium in melanoma cases, which was in keeping with the metastatic nature of the tumors. The morphology was similar to primary cancer in all cases.

The fact that the primary tumor had been identified in all reported cases eases the differential diagnosis with primary tracheal tumors. Nevertheless, when dealing with primary malignant epithelial tumors of the trachea, especially adenocarcinomas, a metastatic disease should be considered in the differential diagnosis, particularly in cases in which sufficient clinical history is not provided.

The prognosis depends on a multitude of factors, including primary tumor site and type, tumor stage, and respiratory function following tracheal luminal narrowing.

References

1. Kaufman G, Klopstock R. Papillomatosis of the respiratory tract. Am Rev Resp Med 1963;88:839–46.
2. Popper HH, Wirsberger G, Juttner-Smoll FM, et al. The predictive value of human papilloma virus (HPV) typing in the prognosis of bronchial squamous cell papillomas. Histopathology 1992;21:323–30.
3. Di Mario AF, Montenegro H, Payne CB Jr, Kwon KH. Papillomas of the tracheobronchial tree with malignant degeneration. Chest 1978;74:464–5.
4. Runckel D, Kessler S. Bronchogenic squamous carcinoma in nonirradiated juvenile laryngotracheal papillomatosis. Am J Surg Pathol 1980;4:293–6.
5. Fechner RE, Fitz-Hugh GS. Invasive tracheal papillomatosis. Am J Surg Pathol 1980;4:79–80.
6. Dail D. Uncommon tumors. In: Dail DH, Hammar SP, editors. Pulmonary pathology. 2nd ed. New York: Springer-Verlag; 1994. p. 1299–300.
7. Hajdu SI, Huvas AG, Goodner JT, et al. Carcinoma of the trachea, clinicopathologic study of 41 cases. Cancer 1970;25:1448–56.
8. Salm R. Primary carcinoma of the trachea. A review. Br J Chest 1964;58:61–72.
9. Grillo HC, Mathisen DJ. Primary tracheal tumors: treatment and results. Ann Thorac Surg 1990;49:69–77.
10. Houston HE, Payne WS, Harrison EG, Olsen AN. Primary cancers of the trachea. Arch Surg 1969;99:132–40.
11. Bennets FE. Tracheal tumors. Postgrad Med J 1969; 45:446–54.
12. Theegarten D, Freitag L. Scar carcinoma of the trachea after tracheostomy. Case report and review of the literature. Respiration 1993;66:250–3.
13. Theegarten D, Philippou S, Freitag L. Tracheal carcinoma and mixed pneumoconiosis. A causal relationship? Respiration 1995;62:49–52.
14. Hatta T, Tsubota N, Matsubara M, et al. Two cases of metachronous cancer of the lung and trachea. Kyoba Geta 1988;41:422–5.
15. Shan L, Nakamura Y, Nakamura M, et al. Synchronous and metachronous multicentric squamous cell carcinoma in the upper aerodigestive tract. Pathol Intern 1997;47:68–72.
16. Saltarelli MG, Fleming MW, Wenig BM, et al. Primary basaloid squamous cell carcinoma of the trachea. Am J Clin Pathol 1995;104:594–8.
17. Thornley PE, Howtson SR. Primary carcinoma of the trachea: mixed squamous and oat cell type. Thorax 1980;35:72–3.
18. Gelder CM, Hetzel MR. Primary tracheal tumours: a national survey. Thorax 1993;48:688–92.
19. Dalby JE, Jones MN. Primary malignant growths of the trachea. Acta Otolaryngol 1961;53:12–20.
20. Zirkin HJ, Tovi F. Tracheal carcinoma presenting as a thyroid tumor. J Surg Oncol 1984;26:268–71.
21. Conti JA, Kemeney N, Klimstra D, et al. Colon carcinoma metastatic to the trachea. Report of a case and a review of the literature. Am J Clin Oncol 1994; 17:227–9.
22. Zerner J. Metastatic carcinoma (endometrial adenoacanthoma) to the trachea. Report of a successful resection and primary anastomosis. J Thorac Cardiovasc Surg 1975; 70:139–42.
23. Mori S, Shinoda M, Hatooka S, et al. A carcinoma arising from benign pleomorphic adenoma of the trachea. Kyobu Geka 1997;50:602–5.
24. Rotenberg D, Lawson VG, van Nostrand AW. Thyroid carcinoma presenting as a tracheal tumor. Case report and literature review with reflections on pathogenesis. J Otolaryngol 1979;8:401–10.
25. Sobin L, Yesner R. Histological typing of lung tumors. International histological classification of tumors. Vol 1. 2nd ed. Geneva: World Health Organization; 1981.
26. Downey RJ, Deschamps C, Asakura S, et al. Large cell carcinoma of the lung: results of surgical treatment. Lung Cancer 1994;11 Suppl:153.
27. Fields JN, Riguad G, Emami BN. Primary tumors of the trachea. Results of radiation therapy. Cancer 1989;15:2429–33.
28. Fishback N, Travis W, Moran C, et al. Pleomorphic (spindle/giant cell) carcinoma of the lung: a clinicopathological study of 78 cases. Cancer 1994;73:2936–45.
29. Onizuka M, Doi M, Mitsui K, et al. Undifferentiated carcinoma with prominent lymphocytic infiltration (so-called lymphoepithelioma) in the trachea. Chest 1990;98:236–7

30. Marigil MA, Pardo-Mindan FJ, Aliaga L, et al. Primary carcinoma of the trachea: combined small cell, squamous cell and giant cell carcinoma. Report of a case and review of the literature. Pathologica 1986; 78:99–105.
31. Passler C, Scheuba C, Prager G, et al. Anaplastic (undifferentiated) thyroid carcinoma (ATC). A retrospective analysis. Langenbecks Arch Surg 1999; 384:284–93.
32. Arrigoni MG, Woolner LB, Bernatz PE. Atypical carcinoid tumors of the lung. J Thorac Cardiovasc Surg 1972;64:413–21.
33. Hulka GF, Rothchild MA, Warner BW, Bove KE. Carcinoid tumor of the trachea in a pediatric patient. Otolaryngol Head Neck Surg 1996;114:822–5.
34. McCaughan BC, Martini N, Bains MS. Bronchial carcinoids. Review of 124 cases. J Thorac Cardiovasc Surg 1985;89:8–17.
35. Kharchenko VP, Galil-ogly GA, Kuz'min IV, et al. Melanocytic carcinoid of the trachea. Arch Pathol 1992;54:38–41.
36. Travis WD, Rush W, Flieder DW, et al. Survival analysis of 200 pulmonary neuroendocrine tumors with clarification of criteria for atypical carcinoid and its separation from typical carcinoid. Am J Surg Pathol 1998;22:934–44.
37. Colby TV, Koss MN, Travis WD. Carcinoid and other neuroendocrine tumors. In: Atlas of tumor pathology. Tumors of the lower respiratory tract. Washington (DC): Armed Forces Institute of Pathology; 1995. p. 287–317.
38. Tartour E, Caillou B, Tenebaum F, et al. Neuroendocrine tumor of the trachea of the intermediate type. Value of its individualization. Presse Med 1992;21:1905–8.
39. Colby TV, Koss MN, Travis WD. Small cell carcinoma and large cell neuroendocrine carcinoma. In: Atlas of tumor pathology. Tumors of the lower respiratory tract. Washington (DC): Armed Forces Institute of Pathology; 1995. p. 248–57.
40. Rostom AY, Morgan RL. Results of treating primary tumors of the trachea by irradiation. Thorax 1978;33:387–93.
41. van Heyningen C, Green AR, MacFarlane IA, Burrow CT. Oncogenic hypophosphatemia and ectopic corticotrophin secretion due to oat cell carcinoma of the trachea. J Clin Pathol 1994;47:82–2.
42. Ma CK, Fine G, Lewis J, Lee MW. Benign mixed tumor of the trachea. Cancer 1979;44:2260–6.
43. Ishida T, Kamachi M, Hanada T, et al. Mucous gland adenoma of the trachea resected with an endoscopic neodymium:yttrium aluminum garnet laser. Intern Med 1996;35:890–3.
44. Ferguson CJ, Cleeland JA. Mucous gland adenoma of the trachea: case report and literature review. J Thorac Cardiovasc Surg 1988;95:347–50.
45. Azar T, Abdulkarim FW, Tacher HM. Adenoid cystic carcinoma of the trachea. Laryngoscope 1998;108: 1297–300.
46. Maziak DE, Todd TR, Keshavjee SH, et al. Adenoid cystic carcinoma of the airway: thirty-two year experience. J Thorac Cardiovasc Surg 1996;112:1522–32.
47. Na DG, Han MH, Kim KH, et al. Primary adenoid cystic carcinoma of the cervical trachea mimicking thyroid tumor: CT evaluation. J Comput Assist Tumor 1995;19:559–63.
48. Nomori H, Kaseda S, Kobayashi K, et al. Adenoid cystic carcinoma of the trachea and main-stem bronchus. A clinical, histopathologic, and immunohistochemical study. J Thorac Cardiovasc Surg 1988;96:271–7.
49. Umeno H, Miyajina Y, Mori K, Nakashima T. Clinicopathological study of 54 cases of adenoid cystic carcinoma in the head and neck. Nippon Jibiinkoka Gakkai Kaiho 1997;100:1442–9.
50. Smetana HF, Iverson L, Swan LL. Bronchogenic carcinoma. Analysis of 100 autopsy cases. Milit Surg 1952;3:335.
51. Liebow AA. Tumors of the lower respiratory tract. In: Atlas of tumor pathology. Tumors of the lower respiratory tract. Washington (DC): Armed Forces Institute of Pathology; 1975. p. 26–53.
52. Yousem SA, Hochholzer L. Mucoepidermoid tumors of the lung. Cancer 1987;60:1346–62.
53. Schanmugarten K. Histological typing of tumors of the upper respiratory tract and ear. Berlin: World Health Organization; 1991. p. 35–6.
54. Lopez-Terrada D, Bloom MGK, Cagle PT, et al. Oncocytic mucoepidermoid carcinoma of the trachea. Arch Pathol Lab Med 1999;123:635–7.
55. Heitmiller RF, Mathisen DJ, Ferry JA, et al. Mucoepidermoid lung tumors. Ann Thorac Surg 1989;47:394–9.
56. Leonardi HK, Jung-Legg Y, Legg MA, Neptune WB. Tracheobronchial mucoepidermoid carcinoma. Clinicopathological features and results of treatment. J Thorac Cardiovasc Surg 1978;76:431–8.
57. Duarte IG, Gal AA, Mansour KA. Primary malignant melanoma of the trachea. Ann Thorac Surg 1998; 65:559–60
58. Kharchenko VP. Primary melanoma of the respiratory tract. Arch Pathol 1998;60:38–41.
59. Eri Z, Stanic J, Stanic M. Primary malignant melanoma of the trachea. Plucne Bolesti 1991;43:75–7.
60. Mori K, Cho H, Som M. Primary "flat" melanoma of the trachea. J Pathol 1977;121:101–5.
61. Divertie MB, Schmidt HW. Tracheal obstruction from metastatic carcinoma of the colon. Report of a case. Mayo Clin Proc 1954;29:403–5.
62. Yeh TJ, Batayias G, Peters H, Ellison RG. Metastatic carcinoma of the trachea. Report of a case of palliation by resection and Marlex graft. J Thorac Cardiovasc Surg 1965;49:886–92.
63. Garces M, Tsai E, Marsan RE. Endotracheal metastasis. Chest 1974;65:350–1.
64. MacMohan H, O'Connel DJ, Cimochowski GE. Pedunculated endotracheal metastasis. Am J Radiol 1978; 131:713–4.
65. Westerman DE, Urbanetti JS, Rudders RA, Fanburg BL. Metastatic endotracheal tumor from ovarian carcinoma. Chest 1980;77:798–800.
66. Baumgartner WA, Mark JB. Metastatic malignancies from distant sites to the tracheobronchial tree. J Thorac Cardiovasc Surg 1980;79:499–503.
67. Andrews AH Jr, Cardarelli DD. Carbon dioxide laser treatment of metastatic melanoma of the trachea and bronchi. Ann Otol 1981;90:310–1.
68. Wicks CM, Pos RH, Emerson GL, Kallay MC. Malignant melanoma metastatic to trachea. Clin Note Resp Dis 1982;21:14–6.
69. Franklin D, Miller RH, Kim Bloom MG, et al. Esthesioneuroblastoma metastatic to the trachea. Head Neck Surg 1987;10:102–6.
70. Nicolai P, Peretti G, Capiello J, et al. Melanoma metastatic to the trachea and nasal cavity: description of a case and review of the literature. Acta Otorhinolaryngol Ital 1991;11:85–92.
71. Ishiyama T, Aoyama T, Hirahara H, et al. Successful resection of endotracheal metastatic lung cancer using percutaneous cardiopulmonary support system: a case report. Kyobu Geka 2001;54:19–23.

3B Mesenchymal Tumors of the Trachea

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Mesenchymal neoplasms, also generally termed "soft tissue tumors," affect the trachea like any other organ or tissue of the body, because the trachea is surrounded by mesenchyme and has mesenchymal cells in its wall and mucosa. These include fibroblasts in the lamina propria, smooth muscle cells in the posterior membranous septum, and chondrocytes in the cartilage. Because of their rarity, mesenchymal lesions are rarely suspected, and the supposition is that a tracheal tumor is epithelial until proven otherwise. The distinction between benign and malignant mesenchymal lesions is in general less distinct than in epithelial lesions. In contrast to epithelial lesions, a greater proportion of mesenchymal lesions of the trachea are benign, or are of such low-grade malignancy that metastasis is unusual. Even malignant lesions, by virtue of their location and circumscription, are still generally amenable to resection and cure. Sampling problems tend to be greater in mesenchymal than in epithelial lesions, so an initial small biopsy is often only a signpost to the final diagnosis established after resection of the specimen.

Lymphomas are rare in the trachea and are included in this section.

Fibroblastic and Fibrohistiocytic Tumors

Fibroblastic and fibrohistiocytic tumors are perhaps the least delineated and clarified area in tracheal tumor pathology. Different names have been given to pathologically identical or very similar lesions, and different histopathologies have been lumped under single names. The confusion between fibroma, fibromatosis, and fibrosarcoma on the one hand, and between benign and malignant fibrous histiocytomas on the other, exemplify this situation. Inflammatory pseudotumor has also been added. It is our belief that the pathologic entities in this group generally should be equated to these lesions in general mesenchymal tumor pathology, unless meaningful data prove otherwise in the future.

Fibroma

Fibroma is found to be one of the most common benign tracheal tumors reported in the older literature, but it is rarely reported in recent publications.[1,2] We believe that most of the reported cases likely represented fibromatosis, low-grade fibrosarcoma, benign fibrous histiocytoma, inflammatory myofibroblastic tumor (inflammatory pseudotumor), or granulation and reactive fibroblastic tissues. Tracheal lesions consisting of fascicles of bland fibroblastic cells should be classified in one of the above-mentioned entities.

Fibromatosis

Fibromatosis, as proposed by Stout, consists of a broad group of benign fibrous proliferations of similar microscopic appearance that are intermediate in their biological behavior between benign fibrous lesions and fibrosarcoma.[3] Like fibrosarcoma, they are characterized by infiltrative growth and a tendency toward recurrence, but unlike this tumor, they never metastasize.[4] Fibromatosis of the trachea is less common than mediastinal fibromatosis invading the trachea. It probably represents most of the previously reported cases of "tracheal fibroma" and is more common in children.

This lesion usually presents as a subepithelial nodule, and is a benign proliferation of bland regular fibroblasts arranged in short fascicles. The margins are infiltrative. The overlying epithelium may show hyperplasia or squamous metaplasia.[5]

The major differential diagnosis is low-grade fibrosarcoma. Fibrosarcoma is usually more cellular, contains long fascicles of more pleomorphic fibroblasts, and exhibits more mitotic activity. Benign fibrous histiocytoma is distinguishable by the presence of histiocytes and inflammatory cells. Inflammatory myofibroblastic tumor (inflammatory pseudotumor) is separated by the presence of inflammatory cells and myofibroblastic differentiation in the proliferative cells.

Because of its infiltrative growth, tracheal fibromatosis commonly recurs after incomplete excision. Prognosis is good after complete resection.

Fibrosarcoma

Very few cases of primary tracheal fibrosarcoma have been reported in the literature, and most have been reported in children.[6,7] It occurs as a neoplastic proliferation of fibroblasts, producing a mass lesion that protrudes into the tracheal lumen. Histologically, tracheal fibrosarcoma consists of spindle cells with somewhat atypical oval nuclei, arranged in long fascicles and in a herringbone pattern (Figure 3-39 [Color Plate 5]). Mitotic figures are seen.

Differential diagnosis includes fibromatosis, benign and malignant fibrous histiocytomas, inflammatory myofibroblastic tumor, and other spindle cell tumors.

Treatment is the same as for fibromatosis, which involves complete excision. Although primary tracheal fibrosarcoma has the potential for metastasis, this did not occur in the few cases reported in the trachea, and prognosis has generally been good.

Benign Fibrous Histiocytoma

This tumor usually presents as a polypoid mass, protruding into the tracheal lumen.

Microscopically, it consists of a poorly circumscribed proliferation of elongated fibroblastic and polygonal histiocytic cells, intermixed with collagen fibers, with occasional multinucleated giant cells and dispersed lymphoid cells. A vague fascicular arrangement or a storiform pattern is often seen. Foam cells and siderophages are less frequently present. Cellular pleomorphism is minimal, and mitotic figures are scant.

A few cases of benign fibrous histiocytoma have been reported in the literature, mostly in children and young adults.[8–10] Cases of this tumor are classified in some publications under the general term "fibrous

histiocytoma" or "malignant fibrous histiocytoma," partly because of its infiltrative nature and recurrence after excision.

Differential diagnosis includes fibromatosis, inflammatory pseudotumor, neurofibroma, fibrosarcoma, and hemangiopericytoma. The distinction from malignant fibrous histiocytoma is often obvious, with the latter showing marked cellular pleomorphism, high mitotic activity, and foci of necrosis.

Benign fibrous histiocytoma is infiltrative at the margins and may recur after incomplete excision. The prognosis is good after proper excision.

Malignant Fibrous Histiocytoma

Very few cases of malignant fibrous histiocytoma (MFH) have been reported in the trachea. One case of postirradiation MFH was reported to have occurred 11 years after radiotherapy to the neck for thyroid papillary carcinoma.[11] It involved almost the entire length of the tracheal wall, and caused death in about 3 months. A co-occurrence of tracheal MFH with thymic carcinoma, producing separate lesions in the mediastinum, has also been reported.[12]

Histology shows pleomorphic spindle cells arranged in storiform and fascicular patterns.

Pleomorphic giant cells are dispersed and inflammatory cells may be seen. Mitotic figures are frequent and foci of necrosis may be present.

Differential diagnosis is mainly with other spindle cell sarcomas.

Muscle Tumors

Leiomyoma

Leiomyoma is a benign smooth muscle tumor that is far less common in the trachea than in bronchi and lung parenchyma. Approximately 21 cases of solitary leiomyoma of the trachea have been reported in the English and Japanese literatures. Multiple leiomyoma of the trachea, as well as other organs such as the esophagus and female genital tract, is associated with Alport's syndrome.[13] A case of esophageal leiomyomatosis involving the trachea has also been reported.[14]

Biology. In one series of 16 patients, the tumors appeared approximately equally in males and females.[15] The age range was 15 to 72 years, with two-thirds of the patients being above 40 years. The mean age was 49 years in another series of 20 cases.[16]

Pathology. More than one-half of the cases have been in the lower third of the trachea, and most have involved the posterior wall, where smooth muscle is most abundant. The tumors have been measured between 1 and 2.5 cm. The tumors usually protrude into the lumen as a nodule (Figure 3-40 [Color Plate 5]). Two cases were pedunculated.

Microscopically, leiomyoma consists of monomorphic spindle cells with blunt-ended nuclei, arranged in interlacing fascicles and storiform patterns. Significant mitotic activity and necrosis are absent (Figure 3-41 [Color Plate 6]).

Differential diagnosis includes leiomyosarcoma, as well as other spindle cell tumors such as hemangiopericytoma and fibrous histiocytoma.

Leiomyosarcoma

This malignant smooth muscle tumor is also rare in the trachea, with only 18 cases reported in the literature so far. It produces an intraluminal mass, which was pedunculated in 2 cases.[17,18] Most of the cases in one series were in the upper third of the trachea.[17]

Microscopically, the tumor consists of interlacing fascicles of spindle cells that are similar to leiomyoma. Distinguishing features from leiomyoma are cellular anaplasia, sometimes with the presence of uni- or multinucleated giant cells, high mitotic activity, and necrosis. The most helpful feature is the mitotic rate, which is more than 1 to 2 per 10 high-power fields, although in most cases, it is more than that; some believe it must be more than 5 per 10 high-power fields.[17–19]

Differential diagnosis includes other spindle cell sarcomas and sarcomatoid carcinomas.

Rhabdomyosarcoma

Rhabdomyosarcoma is a malignant mesenchymal neoplasm with differentiation toward skeletal muscle tissue. The tumor is generally seen in children. Two cases of this tumor in the trachea have been published in the English-language literature. One was in a 65-year-old man, with a polypoid mass in the tracheal lumen causing symptoms.[20] Histologically, the mass was inner to the fibrocartilaginous layer and covered by overlying epithelium. It consisted of anaplastic cells with large hyperchromatic nuclei, and abundant brightly eosinophilic cytoplasm, some showing cross-striations. The tumor was classified as a pleomorphic rhabdomyosarcoma. The second case occurred in a 12-year-old girl, also with an intratracheal polypoid mass. Histologically, this mass consisted of rather small, round, and spindled hyperchromatic cells, and was diagnosed as an embryonal rhabdomyosarcoma.[21] More common is the mediastinal rhabdomyosarcoma with pressure on the trachea.[22]

Lipomatous Tumors

Lipoma-Liposarcoma

Lipoma is a benign mesenchymal neoplasm of fat and is most common in the subcutis. In the usual type, it resembles mature fat, surrounded by a delicate capsule. It is exceedingly rare in the trachea, with only approximately 10 cases reported in the literature. Lipomas produce a polypoid mass covered by respiratory epithelium. One of the reported cases did not produce any symptoms and was found on autopsy,[23] whereas 2 other cases caused airway obstruction.[23–25]

Microscopically, tracheal lipomas are composed of lobules of mature adipocytes, separated by delicate fibrous bands. One case of a well-differentiated liposarcoma in the trachea occurred in a 76-year-old man.[26] It produced a 1 cm polyp, which was histologically composed of mature adipocytes with foci of atypicality. Recurrence or metastasis did not occur in 12 months of follow-up. The current trend is to classify these tumors as "atypical lipoma," because although recurrence occurs, they do not metastasize.[27] Since the recurrence rate is higher in retroperitoneal tumors, some prefer the term "well-differentiated liposarcoma" in that location.

The major differential diagnosis is a hamartoma. In a hamartoma, fat tissue may be prominent, but the presence of cartilage and epithelium-lined clefts help to make the diagnosis.

Cartilaginous Tumors

Chondroma

Chondroma is a benign tumor composed of cartilage and can rarely arise in the trachea. Approximately 10 cases of chondroma are reported in the trachea.[28,29] Grossly, it forms a hard bosselated mass protruding into the lumen.

Microscopically, it is a relatively hypocellular tumor, composed of chondrocytes that sit in lacunae of hyaline cartilage. The chondrocytes may be somewhat hyperchromatic and pleomorphic. Binucleation is exceptional. Foci of ossification may be seen. Differential diagnosis includes, most importantly, chondrosarcoma, as well as hamartoma and tracheopathia osteoplastica. Hamartomas are predominantly composed of hyaline cartilage, but presence of fat, occasional smooth muscle fibers, and epithelium-lined clefts occur. Tracheopathia osteoplastica is multiple and usually distinguishable on bronchoscopy.

Chondroblastoma

Chondroblastoma is a benign tumor of bone and rarely of soft tissue. It usually affects patients in the second decade of life. We have seen 1 case of chondroblastoma occurring in the trachea. To our knowledge, there is no comparable case in the literature.

Our patient was a 20-year-old male with a soft and friable, tan-yellow mass, protruding and partially obstructing the lumen. Grossly, there was hemorrhage and calcification in the tumor (Figure 3-42 [Color Plate 6]). Microscopically, the tumor was mainly in the submucosa. There was focal erosion of cartilage as well as extension to peritracheal adventitia. Sheets of mononuclear cells with the features of chondroblasts were present, including round or oval nuclei and pale eosinophilic cytoplasm. Nuclear grooves were seen in some cells (Figures 3-43, 3-44 [Color Plate 6]).

Occasional multinucleated giant cells were present. A chondroid matrix and mature cartilage were seen. The overlying epithelium showed reactive squamous metaplasia. The tumor was completely excised and did not recur, which is also usually the case with skeletal chondroblastomas.

Chondrosarcoma

Chondrosarcoma, the malignant counterpart of chondroma, is also rare in the trachea, with only 11 cases reported in the literature.[29,30] The age range of the patients was 32 to 87 years with an average of 65 years. The male to female ratio was 8:1.[30] Similar to chondroma, chondrosarcoma produces a hard intraluminal projection, which may be pedunculated. Tracheal obstruction may occur.

Microscopically, the tumor consists of cells with hyperchromatic and sometimes pleomorphic nuclei, and occasionally binucleated cells, some containing prominent nucleoli. The cellularity is increased compared to that of chondroma. The matrix is chondroid and may contain areas of myxoid change. Because mitoses are few and inconsistent, one cannot rely on mitotic activity to separate benign and malignant cartilaginous tumors in the same way as one does for the differentiation of benign from malignant smooth muscle tumors.

The major differential diagnosis is chondroma, which is often not an easy distinction. The criteria for malignancy are given above, but since chondrosarcomas of the larynx and trachea are usually low-grade and slow-growing, the cytologic changes may be subtle. Malignant nature of the tumor has been determined only after recurrence in some published cases. Some propose the term "cartilaginous tumor" for both chondroma and chondrosarcoma of this site because of the difficulty in separation. The treatment of both tumors is the same; that is, excision. After resectional surgery, chondromas and chondrosarcomas infrequently recur.

Vascular Tumors

Hemangioma

Capillary hemangioma affects the larynx and trachea of children far more commonly than it does adults, and because of the narrower airway lumen in this younger age group, it becomes symptomatic early in growth and causes obstructive symptoms such as stridor and dyspnea.[31] Most of the cases are well circumscribed, but some diffusely infiltrate the wall and cause narrowing over a segment, or grow into adjacent mediastinal tissues.[32] Conversely, a few cases of mediastinal hemangioma have been reported with infiltrative growth through the tracheal wall into the lumen.[33] Also published are cases of mediastinal and cervical hemangioma with compression of, but not infiltration into the trachea, causing symptoms.[34,35]

Histologically, capillary hemangiomas consist of closely packed capillary-sized blood vessels with marked endothelial proliferation, often with recognizable lobular architecture.

A major differential diagnosis is granulation tissue, which represents reparative and inflamed connective tissue rich in capillaries, formed in response to trauma or endotracheal intubation.[36] The distinction can be made by the presence of lobularity in hemangiomas, the absence of inflammation in deep areas in hemangiomas, and a history of trauma or endotracheal intubation in cases with granulation tissue.

Recurrence has been reported after incomplete excision.[33]

Kaposi's Sarcoma

Kaposi's sarcoma is a malignant vascular tumor seen in immunocompromised patients, particularly those affected with human immunodeficiency virus (HIV). When the trachea is involved, it is often in a disseminated form of disease with involvement of other sites such as bronchi, the larynx, palate, or distant organs like the skin.[37,38]

Biology. Predisposition to multifocal Kaposi's sarcoma may also develop in drug-induced immunosuppression, as occurred in the trachea and bronchi of a lung transplant patient receiving immunosuppressive therapy.[39] It is rarely seen in patients without immunosuppression.[40] Solitary lesions in the trachea have also been reported.[2] Hemorrhage into the trachea may dominate the clinical presentation.

Pathology. The endoscopic appearance may be suspicious for diagnosis without biopsy. Histologically, Kaposi's sarcoma is composed of proliferating spindle cells with slit-like vascular channels, extravasated red blood cells, and inflammatory cells. Cytoplasmic hyaline bodies may be seen in the cytoplasm of the malignant cells. Ectatic blood vessels may be seen in the surrounding tissue (Figures 3-45, 3-46, 3-47 [Color Plate 6]).

Differential diagnosis includes principally granulation tissue and spindle cell sarcomas.

Hemangiopericytoma

Hemangiopericytoma is a tumor of vascular pericytes, 4 cases of which have been reported in the trachea. Hemangiopericytoma is a potentially malignant tumor and its clinical behavior is not always possible to predict from morphology alone.

Microscopically, the tumor consists of closely-packed round to oval cells with scant cytoplasm, arranged around thin-walled "staghorn" blood vessels (Figure 3-48 [Color Plate 6]). A mitotic rate of generally more than 4 per 10 high-power fields is one indicator of malignant behavior, as was present in a published case, which recurred after primary excision, with massive infiltration of peritracheal cervical and mediastinal soft tissues.[41] Of the 4 cases reported, 2 had aggressive behavior and recurrence, so clinical follow-up is important.

One case of hemangiopericytoma arising in the mediastinum and compressing the trachea, and another case arising in the thyroid gland with invasion of the larynx, have been reported.[42]

Nerve Sheath Tumors

This category of tumors includes neurofibroma, schwannoma, and malignant peripheral nerve sheath tumor. These tumors are among the common tumors in the mediastinum, especially the posterior compartment. They may cause a pressure effect on mediastinal structures including the trachea. Neurofibromas and schwannomas are infrequent as endotracheal tumors. No case of malignant peripheral nerve sheath tumor has been reported in the trachea, but tracheal involvement has occurred in this tumor originating from the vagus nerve.[43]

Neurofibroma

Neurofibroma is a benign tumor of Schwann cell origin and fibroblasts. The axons present within the tumor are continuous with adjacent nerves.

Biology. Neurofibromas may occur as a solitary tumor in the trachea, or as part of von Recklinghausen's neurofibromatosis.[44] The two conditions occur with approximately equal frequency.[45] One case was reported as multiple neurofibromas of the trachea, bronchi, and esophageal wall.[46] The presence of multiple neurofibromas suggests von Recklinghausen's neurofibromatosis.

Pathology. Grossly, the tumor presents as an intraluminal polypoid mass, which is sessile or pedunculated.

Microscopically, the tumor consists of slender spindle cells, sometimes showing wavy nuclei, admixed with fibroblasts in a haphazard fashion. Neurofibromas may be cellular or contain myxoid areas. Mitotic activity is not present. The tumor margins are often ill defined. Differential diagnosis includes schwannoma, fibrous histiocytoma, and other spindle cell tumors.

Schwannoma (Neurilemoma)

Shwannoma is another tumor of Schwann cell origin with rare occurrence in the trachea.

Biology. Unlike neurofibromas, schwannomas occur as solitary lesions in the trachea, and are not associated with von Recklinghausen's neurofibromatosis. In a series of 12 patients, the age range was 6 to 71 years, with almost equal occurrence in males and females.[47]

Pathology. Schwannomas occur most frequently in the lower third of the trachea, followed by the upper and middle thirds.[47,48]

Grossly, schwannomas are encapsulated tumors which may be pedunculated or sessile, and in rare cases, are dumbbell-shaped with an extratracheal component (Figure 3-49 [Color Plate 7]). Schwannomas of the vagus nerve, although extratracheal, may involve the trachea.[49]

Microscopically, schwannoma consists of interlacing fascicles of spindle cells with hypercellular (Antoni type A) and hypocellular (Antoni type B) areas (Figures 3-50 [Color Plate 7]). Nuclear palisade (Verocay bodies) may be seen in hypercellular regions (Figures 3-51 [Color Plate 7]). Bizarre degenerative cells may develop as the tumor ages in a phenomenon termed "ancient schwannoma".

Differential diagnosis includes neurofibroma, fibrosarcoma, fibrous histiocytoma, and other spindle cell tumors. Malignant schwannoma occurs (Figure 3-52 [Color Plate 7]).

Granular Cell Tumor

Granular cell tumor is a tumor of Schwann cell origin and commonly occurs in the tongue, but it has also occurred throughout the body. The trachea is less commonly involved than the larynx and bronchi. Approximately 40 cases have been reported in the trachea.

Biology. In the largest published review of this tumor in the trachea, which comprised 30 cases, there was a female to male ratio of 6.5:1.[50] Blacks were more commonly affected than whites. The age range was between 6 and 56 years with a peak incidence in the fourth decade. In another published series of tracheal tumors in children, granular cell tumor followed after hemangioma as one of the most common benign tumors in this age group.[51]

Pathology. The tumor size is usually between 0.5 to 6 cm (Figure 3-53 [Color Plate 7]). The majority arise from the cervical trachea.[50] Twenty percent of the cases were multiple. Although most of the tumors (73%) were intraluminal lesions, 17% of cases grew extraluminally into surrounding tissues. This extraluminal

growth and potential to penetrate adjacent tissues or organs can cause diagnostic problems, with the tumor presenting as a neck mass, or thyroid or parathyroid nodule.[52–54]

Microscopically, the tumor consists of round, oval, or polyhedral cells with granular eosinophilic cytoplasm, and small and rather central nuclei, occasionally with conspicuous nucleoli (Figure 3-54 [Color Plate 7]). The cytoplasmic granules stain for PAS antigen and neuron specific enolase.

Squamous metaplasia with pseudoepitheliomatous hyperplasia is seen in the overlying epithelium. The cytoplasmic granules have characteristic features on electron microscopy. These are membrane-bound granules, also called "secondary lysosomes," the larger ones having a lamellated structure and the smaller ones a granular content. Malignant granular cell tumors are extremely rare, not exceeding 1 to 2% of all granular cell tumors. They have pleomorphic nuclei with increased mitotic activity and foci of necrosis. Only 1 case has been reported near the trachea, and that was in the retrotracheal space with multiple nodules in both lungs (presumably metastases).

Differential diagnosis includes oncocytoma, and neoplasms with oncocytic (Hurthle cell) change, oncocytic carcinoid tumors, and metastatic carcinomas with oncocytic change. The positive S-100 staining, negative neuroendocrine and keratin markers distinguish this tumor from carcinoid tumors and carcinomas.

Hurthle cell variants of thyroid adenomas and oncocytomas are important because cases of extraluminal growth may present as thyroid nodules. In fine needle aspiration of such a thyroid nodule, the cells contain large, granular eosinophilic cytoplasm resembling Hurthle cells, and may cause misinterpretation.[53] The distinction is by electron microscopy, which shows secondary lysosome rather than mitochondria of Hurthle cells. In malacoplakia, the cells are PAS negative with occasional Michaelis-Gutmann bodies.

Synovial Sarcoma

Synovial sarcoma is primarily a sarcoma of the extremities. It has been reported in the head and neck region on rare occasion and can occur in the pleura and the lung.[55] Only 1 case of this tumor has been reported in the trachea.[56] The patient was a 20-year-old asthmatic white male, who had worsening of respiratory symptoms necessitating bronchoscopy. On bronchoscopy, a smooth pale intratracheal mass was seen. Gross examination of the resected specimen showed extension to paratracheal tissue without fat invasion. The tumor was well circumscribed. No lymph node metastases were detected.

Microscopically, the tumor had a biphasic growth pattern, composed mostly of monomorphic spindle cells, but it also showed numerous foci of cuboidal cells forming pseudoglandular clusters. Immunohistochemistry showed vimentin positivity in the spindle cells and low molecular weight cytokeratin and carcinoembryonic antigen positivity in the epithelial component.

Differential diagnosis is mainly with spindle cell tumors, most of which are separated by the lack of a biphasic pattern. Even if this aspect is not evident on routine histology of synovial sarcomas, it could be highlighted by immunohistochemical studies, as described in this case. The biphasic epithelioid-spindle cell tumors are generally in the differential diagnosis of synovial sarcoma. Within this category in pulmonary pathology, are mixed mesotheliomas, which must be distinguished from primary synovial sarcoma of the pleura, a tumor more recently demonstrated in this location. Sarcomatoid carcinoma of the lung and pulmonary blastoma are also in this category and must be separated from primary or metastatic synovial sarcoma. However, the mentioned biphasic tumors are not reported in tracheae.

Hamartoma

Hamartoma, also called "cartilaginous hamartoma," or less often "benign mesenchymoma," is one of the common benign tumors of the lung, occurring mostly in lung parenchyma, with a minority occurring as bronchial

lesions. Approximately 10 cases have been reported in the trachea, including cases in adults and children.[57] Most of the cases had a polypoid appearance on endoscopy, causing obstruction of the lumen. One extraluminal hamartoma in a child presented as a neck mass attached to the trachea.[58] Two cases were associated with pulmonary hamartomas, one with a similar lesion in lung parenchyma, and the other with multiple tumors in bronchi and lung parenchyma.[59,60]

Microscopically, hamartomas consist of lobules of mature hyaline cartilage and other mesenchymal elements, including undifferentiated mesenchymal tissue, fat, smooth muscle, with entrapped epithelium-lined clefts. The major differential diagnoses are chondroma and tracheopathia osteoplastica. Chondroma, by definition, does not contain mesenchymal elements other than cartilage, and does not have epithelial elements. Tracheopathia osteoplastica usually produces multiple nodules, which carpet the mucosa and consist of columns of bone and cartilage forming struts internal to the tracheal rings. Mediastinal teratoma should be considered in the differential diagnosis of extraluminal tracheal hamartomas.

Paraganglioma (Chemodectoma)

Paraganglioma is a potentially malignant, usually low-grade tumor of the extra-adrenal paraganglia. Only 7 cases have been reported as primary in the trachea.[61,62] It is characterized by organoid nests of epithelioid cells (zellballen pattern) separated by delicate vascularized connective tissue. The cells contain round or oval vesicular or hyperchromatic nuclei and light eosinophilic cytoplasms that are sometimes vacuolated. Neurosecretory granules are demonstrable in the cytoplasm by electron microscopy.

Differential diagnosis includes carcinoid and glomus tumors. Carcinoid tumors show an organoid pattern with high vascularity, but usually also show other features like a trabecular pattern, pseudoglandular acini, and stippled nuclei. Keratin immunostain is positive in the majority of carcinoid tumors, but negative in paragangliomas. Positivity for neuroendocrine markers on immunohistochemistry, and neurosecretory granules on electron microscopy, are not helpful since paraganglioma shares these features with carcinoid tumor. Glomus tumor is another tumor to distinguish from paraganglioma, again because of its nesting pattern and vascularity. The differential point lies mainly on smooth muscle features in glomus cells.

The prognosis for tracheal paragangliomas has been reported to be good, with no case of recurrence reported after surgical excision.

Two cases of thyroid paraganglioma have been reported as behaving in an invasive manner, with secondary involvement of the trachea.[42,63] A paraganglioma arising in the superior mediastinum between the trachea and left subclavian artery, displacing these structures and extending upward to the neck, has also been reported.[62]

Glomus Tumor

This benign neoplasm, which primarily affects the skin, is rare in the trachea. There are approximately 14 cases reported in the literature, and we have encountered a case at Massachusetts General Hospital. The patients were in their fourth to seventh decades of life, and most were men. The tumor usually produces a polypoid mass, protruding into the lumen, and causes partial obstruction.

Histologically, and similar to other sites, the tumor shows nests of round monotonous cells with bland central nuclei and amphophilic or lightly eosinophilic cytoplasms, separated by ectatic thin-walled vascular channels (hence the synonymous term "glomangioma") (Figures 3-55, 3-56 [Color Plate 7]). The tumor cells, in some cases, form solid sheets with inconspicuous vascular channels and little stroma. The stroma is sometimes prominent with hyaline character, in which the tumor cells are embedded. One case of an oncocytic glomus tumor showing intensely eosinophilic cytoplasm has been reported.[64] The glomus cells, which are believed to be modified smooth muscle cells of the glomus body, are positive for desmin and smooth muscle actin immunohistochemically. Ultrastructural features include numerous pinocytotic vesicles, intracytoplasmic bundles of myofibrils, and focal electron dense bodies.[65]

The major differential diagnosis is carcinoid tumor, to which the cytology and architecture may have close resemblance.[66] The distinguishing features are the presence of neuroendocrine differentiation in carcinoid tumor, and the absence of smooth muscle differentiation on immunohistochemical and electron microscopic examinations. The behavior is benign, with no recurrence reported in the literature. The glomus tumor that we saw recurred 3 years after initial surgery.

Lymphomas

Lymphomas are exceedingly rare as primary tumors of the trachea. Somewhat more common is secondary involvement by a nodal lymphoma.

Non-Hodgkin's Lymphoma

Most of the cases of non-Hodgkin's lymphoma are secondary involvement by a nodal lymphoma. In the majority of these cases, neoplastic lymphoid tissue from peritracheal lymph nodes infiltrates the tracheal wall, causing stenosis of the tracheal lumen. Less frequently, non-Hodgkin's lymphoma may arise in the trachea. Excluding plasmacytomas, only about 10 cases of primary tracheal non-Hodgkin's lymphoma have been reported.[67] A few cases of direct invasion from adjacent structures such as the thyroid or thymus are also reported.[68]

Biology. Both primary and secondary tracheobronchial lymphomas occur in both genders, generally in the sixth and seventh decades.[67,69]

Pathology. Grossly, tumoral tissue may infiltrate the wall, causing narrowing and stenosis of the lumen, or protrude into the lumen as a polypoid mass.[67,69] A homogeneous, pale-tan, and fleshy surface is seen on cut section (Figure 3-57 [Color Plate 8]).

Microscopic appearance shows sheets of round or oval lymphoid cells, the details of which depend on the lymphoma type. In both secondary and primary lymphomas, low-grade lymphomas outnumber others. In primary lymphomas, low-grade B-cell lymphoma of mucosa associated lymphoid tissue (MALT), small lymphocytic lymphoma, lymphoplasmacytic lymphoma, and plasmacytoma have been reported. The changes in classification of non-Hodgkin's lymphomas, and increasing diagnostic accuracy with the advent of immunohistochemical and molecular genetic techniques, have affected the diagnosis and categorization of lymphoma cases. Some of these cases may have been categorized under "undifferentiated carcinoma" or "pseudolymphoma" in the past.

Major differential diagnoses include Hodgkin's lymphoma, small cell carcinoma, undifferentiated carcinoma, and benign exuberant lymphoid proliferation (the so-called "pseudolymphoma").

The frequency of progression of primary tracheal lymphoma to systemic lymphoma is not established. In the absence of systemic lymphoma, cases can be treated by surgery or by radiation.

Hodgkin's Lymphoma

Hodgkin's lymphomas occur in the trachea (Figure 3-58 [Color Plate 8]) mostly through direct extension of adjacent peritracheal lymph nodes, involved by the tumor, either at first presentation or in recurrence.[70,71] Many of the reported cases are in children. Tracheal involvement by a thymic Hodgkin's lymphoma has also been reported.[72]

References

1. Elman P, Whittaker H. Primary carcinoma of the trachea. Thorax 1947;2:153–62.
2. Culp OS. Primary carcinoma of the trachea. J Thorac Surg 1938;7:471–87
3. Stout AP. The fibromatoses. Clin Orthop 1961;19:11.
4. Weiss SW, Goldblum JR, editors. Fibromatosis. In: Enzinger and Weiss's soft tissue tumors. 4th ed. St. Louis: Mosby; 2001. p. 309–46.
5. O'Connell JE, Raafat F, Proops D. Congenital subglottic fibromatosis. J Laryngol Otol 1989;103:983–5.

6. Postovsky S, Peleg H, Ben-Itzhak O, Arush MW. Fibrosarcoma of the trachea in a child: case report and review of the literature. Am J Otolaryngol 1999;20:332–5.
7. Garnett JD, Cook CB. Primary bronchopulmonary fibrosarcoma of the trachea in a child. South Med J 1993;86:1283–5.
8. Sculerati N, Mittal KR, Greco MA, Ambrosino MM. Fibrous histiocytoma of the trachea: management of a rare case of upper airway obstruction. Int J Pediatr Otolaryngol 1990;19:295–301.
9. Gonzalez-Campora R, Matilla A, Sanchez-Garrillo JJ, et al. "Benign" fibrous histiocytoma of the trachea. J Laryngol Otol 1981;95:1287–92.
10. Cohen SR, Landing BH, Isaacs H. Fibrous histiocytoma of the trachea. Ann Otol Rhinol Laryngol Suppl 1978;87 Suppl 52:2–4.
11. Louie S, Cross CE, Amott TR, Cardiff R. Postirradiation malignant fibrous histiocytoma of the trachea. Am Rev Respir Dis 1987;135:761–2.
12. Wang YH, Wang JL, Lin AS, Chang L. Malignant fibrous histiocytoma of the trachea combined with thymic carcinoma: a case report. Chang Keng I Hsueh Tsa Chih 1993;16:271–4.
13. Renieri A, Bassi MT, Galli L, et al. Deletion spanning of 5' ends of both the COL4A5 and COL4A6 genes in a patient with Alport's syndrome and leiomyomatosis. Hum Mutat 1994;4:195–8.
14. Kumar P, Breach NM, Goldstraw P. Esophageal leiomyomatosis involving trachea: surgical resection and repair. Ann Thorac Surg 1997;63:531–3.
15. Douzinas M, Sheppard MN, Lennox SC. Leiomyoma of the trachea. Eur J Respir Dis 1987;71:206–9.
16. Shirakawa T, Takenaka S, Matsumoto T, et al. A case of leiomyoma of the trachea. Nippon Kyobu Shikkan Gakkai Zasshi 1991;29:1464–8.
17. Thedinger BA, Cheney ML, Montgomery WW, Goodman M. Leiomyosarcoma of the trachea. Case report. Ann Otol Rhinol Laryngol 1991;100:337–40
18. Firouz-Abadi A, Higgins JP. Leiomyosarcoma of the trachea. Otolaryngol Head Neck Surg 1993;108:184–6.
19. Qutzen KE, Lunding J, Jakobsen J. Leiomyosarcoma of the trachea. J Laryngol Otol 1986;100:979–84.
20. Ho KL, Rassekh ZS. Rhabdomyosarcoma of the trachea. First reported case. Hum Pathol 1980;11 Suppl:572–4.
21. Kedar A, Centrel G, Rosen G. Rhabdomyosarcoma of the trachea. J Laryngol Otol 1988;102:735–6.
22. Azizkhan RG, Dudgeon DL, Buck JR, et al. Life threatening airway obstruction as a complication to the management of mediastinal masses in children. J Pediatr Surg 1985;20:816–22.
23. Hauptman S. Lipoma of the trachea. Z Erkr Atmungsorgane 1989;173:238–41.
24. Tayama K, Takai E, Ueda T, et al. Tracheal lipoma obstructing the right main bronchus: report of a case. Surg Today 1996;26:1017–9.
25. Chen TF, Braidley PC, Shneerson JM, Wells FC. Obstructing tracheal lipoma: management of a rare tumor. Ann Thorac Surg 1990;49:137–9.
26. Van Den Deukel JT, Wagenaar SJ, Vanderschueren R. Liposarcoma of the trachea. Thorax 1979;34:817–8.
27. Weiss SW, Rao VK. Well differentiated liposarcoma (atypical lipoma) of deep soft tissue of extremities, retroperitoneum, and miscellaneous sites: a follow-up study of 92 cases with analysis of the incidence of dedifferentiation. Am J Surg Pathol 1992;16:1051–8.
28. Jortay M, Bisschop P. Chondroma of the trachea. Acta Otolaryngol Belg 1998;52:247–51.
29. Neis RR, McMahon MF, Norris CW. Cartilaginous tumors of the trachea and larynx. Ann Otol Rhinol Laryngol 1989;98:31–6.
30. Farrell ML, Michael L. Tracheal chondrosarcoma: a case report. Head Neck 1998;20:568–72.
31. Dennie CJ, Coblentz CL. The trachea: pathologic conditions and trauma. Can Assoc Radiol J 1993;44:157–67.
32. Bailey CM, Froehlich P, Hoeve HL. Management of subglottic hemangioma. Laryngol Otol 1998;112:765–8.
33. Messineo A, Wesson DE, Filler RM, Smith CR. Juvenile hemangiomas involving the thoracic trachea in children: report of two cases. J Pediatr Surg 1992;27:1291–3.
34. Schwartz MZ. Maintenance of the placental circulation to evaluate and treat an infant with massive head and neck hemangioma. J Pediatr Surg 1993;28:520–2.
35. Yuasa K, Shimizu T, Toyoda T. The giant cavernous hemangioma of the neck. Nippon Kyobu Geka Gakkai Zasshi 1992;40:1274–8.
36. Mills SE, Cooper PH, Fechner RE. Lobular capillary hemangioma: the underlying lesion of pyogenic granuloma. A study of 73 cases from the oral and nasal mucous membranes. Am J Surg Pathol 1980; 4:470–9.
37. Belda J, Canalis E, Gimferrer JM, et al. Subglottic stenosis in an HIV positive patient: an exceptional form of clinical presentation in Kaposi's sarcoma. Eur J Cardiovasc Surg 1997;11:191–3.
38. Miller RF, Tomlinson MC, Cottoril CP, et al. Bronchopulmonary Kaposi sarcoma in patients with AIDS. Thorax 1992;47:721–5.
39. Sleiman C, Mal H, Roue C, et al. Bronchial Kaposi's sarcoma after single lung transplantation. Eur Resp J 1997;10:1181–3.
40. Rajaratnam K, Desai S. Kaposi's sarcoma of the trachea. J Laryngol Otol 1988;102:951–3.
41. Gavilan J, Rodriguez-Peralto JL, Tomas MD, et al. Hemangiopericytoma of the trachea. J Laryngol Otol 1987;101:738–42.
42. Tytor T, Olofsson J. Thyroid tumors invading the larynx and trachea. J Otolaryngol 1986;15:74–9.
43. Fukai I, Masaoka A, Yamakawa Y, et al. Mediastinal malignant epithelial schwannoma. Chest 1995;108:574–5.
44. Meredith HC, Valicenti JF. Solitary neurofibroma of the trachea. Br J Radiol 1978;51:218–9.
45. Lossos IS, Breuer R, Lafair JS. Endotracheal neurofibroma in a patient with von Recklinghausen's disease. Eur Respir J 1988;1:464–5.
46. Fischberg C, Cotting J, Hack I, et al. Fatal double tracheoesophageal vascular compression and neurofibromatosis. Arch Pediatr 1996;3:1253–7.
47. Horovitz AG, Khalil KJ, Verani RR, et al. Primary intratracheal neurilemoma. J Thorac Cardiovasc Surg 1983;85:313–7.
48. Rusch VW, Schmidt RA. Tracheal schwannoma: management by endoscopic laser resection. Thorax 1994; 49:85–6.
49. Yano T, Hara N, Ichinose Y, et al. An intrathoracic vagus nerve schwannoma invading the trachea. Surg Today 1993;23:1113–5.
50. Burton DM, Heffner DK, Patow CA. Granular cell tumor of the trachea. Laryngoscope 1992;102:807–13.
51. Desai RC, Holinger LD, Gonzalez-Crussi F. Tracheal neoplasms in children. Ann Otol Rhinol Laryngol 1998;107:790–6.
52. Gorman RC, Lights V, Furth E, Torosian MH. Unique presentation of granular cell tumor as a paratracheal mass. Oncol Rep 1998;5:1551–4.
53. Kintanar EB, Giordano TJ, Thompson NW, Michael CW. Granular cell tumor of the trachea masquerading as Hurthle-cell neoplasm on fine-needle aspirate: a case report. Diagn Cytopathol 2000;22:379–89.
54. Yang SW, Hong SW, Cho MW, Kang SJ. Malignant granular cell tumor at the retrotracheal space. Yonsei Med J 1999;40:76–9.

55. Amble FR, Olsen KD, Nascimento AG, Foote RL. Head and neck synovial cell sarcoma. Otolaryngol Head Neck Surg 1992;107:631–7.
56. Sykes At, Rokkas CK, Kajdacsy-Balla A, Haasler GB. Primary tracheal synovial cell sarcoma: a first case report. J Thorac Cardiovasc Surg 1997;114:678–80.
57. Nakayama DK, Harrison MR, de Lorimier AA, et al. Reconstructive surgery for obstructive lesions of the intrathoracic trachea in infants and small children. J Pediatr Surg 1982;17:854–68.
58. Gross E, Chen MK, Hollabagh RS, Joyner RE. Tracheal hamartoma: report of a child with a neck mass. J Pediatr Surg 1996;31:1584–5.
59. Suzuki N, Ohno S, Ishii Y, Kitamura S. Peripheral intrapulmonary hamartoma accompanied by a similar endotracheal lesion. Chest 1994;106:1291–3.
60. Dominguez H, Hariri J, Pless S. Multiple pulmonary chondrohamartomas in trachea, bronchi and lung parenchyma. Respir Med 1996;90:111–4.
61. Sing TM, Wong KP, Young N, Despas P. Chemodectoma of the trachea. Thorax 1996;51:341–2.
62. Gallimore AP, Goldstraw P. Tracheal paraganglioma. Thorax 1993;48:866–7.
63. Mitsudo SM, Grajower MM, Balbi H, Silver C. Malignant paraganglioma of the thyroid gland. Arch Pathol Lab Med 1987;111:378–80.
64. Shin DH, Park SS, Lee LH, et al. Oncocytic glomus tumor of the trachea. Chest 1990;98:1021–3.
65. Ito H, Motohiro K, Nomura S, Tahara E. Glomus tumor of the trachea. Immunohistochemical and electron microscopic studies. Pathol Res Pract 1988;183:778–84.
66. Menaissy YM, Gall AA, Mansour KA. Glomus tumor of the trachea. Ann Thorac Surg 2000;70:295–7.
67. Fidias P, Wright C, Harris NL, et al. Primary tracheal non-Hodgkin's lymphoma. Cancer 1996;77:2332–8.
68. Zahradka W, Wuttke WD, Gehrandt F. Primary lymphoblastic lymphoma of the thyroid. Z Gesamte Inn Med 1990;45:58–60.
69. Kaplan MA, Pettit CL, Zukerberg LR, Harris NL. Primary lymphoma of the trachea with morphologic and immunophenotypic characteristics of low-grade B-cell lymphoma of mucosa-associated lymphoid tissue. Am J Surg Pathol 1992;16:71–5.
70. Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 43-1984. A 35-year-old woman with tracheal stenosis after treatment for Hodgkin's disease. N Engl J Med 1984; 311:1105–13.
71. Tanka H, Nakahara K, Sakai S, et al. A case of Hodgkin's disease with endotracheal tumor presenting with severe airflow obstruction. Nippon Kyobu Shikkan Gakkai Zasshi 1992;30:1732–7.
72. Fujimura Y, Kobayashi T, Nawata S, et al. A surgical case of Hodgkin's lymphoma originated from thymus. Nippon Kyobu Geka Gakkai Zasshi 1990;38:126–9.

3C Tumor-Like Lesions of the Trachea

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There are certain infectious, inflammatory, or reactive processes that can cause various degrees of tracheal lumen obstruction by focal nodular or polypoid protrusion into the lumen, or more extensive tracheal wall thickening and luminal narrowing. Infections such as tuberculosis and histoplasmosis, inflammatory lesions such as sarcoidosis, and reactive processes such as post-traumatic granulation tissue formation and fibrosis are amongst these conditions. Herein, we address several of these lesions that merit special consideration on histopathological grounds, and because they are more likely to be mistaken for tracheal tumors on endoscopy.

Inflammatory Myofibroblastic Tumor (Inflammatory Pseudotumor)

Inflammatory myofibroblastic tumor, also called "inflammatory pseudotumor," "plasma cell granuloma," and a battery of other names, is a pseudosarcomatous proliferation of myofibroblastic cells forming a mass lesion. The most common site is the lung, but cases in many other organs including skin, soft tissues, and gastrointestinal tract have been reported. Various etiologies such as inflammatory stimuli and trauma have been proposed, but current data suggest a benign neoplastic process in the lung and in many other organs.[1,2] This differs from myofibroblastic proliferation of the genitourinary tract, which is believed to be reactive and non-neoplastic.[3] Although these lesions in the trachea may be neoplastic, like pulmonary inflammatory myofibroblastic tumors, the exact nature remains to be clarified.

Biology. Inflammatory pseudotumor of the trachea has been reported in all ages, from infancy and childhood to late adulthood.[4–7] It affects both men and women.

Pathology. Inflammatory myofibroblastic tumor typically produces a rather circumscribed polypoid mass in the trachea, which can be sessile or pedunculated.[3]

Microscopically, the lesion is located in the tracheal wall and is covered by reactive or ulcerated epithelium. It consists of proliferated spindle cells arranged more or less in fascicular pattern. These cells contain regular nuclei with open chromatin and minimal pleomorphism. Mitotic figures are not abundant. A variety of inflammatory cells, including lymphocytes, plasma cells, histiocytes, neutrophils, and eosinophils, are infiltrated. Foci of myxoid change may be present. Necrosis does not occur.

Differential diagnosis includes a number of benign and malignant soft tissue tumors such as fibromatosis, benign fibrous histiocytoma, fibrosarcoma, leiomyoma, leiomyosarcoma, neurogenic tumors, hemangiopericytoma, or less frequently, other spindle cell tumors. As the designation "pseudosarcomatous" implies, the histology may closely resemble spindle cell sarcomas. The weak or focally positive immunohistochemical staining for desmin and smooth muscle actin, which is due to myofibroblastic differentiation, distinguishes this lesion from fibromatosis, fibrous histiocytoma, fibrosarcoma, and hemangiopericytoma, all of which lack these markers. The positivity for desmin and smooth muscle actin is much stronger in smooth muscle tumors.

The negative immunostain for S-100 antigen helps in differentiation from neurogenic tumors, which are positive for this marker. Although malignant fibrous histiocytoma is generally among the differential diagnoses, the absence of marked pleomorphism, high mitotic activity, and necrosis rules out this tumor and other high-grade sarcomas.

Intratracheal Thyroid

Although total thyroid ectopia is an extremely rare anomaly, ectopic thyroid tissue has been reported in different organs with the normal thyroid gland in place as well. These include lingual, sublingual, thyroglossal, laryngeal, tracheal, mediastinal, and diaphragmatic sites. Although more than 100 cases were published in Europe, mostly from Germany, only about 25 cases have been reported in the English-language literature.[8]

Biology. In a review of 23 cases, the patients ranged in age from newborn to 56 years, with a mean of 28.3 years, but most were young adults.[9] Females outnumbered males in a ratio of 3.8:1. Clinical presentation varied from asymptomatic cases found on autopsy, to symptoms such as cough, wheezing, stridor, and dyspnea. Symptoms worsened in 3 patients during pregnancy, 1 of them also with menses.[9,10] Another patient had increasing wheezing during labor and died postpartum because she had been treated for asthma and was not suspected to have an intratracheal mass.[10] Increased dyspnea was noted in a girl with menarche. These have led some to suggest hormonal stimulation as a basis for ectopic tissue enlargement during phases of altered female sex hormone production. Several theories have been proposed to explain aberrant location of thyroid tissue including "malformation" and "ingrowth " theories.[11]

Pathology. Grossly, most of the cases are single or multiple nodules or plaques in the tracheal wall, often visible endoscopically as submucosal masses. These usually measure 1 to 3 cm in dimension but may be as large as 5 cm.[12] The most common location is the posterolateral wall of the upper trachea.

Microscopically, this condition often consists of normal thyroid tissue with follicles and intervening stroma. Goiterous enlargement has been observed in some cases, with an associated thyroid gland goiter in 75% of cases.[13] Connection to the thyroid gland with a strand of thyroid tissue has been seen in some cases.

Malignant change may occur in the intratracheal thyroid, and poses a diagnostic problem to the pathologist as to whether the lesion has arisen from intratracheal thyroid or represents invasion by thyroid gland carcinoma. Much more common is direct invasion of papillary carcinoma of the thyroid, usually through the tracheal anterolateral wall directly into the tracheal lumen, where it may cause obstruction.

The area of penetration is between the cartilaginous plates, where blood vessels, lymphatics, and nerves course perpendicular to the tracheal lumen.[14] Biopsy of such tumors may show the characteristic features of papillary carcinoma of the thyroid, for example, nuclear grooves, nuclear overlap with molding, and intranuclear pseudoinclusions. Because of the possibility of surgical resection of a papillary carcinoma invading the trachea in the absence of distant metastases, a staging system has been devised (see Chapter 8, "Secondary Tracheal Neoplasms").[14]

Amyloidosis

Amyloidosis may involve any site in the upper or lower respiratory tract, the larynx being the most common site. It may also affect the esophagus and mediastinal nodes, or produce mass lesions in the neck. Tracheal involvement may occur as part of a generalized amyloidosis, whether primary or secondary, or in a localized form, which itself can be primary or secondary to plasmacytoma.

Biology. The primary localized form affects adults, and only exceptional cases are reported in children.[15] The other forms follow the age distribution of generalized amyloidosis and solitary tracheal plasmacytomas. Tracheal obstruction may occur in severe forms and the obstruction may lead to infections and respiratory failure. Bleeding, although not frequent, may be massive and even fatal, as occurred in a patient with laryngeal amyloidosis.[16] The bleeding problem is probably related to blood vessels, which themselves may have amyloid in their walls and may not constrict normally.

Pathology. Gross appearance, as seen on bronchoscopy, varies from diffusely heaped up edematous mucosa to single or multiple nodules protruding into the lumen. Macroscopically, tracheal amyloidosis has a firm grey, waxy cut surface (Figure 3-59 [Color Plate 8]).

Microscopically, nodules of amorphous, lightly eosinophilic material are deposited in the mucosa (Figure 3-60 [Color Plate 8]) and large deposits may erode into cartilage. Scant chronic inflammation and fibrosis may be present. Vascular amyloidosis may be noted. Where amyloid extends deeply, it may involve the mucosal glands, producing globules of eosinophilic material replacing the atrophic gland acini. Cartilaginous change, calcification, and osseous metaplasia may occur. Foreign body giant cells and granulomas often surround the amyloid. In localized tracheal amyloidosis secondary to solitary plasmacytoma, amyloid material usually comprises a minority of the tumoral tissue, but it may be even more extensive than the neoplastic plasma cells.

Histochemical staining with Congo red shows apple-green birefringence under polarized light, which confirms the diagnosis (Figure 3-61 [Color Plate 8]). Other stains such as methyl violet or thioflavin-T may also be used. Electron microscopy shows the typical fibrillar structure of amyloid. Immunoglobulin light chains were present and mixed with amyloid in one case. Bronchoscopic differential diagnoses include tracheopathia osteoplastica, relapsing polychondritis, inflammatory lesions, and tumors.

With regard to histology, amyloid deposits may be replaced with cartilage and bone. It must be distinguished from other lesions with cartilage and bone, particularly tracheopathia osteoplastica, but also from degenerative and reparative changes that can occur with tracheomalacia. Foreign body reaction to the amyloid should not be mistaken for granulomatous diseases like tuberculosis and sarcoidosis.

Prognosis is generally good in primary localized forms, but the risk of bleeding exists. Laser treatment or surgical excision may be necessary in obstructive lesions. Nodular tracheal amyloidosis may cause obstruction, and postobstructive infection may develop. In diffuse tracheobronchial amyloidosis, postobstructive infection and respiratory failure may occur. In generalized amyloidosis, pulmonary involvement with pulmonary hypertension, cardiac involvement with arrhythmias and failure, or other complications may complicate the clinical course.

Rheumatoid Nodule

A case of tracheal rheumatoid nodules in a 45-year-old Chinese man has been reported.[17] The patient had suffered rheumatoid arthritis for 7 years, with articular manifestations and deformities. Subcutaneous rheumatoid nodules had been present over his elbows and hands.

Endoscopy showed four smooth, whitish nodules, each 3 to 5 mm in diameter. Further specification was not possible on endoscopic findings. Histologic examination showed foci of necrobiosis and vascular fibrinoid necrosis in the vessels surrounded by palisading histiocytes.

Among the histologic differential diagnoses are necrotizing granulomatous diseases such as tuberculosis and histoplasmosis. Amyloid deposits must also be separated from central necrobiosis of this lesion, which may have a similar eosinophilic patternless aspect. Amyloid material, however, is more homogeneous, and stains with Congo red and other special histochemical stains.

Tracheopathia Osteoplastica

Tracheopathia osteoplastica, also known as "tracheobronchopathia osteochondroplastica," is an infrequent disease of the trachea and major bronchi. It produces multiple osseous and cartilaginous projections into the airway lumen.

Biology. The disease affects an age range from 12 to 72 years, with the peak being in the fifth and sixth decades. No gender predominance has been present. Familial occurrence is exceptional but is reported in a mother and daughter.[18] Various theories have been proposed to explain the pathogenesis of this disease, including the outgrowth of normal cartilaginous rings causing ecchondrosis and exostosis, and abnormalities in tracheal elastic tissue causing metaplastic cartilage and bone formation.[19,20] More recently, chronic infection, chemical or mechanical irritation, metabolic abnormalities, and genetic predisposition have been postulated, but the exact cause is unknown.[18,21] Although some earlier reports have associated this disease with tracheobronchial amyloidosis, most patients lack amyloid deposits in airways.[22,23] Many patients are asymptomatic, especially early in the course and in localized forms. Symptomatic patients may have dyspnea, cough, wheezing, stridor, or hemoptysis. Postobstructive pneumonia may develop if significantly symptomatic cases are not diagnosed early in their course.[18]

Pathology. Grossly, lesions are seen bronchoscopically as irregular whitish nodules carpeting the airway lumen, producing a "rock garden" appearance (Figure 3-62 [Color Plate 8]). It is most prominent in the lower two-thirds of the anterolateral tracheal wall and often extends to major bronchi.

Microscopically, cartilaginous and osseous submucosal nodules are seen, covered by a normal or metaplastic squamous epithelium (Figures 3-63, 3-64 [Color Plate 8]). Foci of calcification are sometimes seen. Inflammation is usually absent or sparse, and surface ulceration is not extensive.

Differential diagnoses of bronchoscopic findings include papillomatosis and tumors (especially in localized forms of tracheopathia osteoplastica), and infections such as tuberculosis, sarcoidosis, and others. Microscopic differential diagnosis may include relapsing polychondritis. Osseous metaplasia has also been seen in carcinomas, carcinoid tumors, and tuberculosis. Multinodular amyloidosis may simulate tracheopathia osteoplastica when it has secondary ossification, or it may be part of tracheopathia osteoplastica. The lack or scarcity of inflammation in tracheopathia osteoplastica, and the continuity of a strutwork of osteocartilaginous nodules inner to the tracheal cartilages, help in its differentiation from inflammatory lesions and post-traumatic ossification. Prognosis is usually good, particularly in asymptomatic and localized lesions. Tracheoplasty or surgical resection may be required in extensive and obstructive lesions (see Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions" and Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteopathica, Tracheal Compression, and Staged Reconstruction of the Trachea").

References

1. Pettinato G, Manivel JC, De Rosa N, Dehner LP. Inflammatory myofibroblastic tumor (plasma cell granuloma). Clinicopathologic study of 20 cases with immunohistochemical and ultrastructural observations. Am J Clin Pathol 1990;94:538–46.
2. Weiss SW, Goldblum JR, editors. Inflammatory myofibroblastic tumor. In: Enzinger and Weiss's soft tissue tumors. St Louis: Mosby; 2001. p. 383–9.
3. Weiss SW, Goldblum JR, editors. Benign fibrous tissue tumors. In: Enzinger and Weiss's soft tissue tumors. St. Louis: Mosby; 2001. p. 274–5.
4. Dewar AL, Connett GJ. Inflammatory pseudotumor of the trachea in a ten-month-old infant. Pediatr Pulmonol 1997;23:307–9.
5. Aijaz F, Salam AU, Muzaffar S, et al. Inflammatory pseudotumor of the trachea: report of a case in an eight year old child. J Laryngol Otol 1994;108:613–6.
6. Barker AP, Carter MJ, Matz LR, Armstrong JA. Plasma cell granuloma of the trachea. Med J Aust 1987;146:443–5.
7. Ishii Y, Inoue F, Kamikawa Y, et al. A case report of tracheal inflammatory pseudotumor. Nippon Kyobu Geka Gakkai Zasshi 1993;41:672–7.
8. Myers EN, Panlango IP Jr. Intratracheal thyroid. Laryngoscope 1975;85:1833–40.
9. Brandwein M, Som P, Urken M. Benign intratracheal thyroid, a possible cause for preoperative overstaging. Arch Otolaryngol Head Neck Surg 1998;124:1266–9.
10. Ferlito A, Giarelli L, Silvestri F. Intratracheal thyroid. J Laryngol Otol 1988;102:95–6.
11. Chanin LR, Greenberg LM. Pediatric upper airway obstruction due to ectopic thyroid: classification and case reports. Laryngoscope 1998;98:422–7.
12. Thoren L. Intratracheal goiter. Acta Chir Scand 1947; 95:455–512.
13. Dowling EA, Johnson IM, Collier FD, et al. Intratracheal goiter: a clinicopathological review. Ann Surg 1962;156:258–67.
14. Shin DH, Mark EJ, Suen MC, Grillo HC. Pathologic staging of papillary carcinoma of the thyroid with airway invasion based on the anatomic manner of extension to the trachea. Hum Pathol 1993;24:866–70.
15. Plaza Martos JA, Garcia MH, Garzon MA. Amyloid tumor of the trachea. Presentation of a pediatric case. Ann Esp Pediatr 1989;30:61–2.
16. Chow LT, Chow WH, Shum BS. Fatal massive upper respiratory tract hemorrhage: an unusual complication of localized amyloidosis of the larynx. J Laryngol Otol 1993;107:51–3.
17. Ip SM, Wong MP, Wong KL. Rheumatoid nodules in the trachea. Chest 1993;1:301–3.
18. Young RH, Sandstrom RE, Mark GJ. Tracheopathia osteoplastica. J Thorac Cardiovasc Surg 1980;79:537–41.
19. Virchow R. Die Krankhaften Gesch wulste. Berlin: Hirschwald 1863;1:442–3.
20. Aschoff-Freiburg L. Ueber tracheopathia osteoplastica. Ver Dtsch Ges Pathol 1910;14:125–7.
21 Prakash UBS, McCullogh AE, Edell ES, Nienhuis DM. Tracheobronchopathia osteochondroplastica: familial occurrence. Mayo Clin Proc 1989;64:1091–6.
22. Sakula A. Tracheobronchopathia osteochondroplastica: its relationship to primary tracheobronchial amyloidosis. Thorax 1968;23:105–10.
23. Martin CJ. Tracheobronchopathia osteochondroplastica. Arch Otolaryngol 1974;100:290–3.

Imaging the Larynx and Trachea

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Alfred L. Weber, MD

Normal Anatomy

Larynx

The larynx is divided into supraglottic, glottic, and subglottic parts, whereas the trachea is composed of the cervical extrathoracic trachea and the mid and lower intrathoracic trachea.[1,2] The supraglottic portion of the larynx is constituted by the epiglottis, aryepiglottic folds, arytenoids, and false cords. The glottic portion of the larynx is made up of the laryngeal ventricles and both vocal cords. The crescent-shaped laryngeal ventricles are situated between the false and true cords as lateral invaginations of mucosa. The subglottic space extends from the undersurface of the vocal cords to the inferior margin of the cricoid cartilage, which is also the lower boundary of the larynx. This subglottic area is oval in shape and measures about 1.5 to 2 cm in length (Figures 4-1 through 4-4).[3–5]

Trachea

The cervical trachea extends from the inferior margin of the cricoid cartilage to the thoracic inlet, and the intrathoracic trachea extends from the thoracic inlet to the carina where it divides into the right and left main bronchi.[6,7] There are 14 to 22 (mean 17) C-shaped hyaline tracheal cartilage rings that support the anterior and lateral tracheal walls. The cartilaginous portion of each tracheal ring forms a "C" with the membranous portion found posteriorly, which is unsupported by cartilage. The first ring is partly recessed into the broader ring of the cricoid cartilage. The cartilaginous rings are usually semicircular or horseshoe-shaped and are the chief determinant of cross-sectional shape. Tracheal diameter grows from 3 to 4 mm in infancy to about 20 mm in adulthood.[8,9] The cervical trachea is subject to atmospheric pressure and the intrathoracic trachea is subject to intrathoracic pressure that is equivalent to pleural pressure during quiet breathing. During forced expiration or coughing, the intrathoracic pressure becomes greater than atmospheric pressure, increasing the compressive transmural pressure that narrows the intrathoracic trachea.

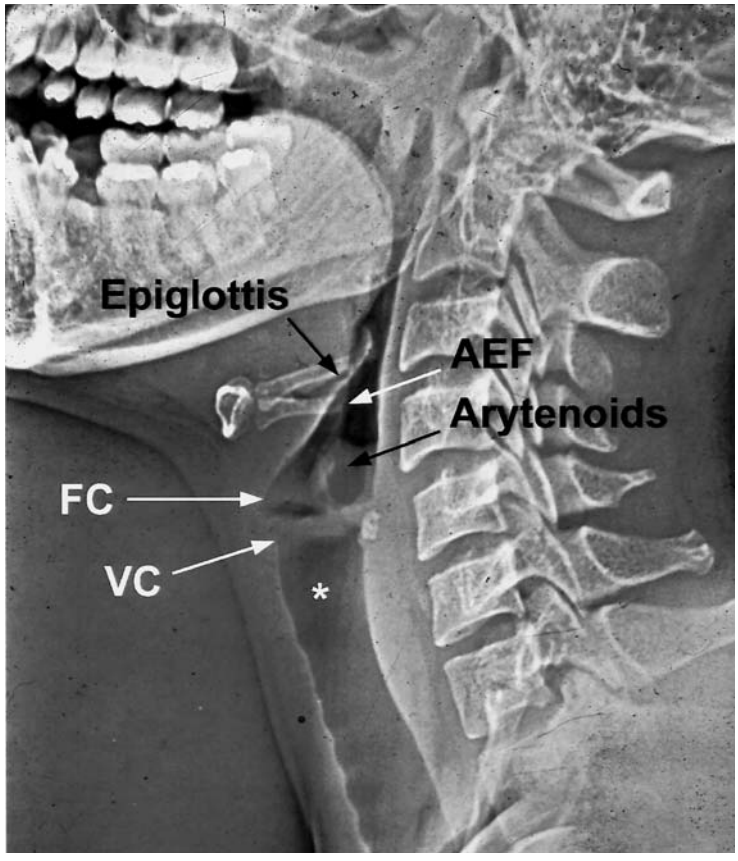


FIGURE **4-1** *Normal laryngeal anatomy. Normal lateral view of the neck demonstrates the epiglottis, aryepiglottic folds* (AEF), *false vocal cords* (FC), *true cords* (VC), *and subglottic space (*asterisk*).*

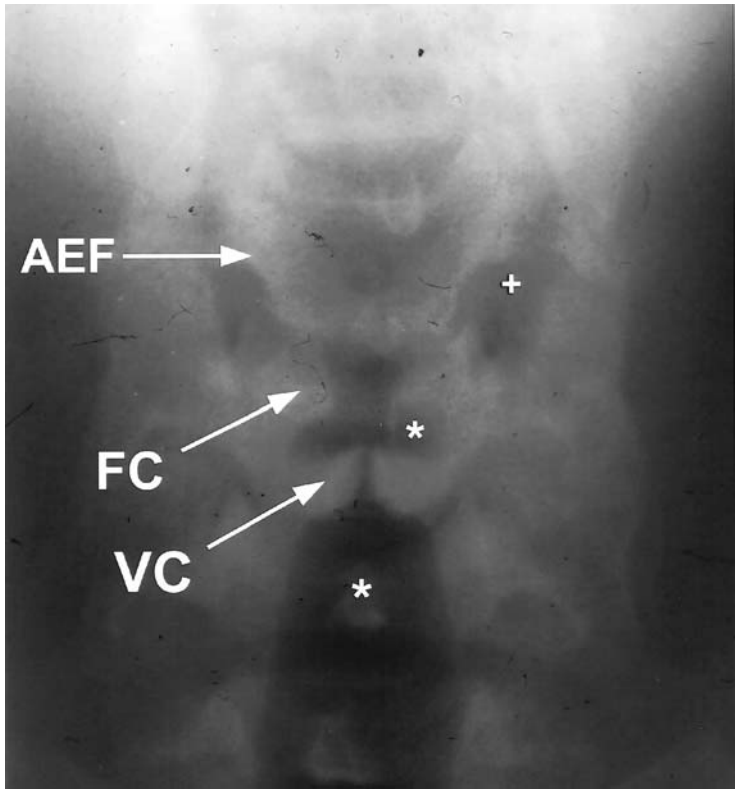
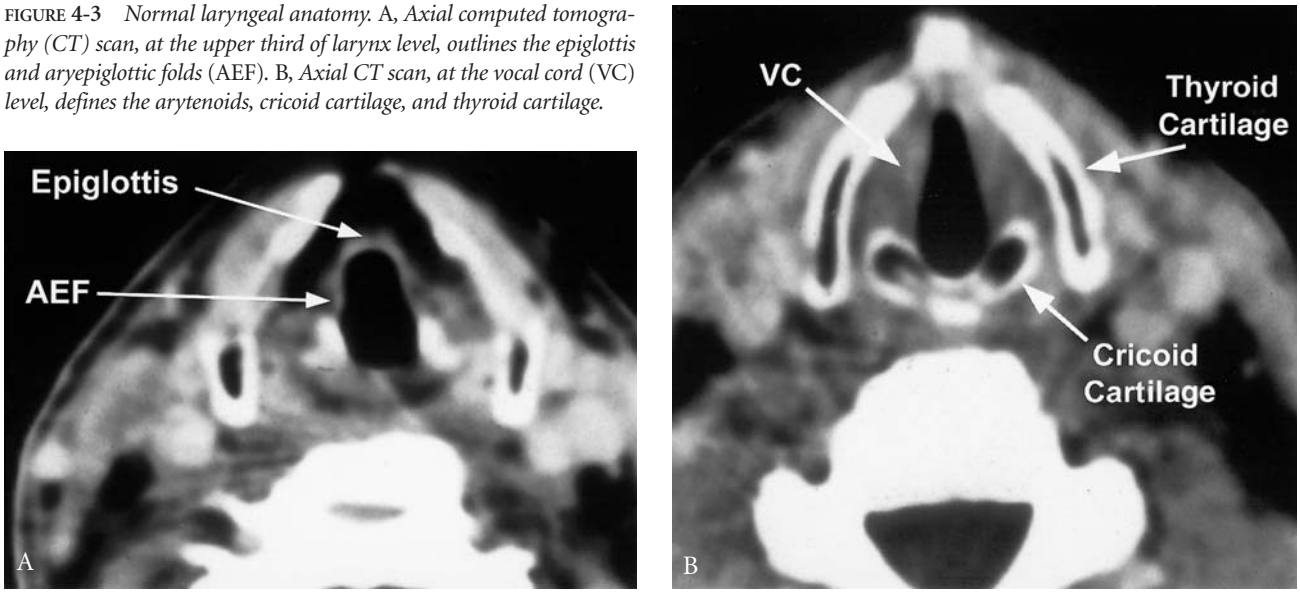


FIGURE **4-2** *Normal laryngeal anatomy. Anteroposterior high-kilovoltage view of the larynx and trachea demonstrates the aryepiglottic folds* (AEF), *false cords* (FC), *vocal cords* (VC), *and subglottic space (*lower asterisk*). Upper asterisk = laryngeal ventricle; plus sign = pyriform sinus.*

FIGURE **4-3** *Normal laryngeal anatomy.* A, *Axial computed tomography (CT) scan, at the upper third of larynx level, outlines the epiglottis and aryepiglottic folds* (AEF). B, *Axial CT scan, at the vocal cord* (VC) *level, defines the arytenoids, cricoid cartilage, and thyroid cartilage.*



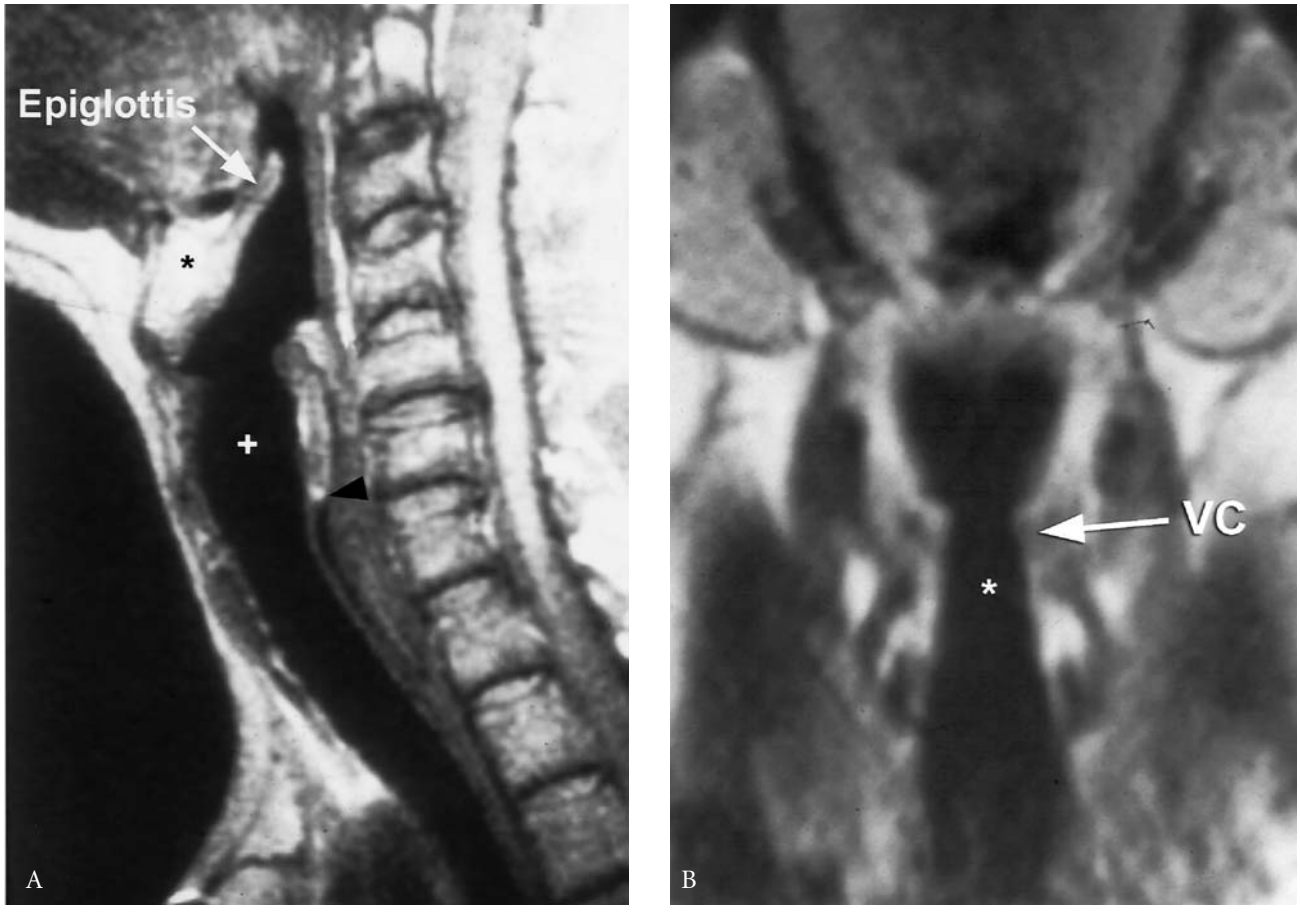


FIGURE **4-4** *Normal laryngeal anatomy.* A, *Normal lateral T1-weighted image of the larynx and cervical trachea illustrates the epiglottis, preepiglottic fat space* (asterisk), *and subglottic space* (plus sign). B, *Coronal T1-weighted image of the larynx and cervical trachea depicts the vocal cord* (VC) *and subglottic space* (asterisk).

Coronal narrowing occurs due to inward bending of the cartilaginous rings, and sagittal narrowing occurs as a result of invagination of the posterior membranous wall. Narrowing of the tracheal diameter up to 50% may be considered normal. Accentuated tracheal collapse may occur if there is abnormal flaccidity as occurs in tracheomalacia.

Normal reflections of the trachea can be identified on chest radiographs.[10,11] The right paratracheal stripe (RPS) is a thin stripe formed by the reflection of the right upper lobe with the right tracheal wall, normally 1 to 4 mm in thickness. Widening of the RPS is indicative of inherent tracheal wall disease or widening of the paratracheal soft tissues or right mediastinal pleural reflection. Normally, the left tracheal wall is not discernible because it has no contact with the left lung. The tracheoesophageal stripe (TES) is formed by the posterior tracheal wall, the anterior wall of the esophagus, and interposed fat and connective tissues, and is seen only when the esophagus contains air. The posterior tracheal band (PTB) is comprised of the posterior membranous wall of the trachea, which is formed by the interface of air in the tracheal lumen and the aerated retrotracheal recess of the right upper lobe. It is present only from the thoracic inlet to the carina, whereas the TES can be discerned from the cervical and intrathoracic regions. The PTB and TES are seen on the lateral chest radiographs, in 80 to 90% of patients. They have a uniform width of 3 to 5 mm. A left-sided aortic arch will make an impression on the left lower tracheal wall. A right-sided aortic arch will make a similar impression on the right lower tracheal wall. The tracheal bifurcation is at the level of the fifth thoracic vertebra. The intrathoracic esophagus lies slightly to the left and behind the trachea. The azygous vein may be seen as a horizontal soft tissue band, which crosses posterior to the trachea toward the right tracheobronchial angle.

Imaging Technique

Radiography

Anteroposterior and Lateral Films of the Neck Including Cervical Trachea. Routine radiologic investigation of the larynx and cervical trachea is composed of anteroposterior (AP) and lateral films of the neck (see Figures 4-1, 4-2) and oblique views of the trachea with the patient in a 45° to 60° rotation.[12,13] The lateral view of the neck is obtained with the head slightly hyperextended to bring the larynx and upper trachea up from the retrosternal position. This lateral view provides useful information about the base of tongue, vallecular area, thyroid and cricoid cartilages, intralaryngeal structures (including the epiglottis, aryepiglottic folds, arytenoids, false cords, ventricles, true cords, and subglottic space), posterior pharyngeal wall, and precervical soft tissues. Diseases that arise or spread in the sagittal plane, including the anterior and posterior tracheal wall, are readily visible. The frontal AP view of the larynx and trachea is obtained by using a high-kilovoltage technique (120 kV) and placing a 1 mm copper filter in front of the x-ray tube.[14] This view provides a survey of the entire airway from the hyoid bone to the tracheal bifurcation and main bronchi. This technique enhances the air–soft tissue interface by obscuring bone shadows. The frontal projection aids in lateralizing disease processes and supplements the lateral view. In cases of a suspected foreign body, a lateral view during swallowing is added to distinguish a foreign body from the calcified cartilaginous structures of the larynx, which move upward. This swallowing film also allows visualization of a foreign body in the upper esophagus that has been obscured by the soft tissue structures of the superimposed shoulders. One to 2 cm of the trachea and esophagus ascend out of the mediastinum during swallowing, depicting additional trachea obscured by soft tissue of the thoracic inlet. As much as 2 to 3 cm of the trachea can move above the suprasternal notch in hyperextension of the neck. This is age dependent and decreases progressively in older patients, especially if they suffer from chronic obstructive pulmonary disease.

Radiography of the Intrathoracic Trachea. The chest radiograph is the traditional screening study of the trachea. Posteroanterior (PA) and lateral views of the chest are routinely employed. The distal cervical and

intrathoracic portions of the trachea are visible on both views; however, overlying mediastinal and bony structures often obscure intrathoracic tracheal abnormalities. Bilateral oblique chest radiographs rotate the spine and mediastinal structures so that the trachea and carina are less obscured (Figure 4-5). High-kilovoltage radiographic technique can improve the visualization of intrinsic airway lesions.[6] Digital radiography can improve the visibility of tracheal walls and mediastinal reflections by virtue of edge enhancement techniques.[7] Conventional tomography of the trachea is no longer routinely employed, having been replaced by multidetector computed tomography (CT) scanning.[15]

Fluoroscopy

Fluoroscopy of the Larynx and Cervical Trachea. In order to assess the dynamics of the larynx and cervical trachea, fluoroscopy in the sitting position is indicated.[12] It supplements all other radiologic studies of the larynx and trachea, including CT scans. A thorough knowledge of the normal roentgenographic anatomy of the larynx and of functional changes encountered during different phonation maneuvers is a prerequisite. Assessment of vocal cord motion is important in the staging of malignant tumors of the larynx. Fixation of the vocal cords or paralysis of the cords from other causes (eg, thyroid carcinoma, lung cancer with mediastinal extension, aortic aneurysm, or iatrogenic trauma) can be assessed easily with phonation maneuvers such as "E" and inspiration. Opening of the ventricles can be accomplished by having the patient phonate "E" during inspiration (reversed "E"). The aryepiglottic folds, true and false cords, and ventricles are always symmetrical. The subglottic space is tubular in shape and is limited superiorly by the vocal cords, which form a right angle with the lateral wall of the subglottic space. The lumen of the subglottic

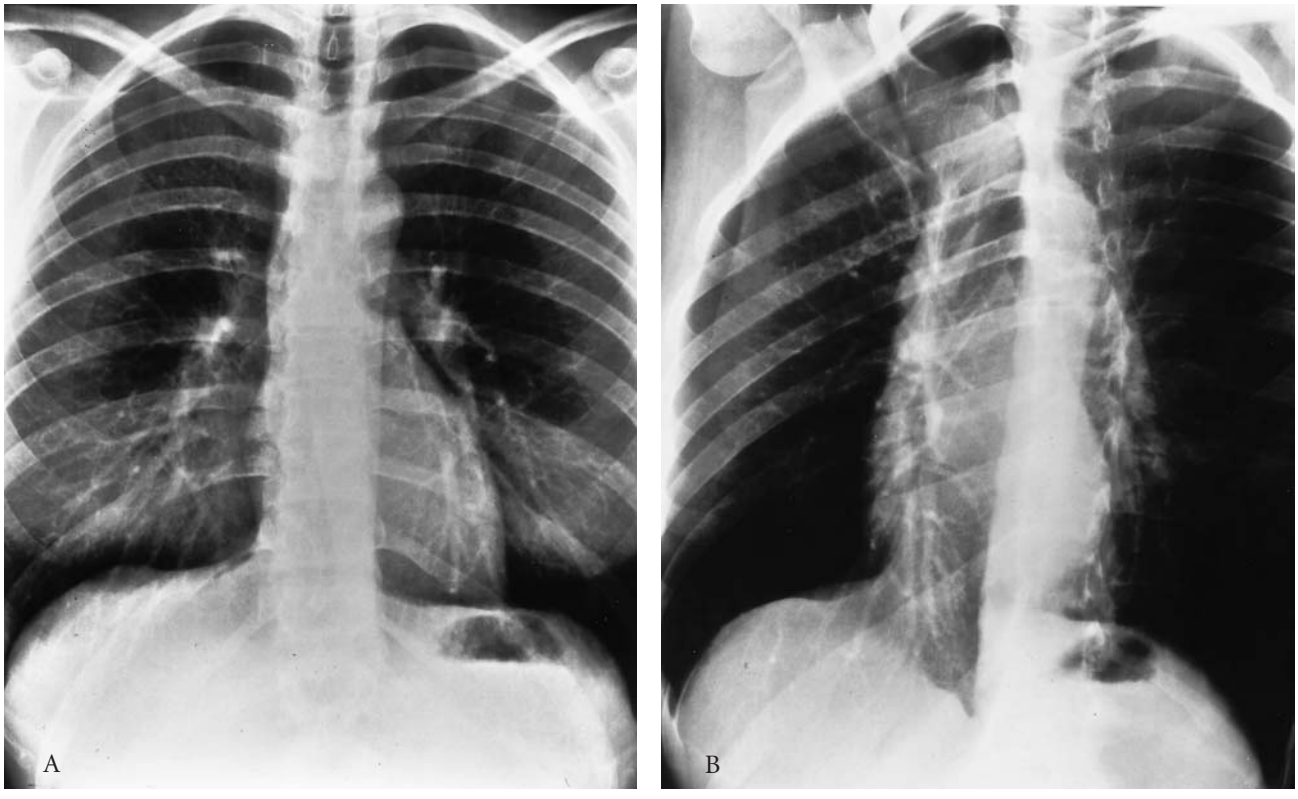


FIGURE **4-5** *Tracheal adenoid cystic carcinoma. Posteroanterior (*A*) and oblique (*B*) views of the chest. A focal soft tissue mass is evident in the midtrachea, superimposed on the thoracic spine on the posteroanterior view and better visualized on the oblique view.*

space is well marginated, and is oval to round in shape. Asymmetry of the pyriform sinuses is a common finding; however, the medial walls of the pyriform sinuses are usually symmetrical.

In the assessment of lesions of the larynx and cervical trachea, the following radiologic parameters should be determined: 1) the location, extent, size, and density of the lesion; 2) definition of the margin, presence of calcification, degree of airway compromise, cartilaginous abnormalities including destruction, and invasion of contiguous structures; 3) distensibility of the pyriform sinuses and ventricles; 4) mobility of the true and false cords; 5) displacement or tilt of the larynx; 6) extralaryngeal masses; 7) calcifications; and 8) presence of an air-filled sac.

In the fluoroscopic study of the larynx, the air–soft tissue interface can be increased easily by mounting a 1 mm copper filter on the tabletop in the field of the x-ray beam. This is especially useful in studying the infant larynx in the frontal view, for assessment of the vocal cords and subglottic space with suspected pathology in this region (eg, hemangioma, cyst, or subglottic stenosis). Simultaneously, spot films on the cervical trachea with different degrees of rotation are taken to free the trachea from superimposed normal anatomic structures at the thoracic inlet.

Fluoroscopy of the Trachea. The dynamic changes of the tracheal wall cannot be evaluated on static imaging.[16]

The nature and severity of tracheal caliber changes are best observed during real-time imaging, such as fluoroscopy. Tracheal compliance can be evaluated by the Valsalva, modified Valsalva, and Müller maneuvers and by coughing. During the Valsalva maneuver, the patient takes a deep inspiration and performs forced expiration against a closed glottis. The modified Valsalva maneuver simulates the action of blowing up a balloon. With both of these maneuvers, a weakness of the cervical trachea will manifest as a bulge (eg, laryngocele or pharyngocele). The Müller maneuver is a sniff test that consists of a forceful inspiration through the nose. With this maneuver, the pleural pressure becomes more negative than with normal inspirations, causing the intrathoracic trachea to widen more than with normal inspiration and the cervical trachea to collapse to a greater degree. When there is obstruction of the cervical trachea, inspiratory collapse may occur during forced inspiration because the pressure around the cervical trachea exceeds the intratracheal pressure. The exact opposite will occur in the intrathoracic trachea. During coughing or forced expiration, the pressure outside the airways is greater than the pressure inside, resulting in a compressive force to the intrathoracic trachea. As a result, expiratory collapse is commonly found in tracheobronchomalacia and in peripheral obstructive airway diseases such as asthma, bronchitis, or bronchiolitis (Figure 4-6).

Barium Esophagogram

The barium esophagogram is an important component of the work-up of congenital and acquired lesions of the airway, due to the close anatomic association of the esophagus with the larynx, trachea, carina, and main bronchi. The esophagogram is invaluable in providing a clue to a malignant laryngeal or tracheal tumor and in identifying primary esophageal tumors that may secondarily invade the trachea. The presence, size, and course of a tracheoesophageal fistula can be established by an esophagogram.

Computed Tomography

CT scanning is the preferred technique for evaluating the larynx, trachea, and main bronchi.[15,17,18] Helical CT scanning has dramatically improved the quality of CT imaging of the airways by acquiring a volumetric data set in a single breath-hold while using a short scanning time. In comparison, conventional CT scanning uses a long scanning time and obtains individual axial scans during separate individual breath-holds. As a result, a major advantage of helical scanning is reduced cardiac and respiratory motion. The quality of two-dimensional (2-D) and three-dimensional (3-D) reformatted images are thus markedly improved. The

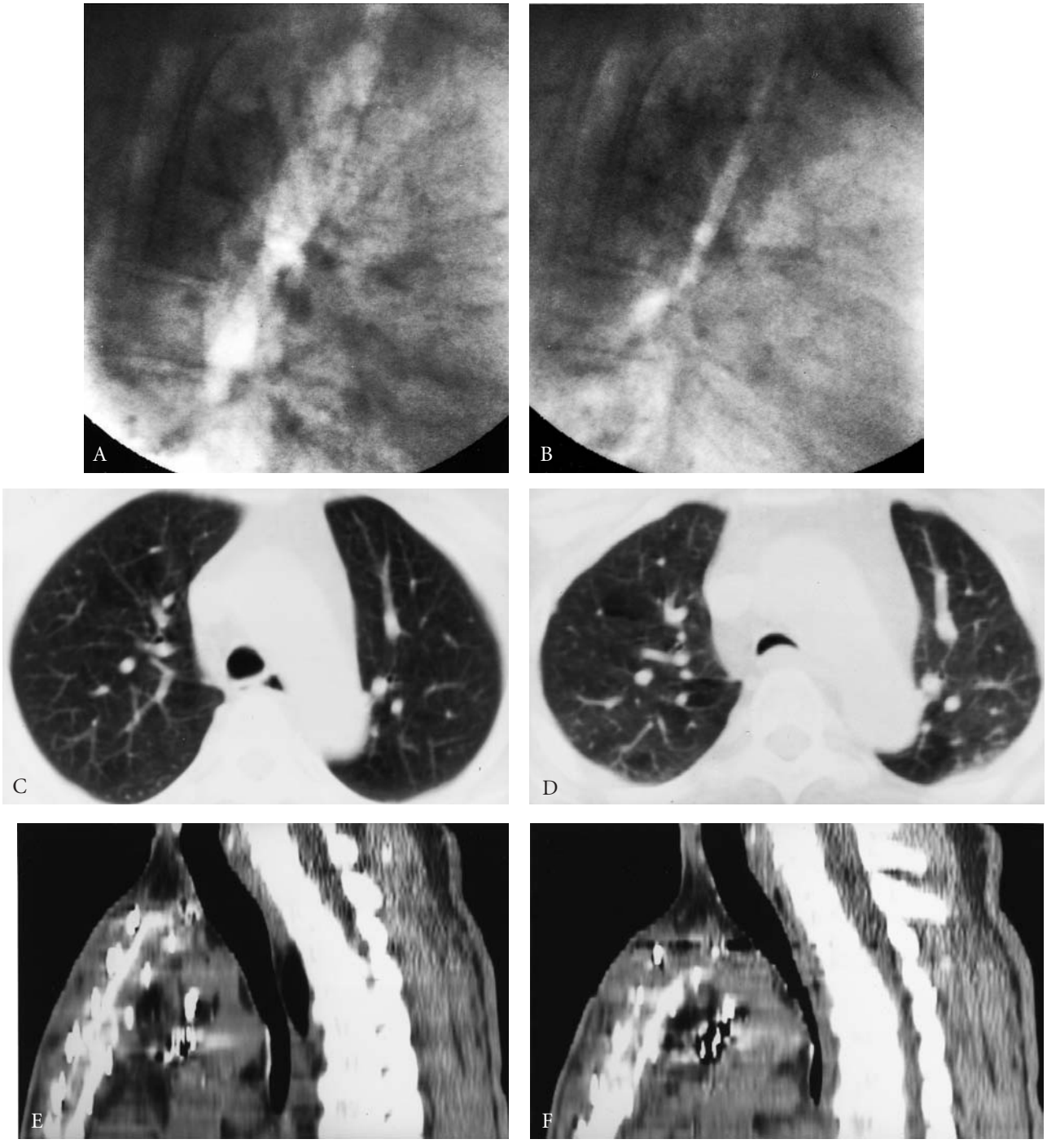


FIGURE **4-6** *Tracheomalacia. Lateral fluoroscopic images of the trachea during inspiration* (A) *and expiration* (B)*, computed tomography (CT) of the midtrachea on inspiration* (C) *and expiration* (D)*, and two-dimensional reformatted sagittal CT images of the trachea on inspiration* (E) *and expiration* (F) *demonstrate > 50% collapse in the anteroposterior diameter of the trachea, consistent with tracheomalacia.*

newest generation multidetector helical CT scanners employ multidetector arrays, which increase the speed of scanning by factors of 4, 8, or 16, thereby decreasing motion artifacts and improving the image quality of reformatted images. In addition, with multidetector helical CT imaging, high quality reconstructed

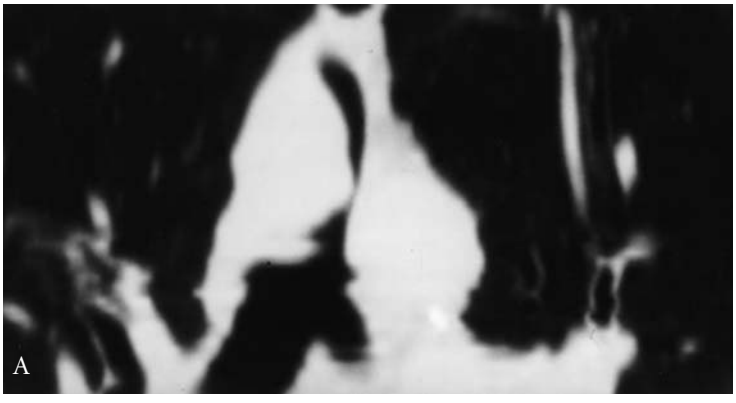
images can be obtained routinely without prospective planning or rescanning the patient. The ability to create 2-D and 3-D reformatted images of the central airways overcomes the inherent limitations of axial images, including the limited ability of detecting subtle stenoses, evaluating the accurate craniocaudad extent of disease, visualizing obliquely oriented airways, and displaying the complex relationship of adjacent mediastinal structures (Figure 4-7*A*). Reformatted 2-D and 3-D images do not offer any new information; rather, they provide a complementary way to view the same data sets.

Although the use of intravenous contrast is not necessary to assess the central airways, it is recommended to evaluate adjacent mediastinal extent of tumor and adenopathy or to assess adjacent mediastinal vascular structures or masses that may compress the airway. CT scanning of the airway is routinely obtained at end-inspiration during a breath-hold. When assessing tracheomalacia, it is helpful to obtain additional scan sequences at the same table levels during end-expiration.

Reconstruction methods that are used for airway imaging include 2-D multiplanar and 3-D internal and external rendering techniques. 2-D images are the easiest to obtain because they can be generated at the CT console, and 2-D reformations can be displayed in the coronal or sagittal planes, orthogonal to a reference point or curved along the axis of the airway. 3-D reformations require the transfer of data to a separate workstation, but 3-D images of the airway can be visible on external 3-D rendered images or on internal renderings that create virtual bronchoscopic images of the central airways.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is another modality being used for the radiologic evaluation of the larynx and trachea.[19] A major advantage of MRI over CT is the acquisition of coronal, oblique, and sagittal sections that demonstrate long segments or the entire length of the trachea (Figure 4-7*B*). With MRI, it is possible to visualize the laryngeal and tracheal structures with great detail in transverse, sagittal, and coronal planes.[3,20] Normal anatomic structures can be differentiated on the basis of different signal intensities on T1-weighted sequences. A bright signal is elicited by fat, hyaline cartilage, submucosal fascial planes



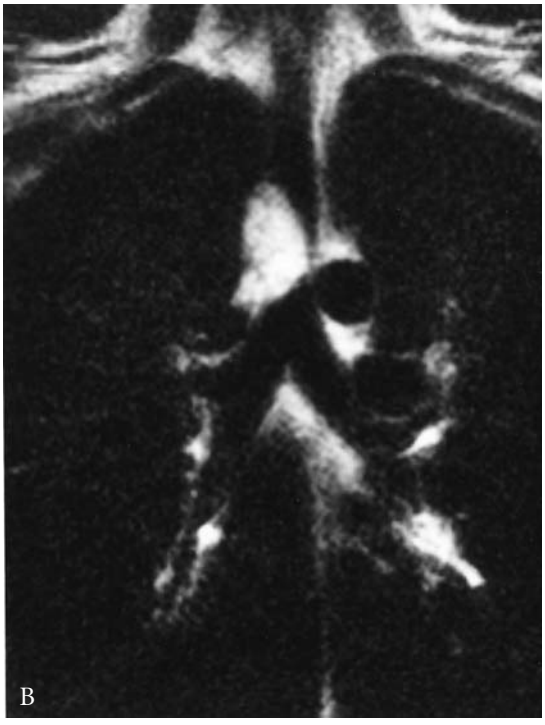


FIGURE **4-7** *Adenoid cystic carcinoma.* A, *Two-dimensional reformatted computed tomography image of the distal trachea and carina demonstrates a smooth intraluminal mass arising from the right lateral wall of the trachea.* B, *T1-weighted coronal magnetic resonance imaging through the trachea and carina in the same patient demonstrates a soft tissue mass in the distal trachea, consistent with adenoid cystic carcinoma.*

(paralaryngeal space), and false cords. Intermediate signal intensities are given off from the true cords, aryepiglottic folds, and the intrinsic laryngeal muscles. Calcified cartilage, the airway, and blood vessels produce low signals. MRI can be used in the assessment of tracheal stenosis and tumors and other paratracheal masses that compress the trachea (Figure 4-8). The examination is performed with a T1 sequence in the axial, coronal, and sagittal planes, supplemented by an axial T2 sequence. The T1-weighted sequence demonstrates the anatomy of the trachea and surrounding structures in great detail. The T2-weighted sequence adds to the characterization of the lesion, for example, differentiation of a cyst from a tumor. Carcinomas are characterized by intermediate signal intensities on the T1 sequences and high signal intensities on heavily T2-weighted images. On T1 sequences, the tumor may obliterate the high signal intensity areas (paralaryngeal and preepiglottic spaces), encroach on the signal void air spaces, and/or may cause erosion of the laryngeal or tracheal cartilaginous structures.

MRI is the preferred method for evaluating paratracheal abnormalities in children because it does not involve ionizing radiation, and is particularly useful in studying vascular rings, slings, and dilated vessels that compress the trachea (Figure 4-9). In addition, MRI is the preferred modality in evaluating patients with paratracheal masses, who have contraindications to iodinated contrast material used for CT scans. Gadolinium, a MRI contrast agent, can be given safely to such patients. Dynamic MRI may also prove to be useful in evaluating tracheomalacia, although at the present time, inspiratory/expiratory CT scanning is still the examination of choice for studying this condition.

Congenital Abnormalities

Tracheal Bronchus

A tracheal bronchus is an anomalous bronchus that arises directly from the right side of the trachea.[21,22] It is present in 0.25 to 1% of the human population. Most tracheal bronchi arise within 2 cm of the carina

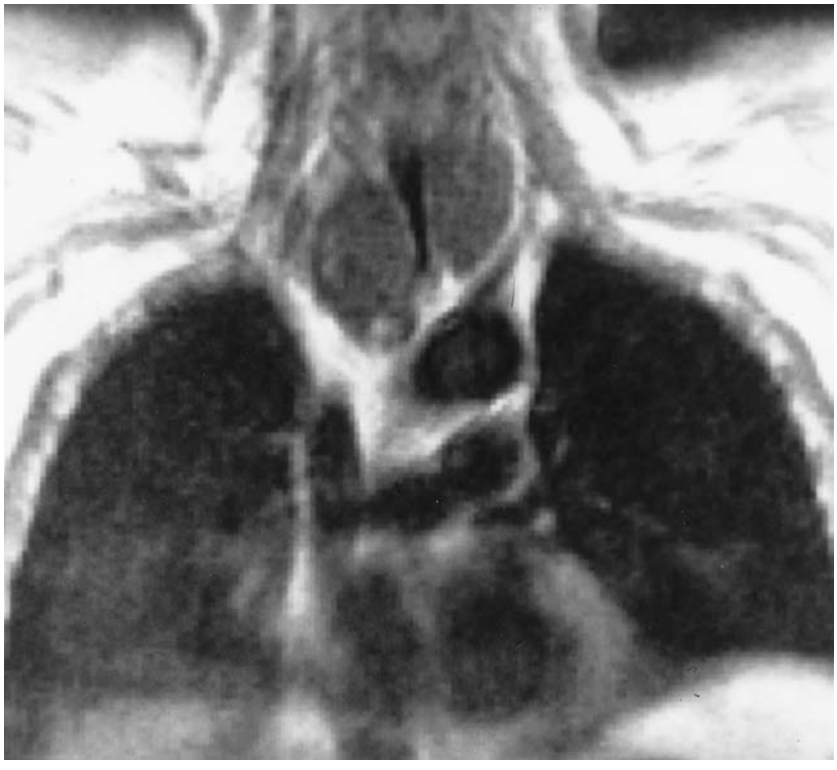
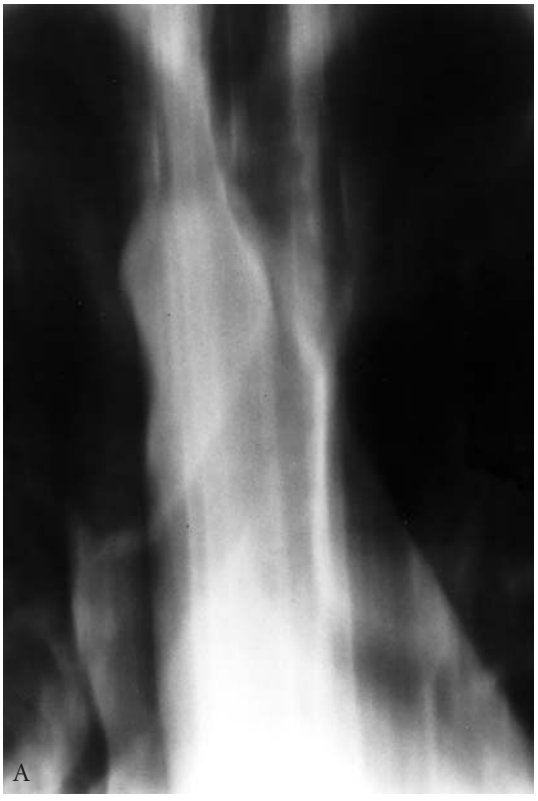


FIGURE **4-8** *Goiter. Coronal T1-weighted magnetic resonance image of the neck and chest reveals an enlarged thyroid gland causing marked tracheal narrowing at the thoracic inlet.*



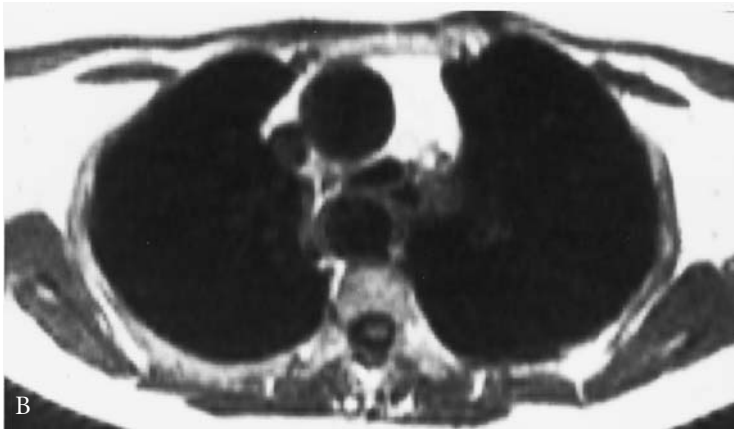


FIGURE **4-9** *Right-sided aortic arch.* A, *Anteroposterior view of the trachea demonstrates marked tracheal narrowing adjacent to the right aortic arch.* B, *Axial T1-weighted magnetic resonance imaging reveals compression of the mid-trachea by a slightly dilated right-sided aortic arch.*

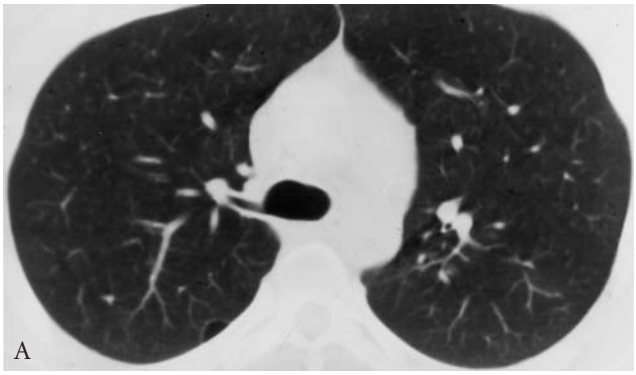
and are usually asymptomatic. Tracheal bronchi are classified into four different types: 1) rudimentary tracheal bronchus, typically arising as a blind outpouching of the right lower side of the trachea; 2) displaced bronchus, the most common anomaly in which one or two of the upper lobe segments are aerated by the tracheal bronchus; 3) supernumerary accessory bronchus, which arises from the trachea in addition to the normal right upper lobe (Figure 4-10); and 4) right upper lobe bronchus, with three normal segments arising above the tracheal bifurcation, sometimes a duplicate of a normal right upper lobe bronchus.

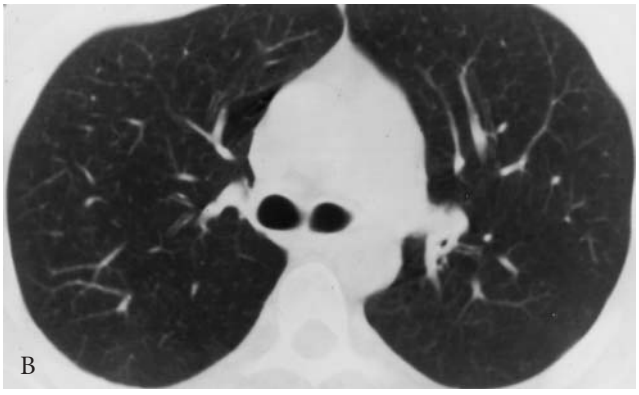
Congenital Tracheal Stenosis

Congenital tracheal stenosis is a rare developmental abnormality of the trachea that may affect any or all parts of the trachea, in which the tracheal cartilages are hypoplastic, forming complete rings without a membranous posterior wall.[23] As a result, the trachea is rigid and nondistensible. On CT scans, the trachea is narrowed, with identifiable calcified complete cartilaginous rings (Figure 4-11). Associated congenital anomalies are present in 80% of cases, including H-type tracheoesophageal fistula, laryngomalacia, subglottic stenosis, bronchial stenosis, hypoplasia or agenesis of the lungs, and other skeletal, cardiovascular, and intestinal anomalies.

Congenital Vascular Anomalies and Rings and Vascular Compression

Vascular rings, slings, or mediastinal great vessels may compress the trachea and/or esophagus.[24–27] In adults, most vascular compressions are related to acquired aneurysmal dilation of the great vessels. Contrast CT, magnetic resonance angiography, and/or angiography is indicated to establish a diagnosis. A barium swallow is necessary in the work-up of a child with respiratory symptoms, because the esophagus is frequently involved in compression syndromes.





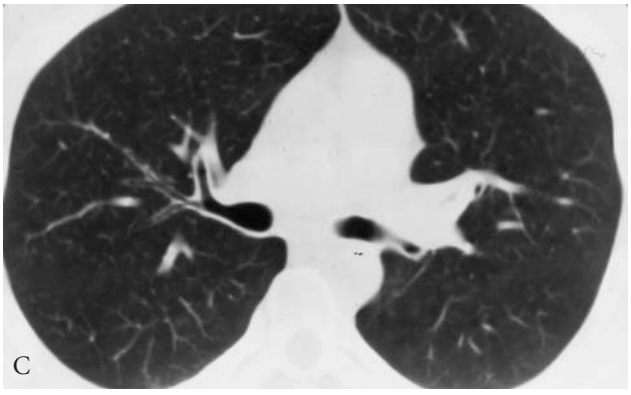


FIGURE **4-10** *Tracheal bronchus. Computed tomography examination demonstrates a tracheal bronchus* (A) *arising from the distal trachea just above the carina* (B). *The normal right upper lobe bronchus is noted* (C).

Vascular rings are the most common symptomatic anomalies causing tracheal and esophageal compression and are classified as 1) double aortic arch or 2) right aortic arch with left ligamentum arteriosum, patent ductus, or aberrant left subclavian artery. The absence of an aortic arch on the left and the presence of a tracheal indentation by the right-sided aorta are critical findings. The distal trachea may be deviated to the left on the frontal view, and anteriorly on the lateral view. A double aortic arch causes both anterior and posterior compression of the trachea.

A *pulmonary artery sling* occurs when an anomalous left pulmonary artery courses over the right main stem bronchus, near its origin from the trachea, and crosses posteriorly and to the left between the esophagus and the trachea, reaching the left hilum above the bronchus. As a result, it may compress the trachea and right main stem bronchus. A strong association exists with both tracheobronchial defects and cardiovascular anomalies. Diagnostic findings on barium swallow, CT, and MRI include indentation of the anterior aspect of the esophagus as well as tracheal deviation to the left just above the carina, by the aberrant pulmonary artery. The left hilum is situated more caudal than normal.[27]

Anterior compression of the trachea may occur by the innominate artery. The degree of compression is usually most severe in expiration. In children, the innominate artery often originates partially or totally to the left of the trachea and crosses in front of it, and clinical symptoms result because of crowding in the mediastinum. The lateral chest radiograph usually demonstrates a typical anterior compression of the trachea. The esophagus is usually not affected on an esophagogram.[26]

Postpneumonectomy Syndrome

Postpneumonectomy syndrome is a rare condition that occurs in children or adults following a pneumonectomy.[28,29] For reasons that are not well understood, in certain patients, the heart and mediastinum shift excessively toward the side of the pneumonectomy and the great vessels rotate significantly. In the case

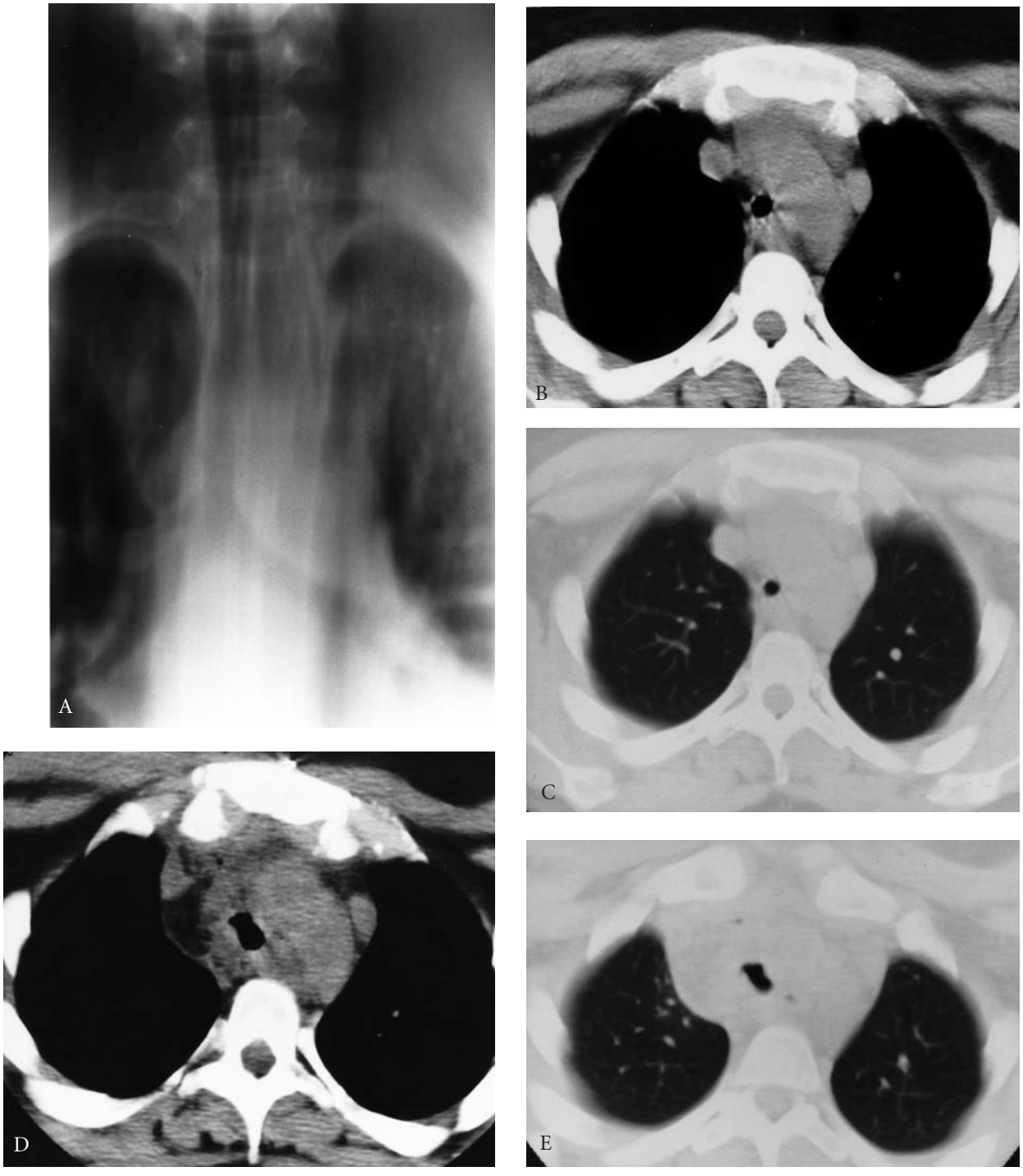


FIGURE **4-11** *Congenital tracheal stenosis.* A, *Coronal tomogram demonstrates diffuse stenosis of the intrathoracic trachea.* B, *Computed tomography image through the midtrachea reveals a complete cartilaginous tracheal ring and significant stenosis of the trachea* (C). D,E, *Following a slide tracheoplasty procedure, the tracheal lumen is significantly greater.*

of a right pneumonectomy with a left-sided aortic arch, the remaining distal trachea and/or left main bronchus becomes interposed between the pulmonary artery anteriorly and the aorta and thoracic spine posteriorly, resulting in a stenosis of the distal trachea or main bronchus (Figure 4-12). A similar compli-

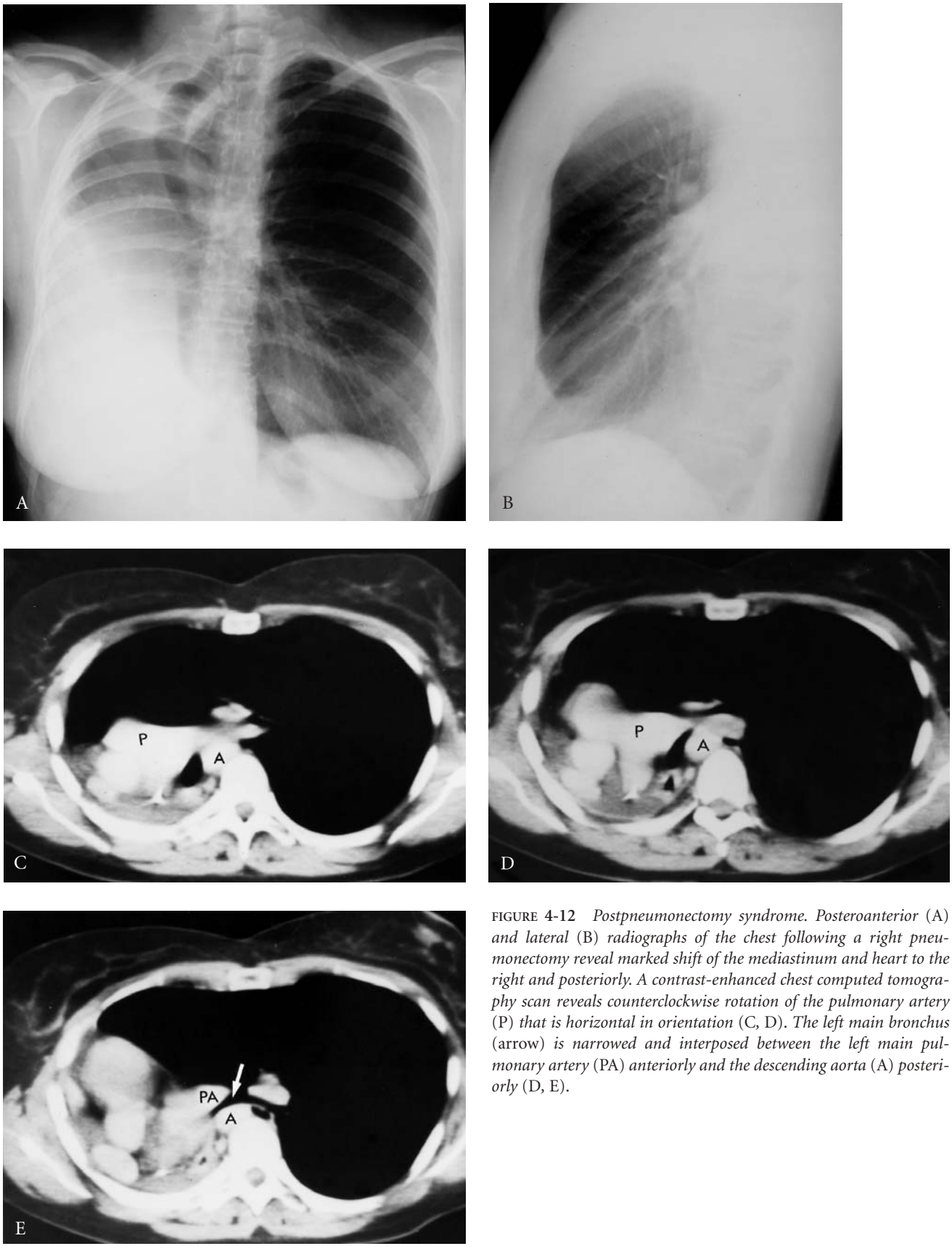


FIGURE **4-12** *Postpneumonectomy syndrome. Posteroanterior* (A) *and lateral* (B) *radiographs of the chest following a right pneumonectomy reveal marked shift of the mediastinum and heart to the right and posteriorly. A contrast-enhanced chest computed tomography scan reveals counterclockwise rotation of the pulmonary artery* (P) *that is horizontal in orientation* (C, D). *The left main bronchus* (arrow) *is narrowed and interposed between the left main pulmonary artery* (PA) *anteriorly and the descending aorta* (A) *posteriorly* (D, E).

cation may be seen following a left pneumonectomy with a right-sided aortic arch. Cross-sectional imaging with CT and/or MRI is essential to establish the vascular and bronchial relationships and to define the position and extent of the tracheobronchial obstruction. Inspiratory/expiratory CT imaging is useful to identify a malacic segment that may rarely occur at the level of the stenosis. Postoperative imaging following repositioning of the mediastinum helps to redefine the vascular and tracheobronchial positions and confirm establishment of a patent airway.

Trauma

Larynx

Laryngeal trauma can be caused by external or internal injuries. External injuries are the result of blunt or penetrating trauma. Internal injuries are usually caused by prolonged intubation or chemical or thermal burns.[30] Trauma is characterized by mucosal disruption of soft tissues, swelling, and collection of air in the soft tissue structures of the larynx including neck and mediastinum. These soft tissue injuries may be associated with cartilaginous fractures, and dislocations of the arytenoids and epiglottis.[31] The blood and edema fluid insinuate along the deep spaces of the larynx, predominantly in the paralaryngeal and epiglottic spaces, followed by a variable degree of airway narrowing. Fractures of the thyroid cartilage may be transverse or vertical, resulting in hemorrhage into the preepiglottic space, with consequent posterior displacement of the epiglottis. Fractures in the cricoid cartilage are often vertical and lead to a variable degree of disruption of the signet ring. Fractures of the cartilaginous structures are associated with a variable degree of displacement and hematoma formation. The arytenoids may be displaced as an isolated incidence or in conjunction with fractures of the cartilaginous structures. They are most often displaced anteriorly and superiorly. Vocal cord motion is usually impaired as a result of hematoma formation, fractures, arytenoid dislocation, fibrosis, or recurrent laryngeal nerve paresis. Disruption of the cricothyroid joint may occur and also lead to dysfunction of the vocal cords. The sequelae of severe laryngeal injuries are variable degrees of stenosis, which may involve the entire larynx or may be localized to the supraglottic, glottic, or subglottic larynx.

CT is the modality of choice for demonstrating the various described findings in laryngeal trauma.[32] CT will also demonstrate the extent of soft tissue edema, hematoma formation, the location and extension of fractures, and the deformity of cartilaginous structures after healing (Figure 4-13).

Trachea and Main Bronchi

Tracheobronchial rupture is a rare injury that results from a decelerating injury. The possible mechanisms of injury include compression of the airway between the sternum and the vertebral column, a sudden deceleration of pendulous lung with a fixed trachea creating shearing forces, and forced expiration against a closed glottis raising intrabronchial pressure.

The site of airway rupture and its extent determine the radiographic findings. The tracheal or bronchial tear is usually complete and will lead to subcutaneous emphysema, pneumomediastinum, or pneumothorax.[33] Tears of the trachea and proximal left main bronchus generally leave the parietal pleura intact and will result in pneumomediastinum (Figure 4-14). Distal left main bronchial tears and right main bronchial tears will generally communicate with the pleural cavity and result in pneumothorax (Figure 4-15). Tears generally occur within 2.5 cm of the carina, and are more commonly seen on the right side. If the central anchoring components of the lung are completely ruptured and the main bronchus disrupted, the lung may collapse peripherally from the hilum, in what has been described as the "fallen lung" sign. In some cases, there may be an incomplete tear and an absence of an air leak when the integrity of the peribronchial or peritracheal connective tissue is maintained, when a cuff occludes the tear, or when fibrin seals the tear. Tears that present in this fashion are often missed initially and have a delayed presentation (Figure 4-16). If healing develops, an

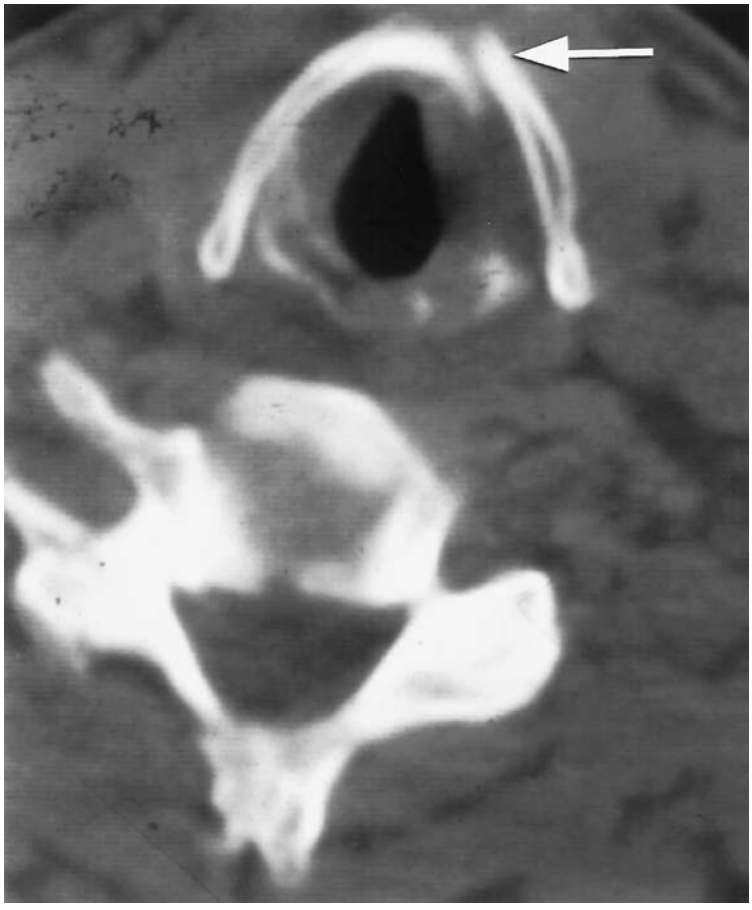
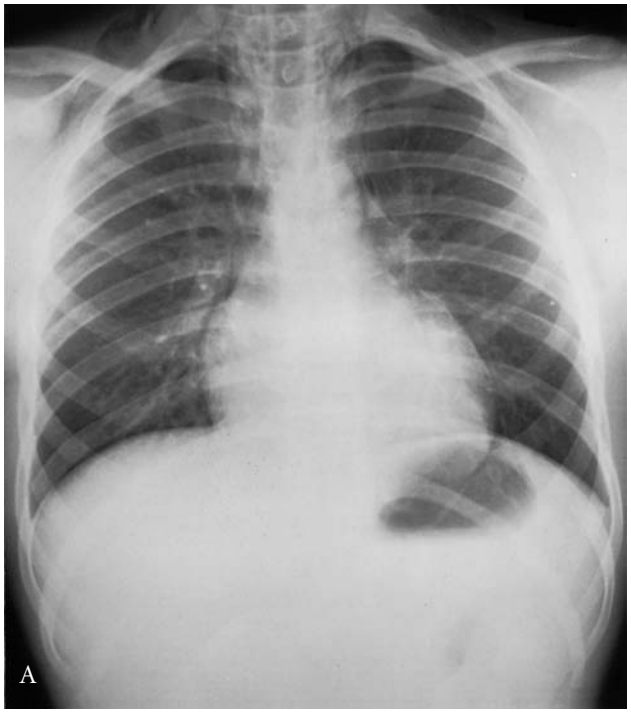


FIGURE **4-13** *Laryngeal trauma. Axial computed tomography section through the subglottic space of the larynx defines a fracture in the anterior thyroid cartilage* (arrow).



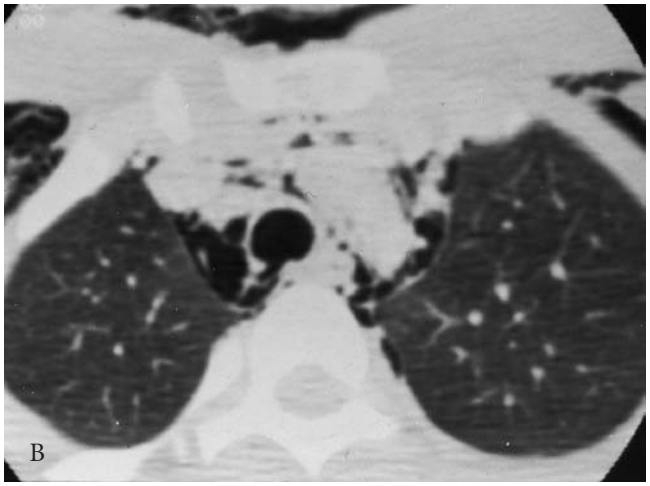


FIGURE **4-14** *Acute tracheal tear.* A, *Posteroanterior chest radiograph demonstrates marked diffuse pneumomediastinum extending into the neck.* B, *Computed tomography scan through the upper chest reveals extensive pneumomediastinum and subcutaneous emphysema. There is a tear of the membranous wall of the trachea.*

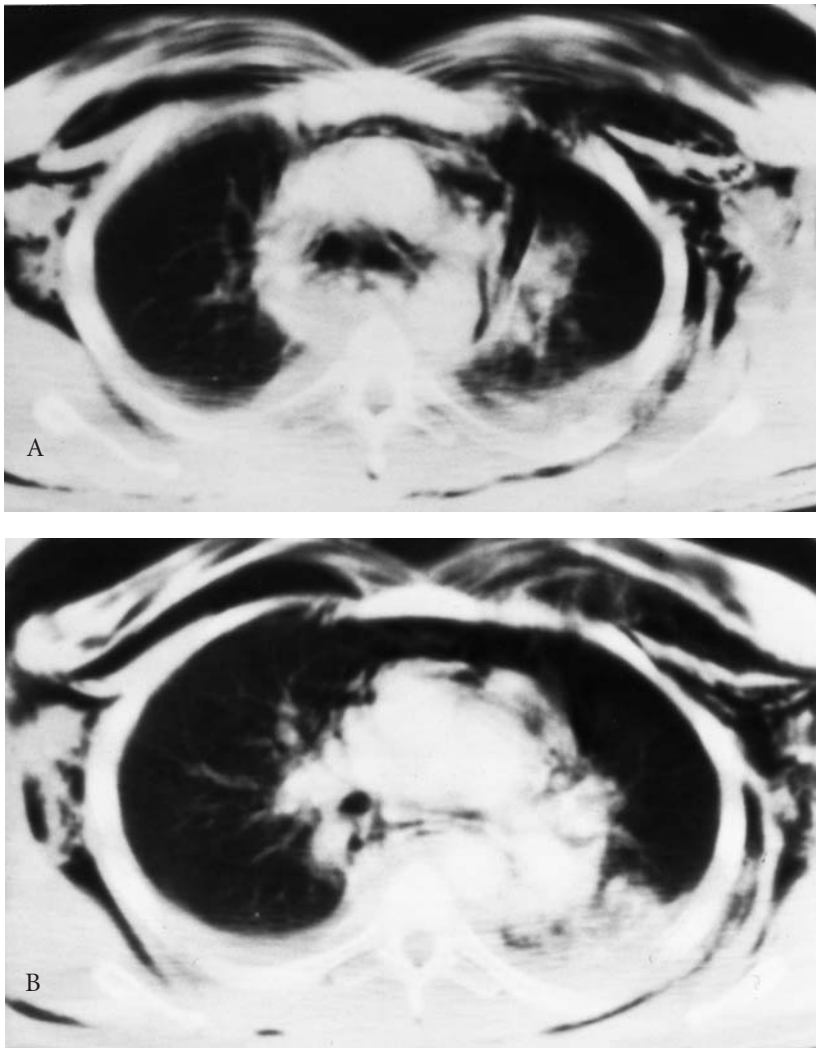


FIGURE **4-15** *Acute left main bronchial rupture. Computed tomography scans through the carina* (A) *and left main bronchus* (B) *reveal extensive pneumomediastinum, subcutaneous emphysema, and a left-sided pneumothorax. There is marked narrowing of the left main bronchus at the site of laceration* (B).

untreated partial laceration will develop a stenosis with a typical hourglass configuration and potentially cause distal collapse of a lung or lobe (Figures 4-17, 4-18). Tracheobronchial trauma is often associated with aortic injury and fractures of the first three ribs or sternum.[34–36]

Traumatic and Postintubation Stenosis

One of the complications to the larynx and trachea, most commonly seen following intubation, is granuloma formation. Granulomas occur primarily in the posterior supraglottic, glottic, and subglottic larynx, and cervical trachea (Figures 4-19, 4-20). They can attain a large size with subsequent airway obstruction. Late changes secondary to trauma represent laryngeal stenoses causing airway obstruction. These stenoses may occur in the supraglottic, glottic, and subglottic larynx,[37] or as a combination of all the sites with extension into the cervical trachea (Figures 4-21, 4-22, 4-23).[38] In severe cases, the entire larynx and/or trachea may become obliterated. Tracheal stenosis is also encountered post-tracheostomy and postintubation.[39] They occur primarily at two sites: 1) the tracheostomy opening or stoma, and 2) in the area of the balloon cuff. In a small percentage of cases, both lesions will occur simultaneously.[40] With the introduction of compliant, extensible, large-volume latex cuffs, this complication has been prevented to a great degree, and, consequently, the incidence of postcuff stenosis has markedly decreased.

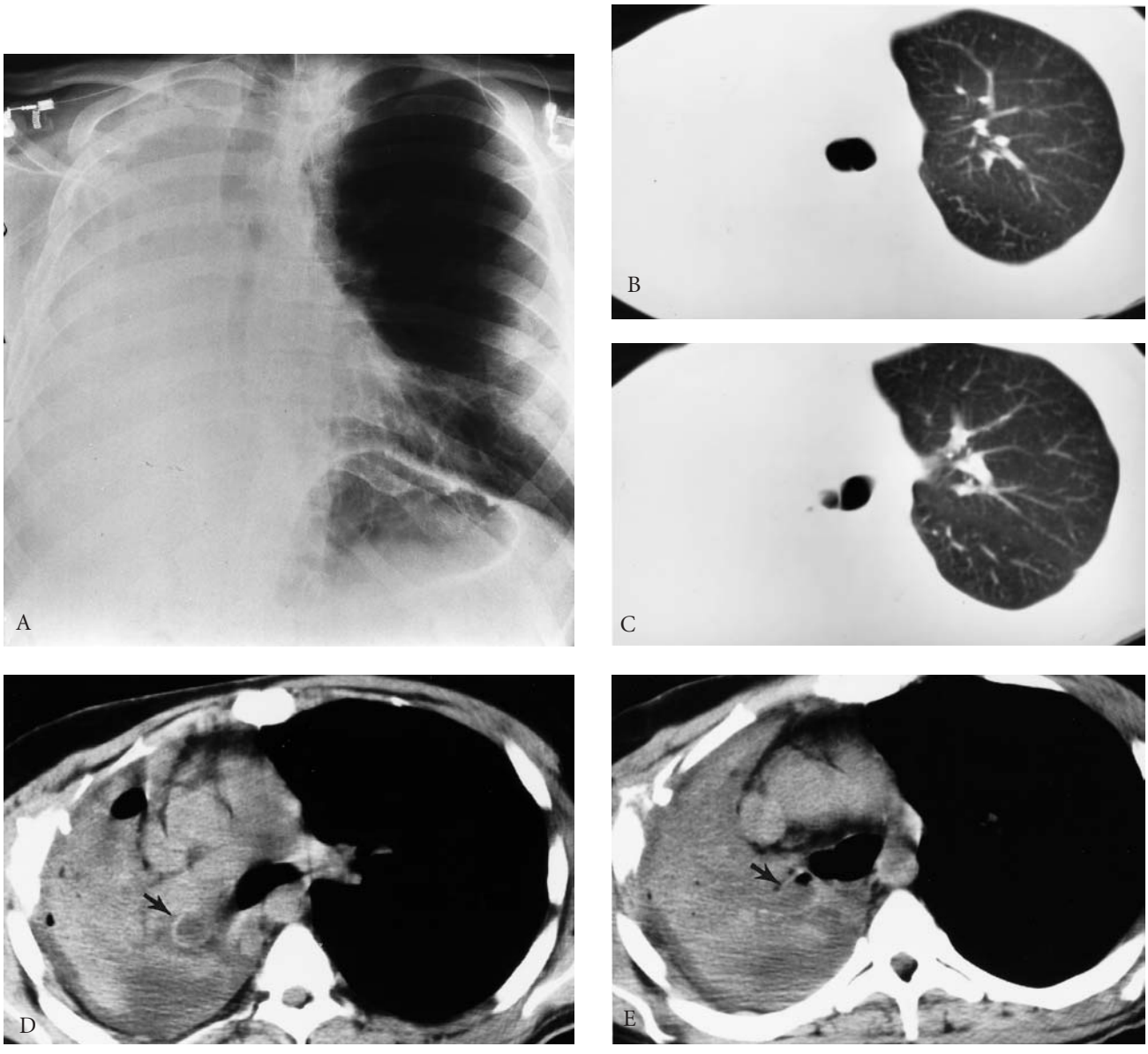
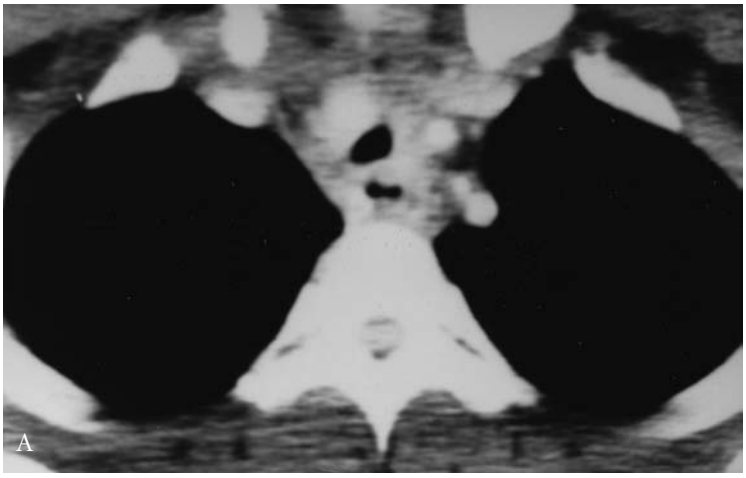


FIGURE **4-16** *Subacute right main bronchial tear.* A, *Anteroposterior chest radiograph reveals complete opacification of the right hemithorax and shift of the trachea and mediastinum to the right, consistent with right lung collapse following contained laceration of the right main bronchus. Computed tomography scans with lung windows at the carina* (B) *and slightly below the carina* (C) *demonstrate narrowing of the lacerated right main bronchus.* D,E, *Soft tissue windows at the same levels reveal a fluid-filled, displaced distal right main bronchus* (arrow).

At the stomal site, a large stoma, superimposed infection, or the use of rigid connecting systems increases the incidence of stomal strictures due to pressure erosion.[41] The stenosis at the tracheostomy stoma frequently involves the anterior and lateral tracheal wall, and forms a triangularly-shaped area of narrowing (Figure 4-24). Changes in the tracheal wall consist of fibrosis, often associated with granulation tissue. In many instances, a variable amount of calcium and bone that are deposited in the tracheal wall are readily demonstrated on the CT scan. Another complication seen at the tracheostomy site is a formation of an anterior tracheal wall flap from above the stoma, caused by inversion of the anterior tracheal wall into the adjacent lumen. Granulation tissue may form on the flap and increase the severity of the obstruction.



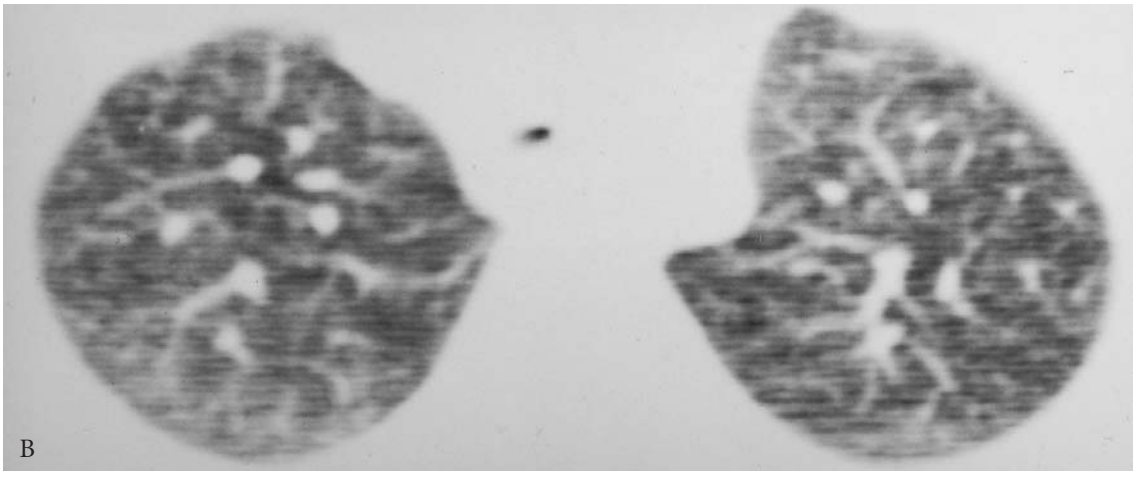


FIGURE **4-17** *Post-traumatic tracheal stenosis.* A, *Computed tomography scan with intravenous contrast material demonstrates a narrowed, misshapen trachea surrounded by soft tissue density, consistent with fibrosis at the site of healed tracheal rupture.* B, *A lung window through the superior mediastinum reveals marked tracheal stenosis.*

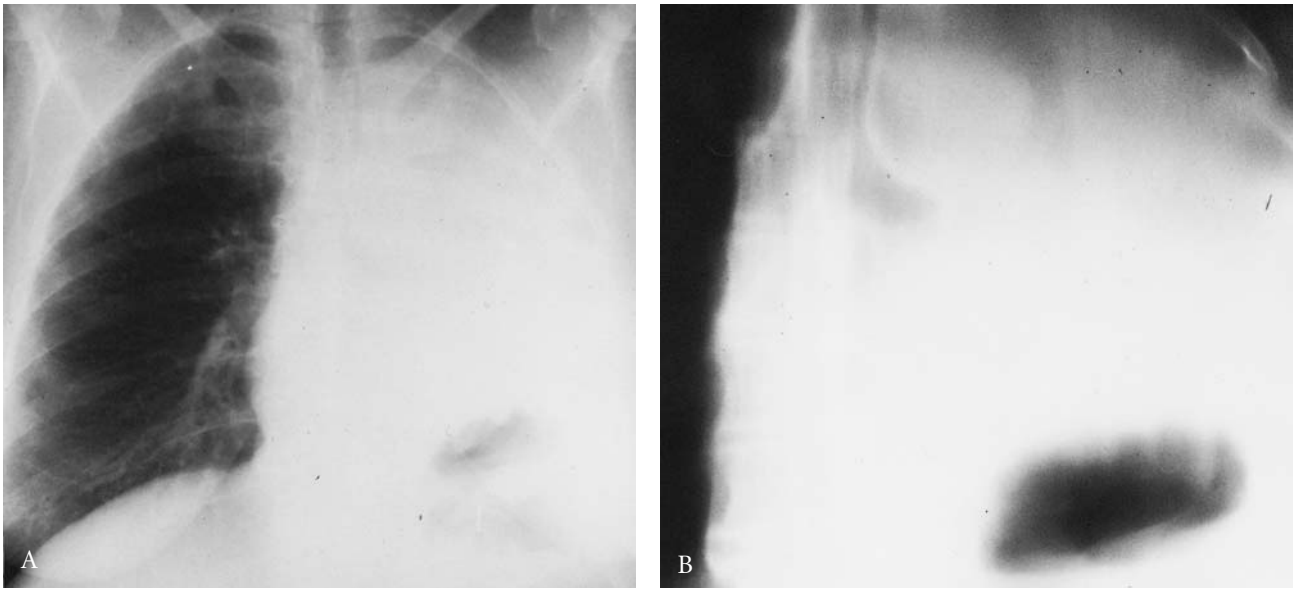


FIGURE **4-18** *Missed left main bronchial stenosis due to missed bronchial rupture. Posteroanterior chest radiograph* (A) *and anteroposterior tomogram* (B) *reveal collapse of the left lung distal to a left main bronchial stenosis.*

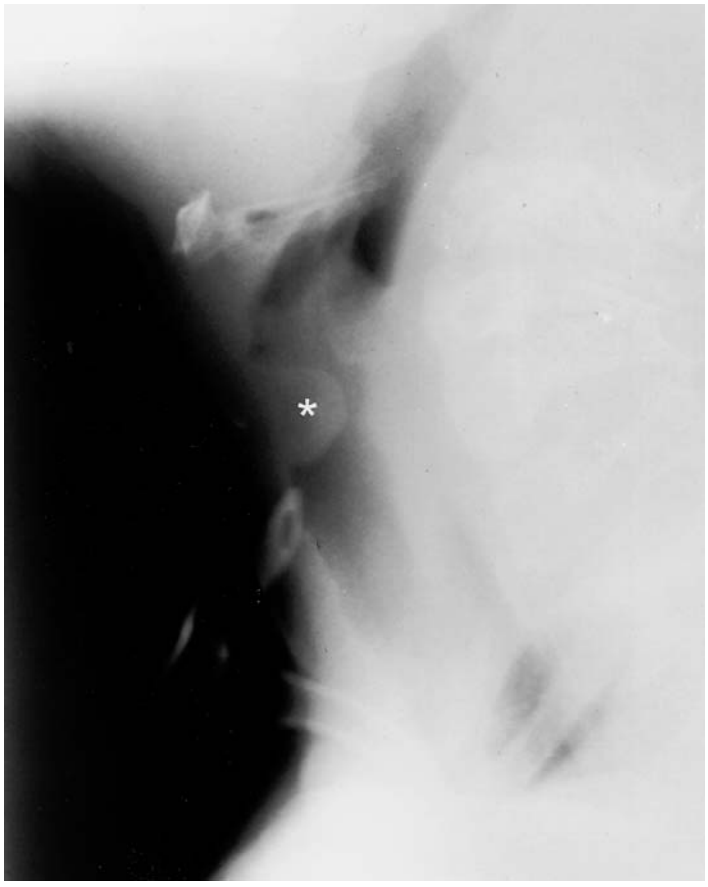


FIGURE **4-19** *Subglottic granuloma. Lateral neck view illustrates a sharply delimited polypoid mass in the anterior subglottic space, consistent with a granuloma* (asterisk).

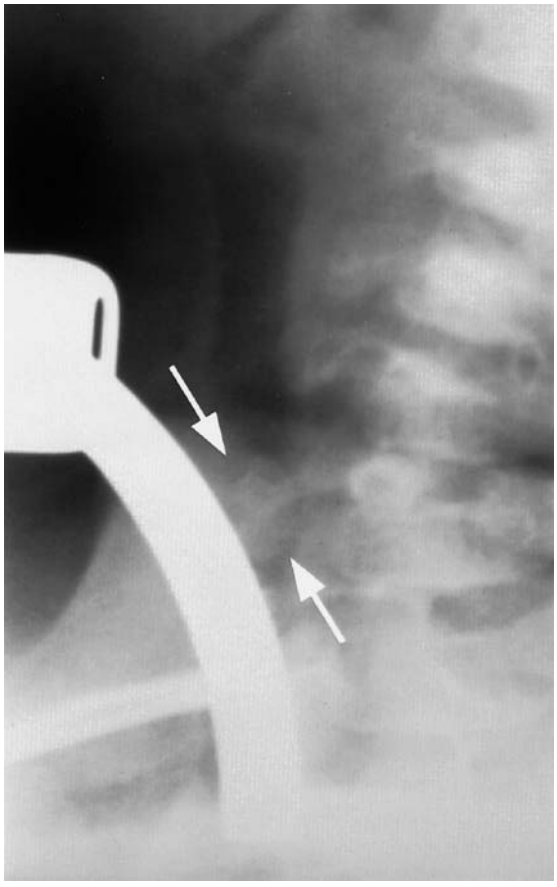


FIGURE **4-20** *Tracheal granuloma adjacent to a tracheostomy canula. Oblique spot film demonstrates a granuloma at the tracheostomy tube* (arrows).

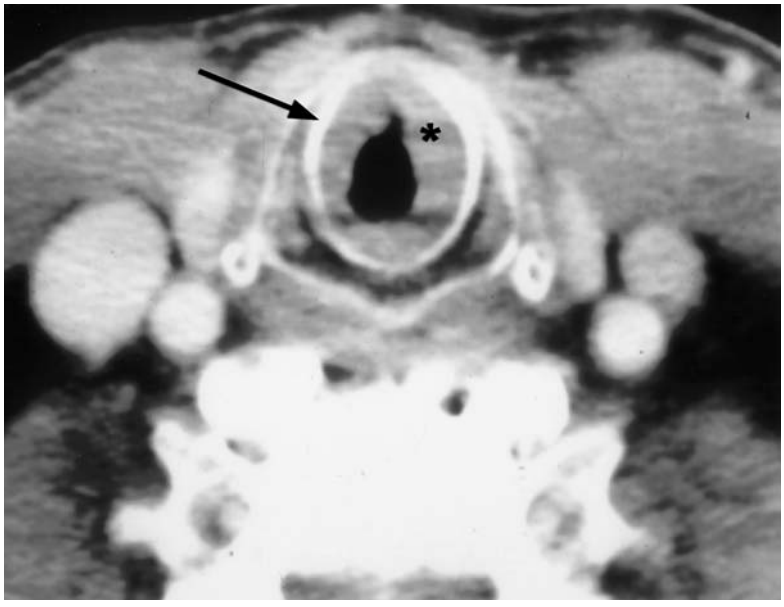


FIGURE **4-21** *Subglottic stenosis. Axial computed tomography scan demonstrates diffuse circumferential thickening of the submucosa in the subglottic space* (asterisk). *Note the normal cricoid cartilage* (arrow).

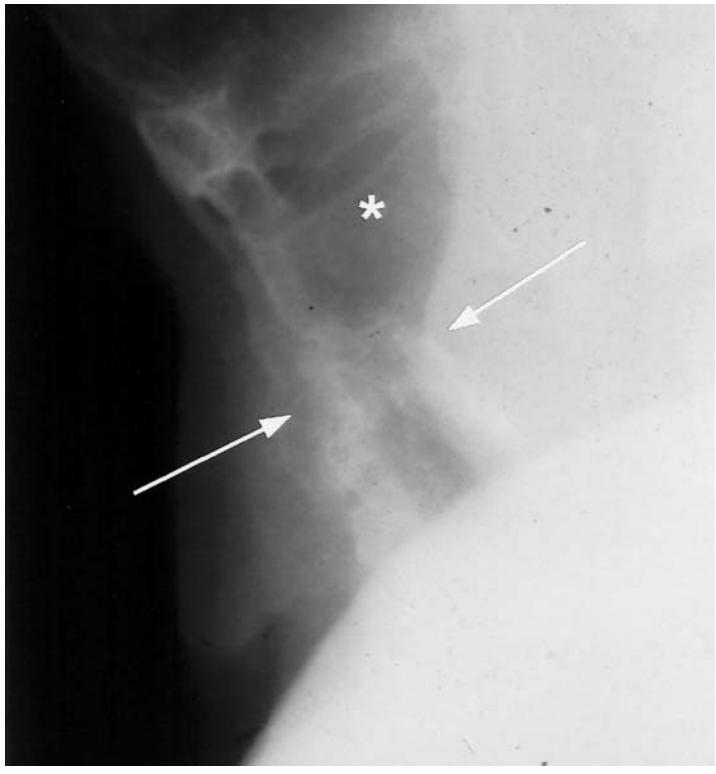


FIGURE **4-22** *Infraglottic and adjacent cervical tracheal stenosis. Lateral view of the neck illustrates slight narrowing of the inferior portion of the subglottic space* (asterisk) *and adjacent trachea posteriorly. There is thickening of the tracheal wall with some calcification* (arrows).

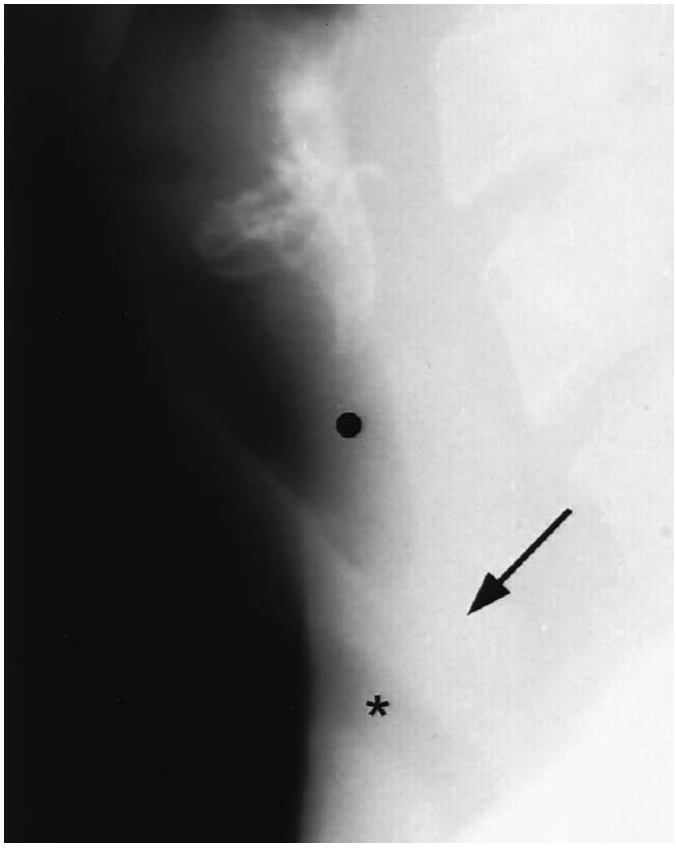


FIGURE **4-23** *Subglottic and cervical tracheal stenosis. Lateral neck view shows a long severe subglottic and cervical tracheal stenosis* (arrow). *Note the tracheostomy tract* (asterisk). *The inferior margin of the larynx is indicated by a* dot.

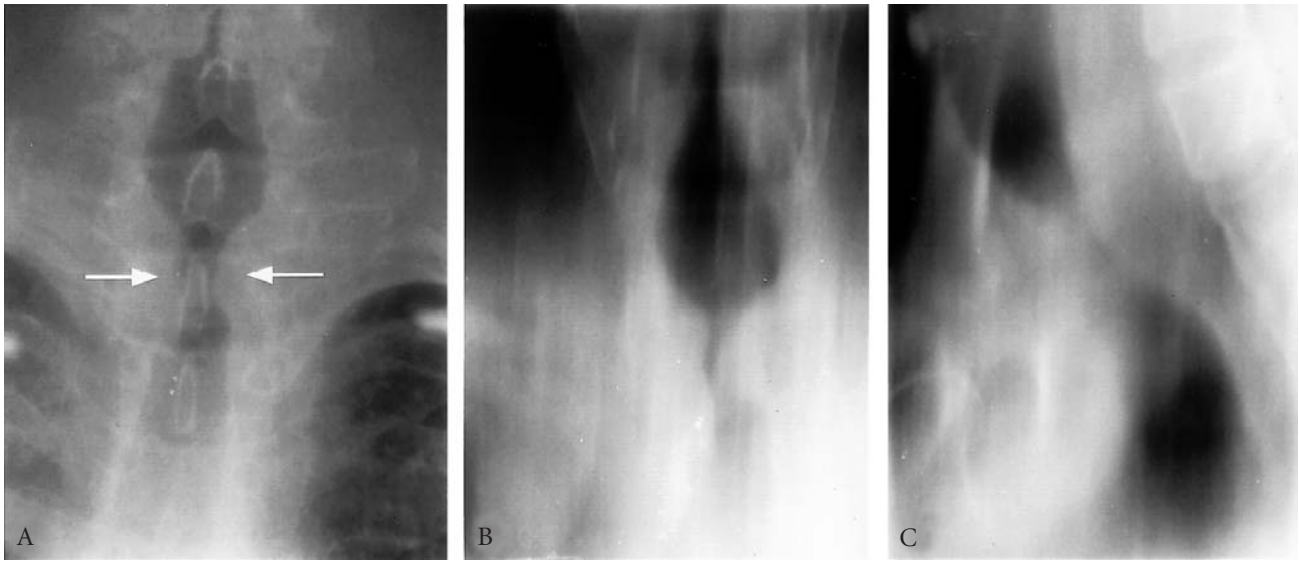


FIGURE **4-24** *Stomal stenosis of the cervical trachea.* A, *Anteroposterior high-kilovoltage view outlines a localized stenosis in the cervical trachea* (arrows). B, *Coronal tomogram shows the polypoid configuration of the stenosis.* C, *Lateral tomogram shows the stenosis chiefly anteriorly and laterally.*

When the tracheostomy tube is removed, it may act as a ball-valve mechanism and obstruct the tracheal lumen. These flaps are optimally demonstrated on lateral neck films as a variable and sometimes mass-like soft tissue density above the stomal opening.

A cuff stenosis occurs 1 to 2 cm below the tracheal stoma and is circumferential in configuration. Inflammatory histologic changes are noted within 24 to 48 hours following intubation. The inflammation leads to superficial tracheitis and mucosal ulceration within 1 week. Deeper mucosal ulceration may develop, along with exposure of the underlying cartilage, in 1 to 3 weeks. If the inflammatory process is not halted, cartilage will be exposed and chondritis will ensue, with fragmentation and eventual total destruction of the cartilaginous supporting structures in a period of 2 to 3 weeks following the intubation. Reparative healing will supersede the inflammatory process and lead to fibrotic change and formation of granulation tissue with tracheal narrowing.[40] The length and severity of the stenosis are related to the pressure within the cuff, size and shape of the cuff, the number of days of intubation, and the peak inspiratory pressure. The stenosis is usually circumferential and from 1 to 4 cm in length. In its fully developed stage, the lesion at the cuff site may vary in severity, from a circumferential diaphragm of fresh granulation tissue to dense rings of mature fibrous tissue partially covered by metaplastic squamous epithelium, extending over a variable length. A preliminary high-kilovoltage oblique film of the trachea defines the level and length of the stenosis (Figure 4-25). For detailed assessment of the lumen, thickness of the tracheal wall, and presence of mural calcification, CT is currently indicated, whereas in previous years, tomography was performed (Figures 4-26, 4-27).

Tracheomalacia develops, in a small percentage of cases, above the cuff stenosis site or at the stomal site. When the full thickness of the tracheal wall has been damaged, so that cartilages are no longer present in the area of injury, malacia may ensue. In a small number of cases, a malacic segment alone may be found at the cuff site. The malacia can be demonstrated by fluoroscopic examination with observation of the trachea during coughing and maximal inspiration and expiration. There is collapse of the entire tracheal wall, especially the anterior wall. Tracheal stenosis may be associated with other findings and complications, such as vocal cord paralysis, and infraglottic stenosis. Subglottic stenosis is usually secondary to damage of the cricoid cartilage from the cuff of an endotracheal tube, or a high tracheostomy tube with erosion of the

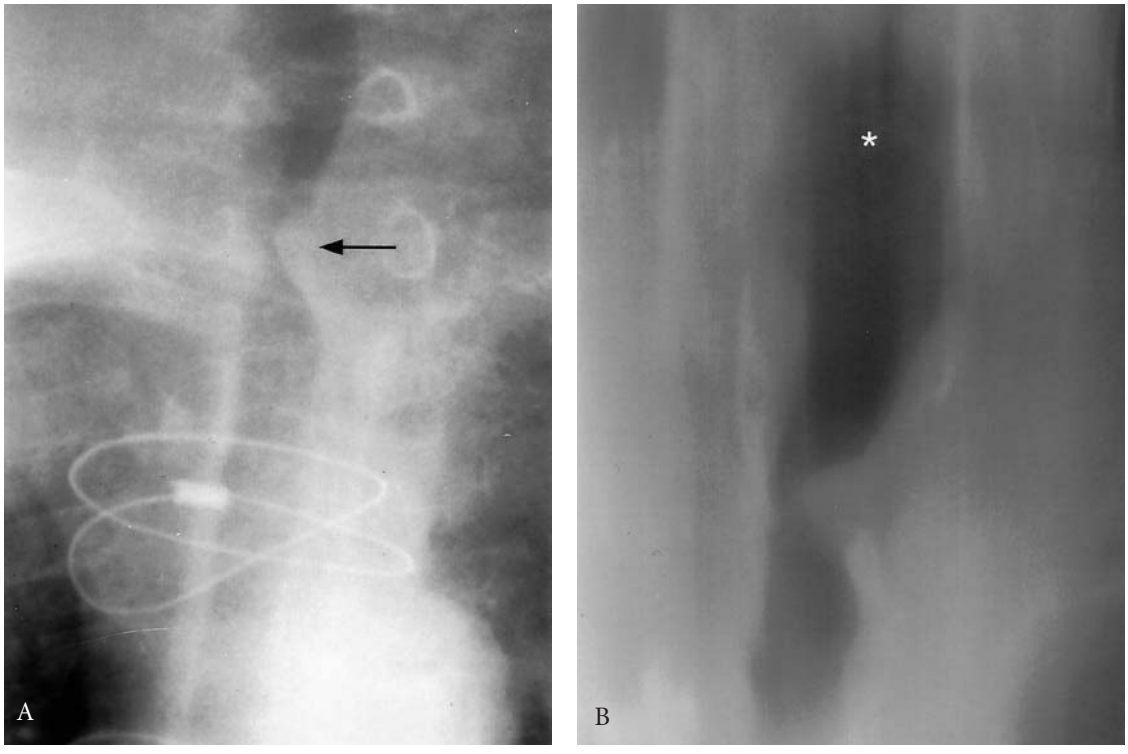


FIGURE 4-25 *Cuff stenosis of the cervical trachea.* A, *Anteroposterior high-kilovoltage view demonstrates narrowing of the trachea* (arrow). B, *Tomographic image reveals circumferential narrowing of the lumen. Note the* asterisk *in the subglottic space of the larynx.*

cricoid, or from a cricothyroidotomy. In a small number of cases, tracheoesophageal fistula may develop, followed by a sudden increase in profuse secretions plus food aspiration.[42] These fistulas are best illustrated with a barium swallow. Different types of foreign bodies may lodge in the trachea, especially in young children, followed by recurrent obstructive pneumonitis, if undetected by either radiographic means or bronchoscopy. Foreign body location in the larynx is uncommon but has occasionally been encountered in adults (Figure 4-28).

Saber-Sheath Trachea

Saber-sheath trachea is a condition closely related to chronic obstructive lung disease, especially chronic bronchitis.[43,44] In this condition, there is coronal narrowing and sagittal widening of the intrathoracic trachea, with a tracheal index (coronal/sagittal diameter) < 0.5 at the level of the aortic arch. It has been theorized that repeated excessive tracheal collapse during chronic coughing leads to degenerative softening, revascularization, and ossification of the tracheal cartilages, resulting in a fixed coronal narrowing.

Radiologically, there is a normal circular cervical tracheal configuration with an abrupt transition to a saber-sheath configuration, beginning at the thoracic inlet and extending to the carina (Figure 4-29). The tracheal cartilages may ossify in this condition, but the trachea otherwise maintains a smooth outline. Tracheomalacia is not a recognized feature.

Granulomatous Lesions

The surface contour of the endolarynx is smooth and symmetrical in inflammatory conditions, with the exception of granulomatous diseases. There is usually preservation of mobility of intralaryngeal structures,

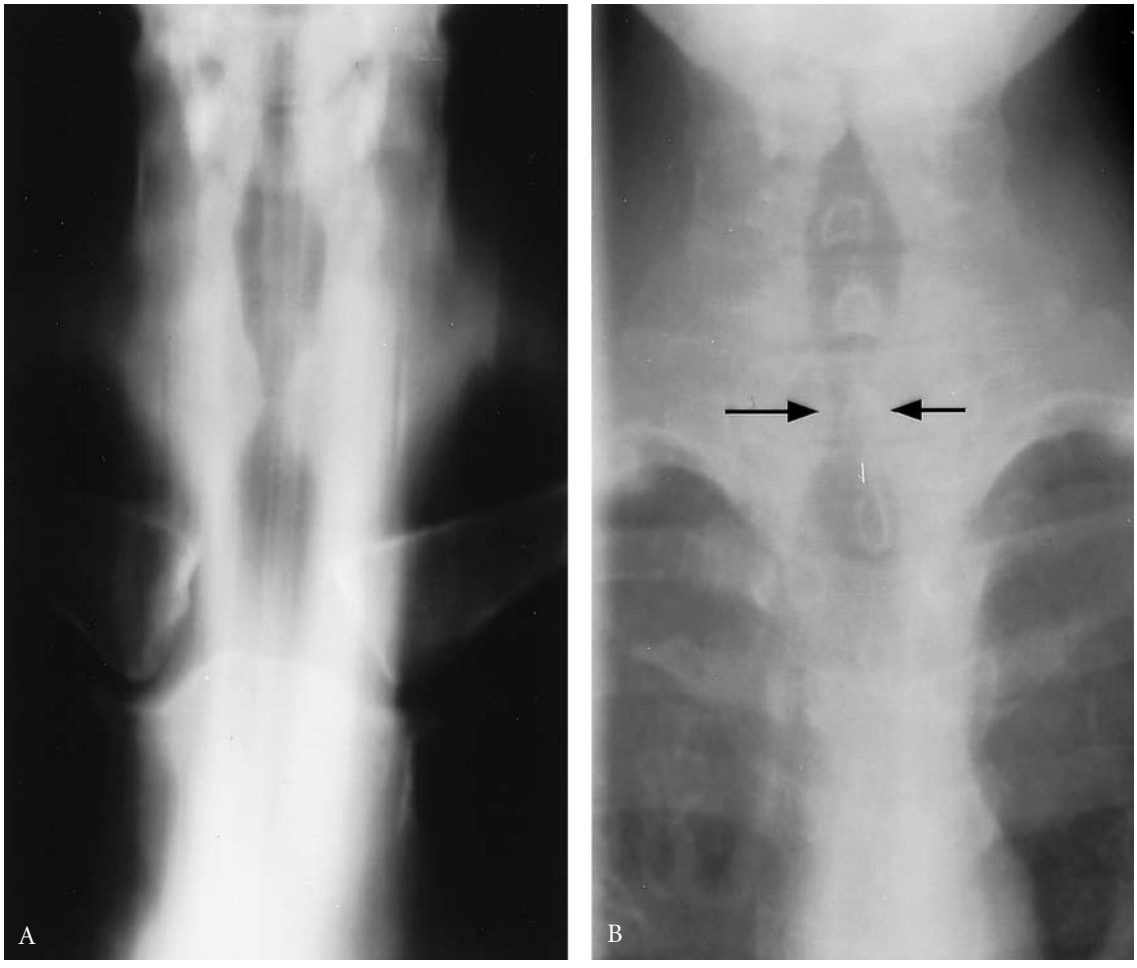


FIGURE **4-26** *Cuff stenosis of the cervical trachea.* A, *Coronal tomogram delineates circumferential narrowing of the trachea.* B, *Anteroposterior high-kilovoltage view illustrates narrowing of the lumen* (arrows).

although slight limitation may occur. Chronic granulomatous lesions of the larynx display a diffuse or localized nodular soft-tissue thickening. Frequently, a malignant tumor cannot be differentiated from the granulomatous process, and a biopsy is often mandatory for a definitive diagnosis. Granulomatous processes may extend from the subglottic part of the larynx into the cervical trachea.

Tuberculosis

Larynx. Tuberculosis of the larynx is usually secondary to pulmonary tuberculosis and commonly affects the posterior structures of the larynx. Diffuse swelling or a localized irregular mass may be found, depending on whether the pathologic process is acute, exudative, or chronic productive.[45]

Tracheobronchial. Tuberculous tracheobronchial stenosis may be caused by extrinsic compression or by adjacent lymphadenopathy or by granulomatous changes within the airway (Figure 4-30). In the hyperplastic stage, tubercles form in the submucosal layer, and ulceration and necrosis of the wall ensues. In the fibrostenotic stage, a smooth stenosis is formed.[46] Radiographically, in the hyperplastic stage, the tracheobronchial walls will be nodular and thickened with variable degrees of stenosis (Figure 4-31). Associated lymphadenopathy may demonstrate rim enhancement with intravenous contrast. There may be parenchymal

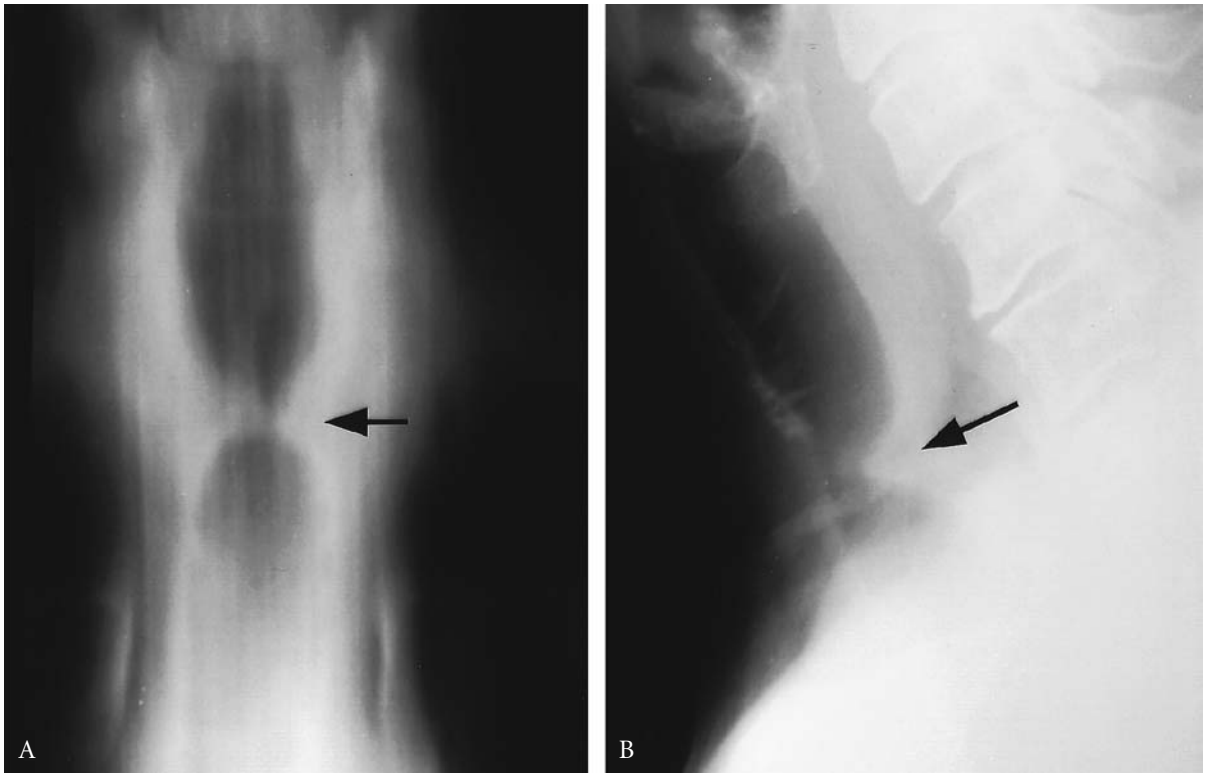


FIGURE **4-27** *Cuff stenosis of the cervical trachea.* A, *Coronal tomographic section reveals a localized well-defined cuff stenosis* (arrow). B, *Lateral view of the cervical trachea demonstrates the stenotic area, chiefly posteriorly* (arrow).

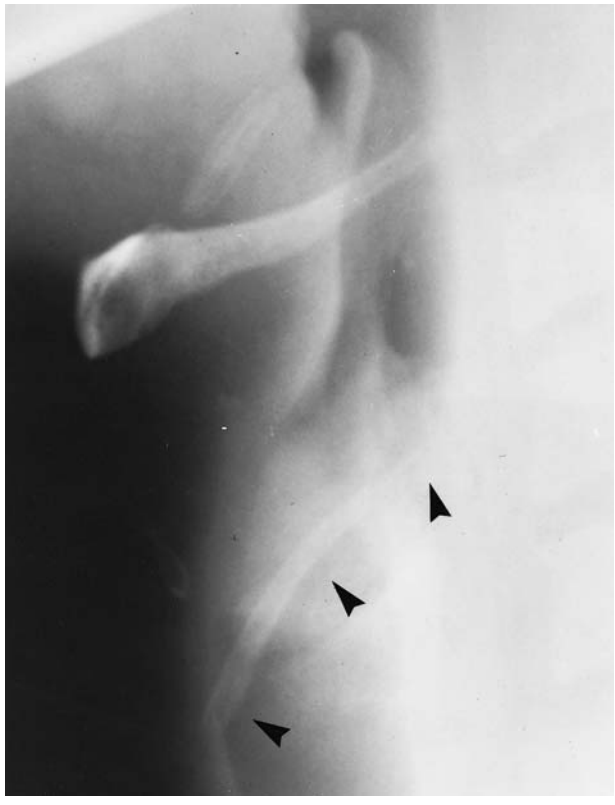


FIGURE **4-28** *Chicken bone in the larynx. Lateral neck view demonstrates a chicken bone in the larynx* (arrows).

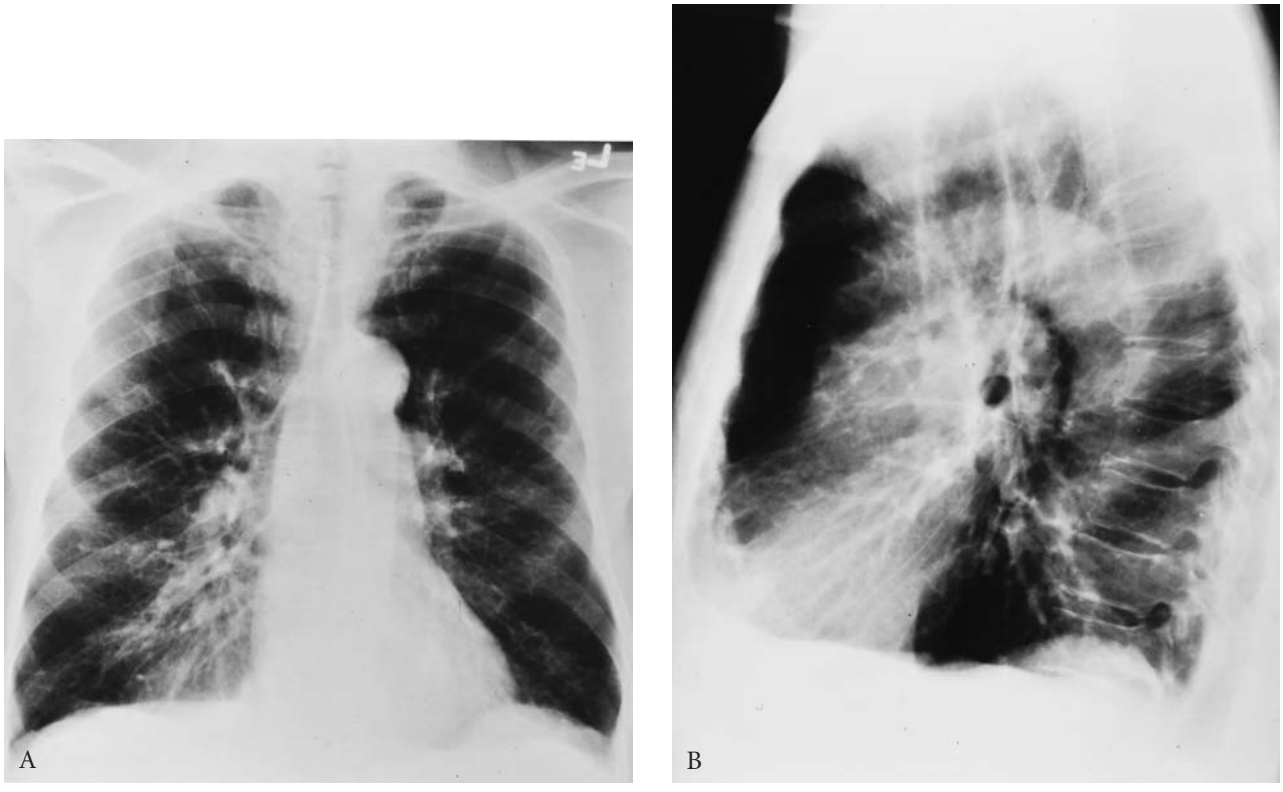


FIGURE **4-29** *Saber-sheath trachea. Posteroanterior* (A) *and lateral* (B) *chest radiographs demonstrate narrowing of the trachea in the coronal plane and widening in the sagittal plane.*

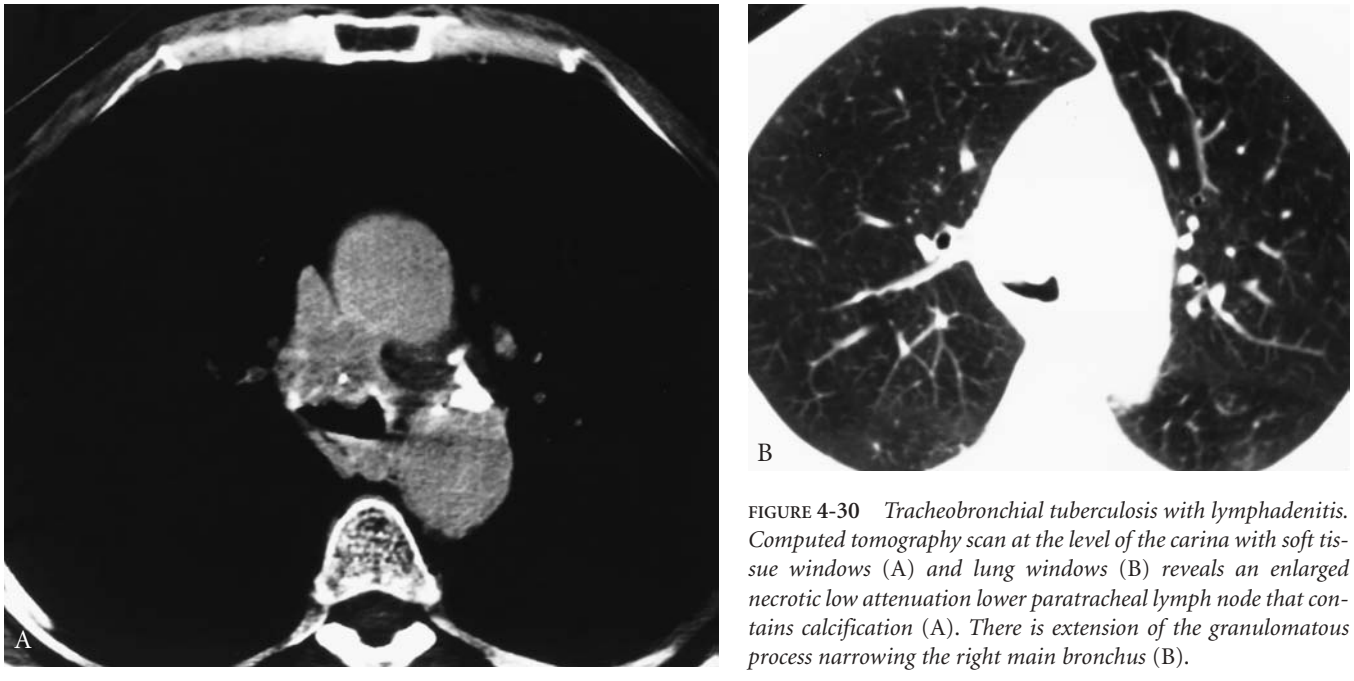


FIGURE **4-30** *Tracheobronchial tuberculosis with lymphadenitis. Computed tomography scan at the level of the carina with soft tissue windows* (A) *and lung windows* (B) *reveals an enlarged necrotic low attenuation lower paratracheal lymph node that contains calcification* (A). *There is extension of the granulomatous process narrowing the right main bronchus* (B).

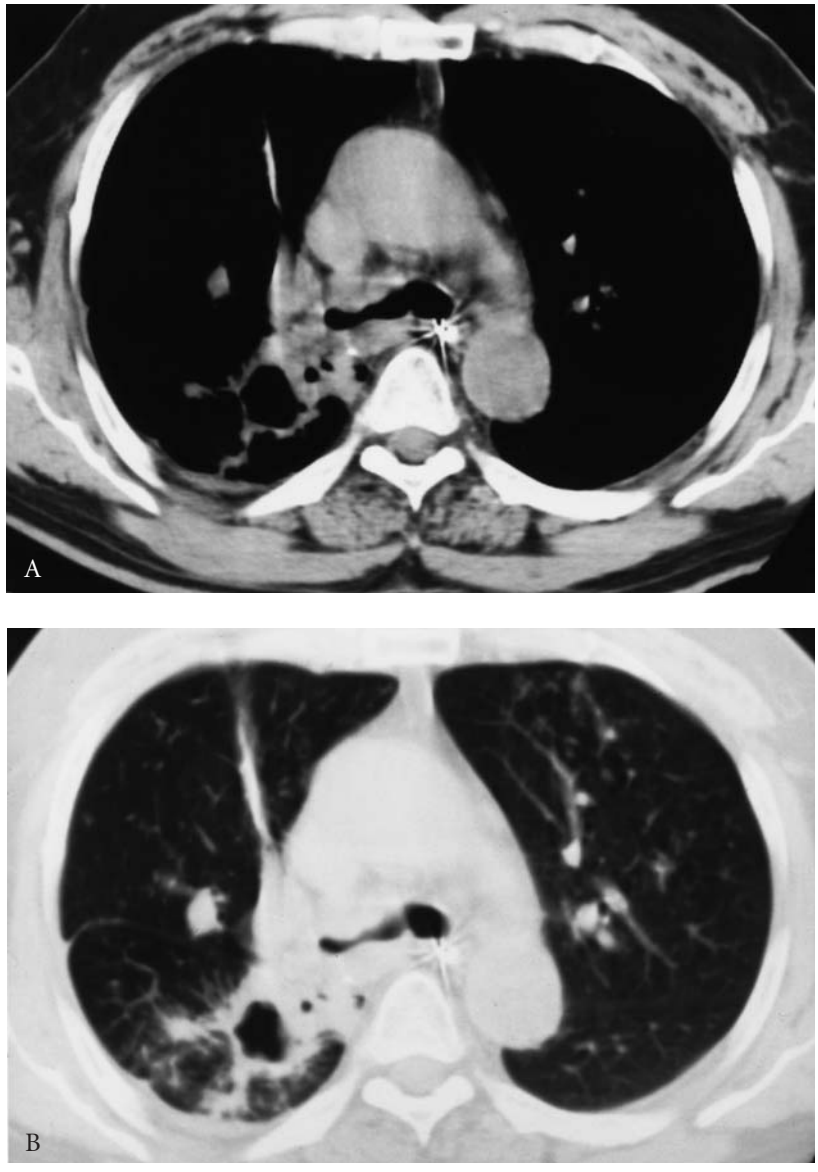


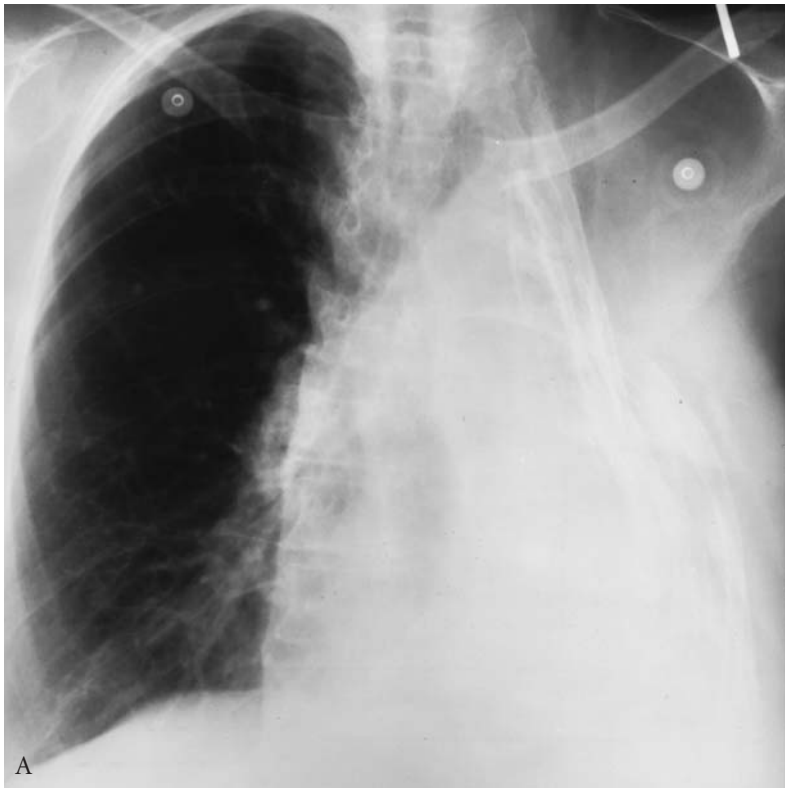
FIGURE **4-31** *Tuberculous bronchial stenosis, hyperplastic stage. Computed tomography scan demonstrates thickening and nodularity of the right main and right upper lobe bronchi associated with right hilar adenopathy* (A) *and cavitation of the right lower lobe* (B).

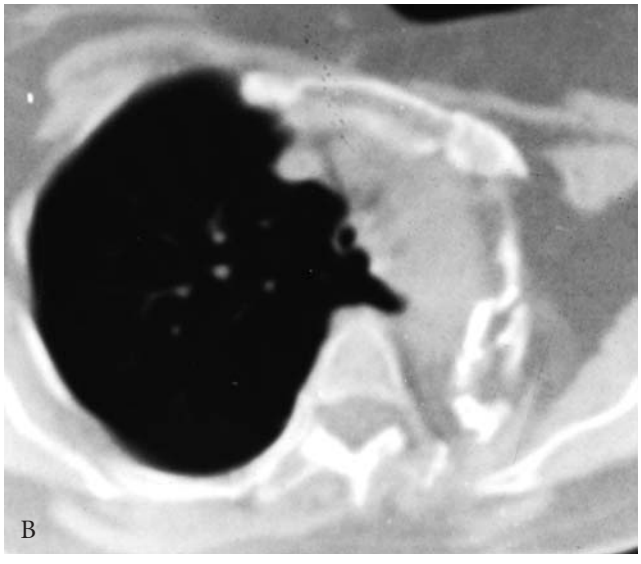
cavitation within the lobes drained by the affected bronchi. In the fibrostenotic stage, the bronchi remain thickened, but have a smooth luminal stenosis (Figure 4-32). If the stenosis is complete, total collapse of a lobe or entire lung will be present.

Broncholithiasis is a late sequela of tuberculosis or histoplasmosis. Rarely, calcified mediastinal nodes will erode into a bronchus, causing obstructive complications such as atelectasis, repeated pneumonia, or bronchiectasis. CT is useful to identify the presence and extent of the obstructing bronchial lesion (Figure 4-33). Calcification within the broncholith and associated mediastinal lymph nodes helps to establish the diagnosis.

Fungal Disease

Fungal diseases, such as blastomycosis, candidiasis, histoplasmosis, mucormycosis, rhinosporidiosis, and coccidioidomycosis, produce radiographic intrinsic stenosis of the central airways similar to those described with tuberculosis (Figure 4-34).





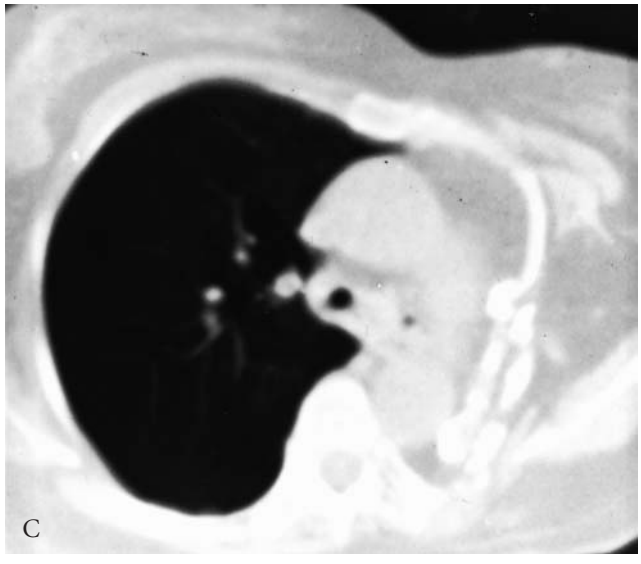


FIGURE **4-32** *Tuberculous stenosis of the trachea and main bronchi, fibrotic stage.* A, *Chest radiograph reveals collapse of the left lung. Computed tomography scans reveal diffuse smooth stenosis of the trachea* (B) *and main bronchi* (C). *There is collapse of the left lung and a calcific left fibrothorax.*

Fibrosing mediastinitis is a complication of granulomatous mediastinitis, resulting from infection from *Histoplasma capsulatum* and, less commonly, *Mycobacterium tuberculosis*. It has also been associated with the use of methysergide. Fibrosing mediastinitis is characterized by a proliferation of fibrous tissue in the mediastinum, which surrounds, invades, and sometimes obliterates normal structures, including the trachea and bronchi, esophagus, vena cava, pulmonary veins and arteries, and thoracic duct. Calcification is often present within the mediastinal fibrosis, identifiable on chest radiographs and CT (Figure 4-35). Contrast enhanced CT or MRI is useful to evaluate vascular invasion or occlusion by the fibrosis.[47,48]

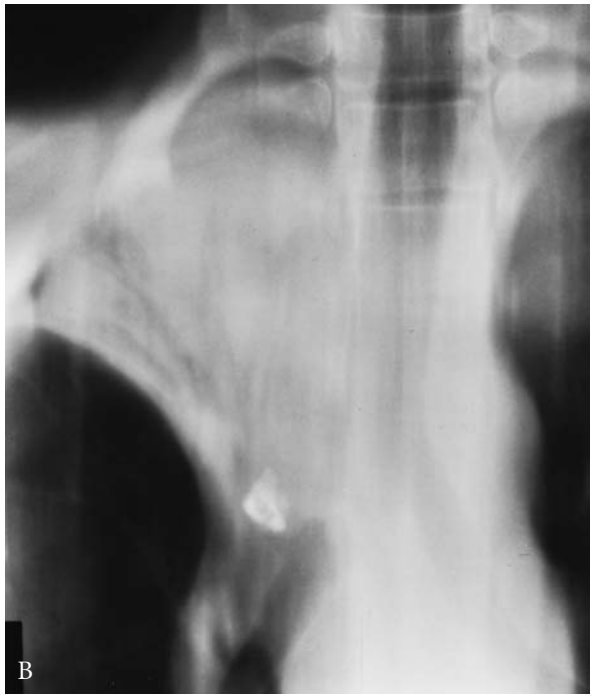
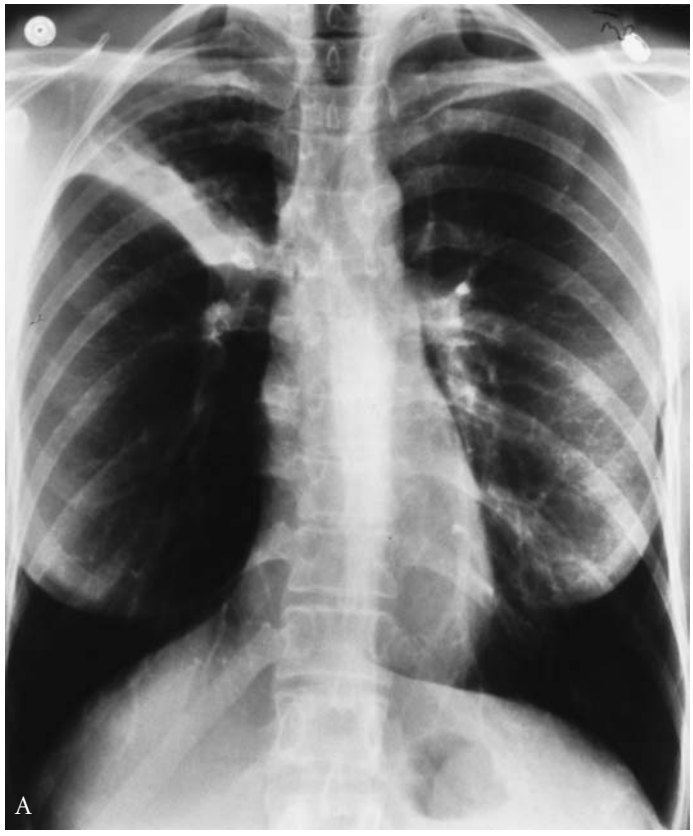
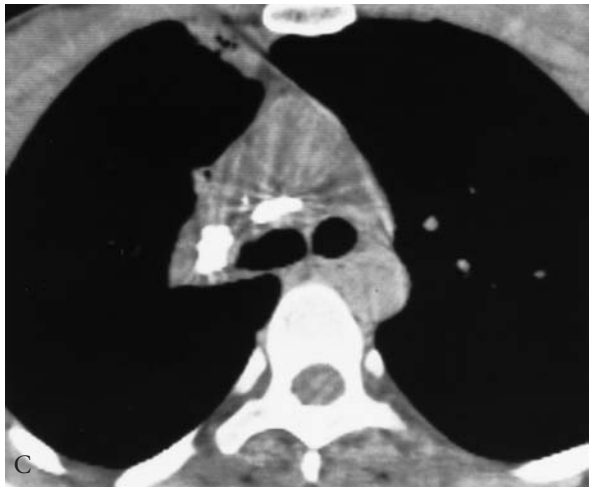


FIGURE **4-33** *Tuberculous broncholithiasis. The posteroanterior chest radiograph* (A) *and anteroposterior tomogram* (B) *reveal collapse of the right upper lobe distal to a calcified right upper lobe-filling defect, consistent with a broncholith.* C, *A computed tomography scan reveals complete obstruction of the right upper lobe bronchus by a calcified endobronchial-filling defect associated with a densely calcified right paratracheal lymph node in a patient with healed tuberculosis.*





Sarcoidosis

Sarcoidosis of the larynx and trachea is characterized by diffuse or nodular thickening, and in some cases, a tumor-like infiltration. The epiglottis is most frequently involved. Other airway involvement includes tracheobronchial mural thickening, which may be smooth, irregular, or nodular luminal narrowing (Figure 4-36). Airway compression by lymphadenopathy may occur. Thickening of the tracheobronchial wall represents the presence of granulomas in the bronchial mucosa and along the bronchovascular interstitium, accounting for the high diagnostic success of transbronchial biopsy (Figure 4-37).[49]

Scleroma

Scleroma is caused by *Klebsiella rhinoscleromatis*, a gram-negative bacterium. It is a chronic granulomatous disorder that commonly involves the subglottic larynx and cervical trachea and is characterized by granu-

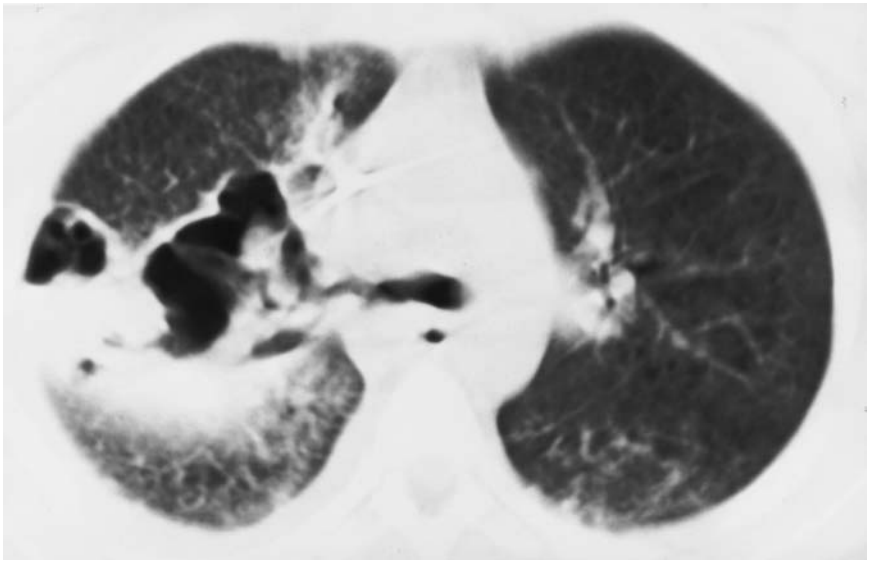


FIGURE **4-34** *Bronchial stenosis due to mucormycosis. A computed tomography scan reveals nodular stenosis of the right upper lobe and main bronchi. There is marked cavitation of the right upper lobe by the infection.*

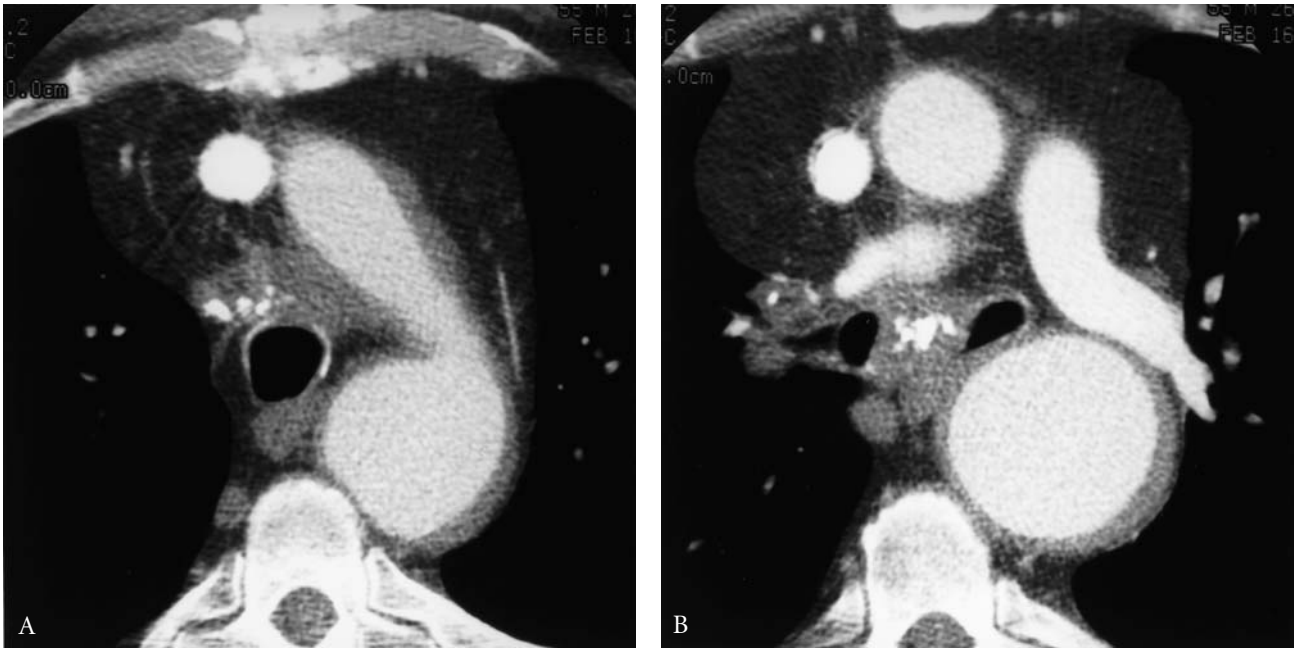


FIGURE **4-35** *Fibrosing mediastinitis. Computed tomography scans through the distal trachea* (A) *and main bronchi* (B) *with intravenous contrast reveal narrowing and distortion of the trachea and bronchi. There is surrounding fibrosis that contains calcification.*

lomatous masses or nodular and diffuse infiltrations. Although the proximal trachea is most commonly involved, the entire trachea and even the main bronchi may be affected by obstruction. The radiologic findings correlate with the three clinical stages of inflammation: 1) the catarrhal stage, 2) the granulomatous proliferative stage, and 3) the sclerotic cicatricial stage.[50]

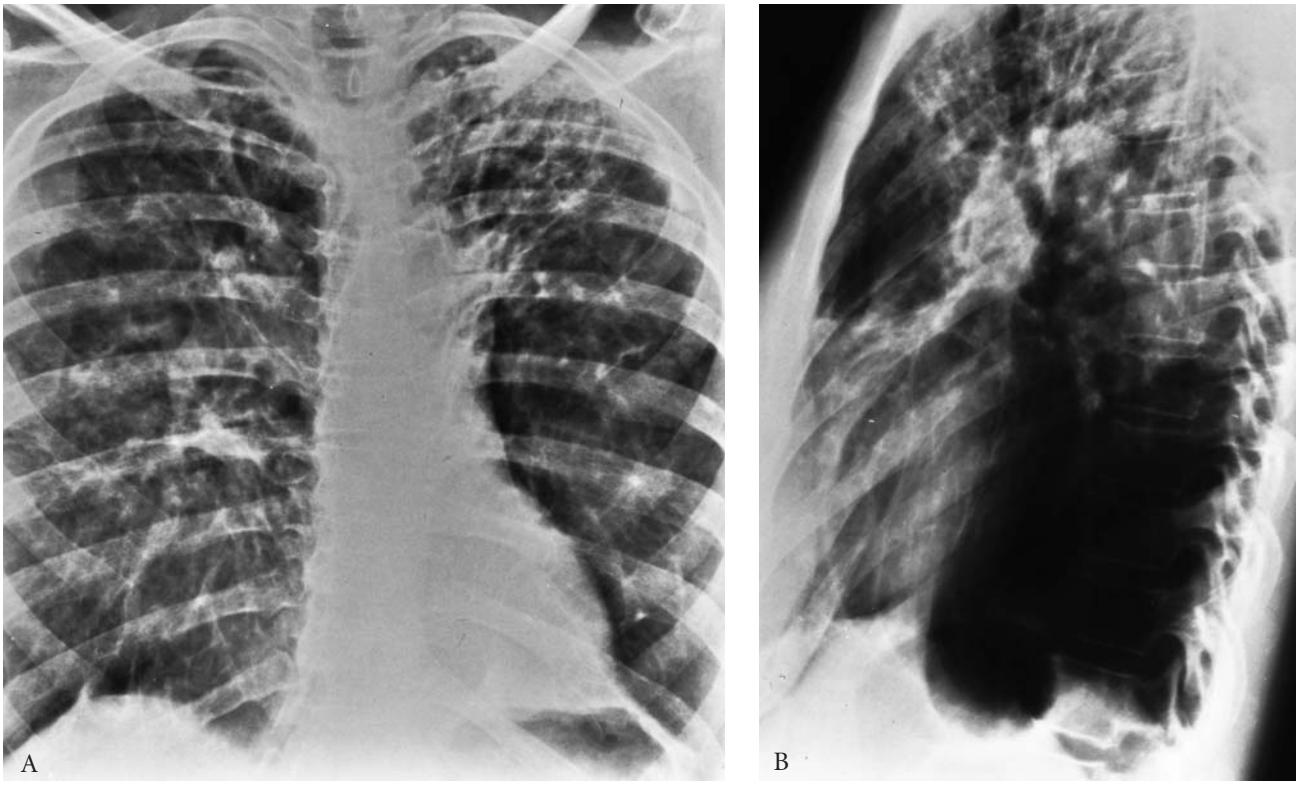


FIGURE **4-36** *Tracheal stenosis in end-stage sarcoidosis. Posteroanterior* (A) *and lateral* (B) *chest radiographs reveal marked tracheal and bronchial stenosis and distortion. There is marked pulmonary fibrosis and traction bronchiectasis related to pulmonary sarcoidosis.*

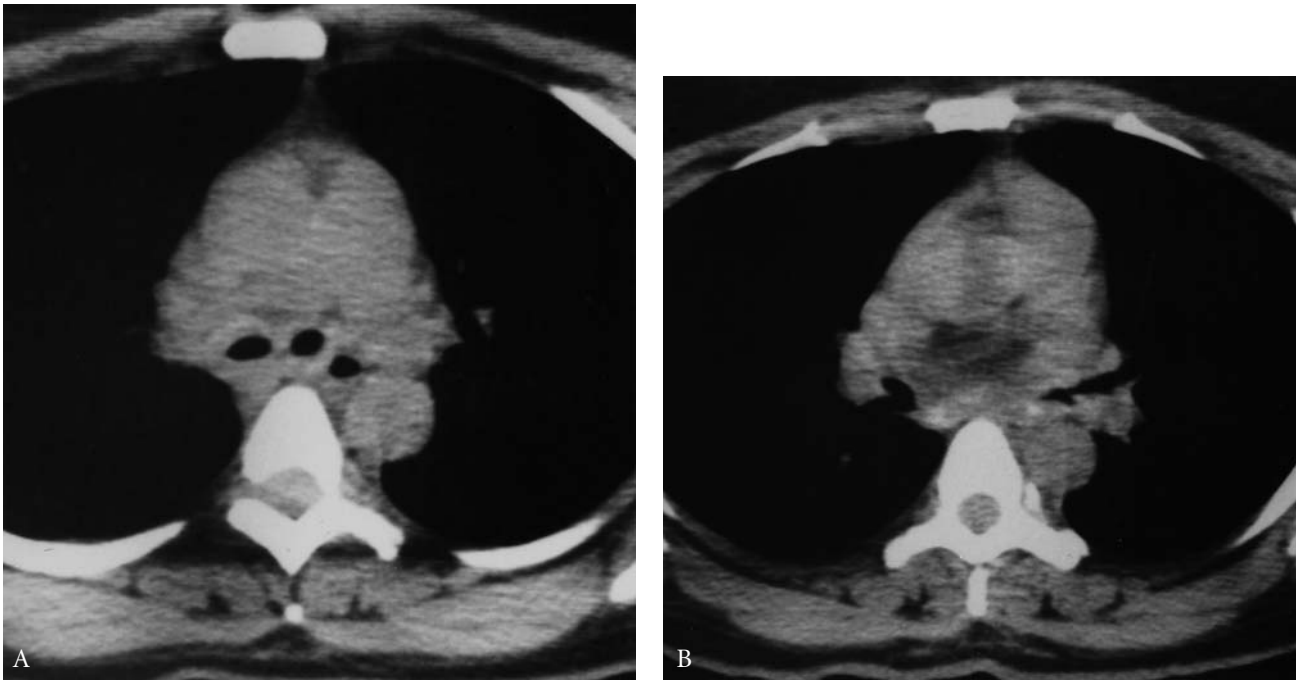


FIGURE **4-37** *Diffuse tracheobronchial stenosis in sarcoidosis. Computed tomography scans through the main bronchi* (A) *and left upper lobe bronchus* (B) *reveal diffuse bronchial wall thickening and calcification. Calcified subcarinal lymph nodes are noted.*

Wegener's Granulomatosis

Wegener's granulomatosis may involve the upper and lower respiratory tract, usually in conjunction with renal and other organ involvement.[51] Wegener's granulomatosis may involve the subglottic larynx and cause diffuse narrowing of the subglottic airway (Figure 4-38). Diffuse tracheobronchial involvement is rare and usually presents late in the disease process. Radiographic findings include tracheobronchial narrowing, thickening, and irregularity that may be focal or diffuse (Figures 4-39, 4-40). Occasionally, granulomatous tissue can obstruct a bronchus, causing atelectasis. Mediastinal and/or hilar adenopathy may be present on CT.

Other Benign Infiltrative Lesions

Idiopathic Laryngotracheal Stenosis

Idiopathic laryngotracheal stenosis is a rare cause of narrowing of the larynx and cervical trachea that typically affects middle-aged women who have no history of trauma, infection, or systemic disorder. The stenotic areas show dense keloid fibrosis involving the adventitia and the lamina propria, sparing the mucosa, muscularis propria, and the cartilages. The radiographic appearance is variable, including smooth and tapered, or irregular, lobulated, and eccentric lesions that are 2 to 4 cm in length (Figure 4-41).[52]

Relapsing Polychondritis

Relapsing polychondritis is an uncommon multisystem disease causing progressive, episodic inflammation and destruction of hyaline cartilage and other tissues. Clinical manifestations include auricular chondritis, arthritis, nasal chrondritis, ocular inflammation, respiratory tract involvement, audiovestibular damage, cardiovascular involvement, and skin disease. Respiratory tract involvement is the major cause of death. Relapsing polychondritis thickens the laryngotracheal cartilages, causing airway narrowing adjacent to the epiglottis, aryepiglottic folds, glottis, subglottis, and upper trachea. Progressive thickening of the distal trachea and bronchi may develop, but this is less common.

There is a spectrum of findings in the airways.[53,54] In the early stages of the disease, mucosal inflammation is present, causing thickening and narrowing of the central airways. As the disease progresses and

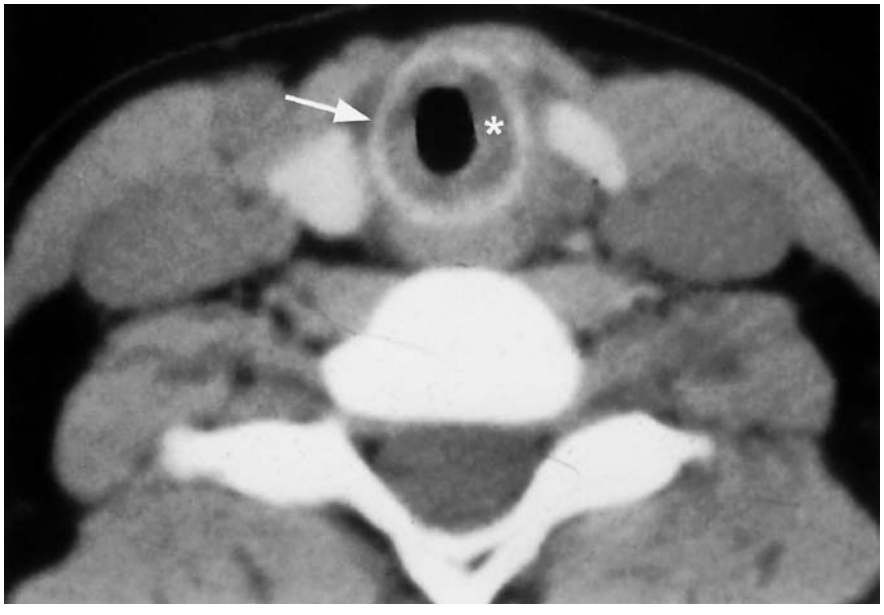


FIGURE **4-38** *Wegener's granulomatosis subglottic larynx. Axial computed tomography section shows diffuse soft tissue thickening of the subglottic larynx* (asterisk) *adjacent to the cricoid cartilage* (arrow).

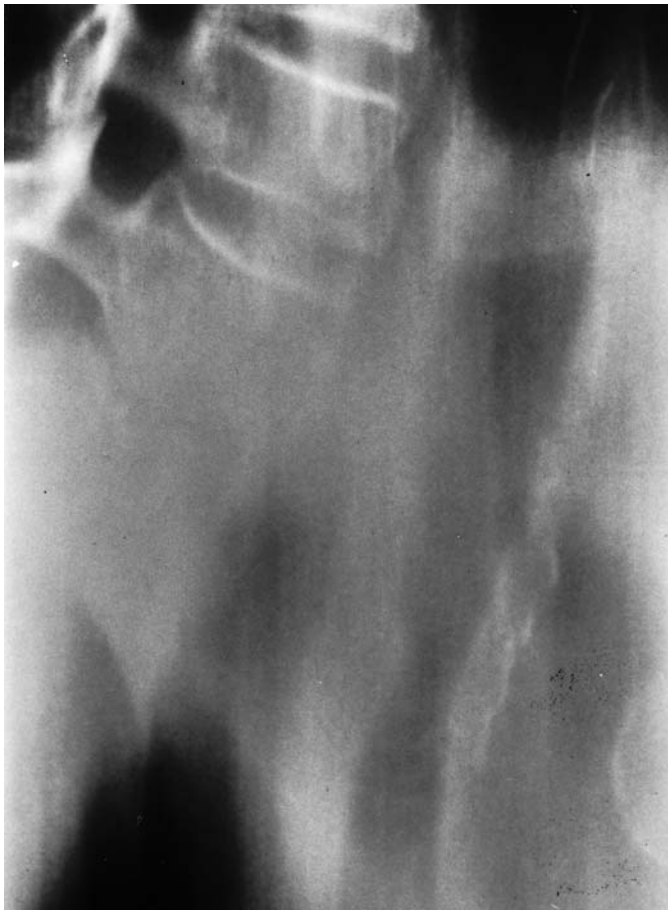


FIGURE **4-39** *Tracheal stenosis due to Wegener's granulomatosis. An oblique tomogram of the trachea reveals a diffuse tracheal stenosis with an hourglass configuration.*

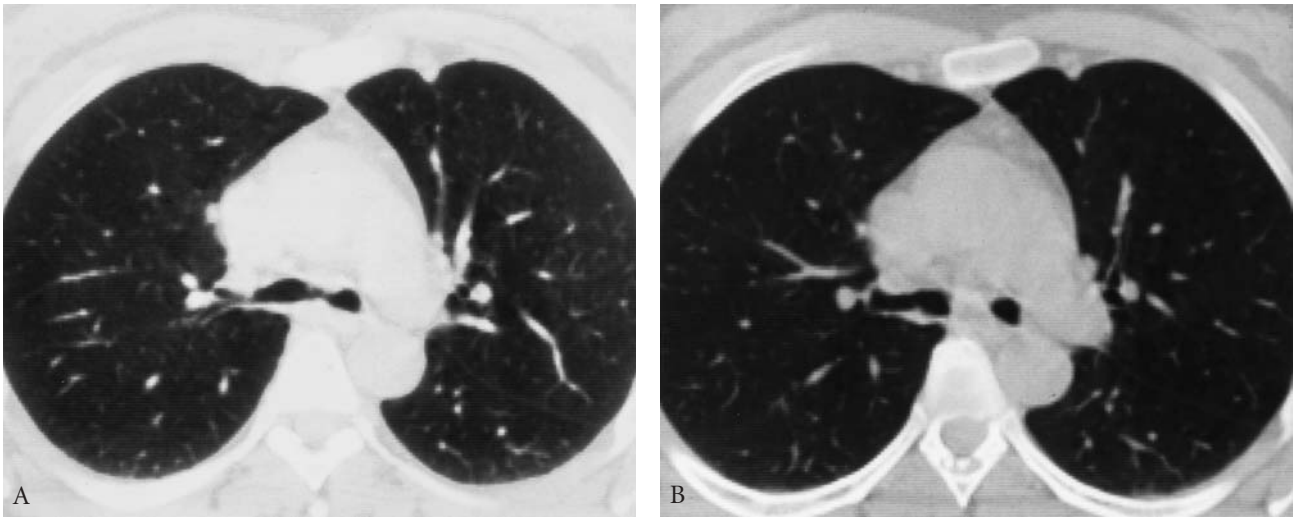


FIGURE **4-40** *Wegener's granulomatosis of the main bronchi. Computed tomography scans through the main bronchi reveal diffuse nodularity of the bronchial walls.*

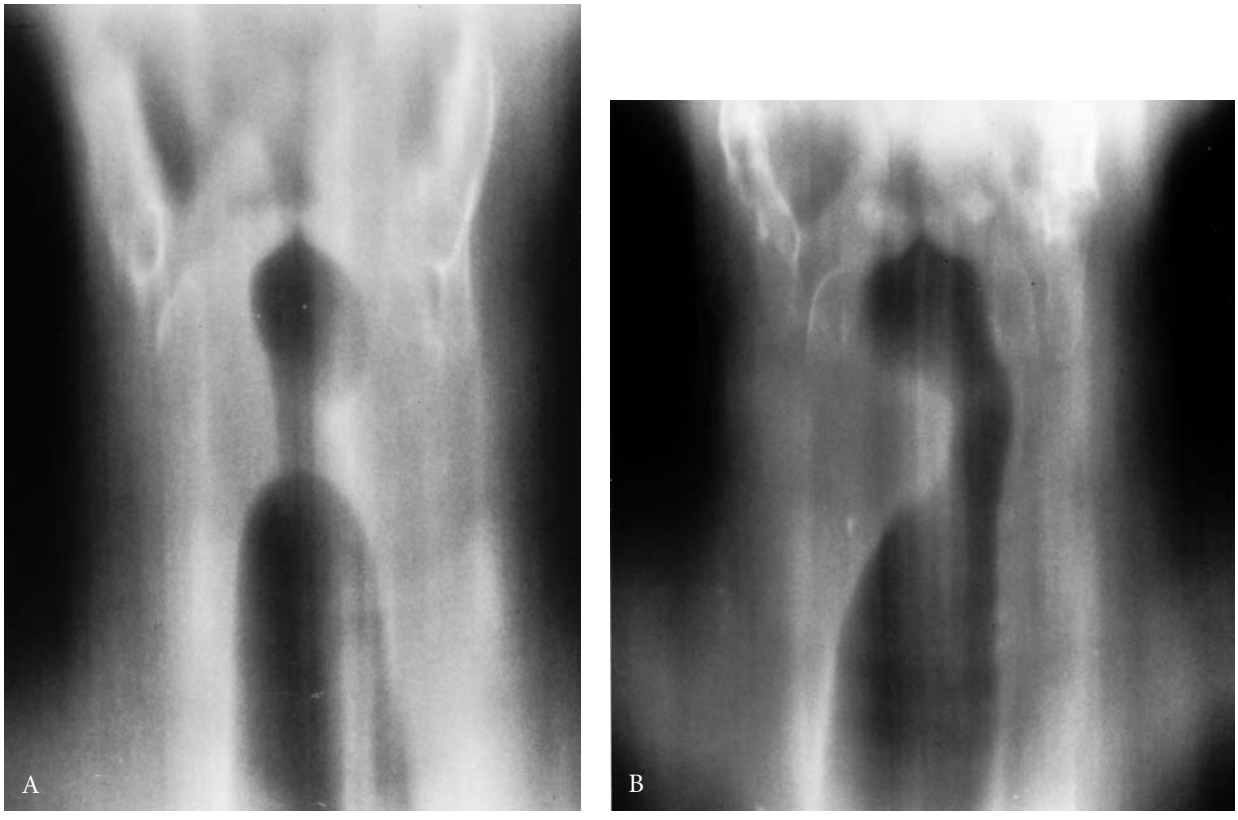


FIGURE **4-41** *Idiopathic tracheal stenosis. Anteroposterior tracheal tomograms in two separate patients reveal subglottic stenosis with a diffuse smooth stenosis* (A) *and a mass-like obstructing lesion of the proximal trachea* (B).

cartilage is destroyed, the airways become collapsible and tracheobronchomalacia develops. Once the destroyed cartilages are replaced by fibrous tissue, a diffuse fixed stenosis develops (Figure 4-42). In such cases, CT demonstrates diffuse smooth thickening of the involved trachea and bronchi (Figure 4-43). Occasionally, calcification is seen within the thickened walls.

Tracheopathia Osteochondroplastica

Tracheopathia osteochondroplastica is a rare benign condition manifested by multiple hard osteocartilaginous masses in the submucosa of the anterior and lateral walls of the cervical and intrathoracic trachea and main bronchi (Figure 4-44). The posterior membranous wall is typically uninvolved, a finding that distinguishes this disease entity from other diffuse infiltrating diseases. The size and distribution of the nodules are best depicted by CT. Submucosal nodules may range in size from a few millimeters to larger obstructing nodules. Ossification within the nodules can be identified on CT scans. The tracheobronchial wall is characteristically rigid on inspiratory/expiratory CT scans and fluoroscopy.[55]

Amyloidosis

Tracheobronchial amyloidosis results from the extracellular deposition of an insoluble protein that stains with Congo red. Airway involvement is most commonly seen in the primary form of the disease. Although any portion of the airway can be involved, distal tracheal and bronchial involvement is more common. Amyloidosis of the airway may be focal or mass-like, mimicking a tumor, or may cause diffuse thickening of the tracheobronchial wall.[56]

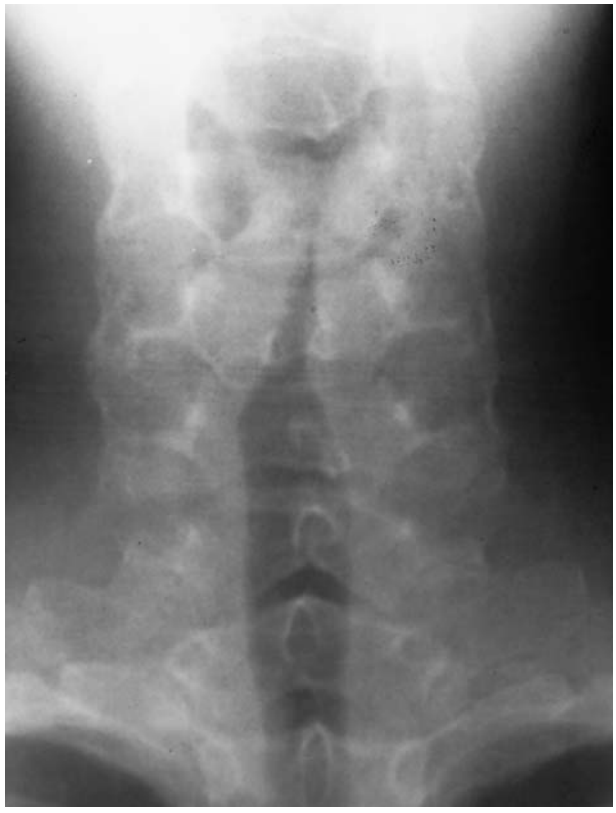
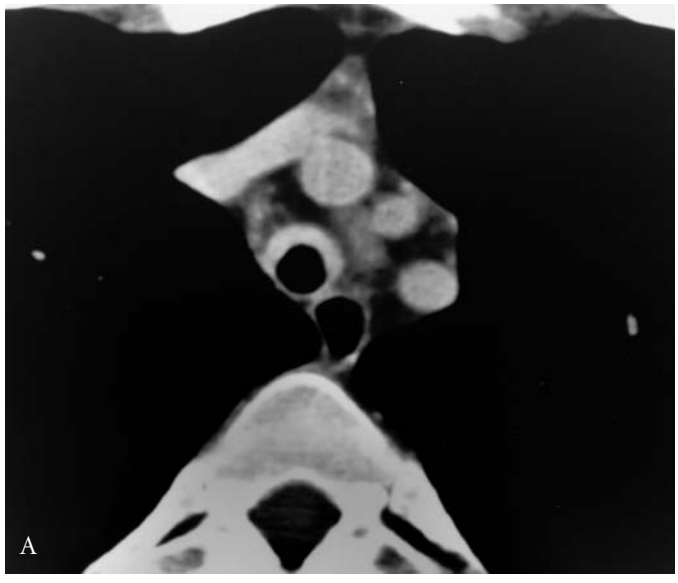


FIGURE **4-42** *Relapsing polychondritis of the glottis and proximal trachea. An anteroposterior tomogram of the proximal trachea and larynx reveals a smooth subglottic stenosis.*



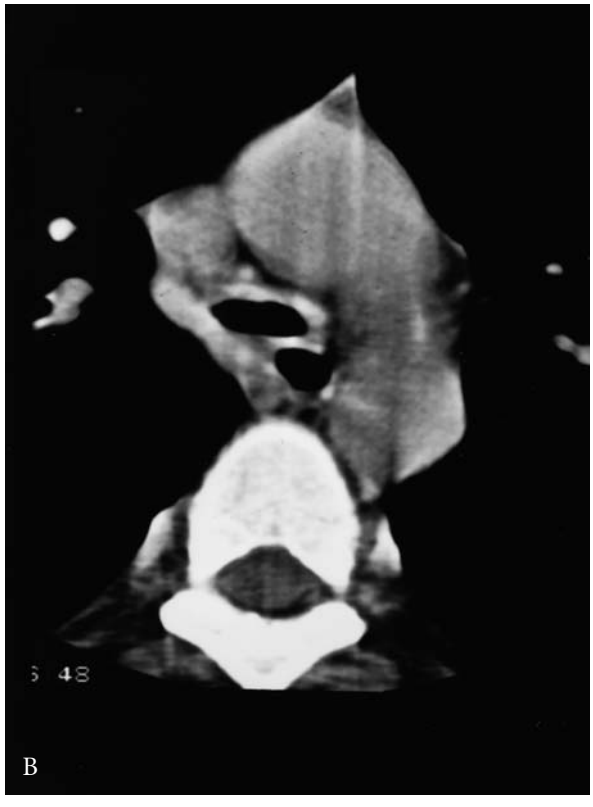


FIGURE **4-43** *Relapsing polychondritis. Computed tomography scans demonstrate diffuse thickening of the trachea* (A) *and bronchial* (B) *cartilages.*

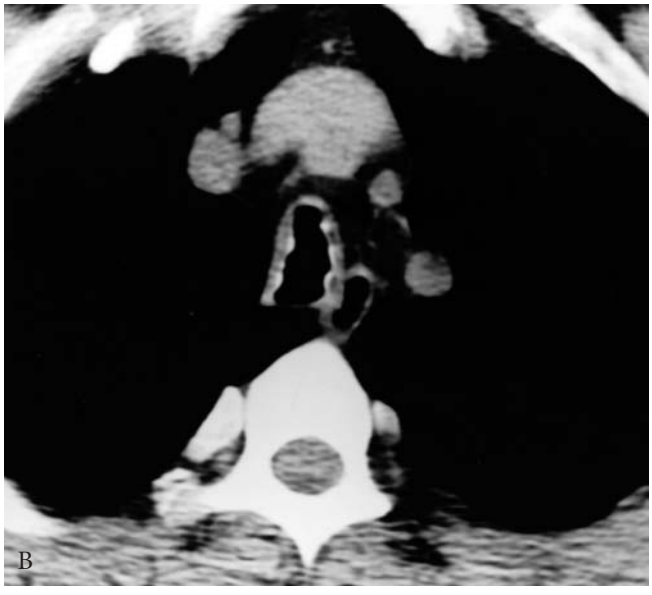


FIGURE **4-44** *Tracheopathia osteochondroplastica.* A, *Anteroposterior tomogram reveals diffuse nodularity of the trachea and bronchi.* B, *Computed tomography scan at the level of the midtrachea demonstrates thickening and nodularity of the tracheal cartilage, sparing the membranous wall.*

Radiographs and CT scans demonstrate tracheobronchial wall thickening and nodularity or, less commonly, a focal endoluminal lesion (Figures 4-45, 4-46). The airway abnormality may calcify and focal lesions may demonstrate intense vascular enhancement with intravenous contrast material on CT, or gadolinium on MRI (Figure 4-47). Associated mediastinal or hilar adenopathy is sometimes present and may contain calcification.

Benign Tumors and Cysts

Benign tumors of the trachea and larynx are very rare and account for 10% of tracheal tumors.[57,58] Ninety percent of tumors which are encountered in the pediatric age group are benign. Benign tumors represent a large group of diverse lesions that manifest as sharply-defined masses with no invasive characteristics.[20,59] They are usually homogeneous, and the majority of lesions have no characteristic features to differentiate them by radiologic means.[60]

Papillomas

Papillomatosis is the result of a multicentric viral infection with the human papilloma virus. Papillomas occur either singly or as multiple, irregular tumor excrescences that generally arise from the true vocal cords.[61] This tumor most often involves the superior surfaces or free margins of the vocal cords and, less commonly, occurs in the supraglottis and subglottis (Figure 4-48). Papillomas are divided into juvenile and adult groups, where those of the juvenile group often manifest as multiple lesions, most commonly found in the larynx. The papillomas may recur or may spread diffusely through the trachea, bronchi, and lungs following excision. In the lungs, sheets of squamous cells proliferate within alveoli, forming nodules that characteristically cavitate. The adult type of lesion often presents as a solitary mass, with a lesser propensi-

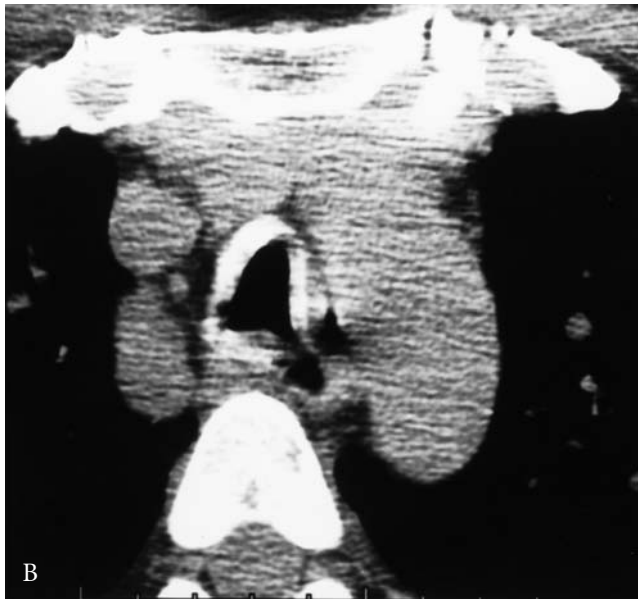
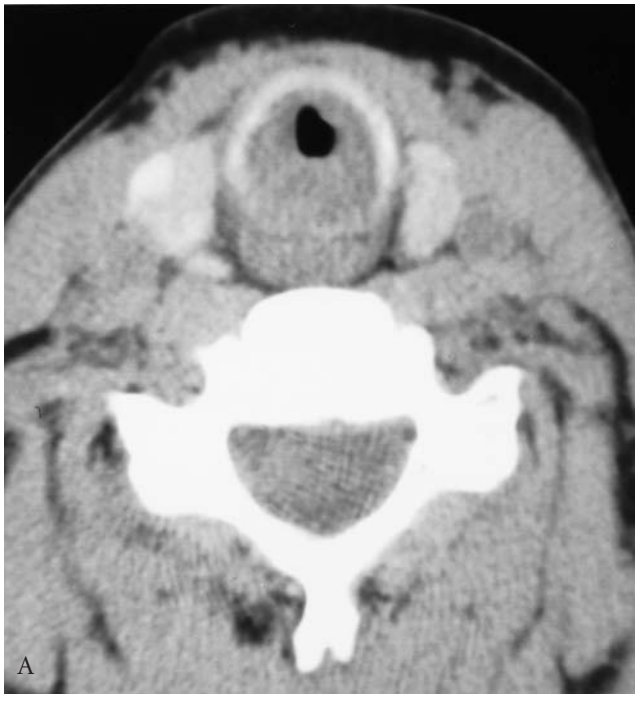
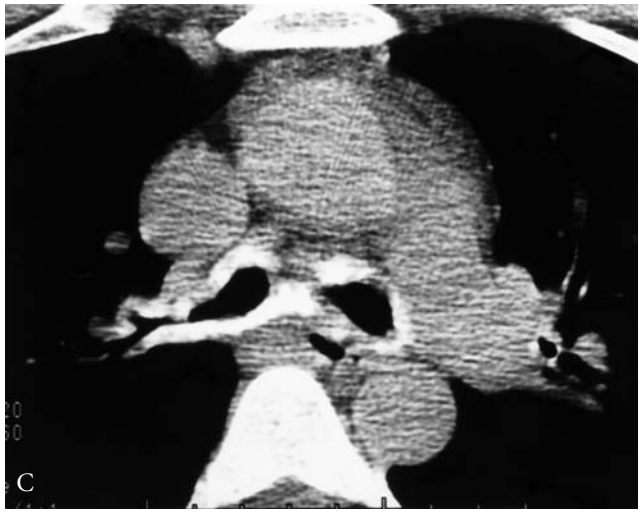


FIGURE **4-45** *Diffuse tracheobronchial amyloidosis. Computed tomography scans at the level of the cricoid cartilage* (A)*, midtrachea* (B)*, and main bronchi* (C) *reveal diffuse tracheobronchial thickening and narrowing, with calcification of the airway walls.*





ty to recur after removal. Transformation of the lesions into invasive squamous cell carcinoma is well known. Radiographically, the tracheal walls may appear thickened and nodular in appearance, either in a focal or diffuse fashion. These are sometimes visible on chest radiographs, but are best demonstrated by CT (Figure 4-49). Multiple pulmonary nodules with and without cavitation can be identified on chest radiographs and CT (Figure 4-50).

Chondromas

Chondromas of the larynx are uncommon lesions that occur most frequently in middle-aged men. The tumor is smooth, submucosal, and firm in consistency.[62] On radiographic and CT examination, the tumor presents as a sharply-defined mass and frequently contains mottled calcifications (Figure 4-51). Radiologically, a chondroma cannot be distinguished from a chondrosarcoma; this distinction may also be difficult on histopathologic examination. The most common location is the inner surface of the cricoid lamina (70%) (Figure 4-52). Less often, chondromas arise from the thyroid, arytenoid, or epiglottic cartilages. On rare occasions, chondromas may be situated on the upper surfaces, free margins, or

undersurfaces of the vocal cords. Chondromas may occur in the trachea and bronchi but do so less commonly than laryngeal chondroma.

Hemangiomas

Hemangiomas are uncommon lesions in the larynx and occur even less commonly in the trachea. The pediatric type of hemangioma usually manifests by 6 months of age, is subglottic in location, and causes signs of airway obstruction.[63] Typically, these children exhibit hoarseness, stridor, and dysphagia with poor feed-

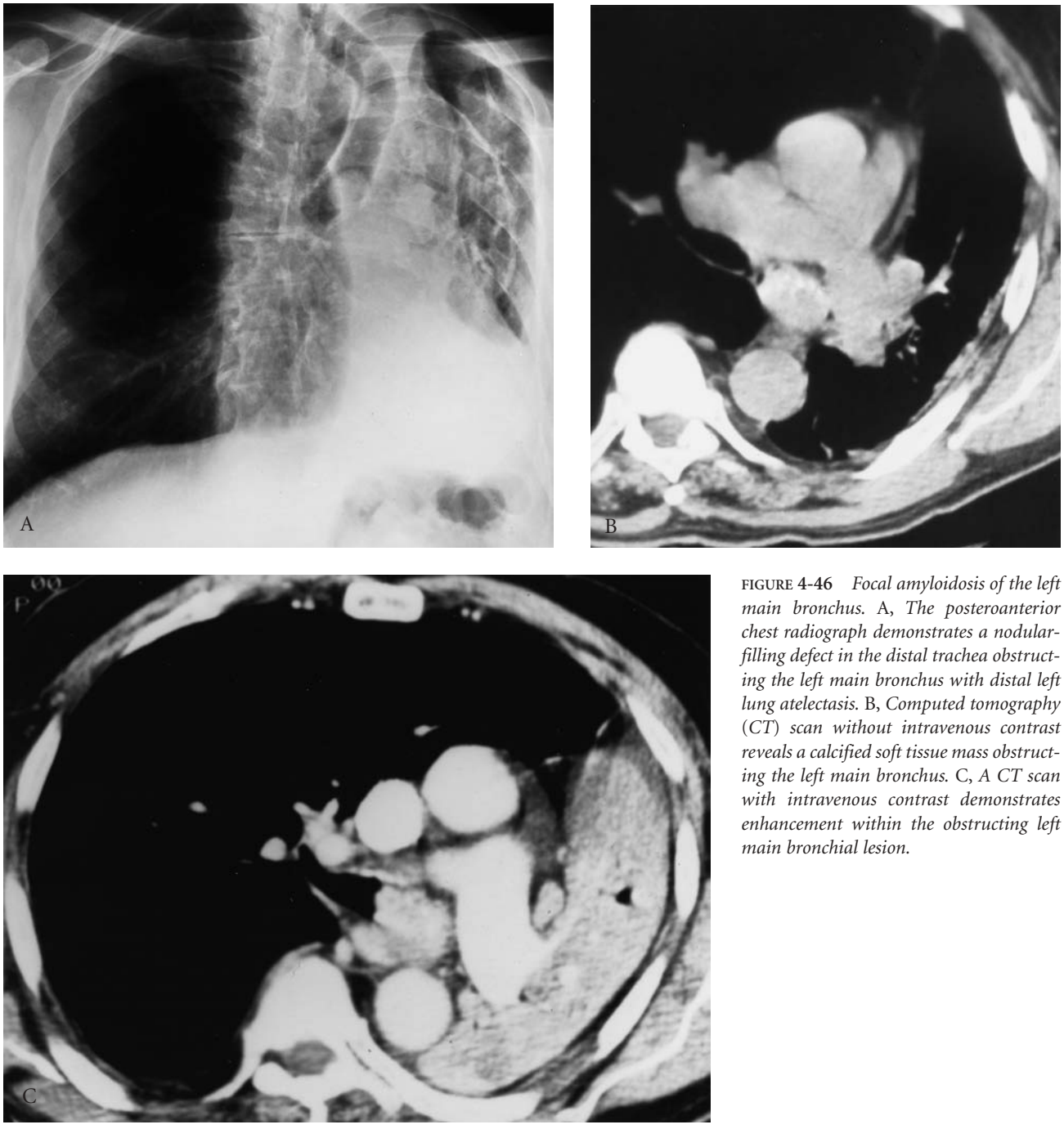
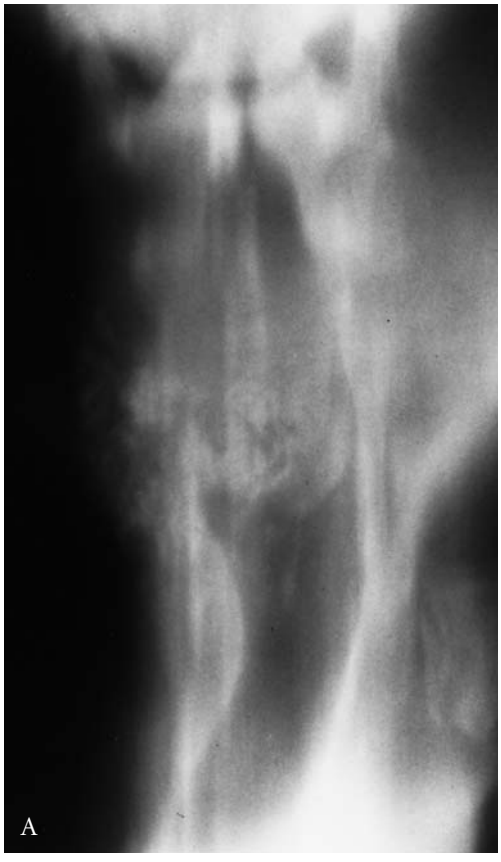
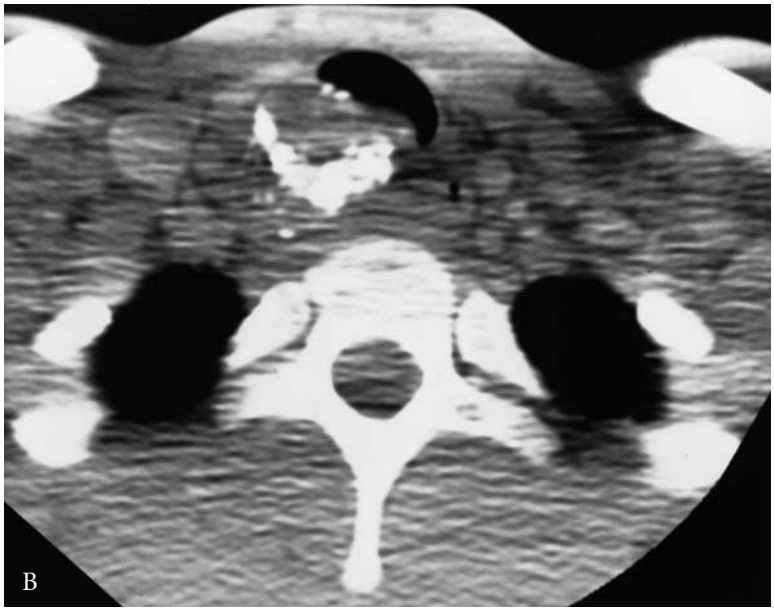


FIGURE **4-46** *Focal amyloidosis of the left main bronchus.* A, *The posteroanterior chest radiograph demonstrates a nodular-filling defect in the distal trachea obstructing the left main bronchus with distal left lung atelectasis.* B, *Computed tomography (CT) scan without intravenous contrast reveals a calcified soft tissue mass obstructing the left main bronchus.* C, *A CT scan with intravenous contrast demonstrates enhancement within the obstructing left main bronchial lesion.*





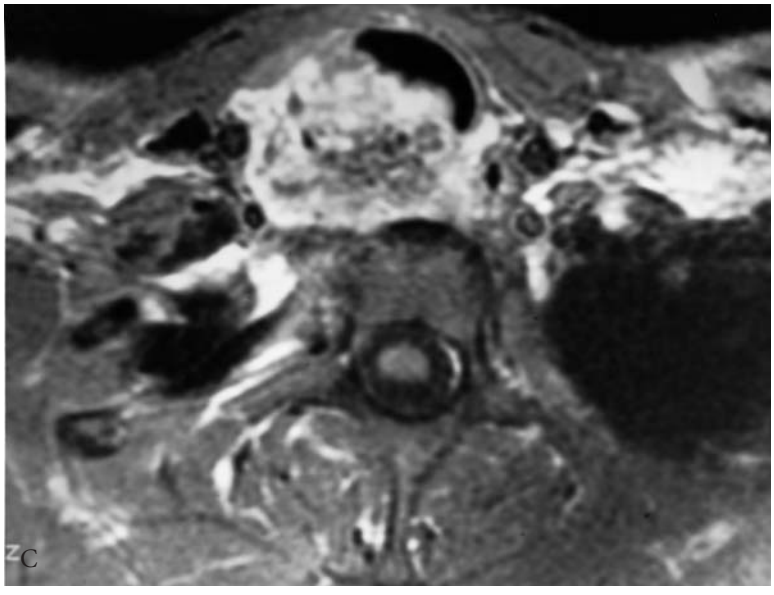


FIGURE **4-47** *Focal tracheal amyloidosis.* A, *Anteroposterior tracheal tomogram reveals a large calcified soft tissue mass nearly obstructing the proximal trachea.* B, *Computed tomography scan without contrast reveals marked calcification within the mass and extension of the mass into the right paratracheal soft tissues.* C, *T1-weighted magnetic resonance image of the proximal trachea with gadolinium reveals marked enhancement of the mass.*

ing. In the anteroposterior radiographic film, there is subglottic and often asymmetrical narrowing, and evidence of a distinctive, homogeneous, sharply-defined soft tissue mass. In adults, laryngeal hemangiomas usually arise in the supraglottic larynx as a sharply-defined, homogeneous mass that may contain phleboliths. Most of the reported cases are of the cavernous variety, as opposed to capillary hemangiomas, which are seen in infancy.

Other Benign Tumors

Miscellaneous benign tumors are encountered in the larynx and trachea. These include neurogenic tumors, pleomorphic adenoma, oncolytic tumor, granular cell tumor, paraganglioma, lipoma, fibrous histiocytoma, rhabdomyoma, and hamartomas (Figure 4-53).[64–72] Conventional radiographs do not usually reveal any characteristic features that allow a specific histopathologic diagnosis. CT can identify fatty attenuation

within hamartomas and lipomas and increased contrast enhancement within paragangliomas. A chondroid matrix can be identified within chondromas and chondrosarcomas.

Laryngoceles

Various cystic lesions are encountered in the larynx that are either retention cysts or laryngoceles. Laryngoceles are air-filled or fluid-filled outpouchings of the mucosa of the laryngeal ventricles. They extend from the ventricle into the adjacent aryepiglottic fold and are then referred to as internal laryngoceles.[73,74] If the air-filled structure herniates through the thyrohyoid membrane, external laryngoceles result, which are usually well defined. Intralaryngeal expansion leads to a variable degree of airway obstruction, depending on the size of the lesion. If these laryngoceles are filled with fluid, they manifest as homogeneous, dense masses that cannot be differentiated from a benign tumor.

Tracheal Cyst or Tracheocele

A tracheal cyst is a thin-walled air-containing paratracheal cavity that is visible on chest radiographs and chest CT scans (Figure 4-54). The tracheal cyst is a circumscribed saccular tracheal outpouching of the posterior wall of the trachea. The cyst may rarely contain an air–fluid level. Dynamic bulging can be observed during a Valsalva maneuver at fluoroscopy or on CT. This condition occurs through a localized weakness of the membranous part of the trachea and is theorized to be associated with obstructive lung disease.[75]

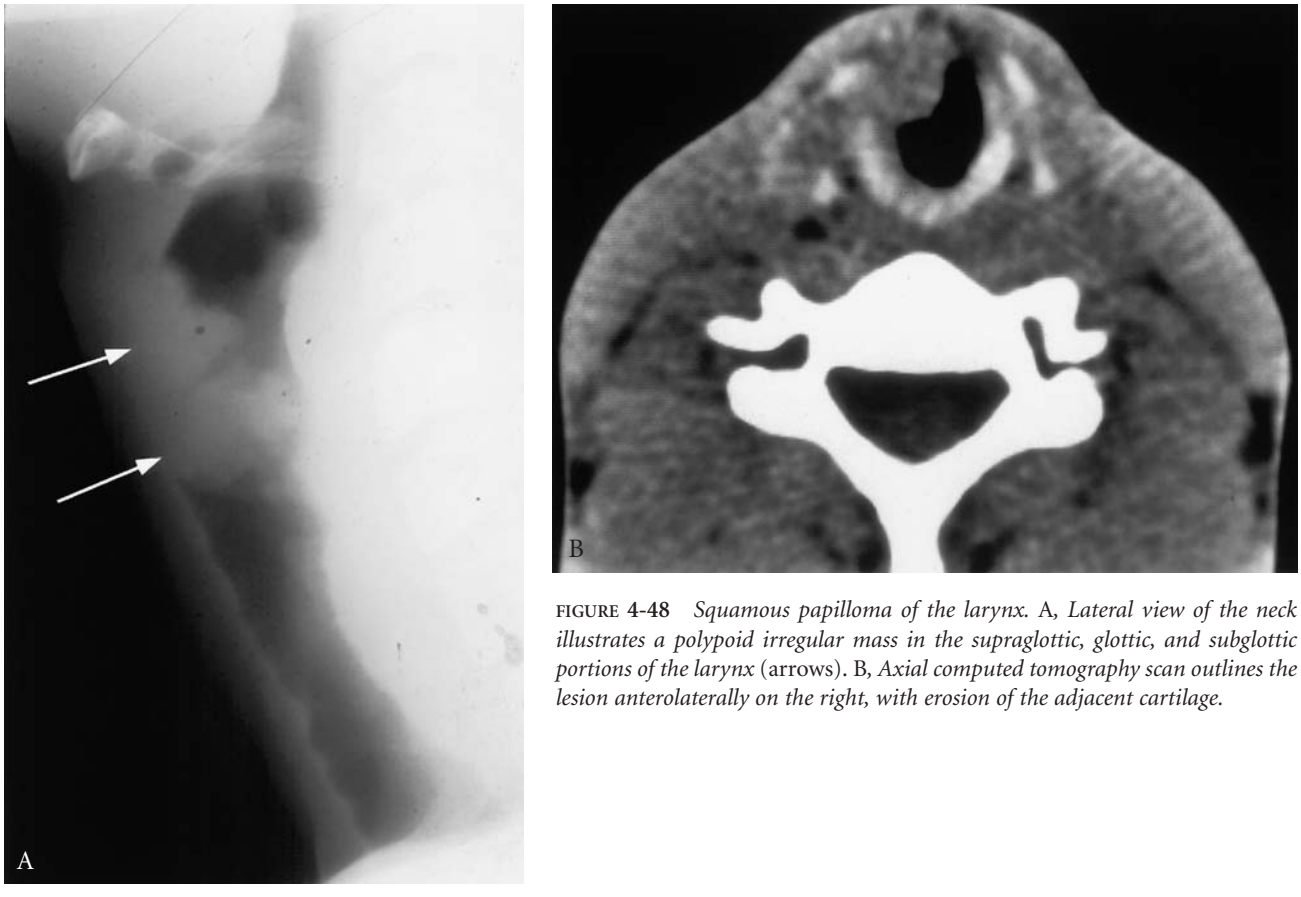


FIGURE **4-48** *Squamous papilloma of the larynx.* A, *Lateral view of the neck illustrates a polypoid irregular mass in the supraglottic, glottic, and subglottic portions of the larynx* (arrows). B, *Axial computed tomography scan outlines the lesion anterolaterally on the right, with erosion of the adjacent cartilage.*

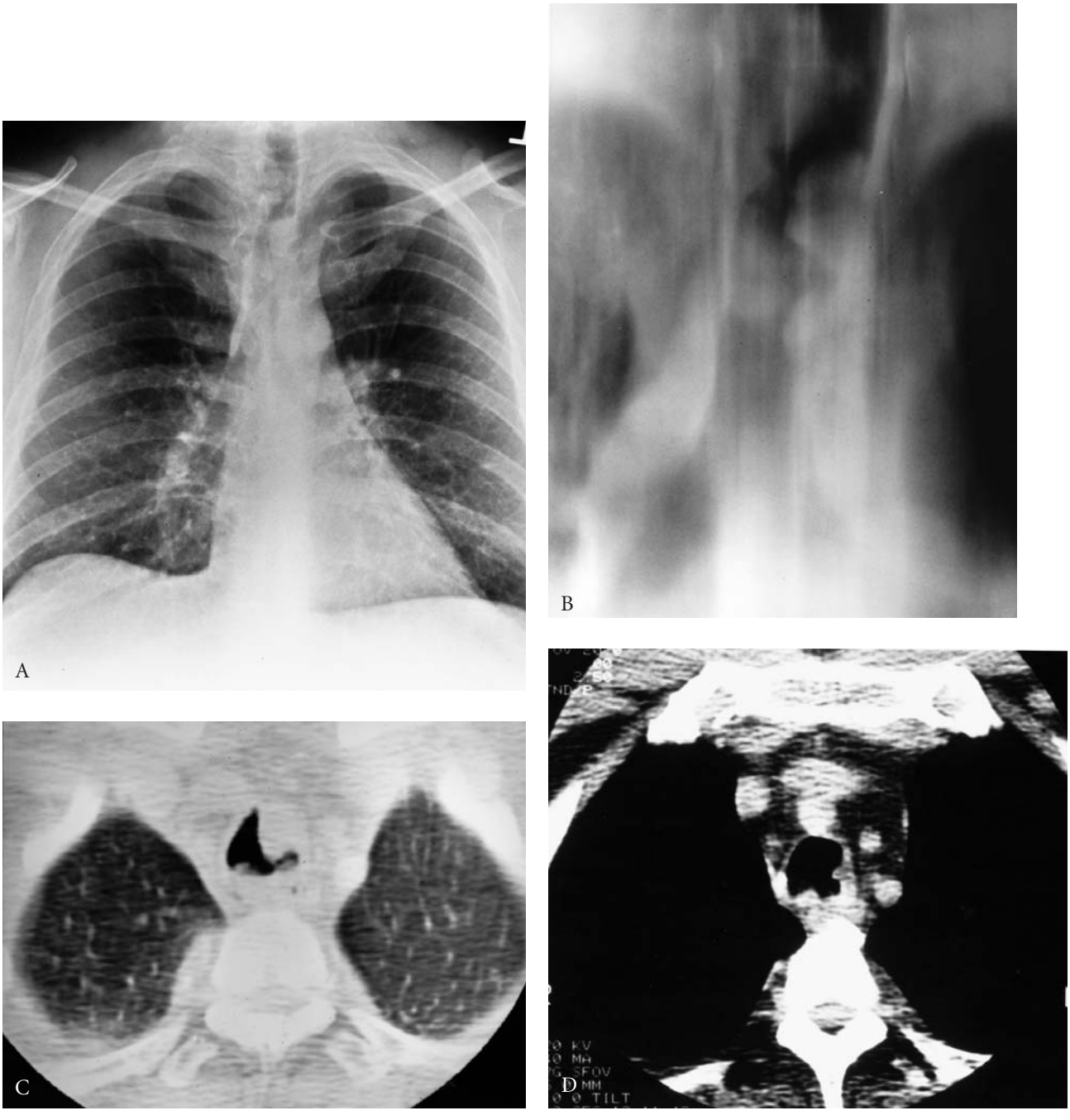
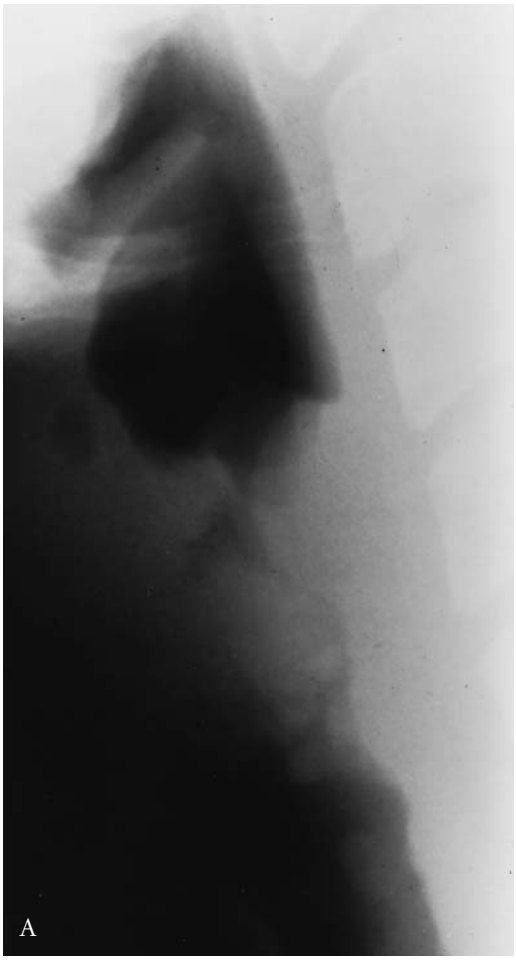
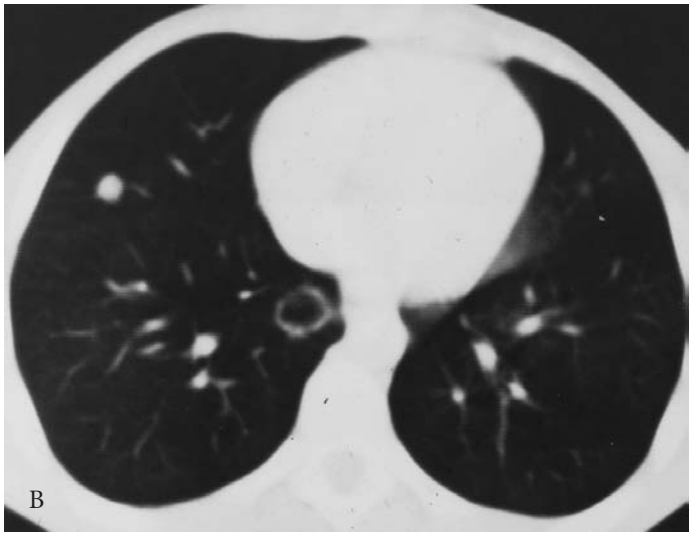
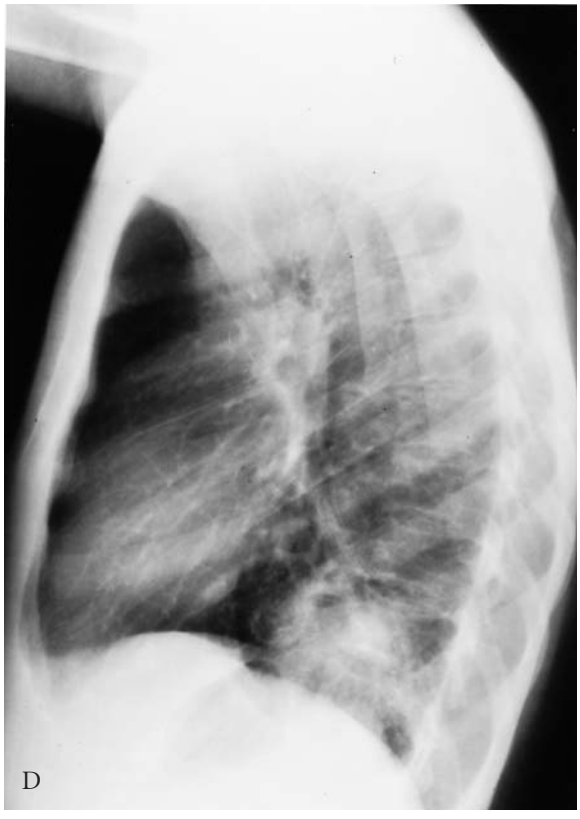


FIGURE **4-49** *Tracheal papillomatosis. Posteroanterior chest radiograph* (A) *and anteroposterior tomogram* (B) *demonstrate diffuse nodularity of the trachea. Computed tomography scans with soft tissue windows* (C) *and lung windows* (D) *demonstrate multiple tracheal wall papillomas that partially obstruct the lumen.*







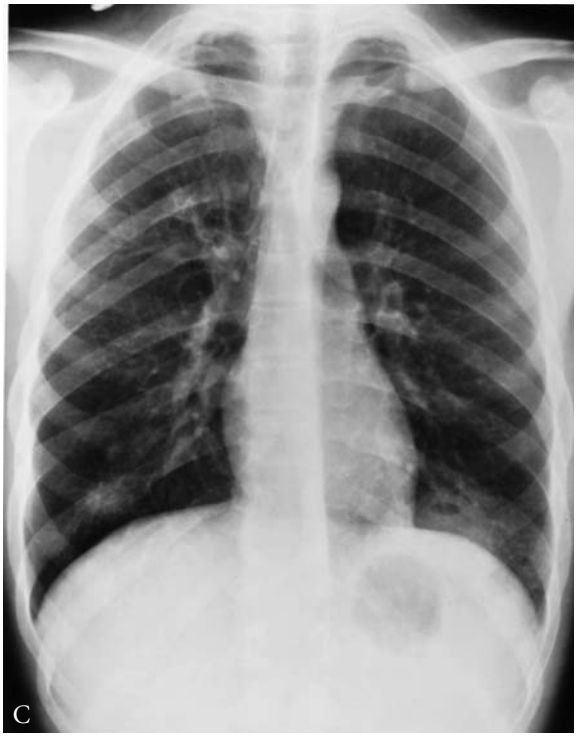


FIGURE **4-50** *Diffuse papillomatosis.* A, *Lateral tomogram of the larynx and cervical region reveals diffuse nodularity throughout the larynx and proximal trachea, with partial obstruction of the lumen.* B, *A chest computed tomography scan reveals small pulmonary nodules and a cavitary nodule in the right lower lobe, consistent with pulmonary papillomatosis. Posteroanterior* (C) *and lateral* (D) *chest radiographs demonstrate bilateral pulmonary nodules, some of which are cavitary.*

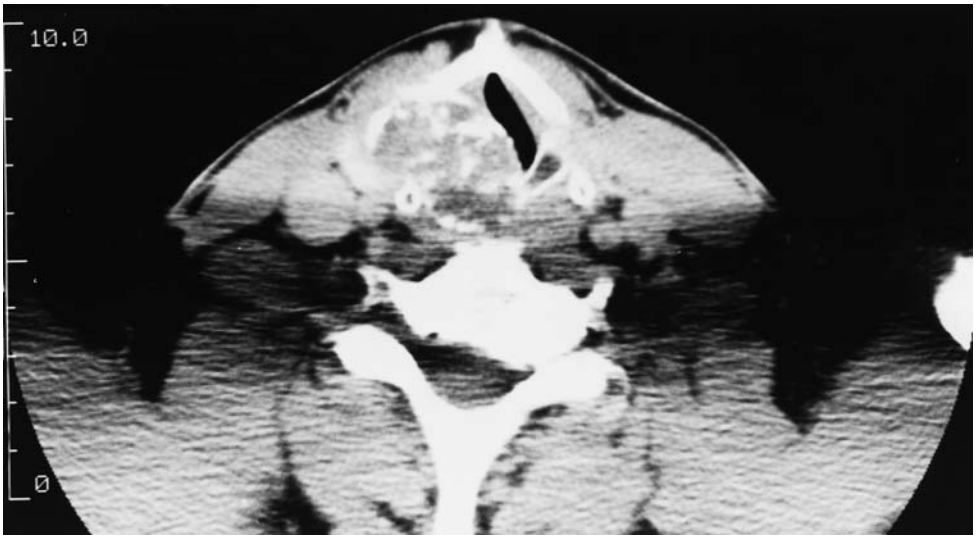
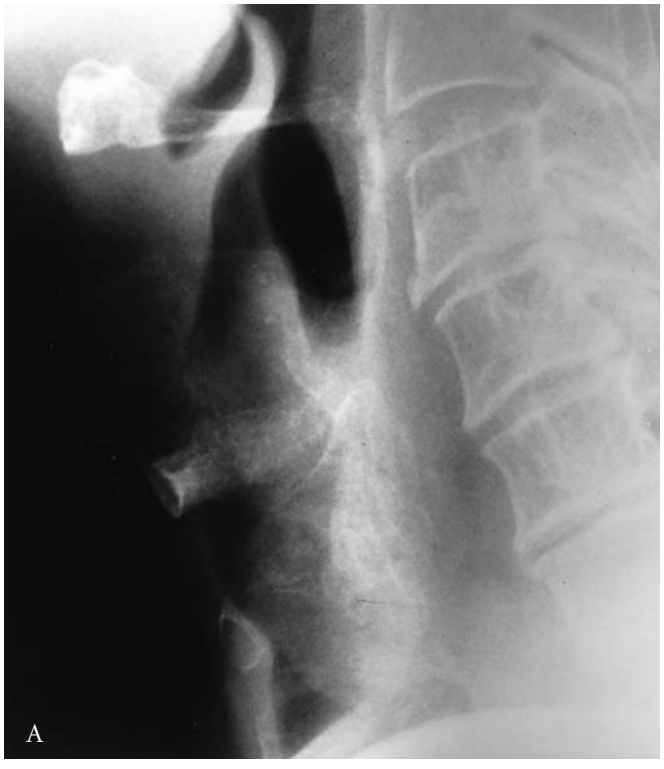
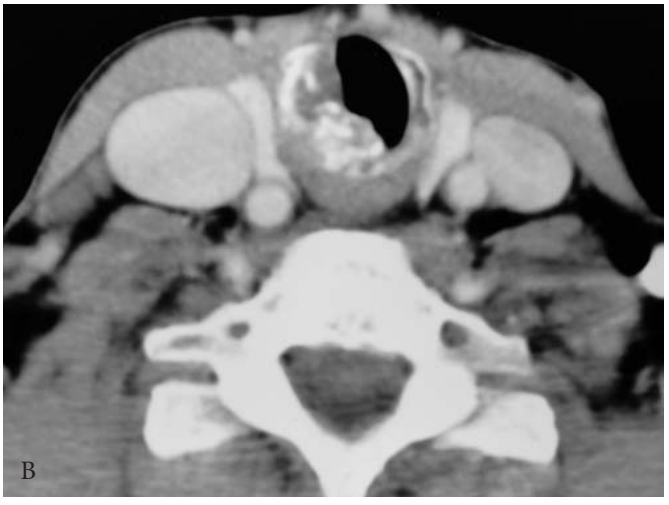


FIGURE **4-51** *Chondroma of the larynx. Axial computed tomography scan shows a calcified mass arising from the right thyroid cartilage and adjacent cricoid cartilage. The tumor bulges into the airspace and displaces the thyroid cartilage laterally.*





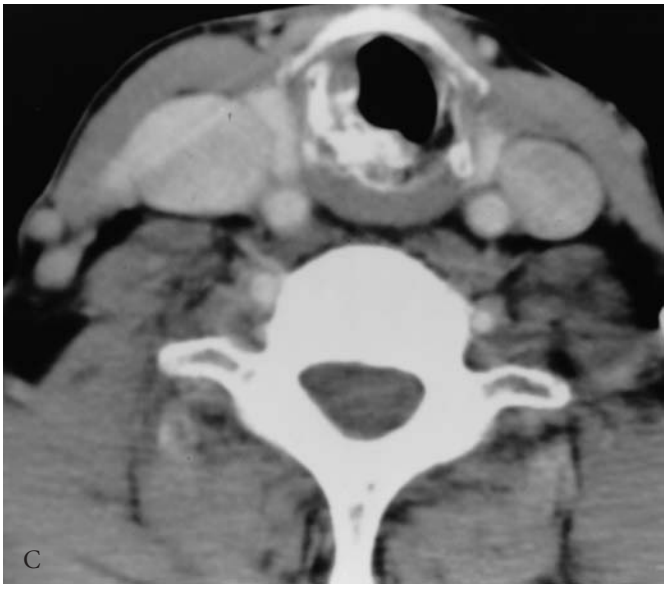


FIGURE **4-52** *Chondroma. Lateral tomogram of the larynx* (A) *and computed tomography scans* (B, C) *through the cricoid cartilage reveal a calcified mass arising from the cricoid cartilage, consistent with a chondroma.*

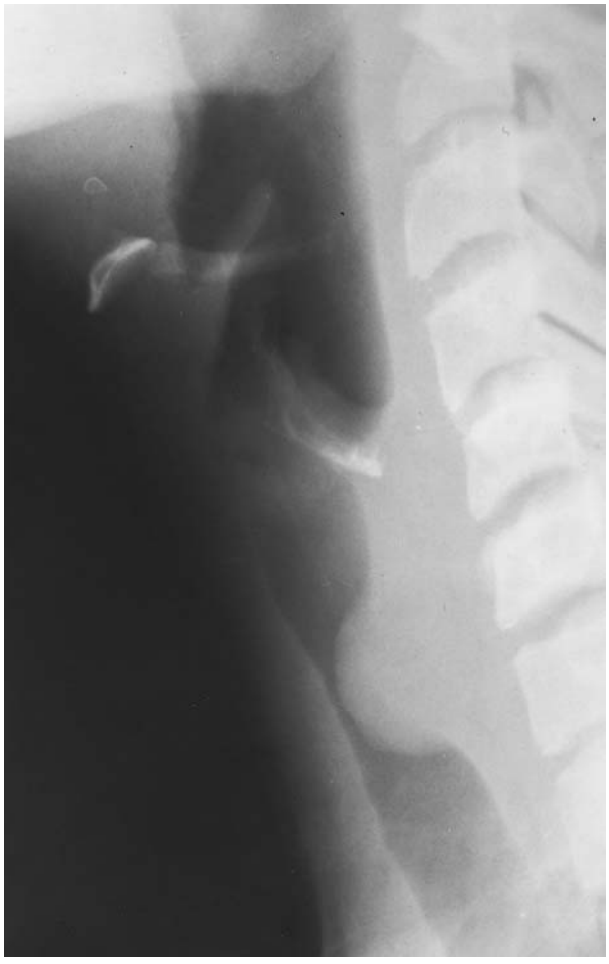
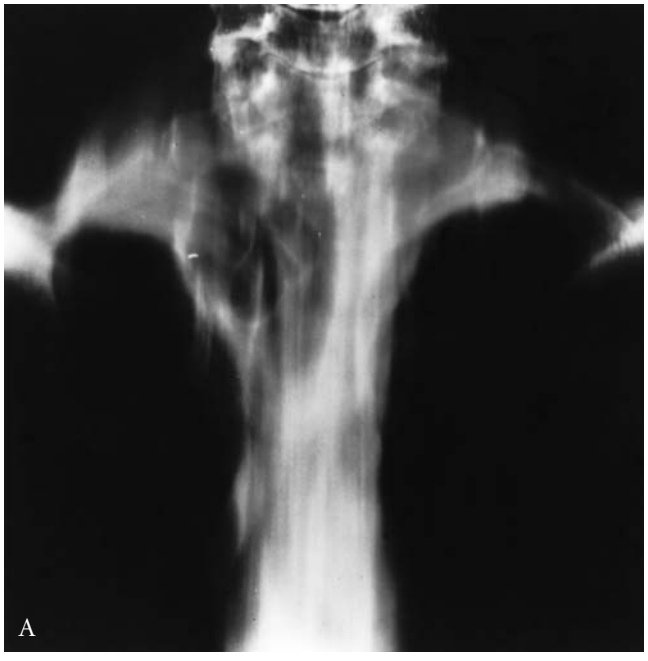


FIGURE **4-53** *Granular cell myoblastoma of the cervical trachea. Lateral view of the neck outlines an oval-shaped, sharply-defined soft tissue mass arising from the posterior cervical tracheal wall.*



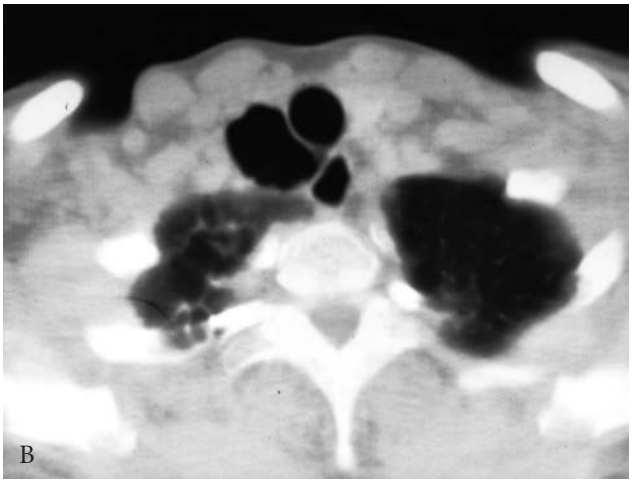


FIGURE **4-54** *Tracheal cyst. Anteroposterior tomogram* (A) *and computed tomography scan of the upper trachea* (B) *demonstrate a thin-walled air-filled right paratracheal cyst.*

Malignant Lesions

Epithelial Tumors

Larynx. The majority of laryngeal malignancies (90%) represent epithelial neoplasms. Among these, 50 to 70% of laryngeal cancers are glottic in nature, 30 to 35% represent supraglottic carcinomas, and 4 to 6% represent subglottic carcinomas.[76,77] Tumor size, location, and histologic grading are important parameters that determine the occurrence of lymph node metastases. Metastatic lymph nodes are more common when the primary tumor in the supraglottic larynx is greater than 2.0 cm in diameter and is poorly differentiated. Supraglottic carcinomas arise from the laryngeal surface and rim of the epiglottis, aryepiglottic folds, arytenoids, false cords, and laryngeal ventricles.[78] They commonly extend across the midline, invade the extralaryngeal structures by direct extension to the pyriform sinuses, extend to the postcricoid region, and potentially extend to the cervical esophagus, valleculae, and base of tongue. Vocal cord cancers arise commonly from the anterior two-thirds of the vocal cords and may spread via the anterior commissure to the subglottic space (20%) and infrequently into the cervical trachea (Figure 4-55).[77] Deep penetration of the cord by tumor into the vocalis muscle causes fixation of the cord.

Carcinomas arising in the cervical trachea may involve by superior extension the subglottic larynx. In larger carcinomas, it is often difficult to determine whether the origin is from the cervical esophagus, the cervical trachea, or an extension of a subglottic carcinoma into the upper trachea. Stomal recurrence postlaryngectomy is encountered in 5 to 15% of cases. The tumor manifests as single or multiple nodules, at or near the stomal margin, involving the skin or tracheal mucosa. Deep invasion is commonly present, associated with ulceration at the skin margin.

CT evaluation provides valuable information concerning extension of malignant tumor to the following areas: 1) the anterior commissure, 2) the paracordal and para-arytenoidal areas, 3) the preepiglottic and subglottic spaces, 4) cartilage invasion (see Figure 4-55), extralaryngeal extension of an endolaryngeal tumor, and 6) extension of pyriform sinus carcinomas through the cricothyroid space to involve the postcricoid region and cervical esophagus.[78] Contrast CT or MRI is used to evaluate the tracheal extension and tumor invasion of the upper mediastinum prior to surgery and/or radiation therapy.[79–81]

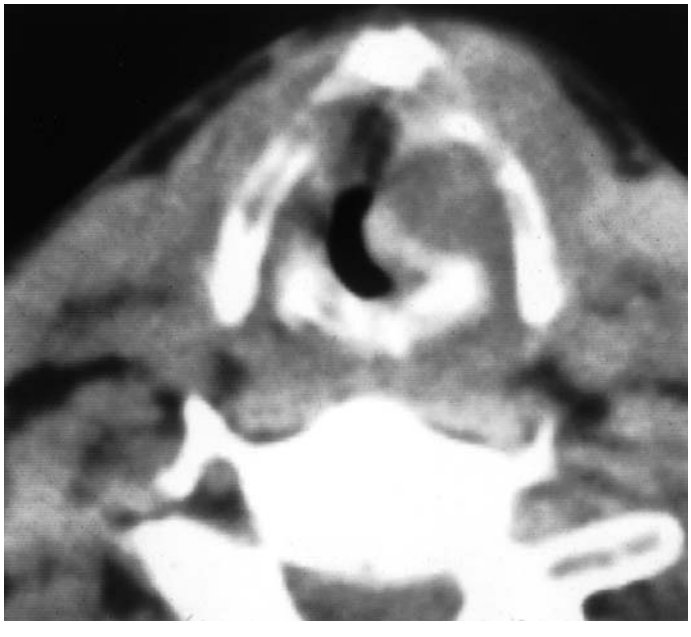


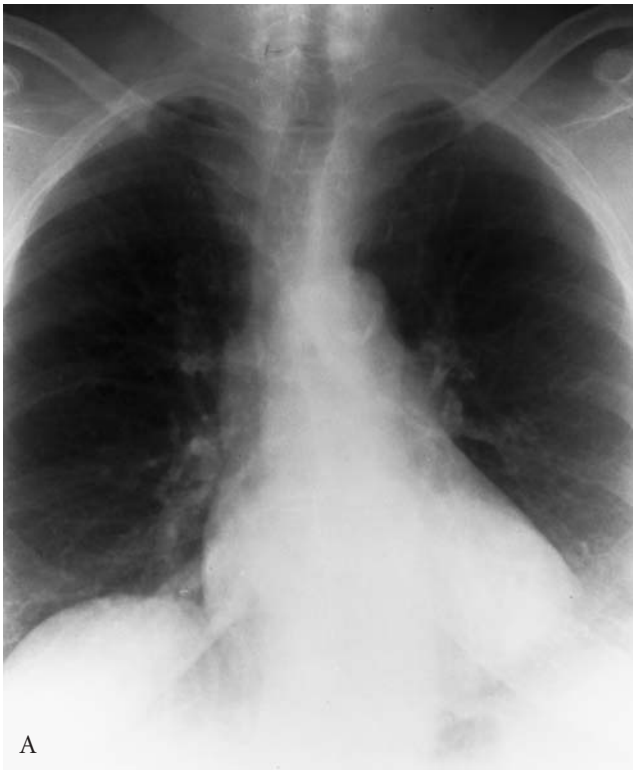
FIGURE **4-55** *Vocal cord carcinoma (left). Axial computed tomography section shows a left vocal cord carcinoma causing cartilaginous erosion and extension into adjacent soft tissue structures.*

Trachea. Although very rare, primary malignancies of the trachea are much more common than benign tumors.[82] Squamous cell carcinoma is the most common tracheal malignancy (50%), followed by adenoid cystic carcinoma (30%) and adenocarcinoma (10%). Other less frequently encountered malignancies include mucoepidermoid carcinoma, undifferentiated carcinoma, small cell carcinoma, and carcinosarcoma.[1,83]

SQUAMOUS CELL CARCINOMA. Squamous cell carcinoma may present as a focal mass with a tendency for exophytic growth and a propensity to invade the mediastinum (Figure 4-56).[84] Synchronous and metachronous squamous cell carcinomas of the larynx, lungs, and esophagus are found in many patients. CT is useful in demonstrating the primary tumor and its extent in the trachea and adjacent mediastinum, as well as associated adenopathy within the mediastinum and hilum.

ADENOID CYSTIC CARCINOMA. Adenoid cystic carcinoma tends to grow with endophytic spread in the submucosal plane of the trachea and bronchi (Figure 4-57).[85,86] On radiographs and CT and MRI scans, the trachea appears thickened with a smooth nodular appearance, associated with luminal narrowing (Figure 4-58). The tumor may extend into the adjacent soft tissues of the neck and mediastinum, depicted on CT or MRI as extension into the adjacent mediastinal fat. Regional lymph nodes in the neck and mediastinum are the first to be involved by metastases. Hematogenous metastases to lung, bones, and liver occur later in the disease progression.

MUCOEPIDERMOID TUMORS. Mucoepidermoid tumors are very uncommon tumors of the trachea, central bronchi, and rarely of the lung.[87] These tumors may be of either high-grade or low-grade malignancy. The radiographic findings are of a focal endoluminal soft tissue mass within a large central airway, without characteristic features to distinguish the mass from other tumors (Figure 4-59).



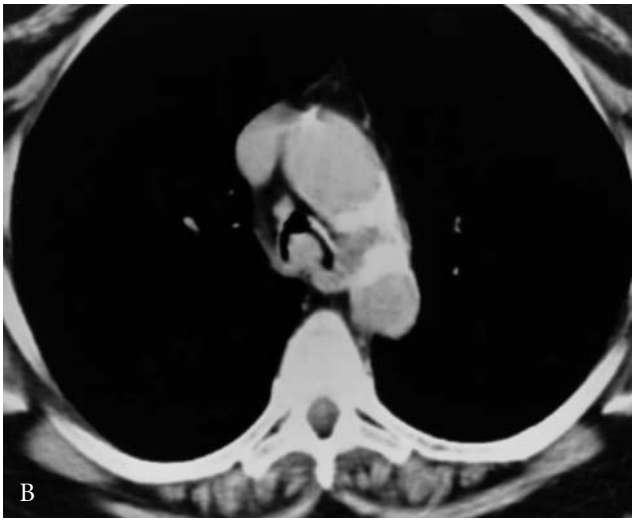


FIGURE **4-56** *Squamous cell carcinoma of the trachea. Posteroanterior chest radiograph* (A) *and computed tomography scan of the distal trachea* (B) *reveal a round soft tissue mass arising from the posterior wall of the trachea.*

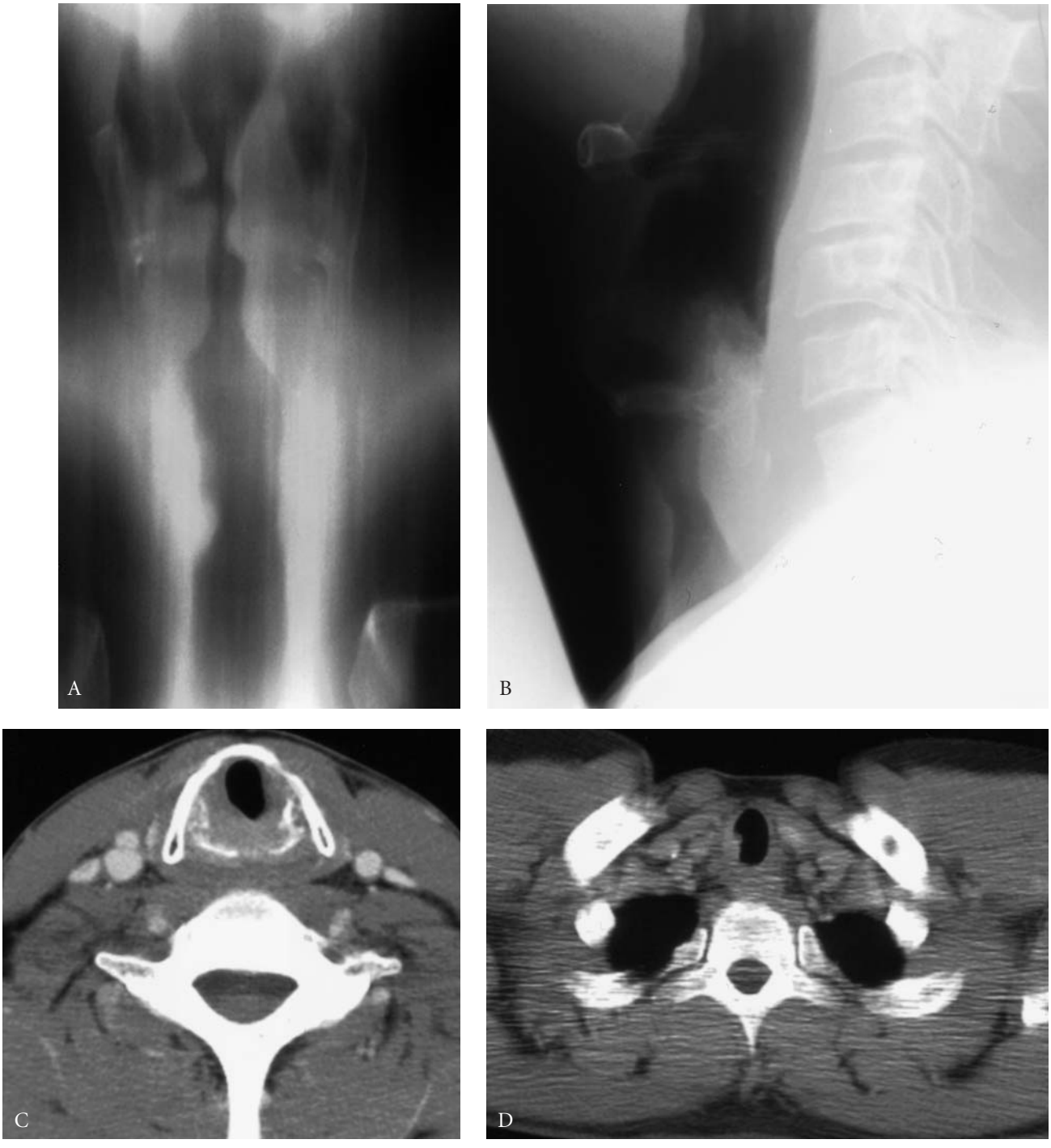


FIGURE **4-57** *Adenoid cystic carcinoma of the larynx and trachea. Anteroposterior* (A) *and lateral* (B) *tomograms of the larynx and proximal trachea and computed tomography scans at the level of the hyoid bone* (C) *and proximal trachea* (D) *reveal a smooth nodular tumor with endophytic growth.*

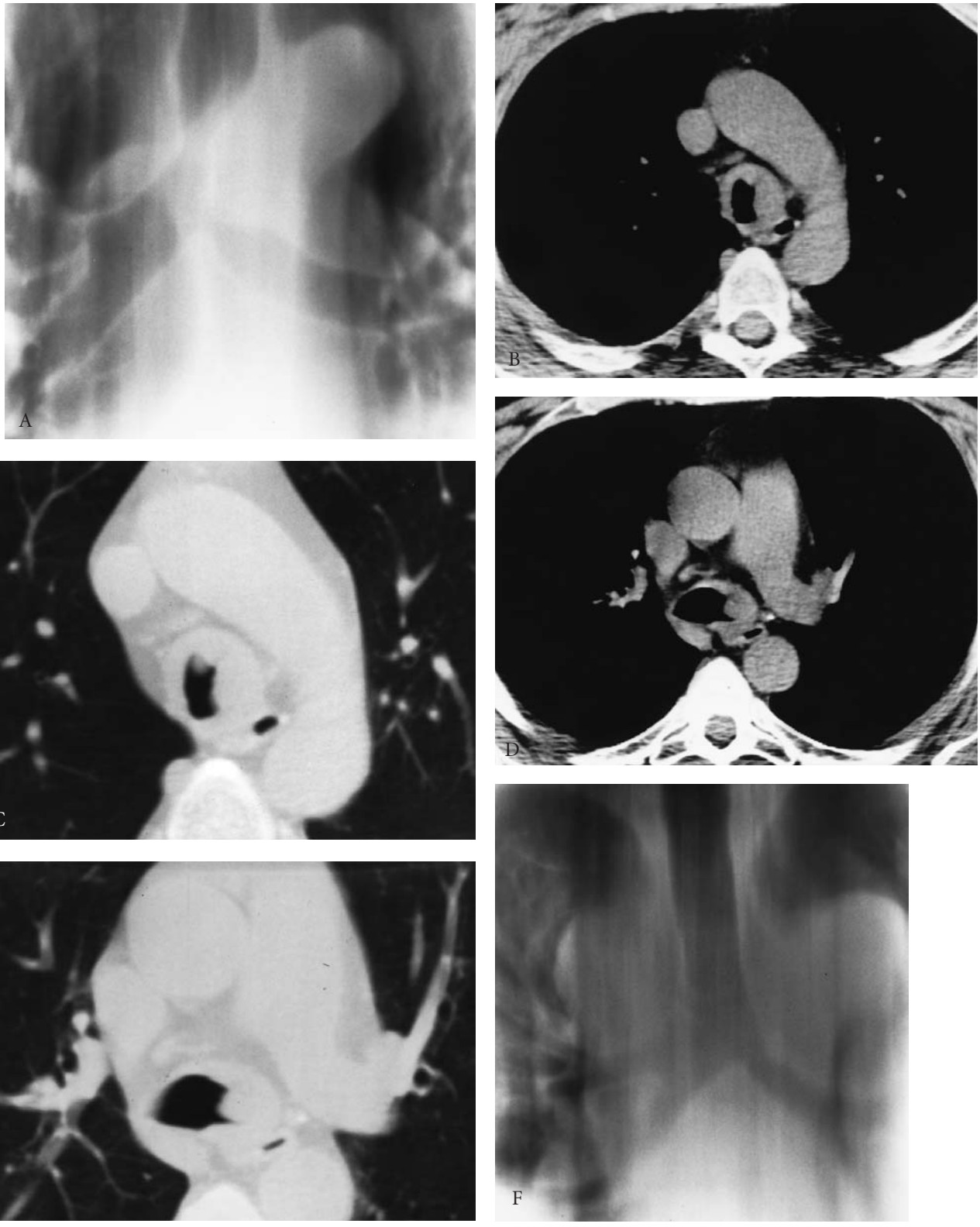


FIGURE **4-58** *Adenoid cystic carcinoma of the distal trachea and proximal left main bronchus.* A, *Anteroposterior tomogram of the trachea and carina reveals a smooth, well-defined mass arising from the left lateral wall of the trachea extending into the proximal left main bronchus. Computed tomography scans of the distal trachea* (B, C) *and proximal left main bronchi* (D, E) *demonstrate nodular thickening of the tracheal and left main bronchial walls.* F, *A postoperative anteroposterior tomogram of the trachea demonstrates a patent anastomosis following a carinal reconstruction.*

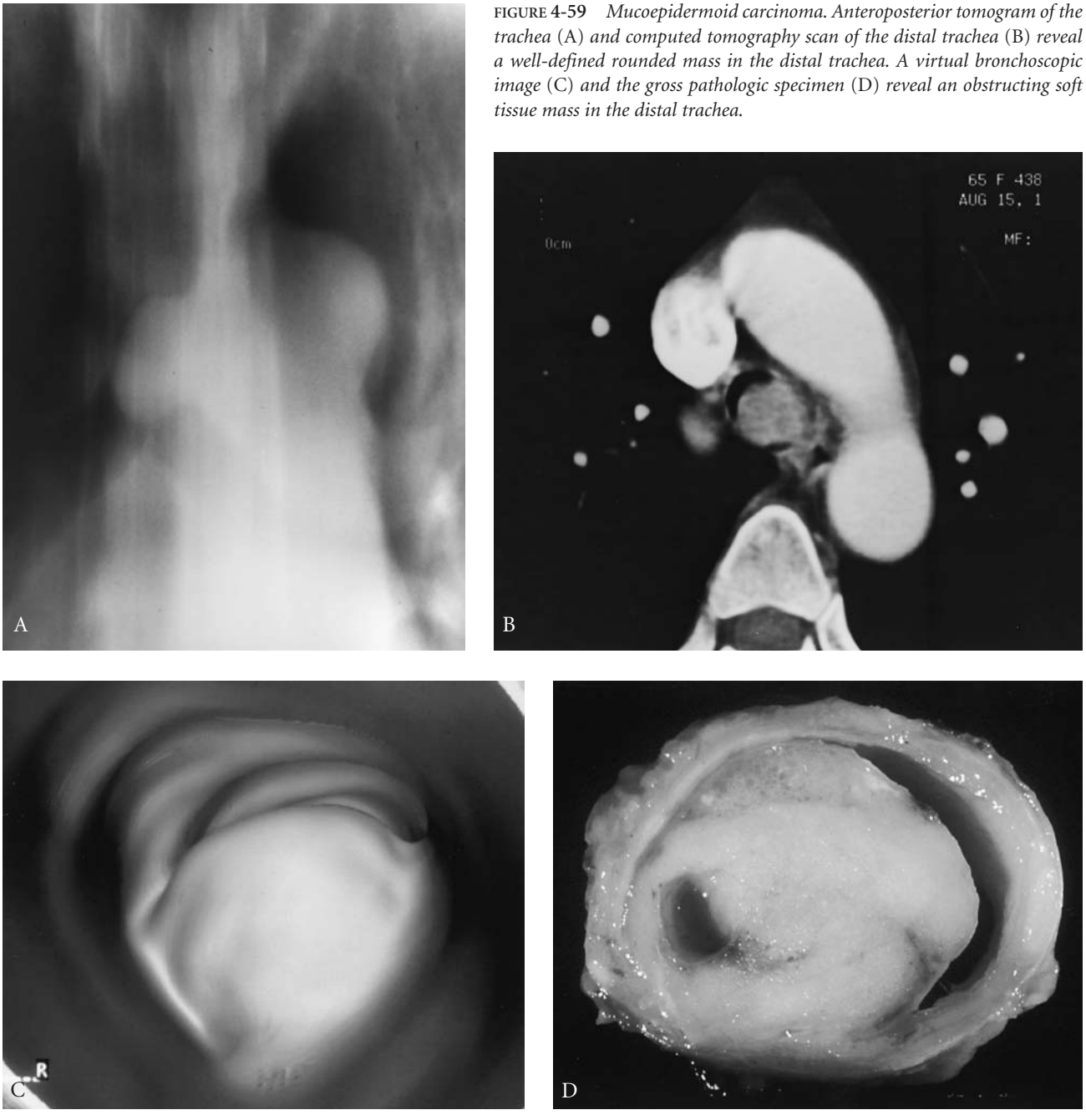


FIGURE **4-59** *Mucoepidermoid carcinoma. Anteroposterior tomogram of the trachea* (A) *and computed tomography scan of the distal trachea* (B) *reveal a well-defined rounded mass in the distal trachea. A virtual bronchoscopic image* (C) *and the gross pathologic specimen* (D) *reveal an obstructing soft tissue mass in the distal trachea.*

CARCINOID TUMORS. Carcinoid tumors are neuroendocrine tumors derived from Kulchitsky's cells.[88] The typical carcinoid tumor represents the lowest grade subtype of a spectrum of tumors that includes the more aggressive atypical carcinoid tumor and the highly malignant small cell carcinoma. *Typical carcinoids* present in the fifth and sixth decades and tend to arise in the central bronchi, peripheral lung (10%), and rarely in the trachea. They tend to be smooth, well-defined, round masses that present as a nodular-filling defect, and may be associated with atelectasis, distal pneumonia, and/or bronchiectasis if they cause bronchial obstruction (Figure 4-60). *Atypical carcinoid tumors* tend to present in the sixth and seventh decades of life, may be either central or peripheral in the lung, and have a tendency to metastasize

to regional hilar and mediastinal lymph nodes (Figure 4-61). *Small cell carcinomas* are extremely malignant tumors that present in the seventh and eighth decades. They are usually associated with large, bulky central hilar and mediastinal lymphadenopathy and distant metastases at the time of diagnosis. CT scans often reveal a small peripheral primary tumor within the lung, generally not visible on routine chest radiographs.

Carcinoid tumors have several distinguishing features on imaging studies. Typical carcinoid tumors generally exhibit slow growth and may contain calcifications. Carcinoid tumors are highly vascular and will demonstrate a high degree of contrast enhancement with iodinated contrast on CT scans (Figure 4-62).

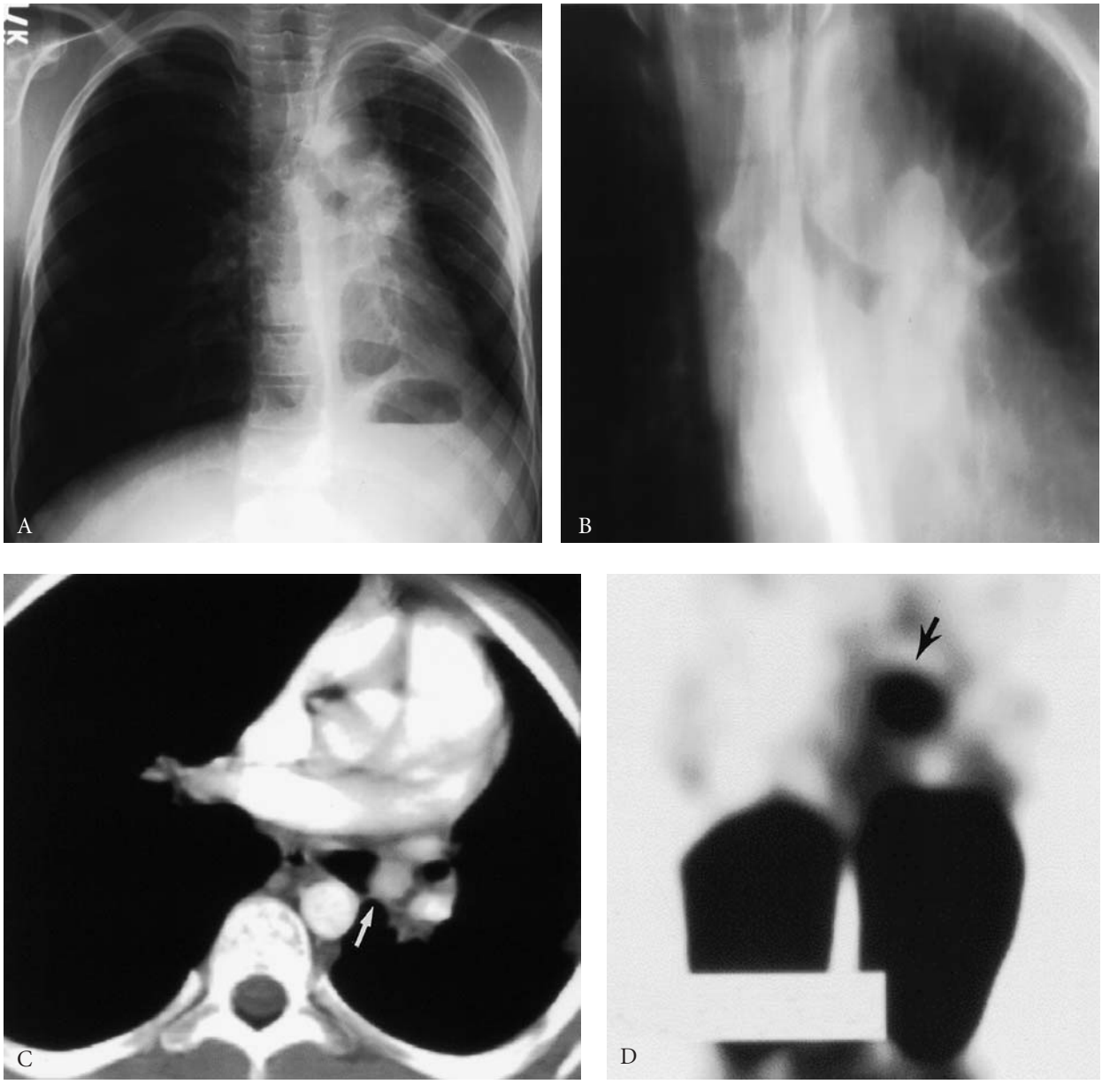


FIGURE **4-60** *Typical carcinoid tumor. Posteroanterior chest radiograph* (A) *and anteroposterior tomogram* (B) *reveal an obstructing mass in the left main bronchus with partial volume loss in the left lung.* C, *A contrast enhanced computed tomography scan demonstrates a focal mass within the left main bronchus* (arrow). D, *An octreotide scan of the chest demonstrates a focal area of intense uptake in the left hilum* (arrow).

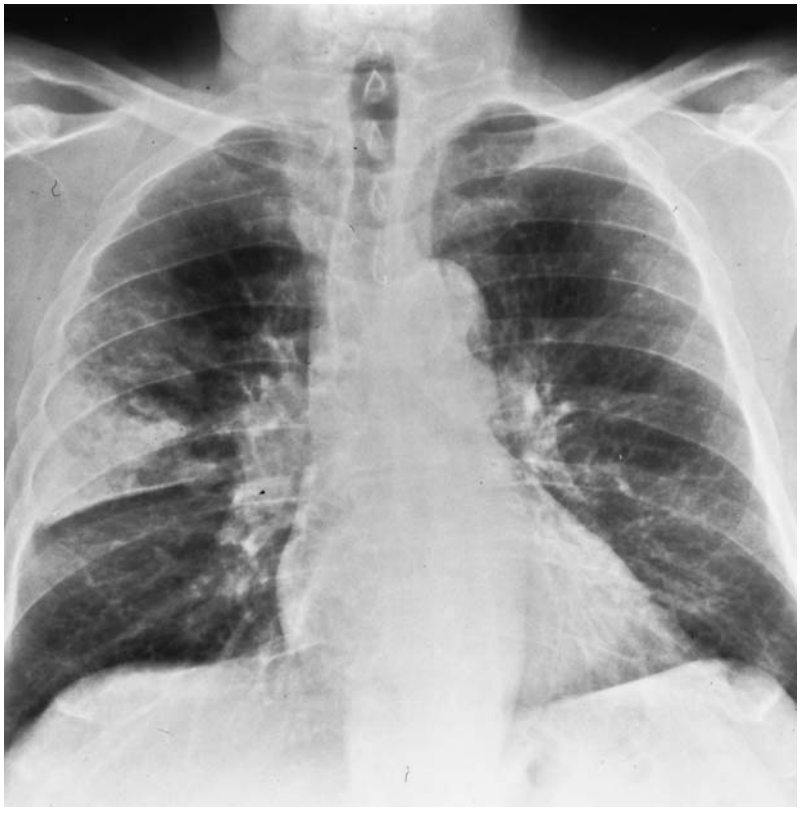


FIGURE **4-61** *Atypical carcinoid tumor. Posteroanterior chest radiograph demonstrates a peripheral mass in the right upper lobe, associated with right hilar adenopathy.*

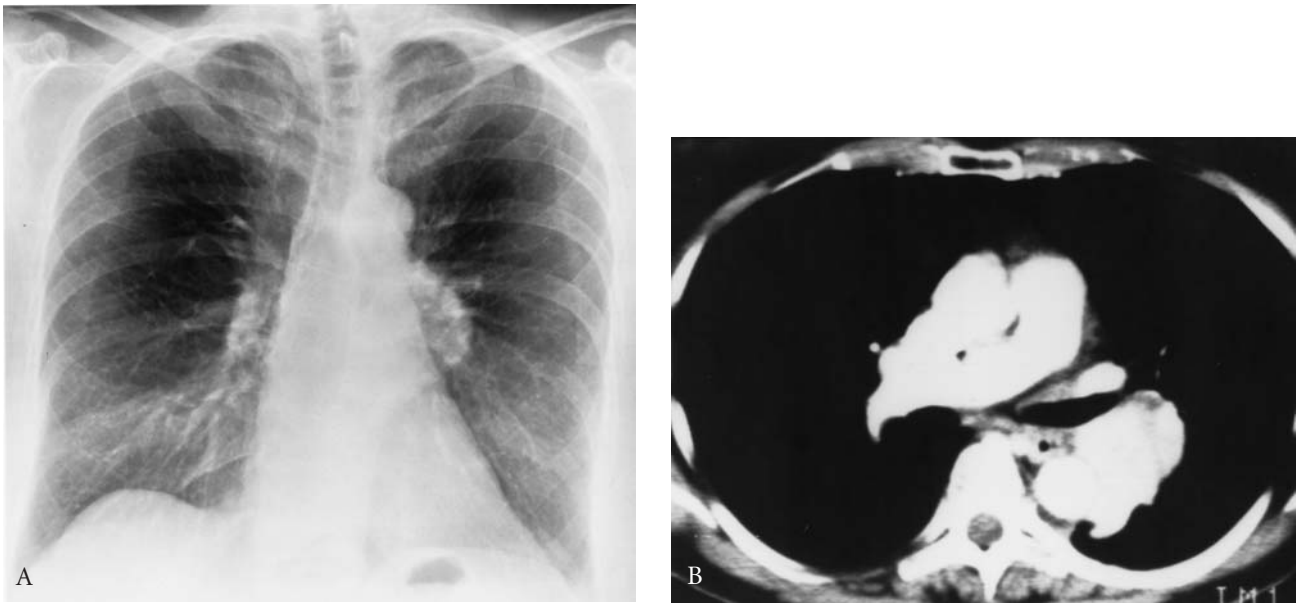


FIGURE **4-62** *Typical carcinoid tumor.* A, *Posteroanterior chest radiograph demonstrates a left hilar mass.* B, *Computed tomography scan with contrast enhancement reveals intense enhancement of the carcinoid tumor in the left lower lobe bronchus.*

Because somatostatin receptors are found in carcinoid tumors, radionuclide-coupled somatostatin analogues such as ^{123}I-Tyr3-octreotide and ^{111}In-octreotide can be used to identify carcinoid tumors. This diagnostic approach is helpful in identifying occult carcinoid tumors in those patients who present with clinical symptoms referable to serotonin, adrenocorticotropic hormone, or bradykinin production.

Mesenchymal Tumors

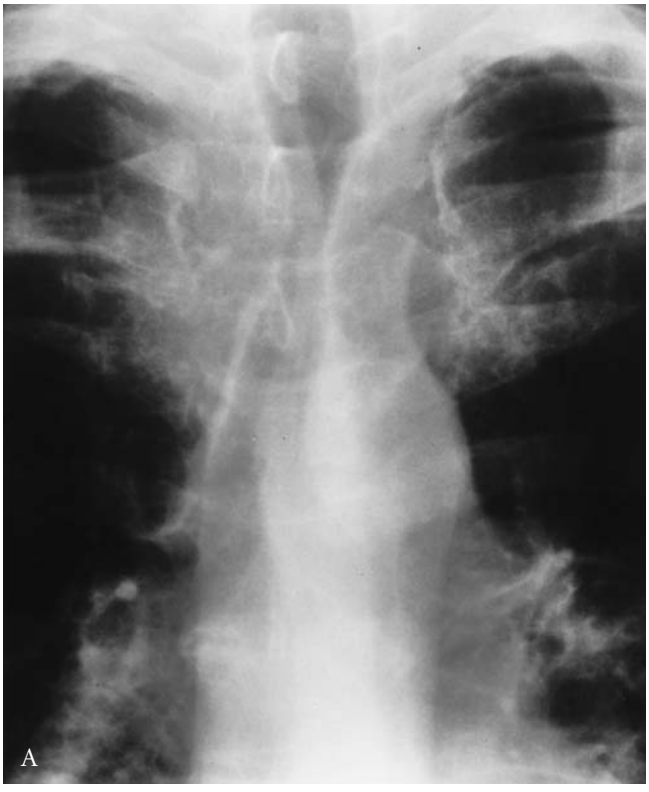
Mesenchymal tumors are rarely reported to occur in the trachea, and tend to occur in young adults. Fibrosarcoma, leiomyosarcoma, chondrosarcoma, hemangioendotheliosarcoma, and lymphomas have been reported (Figure 4-63).[89] Except for calcifications in chondrosarcomas, there are no specific characteristics with which to differentiate mesenchymal tumors from other malignancies (Figure 4-64).

Secondary Malignant Tumors

Carcinomas, especially papillary and follicular types arising from the thyroid gland, may invade the larynx and cervical trachea in up to 5% of cases. The trachea may also be invaded by tumors of the esophagus and lung. The delineation of the extent of these tumors is best accomplished with CT and MRI (Figure 4-65).[79,80]

Tracheobronchomegaly

Tracheobronchomegaly, seen in Mounier-Kuhn syndrome, is an abnormal diffuse dilatation of the trachea and main bronchi.[90–92] The diagnosis of tracheobronchomegaly is usually apparent on chest radiographs, CT, or MRI. Typically, there is protrusion of the mucosa through the trachealis muscle between the cartilaginous rings, producing a scalloped or corrugated appearance of the trachea and main bronchi (Figure 4-66). There is often an abrupt transition between the dilated lobar bronchi and normal segmental bronchi. Patients with this disorder may have repeated respiratory infections leading to peripheral bronchiectasis.



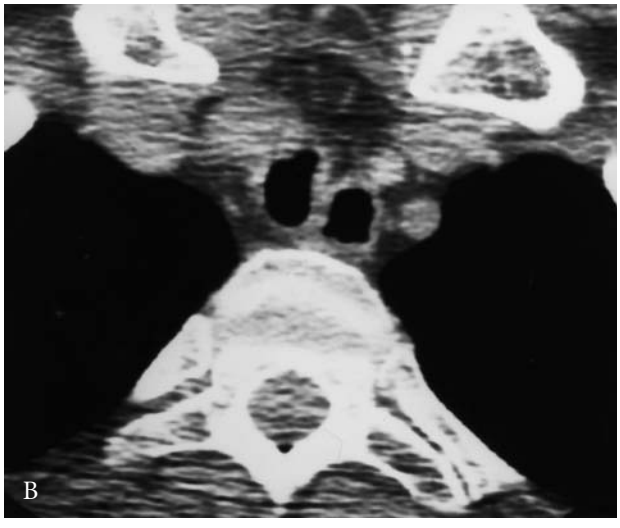


FIGURE **4-63** *Tracheal lymphoma.* A, *Anteroposterior tracheal tomogram reveals a smooth stenosis of the proximal trachea with an hourglass configuration.* B, *Computed tomography scan demonstrates thickening of the tracheal wall.*

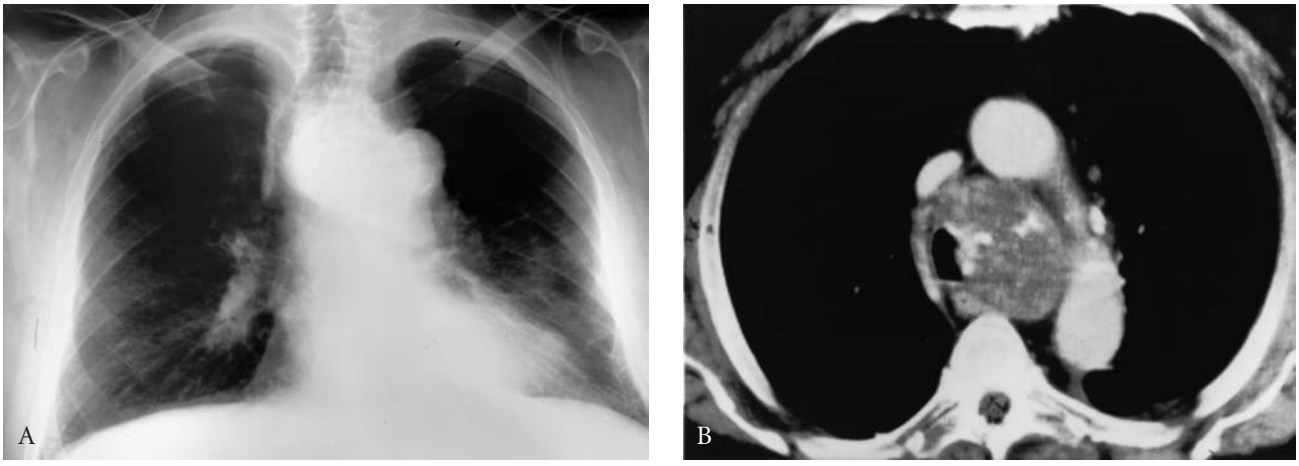


FIGURE **4-64** *Chondrosarcoma of the trachea.* A, *Posteroanterior chest radiograph demonstrates a large mediastinal mass that deviates and partially obstructs the mid and distal trachea.* B, *Computed tomography scan through the midtrachea reveals a large soft tissue mass containing calcification that encircles and narrows the trachea, consistent with a chondrosarcoma arising from the trachea.*

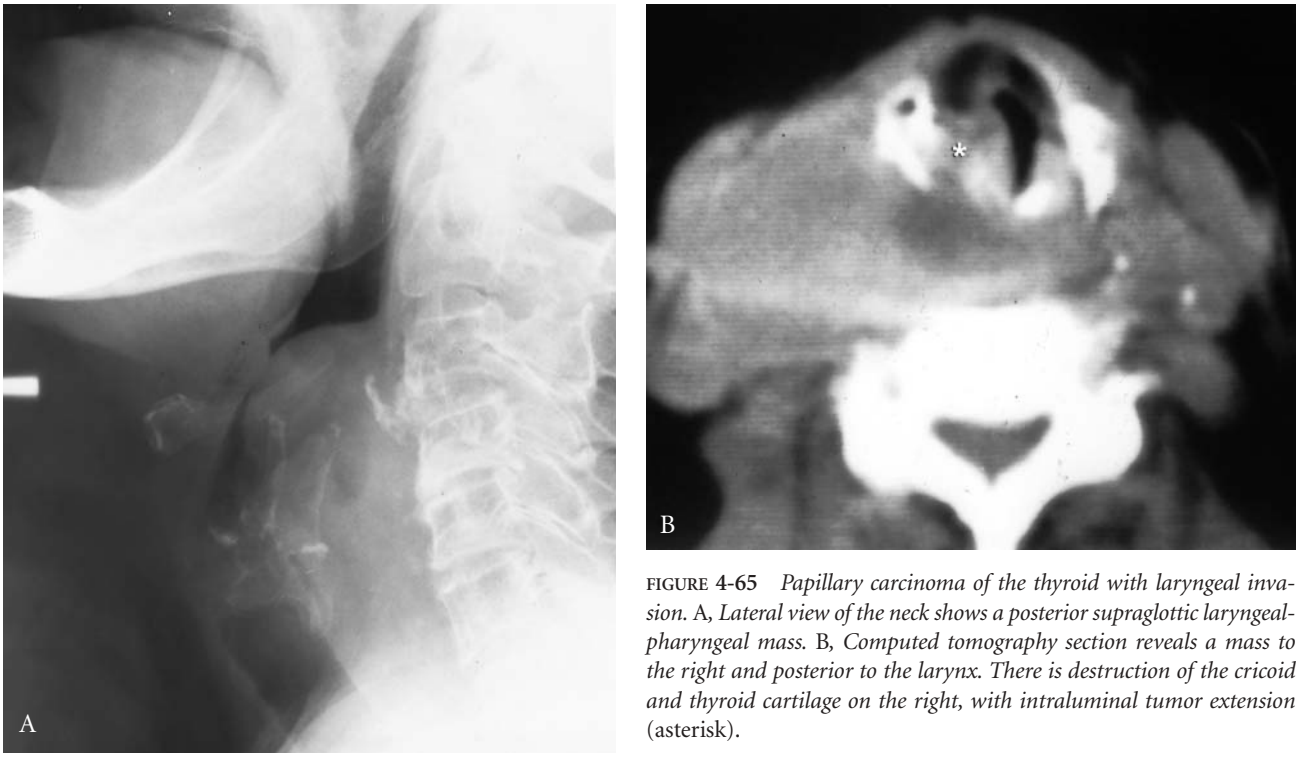


FIGURE **4-65** *Papillary carcinoma of the thyroid with laryngeal invasion.* A, *Lateral view of the neck shows a posterior supraglottic laryngealpharyngeal mass.* B, *Computed tomography section reveals a mass to the right and posterior to the larynx. There is destruction of the cricoid and thyroid cartilage on the right, with intraluminal tumor extension* (asterisk).

Tracheobronchomegaly is also seen in association with Ehlers-Danlos syndrome, cutis laxa, and ataxia telangiectasia (immune deficiency syndrome), and with diffuse pulmonary fibrosis.

Tracheobronchomalacia

Tracheobronchomalacia refers to a weakness in the tracheobronchial walls due to a primary immaturity or secondary softening or destruction of the cartilaginous rings, or excessive flaccidity of the posterior mem-

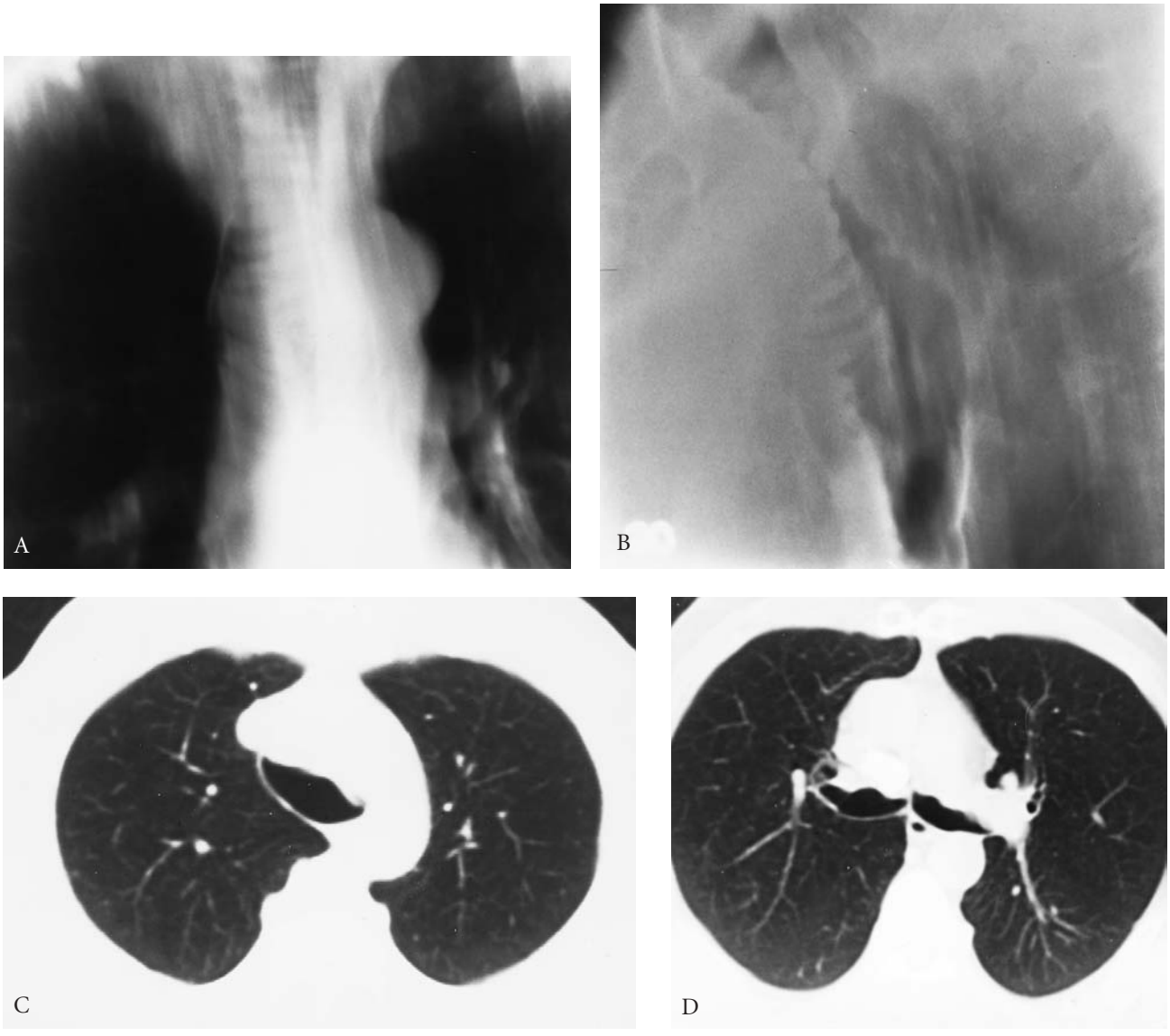


FIGURE **4-66** *Mounier-Kuhn syndrome. Anteroposterior* (A) *and lateral* (B) *tomograms of the trachea demonstrate dilatation of the trachea and main bronchi, with a scalloped or corrugated appearance. Computed tomography scans of the midtrachea* (C) *and main bronchi* (D) *demonstrate dilated scalloped-appearing airways.*

branous wall with resultant collapsibility. Tracheobronchomalacia may be focal or diffuse.[93,94] There are various causes of tracheobronchomalacia, including 1) congenital absence or hypoplasia of the cartilaginous rings, 2) traumatic causes related to mechanical ventilation with cuffed tubes or blunt chest trauma, 3) inflammatory conditions such as relapsing polychondritis, or 4) compression by vascular structures such as aberrant vessels or aneurysms or external mediastinal masses such as long-standing goiters. The location and extent of malacia is best evaluated radiographically by inspiratory/expiratory CT scans (Figure 4-67).

Acquired Tracheobronchoesophageal Fistulae

Tracheobronchoesophageal fistulae may be congenital or acquired.[95,96] The most common congenital tracheoesophageal fistula, accounting for 80 to 90% of cases, is esophageal atresia with a low tracheoesophageal

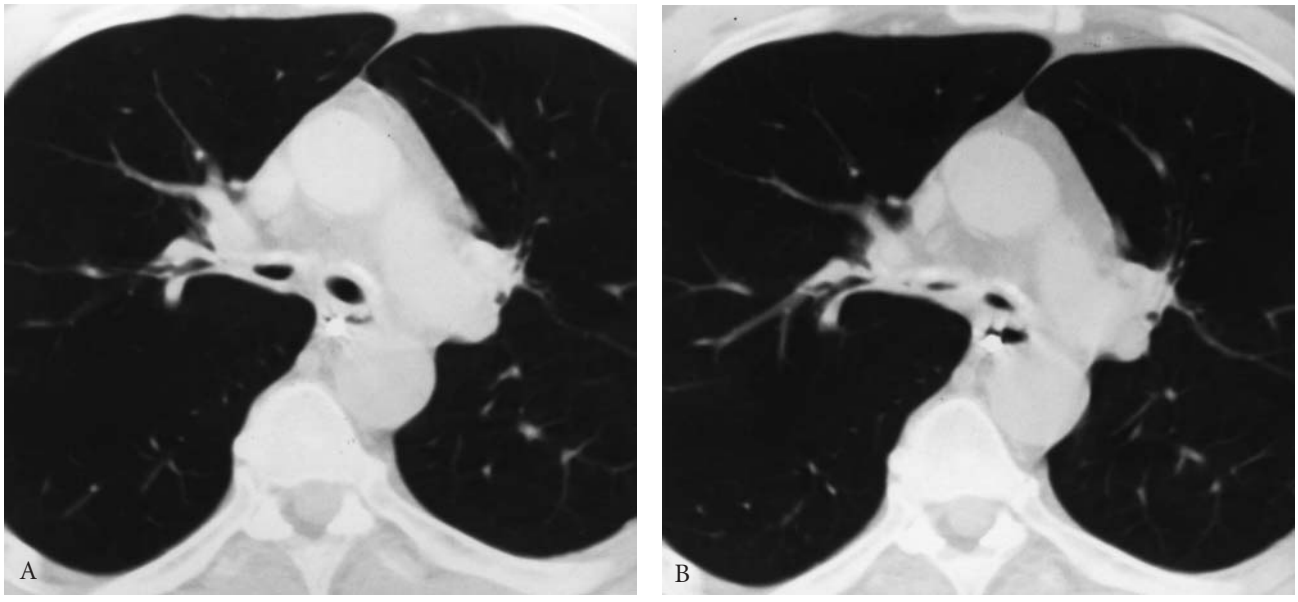


FIGURE **4-67** *Tracheobronchomalacia. Inspiratory* (A) *and expiratory* (B) *computed tomography scans of the main bronchi reveal marked collapse of the bronchi on expiration. There is also diffuse thickening and calcification of the airways, typical of relapsing polychondritis.*

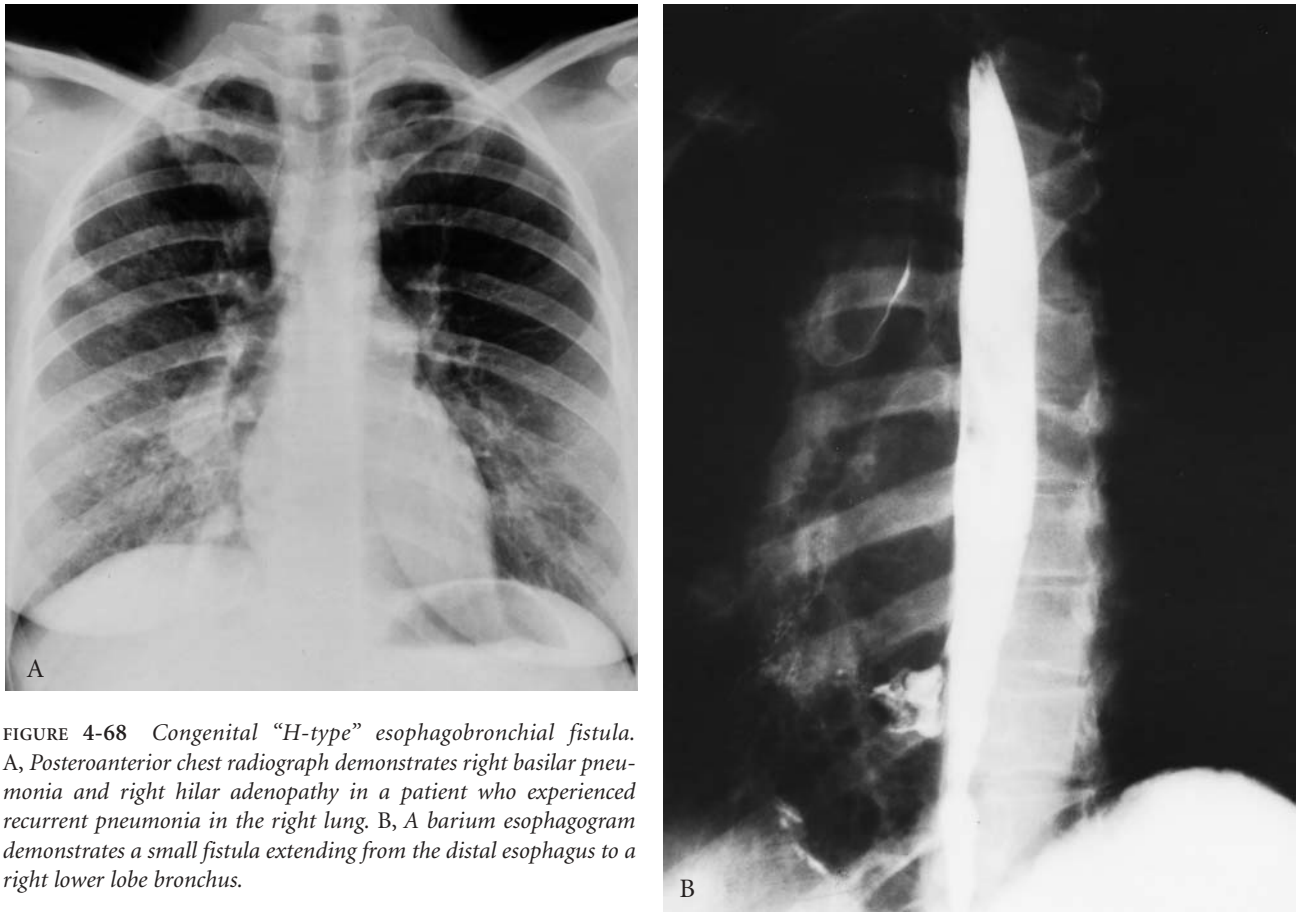
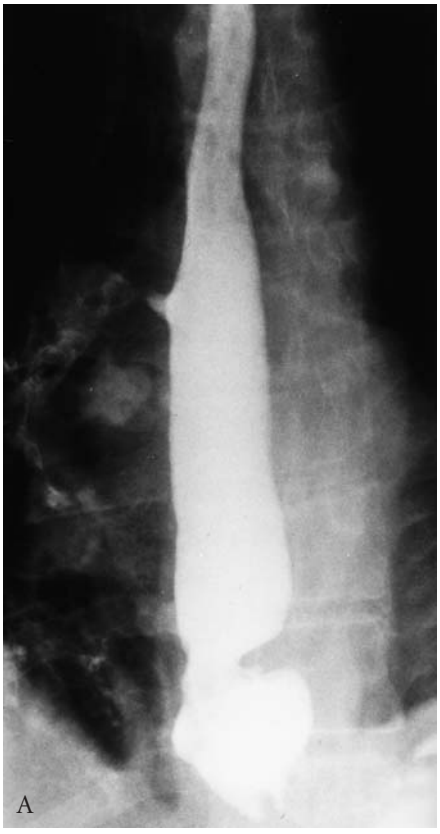


FIGURE **4-68** *Congenital "H-type" esophagobronchial fistula.* A, *Posteroanterior chest radiograph demonstrates right basilar pneumonia and right hilar adenopathy in a patient who experienced recurrent pneumonia in the right lung.* B, *A barium esophagogram demonstrates a small fistula extending from the distal esophagus to a right lower lobe bronchus.*



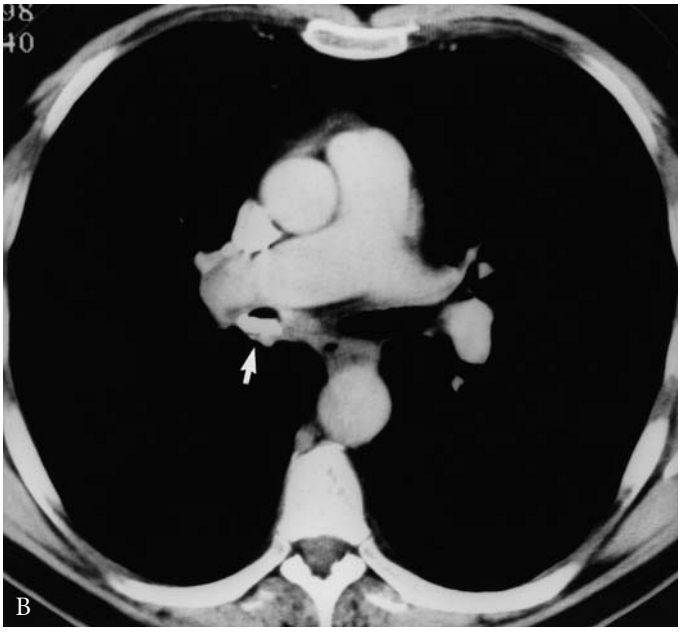


FIGURE **4-69** *Esophagobronchial fistula and broncholith due to histoplasmosis. An esophagogram* (A) *reveals an esophagobronchial fistula extending from the distal esophagus to the bronchus intermedius, and as demonstrated by a chest computed tomography scan* (B)*, this patient had a broncholith* (arrow) *in the bronchus intermedius.*

fistula, presenting at birth. The "H-fistula" without atresia may be difficult to diagnose and may go undetected until adulthood (Figure 4-68). The majority of tracheobronchoesophageal fistulae in adults are acquired. Such acquired fistulae result from several causes, including 1) malignancy of the esophagus, trachea, bronchi, thyroid, and lymphomas; 2) radiation injury; 3) fungal infections such as histoplasmosis and actinomycosis, tuberculosis, syphilis, and bacterial infections (Figure 4-69); and 4) traumatic causes such as those from mechanical ventilation with cuffed tubes and indwelling nasogastric tubes, blunt or penetrating trauma, lye burns of the esophagus, instrumentation, or esophageal foreign bodies (Figure 4-70). The diagnosis of a fistula can be established with the judicious use of an esophagogram, using low osmolar contrast material. High osmolar contrast should be avoided, because if the lung becomes flooded with contrast, life-threatening pulmonary edema and bronchospasm may develop.

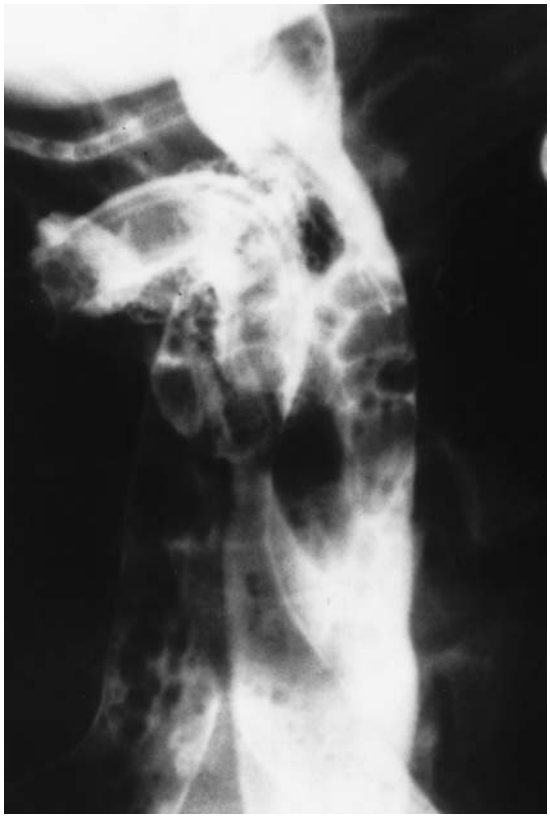


FIGURE **4-70** *Postintubation tracheoesophageal fistula. Anteroposterior view of an esophagogram reveals a fistula of the proximal esophagus extending to the trachea in a patient with a tracheostomy tube.*

References

1. Weber AL, Grillo HC. Tracheal tumors: a radiological, clinical, and pathological evaluation of 84 cases. In: Weber AL, editor. Symposium on the larynx and trachea. Radiol Clin North Am 1978;16:227–46.
2. Fried MP, Meller SM. Adult laryngeal anatomy. In: Fried MP, editor. The larynx: a multidisciplinary approach. 2nd ed. St. Louis: CV Mosby; 1996. p. 33–43.
3. Castelijns JA, Doornbos J, Verbeeten BJ, et al. MR imaging of the normal larynx. J Comp Tomogr 1985; 9:919–25.
4. Castelijns JA, van den Brekel MW, Niekoop VA, Snow GB. Imaging of the larynx. Neuroimaging Clin North Am 1996;6:401–15.
5. Curtin HD. Larynx. In: Som PM, Curtin HD, editors. Head and neck imaging. St. Louis: CV Mosby; 1996. p. 612–707.
6. Momose KJ, Macmillan AS Jr. Roentgenologic investigations of the larynx and trachea. Radiol Clin North Am 1978;16:321–41.
7. Proctor DF. State of the art: the upper airway. II. The larynx and trachea. Am Rev Respir Dis 1977;115:315–42.
8. Effman EL, Fram BK, Yock P, et al. Tracheal cross-sectional area in children: CT determination. Radiology 1983; 149:137–40.
9. Gamsu G, Webb WR. Computed tomography of the trachea: normal and abnormal. Am J Roentgenol 1982;139:321–6.
10. Kormano M, Yrjana J. The posterior tracheal band: correlation between computed tomography and chest radiography. Radiology 1980;136:689–94.
11. Savoca MCJ, Austin JHM, Goldberg HI. The right paratracheal stripe. Radiology 1977;122:295–301.
12. Weber AL. Radiology of the larynx. Otolaryngol Clin North Am 1984;17:13–28.
13. Takashima S, Takayama F, Wang Q, et al. Radiologic evaluation of the trachea. Chest Surg Clin North Am 1996;6:637–73.
14. Maguire GH. The larynx: simplified radiological examination using heavy filtration and high voltage radiography. Radiology 1966;87:102–9.
15. Boiselle PM, Ernst A. Recent advances in central airway imaging. Chest 2002;121:1651–60.
16. Greene RME, Shepard JO, Lechner G. Radiology of the trachea. In: Traveras JM, Ferrucci JT, editors. Radiology: diagnosis/imaging/intervention. Vol 1. Philadelphia: JB Lippincott Company; 2000.
17. Holbert JM, Strollo DC. Imaging of the normal trachea. J Thorac Imaging 1995;10:171–9.
18. Hu H, He HD, Folley WD, Fox SH. Four multidetector-row helical CT: image quality and volume coverage speed. Radiology 2000;215:55–62.
19. Sakai F, Gamsu G, Dillon WP, et al. MR imaging of the larynx at 1.5 T. J Comput Assist Tomogr 1990; 14:60–71.
20. Becker M. Larynx and hypopharynx. Radiol Clin North Am 1998;36:891–920.
21. Landing BH. Congenital malformation and genetic disorders of the respiratory tract (larynx, trachea, bronchi and lungs). Am Rev Respir Dis 1979;120:151–85.
22. Siegel MJ, Shakelford GD, Francis RS, et al. Tracheal bronchus. Radiology 1979;130:353–5.

23. Benjamin B, Pitkin J, Cohen D. Congenital tracheal stenosis. Ann Otol Rhinol Laryngol 1981;90:364–71.
24. Berdon WE, Baker DH. Vascular anomalies in the infant lung: rings, slings and other things. Semin Roentgenol 1972;7:39–64.
25. Park CD, Waldhausen JA, Friedman S, et al. Tracheal compression by the great arteries in the mediastinum: report of 39 cases. Arch Surg 1971;103:626.
26. Strife JL, Baumel AS, Dunbar JS. Tracheal compression by the innominate artery in infancy and childhood. Radiology 1981;139:73.
27. Gumbiner CH, Mullins CE, McNamara DG. Pulmonary artery sling. Am J Cardiol 1980;45:311.
28. Shepard JO, Grillo HC, McLoud TC, et al. Right pneumonectomy syndrome: radiologic findings and CT correlation. Radiology 1986;161:661–4.
29. Grillo HC, Shepard JO, Mathisen DJ, Kanarek DJ. Post pneumonectomy syndrome: diagnosis, management, and result. Ann Thorac Surg 1992;54:638–51.
30. Peralta R, Hurford WE. Airway trauma. Int Anesthesiol Clin 2000;38:111–27.
31. Montgomery WW, Cheney ML. Laryngeal trauma. In: Fried MP, editor. The larynx: a multidisciplinary approach. 2nd ed. St. Louis: CV Mosby; 1996. p. 387–94.
32. Mancuso TA, Hanafee WN. Computed tomography of the injured larynx. Radiology 1979;133:139–44.
33. Unger JM, Schuchman GG, Grossman JE, Pellett JR. Tears of the trachea and main bronchi caused by blunt trauma: radiologic findings. Am J Roentgenol 1989;153:1175–80.
34. Dee PM. The radiology of chest trauma [review]. Radiol Clin North Am 1992;30:291–306.
35. Groskin SA. Selected topics in chest trauma [review]. Radiology 1992;183:605–17.
36. Mirvis SE, Templeton P. Imaging in acute thoracic trauma [review]. Semin Roentgenol 1992;27:184–210.
37. Ohlms SA. Subglottic stenosis. In: Fried MP, editor. The larynx: a multidisciplinary approach. 2nd ed. St. Louis: CV Mosby; 1996. p. 165–9.
38. Montgomery WW. The surgical management of supraglottic and subglottic stenosis. Ann Otol Rhinol Laryngol 1968;77:534–46.
39. Mathisen DJ. Surgery of the trachea. Curr Prob Surg 1998;35:453–42.
40. Cooper JD, Grillo HC. The evolution of tracheal injury due to ventilatory assistance through cuffed tube. A pathologic study. Ann Surg 1969;169:334.
41. Wood DE, Mathisen D. Late complications of tracheotomy. Clin Chest Med 1991;52:759–65.
42. Mathisen DG, Grillo NC, Wain JC, Hilgenberg AD. Management of acquired nonmalignant tracheoesophageal fistula. Ann Thorac Surg 1991;52:759–65.
43. Greene R, Lechner G. Saber-sheath trachea: a clinical and functional study of marked coronal narrowing of the intrathoracic trachea. Radiology 1975;115:265–8.
44. Greene R. Saber-sheath trachea: relation to chronic obstructive pulmonary disease. Am J Roentgenol 1978;130:441–5.
45. Bailey CM, Ninde-Taylor PC. Tuberculous laryngitis: a series of 37 patients. Laryngoscope 1981;91:93–100.
46. McAdams HP, Erasmus J, Winter JA. Radiologic manifestations of pulmonary tuberculosis. Radiol Clin North Am 1995;33:655–78.
47. Farmer DW, Moore E, Amparo E, et al. Calcific fibrosing mediastinitis: demonstration of pulmonary vascular obstruction by magnetic resonance imaging. Am J Roentgenol 1984;143:1189.
48. Weinstein JB, Aronberg DJ, Sagel SS. CT of fibrosing mediastinitis: findings and their utility. Am J Roentgenol 1983;141:247–51.
49. Lenique F, Brauner MW, Grenier P, et al. CT assessment of bronchi in sarcoidosis: endoscopic and pathologic correlations. Radiology 1995;194:419–32.
50. Feldman F, Seaman WB, Baker DC. The roentgen manifestations of scleroma. Am J Roentgenol 1967; 101:807–813.
51. Stern MG, Gamsu G, Webb WR, Stulbarg MS. Computed tomography of diffuse tracheal stenosis in Wegener granulomatosis. J Comput Assist Tomogr 1986; 10:868–70.
52. Bhalla M, Grillo HC, McLoud TC, et al. Idiopathic laryngotracheal stenosis: radiographic findings. Am J Roentgenol 1993;1661:515–7.
53. Mendelson DS, Som PM, Crane R, et al. Relapsing polychondritis studied by computed tomography. Radiology 1985;157:489–90.
54. Im J-G, Chung JW, Han SK, et al. ST manifestations of tracheobronchial involvement in relapsing polychondritis. J Comput Assist Tomogr 1988;12:792–3.
55. Onitsuka H, Hirose N, Watanake K. Computed tomography of tracheopathia osteoplastica. Am J Roentgenol 1983;140:268–70.
56. Urban BA, Fishman EK, Goldman SM, et al. CT evaluation of amyloidosis: spectrum of disease. Radiographics 1991;13:1295–308.
57. McCarthy MJ, Rosado-de-Christenson ML. Tumors of the trachea. J Thorac Imaging 1995;10:180–98.
58. Caldarola VT, Harrison EG Jr, Clagett OT, et al. Benign tumors and tumorlike conditions of the trachea and bronchi. Ann Otol 1974;73:1042–61.
59. Jones SR, Mers EN, Barnes L. Benign neoplasms of the larynx. Otolaryngol Clin North Am 1984;17:151–78.
60. Aspestrand F, Kolbenstvedt A, Boysen M. CT findings in benign expansions of the larynx. J Comput Assist Tomogr 1989;13:222–5.
61. Aywlard TD, Glege JB Jr. Primary papilloma of the trachea. Ann Thorac Surg 1973;16:620–3.
62. Hyams VJ, Rabuzzi DD. Cartilaginous tumors of the larynx. Laryngoscope 1970;80:755–67.
63. Sutton TJ, Nogrady MB. Radiologic diagnosis of subglottic hemangioma in infants. Pediatr Radiol 1973; 1:211–6.
64. Cummings CW, Montgomery WW, Balogh K Jr. Neurogenic tumors of the larynx. Ann Otol Rhinol Laryngol 1969;78:76–95.
65. Cotelingam JD, Barne L, Nixon VB. Pleomorphic adenoma of the epiglottis. Arch Otolaryngol 1977; 103:245–7.
66. Som PM, Nagel BD, Feuerstein SS, Strauss L. Benign pleomorphic adenoma of the larynx. Ann Otol Rhinol Laryngol 1979;88:111–2.
67. Oliveira CA, Roth JA, Adams GL. Oncolytic lesions of the larynx. Laryngoscope 1977;87:1718–25.
68. Agarwal RK, Blitzer A, Perzin KH. Granular cell tumors of the larynx. Otolaryngol Head Neck Surg 1979;87:807–14.
69. Schaefer SD, Blend BL, Denton JC. Laryngeal paragangliomas: evaluation and treatment. Am J Otolaryngol 1980;1:451–5.
70. Pahor AL. Lipoma of the larynx. Ear Nose Throat J 1976;5:341–2.
71. Johnson JT, Pouschter DL. Fibrous histiocytoma of the subglottic larynx. Ann Otol Rhinol Laryngol 1977; 86:243–6.
72. Modlin B. Rhabdomyoma of the larynx. Laryngoscope 1982;92:580–2.
73. Glazer HS, Mauro MA, Aronberg DJ, et al. Computed tomography of laryngoceles. Am J Roentgenol 1998;140:549–52.
74. Silverman PM, Korobkin M. Computed tomographic evaluation of laryngoceles. Radiology 1982;145:104.

75. Tanaka H, Mori Y, Kurokawa K, et al. Paratracheal air cysts communicating with the trachea: CT findings. J Thorac Imaging 1997;12:38–40.
76. Sessions DG, Ogura JH, Fried MP. Carcinoma of the subglottic area. Laryngoscope 1975;85:1417.
77. Pilch BZ, Dorfman DM, Brodsky GI, et al. Pathology of laryngeal malignancies. In: Fried MP, editor. The larynx: a multidisciplinary approach. 2nd ed. St. Louis: CV Mosby; 1996.
78. Becker M, Moulin G, Kurt AM, et al. Non-squamous cell neoplasms of the larynx: radiologic-pathologic correlation. Radiographics 1998;18:1189–209.
79. Takashima S, Takayama F, Wang Q, et al. Differentiated thyroid carcinomas. Prediction of tumor invasion with MR imaging. Acta Radiol 2000;41:377–83.
80. Wang JC, Takashima S, Takayama F, et al. Tracheal invasion by thyroid carcinoma: prediction using MR imaging. Am Roentgenol 2001;177:929–36.
81. Grillo HC, Suen HC, Mathisen DJ, Wain JC. Resectional management of thyroid carcinoma invading the airway. Ann Thorac Surg 1992;54:3–9.
82. Hajdu SI, Huvos AG, Goodner JT, et al. Carcinoma of the trachea: clinicopathologic study of 41 cases. Cancer 1970;25:1448–56.
83. Felson B. Neoplasms of the trachea and main stem bronchi. Semin Roentgenol 1983;18:23–37.
84. Janower ML, Grillo HC, MacMillan AS Jr, et al. The radiologic appearance of carcinoma of the trachea. Radiology 1970;96:39–43.
85. Cleveland RH, Nice CM Jr, Ziskind J. Primary adenoid cystic carcinoma (cylindroma) of the trachea. Radiology 1977;122:597–600.
86. Stark P. Das adenoidzystische Karzinom (Zylindrom) der Trachea. Fortschr Rontgenstr (RoFo) 1982;136:31–5.
87. Heitmiller RF, Mathisen DJ, Ferry JA, et al. Mucoepidermoid lung tumors. Ann Thorac Surg 1989; 47:394–9.
88. Zwiebel BR, Austin JH, Grimes MM. Bronchial carcinoid tumors: assessment with CT of location and intratumoral calcification in 31 patients. Radiology 1991; 179:484–6.
89. Weber AL, Shortsleeve M, Goodman M, et al. Cartilaginous tumors of the larynx and trachea. Radiol Clin North Am 1978;16:261–71.
90. Dunne MG, Reiner B. CT features of tracheobronchomegaly. J Comput Assist Tomogr 1988;12:388–91.
91. Katz I, LeVine M, Herman P. Tracheobronchomegaly: the Mounier-Kuhn syndrome. Am J Roentgenol Radium Ther Nucl Med 1962;88:1084–94.
92. Shin MS, Jackson RM, Ho K-J. Tracheobronchomegaly (Mounier-Kuhn Syndrome); CT diagnosis. Am J Roentgenol 1988;150:777–9.
93. Feist JH, Johnson TH, Wilson RJ. Acquired tracheomalacia: etiology and differential diagnosis. Chest 1975; 68:340.
94. Aquino S, Shepard JO, Ginns LC, et al. Acquired tracheal malacia: detection by expiratory CT scan. J Comput Assist Tomogr 2001;25:394–9.
95. Judd DR, Dubuque T Jr. Acquired benign esophagotracheobronchial fistula. Dis Chest 1968;54:237–40.
96. Berkmen YM, Young HA. CT diagnosis of acquired tracheoesophageal fistula in adults. J Comput Assist Tomogr 1985;9:302–4.

Diagnostic Endoscopy

Hermes C. Grillo, MD

General Considerations

Laryngoscopy and bronchoscopy are always required to assess airway lesions. Esophagoscopy is added if the lesion is a tumor that may involve the esophagus, if there is a question of fistula, or if there is another reason to suspect esophageal pathology. Mediastinoscopy is not often done as a separate procedure for the assessment of primary tracheal lesions.

Photographs of some lesions, as observed through bronchoscopy, are included in the color plates.

Laryngoscopy

This procedure is performed to determine if there is pathology in the larynx, either as an extension of or in addition to tracheal pathology, and also to assess glottic function and competence (see Chapter 35, "Laryngologic Problems Related to Tracheal Surgery"). Functional evaluation of the larynx is made by indirect examination, or, now more commonly, with a flexible laryngoscope or bronchoscope. The conscious patient can cooperate in maneuvers necessary to show whether the vocal cords move normally. Glottic function may be assessed under general anesthesia by lightening the anesthesia to a point where vocal cord reflexes return, but this is not nearly as satisfactory. Mass movement may be difficult to interpret. Examination must be accurate and is often best performed by a consulting otolaryngologist. If any complexity is suspected, I usually arrange for an endoscopy as a joint effort with the otolaryngologist. Degrees of aspiration on deglutition are assessed by barium studies, conventional or modified.

If patients have pathology in both the larynx and trachea, it is critically important to define both lesions at the outset, in order to plan effective treatment. Generally, a significant non-neoplastic laryngeal lesion should be corrected or made manageable by an initial laryngeal procedure before the tracheal lesion is surgically treated. Alternatively, both laryngeal and tracheal lesions may be repaired concurrently, although this usually requires concomitant tracheostomy for safety, which is not always desirable because the surgeon might correct a tracheal lesion by extensive resection, possibly bringing the larynx down to the sternal notch. If an obstructive lesion in the larynx is only then detected, tracheostomy may be difficult or

dangerous to the fresh anastomosis, either to the larynx itself or to an adjacent brachiocephalic artery. Intubation, preferably avoided following fresh tracheal reconstruction, could be necessary.

Laryngoscopic examination is particularly important in patients with trauma to the upper airway, where recurrent laryngeal nerve injuries are likely. Many patients with postintubation tracheal stenosis have inflammatory lesions of the larynx as well, which may cause obstruction in concert or independently. Vocal cord paralysis may also be present in these patients with or without a history of prior tracheal reconstruction or of direct injury to the larynx. Cord paralysis also occurs with primary or secondary tracheal neoplasms, and high tumors may invade the larynx.

Bronchoscopy

For a tracheal lesion, bronchoscopy has two purposes. The first is to obtain precise diagnosis of the type and extent of the lesion. The second is to treat obstruction, if present, in order to provide an airway adequate for immediate resection, to allow delay for further studies or treatment prior to resection, or to permit safe nonsurgical treatment such as stenting or irradiation (see Chapter 40, "Tracheal and Bronchial Stenting" and Chapter 41, "Radiation Therapy in the Management of Tracheal Cancer"). For the first purpose, I prefer rigid bronchoscopy. For therapeutic purposes, the rigid instrument is essential. Hopkins telescopes used through the rigid bronchoscope provide a magnified image, which is very much clearer optically than that provided even with improved flexible fiber optics. This provides precise delineation of the extent of glottic and subglottic injury and of the tracheal lesion. Measurements of distances between major normal and pathologic landmarks are more accurately made with the rigid bronchoscope. Furthermore, the rigid bronchoscope affords better control of the airway while these observations are being made. The flexible bronchoscope is useful for visualization and for diagnostic maneuvers such as biopsy, brushing, and aspiration. It is especially useful for precise positioning of endotracheal tubes, guiding catheters or stents for intraoperative localization of lesions, and for examination of a new anastomosis.

Usually, radiologic examination of the airway precedes bronchoscopy. These images serve as a road map for the bronchoscopist. Obviously, there are cases of suspected airway obstruction where bronchoscopy is done initially and sometimes urgently. If significant obstruction is identified, it is preferable not to instrument the lesion unless urgently required to, in order to avoid precipitating acute obstruction, unless the operator is prepared to proceed with whatever is necessary to relieve the obstruction, either by endoscopy or surgically. Definitive endoscopic examination is best accomplished under general anesthesia with rigid instruments, and most often immediately before a surgical operation to correct the lesion. This avoids any hazard of precipitating obstruction during diagnostic examination and avoids a second anesthesia. Definitive histologic diagnosis may be obtained prior to resection by dependable frozen sections from the generous biopsies permitted via rigid bronchoscopy. A limited lesion that clearly needs resection may not require preliminary biopsy. Control of post-biopsy bleeding under general anesthesia with a rigid bronchoscope in place has, in our hands, never presented a major problem. If this were to occur, tamponade with a cuffed endotracheal tube should provide control, followed if need be by surgery. Lesions that appear excessively vascular on telescopic examination should best not be biopsied. Aneurysmal vessels or hemangiomatous appearance are indications for angiography.

Bronchoscopy, as a separate procedure prior to surgery, is justifiable where the lesion is particularly complex and requires special planning, where reparative or extirpative surgery seems highly unlikely, if the patient will require a prolonged period of preparation for surgery, or if frozen sections are unlikely to define the type or extent of an unusual tumor.

Virtual bronchoscopy using three-dimensional reconstruction from helical computed tomography images offers a noninvasive technique for initial evaluation of tracheal or bronchial lesions, but it does not replace the refinement and precision of an actual bronchoscopy.[1]

Therapeutic bronchoscopy for obstruction can be a critical procedure and is discussed in Chapter 19, "Urgent Treatment of Tracheal Obstruction." I do not believe a surgeon should attempt tracheal reconstructions unless he is competent in and equipped to perform rigid bronchoscopy.

Esophagoscopy

This technique is performed when any preceding imaging studies, usually with relation to a tumor, indicate deformation or possible involvement of the esophagus. Often, tumors deform the esophageal wall without actually extending into or through the mucosa. Nonetheless, it is wise to add esophagoscopy, following bronchoscopy. Esophagoscopy may be useful to visualize tracheoesophageal fistulae after first examining the defect bronchoscopically. In the rare patient with a concomitant esophageal stricture at the level of a tracheal stricture, or with an unrelated distal stricture, these lesions should be evaluated, and if necessary, dilated.

Mediastinoscopy

This procedure has shown little value for evaluation of primary tracheal tumors, particularly adenoid cystic carcinoma. The finding of involved lymph nodes adjacent to the tumor on one or both sides of the trachea does not make a patient unsuitable for surgical resection. These paratracheal lymph nodes are the first regional lymph node stations involved by a tracheal tumor and do not seem to carry the same significance for adenoid cystic carcinoma of the trachea as they do for carcinoma of the lung. I consider these to be N1 nodes with respect to a tracheal tumor. If the primary tracheal tumor is large, and invasion of other mediastinal structures is suspected, then mediastinoscopy may be performed immediately prior to a planned exploration for resection under the same anesthesia. Surgical planes will thus not be confused by inflammatory scarring, which results if resection is delayed following mediastinoscopy.

Histologic information from mediastinoscopy is obtained by frozen section. This permits surgeons with final responsibility for tracheal or carinal resection and reconstruction to make their own decisions, and to operate in a freshly dissected field, where iatrogenic changes do not confuse basic pathology. Mediastinoscopy does not provide any better mobilization of the trachea than can be obtained by retrograde pretracheal dissection at thoracotomy.

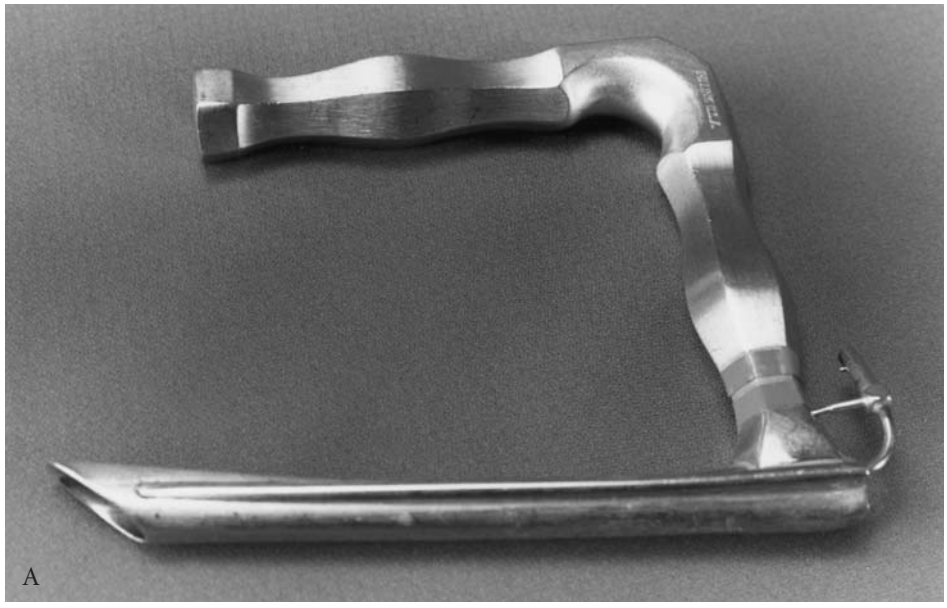
In evaluating bronchogenic carcinomas that invade the main bronchus or carina, it is also preferable to defer the necessary mediastinoscopy to the time of a potential carinal resection. If N2 lymph node involvement is discovered, which might dictate delay for a program of adjunctive preoperative treatment, consequent inflammatory changes must be accepted.

Techniques

This chapter assumes familiarity and competence with the endoscopic techniques and instruments discussed herein, including facile use of the rigid bronchoscope.

Laryngoscopy

If it is to be performed concurrently with rigid bronchoscopy, laryngoscopy is done first, almost always under general anesthesia. Where laryngeal complexities are suspected or where it is possible that a laryngeal surgical procedure will have to be done at the time of the examination, or later as an independent but preceding procedure, a consulting otolaryngologist should be present. Often, the consulting otolaryngologist will have examined the patient previously, and already have performed an indirect or flexible laryngoscopy. We find the Holinger anterior commissure laryngoscope to be particularly useful for examining the glottis and subglottic larynx under anesthesia (Figure 5-1*A*). The Lewy suspension apparatus is frequently useful (Figure 5-1*B*). Anesthesia or ventilation may be easily maintained by intermittent placement of an endotracheal tube through the laryngoscope. Hopkins optical telescopes provide a clear, magnified view



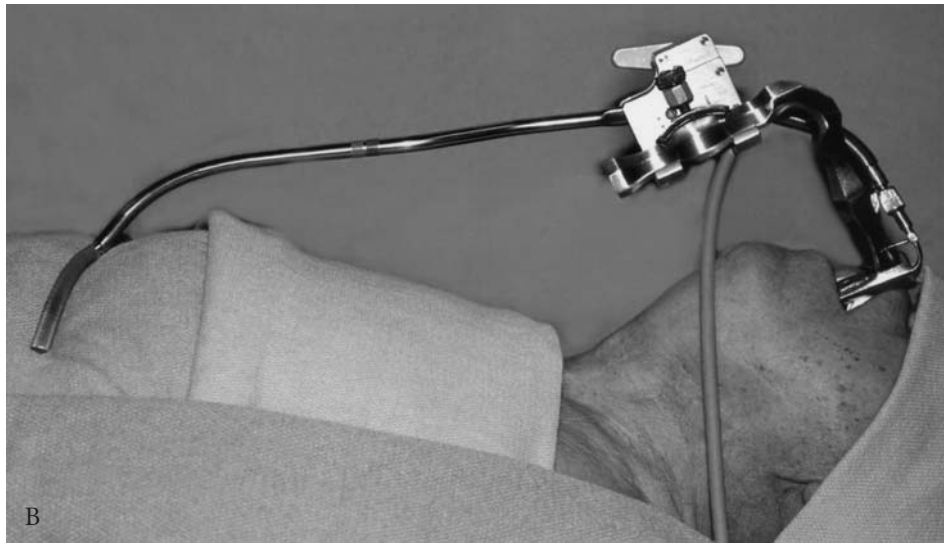


FIGURE **5-1** A, *A Holinger anterior commissure laryngoscope, especially useful for direct visualization of the glottis and subglottic larynx.* B, *The Lewy suspension apparatus in use. The angle of the laryngoscope is fully adjustable.*

through the laryngoscope. Minor laryngeal procedures, such as removal of vocal cord polyps or granulomas, or even division of a posterior commissural stricture between the arytenoids, may be accomplished at this time. If considerable manipulation is required, it is preferable not to proceed at once to a further tracheal procedure, since glottic edema may become a problem postoperatively. In this way, tracheostomy may be avoided.

If the laryngeal lesion is of great complexity, such as a complete subglottic stenosis that will require laryngofissure and stenting, the patient may be transferred to the care of the otolaryngologist until that problem is resolved. Endoscopic examination may reveal details that were not evident from radiologic images, particularly in a high subglottic laryngotracheal lesion. I prefer to be certain that a complex laryngeal reconstruction has truly succeeded before proceeding to an extended tracheal or laryngotracheal reconstruction. Such patients often already have a distal tracheostomy in place, which is removed at the time of the subsequent reconstruction.[2] Maddaus and colleagues preferred to perform repair of both lesions concurrently, establishing a prolonged tracheostomy with stent placement for later removal.[3]

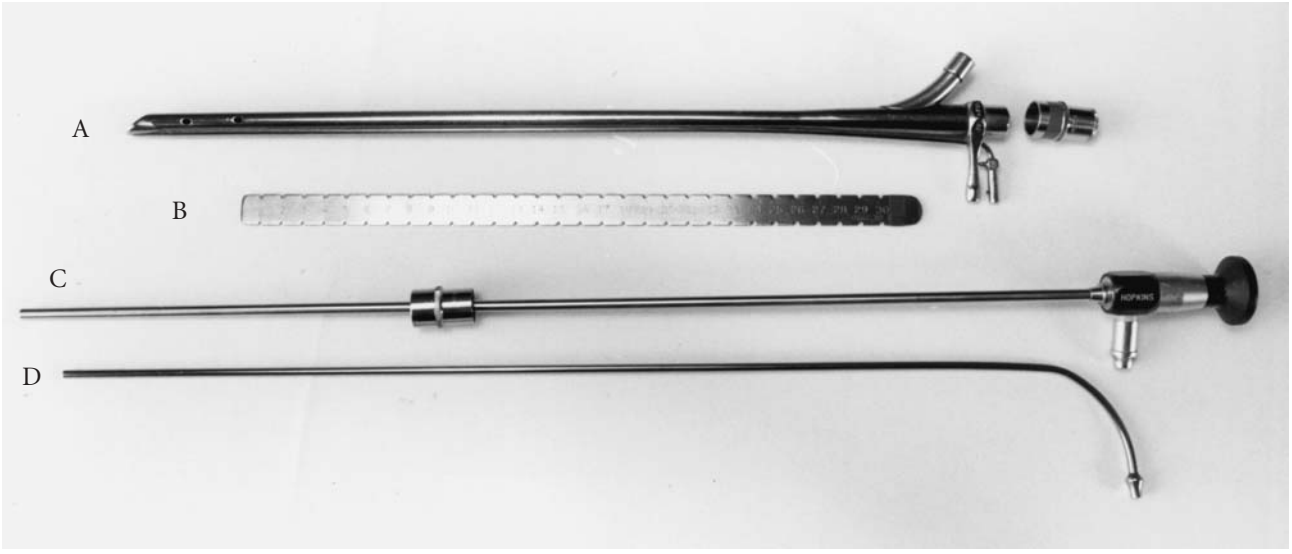


FIGURE 5-2 *Basic equipment for a rigid bronchoscopy. From top to bottom:* A, *Jackson bronchoscope (Pilling) with a ventilatory sidearm and eyepiece cap at the right. Bronchoscopes of internal diameter 7, 8, and 9 mm are routinely provided for adult examination.* B, *Metal centimeter ruler.* C, *Storz Hopkins telescope with a "gasket" to seal the bronchoscope. Telescopes of 0°, 30°, and 90° are made available in the kit.* D, *Adequate large-bore suction tip, especially useful for brisk or massive hemoptysis, thick secretions, biopsy, or coring-out procedures.*

Rigid Bronchoscopy

This technique best entails general anesthesia, which may be given safely even in the face of severe airway obstruction (see Chapter 18, "Anesthesia for Tracheal Surgery"). Numerous techniques have been described for the anesthetic management of rigid bronchoscopy under general anesthesia, including jet ventilation. I have found the Jackson ventilating bronchoscope, with a side port for attachment of an anesthesia tube and window eyepiece or telescopic gasket, to be highly satisfactory. The surgeon must be on hand during induction of anesthesia, especially when there is any degree of airway obstruction. Equipment for relief of obstruction must be immediately available if obstruction is present, and the anesthetist must have confidence that the surgeon can establish an airway at any moment. This presupposes complete competence in rigid bronchoscopy, which unfortunately seems in danger of becoming a lost art.

Our bronchoscopic set-up for examination of an adult includes 7, 8, and 9 mm rigid Jackson ventilating bronchoscopes (Figure 5-2). The tips of these bronchoscopes are oblique with rounded edges, and are easier to introduce through a tight stenosis or past a tumor than are the spade-like tips of the Storz instruments, which may also elevate a flap of mucosa (Figure 5-3). A large bore suction tip must be available. The Storz Hopkins telescopes are introduced through adapters placed over the open end of the rigid bronchoscope to provide a seal and permit ventilation (see Figure 5-2). When the telescope is not in place, a window cap is placed over the proximal end of the bronchoscope. Also available in the endoscopic room or immediately adjacent should be a kit of sterile pediatric Jackson rigid bronchoscopes of the following sizes: 3.5, 4, 5, and 6 mm. These may be used serially to dilate tightly stenotic lesions, as described in Chapter 19, "Urgent Treatment of Tracheal Obstruction." The Storz pediatric bronchoscopes are superb for diagnosis in children, but for dilation, the tip of the Jackson pediatric bronchoscope is preferable. If it is known that a high degree of obstruction is present, these bronchoscopes should be on the instrument table initially, along with selected small bougies (see Figure 19-1 in Chapter 19, "Urgent Treatment of Tracheal Obstruction").

Even if the patient has a preexisting tracheostomy, bronchoscopic examination is commenced through the mouth, passing the rigid bronchoscope into the larynx and through the glottis as far distally as

FIGURE 5-3 *Comparative tips of Jackson* (above) *and Storz* (below) *rigid bronchoscopes. The Jackson model is more easily introduced into a tightly stenotic lesion with a rotatory motion than the Storz model, with its more shovel-like tip.*

is easily permitted. This provides a picture of the anatomy of the entire airway and more precise measurements of lengths of lesions and normal structures. If the obstructing lesion is not densely fibrotic, or if it is a tumor, then the bronchoscope is passed beyond the lesion. If the lesion is densely stenotic, particularly if it involves the subglottic larynx, or if there is discontinuity between the proximal airway and the more distal trachea, as occurs sometimes with postintubation stenoses or after trauma, then maximum information is obtained by examining the airway up to the point of obstruction, followed by completion of the examination of the distal trachea through the tracheostomy present.

Once the bronchoscope is passed and the airway is cleared of secretions (which may pool distal to a partially obstructing lesion), and also cleared of blood that may be incited by the examination, the 0° telescope is used first for visualization. Desired information about the larynx includes 1) status of both the structure and function of the glottis, including vocal cords, arytenoids, and the anterior and posterior commissures; and 2) airway diameter, deformities, stenosis, inflammation, edema, or involvement by tumor of the subglottic larynx down to the inferior margin of the cricoid cartilage.

The cricoid cartilage may be directly visualized, if not altered by pathology. It presents as a broad ring immediately above the narrower tracheal rings (Figure 5-4). If pathology has blurred this differential, gentle external pressure on the palpable cricoid cartilage by the bronchoscopist's finger, while looking through the 0° telescope, usually defines the exact level. For greatest precision, a no. 25 hypodermic needle may be passed through the skin and wall of the trachea or larynx, just below the cricoid, and its entry point into the airway noted. In adult males of average size, an 8 or 9 mm bronchoscope (40 cm long) is used initially. In smaller males or in females, the 7 mm bronchoscope is used initially. For visualization and manipulation of upper tracheal lesions (such as injection of triamcinolone or Depo-Medrol into lesions with special long hypodermic needles), a 9 mm bronchoscope, which is only 25 cm long, is useful (Figure 5-5).

After the larynx has been fully examined, the bronchoscope is passed distally, observing the tracheal configuration in anteroposterior and lateral directions and for abnormalities in the cartilaginous architecture. The membranous wall is characteristically smooth. The mucosa is examined for inflammation, easy bleeding or increased vascularity, extrinsic or submucosal deformities, and mucosal lesions. Areas of malacia can sometimes be detected, assisted by gentle palpation in the neck or by observing collapse with respiration as the patient's anesthesia is lightened. Malacia is better identified with the flexible bronchoscope

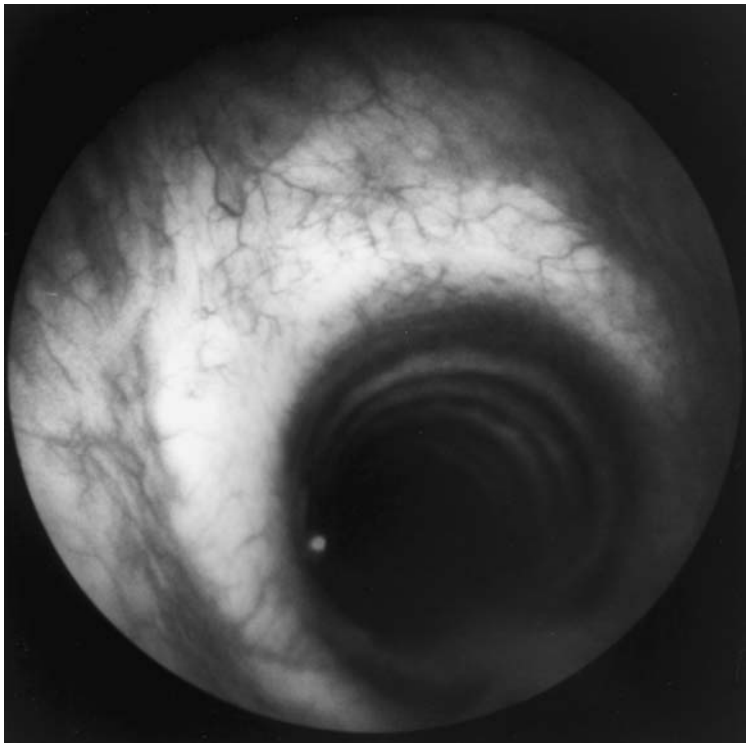


FIGURE **5-4** *Endoscopic delineation of the border between the inferior margin of cricoid cartilage and the uppermost tracheal rings. The cricoid is very broad compared to the narrow tracheal rings below. Bronchoscopic measurements of the tracheal length accurately use this point proximally. However, if the cricoid is indistinct or obscured by pathologic change, the glottis is used as the proximal landmark, but the approximate length of the subglottic larynx (to bottom of cricoid) must be subtracted to obtain the tracheal length.*

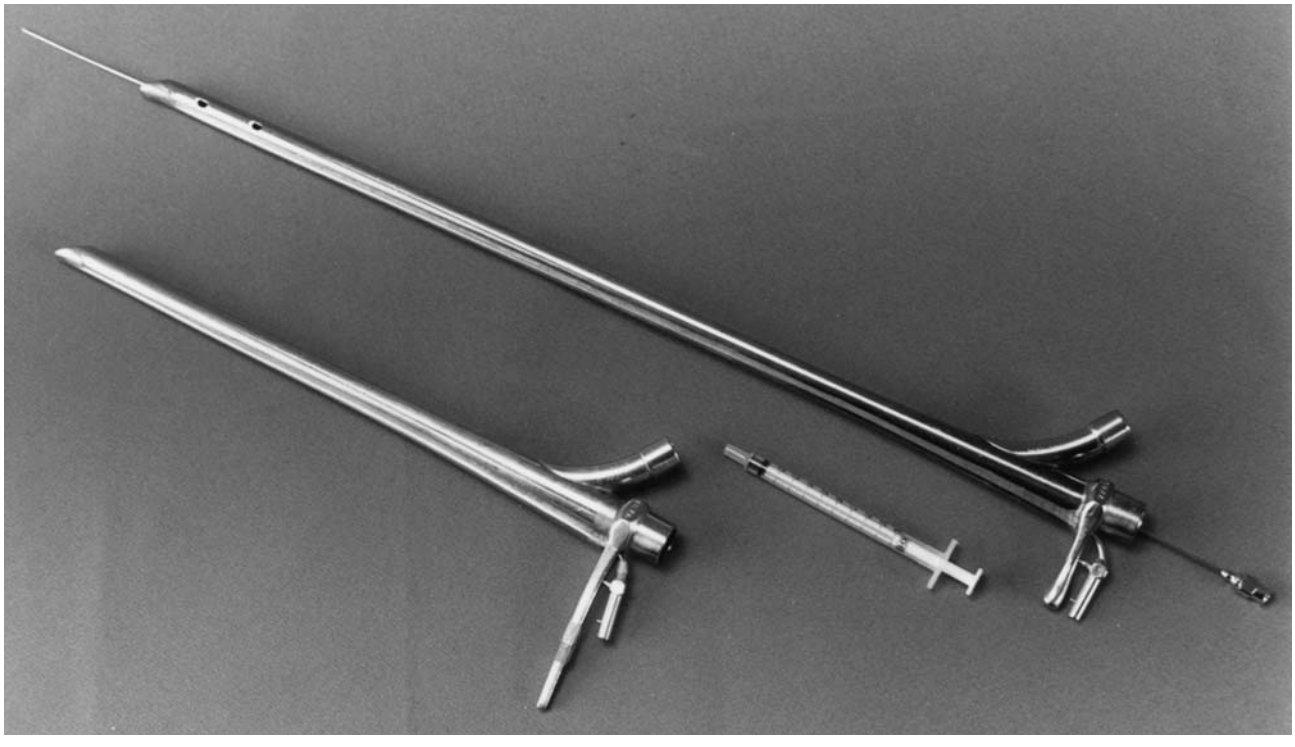


FIGURE **5-5** *Equipment for intralaryngeal or intratracheal injection. A no. 20 needle with no. 25 tip is long enough to reach through an adult rigid bronchoscope (8 mm, 40 cm shown). A tuberculin syringe is necessary to develop sufficient pressure to inject fluid via the long needle with this fine tip. Also shown is a short bronchoscope (9 mm, 25 cm) to facilitate access to laryngeal and subglottic areas.*

under topical anesthesia so that the patient may cooperate in maneuvers that emphasize collapse, usually cervical inspiratory collapse or intrathoracic expiratory collapse. Complete bronchoscopic examination to segmental levels is particularly important in patients with squamous cell cancer of the trachea, since they may well have concurrent squamous cell lesions of the aerodigestive tract. A flexible bronchoscope is easily passed through the telescopic "adapter" via the rigid bronchoscope to complete a distal survey of segmental bronchi. These patients also deserve complete endoscopic examination of the pharynx and esophagus, in addition to laryngoscopy and bronchoscopy.

The endoscopist should be familiar with normal and abnormal variations in the trachea (see Figure 1-2 in Chapter 1, "Anatomy of the Trachea"). Sabre-sheath trachea has been mistakenly identified as stenosis (see Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions"). Tracheopathia osteoplastica is very rare and may not be recognized when observed (see Chapter 15, "Tracheobronchial Malacia and Compression"). Findings in children and in congenital lesions of the trachea are described in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children." Even the most severe deviations of the trachea due to extrinsic pressure, as seen with huge substernal and intrathoracic goiters, usually allow easy passage of a rigid bronchoscope (see Chapter 15, "Tracheobronchial Malacia and Compression"). The trachea widens and straightens out as the rigid bronchoscope is passed. Normal contraction of the posterior membranous wall, and its protrusion forward in response to cough, either in the awake patient or under lightened anesthesia, should not be confused with malacia. Pulsatile compression should especially be noted. Pulsation and extrinsic deformation is commonly seen in the left lateral wall of the lower trachea due to the adjacent aorta.

Bronchoscopes should not be pushed through a fibrous stenotic lesion with excessive force. Dilation should be carried out systematically (see Chapter 19, "Urgent Treatment of Tracheal Obstruction") to avoid splitting or perforating the tracheal wall proximal to the tough fibrous stricture of a postintubation stenosis. In the case of tumors, one can *always* initially pass the rigid bronchoscope beside the tumor, on the side where tumor is not attached to the tracheal wall, no matter how complete the obstruction appears to be. Cartilage will yield and permit the bronchoscope to pass, partly displacing the tumor. The bronchoscope can be passed through the center of the circumferential tumor, carefully following even a tiny opening. A small bougie can serve as a guide. Cardiopulmonary bypass has been unnecessarily employed to resect tumors that appear to be causing nearly complete obstruction. A severely obstructing tumor can be cored out using the rigid bronchoscopes, with additional trimming done with biopsy forceps (see Chapter 19, "Urgent Treatment of Tracheal Obstruction"). The technique is so simple that we have found it unnecessary to use the laser for this purpose.[4] Bleeding has not been a problem, despite cautions and alarms cited as rationale for laser removal of obstructing tumor. I routinely measure findings, including the location of normal structures and of the upper and lower limits of lesions. This is performed by difference, as illustrated in Figure 5-6. *Systematic measurement* is begun with the tip of the bronchoscope touching the carina, as seen through the 0° telescope, which is kept just *proximal* to the tip of the bronchoscope. Measurement is made of the distance from a selected point on the upper teeth or gingival ridge to a fixed point at the hub of the bronchoscope, using a centimeter rule. The bronchoscope is then withdrawn until its tip is at the lower border of the lesion. Measurement is again made at this point and is repeated with the bronchoscope's tip at the upper border of the lesion. Other points also noted are the location of a tracheal stoma, the level of the inferior margin of the cricoid cartilage, and the level of the vocal cords. Distances are obtained by subtraction from the figures recorded (Figure 5-7). Even with the most meticulous measurement, such figures are accurate only to about 0.5 cm because of the imprecise level of points of measurement and the flexibility of tissues. I find it helpful to construct a diagram of the airway and its lesions (see Figure 5-7*F*). It is posted in the operating room for reference and a copy is recorded in the patient's chart.

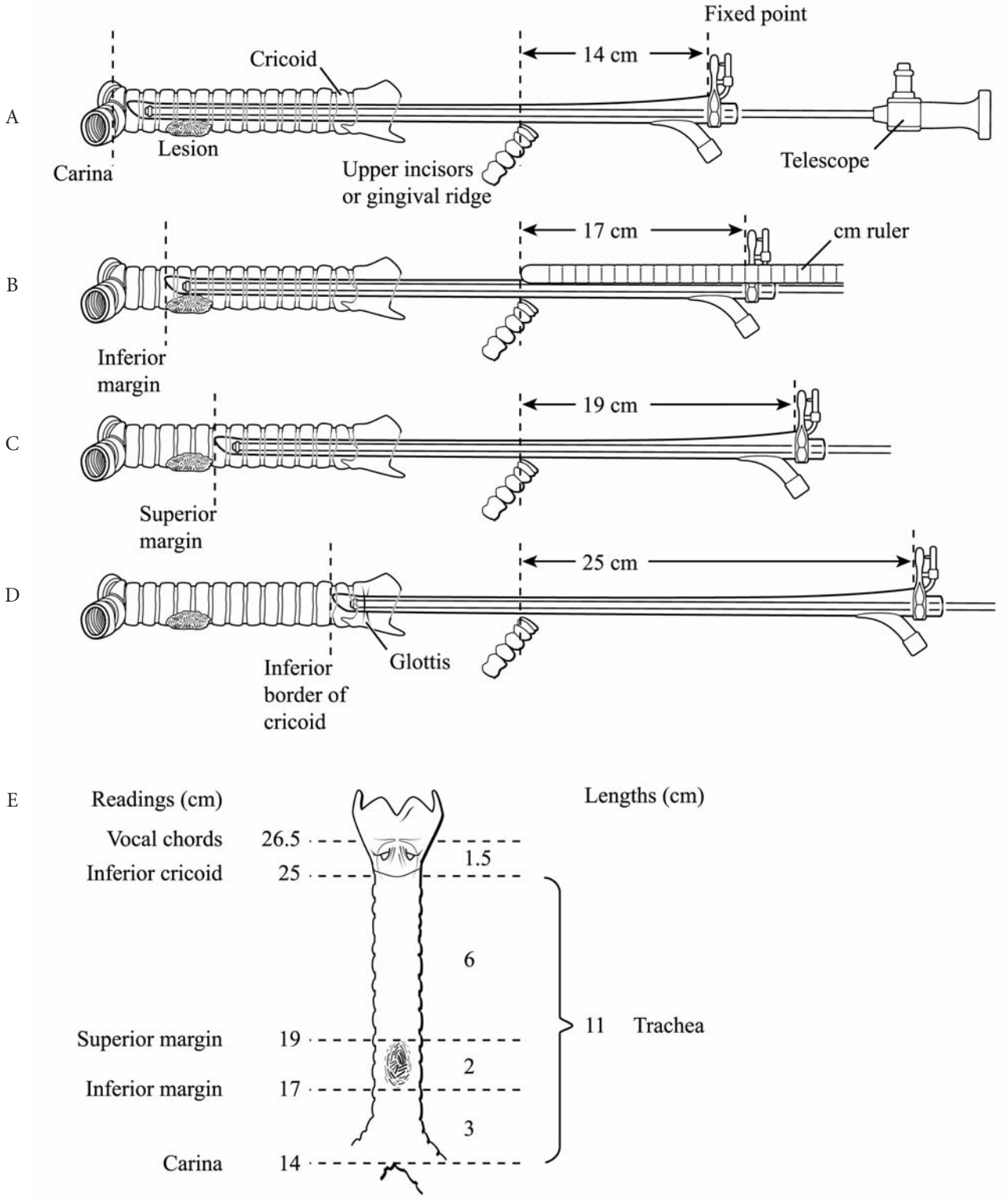
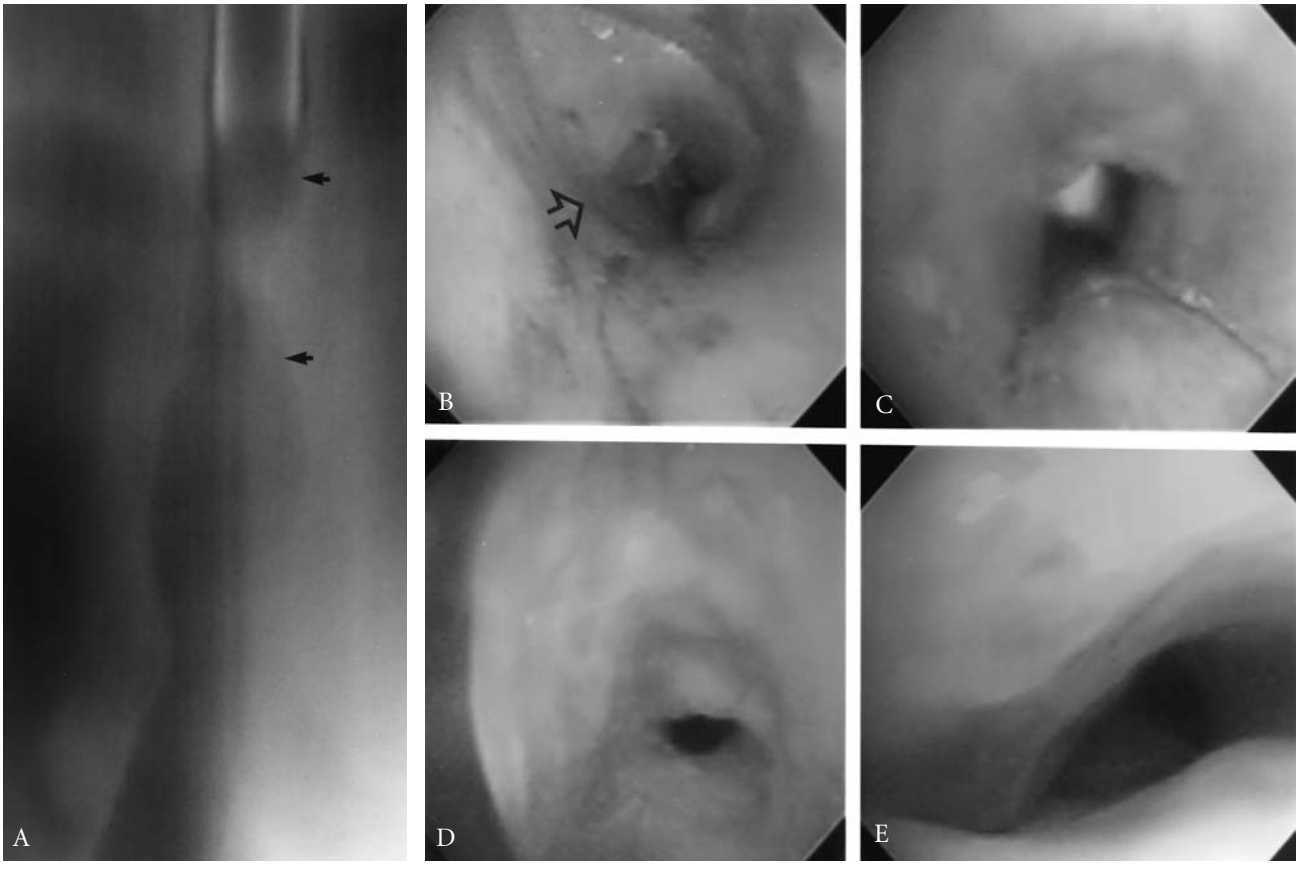


FIGURE **5-6** *Localization and measurement of the length of components of the upper airway and of a lesion. A telescope is just within the bronchoscope to locate the tip of the bronchoscope precisely.* A, *With the tip of the bronchoscope at the carinal spur, distance is measured and recorded between the incisor teeth (or upper gingival ridge) and a proximal fixed point on the bronchoscope. Similar measurements are made with the bronchoscopic tip successively at the lower edge of the lesion* (B), *the upper edge of the lesion* (C), *the lower edge of the cricoid* (D), *and/or at the vocal cords.* E, *Recording of measurements and approximate lengths (by subtraction).*



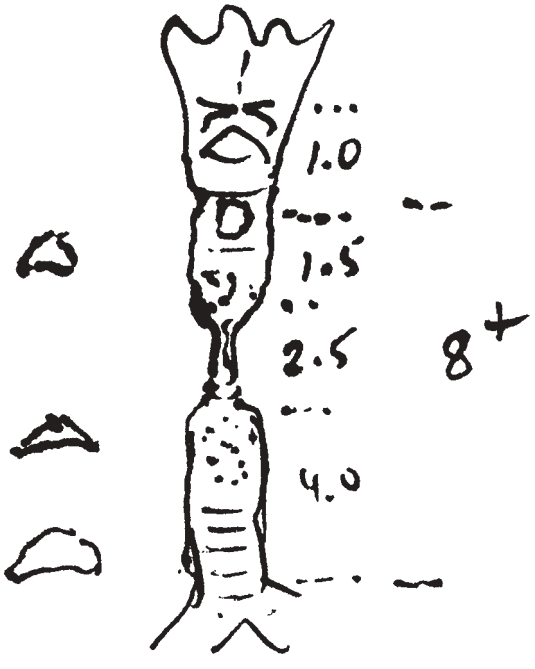


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FIGURE 5-7 *Quantitative recording of tracheal pathology.* A, *Tomographic cut of a trachea with the tracheostomy tube in place, stenosis and granuloma below* (between arrows), *a widened tracheal segment below this, and a short distal normal segment above the carina. This is a road map for the bronchoscopist. Bronchoscopic views correspond. View at:* B, *glottis* (arrow) *to site of stoma;* C, *stoma with tube in place;* D, *stenosis distal to stoma; and* E, *malacic and dilated trachea below stenosis with carina distantly seen.* F, *Diagram sketched in the operating room, from bronchoscopic measurements, using a marking pen on glove paper. Noted are the distances between vocal cords and tracheostome (*dotted lines; *1 cm), stoma and stenosis (1.5 cm), length of stenosis (2.5 cm), and distal trachea from stenosis to carina (4 cm). A granuloma is roughly outlined above the stenosis, and stippling distal to the stenosis locates malacia above a few remaining, relatively normal tracheal rings. The outlines at the left represent configuration of this severely damaged trachea at levels where they were observed. Total tracheal length is about 8 cm.*

During or following extensive rigid laryngoscopy and bronchoscopy, especially if manipulation has been necessary, Decadron is administered for 24 to 48 hours, beginning intraoperatively, in an effort to minimize edema. Racemic epinephrine is also often added.

Flexible Bronchoscopy

The flexible bronchoscope does not replace the rigid instrument in diagnosis nor in management of airway lesions, but it is a very useful adjunctive tool. It should be used liberally by pulmonologists to rule out organic obstruction in patients thought to have "adult onset asthma," to clarify the origin of hemoptysis (however minor), and to investigate the possible causes of recurrent or unyielding volume loss, atelectasis, or pneumonitis. Intubation, for any reason, is facilitated by using the flexible bronchoscope as a guide. Difficult intubations are made simple in this way. Traumatic tracheal separation may respond to this technique.

Rigid bronchoscopy is truly impossible in only very few patients. Examples included 2 patients with achondroplastic dwarfism with prognathous jaws, which produced a deep right angle between the oropharynx and the trachea. Another patient suffered severe fixed cervical deformity as a result of radical neck surgery and remote high-dose irradiation. In such patients, only flexible bronchoscopy is possible. If general anesthesia is elected, intubation may be avoided by passing the flexible bronchoscope through a laryngeal mask airway (Figure 5-8*A*).[5]

The flexible instrument may be used with the sealing cap used for telescopes through a rigid bronchoscope, in order to expand examination to include segmental bronchi. It is also introduced via an adapter through an endotracheal tube to identify a precise point in the airway during surgery (Figure 5-8*B*). If necessary, the endotracheal tube is partly withdrawn and the bronchoscopic light transilluminates the trachea to the operative field (with operating table lights deflected). Thus, the extremities of a postintubation stenosis, not clearly defined visually from the outside of the trachea, can be identified precisely. The surgeon passes a no. 25 needle through the tracheal wall into the lumen, and the needle is adjusted precisely by bronchoscopic confirmation (see Figure 24-10 in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection").

The flexible bronchoscope is also invaluable in placing and replacing either double or single lumen tubes in the left or right main bronchus during bronchial or carinal resections. A pediatric flexible bronchoscope can be passed through one of the channels of a double lumen tube. Tracheobronchial anastomosis can be examined intraoperatively in this way. Correction of postpneumonectomy syndrome, of splinting procedures for tracheobronchomalacia, and assessment for malacia after excision of huge compressive goiters are established by repeated intraoperative flexible endoscopy. Transillumination may help to identify the location of a bronchial stump, in the event of a chronic bronchopleural fistula being buried in dense, irradiated mediastinal cicatrix. Flexible bronchoscopy has become our routine method for examination of an anastomosis, prior to discharge of a patient recovering from tracheal reconstruction. It is much more dependable than any other imaging technique for the early identification of anastomotic defects of any type.

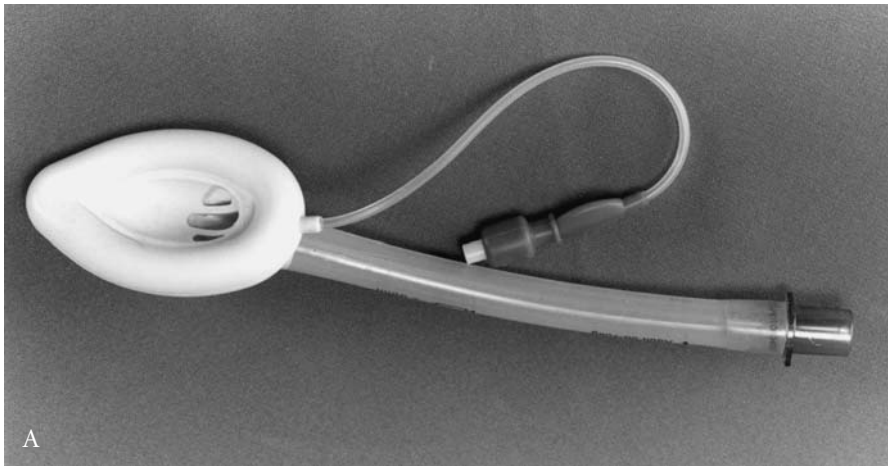

FIGURE **5-8** A, *Laryngeal mask airway (LMA). A flexible bronchoscope is easily passed through the LMA.* B, *Adapter for bronchoscopy via endotracheal tube or LMA (Portex).*

References

1. Finkelstein SE, Summers RM, Nguyen DM, et al. Virtual bronchoscopy for evaluation of malignant tumors of the thorax. J Thorac Cardiovasc Surg 2002;123:967–72.
2. Grillo HC, Mathisen DJ, Wain JC. Laryngotracheal resection and reconstruction for subglottic stenosis. Ann Thorac Surg 1992;53:54–63.
3. Maddaus MA, Toth JLR, Gullane PJ, Pearson FG. Subglottic tracheal resection and synchronous laryngeal reconstruction. J Thorac Cardiovasc Surg 1992;104:1443–50.
4. Mathisen DJ, Grillo HC. Endoscopic relief of malignant airway obstruction. Ann Thorac Surg 1989;48: 469–75.
5. Roberts JT. Clinical management of the airway. Philadelphia: WB Saunders; 1994. p. 365.

Congenital and Acquired Tracheal Lesions in Children

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Congenital tracheal lesions are rare, and acquired tracheal lesions in children are also relatively uncommon. Experience in their management is consequently quite limited and widely dispersed. The small diameter of the juvenile trachea can easily become obstructed following surgery. One millimeter of subglottic swelling can reduce a newborn airway to one-third of its normal cross-sectional area. The danger of separation or stenosis is increased after reconstruction, because the delicate structure of the tracheal wall tolerates anastomotic tension less well. The great length of many congenital lesions prohibits resection and presents special problems in correction. Earlier concerns about tracheal growth following anastomosis have been allayed by experimental and clinical observations.

Embryology of the Trachea

The laryngotracheal groove or sulcus appears in the proximal foregut at the third week (3 mm embryo, stage 10).[1] The laryngotracheal groove progresses caudad and the lateral ridges progress cephalad to form the primordium of the trachea (Figure 6-1). The pulmonary primordium appears and bulges ventrally from the foregut. Complete separation of trachea and esophagus occurs by 11 to 14 mm (sixth week). The tip of the tracheal primordium buds asymmetrically, left and right, at the 4 mm stage, to provide bronchial primordia. Mesenchymal proliferation by cells lining the coelomic cavity provides the tissue from which cartilage, muscle, and connective tissue will develop. Epithelial–mesenchymal interrelationships are essential for bronchial and pulmonary development to occur. The tracheal bifurcation moves gradually downward from the neck to the level of the fourth vertebra. Cartilage appears in the trachea at 10 weeks.

When the laryngotracheal groove appears, the forerunner of the glottis also appears as a median slit in the pharyngeal floor between the fourth and sixth branchial arches. The epiglottic primordium lies anteriorly and the arytenoid swellings lie laterally prior to their more medial migration. Ventricular buds are solid at first. A T-shaped slit appears, which opens into a lumen by the eighth week. Vocal cords are seen at 3 months. Thyroid and cricoid cartilages appear between 5 and 7 weeks. The laryngeal cartilages derive from the fourth and fifth arches.

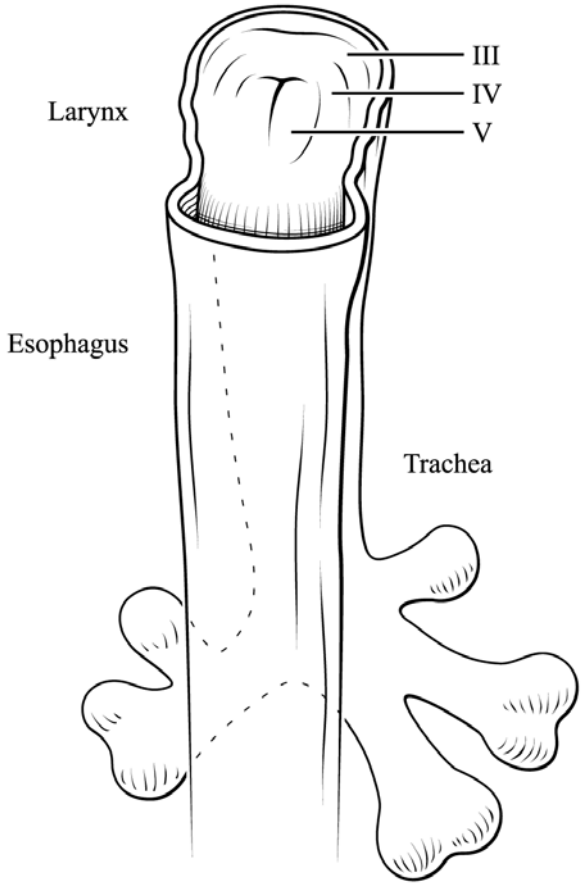


FIGURE 6-1 *Development of larynx and trachea at about the sixth to eighth weeks. The laryngeal slit lies in the floor of the pharynx. Numerals indicate arches. The foregut is separating into the trachea and esophagus. Stem bronchi and branches are present. Specific stages are altered to show general relationships. Adapted from Gray SW and Skandalakis JE.*[1]

Failure of complete separation of the foregut into respiratory and alimentary components is the most common defect and produces tracheoesophageal fistula (TEF). At the upper end, the larynx may fail to reopen, producing atresia (a fatal anomaly), or it may fail to form a complete posterior septum, producing a laryngotracheoesophageal cleft. Tracheal atresia, stenosis, esophageal atresia, and tracheoesophageal fistula occur more distally. The relatively separate processes of laryngeal development and budding of bronchi and pulmonary development allow for malformations of the trachea, such as agenesis and stenosis in the presence of a normal larynx and bronchial tree.

Congenital Lesions

Tracheal agenesis or *atresia* is usually fatal at birth. The larynx may form normally. The lungs may or may not be normal, and with or without bronchial communications to the esophagus (Figure 6-2). These malformations are extremely rare. The most common presentation is with normal bronchi, communicating centrally to the esophagus.[2] Other congenital anomalies are common in these patients. Fonkalsrud and colleagues described a newborn of type C, who survived for a short term by using the esophagus as an airway.[3] A major bronchus may also communicate directly with the esophagus while the balance of the lung is served by anomalous bronchi from a partly stenotic trachea. Microgastria is a common concomitant feature. No systematic surgical treatment has evolved, doubtlessly due to the rarity and variations in the anomalies as well as the complexity of the defects.

Hiyama and colleagues described two such patients in whom diagnosis was suspected due to respiratory distress without audible cry and difficulty in intubation.[4] One infant was successfully treated by the

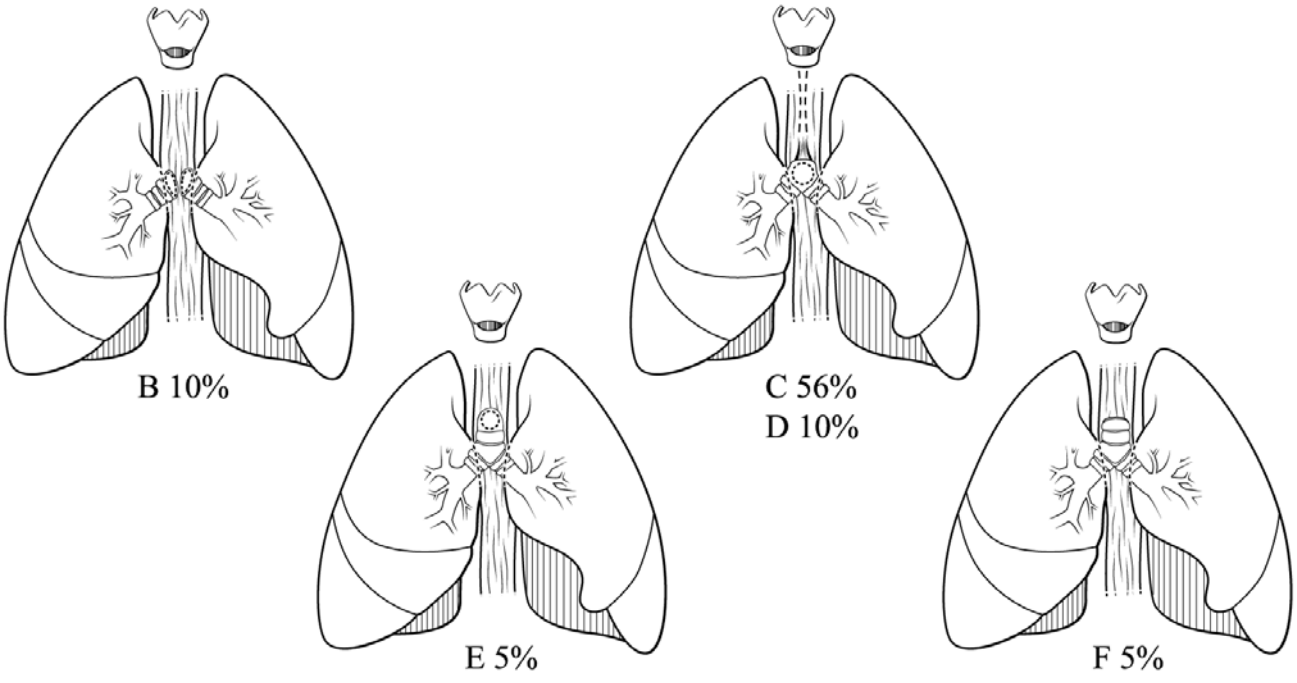


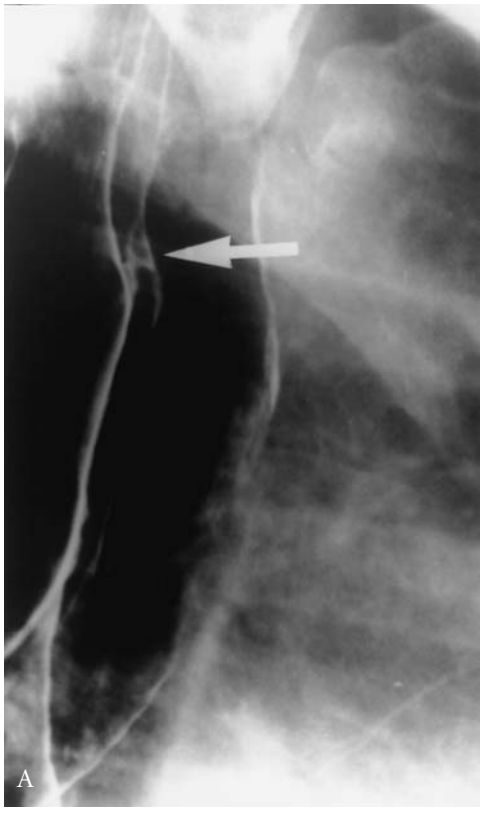
FIGURE **6-2** *Tracheal agenesis, redrawn according to Faro and colleague's classification.[2] In addition to types diagrammed, type A (8%) shows total pulmonary agenesis and type G (5%) has short segment tracheal agenesis. In type B, both main bronchi connect to esophagus separately, whereas in type C, bronchi are fused but a bronchoesophageal fistula (BEF) is present. Type D additionally has an atretic band* (dashed line) *from larynx to BEF. Type E has a tracheoesophageal communication, but in type F, communication with the esophagus is absent. Distribution of types in Faro and colleague's collected series of 39 patients is noted.*

following procedures: gastrostomy and abdominal esophageal banding, translaryngeal and esophageal ventilation by endotracheal tube, tracheostomy and later T tube, pharyngeal sump drainage followed by establishment of cervical esophagostomy (proximal tracheal segment present), and esophageal reconstruction by colonic interposition at age 3.

In the more common anomaly of *tracheoesophageal fistula*, the tracheal problem is usually managed by division and closure of the communication. Congenital TEF and the complexities of esophageal atresia have been well described and categorized. The reconstructive challenge is principally esophageal, and congenital TEF is not further detailed here. Rarely, there is accompanying tracheal stenosis. Adjacent local tracheomalacia, especially after repair of TEF, may cause respiratory problems, and is described later. Less commonly found is an *H-type congenital tracheoesophageal fistula* without concomitant esophageal atresia, often high in the trachea and usually small in diameter. It is sometimes discovered in the adult and is usually managed by transcervical division and closure (Figure 6-3).[5,6] Coughing is the usual symptom, especially after ingestion of liquids. A large fistula may be treated by limited tracheal resection and anastomosis, with esophageal closure.

Congenital *fistula between biliary and respiratory tracts* is extraordinarily rare.[7] Respiratory problems begin with cough and progress to intractable pneumonia. The most common location of the fistula is at the carina, but right and left main bronchial connections have been noted. Yellow fluid is identified bronchoscopically. Contrast will identify a long paraesophageal tract connecting to a hepatic duct. It has also been seen in a young adult.[8] Excision of the intrathoracic segment with closure at the carina (or bronchus) and at the diaphragmatic level cures the problem.

Congenital *bronchoesophageal fistula* is a rare anomaly with fewer that 150 cases reported (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula").[9] Symptoms may not occur until



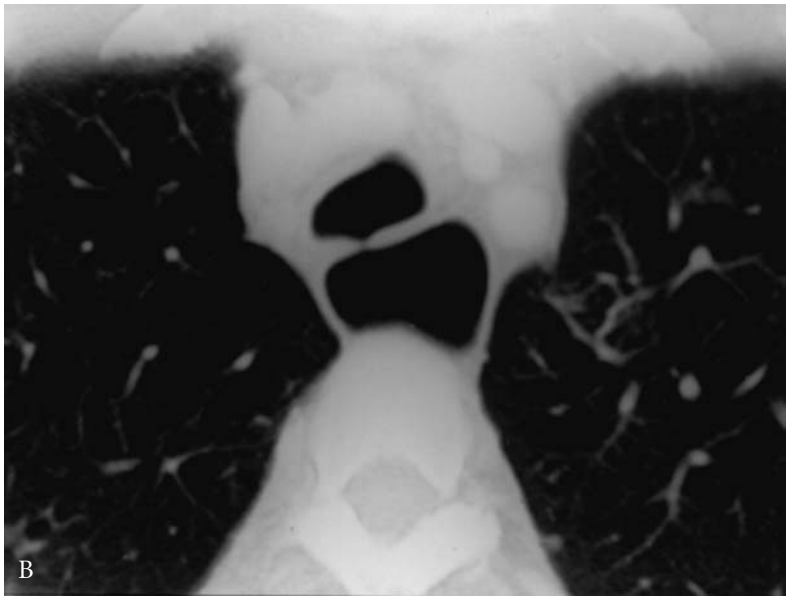


FIGURE **6-3** *H-type tracheoesophageal fistula in a 49-year-old woman.* A, *Fistula demonstrated* (arrow) *by barium contrast swallow. The trachea is clearly outlined.* B, *Computed tomography scan showing the small fistula. The fistula was divided and repaired easily through a low collar incision. Also, see Figure 6-21*B. *(Courtesy of Dr. Hon Chi Suen.)*

later in life (reported from age 9 to 83 years) but 25% of cases present before age 17. These fistulas connect to the middle or lower esophagus, from right upper lobe, from left lower lobe, from bronchus intermedius, from right middle or lower lobe, and from left upper lobe. The fistula usually slopes downward from the bronchus to esophagus, perhaps accounting in part for the lack of earlier symptoms, but it may also connect from a small diverticulum of esophagus, or on the pulmonary side, to a cyst or to a sequestrated lobe.[10] (See Figure 12-10 in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula.") Cough, especially after drinking, is the most common symptom, with respiratory infection and hemoptysis, and even hematemesis (seen less often). Barium esophagogram is the most accurate method of diagnosis. Endoscopy or bronchography are less helpful. Methylene blue in the esophagus aids bronchoscopic identification. The condition is permanently corrected by surgical excision and closure of the fistula, with interposition of healthy tissue between the bronchus and esophagus. Chronic pulmonary infection may dictate limited resection of the lung (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula" and Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula").

A very rare anomaly is the *laryngotracheoesophageal cleft*. Varying degrees of incompleteness of the wall between the larynx and trachea and the esophagus present (Figure 6-4).[11,12] A minor interarytenoid cleft reaching through cricoid cartilage, a deeper defect reaching to the upper trachea, and a complete cleft extending to the carina have been designated as types I, II, and III, respectively.[11] Ryan and colleagues added type IV, where the defect extends into both main bronchi.[12] Type I may not require repair. Type II has been repaired through laryngofissure or cervical or cervicomediastinal approach. Types III and IV are best approached laterally transcervically and transthoracically (see Chapter 33B, " Repair of Congenital Tracheal Lesions: Larynogotracheoesophageal Cleft Repair"). Donahoe and Gee first successfully repaired a type III cleft, with long-term survival.[13]

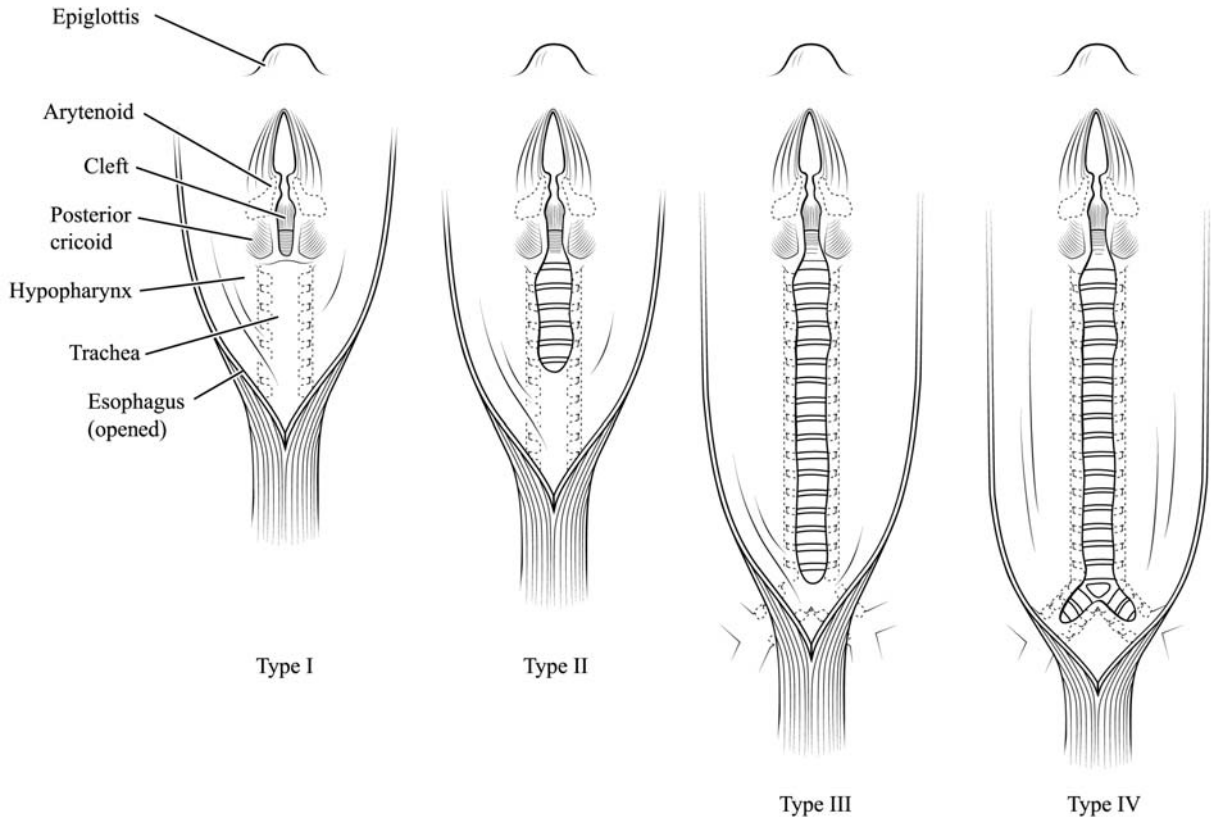


FIGURE **6-4** *Laryngotracheoesophageal cleft of increasing severity. Posterior view, with esophageal wall opened to show extent of cleft. Based on Petterson's classification.[11] Adapted from Ryan DP et al.[12]*

Tracheal webs sometimes occur in the neonatal and juvenile trachea at the cricoid level. Laryngeal webs at the glottic level are more common. The tracheal web does not involve any significant length of airway and is consequently usually treated endoscopically (Figure 6-5). Congenital cartilaginous cricoid stenosis is an extremely rare lesion.[14] Subglottic stenosis is more common as a consequence of postintubation injury. Other purely laryngeal congenital lesions, such as glottic and subglottic stenosis and atresia, are not considered here.

Segmental or diaphragm-like congenital main bronchial stenosis, principally on the right, has been very rarely encountered.[15] Segmental bronchial resection or even wedge resection in the instance of a thin web is an effective treatment. In the latter case, bronchoscopic management might be considered.

Congenital tracheal stenosis has been classified into three principal types: 1) generalized hypoplasia, 2) funnel-like narrowing, and 3) segmental stenosis (Figure 6-6).[16] The stenotic segment is most often composed of completely circular "O" rings of cartilage (Figure 6-7). Alternatively, disorganized cartilages, ridges, or plates of cartilages may occur (Figure 6-8). In type I, the larynx is of normal diameter but the entire trachea, or much of the trachea, is narrowed (1 to 3 mm diameter in the newborn) to a point just above the carina. The main bronchi are often normal in diameter, but may be more transverse than usual and frequently malacic. In some cases, the bronchi are also stenotic and composed of "O" rings of cartilage. In type II, the trachea begins with normal diameter, but then funnels down over a variable length to a tight stenosis. The location of the funneling and of the tight stenosis varies widely. It may be located proximally with a more distal normal segment. In many cases, the stenosis is long, involving more than half of the trachea. In type III,

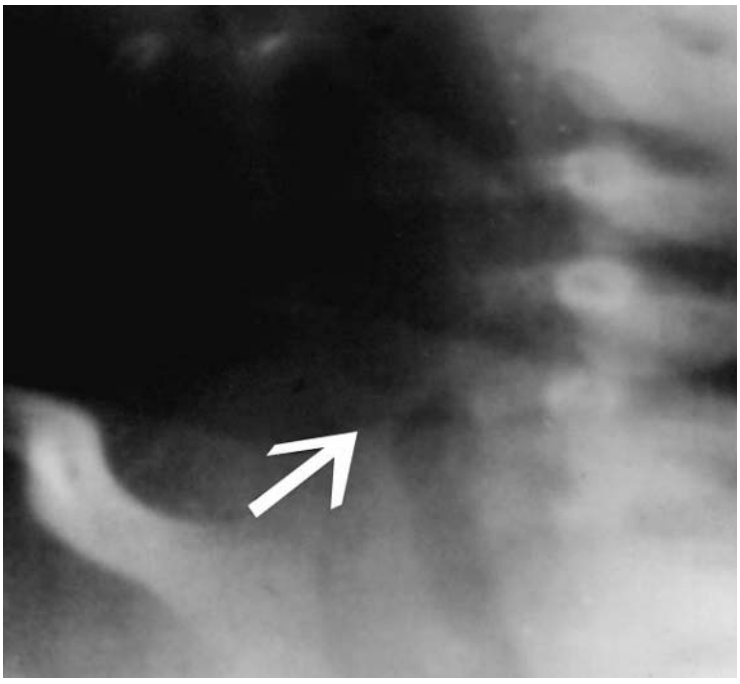


FIGURE 6-5 *Congenital web at cricoid level. The* arrow *marks the thin mucosal structure.*

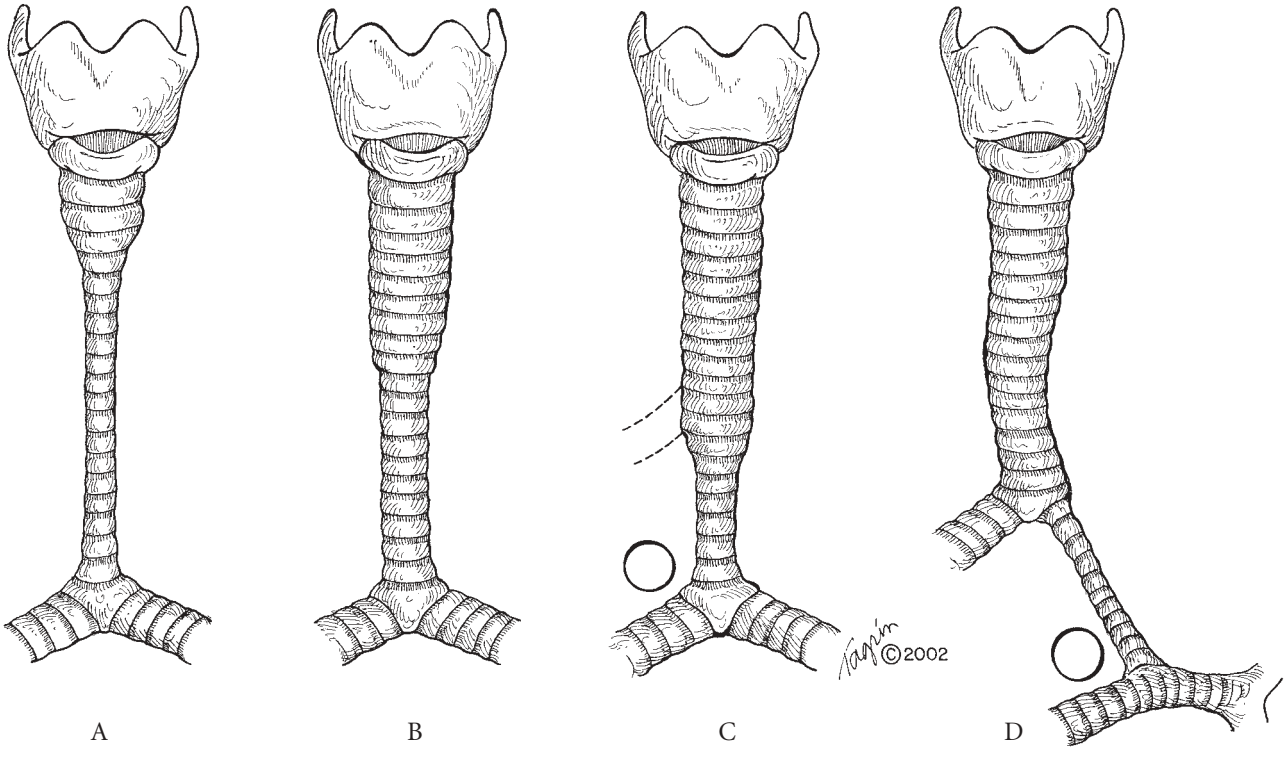
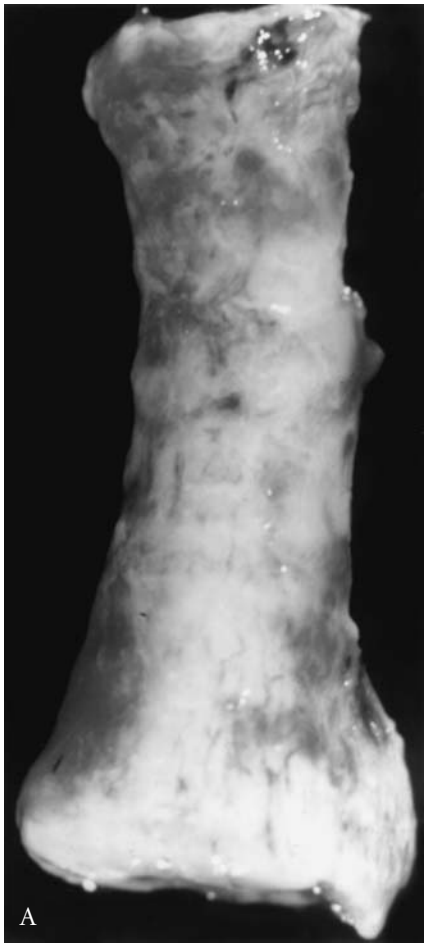


FIGURE 6-6 *Congenital tracheal stenosis. General categories of stenosis are diagrammed. Types I–III redrawn from Cantrell JR and Guild HC.*[16] A, *Type I: All or most of the trachea is stenosed.* B, *Type II: Funnel stenosis variously located and of variable length.* C, *Type III: Short segmental stenosis, sometimes below an anomalous right upper lobe bronchus.* D, *Type IV: Anomalous right upper lobe bronchus with a "bronchus" to horizontally branching bronchi to the rest of the lung. The right upper lobe bronchus is at the normal carinal level. The bridge bronchus is stenotic, and lesser stenosis may involve part of the trachea above. In some cases, the trachea is elongated as shown, but the upper lobe bronchus is absent.* Circles *indicate locations of left pulmonary artery sling when present.*

segmental stenosis may occur at any level in the trachea and be of any length, but it occurs most often in the lower trachea. Bronchial anomalies may be present, such as a misplaced right upper lobe bronchus (*bronchus suis*), which takes off from the trachea above an area of segmental stenosis. Tracheal stenosis may also be present above an anomalous right upper lobe bronchus, with tighter stenosis or "bridge bronchus" below the lobar bronchus. The right main bronchial anomaly is also found as an isolated variant apart from congenital stenosis, and is then usually closer to the carina. With some frequency, patterns are seen which manifest variable groupings of funnel tracheal stenosis, bronchus suis, a "bridging bronchus" (often the most severely stenotic segment), and distal branching into horizontal bronchi to right lower lobe and left lung, forming an inverted "T" (Figure 6-9).[17] The point of relatively transverse bronchial branching is held not to be the true carina, because the trachea plus "bridging bronchus" to this point is much longer for age than normal and contains many more rings than the normal trachea. These patients are more difficult to correct surgically than patients with stenosis in a trachea with a more normal pattern. Repair is described in Chapter 33A, "Repair of Congenital Tracheal Lesions: Tracheoplasty for Congenital Tracheal Stenosis."

In over half of the patients, congenital tracheal stenosis may be accompanied by many other malformations, including cardiac anomalies, hyaline membrane disease, pulmonary anomalies, inguinal hernias, imperforate anus, radial aplasia, and megaureters. Segmental stenosis of the distal trachea may be associated with an aberrant left pulmonary artery, the so-called "pulmonary artery sling" ("ring sling complex") (see Figure 6-9).[18,19] The left pulmonary artery originates from the proximal portion of the right artery and passes behind the trachea to the left lung (Figure 6-10). In this course, it indents, but rarely obstructs the



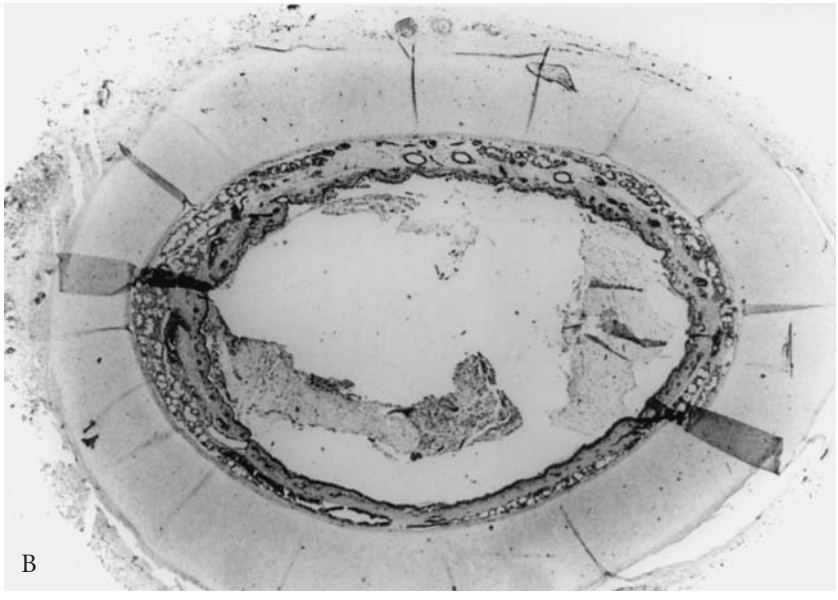


FIGURE **6-7** *Congenital tracheal stenosis with complete cartilage rings.* A, *Resected specimen of stenotic segment. The regular ring structure is evident. The ends flare toward normal diameters.* B, *Photomicrograph of cross section of trachea, demonstrating the complete "O" ring of cartilage.*

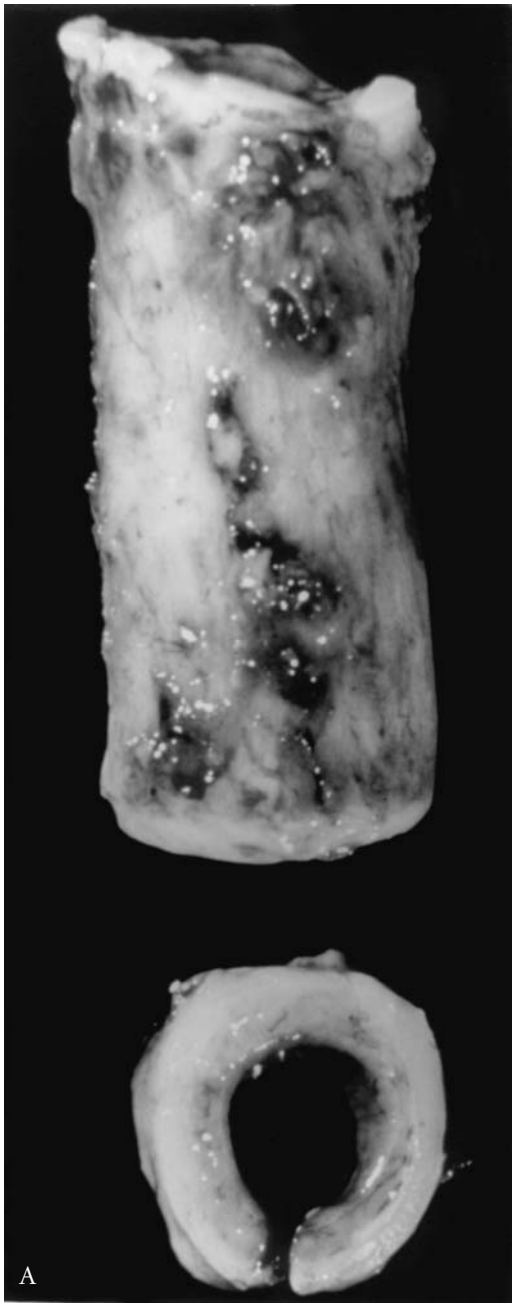
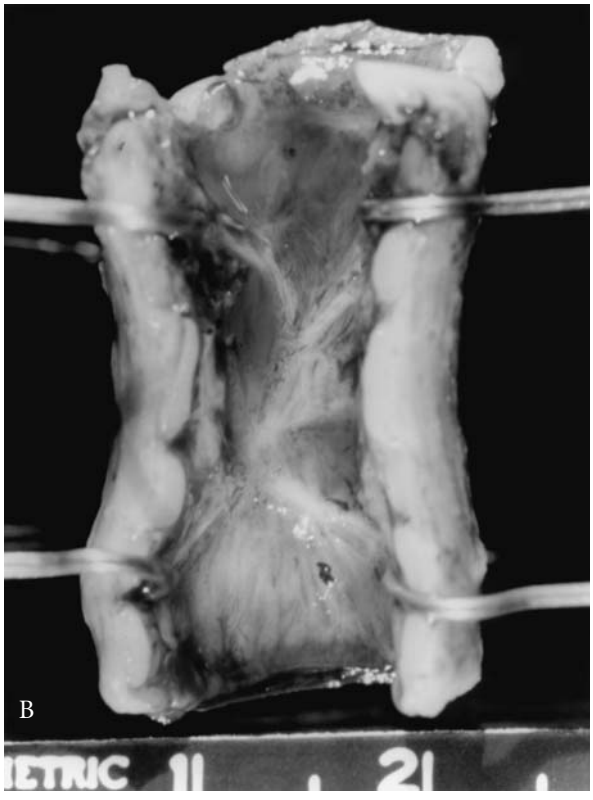


FIGURE **6-8** *Congenital stenosis with irregular, confluent plates of cartilage, a very rare malformation.* A, *Resected specimen. Note the absence of visible rings externally, in comparison with the usual structure of congenital stenosis shown in Figure 6-7*A. *A cross-sectional specimen below clearly shows completely circular cartilage.* B, *Luminal surface of* A. *Note the irregular "haustra" of disordered cartilage plates covered with mucosa.*



esophagus. In most of these patients, completely circular "O" rings of cartilage are found in the stenotic segment. The length of tracheal stenosis most often extends beyond the region of the anomalous pulmonary artery sling. Where stenosis is not present, there may instead be a malacic segment at the level of the artery.[19] The artery alone can also obstruct the right main bronchus.

Division of the anomalous pulmonary artery and reimplantation into the main pulmonary artery anterior to the trachea fails to relieve the airway obstruction when stenosis or malacia are present.[19,20] The ligamentum arteriosum, which effectively makes this a ring, is also divided. Sometimes, it has been possible to resect the stenotic tracheal segment and shift the artery anteriorly prior to anastomosing the trachea, as suggested might be possible by Grillo.[21,22] This is desirable because of a high rate of stenosis of reimplanted

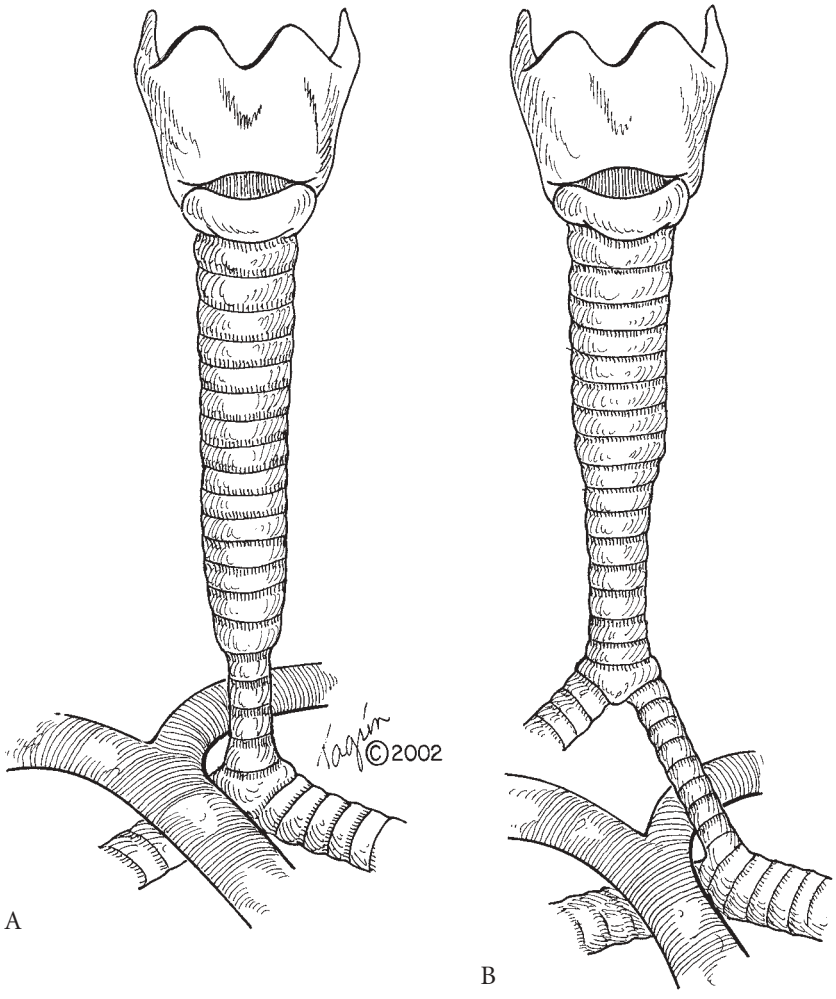
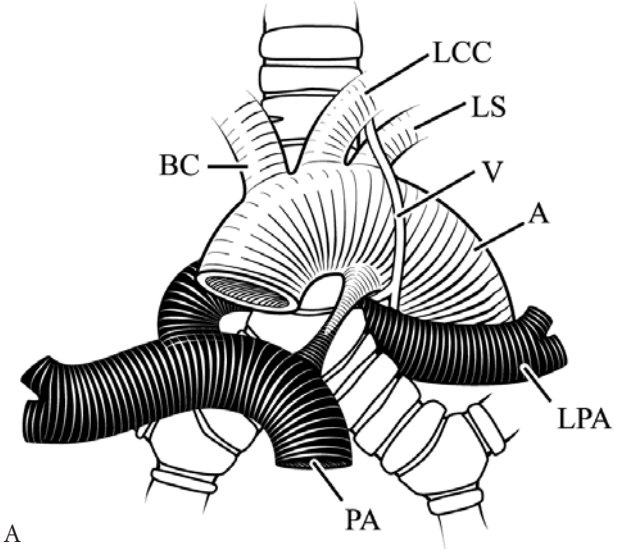


FIGURE **6-9** *Congenital stenosis with associated left pulmonary artery sling, the ring sling complex.* A, *A short segment distal stenosis is present.* B, *A bridge bronchus is adjacent to the anomalous artery. The apparent length of "trachea" is much greater than that in* A, *and the bronchial branching approximates an inverted "T."*

pulmonary arteries in children. The anatomic disposition of the pulmonary arterial sling, however, does not generally permit this transposition without division and reimplantation of the anomalous artery. The distortion of the artery may also compress the resected trachea and cause recurrent obstruction.[23]

Congenital tracheomalacia that is not the result of compression by vascular structures, by an extrinsic mass, or related to congenital TEF, occurs only rarely. It is infrequently documented in convincing fashion. Primary congenital tracheomalacia is recognized early in life, sometimes in previously apparently healthy infants. It is characterized by progressively noisy respiration, a "seal-bark" cough, episodic cyanosis, increased respiratory rate, intercostal retraction, and stridor which is most noticeable on expiration.[24,25] Symptoms worsen with agitation and respiratory infections. Apneic spells may occur. Fluoroscopy may be of diagnostic help but bronchoscopy (preferably rigid) is the key diagnostic technique, showing tracheal narrowing from front to back, indistinct tracheal rings, and expiratory collapse of distal trachea and main bronchi (Figure 6-11 and Figure 3 [Color Plate 12]). Symptoms of primary tracheomalacia often clear by the second year of life, with stability of the trachea then seen on bronchoscopy. In severe cases, prolonged stabilization of the airway is obtained by tracheostomy. Growth of cartilage eventuates in recovery in most cases, in 2 or 3 years. In others, silicone stents (Y stent for lower trachea and main bronchi) can provide long-term patency. Expandable stents are not advisable because of possible permanent tracheal injury and growth problems.

FIGURE **6-10** *Anatomic relationships with anomalous left pulmonary artery sling.* A, *Anterior view showing the aortic arch and ligamentum arteriosum. Note position of vagus and recurrent laryngeal nerve.* B, *Transverse-sectional view of the left pulmonary artery sling behind the trachea. A = aorta; BC = brachiocephalic artery; E = esophagus; LCC = left common carotid; LPA = left pulmonary artery; LS = left subclavian artery; PA = pulmonary artery; T = trachea; V= vagus nerve.*



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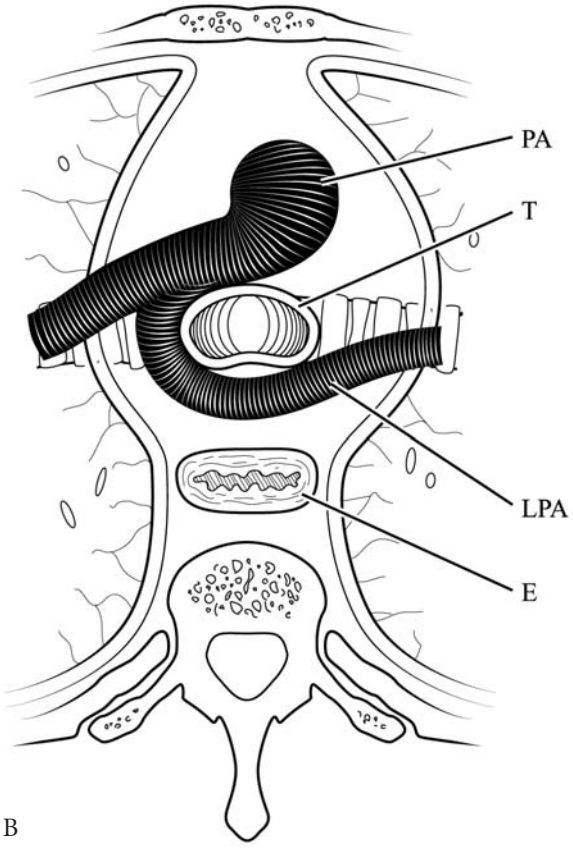


B

Focal malacia may also occur in relation to a widened membranous wall or residual pouch after repair of TEF and esophageal atresia (Figure 6-12). Defective cartilaginous rings may also be found at this level. Filler and colleagues considered this association to be the most common cause of tracheomalacia in infants, although precise explanation is lacking.[26] If severe collapse follows TEF repair, aortopexy may have to be considered.[27] Since gastroesophageal reflux can also be present, antireflux surgery may be needed. In a rare case, agenesis or hypoplasia of the right lung may result in mediastinal displacement and rotation severe enough to result in compression of the remaining bronchus at its origin, which is analogous to postpneumonectomy syndrome (see Chapter 15, "Tracheobronchial Malacia and Compression").[28,29]

When a short segment of malacic trachea accompanies pulmonary artery sling rather than segmental congenital stenosis, it may be managed by tracheopexy (or aortopexy) in conjunction with division and reimplantation of the anomalous left pulmonary artery.[30]

Laryngomalacia quite often causes inspiratory, fluttering stridor in the newborn, but usually corrects itself in 1 or 2 years, although it may require tracheostomy—which in turn may lead to later tracheal stenosis. Since this book does not pretend to give comprehensive coverage of laryngologic problems, no more will be mentioned of congenital laryngeal lesions such as absence or deformities of laryngeal cartilages, cysts and laryngoceles, congenital nerve paralysis, or of hemangioma and lymphangioma of the larynx, the latter an extension of cervical cystic hygroma.

Tracheobronchomegaly (Mounier-Kuhn disease) appears to be of congenital origin, but most patients have not become overly symptomatic until midlife.[31,32] Then, symptoms can often be traced back to youth. It has also been discovered in children. The condition is very rare and thought possibly to be due to absence

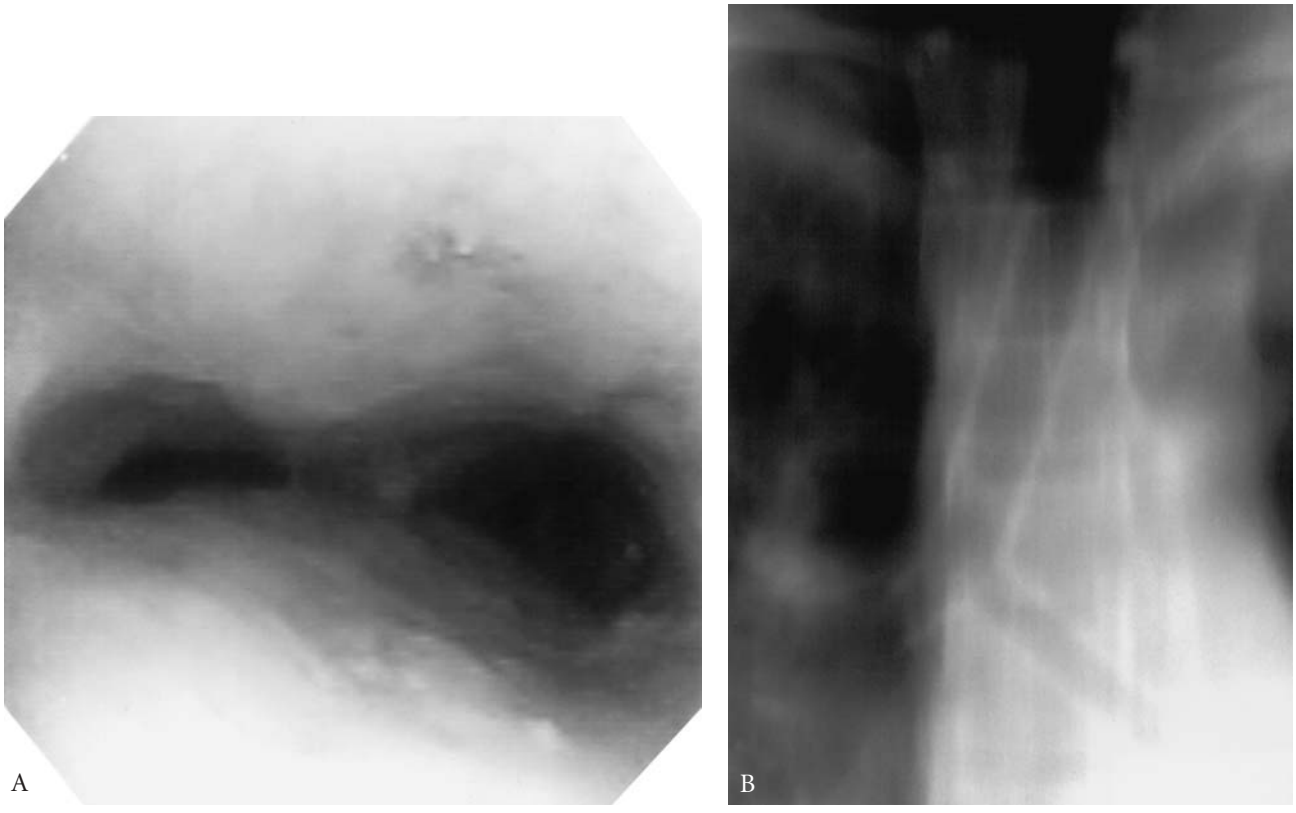


FIGURE **6-11** *Congenital tracheomalacia causing difficulty early in life. Malacia of the lower 45% of the trachea and of the left main bronchus was identified at 6 months. At age 12, a 12 mm silicone Y stent was placed to allow the patient to be more active. A 14 mm Y stent was fitted at age 15. Minor granulation tissue in the left main bronchus was removed and that limb of a new stent was shortened at age 19. The stent continues to be well tolerated. Resection and primary reconstruction would be too hazardous. The lack of visible cartilage discourages posterior wall splinting, and the location deters the use of external circumferential splinting.* A, *Bronchoscopic view at carina. Note the absence of a visible ring structure in the anterior tracheal wall. The left main bronchus shows collapse. The right maintains its patency.* B, *Tomogram demonstrating lower trachea and left main bronchus, held patent by a silicone Y stent. Also, see Figure 3 in the Tracheobronchial Endoscopic Atlas (Color Plate 12).*

of the trachealis muscle. The anterior tracheal wall may become indented as the rings fold backward (see Chapter 15, "Tracheobronchial Malacia and Compression").

Vascular rings are associated with tracheal and esophageal compression and, if of long enough duration, with tracheomalacia. Tracheal compromise is seen most commonly with double aortic arch, right aortic arch with retroesophageal left subclavian artery and left ligamentum arteriosum, and right arch with mirror image brachiocephalic artery and left ligamentum (Table 6-1) (Figure 6-13). The multiple patterns of vascular rings that occur have been well described and will not be cataloged here again.[33,34] Right aberrant subclavian artery from left aortic arch may be associated with dysphagia due to esophageal compression (*dysphagia lusoria*), but infrequently causes tracheal symptoms. Aneurysm of an aberrant artery late in life, however, can cause tracheal compression.

In infants, severe respiratory symptoms may result from vascular rings, with stridor, "crowing," and repeated respiratory infections. Dysphagia and aspiration may occur. Diagnosis was traditionally made by barium esophagogram. However, vessels are now identified by computed tomography (CT) with contrast or by magnetic resonance imaging (MRI). Division of the ring and ductus or ligamentum arteriosum plus excision of a Kommerell's diverticulum, if present, usually serves to correct the basic constriction.[35–39]

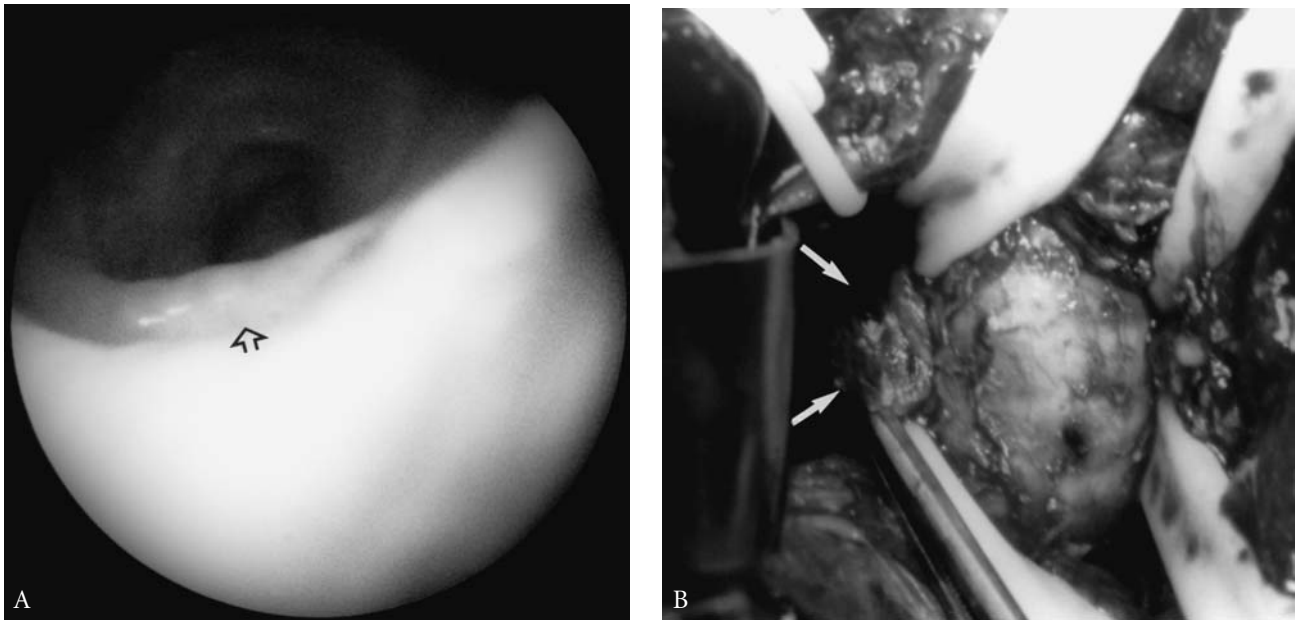
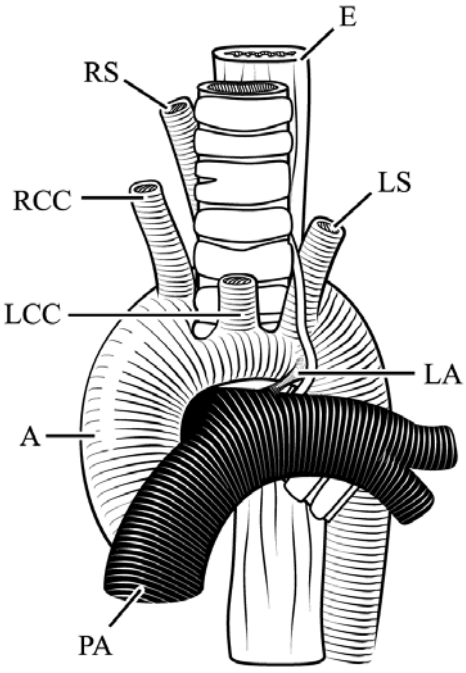


FIGURE **6-12** *Symptomatic tracheal diverticulum remaining after repair of a congenital tracheoesophageal fistula. Although the cartilaginous rings were not malacic, the combination of a patulous membranous wall and a diverticular pouch created obstruction.* A, *Bronchoscopic view of superior* (arrow) *and inferior lips of the wide-mouthed diverticulum, both of which extend obliquely across the field of view. The distal trachea is above and the patulous membranous tracheal wall below the slit-like mouth of the diverticulum* (arrow). B, *Diverticulum exposed by sternotomy. Penrose drains encircle and retract the trachea, allowing the posterior diverticulum to protrude* (arrows). *Forceps point to the diverticulum. A vascular loop encircles the brachiocephalic vein. The diverticulum was excised and the membranous wall was closed in a linear fashion, pulling the splayed rings into normal configuration to provide a normal tracheal lumen. Symptoms were relieved.*

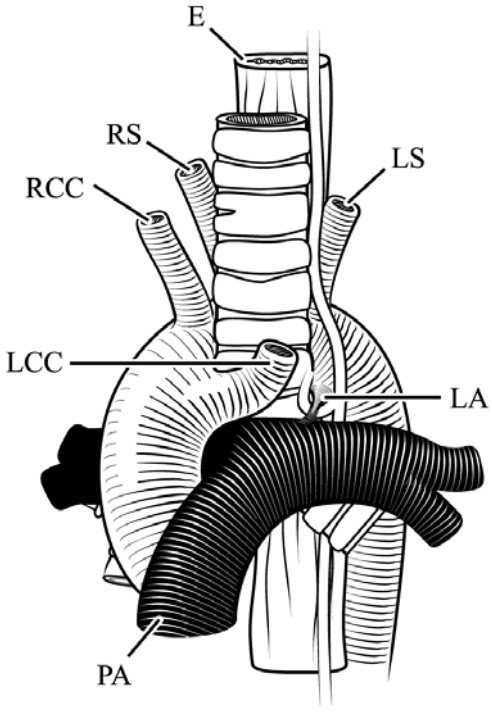
An aberrant subclavian artery may be best divided and reimplanted or possibly just divided in an infant. Continued tracheal obstruction after surgery may be due to malacia and require intubation or splinting. This must be differentiated from residual obstruction due to an unresected aortic diverticulum or continued vascular compression, as described below.

Table 6-1 Congenital Vascular Rings Which Obstruct Trachea

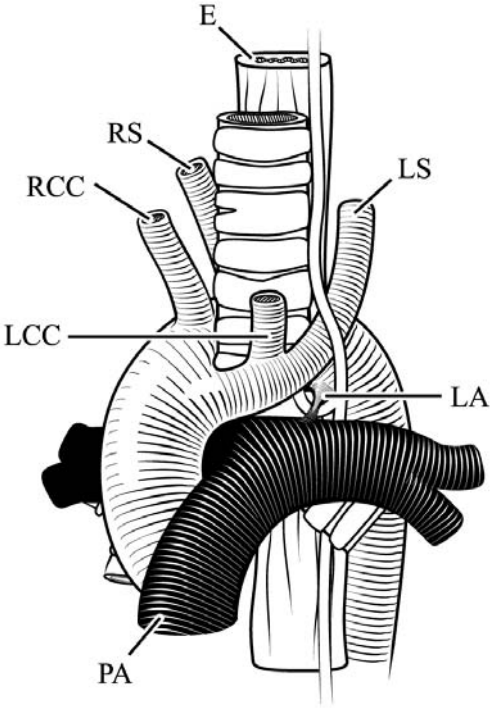
	Anomaly		
	Double aortic arch	*Right arch, retroesophageal Left subclavian artery Left ligamentum arteriosum*	*Right arch, mirror image brachiocephalic artery Left ligamentum arteriosum*
Mayo Type[33]	IA	IIIB	IIIA
Reference			
33	19	4	4
34	36	13	3
35	17	1	18
36	61	34	18
37	11	11	4
Total	144	63	47
%	57	25	18



A



B



C

FIGURE **6-13** *Principal vascular rings which may compress trachea.* A, *Mayo type IA: double aortic arch, dominant right arch.* B, *Mayo type III B1: right aortic arch, left ligamentum arteriosum, retroesophageal left subclavian artery.* C, *Type IIIA: right aortic arch, left ligamentum arteriosum, mirror image left brachiocephalic artery. Ligamentum may connect to the brachiocephalic artery or to the upper descending aorta. Adapted from Stewart JR et al.*[33] *A = aorta; E= esophagus; LA = ligamentum arteriosum; PA = pulmonary artery; RCC, LCC = right, left common carotid arteries; RS, LS = right, left subclavian arteries; V = vagus nerve.*

A variant of vascular right tracheobronchial compression has been observed in a small number of patients with *right aortic arch* and *a right descending aorta*, ligamentum arteriosum, and anomalous left subclavian artery with a Kommerell's diverticulum at its origin (Figure 6-14). The aortic arch may be high, and bends sharply in a "hairpin" configuration as it descends either to the right of or directly over the vertebral spine, leaving little space beneath the arch for the airway (Figure 6-15). All patients with this anomaly had chest wall abnormalities, which markedly narrowed the space between the sternum and vertebrae. Previous division of the ligamentum, excision of the aortic diverticulum, and division of the anomalous subclavian artery in some patients, in different combinations elsewhere, had failed to relieve airway compression. Two patients who had failed to improve after the procedures mentioned above, obtained relief only after division of the aortic arch with pexy of the ends following bypass grafting from the ascending to descending aorta (see Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea"). In a third patient, division of ligamentum, excision of the aortic diverticulum, transposition of the aberrant subclavian, and arch aortopexy sufficed. This was an initial corrective operation without prior surgery. If, however, severe malacia has already developed in the compressed airway, additional measures such as tracheal splinting or resection may be needed. A fourth patient with such malacia, after multiple prior operations, needed a tracheobronchial stent. These problems appear to be highly individual. The general principle of correcting cardiovascular lesions as early as feasible may offer the additional advantage of preventing severe tracheomalacia.

Backer and colleagues reported 8 children with a similar anomaly, who had recurrent respiratory symptoms and/or dysphagia, despite prior division of a left ligamentum arteriosum in 7 of the children.[40] All had a Kommerell's diverticulum still present. Resection of the diverticulum and reimplantation of a retroesophageal left subclavian artery into the left carotid artery relieved tracheal compression. The aorta is presumed to have descended on the left in these patients and no chest wall deformity was described. Konstantinov has noted, as we did, that symptoms are not always relieved by this limited procedure, although he found this to occur primarily with an encircling right aortic arch which descended on the

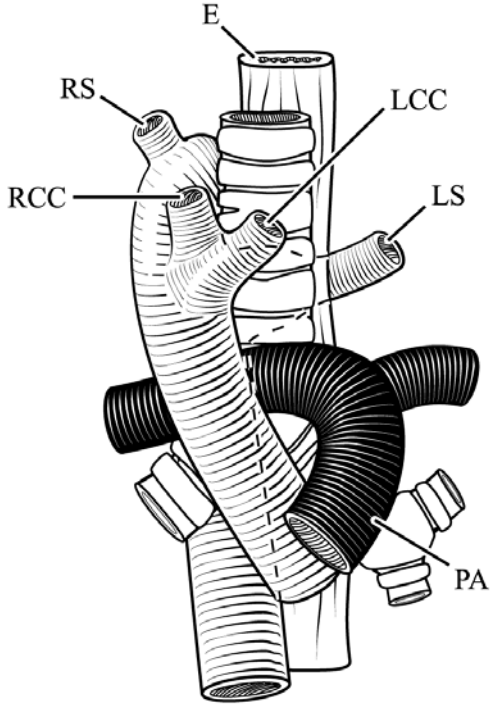


FIGURE **6-14** *Right aortic arch, right descending aorta, aberrant left subclavian artery, and Kommerell's diverticulum. The airway compression is not necessarily relieved by excising the aortic diverticulum and transplanting the aberrant subclavian because of the narrow space between ascending and descending aorta. Left ligamentum arteriosum is usually present to the diverticulum or LS. E= esophagus; PA = pulmonary artery; RCC, LCC = right, left common carotid arteries; RS, LS = right, left subclavian arteries.*

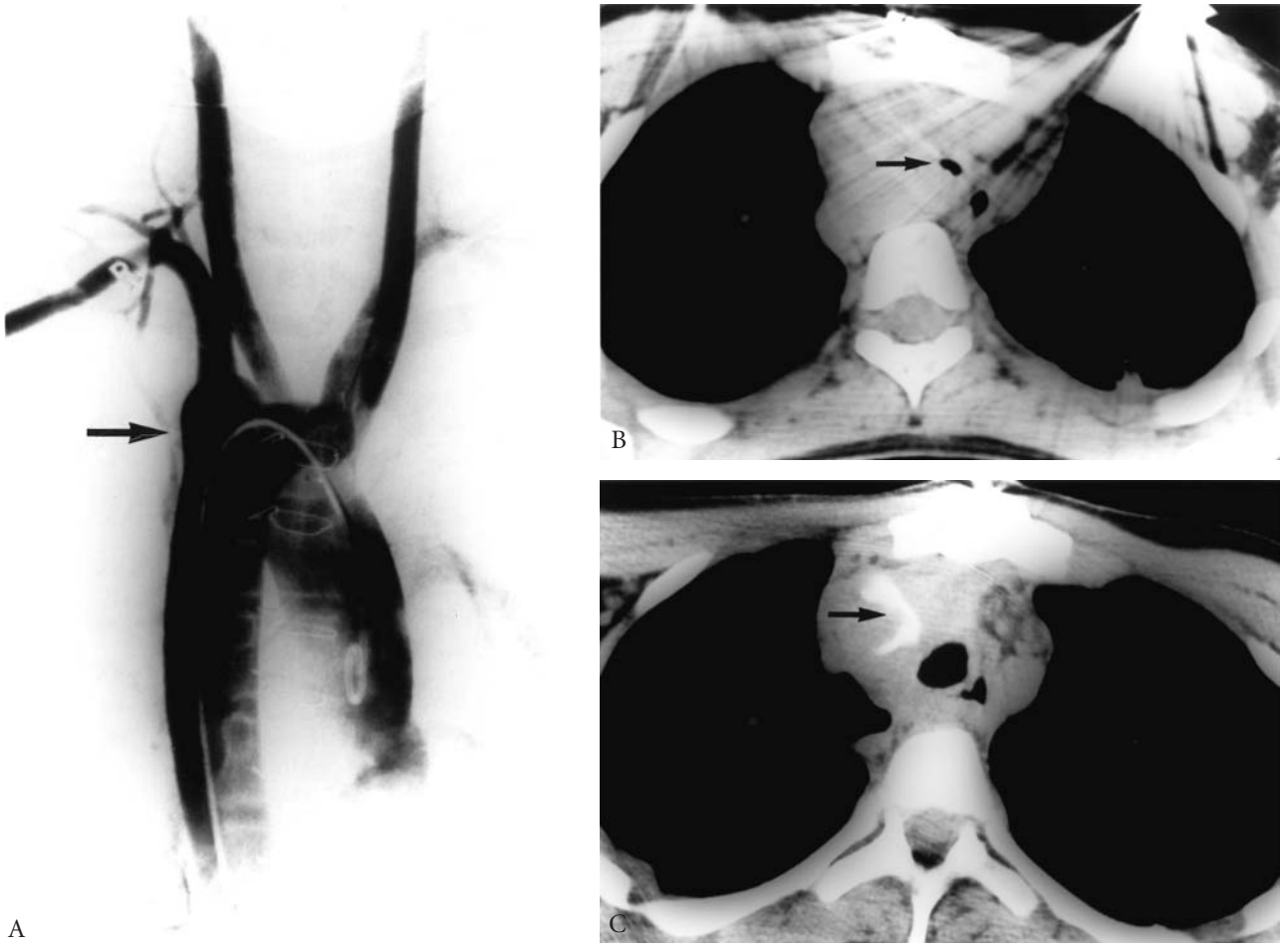


FIGURE **6-15** *Tracheal obstruction by "hairpin" aortic arch: right aortic arch, right descending aorta, anomalous left subclavian artery originating from aortic diverticulum.* A, *Aortagram showing a high and narrow arch with a large Kommerell's diverticulum* (arrow) *from which the left subclavian artery originates.* B, *Computed tomography (CT) scan showing the trachea* (arrow) *compressed and displaced by the right aortic arch. Note the short distance between the sternum and vertebral bodies.* C, *Postoperative CT after aortic bypass, division of arch, and pexy of ascending arch by a Goretex sling* (arrow) *passed around an anterior rib. The tracheal lumen is widely opened.*

left.[41,42] Intraoperative bronchoscopy can monitor the degree of correction achieved by successive procedures, beginning with lesser ones, as in one of our patients cited above.

Compression of the trachea by a *prominent innominate artery* has been relieved by pexy of the artery and aortic arch to the sternum, leaving the trachea attached so that it is pulled forward and suspended.[43,44] The validity of this diagnosis has been questioned, but there are instances of obstruction relieved by surgery.[45] The artery has been divided and reimplanted more proximally on the aorta to successfully remove the point of tracheal compression and also the possibility of recurrence following suspension.[46,47]

Acquired Lesions

Postintubation stenoses are preponderant among acquired airway lesions in children.[48] Unfortunately, these injuries include laryngotracheal stenoses resulting from the preferential use in this age group of endotracheal tubes, often uncuffed, for ventilatory support. Laryngotracheal stenosis is much more difficult to correct than a purely tracheal lesion. In a study of 854 newborns who required intubation and ventilation (1979–1983), Marcovich and colleagues discovered a 0.6% incidence of severe subglottic stenosis, where

uncuffed, small diameter tubes had been used nasotracheally.[49] As in adults, the length of the period of intubation does not seem to be critical, although the period of risk is obviously greater with prolonged intubation. The reported incidence of injury has varied widely, and it remains difficult to define specific conditions that produce sufficient erosion to cause stenosis in one patient and not in another (see Chapter 11, "Postintubation Stenosis").

Tracheostomy, used either by choice to limit the period of endotracheal intubation or as treatment for laryngeal or subglottic obstruction resulting from endotracheal intubation, may produce its own stenotic or other obstructive lesions in infants and children. The uses of carefully placed vertical incisions in cartilage and specifically designed pediatric uncuffed tracheal tubes for tracheostomy in the infant have reduced the incidence of stomal tracheal stenosis (see Chapter 10, "Tracheostomy: Uses, Varieties, Complications"). Obstruction also occurs, although less often, by compression deformity of the soft tracheal wall of infants, just proximal to the stoma, due to pressure by the curvature of the tube. This is correctable by use of T tubes, which, however, may be difficult to care for in infants due to their tiny diameter. Overall, nasotracheal intubation has more often been selected for prolonged ventilation in these age groups.

A small number of *postintubation tracheoesophageal fistulae* have been seen, and, fortunately, only a rare tracheo-innominate artery fistula (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula" and Chapter 13, "Tracheal Fistula to Brachiocephalic Artery"). Post-traumatic stenosis from external injury is uncommon except in older children. Stenosis due to inhalation burns is difficult to treat because of the basic pathology of burned tissue (see Chapter 9, "Tracheal and Bronchial Trauma").

Tumors, benign or malignant, occur uncommonly. In 198 primary tracheal tumors, only 4 were in patients under the age of 10 years, and 8 occurred in patients between 11 and 19 years of age.[50] These included granular cell tumor, fibrous histiocytoma, neurofibroma, mucoepidermoid tumor, carcinoid (most common), adenoid cystic carcinoma, and rhabdomyosarcoma. Other tracheal tumors noted in childhood were fibrosarcoma, squamous cell carcinoma, hemangiopericytoma, and chondroma. "Congenital" tumors compressing the trachea or bronchi included cystic hygroma, hemangioma, teratoma, and thymic cyst.[51]

Recurrent respiratory *papillomatosis* due to the human papilloma virus causes wart-like excrescences in the larynx and trachea, in children as well as in adults. Patients present with hoarseness, a weakened cry, cough, respiratory infections, choking, and obstruction of progressive severity. The disease is vertically transmitted in children. Present treatment is repeated laser vaporization. Multiple antiviral adjuvants have been tried, including interferon. Although most children enter spontaneous remission, recurrences do emerge. Malignant degeneration has rarely been reported in children.[52]

Hemangiomas of the airway are frequently associated with cutaneous hemangiomas of the chin, lips, mandibular region, and neck.[53] In the subglottic region, proliferation produces hoarseness and stridor. Respiratory failure can follow, often at between 6 to 12 weeks of age. Management includes corticosteroids and tracheostomy since the lesions usually regress spontaneously. Cryotherapy and laser therapy have been used, but scarring and stenosis can follow, especially if the subglottic lesion is extensive.

Rarely, an *extrinsic lesion* such as a bronchogenic cyst or mediastinal tumor will produce compression of the trachea or secondary malacia in early childhood.[54] For example, apparent malacia over a long tracheal segment, seen in a 2½-year-old child, proved to be due to compression by a large thymic cyst (Figure 6-16). Because of prolonged compression of the trachea by the cyst, which had apparently been present since birth, removal alone did not immediately restore a satisfactory airway. Splinting with a tracheostomy tube was required until the cartilages became firm with further growth. Rarely, a bronchogenic cyst at the carina may produce compressive symptoms in children. Adenocarcinoma was discovered in the base of an obstructing cyst in an 8-year-old girl, and curative carinal resection was secondarily performed (Figure 6-17).

Obstruction of the trachea or bronchi has resulted from *infection* with tuberculosis, histoplasmosis, diphtheria, rhinoscleroma, and syphilis (see Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic,

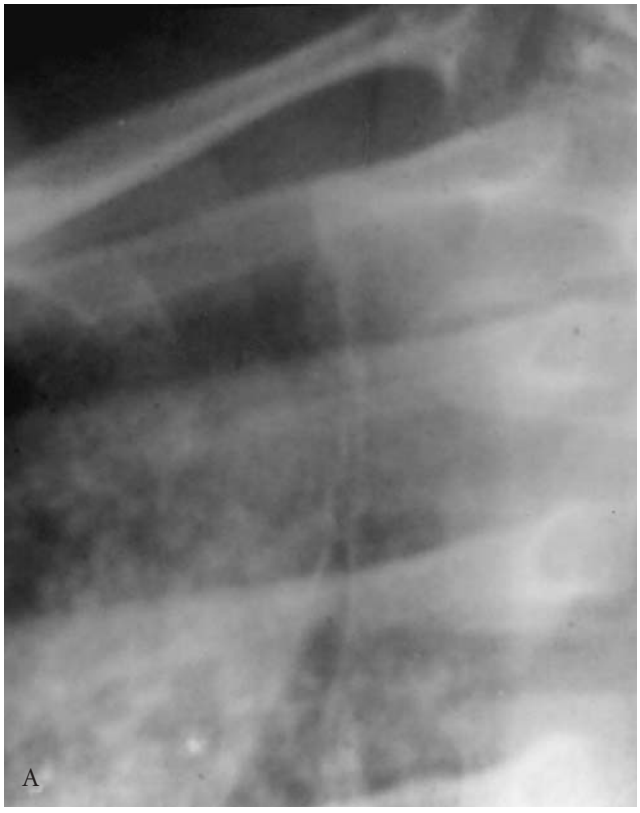


FIGURE **6-16** *Thymic cyst compressing the trachea in a 2½ -year-old boy.* A, *The lateral roentgenogram demonstrates tracheal deformation.* B, *The cyst as it was being extracted from the mediastinum via cervical incision.*

and Miscellaneous Tracheal Lesions"). All except histoplasmosis (in adults) are vanishingly uncommon in developed countries. Bulky tuberculous enlargement of mediastinal or hilar lymph nodes can compress and erode the relatively soft trachea or bronchi of children.[55] Subcarinal nodes are commonly involved.

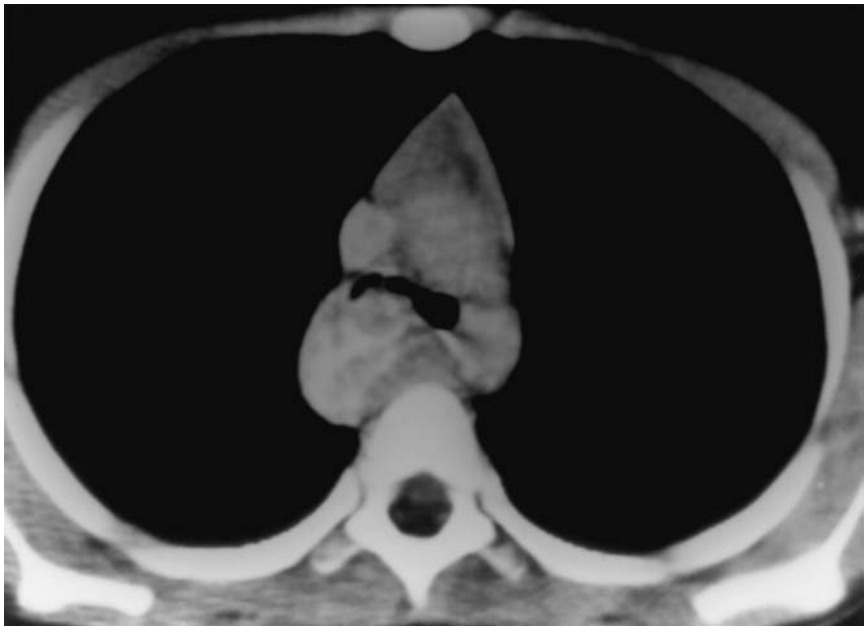


FIGURE **6-17** *Computed tomography scan in an 8-year-old girl, showing a bronchogenic cyst deforming the carina, but with irregular protrusion into the lumen, which proved to be adenocarcinoma in the base of the cyst. After secondary carinal resection, she remained disease free in 16-year follow-up.*

Sequelae include pulmonary collapse or hyperinflation, airway perforation, bronchostenosis, and caval or esophageal obstruction. Glandular enlargement can occur despite antituberculosis drug treatment, and indeed has been observed to likely increase on initial treatment.

Surgical treatment is reserved for symptomatic patients. Acute obstruction may demand emergent surgery. Enucleation and curettage of obstructing nodes and of caseous material, suture and muscle flap repair of a bronchial opening, and conservation of all but destroyed lung are effective.[56] Lymph node excision is avoided in order to minimize complications. Late fibrous bronchostenosis is best treated by segmental bronchial resection and anastomosis with preservation of the lung, unless it is irretrievably damaged. Corticosteroids are unlikely to improve gross airway obstruction.

Pneumonectomy has become a rare procedure in childhood, so that *postpneumonectomy syndrome*, once thought to occur principally in childhood, is now seen largely in adults (see Chapter 15, "Tracheobronchial Malacia and Compression").

Assessment

Clinical Findings

The diagnosis of *congenital tracheal stenosis* and other obstructive anomalies is based on a high degree of suspicion in infants and children with respiratory distress. Inspiratory and/or expiratory stridor may be present, accompanied by retraction. Recurrent or persistent cough and exercise intolerance occur. There may be a history of respiratory difficulties of lesser intensity since birth, or shortly after birth, or of repeated and stubborn respiratory infections. Strangely, dyspnea may be episodic. Cyanosis and apneic episodes may occur. In some cases, difficulty in intubation had led to the diagnosis. Late manifestations of congenital stenosis may represent the child's respiratory demands outpacing the ventilation permitted by the stenotic airway. Only then may a clinical history be retrospectively traced to a much earlier time. Other obstructive lesions are manifest in similar ways.

The clinical presentation of congenital TEF is described in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula." Caution about delayed manifestation of small congenital H-type tracheoesophageal fistulae and bronchoesophageal fistulae must be repeated.

Acquired stenosis due to intubation for ventilation is signaled by shortness of breath on exertion and stridor in the wake of a history of intubation, usually for respiratory support, with or without tracheostomy. Children diagnosed with "asthma," who fail to respond to treatment, must be suspected of an organic airway lesion. *If the child has been previously ventilated, the working diagnosis should be airway stenosis, until proved otherwise.*

Imaging

Air tracheograms and careful fluoroscopy provide precise information about the presence of an anomaly, its location, and type (see Chapter 4, "Imaging the Larynx and Trachea") (Figure 6-18). Contrast media are usually unnecessary and may cause complications when used in tiny airways. Tomograms offer specific additional detail, but in many hospitals are no longer available of useful quality. CT scanning provides precise information on the cross-sectional area and extent of lesions, with three-dimensional reconstruction available. An advantage of spiral CT is the rapidity of examination, which is important in small children. MRI offers similar information (Figure 6-19) and is especially useful to delineate associated cardiovascular anomalies. The role of cardiac catheterization to delineate such lesions has thus diminished. MRI may be difficult to accomplish in infants and small children. Angiography is also used less often, but it still provides a "gold standard" for precise and complete delineation of vascular anomalies. Echocardiography is also very helpful to identify cardiovascular anomalies. Left pulmonary artery sling must be identified preoperatively

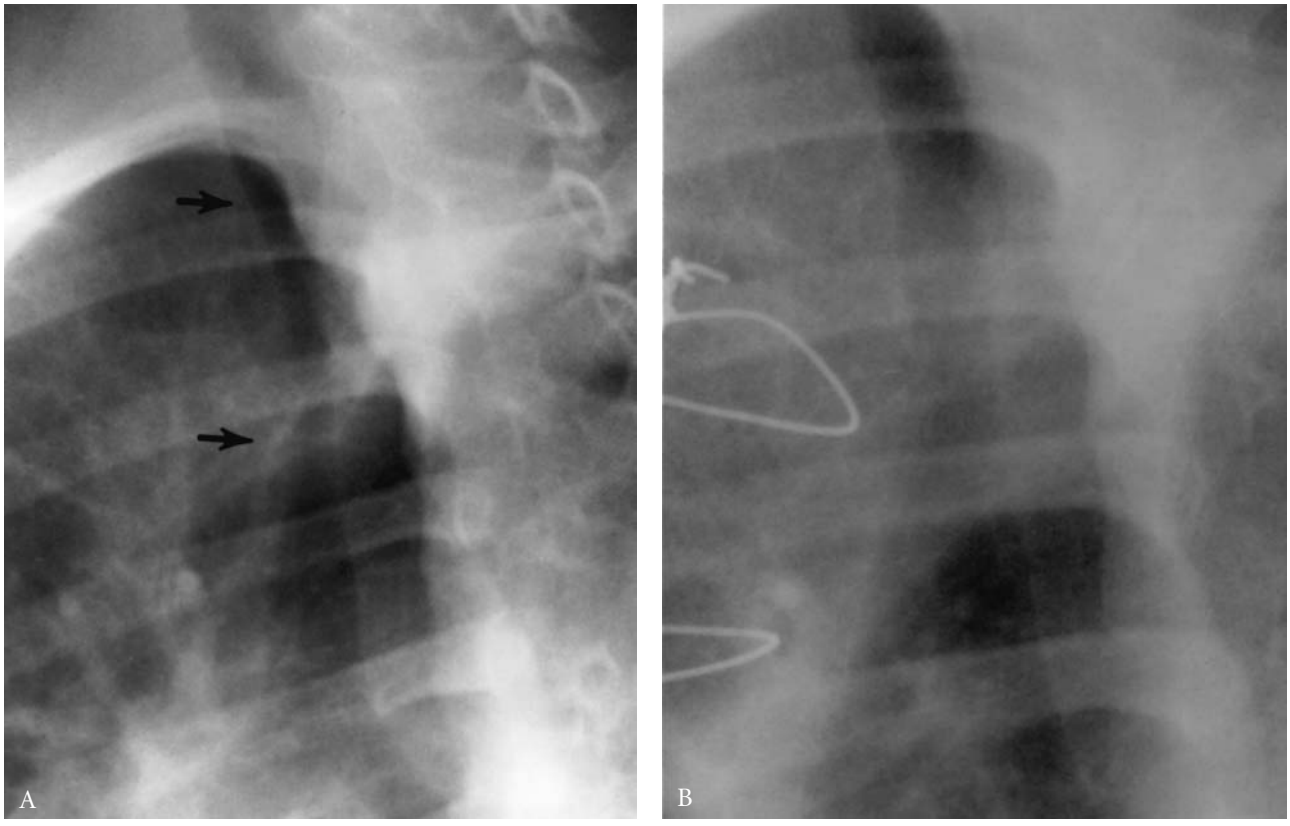


FIGURE **6-18** *Imaging of congenital tracheal stenosis in an 11-year-old boy.* A, *The simple oblique roentgenogram shows the location* (arrow) *and length of stenosis as well as the length of normal trachea from larynx to carina—all critical information for the surgeon.* B, *Postoperative view of the same patient. Note the increased acuteness of the carinal angle.*

because its approach and anesthesia can be different from that in a patient who has an isolated congenital tracheal stenosis. Barium swallow is still an important diagnostic method in identifying a vascular ring or aberrant subclavian artery.

If a child has a tracheostomy in place, it is preferable to obtain initial roentgenograms with the tube temporarily removed, if the patient can and will tolerate removal. A physician who is able to replace the tube promptly must be in attendance, with adequate preselected equipment on hand. Fistula to the esophagus requires fluoroscopy, with careful administration of a small volume of contrast medium to judge the exact location and approximate size of the communication. If a pulmonary artery sling has been previously corrected by reimplantation, a perfusion scan or pulmonary angiogram should be considered, since this vascular anastomosis in infants has a tendency to stenose and, at best, perfusion will be diminished on the left side. The information could be critical since operative correction of a tracheal stenosis might otherwise be conducted with left main bronchial intubation, thus ventilating an unperfused or underperfused lung.

Endoscopy

Careful use of a flexible pediatric bronchoscope can clarify much about a lesion. The bronchoscope should not be passed into a tightly stenotic lesion in order to avoid causing edema and inflammation, which might precipitate acute obstruction. Indeed, in a significantly symptomatic child, in whom the presence of stenosis is already known from radiologic study (even if definition is not wholly complete), bronchoscopy is usu-

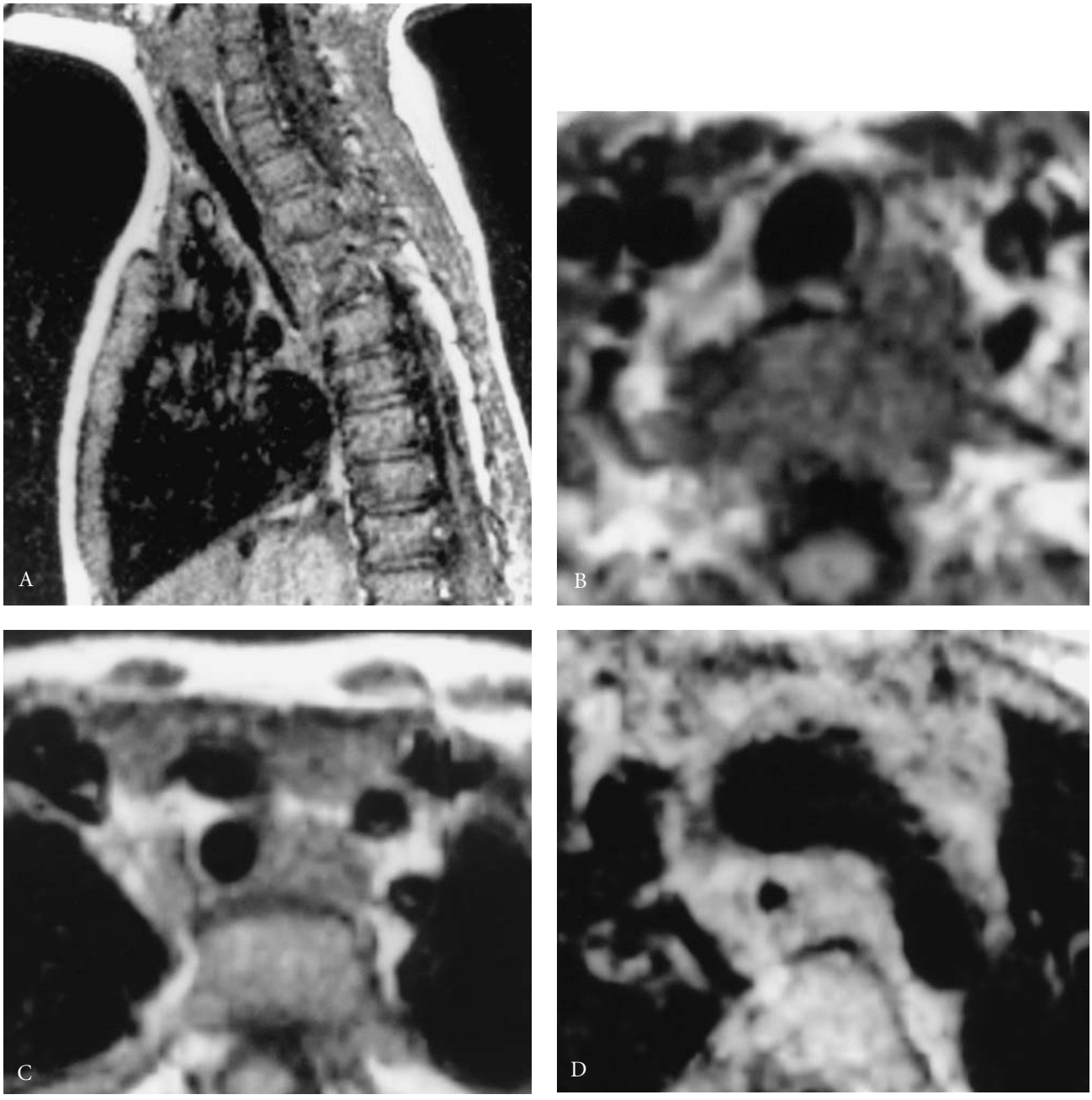


FIGURE **6-19** *Congenital stenosis in a 3½-year-old boy.* A, *Sagittal magnetic resonance study showing narrowed supracarinal segment.* B, C, *and* D *clarify tracheal diameters in a normal segment, at the beginning of cartilaginous "O" rings and at maximum stenosis.*

ally best deferred to the time of planned surgical repair. This also applies to less critical patients. Surgeons must always do the bronchoscopy themselves, irrespective of any prior examinations.

Definitive bronchoscopy, ideally performed just prior to a planned surgical procedure for correction of the lesion, is best accomplished with rigid Storz ventilating pediatric bronchoscopes. The 3.5 mm rigid bronchoscope (OD 5.7 mm) will not pass through a tiny stenosis. However, either a flexible pediatric bronchoscope (2.7 mm) or a long telescope (OD 2 mm) allows a more distal examination. These may be inserted through a larger rigid (ventilating) bronchoscope seated proximally, or through a pediatric operating

laryngoscope. Bronchoscopes should not be forced into a stenosis, nor should any attempt be made to dilate the narrowing, if it is congenital. Ventilation is maintained by intermittent placement of an endotracheal tube into the laryngoscope. Fine suction devices are necessary. Circular "O" rings of cartilage, found in many cases of congenital stenosis, are clearly visible bronchoscopically, if looked for (Figure 6-20*A* and Figure 1 [Color Plate 11]). Disordered or fused cartilages are present in congenital stenosis, but far less often (Figure 6-20*B* and Figure 2 [Color Plate 11]). As in adults, the larynx must be examined, especially in the presence of a postintubation lesion. The glottis is visualized in passing with the rigid bronchoscope and telescope. However, the pediatric Holinger laryngoscope, used in conjunction with a telescope, provides a superior view of laryngeal structures. If any complexity is identified, consultative examination by a pediatric otolaryngologist is advisable. Cooperation in the initial endoscopic examination of these patients is beneficial for all.

Tracheoesophageal fistula is identified in the membranous wall of the trachea (Figure 6-21). Instillation of methylene blue-colored saline into the esophagus via a high placed nasogastric tube may conclusively identify a small H fistula. Confusion can result if a large volume of dyed saline refluxes above the cricopharyngeus and spills over the arytenoids into the airway. Esophagoscopic identification of a fistula may be more difficult unless the fistula is large or distended by a cuffed tube in the trachea.

Treatment and Results

Healing of the Juvenile Trachea: Growth and Tension

The question of whether anastomosis performed in the juvenile trachea will grow adequately has been answered positively. As an example, Maeda and Grillo experimentally demonstrated that tracheal anastomosis in puppies resulted in some narrowing at the anastomotic site in the adult, caused by infolding of adjacent rings and restriction of growth by cicatrization of the membranous wall (Figure 6-22).[57] At the anastomotic site, the average growth was 82% of normal sagitally, and 75% coronally, values equivalent to

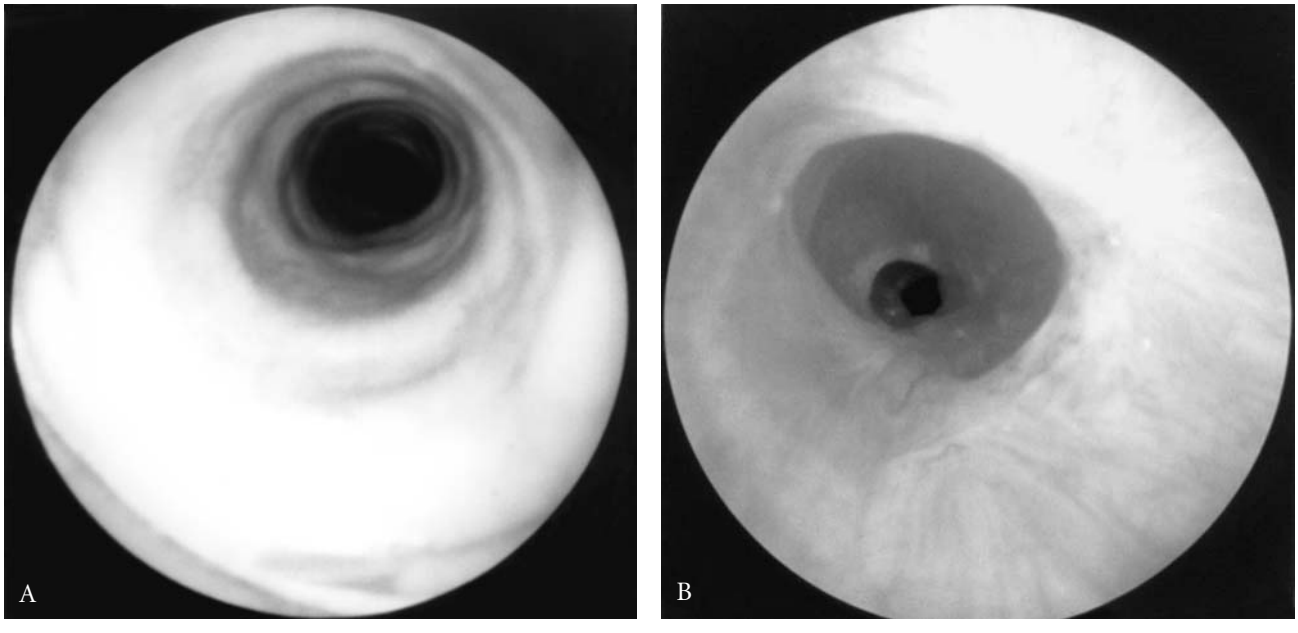


FIGURE **6-20** *Bronchoscopic observations in congenital stenosis.* A, *Complete cartilaginous "O" rings are easily identified visually.* B, *Very rarely, irregular cartilaginous walls such as in this case are seen in a stenotic segment. Resected at age 9. See also Figures 1 and 2 in the Tracheobronchial Endoscopic Atlas (Color Plate 11).*

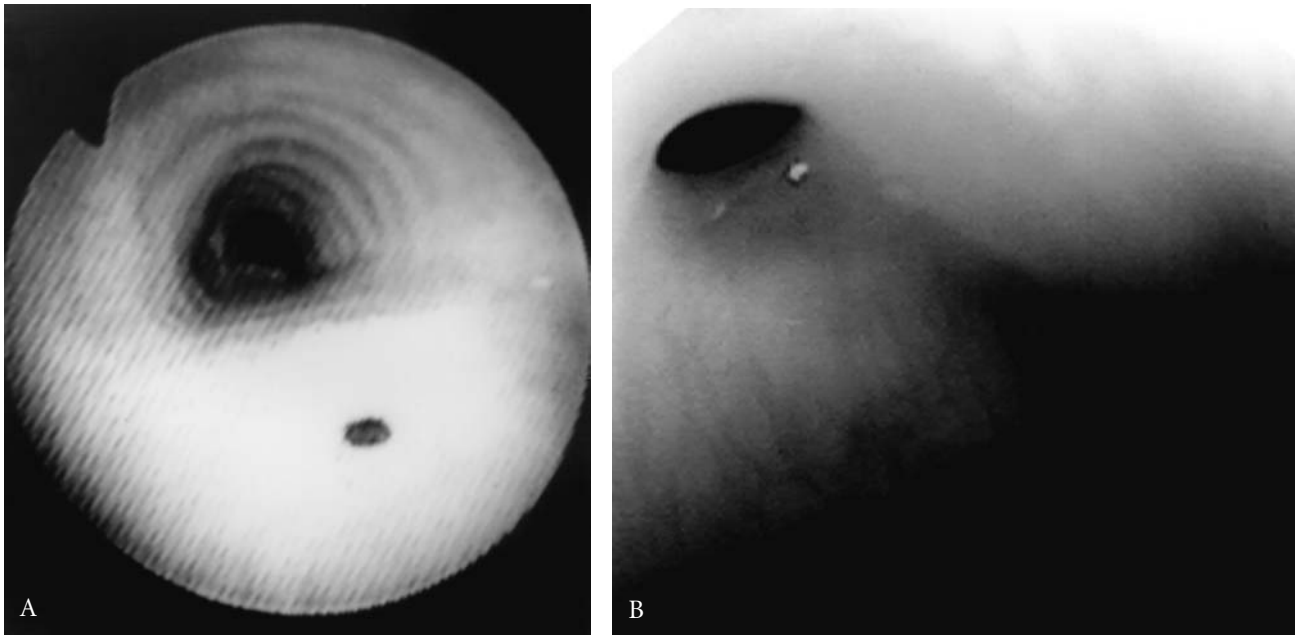


FIGURE **6-21** A, *Congenital H-type tracheoesophageal fistula, visualized bronchoscopically posteriorly.* B, *Esophageal opening of the same fistula, on the anterior wall. The dark area below is the esophageal lumen. (Courtesy of Dr. Hon Chi Suen.)*

20% stenosis of the tracheal lumen. Tension on the anastomosis caused by resection of progressively longer segments of trachea led to greater anastomotic narrowing when performed in puppies rather than in adult animals.[58] Puppies were more susceptible to tension than adult animals. Levels allowing safety from disruption were 1,000 g in puppies and 1,750 g in adult dogs. The range of permissible resection was about half that of the adult, about 25% of the entire length of the juvenile trachea. Within permissible tension limits, fully adequate airways resulted (Figure 6-23). The use of a few spaced sutures from tracheal rings proximal and distal to the anastomosis, to reduce tension on the suture line, lessened postoperative narrowing.[59] This is effectively accomplished clinically by our present use of lateral traction sutures (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). Other laboratory work has generally confirmed this postanastomotic tracheal growth, with variations probably being due to experimental design.[60–62]

As experience with tracheal resection and reconstruction in infants and children increased, clinical results confirmed these expectations of anastomotic growth.[63–66] Couraud and Monnier and their colleagues observed growth following similar laryngotracheal procedures.[67,68] This contrasted with a diminished tracheal diameter and the need for later revisions or resection in children treated endoluminally or by plastic procedures. Further evidence of lesser tolerance of anastomotic tension in the juvenile trachea has been adduced, confirming the desirability of confining resection in children to 25 to 30% of the tracheal length.[69]

Growth of tracheal cartilage appears to occur continuously on the convex or outer side of a ring, with resorption taking place on the concave surface, without identifiable growth centers.[70] Personal serial bronchoscopic observations of unoperated congenital stenosis showed that the stenotic segment grew proportionally to the normal trachea. Manson and colleagues demonstrated growth radiologically.[71] These observations led to the expectation that growth would occur after slide tracheoplasty. Macchiarini and colleagues observed growth in an experimental model approximating slide tracheoplasty for long congenital stenosis.[72] Such growth has now been clinically confirmed by Grillo and colleagues.[73]

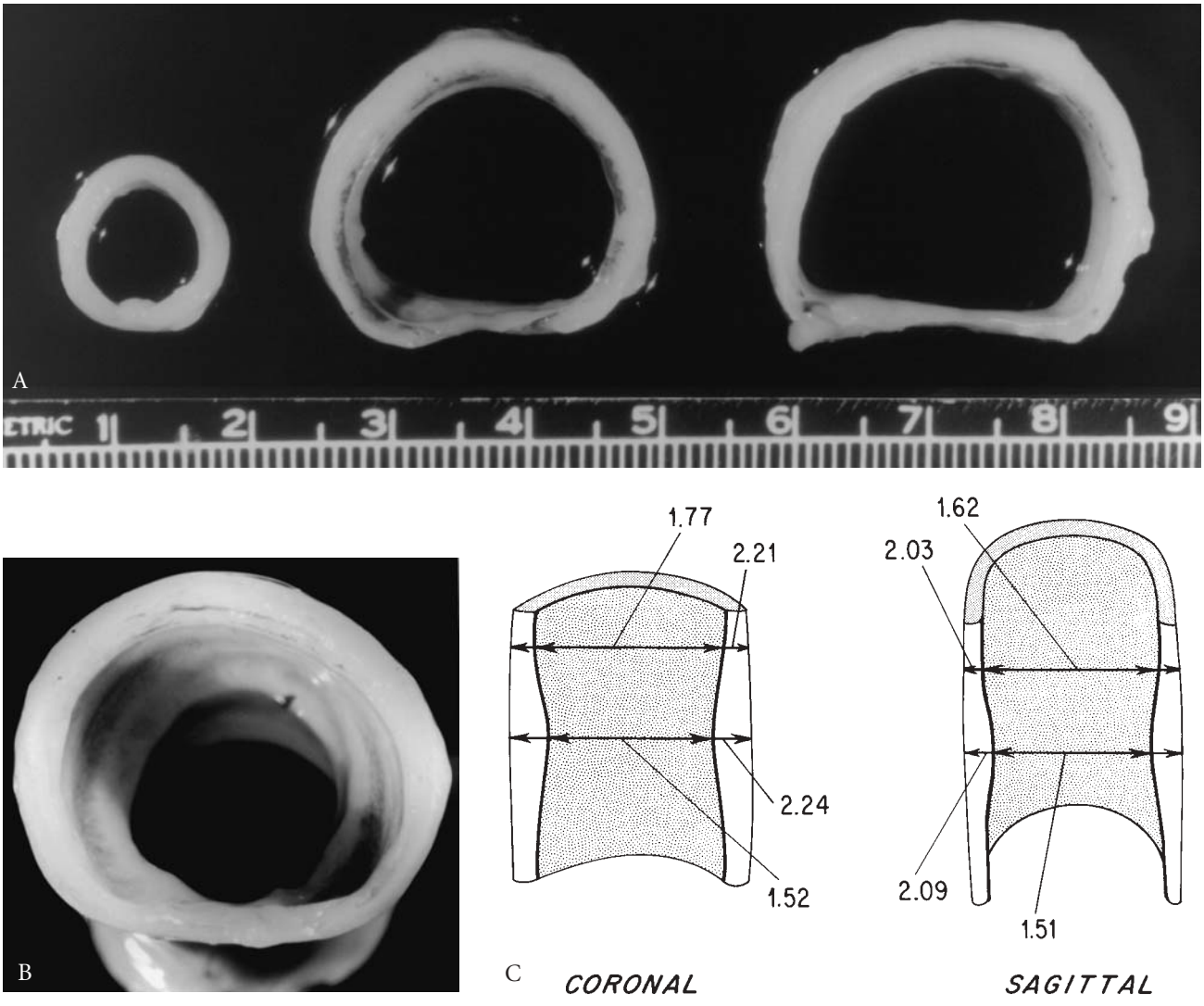


FIGURE **6-22** *Growth of trachea after experimental division and anastomosis.* A, *Differences in cross-sectional shape in a puppy, a 7-month-old dog that underwent anastomosis as a puppy, and an adult dog. Differences are minor in those of adults.* B, *Circular narrowing at the anastomotic site after full growth. The lumen remains completely adequate.* C, *Measurements of internal and external diameters and wall thickness (mm) at and above anastomoses after growth. Reproduced with permission from Maeda M and Grillo HC.*[57]

Non-Operative Treatment

Growth has also provided numerous instances where lesser acquired stenotic lesions or limited areas of malacia improved or became sufficiently corrected over time to obviate need for reconstruction. For this reason, and since anastomosis is more safely performed in larger airways, I have approached many pediatric patients conservatively, even accepting prolonged presence of a tracheostomy tube in the interim. Although a silicone T tube has many functional advantages over tracheostomy for prolonged interim treatment, the failure rate of T tubes in very small tracheae is much greater than in the adult, due to easy obstruction of the tube.[74] A particularly favorable application of the T tube is in a child with obstruction from a residual depressed flap in the anterior tracheal wall due to the tracheostomy tube lying just below. A T tube splints the deformed wall flap forward, allowing cartilage to remodel in the corrected position. The tube is later removed and the stoma allowed to close. Permanent stents of small caliber or expandable

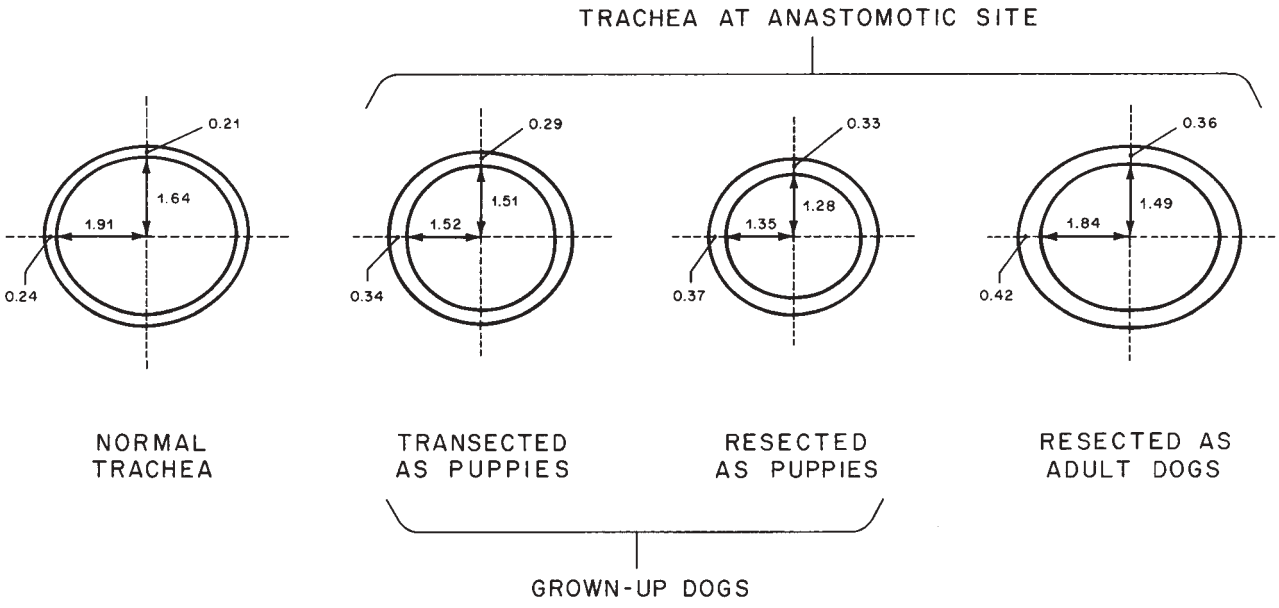


FIGURE 6-23 *Diagram of tracheal cross sections at anastomotic sites when fully grown, from puppies that were transected or resected, and in dogs resected as adults. Sagittal diameters are vertical (cm). Fully adequate airways resulted within the range of permissible resection. Reproduced with permission from Maeda M and Grillo HC.*[58]

stents should be avoided, because of their failure to account for growth, their ease of occlusion, their potential to cause additional injury, and the difficulty of removal.

Dilation of acquired stenosis, just as in the adult, provides temporary relief at best in most patients, with or without use of the laser. An exception may be web-like congenital strictures (rarely seen as acquired lesions). Kim and Hendren noted satisfactory results in selected cases with electrocautery excision of subglottic webs.[75] Congenital segmental stenosis should not be dilated since this can split the "O" rings, which are usually present.

Resection and Reconstruction of the Trachea

Increasingly, tracheal resection and anastomosis, both for acquired lesions and short congenital stenoses, has been performed with considerable success (Figure 6-24*A*). Procedures developed for adults have served well in children, but principally for acquired stenosis, since the length of most congenital stenoses precludes resection and reconstruction. Warnings about intolerance of greater anastomotic tension and danger of postoperative obstruction due to edema and secretions remain valid. Technique, as ever, must be precise and meticulous. For anastomosis in children, I prefer using interrupted 5-0 Vicryl sutures because of their ease of handling, strength, and lack of granulation formation. Conclusions from experiments purporting to demonstrate the superiority of polydioxanone monofilament sutures (PDS) over Vicryl braided sutures, were based on a scale of early histologic reaction to sutures and not on long-term results.[76] In large numbers of cases, Vicryl sutures have been shown to be as close to an ideal tracheal anastomotic suture as has yet been offered, in terms of ease of use, strength, minimal reactivity and, most importantly, absence of long-term complications such as granulomas, suture erosion into the lumen, and anastomotic separation and stenosis.[77] PDS is somewhat more difficult to handle and has no positive advantages to highlight its recommendation.

Anesthetic management by the cross-field intubation technique, similar to that used in adults, has worked extremely well in skilled hands. Some cardiac surgeons, however, used to using cardiopulmonary bypass in infants, feel that this simplifies their operative field. My principle has been to avoid possible complications from unnecessary additional procedures.

Carcassonne and colleagues published their experience in pediatric tracheal reconstruction in 1973, and Nakayama and colleagues reported reconstruction in infants and small children in 1982, updating the series in 1990 to 45 patients.[63,6678] Grillo and Zannini described the management of obstructive tracheal disease in children in 1984, including 20 who underwent resection and reconstruction: 3 for congenital stenosis, 9 for postintubation stenosis, 1 for TEF, 2 for post-traumatic stenosis, 1 for idiopathic stenosis, and 4 for primary tumors.[64] Two had carinal resections. One death occurred in the congenital group and one in the acquired group. Seventeen enjoyed good results. Gaissert and colleagues extended these observations to carinal and main bronchial reconstruction.[79] The series was updated in 2002.[69] Success of over 80% in 73 major operations was considerably less than in adults, however.

Repair of Subglottic Laryngotracheal Stenosis

When the subglottic larynx and, usually, the adjacent upper trachea is stenotic, most often due to ventilation through an endotracheal tube, correction is more difficult than when the problem is purely tracheal. A simple sleeve resection is not possible if the recurrent laryngeal nerves are to be saved, just as in the adult. Conservative measures, such as repetitive dilation, local and systemic steroids, laser or electrocoagulation ablation, and prolonged stenting, have only occasionally been successful. A host of complex operative procedures usually associated with laryngofissure, buccal, cutaneous, or cartilage grafts with stents, anterior and posterior cricoid splits, and castellated incisions, have also produced variable results. Despite failure rates of 20 to 50% in some series of such procedures, satisfactory success with the cricoid split and cartilage graft has been reported.[48,80,81] However, more recently, the effectiveness and safety of primary resection and anastomosis have been acknowledged.[82]

Single stage methods of repair (see Chapter 25, "Laryngotracheal Reconstruction") involve resection of the anterior cricoid arch and stenotic scar anterior to the posterior cricoid plate, with preservation of the posterior plate and perichondrium to protect the recurrent nerves, tailoring the distal trachea to reconstruct the subglottic airway and to resurface the posterior cricoid plate. Developed initially for adults, these techniques have been applied successfully in children, with considerable success.[83–89] Monnier and colleagues described 15 children so treated, with 14 successes.[90] Three were of congenital origin and 12 resulted from intubation. Decannulation was achieved in a single procedure in 14 of the patients. Follow-up in 10 of the children from 5 to 14 years showed good laryngeal growth despite removal of the anterior cricoid.[68] Couraud and colleagues noted a similar experience, also with laryngeal growth.[67] More recent experience further supports this approach, which seems on a rational basis to be superior to the cricoid split with cartilage graft insertion, leaving the stenosis in place.

Congenital Stenosis

In 1982, Kimura and colleagues described anterior patch tracheoplasty for treatment of congenital stenosis involving the entire trachea (see Chapter 33, "Repair of Congenital Tracheal Lesions").[91] The technique followed the principle of Gebauer's wire reinforced dermal grafts for tracheal and bronchial stenosis.[92] Stenosis too long to be treated by resection and anastomosis was incised vertically throughout its length and the tracheal diameter widened by fitting a long cartilaginous graft (Figure 6-24*B*). An endotracheal tube was left in place until the patch became firm. A later report augmented their experience.[93] Idriss and colleagues modified the procedure by using pericardium, which required not only postoperative splinting with an endotracheal tube but suture suspension of the pliable pericardial patch to mediastinal structures as well.[94] Considerable difficulty was encountered, with granulation tissue formation, necessitating multiple postoperative bronchoscopies (mean 3.8), especially for grafts extending far distally (mean 16).[95] Twenty-one patients underwent pericardial patch repair, with 2 operative deaths and 3 late deaths. Six needed later tracheostomy, 2 for airway stenting. Troubled by the lack of intrinsic sup-

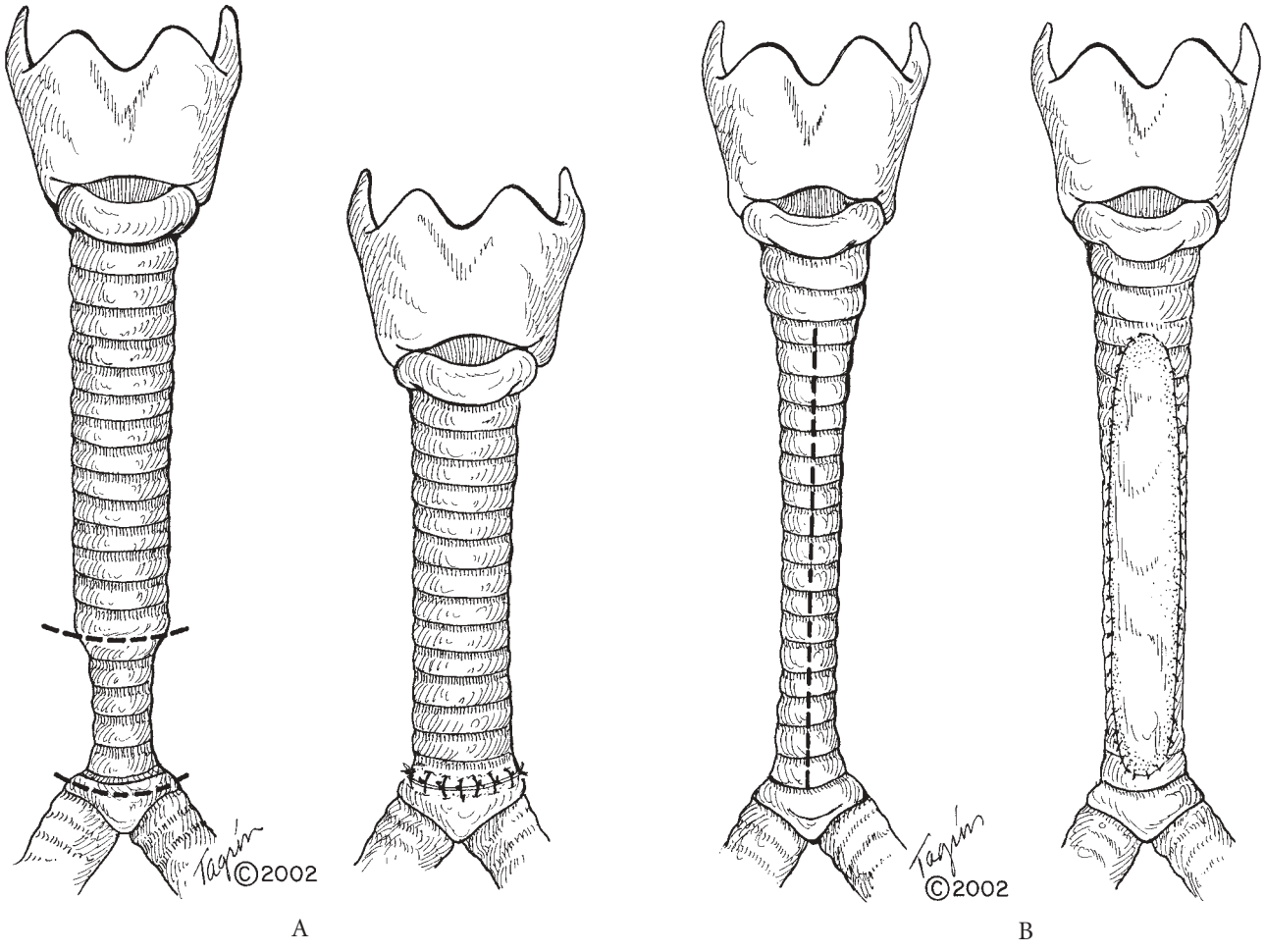


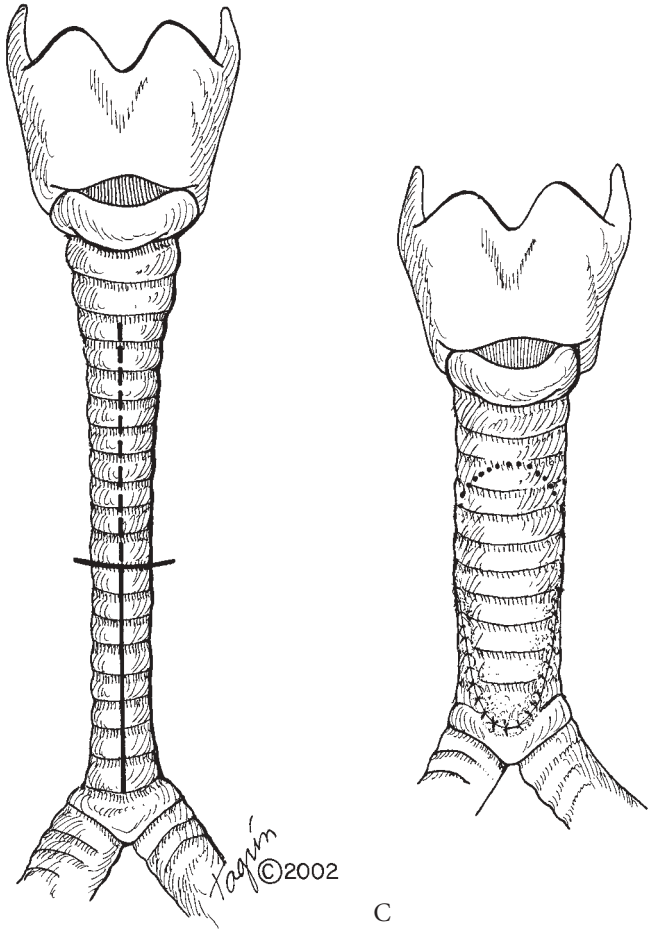
FIGURE **6-24** *Surgical techniques for treatment of congenital tracheal stenosis.* A, *Resection and reconstruction, suitable only for shorter stenosis.* B, *Patch tracheoplasty. Incision of long stenosis and widening of trachea with free cartilage or pericardial grafts.*

port, a later modification proposed by this group was anterior division of the stenosis, and excision of a central piece of trachea to be used for part of the "gusset," augmented with pericardium. This complicated repair offers little over simple slide tracheoplasty, described below.

Heimansohn and colleagues also used pericardium in a group of 12 patients, and noted only 2 patients with granulations and requiring re-operation of the 10 patients who survived long term.[96] Importantly, normal growth of the trachea was noted in longer-term results (1 to 11 years, mean 5 years).[97] In 2 patients examined postmortem 13 and 18 months, respectively, after pericardial patch tracheoplasty, it was found that the pseudostratified columnar epithelium had lined the dense mature collagenous scar tissue that had replaced the pericardial patch.[98] Jaquiss and colleagues returned to cartilage patch tracheoplasty, with granulation and dehiscence in 1 of 6 patients, all of whom survived.[99] Mechanical ventilatory support was provided for a mean of 11 days, with a median postoperative hospitalization of 17 days. In time, cartilage grafts are also replaced by mature scar tissue and reepithelized by ciliated columnar epithelium.[100] The luminal enlargement is maintained. Linear sections of cadaver trachea, chemically fixed so that the tissue is not viable, have been used as a patch to widen long stenoses of various etiologies.[101] Both the biological processes of healing and the benefits of this technique compared with the use of native tissues are unclear (see Chapter 45, "Tracheal Replacement").

Slide tracheoplasty (see Chapter 33, "Repair of Congenital Tracheal Lesions"), first proposed by Tsang and colleagues and successfully employed by Grillo, meets the problems of long segment congenital stenosis by using tracheal tissue alone to widen the lumen, giving stability, minimizing granulation tissue formation, and assuring more likely prompt healing.[102,103] A complete epithelial surface is also immediately provided. The stenosis is divided horizontally in its midpoint, and the upper and lower segments of stenosis are incised vertically through their entire extent, one anteriorly and one posteriorly. Corners are trimmed and the two segments slid together for suturing (Figure 6-24*C*). The circumference of the trachea is doubled and the cross-sectional area approximately quadrupled (Figure 6-25). Since the ends of the cartilaginous walls tend to curl inward, a lobulated cross-sectional appearance of a figure 8 character may be produced, so that the area is slightly less than quadrupled (Figure 6-26). Since the affected segment of the trachea is halved in length, even stenosis of the entire length of trachea does not preclude the procedure.

This operation was performed in 8 patients ranging in age from 9 days to 19 years (Figure 6-27).[73] Five were done with simple cross-field ventilation and 3 with a period of bypass necessary to reimplant a divided pulmonary artery sling, to perform other cardiac procedures or because of myocardial disease. Segments resected were between 36 to 83% of tracheal length. Two had anomalous right upper lobe bronchi. Only 1 required 3 days of intubation for ventilatory support, principally for airway clearance. Hospitalization ranged from 8 to 13 days. One minute granuloma appeared late and was removed bronchoscopically. Follow-up between 1 and 10 years shows good growth of the reconstructed trachea in the younger patients.[73] Functional results, which can be measured in older patients, are excellent (Table 6-2) (Figure 6-28).



C

FIGURE **6-24** (CONTINUED) C, *Slide tracheoplasty, reconstructing the trachea with (unresected) native trachea, providing fourfold widening of the lumen. Probably preferable even for most shorter stenoses as well as for long stenosis.*

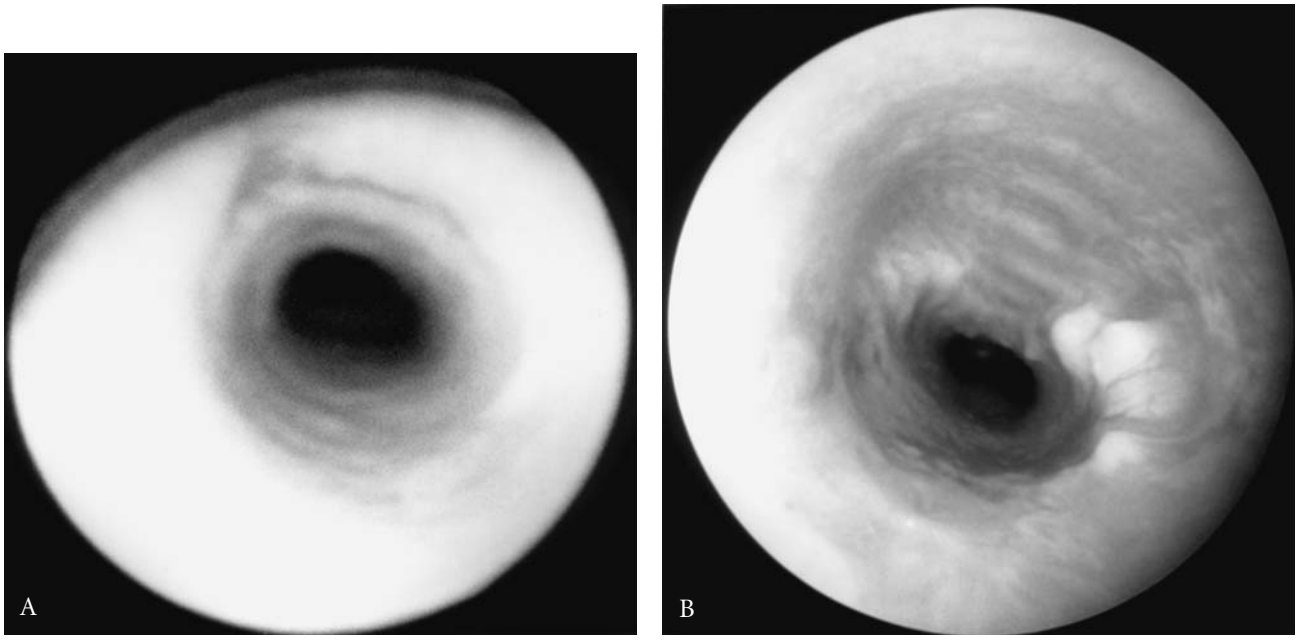


FIGURE **6-25** *Bronchoscopic views before* (A) *and after* (B) *slide tracheoplasty in a 3-month-old boy with stenosis of one-half of his tracheal length. In* A, *"O" rings are clearly visible. In* B, *the widening of the lumen allows the carina to be seen distantly. The irregular margins of the tracheoplasty are evident. The child was completely relieved of severe symptoms and his trachea grew well.*

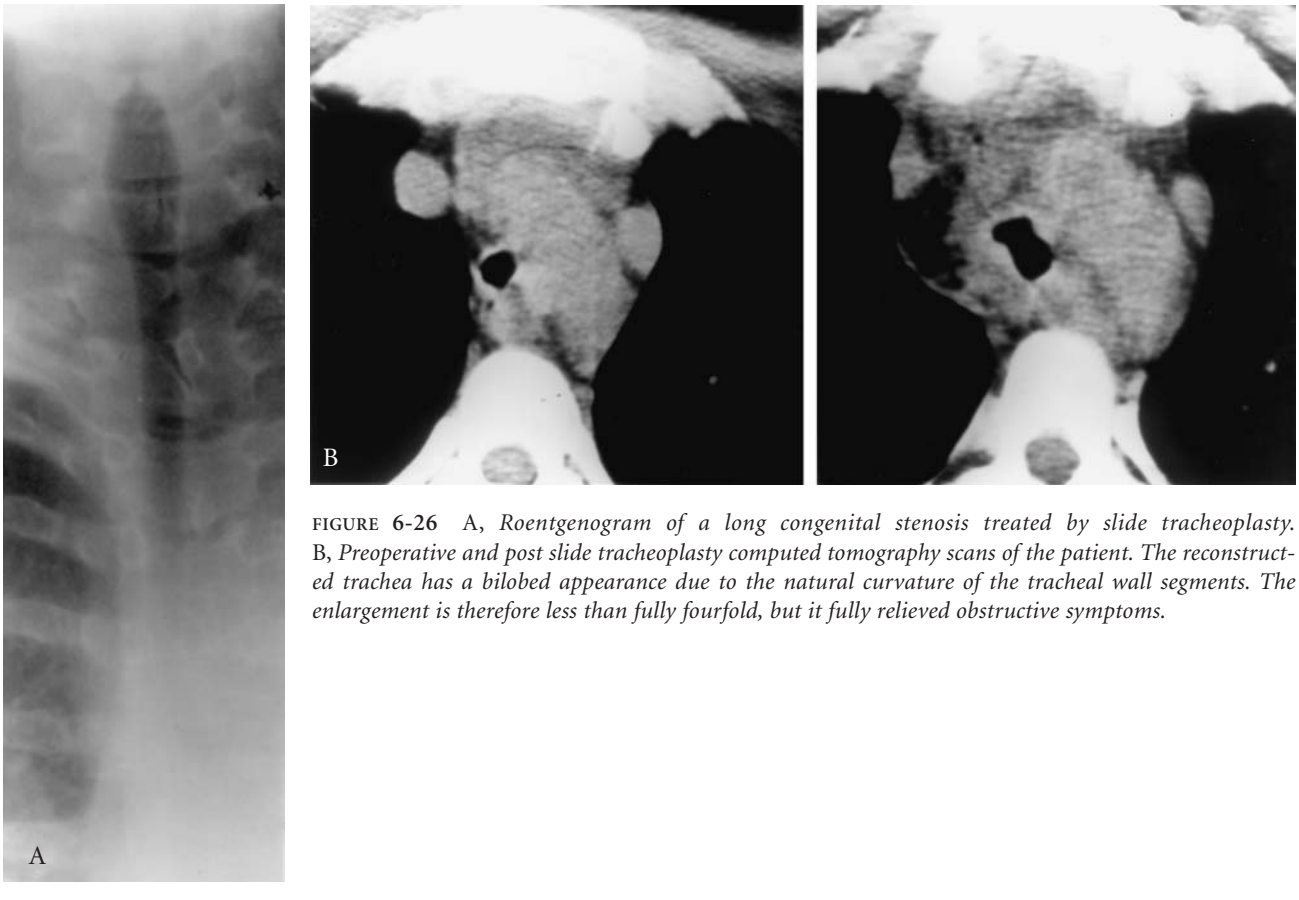


FIGURE **6-26** A, *Roentgenogram of a long congenital stenosis treated by slide tracheoplasty.* B, *Preoperative and post slide tracheoplasty computed tomography scans of the patient. The reconstructed trachea has a bilobed appearance due to the natural curvature of the tracheal wall segments. The enlargement is therefore less than fully fourfold, but it fully relieved obstructive symptoms.*

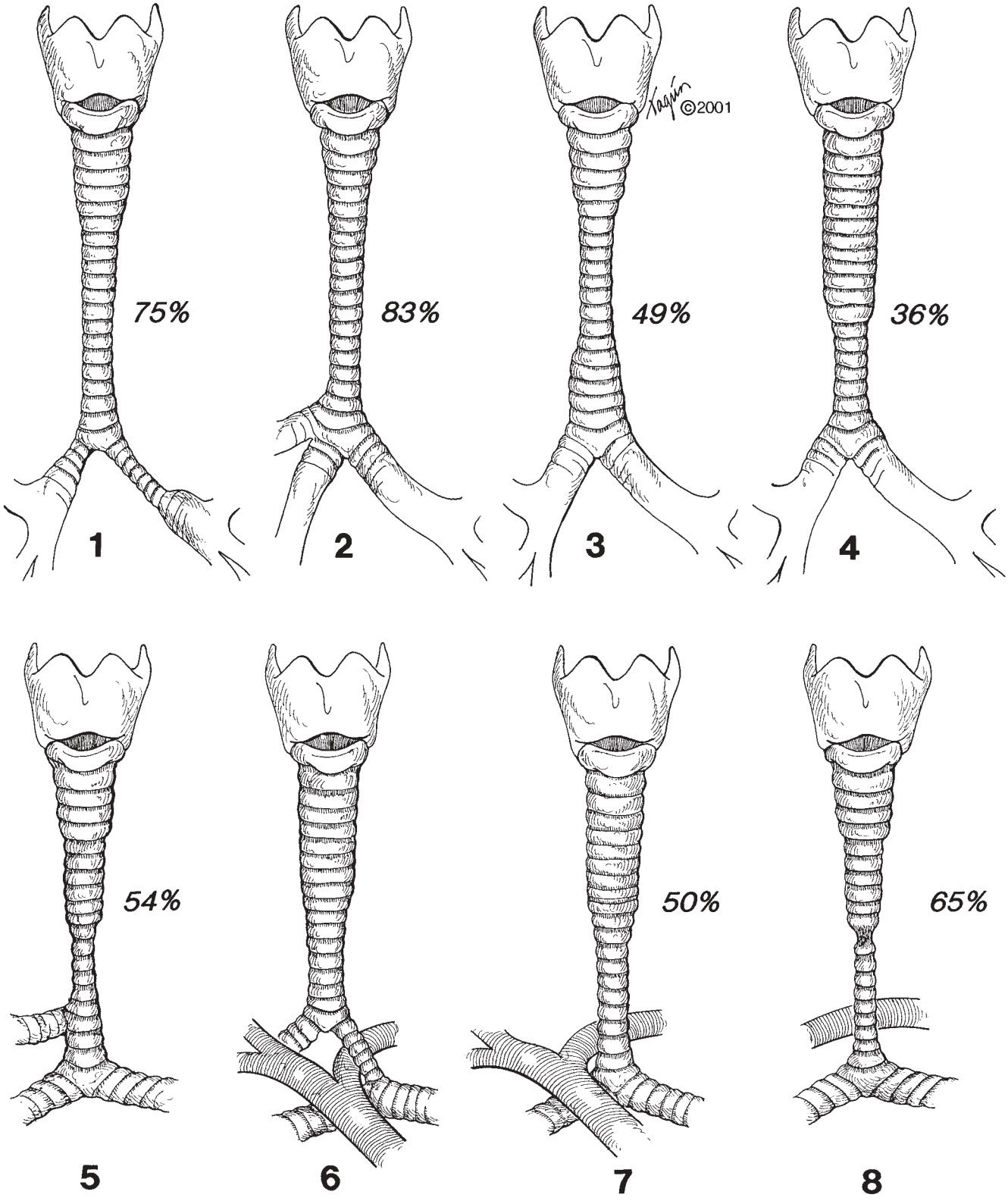


FIGURE **6-27** *Application of slide tracheoplasty. Diagrams of our first 8 patients, with percentages of trachea affected by stenosis treated by this technique. In number 6, the lesser supracarinal stenosis did not require correction, and the slide procedure was applied to the "bridge" bronchus. True tracheal length naturally varied with age. Reproduced with permission from Grillo HC et al.*[73]

Table 6-2 Pulmonary Function Before and After Slide Tracheoplasty for Congenital Stenosis

	Patient 1		*Patient 2*	
Variable	*Preoperative (%)*	*Postoperative (%)*	*Preoperative (%)*	*Postoperative (%)*
FEV_1 (L)	1.26 (40)	1.51 (52)	3.0 (79)	3.37 (93)
VC (L)	1.3 (37)	1.68 (47)	4.10 (92)	4.28 (102)
FEV_1/VC	0.97 (107)	0.90 (110)	0.74 (85)	0.79 (91)
PEFR	2.3 (37)	3.18 (51)	4.39 (63)	5.4 (80)
MBC	50 (40)	44 (40)	121 (79)	135 (93)
RV/TLC	0.62 (238)	0.52 (217)	0.28 (93)	0.13 (44)

FEV_1 = forced expiratory volume in 1 second; MBC = maximum breathing capacity; PEFR = peak expiratory flow rate; RV = residual volume; TLC = total lung capacity; VC = vital capacity.

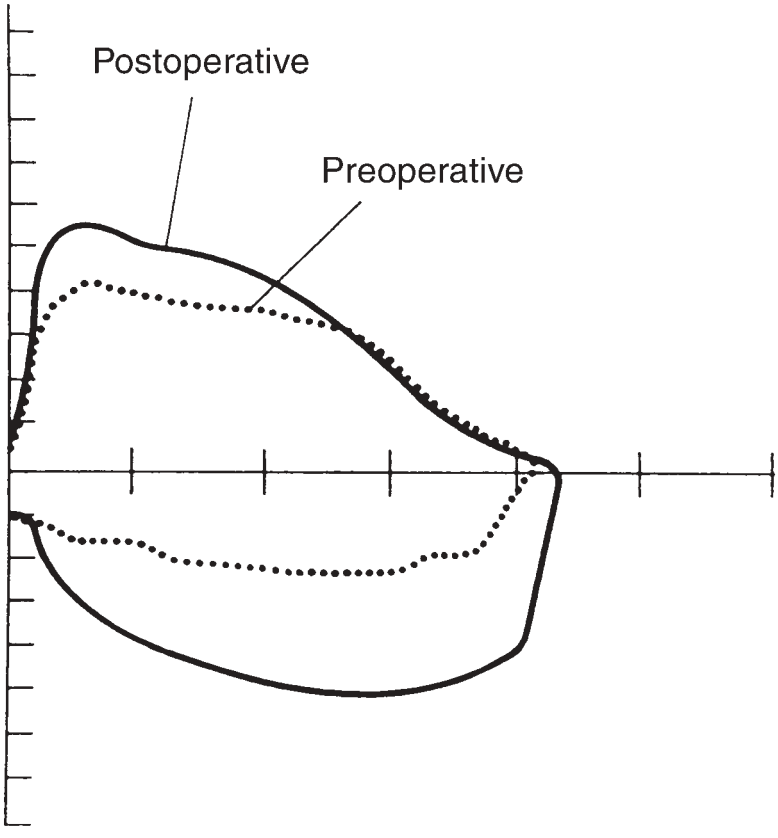


FIGURE **6-28** *Flow volume loop in a 19-year-old patient who underwent slide tracheoplasty, preoperatively and 1 year postoperatively. She was totally relieved of symptoms.*

The results of slide tracheoplasty suggest its superiority over patch tracheoplasty.[73,103] Since reports from three major children's hospitals show an average incidence of only one or two such cases yearly in each institution, a long time may be needed to establish a preferable method statistically. Nevertheless, a number of recent reports have indicated a growing acceptance of slide tracheoplasty.[73] I recommend this technique even over resection and reconstruction, if there is any likelihood of too much tension after resecting a lesion of borderline length. It should be unnecessary to point out that this technique has no application in acquired fibrous stenosis, where the absence of a "normal" tracheal wall would result in restenosis.

Resection and reconstruction of congenital stenosis is still the method of choice for short segments of stenosis, where lack of anastomotic tension may be confidently predicted. Resection is also uniquely applicable in the case of a *short* bridging bronchus, especially where maximum narrowing is located at one end of the stenotic bridging bronchus. Cantrell and Guild described this procedure and I have found it useful.[16,73] Extreme caution must be used in designing the exact size and location of the anastomotic apertures proximally and distally, to avoid angulation of the bronchi (see Chapter 33, "Repair of Congenital Tracheal Lesions").

Transplantation of trachea remains impractical at present. Problems of maintenance of viability of the epithelium and cartilage, tissue rejection, and hazards of prolonged immunosuppression are discussed at length in Chapter 45, "Tracheal Replacement." A further problem with any form of transplant in juveniles, as well as with synthetic or composite grafts to replace lost trachea, is the need for growth as the child grows. Experimenters tend to forget this in their enthusiasm for "new" technology. For similar reasons, I believe that permanent or semipermanent expandable stents are to be avoided in the juvenile trachea, if the disease is not soon likely to be lethal. In addition, such stents can irreversibly damage the trachea and prove to be nearly irremovable. Silicone inlying stents, such as the Dumon stent, should be used with measured judgement. I have surgically corrected children who were seemingly condemned to indefinite periodic replacement of Dumon stents. Silicone stents cause reactive granulomas and thus extend the original pathology. The inadvisable path of repeated laser treatments should also be avoided.

References

1. Gray SW, Skandalakis JE. Embryology for surgeons. The embryological basis for the treatment of congenital defects. Philadelphia: WB Saunders; 1972. p. 293–322.
2. Faro RS, Goodwin CD, Organ CH, et al. Tracheal agenesis. Ann Thorac Surg 1979;28:295–9.
3. Fonkalsrud EW, Martelle RR, Maloney JV Jr. Surgical treatment of tracheal agenesis. J Thorac Cardiovasc Surg 1963;45:520–5.
4. Hiyama E, Yokoyama T, Ichikawa T, Matsuura Y. Surgical management of tracheal agenesis. J Thorac Cardiovasc Surg 1994;108:830–3.
5. Acosta JL, Battersby JS. Congenital tracheoesophageal fistula in the adult. Ann Thorac Surg 1974;17:51–7.
6. Chavrier Y, Revillon Y. Les fistules resotrachéales congénitales chez l'enfant. Chir Pédiatr 1984;25:228–33.
7. Dyon JF, deMarliave H, Bacle B, BenSalah S. Les fistules aérodigestives congénitales rares. Chir Pédiatr 1984;25:234–44.
8. Levasseur P, Navajas M. Congenital tracheobiliary fistula. Ann Thorac Surg 1987;44:318–9.
9. Azoulay D, Regnard JF, Magdeleinat P, et al. Congenital respiratory-esophageal fistula in the adult. J Thorac Cardiovasc Surg 1992;104:381–4.
10. Braimbridge MV, Keith HI. Oesophago-bronchial fistula in the adult. Thorax 1965;20:226–33.
11. Petterson G. Laryngo-tracheo-esophageal cleft. Z Kinderchir 1969;7:43–9.
12. Ryan DP, Muehrcke DD, Doody DP, et al. Laryngotracheoesophageal cleft (type IV): management and repair of lesions beyond the carina. J Pediatr Surg 1991;26:962–70.
13. Donahoe PK, Gee PE. Complete laryngotracheoesophageal cleft: management and repair. J Pediatr Surg 1984;19:143–8.
14. Berkovits RNP, Van der Schams EJ, Molenaar JC. Treatment of congenital cricoid stenosis. In: Wurnig P, editor. Trachea and lung surgery in childhood. Berlin: Springer-Verlag; 1991. p. 20–8.
15. Jaubert de Beaujeu M, Louis D. Obstruction par sténoses trachéales et bronchiques congénitales. II Sténoses congénitales des bronches souches. Chir Pédiatr 1984;25:256–60.
16. Cantrell JR, Guild HC. Congenital stenosis of the trachea. Am J Surg 1964;108:297–305.
17. Wells TR, Gwinn JL, Landing BH, Stanley P. Reconsideration of the anatomy of sling left pulmonary artery: the association of one form with bridging bronchus and imperforate anus. Anatomic and diagnostic aspects. J Pediatr Surg 1988;23:892–8.
18. Clarkson PM, Ritter DG, Rahimtoola SH, et al. Aberrant left pulmonary artery. Am J Dis Child 1967; 113:373–7.
19. Sade RM, Rosenthal A, Fellows K, Castaneda AR. Pulmonary artery sling. J Thorac Cardiovasc Surg 1975; 69:333–46.
20. Backer CL, Idriss FS, Holinger LD, Mavroudis C. Pulmonary artery sling. Results of surgical repair in infancy. J Thorac Cardiovasc Surg 1992;10:683–91.
21. Jonas RA, Spevak PJ, McGill T, Castaneda AR. Pulmonary artery sling: primary repair by tracheal resection in infancy. J Thorac Cardiovasc Surg 1989;97:548–50.
22. Grillo HC. Discussion of Dunn JM, Gordon I, Chrispin AR, et al. Early and late results of surgical correction of pulmonary artery sling. Ann Thorac Surg 1979;28:230–8.
23. Van Sohn JAM, Hamlisch J, Haas GS, et al. Pulmonary artery sling: reimplantation versus antetracheal translocation. Ann Thorac Surg 1999;68:989–94.
24. Cogbill TH, Moore FA, Accurso FJ, Lilly JR. Primary tracheomalacia. Ann Thorac Surg 1983;35:538–41.
25. Filler RM, deFraga JC. Tracheomalacia. Semin Thorac Cardiovasc Surg 1994;6:211–5.
26. Filler RM, Messineo A, Vinograd I. Severe tracheomalacia associated with esophageal atresia: results of surgical treatment. J Pediatr Surg 1992;27:1136–41.
27. Vazquez-Jimenez JF, Sachweh JS, Liakopoulos OJ, et al. Aortopexy in severe tracheal instability: short-term and long-term outcome in 29 infants and children. Ann Thorac Surg 2001;72:1898–901.
28. Harrison MR, Hendren WH. Agenesis of the lung complicated by vascular compression and bronchomalacia. J Pediatr Surg 1975;10:813–7.
29. McCormick TL, Kuhns LR. Tracheal compression by a

normal aorta associated with right lung agenesis. Radiology 1979;130:659–60.
30. Conti VR, Lobe TE. Vascular sling with tracheomalacia: surgical management. Ann Thorac Surg 1989; 47:310–1.
31. Mounier-Kuhn P. Dilatation de la trachée; constatations radiographiques et bronchoscopiques. Lyon Med 1932;150:106–9.
32. Katz I, Levine M, Herman P. Trachiobronchiomegaly: the Mounier-Kuhn syndrome. Am J Roentgen 1962;88: 1084–94.
33. Stewart JR, Kincaid OW, Edwards JE. An atlas of vascular rings and related malformations of the aortic arch system. Springfield (IL): Charles C. Thomas; 1964.
34. Gross RE. Arterial malformations which cause compression of the trachea or esophagus. Circulation 1955;11:124–34.
35. Lincoln JCR, Deverall PB, Stark J, et al. Vascular anomalies compressing the esophagus and trachea. Thorax 1969;24:295–306.
36. Arciniegas E, Hakimi M, Hertzler JH, et al. Surgical management of congenital vascular rings. J Thorac Cardiovasc Surg 1979;77:721–7.
37. Richardson JV, Doty DB, Rossi NP, Ehrenhaft JL. Operation for aortic arch anomalies. Ann Thorac Surg 1981;31:426–32.
38. Backer CL, Ilbawi MN, Idriss SS, DeLeon SY. Vascular anomalies causing tracheoesophageal compression: review of experience in children. J Thorac Cardiovasc Surg 1989;97:725–31.
39. Chun K, Colombani PM, Dudgeon DL, Haller JA Jr. Diagnosis and management of congenital vascular rings: a 22-year experience. Ann Thorac Surg 1992;53:597–603.
40. Backer CL, Hillman N, Mavroudis C, Holinger LD. Resection of Kommerell's diverticulum and left subclavian artery transfer for recurrent symptoms after vascular ring divsions. Eur J Cardiothorac Surg 2002;22:64–9.
41. Konstantinov IE. Resection of Kommerell's diverticulum and left subclavian artery transfer does not always relieve symptoms. Eur J Cardiovasc Surg 2002;22: 1038–9.
42. Robotin MC, Bruniaux J, Serraf A, et al. Unusual forms of tracheobronchial compression in infants with congenital heart disease. J Thorac Cardiovasc Surg 1996;112:415–23.
43. Gross RE, Neuhauser EBD. Compression of the trachea by an anomalous innominate artery. An operation for its relief. Am J Dis Child 1948;75:570–4.
44. Mustard WT, Bayliss CE, Fearon B, et al. Tracheal compression by the innominate artery in children. Ann Thorac Surg 1969;8:312–9.
45. Jones DT, Jonas RA, Healy GB. Innominate artery compression of the trachea in infants. Ann Otol Rhinol Laryngol 1994;103:347–50.
46. Langlois L, Binet J-P, Planche C, et al. La compression de la trachée par le tronc artérial brachio-cephalique chez le nourrisson. Ann Chir 1982;36:157–63.
47. Hawkins JA, Bailey WW, Clark SM. Innominate artery compression of the trachea. Treatment by re-implantation of the innominate artery. J Thorac Cardiovasc Surg 1992; 103:678–82.
48. Cotton RT, Gray SD, Miller RP. Update of the Cincinnati experience in pediatric laryngotracheal reconstruction. Laryngoscope 1989;99:1111–6.
49. Marcovich M, Pollauf F, Burian K. Subglottic stenosis in newborns after mechanical ventilation. In: Wurnig P, editor. Trachea and lung surgery in childhood. Berlin: Springer-Verlag; 1991. p. 8–19.
50. Grillo HC, Mathisen DJ. Primary tracheal tumors: treatment and results. Ann Thorac Surg 1990;49:69–77.
51. Chappius JP. Les compressions trachéo-bronchiques par tumeurs congénitales. Chir Pédiatr 1984;25:279–83.
52. Bauman NM, Smith RJH. Recurrent respiratory papillomatosis. Pediatr Clin North Am 1996;43:1385–401.
53. Drolet BA, Esterly NB, Friedan J. Hemangiomas in children. N Engl J Med 1999;341:173–81.
54. Balquet P. Kystes bronchogéniques comprimant la trachée et les bronches souches. Chir Pédiatr 1984;25:270–5.
55. Papagiannopoulos KA, Linegar AG, Harris DG, Rossouw GJ. Surgical management of airway obstruction in primary tuberculosis in children. Ann Thorac Surg 1999;68:1182–6.
56. Worthington MG, Brinkj JG, Odell JA, et al. Surgical relief of acute airway obstruction due to primary tuberculosis. Ann Thorac Surg 1993;56:1054–62.
57. Maeda M, Grillo HC. Tracheal growth following anastomosis in puppies. J Thorac Cardiovasc Surg 1972;64:304–13.
58. Maeda M, Grillo HC. Effect of tension on tracheal growth after resection and anastomosis in puppies. J Thorac Cardiovasc Surg 1973;65:658–68.
59. Kotake Y, Grillo HC. Reduction of tension at the anastomosis following tracheal resection in puppies. J Thorac Cardiovasc Surg 1976;71:600–4.
60. Sørensen HR, Moesner J, Nielson PA. Influence of growth upon the development of stenosis following experimental resections of the trachea in puppies. Scand J Thorac Cardiovasc Surg 1971;5:202–5.
61. Murphy DA, Dunn GL, Poirier N, Martin M. Growth of tracheal anastomoses. Ann Thorac Surg 1973;16:158–62.
62. Mendez-Picon G, Hutchen NE, Neifield J, Salzberg AM. Long term study of tracheal growth after segmental resection in puppies. J Pediatr Surg 1974;9:615–9.
63. Carcassonne M, Dor V, Aubert J, Kreitman P. Tracheal resection with primary anastomosis in children. J Pediatr Surg 1973;8:1–8.
64. Grillo HC, Zannini P. Management of obstructive tracheal disease in children. J Pediatr Surg 1984;19:414–6.
65. Alstrup P, Sørensen HR. Resection of acquired tracheal stenosis in childhood. J Thorac Cardiovasc Surg 1984;87:547–9.
66. DeLorimier AA, Harrison MR, Karen H, et al. Tracheobronchial obstruction in infants and children: experience with 45 cases. Ann Surg 1990;212:277–89.
67. Couraud L, Moreau JM, Velly JF. The growth of circumferential scars of the major airways from infancy to adulthood. Eur J Cardiothorac Surg 1990;4:521–6.
68. Monnier P, Savary M, Chapuis G. Cricotracheal resection for pediatric subglottic stenosis: update of the Lausanne experience. Acta Otorhinolaryngol Belg 1995;49:373–82.
69. Wright CG, Grahm BB, Grillo HC, et al. Pediatric tracheal surgery. Ann Thorac Surg 2002;74:308–14.
70. Burrington JD. Tracheal growth and healing. J Thorac Cardiovasc Surg 1978;4:453–8.
71. Manson D, Filler R, Gordon R. Tracheal growth in congenital tracheal stenosis. Pediatr Radiol 1996;26:427–30.
72. Macchiarini P, Sulmet E, de Montpreville V, et al. Tracheal growth after slide tracheoplasty. J Thorac Cardiovasc Surg 1997;113:558–66.
73. Grillo HC, Wright CD, Vlahakes GJ, MacGillivray TE. Management of congenital tracheal stenosis by means of tracheoplasty or resection and reconstruction, with long term follow-up of growth after slide tracheoplasty. J Thorac Cardiovasc Surg 2002;123:145–52.
74. Gaissert HA, Grillo HC, Mathisen DJ, et al. Temporary and permanent restoration of airway continuity with the tracheal T-tube. J Thorac Cardiovasc Surg 1994; 107:600–6.
75. Kim SH, Hendren WH. Endoscopic resection of obstructing airway lesions in children. J Pediatr Surg 1976;11:431–41.

76. Friedman E, Perez-Atayde AR, Silvera M, Jonas RA. Growth of tracheal anastomoses in lambs: comparison of PDS and Vicryl suture material and interrupted and continuous techniques. J Thorac Cardiovasc Surg 1990;100:188–93.
77. Grillo HC, Donahue DJ, Mathisen DJ, et al. Postintubation tracheal stenosis. Treatment and results. J Thorac Cardiovasc Surg 1995;109:486–93.
78. Nakayama DK, Harrison MR, De Lorimier AA, et al. Reconstructive surgery for obstructing lesions of the intrathoracic trachea in infants and small children. J Pediatr Surg 1982;17:854–71.
79. Gaissert HA, Mathisen DJ, Grillo HC, et al. Tracheobronchial sleeve resection in children and adolescents. J Pediat Surg 1994;29:192–8.
80. Cotton RT, Myer CM, O'Connor DM, Smith ME. Pediatric laryngotracheal reconstruction with cartilage grafts and endotracheal tube stenting: the single stage approach. Laryngoscope 1995;105:818–21.
81. April MM, Marsh BR. Laryngotracheal reconstruction for subglottic stenosis. Ann Otol Rhinol Laryngol 1993;102:176–81.
82. Rutter MJ, Hartley BEJ, Cotton RT. Cricotracheal resection in children. Arch Otolaryngol Hand Neck Surg 2001;127:289–92.
83. Ogura JH, Roper CL. Surgical correction of traumatic stenosis of larynx and pharynx. Laryngoscope 1962;72:468–80.
84. Gerwat J, Bryce DP. The management of subglottic laryngeal stenosis by resection and direct anastomosis. Laryngoscope 1974;84:940–57.
85. Pearson FG, Cooper JD, Nelems JM, et al. Primary tracheal anastomosis after resection of the cricoid cartilage with preservation of recurrent laryngeal nerves. J Thorac Cardiovasc Surg 1975;70:806–16.
86. Grillo HC. Primary reconstruction of the airways after resection of subglottic laryngeal and upper tracheal stenosis. Ann Thorac Surg 1982;33:3–8.
87. Grillo HC, Mathisen DJ, Wain JC. Laryngotracheal resection and reconstruction for subglottic stenosis. Ann Thorac Surg 1992;53:54–63.
88. Couraud L, Hafez A, Velly JF. Current reconstructive management of subglottic stenosis of the larynx with reference to 60 consecutively treated cases. J Thorac Cardiovasc Surg 1985;33:263–7.
89. Maddaus AAA, Toth JLR, Gullane PJ, et al. Subglottic tracheal resection and synchronous laryngeal reconstruction. J Thorac Cardiovasc Surg 1997;104:892–9.
90. Monnier P, Savary M, Chapuis G. Partial cricoid resection with primary tracheal anastomosis for subglottic stenosis in infants and children. Laryngoscope 1993;103:1273–83.
91. Kimura K, Mukohara N, Tsugawa C, et al. Tracheoplasty for congenital stenosis of the entire trachea. J Pediatr Surg 1982;17:869–71.
92. Gebauer PW. Reconstructive surgery of the trachea and bronchi: late results with dermal grafts. J Thorac Surg 1951;22:568–84.
93. Tsugawa C, Kimura K, Muraji T, et al. Congenital stenosis involving a long segment of the trachea: further experience in reconstructive surgery. J Pediatr Surg 1988;23:471–5.
94. Idriss FS, DeLeon SY, Ilbawi MN, et al. Tracheoplasty with pericardial patch for extensive tracheal stenosis in infants and children. J Thorac Cardiovasc Surg 1984;88:527–36.
95. Dunham ME, Holinger LD, Backer CL, et al. Management of severe congenital tracheal stenosis. Ann Otol Rhinol Laryngol 1994;103:351–6.
96. Heimansohn DA, Kesler KA, Turrentine MW, et al. Anterior pericardial tracheoplasty for congenital tracheal stenosis. J Thorac Cardiovasc Surg 1991;102:710–6.
97. Bando K, Turrentine MW, Sun K, et al. Intermediate to long-term outcomes after anterior pericardial tracheoplasty for congenital tracheal stenosis. Ann Thorac Surg 1996;62:981–9.
98. Cheng ATL, Backer CL, Holinger LD, et al. Histopathologic changes after pericardial patch tracheoplasty. Arch Otolaryngol Head Neck Surg 1997;123:1069–72.
99. Jaquiss RDB, Lusk PR, Spray TL, et al. Repair of long-segment tracheal stenosis in infancy. J Thorac Cardiovasc Surg 1995;110:1504–12.
100. Oue T, Kamata S, Usui N, et al. Histopathologic changes after tracheobronchial reconstruction with costal cartilage graft for congenital tracheal stenosis. J Pediatr Surg 2001;36:329–33.
101 Jacobs JP, Elliott MJ, Haw MP, et al. Pediatric tracheal homograft reconstruction: a novel approach to complex tracheal stenosis in children. J Thorac Cardiovasc Surg 1996;112:1549–60.
102. Tsang V, Murday A, Gilbe C, Goldstraw P. Slide tracheoplasty for congenital funnel-shaped tracheal stenosis. Ann Thorac Surg 1989;48:632–5.
103. Grillo HC. Slide tracheoplasty for long-segment congenital stenosis. Ann Thorac Surg 1994;58:613–21.

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Primary Tracheal Neoplasms

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Primary tracheal neoplasms are still often diagnosed long after the onset of symptoms or signs, particularly in the absence of hemoptysis. Benign neoplasms may be unrecognized for many months or even for several years. The duration of symptoms for malignant lesions prior to diagnosis is 6 to 18 months, reflecting more rapid growth, and especially, onset of hemoptysis. Even pulmonologists remain unfamiliar with tracheal tumors because of their rarity and a corresponding dearth of information about their occurrence and behavior. Reassured by a chest radiograph, which is often read as normal, the physician assigns the reason for wheezing and exertional dyspnea caused by a tumor to "adult onset asthma" or to chronic lung disease. Inappropriate treatment, once the lesion has been recognized, is an equally serious problem. It is still not widely appreciated that modern techniques of tracheal surgery, combined with radiotherapy, can produce cure or long-term palliation in many patients with primary malignant tumors.[1–6] Overall results of treatment are more encouraging than oncologic results of management of carcinoma of the lung, to which major therapeutic efforts are regularly directed.

As with most tumors, the best opportunity for cure presents at the time of diagnosis. The physician who deals with a primary tracheal tumor should, therefore, be fully aware of its potential clinical course and the best methods available for treatment. If expertise in management is not available in the physician's area, the patient should be referred to an appropriate center. Patients continue to be seen with resectable tracheal tumors that are presumed on the one hand to be of limited malignancy or on the other hand to be incurable and that are treated by transbronchial ablation or irradiation alone. Laser treatment has been employed repetitively on resectable tumors—a treatment that can never cure a malignant tracheal neoplasm and only rarely a benign one. When such patients are seen many months and sometimes years after initial diagnosis, longitudinal spread, particularly of adenoid cystic carcinoma, has become so extensive that surgical resection and reconstruction is too often no longer possible. High-dose "curative" irradiation administered without initially considering surgical excision may make later resection prohibitively risky. Even benign tumors present increasingly greater technical problems for definitive surgical resection if their management has been delayed by ill-conceived attempts at local management—often with a laser. Simple microscopic exam-

ination makes it clear that only full-thickness ablation of the wall of the trachea, which contains the base even of most benign tumors, could possibly lead to extirpation (see Figure 3-12 [Color Plate 2] from Chapter 3, "Pathology of Tracheal Tumors").

Unfortunately, at present in the United States, considerations of cost and jurisdiction under managed care can keep patients from proper treatment. This could worsen if the web of managed care and capitation spreads.

Characteristics

The incidence of primary tracheal tumors in the general population is not precisely known. Ranke and colleagues found 2 patients with tracheogenic carcinoma in 1,744 cancer deaths.[7] Culp noted 4 patients with primary tracheal tumors in 89,600 autopsies.[8] It is not a surprise that the diagnosis is seldom considered, even by pulmonologists.

The majority of primary tracheal tumors in adults are malignant. Thirty-six percent of a series of 198 patients with primary tracheal tumors seen at the Massachusetts General Hospital (MGH) over a 26-year period presented with primary squamous cell carcinoma (SCC) and 40% with adenoid cystic carcinoma (ACC).[1] The remaining 24% of the total included 9 other malignant lesions, 17 of intermediate character, such as carcinoid and mucoepidermoid tumors, and 21 were clearly benign. None of the SCCs were secondary to laryngeal, bronchogenic, or esophageal carcinoma. By 2002, the number of ACCs and of SCCs seen had risen to 135 each, a total of 270 patients in these categories alone. The very wide variety of tumors other than SCC and ACC is noteworthy (Table 7-1). The pathology of primary tracheal tumors is reviewed in Chapter 3, "Pathology of Tracheal Tumors," and illustrated in a color fascicle. Bronchoscopic views of some tumors are also to be found in the color section. Tracheal tumors are even rarer in children. Two-thirds are benign, since of the most common adult tumors, ACC is only occasional in children and SCC is nearly unique. In a review of the literature and from their own experience, Desai and colleagues found only 38 children with tracheal tumors over a 30-year period.[9] Over half were diagnosed initially as "asthma"; 39% were more than 50% obstructed when diagnosed. Hemangiomas and granular cell tumors were most common in the benign category, and mucoepidermoid tumors and histiocytomas in the malignant. Malignant tumors usually appeared in adolescence.

Primary SCC of the trachea may be exophytic or ulcerative, localized or longitudinally infiltrating, or less commonly, may show multiple areas of involvement scattered throughout the trachea (Figures 7-1, 7-2 and Figures 4 and 5, Color Plate 12). Invasive squamous cancer may also be found deep within what appears to be an area of papillomatous change, which on superficial biopsy reveals apparently in situ carcinoma. If such a lesion is grossly visible, it often does have deeper areas of invasive carcinoma.

As SCC grows, it extends longitudinally and circumferentially in the tracheal wall, and may penetrate extraluminally to involve adjacent structures. Tumors may occur at any level of the trachea or carina. Adjacent recurrent laryngeal nerves and the esophagus may be invaded directly. The most common sites of metastases are adjacent peritracheal lymph nodes. Hematogenous metastases to the lung, bone, liver, or adrenals are less common initially. Age and gender incidences of SCC of the trachea are similar to those of carcinoma of the lung (Table 7-2), with peak incidence between 50 and 70 years, predominating in males. The etiologies seem to be identical. Except in 4 cases, every patient we have seen with SCC of the trachea has been a cigarette smoker, usually for many years. One exception had received arsenicals for dermatologic treatment in youth and suffered multiple squamous skin cancers on exposure to sunlight. Another had worked for many years with a multitude of organic chemicals and had previously suffered squamous carcinoma of his tongue base. In 2 other nonsmokers, no etiology was determined, although the extent of involvement of the overlying thyroid gland in 1 raised a question of whether that tumor might have been a primary squamous carcinoma of the thyroid invading the trachea. Forty percent of our patients with SCC

Table 7-1 Primary Tracheal Tumors other than Adenoid Cystic and Squamous Carcinomas

Benign	Malignant
Squamous papilloma	Adenocarcinoma
Multiple	Adenosquamous carcinoma
Solitary	Small cell carcinoma
Pleomorphic adenoma	Basaloid squamous cell carcinoma
Granular cell tumor (myoblastoma)	Atypical carcinoid
Glomus tumor	Malignant fibrous histiocytoma
Fibroma	Melanoma
Fibrous histiocytoma (pseudotumor, plasma cell granuloma, xanthoma)	Chondrosarcoma
	Spindle cell sarcoma
Lipoma	Rhabdomyosarcoma
Leiomyoma	Fibrosarcoma
Hamartoma	Leiomyosarcoma
Chondroma	Kaposi's sarcoma
Chondroblastoma	Lymphoma
Schwannoma	Lymphoepithelial carcinoma
Neurofibroma	Angiosarcoma
Paraganglioma	
Hemangioma	
Hemangioendothelioma	
Vascular malformation	
Intermediate Malignancy	
Carcinoid	
Mucoepidermoid	
Plexiform neurofibroma	
Pseudosarcoma	
Plasmacytoma	
Acinic cell carcinoma	

of the trachea who underwent resection had either a previous history, a concurrent finding, or a later occurrence of SCC of the respiratory tract.[1] Cancer arose in the tongue, tonsil, larynx, trachea (second primary lesion), and lung.

Basaloid squamous cell carcinoma, a rare and aggressive variant consisting of basaloid cells with either dysplastic epithelium or in situ squamous epithelium or invasive tumor, usually occurring in the upper aerodigestive tract, may be found in the trachea and may be deeply ulcerative, invasive, and metastatic.[10] It may be confused with ACC histologically and with neuroendocrine carcinoma.

Adenoid cystic carcinoma may appear deceptively benign. Its former appellation, "cylindroma," masked its truly malignant character.[1,11] Indeed, it used to be described clinically and pathologically under a general heading of "bronchial adenoma." This included a heterogeneous collection of "cylindroma," carcinoid, and mucoepidermoid tumors plus a few true adenomas. Both terms are best abandoned. Adenoid cystic carcinoma of the trachea occurs over a wide age range, from the twenties through the seventies (see Table 7-2). Distribution between male and female is quite even but with female predominance (72 to 63 in 135 patients). No relationship has been discerned with cigarette smoking or other known carcinogenic factors. Thirty-three percent were smokers compared with at least 66% of patients with SCC. Although the

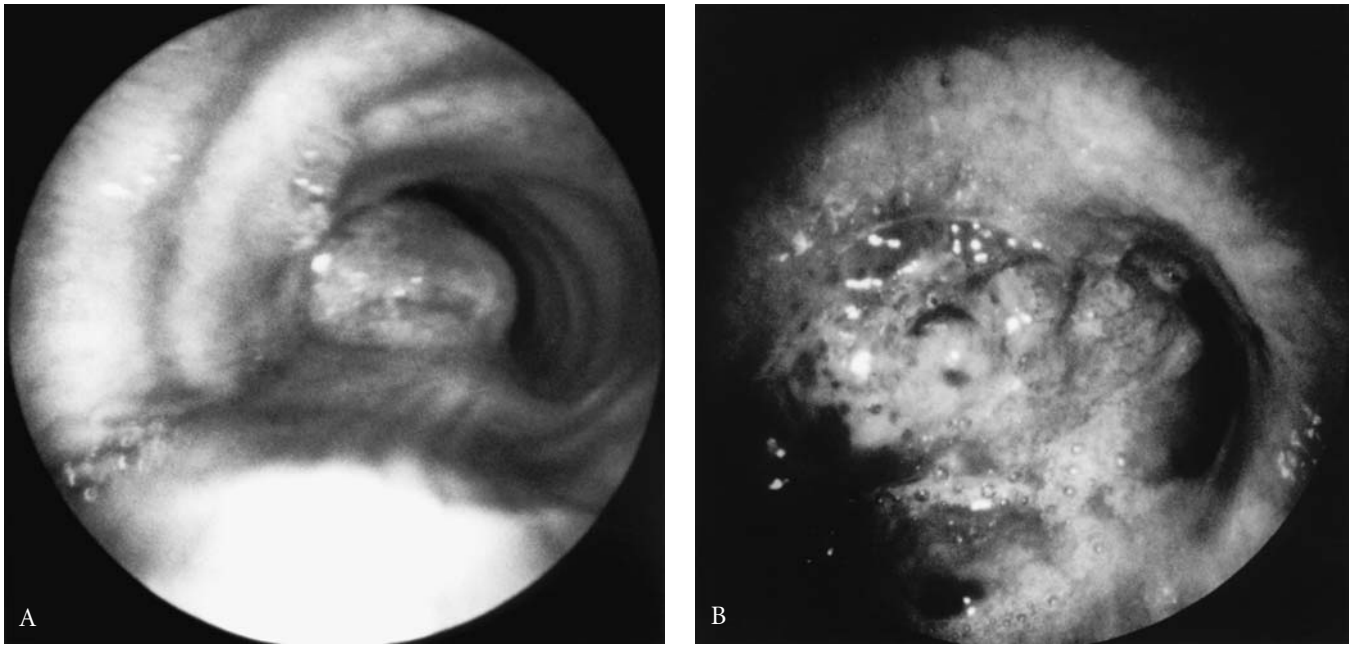


FIGURE 7-1 *Squamous cell carcinoma of trachea. Bronchoscopic views.* A, *Small tumor in the upper trachea.* B, *Large tumor at the carina.*

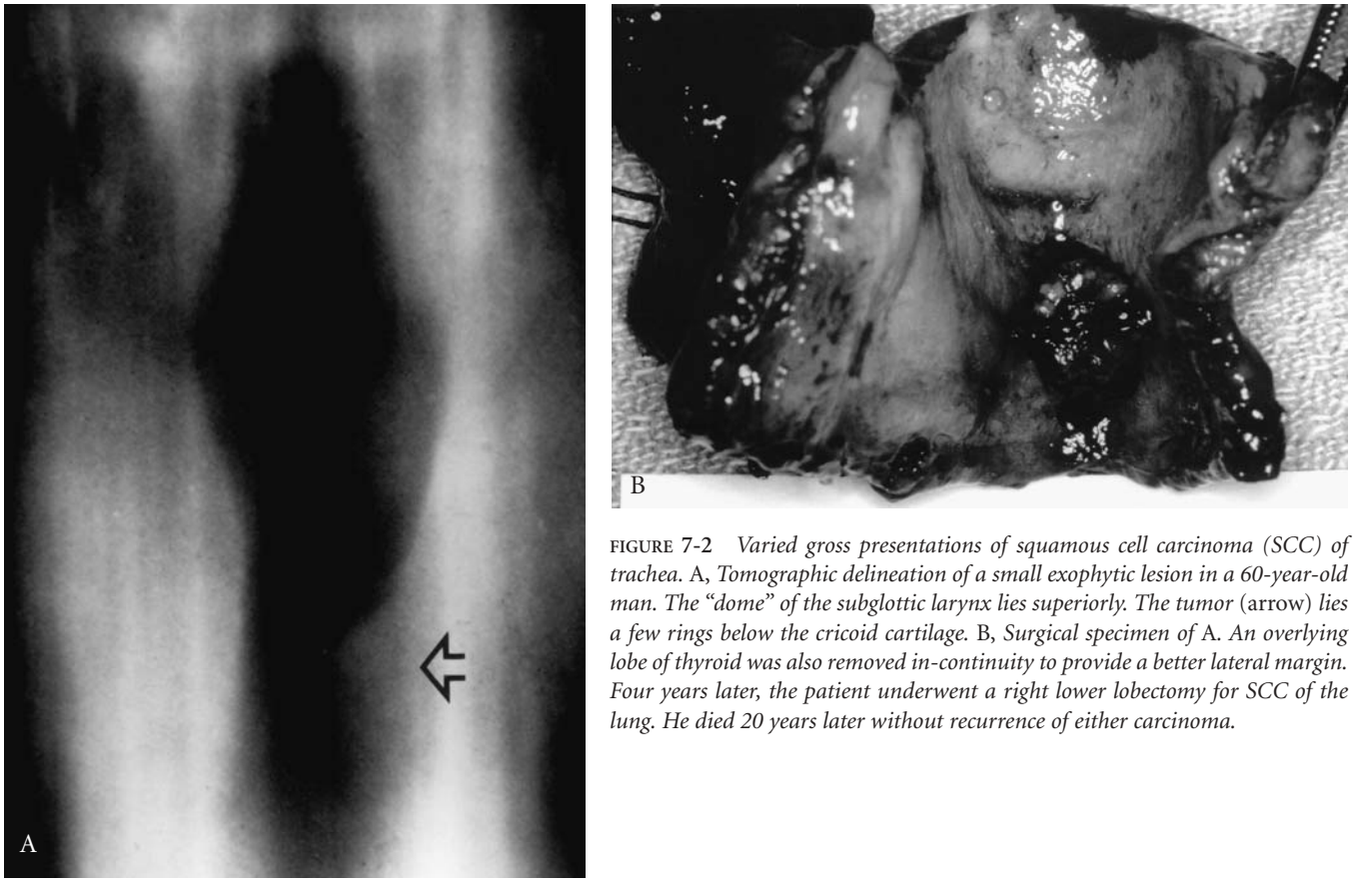
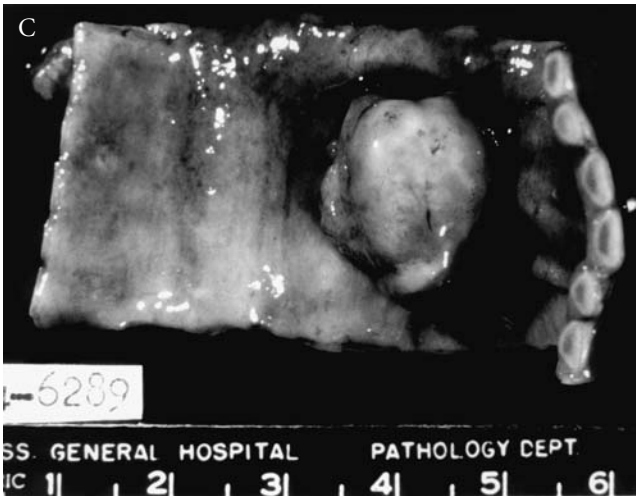
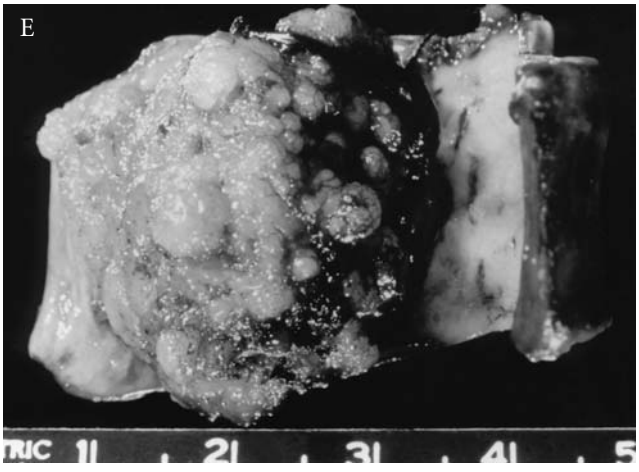


FIGURE 7-2 *Varied gross presentations of squamous cell carcinoma (SCC) of trachea.* A, *Tomographic delineation of a small exophytic lesion in a 60-year-old man. The "dome" of the subglottic larynx lies superiorly. The tumor* (arrow) *lies a few rings below the cricoid cartilage.* B, *Surgical specimen of* A. *An overlying lobe of thyroid was also removed in-continuity to provide a better lateral margin. Four years later, the patient underwent a right lower lobectomy for SCC of the lung. He died 20 years later without recurrence of either carcinoma.*





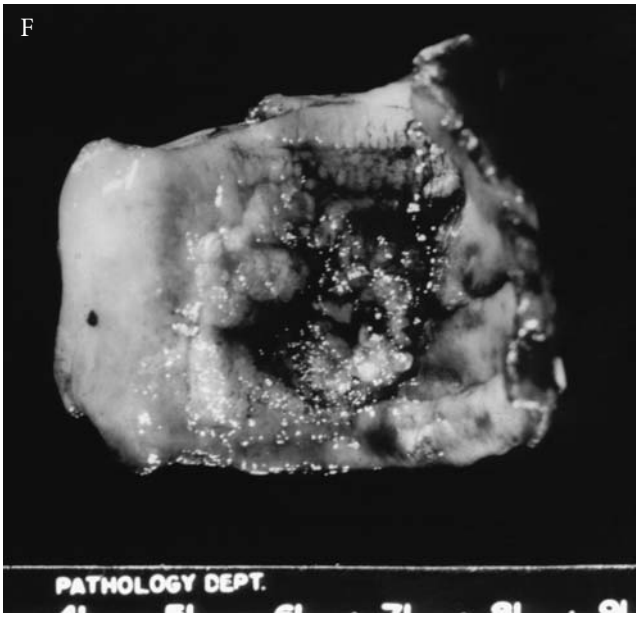


FIGURE 7-2 (CONTINUED) C, *Smooth-appearing exophytic lower tracheal squamous carcinoma in a 56-year-old male. Right upper lobectomy was done 15 years later for squamous carcinoma. Neither tumor recurred.* D, *Tomogram showing a large squamous lesion of the lower trachea* (between arrows) *in a 55-year-old male . The lower* open arrow *marks the carina.* E, *Surgical specimen from* D. *The base of tumor was less extensive than gross tumor and provided a microscopically clear margin.* F, *Ulcerating squamous cell carcinoma in a 57-year-old man. Adjacent lymph nodes were involved. Three years later, he developed pulmonary metastases and a second primary squamous carcinoma of the tongue.*

Table 7-2 Incidence of Primary Tracheal Tumors by Age

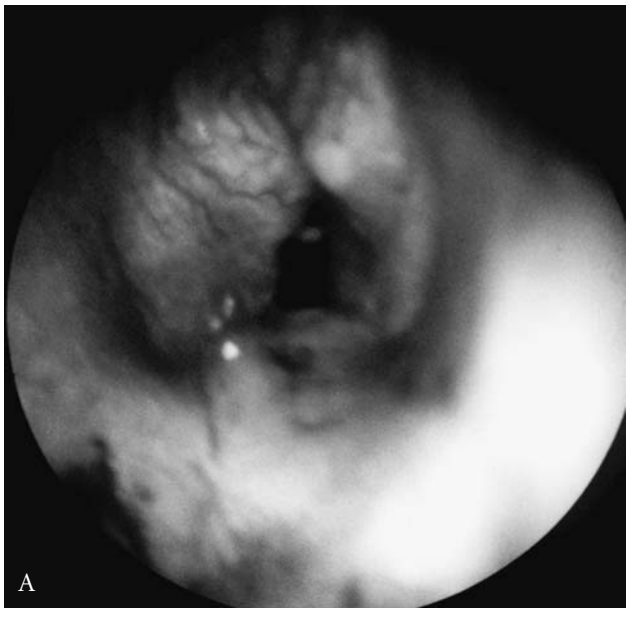
	Number of Tumors		
Age (years)	*Squamous*	*Adenoid Cystic*	*Other*
1–10	—	—	4
11–19	—	—	8
20–29	1	13	11
30–39	1	16	9
40–49	9	19	5
50–59	29	15	6
60–69	24	13	4
70–79	6	5	1

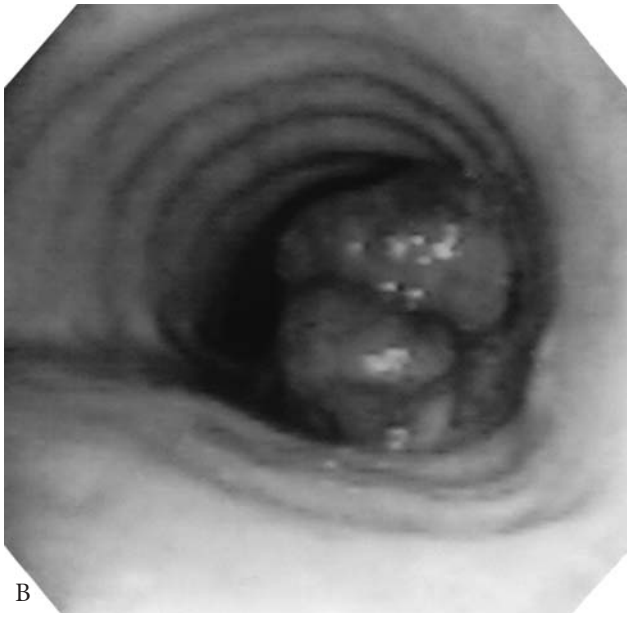
Adapted from Grillo HC and Mathisen DJ.[1]

tracheal tumor is histologically indistinguishable from that found in salivary glands, it is indeed rare for a single patient to present with tumors, either synchronous or metachronous, both in the salivary gland and in the trachea. The airway tumor may occur at any level of the trachea, but seems to be more prevalent in the lower trachea and carina. It occurs much less often in main bronchi and very rarely distal to that. Multiple tumors are extremely rare.

Adenoid cystic carcinoma presents grossly in a variety of ways (Figures 7-3, 7-4, 7-5, and Figures 7 through 11 [Color Plates 12, 13]). The lesions may be red and rubbery in appearance or granular and friable. One or all borders may appear sharply defined or the tumor may infiltrate diffusely. Frequently, adjacent to the main mass of tumor, which clearly projects from the mucosa, there is evidence of tumor infiltration beneath the mucosa or in the tracheal wall. The margins are often indistinct when examined with a magnifying telescope through a rigid bronchoscope. Mucosal elevation or friability is seen and new tumor vessels may be identified in the mucosa. Commonly, these cancers invade microscopically submucosally and perineurally for long distances beyond grossly visible disease. Microscopic pathology is described in Chapter 3, "Pathology of Tracheal Tumors." The lesion may be essentially circumferential within the trachea or may involve both main bronchi at the carinal level. Proximal tumors may involve the larynx in varying degrees. The extratracheal tumor mass may become bulky. Although these tumors may grow to considerable size extramurally into the mediastinum prior to recognition, they often displace adjacent structures before invading them, including the esophagus and pulmonary artery. I have seen the left pulmonary artery shown to be completely occluded by the pulmonary angiogram, but at operation found only compression, without invasion of the pulmonary arterial wall. The esophagus is more likely to be directly invaded by a posterior tumor, especially in its muscular wall. A recurrent laryngeal nerve may be defunctioned by direct invasion. Adjacent paratracheal regional lymph nodes on both sides may be involved, but apparently not as frequently as by SCC of the trachea. For a tracheal tumor, these should be considered as N1 nodes.

Late local recurrence of ACC, even 15, 20, or more years after apparent cure by surgery and irradiation, is a discouraging characteristic. Despite almost uniformly excellent early response to external irradiation treatment, recurrence usually follows if irradiation alone is used for treatment. Recurrence is seen at the prior site of the maximum bulk of tumor, usually between 3 to 7 years following treatment. Improved long-term outcome seems to follow combined surgical resection and full-dose radiotherapy, but these results must be regarded with reservation in view of the tumor's indolent clinical behavior and the small amount of information about long-term behavior.[6,11,12] Metastases to the lung occur all too frequently, but





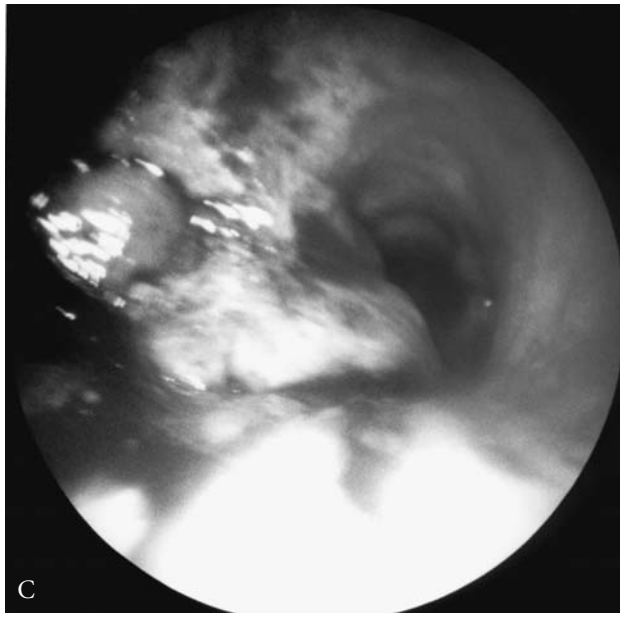


FIGURE **7-3** *Adenoid cystic carcinoma. Bronchoscopic views.* A, *Tumor invading the subglottic larynx in a 54-year-old man, at and above the cricoid level, with essentially circumferential extension. Resection with laryngeal salvage was impossible. The patient elected palliative neutron beam radiation to preserve laryngeal speech. Tumor recurred, as expected, in 6 years.* B, *Adenoid cystic carcinoma confined to midtrachea, 2 × 2.7 cm, in a 64-year-old man. The tumor, 4.5 cm from the carina, was removed by cervicomediastinal tracheal resection.* C, *Tumor at the carina and in the left main bronchus, also extending into the proximal right main bronchus. Left carinal pneumonectomy was done via a "clamshell" incision.*

may emerge late and enlarge slowly. Patients may remain asymptomatic for many years with multiple enlarging pulmonary metastases. Indeed, such behavior may justify surgical removal of a tracheal lesion despite the presence of numerous lung metastases, in order to prevent airway obstruction. Experience is small, but excision of pulmonary metastases has almost never produced cure or seemed to alter the course of the disease. Usually, metastases are too numerous for complete removal. Where a few metastases have been resected, other metastases have usually appeared later. Metastases to bone and other organs also occur.

In a minority of cases, although no unusual histologic characteristics were identified, ACC proved to be rapidly and widely aggressive. Manifestations in these few patients included local invasion of the central pulmonary artery, intrapericardial extension, multiple pleural metastases, and multiple rapidly-growing pulmonary or osseous metastases. The primary tumor in such a patient is not necessarily large.

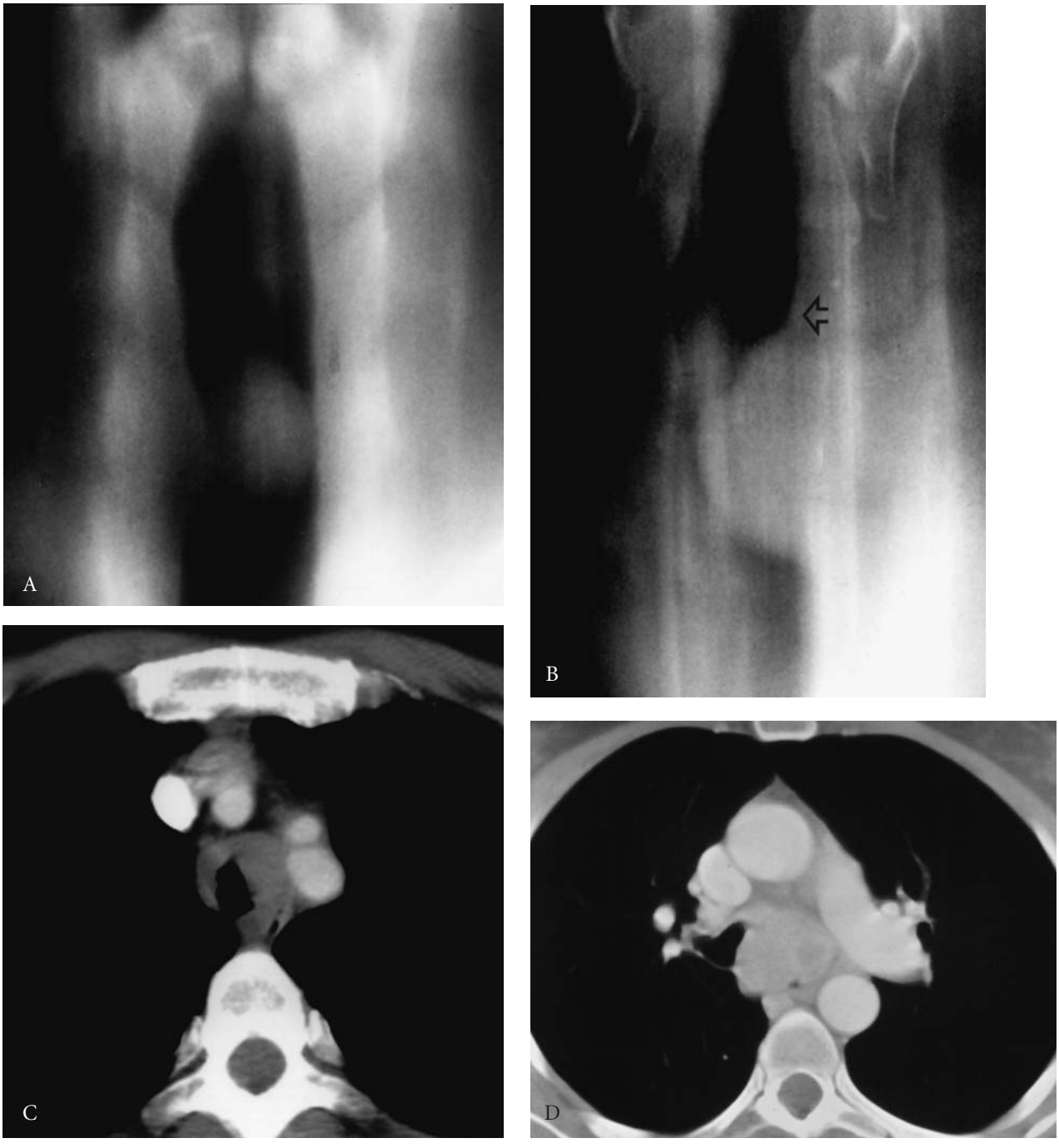
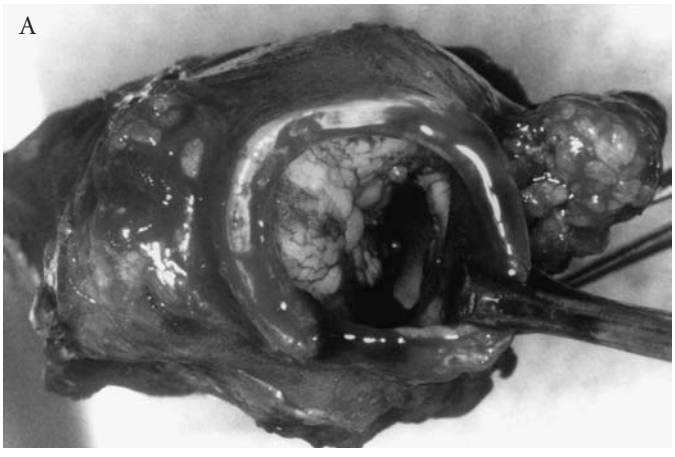
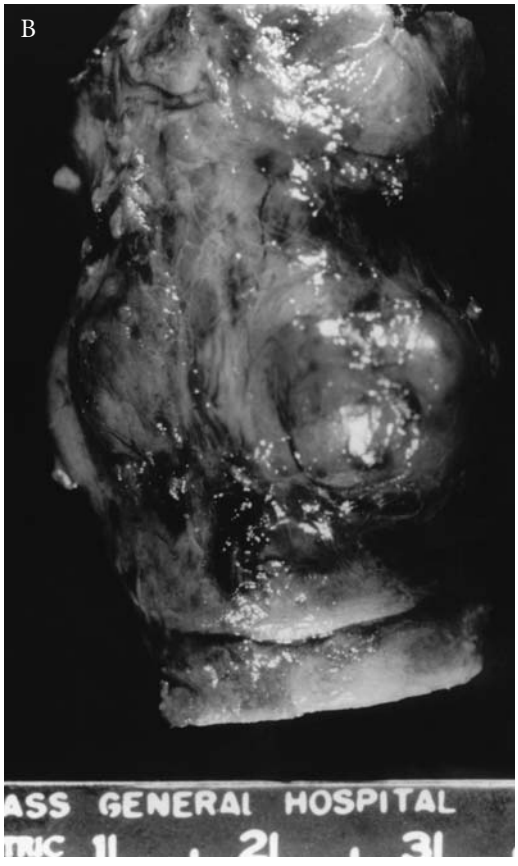
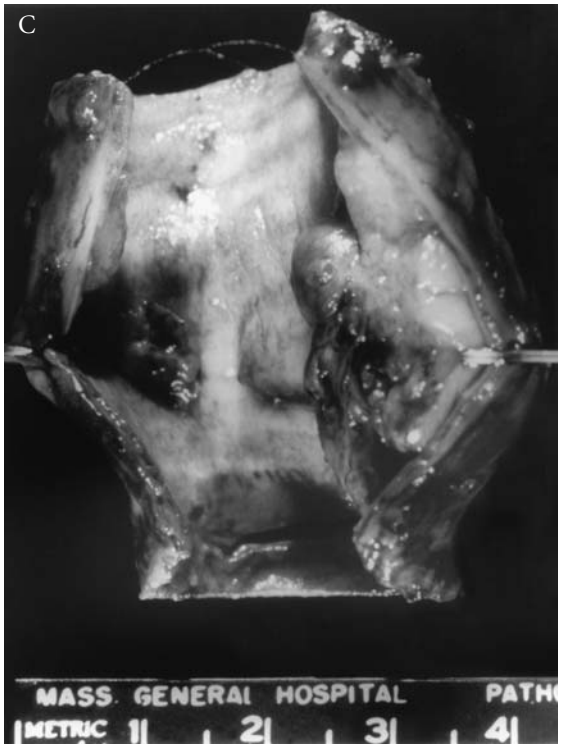


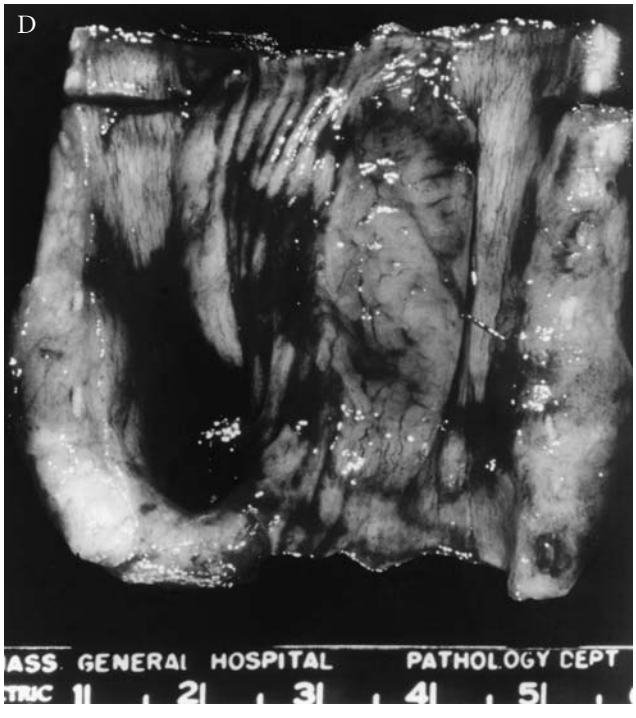
FIGURE 7-4 *Radiologic presentation of adenoid cystic carcinoma (ACC).* A, *Small, well-defined tumor of the upper trachea, delineated by tomography. The vocal cords and subglottic larynx are well outlined. The involved right wall of trachea is deformed. The right recurrent laryngeal nerve was involved. This 25-year-old man underwent cervicomediastinal resection and also received 5,000 cGy of irradiation postoperatively. He remained well and without recurrence 30 years later.* B, *Tomographic detail. Broad-based ACC in a 40-year-old woman extending into the cricoid cartilage on the left* (arrow). *The left side of the lower larynx was included in the resection. The left recurrent laryngeal nerve had to be resected. The trachea was bevelled to fit the line of laryngeal resection. Laryngeal structures are visible above. Vocal cords are widely abducted.* C, *Computed tomography scan in a 39-year-old woman shows the midtrachea encircled by ACC. She was treated by segmental tracheal resection.* D, *Large tumor at the carina obstructing the left main bronchus totally and the right bronchus subtotally. Left carinal pneumonectomy was necessary.*

FIGURE **7-5** *Gross surgical specimens of adenoid cystic carcinoma.* A, *Tumor with large extratracheal component and involved peritracheal lymph nodes.* B, *A 6.5 cm resection for an extensive tumor of the midtrachea in a 43-year-old man. Complete sternotomy plus extension into the right thorax was necessary for removal. Laryngeal release was also required. A tentative initial distal incision in the trachea was made, but was too close to the tumor.* C, *Open specimen of* B, *showing the intraluminal extent of tumor.* D, *Another extensive tumor in a 27-year-old man, involving distal trachea, carina, and left main bronchus. Left carinal pneumonectomy was necessary via bilateral thoracotomy, with elevation of the right main bronchus to proximal trachea. Laryngeal release helped because of the high level of anastomosis. Irradiation followed. The patient is now 22 years after resection.*









Over 40 years, between 1962 and 2002, 135 patients with ACC and 135 with SCC were seen at MGH.[6] In the second 20-year period, 97 with ACC and 98 with SCC were seen. Of these, 101 (75%) with ACC and 90 (67%) with SCC were resected.

A miscellaneous group of other *primary tracheal carcinomas* has also been encountered, usually with so few examples of each type that little can be said about general behavior patterns. These neoplasms include *adenocarcinoma* in the carina of a child at the base of a bronchogenic cyst. Carinal excision following prior removal of the cyst at another hospital led to cure. *Adenosquamous carcinoma* was encountered, involving both the trachea and the lower larynx, and was treated by cervical mediastinal excision with mediastinal tracheostomy. Another such tumor, involving the right main bronchus and carina, was treated successfully (15-year survival) by carinal resection and right upper lobectomy with anastomosis of the bronchus intermedius to the left main bronchus and the left main bronchus to the trachea. Two instances of *small cell carcinoma* of the trachea were encountered, reconfirmed on pathological review, with no involvement of adjacent lung and no nodal metastases. These patients remained disease free over many years after surgical resection plus conventional adjuvant therapy for small cell carcinoma. The rarity of primary tracheal small cell carcinoma was observed elsewhere.[13] A greater frequency of small cell carcinoma in the trachea, which was described in the past, probably included secondary invasion from the lung.[14]

Lymphoepithelial carcinoma (Schmincke tumor), more often occurring in the nasopharynx, has been seen in the trachea.[15] Treatment was resection and irradiation.

It appears that *melanoma* can occur primarily in the trachea. As in the patient reported by Duarte and colleagues,[16] our single patient had undergone removal of a small cutaneous lesion, without histologic record, many years before. Lower tracheal resection was done. However, the patient had no subsequent recurrence of melanoma in 13-year follow-up.

Except for 11 patients with *carcinoid tumor* of the trachea and carina, which we noted in 1978, carcinoid has not often been reported in the trachea.[17] Behavior of carcinoid in the trachea and carina appears to be much the same as in the bronchi, where it is much more common (Figure 7-6, and Figures 12 through 15 [Color Plate 13]).[18] The rarer atypical variety may be highly malignant. Typical carcinoids, when resected with limited but negative histologic margins, did not recur. In these cases, no regional nodes were involved. Carcinoid syndrome is seen only rarely with bronchial carcinoid, often, but not uniformly, in the presence of hepatic or other metastases. One patient with carcinoid syndrome from a tracheal tumor was referred with massive mediastinal lymph node metastases following resection of an atypical tracheal carcinoid. Block removal of lymph nodes temporarily controlled the syndrome until tumor recurred. Carcinoid appears to respond poorly to irradiation. We have not encountered Cushing's syndrome with tracheal carcinoids, possibly a reflection of the low incidence of this hormonal effect in these tumors.[19]

Histological examination of the wall of the trachea from which a typical carcinoid tumor arises usually demonstrates deep enough origin that it is impossible to remove the tumor definitively intraluminally with a laser or by other means (see Figure 3-12 [Color Plate 2] from Chapter 3, "Pathology of Tracheal Tumors"; Figure 7-6*F*). Only transient relief of obstructive symptoms can be obtained by debulking the tumor within the lumen. Full-thickness excision of the tracheal wall is required for complete removal. On the membranous wall, a carcinoid tumor of moderate size often extends through the posterior wall. Claims of cure appear to be either an illusion or such a rarity that harm may be done by encouraging delay in accomplishing simple curative surgical extirpation.

Mucoepidermoid tumor presents in the trachea and main bronchi, with malignant behavior in a small proportion of cases in most series.[20,21] In Heitmiller and colleagues' series from MGH, 3 of 18 patients with mucoepidermoid tumors of the trachea and bronchi were high grade and fatal within 16 months.[21] All the others survived (Figure 7-7). Nodal spread was infrequent in low-grade tumors. Two tracheal tumors were treated by sleeve resection, one by laryngotracheoplastic resection, and another underwent carinal

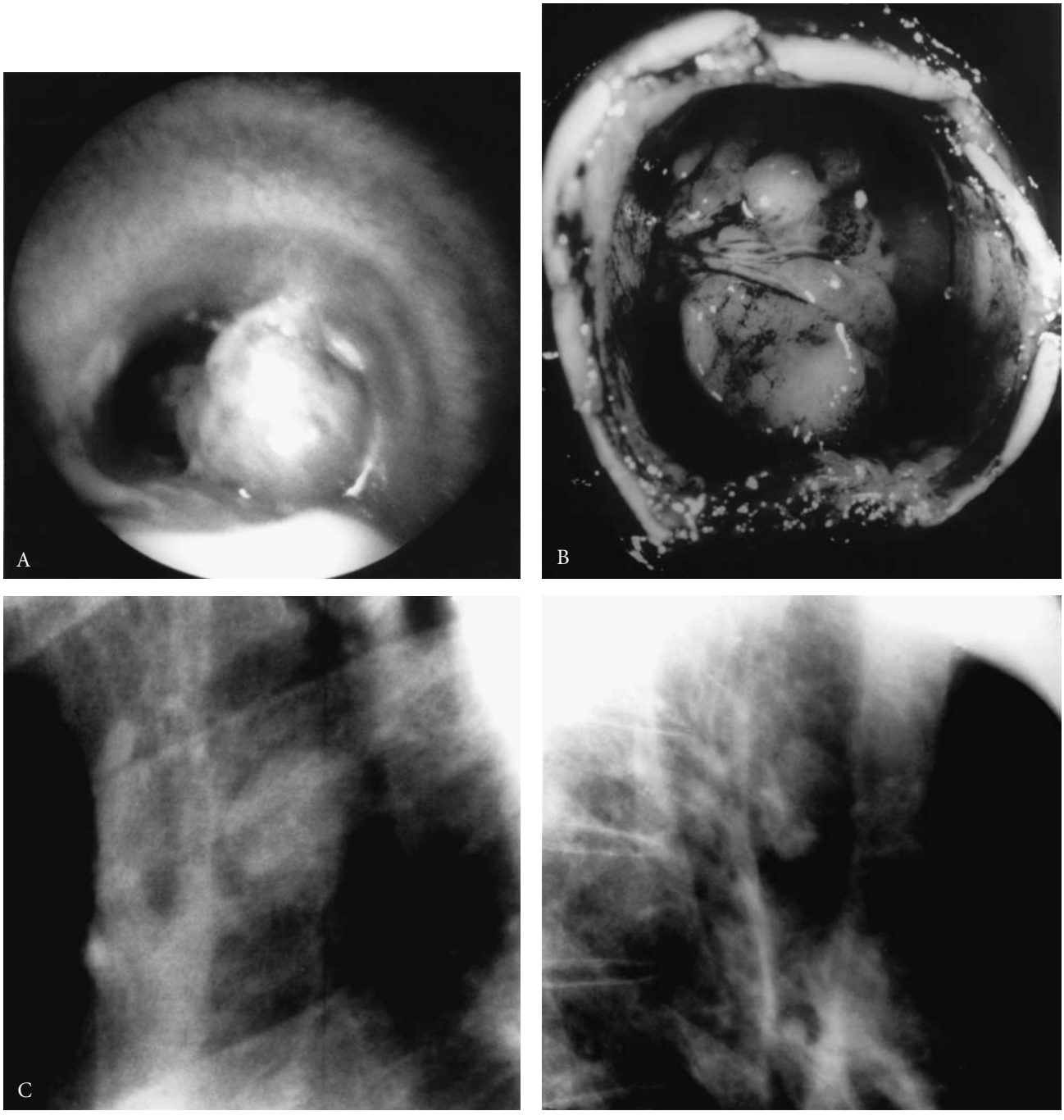
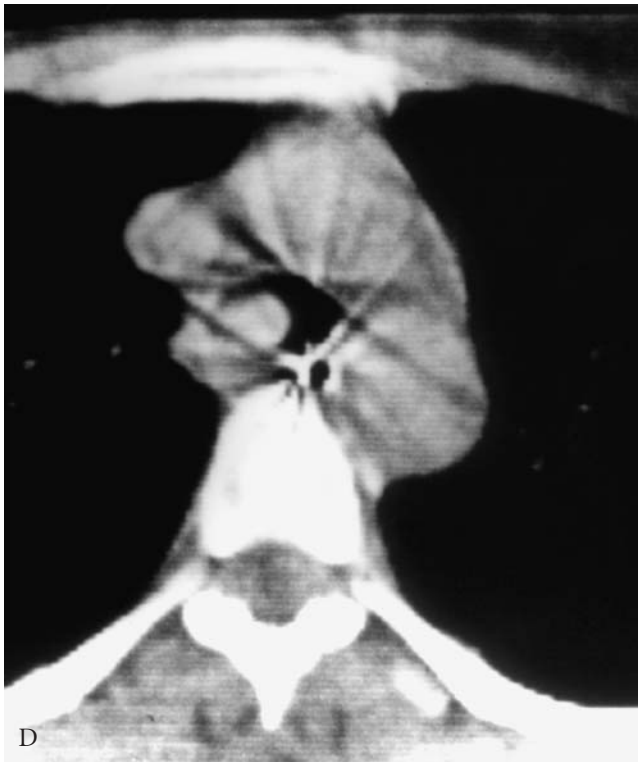
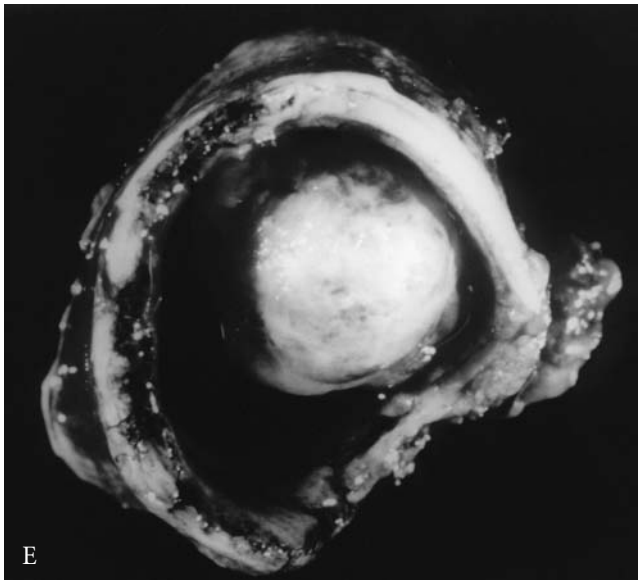


FIGURE **7-6** *Carcinoid tumors.* A, *Bronchoscopic view. Carcinoids are often smooth, rounded, and vascular in appearance.* B, *Irregular carcinoid located just above the carina.* C, *Radiographic details of the carina of a 31-year-old woman treated for "asthma" with progressive dyspnea for over a year. Anteroposterior view on* left radiograph *and lateral view on* right radiograph. *Both show the lower tracheal tumor clearly.*

pneumonectomy. Bronchial sleeve resection sufficed for another 5 patients, and lobectomy was needed in 7. Jensik, Faber, and colleagues made similar observations earlier. Irradiation treatment of high-grade tumors seemed ineffective.[20] In 31 children and adolescents, in whom the tumor was located in the bronchi, prognosis appeared to be better than in adults, with no recurrence seen after resection alone.[22]





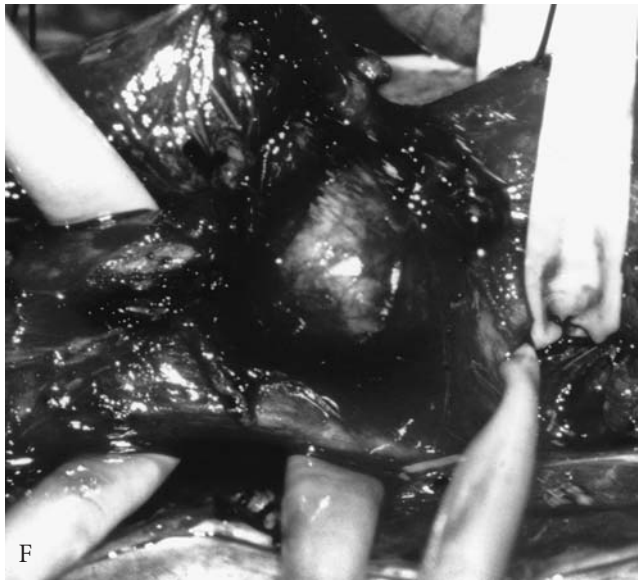
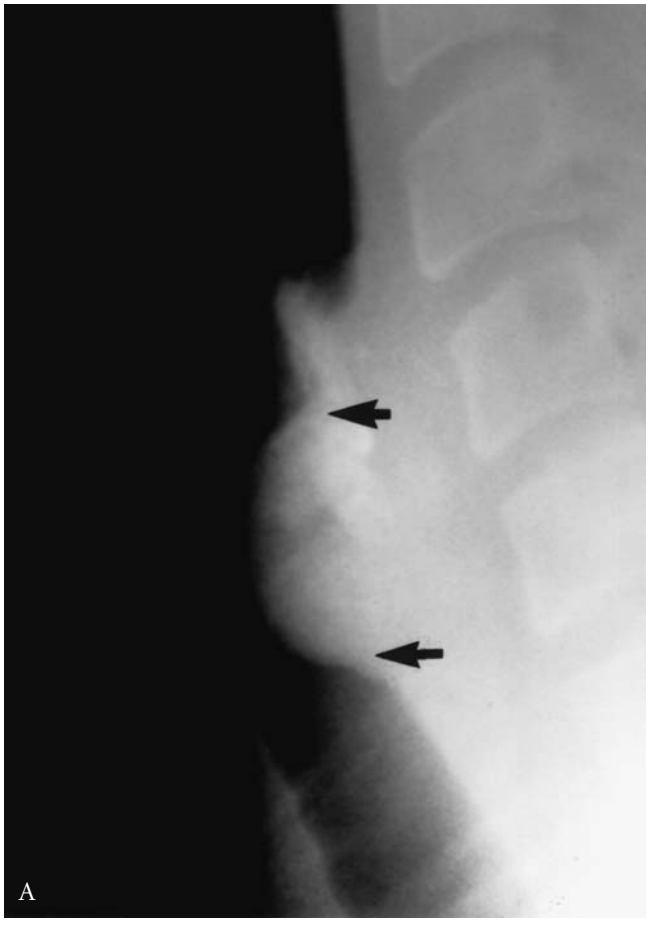


FIGURE 7-6 (CONTINUED) D, *Computed tomography scan of the patient described in* C. E, *Gross specimen of the patient in* C, *exhibiting a typical carcinoid in character and appearance.* F, *Carcinoid invading the carina in a 19-year-old female. Operative photograph shows protrusion of tumor through the membranous wall of the trachea. The left-hand tape encircles the trachea. The two on the right encircle the right main* (above) *and left main* (below) *bronchi. The futility of laser treatment, which she had on several occasions, is obvious.*

Two *pleomorphic adenomas* in the trachea were excised in our series (Figure 7-8, and Figure 18 [Color Plate 13]). These tumors are rare, may occur in children, and produce obstructive symptoms. They are mixed tumors of salivary gland type with varied mixtures of epithelial and stromal components. By 1992, 19 cases had been gathered.[23] Similar tumors in the bronchus were first described by Payne and colleagues.[24] Complete surgical excision and reanastomosis is the proper treatment. One of our patients had a similar tumor excised years before from a salivary gland, but there was no evidence then or since of metastatic disease. It was, therefore, classified as a primary tracheal tumor. Malignant features evidently occur, but were not present in these patients.



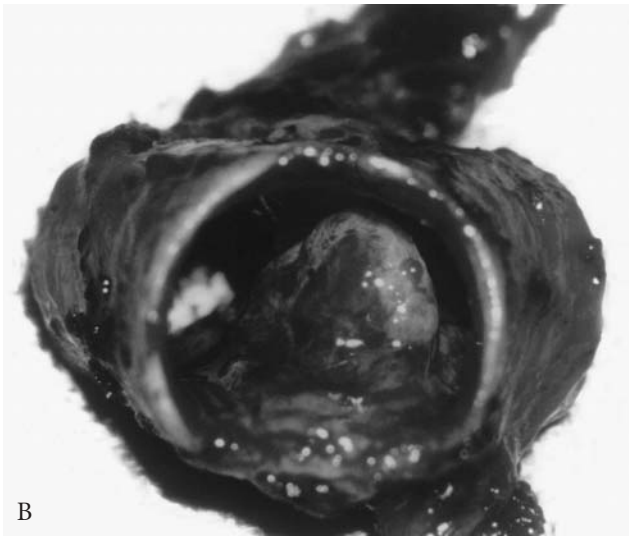
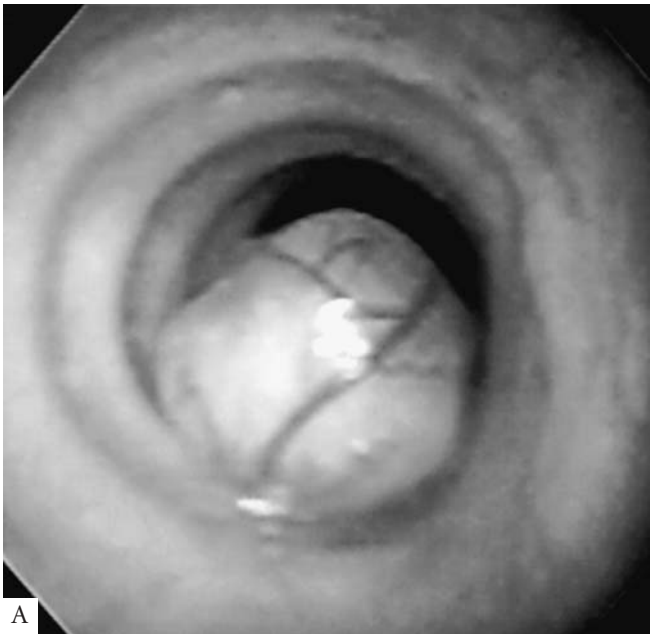


FIGURE 7-7 *Mucoepidermoid tumors.* A, *Lateral neck roentgenogram in a 28-year-old woman showing a posterior lesion* (arrows) *involving the subglottic larynx and upper trachea. Laryngoplasty was done, advancing the posterior membranous tracheal wall to resurface the posterior larynx after tumor excision. Both recurrent nerves remained intact.* B, *Carinal resection for mucoepidermoid tumor in a 34-year-old man, viewed from the tracheal end of the specimen. Twelve year follow-up was without recurrence.*



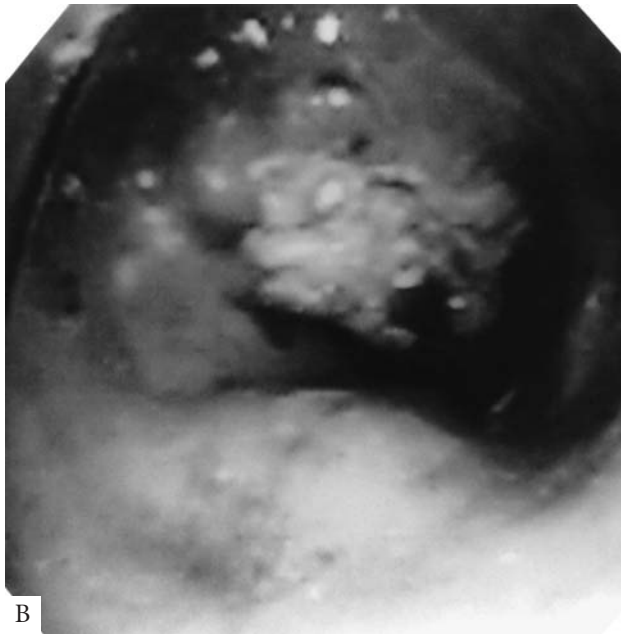


FIGURE 7-8 A, *Bronchoscopic view of pleomorphic adenoma. This true adenoma of the trachea is very rare.* B, *Another pleomorphic adenoma, with areas of adenocarcinoma present.*

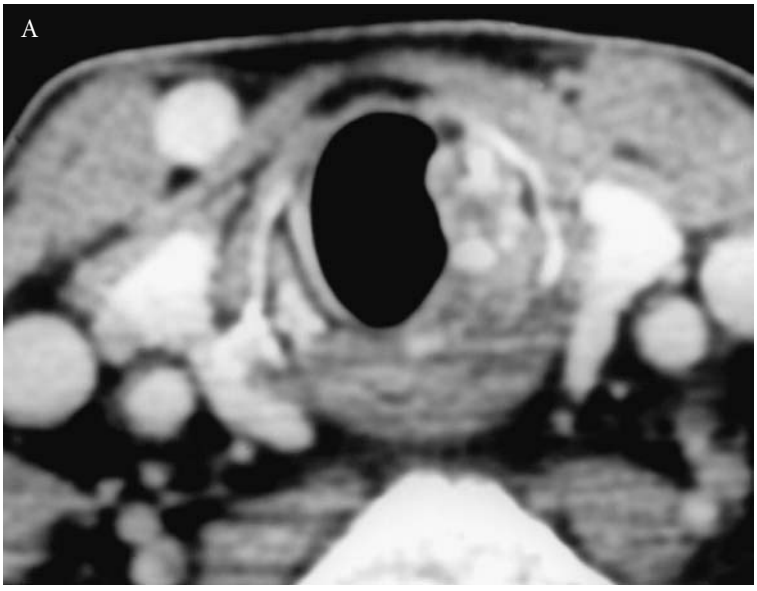
Squamous papilloma of the trachea may occur as a solitary, moderate-sized lesion, which should be removed by segmental tracheal resection. Naka and colleagues in 1993 found reports of 5 solitary tracheal papillomas, 7 in the main bronchus, and 41 in a lobar bronchus.[25] They proposed surgical resection for wide-based or poorly-defined tumors or for suspected malignancy. Tracheal polyps of inflammatory origin which occur as solitary lesions may also be obstructive.[26]

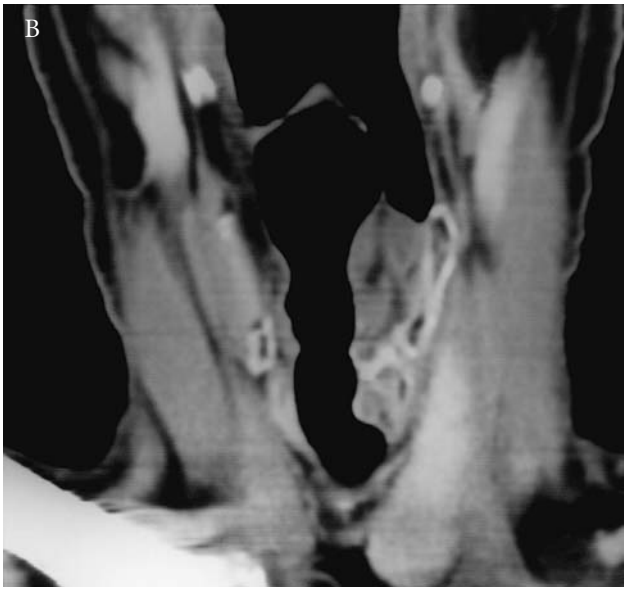
Multiple squamous papillomatosis also occurs in many locations in the trachea and at the carina. A fortunately-rare form produces multiple papillomas throughout the distal tracheobronchial tree, with obstructive septic consequences. In children, squamous papillomas of the trachea behave in benign fashion and tend to regress with adolescence (see Chapter 6, "Congenital and Acquired Tracheal Lesions in Children").[27] Their occurrence is related to human papilloma virus and antiviral treatment has been employed. These tumors were treated by repeated endoscopic removal using, in various eras, electrocoagulation, cryosurgery, and now, laser—all seemingly effective. Multiple lesions in adults, on the other hand, may be verrucous lesions, which cover large areas. The process may be so extensive that surgical resection is not possible. This is one of the few clear-cut indications for repetitive laser treatment. The role of photodynamic therapy remains to be clarified. Invasive carcinoma is not uncommon deep within these lesions.

Cartilaginous tumors, including *osteochondroma*, *chondroma*, *chondroblastoma*, and *chondrosarcoma*, are very rare in the trachea and only somewhat more frequent in the larynx.[28] Huizenga and Balogh found 10 cartilaginous tumors in 5,000 laryngeal neoplasms.[29] Peak incidence is from 40 to 70 years of age. In the larynx, about 70% arise from the cricoid, most frequently from the posterior plate. The tumor is submucosal and produces obstructive symptoms (Figure 7-9, and Figures 16, 17 [Color Plate 13]). A mass may be palpated in the neck. Biopsy is difficult because the tumor is firm and normal mucosa is likely to be obtained. The recurrent nerve is not initially affected, but voice change occurs due to tumor bulk. Hemoptysis is very rare. These lesions are usually chondrosarcomas of low grade and often are indolent in behavior. Treatment is surgical excision with every effort made to save the larynx. Techniques must be individualized. The reconstructive approach is outlined in Chapter 25, "Laryngotracheal Reconstruction." With conservative approach to maintain laryngeal function, recurrence must be watched for over a long term, with likelihood of later re-resection necessary years later. Salvage laryngectomy is eventually needed in some cases. With recurrences, atypical areas and dedifferentiation are seen.[28,30,31] Chondrosarcoma does not respond to radiotherapy.

Tracheal chondral tumors seem to be even less common than laryngeal tumors. Limited experience suggests that these are most common in older males.[32] They may vary from benign chondroma to chondrosarcoma of increasing malignancy (Figure 7-10). Lesions may be largely intraluminal or extend through the tracheal wall.[33–35] Nonproductive cough is followed by dyspnea, first on exertion and then at rest, with wheezing—especially on recumbency—late in the course. With chest x-rays initially "clear," the diagnosis of asthma is frequently carried for a long period. A medical student with a tracheal chondroblastoma was retrospectively identified as having had tumor present radiologically for 7 years (Figure 7-11). Cartilaginous tumors are radiologically defined on all imaging modalities. Calcification is frequently seen as well as destruction of normal cartilages, especially in the larynx. A bulky extraluminal mass may be visible. Treatment is surgical excision with tracheal reconstruction. The approach is directed by the location and extent of tumor (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection," Chapter 25, "Laryngotracheal Reconstruction," and Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). Complete excision is necessary to prevent recurrence and hence should be a patient's first procedure. Bronchoscopic debulking is not definitive treatment. Further, tumors apparently progress in degree of malignancy or transform from being benign to malignant.[36]

True cartilaginous tumors are distinguished from hamartomas by the absence of other tissue elements that are seen in hamartomas: lipomatous, epithelial, and lymphoid. Our patients with chondroma





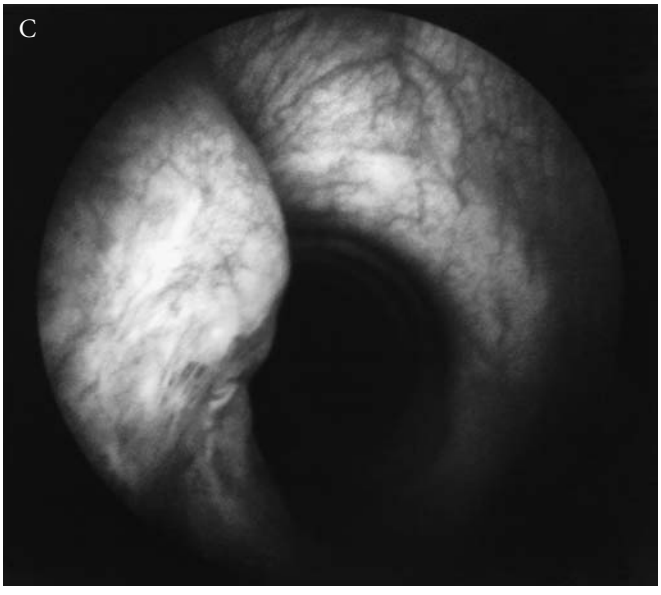


FIGURE **7-9** *Chondrosarcoma of larynx.* A, *Cervical computed tomography (CT) scan showing the low-grade tumor invading the laryngeal wall, notably the left cricoid cartilage, in a 64-year-old man. Calcification is notable in the tumor. Treated by laryngoplastic resection and reconstruction.* B, *CT reconstruction showing the extent of tumor.* C, *Bronchoscopic view of the same tumor. The tumor is covered by mucosa. Note tracheal rings distally on the right.*

and chondroblastoma of the trachea had no recurrence after complete resection with primary anastomosis. A low-grade, bulky chondrosarcoma treated by resection developed multiple, slow-growing pulmonary metastases over many years, but had no tracheal recurrence.

Hemangioma of the airway occurs, especially in children, in the subglottic region of the airway.[37] Half of these children also have hemangiomas elsewhere. The lesions are generally characterized by proliferative endothelial cells. Although lesions regress with age, they may require treatment because of their location either in the lumen of the airway or as an extrinsic compressive mass. Lymphangioma can on rare occasions similarly obstruct the trachea by pressure. Biopsy is to be avoided. A conservative approach is advised. Treatment has included temporizing tracheostomy to await regression, laser, corticosteroids, and in the past, irradiation. Laser therapy generally leads to remission.[38,39] Interferon has shown effect but neurological complications may occur.[40]

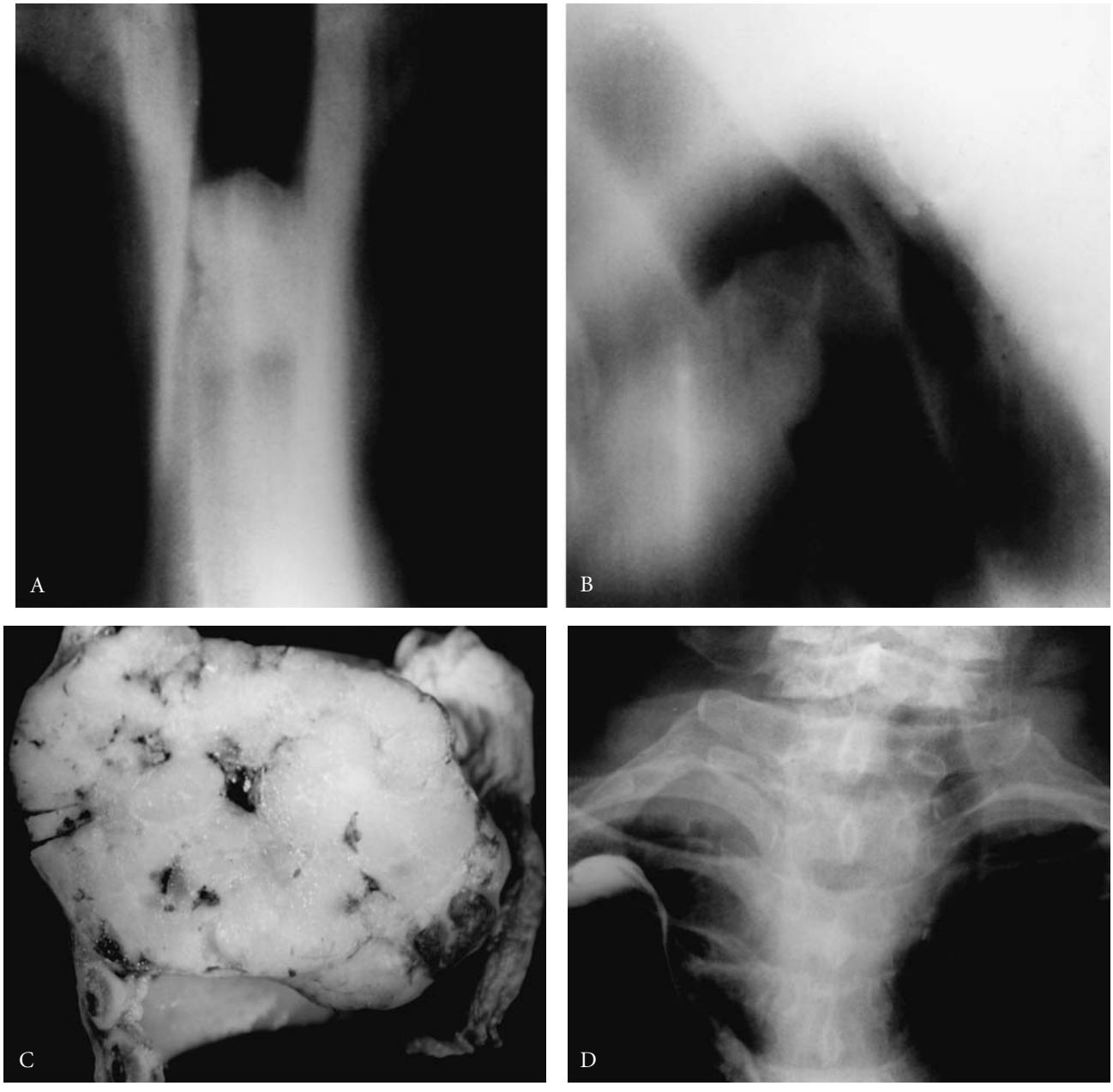


FIGURE **7-10** *Chondral tracheal tumors. Benign chondroma causing nearly total obstruction, in a 69-year-old musician who suffered dyspnea and stridor for 9 months before diagnosis of obstruction by flow volume loop. Tomograms show in* A, *anteroposterior and* B, *lateral views, how completely the lesion filled the tracheal lumen.* C, *The cut surgical specimen shows tumor originating from the anterior tracheal wall and abutting the membranous wall.* D, *Roentgenogram shows a slowly progressive low-grade chondrosarcoma, which originated in the tracheal wall, markedly displacing the trachea, brachiocephalic vein, and filling the upper mediastinum.*

An extremely rare lesion, but quite different, is *arteriovenous vascular malformation* in the anterior and middle mediastinum, which may present within the tracheal lumen as an obstructive lesion (Figure 7-12). These are not mediastinal hemangiomas, which are most often quite discrete.[40,41] The arteriovenous vascular malformation is fed by multiple arteries and entwines the mediastinum with a network of vasculature.[40,42] Such malformations are presumed to be congenital and not true tumors. Unless thrombosis has occurred, the vascular endothelium is quiescent, rather than proliferative as in hemangiomas.

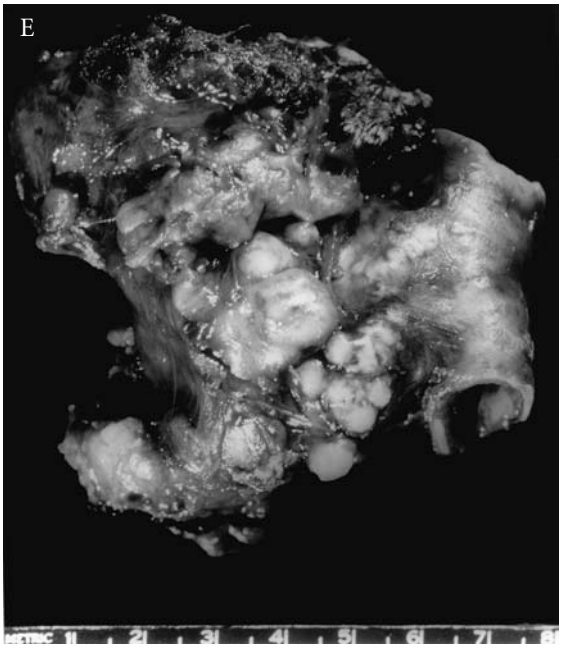
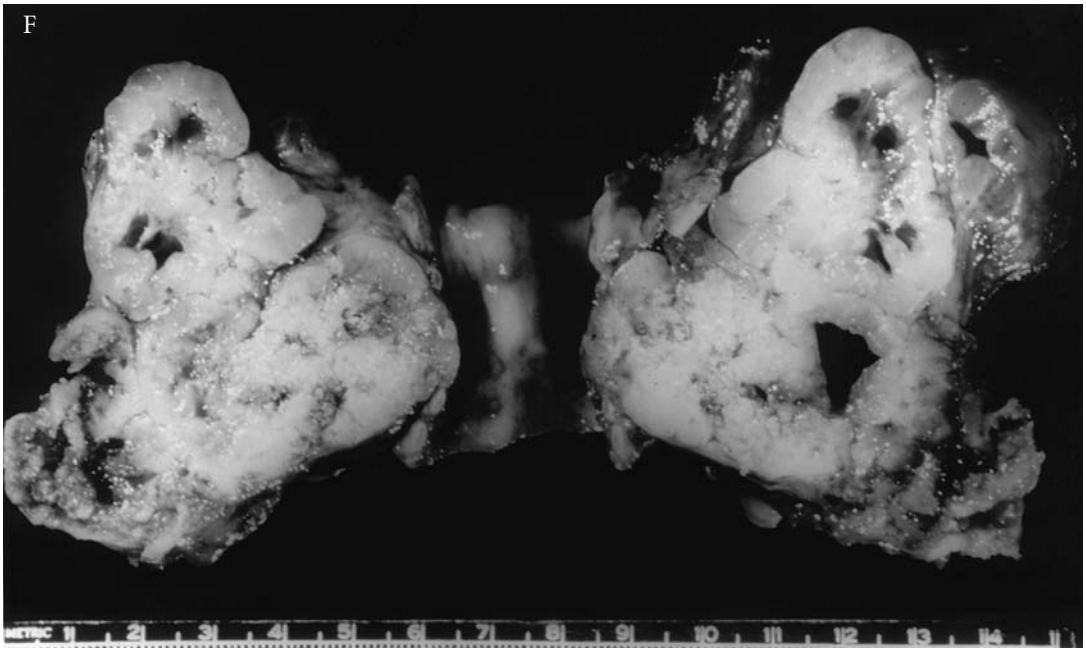


FIGURE **7-10** (CONTINUED) E, *Gross specimen, excised via a cervicomediastinal approach. Concurrent carotid endarterectomy was performed.* F, *The same specimen opened. The patient developed pulmonary metastases and died many years later, but without airway obstruction.*



In 2 patients, the malformation extended from the thoracic inlet to the diaphragm and was fed by multiple arterial branches. These included thyrocervical, internal thoracic, right coronary, a branch of right subclavian, and bronchial arteries. The huge resulting shunt enlarged the azygos vein and the superior and inferior vena cava. The malformation protruded through the wall of the trachea in both, visible bronchoscopically as a plexus of pulsating vessels. One patient had undergone right upper and middle lobectomies in childhood for hemangiomatous lesions not further defined in prior records, and the other patient had had cutaneous vascular lesions removed in childhood. Both were 25 years of age when seen. In one patient, who had severe obstructive symptoms, management was commenced with an extensive program of embolic obliteration of multiple feeding vessels from the neck, mediastinum, and right coronary artery. After

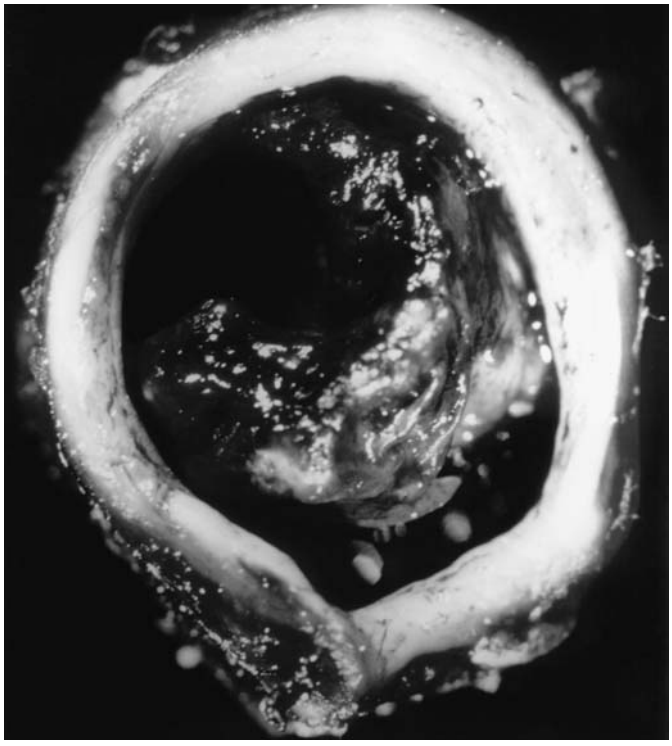


FIGURE 7-11 *Chondroblastoma in a 22-year-old medical student, treated for years for "asthma." Retrospectively, a tracheal tumor was identified on x-rays taken 7 years earlier. The impression of the endotracheal tube on the soft intraluminal tumor is seen.*

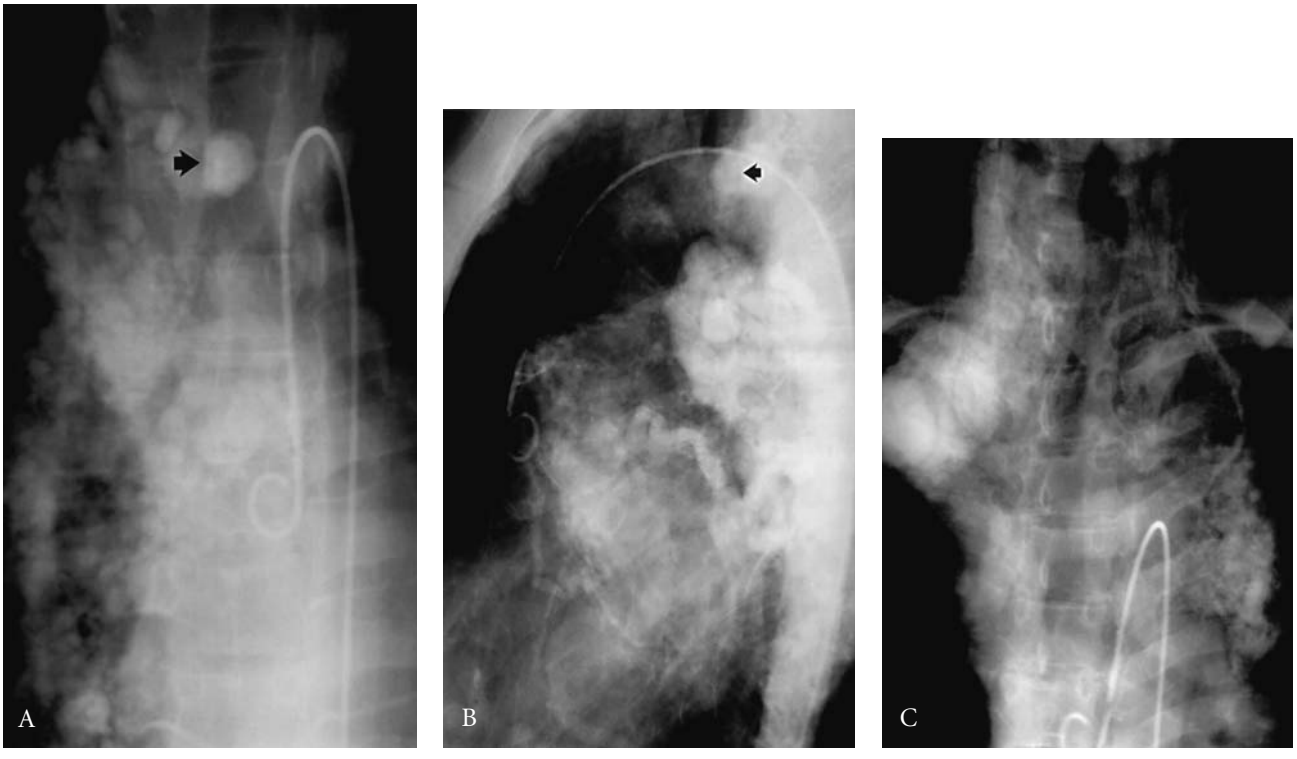


FIGURE 7-12 *Arteriovenous vascular malformations (AVM) of mediastinum involving trachea. Angiograms show widespread involvement of the mediastinum and neck.* A, *A 25-year-old man with protrusion of AVM into the lower trachea* (arrow) *producing respiratory obstruction. Managed by multiple, repeated embolization of feeding arteries followed by successful tracheal segmental resection. His airway remained clear 19 years later. See text.* B, *Lateral view of the patient in* A. *The* arrow *indicates tracheal invasion.* C, *A 25-year-old woman with more diffuse tracheal compression by very extensive AVM. Intratracheal protrusion of vessels was also present. See text.*

reducing flow in this way, meticulous dissection accomplished resection of the involved portion of the trachea. The basic malformation was not resectable in its entirety. Seven years later, multiple coronary arterial venous fistulae were ligated at another hospital. Since then, the patient has continued to do well. The second patient was not initially sufficiently symptomatic to justify such an extensive and somewhat perilous approach. Years later, she presented at another hospital where embolization failed and surgery was unsuccessful. She died there from intratracheal hemorrhage adjacent to a tracheostomy.

Glomus tumor, found most commonly in skin and subcutaneous tissue and especially in fingers under the nails, occurs in the trachea only rarely. It arises from cells related to the glomus bodies, which have smooth muscle characteristics.[43] Symptoms in trachea are cough, dyspnea, and hemoptysis. The tumors are benign and treated by limited but complete resection in most cases. Fourteen tracheal cases had been collected by the year 2000.[44] The tumors may be very vascular. Initial examination may suggest carcinoid or hemangiopericytoma. Immunohistology clarifies the differential diagnosis. We have also encountered a very diffuse form, which contiguously involved most of the trachea with extension into right and left main bronchi (Figure 7-13). The patient showed early response to external irradiation and brachytherapy, but required a stent to maintain a satisfactory lumen. Long-term palliation cannot yet be assessed, although he continues to do well 3½ years after treatment.

Granular cell tumor, formerly termed *granular cell myoblastoma*, is predominantly believed to arise from Schwann cells, and is of muscle origin (Figure 7-14). The tumors arise most commonly in the tongue and head and neck area. A small number occur in the airways, and most of these are in the larynx or bronchi. Burton and colleagues collected 30 tracheal instances in 1992.[45] Age of occurrence was from 6 to 56 years and the tumor was more often found in women. Many patients are black.[46] Granular cell tumors can be multiple in the airway or elsewhere, and malignant manifestation is only very rarely described. Tracheal lesions appear most often intraluminally, but can be located or extend extraluminally. Tumor cells are present deep in the tracheal wall in all the larger tumors.[47] Endoscopic resection of the smaller tumors has produced some long-lasting clearances. However, the frequency of recurrence (over half), lack of knowledge of the tumor depth in unresected patients, the indolent course of tumor, late recurrences, and the need

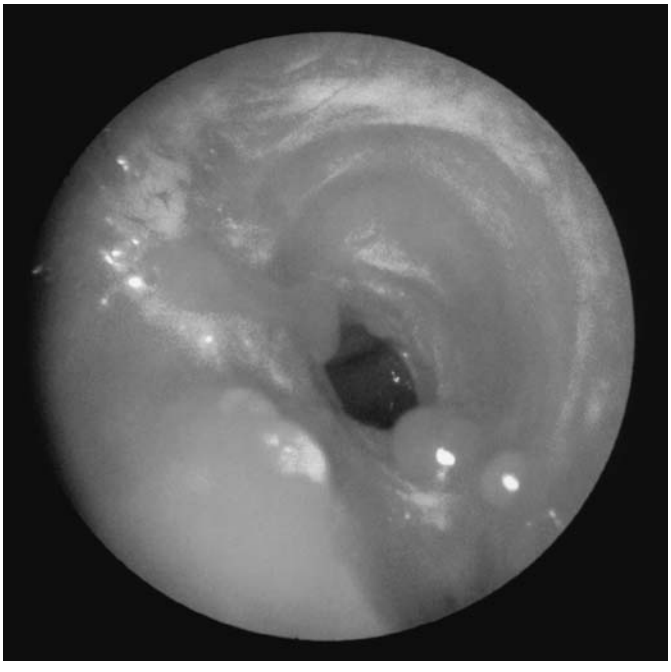


FIGURE **7-13** *Diffuse glomus tumor. Bronchoscopic view. Widespread nodular disease is evident. Treated by irradiation because of extent. See text. Also, see Figure 19 (Color Plate 14).*

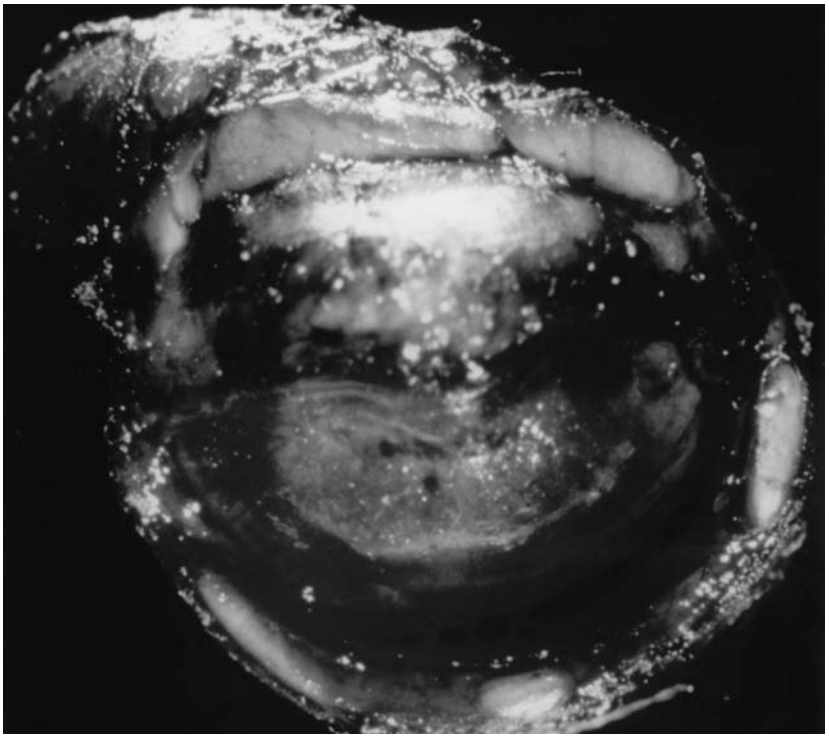


FIGURE 7-14 *Granular cell tumor of the upper trachea, posteriorly based and extending over the cricoid cartilage. An arc of posterior laryngeal mucosa and submucosa is visible at the top of the photograph. Only a slit of airway remains anterior to the tumor. The patient, a 14-year-old girl, was critically obstructed.*

for very long-term bronchoscopic follow-up after endoscopic removal, favor primary limited but complete sleeve resection of the trachea or bronchus. Patients often present with "asthma," that is, shortness of breath and wheezing, rather than hemoptysis. One 14-year-old girl with critical obstruction urgently required laryngotracheal resection and reconstruction (see Figure 7-14). Follow-up bronchoscopy revealed a second granular cell tumor in the bronchus intermedius, which was resected without loss of lung tissue. Multiple granular cell tumors elsewhere have also been reported. The question of irradiation hardly arises with this particular pattern of behavior, and when irradiation has been given, its effect has been uncertain.

A lesion that occurs infrequently in the lung and sometimes in the gastrointestinal tract is variously termed *postinflammatory tumor*, *pseudotumor*, *plasma cell granuloma*, *xanthoma*, *fibrous histiocytoma*, and by composite names formed from these terms (xanthomatous pseudotumor, plasma cell tumor, fibroxanthoma, xanthogranuloma). Histologically, the lesions show proliferative mature plasma cells, reticuloendothelial cells, granulation tissue stroma with proliferative fibroblasts, and contain lymphocytes and fat-laden mononuclear cells. What is still not clear is whether these lesions are true tumors or in fact inflammatory in nature (Figure 7-15). Tumors which might be considered inflammatory on bronchoscopic biopsy may prove to be neoplastic and invasive on final examination.[48] Depth of invasion into and through the cartilage varies. Matsubara and colleagues have subdivided these lesions in the lung into three groups.[49] They question the relationship of these lesions, which they consider to be of inflammatory origin, with malignant fibrous histiocytoma. In the airway, the tumor causes cough, dyspnea, wheeze, hemoptysis, and pulmonary changes due to obstruction.[50,51] Tumors occur at any age but more often in children and young adults. Intraluminal tumor may be polypoid or sessile.

Criteria for benignity or malignancy do not seem to be well established.[52] The lesions can extend into the mediastinum and the thyroid gland, giving rise to diagnosis of *malignant fibrous histiocytoma*.[53,54] This term is also used to describe high-grade sarcoma, adding to confusion. Laryngeal tumors may more often appear malignant. On the other hand, complete excision of tracheal histiocytoma usually results in

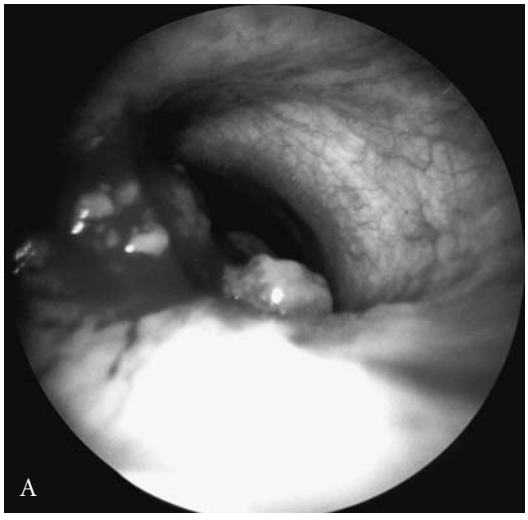
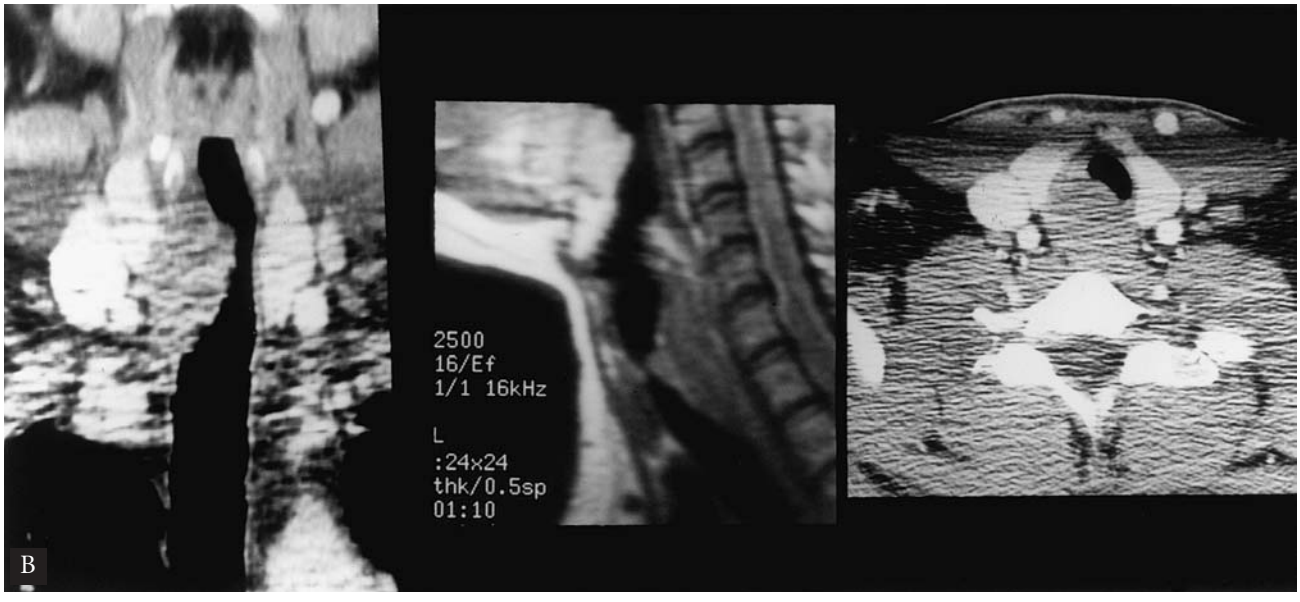


FIGURE **7-15** *Inflammatory pseudotumor of the trachea in a 39-year-old woman, extending into the posterior subglottic larynx.* A, *Endoscopic view. The cricoid is visible anteriorly, tracheal rings distally, and proximal extension of lesion posteriorly.* B, *Magnetic resonance scan and reconstructions showing localization of the lesion. From* left *to* right*:* 1) *subglottic and linear extent, anterior view;* 2) *lateral view showing the posterior mass high in the airway;* 3) *cross section of airway partly obstructed by bulky mass, with thyroid lobes on either side. Removal of tumor required resection of 4 cm of trachea with posterior laryngeal mucosa and submucosa, and sacrifice of invaded right recurrent laryngeal nerve. Temporary tracheostomy was done. There has been no recurrence.*



cure.[47,48,53–55] Incomplete excision is likely to result in local recurrence. Increasing the etiologic mystery are reports of resolution of pulmonary lesions following treatment with cortisone or spontaneously.[56,57] This last raises a question about the suggested beneficial effect of irradiation or of cortisone.[58]

In 2 patients with very large pseudotumors of the trachea, excision produced relief without recurrence. Extension of the lesion proximally over the posterior cricoid plate submucosally required excision of the lesion anterior to the cartilage and reconstruction with a tongue of distal tracheal membranous wall (see Figure 7-15). Sleeve resection of an obstructed left main bronchus for another lesion allowed salvage and gradual effective recovery of the chronically inflamed lung. A measured decision must always be made on whether a long obstructed lung can be usefully salvaged.

Primary *neurogenic tracheal tumors* are also uncommon. Horowitz and colleagues found reports of 3 *neurofibromas* (not with von Recklinghausen's disease) and 12 *neurilemomas* (Schwannoma), to which they added another.[59] Most were in the lower trachea and produced cough, symptoms of tracheal obstruction, or hemoptysis. Segmental resection was curative. Endoscopic resection may well result in long-delayed recur-

rence.[59] Such a recurrent tumor was dumbbell in form and necessitated carinal resection and reconstruction. Stack and Steckler added an upper tracheal case in 1990, the nineteenth reported patient.[60] All were benign. A patient with recurrent *plexiform neurofibroma* of the lower trachea, following previous local enucleation elsewhere, attained cure (24-year follow-up) by extended resection of the trachea, including a portion of esophageal wall and an isolated area of metastatic seeding in the prior thoracotomy wound (Figure 7-16). Local enucleation of tumors from the tracheal wall is generally not an appropriate method of management.

Two patients with tracheal *paraganglioma*, which involved the posterior wall of the trachea intimately adjacent to the cricoid cartilage, were treated by segmental excision of the upper trachea, removal of a portion of the posterior cricoid plate, and in one, a portion of the muscular wall of the esophagus. There was no local recurrence. One, however, affected with familial incidence of multiple tumors, later presented with bilateral carotid body tumors. His trachea remained clear. This tumor is not common and was first reported in the trachea in 1956.[61]

Other mesenchymal tumors occur in the trachea. These include *leiomyoma*, *lipoma*, and *fibroma* (Figure 7-17).[62–64] Eleven tracheal leiomyomas have been reported, with calcification in one, just as occasionally occurs in esophageal leiomyomas.[63] The majority of *hamartomatas* are found in pulmonary parenchyma, with less than a quarter presenting endobronchially and rarely in the trachea.[65,66] Since these lesions rarely produce hemoptysis, the patients often present with a high degree of obstruction after prolonged treatment for "asthma." In a rare case where a benign connective tissue tumor is based on a narrow pedicle, endoscopic removal alone may produce cure. Such patients, however, should be observed bronchoscopically for years for recurrence. Where there is any degree of involvement of tracheal wall, segmental tracheal resection is advisable. Resection is definitive, and given the limited resection required, carries little morbidity in experienced hands.

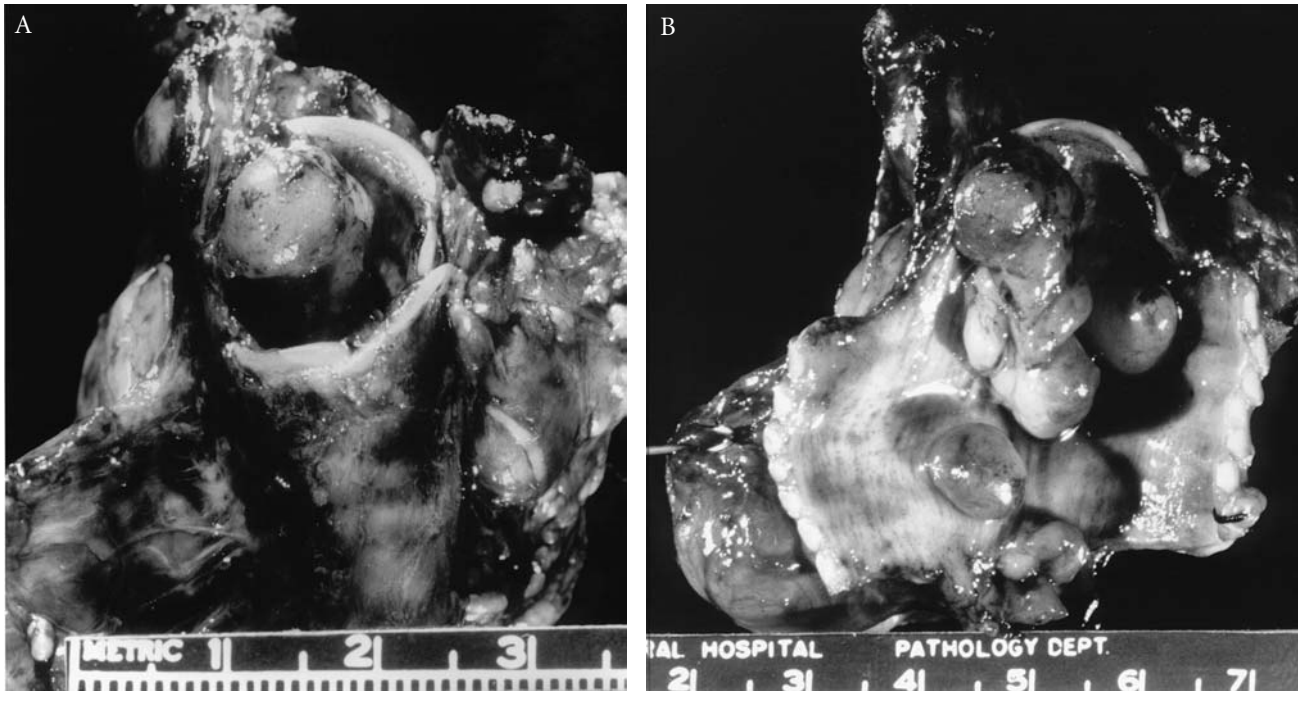
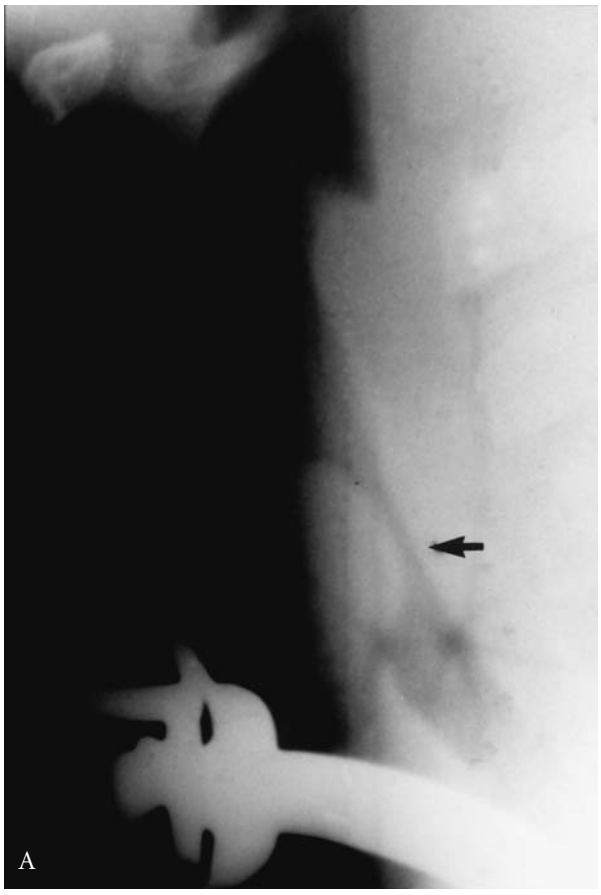


FIGURE 7-16 *Plexiform neurofibroma of the lower trachea, in an 11-year-old boy. Resected by enucleation 6½ years previously. There was also an incisional recurrence. The patient is tumor-free 24 years after sleeve resection.* A, *Resected 4.5 cm specimen with surrounding tissue. Lobule of tumor protrudes from the cut end of the trachea.* B, *Opened specimen showing the extent of recurrent tumor.*



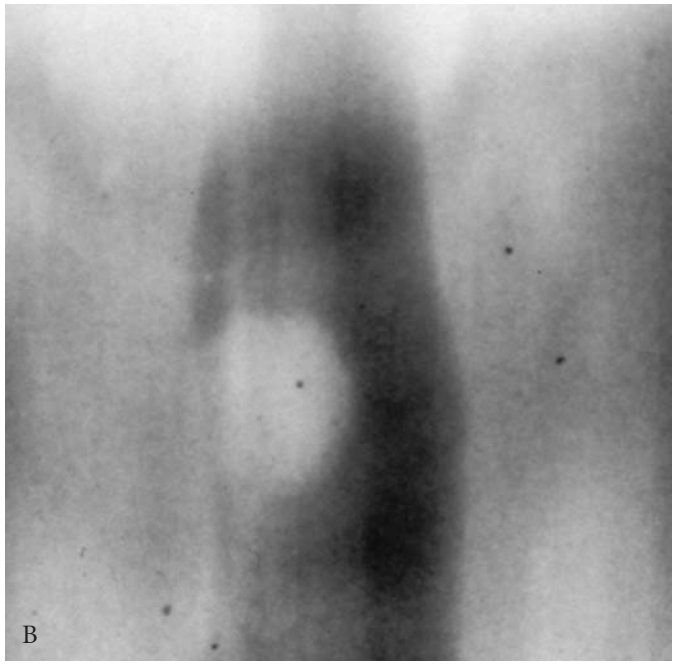


FIGURE 7-17 A, *Lateral tomogram of fibroma high in the trachea* (arrow). *A tracheostomy had been done unnecessarily prior to referral.* B, *Antero-posterior tomogram showing a low-grade spindle cell sarcoma in the uppermost trachea, just below the cricoid, in a 15-year-old female. The right side of the cricoid was bevelled to provide a wider margin. No recurrence occurred in 10-year follow-up.*

Extramedullary solitary *plasmacytomas* occur most often in the head and neck. In 1995, Logan and colleagues reported the eleventh such tracheal patient.[67] It is not known how likely these patients are to develop multiple myeloma. Resection and subsequent irradiation are advised for the apparently solitary tracheal lesion.

Non-Hodgkin's lymphoma (NHL) is rarely confined primarily to the trachea or bronchi. In addition to lymphocytic lymphoma, mucosa associated lymphoid tissue, and anaplastic histologic subtypes of NHL, lymphoplasmacytoid lymphoma has been found in the trachea.[68] Symptoms of tracheal lymphoma are cough and those of airway obstruction, frequently misdiagnosed as asthma. When other anatomic sites are not involved, the disease is often of lower grade histology. Fidias and colleagues identified 5 patients with primary tracheal disease, variously treated with chemotherapy, resection, and irradiation.[69] An additional 36 were found to have secondary tracheal involvement. The tumors may arise from mucosal lymphoid tissue, forming "lymphoepithelial" lesions. Since the cases are few, treatment regimens have varied widely. Long-term freedom from disease (12 to 64 months) resulted in 4 of 5 patients followed. One patient required tracheal resection for malacia following complete response to chemotherapy. Irradiation was then added. Maeda and colleagues reported survival over 5 years after tracheal resection only for primary tracheal malignant lymphoma.[70] Although experience is sparse, appropriate treatment for stage I extranodal tracheal lymphoma would seem to be chemotherapy, surgery where feasible, and irradiation. Resection may not prove to be necessary in all cases, but when needed, it is best done prior to irradiation. Recurrence may follow after a long interval. Thus, one of my patients, who had tracheal resection with laryngotracheoplas-

ty for diffuse mixed cell and large cell malignant lymphoma, followed by irradiation, suffered recurrence retroperitoneally 11 years later, without airway disease.

Hodgkin's lymphoma in mediastinal lymph nodes has affected the trachea in late stages from recurrence with perforation (tracheoesophageal or tracheomediastinal fistula) or from fibrous stenosis years after chemotherapeutic and irradiation treatments.[71,72] In the latter case, surgical resection seems contraindicated (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation").

Fortunately, *sarcomas* are uncommon. This group includes leiomyosarcoma, fibrosarcoma, and rhabdomyosarcoma.[73–75] All are uncommon in the trachea and bronchi. In 1999, Vinod and colleagues discovered in the literature 8 cases of leiomyosarcoma, 6 of malignant fibrous histiocytoma, and 1 each of rhabdomyosarcoma, fibrosarcoma, liposarcoma, and non-human immunodeficiency virus (HIV) Kaposi's sarcoma, all primary in the trachea.[54] Of these 18 cases followed for varied time periods, only 3 died of disease and 1 of other causes. The behavior of sarcomas is variable (see Figures 7-17*B*, 7-18). Although in our series a low-grade spindle cell sarcoma and a fibrosarcoma did not recur after resection, other sarcomas have been of high malignancy, and, in several cases, have been too extensive for resection when first identified. Irradiation and chemotherapy should probably be considered after surgical extirpation, but data are few for each cell type. In the case of a highly malignant sarcoma, postoperative irradiation has not appeared to delay recurrence. Pulmonary metastases seemed to occur early. Obstructing *Kaposi's sarcoma* was also identified exclusively in the subglottic region and trachea of an HIV-positive patient, and treated by laser and irradiation.[76] Endobronchial lesions are often present in acquired immunodeficiency syndrome (AIDS) patients with hilar adenopathy and perihilar infiltration, who show Kaposi's sarcoma of the skin and mucous membranes.[77,78] Obstruction may occur. Prognosis is very poor.

Clinical Presentation

Patients with tracheal tumors commonly have a long history of persistent *coughing*, which may steadily worsen. A patient notices gradual onset of *shortness of breath* on exertion, which will progress to dyspnea

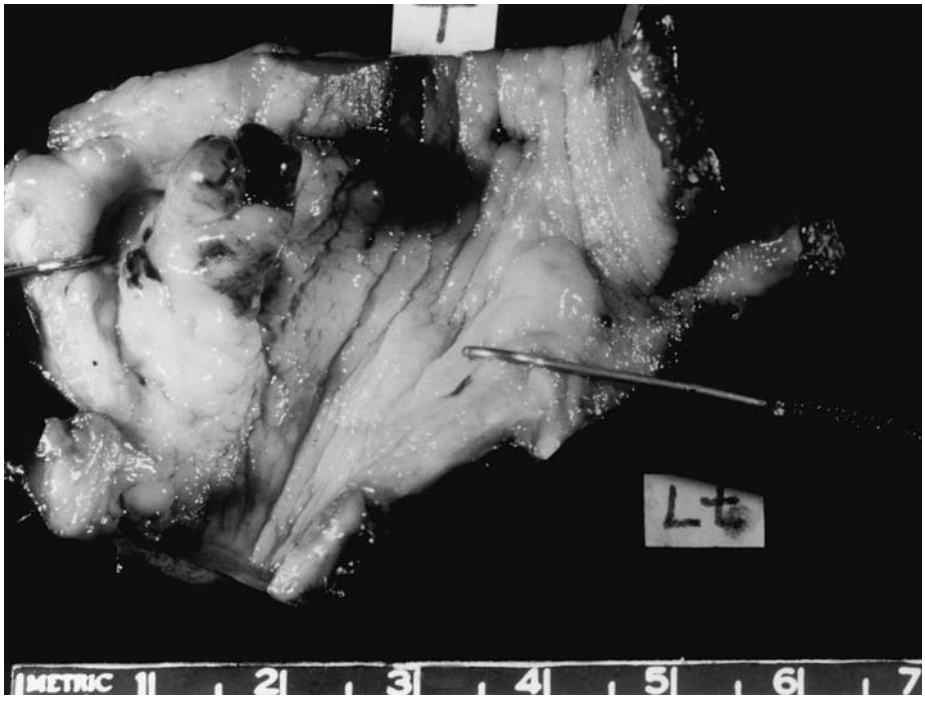


FIGURE **7-18** *Infiltrating spindle cell sarcoma with myxoid stroma, involving the lower trachea* (T), *carina, and much of the left main bronchus* (Lt), *in a 28-year-old man. Carinal resection and left pneumonectomy were followed by 6,000 cGy irradiation and chemotherapy. The hook in the gross specimen is at the carina. The patient died 3 years later of tumor progression.*

at rest. At this point, the airway is reduced to 30 to 50% of its normal cross-sectional area. Dyspnea may be aggravated by eating and by position. *Wheeze* is followed by true *stridor* (Table 7-3).[79] Chest roentgenograms are frequently interpreted as normal on the basis of clear lung fields, although the lesion may well be visible if the tracheal air column is examined critically. Unfortunately, a radiologist's index of suspicion of a tumor in the presence of such symptoms is all too often no greater than that of a pulmonologist or thoracic surgeon. *In a patient with signs of upper airway obstruction—dyspnea on exertion, wheeze, or stridor, with or without cough—organic obstruction of the upper airway should be suspected, even if the lung fields appear to be clear on standard roentgenograms*. The patients are all too often treated for "adult onset asthma" or for other imprecise diagnoses for long periods of time. Some present with Cushingoid appearance due to prolonged administration of high-dose steroids for treatment of presumed asthma. *Such patients should be bronchoscoped early*. A benefit of increasing use of computed tomography (CT) scans is earlier identification of tracheobronchial lesions.

Hemoptysis occurs sooner or later with many tumors, especially epithelial. It is more common with SCC of the trachea, somewhat less common with ACC and carcinoid tumors, and may not occur at all with many tumors, especially of mesenchymal origin, benign or malignant. Since the warning sign of hemoptysis is less frequent in ACC than in SCC (Table 7-4), the duration of symptoms is usually longer in the former and wheeze has more often developed.[6] As expected, resectability diminishes in patients with a longer average duration of symptoms prior to diagnosis in both types of carcinomas (Table 7-5). Hemoptysis mandates bronchoscopy, even with apparently clear lung fields radiologically, and is explained away distressingly often as due to strenuous cough, tracheobronchitis, or pneumonia. The bronchoscopist must think of the possibility of tracheal tumor. In particular, if the tumor is in the upper trachea, the bronchoscopist, even with a flexible bronchoscope, may pass too quickly from the vocal cords to a supracarinal, carinal, and bronchial examination and overlook a small proximal tracheal lesion. If examination is made with the flexible bronchoscope through an endotracheal tube, the tube must be withdrawn to the level of the glottis as the examination is completed so that the entire trachea is visualized.

Unilateral or bilateral *pneumonitis* may occur. Episodes of pneumonia or pneumonitis may respond to treatment, only to recur. Recurrent pneumonia or persisting pulmonary infiltrates, particularly in an otherwise healthy patient, are indications for bronchoscopy. A small tumor low in the trachea may produce recurrent unilateral pneumonitis and yet be of such small size that it is not visualized on tomography. A thin-section CT scan, however, will demonstrate even tiny tracheobronchial lesions. *Hoarseness* results

Table 7-3 Symptoms and Signs Associated with Tracheal Tumors

	Number from 84 Patients
Dyspnea	44
Hemoptysis	28
Cough	22
Wheezing	16
Dysphagia	13
Change in voice and/or hoarseness	13*
Stridor	12
Pneumonia	10
Emphysema and/or asthma	9

Adapted from Weber AL and Grillo HC.[79]

*Eight of 13 had vocal cord paralysis.

Table 7-4 Comparative Symptoms in Adenoid Cystic Carcinoma (ACC) and Squamous Cell Carcinoma (SCC) of Trachea

	Number from 135 Patients of each Diagnosis		
	ACC	*SCC*	χ^2
Dyspnea	65	50	0.014
Cough	55	52	NS
Hemoptysis	29	60	< 0.001
Wheeze	44	27	0.003
Stridor	21	27	NS
Hoarseness	10	13	NS
Dysphagia	7	7	NS
Fever	7	4	NS
Other	12	14	NS

Adapted from Gaissert HA et al.[6]

NS = not significant.

from involvement of a recurrent laryngeal nerve by tumor. *Dysphagia* is a late symptom caused by esophageal invasion. Untreated, death results from asphyxia, pneumonia, or hemorrhage.

Diagnostic Studies

Only very rarely will the *imaging studies* described in Chapter 4, "Imaging the Larynx and Trachea," fail to define the location and extent of a tracheal tumor (Figure 7-19). Oblique tomography or thin-section CT scan will usually identify small and otherwise subtle tracheal lesions. Virtual bronchoscopy may add a pictorial dimension, although it is not essential. If pulmonary lesions are also discovered, a fine needle percutaneous biopsy is useful to determine their nature.

Bronchoscopy is essential sooner or later (see Chapter 5, "Diagnostic Endoscopy"). The number of tracheal lesions overlooked or diagnosed late will be minimized if bronchoscopy is routinely used in the following circumstances: 1) patients who suffer from prolonged cough, dyspnea on exertion, and wheezing or stridor—without precise and proven diagnosis; 2) patients with hemoptysis; 3) patients with recurrent atelectasis, pneumonitis, pneumonia, or persistent unexplained pulmonary infiltrate. Where a lesion has been identified radiologically, is not unusually extensive, and clearly would be best treated by resection, a rigid bronchoscopy is often deferred to the general anesthesia under which resection is planned. The endoscopic management of severe acute obstruction by tumor is outlined in Chapter 19, "Urgent Treatment of Tracheal Obstruction." If there is possible hazard from an attempt at biopsy and the patient is to be referred to a center for definitive surgical resection, biopsy is best deferred.

Table 7-5 Duration of Symptoms and Resectability

Tumor Type	*ACC (months)*	*SCC (months)*
Resectable	18.3(14.6–21.9)	4.5(3.6–5.5)
Unresectable	23.7(15.1–32.3)	7.6(2.8–12.4)

Adapted from Gaissert HA et al.[6]

The range is given in parenthesis.

ACC = adenoid cystic carcinoma; SCC = squamous cell carcinoma.

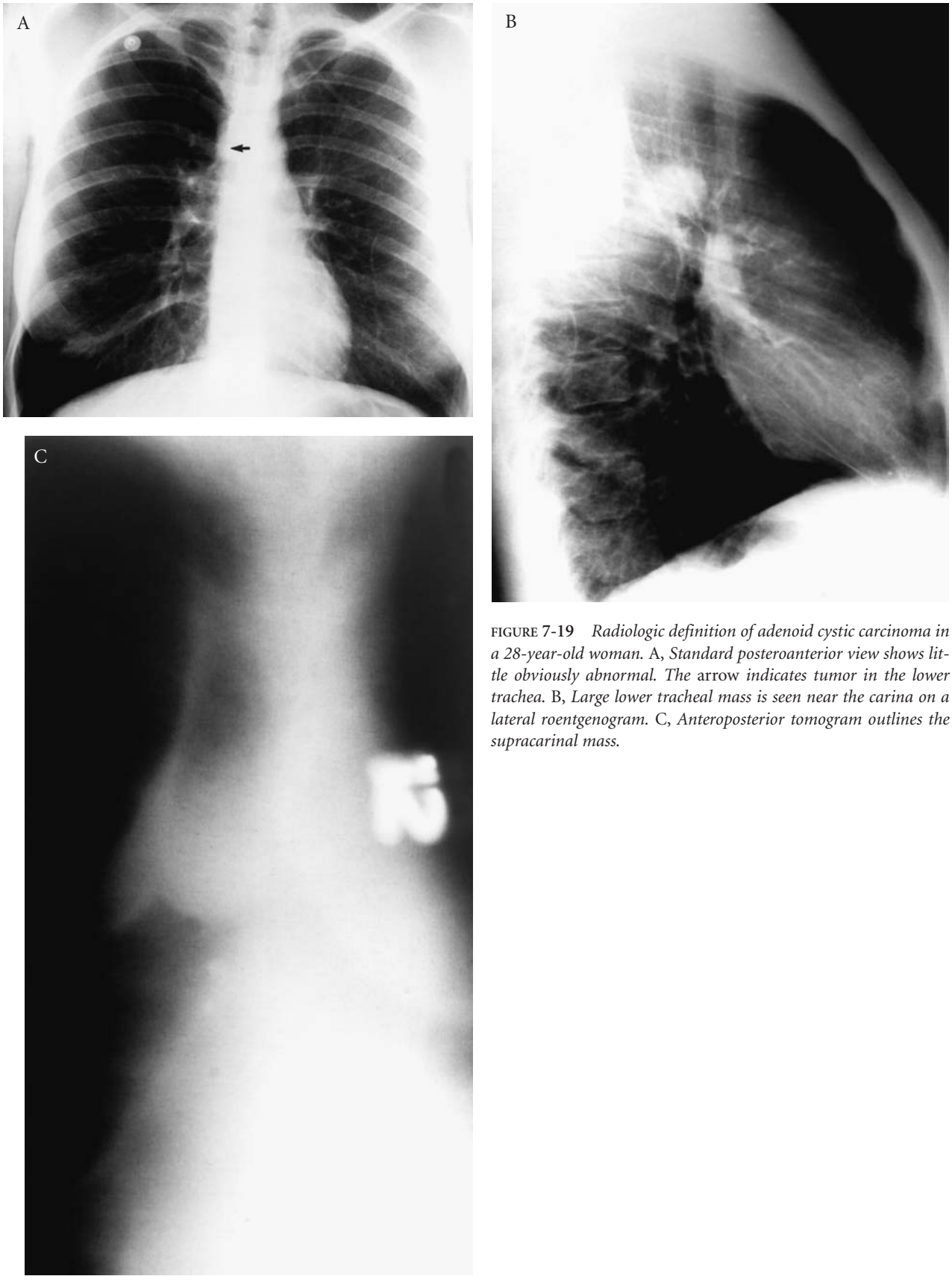


FIGURE **7-19** *Radiologic definition of adenoid cystic carcinoma in a 28-year-old woman.* A, *Standard posteroanterior view shows little obviously abnormal. The* arrow *indicates tumor in the lower trachea.* B, *Large lower tracheal mass is seen near the carina on a lateral roentgenogram.* C, *Anteroposterior tomogram outlines the supracarinal mass.*

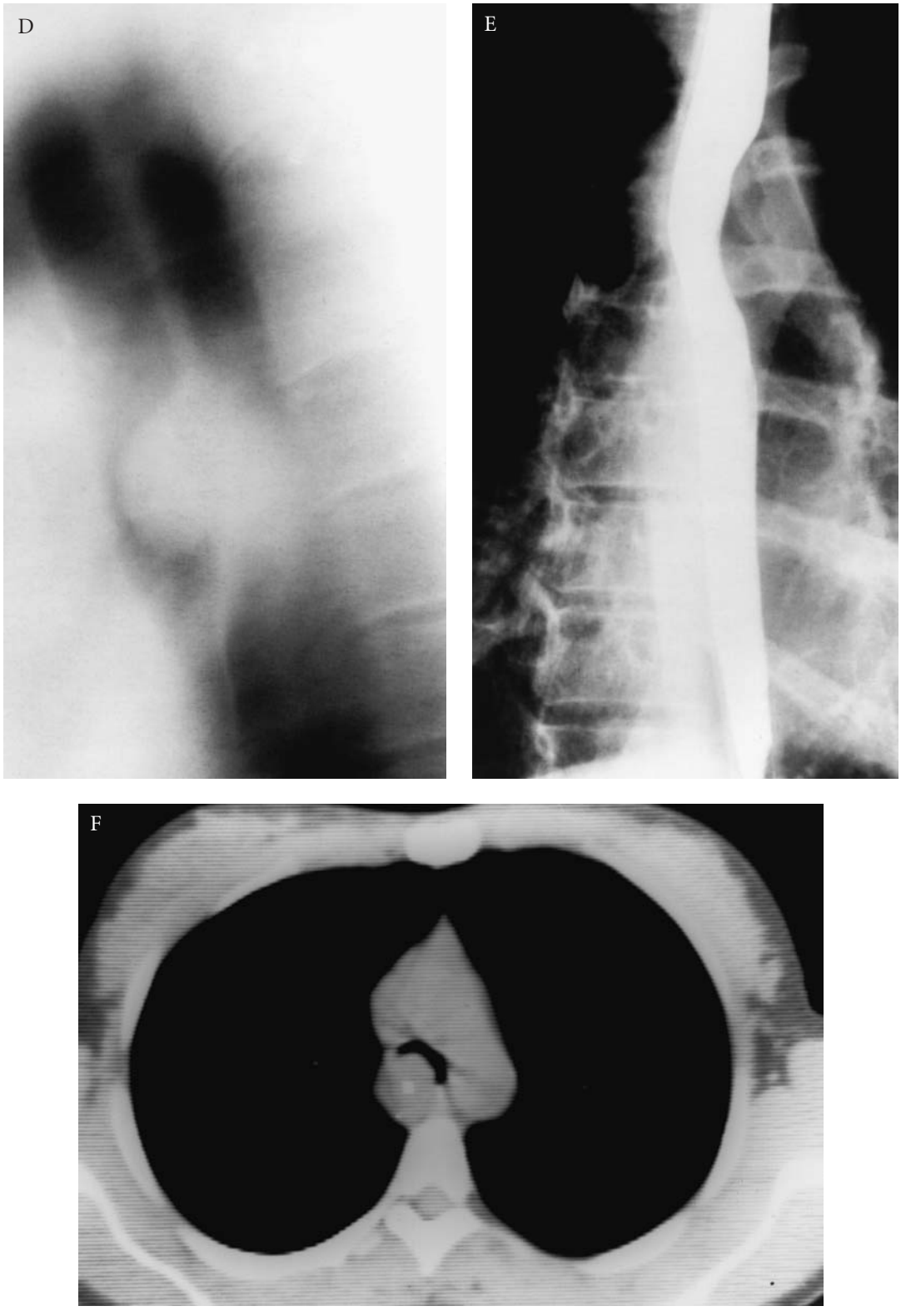


FIGURE 7-19 (CONTINUED) D, *Lateral tomogram demonstrates the posterior location of the base of tumor.* E, *Barium esophagogram shows smooth indentation of the esophagus by the mass.* F, *Computed tomography scan clarifies the component of tumor extending beyond the tracheal lumen. Carinal resection was required for an adequate margin, and was followed by radiotherapy. She remained well and without recurrence 19 years later.*

Functional studies are rarely of much practical use. Obstruction of the central airway requires relief. No matter how diminished the pulmonary reserve is, the patient will be improved by relief of the proximal airway obstruction. The surgical approach to distal tracheal lesions may be altered in the light of very poor pulmonary parenchymal function, however.

Treatment and Results

Surgical Management

Until the development of current techniques of tracheal resection and reconstruction, prospects for cure of tracheal tumors were small. Even when a lesion seemed potentially curable, some surgeons were often so inhibited by fear of inability to reconstruct the trachea that they settled for very limited local resection, often lateral resection, in an effort to maintain tracheal continuity. Local recurrence frequently followed such limited resection. Radiotherapy almost uniformly resulted in local recurrence even of radiosensitive tumors after varying periods of time, longer with ACC (3 to 7 years) than with squamous carcinoma (1 to 2½ years). Characteristic reports from this era were those by Houston and colleagues in 1969, listing 53 primary tracheal cancers seen over a 30-year period, and by Hajdu and colleagues in 1970, noting 41 patients over a 33-year period.[80,81] Because data is limited—due to the still relatively small number of cases reported, the absence of many single institutional series of significant size with prolonged follow-up, and the wide range in types and behavior of tracheal tumors—it is difficult to be categorical about management. However, enough information has accumulated in the past 30 years to formulate preliminary conclusions. In 1978, Grillo reported 63 patients, and in 1990, Grillo and Mathisen reported 198 patients with primary tracheal tumors treated at MGH, the latter report spanning 26 years.[1,82] Eschapasse, Pearson, and Perelman, and their colleagues offered significant series.[3,4,83] The combined experience of 26 French, German, and Italian hospitals was reported in 1996 by Regnard and colleagues.[5]

The distribution of primary tumors has been noted previously. In a 1990 series, 132 (66%) of our patients underwent resection with primary reconstruction of the airway (Table 7-6).[1] In 9 of these (adenoid cystic 3, squamous 1, other 5), involvement of the lower larynx by a high tracheal tumor required removal of a portion of the larynx with suitable tailoring of the distal trachea to accomplish reanastomosis with a remaining functional larynx (see Chapter 25, "Laryngotracheal Reconstruction"). In an additional small

Table 7-6 Primary Tracheal Tumors (1990)

Variable	*Squamous*	*Adenoid Cystic*	*Other*	*Total*
Number of lesions	70	80	48	198
Percentage of total	36	40	24	100
Surgical treatment	50	65	43	158
Excised	44	60	43	147
Percentage of type	63	75	90	74
Explored	6	5	0	11
Resection				
With reconstruction	41	50	41	132
Trachea	32	22	28	82
Carina	9	28	13	50
Laryngotracheal resection	1	4	2	7
Staged procedure	2	6	0	8

Adapted from Grillo HC and Mathisen DJ.[1]

number of patients (7), the larynx was so extensively involved by a high tracheal tumor that salvage was impossible and laryngotracheal resection was performed (Figure 7-20). Eight staged resections were performed where primary reconstruction was not possible and where it was planned ultimately to reconnect the larynx to the distal trachea in secondary procedures. Since so few of these succeeded optimally, staged procedures were abandoned. Sixty-three percent of all SCCs were resected, as well as 75% of all ACCs and 90% of others. The high resectability rate of the last group was due to the large number of benign lesions (see Table 7-6). Resectability rates remained stable at 68% for SCC and 73% for ACC as the series grew.[6]

Principal contraindications to resection are 1) extensive linear involvement of the airway such that primary end-to-end resection would not be possible without excessive tension, 2) mediastinal invasion of nonresectable organs, or 3) remote metastases. Although palliative resection may be advisable in some patients with ACC or differentiated thyroid carcinoma, to remove a potentially obstructing lesion even in the face of pulmonary metastases, I usually prefer to embark upon tracheal resection and reconstruction where there is a chance of cure. This applies even more to carinal resection because of increased risks of major surgical morbidity and higher mortality (see Chapter 21, "Complications of Tracheal Reconstruction").

Emergency management of patients with severe obstruction is described in Chapter 19, "Urgent Treatment of Tracheal Obstruction." If resection and reconstruction is not deemed to be possible because of linear extent of the lesion, primary radiotherapy is employed (see Chapter 41, "Radiation Therapy in the Management of Tracheal Cancer"). Brachytherapy has, on occasion, been added to external beam irradiation in selected patients. In the presence of bulky tumor with extrinsic compression of the tracheal wall, often after failure of radiotherapy, a T tube or a solid or coated expandable stent may span the area of obstruction for a time (see Chapter 40, "Tracheal and Bronchial Stenting").

Surgical approaches (see Tables 7-6, 7-7) and techniques for resection are described in Part 2, "Therapeutic Techniques and Management" (see Chapters 23–25, 28, 29, 34). The differing distribution of tumors is highlighted by the fact that 9 of 41 (22%) patients undergoing primary resection and reconstruction for SCC underwent resection of the carina, whereas 28 of 50 (56%) patients who had ACC were treated by carinal resection. By the year 2002, these figures were 22% and 41%, respectively. Laryngeal release was used 7 times in 82 (8.5%) patients undergoing tracheal resection for tumor. It was earlier also used 5 times in those undergoing carinal resection. I have since concluded that it can be useful in carinal resection, only where a large portion of the trachea itself has been removed, since laryngeal release only assists in advancement of the upper half of the trachea. This was confirmed by Valesky and colleagues in anatomic studies.[84] Hilar release, on the other hand, particularly the inferior portion of release at the level of the inferior pulmonary vein, was used in 12 of 32 (38%) patients undergoing transthoracic tracheal resection and 23 of 50 (46%) patients treated by carinal resection. Additional structures removed in these resections included a lobe of the thyroid, portions of the esophageal wall, and recurrent laryngeal nerve. Where extensive radiotherapy had been used prior to the operation, either remotely or recently, an omental pedicle flap was used to wrap the anastomosis (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation").

Gaissert and colleagues commenced review of the MGH experience with tracheal tumors to the year 2002.[6] Figures cited here are therefore somewhat preliminary. In 40 years, 135 patients were treated for ACC and 135 for SCC. Overall resectability rates were 78% for ACC and 68% for SCC. Given that twice as many patients suffering from SCC were smokers, the significantly increased incidence of prior carcinoma of the lung (15%), larynx (7%), and other head and neck lesions (4%) is not surprising. The overall incidence of prior upper respiratory tract cancer in patients with SCC of the trachea was 27%. The principal reasons for nonresectability were extent of airway involvement (ACC 68%, SCC 67%) and extent of regional disease (ACC 23%, SCC 24%). Other causes were distant disease (ACC 6%, SCC 7%), medical contraindication (ACC 0, SCC 2%), and patient's choice (ACC 3%, SCC 0).

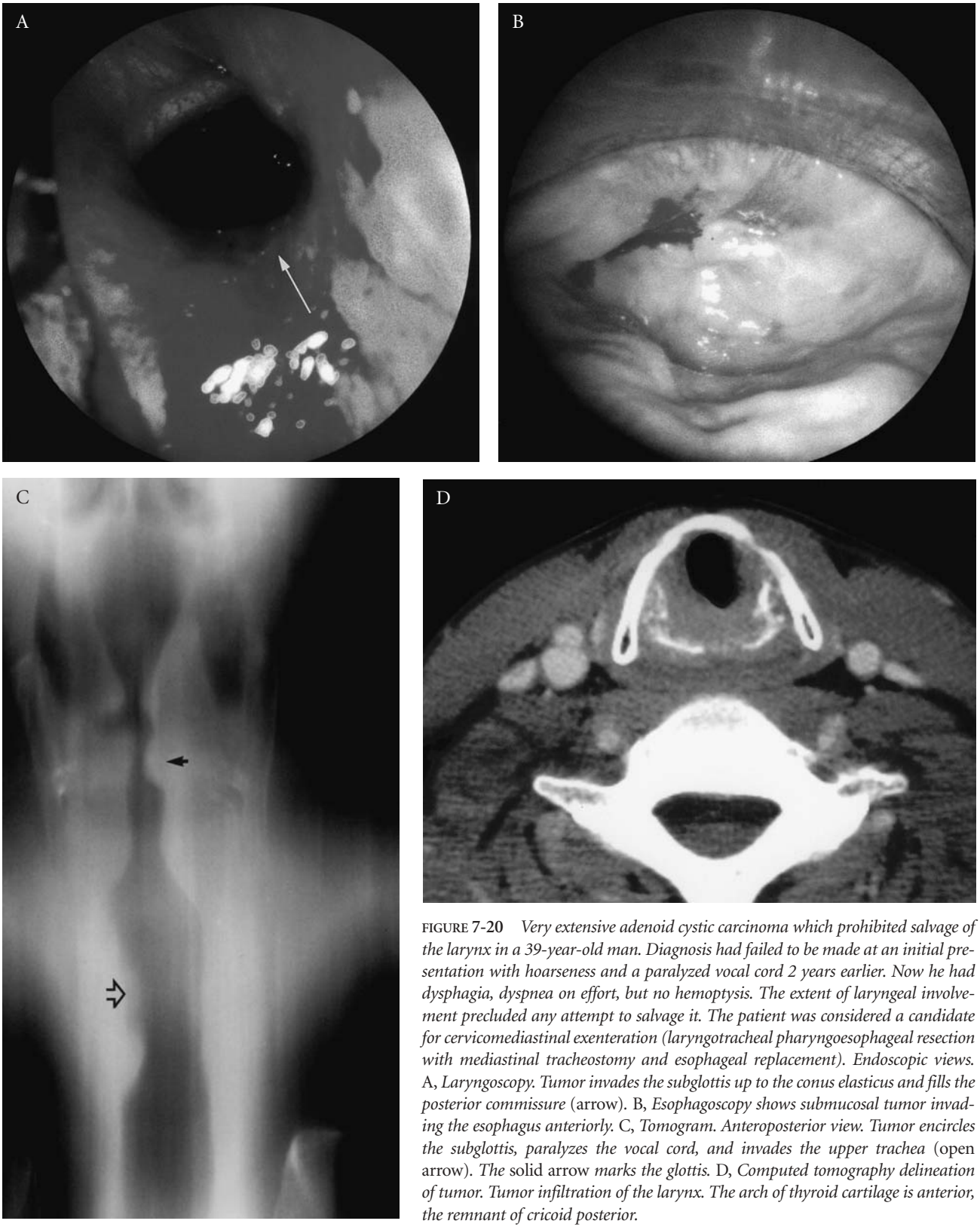


FIGURE **7-20** *Very extensive adenoid cystic carcinoma which prohibited salvage of the larynx in a 39-year-old man. Diagnosis had failed to be made at an initial presentation with hoarseness and a paralyzed vocal cord 2 years earlier. Now he had dysphagia, dyspnea on effort, but no hemoptysis. The extent of laryngeal involvement precluded any attempt to salvage it. The patient was considered a candidate for cervicomediastinal exenteration (laryngotracheal pharyngoesophageal resection with mediastinal tracheostomy and esophageal replacement). Endoscopic views.* A, *Laryngoscopy. Tumor invades the subglottis up to the conus elasticus and fills the posterior commissure* (arrow). B, *Esophagoscopy shows submucosal tumor invading the esophagus anteriorly.* C, *Tomogram. Anteroposterior view. Tumor encircles the subglottis, paralyzes the vocal cord, and invades the upper trachea* (open arrow). *The* solid arrow *marks the glottis.* D, *Computed tomography delineation of tumor. Tumor infiltration of the larynx. The arch of thyroid cartilage is anterior, the remnant of cricoid posterior.*

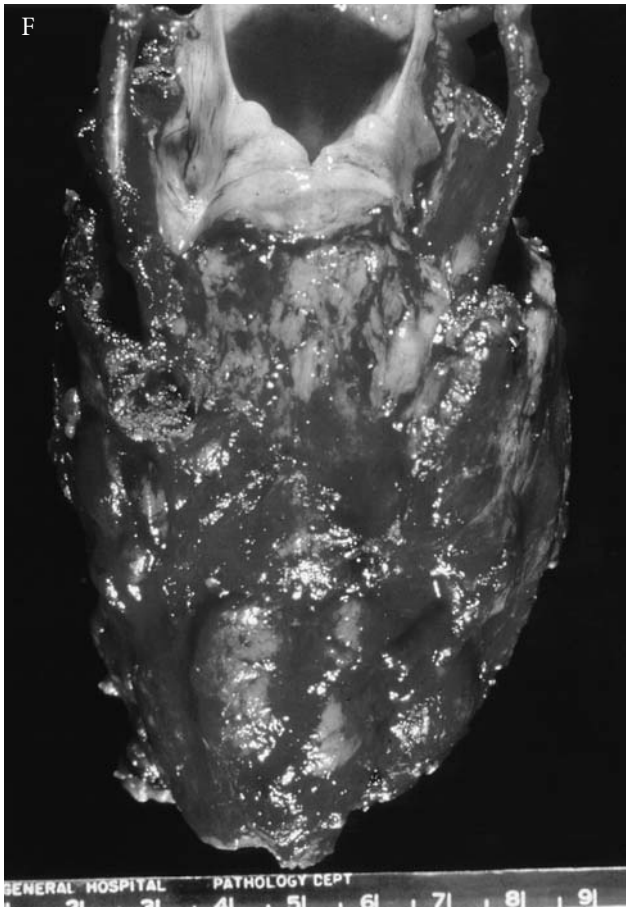
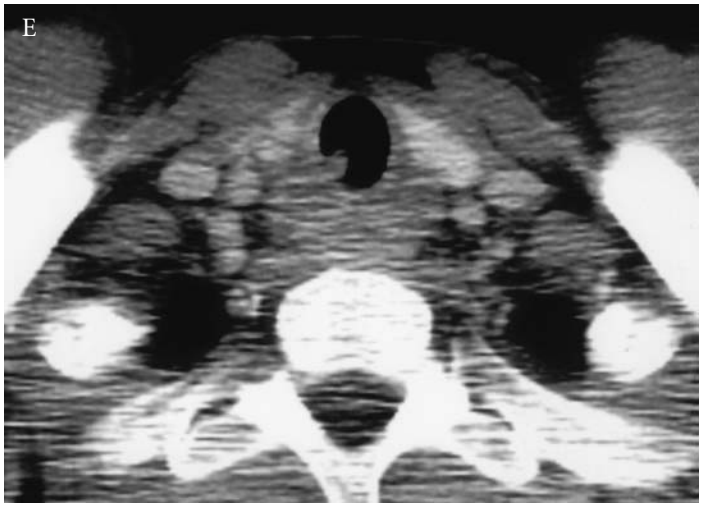


FIGURE 7-20 (CONTINUED) E, *Extension of tumor in the trachea, upper mediastinum, and around the esophagus. Carotid arteries are not invaded.* F, *Surgical specimen from another patient with adenoid cystic carcinoma, a 41-year-old man, viewed posteriorly. Tumor extends from the arytenoids downward. The esophagus was not involved. Total thyroidectomy, parathyroidectomy, and laryngotracheotomy were done. Because of the low level of mediastinal tracheostomy necessary, the brachiocephalic artery was divided and the omentum advanced. The patient later developed bone, lung, and liver metastases, which progressed slowly. He died from brain metastases 10 years later.*



Nearly 75% of patients undergoing tracheal resection for ACC and SCC were operated upon in the second two decades of this study (1982 to 2001), reflecting a variety of likely factors. The distribution of types of operation performed overall is listed in Table 7-7. The mean tumor lengths resected were 3.1 cm for all (standard deviation [SD] 1.7), 3.5 cm for ACC (SD 1.9), and 2.6 cm for SCC (SD 1.4).

Operative Results

Complications that occurred in the series of 132 resections and reconstructions, which was comprised of consecutive experience from my first tracheal resections for tumors in 1962 until 1989, included the following: anastomotic stenosis in 2 after tracheal resection and in 4 after carinal resection; 3 air leaks; 4 suture line granulomas; 1 esophageal fistula after partial excision of the esophageal wall; unintended vocal cord paralysis in 8; and problems with aspiration or laryngeal function in 6, principally after laryngeal release. Stenoses were most likely due to excessive tension on the anastomosis, particularly following extended resection, abetted in one patient almost certainly by lengthy preoperative treatment with prednisone in high doses. All anastomotic stenoses were treated successfully by re-resection at a later date, although with a loss of reimplanted right upper lobe in 2 patients who had carinal reconstructions. Suture line granulomas antedated the use of absorbable suture material. Since absorbable Vicryl has been solely used for tracheal anastomosis, suture line granulomas have all but disappeared. Pulmonary edema occurred in 2 patients after right carinal pneumonectomy. Surgical complications are dealt with in detail in Chapter 21, "Complications of Tracheal Reconstruction."

Table 7-7 Types of Resection Performed For ACC and SCC of Trachea (2002)

	ACC		*SCC*		*Total*	
Type of Resection	n	%	n	%	n	%
Laryngotracheal	8	8	8	9	16	8.4
Tracheal	45	45	57	63	102	53
Tracheal with permanent tracheostomy	7	7	5	6	12	6.3
Carinal without pulmonary resection	23	23	18	20	41	21
Carinal with pulmonary resection	18	18	2	2	20	10
Total	101	100	90	100	191	100

Adapted from Gaissert HA et al.[6]

ACC = adenoid cystic carcinoma; SCC = squamous cell carcinoma.

In our 2002 review of all resections done over 40 years for ACC and SCC of the trachea, 4 of 101 patients with ACC and 7 of 90 with SCC required postoperative ventilation. Anastomotic separations occurred in 5 (5%) with ACC and 7 (8%) with SCC or 6.3% for both groups.

Seven *operative deaths* in 132 patients undergoing tracheal and carinal resection and reconstruction totaled 5% (Table 7-8).[1] One death (1%) occurred in the 82 patients undergoing tracheal resection; 6 deaths (12%) occurred among the 50 undergoing carinal reconstruction. The single death after tracheal reconstruction was due to anastomotic leakage and pneumonia. The deaths after carinal reconstruction were due to respiratory failure after anastomotic leakage, pulmonary edema, and pneumonia, and 1 from hemorrhage was probably due to pulmonary artery erosion. He was the single such patient in whom tissue had not been interposed between the anastomosis and pulmonary artery. Mortality was also high among the 9 patients who underwent exploration only, probably related to the extent of their malignant disease. As noted, failure and mortality rates in the group of staged reconstructions were high enough to discourage further use of this approach, except in extraordinary circumstances. Prostheses were not employed.

The beneficial effect of growth of experience—patient selection, operative approach, improved techniques, increased comprehension of operative limits—is reflected in the progressive diminution of operative mortality for resection of ACC and SCC in successive decades: 21%, 11%, 5%, 3% (Table 7-9).

Oncologic Results

In 1990, 135 of 147 patients were reported to have survived tumor resection.[1] At the time of the survey, 49% of the patients with SCC, 75% of those with ACC, and 83% of patients with other tumors were alive and free of disease. These figures were difficult to interpret since the patients were spread over many years and accrued at a variable rate. The higher survival in the third group obviously reflected the large number of benign tumors and tumors of low malignancy in this group.

In 2002, an analysis of the cumulative results of resection plus postoperative irradiation in nearly every case showed an absolute survival rate of 88% from operation for *ACC*; 86% at 1 year, 64% at 5 years, and 45% at 10 years.[6] Treatment by irradiation only showed 44% survival at 5 years and 15% at 10 years (Table 7-10). Resection of *SCC*, with 95% survival of operation, gave survivals of 91% at 1 year, 46% at 5 years, and 25% at 10 years. Unresected survival was 8% at 5 years and 5% at 10 years (with irradiation) (see Table 7-10; Figure 7-21). A few unresected patients of both histologies underwent exploration only.

The results cited for both types of cancers are for patients followed to the time of their death or within 18 months of enumeration (up to 40 years follow-up). In our earlier study, SCC of the trachea recurred in a pattern similar to that of squamous carcinoma of the lung; that is, within 5 years, and frequently with-

Table 7-8 Results of Surgical Treatment of Primary Tracheal Tumors (1990)

Variable	*Squamous*	*Adenoid Cystic*	*Other*	*Total*
Number of tumors resected	44	60	43	147
Operative deaths (resection)	3	8	1	12
Resection, reconstruction				
Trachea	1	0	0	1
Carina	1	4	1	6
Percent (resection reconstruction)	5	8	2	5
Laryngotracheal	0	0	0	0
Staged procedures	1/2	4/6	0	5
Exploration only	2/6	1/3	0	3
Survival (resected tumors)				
Dead				
Of tumor	13	7	3	23
Of other cause	6	5	1	12
Alive				
With tumor	0	1	0	1
Without tumor	20	39	35	94
Lost to follow-up	2	0	3	5

Adapted from Grillo HC and Mathisen DJ.[1]

Table 7-9 Surgical Resection and Mortality Rates by Decade for Adenoid Cystic and Squamous Cell Carcinomas of Trachea and Carina

Decade	*Years*	*Number*	*Resection Rate (%)*	*Mortality Rate (%)*
1	1962–1971	19	68	21
2	1972–1981	54	61	11
3	1982–1991	107	66	5
4	1992–2001	88	82	3
		268	71	7

in 3 years (Table 7-11). Adenoid cystic carcinoma, on the other hand, recurred later, but continued to occur, sometimes many years later (see Table 7-11). Local recurrence, as well as metastases, have been observed 17 and 20 or more years after presumed complete resection. The slower rate of tumor progression and greater susceptibility to irradiation are probably both determining factors.

As in most malignant neoplasms, a complete resection promises more favorable survival (Table 7-12). Five- and 10-year survival rates for ACC were 84% and 68%, respectively, with negative proximal and distal resection margins against 50%, and 28% with positive margins. Even more striking are the corresponding figures for squamous cell cancer of 52% and 30% versus 13% and 0% (Figure 7-22).

Eschapasse collected 152 primary tumors from multiple teams in France and in the USSR.[83] In 1974, he reported 121 patients treated surgically, 75 reconstructed after resection (47 trachea, 28 carina), with 13 deaths. Five of 19 patients with adenoid cystic tumors were alive and free of disease from 3 to 9 years, and 11 of 27 with SCC were alive and free of disease from 7 months to 16 years. In 1987, Perelman and

Table 7-10 Absolute Survival from Adenoid Cystic or Squamous Cell Carcinoma of Trachea and Carina With and Without Surgical Resection

		Survival (%)			
	Number	*Operation*	*1-year*	*5-year*	*10-year*
Adenoid Cystic					
Resected	81	88	86	64	45
Unresected	30	97	86	44	15
Squamous					
Resected	87	95	91	46	25
Unresected	41	95	68	8	5
Total	239	93	84	84	26

Adapted from Gaissert HA et al.[6]

All patients were followed up to their death or within less than 18 months. Nearly all received postoperative irradiation, but dosage varied.

Koroleva reported 116 open operations on 153 patients with all types of tumors.[85] Seventy-five underwent sleeve resection ($n = 41$) or carinal resection ($n = 34$), with 11 deaths. The actuarially calculated survival was 13% for SCC at 5 years and 66% for ACC at 5 years.

Pearson and colleagues described 29 resections in 44 patients with primary tracheal tumors (16 sleeve resections, 13 carinal resections, 2 deaths).[3] Nine of the patients with ACC were alive without disease from

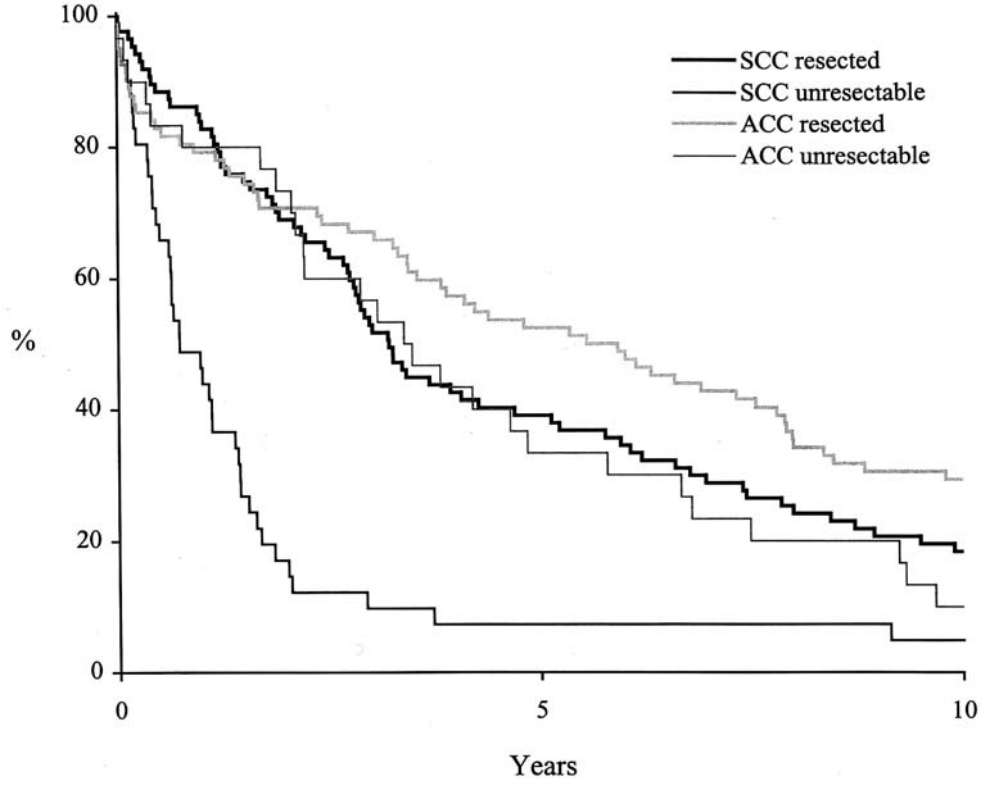


FIGURE 7-21 *Absolute survival of patients with adenoid cystic carcinoma (ACC) and squamous cell carcinoma (SCC), resected and unresected. Chart includes only patients with complete follow-up.*

Table 7-11 Survival after Resection of Tracheal Carcinoma (1990)

Variable	*Squamous*	*Adenoid Cystic*
Operative deaths	3	8
Alive without carcinoma		
> 10 yr	3(9)*	5(11)
5–10 yr	4(10)	11(12)
3–5 yr	3(6)	10(11)
0–3 yr	10(11)	13(13)
Died of carcinoma		
> 10 yr	0	4
5–10 yr	0	1
3–5 yr	5	2
0–3 yr	8	0
Died without carcinoma; lost		
> 10 yr	1	0
5–10 yr	3	0
3–5 yr	1	1
0–3 yr	3	4
Alive with carcinoma	0	1

Adapted from Grillo HC and Mathisen DJ.[1]

*Original number of operative survivors, excluding those who died of other causes later, is shown in parentheses.

1 to 20 years after operation. Three died of other disease 6 to 18 years postoperatively. Two were alive with disease. Four of 6 patients with squamous cell cancer were alive 6 to 56 months after resection. In 1996, Maziak and colleagues reported on 30 resections of 36 (83%) patients with ACC, with 7% mortality and survival rates of 79% and 51% at 5 and 10 years, respectively.[11] Similar results were obtained in a smaller series.[12]

A collected series of 208 patients with primary tracheal tumors, from 26 institutions in France, Germany, and Italy, was composed of 94 with SCC, 65 with ACC, and 49 with other tumors.[5] Of these

Table 7-12 Survival after Resection of Adenoid Cystic or Squamous Cell Carcinoma of Trachea and Carina With Negative or Positive Margins (2002)

		Survival (%)			
	Number	*Operation*	*1-year*	*5-year*	*10-year*
Adenoid Cystic					
Negative margins	30	87	90	84	68
Positive margins	49	88	83	50	28
Squamous					
Negative margins	67	97	94	52	30
Positive margins	17	88	75	13	0
Total	163	91	88	53	33

Adapted from Gaissert HA et al.[6]

Nearly all received postoperative irradiation of varied dosage. Margins are at proximal and distal resection lines. Lateral margins and lymph nodes are not noted here.

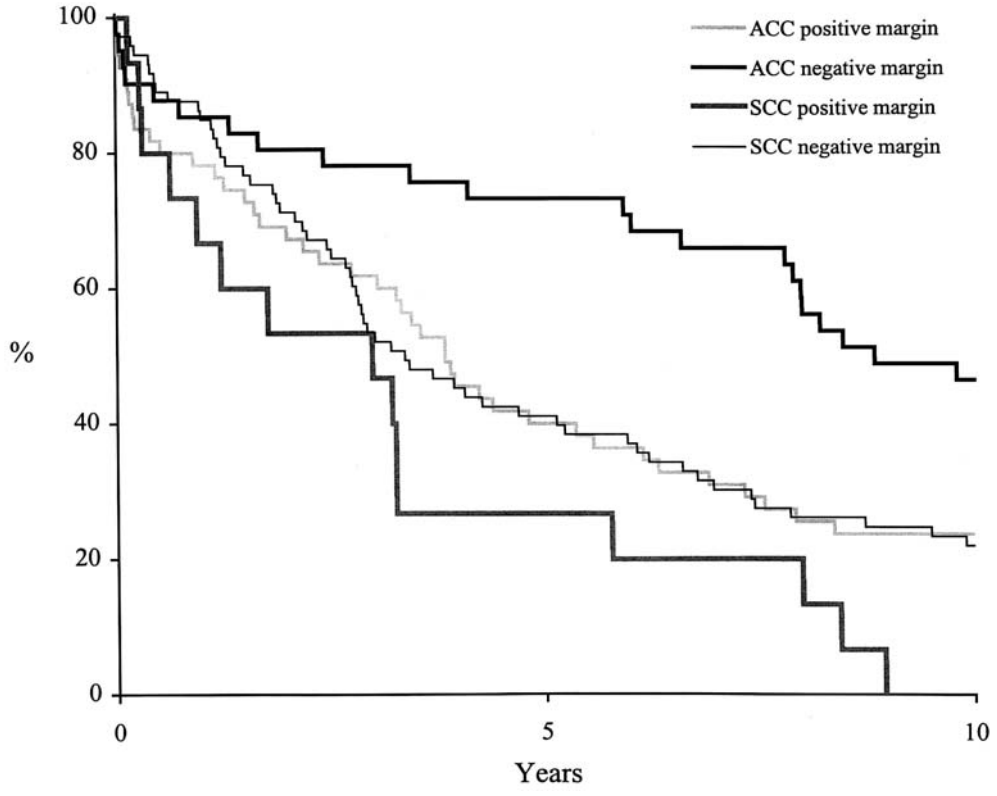


FIGURE 7-22 *Survival of patients resected for adenoid cystic carcinoma (ACC) and squamous cell carcinoma (SCC) with negative and positive margins. Margins are proximal and distal lines of resection.*

patients, 165 underwent tracheal resection and primary anastomosis, 24 underwent carinal resection, and 19 underwent laryngotracheal resection, with an overall mortality of 10.5%. Survival at 5 and 10 years for ACC was 73% and 57%, and 47% and 36% for other tracheal cancers (squamous plus 4 with adenocarcinoma), respectively. Comparative observations on treatment of ACC by several groups are summarized in Table 7-13. The behavior of our remaining heterogeneous group of 48 tumors other than squamous cell or adenoid cystic carcinomas may be summarized as follows.[1] Typical carcinoids did not recur, mucoepidermoid tumors behaved benignly in this series, and all benign tumors were cured. The few aggressive sarcomas generally suffered early recurrence. On the other hand, malignant fibrous histiocytoma, pseudosarcoma, and low-grade spindle cell sarcoma did not recur. Two small cell carcinomas, which had no evidence of nodal spread and were treated with additional standard chemotherapy, showed no recurrence in over 5 years. Unfortunately, these experiences are in anecdotal numbers only.

Radiotherapy

Nearly all our patients with SCC, ACC, or sarcoma received *postoperative irradiation*. Radiotherapy was given since it is impossible to obtain sufficiently generous margins beyond the tumor in most tracheal resections because of the limited length of the trachea. In many cases, it was necessary to accept microscopically positive margins, especially with ACC, to permit reanastomosis. In some, adjacent lymph nodes were positive. Irradiation was given in an attempt to "sterilize" microscopic disease. The dose advised was in the range of 5,500 cGy (see Chapter 41, "Radiation Therapy in the Management of Tracheal Cancer"). The actual doses given varied since most patients were returned to their home communities for radiotherapy.

Table 7-13 Treatment of Adenoid Cystic Carcinoma of Trachea

	Author (reference)				
	Perelman (85)	*Grillo (1)*	*Regnard (5)*	*Maziak (11)*§	*Gaissert (6)*
Year of report	1987	1990	1996	1996	2002
Years included	20	26	23	32	40
Number of patients	—	80	—	38	135
Number of resected*	56	60	65	32	101
% resected	—	75	—	84	75
Operative mortality (%)	14†	13	6	9	11‡
Radiotherapy also (%)	41	100	43	81	100
Survival (%)					
5 years	66	79	73	79	64
10 years	56	42	57	51	45

*Tracheal and carinal resections.

†All tracheal tumor resections.

‡3% for 1992–2001.

§ACC only

In our 1990 study, not unexpectedly, we observed that positive lymph nodes in SCC were more commonly present in patients who later died with cancer than in those who survived without cancer.[1] Of 13 patients who died of cancer, 6 had positive lymph nodes. Of 22 disease-free survivors, only 2 had positive nodes. Invasive carcinoma at the resection margin had different consequences from in situ carcinoma. Four of 5 patients with invasive carcinoma died, whereas 6 with in situ carcinoma remained disease free. Parallel observations have been made in bronchogenic squamous carcinoma with respect to additional in situ lesions.

Both lesions are sensitive to radiotherapy, in particular, ACC. Since it has been argued that radiotherapy might be considered as primary treatment for tracheal tumors, we examined the outcome of patients treated by resection followed by irradiation in comparison with those patients treated by irradiation alone (including patients explored but not resected).[86,87] Although these are not totally comparable groups, the principal reason for not resecting the trachea in ACC was that the linear extent of the tumor was too great, rather than because of lateral bulk. In that sense, both shorter tumors and longer tumors may be considered somewhat comparable in accessibility to radiotherapy. Table 7-10 shows that there is a clear difference in survival of patients with both squamous and adenoid cystic tracheal carcinomas, when treated by resection followed by irradiation, when compared with irradiation alone (see Figure 7-21).[1,6] Further, there appeared to be palliative benefit in giving irradiation after resection, even in those patients who ultimately died of recurrent carcinoma. The median survival of patients with SCC undergoing irradiation alone was 10 months, compared with 34 months for those who also were resected.[1] Comparable figures for ACC are 28 months and 118 months, respectively. Cheung found primary radiotherapy for squamous cell and adenoid cystic carcinomas of the trachea in doses from 4,000 to 6,000 cGy to be ineffective in obtaining complete local control.[88] Preoperative irradiation was briefly proposed by Pearson and colleagues but no data are available to support this use.[3] The roles of external radiotherapy and brachytherapy are more fully expounded in Chapter 41, "Radiation Therapy in the Management of Tracheal Cancer."

Recommendations for Treatment

Based on our experience cited, and the experiences of other groups, we make the following recommendations for management of primary tumors of the trachea.

1) All benign primary tumors of the trachea and tumors of intermediate aggressiveness are best treated by complete surgical resection with primary reconstruction.
2) Primary SCC and ACC of the trachea are best treated by resection followed by irradiation, as long as primary reconstruction is judged to be safely possible.
3) Malignant primary tumors of other types should be resected if technically safe to do so, and irradiation most likely should be added despite small experience with many of these tumors.

The limited information that has accrued in the literature seems to support these recommendations.[1,3–6,83,88,89]

A final cautionary note is in order. During many years of treating tracheal tumors, I have seen numerous patients who have suffered prolonged delay in surgical management. Delay has first been due to the failure to make a timely diagnosis. The second significant cause of delay has been failure to offer definitive therapy because of lack of appreciation of the availability and effectiveness of current surgical techniques, assisted by irradiation. A third group of patients has been delayed for even less acceptable reasons: repetitive use of laser management without any clear rationale for continued use of this modality. These treatments were not given for emergency relief of obstruction, but rather as continuing management. Since the cure even of benign tracheal tumors is very rarely achieved by laser (or other endoscopic removal), such treatment is not justified, except perhaps for palliation of acute obstruction.[90] Even then, there are simpler methods.[91] Surgical resection, as early as possible, must be considered as the preferred initial treatment in almost every case.

References

1. Grillo HC, Mathisen DJ. Primary tracheal tumors. Treatment and results. Ann Thorac Surg 1990;49:69–77.
2. Grillo HC. Primary tracheal tumours [editorial]. Thorax 1993;48:681–2.
3. Pearson FG, Todd TRJ, Cooper JD. Experience with primary neoplasms of the trachea. J Thorac Cardiovasc Surg 1984;88:511–8.
4. Perelman MI, Koroleva N. Surgery of the trachea. World J Surg 1980;4:583–91.
5. Regnard JF, Fourquier P, Levasseur P. Results and prognostic factors in resections of primary tracheal tumors: a multicenter retrospective study. J Thorac Cardiovasc Surg 1996;111:808–14.
6. Gaissert HA, Grillo HC, Shadmehr B, et al. Comparative long term survival after resection of adenoid cystic and squamous cell carcinoma of trachea and carina.[In preparation]
7. Ranke EJ, Presley SS, Holinger PH. Tracheogenic carcinoma. JAMA 1962;182:519–22.
8. Culp OS. Primary carcinoma of the trachea. J Thorac Surg 1938;7:471–87.
9. Desai DP, Holinger LD, Gonzalez-Crussi F. Tracheal neoplasms in children. Ann Otol Rhinol Laryngol 1998;107:790–6.
10. Saltarelli MG, Fleming MV, Wenig BM, et al. Primary basaloid squamous cell carcinoma of the trachea. Am J Clin Pathol 1995;104:594–8.
11. Maziak DE, Todd TRJ, Keshavjee SH, et al. Adenoid cystic carcinoma of the airway: thirty-two year experience. J Thorac Cardiovasc Surg 1996;112:1522–32.
12. Prommegger R, Salzer GM. Long term results of surgery for adenoidcystic carcinoma of the trachea and bronchi. Eur J Surg Oncol 1998;24:440–4.
13. Soorae AS, Gibbon JRP. Primary oat-cell carcinoma of the trachea. Thorax 1979;34:130–1.
14. Gelder CM, Hetzel MR. Primary tracheal tumours: a national survey. Thorax 1993;48:688–92.
15. Onizuka M, Doi M, Mitsui K, et al. Undifferentiated carcinoma with prominent lymphocytic infiltration (so-called lymphoepithelioma) in the trachea. Chest 1990;98:236–7.
16. Duarte IG, Gal AA, Mansour KA. Primary malignant melanoma of the trachea. Ann Thorac Surg 1998; 65:559–60.
17. Briselli M, Mark EJ, Grillo HC. Tracheal carcinoids. Cancer 1978;42:2870–9.
18. Wilkins EW, Scannell JG, Grillo HC, Moncure AC. Changing times in surgical management of bronchopulmonary carcinoid tumor. Ann Thorac Surg 1984;38:339–44.
19. Shrager JB, Wright CD, Wain JC, et al. Bronchopulmonary carcinoid tumors associated with Cushing's syndrome: a more aggressive variant of typical carcinoid. J Thorac Cardiovasc Surg 1997;114:367–75.
20. Breyer RH, Dainauskas JR, Jensik RJ, Faber LP. Mucoepidermoid carcinoma of the trachea and bronchus: the case for conservative resection. Ann Thorac Surg 1980;29:197–204.
21. Heitmiller RF, Mathisen DJ, Ferry JA, et al. Mucoepidermoid lung tumors. Ann Thorac Surg 1989;47:394–9.
22. Welsh JH, Maxson T, Jaksic T, et al. Tracheobronchial mucoepidermoid carcinoma in childhood and adolescence: case report and review of the literature. Int J Pediatr Otorhinolaryngol 1998;45:265–73.
23. Heifetz SA, Collins B, Matt BH. Pleomorphic adenoma (benign mixed tumor) of the trachea. Pediatr Pathol 1992;12:563–74.
24. Payne WS, Schier J, Woolner LB. Mixed tumors of the bronchus (salivary gland type). J Thorac Cardiovasc Surg 1965;49:663–8.
25. Naka Y, Nakao K, Hamaji Y, et al. Solitary squamous papilloma of the trachea. Ann Thorac Surg 1993;55:189–93.
26. Gamblin TC, Farmer LA, Dean RJ, et al. Tracheal polyp. Ann Thorac Surg 2002;73:1286–7.

27. Bauman NM, Smith RJH. Recurrent respiratory papillomatosis. Pediatr Clin North Am 1996;43:1385–401.
28. Weber AL, Shortsleeve M, Goodman M, et al. Cartilaginous tumors of the larynx and trachea. Radiol Clin North Am 1978;16:261–71.
29. Huizenga C, Balogh K. Cartilaginous tumors of the larynx: a clinicopathologic study of 10 new cases and a review of the literature. Cancer 1970;26:201–10.
30. Bogden CJ, Maniglia AJ, Eliachar I, Katz RL. Chondrosarcoma of the larynx: challenges in diagnosis and management. Head Neck 1994;16:127–34.
31. Brandwein M, Moore S, Som P, Biller H. Laryngeal chrondrosarcomas. Laryngoscope 1992;102:858–67.
32. Farrell MI, Gluckman JL, Biddinger P. Tracheal chondrosarcoma: a case report. Head Neck 1998;20:568–72.
33. Brewster DC, MacMillan IKR, Edwards FR. Chondroma of the trachea: report of a case and review of the literature. Ann Thorac Surg 1975;19:576–84.
34. Kiriyama M, Masaoka A, Yamakawa Y, et al. Chondrosarcoma originating from the trachea. Ann Thorac Surg 1997;63:1772–3.
35. Jortay AM, Bisschop P. Chondroma of the trachea. Acta Otorhinolaryngol Belg 1998;52:247–51.
36. Salminen US, Halttumen P, Taskinen E, Mattila S. Recurrence and malignant transformation of endotracheal chondroma. Ann Thorac Surg 1990;49:830–2.
37. Drolet BA, Esterly NB, Friedan J. Hemangiomas in children. N Engl J Med 1999;341;173–81.
38. Wenig BL, Abramson AL. Congenital subglottic hemangiomas: a treatment update. Laryngoscope 1988;98: 190–2.
39. Weber TR, Connors RH, Tracu TF, Bailey PV. Complex hemangiomas of infants and children. Individualized management in 22 cases. Arch Surg 1990;125:1017–21.
40. Fishman SJ. Vascular anomalies of the mediastinum. Semin Pediatr Surg 1999;8:92–8.
41. Attar S, Cowley RA. Hemangioma of the mediastinum: collective review and case report. Am Surg 1964;30:141–52.
42. Lunde P, Sörlie D, Bolz KD, Eide TJ. Huge arteriovenous malformation in the mediastinum. Scand J Thorac Cardiovasc Surg 1984;18:75–80.
43. Garcia-Prats MD, Sotelo-Rodriguez MT, Ballestin C, et al. Glomus tumour of the trachea: report of a case with microscopic, ultrastructural and immunohistochemical examination and review of the literature. Histopathology 1991;19:459–64.
44. Lange TH, Magee MJ, Boley TM, et al. Tracheobronchial glomus tumor. Ann Thorac Surg 2000;70:292–5.
45. Burton DM, Heffner DK, Papone CA. Granular cell tumors of the trachea. Laryngoscope 1992;102:807–13.
46. Desai DP, Maddalozzo J, Holinger LD. Granular cell tumor of the trachea. Otolaryngol Head Neck Surg 1999;120:595–8.
47. Daniel TM, Smith RH, Faunce HF, Sylvest VM. Transbronchoscopic versus surgical resection of tracheobronchial granular cell myoblastomas. J Thorac Cardiovasc Surg 1980;80:898–903.
48. Tan-Liu NS, Matsubara O, Grillo HC, Mark EJ. Invasive fibrous tumor of the tracheobronchial tree: clinical and pathologic study of seven cases. Hum Pathol 1989;20:180–4.
49. Matsubara O, Tan-Liu NS, Kenney RM, Mark EJ. Inflammatory pseudotumors of the lung: progression from organizing pneumonia to fibrous histocytoma or to plasma cell granuloma in 32 cases. Hum Pathol 1988;19:807–14.
50. Hakimi M, Pai RP, Fine G, Davila JC. Fibrous histiocytoma of the trachea. Chest 1975;68:367–9.
51. Sputo G, Rossi NP, Allsbrook WC. Tracheobronchial plasma cell granuloma. J Thorac Cardiovasc Surg 1977; 73:804–6.
52. Ozturk S, Kutlay H, Sak SD, Yavuzer S. Fibrohistiocytic tumor of the trachea in a child. Eur J Cardiothorac Surg 1999;16:464–8.
53. Sandstrom RE, Proppe KH, Trelstad RL. Fibrous histiocytoma of the trachea. J Clin Pathol 1978;70:429–33.
54. Vinod SK, MacLeod CA, Barnes DJ, Fletcher J. Malignant fibrous histiocytoma of the trachea. Respirology 1999;4:271–4.
55. Gonzalez-Campora R, Matilla A, Sanchez-Carrillo J, et al. 'Benign' fibrous histiocytoma of the trachea. J Laryngol Otol 1981;95:1287–92.
56. Bando T, Fujimura M, Noda Y, et al. Pulmonary plasma cell granuloma improves with corticosteroid therapy. Chest 1994;105:1574–5.
57. Mandelbaum I, Brashear RE, Hull MT. Surgical treatment and course of pulmonary pseudotumor (plasma cell granuloma). J Thorac Cardiovasc Surg 1981;82:77–82.
58. Imperato JP, Folkman J, Sagerman RH, Cassidy JR. Treatment of plasma cell granuloma of the lung with radiation therapy. Cancer 1986;57:2127–9.
59. Horowitz AW, Khalil KG, Verani RR, et al. Primary intratracheal neurilemoma. J Thorac Cardiovasc Surg 1983;85:313–20.
60. Stack PS, Steckler RM. Tracheal neurilemmoma: case report and review of the literature. Head Neck 1990;12:436–9.
61. Zeman MS. Carotid body tumor of the trachea. Ann Otol 1956;65:960–2.
62. Choi G, Kim HY, Kim A, Choi Jo. Tracheal leiomyoma. J Otolaryngol 1998;27:87–9.
63. Borski TG, Stucker FJ, Grafton WD, Nathan CO. Leiomyoma of the trachea: a case report and a novel surgical approach. Am J Otolaryngol 2000;21:119–21.
64. Chen TF, Braidley PC, Shneerson JM, Wells FC. Obstructing tracheal lipoma: management of a rare tumor. Ann Thorac Surg 1990;49:137–9.
65. Bateson EM. Relationship between intrapulmonary and endobronchial cartilage-containing tumors (so-called hamartomata). Thorax 1965;20:447–61.
66. Butler C, Kleinerman J. Pulmonary hamartoma. Arch Pathol 1969;88:584–92.
67. Logan PM, Miller RR, Muller NL. Solitary tracheal plasmacytoma. Can Assoc Radiol J 1995;46:125–6.
68. Gomez-Roman JJ, Perez-Montes R, Perez-Exposito MA, et al. Primary lymphoplasmacytoid lymphoma of the trachea with immunoglobulin G paraprotein. Pathol Int 1999;49:1100–4.
69. Fidias P, Wright C, Harris NL, et al. Primary tracheal non-Hodgkin's lymphoma. A case report and review of the literature. Cancer 1996;77:2332–8.
70. Maeda M, Kotake Y, Monden Y, et al. Primary malignant lymphoma of the trachea. Report of a case successfully treated by primary end-to-end anastomosis after circumferential resection of the trachea. J Thorac Cardiovasc Surg 1981;81:835–9
71. Tse DG, Summers A, Sanger JR, Haasler GB. Surgical treatment of tracheomediastinal fistula from recurrent Hodgkin's lymphoma. Ann Thorac Surg 1999;67:832–4.
72. Grevend KM, Evans LS. The occurrence and management of esophageal fistulas resulting from Hodgkin's disease. Cancer 1992;69:1031–3.
73. Reichert K, Helling K, Mensen HD, et al. Primary tracheal leiomyosarcoma. J Laryngol Otol 2000;114:64–6.
74. Postovsky S, Peleg H, Ben-Itzhak O, Arush MW. Fibrosarcoma of the trachea in a child: case report and review of the literature. Am J Otolaryngol 1999;20:332–5.
75. Kegar A, Cantrel G, Rosen G. Rhabdomyosarcoma of the trachea. J Laryngol Otol 1988;102:735–6.
76. Belda J, Canalis E, Gimferrer JM, et al. Subglottic stenosis in an HIV positive patient: an exceptional form of clinical presentation in Kaposi's sarcoma. Eur J Cardiothorac Surg 1997;11:191–3.

77. Zibrak JD, Silvestri RC, Costello P, et al. Bronchoscopic and radiologic features of Kaposi's sarcoma involving the respiratory system. Chest 1986;90:476–9.
78. Judson MA, Sahn SA. Endobronchial lesions in HIV-infected individuals. Chest 1994;105:1314–23.
79. Weber AL, Grillo HC. Tracheal tumors. A radiological, clinical and pathological evaluation in 84 cases. Radiol Clin North Am 1978;16:227–46.
80. Houston HE, Payne WS, Harrison EG Jr, Olsen AM. Primary cancers of the trachea. Arch Surg 1969;99:132–40.
81. Hajdu SI, Huvos AC, Goodner JT, et al. Carcinoma of the trachea. Clinico-pathologic study of 41 cases. Cancer 1970;25:1448–56.
82. Grillo HC. Tracheal tumors: surgical management. Ann Thorac Surg 1978;26:112–25.
83. Eschapasse H. Les tumeurs trachéales primitives. Traitement chirurgicale. Rev Fr Malad Resp 1974;2:425–46.
84. Valesky A, Hohlbach G, Schildberg FW. Wertigkeit unterschiedlicher massnahmen zur minderung der anastomosenspannung nach kontinuitätsresektion der trachea. Langenbecks Arch Chir 1983;360:59–69.
85. Perelman MI, Koroleva NS. Primary tumors of the trachea. In: Grillo HC, Eschapasse H, editors. International trends in general thoracic surgery. Vol 2. Philadelphia: WB Saunders; 1987. p. 91–106.
86. Rostom AY, Morgan RL. Results of treating primary tumours of the trachea by irradiation. Thorax 1978; 33:387–93.
87. Makarewicz R, Mross M. Radiation therapy alone in the treatment of tumours of the trachea. Lung Cancer 1998;20:169–74.
88. Cheung AYC. Radiotherapy for primary carcinoma of the trachea. Radiother Oncol 1989;14:279–85.
89. Fields JN, Rigand G, Emami BN. Primary tumors of the trachea: results of radiation therapy. Cancer 1989; 63:2429–33.
90. Daddi G, Puma F, Avenia N, et al. Resection with curative intent after endoscopic treatment of airway obstruction. Ann Thorac Surg 1998;65:203–7.
91. Mathisen DJ, Grillo HC. Endoscopic relief of malignant airway obstruction. Ann Thorac Surg 1989; 48:469–75.

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Secondary Tracheal Neoplasms

Hermes C. Grillo, MD

Secondary neoplastic invasion of the trachea by direct extension is most often due to carcinomas of the esophagus, thyroid, and lung. Carcinoma of the larynx may also invade the trachea directly or it may recur at the margin of the stoma from lymphatics after laryngectomy. Less commonly, hematogenous metastases involve the trachea or carina. Sites of origin include the breast, melanoma, kidney, and thyroid. Carcinoma metastatic to the mucosa of the trachea from distant primary sites is less common than metastases to the bronchial mucosa, which is in itself an uncommon phenomenon.

The goals of major resection of the trachea or carina for secondary neoplasms should be the possibility of cure, or otherwise, prolonged palliation. This excludes most hematogenous metastases. Palliation of irresectable obstructing tumor may be achieved by endobronchial curettage, laser therapy, external beam irradiation, brachytherapy, or sometimes by stenting. The limited place for tracheal resection and reconstruction, when the airway is invaded by adjacent neoplasm, is considered below. The two most appropriate categories for surgical treatment are thyroid and bronchogenic carcinomas. Invasion by esophageal carcinoma is almost never an indication for tracheal resection.

Thyroid Carcinoma

Intraluminal airway invasion by differentiated thyroid carcinoma is rare, especially as a primary presentation, with estimates ranging from 0.5 to 7% or higher.[1,2] Invasive well-differentiated thyroid carcinoma may present initially with hoarseness, hemoptysis, or dyspnea. Frequently, however, the invasive carcinoma is identified by a surgeon at thyroidectomy. The thyroid surgeon who finds the trachea invaded at the time of thyroidectomy commonly "shaves off" the tumor from the tracheal wall. Treatment with ^{131}I or external irradiation may follow. Unfortunately, such treatment may not be successful in preventing later airway obstruction. It has been calculated that 82% of deaths from thyroid carcinomas may be due to asphyxia or pneumonia from local recurrence.[3–5] Invasion of the airway by differentiated thyroid carcinoma, either papillary or mixed follicular and papillary (which generally behaves like papillary carcinoma), is more common in older patients, but it is by no means restricted to this age group.

Anaplastic or very poorly differentiated tumors frequently involve the trachea and larynx, to such degree at initial presentation that laryngeal salvage is not possible. The pharynx and esophagus may also be invaded in such advanced cases. Late, massive recurrence of differentiated carcinoma can produce a similar picture.

Tumor Behavior

A well-differentiated thyroid carcinoma usually runs an indolent course, frequently with long-term survival.[6,7] Airway invasion, however, is directly responsible for many late deaths due to thyroid cancer and is a source of profound morbidity from hemorrhage and suffocation.[3] The cancer directly invades the closest portion of the airway. Tsumori and colleagues reported that 50% of papillary and follicular carcinomas which invaded the airway showed poor differentiation, whereas only 11.4% of noninvasive thyroid cancers of similar histology were poorly differentiated.[8] Invasion also occurs most often in older patients, where papillary and follicular thyroid cancers may be more aggressive, although the spectrum is broad. Nomori and colleagues found that the nuclear area of tumor cells was significantly increased in cases with tracheal invasion compared to those without.[9] Both groups noted that the tumors had sometimes become less well differentiated than at initial presentation.[8,9]

The prognosis of thyroid cancers invading the airway appears to correlate with the site and depth of invasion. Shin and colleagues classified papillary thyroid cancer invading the trachea as follows[10] (Figure 8-1):

Stage 0:	tumor confined to the thyroid gland;
Stage I:	extension through the capsule to abut the perichondrium but without cartilaginous erosion or intercartilaginous invasion;
Stage II:	destruction of cartilage or intercartilaginous invasion;
Stage III:	extension into the lamina propria of the tracheal mucosa;
Stage IV:	extension through the tracheal mucosa.

Clinical results correlated well with these stages, and in general confirmed the 1987 observations of Tsumori and colleagues.[11]

The manner in which papillary carcinoma invades the thyroid is by dissection along blood vessels and collagen fibers, oriented perpendicularly to the tracheal lumen between the cartilaginous rings.[10] The perpendicular fibers course from the collagen fibers of the peritracheal fascia, which are parallel to the tracheal wall and are contiguous with those of the isthmus of the thyroid gland. Beneath the tracheal mucosa, these intercartilaginous fibers spread into a network of reticulum. Nerve fibers and lymphatics run parallel to the perpendicular collagenous fibers between cartilages. In a series of 22 patients with papillary cancer invading the trachea, these lymphatics rarely contained tumor. In the region of the isthmus, arteries emanate from the thyroid gland. The source of tracheal invasion is most often directly from the thyroid gland, along the anatomic pathways noted, and not primarily by lymphatic metastasis. Only en bloc excision will remove such invasion. Submucosal penetration indicates a poor prognosis.

Because of the location of the thyroid gland, the subglottic larynx may also be invaded, most often in the cricoid cartilage (Figure 8-2). The corresponding recurrent laryngeal nerve is often paralyzed, paretic, or encircled by tumor. The adjacent esophagus and cricopharyngeus may be involved. The tumor may penetrate to any depth of the airway (Figure 8-3). Recurrent tumors are too often permitted to grow to large sizes before further surgery is contemplated, even though they may respond little to radioactive iodine uptake or to external radiotherapy. The added negative point of giving therapeutic radiotherapy where surgical reconstruction may later be necessary is obvious.

Diagnosis

The patient with differentiated thyroid cancer involving the airway may present with classical symptoms and signs of airway neoplasm, namely, hemoptysis, wheezing, dyspnea on exertion, and, additionally, hoarseness.

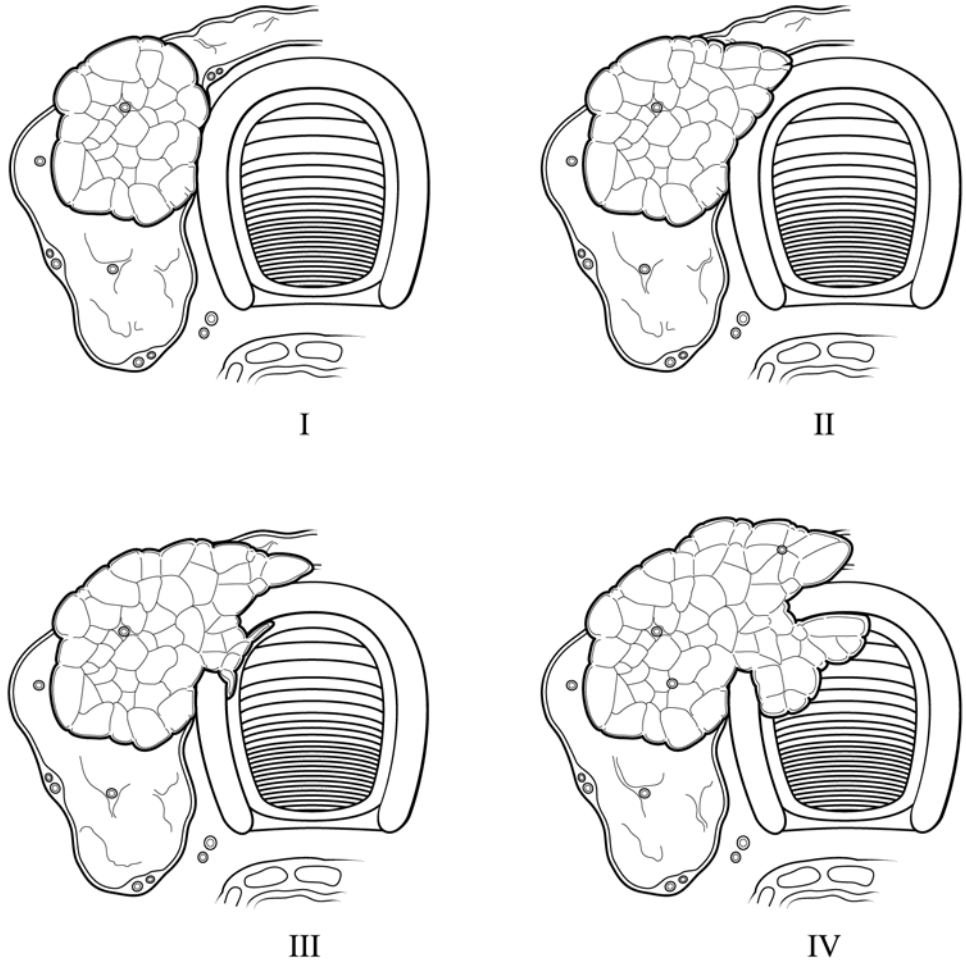

FIGURE **8-1** *Stages of papillary carcinoma of thyroid invading the trachea, based on the histopathologic extent of invasion. Adapted from Shin DH et al.*[10]

More often, airway involvement produces no symptoms, since the tumor has not yet penetrated the mucous membrane or projected any distance into the lumen. A firm mass may be palpated, which is not freely movable over the trachea. Often, tracheal and laryngeal involvement are discovered at thyroidectomy.

In my opinion, in addition to the usual diagnostic approach to thyroid cancer (thyroid function studies, thyroid scan, and needle biopsy), flexible bronchoscopy is advisable for *every* such patient, despite the rarity of visible airway invasion. However, there is not yet a completely accurate method to distinguish close abutment of the tumor to the tracheal wall from actual early invasion. Linear x-ray studies of the trachea include filtered views and crisp tomography, which are of great use in determining the extent of gross involvement of the larynx and trachea, and also the relative portion of the airway that is not involved (Figure 8-4). Fluoroscopy of the larynx adds information about the function of the vocal cords to that obtained by direct laryngoscopy (Figure 8-5). The neck should be imaged using thin section computed tomography (CT) scans, which are most likely to identify involvement of the tracheal wall or intrusion into the lumen (Figure 8-6). CT scanning should include the chest, to search for pulmonary metastases. Magnetic resonance imaging (MRI) is also useful in defining these lesions. Barium swallow may define the bulk of tumor and suggest esophageal involvement. Preoperative studies that are appropriate for exenteration, which may be considered for massive invasive tumors, are described in Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy."

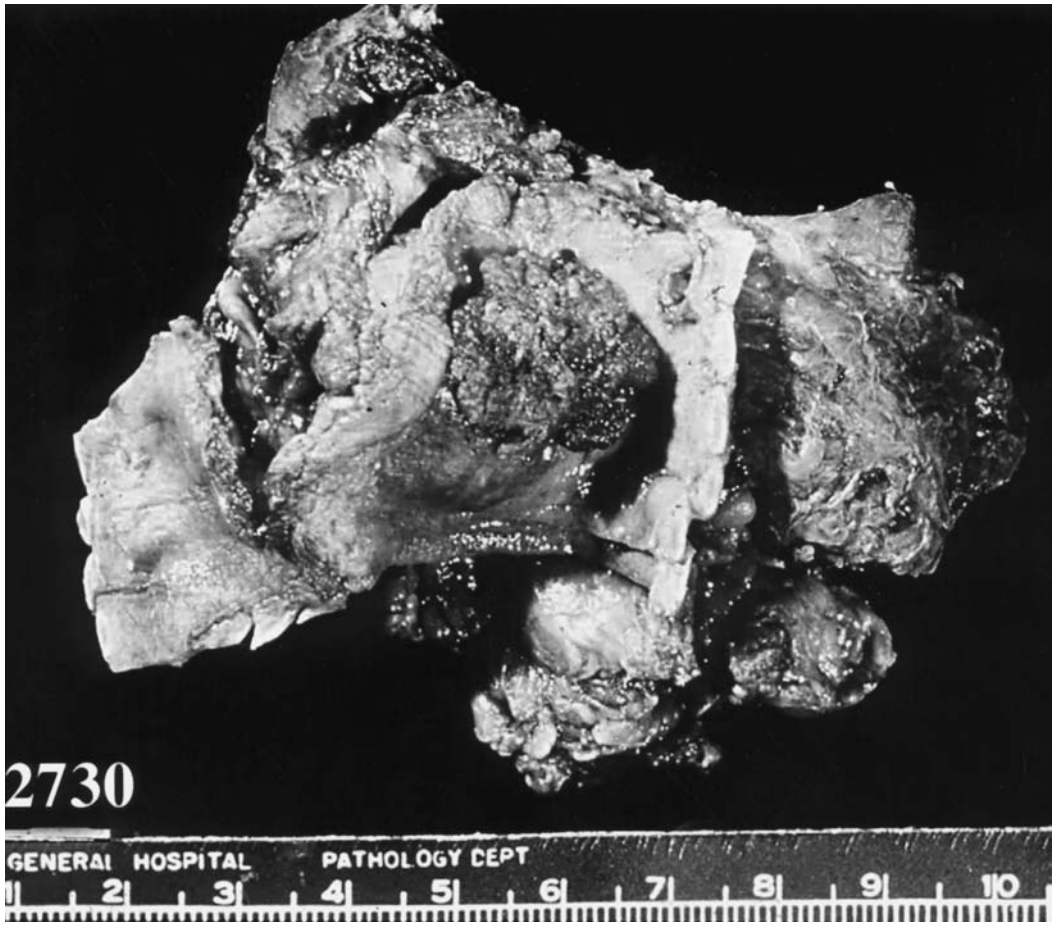

FIGURE **8-2** *Papillary thyroid carcinoma in a 68-year-old male invading the upper trachea and left side of the cricoid.*

Principles of Surgical Treatment

The currently accepted principles of surgery for differentiated thyroid cancer call for complete removal of the local lesion and extensions in the neck. Longer survival and better control of symptoms are obtained if gross tumor is fully removed. This is usually interpreted to mean thyroidectomy and excision of involved regional lymph nodes, with persisting differences of opinion about the need for total thyroidectomy. Because of the pathological behavior of these tumors, nodal metastases are excised by limited regional dissection rather than by standard radical neck dissection. Even in extended node dissection, adjacent structures, such as the sternocleidomastoid muscle and internal jugular vein, are spared whenever possible. The submandibular triangle is rarely involved, but on the other hand, positive nodes do occur pretracheally, in the tracheoesophageal groove, along the length of the internal jugular chain, in the "V" between the innominate and left carotid arteries, and in the posterior cervical triangle. The goal of surgery, in addition to cure, is to prevent airway obstruction and death from asphyxiation.

The otherwise guiding surgical principle of complete local removal of thyroid neoplasm is all too often broken when a tumor invades the upper trachea or the junction of the larynx and trachea. Frequently unfamiliar with techniques of airway surgery, the thyroid surgeon regards the addition of tracheal resection as "radical surgery," potentially fraught with morbid or fatal consequences. Hence, "shave" techniques have been advocated.[12–16] In experienced hands, however, airway reconstruction is *not* radical surgery. Addition of

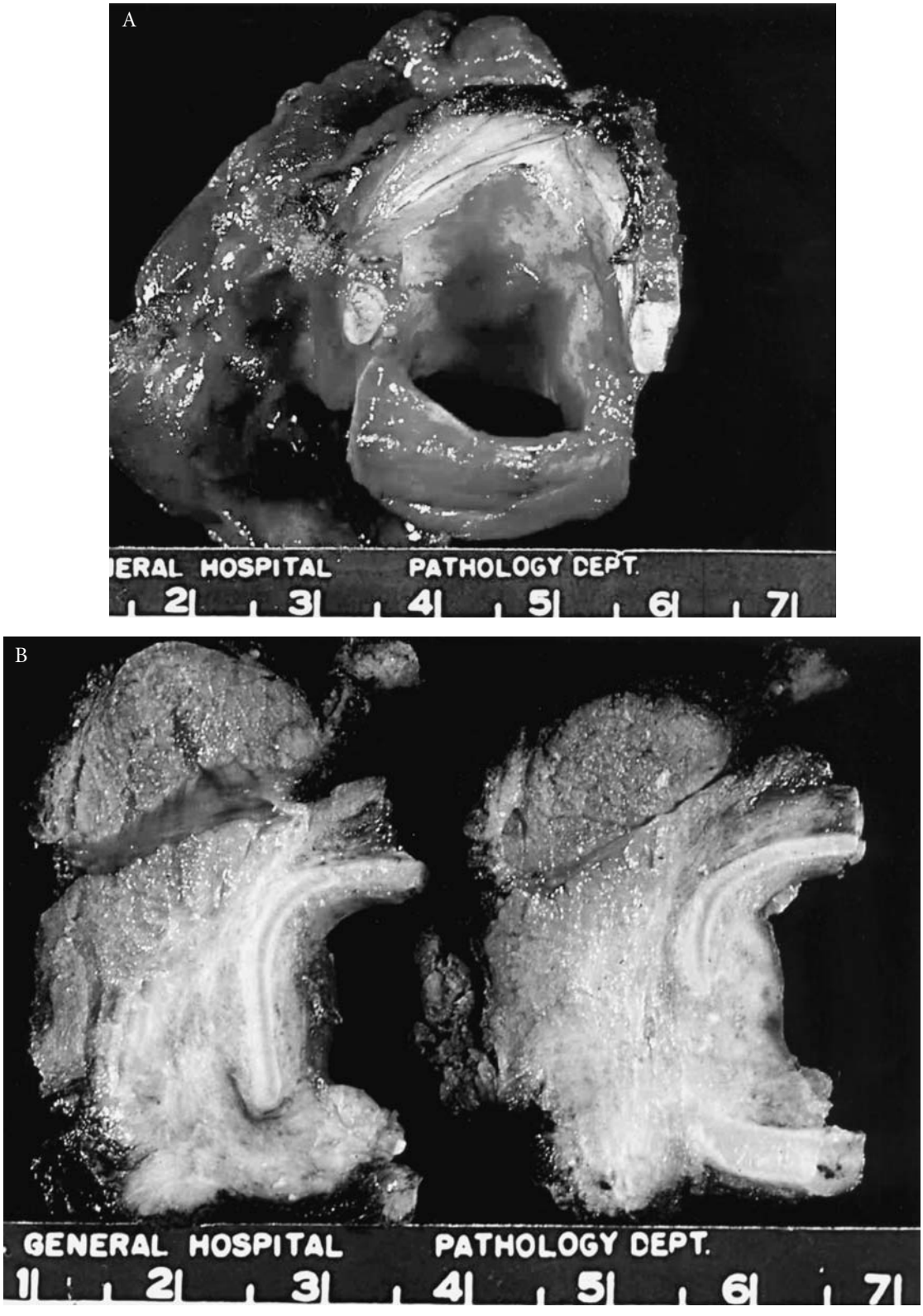

FIGURE **8-3** A, *Surgical specimen (mixed papillary and follicular carcinoma) of a patient, whose x-rays are shown in Figure 8-4, viewed from above. The broad arc of the partially resected cricoid is above in the photograph. Invading tumor is just below.* B, *The tumor extends between and through the cartilages. Seven years after resection of the obstructing tumor, the patient developed pulmonary metastases, which were treated with* ^{131}I.

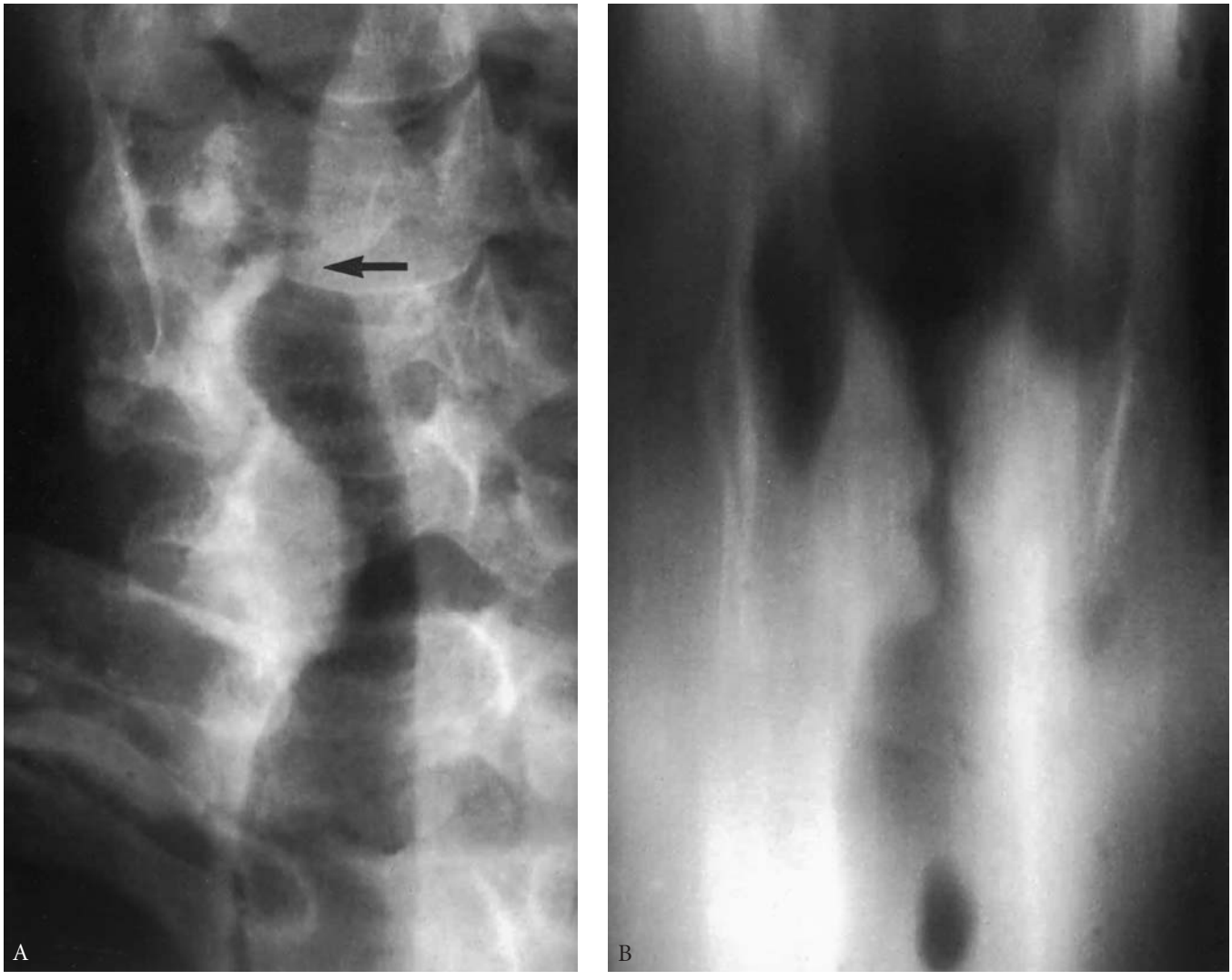

FIGURE **8-4** *Radiologic delineation of linear extent of tracheal invasion by thyroid carcinoma.* A, *Filtered view of tracheal invasion by mixed papillary and follicular carcinoma in a 59-year-old man with identified pulmonary metastases.* Arrow *marks the glottis. Nonetheless, the patient enjoyed 14 years of life after tracheal resection and reconstruction, and had no further airway disease.* B, *Tomographic cut showing high invasion in the subglottic larynx by papillary carcinoma in a 36-year-old man. The right vocal cord is paralyzed.*

tracheal resection following dissection for thyroidectomy adds little length or complexity to the operation and does not increase morbidity or mortality much.[17,18] Voice, airway, and deglutition are all preserved.

What *is* lacking are firm criteria about what constitutes an adequate "shave," histologic identification of complete or incomplete tumor removal by shaving, the decades of follow-up necessary to validate this unusual oncologic approach, or consideration of the potential for change in the histology and aggressiveness of thyroid cancer.[8,9] Many of our patients had been previously subjected to shaving procedures as initial and ultimately unsuccessful treatment, years before recurrence.[17] Added to these considerations is the indication from our data that excision of airway involvement at initial thyroidectomy, or immediately thereafter, leads to better long-term results than late removal of a recurrent invasive tumor.[17] The purposes of complete resection of thyroid cancer that invades the airway are, therefore, 1) to relieve or prevent airway obstruction in patients with slowly progressing neoplasm, 2) to prevent tortured death by asphyxiation or hemorrhage, and 3) perhaps to achieve cure by early complete resection of the tumor (Figure 8-7).

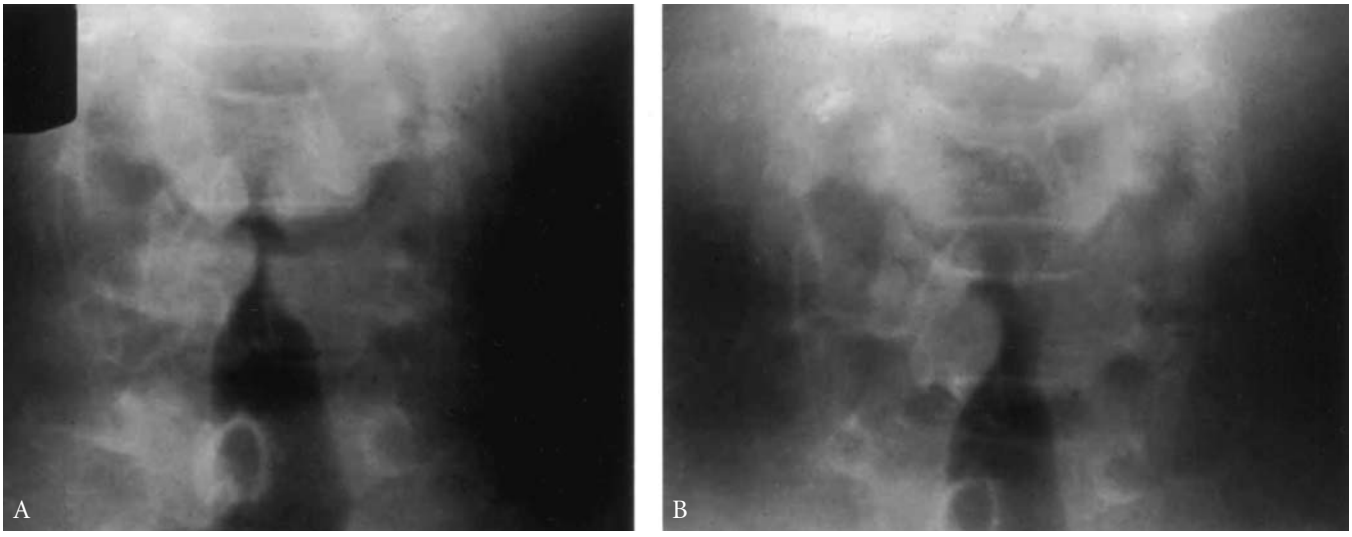

FIGURE **8-5** *Vocal cord paralysis or dysfunction due to recurrent laryngeal nerve invasion by papillary thyroid carcinoma, in a 36-year-old man, is well demonstrated on fluoroscopy or by direct examination. On these spot films taken during fluoroscopy,* A, *a paralyzed right cord is evident on attempted cord adduction. Note the asymmetry of the vocal cords.* B, *On inspiration, the tumor is also seen just below the glottis.*

Radical removal of the larynx, trachea, and other affected tissues en bloc may be justified only in rare cases of seemingly confined undifferentiated carcinoma (Figure 8-8) and for palliation of longstanding massively recurrent and severely symptomatic differentiated carcinoma (Figure 8-9).[17]

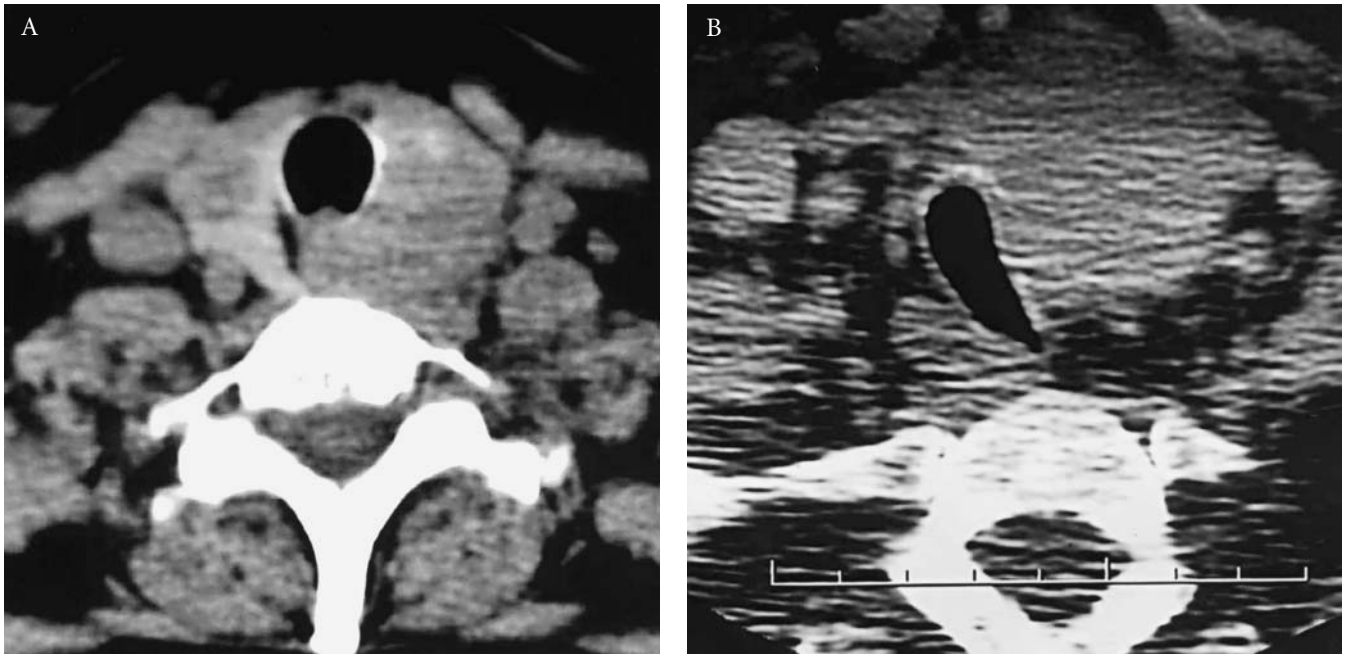

FIGURE **8-6** *Computed tomography scans of invasion by differentiated carcinoma.* A, *In a 65-year-old woman with papillary carcinoma invading the trachea, esophagus, cricoid, and right recurrent laryngeal nerve. Treated by thyroidectomy, with tracheal resection and reconstruction.* B, *In a 51-year-old man with a very large papillary lesion invading and compressing the trachea. Resection with reconstruction was nonetheless possible because the length of trachea invaded was less than the total extent of tumor apparent on the scan. Six years later, the patient remained free of recurrence.*

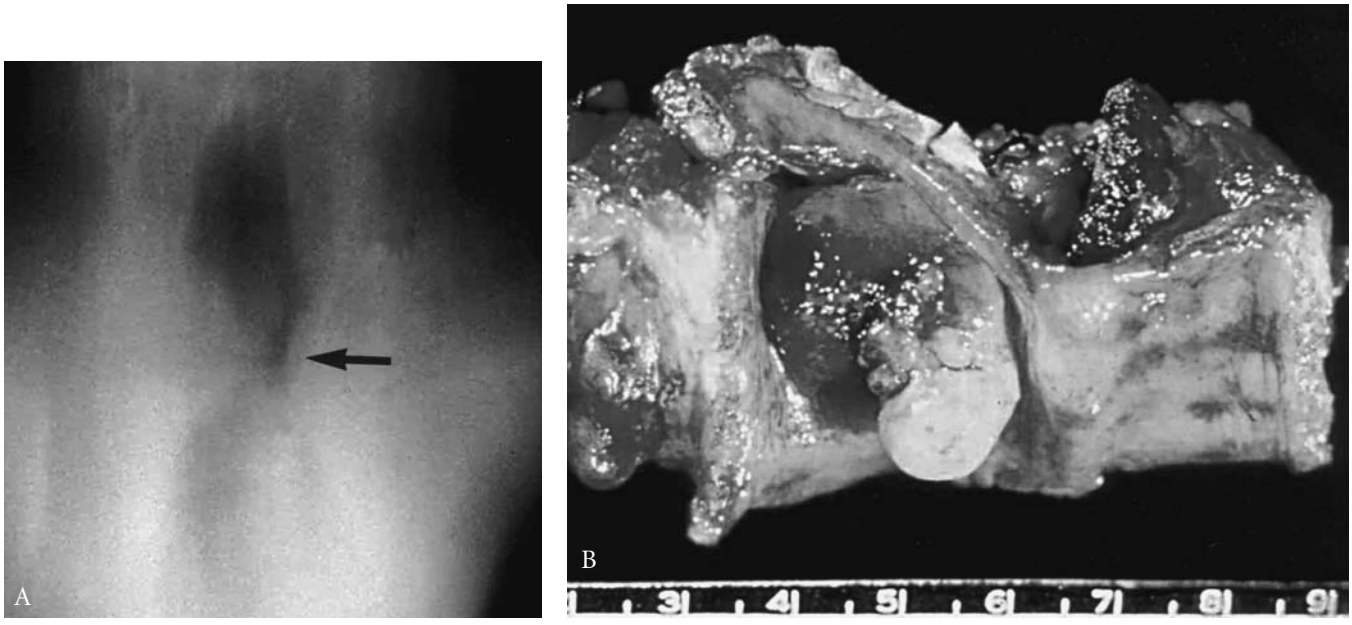

FIGURE **8-7** *Severely obstructive papillary carcinoma in a 62-year-old man who had undergone thyroidectomy 6 years and 4 years earlier, followed by* ^{131}I. A, *Tomogram shows marked occlusion of the proximal trachea* (arrow). B, *Resected specimen includes a portion of the cricoid cartilage.*

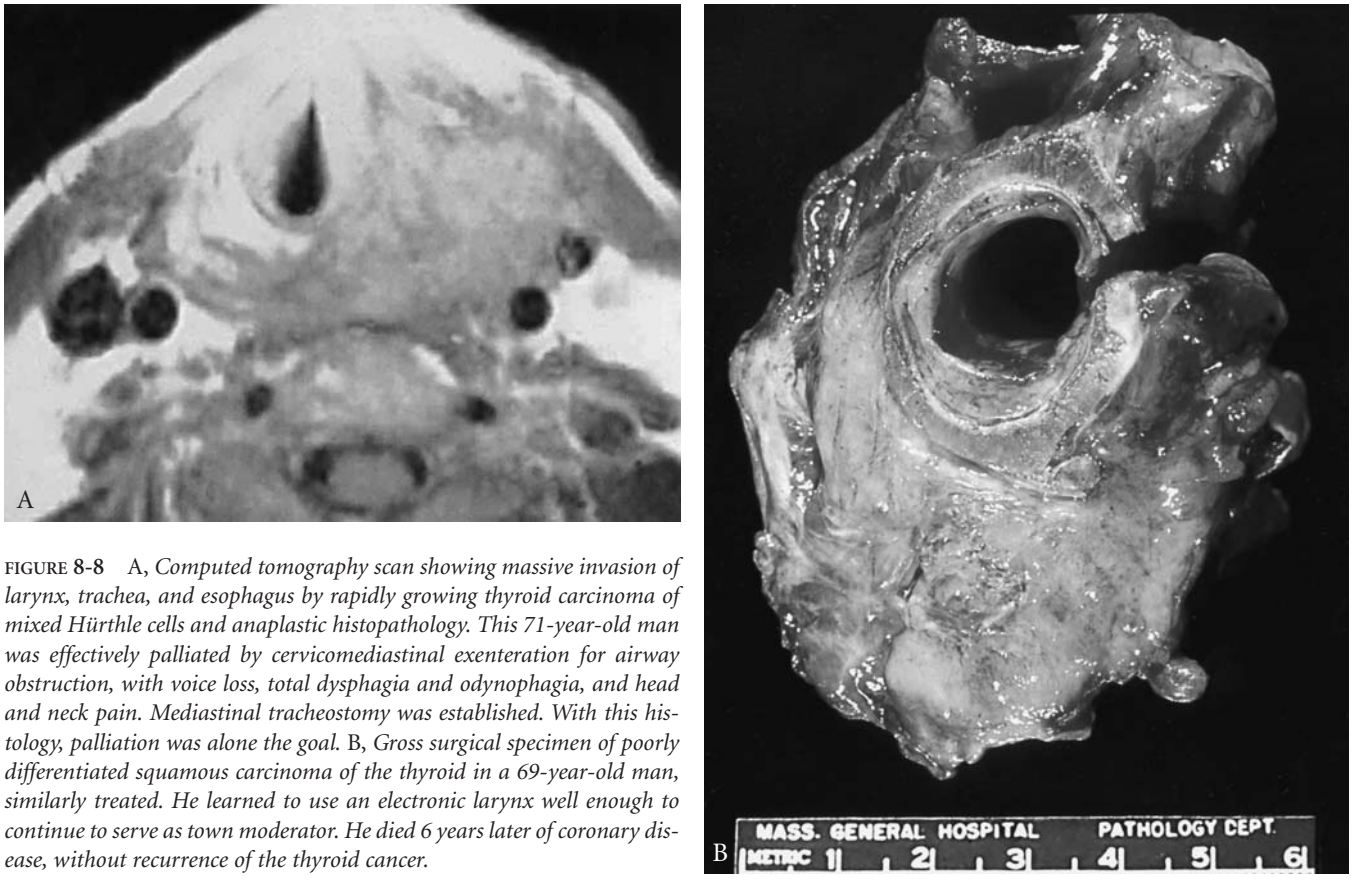

FIGURE **8-8** A, *Computed tomography scan showing massive invasion of larynx, trachea, and esophagus by rapidly growing thyroid carcinoma of mixed Hürthle cells and anaplastic histopathology. This 71-year-old man was effectively palliated by cervicomediastinal exenteration for airway obstruction, with voice loss, total dysphagia and odynophagia, and head and neck pain. Mediastinal tracheostomy was established. With this histology, palliation was alone the goal.* B, *Gross surgical specimen of poorly differentiated squamous carcinoma of the thyroid in a 69-year-old man, similarly treated. He learned to use an electronic larynx well enough to continue to serve as town moderator. He died 6 years later of coronary disease, without recurrence of the thyroid cancer.*

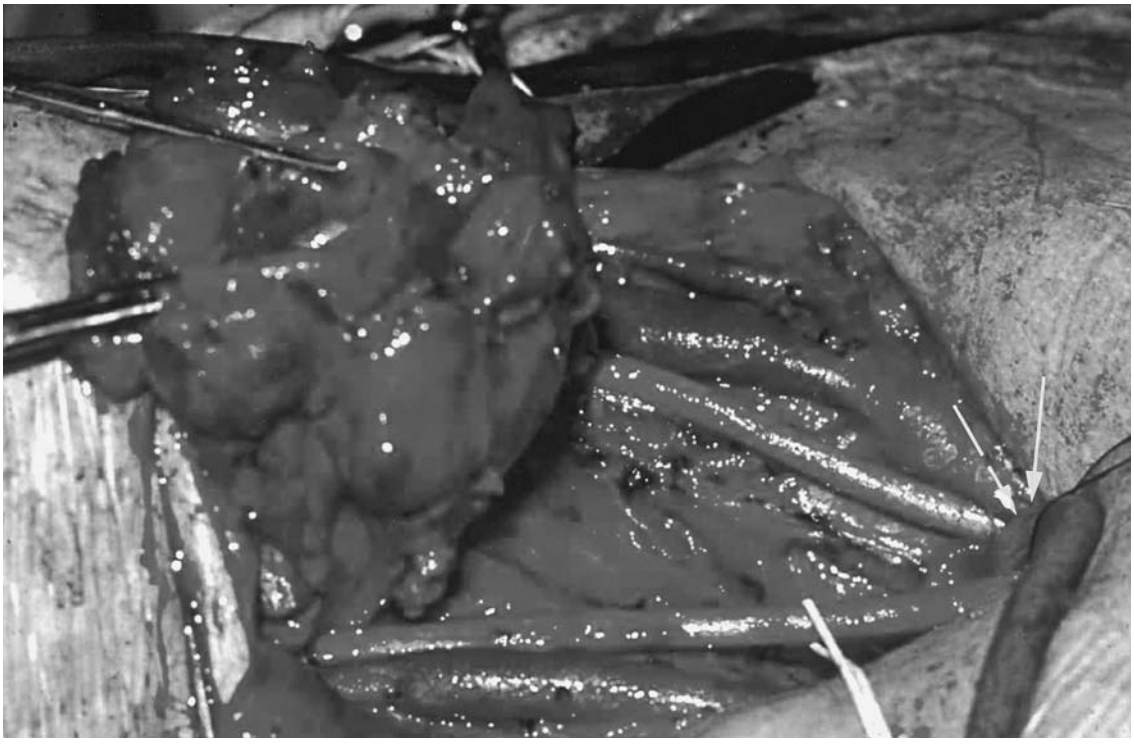

FIGURE **8-9** *A 62-year-old man with massive recurrent papillary thyroid carcinoma following thyroidectomy, 4 years previously, and subsequent treatment with ^{131}I. The disease progressed to cause pain, bleeding, airway and esophageal obstruction, and loss of voice. The operative photograph shows the mass specimen including thyroid, larynx, trachea, and esophagus being removed by en bloc dissection. The neck is at the left. On the right, the upper sternum, heads of clavicles, and upper two costal cartilages have been excised to provide access for mediastinal tracheostomy. The flexible endotracheal tube in the right lower corner is in the proximal end of the trachea* (arrow). *The floor of dissection reveals carotid arteries, internal jugular veins, and prevertebral fascia. Good palliation was attained for a number of years. Pulmonary metastases appeared.*

Resection and reconstruction of the involved airway as part of a complete local excision of thyroid cancer, especially as an initial procedure, accomplishes the primary goals of conventional thyroid cancer surgery. It does *not* represent a radical extension of surgery attended by great hazards. Follow-up results strongly suggest that the best long-term results are obtained either 1) in those patients in whom the involved airway is removed at the initial resection of tumor or 2) where invaded airway is removed as soon as possible after identification at initial thyroidectomy. Prolonged palliation has been achieved by late removal of the airway invaded by a recurrent tumor, but it rarely provides a cure, even though patients had appeared earlier to run an indolent course. Palliative resection of an obstructed airway seems justified, even in the face of pulmonary metastases when the tumor is known to be slowly progressive.[17] Pulmonary metastasectomy has not been shown to be of value in thyroid cancer.[19] Most often, the recurrent laryngeal nerve that has to be sacrificed is already involved by tumor, and so no further functional loss follows.

Management

Resection of the airway may require 1) simple circumferential removal of a segment of the upper trachea, 2) bevelled resection of one side of the anterolateral cricoid if it is involved, or 3) complex resection in which a portion of subglottic larynx on the invaded side is removed in a "bayonet" fashion and the distal trachea is tailored to repair the defect (Figure 8-10). "Window" resections are to be avoided because of the increased likelihood of leaving residual tumor and the less kindly healing of a trachea patched with

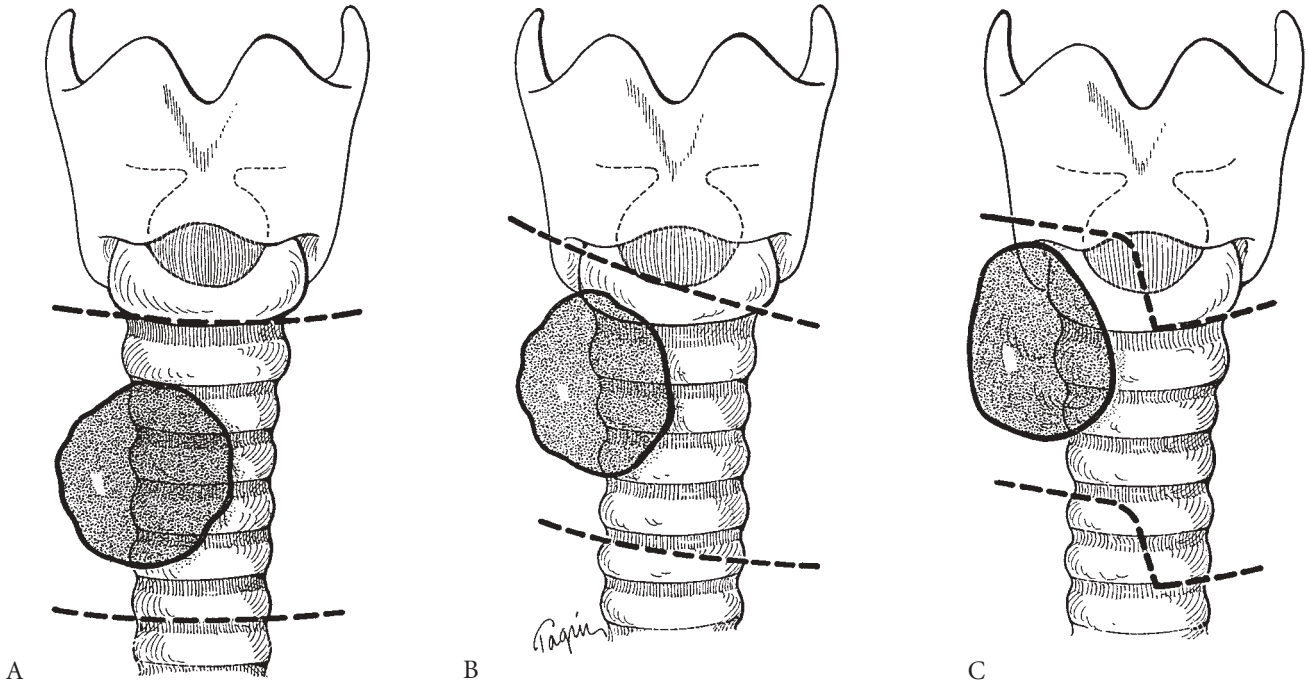

FIGURE **8-10** *Modes of resection of thyroid cancer invading tracheae.* A, *Cylindrical tracheal resection. Because of the location of the thyroid gland, invasion most frequently requires that proximal transection of the trachea be just below the cricoid cartilage.* B, *Varying amounts of cricoid must often be removed on the side of the tumor, from a slightly oblique bevelled resection to a nearly complete lateral excision, as diagrammed.* C, *"Bayonet" resection, where invasion of the cricoid is so extensive that the line of transection must lie somewhere beneath the vocal cord on that side. The inferior line of tracheal transection in this case is fashioned to fit the proximal laryngeal defect.*

autologous mesenchymal tissue. The mesenchymal surface encourages granulation tissue formation and subsequent contraction. If such a window can be managed by insertion of a tracheostomy tube alone, the resection is usually of an inadequate extent. Surgical approach and techniques of resection and reconstruction are described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection," and Chapter 25, "Laryngotracheal Reconstruction." The technique of cervicomediastinal exenteration is described in Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy."

Cervicomediastinal exenteration should be applied selectively; that is, only in the rare case where an invasive anaplastic or undifferentiated carcinoma appears to be totally resectable by such en bloc resection, or where there is highly symptomatic massive late recurrence of differentiated carcinoma, usually after multiple unsuccessful treatment by thyroidectomy, ^{131}I, and sometimes external beam irradiation. Such an effort is principally palliative, but it does indeed provide the patient with a measure of comfort, as an alternative to the misery caused by a progressively extensive local disease.

Results of Treatment

Total resection of the larynx and upper trachea was early performed for both well and poorly differentiated extensively invasive carcinoma of the thyroid, in some patients with surprisingly long-term palliation or apparent cure.[20,21] Patients with a well-differentiated carcinoma involving the trachea or adjacent larynx, in limited enough fashion to be resectable along with the involved airway and yet permit primary reconstruction, were of course not completely resected prior to development of contemporary airway surgery. The techniques of airway reconstruction that evolved were only slowly applied in these patients. Grillo reported a case in 1965,[22] whereas Ishihara and colleagues reported 11 patients in 1982.[5] In 1986, Grillo and

Zannini described 19 patients who underwent resection for papillary or mixed papillary and follicular carcinoma of the thyroid, and 3 patients for undifferentiated carcinoma.[23] Sixteen of the patients had primary reconstruction performed and 6 were treated with en bloc cervicomediastinal resection with mediastinal tracheostomy. Fifteen of the 16 patients who underwent airway reconstruction had good surgical results and speech preservation. Eight patients were alive without disease up to 9 years later, and only 2 developed airway recurrence. In 1989, Maeda and colleagues recorded 151 patients with tracheoplasty for thyroid carcinoma in Japan, which represented nearly 27% of all tracheoplasties done in Japan up to that date.[24]

By 1991, Ishihara and colleagues had performed 60 resections for thyroid cancer, with 41 needing laryngotracheal anastomosis.[18] Five- and 10-year survival rates were both at 78% with complete resection, and at 44 and 24% respectively with incomplete resection. In 1992, Grillo and colleagues described 34 of 52 patients who underwent resection, 27 with airway reconstruction and 7 with cervicomediastinal exenteration (Figure 8-11).[17] Older patients predominated (Figure 8-12). Ten of the 27 reconstructed airways required laryngotracheal resections. The length of airway resection averaged 3.5 cm, a length that normally permits reconstruction without difficulty (Figure 8-13). Eighteen were not resected for reason of either distant metastases, excessive local extension, or to preserve laryngeal function where the only alternative was laryngectomy. In 2 of these latter patients, laryngotrachiectomy was deferred for a number of years. Of the 27 patients with reconstructed airways, it is notable that 9 had no prior treatment, 5 had been referred immediately after a surgeon identified invasion by tumor intraoperatively, and 13 arrived following recurrence at 1 to 47 years after the tumor had initially been shaved off the trachea. One patient died from local necrosis related to 4,800 cGy of irradiation, given 6 years previously, prior to the use of an omentum for vascular augmentation in previously irradiated tracheas (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation"). In a second patient, earlier postoperative tracheostomy would have avoided respiratory arrest due to edematous airway obstruction.

The long natural history of differentiated thyroid carcinoma cautions on conclusions about long-term results (Figure 8-14). In the Massachusetts General Hospital (MGH) series, 11 of 25 patients died of cancer in 3 months to 10.25 years but only 2 had airway difficulty, indicating achievement of the principal goals—long-term palliation and obviation of death by airway obstruction.[17] Twelve patients were without cancer for up to 14.5 years and 1 had pulmonary metastases. The average survival for 7 patients operated upon with known pulmonary metastases was 4.2 years, with the longest surviving at 10.5 years. Nine of 13 survivors without cancer had an airway resection done either as an initial treatment or were referred immediately after a surgeon discovered the invasion, which was far more promising than those treated for late recurrence after a remote prior thyroidectomy.

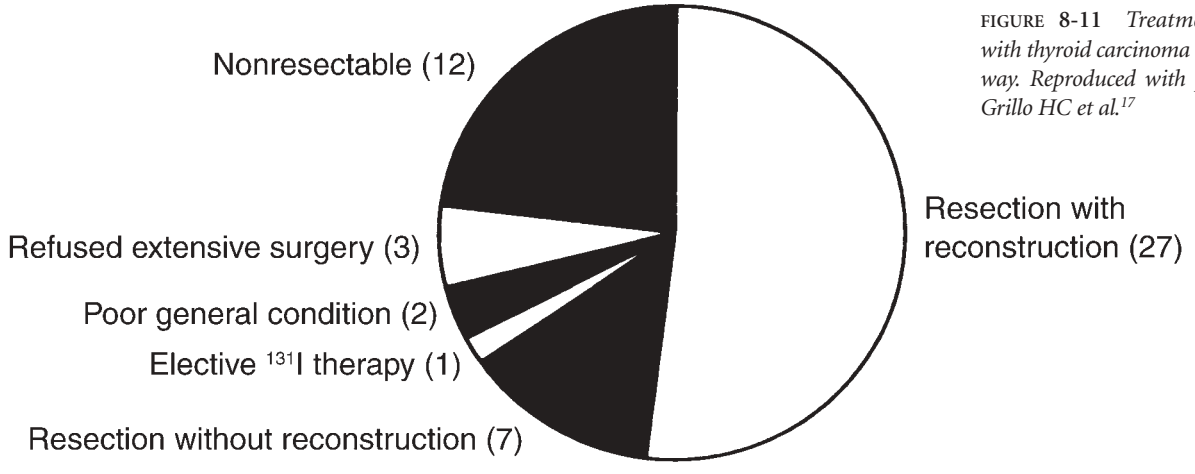

FIGURE **8-11** *Treatment of patients with thyroid carcinoma invading the airway. Reproduced with permission from Grillo HC et al.*[17]

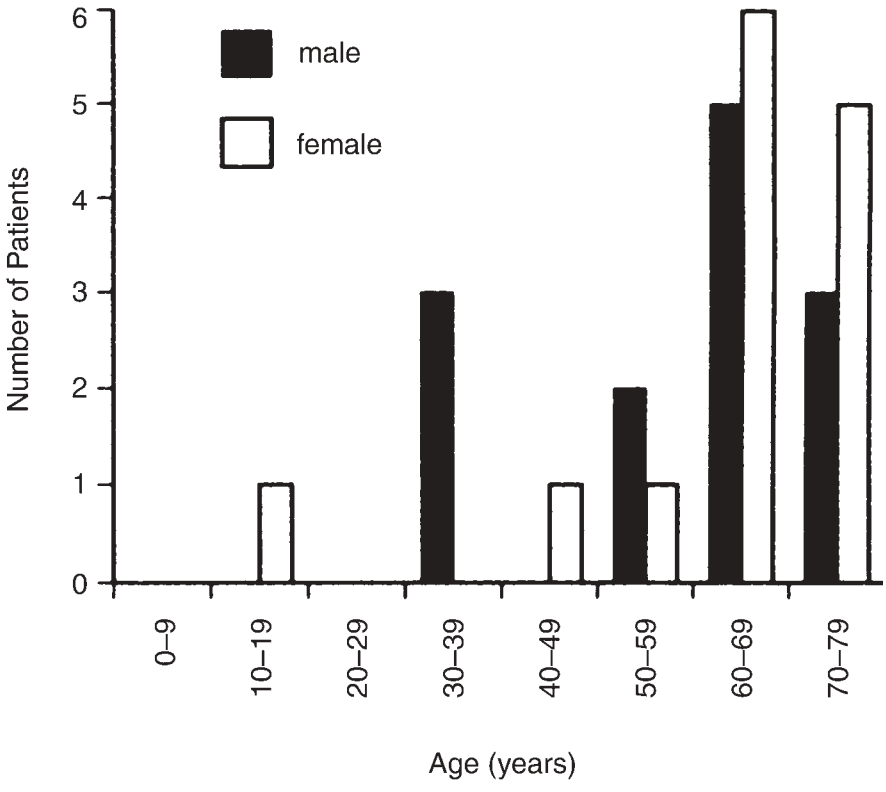

FIGURE **8-12** *Distribution by age and gender of patients undergoing resection and reconstruction for thyroid carcinoma. Reproduced with permission from Grillo HC et al.*[17]

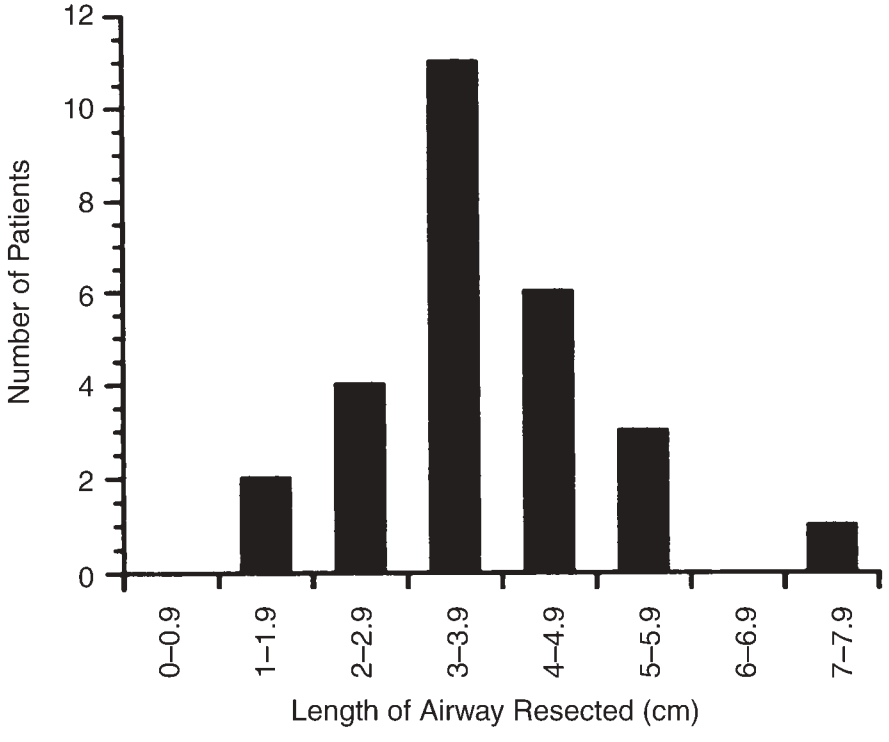

FIGURE **8-13** *Distribution of length of airway resected, where tracheal reconstruction was done. Reproduced with permission from Grillo HC et al.*[17]

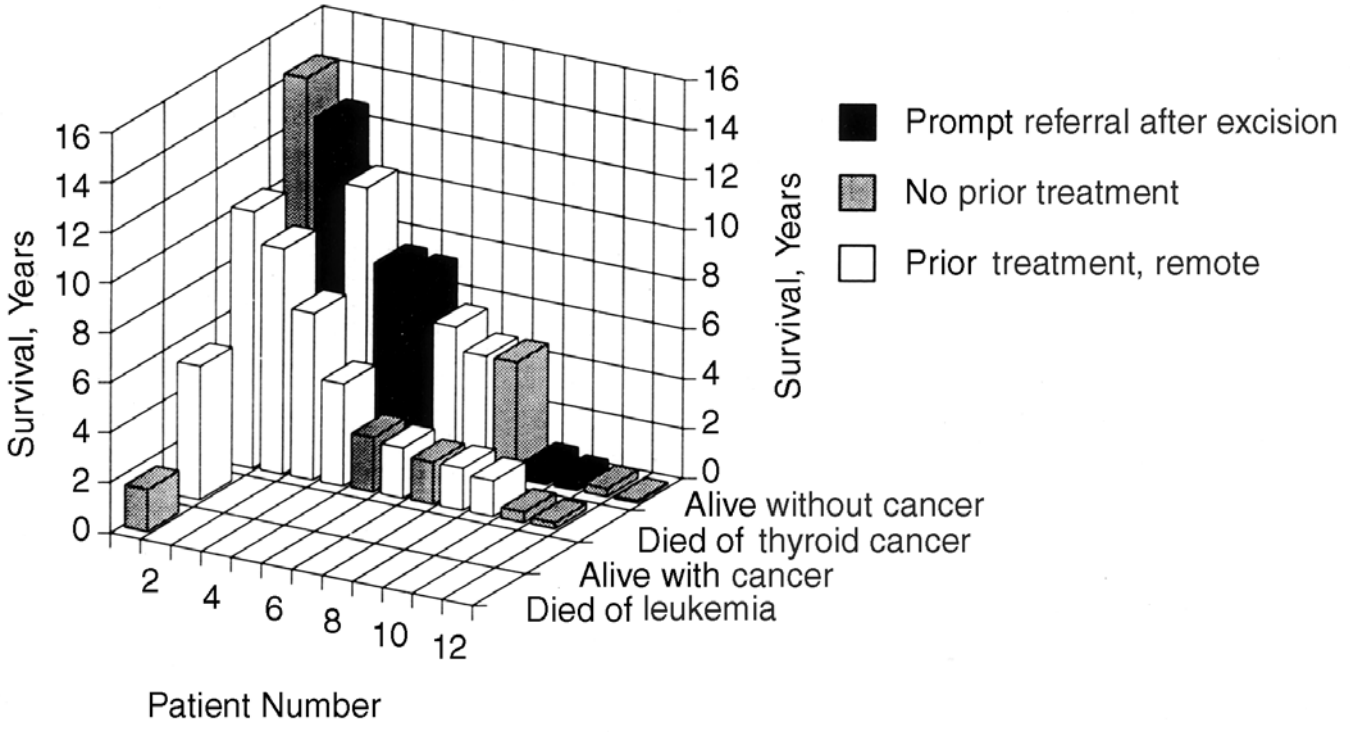

FIGURE **8-14** *Performance of 25 patients who survived tracheal resection and reconstruction for thyroid carcinoma. Reproduced with permission from Grillo HC et al.*[17]

Recommendations

The illustrative examples by Grillo and Ishihara and their colleagues[17,18] have been amply confirmed by others in the last decade.[17,18,25–30] Airway resection and reconstruction for differentiated thyroid cancer is safe, has a low morbidity and mortality and provides prolonged palliation, prevents death by asphyxiation or hemorrhage, and probably achieves cure in some patients.

At issue with recommendation of the shave technique as a procedure of choice for stage I patients, is whether appropriate patients can be accurately selected by imprecise operative standards or by stratification of patients with presumed low-risk tumors, and the latter with certainty that the degree of differentiation will not change over the years. Also in question is the validity of surgical motivation to avoid simple extirpative surgery on the mistaken premise of high risk, where in fact added risk has been shown to be minimal and surgical results to be very good.[12–16]

Although this controversy will not be settled for some time, I support a consistent policy of complete local resection of all known tumor at the initial operation. Preoperative study to include CT and bronchoscopy will identify many patients who require airway resection and reconstruction. If the operator first discovers invasion at surgery and is uncomfortable with airway surgery, early referral for reoperation has proved to be effective.[17] The larynx should be salvaged wherever possible, even if later removal might become necessary, even accepting an unknown increase in hazard of metastasis. ^{131}I and especially external beam irradiation are better employed *after* thyroidectomy and airway reconstruction.

Airway resection and reconstruction for late recurrent tumor in the airway provides the best palliation and prevention of bleeding and obstruction. In highly selected patients, radical resection including laryngectomy may be indicated.[17,31]

Bronchogenic Carcinoma

Bronchogenic carcinoma involving the proximal main bronchus (within 2 cm of the carina) is classified as a T3 lesion and that extending to the carina as T4. As surgical experience has grown, T3N0M0 lesions (including T3 lesions other than bronchial) have been moved to stage IIB, but T4N0M0 lesions remain in stage IIIB. The latter was based upon lack of familiarity with techniques of carinal resection and, in particular, of carinal pneumonectomy. A T4N0M0 lesion, by virtue of the carinal location, should be stage IIIA. T3 lesions due to main bronchial involvement, especially on the right, also usually require carinal resection in order to obtain an acceptable surgical margin. Squamous cell lesions centered at the carina may perhaps be considered as primary carinal neoplasms, in the way that a slightly more proximal lesion is a primary low tracheal carcinoma. The division is arbitrary but a localized central lesion is potentially treatable without loss of lung rather than by carinal pneumonectomy.

Tumors suitable for excision by carinal pneumonectomy (or tracheal sleeve pneumonectomy) are predominantly squamous in type and right sided. Mathey and Jensik and their colleagues were among the earliest to practice sleeve pneumonectomy for bronchogenic carcinoma other than episodically.[32,33] (For a fuller account, see Introduction, "Development of Tracheal Surgery: An Historical Review.") Over a 15-year period, carinal resection for bronchogenic carcinoma was reported in North America by Deslauriers and colleagues, Jensik and colleagues, and Mathisen and Grillo, in Europe by Dartevelle and colleagues, Perelman and Koroleva, and Roviaro and colleagues, and in Japan by Isihara and colleagues and Watanabe and colleagues, among others.[34–41] Excessive mortality initially accompanied this surgery but has improved with time (Table 8-1).

Governing Principles

When bronchogenic carcinoma of the upper lobes invades the trachea directly from parenchymal contiguity, the disease is usually so extensive that segmental resection of the trachea is precluded. In a very rare circumstance, an extensive disease that invades the superior vena cava and adjacent edge of the trachea may still be grossly completely resectable. If the linear extent of tracheal invasion obviates segmental resection, lateral resection, despite its contraindications, may be considered. Repair may be made with a pedicled pericardium to preserve its viability, supported by Marlex. This repair must not be circumferential. Even so, granulations are likely to result and the hazard of mediastinal leakage is enhanced. A pedicled intercostal muscle may also be considered. On the other hand, when bronchogenic carcinoma is centered in the main bronchus or extends up to the carina, the patient should be considered for resection (see Chapter 29, "Carinal Reconstruction").

Carcinoma in the right upper lobe bronchus easily extends up the short length of the right main bronchus to the main carina. Carcinoma of the left upper lobe, with its considerably longer bronchus, is less likely to invade the carina. The number of right carinal pneumonectomies for bronchogenic carcinoma is therefore far greater than of the left. The disease must be localized enough so that resection of the

Table 8-1 Representative Results of Carinal Resection for Bronchogenic Carcinoma

Author (Year)	*Reference*	*Operative Number*	*Mortality %*	*5-Year Survival %*
Jensik (1982)	35	34	29	15
Deslauriers (1989)	34	38	29	13
Watanabe (1990)	41	12	17	
Mathisen, Grillo (1991)	36	37	19	19
Roviaro (1994)	39	28	4	20
Dartevelle (1996)	43	60	7	43
Mitchell (2001)	45	60	15	42

carina will not lead to reconstruction under tension. The usually safe limit for resection by right carinal pneumonectomy is approximately 4 cm in length between the points of the tracheal and left main bronchial division. Elevation of the left main bronchus is limited by the aortic arch. Carinal resection alone, with preservation of both lungs, can be somewhat more extensive because of the possibility for intrapericardial mobilization of the right lung, which is unrestricted by the aortic arch. In this case, the right main bronchus may be elevated in the thorax to reach the distal tracheal stump. The technique depends upon the length of tracheal resection. This also applies to the rarer left carinal pneumonectomy for bronchogenic carcinoma. In a few appropriate patients, the right lower or lower and middle lobes may be salvaged. Special precautions apply, as noted in Chapter 29, "Carinal Reconstruction."

Considerations regarding lymph node involvement are the same as for any resection of carcinoma of the lung. N3 disease conventionally places the disease out of bounds. N2 disease should be resected only as part of a protocol approach with neoadjuvant therapy, as in other cases of bronchogenic carcinoma. In these patients, the outlook remains poor. Because of the extent of the operation and a higher mortality rate, which is greater than for pneumonectomy alone, it is always important to search exhaustively for possible remote metastases prior to performing the resection. N2 disease heightens the probability of distant metastases. Carinal resection because of tumor invasion from metastases in subcarinal lymph nodes is not advised.

In our series of 58 patients, 42 had squamous carcinoma, 10 had adenocarcinoma, 4 had large cell carcinoma, 1 had small cell carcinoma, and 1 had bronchoalveolar cell carcinoma.[42] Particularly in right carinal pneumonectomy, significant interruption of tracheobronchial lymphatics necessarily follows, even where effort has been made to avoid a radical mediastinal lymphadenectomy.

Diagnosis and Evaluation

Involvement of the carina by bronchogenic carcinoma must be assessed with great care by conventional imaging, which includes CT scan of the chest and upper abdomen. Crisp carinal tomograms can be useful to demonstrate the gross extent of the lesion, both within and without the lumen of the trachea, and to make clear the relative portion of airway that seems to be uninvolved by tumor (Figure 8-15). Final bronchoscopic assessment is best made with the Storz Hopkins magnifying telescopes through a rigid bronchoscope. Biopsies of tracheal mucosa proximal to the visible tumor may help to establish the feasibility of resection. Mediastinoscopy is very important for assessment of lymph nodes beyond the information obtained from CT scan. Mediastinoscopy is preferably performed concurrently with a planned resection so that tissue planes and definition of the tumor will not become obscured by inflammation and scar. Should preoperative adjunctive therapy be given following mediastinoscopy because of the finding of N2 lymph nodes, a further en bloc resection at a later date will have to encompass all node-bearing tissue, and accept partly obscured tissue planes. The role of positron emission tomography (PET) scanning in comparison with mediastinoscopy has yet to be clarified, but is likely to remain less exact.

Metastases must be carefully sought by CT examination of the liver and brain and by bone scan. The increased surgical risk of the operation makes these studies mandatory.

Patients being considered for carinal resection for bronchogenic carcinoma must be evaluated for total pulmonary function and distribution of ventilation and perfusion and blood gases. Smoking must have stopped. Sometimes, the involved lung contributes little to total respiratory function if the bronchus is severely obstructed. Cardiac function is also carefully assessed. In a few patients where pneumonectomy would not be tolerated, but where tumor is sufficiently localized, it is possible to perform carinal resection and right upper lobectomy with reimplantation of the middle and lower lobes or of the lower lobe (Figure 8-16). In such a case, involvement of the pulmonary artery is a crucial matter, and angiography may be needed. The final decision to proceed is made only after thorough exploration and before irrevocable surgical steps are

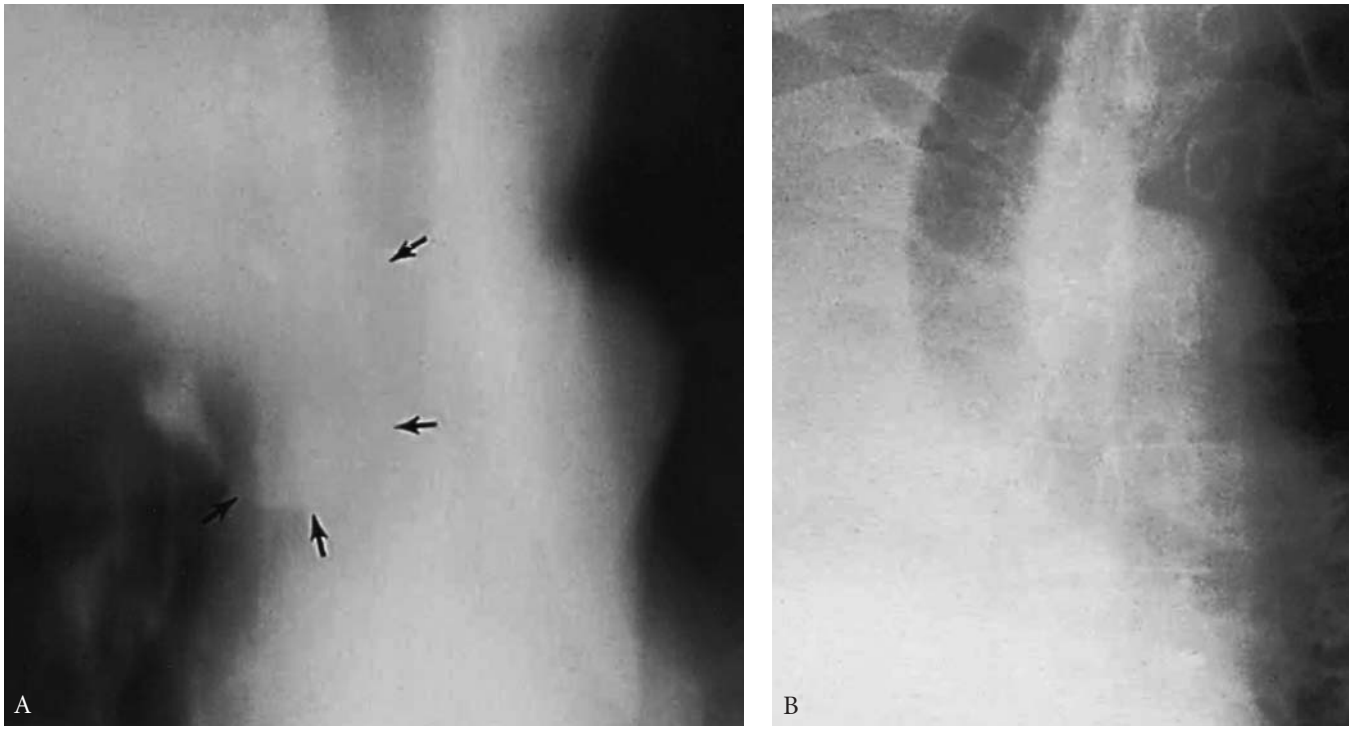

FIGURE **8-15** A, *Tomogram of large cell carcinoma* (arrows) *involving the lower trachea, carina, and right main bronchus in a 66-year-old man.* B, *Postoperative tomogram. The patient died 9 years later of other causes and without recurrence.*

taken. There are special hazards in such complex reconstruction that require precautions in order to avoid excessive anastomotic tension.

Management

The technique for carinal resection (tracheal sleeve pneumonectomy) for bronchogenic carcinoma is described in Chapter 29, "Carinal Reconstruction," and anesthesia in Chapter 18, "Anesthesia for Tracheal Surgery." Resection of a carina and a bronchial stump for residual tumor or for strictly localized recurrence may be indicated, but only after rigorous preoperative and intraoperative assessment. Dartevelle and Macchiarini additionally recommend introducing latissimus dorsi and serratus anterior muscles to buttress the anastomosis, and performing a tailoring thoracoplasty to obliterate the pleural space for infection control.[43] We have not done this in a "clean" operation.

Initial experience with carinal resection for bronchogenic carcinoma was discouraging, with mortality rates of nearly 30% reported by Jensik, Deslauriers, and their teams.[33,34] Our initial early postoperative mortality rate was 8%, but delayed mortality was 11%, due principally to anastomotic complications from tracheal reimplantation of the residual right lung, hence totalling a mortality rate of 19%.[36] This contrasted with an 8 to 12% mortality rate for carinal resections for primary tracheal tumors.[44] Nonetheless, inclusive of the unfavorable early cases noted, our total mortality rate dropped to 15.5% by 1998.[42] The mortality rate of 11% reported by Dartevelle and colleagues in 1988 is assuring.[37] Our surgical mortality dropped to 10% in the second half of our series, varying with type of resection.[45] A very significant part of early postoperative mortality was due to a particularly aggressive and rapidly moving adult respiratory distress syndrome (ARDS), which has been labelled postpneumonectomy pulmonary edema or noncardiogenic pulmonary edema. The operation may go smoothly, and the patient who was extubated early will appear to be in fine

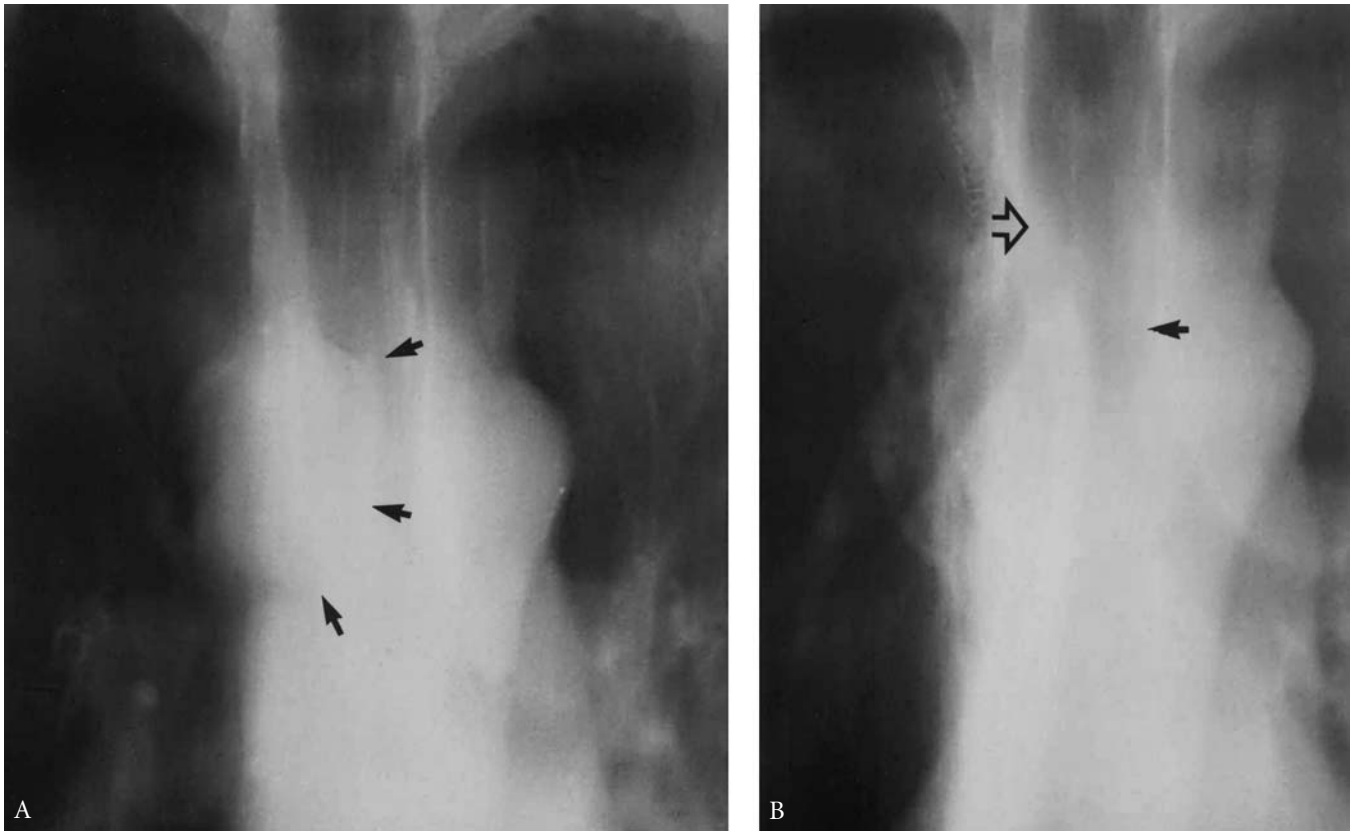

FIGURE **8-16** A, *Adenocarcinoma* (arrows) *of the right upper lobe invading the carina in a 40-year-old patient with insufficient pulmonary reserve to tolerate pneumonectomy.* B, *Reconstruction with end-to-side anastomosis of the bronchus intermedius to lower trachea* (open arrow) *just above the end-to-end joining of distal trachea and left main bronchus* (arrow). *Eight years later, an adrenal metastasis appeared. More often the bronchus intermedius is implanted into the medial side of the left main bronchus.*

condition for 24 hours. At 36 to 48 hours, a diffuse infiltrate appears in the remaining lung. This progresses almost relentlessly to opacification of the residual left lung, and ultimately to death (see Figure 21-1 in Chapter 21, "Complications of Tracheal Reconstruction"). At postmortem examination, the lung is wet and heavy but only nonspecific bacteria, if any, are cultured. This does not support a postmortem diagnosis of bronchopneumonia. The syndrome also follows conventional right pneumonectomy less often, and left pneumonectomy or lobectomy even less frequently. Initially, it was attributed to perioperative intravenous fluid overload.[46] We and others have not found any correlation between the amount of perioperative fluid administered and the occurrence of this dreaded complication.[47] Nonetheless, it seems prudent to manage pneumonectomy patients with minimum fluid administration. Interference with pulmonary lymphatics may impair the ability of the remaining lung to clear interstitial fluid. Barotrauma is likely implicated. The low incidence of ARDS in the series of Dartevelle and Macchiarini suggested a difference in anesthesiologic techniques.[43] The declining incidence of this complication is likely due to adjustment of intraoperative airway ventilatory pressure and tidal volumes to avoid pulmonary barotrauma.

Until recently, the syndrome was nearly uniformly fatal. Addition of inhaled nitric oxide to the therapeutic regimen of fluid restriction, diuresis, ventilatory support, and steroids give promise of better results.[47] Ten consecutive patients with severe ARDS (ARDS score 3.1), treated with inhaled nitric oxide at 10 to 20 ppm, showed immediate improvement in the mean ratio of partial pressure of arterial oxygen to fraction of inspired oxygen from 95 to 128 mm Hg (32% improvement), with further improvement there-

after. Chest x-rays improved in 8 patients, and 7 patients survived. The 3 who died were late deaths related to sepsis after recovery from initial ARDS.

Early in my experience, I tried to save the right lower or middle and lower lobes by bronchial implantation, after intrapericardial hilar mobilization, into either the side of the trachea above the end-to-end trachea to left main bronchus anastomosis, or into the medial side of the left main bronchus. Although the right *main* bronchus may be quite easily anastomosed in this way, excessive tension results if the right lower lobe bronchus or bronchus intermedius is pulled up to the trachea. Stenosis or separation resulted in a number of instances, with some fatal anastomotic complications.[42] Clearly, this should not be done. If it is essential to save parenchyma because of borderline function, anastomosis of the bronchus intermedius or right lower lobe bronchus should be made to the medial side of the left main bronchus. Ishihara and colleagues also reattached the bronchus intermedius and left main bronchus to the trachea side-by-side, in 2 patients.[40] Where functional status permits, pneumonectomy is probably preferable for safety.

Unilateral node dissection does not seem to affect anastomotic healing. Excessive bronchial stripping is best avoided. As in all airway surgery in the thorax, a second tissue layer is advised over the anastomosis. If irradiation has been given remotely, omental coverage will help to provide healing elements to the inert bronchial tissues (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation"). Since these tumors are so central, resection will often be made intrapericardially, and portions of esophageal wall or superior vena cava may also have to be excised. Extensive surgery, including our earlier adverse techniques described, produced an operative morbidity of 47% compared with 27% for carinal reconstruction without pneumonectomy.[42]

Results

At the outset, mortality rates from carinal pneumonectomy for bronchogenic carcinoma exceeded long-term survival rates (see Table 8-1). Jensik and colleagues reported a 5-year survival rate of 15% in 1982,[35] wheras that from Deslauriers and colleagues was 13% in 1989,[34] that by Dartevelle and colleagues was 23% in 1988,[37] and that by Mathisen and Grillo was 19% in 1991.[36] The figure of Dartevelle and Macchiarini rose to 43% in 1996.[43] The overall survival rate from these authors and others was 22±12%. Dartevelle and Macchiarini found in 60 patients that long-term survival was significantly influenced by nodal status, with 5-year rates of 41, 54, and 0%, respectively, for N0, N1, and N2 lesions.[43] Squamous cancer was more favorable than nonsquamous cancer. A 2001 review of the MGH series of resections of the carina by various surgical modes for bronchogenic carcinoma produced an overall 5-year survival rate of 42% (including operative mortality), with 19 absolute 5-year survivors.[45] Survival was highest after isolated carinal resection (51%). Nodal involvement was a strong influence in this series as well. Five-year survival rates were 51% for N0, 32% for N1, and 12% for N2 or N3 lesions.

Comment

With the development of carinal surgery, carinal pneumonectomy for bronchogenic carcinoma has become feasible, with growing safety. Involvement of the carina should not, therefore, in itself exclude consideration of surgery. Carinal resection is also appropriate to obtain an adequate margin for very proximal main bronchial carcinoma. Patients must be carefully appraised for the extent of local and distant disease and for the anatomic and functional feasibilities of safe resection. With careful attention to the selection of patients, surgical technique, and perioperative management, complications from the surgery should continue to decrease. Postpneumonectomy pulmonary edema or ARDS remains an ominous, if decreasing, threat. Its cause is not yet fully understood. Five-year survival rates of 30% or greater may be anticipated, if patients with N2 disease are excluded. Inclusion of N2 patients with neoadjuvant treatment will require careful prospective study.

Other Tumors

Recurrence of *squamous cell carcinoma of the larynx* at the tracheal stoma after laryngectomy has been approached by radical resection.[48] This usually follows either postlaryngectomy irradiation, which failed to prevent recurrence, or failed irradiation treatment of recurrence. Resection frequently requires radical en bloc resection, which includes adjacent cervical tissues and, sometimes, the esophagus. Cervicomediastinal exenteration is described in Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy." Due to local recurrent tumor and prior irradiation, a wide excision may be necessary, with rotation of unirradiated myocutaneous flaps to cover the lower neck and upper mediastinum and with establishment of a low mediastinal tracheostomy. All too often, however, cancer recurs soon after resection. Paratracheal lymphatics are often permeated with cancer distally. Justification for such extensive surgery may therefore be questioned. For this reason, I have only performed a few cervical exenterations for recurrent laryngeal carcinoma.

Cervical exenteration has also been performed for *postcricoid squamous cell carcinoma of the esophagus*, which involves the larynx and upper trachea, rendering laryngeal salvage impossible. Restoration of esophageal continuity may be accomplished by a variety of techniques.[31] This, however, is beyond the scope of a book on airway reconstruction. Low cervical tracheostomy rather than mediastinal tracheostomy is usually possible in these patients.

If abnormality is suggested, bronchoscopy and biopsy should be performed on all patients with upper and midesophageal carcinoma. Generally, segmental resection of the trachea, for direct invasion by *esophageal carcinomas* other than postcricoidal esophageal carcinoma, is not advised. The extent of involvement is usually so great that curative resection is unlikely. Tracheoesophageal fistula due to esophageal carcinoma is an extreme example. However, in a rare patient, following preoperative neoadjuvant treatment, if the only point of nonresectability is a short segment of trachea, it may be worthwhile to resect this segment and perform a direct tracheotracheal anastomosis. There is, however, a particular danger of tracheal necrosis, since the blood supply of the trachea is markedly diminished by adjacent esophagectomy. (See Chapter 1, "Anatomy of the Trachea," describing tracheal blood supply.) Segmental arteries supplying anterior branches to the trachea and posterior to the esophagus are removed by esophagectomy. An undivided trachea retains viability via collateral vessels. However, when divided for anastomosis, the intramural blood supply may be insufficient for viability, and necrosis may occur adjacent to this anastomosis. To avoid complications, Matsubara and colleagues resected a limited portion of involved posterior tracheal wall and repaired the defect with a muscle flap.[49] If combined resection is performed, advancement of the omentum is advisable. It is brought up with the stomach if this is used to reconstruct the esophagus, and separately if colon is used. Extension of a radical esophageal resection to include a tracheal segmental resection in any programmatic way is probably unwise. The palliation of malignant tracheoesophageal fistula, considered in Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula," is not accomplished by tracheal resection.

A unique case of *esophageal leiomyomatosis* involving the trachea has been recorded, necessitating esophagectomy and tracheal resection.[50]

Metastases from remote sites occur to the bronchi, but rarely to the trachea. King and Castleman found that over 185 of patients with pulmonary metastatic tumor also showed bronchial invasion by extension or metastatic deposit.[51] Baumgartner and Mark described 2 endotracheal metastases from breast cancer, and cited 6 prior reports with primary sites in the breast, colon, kidney, and uterus.[52] Heitmiller and colleagues found 1 tracheal metastasis in 23 patients with tracheobronchial metastases.[53] Primary sites, in descending order of frequency, were the breast, kidney, and colon, with others from the bladder, thyroid, ovary, and nasopharynx. In most patients (87%), extrabronchial disease was also present. Although tracheobronchial metastases occurred late after primary tumor (mean 5 years), survival was short (mean 1 year).

References

1. Lawson W, Som MP, Biller HF. Papillary carcinoma of the thyroid invading the upper air passages. Ann Otol Rhinol Laryngol 1977;86:751–5.
2. Zannini P, Melloni G. Surgical management of thyroid cancer invading the trachea. Chest Surg Clin North Am 1996;6:777–90.
3. Silliphant WM, Klinck GH, Levitin MS. Thyroid carcinoma and death: a clinicopathological study of 193 autopsies. Cancer 1964;17:513–25.
4. Silverberg SG, Hutter RVP, Foote FW Jr. Fatal carcinoma of the thyroid: histology, metastases and causes of death. Cancer 1970;25:792–802.
5. Ishihara T, Yamazaki S, Kobayashi K, et al. Resection of the trachea infiltrated by thyroid carcinoma. Ann Surg 1982;195:496–500.
6. Cady B, Rossi R. An expanded view of risk-group definition in differentiated thyroid carcinoma. Surgery 1988;104:947–53.
7. Grant CS, Hay ID, Gough IR, et al. Local recurrence in papillary thyroid carcinoma: is extent of surgical resection important? Surgery 1988;104:954–62.
8. Tsumori T, Nakao K, Miyata M, et al. Clinicopathologic study of thyroid carcinoma infiltrating the trachea. Cancer 1985;56:2843–8.
9. Nomori H, Kobayashi K, Ishihara T, et al. Thyroid carcinoma infiltrating the trachea: clinical, histologic, and morphometric analyses. J Surg Oncol 1990;44:78–83.
10. Shin DH, Mark EJ, Suen HC, Grillo HC. Pathological staging of papillary carcinoma of the thyroid with airway invasion based upon the anatomic manner of extension to the trachea. Hum Pathol 1993;24:866–70.
11. Tsumori T, Nakao K, Miyata M, et al. Clinicopathologic study on the mode and degree of invasion of the trachea by thyroid carcinoma. Nippon Geka Gakkai Zasshi 1987;88:600–6.
12. Czaja JM, McCaffrey TV. The surgical management of laryngotracheal invasion of well-differentiated papillary thyroid carcinoma. Arch Otol Head Neck Surg 1997; 123:484–90.
13. McCaffrey TV, Bergstralh EJ, Hay ID. Focally invasive papillary thyroid carcinoma: 1940–90. Head Neck 1994;16:165–72.
14. Friedman M, Danielzadeh JA, Caldarelli DD. Treatment of patients with carcinoma of the thyroid invading the airway. Arch Otol Head Neck Surg 1994;120:1377–81.
15. Nishida T, Nakao K, Hamaji M. Differentiated thyroid carcinoma with airway extension: indication for tracheal resection based on the extent of cancer invasion. J Thorac Cardiovasc Surg 1997;114:84–92.
16. McCarty TM, Kuhn JA, Williams WL, et al. Surgical management of thyroid cancer invading the airway. Ann Surg Oncol 1997;4:403–8.
17. Grillo HC, Suen HC, Mathisen DJ, Wain JC. Resectional management of thyroid carcinoma invading the airway. Ann Thorac Surg 1992;54:3–10.
18. Ishihara T, Kobayashi K, Kikuchi K, et al. Surgical treatment of advanced thyroid carcinoma invading the trachea. J Thorac Cardiovasc Surg 1991;102:717–20.
19. Protopapas AD, Nicholson AG, Vini L, et al. Thoracic metastasectomy in thyroid malignancies. Ann Thorac Surg 2001;72:1906–8.
20. Frazell EL, Foote FW Jr. Papillary cancer of the thyroid: a review of 25 years of experience. Cancer 1958;11: 895–922.
21. Hendrick JW. An extended operation for thyroid carcinoma. Surg Gynecol Obstet 1963;116:183–8.
22. Grillo HC. Circumferential resection and reconstruction of the mediastinal and cervical trachea. Ann Surg 1965;162:374–88.
23. Grillo HC, Zannini P. Resectional management of airway invasion by thyroid carcinoma. Ann Thorac Surg 1986;42:287–98.
24. Maeda M, Nakamoto K, Ohtea M, et al. Statistical survey of tracheobronchoplasty in Japan. J Thorac Cardiovasc Surg 1989;97:402–14.
25. Mellière DJM, Ben Yahia NE, Becquemin JP, et al. Thyroid carcinoma with tracheal or esophageal involvement: limited or maximal surgery. Surgery 1993;113: 166–72.
26. Ozaki O, Sugino K, Miura T, Ito K. Surgery for patients with thyroid carcinoma invading the trachea: circumferential sleeve resection followed by end-to-end anastomosis. Surgery 1995;117:268–71.
27. Bayles SW, Kingdom TT, Carlson GW. Management of thyroid carcinoma invading the aerodigestive tract. Laryngoscope 1998;108:1402–7.
28. Musholt TJ, Musholt PB, Behrend M, et al. Invasive differentiated thyroid carcinoma: tracheal resection and reconstruction procedures in the hands of the endocrine surgeon. Surgery 1999;126:1078–87.
29. Park CS, Suh KW, Min JS. Cartilage-shaving procedure for the control of tracheal cartilage invasion by thyroid carcinoma. Head Neck 1993;15:289–91.
30. Kim KH, Sung MW, Chang KH, Kang BS. Therapeutic dilemmas in the management of thyroid cancer with laryngotracheal involvement. Otol Head Neck Surg 2000;122:763–7.
31. Grillo HC, Mathisen DJ. Cervical exenteration. Ann Thorac Surg 1990;49:401–9.
32. Mathey J, Binet JP, Galey JJ, et al. Tracheal and tracheobronchial resections. Technique and results in 20 cases. J Thorac Cardiovasc Surg 1966;51:1–13.
33. Jensik RJ, Faber P, Milloy FJ, Goldin MD. Tracheal sleeve pneumonectomy for advanced carcinoma of the lung. Surg Gynecol Obstet 1972;134:231–6.
34. Deslauriers J, Beaulieu M, McClish A. Tracheal sleeve pneumonectomy. In: Shields TW, editor. General thoracic surgery. 3rd ed. Philadelphia: Lea and Febinger;1989. p. 382.
35. Jensik RJ, Faber LP, Kittle CF, et al. Survival in patients undergoing tracheal sleeve pneumonecotmy for bronchogenic carcinoma. J Cardiovasc Thorac Surg 1982;84:489–96.
36. Mathisen DJ, Grillo HC. Carinal resection for bronchogenic carcinoma. J Thorac Cardiovasc Surg 1991; 102:16–23.
37. Dartevelle PG, Khalife J, Chapelier A, et al. Tracheal sleeve pneumonectomy for bronchogenic carcinoma. Report of 55 cases. Ann Thorac Surg 1988;46:68–72.
38. Perelman M, Koroleva N. Surgery of the trachea. World J Surg 1980;4:583–91.
39. Roviaro GC, Varoli F, Rebuffat C, et al. Tracheal sleeve pneumonectomy for bronchogenic carcinoma. J Thorac Cardiovasc Surg 1994;107:13–8.
40. Ishihara T, Ikeda T, Inoue H, Fukai S. Resection of cancer of lungs and carina. J Thorac Cardiovasc Surg 1977; 73:936–43.
41. Watanabe Y, Shimizu J, Oda M, et al. Results in 104 patients undergoing bronchoplastic procedures for bronchial lesions. Ann Thorac Surg 1990;50:607–14.
42. Mitchell JD, Mathisen DJ, Wright CD, et al. Clinical experience with carinal resection. J Thorac Cardiovasc Surg 1999;117:39–53.
43. Dartevelle P, Macchiarini P. Carinal pneumonectomy for bronchogenic carcinoma. Sem Thorac Cardiovasc Surg 1996;8:414–25.
44. Grillo HC, Mathisen DJ. Primary tracheal tumors: treatment and results. Ann Thorac Surg 1990;49:69–77.

45. Mitchell JD, Mathisen DJ, Wright CD, et al. Resection for bronchogenic carcinoma involving the carina: long-term results and effect of nodal status on outcome. J Thorac Cardiovasc Surg 2001;121:465–71.
46. Zeldin RA, Normandin ED, Landtwing BD, Peters RM. Postpneumonectomy pulmonary edema. J Thorac Cardiovasc Surg 1984;87:359–65.
47. Mathisen DJ, Kuo EY, Hahn C, et al. Inhaled nitric oxide for adult respiratory distress syndrome after pulmonary resection. Ann Thorac Surg 1998;65:1894–902.
48. Krespi YP, Wurster CF, Sisson GA. Immediate reconstruction after total laryngopharyngoesophagectomy and mediastinal dissection. Laryngoscope 1985;95:156–61.
49. Matsubara T, Ueda M, Nakajima T, et al. Can esophagectomy cure cancer of the thoracic esophagus involving the major airways? Ann Thorac Surg 1995;59:173–7.
50. Kumar P, Breach NM, Goldstraw P. Esophageal leiomyomatosis involving trachea: surgical resection and repair. Ann Thorac Surg 1997;63:531–3.
51. King DS, Castleman B. Bronchial involvement in metastatic pulmonary malignancy. J Thorac Cardiovasc Surg 1943; 12:305–15.
52. Baumgartner WA, Mark JB. Metastatic malignancies from distant sites to the tracheobronchial tree. J Thorac Cardiovasc Surg 1980;79:499–503.
53. Heitmiller RF, Marasco WJ, Hruhan RH, Marsh BR. Endobronchial metastasis. J Thorac Cardiovasc Surg 1993;106:537–42.

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Tracheal and Bronchial Trauma

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Mechanical Injuries

Injuries to the trachea and bronchi, whether blunt or penetrating, are uncommon. Figures for frequency depend upon the numerator (type and location of injury, venue) and the denominator (autopsy series, multiple trauma cases, survivors).[1]

Types of Injuries

Airway trauma may be life threatening, immediately or in the hours following acute injury. Penetrating or blunt trauma to the neck may injure the larynx or cervical trachea, whereas injuries to the thorax may damage the thoracic trachea and bronchi. In civilian practice, *blunt trauma* to the airway from motor vehicle accidents used to be the most common cause of such injuries. Unfortunately, penetrating wounds from stabbing and gunshot have increased rapidly in urban areas, with firearms in the lead (Table 9-1).[1] Cervical injuries also result from striking wires or cables while driving motorcycles or snowmobiles, or from strangulation by seat belt.[2,3] In motor vehicle injuries, the larynx and cervical trachea are damaged by direct impact of the steering wheel or dashboard against the cervical vertebra, often with the neck extended. Direct blows to the neck also produce laryngotracheal injuries. Blunt trauma to the anterior thorax during motor vehicle accidents, or less often from industrial crush injuries, produces a variety of injuries to the thoracic trachea, carina, and main bronchi.

Penetrating injuries to the trachea are most often due to bullet or stab wounds and may be accompanied by major vascular trauma. Because of the lethal nature of missile or stab wounds to the surrounding major structures, penetrating wounds of the thoracic trachea are not often seen in the living. The esophagus is at risk in all of these injuries, cervical and thoracic.

Tracheobronchial lacerations, which result from endotracheal intubation or other per oral instrumentation, are included here. *Postintubation injuries*, which are late results of treatment of respiratory failure, are described in Chapter 11, "Postintubation Stenosis."

Table 9-1 Type of Injury and Location

	Blunt			*Penetrating*			*Total*
Cervical airway	28%	65	29%	77%	161	71%	226 (100%)
Thoracic trachea	27%	62	63%	17%	36	37%	98 (100%)
Bronchi	45%	105	90%	6%	12	10%	117 (100%)
	100%	232		100%	209		441

Reproduced with permission from Bacha EA et al.[1]

Cervical Injury. Damage to the *larynx* and *cervical trachea* from *blunt injury* may include fracture with or without displacement of the hyoid, the supraglottic larynx, the infraglottic larynx, the cricoid cartilage, and the cervical trachea (Figure 9-1). A variable amount of trachea may be elevated into the neck by cervical hyperextension at the time of injury, depending on the age of the patient. In the young, the larynx may be severely contused or otherwise injured without actual fracture of the more flexible cartilages. In older patients, stiffening and calcification render the larynx more vulnerable to fracture. Mucosal tears and avulsions are seen. Vocal cords and arytenoids may be torn and displaced. It is important to consider the spectrum of possible injuries when examining an acutely traumatized patient.[4] Separation of the airway may be partial or complete. The points of actual rupture of the cervical airway are most commonly between the cricoid and trachea and in the upper trachea. Blunt trauma rarely produces clean cuts, but rather produces complexes of injuries, including, for example, cricotracheal separation with concurrent fracture of the cricoid cartilage, and avulsion of mucosa from the anterior surface of the posterior cricoid plate (Figure 9-2).

In 19 patients with laryngotracheal disruption, 11 due to direct impact and 8 due to strangulation, Couraud and colleagues identified 14 complete separations below the cricoid or first ring.[3] Nine cricoid fractures were seen. The mucosa retracted in all patients to expose cricoid cartilage. One or both *recurrent laryngeal nerves* may be temporarily or permanently damaged. Fourteen of Couraud's patients suffered bilateral recurrent nerve damage and 4 were unilateral. Concomitant tears of the *esophagus* may occur. Avulsion of the trachea from the cricoid may be accompanied by transverse laceration of the anterior esophagus from the pharynx, where it is attached to the cricoid posteriorly, or by completely circumferential separation. Subluxation of *cervical vertebrae*, with or without injury to the spinal cord, may occur concomitantly. With penetrating wounds of the neck and thoracic inlet, and even with blunt trauma, the spectrum of potential injuries includes major *vascular injuries*.

Penetrating cervical wounds, chiefly due to stab or gunshot wounds, may injure the trachea as one of several structures damaged. Bilateral recurrent laryngeal nerve division is less common than in complete tracheal separation due to blunt injury. Although dissenting voices are raised, surgical exploration of penetrating cervical wounds still seems to be the judicious course.

Thoracic Trachea and Bronchi. Fracture or laceration of the thoracic trachea, carina, or main bronchi following closed chest injury may be seen in children and young adults without rib or sternal fractures. The young thorax can absorb major compressive trauma and rebound without skeletal fractures. In the older patient, clavicular and upper rib fractures and the number of rib fractures correlate with the likelihood of tracheobronchial injury.[5] It is likely that a sudden increase of intrabronchial pressure against the closed glottis, or rapid deceleration, produces some airway injuries. Blunt injuries to the thoracic trachea are most common in the lower trachea and vary widely from complete transection to a partial horizontal tear. Vertical splitting of the trachea from the carina upward occurs, running anteriorly through cartilages and/or posteriorly up the membranous wall, either in its center or laterally along the junction with the cartilages. Injury to the lower trachea may be accompanied by partial or complete shearing of one or both main bronchi.

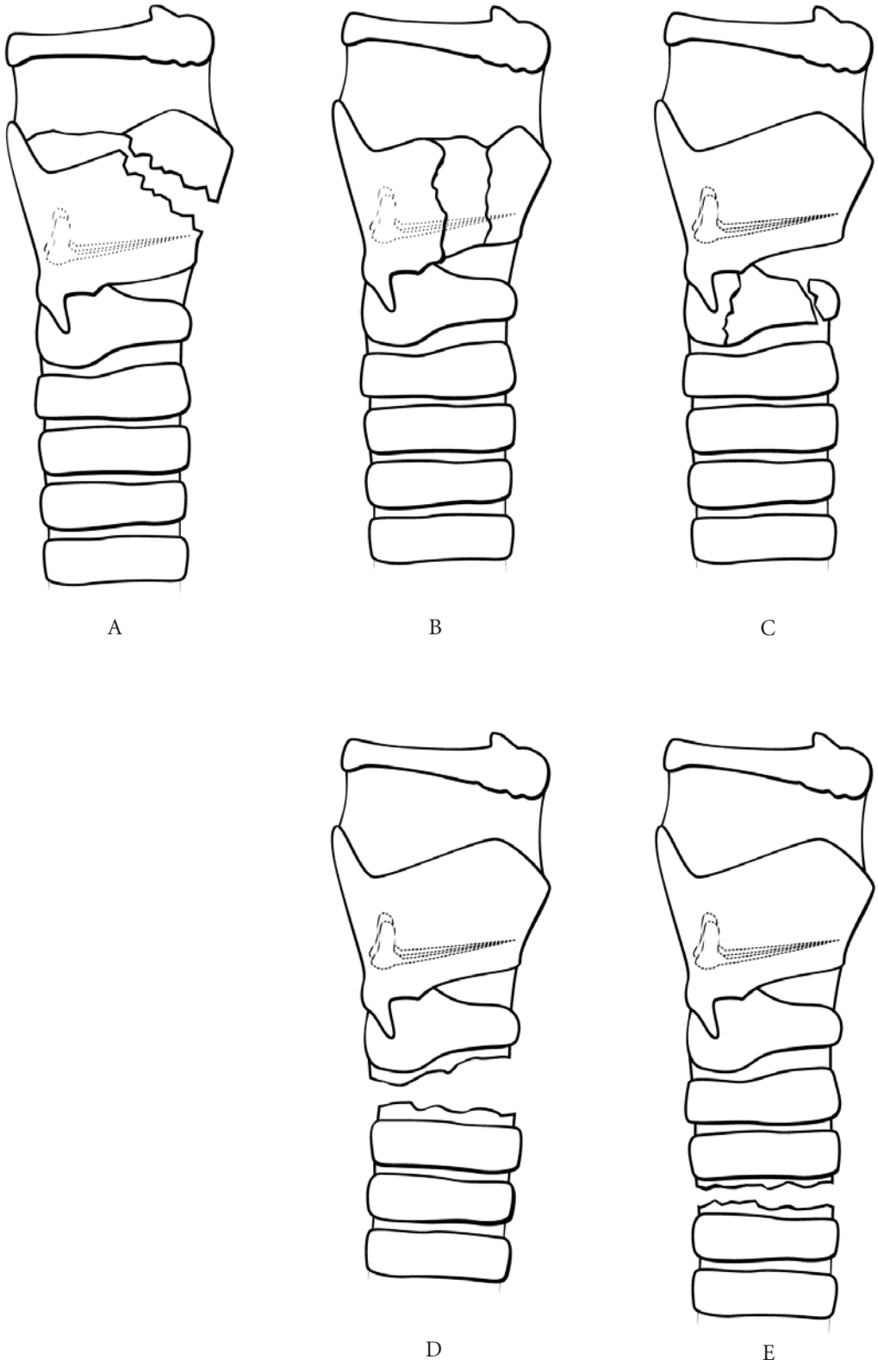


FIGURE **9-1** *Cervical laryngotracheal blunt trauma.* A, *Supraglottic tears and fractures.* B, *Transglottic injuries.* C, *Cricoid fracture.* D, *Avulsion of trachea from cricoid.* E, *Laceration or tear of trachea. Adapted from Harris HH. Management of injuries to the larynx and trachea. Laryngoscope 1972;82:1924–9.*

FIGURE 9-2 *Blunt injury to the neck in motor vehicle accident. The distal end of the trachea is completely separated from the larynx. The forceps holds a posterior and lateral full thickness mucosal flap* (arrow) *which was avulsed from the anterior surface of the posterior cricoid below the arytenoids. The endotracheal tube (ET) has been introduced through a tracheostomy below the level of transection. Since both recurrent laryngeal nerves were severed, it was evident that a tracheostomy would be necessary postoperatively and hence the ET was placed through this location. The mucosal flap was resutured into the posterolateral laryngeal defect. A laryngeal mold was also used. The esophagus, which had been separated from the pharynx, was reanastomosed. Strap muscle was interposed between laryngotracheal and pharyngoesophageal suture lines.*

Lobar or segmental bronchi may also be lacerated or separated by crush injuries, usually accompanied by deep parenchymal laceration. From a review of 183 tracheobronchial blunt injuries reported between 1970 and 1990, Symbas and colleagues noted that 74% were transverse ruptures with 4% in cervical and 12% in thoracic trachea, 25% in the right main bronchus, 17% in the left main bronchus, and 16% in lobar bronchi (Figure 9-3).[6] Of longitudinal tears (18%), 6.5% were in the cervical trachea, 10% in the thoracic trachea, and 1.5% in main bronchi. The 8% remaining were complex, involving the trachea and right or both main bronchi. Most injuries occur within 2.5 cm of the carina. Kiser and colleagues, in a review of 265 patients who suffered blunt tracheobronchial injuries, confirmed a greater frequency of right-sided injuries, both overall and at the time of diagnosis and treatment.[7] They found that bronchial rupture occurred within 2 cm proximal to the carina in 76%, and that 43% occurred in the right main bronchus.

Recurrent laryngeal nerve injury is rare in thoracic tracheal trauma. Also rare but equally as important is concurrent *laceration of the esophagus*, often longitudinally. Esophageal injury should be considered in every posterior laceration of the trachea, since esophageal injury from blunt trauma is unlikely to occur by itself. It probably results from sudden forceful compression of the trachea and esophagus against the vertebrae, such as that from steering wheel impact. The injury occurs more often in young patients, with or without upper rib fractures. An elastic chest wall seems to favor such injury. Injury is most common in the lower trachea but may occur in the neck. The communication may be instantly established or occur later as traumatized tissues necrose. Potentially lethal mediastinitis may be a consequence of an overlooked intrathoracic esophageal laceration.

Failure to recognize acute injury to the airway because of distraction due to catastrophic associated injuries, or failure to manage acute airway injury appropriately, may lead to cicatricial obstruction and other sequelae, days or months later. Such problems are often correctable but with greater difficulty and complications.

Tracheobronchial Lacerations after Intubation. Lacerations of the trachea and bronchi may be produced by single and double lumen endotracheal tubes. The injuries are in the membranous wall and are usually

linear. Postoperative mediastinal or subcutaneous emphysema may indicate an unrecognized intraoperative endotracheal intubation laceration. In a careful analysis of these lacerations, Massard and colleagues found predominant injuries in the lower trachea and main bronchi, and in the case of single lumen tubes, along the right membranous cartilaginous junction.[8] Cervical laceration occurs less often. Overinflation of cuffs rather than stylets or tube tips appears to cause the injuries. Repositioning a tube that was originally placed in the right main bronchus without deflating the cuff may be an important factor. Short women, with correspondingly narrower airways, appear to be more at risk. Lacerations also occur from the placement of tracheostomy tubes when insertion is difficult. Mediastinal or subcutaneous emphysema and pneumothorax are harbingers.

The modalities of surgical repair and conservative treatment are discussed in Chapter 31, "Repair of Tracheobronchial Trauma." In general, small lacerations may be safely managed conservatively, but larger ones are best repaired surgically.[8,9]

Injury to the membranous wall of the trachea above the carina or of the left main bronchus has been noted in about 1 to 2% of patients undergoing transhiatal esophagectomy.[10] These have been treated by suture, reinforcement with pleura or pericardium, and effective buttressing with the gastric tube neoesophagus.

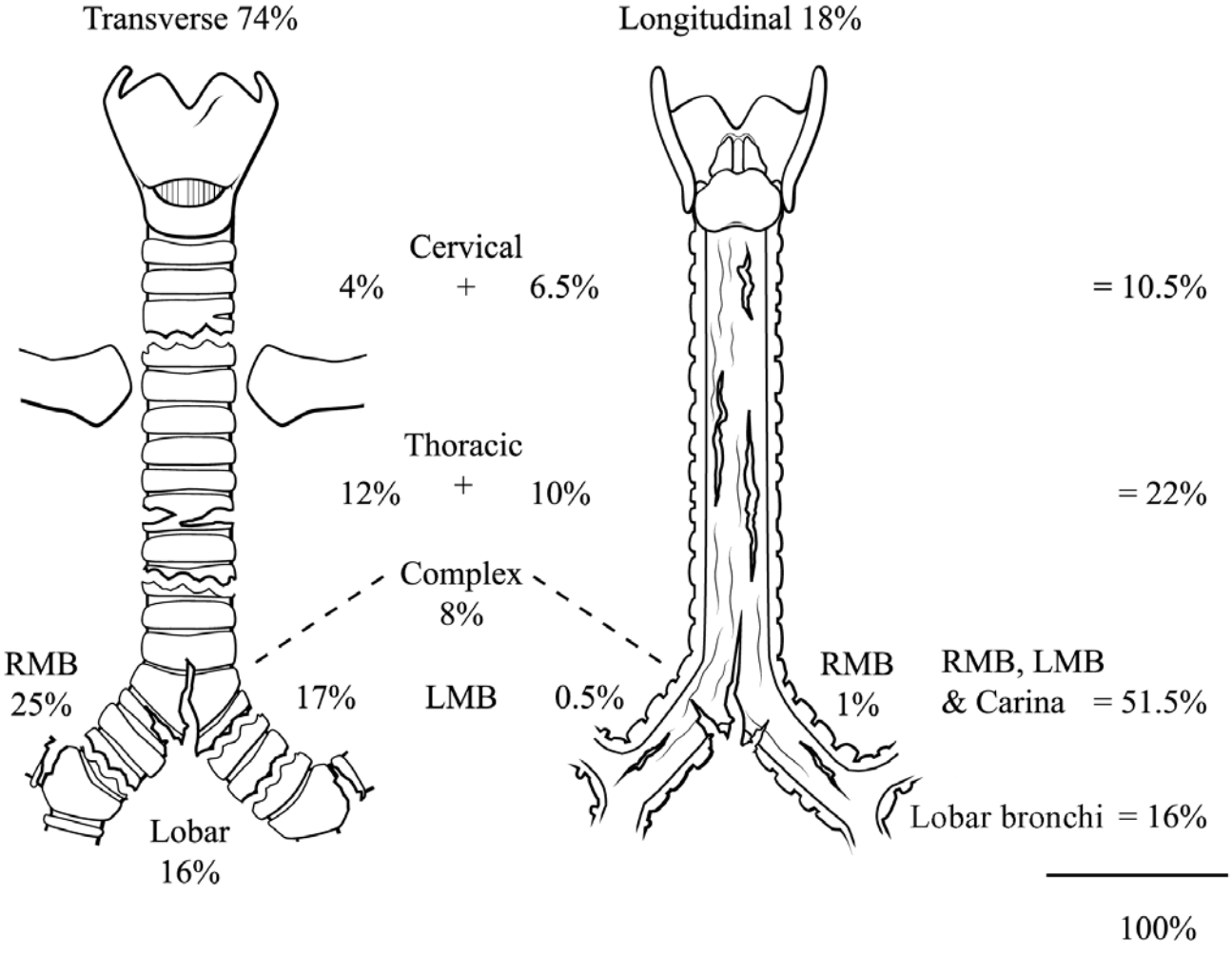


FIGURE 9-3 *Blunt tracheobronchial injuries: type and location. Adapted from Symbas PN et al.[6] LMB = left main bronchus; RMB = right main bronchus.*

Clinical Characteristics

Acute Injury. *Cervical tracheal injury* presents most commonly with palpable subcutaneous emphysema. Contusion, abrasion, or laceration of skin may or may not be present. Hemoptysis is a common finding and hoarseness, inspiratory stridor, and dysphonia or aphonia may occur. The patient may be in acute respiratory distress with asphyxia or show little initial difficulty.[11,12] Slight respiratory distress which is present initially may suddenly precipitate into severe obstruction. Subcutaneous emphysema may increase with coughing or swallowing. Deep cervicomediastinal emphysema is characteristic and pneumothorax may be present.[2,3]

Laceration of the *thoracic trachea*, the *main bronchi*, or both may produce dyspnea, subcutaneous emphysema, massive cervical and mediastinal emphysema, or uni- or bilateral pneumothorax. Pneumo mediastinum or cervical emphysema is a warning of probable tracheal rupture. Bilateral pneumothorax often indicates tracheal or carinal rupture. Unilateral or tension pneumothorax may occur, particularly with bronchial rupture. Hemopneumothorax may be present. Mild hemoptysis occurs. A mediastinal "crunch" may be heard, synchronous with heartbeat (Hamman's sign). Response to chest tube suction varies. There may be partial resolution of pneumothorax or none, continued brisk air leak, and even increased dyspnea on suction. These responses should alert the surgeon to a probable airway rupture. On the other hand, the main bronchus may be torn without pneumothorax, especially if there is preexisting pleural obliteration. These tears are more often in the lower trachea or carina. If the esophagus is also torn, it may not immediately be manifest clinically.[13] The earliest sign of esophageal injury may be cough on swallowing, becoming noticeable in the days following injury, or the onset of mediastinitis. Concurrent injuries to organs in the traumatized region must therefore be sought and treated at the outset. These may include injury to the aorta, brachiocephalic and/or carotid arteries. Thirteen of 15 patients with early and late blunt tracheobronchial trauma at all levels had the following associated injuries: rib fractures (10), esophageal injuries (7), pulmonary contusion (5), vascular injury (4), head injury (4), and extremity fractures (4).[14]

Chronic Injury. In 3 of 17 patients, whom we saw for late management of *upper tracheal trauma*, diagnosis had not been made at the time of injury.[2] In 10 of these patients, tracheostomy had been performed to establish a secure airway but no attempt at primary repair had been made. In these patients, complete airway stenosis had usually evolved above the tracheostomy at the level of the injury, and in addition, vocal cord paralysis was present. In 4 cases, primary repair had been attempted but failed, resulting in airway stenosis. Among the 3 patients whose diagnosis was overlooked, progressive airway obstruction developed between 1 to 4 weeks after injury. One patient also developed a tracheoesophageal fistula, with such acute surrounding inflammation that proximal esophageal defunctioning, a procedure we rarely use, was required before correction could be attempted.

The absence of pneumothorax following injury or its apparently successful management by intrapleural suction may lead to failure to diagnose *intrathoracic tracheal* or *bronchial injury*. The area of separation heals by cicatrization, causing tracheal obstruction or bronchial stenosis with atelectasis or obstructive lung infection.

Diagnosis

In many patients with *acute injury* to the trachea, and to the cervical trachea in particular, clinical signs will strongly suggest the diagnosis. However, tracheobronchial injuries should be considered in *every* patient with severe trauma to the neck or chest in order to avoid overlooking any injuries. Bronchoscopy should be performed whenever the possibility of injury is suspected. With *cervical injuries*, anteroposterior and lateral radiographs of soft tissues of the neck as well as chest radiographs may reveal dissection of air in the cervical tissues up to the base of the skull or into the mediastinum (Figure 9-4). Mediastinal emphysema,

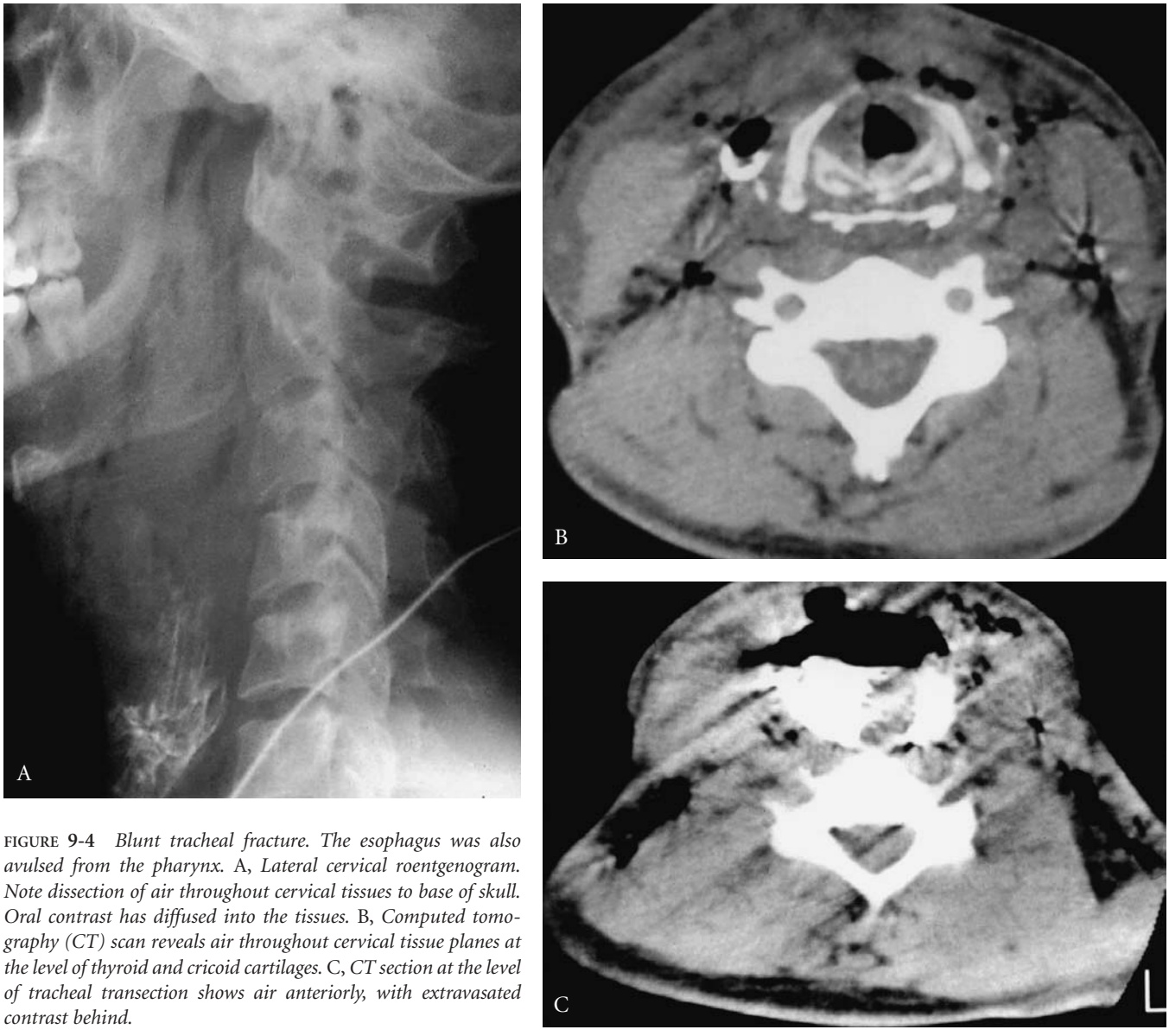


FIGURE **9-4** *Blunt tracheal fracture. The esophagus was also avulsed from the pharynx.* A, *Lateral cervical roentgenogram. Note dissection of air throughout cervical tissues to base of skull. Oral contrast has diffused into the tissues.* B, *Computed tomography (CT) scan reveals air throughout cervical tissue planes at the level of thyroid and cricoid cartilages.* C, *CT section at the level of tracheal transection shows air anteriorly, with extravasated contrast behind.*

with cervical dissection of air and subcutaneous emphysema, is usually present in *intrathoracic tracheal rupture*. Pneumothorax, sometimes bilateral, will often be present unless there is pleural obliteration. Complete *bronchial rupture* may reveal the lung collapsed at the bottom of the thorax, when only the pulmonary vessels retain continuity. Partial but significant collapse, unresponsive or poorly responsive to suction, may present when bronchial mediastinal pleural investments are partially intact. At other times, the lung will fully expand on suction, sometimes without subsequent air leak, which may be clinically deceptive. Pleural obliteration may limit even mediastinal emphysema (Figure 9-5).

Laryngoscopy is necessary to assess possible damage in the uppermost airway. This is facilitated by the flexible nasopharyngoscope. In acute injury, accurate laryngeal assessment may be very difficult because of edema, hemorrhage, and tissue trauma. If the first manifestation of the injury is airway obstruction, initial treatment also serves as a diagnostic procedure. Bronchoscopy should be done early for direct visual-

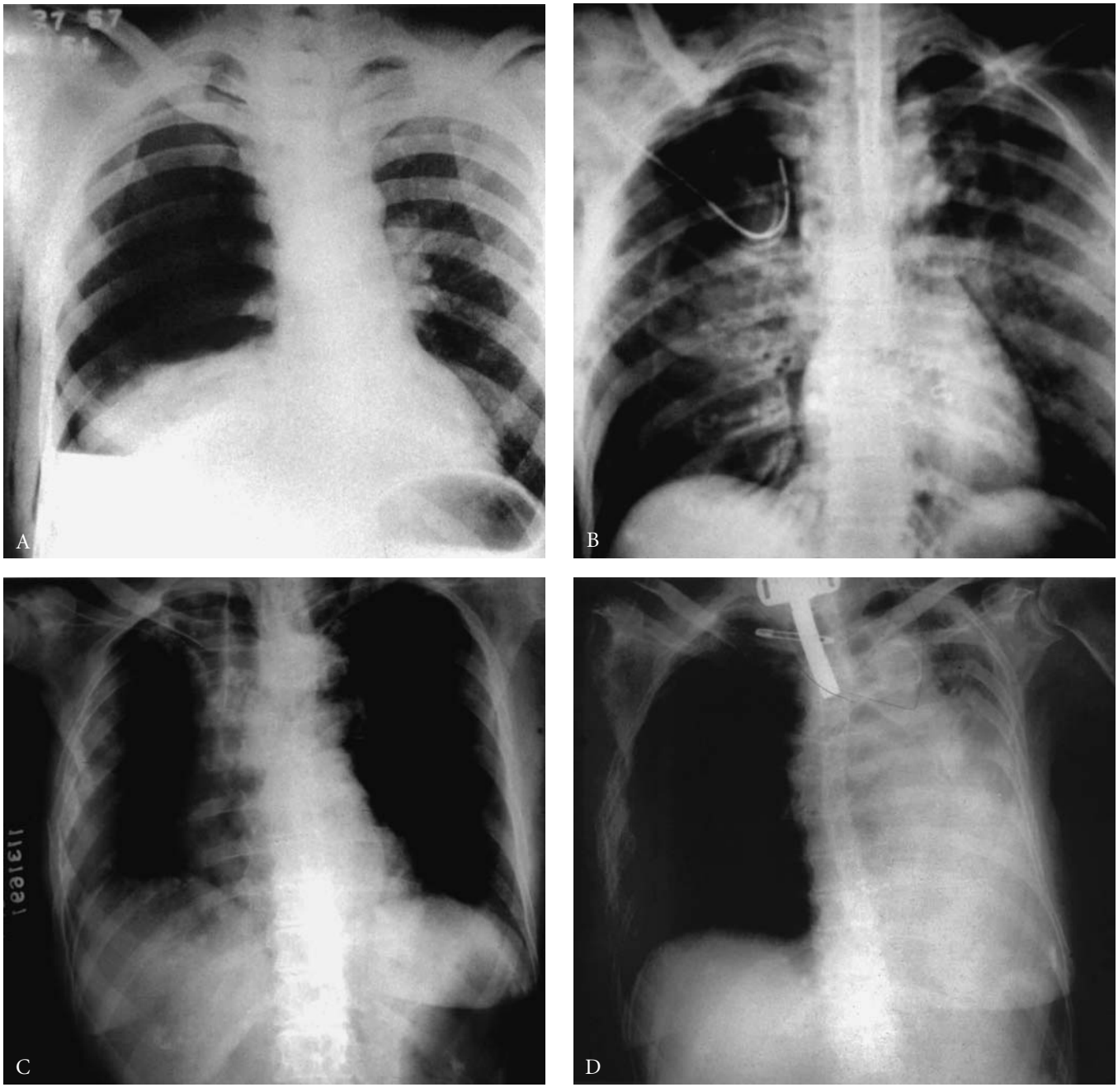


FIGURE **9-5** *Variations in radiographic picture obtained following main bronchial rupture.* A, *Complete right main bronchial rupture with lung collapsed at the base of hemithorax, since it remains connected to the hilum only by pulmonary vessels. Tension is indicated by mediastinal displacement. Hemothorax is present.* B, *Partial expansion of lung on pleural suction after right main bronchial fracture, because peribronchial tissues maintain a partially "intact" air channel to the lung. This presentation is most commonly seen. Note the mediastinal emphysema and air in the muscle planes.* C, *Chest roentgenogram of a 69-year-old woman, run over by a truck. Multiple bilateral rib fractures and unstable chest wall. Mediastinal shift to right. Pleural obliteration prevented pneumothorax.* D, *Same patient after partial atelectatic collapse of left lung. Air delineates the transected left main bronchus.*

ization of the airway. Flexible bronchoscopy with an endotracheal tube threaded over it may be the method of choice (see Chapter 31, "Repair of Tracheobronchial Trauma", and Figure 10-1 in Chapter 10, "Tracheostomy: Uses, Varieties, Complications"). Pneumothorax may be severe or accompanied by tension and must be treated immediately.

If there is suspicion or any possibility of *cervical spine injury*, urgent airway assessment and establishment must be done with this concern in mind. Adequate splinting must be provided during radiographic examination. Intubation over a flexible bronchoscope avoids cervical flexion or extension. If that fails, urgent tracheostomy becomes necessary. This is one of the few remaining indications for emergency tracheostomy. Equipment for tracheostomy should be at hand when bronchoscopy is first attempted, in case of loss of airway during endoscopy. In a series of acute tracheal injuries, only 4 patients needed no intubation, whereas 11 required oral endotracheal intubation—2 with the aid of a flexible bronchoscope, 2 intubated through the open neck, and 2 with a rigid bronchoscope.[14] Eight airways were controlled by tracheostomy. Computed tomography (CT) adds detail but is usually not critical to diagnosis, and it certainly should not be routinely obtained if the patient is unstable or might lose the airway (Figure 9-6).

In an urgent situation, sufficient information is usually at hand after endoscopy and on the x-rays to proceed to surgical treatment, unless there is strong indication for additional imaging such as angiography.

If *esophageal injury* is suspected, contrast esophagography is performed after the airway is secure, and other life-threatening injuries, such as aortic rupture, have been evaluated. A small amount of barium or Gastrografin is used. Contrast studies may fail to show esophageal injury. Therefore, rigid esophagoscopy is performed if cervical spine injury is absent. The proximity of a penetrating injury is sufficient to raise the question of esophageal injury.[13] If the injury on the initial chest x-ray suggests the possibility of *vascular injury*, then a CT scan with contrast or, more definitively, angiography is advised to delineate the lesion precisely (Figure 9-7).

In injuries where diagnosis has been *delayed* or only an emergency tracheostomy was established, the larynx is first completely and carefully assessed. This is best done by an experienced otolaryngologist on the awake patient, so that glottic function is fully observed. Complete radiographic studies of the larynx and trachea are performed (see Chapter 4, "Imaging the Larynx and Trachea"). If indicated, esophagography is included (Figure 9-8). Endoscopic examination is next, made of all portions of the airway and of the esophagus if necessary. In many patients in whom treatment is delayed, cicatrization produces total discontinuity between the larynx or upper trachea and the distal trachea. Access to the lower airway is via tracheostomy only (Figure 9-9). Frequently, there will appear to be a long gap between the two ends of the airway. Since the distal end of a separated trachea drops into the mediastinum, whereas the upper segment remains fixed to the less

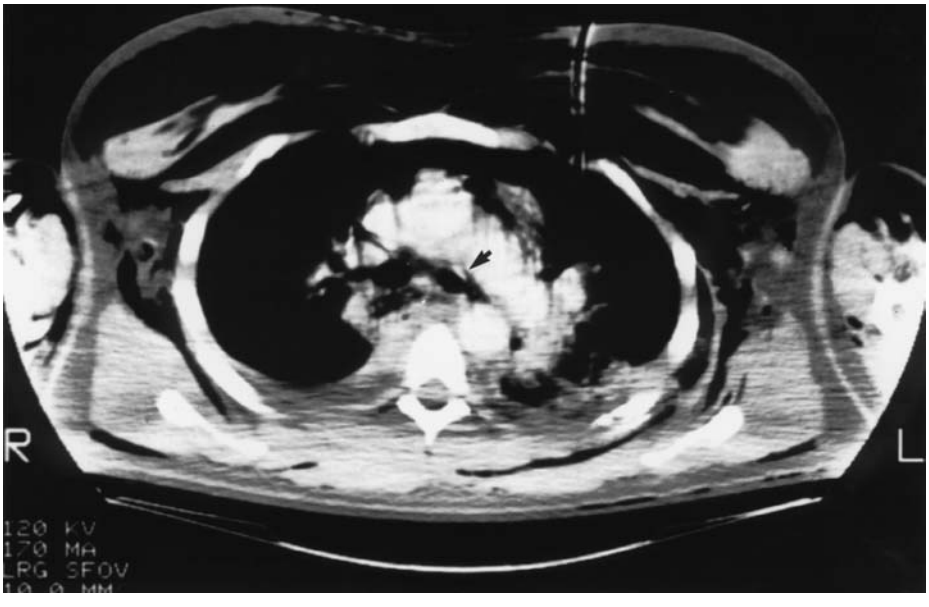


FIGURE **9-6** *Computed tomography scan at and just below the carina showing a separated left main bronchus following blunt chest trauma* (arrow). *Massive emphysema is seen in all layers of the chest wall.*

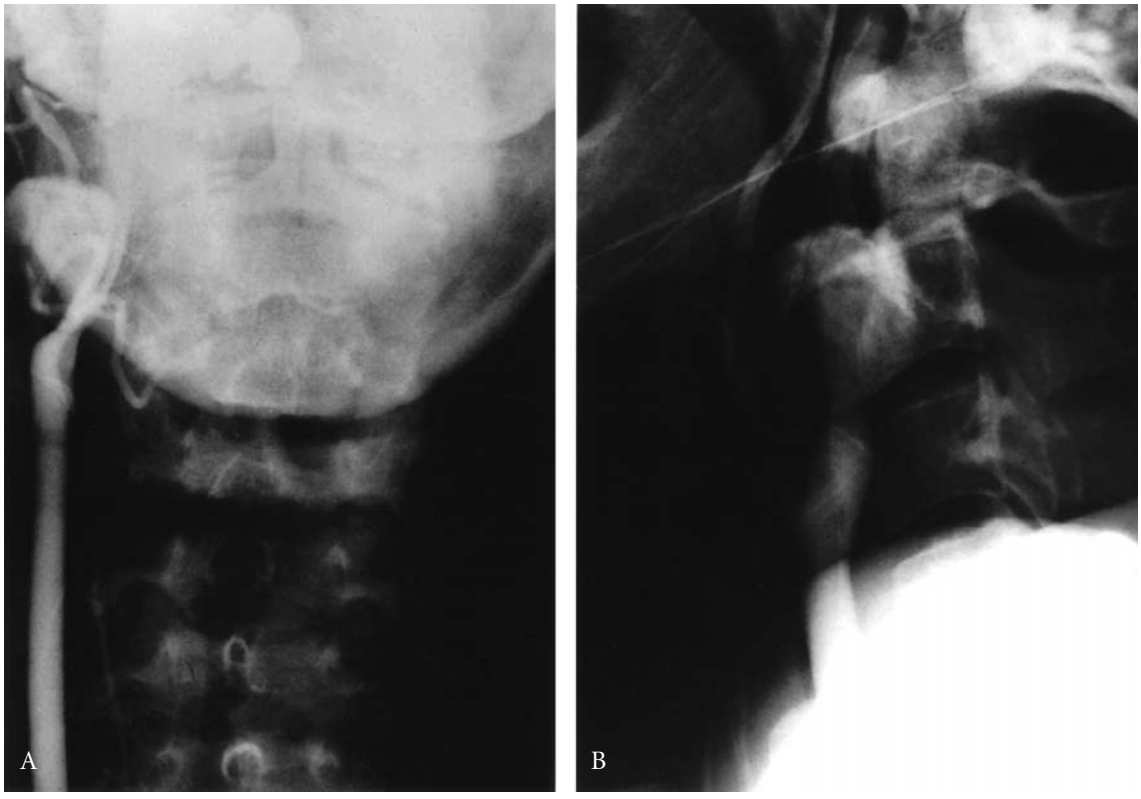


FIGURE 9-7 *Blunt injury to the neck caused separation of the cervical trachea with recurrent laryngeal nerve injury and damage to the right carotid artery resulting in aneurysm, demonstrated in* A, *anteroposterior and* B, *lateral arteriograms. The injury occurred 2 weeks earlier.*

mobile larynx, the apparent major airway gap is usually illusory. In 18 patients seen, late repair was occasioned by failure of initial diagnosis in 9, failed repairs in 8, and due to tracheoesophageal fistula and respiratory failure in 1.[14]

The importance of correct laryngeal assessment cannot be overemphasized. It is sometimes necessary that corrective procedures be applied to the damaged or paralyzed larynx prior to reconnection to the distal trachea (see Chapter 35, "Laryngologic Problems Related to Tracheal Surgery"). If the tracheal separation were corrected first, and only then was it discovered that the airway at laryngeal level was inadequate, the patient would require immediate reintubation through the larynx or a tracheostomy. This could adversely affect the integrity of the tracheal repair. The proper order of business, therefore, is restoration of the larynx to anatomical and functional adequacy, prior to repair of the trachea or sometimes concurrently with airway restoration. *Management* of acute and chronic airway trauma is described in Chapter 31, "Repair of Tracheobronchial Trauma."

If early repair of tracheal separation is not done, it is best to wait 4 to 6 months before delayed repair is performed so that inflammation may subside and scar may mature, providing that obstruction is not present.

Results

Larynx and upper trachea. If principles of management are strictly observed for isolated laryngeal and upper tracheal trauma (see Chapter 31, "Repair of Tracheobronchial Trauma"), the results of treatment of *acute injuries* are generally very good (Table 9-2). Since tissues beyond the actual area of injury are essentially

normal, precise reconstruction with accepted techniques generally produces a permanently satisfactory airway. In a series of 10 patients treated promptly for acute injuries, all achieved an excellent airway, and there were no instances of later tracheal stenosis.[2] Couraud and associates treated 19 patients with laryngotracheal disruption; 11 within 5 days of injury, 17 by similar repairs but with stenting in 13, and 2 by laser and stenting.[3] Excellent respiratory results were achieved in all. Phonation was good in 7 of the patients and fair in 13, reflecting the impossibility of restoring true vocal cord normalcy. Schaeffer reported excellent results in treatment of acute laryngeal trauma.[4] Early definitive treatment was emphasized based on classification by severity of the laryngeal injury, with observation only in the absence of mucosal laceration or cartilaginous fracture and displacement. Functional results are generally better, following early rather than delayed repair.

Delayed management of these injuries presents a multitude of problems, depending on the individual injury and the nature of the prior treatment. Where both recurrent laryngeal nerves are permanently damaged, a wholly adequate albeit husky voice can be obtained. A paralyzed larynx can still function satisfactorily for speech. The glottic aperture must be fixed at approximately 4 mm (see Chapter 35, "Laryngologic Problems Related to Tracheal Surgery"). This provides for clear speech, which is produced largely by the pharyngeal musculature, with the lung bellows providing an air column in a more efficient way than the stomach and esophagus do for so-called "esophageal speech" after laryngectomy. Modulation of voice is lacking. A high school senior who had suffered tracheal separation with bilateral cord paralysis reported, after repair of glottis and trachea, that he was able to return to the debating team but not to the glee club. If the glottic aperture is made narrower, speech may be improved, but the patient will not be able to move

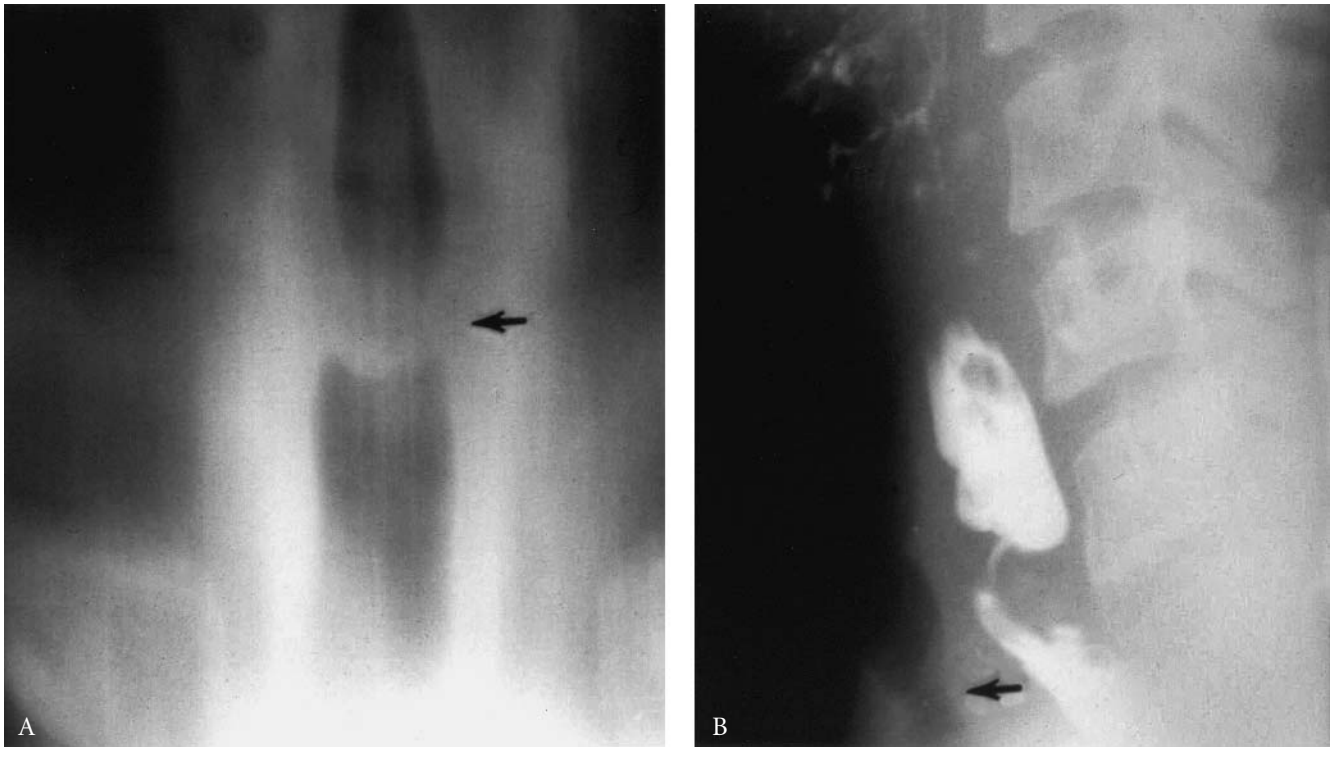


FIGURE **9-8** *Late findings 6 months after a motorcycle-cable injury in a 15-year-old, resulting in high tracheal separation and esophageal avulsion at the cricopharyngeus. The left vocal cord was paralyzed and the right functioned suboptimally.* A, *Anteroposterior tomographic section showing severe stenosis of cervical trachea. The subglottic larynx lies at the top.* Arrow *at site of stricture.* B, *Lateral neck view with swallow of barium.* Arrow *indicates stricture of the trachea. Severe stenosis of the esophagus is demonstrated by contrast medium. Both injuries were successfully corrected surgically in a single procedure.*

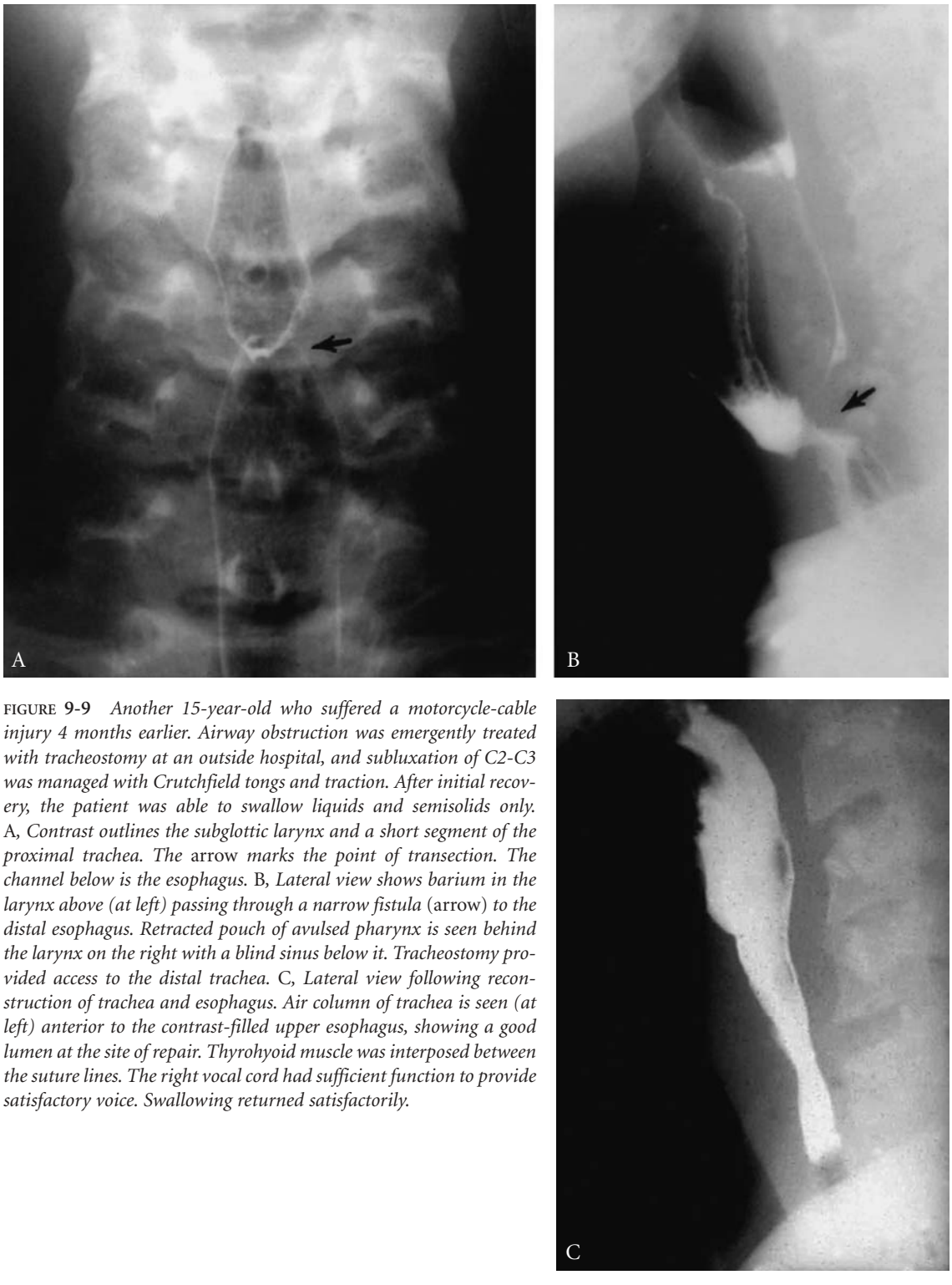


FIGURE **9-9** *Another 15-year-old who suffered a motorcycle-cable injury 4 months earlier. Airway obstruction was emergently treated with tracheostomy at an outside hospital, and subluxation of C2-C3 was managed with Crutchfield tongs and traction. After initial recovery, the patient was able to swallow liquids and semisolids only.* A, *Contrast outlines the subglottic larynx and a short segment of the proximal trachea. The* arrow *marks the point of transection. The channel below is the esophagus.* B, *Lateral view shows barium in the larynx above (at left) passing through a narrow fistula* (arrow) *to the distal esophagus. Retracted pouch of avulsed pharynx is seen behind the larynx on the right with a blind sinus below it. Tracheostomy provided access to the distal trachea.* C, *Lateral view following reconstruction of trachea and esophagus. Air column of trachea is seen (at left) anterior to the contrast-filled upper esophagus, showing a good lumen at the site of repair. Thyrohyoid muscle was interposed between the suture lines. The right vocal cord had sufficient function to provide satisfactory voice. Swallowing returned satisfactorily.*

an adequate amount of air and will be short of breath on exercise. If the aperture is much larger, the patient will be able to exercise more intensely but will lose adequacy of speech and be more susceptible to aspiration. Dysphonia is the most important late disability after laryngotracheal injury.[12]

Table 9-2 Outcome of Airway Injuries

	Injuries	*Deaths*	*Mortality (%)*
Blunt			
Cervical	16	0	0
Thoracic trachea	14	2	14
Bronchial	32	8	25
Total	62	10	16
Penetrating			
Cervical	151	11	7
Thoracic trachea	31	11	35
Bronchial	11	3	27
Total	193	25	13

Reproduced with permission from Bacha EA et al.[1]

In 17 patients on whom delayed reconstruction of the airway was done, 16 attained a good airway without limitation of activities.[2] One patient preferred to maintain a tracheal cannula so that he could open it intermittently during strenuous exercise. Variable results were obtained with vocal function. A good voice was attained in 10 patients, 3 of whom had unilateral and 4 of whom had bilateral vocal cord paralysis. Six patients had a husky but functional voice. Three of these had unilateral paralyses and 3 had bilateral paralyses. In 1 case, adequate vocal function was not achieved. This patient had one paralyzed cord and another with partial paresis. He was able to speak only in a whisper. No patient with tracheoesophageal fistula repaired by the technique described (see Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula") had subsequent recurrence of the fistula.

Thoracic Trachea and Bronchi. High mortality is often associated with intrathoracic tracheal and bronchial trauma but this is due to severe associated injuries or widespread body trauma (see Table 9-2).[11] Blunt injuries of sufficient force to damage the tracheobronchial tree are likely to injure other organs. Penetrating wounds easily injure major structures centered in the thorax. Good anatomic and hence functional results can now be expected following tracheobronchial repair of *acute injuries*, whether linear or anastomotic,[6,12] if performed in accordance with current techniques. Poorly conceived or executed reconstructions are likely to fail.

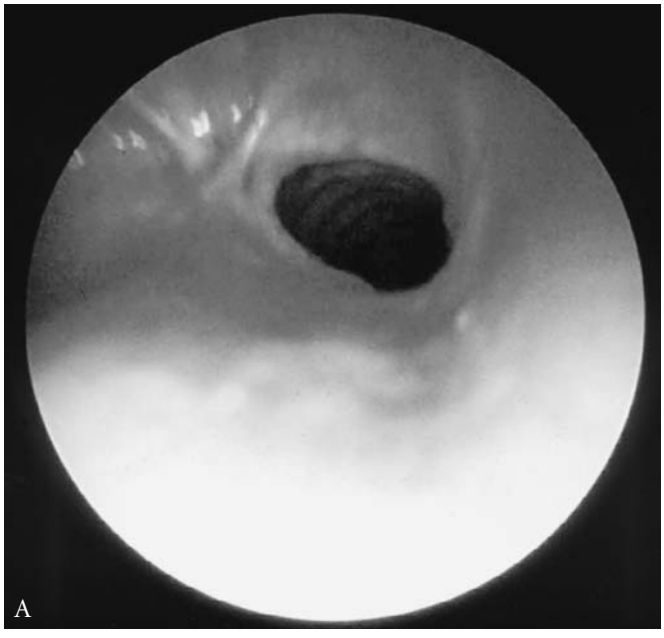
Late tracheal injuries, usually presenting as a stenosis, are successfully managed by resection and reconstruction (Figure 9-10).[2,14] Main bronchial stenosis from *overlooked injuries* (Figure 9-11) or failed repair should be managed by careful dissection and reanastomosis, even if the lung has been functionless for a long time. It appears that the proportional return of function is roughly inversely proportional to the length of time elapsed. Deslauriers and colleagues demonstrated a quite respectable return of lung function after reanastomosis.[15] If chronic sepsis or fibrosis has supervened, pneumonectomy is indicated to remove a septic source. Lesser treatments, including dilation, laser, and steroid injection, have no lasting benefit.

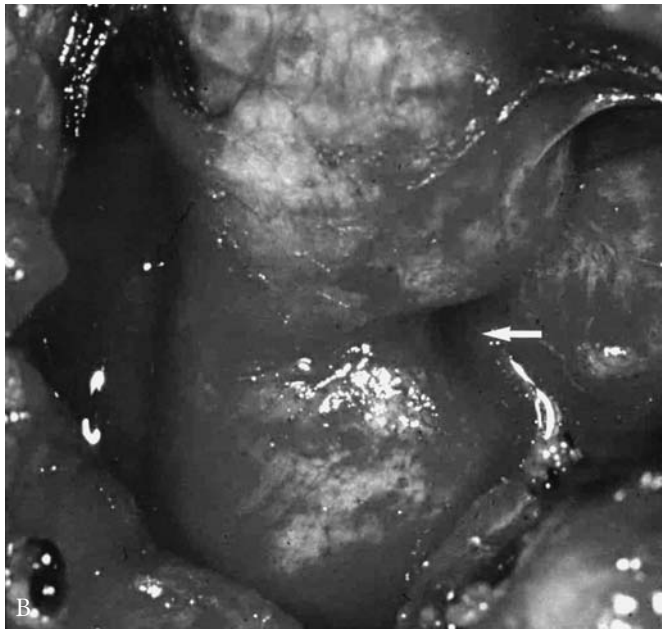
Generally, repair of fresh injuries is accomplished most often by suture repair, with minimal debridement, and resection is rarely required. Late repairs most often need resection with reconstruction.[14] In 28 patients with early and late repairs, at all levels, only 3 suffered airway complications; 1 early dehiscence was promptly repaired and 2 late separations were treated with T tubes, one of which remained permanently.[14] Death from liver failure occurred in 1 cirrhotic patient. Individual patients present special challenges, both with respect to type and location of injury, concomitant trauma, and prior treatment and timing (Figure 9-12).[6]

Burns

Inhalation Burns

Burns of the larynx, trachea, and bronchi may be caused by inhalation of hot gases, steam, particulate matter in smoke, or of chemical substances released in industrial explosions or combustion. We have encountered burns due to house fires, motor vehicle accidents, electrical exposure, gas explosion, airplane crash, TV set explosion, and chemical inhalation (ammonia and hydrochloric acid), among many other origins of burns.[16] It is often difficult to know precisely what the mixture of damaging agents was. In addition to heat, injury may be produced by irritant gases such as aldehydes, ammonia, and hydrochloric acid, and by particulate matter. Moylan and Chan observed by bronchoscopy that one-third of burn patients had evidence of inhalation injury.[17] Ninety-seven percent of these had facial burns and 75% were injured in a





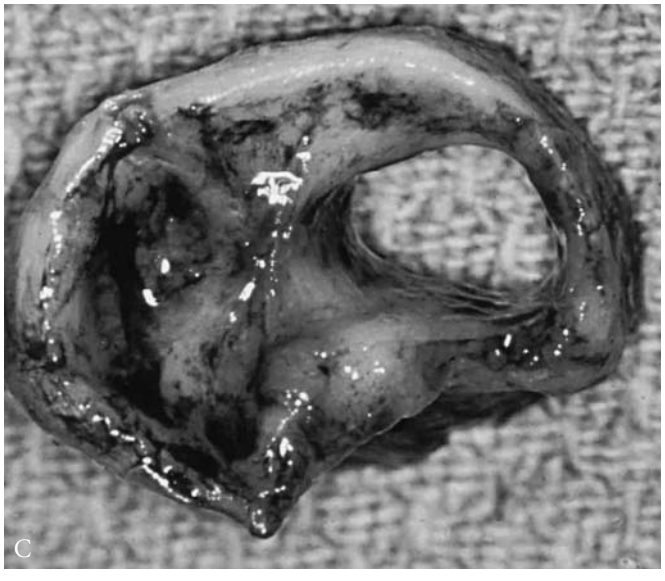
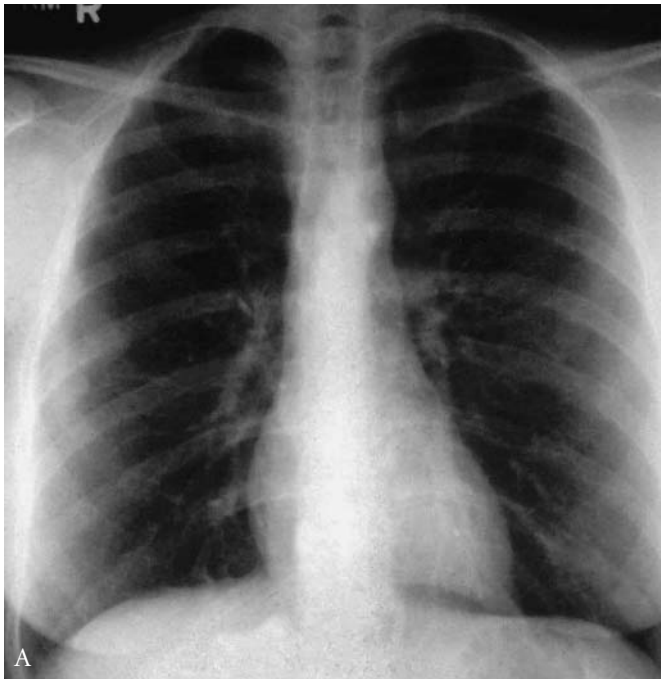
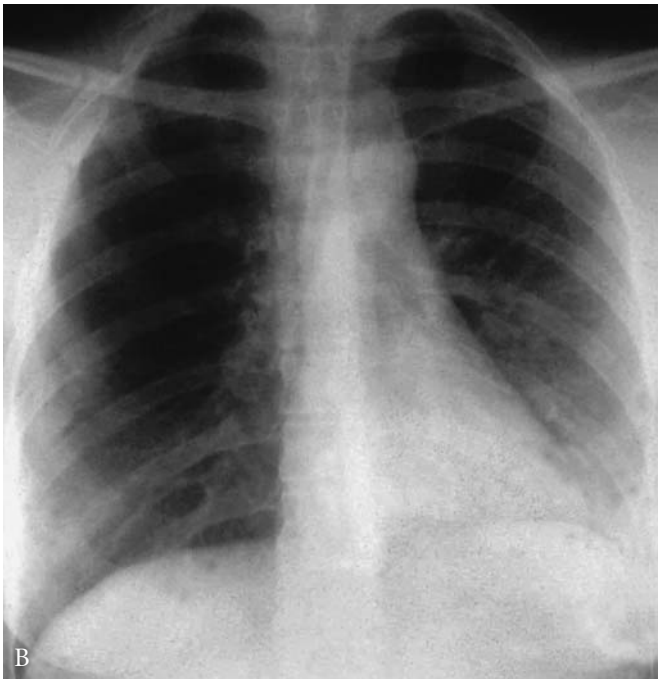


FIGURE **9-10** *Findings in a patient who developed progressively severe dyspnea late after anterior chest trauma.* A, *Bronchoscopic examination shows tight, well-healed stenosis in midtrachea.* B, *Gross appearance of the site of left-sided transverse laceration of mediastinal trachea* (arrow). *The wedge defect brings to mind a woodsman's attack on a tree trunk.* C, *Resected specimen of stenosis. The stenotic airway is about one-fourth or less of the normal tracheal cross section. Also, see Figures 21 and 22 (Color Plate 14).*





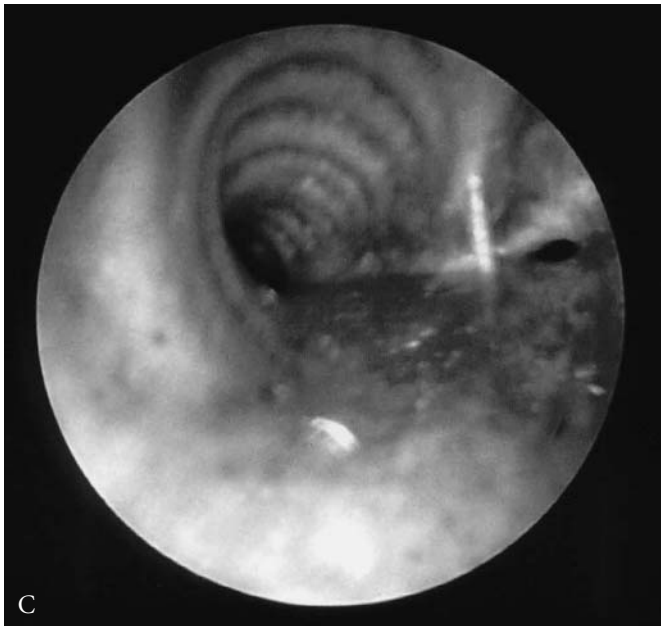


FIGURE **9-11** *A 26-year-old patient suffered multiple trauma in a motor vehicle accident 5 months earlier, with initial bilateral pneumothoraces which responded to tube drainage. With late onset of dyspnea, a right main bronchial stenosis with nearly complete occlusion was identified. Two attempts were made at different institutions to dilate the stenosis.* A, *Inspiratory chest x-ray shows apparently good bilateral expansion.* B, *Expiratory film shows continued full expansion on the right due to air trapping.* C, *Bronchoscopy reveals nearly complete occlusion of the right main bronchus at the origin. Obstruction is not complete, however. Resection of the stenosis and reanastomosis of the right main bronchus allowed her to return to climbing in the Grand Tetons. Also, see Figure 23 (Color Plate 14).*

closed space. Seventy-five percent of these patients developed severe respiratory complications and one-third of them died as a result of airway burns.

The patient often also suffers from cutaneous burns of varying extent and depth. When first examined, the patient may have significant burns of the oropharynx, largely of thermal origin. These commonly heal and regress so that the glottis may soon appear quite normal. Varying degrees of persisting injury are observed in the subglottic larynx and upper trachea, the worst being just beneath the glottis with gradual diminution of the effects of the burn proceeding distally.[16] These burns appear to result more from chemicals and particulates in smoke, except in the case of steam burns. An exaggerated necrotizing process

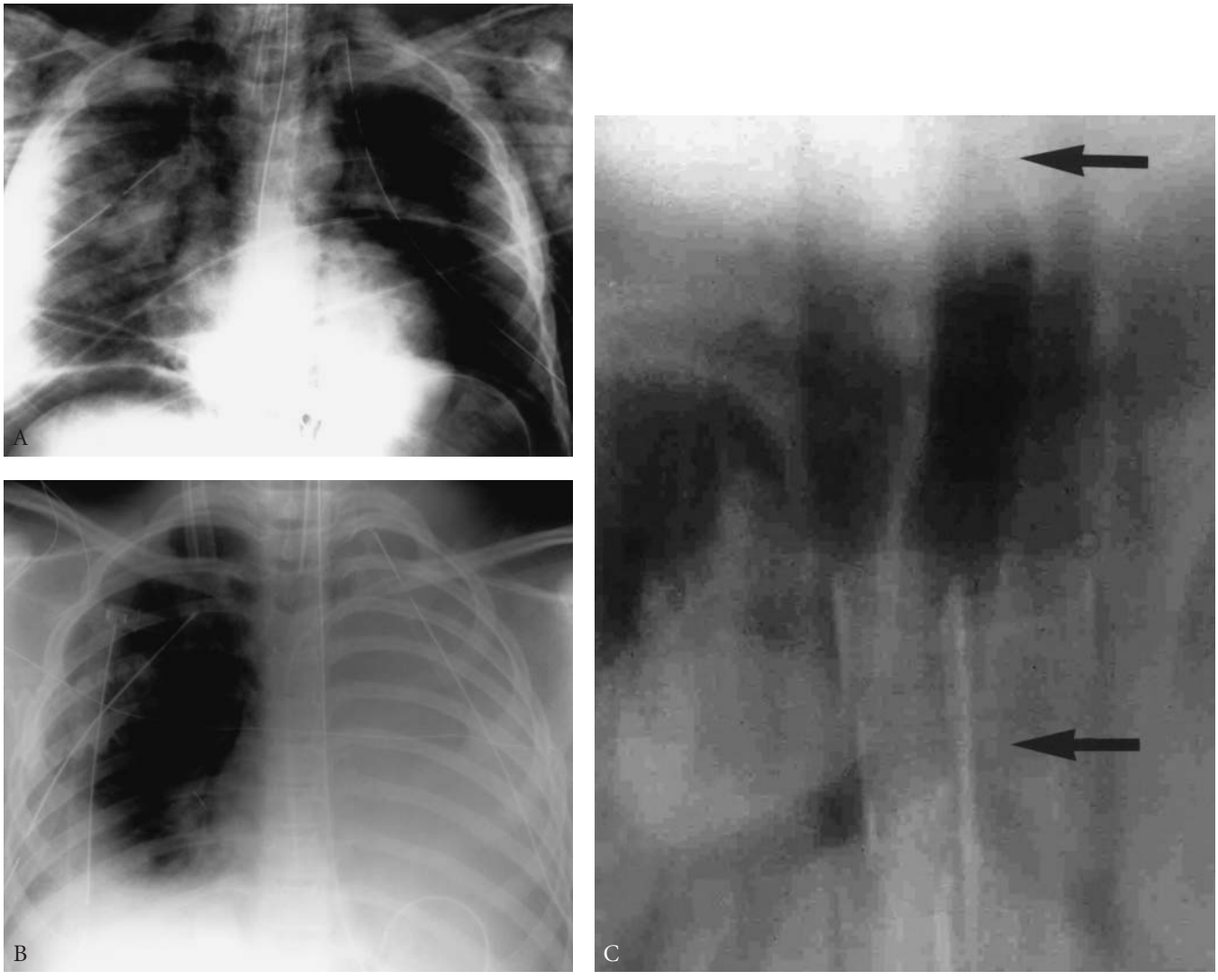


FIGURE **9-12** *Example of complex tracheobronchial injury in an 8-year-old girl, thrown from a vehicle and crushed as it rolled over.* A, *Initial chest roentgenogram. Bilateral pneumothoraces (intubated) and extensive emphysematous dissection of chest wall muscles and planes. There is little evidence of rib fractures or displacement, due to flexibility of the rib cage in childhood. At the initial hospital, laparotomy was negative. Tracheal transection was identified and presumptive repair was done under cardiopulmonary bypass.* B, *Subsequent chest film shows complete opacification of the left chest. Eight days after injury, she was urgently transferred. The initial repair was found to be inadvertent anastomosis of trachea to right main bronchus rather than to trachea. The concurrent rupture of the left main bronchus went unrecognized. At right thoracotomy, the tracheobronchial repair and right hilum were mobilized intrapericardially. The left bronchial stump could not be freed under the aortic arch because of inflammation and early scar formation. This required mobilization via left thoracotomy. Left intrapericardial mobilization was also done and the aortic arch was freed. Finally, via sternotomy and transpericardial dissection between the vena cava and aorta, and after proximal tracheal mobilization, it was possible to implant the debrided left main bronchus in the left lateral wall of trachea above the prior anastomosis. A second anesthesia machine was used to reinflate the left lung prior to anastomosis, vastly improving oxygenation. If the pathologic findings after 8 days could have been anticipated, alternative incisions might have been possible.* C, *Tomogram showing final reconstruction. The* upper arrow *marks the glottic level. In the neocarina, the right main bronchus appears long due to deviation of the distal trachea to the right, below the left bronchial anastomosis* (lower arrow). *The patient's further course was excellent.*

immediately below the cords was first noted in victims of The Cocoanut Grove fire and was attributed to eddy currents. Some unfortunate patients sustain injury extending into the main bronchi and below. The intensity of damage varies. Severe tracheobronchitis may be followed by mucosal sloughing. If the basal cell

layer remains intact, early repair is accomplished rapidly, both clinically and experimentally. If the basal membrane is destroyed, granulations, cicatrization, and stenoses may follow.

A late complication of inhalation burn, 2 to 6 months after acute injury, is the formation of endobronchial polyposis.[18,19] Significant hemoptysis occurs. Polyps regressed over 6 months without treatment in one patient and while receiving corticosteroids in another. Fatal bronchiolitis obliterans has been described as a late complication of inhalation burn following explosion in a confined space.[20]

Effects of inhalation injury and the intubation injury resulting during treatment are difficult to separate, especially since intubation is often performed early in the presence of respiratory symptoms. Burn injury may well make the trachea more susceptible to intubation injury. Stenoses are often of greater length than those resulting from intubation injury alone. Injury may also extend beyond the level of grossly visible changes. Often, the cartilages of the trachea appear to be only slightly injured. Peritracheal fibrosis is almost always found.

The incidence of stenosis due to burn injury is impossible to determine, given the variety and intensity of agents and the widespread use of intubation in management. Clinical reports are largely composed of patients who were intubated. Two of 38 survivors of burns treated with intubation developed subglottic stenosis in one series, and in another, 6 of 25 survivors of airway complications treated with tracheostomy developed tracheal stenosis. In a search for later sequelae of inhalation burns in 17 survivors, 4 had tracheal stenosis and 5 had significant tracheal granulomas. Gaissert and colleagues treated 18 patients with chronic airway compromise after inhalation burns; there were 18 tracheal stenoses, 14 subglottic strictures, and 2 main bronchial stenoses.[16] Three patients developed laryngotracheal stenoses *without* intubation.

Evaluations of patients with inhalation burns include tracheal and laryngeal radiography followed by laryngoscopy and bronchoscopy under general anesthesia. In our 18 chronic patients, 14 had subglottic as well as tracheal stenoses, and in 4, the two areas were separated by a tracheal segment which was not stenosed.

If airway obstruction occurs, whether by laryngeal edema, inflammatory swelling, granulations, or later stenosis of the burned subglottic larynx and trachea, an airway is best established urgently by endotracheal intubation. Obstruction may also occur 3 weeks to 5 months after injury. If the patient has pulmonary damage that requires ventilation, a cuffed tube is necessary. Otherwise, it is preferable to avoid an inflated cuff.

For long-term management, tracheostomy becomes necessary if the patient's neck is not damaged by the burn. The tracheostomy usually lies within the damaged area of the trachea. As inflammation subsides, a T tube may be inserted to span the entire area of injury (see Chapter 39, "Tracheal T Tubes"). If the subglottic larynx is involved, as it often is, the T tube must extend up through the glottis. This is frequently necessary because the intensity of an inhalation burn is often greatest in the subglottic larynx. A T tube, with its upper end between the false and true vocal cords, usually permits hoarse or whispered speech as well as swallowing without aspiration. Training by a speech pathologist is advisable. If the proximal end of the T tube is sited in the subglottic location where there is burn injury, it will repeatedly become obstructed by granulation tissue.

The T tube maintains airway patency, preserves understandable phonation, and permits gradual resolution of burn injury to the mucosa and submucosa. Burn injury resolves only very slowly, and resulting cicatrization matures slowly, which is entirely parallel with the evolution of cutaneous burns. Attempts to perform surgical resection and early reconstruction of the airway are likely to fail.[16] With patience and persistence by both the patient and surgeon, conservative management is most likely to result in a satisfactory airway, although not a normal one (Figure 9-13). Since cartilages remain basically intact, the goal is regression of granulations and stabilization of the mucosal and submucosal process. In 5 patients treated with T tubes only, and 4 with laryngofissure and T tube, decannulation was achieved between 4 to 61 months (mean 28 months) after injury. Four patients required permanent tracheal tubes (2 T tubes and 2 tracheostomy tubes).

Specific criteria for discontinuance of the T tube based on bronchoscopic observation or biopsy do not exist. Our management has been to attempt to remove the T tube when regression seemed adequate,

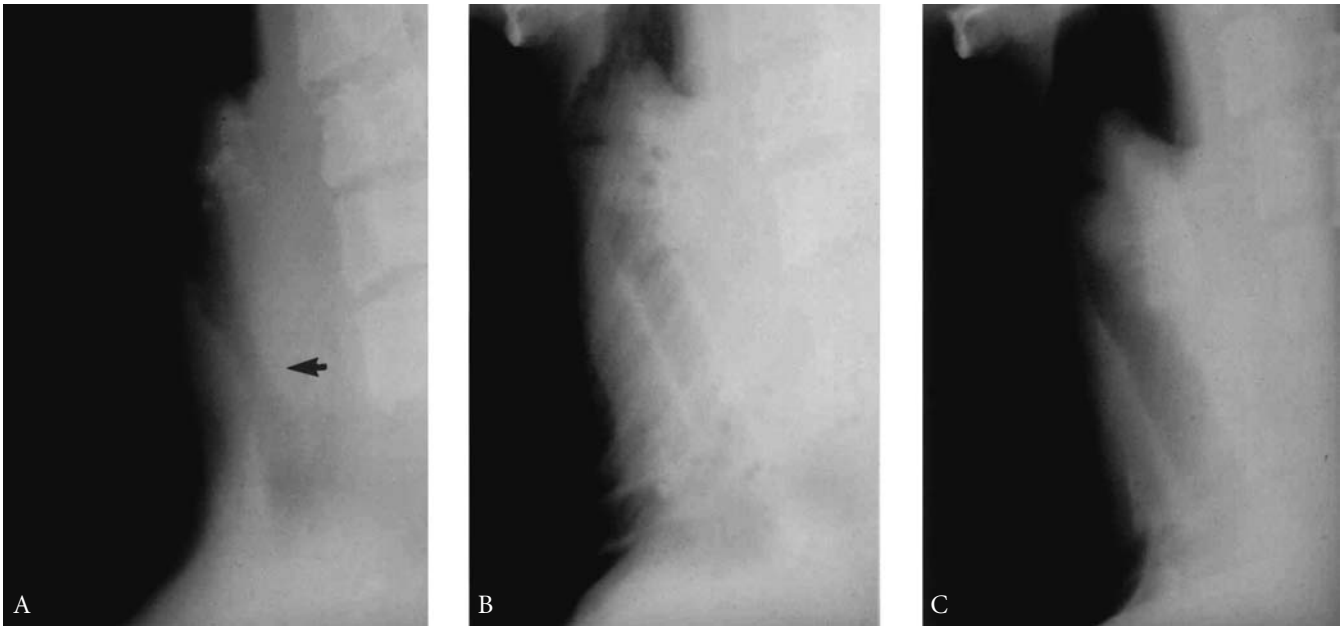


FIGURE **9-13** *Lateral cervical roentgenograms from an 18-year-old female suffering inhalation burn by toxic gases from combustion of plastic building materials.* A, *Diffuse upper tracheal stenosis* (arrow). *Posterior laryngeal calcification is visible superiorly.* B, *T tube in place, extending from the subglottic larynx into normal trachea. The sidearm of the T lies at the base of the neck.* C, *Result after 6 years of splinting is an apparently stable although narrowed subglottic larynx and proximal trachea. Dense reactive scar tissue appeared to have resolved to a degree. However, the patient went on slowly to laryngotracheal stenosis, which was successfully treated by laryngotracheal resection and reconstruction 13 years after decannulation* (C). *She has required several procedures for posterior commissural glottic stenosis and unilateral arytenoid fixation over subsequent years. The laryngotracheal repair has remained stable.*

leaving a cannula or "button" in place to maintain the stoma during the test period. If resection of a limited residual stenosis is necessary, it is preferably done later than earlier, although good response has been reported to earlier repair on occasion.

Operative management is even more hazardous because so many burns involve the subglottic space, where airway reconstruction is more difficult, whether by single-stage laryngotracheal reconstruction or by laryngofissure with resurfacing and stenting. Four of 6 patients who ultimately underwent open repair of a subglottic stenosis had good results. Management of stenosis extending into the carina and main bronchi, fortunately rare, is even more difficult. We hesitate to use a T-Y tube since the bronchial ends of the tube may stimulate more reaction if they lie in areas of burn injury. Isolated bronchial stenosis has been managed by repeated dilation. An inlying stent would pose the danger of inciting granulomas.

Successful outcome of treatment does not result in a normal airway. The quality of voice is often diminished and a degree of hoarseness is present. Mild chronic wheezing and recurrent episodes of respiratory tract infections occur. We have seen a case of late recurrent obstruction, but there is no large experience with these injuries.

Ingestion Burns

Ingestion of caustic substances such as lye may produce burns of the oropharynx, larynx, and esophagus. The epiglottis may be destroyed, and the vocal cords injured severely enough to fibrose. Tracheal ingestion is not common, perhaps due to the protective reflexes of the glottis. Severe caustic injury to the upper esophagus may on rare occasion penetrate by necrosis through the back wall of the trachea or left main bronchus. Tracheal injury is usually distal.

Attempts at conservative management of such injuries penetrating from the esophagus have not succeeded. Appropriate treatment, although not sufficiently documented, would seem to be removal of the destroyed esophagus, conservative debridement of the airway injury, and patching with appropriate tissue. A carefully sutured intercostal muscle pedicle flap, using multiple fine sutures placed as if in an anastomosis and not simply as "tacking" sutures, should provide protection. The omentum should also be considered for buttressing in such a catastrophic situation.

Laser Burns

Despite warnings and precautions, occasionally, a plastic endotracheal tube has been ignited by a laser, producing disastrous tracheal and bronchial thermal burns.[21] Such a lesion is managed in the same fashion as inhalation burns, but the extent and depth may well be fatal, especially if the injury extends into the bronchial tree. Even Y or T-Y stents become useless. Similar burns have been produced by inept use of the cautery during tracheostomy.

A laser burn of the left main bronchus was incurred in an ill-advised and ill-executed attempt to destroy an area of dysplasia. The resulting complete stenosis of the bronchus was successfully treated by bronchial resection and anastomosis (see Figure 38 [Color Plate 16]). A tracheoesophageal fistula produced by laser treatment of cuff stenosis was managed by standard reconstructive techniques. Since the laser damage was localized, reconstruction was feasible in this case.

Injuries caused by *external irradiation* are discussed in Chapter 41, "Radiation Therapy in the Management of Tracheal Cancer." I have encountered an irremediable main bronchial stenosis due to a misguided use of *brachytherapy* to irradiate peribronchial tissue rather than the intralumenal tumor. The resulting complete stenosis was fused to the adjacent pulmonary vessels.

References

1. Bacha EA, Mathisen DJ, Grillo HC. Airway trauma. In: Westaby S, Odell JA, editors. Cardiothoracic trauma. London: Arnold; 1999. p. 265–79.
2. Mathisen DJ, Grillo HC. Laryngotracheal trauma. Ann Thoracic Surg 1987;43:254–62.
3. Couraud L, Velly JF, Martigne C, N'Diaye M. Posttraumatic disruption of the laryngotracheal junction. Eur J Cardiothorac Surg 1989;3:441–4.
4. Schaeffer SD. The acute management of external laryngeal trauma. A 27-year experience. Arch Otolaryngol Head Neck Surg 1994;118:598–604.
5. Burke JF. Early diagnosis of traumatic rupture of the bronchus. JAMA 1962;181:682–6.
6. Symbas PN, Justicz AG, Ricketts RR. Rupture of the airways from blunt trauma: treatment of complex injuries. Ann Thorac Surg 1992;54:177–83.
7. Kiser AC, O'Brien SM, Detterbeck FC. Blunt tracheobronchial injuries: treatment and outcomes. Ann Thorac Surg 2001;71:2059–65.
8. Massard G, Rougé C, Dabbagh A, et al. Tracheobronchial lacerations after intubation and tracheostomy. Ann Thorac Surg 1996;61:1483–7.
9. Mussi A, Ambrogi MC, Menconi G, et al. Surgical approaches to membranous tracheal wall lacerations. J Thorac Cardiovasc Surg 2000;120:115–8.
10. Hulscher JBF, Hofstede E, Kloek J, et al. Injury to the major airways during subtotal esophagectomy: incidence, management and sequelae. J Thorac Cardiovasc Surg 2000;120:1093–6.
11. Angood PB, Attia EL, Brown RA, Mulder DS. Extrinsic civilian trauma to the larynx and cervical trachea — important predictors of long-term morbidity. J Trauma 1986;26:869–73.
12. Chagnon FP, Mulder DS. Laryngotracheal trauma. Chest Surg Clin North Am 1996;6:733–48.
13. Kelly JP, Webb WR, Moulder PV, et al. Management of airway trauma II: combined injuries of the trachea and esophagus. Ann Thorac Surg 1987;43:160–3.
14. Wright CD, Wain JC, Donahue DM, et al. Tracheobronchial trauma: management of early and late airway disruption. [In preparation]
15. Deslauriers J, Beaulieu M, Archambault G, et al. Diagnosis and long-term follow-up of major bronchial disruptions due to nonpenetrating trauma. Ann Thorac Surg 1982;33:32–9.
16. Gaissert HA, Grillo HC, Lofgren R. Upper airway compromise after inhalation injury: complex strictures of larynx and trachea and their management. Ann Surg 1993;218:672–8.
17. Moylan JA, Chan CK. Inhalation injury — an increasing problem. Ann Surg 1978;188:34–7.
18. Adams C, Moisan T, Chandrasekhar AJ, Warpeha R. Endobronchial polyposis secondary to thermal inhalational injury. Chest 1979;75:643–5.
19. Williams DO, Vanecko RM, Glassroth J. Endobronchial polyposis following smoke inhalation. Chest 1983;84:774–6.
20. Perez-Guerra F, Walsh RE, Sagel SS. Bronchiolitis obliterans and tracheal stenosis. Late complications of inhalation burn. JAMA 1971;218:1568–70.
21. Cros A-M. Lasers and the airway. In: Roberts JT, editor. Clinical management of the airway. Philadelphia: WB Saunders; 1994. p. 355–67.

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Tracheostomy:
Uses, Varieties, Complications

Hermes C. Grillo, MD

Tracheostomy

Other than being a surgical technique, tracheostomy has many aspects that merit discussion; hence, I thought it best to consider these in a section of this book which is otherwise devoted to diseases of the trachea. Included elsewhere are historical notes on tracheostomy, surgical technique (see Chapter 22, "Tracheostomy, Minitracheostomy, and Closure of Persistent Stoma"), tracheostomy devices (see Chapter 38, "Tracheal Appliances" and Chapter 39, "Tracheal T Tubes"), and stents (see Chapter 40, "Tracheal and Bronchial Stenting").

Indications

For many years, tracheostomy was the primary means of providing emergency relief for upper airway obstruction. In the period following World War II, tracheostomy was considered to have three primary purposes: 1) emergency relief of airway obstruction, 2) management of secretions, especially after chest and central nervous system injury, and 3) to decrease respiratory dead space in order to improve ventilation. During the poliomyelitis epidemics of the early 1950s, tracheostomy was increasingly used to provide a route for administration of positive pressure ventilation for respiratory failure or impending respiratory insufficiency. This widespread use increased awareness of the many potential complications of tracheostomy and also introduced a new spectrum of lesions that resulted from intubation and mechanical ventilation. The immediate complications of tracheostomy, now very much reduced in incidence, are noted in this chapter, but the later complications, largely of postintubation origin, are dealt with in Chapter 11, "Postintubation Stenosis," Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula," and Chapter 13, "Tracheal Fistula to Brachiocephalic Artery." Also considered in this chapter is the special topic of tracheostomy in children.

Today, tracheostomy is rarely performed to provide an *emergency airway*. The airway is usually reestablished emergently by speedy insertion of an endotracheal tube, usually orally. Benign stenosis, such as that resulting from intubation, is best managed emergently by systematic dilation, without tracheostomy. Even in the presence of organic upper airway obstruction by a tumor, it is usually possible to slip an endotracheal

tube into the airway, above or past an obstruction, in order to provide emergency ventilation until the obstruction can be dealt with bronchoscopically (see Chapter 19, "Urgent Treatment of Tracheal Obstruction"). For *difficult intubations*, a flexible bronchoscope is frequently of great help (Figure 10-1). The endotracheal tube is passed over the bronchoscope, which has been advanced beyond the obstruction. This technique is also valuable in patients with anatomically difficult airways or in those whose access is limited by severe cervical arthritis or temporomandibular arthritis, or by malformations. The rigid bronchoscope may also be a last resort for airway access, and in most cases, except the last mentioned, it can be introduced perorally by a skilled bronchoscopist. On rare occasions, an endotracheal tube may be threaded over a rigid pediatric bronchoscope, with a short length of proximal "pusher" tube also threaded proximal to the endotracheal tube (see Figure 10-1). With the aid of a straight bladed laryngoscope (Miller blade), the assembly is passed into the airway, the endotracheal tube pushed into the trachea, and the bronchoscope and "pusher" tube withdrawn. The laryngeal mask airway is a useful option if all else fails (see Figure 10-1). Roberts discusses the many aspects of these problems in *Clinical Management of the Airway*.[1] Finally, if obstruction is high, a large bore needle or a small bore catheter may be inserted through the cricothyroid membrane to provide emergent oxygenation while either intubation or tracheostomy is accomplished. Many previously seen immediate complications of tracheostomy were incurred during the emergent placement of a tra-

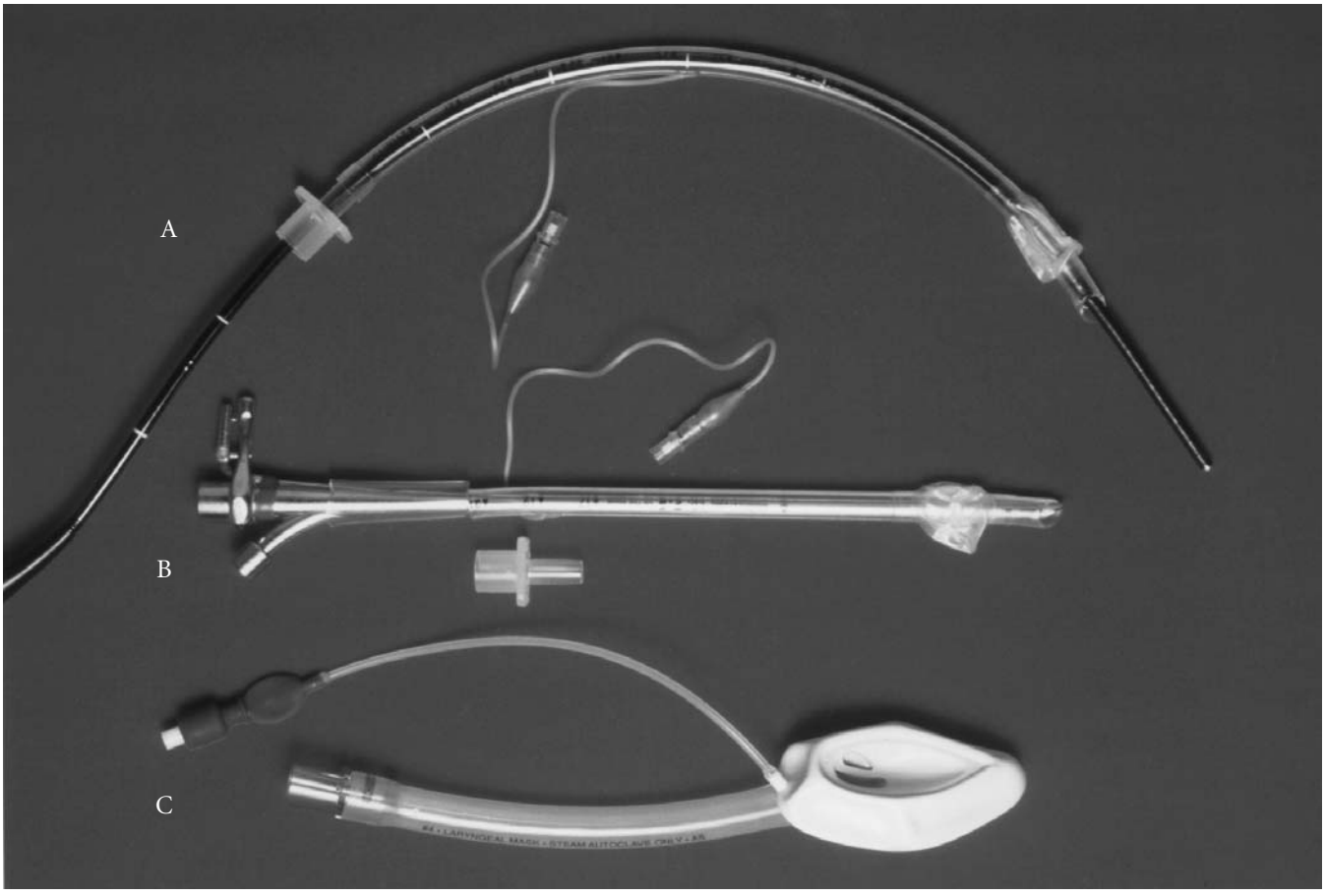


FIGURE **10-1** *Tools for the management of difficult intubations. From top to bottom.* A, *Endotracheal tube (ET) threaded over a flexible bronchoscope.* B, *Endotracheal tube over a pediatric rigid bronchoscope. Note the short segment of plastic "pusher" tube on the bronchoscope proximal to the ET. The assembly is introduced with a straight-bladed, open laryngoscope. The ET adapter is replaced after intubation is effected.* C, *Laryngeal mask airway, which is introduced to rest upon and seal over the larynx.*

cheostomy tube under poor conditions. With tracheostomy now being almost always an elective procedure, these early complications, such as injury to carotid vessels or pneumothorax, have vanished.

The second classic indication for tracheostomy, the *management of secretions*, has not been entirely replaced. However, with adequate humidification of the airway, effective employment of skilled pulmonary physiotherapy, endotracheal catheter suctioning, and more commonly, frequent flexible bronchoscopic aspiration, intubation is rarely necessary for secretions alone. Minitracheostomy, considered in detail later in this chapter, is an effective method for the management of persistent copious secretions.

With the advent of positive pressure ventilatory support, tracheostomy is no longer used to facilitate ventilation by reducing dead space.

A current major use of tracheostomy is as a route for *mechanical ventilatory support*. Endotracheal tubes are used for prolonged periods of time for ventilatory support. Although endotracheal tubes may produce their unique spectrum of complications, tracheostomy is often deferred for some time.[2] No definitive studies are available to dictate a universally accepted management policy. If an endotracheal tube must be left in for more than 48 hours, it is often replaced with a more comfortable nasotracheal tube. Secretions are more easily managed by nursing staff through a tracheostomy tube than through an endotracheal tube. For this reason, as well as for patient comfort, and, importantly, in order to avoid serious injury to the glottis and subglottis, we usually replace an endotracheal tube with a tracheostomy tube in an adult in between 5 to 7 days, unless it appears that the patient may be weaned in a further short period of time. If it is clear that a patient will require prolonged mechanical ventilation, as in polyneuritis, then tracheostomy is done earlier.

Tracheostomy continues to be useful to establish an airway temporarily or permanently wherever *chronic obstruction* presents, and definitive correction must be postponed or is not possible. In many such situations, a T tube will be preferable (see Chapter 39, "Tracheal T Tubes").[3] In the case of an obstructive benign stenosis which is accessible in the neck, it is mandatory that the tracheostomy or T tube stoma be located *in the stenotic segment*. In this way, further damage to the trachea is prevented, and if resection is later possible, the stoma and stenotic lesion will be simultaneously resected. Unfortunately, surgeons continue to place tracheostomies below such lesions, thereby damaging a further centimeter or two of the trachea (Figure 10-2*A*). Often, it becomes necessary to relocate such a misplaced stoma to the stenotic segment and allow the initial stoma to heal, in order to recapture this length of trachea and so facilitate or make possible later reconstruction. If a stenotic lesion is very low, even retrosternal, the stoma is carefully placed at the conventional level (second and third rings), leaving a generous segment of normal trachea between the stoma and lesion. The tracheostomy tube must then be long enough to pass through the dilated stenosis to splint it open. If the tracheostomy tube tip lies above the stenosis, it will only create an illusion of airway control (Figure10-2*B*).

Additional indications for tracheostomy include complementary use with laryngeal anastomoses that may suffer transient glottic edema and for management of aspiration due to laryngeal dysfunction. Postoperative tracheal anastomotic complications may require tracheostomy (or T tube placement), sometimes for definitive management, but most often to permit resolution until reconstruction may again be safely considered (see Chapter 21, "Complications of Tracheal Reconstruction"). Tracheostomy should generally not be used for an obstructing tumor. Tumor is best managed in emergency by intubation past the tumor and then by the coring-out of tumor bronchoscopically (see Chapter 19, "Urgent Treatment of Tracheal Obstruction"). Definitive treatment then follows, either surgical resection or irradiation. Occasionally, under *emergency circumstances in non-hospital settings*, the need arises for external opening into the airway. Most often, the Heimlich maneuver accomplishes dislodgement of an acute supraglottic obstruction, such as that caused by an aspirated chunk of food. If this fails, and in the absence of laryngoscopic equipment, cricothyroidotomy should be employed. The cricothyroid membrane is the most superficial portion of the cervical airway. Even in obese individuals, this area can usually be palpated with the neck in hyperexten-

sion. In my opinion, cricothyroidotomy should almost never be used as an elective route of airway intubation because of the likelihood of serious and possibly irreversible laryngeal damage. Emergency cricothyroidotomy is preferably converted to tracheostomy if its use will be prolonged.

Jackson in 1921 cautioned against "high tracheostomy" (cricothyroidotomy) as a cause of subglottic stenosis (Figure 10-2*C–E*).[4] The precept was challenged by Brantigan and Grow in 1976, although they later reported an incidence of subglottic stenosis following the elective procedure.[5,6] Antecedent endotracheal intubation for a length of time appeared to predispose to such a stenosis.[7,8] An overall incidence of at least 2% stenosis and up to 32% permanent voice changes followed cricothyroidotomy.[7] Since there are few real advantages, if any, to cricothyroidotomy over tracheostomy, and since subglottic laryngeal stenosis is sometimes poorly correctable or impossible to correct, whereas nearly all postintubation tracheal stenoses are initially correctable, there is no reason to subject a patient to the hazard of cricothyroidotomy. The concern which led to the use of cricothyroidotomy, that is, separation of the stoma from median sternotomy for cardiac surgery, may be addressed by a few days of endotracheal tube ventilation to allow tissue planes to seal, thereby permitting tracheostomy to be done safely. However, the question of whether cricothyroidotomy poses a threat to median sternotomy remains controversial.[9,10]

Technique

The technique of tracheostomy is described in Chapter 22, "Tracheostomy, Minitracheostomy, and Closure of Persistent Stoma." Elective tracheostomy is best done in the operating room with adequate instruments and under ideal conditions. This minimizes the occurrence of both early and late complications.

Percutaneous tracheotomes of various designs have appeared repeatedly over the decades, each seeming to lead eventually to a series of unnecessary and often serious complications, such as obstruction due to hemorrhage, damage to major blood vessels in the neck, intubation into the mediastinum rather than the airway, pneumothorax, tracheal wall laceration, and injury to recurrent laryngeal nerves. Recent techniques require placement of a guide wire or catheter and endoscopic confirmation of its location before plunging a tracheotome into the neck.[11] A so-labelled "fingertip subcricoid minitracheostomy" (done at bedside) is a combination of a very small incision for dissection to the tracheal wall with wire-guided dilation and tube insertion, still generally called a percutaneous tracheostomy.[12] With these precautions, it is hoped that many of the previously seen complications will not result. The presumed advantage over fully open procedures remains unsettled. Melloni and colleagues compared surgical versus percutaneous dilational tracheostomy prospectively in a consecutive series of 50 patients.[13] They concluded that the percutaneous method was simpler and quicker, and had many fewer early complications, but had two late complications (malacia and stenosis), compared with no late complications surgically.

Complications

The majority of *immediate complications* of tracheostomy incurred were often due to hurried performance of tracheostomy under inadequate emergency conditions, with poor definition of anatomic landmarks. These complications have largely disappeared.[14] Hypoxia, which occurs during an urgent performance of tracheostomy and is sometimes accompanied by cardiac arrest, is eliminated when the procedure is done under elective conditions after establishment of an adequate airway. Laceration of the membranous tracheal wall can occur as a result of excessively forceful insertion of an inappropriate tube. The procedure of tracheostomy itself nowadays almost always follows establishment of an airway. A rare exception may be necessary, in separation of the cervical trachea following blunt trauma, where emergency bronchoscopy fails to reveal a channel to the distal trachea (see Chapter 9, "Tracheal and Bronchial Trauma"). In such a case, preparation is always made prior to endoscopy for emergent tracheostomy. Operative damage to structures such as the recurrent laryngeal nerves, the great vessels of the neck, and the esophagus has been

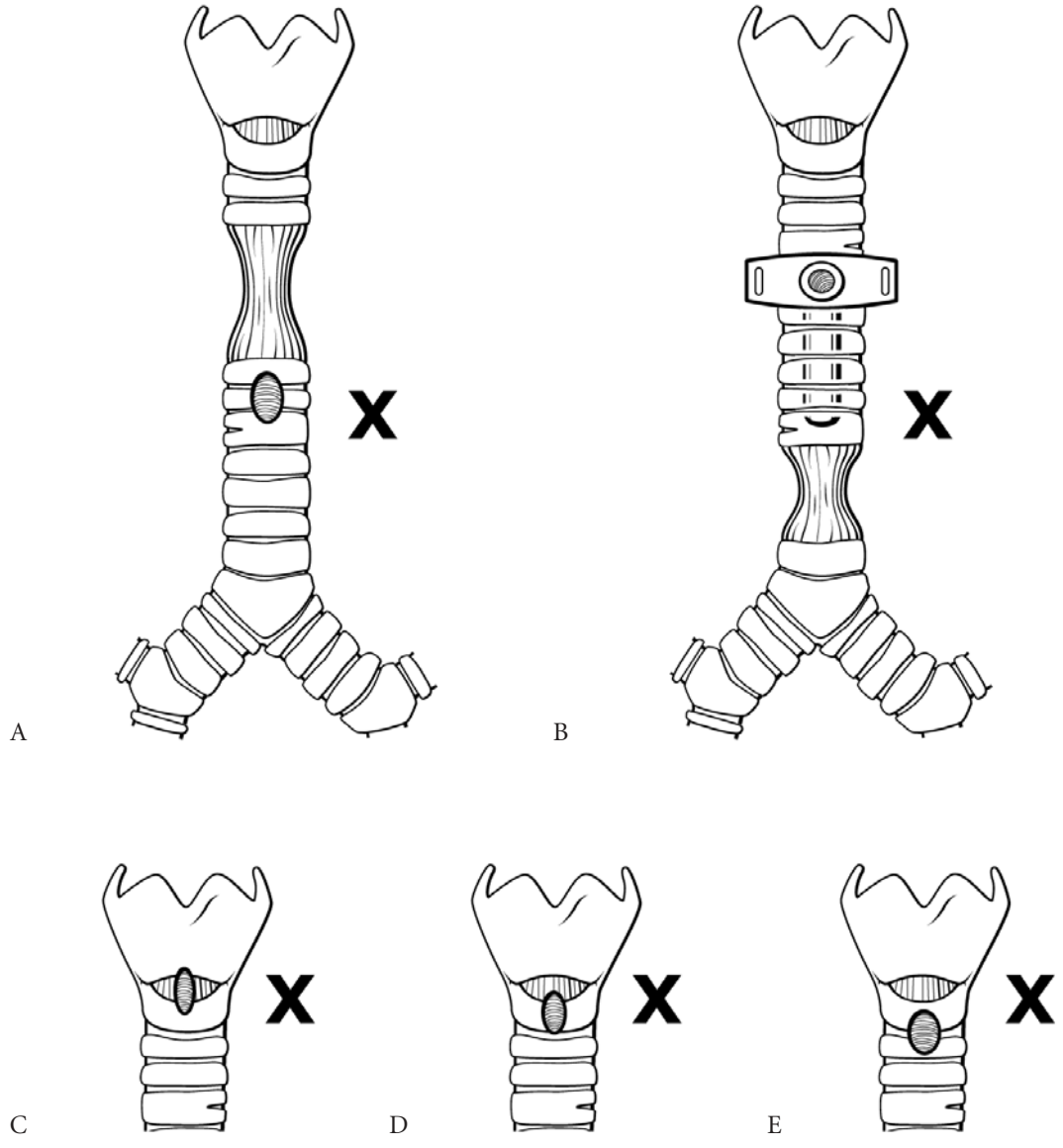


FIGURE **10-2** *Errors in location of the tracheostomy.* A, *Placement of the stoma below a cervical stenosis lengthens the extent of damaged trachea. A needed stoma should be located in the stenosis, an already damaged segment. In some patients, it is judicious or necessary to relocate a stoma correctly and allow the prior stoma to heal, in order to recapture usable trachea for reconstruction.* B, *An insufficiently long tube that fails to pass through a low stenotic lesion fails in its purpose. The solution is* not *to lower the stoma but to use a longer tube. In urgent cases, a modified endotracheal tube may be used.* C, D, *The stoma should not be located, whether by intent or error, in the cricothyroid membrane or through the cricoid cartilage.* E, *A stoma located just below the cricoid, especially in a kyphotic patient, may erode the central cricoid cartilage and result in a partly intralaryngeal subglottic stenosis.*

largely eliminated with adequate exposure, good lighting, adequate anesthesia, and precise surgical technique. Pneumothorax during tracheostomy has become very rare. It tended to occur in small children. Obese, short-necked, kyphotic individuals may still present technical challenges.

Longer-term complications present chiefly as sepsis, hemorrhage, and obstruction. Additional complications include acquired tracheoesophageal fistula (TEF) and persistence of tracheal stoma. *Sepsis* of invasive or necrotizing type is surprisingly rare, even though all tracheostomies are soon contaminated, most

often with *Staphylococcus aureus* (often a resistant strain) and *Pseudomonas aeruginosa*. *Streptococcus* and *Escherichia coli* are frequently present. This will occur despite sterile surgical technique and careful management of stoma, tubes, and suctioning. Antibiotics are not employed unless there is evidence of local invasion or pulmonary infection, to minimize overgrowth of other organisms. The contamination clears when the device is removed and the stoma is permitted to heal.

Hemorrhage, *obstruction* at the laryngeal and tracheal level due to granuloma, stenosis, and malacia, *TEF*, and *persistent stoma* are detailed in subsequent chapters, and their surgical treatment described (see Chapter 11, "Postintubation Stenosis," Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula," and Chapter 13, "Tracheal Fistula to Brachiocephalic Artery"). Prevention is also stressed.

Dysphagia and a tendency to aspiration, more pronounced in the elderly, may follow tracheostomy. Usually, this will ameliorate with time.

Minitracheostomy

Matthews and Hopkinson described minitracheostomy as a method for removal of secretions from large airways while preserving glottic function for cough and phonation.[15] A small-bore cannula (OD 5.4 mm) is placed into the trachea through the cricothyroid ligament for ease in suctioning. The technique is described in Chapter 22, "Tracheostomy, Minitracheostomy, and Closure of Persistent Stoma." There is no interference with glottic competence, and the cough, which is stimulated by suctioning, is effective. The cannula may also be used for the intratracheal administration of medications and for access for jet ventilation. It may also be used in an emergency for temporary access to the airway. Minitracheostomy does not carry with it the potential complications of endotracheal intubation or of formal tracheostomy, since it does not interfere with the normal physiology of glottic function and cough, nor does it produce the same kind of potential for local tissue injury. It also seems far less likely to injure the subglottic larynx than conventional cricothyroidotomy does with placement of a full sized cannula. It is applicable to any patient who has excessive tracheobronchial secretions or ineffective cough. If a patient does not respond to the usual postoperative techniques for clearing of secretions, namely, chest physiotherapy, blind endotracheal suctioning, or flexible bronchoscopy after a limited number of trials, then minitracheostomy offers an additional avenue. It is best used early in patients in whom such difficulties may be anticipated. Minitracheostomy was effective when electively used prophylactically, to control sputum retention after lung operation in high-risk patients.[16] A no. 10 French suction catheter is used for aspiration, and a small amount of saline (3–5 cc) is instilled prior to suctioning to stimulate cough and to loosen secretions. The cannula is otherwise capped to maintain humidification and to prevent drying of secretions. After initial placement, the catheter is suctioned frequently, then every 4 hours, and subsequently as needed. The cannula is well tolerated and does not limit the patient's mobility, speaking, breathing, or swallowing. It does not stimulate aspiration, as sometimes a tracheostomy will. Suctioning through a minitracheostomy is more comfortable than blind endotracheal suctioning and it is certainly more comfortable than flexible bronchoscopy. Minitracheostomy has been particularly useful in postoperative patients who demonstrate difficulty in clearing secretions. In our experience, over 70% of the patients who required minitracheostomy responded to this technique alone.[17] The rest of the patients required endotracheal intubation or formal tracheostomy because of progressive pulmonary insufficiency.

Minitracheostomy does not provide an airway of sufficient diameter for spontaneous nonassisted ventilation with complete glottic obstruction. Although it may be used in an emergency situation for insufflation of oxygen, it can only be used as an airway with jet ventilation, using ventilators to provide the proper humidification, as described by Matthews and colleagues.[18] Campbell and colleagues demonstrated no permanent changes in laryngeal function or adverse effects on the larynx following minitracheostomy.[19]

Clinical Experience

Wain and colleagues analyzed the first 56 patients in whom minitracheostomy was used at the Massachusetts General Hospital.[17] Twenty-four of the patients were general thoracic, 9 were cardiovascular, 18 were general surgical, and 5 were medical. Their preceding surgical procedures and conditions included thoracotomy (20), esophagectomy (8), coronary artery bypass grafting (8), thoracic or abdominal aortic aneurysm repair (7), gastrointestinal (7), and subdural hematoma (1). Sixty minitracheostomies were placed, with repeat procedures performed at discrete intervals for different indications during complex hospital courses rather than because of malfunction of the minitracheostomy itself. The principal indication was excessive postoperative secretions in 46 patients. Twenty-five of these patients who were extubated immediately had minitracheostomy placed at a mean of 4 days after surgery, and in 21 patients who were ventilated postoperatively, the minitracheostomy was placed at a mean of 2 days after extubation. Additional indications were excessive postpneumonic secretions (5), preoperative secretions (4), and difficulty with standard blind endotracheal suctioning (4). In 1 patient, a minitracheostomy was used as an emergency means of establishing an airway until formal tracheostomy was done. The procedure was done at bedside in the intensive care unit in 32 patients and in a medical or surgical unit in 28 patients.

Complications were seen in 1 in 6 patients. Intratracheal bleeding of significance occurred in 2 patients and emergency intubation was required. If intratracheal bleeding of significance occurs, an endotracheal tube must be placed immediately, over a flexible bronchoscope, before withdrawing the minitracheostomy catheter. Two cases of hemorrhage were probably related to injury to veins anterior to the cricothyroid membrane, with bleeding into the trachea, following removal of the cannula. If the cannula has been successfully placed in the trachea, it is left in place and the superficial hemorrhage is controlled by pressure or other conservative measures. Extratracheal placement of the catheter has been described by others, but it is our belief that careful attention to anatomic landmarks will prevent this. Serious complications may be recognized or prevented by performing flexible bronchoscopy *routinely* with every placement of a minitracheostomy tube.

Tracheostomy in Children

The spectrum of diseases requiring intubation or tracheostomy in infants and small children differs from that of the adult. Congenital malformations of the nasopharynx, oral cavity, neck, larynx, and trachea or laryngeal hemangioma may be indications for urgent intubation. Acute laryngeal inflammatory processes including acute epiglottitis and laryngotracheal bronchitis, as well as acute laryngeal edema, sometimes due to allergic phenomena, may be emergency problems. Diphtheria is fortunately very rare and poliomyelitis has become so as well. The tiny size of the airway in the newborn or young child requires special skill in intubation. Nasotracheal tubes are preferred for comfort, especially if respiratory support is necessary. Cuffed tubes are not necessary for ventilation of infants and small children. In order to avoid erosive damage, the diameter of the tube is selected so that it will not impinge on the airway throughout its length. Severe and lengthy malacia or stenosis may result if a tube of excessive diameter remains in firm contact with the trachea for long. A tight fit at the glottic level may injure the cords and commissures, and result in stenosis. A tight fit at the cricoid level may produce subglottic stenosis.[20] Infants and small children are carried for longer periods with endotracheal tubes, in order to avoid tracheostomy with its additional problems. On the other hand, the same trade-off of laryngeal injury is seen in children as in adults, although problems at the tracheal stoma may be eliminated (see Chapter 11, "Postintubation Stenosis").

Tracheostomy in an infant or child should be done over a previously established airway and under general anesthesia. Hendren and Kim recommended insertion of a rigid bronchoscope, which elevates the trachea and makes it easy to identify.[21] A limited horizontal skin incision is preferred, placed below the

cricoid. It is often necessary to divide the thyroid isthmus. The cartilaginous rings of the juvenile trachea are so tiny and soft that it seems preferable to divide vertically the third and fourth rings rather than the second and third as in the adult. It is important to avoid subglottic laryngeal injury. No tracheal wall is excised. Tracheostomy tubes fashioned specifically for infants and small children, such as those introduced by Aberdeen, are selected, avoiding tubes of too large a diameter (Figure 10-3).[22] The curve of such tubes is unlikely to produce anterior granuloma or erosion from the tube tip. A degree of flexibility allows the tube to adapt to the curve of the airway. Modern humidification techniques permit the elimination of the inner cannula. The tube flanges slope upward so that the tapes do not tend to pull out the tube. The tubes are designed to accept a tracheostomy connector proximally, regardless of their basic internal diameter.

With such precautions and with use of tubes especially designed for pediatric use, decannulation problems are reduced. The underlying lesion or a complication of the tracheostomy is probably the most common cause for difficulty in decannulation. Gradual progression to smaller tracheostomy tubes to smoothen the process of decannulation is sometimes advisable.

The soft, thin wall of the infant trachea is easily deformed by a tracheostomy tube. In a number of children who have had tracheostomy tubes in place for some time, the anterior wall of the trachea just superior to the stoma becomes depressed by tube pressure. This deformity, together with thickening of the lower margin of the depressed flap, may cause obstruction on decannulation. Insertion of a small sized Montgomery Silastic T tube, with or without minimal excision of the tip of thickened scar at the lower end of the flap, restores the lumen. The tube is left in place for 3 months or more to allow the flap to become fixed in this more normal position. The T tube is then withdrawn and the child observed carefully for airway obstruction. A further period of splinting may be necessary. Residual cartilaginous structure must be

FIGURE **10-3** *Pediatric tracheostomy tube. Pictured is a size 4 (ID 5.5 mm, OD 8 mm, length 4.6 cm) silicone tube (Shiley, Mallinckrodt Inc., St. Louis, MO). The curve of the tube and arrangement of the flanges are adapted for infants and small children, following the Great Ormond St. design. Note the standard size of the proximal adaptor end.*

present in the depressed flap to obtain permanent correction in this way. If the tracheal wall has been extensively replaced by scar, a stenosis will follow, requiring resection. It must be cautioned that T tubes are sometimes poorly tolerated in small children, probably because of the tiny diameters of their tracheae.[3]

Persistent Stoma

Most tracheostomies close promptly after extubation. Indeed, reinsertion of a tracheostomy tube may become difficult within minutes or hours after its removal. In a few patients, the stoma persists, most often in those who have carried a tracheostomy tube for a very long time, who are aged or debilitated, who suffer from metabolic diseases, or who have received prolonged corticosteroid treatment. In most of these patients, the cutaneous epithelium has healed to the tracheal epithelium. A stoma is considered to be persistent if it remains patent 3 to 6 months after extubation. Some stomas contract after removal of the tube, but reach an end point without complete healing. The persistent stoma varies from full size to a sinus through which air and secretions escape. The persistent stoma may be a source of both difficulty and annoyance to the patient. Such patient may have to occlude the stoma in order to speak properly or to cough effectively, may be troubled by secretions, and breath with a whistling sound. I have not seen proven instances of increased susceptibility to pulmonary infection in such patients, but this concern is frequently raised. A patient with chronic obstructive pulmonary disease, dependent on nasal oxygen, found that she could breathe with much more comfort when the vent of the persistent stoma was closed.

In most cases, closure of a persisting stoma is a minor procedure, easily done by techniques such as approximation of the strap muscle over the stoma. With a larger stoma, however, such a mesenchymal seal may lead to granuloma formation on the inner surface of the closed stoma. Our technique, described in Chapter 22, "Tracheostomy, Minitracheostomy, and Closure of Persistent Stoma," provides immediate epithelial closure within the lumen and also corrects the cosmetic deficit that is usually present.[23] Needless to say, closure is done only if the patient no longer requires a stoma for any purpose. If the patient still needs a stoma for suctioning, or if there is immediate likelihood of another tracheostomy, then closure is pointless. The single complication in over 30 patients was a subcutaneous hematoma in 1 patient with an undrained wound.

This technique of closure has also been used in conjunction with resection of a more distal cuff stenosis of the trachea, where healing of skin to tracheal epithelium was present at the stoma. If granulation tissue rings the stoma, then this closure should not be attempted. In such patients, spontaneous closure will generally follow.

References

1. Roberts JT. Clinical management of the airway. Philadelphia: WB Saunders; 1994.
2. Stauffer JL, Olson DE, Petty TL. Complications and consequences of endotracheal intubation and tracheostomy. Am J Med 1981;70:65–75.
3. Gaissert H, Grillo HC, Mathisen DJ, Wain JC. Temporary and permanent restoration of airway continuity with the tracheal T-tube. J Thorac Cardiovasc Surg 1994; 107:600–6.
4. Jackson C. High tracheostomy and other errors: the chief causes of chronic laryngeal stenosis. Surg Gynecol Obstet 1921;32:392–8.
5. Brantigan CO, Grow JB Sr. Cricothyroidotomy: elective use in respiratory problems requiring tracheostomy. J Thorac Cardiovasc Surg 1976;71:72–81.
6. Brantigan CO, Grow JB Sr. Subglottic stenosis after cricothyroidotomy. Surgery 1982;91:217–21.
7. Cole RR, Aguilar EA III. Cricothyroidostomy versus tracheotomy: an otolaryngologist's perspective. Laryngoscope 1988;98:131–5.
8. Weymuller EA, Cummings CW. Cricothyroidotomy: the impact of antecedent endotracheal intubation. Ann Otol Rhinol Laryngol 1982;91:437–9.
9. Stamenkovic SA, Morgan IS, Pontrefact DR, Campanella C. Is early tracheostomy safe in cardiac patients with median sternotomy incisions? Ann Thorac Surg 2000;69:1152–4.
10. Curtis JJ, Clark NC, McKenney CA, et al. Tracheostomy: a risk factor for mediastinitis after cardiac operation. Ann Thorac Surg 2001;72:731–4.
11. Hazard PB, Garrett HE Jr, Adams JW, et al. Bedside percutaneous tracheostomy: experience with 55 elective patients. Ann Thorac Surg 1988;46:63–7.
12. Ciaglia P, Graniero K. Percutaneous dilatational tracheostomy — results and long-term follow-up. Chest 1992;101:464–7.
13. Melloni G, Muttini S, Gallioli G, et al. Surgical tracheostomy versus percutaneous dilational tracheostomy. A prospective randomized study with long-term follow-up. J Cardiovasc Surg 2002;43:113–21.

14. Grillo HC, Mathisen DJ. Tracheostomy and its complications. In: Sabiston DC, editor. Textbook of surgery. 15th ed. Philadelphia: WB Saunders; 1997. p. 1815–20.
15. Matthews HR, Hopkinson RB. Treatment of sputum retention by minitracheostomy. Br J Surg 1984;71: 147–50.
16. Bonde P, Papachristos I, McCraith A, et al. Sputum retention after lung operation: prospective, randomized trial shows superiority of prophylactic minitracheostomy in high-risk patients. Ann Thorac Surg 2002;74:196–203.
17. Wain, JC, Wilson DJ, Mathisen DJ. Clinical experience with minitracheostomy. Ann Thorac Surg 1990;49:881–6.
18. Matthews HR, Fisher HBJ, Smith BE, Hopkinson RB. Minitracheostomy: a new delivery system for jet ventilation. J Thorac Cardiovasc Surg 1986;92:673–5.
19. Campbell JB, Watson MG, Povey L, Shenoi PM. Minitracheostomy and laryngeal function. J Laryngol Otol 1988;102:49–52.
20. Lindholm CE. Prolonged endotracheal intubation. Acta Anaesth Scand 1969;Suppl 33:1–131.
21. Hendren WH, Kim SH. Pediatric thoracic surgery. In: Scarpelli EM, Auld PAM, Goldman HS, editors. Pulmonary disease of the fetus and newborn and child. Philadelphia: Lea & Febiger; 1978. p. 166–234.
22. Aberdeen E. Tracheostomy and tracheostomy care in infants. Proc R Soc Med 1965;58:900–2.
23. Lawson DW, Grillo HC. Closure of a persistent tracheal stoma. Surg Gynecol Obstet 1970;130:995–6.

Postintubation Stenosis

Hermes C. Grillo, MD

Acute tracheal lacerations resulting from intubation with endotracheal tubes or tracheostomy tubes are described in Chapter 9, "Tracheal and Bronchial Trauma." Late secondary lesions, principally stenosis of the larynx and trachea, are discussed here. Tracheoesophageal and tracheoarterial damage due to intubation are treated in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula," and Chapter 13, "Tracheal Fistula to Brachiocephalic Artery."

Characteristics and Origin of Lesions

Since the 1960s, the steadily increasing use of endotracheal, tracheostomy, and cricothyroidostomy tubes for the management of secretions, prevention of aspiration and, most importantly, delivery of mechanical ventilatory support for respiratory failure have produced a spectrum of upper airway lesions that range in location from the nostril to the lower trachea, and in severity from pharyngitis to complete obstruction of the airway or asphyxiating hemorrhage (Figure 11-1). Immediate and early complications of tracheostomy are described in Chapter 10, "Tracheostomy: Uses, Varieties, Complications." The majority of these lesions may be avoided by elective performance, use of careful technique, and proper management of tracheostomy. The later complications detailed in this chapter at first seemed unavoidable and unpredictable; in large part, they no longer are. Despite great strides that have been taken in their prevention, postintubation lesions continue to be the most frequently seen surgical tracheal problems. Their clinical characteristics and nature must be made better known so that patients will not continue to suffer delay in recognition of the lesions and so that optimal treatment is given.

The tracheal surgeon must become familiar with laryngeal lesions that result from intubation. Since most patients are initially ventilated through an endotracheal tube, the larynx may also sustain lasting laryngeal injury, even though the patient presents clinically with a tracheal lesion or a tracheostomy. Serious complications may result if a surgeon repairs the trachea without prior assurance of laryngeal competence. For example, an inadequate glottis that is not recognized because of the presence of a tracheostomy preoperatively, and is diagnosed only after tracheal reconstruction, may require either endotracheal intuba-

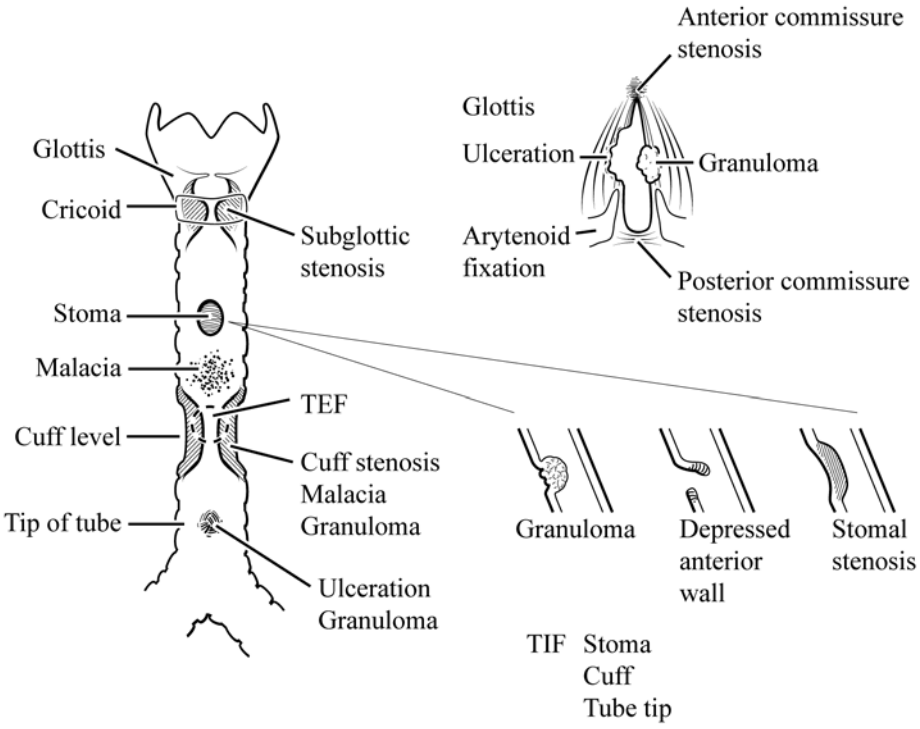


FIGURE **11-1** *The spectrum of postintubation laryngeal and tracheal lesions. Various combinations of these lesions are seen in a single patient. Focus on a laryngeal or tracheal lesion should not lead to overlooking a lesion elsewhere in the airway. TEF = tracheoesophageal fistula; TIF = tracheoinnominate artery fistula.*

tion or another tracheostomy to provide a competent airway, pending glottic correction. Either mode of intubation may compromise the healing of a freshly repaired trachea. A brief catalogue of common postintubation laryngeal lesions follows.

Laryngeal Problems Following Intubation

Laryngologic problems related to tracheal surgery are described in Chapter 35, "Laryngologic Problems Related to Tracheal Surgery." In 1950, Briggs used a plastic endotracheal tube for 42 days to administer prolonged respirator therapy.[1] It remains common practice to replace an endotracheal tube used for ventilator treatment in a much shorter period with a tracheostomy tube. A tracheostomy tube is more easily managed by nursing personnel, with less danger of obstruction and more comfort for the patient than an oro- or nasotracheal tube. Endotracheal intubation provides an airway promptly and can avoid tracheostomy when prolonged mechanical ventilation is unnecessary. It is because of this lessened temporal exposure that fewer cuff lesions were seen following endotracheal intubation than after tracheostomy. A cuff that exerts high pressure on the trachea, either because of its innate characteristics or its usage, is as damaging when on an endotracheal tube as when it is on a tracheostomy tube or a cricothyroidostomy tube. Lesions due to cuffs on endotracheal tubes are usually located higher in the trachea because the cuff is seated higher in the trachea than it is with a tracheostomy tube. Tracheal stomal lesions are obviously avoided if tracheostomy is not done. In using prolonged endotracheal intubation, however, the physician exchanges the absence of potential stomal complications for complications at higher levels (nostril, pharynx, and principally larynx).

Although Briggs, in his original case, found only minor ulcerations over the arytenoid and in two small areas of the trachea at autopsy, a spectrum of more severe lesions occurs.[2,3]

In a series collected from the literature, Lindholm reported approximately 1 death in 120 children as a probable complication rate of prolonged endotracheal intubation.[2] Most deaths occurred during the period of intubation as a result of obstruction of the airway and this probably reflects the small caliber of tubes

necessarily used in small children. Laryngeal mucosal changes were prospectively observed, without exception, after prolonged endotracheal intubation. These were located on the medial sides of the arytenoid cartilages in the interarytenoid region and against the posterolateral portion of the cricoid. Changes in children were less pronounced. The lesions varied from superficial epithelial damage with only slight inflammation to deep ulceration. Donnelly detailed the histopathology of intubation.[4] Within 48 hours, the perichondrium of the vocal processes and cricoid laminae were focally ulcerated, and the severity increased with time. Bergström and colleagues had earlier reported similar damage.[5] Injury to the mucosa was seen by scanning electron microscopy, as early as 4 hours after endotracheal intubation.[6] Lindholm found that 6 of 225 adult patients showed respiratory obstruction after extubation, and in 4 of these patients, the difficulty lay in the larynx.[2] Five of 38 children had respiratory obstruction following extubation, and all required tracheostomy. The precise incidence of postintubation lesions of the larynx or trachea has never been satisfactorily determined, nor are such figures very meaningful, given the changing and varied standards of equipment and care both in time and place. The lesions continue to occur today.

Nearly two-thirds of adult patients with erosive lesions healed by primary epithelization within a month. In a third of the patients, a granuloma formed during healing, located largely on the medial side of the arytenoid cartilages. In many cases, the granuloma regressed spontaneously in 1 to 10 months, with a median of 60 days. The symptoms of a granuloma are irritative cough, hoarseness, and transient sensations of suffocation. Granuloma also occurs on the anterior portion of the vocal cord. In his study, Lindholm found two children who formed fibrous scars with circumferential stenosis at the level of the cricoid and a third with a posterior commissural scar bridging the interarytenoid space. Localization of damage is likely related to the curve of the endotracheal tube. When a relatively straight or slightly arched endotracheal tube is reshaped by the patient's airway, considerable force is exerted posteriorly against the medial sides of the arytenoid cartilages and the posterior surface of the cricoid (Figure 11-2).[2,7] Another factor may be movement between the larynx and endotracheal tube. More prolonged exposure to pressure by the tube seems to lead to a greater incidence and depth of injury. Lindholm recommended a preshaped tube with a gently curved right angle to try to avoid such pressure.

Subglottic erosions at the cricoid level appear to be caused in large part by tubes that are too large for the particular airway. This is supported by the observation of a probably greater frequency of stenosis at this level in women and smaller males, who tend to have smaller airways. Since the cricoid cartilage is nor-

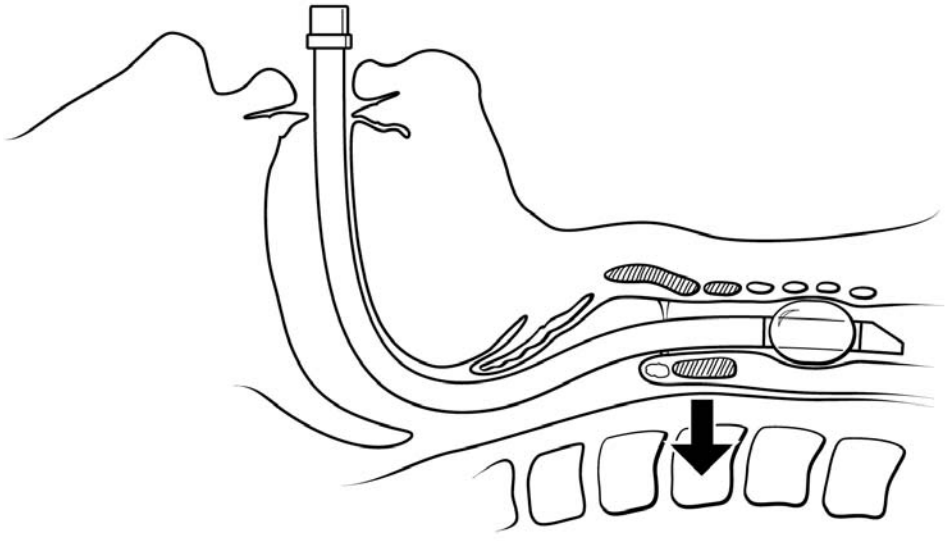


FIGURE **11-2** *Pressure on the larynx is exerted by an endotracheal tube, chiefly posteriorly, against medial arytenoid cartilages and posterolateral cricoid. Adapted from Lindholm C-E.*[2]

mally the only complete cartilaginous ring in the upper airway, the absolute limit of the airway's diameter is set by that cartilage. If pressure results only in edema, the condition is reversible. If ulceration occurs following pressure, then a cicatricial stenosis may result during healing. Fortunately, the cricoid cartilage is a rather rugged structure, and it is unusual for it to be eroded very deeply or through its full thickness by an endotracheal tube. This probably accounts for the fact that prolonged stenting of cicatricial lesions in this region may occasionally lead to success, in contrast to the usual failure of stenting for circumferential lesions in the trachea. It may also account for the greater potential for occasional success in laser treatment of obstructive lesions at this level, in contrast with the trachea where laser treatment often fails.

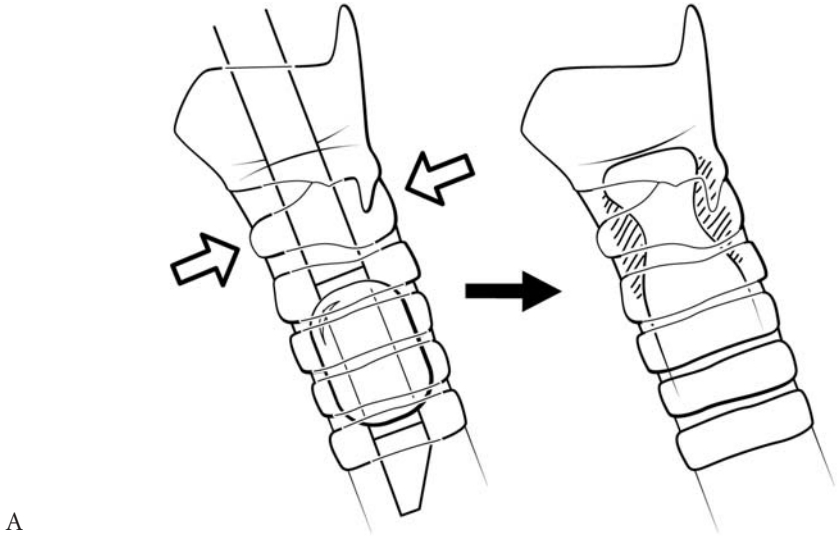
Vocal cord disturbances are common in patients who have undergone prolonged endotracheal intubation. In a small percent, these disturbances may go on for over a month, but frequently regress. Permanent laryngeal paresis does occur in a small number of patients, presumably due to inflammatory involvement of recurrent laryngeal nerves. Such disability is likely to be unilateral.

Postmortem studies have shown the precise location of postintubation ulceration or necrosis of the larynx in 33 patients to be as follows: 3 had ulcers in the interarytenoid area, 26 had ulceration or necrosis on the medial side of the arytenoid cartilages, and 33 ulcerative lesions were seen on the inner posterolateral part of the cricoid.[2,5] Eight of 26 patients also had tracheal lesions. In other reported series, the incidence of subglottic stenosis of the larynx following prolonged endotracheal intubation in children has run from 0 to 8%, with one series reporting as high as 20%.

In clinical practice, one may see the following at the *glottic* level: arytenoid fixation, interarytenoid posterior commissural scar preventing separation of the cartilages, anterior commissural stenosis, and vocal cord thickening, ulceration, or granulomas. Unilateral vocal cord paralysis that is present as a consequence of endotracheal intubation, tracheostomy, or prior surgical procedures may affect the adequacy of the glottic aperture or contribute to aspiration on deglutition. The rare presence of a bilateral cord paralysis, most often post-traumatic or postsurgical, may cause airway inadequacy or aspiration. *Subglottic* intralaryngeal narrowing due to cicatricial stenosis may begin immediately below the glottis or at any level below that. It is often maximal at the cricoid level for the reasons explained. Often, this subglottic laryngeal stenosis is confluent with a stenosis that extends into the upper trachea. In 50 patients treated surgically for postintubation subglottic stenosis with such a major component in the larynx, 31 resulted from endotracheal intubation alone, 16 from high or eroded tracheostomy, and 3 from cricothyroidostomy (Figure 11-3).[8]

The obvious statement must be made that wherever a foreign body impinges forcefully on the airway, whether a stoma is present or not, erosion may occur and be followed by cicatricial stenosis. Cuff lesions are common to all types of tubes and independent of them, except with regard to the level of cuff impingement. Stenotic injuries unique to endotracheal and cricothyroidostomy tubes are necessarily within the larynx. The stomal lesion from a tracheostomy lies within the trachea, except when a stoma is placed too high or, more often, in an older kyphotic patient, where erosion of the anterior cricoid occurs. "Subglottic stenosis," that is, laryngotracheal stenosis beginning below the cords and usually extending into the upper trachea, is circumferential following injury from endotracheal tubes (see Figure 11-3*A* and Figure 28 [Color Plate 15]). Those stenoses that are caused by cricothyroidostomy tubes or eroding tracheostomy tubes may be largely anterior or also circumferential (see Figures 11-3*B*,*C*). In all cases, we are dealing with laryngotracheal "bed sores," since pressure necrosis is the primary damaging factor (Figures 11-4*A*–*E*).

Brantigan and Grow proposed eliminating tracheal stomal stenosis by transferring the stoma to the cricothyroid membrane.[9] Although they were fortunate that their series had no stomal complications (or cuff stenosis), clearly, this approach merely transfers the location from the trachea to the larynx (Figures 11-5*A*,*B*).[3,8] Subsequent studies by these and other authors showed that cricothyroidostomy contributes to a significant and often irreversible subglottic stenosis, with an overall airway coimplication incidence of up to 52%.[10] Kuriloff and colleagues pointed out that the short length of the cricothyroid membrane (5 to 12 mm)



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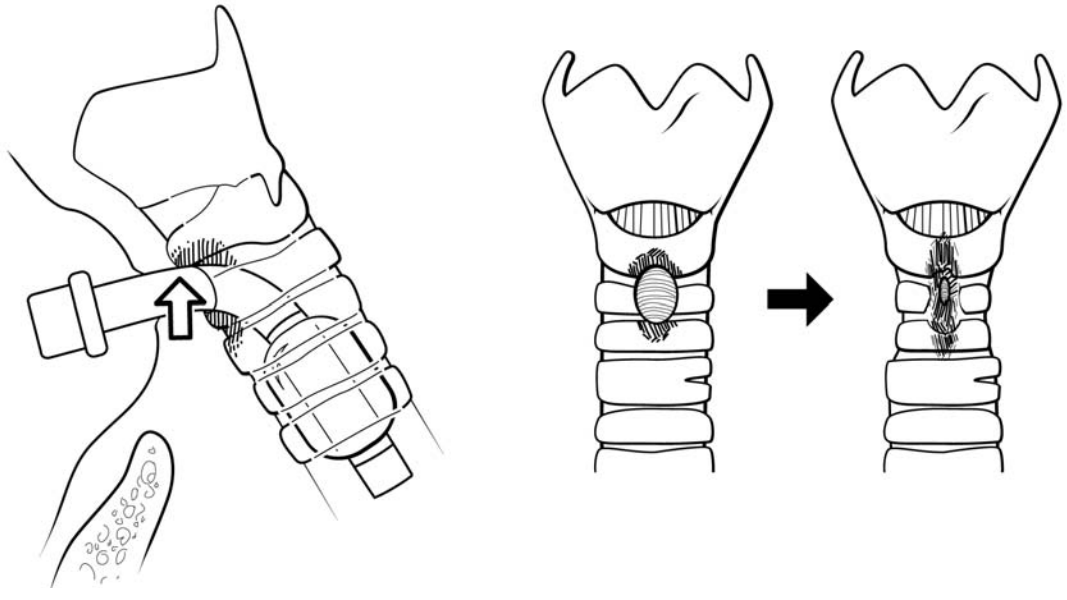

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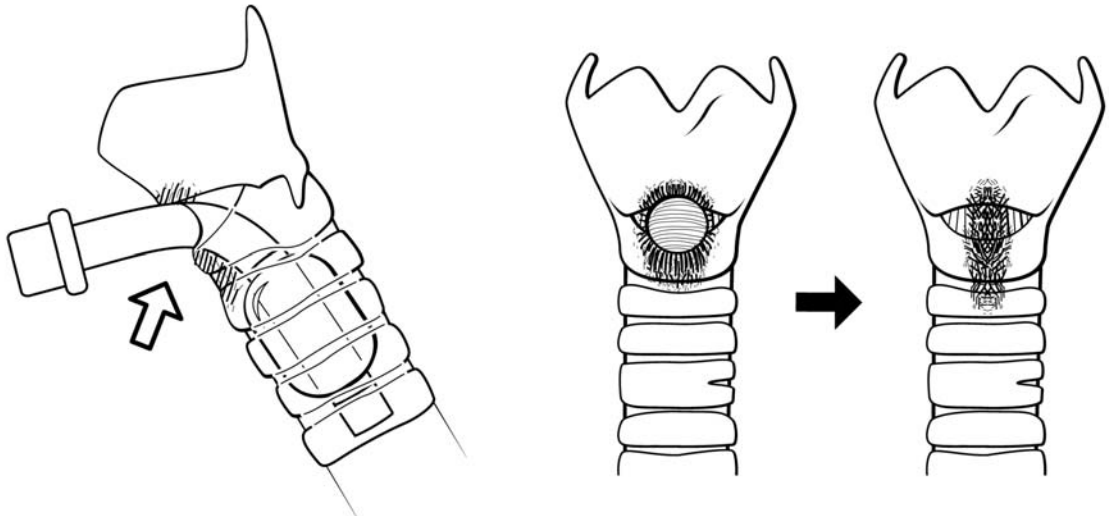


C

FIGURE 11-3 *Origin of a subglottic postintubation laryngotracheal stenosis.* A, *Circumferential cricoid erosion by the endotracheal tube.* B, *Proximal erosion of cricoid by a high tracheostomy in a kyphotic individual. Anterolateral stenosis results.* C, *Subglottic stenosis secondary to anterior cricoid erosion by the cricothyroidostomy tube residing in the cricothyroid membrane.*

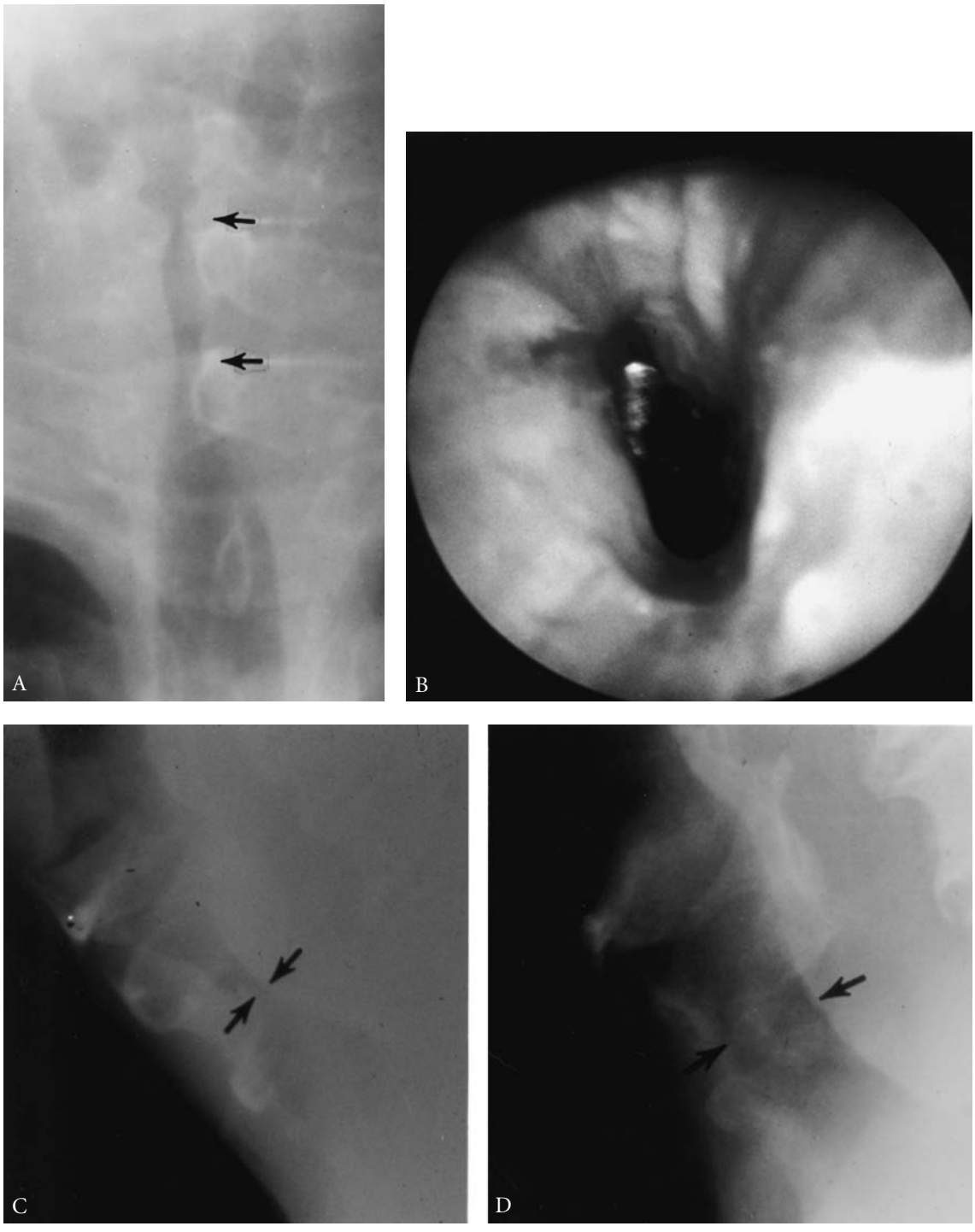


FIGURE **11-4** *Subglottic postintubation stenoses.* A, *Roentgenogram of the larynx and upper trachea. A slight 25-year-old man was ventilated with an endotracheal tube for head injury.* Upper arrow *indicates the vocal cord level.* Lower arrow *is at the cricoid level, also the point of maximum stenosis. The entire subglottic space is markedly narrowed.* B, *Laryngoscopic view just below the glottis in the same patient. The stenosis is severe, irregular, but circumferential. The glint of a tracheostomy tube is seen distally. Tracheostomy was necessary prior to laryngotracheal resection and reconstruction.* C, *Laryngotracheal stenosis following ventilation via "high tracheostomy," probably cricothyroidostomy, after repair of ruptured chordae tendineae in a 71-year-old man. Lateral cervical roentgenogram.* Arrows *indicate severe constriction of the lower larynx and trachea.* D, *Airway restoration following laryngotracheal reconstruction. Normal lumen is restored* (arrow). *See also Figure 28 (Color Plate 15).*

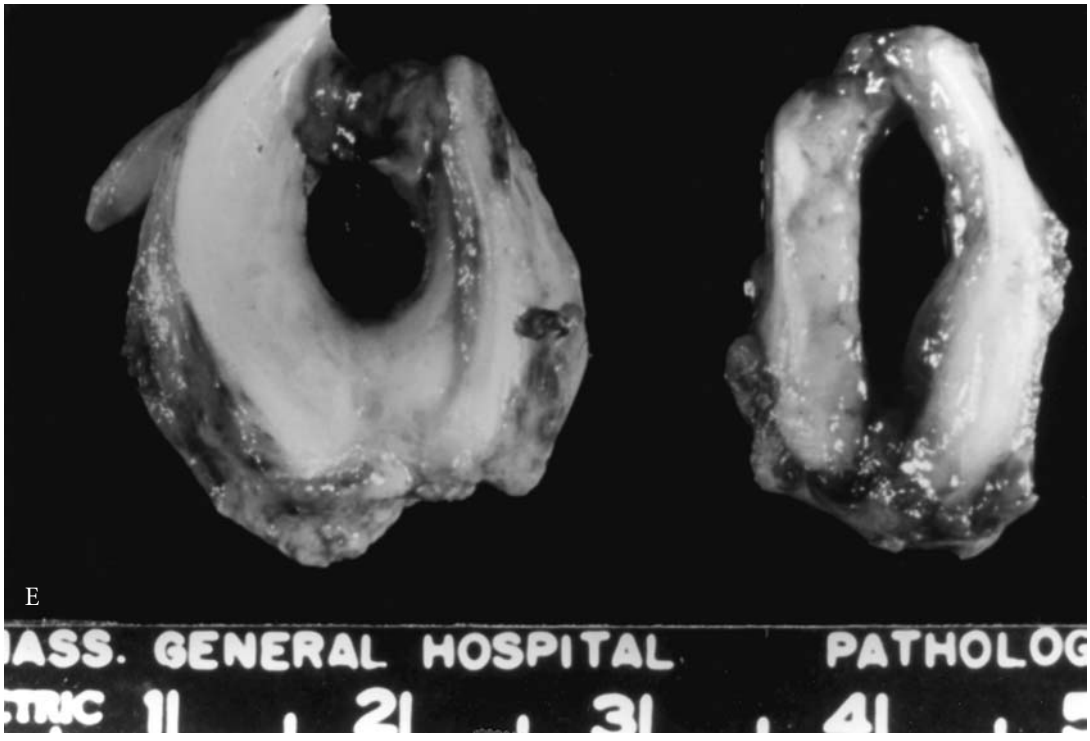


FIGURE **11-4** (CONTINUED) E, *Surgical specimens from a young woman who was ventilated after multiple injuries for a long period via an endotracheal (ET) tube, continued via a tracheostomy tube. Subglottic stenosis from ET tube injury is shown at left and result of the combined ET cuff and tracheal stomal injury is on the right.*

is less than the outer diameter of most commonly used tracheostomy tubes (10 to 12 mm).[10] Although most purely tracheal stomal stenoses can be corrected surgically on a first attempt with almost certain success, many laryngeal lesions are not correctable or only partially correctable from the outset. I, therefore, deplore use of elective cricothyroidostomy for ventilation. It should also be noted that concern about contaminating sternotomy incisions after cardiac surgery, by an immediately adjacent tracheostomy tube, may be obviated by using an endotracheal tube for ventilation for a few days. If tracheostomy is needed subsequently, tissue planes will have already sealed.

Preexisting neurologic deficits unrelated to the postintubation injury, particularly in patients who have suffered central nervous system trauma, may affect protective laryngeal reflexes and result in aspiration on deglutition. Such a deficit may preclude airway repair since tracheostomy may then be necessary to protect the airway and to clear secretions. Such functional deficits *must* be identified prior to a tracheal reconstruction.

Obstructive Lesions of the Trachea

Obstructive lesions of the trachea following intubation occur at four levels, depending on the source of injury. At each level, one or more distinct lesions may produce obstruction. The levels are 1) stomal, 2) the site where the inflatable cuff rested, 3) the segment between the stoma and the level of the cuff, and 4) the locus where a tip of the tube may impinge on the tracheal wall (see Figure 11-1).

Lesions at Stomal Level. Since tracheostomy creates a defect in the wall of the trachea, whether the opening is made by a vertical, horizontal, cruciate, or T incision, by excision of a segment or segments of carti-

lage, or by turning a flap, some scarring is inevitable during healing. Long after healing is complete, inspection, both bronchoscopically and radiologically, will demonstrate dimpling or deformity, an anterior shelf-like projection, or softness of the anterior wall at the site of prior tracheostomy. A surprising degree of asymptomatic narrowing may occur. Nearly 50% narrowing of the cross-sectional area of the trachea, or even more, is necessary before a sedentary person experiences dyspnea. Three stomal lesions, seen alone or in combination, may cause obstruction. These are 1) granuloma, 2) a posteriorly depressed flap of tracheal wall above the stoma, and 3) anterolateral stenosis.

Granulation tissue forms at the stoma before and during healing. *Granulomas* may be noted weeks or months after extubation. As healing progresses, ebullient granulation tissue may form on the inner surface of the trachea at the site of the stoma and become sufficiently bulky to obstruct the airway. Accumulation of this type of papillomatous granulation tissue often occurs in conjunction with deformity at the healing stomal site (see Figure 11-5). If a large granuloma is already present, immediate airway obstruction may follow removal of the tracheostomy tube.

The curve of the tracheostomy tube may produce a *depressed tracheal wall flap* just above the stoma. The tip of the flap may be thickened or granulomatous, but in most cases, this alone is insufficient to cause serious obstruction when the tube is withdrawn. When the tracheostomy tube has been in place for a long time, the upper flap may remain positioned posteriorly and produce partial or even subtotal obstruction (see Figure 11-1). This tissue may even become calcified prior to removal of a long-standing tracheostomy tube. We do not know whether this flap effect can be avoided by choice of incision for tracheostomy. It seems to occur most commonly in children who have had prolonged tracheostomy, perhaps because of the thinness and pliability of the juvenile trachea.

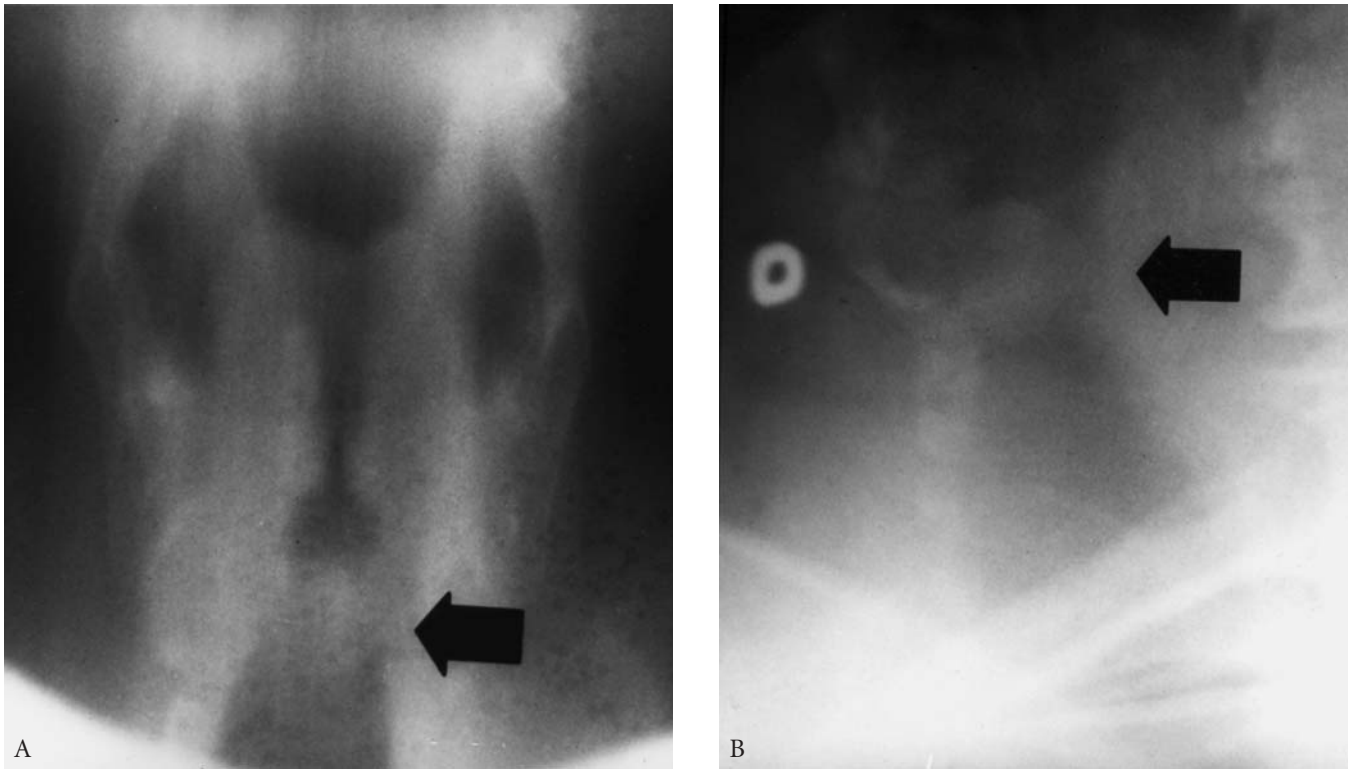


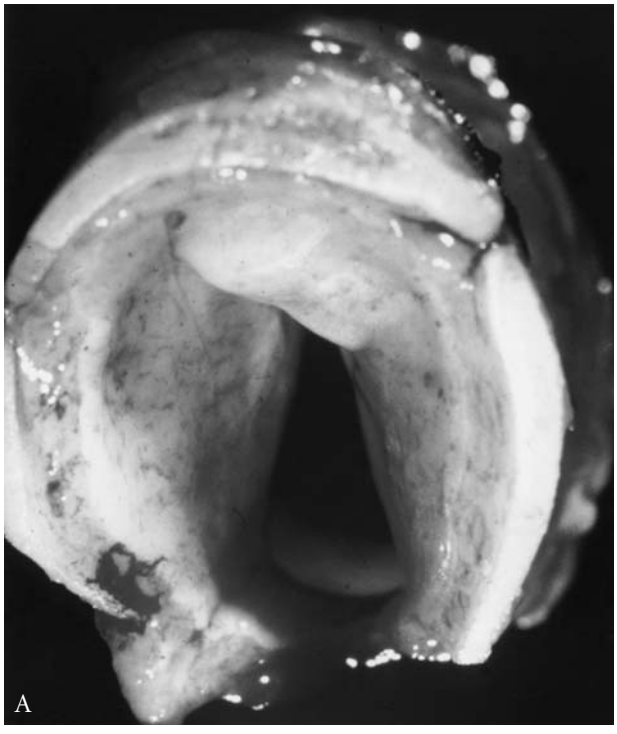
FIGURE **11-5** A, *Anteroposterior tomogram of the larynx showing stenosis and granuloma* (large arrow) *at the site of cricothyroidostomy. Note the proximity of the vocal cords just above the lesion.* B, *Lateral view showing narrowing of the airway, deformity, and large granuloma. The "O" marks the stomal site on the skin. Laryngotracheal resection and reconstruction was necessary.*

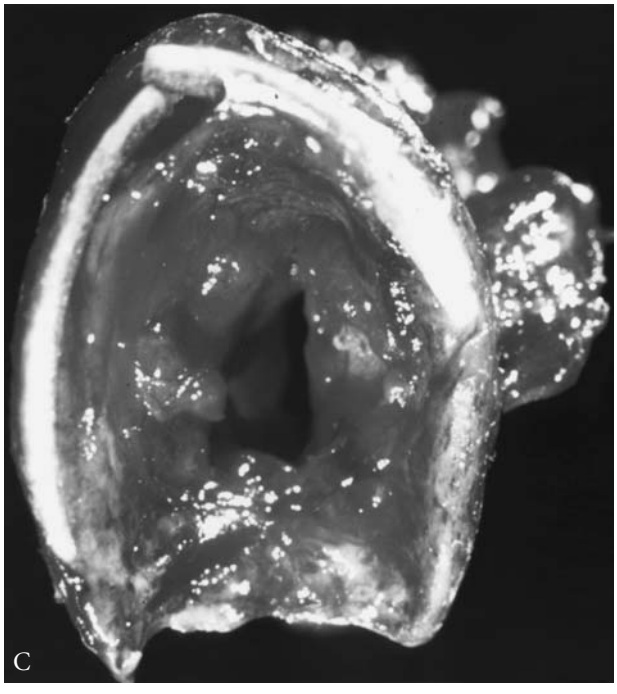
The most common lesion of significance at the stomal level is *anterolateral stenosis*. Following removal of the tracheostomy tube, the patient gradually develops obstructive symptoms. The patient is found bronchoscopically to have an A-shaped stricture with an apex anteriorly, which involves the anterolateral walls of the trachea (Figures 11-6*A*,*B* and Figures 24 and 25 [Color Plate 14]). The membranous wall is usually spared but irritative granulation tissue may be present posteriorly in some cases (Figure 11-6*C*). The membranous wall may be shortened by deformity of the lateral walls, which are pulled together by the anterior scar. Stomal stenosis results from cicatricial healing of what was or has become a large stomal defect (Figure 11-7). A number of factors appear to play an etiologic role. Occasionally, a surgeon makes a much too generous opening in the tracheal wall, failing to realize that loss of tracheal substance will ultimately be healed by the natural process of contraction of the scar. Tracheostomies probably erode toward the size of their inlying tracheostomy tube due to local pressure necrosis, no matter how the stoma is made. Any opening which is larger than this may only add to the destructive process. All tracheal stomas are inevitably contaminated bacterially. Although invasive sepsis is not frequent, bacterial activity may lead to further local tissue destruction. The most important factor is the weight of unsupported tubing which levers the tracheostomy tube against the margins of the stoma, producing pressure necrosis. Evidence to support this was the decrease in incidence of stomal stenosis in a single respiratory care unit, before and after the introduction of lightweight swivel connectors to tracheostomy tubes.[11] Careful suspension of tubes and connectors has essentially eliminated such lesions at Massachusetts General Hospital (MGH).

As noted earlier, an especially complex stomal lesion results if the cricoid cartilage is eroded by upward pressure of the tracheostomy tube. If the anterior cricoid cartilage loses its integrity, anterior subglottic laryngeal stenosis occurs in conjunction with upper tracheal stenosis. Even if the first tracheal ring has not been mistakenly divided during tracheostomy, a tube that impinges against it may erode through it and into the cricoid cartilage. This is most likely to occur in older patients with a degree of kyphosis, where hyperextension of the cervical spine fails to draw the larynx far above the sternal notch. Although the tracheostomy tube may be correctly placed at the level of the second tracheal ring, it may have to arch across the cervical tissues to reach the skin surface, exerting pressure against the cricoid (see Figure 11-3*B*). It is important to recognize the extent of such lesions prior to surgery, since the technique of repair of a purely *tracheal* stenosis is very different from that for subglottic *laryngotracheal* stenosis (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection," and Chapter 25, "Larygotracheal Reconstruction"). A comment on terminology is in order. "Subglottic" is often used to describe lesions anywhere from just below the vocal cords to the upper or even midtrachea. Surgical problems and prognosis are quite different at different levels. I prefer to describe a lesion that lies in the larynx between the vocal cords and the lower border of the cricoid cartilage as a *subglottic* lesion (intralaryngeal). If the upper border of the lesion lies just below the lower border of the cricoid cartilage, it is clearly an *upper tracheal* lesion, and *laryngotracheal* spans the subglottic level and upper trachea.

Infrastomal Obstructive Lesions. The principal infrastomal lesion that results from intubation for respiratory support is *tracheal stenosis at cuff level* or *cuff stenosis*. It is the most common lesion complicating modern respiratory care and it is clearest in the minds of most physicians (Figure 11-8 and Figures 26 and 27 [Color Plates 14 and 15]). The origin of stenosis at cuff level was obscure when the lesion was first recognized. It originates from circumferential erosion of the tracheal wall due to the pressure of the cuff and is common to all forms of access to the trachea; endotracheal tubes, tracheostomy tubes, or cricothyroidostomy tubes (Figure 11-9). In the extreme, a tracheo-innominate artery fistula can result if erosive pressure is maximum anteriorly, or a tracheoesophageal fistula if the erosion penetrates posteriorly. Conventional high-pressure cuffs formerly in use, whether on endotracheal tubes or tracheostomy tubes, exerted enormous pressures on the tracheal wall. They almost uniformly produced some degree of tracheal injury

within 48 hours of placement. The depth and severity of damage is roughly, but not uniformly, related to the duration of exposure to pressure injury. The key etiological factor in the production of stenosis is pressure necrosis caused by the cuff, compressing the tracheal mucosa and, later, the deeper structures of the tracheal wall. The principal evidence supporting these conclusions has been derived from autopsy study of patients who received ventilatory support,[12,13] from prospective studies by direct visualization of the tracheae of patients receiving ventilatory assistance,[11,14] and from experimental production of identical





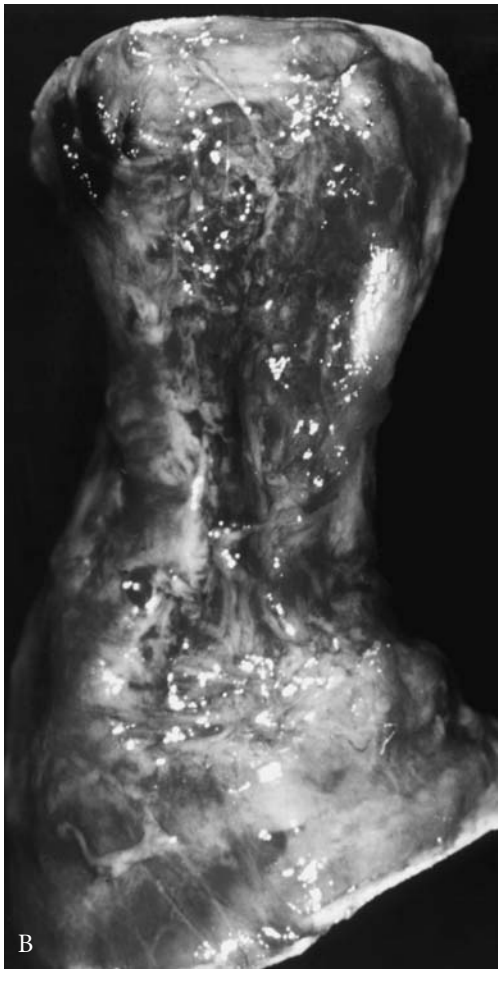


FIGURE **11-6** A, *Surgical specimen of stomal stenosis showing the typical triangular lumen with apex anterior. A granuloma is also present where the stoma lay. The membranous wall is smooth.* B, *Longitudinal view of the same specimen demonstrating that the tracheal walls are pulled together by the cicatricial closure of the stomal defect.* C, *Another specimen showing stomal stenosis with active inflammatory granulation tissue everywhere, including the membranous wall of the trachea. An endotracheal tube had been wedged into the stenosis on several occasions. Also, see Figures 24 and 25 (Color Plate 14).*

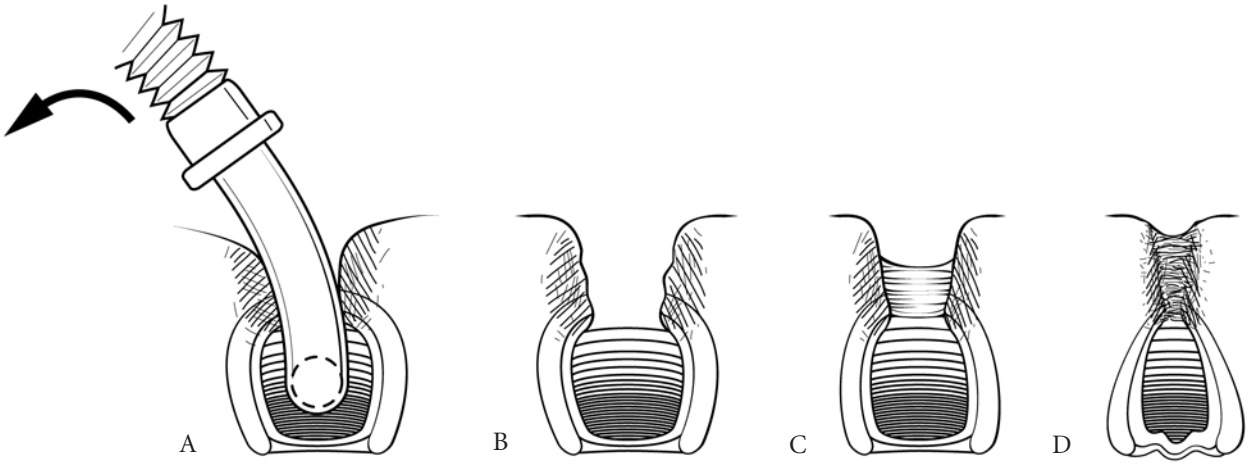


FIGURE **11-7** *Evolution of stomal stenosis. Cross-sectional diagrams of the trachea at the stomal site.* A, *The tracheostomy tube may be leveraged against the stomal margins, causing their erosion.* B, *The resulting large stoma exhibits granulations and inflammation at its margins.* C, *Contraction of scar tissue forming across the defect pulls the tracheal stomal margins to the midline.* D, *The result is an A-shaped lumen. The posterior wall is also often shortened. Scar tissue is present, in most anteriorly only.*

lesions.[15] Correlation of these observations with surgically removed specimens further supports this thesis.[16] Final confirmation is that removal of excessive cuff pressure has eliminated the lesions in a very large number of patients at risk over a long period of time, at MGH.

Cooper and Grillo examined the tracheae of 30 patients who died while receiving ventilatory assistance through cuffed tracheostomy tubes as well as 4 additional patients who had received such assistance through cuffed endotracheal tubes only for short periods of time.[12] Both metal and plastic tracheostomy tubes and plastic and rubber cuffs had been used. At that time, all cuffs were of designs that produced high intracuff pressures. A consistent pattern of tracheal damage was observed, with the major damage located at the site of the cuff. The period of mechanical ventilation and the degrees of damage generally correlated. Superficial tracheitis and fibrin deposits appeared within 48 hours of placement of the tube. Small, shallow ulcerations were then seen, overlying the cartilaginous rings. With time, the size of the ulcers increased and cartilages were exposed. The inflammatory process spread laterally and deeply, followed by fragmentation of cartilage (Figure 11-10). The tracheal wall in many cases bulged where the balloon was located. These lesions usually began approximately 1.5 cm below the inferior margin of the tracheostomy stoma and extended downward for a length of about 2.5 cm, that is, the location of the cuff. Usually, between two to four cartilages were completely bared in time. Eventually, segments of cartilage sloughed and, in advanced cases, the balloon site was completely devoid of cartilages. Severe damage was observed between 10 days to 2 weeks after placement of the cuff. Additional ulcerations were occasionally seen, corresponding to the tip of a tracheostomy tube below the cuff injury. Changes that occurred at cuff sites from endotracheal tubes compared closely to those seen from cuffs on tracheostomy tubes with similar duration of intubation. These lesions were located more proximally in the trachea, since the cuff of an endotracheal tube is sited more proximally than that from a tracheostomy tube (see Figure 11-9).

Microscopic examination elaborated the progressive changes that were found grossly (Figure 11-11). Acute inflammation and fibrin appeared early. Microscopic ulceration followed, overlying the cartilaginous rings where the mucosa had been compressed between the balloon and the underlying cartilage (see Figure 11-11*A*). As the ulcers deepened, the surfaces of the cartilages were bared, and inflammatory degeneration ensued. Inflammatory cells infiltrated beneath the cartilages (see Figure 11-11*B*). Fragmentation of

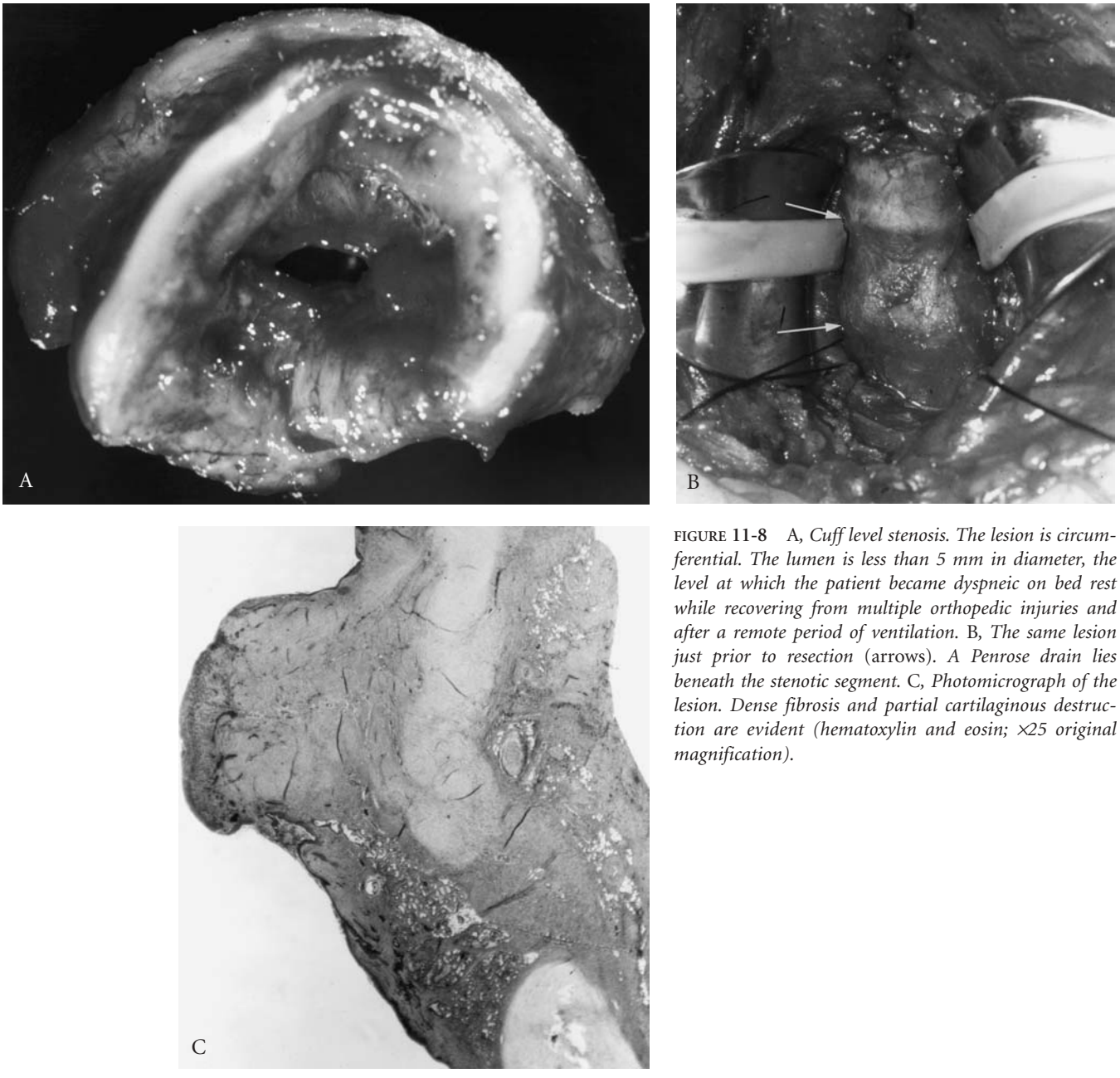


FIGURE **11-8** A, *Cuff level stenosis. The lesion is circumferential. The lumen is less than 5 mm in diameter, the level at which the patient became dyspneic on bed rest while recovering from multiple orthopedic injuries and after a remote period of ventilation.* B, *The same lesion just prior to resection* (arrows). *A Penrose drain lies beneath the stenotic segment.* C, *Photomicrograph of the lesion. Dense fibrosis and partial cartilaginous destruction are evident (hematoxylin and eosin; ×25 original magnification).*

cartilage occurred next (see Figure 11-11*C*) and, finally, only an ulcerated bed remained where the cartilage had sloughed (see Figure 11-11*D*). In some cases, the tracheal wall was totally replaced with granulation tissue as repair competed with erosion (see Figure 11-11*E*). At the margins where the epithelium remained, squamous metaplasia was evident. Although the membranous wall sometimes escaped changes as severe as those in the noncompliant cartilaginous portion of the tracheal wall, severe erosive inflammatory changes were produced nonetheless. The circumferential lesion reflected circumferential pressure injury. In several cases, the membranous wall was reduced to paper thinness, and there were inflammatory changes in the adjacent esophageal wall. Complete fistulization occurred in other specimens. Florange and colleagues reported similar pathologic findings.[13]

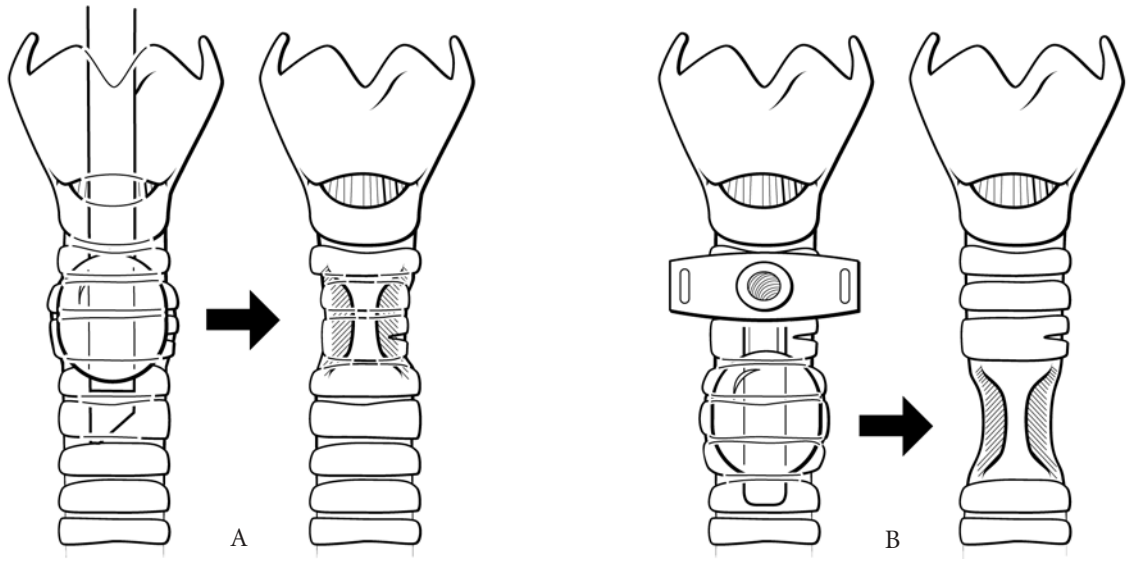


FIGURE **11-9** *Evolution of a cuff stenosis. Excessively high pressure exerted on the trachea produces circumferential erosion, which heals by cicatrization of granulation tissue. The resulting scar is circumferential.* A*, Cuff lesion from an endotracheal tube.* B*, Cuff lesion from tracheostomy tubes. The lesion is usually lower than that from an endotracheal tube.*

These changes were compared with a group of surgically resected specimens of cuff stenosis, where a circumferential ring of dense fibrous tissue resulted at 1 to 3.5 cm below the tracheostomy site. In these resected specimens, the residual effective airway often measured 2 to 5 mm in diameter. Externally, the area of stenosis was usually demonstrable, frequently hourglass in shape, but without external indication of the extreme narrowness of the minimal internal airway (see Figures 11-8*A*,*B*). In longstanding cases, metaplastic squamous epithelium was present. In most, the scar tissue of the stenotic ring remained unepithelialized. In some, residual pieces of tracheal cartilage were present in a greater or lesser degree (Figure 11-12). In fully advanced cases, not even vestigial remnants of tracheal architecture were identifiable. Patients with cuff stenoses following endotracheal intubation often had marked cicatricial stenosis, but relatively intact outer cartilages, presumably because the length of exposure to pressure had been too brief to necrose the cartilages completely. A striking finding was that *all* patients with the then-conventional cuffs in place had notable changes. These findings were confirmed in a prospective study by Andrews and Pearson, who examined the tracheal wall endoscopically through the stoma at the time of removal of the tracheostomy tube.[11] We made similar endoscopic observations in a study of the comparative effects of standard cuffs and an experimental low-pressure cuff.[14]

Many etiologic possibilities for these lesions had earlier been suggested, including the influence of sepsis, the fact that many patients had periods of hypotension during their illness which could impair circulation in the compressed mucosa, damage by toxic materials in the tubes, and cuffs and from gas sterilization.[17] Shelly and colleagues questioned the effect of systemic hypotension, on the basis of animal experiments.[18] Experimental reproduction of the lesions helped to clarify these questions.[15] Murphy and colleagues attempted to reproduce the lesions in dogs, but were able to do so only with the combination of cuff and tracheostomy.[19] We placed short segments of Portex endotracheal tubes with cuffs perorally into the tracheae of dogs, and fixed them with percutaneous wires.[15] Tracheostomy was avoided to minimize infection. The tubes were clean but not gas sterilized. The cuff was inflated just sufficiently to provide a seal at 25 cm of water ventilatory pressure, and this pressure was then maintained throughout the experiment. Destructive lesions were uniformly produced within 1 week (Figures 11-13*A*,*B*). Removal of the tube was

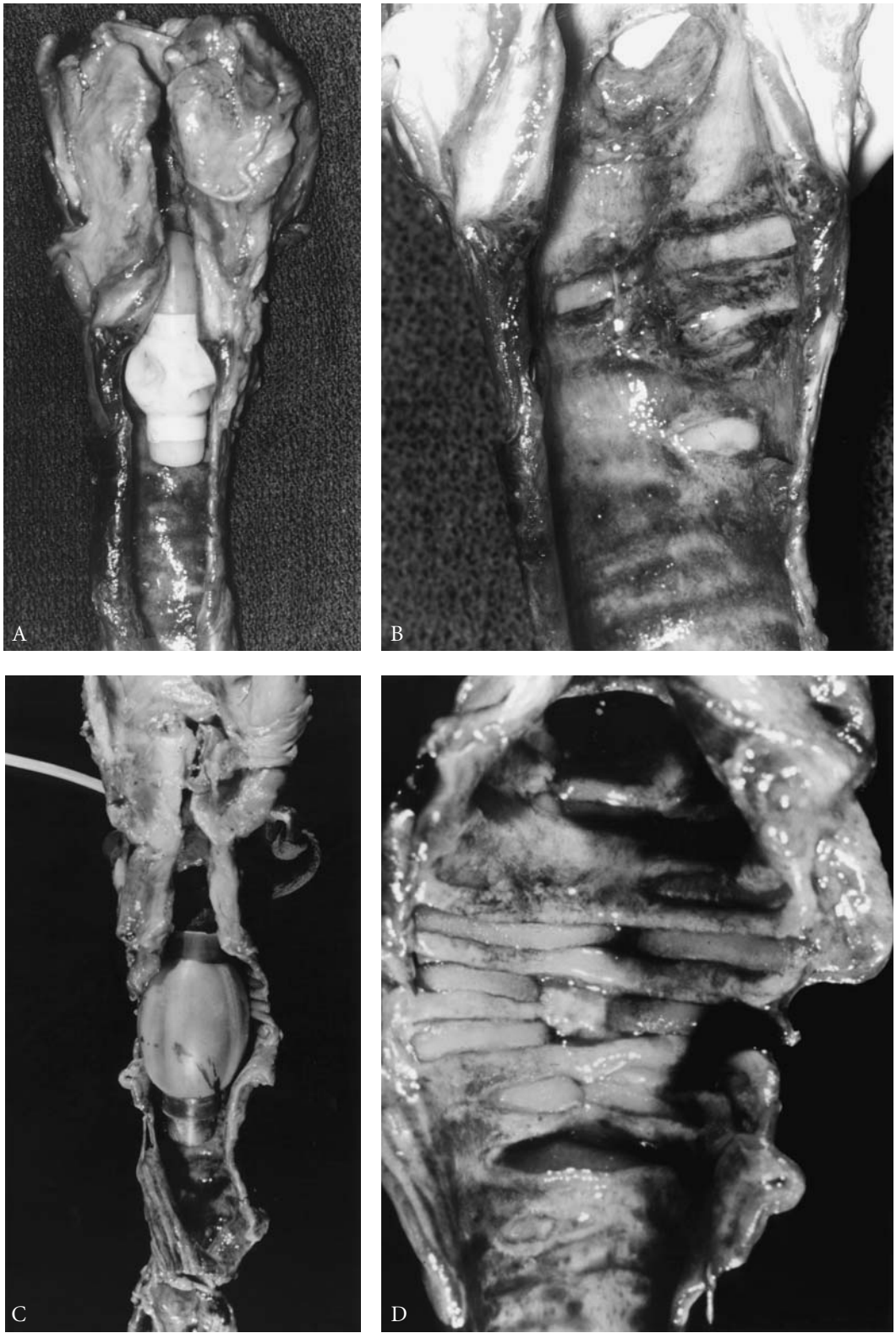


FIGURE **11-10** *Postmortem tracheal specimens of patients ventilated with high-pressure cuffs then in use.* A, *Trachea opened posteriorly. Portex tube and cuff are evident. Ventilation was for 19 days. The patient was age 55 years.* B, *Ulceration and loss of cartilage at the cuff site.* C, *Trachea of a 69-year-old woman, ventilated for 16 days. Metal tracheostomy tube with latex cuff are evident.* D, *Mucosa is destroyed at the cuff site and cartilages are bared and fragmented.*

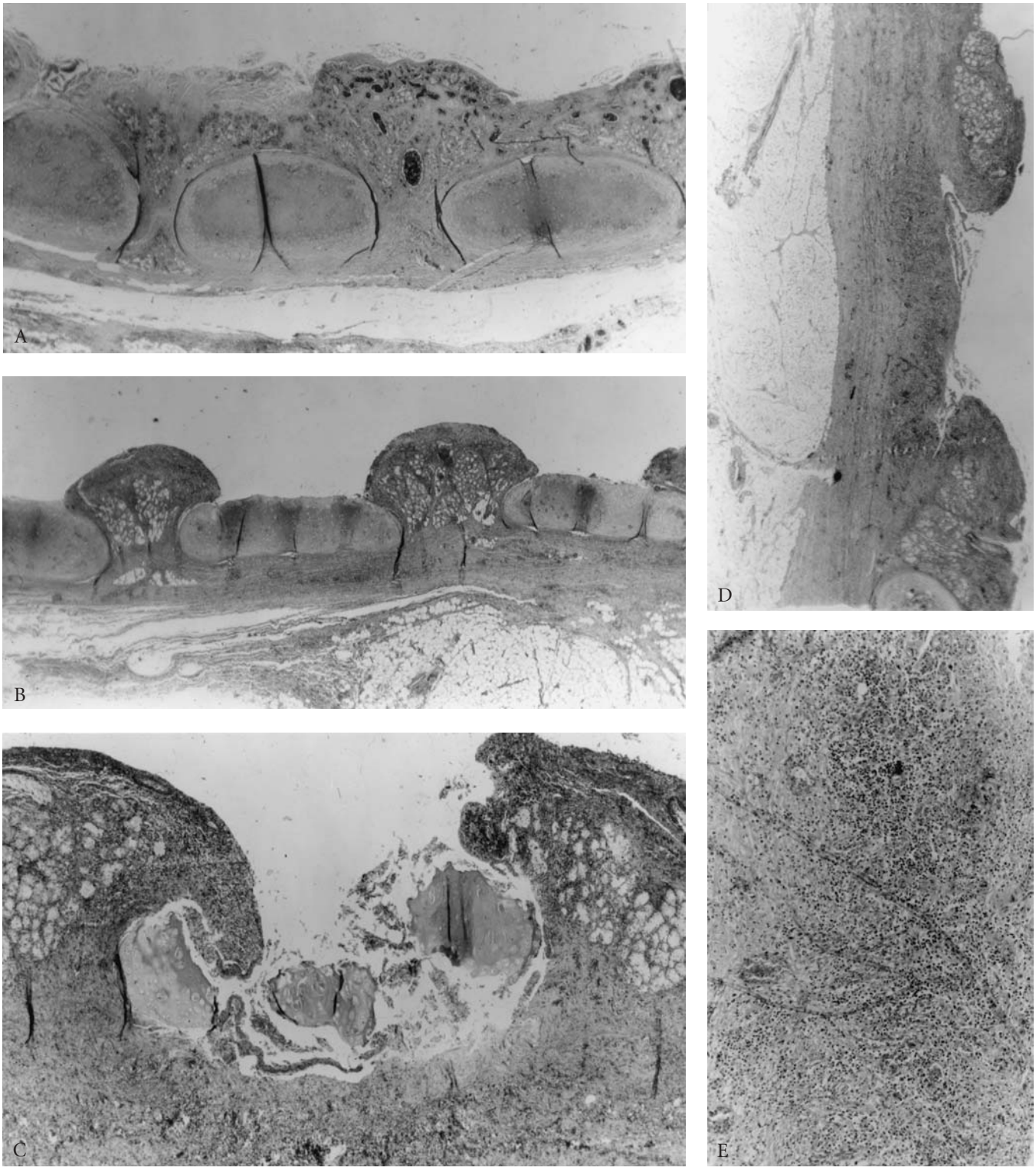


FIGURE **11-11** *Photomicrographs from postmortem specimens of tracheae injured by ventilation using high-pressure cuffs. Tracheal lumen is above in* A, B, *and* C. *The lumen is at the right in* D *and* E. A, *Erosion of mucosa over cartilages due to cuff compression. Submucosal inflammation.* B, *Cartilages now bared by progress of mucosal and submucosal necrosis. Lamina propria has been thickened by inflammation.* C, *Necrosis of cartilage follows with increased inflammatory intensity in surrounding tissues.* D, *Total destruction of cartilages occurs. Inflammation extends deeply into tracheal wall.* E, *The tracheal wall is now essentially replaced with inflammatory tissue and beginning reparative response of granulation tissue (hematoxylin and eosin; ×25 original magnification).*

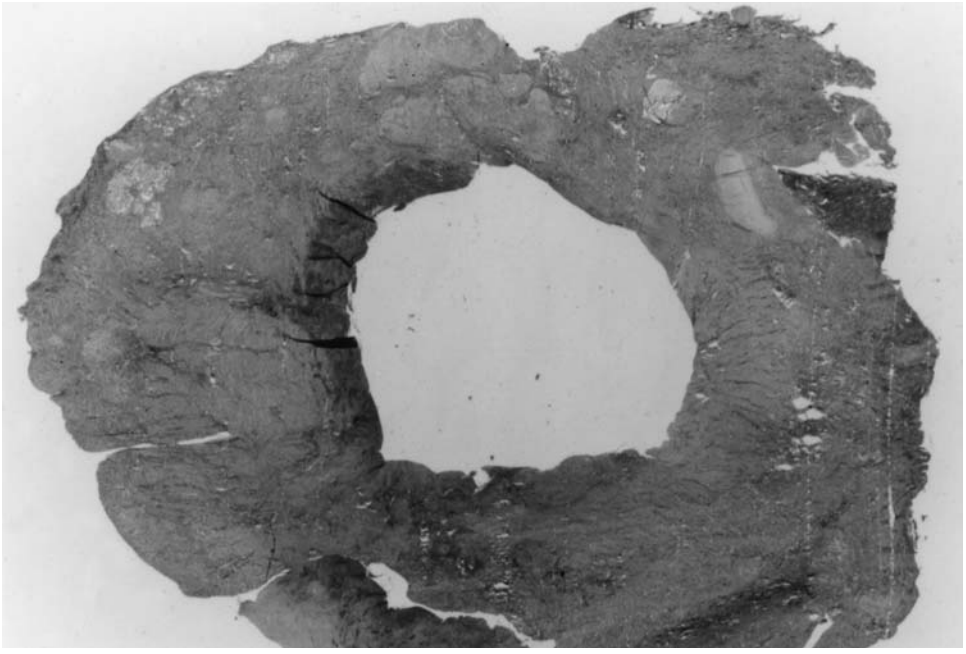


FIGURE 11-12 *Photomicrograph of transverse section of a fully developed cuff stenosis. A fragment of degenerated cartilage is essentially the only immediately recognizable remnant of the tracheal architecture. Otherwise, a ring of scar tissue has replaced the trachea, and its contraction leaves only a very reduced lumen. Mucosa is lacking (hematoxylin and eosin; ×11 original magnification).*

followed by cicatricial stenosis and airway obstruction (Figure 11-13*C*). Although the experimental and clinical evidence do not rule out possible additive contributions by other factors mentioned as well as others unknown, the principal common denominator appears to be necrosing pressure on tissue. We saw *no* cuff stenoses in over 5 years and in many hundreds of patients, following the design and introduction of a latex low-pressure cuff, although no other factors changed during this period. Since then, despite the necessary but careful use of plastic low-pressure cuffs, since latex cuffs are not available, no cuff stenoses have been produced at MGH in thousands of ventilated patients.

Varying degrees of tracheitis occur in the *segment between the level of the stoma and the level of the cuff*. The segment is usually short, but it lies in close proximity to two areas of damaging influences. In many cases, secretions puddle above the cuff, despite intermittent deflation. Heavy bacterial colonization is routine around tracheostomies. Varying degrees of gross and microscopic inflammation are seen in this segment of trachea. The cartilages may be thinned and inflamed while the mucosa, although inflamed, is intact. At operation, the tracheal wall at this point may be markedly inflamed and its architecture partly destroyed. This becomes evident once part of the trachea is detached from surrounding supporting tissues.

Tracheal malacia occurs in this segment and is demonstrable fluoroscopically or bronchoscopically. Such changes can be of great importance in planning surgical excision of cuff stenoses, since the segment of trachea requiring removal may be almost double the length apparent in preoperative static images of a cuff stenosis. In a few patients, the area of cuff damage itself may be primarily malacic rather than firmly stenotic, producing valve-like obstruction on deep breathing or coughing (Figures 11-14*A*,*B*). Routine tracheal x-rays may show only slight or no deformity at cuff level. Functional obstruction becomes evident only when deliberately sought for fluoroscopically or during an awake flexible bronchoscopy. In these patients, cartilaginous rings are absent, and the fibrous wall is covered with squamous metaplastic epithelium (Figure 11-14*C*). The evolution of malacia rather than fibrous stenosis at cuff level has not been explained.

Lesions may overlap and it may be difficult to ascertain the precise etiology, especially if a stenosis appears late. In obese or aged individuals, for example, the cuff may reside immediately within the stoma, with confluence of stomal and cuff injuries. Multiple tracheostomies also serve to confuse the issue, since records of prior treatment are often imprecise. Stenosis resulting from prior endotracheal intubation may be lost sight of after a series of therapeutic tracheostomies and laser treatments.

Occasionally, *trachiectasis* occurs. In a few patients with persistent respiratory failure from chronic lung disease, who are managed for lengthy periods of time with conventional equipment, the requirement for volume of air to seal the cuff gradually increases. Generally, high ventilatory pressures are needed. The trachea in one patient required a balloon volume of approximately 200 cc for a seal.

Granuloma formation at a site of ulceration by the tip of a tracheostomy tube can also cause obstruction, although rarely. With the older type of high-pressure cuffs that expanded eccentrically, the tip of the tracheostomy tube could easily be angulated against the tracheal wall. A tracheostomy tube with a 90° angle accentuates this possibility. This also happened in children where no cuff was used at all, where slight angulation of a tracheostomy tube levered the tip against the tracheal wall (Figure 11-15). After the tube is removed, an ulcer may heal, with profuse connective tissue formation, producing an inflammatory granuloma. The incidence of such lesions in children has diminished remarkably with the development and use of improved pediatric tracheostomy tubes (see Chapter 10, "Tracheostomy: Uses, Varieties, Complications").

Granulation tissue may form around the lower end of a tracheostomy tube while a patient is still on a ventilator. This occurs most often in patients who have long been on mechanical assistance, who have

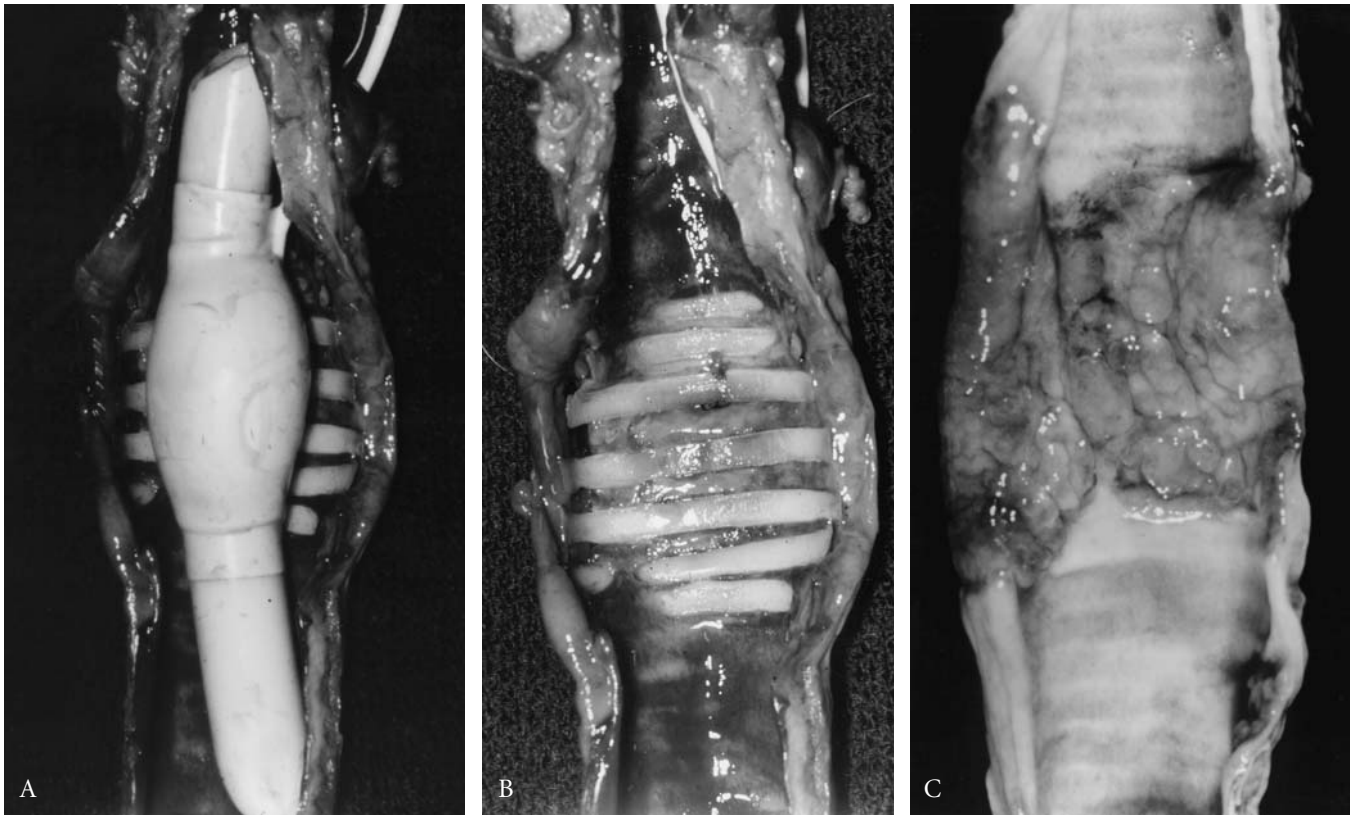


FIGURE **11-13** *Experimental production of cuff stenosis in dogs.* A, *Segment of the endotracheal tube with high-pressure (standard) cuff after 7 days of exposure to the seal at standard ventilatory pressure.* B, *Mucosal destruction has occurred, cartilages are bare, and the trachea is distended.* C, *In another specimen, 13 days of exposure has produced copious granulation tissue, replacing the normal tracheal structure.*

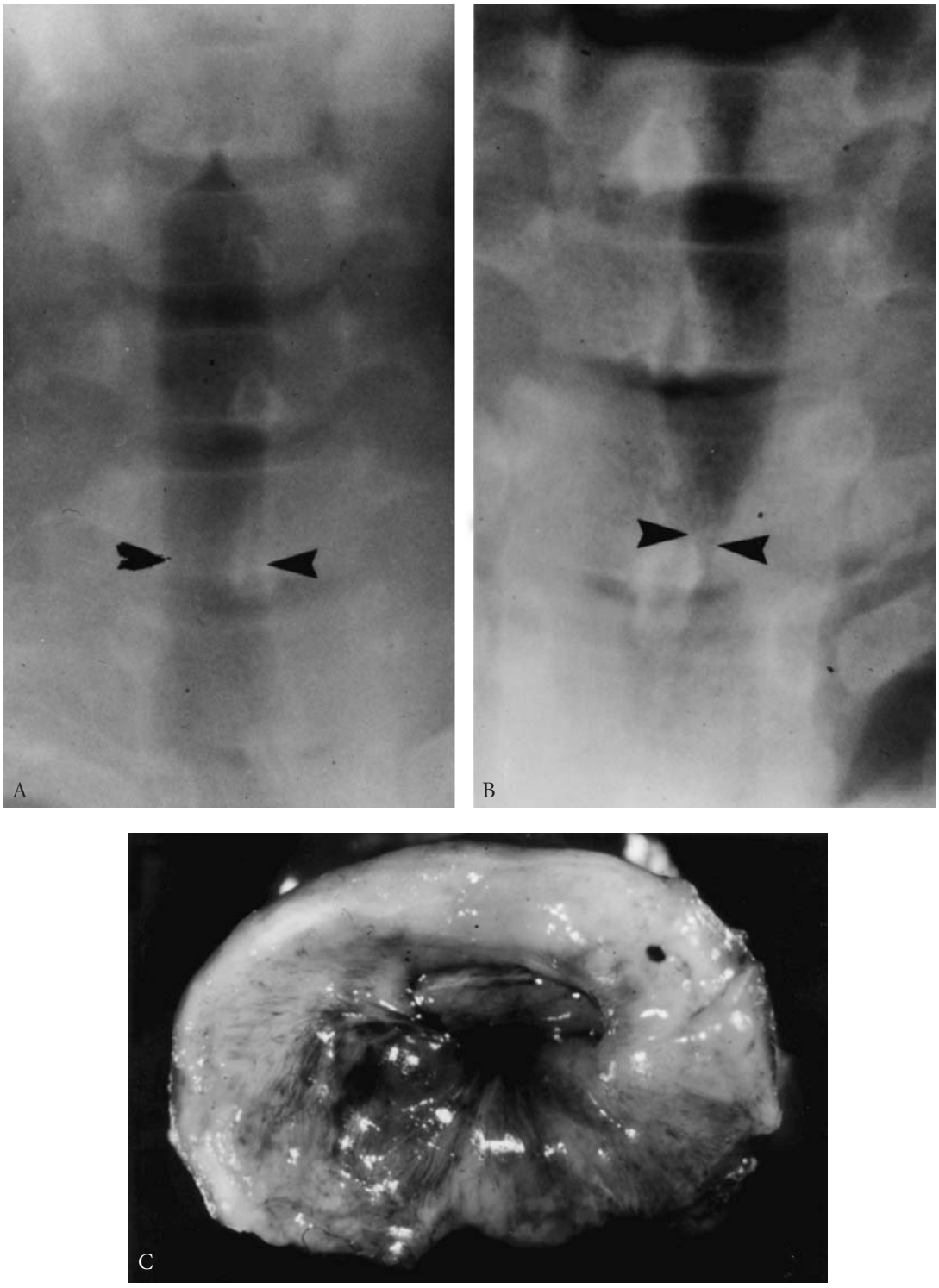


FIGURE **11-14** *Diagnostic images from a 38-year-old woman with attacks of dyspnea and changes of voice worsening over a year, following emergency tracheostomy and ventilation for obstructive laryngitis.* A, *"Spot film" from fluoroscopy demonstrating an apparently normal upper tracheal diameter* (arrows). *The adducted vocal cords and subglottic larynx are clearly defined above. Symptoms were thought to be of psychiatric origin because of similar static x-rays of the trachea elsewhere.* B, *Fluoroscopic picture of the segmental malacic tracheal collapse* (arrows) *on cough. Resection of the damaged trachea fully relieved her complaints.* C, *Surgical specimen from a 70-year-old woman who was twice ventilated for long periods for respiratory failure. Circumferential cuff lesion was principally malacic, with some fibrosis.*

diffuse tracheitis, and who may have significant injury at cuff level. Bronchoscopic removal of granulations provides only transient relief. A longer tube or one with a different angle may be inserted, but the danger remains that the process will extend distally, ultimately to the carina, unless the patient can be weaned. Granulations in a supracarinal location present a special hazard, since a tube may slip back only a few millimeters and obstruction from the granulations may slowly form below the tip of the tube while the physician has an illusion of safety.

Incidence

It has been difficult at any time to establish the incidence of lesions that follow tracheal intubation. In 1967, 17% of a vulnerable population from a respiratory care unit at the Massachusetts General Hospital developed clinical evidence of upper airway obstruction. This selected population consisted of survivors of relatively prolonged treatments of the most severe respiratory failures and the study occurred in the era preceding the development of low-pressure cuffs. Large numbers of patients who had received respiratory support for lesser problems, often through an endotracheal tube, were not included. The figure compared quite closely with the range then described from other institutions: 20% from the Toronto General Hospital with a similar population, 12% of a group of cardiac surgical patients from Mount Sinai Hospital in New York, and 16% of a group of 50 patients from Australia. Harley attempted to establish the incidence of laryngotracheal stenosis following tracheostomy and assisted ventilation, by analyzing reported series.[20] The range was from 0 to 22%, with an average of 3.27% for 3,793 tracheostomies.

Introduction of low-pressure cuffs of varying efficacy and closer attention to avoidance of stomal erosion greatly diminished the occurrence of injury in succeeding years. Following introduction of our prototype of the low-pressure latex cuff, cuff stenosis vanished at Massachusetts General Hospital. Careful attention to the use of currently available plastic large-volume, low-pressure cuffs has continued this record. Stomal strictures were reduced to well under 1% (see "Prevention of Postintubation Lesions" below). In recent years, further attention to tube support has eliminated these lesions as well. Unfortu-

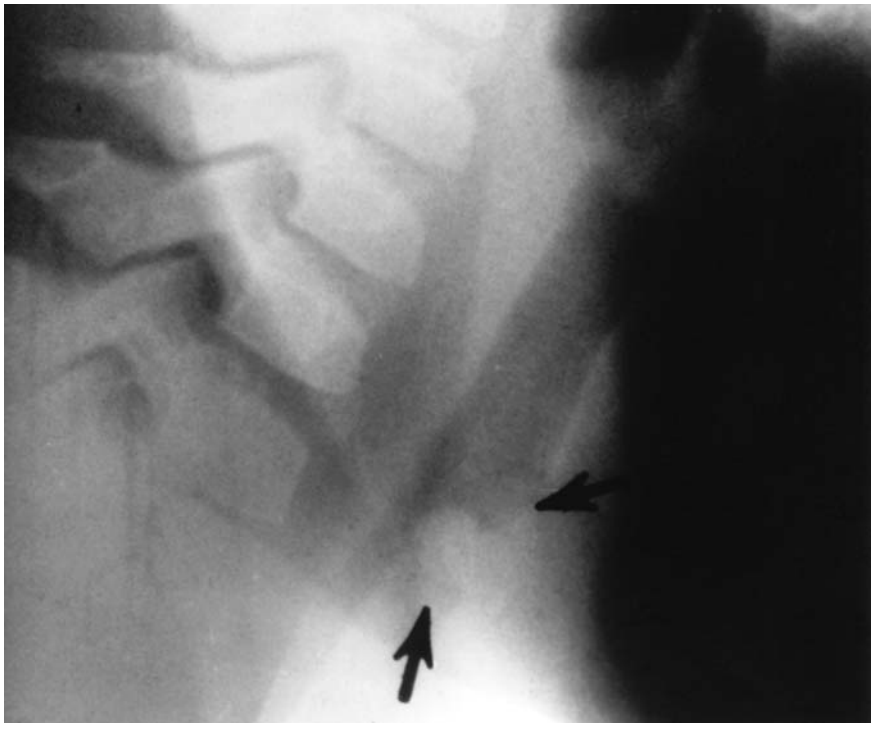


FIGURE **11-15** *Obstructing granuloma in a child, caused by erosion by the tip of a tracheostomy tube. The angle of many adult tubes is inappropriate for pediatric use. Bronchoscopic removal of the granuloma sufficed.*

nately, this is not universally true, for reasons described later. Postintubation lesions of all types continue to be produced worldwide, and they continue to be the leading indication for tracheal surgery.

Tracheoesophageal fistula (TEF) and *tracheo-innominate arterial fistula* resulting from intubation and ventilation are described in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula," and Chapter 13, "Tracheal Fistula to Brachiocephalic Artery."

Prevention of Postintubation Lesions

Prevention of all postintubation lesions of the trachea is unlikely until totally new methods of respiratory support are developed that do not require a foreign body in the trachea as part of the support system. Noninvasive techniques remain in the future.[21] Enough has been learned about the origin of postintubation lesions so that their frequency has been markedly reduced and further improvement is to be expected.

Stenosis at Stomal Site

Stomal stenosis may be minimized or avoided altogether by attention to details, performance, and management. First, the surgeon should make no larger an opening for the tracheostomy tube than is necessary. The tube should not be too large for the particular patient. Its curve should be appropriate. In order to minimize the size of the stoma and destruction of tracheal tissue, I prefer a simple linear vertical incision in the trachea (see Chapter 22, "Tracheostomy, Minitracheostomy, and Closure of Persistent Stoma"). The procedure is done in an operating room with aseptic technique. Bacteria are always present in the tracheal lumen and further colonization will occur after tube placement, despite exquisite postoperative care. *Staphylococcus aureus* and *Pseudomonas aeruginosa* are the most common. However, invasive sepsis may be limited by scrupulous postoperative care. The tracheostomy tube should be well seated and fastened securely to the patient's neck. Avoidance of *leverage on the tracheostomy tube* is most important. The weight of connecting tubing and adapters, transmitted through the tracheostomy tube against the tracheal wall, causes erosion of the stomal margin. Long-term exposure to ventilation and other factors such as diabetes and corticosteroids are additional likely agents. Lightweight swivel adapters attached to the tracheostomy tube move in multiple planes and are connected by light, flexible corrugated tubing to the ventilator. It has been hypothesized that accordion tubing helps to avoid transmission of the respirator's thrust to the stomal edges, and to the cuff itself, but conclusive experimental data is required. The ventilating connecting tubes are in turn suspended from supports. Clinical results support this protocol overall. Progressive careful attention to the points noted has eliminated stomal lesions at Massachusetts General Hospital.

Stenosis at Cuff Level

Evidence pointing to pressure necrosis as the most important etiologic agent has been presented. Adriani and Phillips noted that variables such as cuff site, materials, and tracheal shape affected intracuff pressure.[22] They found that intracuff pressure was not a direct index of pressure exerted on the tracheal wall. Knowlson and Bassett found that small increments over the minimal occlusive volume required to effect a seal in patients with a conventional endotracheal cuff, with 20 cm of water inspiratory pressure, caused a rapid rise in the pressure exerted on the tracheal mucosa.[23] This exceeded arterial capillary pressure, especially against the anterior tracheal wall. Carroll and colleagues correlated intracuff pressures with pressures exerted on the tracheal wall by a variety of cuffs and found the relationship to be generally proportional.[24] They set forth as criteria for ideal cuffs that these should have "large sealing areas, inflate evenly, and center the tube within the tracheal lumen;. . . . have large residual volumes requiring small additional volumes for 'seal,' low tracheal wall sealing pressure with overinflation." Lomholt described a large-volume Teflon cuff with an attached trap, intended to maintain a constant cuff inflation pressure.[25]

Cooper and Grillo proposed the use of a large-volume, low-pressure cuff which would conform to the irregular shape of the trachea when inflated, rather than establish a seal by expanding circumferentially and deforming an elliptical trachea to the shape of the cuff (Figure 11-16).[15] The prototype of such a cuff was tested in experimental canine preparations. Conventional cuffs produced erosive and stenotic lesions (see Figure 11-13), whereas in equivalent time periods, the prototype large-volume, low-pressure cuffs resulted in no injuries other than slight submucosal inflammation (Figure 11-17). A latex cuff suitable for clinical use was designed, and when tested in humans for its sealing characteristics, it was found to require a tenth of the pressure required by a conventional high-pressure Rusch cuff.[14] The cuff, roughly cylindrical in shape, measured approximately 2.4 cm in length and 3.0 cm in diameter when inflated to a pressure of 1 cm of water. At this point, the latex wall was unstretched, and the total volume of air that the cuff accepted prior to stretching was 12 cc (Figures 11-18*A*,*B*). The cross-sectional area of the filled but undistended cuff was greater than that of most adult tracheas, and could therefore fill out the configuration of the normal ovoid tracheal shape without applying stretch to the wall of the cuff itself. Initially, these cuffs were placed on conventional metal Jackson tracheostomy tubes, with care taken to prevent slippage.

Randomized clinical trials following tests for safety compared the cuff's performance in 25 patients, with 20 having standard cuffs.[14] "Blind" endoscopic evaluation of damage to the tracheal wall on a scale of 0

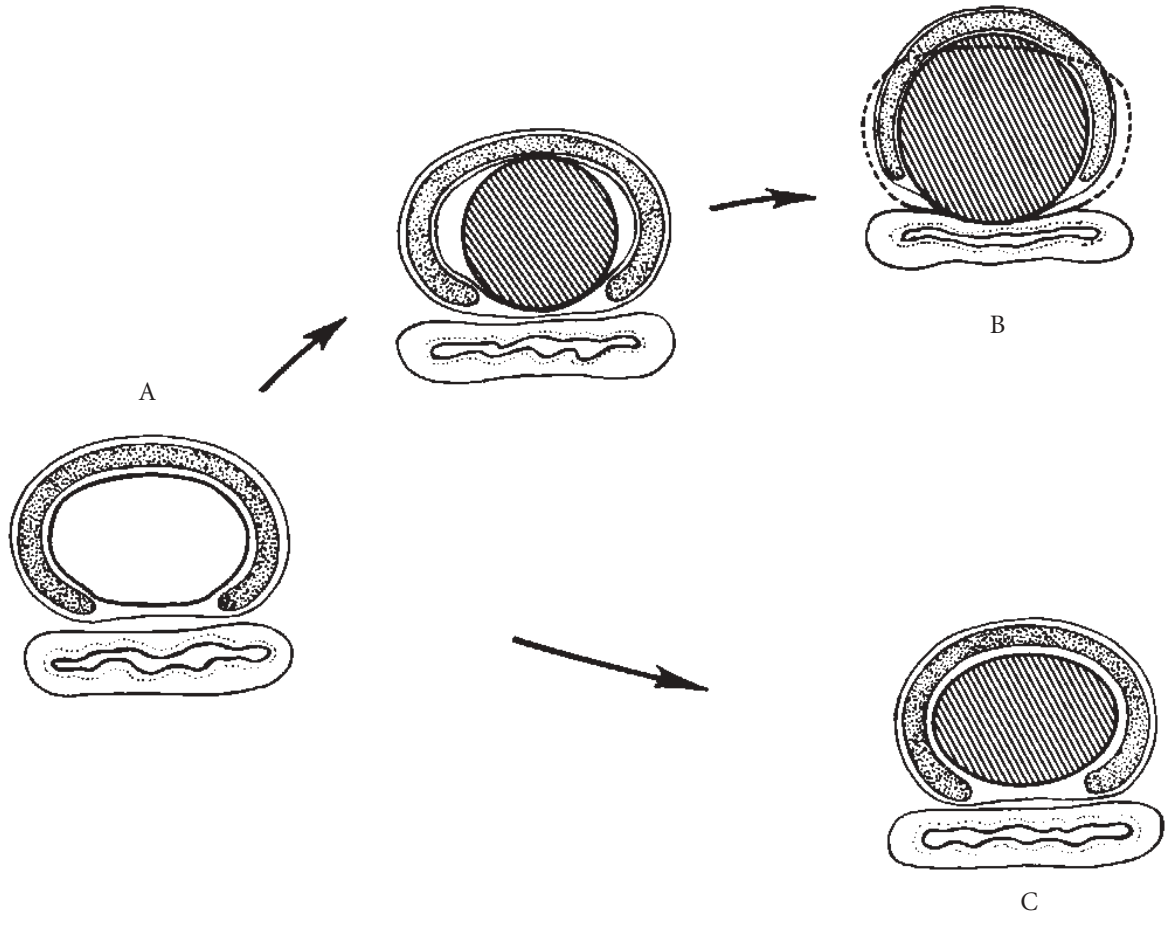


FIGURE **11-16** *Diagram of a tracheal seal, attained by a high-pressure cuff versus a large-volume, low-pressure cuff.* A, *Cross section of the trachea and esophagus.* B, *The low-volume cuff is necessarily inflated to a high pressure to occlude the irregularly shaped tracheal lumen.* C, *The large-volume cuff expands to occlude the lumen, conforming to the shape of the lumen at low inflation pressure.*

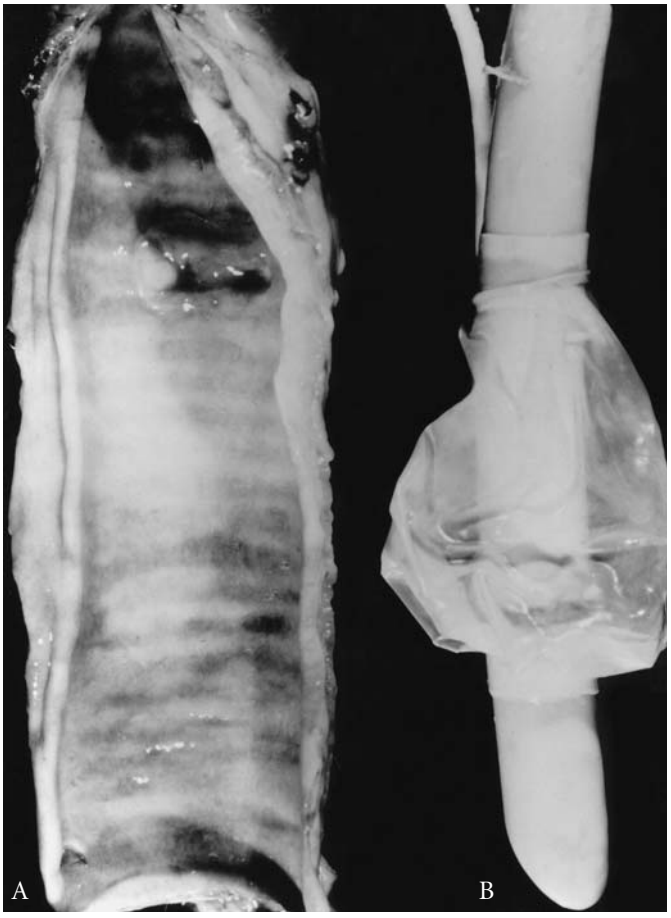


FIGURE **11-17** *Experimental use of large-volume, low-pressure cuff in dogs.* A, *After 2 weeks of seal at the same ventilatory pressure used with high-pressure cuffs, only minimal mucosal inflammation results. Compare with Figures 11-13*B,C. B, *Experimental cuff, deflated.*

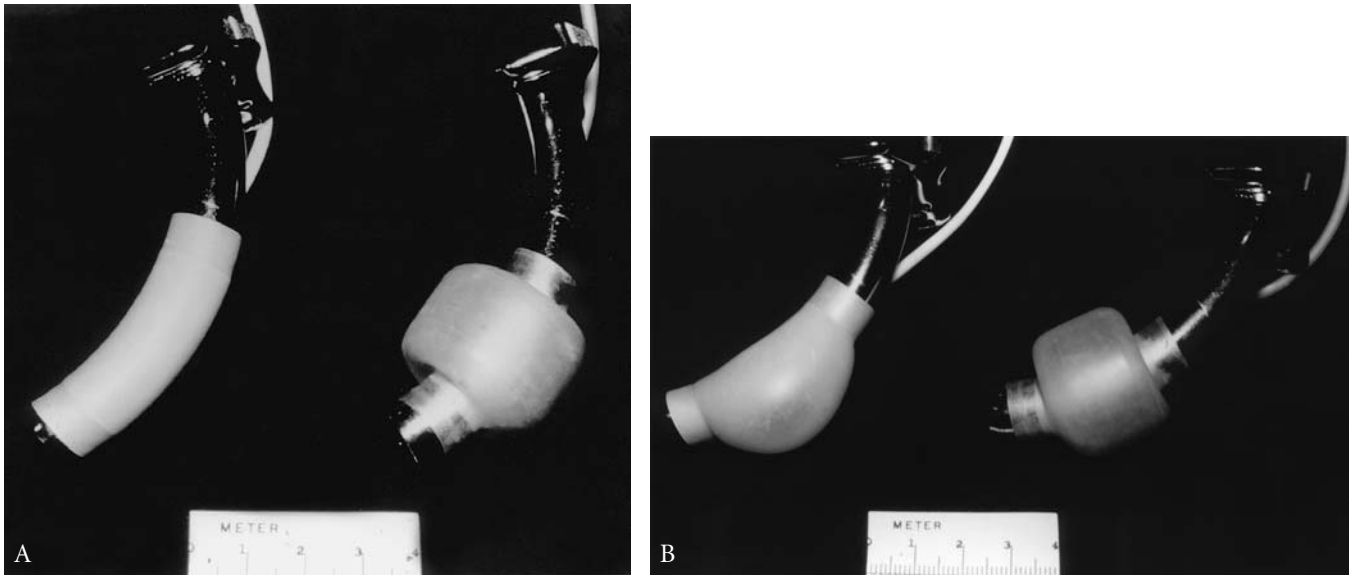


FIGURE **11-18** A, *Rusch standard cuff (1971) at the left and experimental latex cuff on the right, mounted on Jackson metal tracheostomy tubes, both in a "resting" state at resting volume. The experimental cuff is deflated for insertion.* B, *Standard cuff (left) is inflated with 8 cc of air and has high intracuff pressure, and is asymmetric and quite rigid. The large-volume, low-pressure cuff is undistended with a similar volume of air, has no intracuff pressure, and is soft and symmetrical.*

to 4 was made immediately after deflation of the cuff as weaning began. Patients with the new cuffs had an average rating of 1.3 (median 1.0) in comparison with an average rating of 2.6 (median 2.5) for those with the standard cuffs. All patients in the minimum injury group had the new cuff in place. Few with the new cuff were in a category showing more serious damage. In contrast, the bulk of patients with the standard cuff fell into groups with progressively more severe damage, many lying in the ranges which were predictably likely to go on to clinical stenosis (Figure 11-19). The average intracuff pressure developed in the experimental cuff was 33 mm Hg compared to an average intracuff pressure of 270 mm Hg in the standard cuff. During the period of development of the cuff, following initial indicative experiments, Geffin and Pontoppidan proposed the interim use of prestretched Portex cuffs to approximate these conditions.[26] Despite the limitations of this method, the incidence of cuff strictures in our respiratory care unit dropped noticeably with the prestretched cuffs, and totally disappeared following routine use of large-volume latex cuffs.

Despite this clear enunciation of desirable standards for sealing cuffs for ventilation, followed by years of favorable experience, clinically available equipment still varies in characteristics. Latex is almost indefinitely extensible so that damaging pressures are not developed. However, the short shelf life of latex and the cost of attaching it to plastic tubes led to its abandonment. Large-volume cuffs are now generally available, but are made of relatively inextensible plastic materials. When the resting volume of the fully inflated, but unstretched, cuff is exceeded by only a few cc of overinflation, the lack of extensibility of the material leads to a rapid climb in intracuff pressure. The margin of safety is thus reduced with the relatively nonextensible material now used to fabricate cuffs. Ching and Nealon, and Ching and colleagues analyzed comparative characteristics of cuffs in several studies, confirming these findings (Figure 11-20).[27,28] More extensible plastic would further improve safety. Large-volume cuffs should not be inflated beyond the minimum pressure that is adequate to provide ventilation without leakage. Personnel must also understand that cuffs have to be reinflated with care after routine deflation. Otherwise, the inflation volume, and consequently the pressure, creeps upward. *It is principally the failure of proper management of cuff volume that continues to produce cuff stenoses today*.

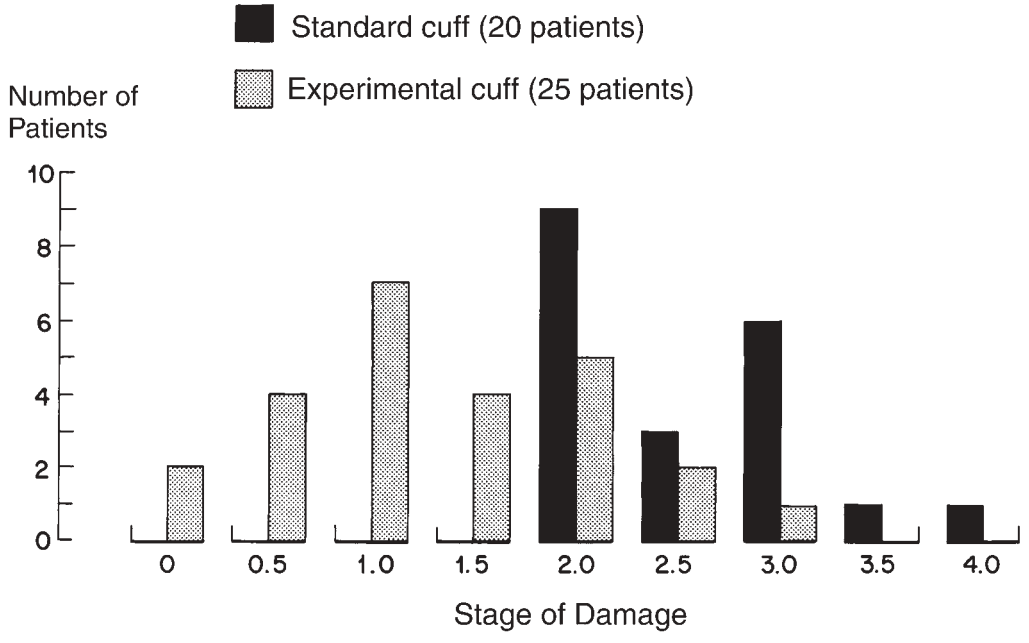


FIGURE **11-19** *Damage to trachea from standard low-volume, high-pressure cuffs versus large-volume, low-pressure tracheostomy tube cuffs. Sixty-eight percent of patients with the experimental cuff showed no exposed cartilage (rating less than 2.0). No patient with standard cuff was in this category. Reproduced with permission from Grillo HC et al.*[14]

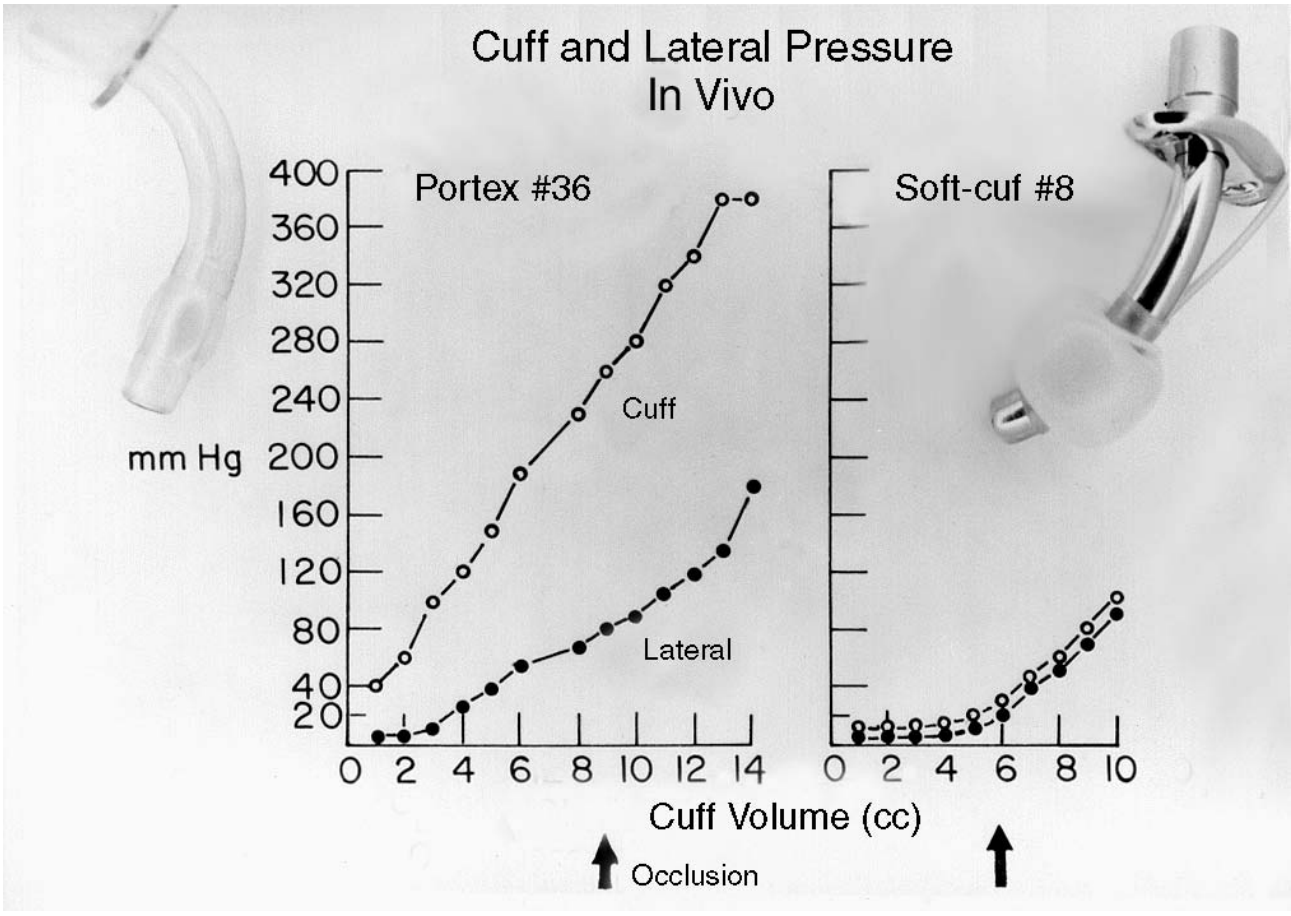


FIGURE **11-20** *Comparison of intracuff and lateral tracheal wall pressures engendered by a then-standard Portex plastic cuff on the left and the newly developed latex large-volume cuff on the right. The occlusion volume for the trachea is indicated by an* arrow *in each case. If volume is exceeded, high intracuff and lateral tracheal wall pressures do not result in the large-volume cuff. (Illustration courtesy of Dr. NPH Ching and Dr. TF Nealon Jr.)*

Seals other than air-filled balloons have been proposed, including flexible discs and a compressible synthetic sponge in an outer bag, which fills by expansion of the sponge matrix.[29] A low-pressure pilot balloon was designed to relieve pressure in excess of a sealing level of 25 cm of water.[30] It was also proposed to reduce the time of exposure of an area of trachea to pressure, by alternate inflation of double cuffs in series. This proved to be unsatisfactory and, if anything, produced longer stenoses. Intermittent cuff inflation cycled to inspiration was also tried, with no instances of damage seen.[31] None of these methods gained currency perhaps because of their relative complexity. Furthermore, they provide less protection against aspiration than sealing cuffs. Substitution of high-volume flow respirators has been suggested. Although it is possible to maintain children without sealing cuffs, adults with poor compliance and severe degrees of respiratory failure cannot currently be managed without a tracheal seal.

Thus, we see that stomal strictures may be reduced to a minimum and perhaps eliminated. Information is available to eliminate cuff strictures. No cuff strictures have been produced at MGH since the initial introduction of a large-volume cuff. The problems that remain are the dissemination of information on management, coordination of manufactured equipment, and evolution of better materials. Prop-

er use of large-volume, low-pressure cuffs and cessation of prolonged use of stiff nasogastric tubes should prevent tracheoesophageal fistulae (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula). Tracheo-innominate artery fistulae from cuff injury would also disappear. Those at the stomal level can be avoided by choosing the proper level for tracheostomy (see Chapter 13, "Tracheal Fistula to Brachiocephalic Artery").

Clinical Presentation and Diagnosis

Clinical Characteristics

The majority of patients with postintubation tracheal lesions present clinically with obstruction. Principal manifestations are 1) progressive dyspnea, 2) wheezing and stridor, and 3) intermittent obstruction with retention of secretions. Pneumonitis or frank pneumonia may occur unilaterally or bilaterally.

As the airway narrows, *dyspnea on effort* is noted first. This appears initially with marked effort, depending on the respiratory reserve of the patient. In time, dyspnea appears with less exertion. Many patients with benign tracheal stenosis remain sedentary or bedridden for a long time due to their original illnesses. Severe degrees of obstruction may therefore occur before clinical symptoms become obvious. In a patient on bed rest, the airway may contract to a diameter of 5 or 6 mm before symptoms are recognized. Other patients with a severe but fixed stenosis that is no longer progressing are dyspneic, only as they become more active during recovery from illnesses such as polyneuritis. Slow progression of stenosis may lessen a patient's awareness that a change in airway function is occurring. In most cases, however, the rate of closure is relatively swift. Sometimes, symptoms follow immediately or within days after removal of a tracheostomy tube. Obstruction may also occur from granulation tissue while the patient is still tracheostomized. With the most severe degrees of airway obstruction, the patient may be unable to lie down or complete a sentence without gasping for breath.

As the airway narrows, *wheezing* occurs, followed by frank *stridor*. Classically, an upper tracheal obstruction outside of the thorax will present with severe inspiratory stridor, and a low intrathoracic stenosis with expiratory wheezing. Usually, stridor may be produced in either phase on deep breathing with effort. Later, the wheeze is present at rest. A marked inspiratory high-pitched sound may be heard across the room even when the patient is quietly seated. When stridor becomes audible at rest, a high degree of obstruction is usually present, with the airway measuring less than 6 or 7 mm in diameter. At this point, action is urgently demanded. The stridor is elicited by having the patient breathe in slowly and deeply through an open mouth, and then forcing the breath out rapidly, with mouth still open. An attempt to inspire deeply and suddenly will often lead to severe coughing. Auscultation over the trachea and upper chest will further identify stridor. It may be heightened by the forced expiratory maneuver described. Although some of these sounds are transmitted peripherally, they are more remote on auscultation over the peripheral lung fields. In contrast, wheezing due to asthma and bronchitis is peripheral and is not heard maximally over the trachea itself.

As the airway narrows it becomes increasingly *difficult to clear secretions*. Plugs of mucous accumulate, occasioning transient episodes of worsened obstruction. The patient may cough violently in an effort to clear the airway, becoming plethoric and then cyanotic. Episodes of transient obstruction usually signal a marked degree of airway obstruction with an aperture that may measure less than 5 mm in diameter. The fact that such an episode may be cleared with chest physiotherapy or suctioning does not lessen the gravity of the warning. A subsequent obstructive episode may well be fatal.

Pneumonitis and *pneumonia* occur in the presence of tracheal obstruction. Most commonly, however, the lung fields remain clear on the x-ray. This is the reason why so many patients with severe obstructive tracheal lesions fail to be diagnosed promptly. Assumption is made that the disease must be bronchi-

tis or asthma, and too many patients have been treated over long periods for "adult onset asthma," with an illusion of response. Some have been placed on high doses of prednisone prior to referral. The basic diagnostic rule to be remembered is that *any patient who presents with dyspnea on effort, wheezing, or episodes of airway obstruction, and who has been intubated and ventilated at any time in the recent past, must be considered to have organic upper airway obstruction until proved otherwise.* The corollary should be added that if a history of intubation is lacking, tumor or other obstructing disease of the upper airway should be excluded. With this rule in mind, diagnosis is not difficult, especially with the ready availability of the flexible bronchoscope.

Analysis of a group of our early patients with stenosis following tracheostomy for respiratory care showed an equal gender distribution and a mean age of 47 years (range 16 to 79 years). The causes of the original respiratory failure were diverse, including chest trauma, drug ingestion, myasthenia gravis, polyneuritis, head injury, pickwickian syndrome, pneumonia, and following cardiac surgery. Tracheostomy had been required in these patients for periods ranging from 2 to 119 days, with a mean duration of 42 days. Several different types of tracheostomy tubes had been used in this group of patients, but metal tubes (silver or stainless steel) with rubber cuffs, or plastic tubes with plastic cuffs, predominated in this era prior to the development of low-pressure cuffs. In many cases, at least two kinds of tube and cuff had been used. In most, an endotracheal tube had been employed for a period up to 4 or 5 days prior to tracheostomy. In 3 patients, the inflatable cuffs had been used without ventilator assistance to prevent aspiration of foreign material into the lungs. In the remaining patients, the cuffs were used in association with artificial ventilation. Ventilation was administered for 3 to 112 days with a mean duration of 33 days. Although many of the patients had been treated at other hospitals prior to referral for correction of their stenosis, the care of patients who developed stenosis at the Massachusetts General Hospital included hourly deflation and reinflation with no more air than was required to effect a seal at peak airway pressure. All procedures and suctioning were done with aseptic precautions.

In patients in whom precise information was available on the time interval between extubation and the onset of symptoms, it was found that 18 had symptoms within 30 days and 24 had symptoms within 90 days of extubation. One patient was seen 18 months following extubation. In some patients, the symptoms were evident within a few days after removal of the tube. A 30- to 90-day interval, however, meant that many of these patients were discharged from the hospital prior to development of symptoms. This undoubtedly accounts for the fact that so many were treated for "adult onset asthma" or other vague diagnoses. Clinical history is a most important element in diagnosis.

A later analysis of 156 postintubation lesions treated in the decade between 1965 and 1975 showed that 14 patients had never had tracheostomy, but had developed stenosis from the cuff on endotracheal tubes. Several had been intubated for periods less than 48 hours and one case was for less than 36 hours. Of the lesions related to tracheostomy tubes, 72 were due to cuff stenoses, 51 to stomal strictures, 9 had had both lesions present, and the etiology was uncertain in 1 other case. Two patients had segments that demonstrated malacia only; both were located in areas of cuff damage. There were 6 tracheoesophageal fistulae due to cuffs and 1 fistula to the brachiocephalic artery due to a cuff erosion.

A review carried out in 1995 showed an increasing ratio of stomal over cuff lesions in patients with tracheostomy tubes (from 1:1.4 in the decade of 1965–1975 to 2.3:1 in the two decades from 1975–1995), probably reflecting the introduction of low-pressure cuffs and their correct usage.[32] Increasing use of endotracheal tube ventilation is suggested by an increasing ratio of cuff stenoses from endotracheal tubes over those from tracheostomy tubes (1:5.1 in 1965–1975 compared to 1.8:1 in 1975–1995). Increased incidence in laryngotracheal subglottic stenosis, chiefly the result of ventilation with endotracheal tubes, also reflects this changing preference in chosen route of administration of mechanical ventilation.

Diagnosis

When the presence of stenosis is suspected on the basis of the history, symptoms, and signs, appropriate *imaging studies* will quickly define the location and extent of the lesion. Once a patient begins to have stridor and shortness of breath on minimal exertion, the lesion may progress rapidly toward complete obstruction. A small plug of mucous or edema may close the airway completely. Such patients must be hospitalized at once, watched carefully in a respiratory care unit, and studies completed urgently. Conventional radiologic images are often more useful than a computed tomography (CT) scan or CT-derived reconstructed images (see Chapter 4, "Imaging the Larynx and Trachea") (Figures 11-21*A–E*). Contrast medium is not necessary and may cause some difficulty in patients who have high degrees of obstruction. Fluoroscopy is essential as an added means of assessing glottic function and to detect tracheomalacia (see Figures 11-14*A*,*B*). It is extremely important to define all tracheal lesions and to analyze the status of the larynx precisely, since concurrent lesions do occur. The treatment of specific lesions varies and a total plan should be based on complete information. In particular, it is important that an effective functional laryngeal airway be assured before tracheal reconstruction is undertaken. It may be necessary to temporize even with a tracheostomy while an affected larynx is initially repaired. Synchronous repair of the larynx and trachea is possible but can add risk.[33] High tracheal lesions must be differentiated from those that also involve the subglottic larynx.[8] An early surgical failure in our series was due to failure to recognize a significant degree of proximal malacia in addition to stenosis. Another failure was due to a lack of appreciation of glottic inadequacy, in a patient with a complex tracheal stenosis.

Informative tracheal x-rays should be available prior to endoscopy. Findings on bronchoscopy may be somewhat confusing to a surgeon who has not seen many of these lesions. X-rays serve as a road map for the bronchoscopist. Also, malacia may not be recognized bronchoscopically, especially under general anesthesia, without prior warning of its presence from fluoroscopic examination. Diagnostic *bronchoscopy* is discussed in Chapter 5, "Diagnostic Endoscopy." It is my firm belief that *every patient with a diagnosis of "adult onset asthma" should be examined bronchoscopically* to rule out organic obstruction.

Management and Results

Urgent Management

A number of patients will have been treated previously for acute respiratory arrest in a hospital from which they were referred. Others will arrive with high degrees of obstruction in very tenuous status. Respiratory decompensation can occur very soon after admission.

If a hospitalized patient develops nearly complete obstruction of the airway after failure to recognize the severity of the problem, or suddenly deteriorates while under observation, emergency endotracheal intubation may be required. No effort should be made to push a tube through the stenosis. Rather, the endotracheal tube should be placed above the stricture and the airway suctioned. With positive pressure ventilation, it is almost always possible to maintain the patient with a Venturi-like flow through the stenosis. A laryngeal mask airway can be used for a subglottic stenosis where proximal intubation is not possible. The patient should be moved promptly to the operating room, where a rigid bronchoscopy and dilation of the stenosis can be done under general anesthesia without respiratory paralysis.

Very few patients, even those in borderline state, require such urgent intubation. Immediately upon arrival to the hospital, the patient should be placed in a high-level intensive care facility, preferably a respiratory unit, where intubation can be done at a moment's notice and where there is constant attendance by appropriately trained physicians. With gentle physiotherapy, suctioning, adequate humidification, and supplemental oxygen or heliox, and with light medication to control anxiety, the patient usually settles down and is quite comfortable. The time gained may be used for obtaining appropriate diagnostic x-rays and ini-

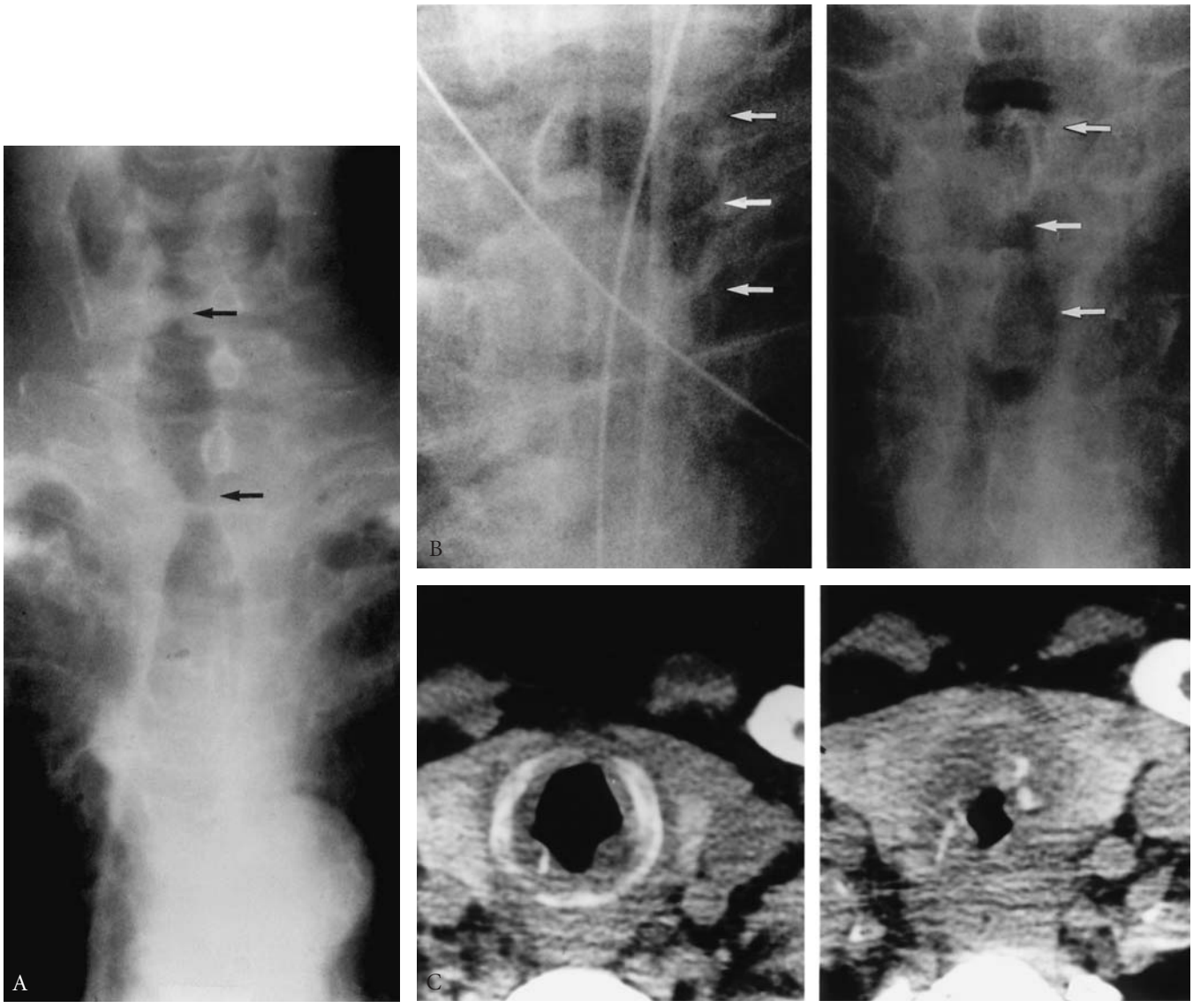


FIGURE **11-21** *Roentgenographic delineation of postintubation stenosis.* A, *Filtered view of entire upper airway, in a 60-year-old man with recurrent upper tracheal stenosis after prior resection of the stomal stenosis elsewhere. Stenosis followed ventilation via tracheostomy for respiratory failure after coronary artery surgery. Easily identifiable are the false vocal cords, glottis* (arrow), *subglottic larynx, stenosis* (arrow), *and the length of normal trachea remaining.* B, *Evolution of a cuff stenosis for ventilation with an endotracheal tube only. On the* left *is a detail from a chest x-ray showing the endotracheal tube and the distended balloon cuff* (arrows). *On the* right *is the stenosis* (arrows) *in the same tracheal segment, from another plain chest x-ray.* C, *The stenotic lesion from* B *shown on a computed tomography scan. The* left image *shows the beginning of the stenosis whereas the* right image *shows the middle of the lesion. Note the nearly complete destruction of the cartilage, and the massive scar tissue contracting the lumen to a fraction of normal cross section.*

tial clarification of the patient's medical condition. An elective corrective surgical procedure may be planned and performed under ideal circumstances. This will minimize errors in appraisal of the lesion and of the larynx. If the patient's condition fails to improve or deteriorates, the surgical team should move promptly to bronchoscopic evaluation and dilation under general anesthesia.

A technique for safe emergency *tracheal dilation* is detailed in Chapter 19, "Urgent Treatment of Tracheal Obstruction." Dilation is also a method of temporizing while a patient is further evaluated and

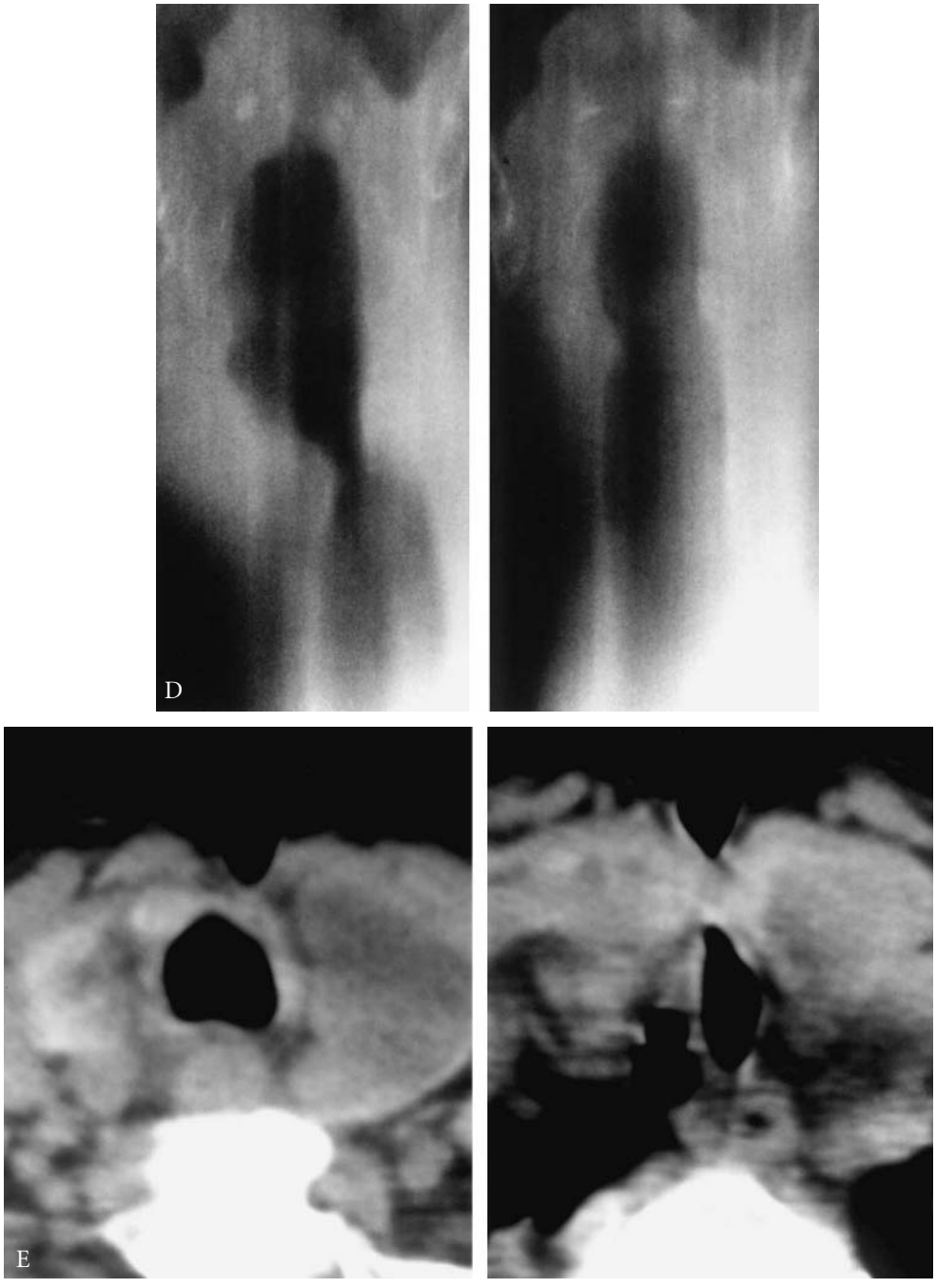


FIGURE **11-21** (CONTINUED) D, *Tomograms outlining areas of stomal stenosis and anastomosis pre- and postoperatively (*left *and* right images*, respectively). Note the irregularity of the right side of the subglottic larynx preoperatively. There is slight narrowing at the anastomosis.* E, *Computed tomography scans comparing a nearly normal trachea at* left image *with side-to-side narrowing of a stomal stenosis on the* right image. *Indentation of the cutaneous scar is seen. At bronchoscopy and operation, the anterior cricoid cartilage proved to be completely eroded, and thus required laryngotracheal resection and reconstruction. Imaging and endoscopy are both essential before an operation.*

medical or other surgical problems are corrected. Dilation may be ineffective in a patient with stomal stenosis, but such patients usually do not obstruct so totally. In circumferential cuff stenosis, dilation is essentially always only transiently effective. In most patients, dilation is effective for days to weeks or longer. Tra-

cheostomy is best avoided if it appears possible to move ahead soon to a definitive surgical treatment. A fresh tracheostomy usually delays surgical repair and, worse, may damage normal trachea necessary for reconstruction, if the tracheostomy is mistakenly placed other than through the lesion itself. Dilation, performed carefully by an experienced endoscopist, is also a procedure that may be used in an institution where there is little experience in tracheal reconstruction. A patient may then be transferred safely to a center where such work is done regularly, without the delaying or potentially damaging effect of tracheostomy. Transfer should be expeditious since the duration of relief from dilation is unpredictable. A small endotracheal tube may be inserted through the dilated lesion, if necessary, for transportation. Dilation through an existing stoma is described in Chapter 19, "Urgent Treatment of Tracheal Obstruction."

There is little practical use for lasers in these situations. Some improvement may be obtained by lasering away small amounts of granulation tissue or scar, but this should be no more than is usually obtained by dilation with, sometimes, use of bronchoscopic biopsy forceps. Radial lasering and dilation seem to have little benefit greater than dilation alone. It must be recalled that the pathology of a tracheal stenosis most often involves hourglass or side-to-side narrowing of the trachea, so that aggressive destruction of tracheal wall can result in perforation.

If for any reason surgical treatment is not elected, or if the patient is nontransportable, a tracheostomy may be necessary for longer-term management, even if not as the definitive treatment. Bronchoscopic dilation is done first under general anesthesia. The rigid ventilating bronchoscope used for dilation should remain in place for ventilation and as a guide for tracheostomy. If there was a prior tracheostomy, it is often advisable to reinstitute the tracheostomy at precisely the same point where the previous opening was made, especially if it lies in the stenotic segment (Figure 11-22*A*). If the stricture is stomal, no additional trachea will be damaged by a second tracheostomy. A cuff stenosis that lies in the cervical trachea is the best site for a necessary tracheostomy (Figure 11-22*B*). If the lesion is a cuff stenosis and lies at or below the sternal notch, then the least damage will be done, with few exceptions, if the old tracheostomy site is carefully reopened (Figure 11-22*C*). The procedure is minimal since the scar leads directly to the trachea. A transverse incision about 1 cm in length in the prior tracheostomy scar will usually suffice. The tracheostomy tube must be passed *through* the stenosis, confirmed by flexible bronchoscopy through the tracheostomy tube. Otherwise, a false sense of security will be obtained while the stenosis may close down below the tip of the tracheostomy tube. If a prior surgeon had recently placed a new tracheostomy inferior to an obstructing stenosis, which threatens to increase the extent of tracheal damage, then this length of potentially useful trachea may be recaptured, by replacing a tracheostomy through the stenotic segment and permitting the new, inferior stoma to heal (Figure 11-22*D*). If operation must be delayed a T tube provides a patient with a more normal airway and with voice. Dilation of a tracheal stenosis, reinstitution of a tracheostomy at an appropriate level, and reinsertion of a tube of proper length or a T tube is also a method of permanent management of a tracheal stenosis where repair is contraindicated. If early reconstruction is envisaged, peroral dilation is the method of choice. Rarely would a silicone stent be considered as a bridge to later resection, and *never* should an expandable stent be considered at all.

Postintubation *tracheoesophageal fistula* and *tracheo-innominate fistula* are discussed in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula," and Chapter 13, "Tracheal Fistula to Brachiocephalic Artery."

Selection of Patients for Reconstruction

Most patients with stenotic lesions of the trachea can be managed indefinitely by reinstituting a tracheostomy and placing an appropriate splinting tube, such as a Montgomery silastic T tube, through the stenosis. This places a great burden on a surgeon proposing reconstruction, who must then confidently be able to offer a very high chance of success for a proposed surgical alternative to a T tube. This is heightened by the fact that

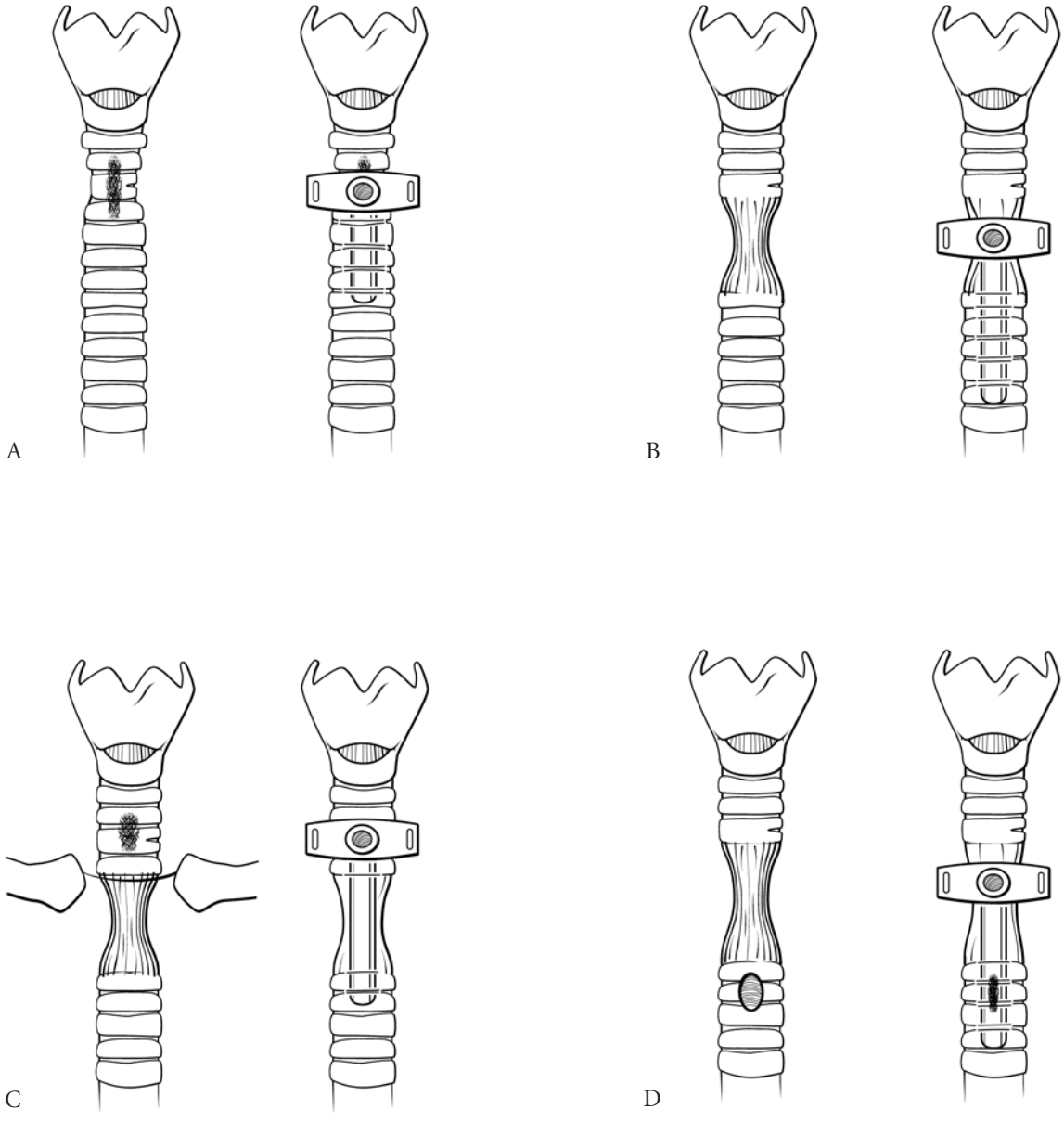


FIGURE **11-22** *Correct placement of tracheostomy tubes, when necessary, to manage postintubation tracheal stenoses.* A, *A stomal stenosis is best treated by locating the new tracheostomy in the stenotic segment, never below it.* B, *A high cervical cuff stenosis is best treated by locating the new tracheostomy in the stenotic segment, never below it.* C, *A low substernal cuff stenosis is managed by tracheostomy in a prior cervical site or at the conventional (2d–3d ring) site, with placement of a tube long enough to extend through the distal stenosis.* D, *If a cervical stenosis of considerable length has been managed by tracheostomy placed distal to it, the tracheostomy site should be relocated to the stenosed segment. The inferior tracheostomy is allowed to heal, thus recapturing a usable distal tracheal length. T tubes are also placed in the same locations in each case for longer-term management of patients, providing voice and more normal respiration.*

the best opportunity for successful reconstruction lies in the initial surgical attempt. Second trials may or may not succeed and a third attempt entails even more risk. Tracheal reconstructive procedures should not be undertaken without considerable study and experience. Silicone or expandable stents, even if coated, appear inadvisable to treat benign stenosis, since they not only produce severe stenotic lesions but may make future definitive repair impossible (see Chapter 40, "Tracheal and Bronchial Stenting").[34]

As techniques evolved and experience increased, I rejected few patients for "medical reasons," particularly those with poor respiratory reserve or marginal cardiac status. Essentially, all postintubation tracheal stenoses may be repaired through an anterior approach, avoiding entry into the pleura or pericardium, even with lesions at the supracarinal level (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). With skilled anesthesia and with exquisite intra- and postoperative management of the airway, few patients need to be denied curative treatment. The patient who remains in need of a respirator, however, should *not* be considered for surgical reconstruction unless there is absolutely no alternative way to maintain the airway, a situation which rarely pertains. A patient who will predictably require prolonged mechanical respiratory support postoperatively is also best deferred as a surgical candidate. Even a low-pressure cuff in contact with a fresh anastomosis for a long period of time will incite inflammation, which may lead to dehiscence and death. Following extensive tracheal resection, it may be impossible to seat a cuff that will not impinge on the anastomosis in the shortened trachea.

I have preferred not to perform reconstructions in patients who have basic diseases that will almost certainly require another tracheostomy within a very short period of time. An example of this is a patient who suffers from severe myasthenia gravis and has required multiple tracheostomies over the course of illness. For such a patient, a fenestrated tracheostomy tube or a silastic T tube seems preferable to provide airway and speech. I do consider patients with a borderline pulmonary status for reconstruction. In these patients, a calculated risk is taken, after full discussion with the patient, with the understanding that should another attack of respiratory failure in the future occur, then another tracheostomy, temporary or permanent tracheostomy may be required. Thus far, such patients have been satisfied to obtain a prolonged or, most often, indefinite relief from tracheostomy. With proper consultation and management, stable coronary artery disease is not a contraindication to reconstruction. Proponents of lasers and stents have all too readily accepted these conditions as reasons to deny patients definitive surgical treatment.

Elective tracheal reconstruction is best deferred in patients who are on chronic high-dose corticosteroid therapy. Although healing does occur slowly in the presence of significant doses of steroids, the chance of dehiscence is increased. Even with only moderately long resection, and hence moderate anastomotic tension, the result may be slow distraction due to delayed collagenous healing, slow increase in tensile strength, and subsequent restenosis. In 2 patients who suffered anastomotic stenosis following reconstruction, while on about 50 mg of prednisone daily, a successful re-resection was later accomplished when they were no longer steroid dependent. Where possible, it is preferable to wean the patient from the drug completely or to low doses before a tracheal operation. In the interim, the patient may be carried by repeated dilations, or with a T tube, if dilation is required too frequently. A paradoxical problem arises in a patient whose myasthenia gravis is successfully controlled with chronic steroid treatment, but who has a tracheal stenosis. Here, conservative management with a T tube may be preferable.

Demonstrated subtotal destruction of the trachea contraindicates reconstruction. An example is a patient who has only 2 or 3 cm of adequate trachea remaining. In postintubation lesions, this is almost always a result of inappropriate attempts at surgical repair of the trachea, and almost never from the original lesion. Although it may be possible to try to rebuild such tracheae in stages or to attempt replacement with a prosthesis, the first method is complex and the second is experimental. Failure is frequent and may be fatal (see Chapter 45, "Tracheal Replacement"). A safe and relatively satisfactory alternative is to splint the trachea with a Montgomery silastic T tube (see Chapter 39, "Tracheal T Tubes"). Pearson and Andrews preferred to defer operation until a fresh stenosis had "matured" and acute inflammatory changes had subsided.[35] In the presence of florid granulations and acute inflammation, I completely agree that it is judicious to delay surgery until the inflammatory reaction subsides. This may require weeks, months, or longer. Following a prior failed attempt at reconstruction, at least 4 months, and preferably 6, should pass before a second operation. Even then, surgical planes will be difficult and the chance of success somewhat diminished.

A variety of treatments have been used for postintubation stenosis, including repetitive dilation with or without injection of steroids into the area of inflammation, lasering, or, usually more effectively, the insertion of conventional internal splints (a tracheostomy tube or silastic T tube). Rarely, a patient with a lesion characterized by incomplete destruction of the tracheal wall in depth, extent, or circumference, may achieve a permanently open airway after a prolonged period of stenting. Lesions that respond to such treatment are usually of lesser severity. As the inflammatory process subsides and the scar becomes more mature, a partial airway is obtained. In order for this to occur in the trachea, it must be assumed that part of the structural integrity of the cartilaginous wall remained. Treatment requires a prolonged period of time and often results in a less than ideal airway. When the splinting tube is removed, the patient must remain under close observation, since in most cases, the stenosis will contract again over time and ultimately require surgical reconstruction. Stenting should *not* be a routine step in treating tracheal stenosis, since success by this method is so rare. Furthermore, expandable stents, even when coated, may cause severe proximal and distal granulation and stenosis, extending the original lesion to unresectable lengths (Figures 29 and 30 [Color Plate 15]).[34] Such stents should not be used, especially where the lesion is a correctable one. In most patients, indications for surgical correction are evident when the patient is first seen.

For most patients, there is almost no hope for success of a "conservative" treatment. The alternatives are a permanent tube or surgical reconstruction. Patients who have had splinting tubes in place for months, or even years, will usually close their airway acutely in the 40 or 50 minutes required for tracheal radiography to be completed with a tracheostomy tube removed. Following removal of a T tube, distress usually occurs in days to weeks. As surgical techniques and experience with tracheal reconstruction grew, I felt increasingly justified in advising early reconstruction rather than tedious trials of uncomfortable conservative treatment, which could rarely succeed. Repair has only been deferred in the special categories described. Especially to be avoided are inlying stents, which make lesions surgically incurable. Even silicone stents for lesions high in the trachea may incite granulation tissue if they impinge on the conus elasticus of the subglottic larynx.

Definitive Treatment

Surgical techniques, applicable to correction of postintubation stenosis are detailed in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection," and Chapter 25, "Laryngotracheal Reconstruction." The surgical management of postintubation tracheoesophageal and tracheoarterial fistulae is related in Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula," and Chapter 27, "Repair of Tracheobrachiocephalic Artery Fistula." The rare lesion of isolated malacia resulting from intubation is best treated by segmental resection, where its length is not excessive.

Results of Surgical Treatment

Patients. In the 27 years between 1965 and 1992, 503 patients underwent tracheal resection and reconstruction for postintubation lesions at Massachusetts General Hospital.[32] There were 266 males and 237 females with an age range of 6 to 85 years (average 44 years). Of these, 251 lesions resulted from the sealing cuff of an endotracheal or tracheostomy tube, 178 were at the site of a tracheostomy, and 38 had evidence of both lesions. In 36 cases, the exact site of origin was not certain, often because of previous attempts at treatment, including multiple tracheostomies. In 441 patients, the lesions were principally tracheal. Sixty-two had involvement of the subglottic larynx as well as the upper trachea. In 123 patients, ventilation had been provided only with an endotracheal tube. Most patients had received ventilatory assistance initially via an endotracheal tube for varying periods of time before tracheostomy, and 380 had either already had a tracheostomy or were given one when seen. Of note is the fact that 2 patients had been intubated for less than 18 hours, several for 48 hours or less. Nearly all had received ventilatory assistance.

Many patients had undergone prior attempts at surgical treatment before referral. These included resection (53), tracheal operations such as wedge resections, splinting, or fissure (31), and laryngeal procedures such as stenting, grafting, or fissure (20). Sixty had had T tubes placed and at least 45 had undergone laser treatment. Eight patients had had prior repairs of tracheoesophageal fistula, 3 of which had failed.

Operative Treatment. At MGH, 521 tracheal resections were done on the 503 patients described above. Thirteen of them who had restenosis after initial operation were later reoperated upon. Five patients with immediate failure because of residual malacia were reoperated on within hours of the initial operation. These cases are not counted as additional reconstructions but as complications. Since this series includes our entire experience, the surgical technique had evolved over the 27 years spanned. Surgical procedures are described in Part 2 of this book. Approach was through a cervical incision in 350 patients, through a partial upper sternotomy to a point below the sternal angle (cervicomediastinal) in 145 patients, with 2 additional patients requiring extension of the cervicomediastinal incision into right anterior thoracotomy. Earlier, a right posterolateral thoracotomy was used for 6 patients. Few if any reconstructions for postintubation stenosis would now be done through thoracotomy. A cervical or upper cervicomediastinal incision suffices, even for lesions at the supracarinal level. Complete sternotomy is unnecessary. The amount of trachea resected most commonly measured between 2 to 4 cm with a range from 1 to 7.5 cm. Anastomotic tension was routinely lessened by complete dissection of the pretracheal plane and by cervical flexion at the time of anastomosis. Laryngeal release was used in 49 patients, the first 9 by the thyrohyoid technique of Dedo and Fishman,[36] which Dedo has now abandoned, and the remaining 40 by the suprahyoid technique of Montgomery.[37] Laryngeal release was used in 9.7% of patients. Only 8% of the 450 patients who had not undergone prior tracheal resection were thought to require laryngeal release to reduce anastomotic tension, in comparison with 24% of the 53 who had had prior resection and reconstruction. In only one complex case, an intrapericardial hilar release was added.

Trachea-to-trachea anastomosis was performed in 324 patients, trachea-to-cricoid anastomosis (with horizontal removal of varying amounts of the anterior cricoid cartilage) in 117 patients, and laryngotracheal anastomosis (with removal of the anterior cricoid arch of the subglottic larynx) in 62. Laryngeal release was used in 29 of 324 (9%) patients with trachea-to-trachea anastomosis, in 12 of 117 (10.3%) with trachea-to-cricoid anastomosis, and in 8 of 62 (12.9%) with trachea-to-thyroid cartilage anastomosis. The use of laryngeal release was dictated by the extent of resection and tracheal mobility in each patient. It was necessary more often in patients who had undergone prior resection or in whom the lesion extended into the lower larynx. Laryngeal release should not be employed routinely.

Although the technique of placement of sutures varied little over the period described, suture material progressively changed. Prior to 1978, 4-0 Dacron polyester, Tevdek polyester, Mersilene polyester, and Prolene polypropylene sutures were used in a search for improvement. Sufficiently fine, absorbable catgut was not strong enough for use. Wire presented a threat to an adjacent brachiocephalic artery. Since 1978, Vicryl polyglactin 910 has been used. The change was dictated by the frequency of granulomas at the suture line with all nonabsorbable sutures listed. Granulations essentially vanished following change to the use of absorbable Vicryl. Suture line granulations dropped from an incidence of 23.6 to 1.6%, and most are now not of clinical importance. Monofilament PDS polydioxanone was tried and discarded since no advantage over Vicryl was found and it was somewhat more difficult to use.

Twenty-five patients in the series had tracheoesophageal fistulae as well. Seven patients with stenosis accompanied by extensive tracheomalacia were managed with the placement of one or more polypropylene rings around the malacic segment of the trachea, usually above the level of the tracheal resection for stenosis. In 84 patients, the anastomoses were covered with adjacent tissues: thyroid isthmus in 50, cervical strap muscle in 26, and other tissue including thymus and pericardial fat pad in 8. If the pathology and, therefore, the anastomosis lay adjacent to the innominate artery, or if the artery had been dissected in a

prior operation, then the tissue (usually pedicled sternohyoid muscle) was interposed between the anastomosis and the artery. Routine wrapping of the anastomoses was not deemed necessary in the cervical or mediastinal region with these approaches, in contrast to intrathoracic anastomoses.

Results. In the MGH series, results were classified as *good*, *satisfactory*, *failure*, and *death*. *Good* indicates a patient being functionally able to perform usual activities, with an anatomically, essentially normal airway, as determined by postoperative roentgenograms or bronchoscopy. Results are considered *satisfactory* if the patient can perform normal activities but is stressed on exercise, or if there exists an abnormality such as a paralyzed or paretic vocal cord, or where significant airway narrowing is evident on endoscopy or roentgenograms, even if the patient's level of activity does not clinically evidence this. Patients were considered *failures* if they required a permanent tracheostomy or T tube to maintain an airway. The average length of follow-up was 3 years. In our experience, patients who remain stable after 2 months are very unlikely to have further difficulties. By 6 months to a year, the result may be considered to be final. Later change in clinical status was not observed.

Results were good in 440 patients and satisfactory in 31 patients, and there were 20 failures and 12 deaths (Table 11-1). Of the failed patients, 11 were treated with a tracheostomy, 7 with a T tube, and dilations in 2.

Factors in the Results. *Prior resection and reconstruction* led to 72.2% good results in comparison with 85% good results in those without prior resection and reconstruction (see Table 11-1).[32] Combined good and satisfactory results were 90.2% and 94.4%, respectively, with and without prior resection. The failure rate of the first operation was 3.8% with a 2.4% mortality, compared with 5.6% failure with a prior resection. Despite a surprisingly high proportion of good results (87%), reconstruction after prior complex tracheal operations such as insertion of a Marlex prosthesis, cartilage grafts, hyoid bone grafts, cutaneous trough operations, and stented cutaneous grafts, led to the highest failure rate (9.7%) (Table 11-2). A previous T tube, laser therapy, or prior repair of tracheoesophageal fistula did not seem to affect outcome adversely.

The failure rate increased with a higher *level of anastomosis* (Table 11-3): trachea-to-trachea anastomosis 2.2%, trachea-to-cricoid 6.0%, and trachea-to-thyroid cartilage 8.1%. Minor complications became more prevalent with each level (from 16% to 17.1% to 21%) but major complications were unchanged (13.9%, 15.4%, 12.9%, respectively).

Laryngeal release was performed in 46 patients with an average length of resection of 4.4 cm. Forty-one were done at the initial operation and 5 at reoperation. Three additional patients had release before referral. Of the 41 released initially, results were good in 77%, satisfactory in 9.1%, and failure or death occurred in 6.8%. The 5 who underwent release with reoperation and the 3 with a prior release all had good outcomes.

Of the 20 patients with *tracheoesophageal fistulae* also, including 3 with failed prior repairs, 18 had a good outcome, 1 was satisfactory with reoperation, and 1 died. Patients with short segments of *tracheomalacia* were treated by resection. However, 7 who underwent tracheal resection for stenosis had extensive

Table 11-1 Results of Surgical Treatment of Postintubation Tracheal Stenosis

	Number of Patients	*Good*		*Satisfactory*		*Failure*		*Death*		*Reoperation*	
		Number	*%*	*Number*	*%*	*Number*	*%*	*Number*	*%*	*Number*	*%*
Initial operation	503	427	84.9	27	5.3	19	3.8	12	2.4	18	3.6
Reoperation	18	13	72.2	4	22.2	1	5.6	0	0	0	0
Overall	503	440	87.5	31	6.2	20	3.9	12	2.4	0	0

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Table 11-2 Effect of Prior Treatment on Results of Surgical Treatment of Tracheal Stenosis

		Good		*Satisfactory*		*Failure*		*Death*		*Reoperation*	
Treatment	*Total*	*Number*	*%*	*Number*	*%*	*Number*	*%*	*Number*	*%*	*Number*	*%*
T tube	60	51	85	0	0	3	5	2	3.3	4	6.7
Laser	45	40	88.9	1	2.2	1	2.2	0	0	3	6.7
Resection and reconstruction	53	40	75.5	6	11.3	3	5.6	2	3.8	2	3.8
Other tracheal surgery	31	27	87.1	1	3.2	3	9.7	0	0	0	0
Laryngeal surgery	20	16	80	2	10	0	0	1	5	1	5
TEF repair	8	7	87.5	0	0	0	0	0	0	1	12.5
No prior treatment	342	295	86.2	18	5.3	12	3.5	8	2.3	9	2.7
No prior resection and reconstruction	450	387	86	21	4.7	16	3.6	10	2.1	16	3.6

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additional malacic segments splinted with polypropylene rings. Three had a good outcome, 1 failure required a permanent T tube, and 1 died. Two reoperations resulted in 1 good and 1 satisfactory result. This procedure is *not* a standard operation, is not recommended, and requires further study and development. The danger points are potential loss of blood supply to the malacic segment and infection around the foreign material in a procedure where the trachea is also opened.

Postoperative reintubation was necessary in 23 patients: 9 on the day of surgery, 11 in the first postoperative weeks, and 1 at 30 days. Proximity of repair to the glottis increased the likelihood: 9 reintubations in 324 trachea-to-trachea, 8 in 117 trachea-to-cricoid, and 6 in 62 trachea-to-thyroid cartilage anastomoses. Since reintubation indicated a major problem, it is not surprising that 4 of these patients needed permanent tracheostomy or T tube and that 5 patients died.

In 27 patients, a *concurrent tracheostomy* was deemed advisable at completion of the reconstruction for factors such as laryngeal edema, vocal cord paralysis, or severe and uncorrectable narrowing immediately below the glottis. Tracheostomy was initially routinely performed in our early series of laryngotracheal reconstructions but is now used only occasionally for specific indications. Four T tubes were also placed, for such reasons as control of a high second stenosis after resection of a lower one. Twenty-one obtained

Table 11-3 Effect of Level of Anastomosis, Presence of TEF, and Use of Ring Supports on Results of Surgical Treatment of Tracheal Stenosis

		Good		*Satisfactory*		*Failure*		*Death*		*Reoperation*	
Treatment	*Total*	*Number*	*%*	*Number*	*%*	*Number*	*%*	*Number*	*%*	*Number*	*%*
Anastomosis											
Trachea-trachea	324	275	85.9	18	5.5	7	2.2	9	2.8	15	4.6
Trachea-cricoid	117	101	86.3	4	3.4	7	6.0	2	1.7	3	2.6
Trachea-thyroid	62	51	82.2	5	8.1	5	8.1	1	1.6	0	0
TEF repair	20	18	90.0	0	0	0	0	1	5.0	1	5.0
Plastic ring supports used	7	3	42.9	0	0	1	14.3	1	14.3	2	28.5

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good results, 3 satisfactory, whereas 1 required reoperation and 2 required permanent tracheostomy. The placement of a complementary tracheostomy and its compartmentalization from the fresh anastomosis and from the innominate artery are detailed in Chapter 25, "Laryngotracheal Reconstruction."

Complications of operations are summarized in Table 11-4 and discussed in detail in Chapter 21, "Complications of Tracheal Reconstruction."

Deaths occurred in 12 patients perioperatively. Anastomotic dehiscence and its complications accounted for 7 deaths, including 2 with consequent innominate artery hemorrhage. One of these had undergone a resection through massive fibrosis of prior irradiated Hodgkin's disease, and reconstruction failed despite omentoplasty. Retrospectively, healing was not possible. Subsequent patients with this lesion have been managed with T tubes. Early in our series, 2 patients with previously failed resections elsewhere were reoperated on, while on respirators, a situation that should be avoided. Reintubation for secretions and flail chest accounted for the other dehiscences. In the era when only high-pressure cuffs were available, reintubation after extensive resection soon led to dehiscence. Low-pressure cuffs resting on a fresh anastomosis also encourage separation, but after a longer interval of irritation. If ventilation is needed after tracheal reconstruction, it is best to site the cuff above or below the anastomosis, if possible.

One patient died from a postoperative innominate artery fistula, 2 from malacic tracheal obstruction, 1 from myocardial infarction, and 1 from respiratory failure late at home without clarification of cause.

Reconstruction after an Unsuccessful Repair

Reconstruction following a prior attempt is daunting because of reduced length of normal trachea available, and surgical scar, which makes dissection difficult, endangers recurrent laryngeal nerves, limits tracheal mobility, and may affect tracheal blood supply. Reoperation should be delayed for 4 to 6 months after a prior reconstruction to allow subsidence of tissue inflammation and edema, and maturation of scar. We

Table 11-4 Complications of Operations for Postintubation Tracheal Stenosis

	Major	*Minor*	*Total*
Granulations	11	38	49
Before 1978	10	34	44
After 1978	1	4	5
Dehiscence	28	1	29
Laryngeal dysfunction	11	14	25
Malacia	10	0	10
Hemorrhage	5	0	5
Edema (anastomosis)	3	1	4
Infection	12	22	34
Wound	7	8	15
Pulmonary	5	14	19
Myocardial infarction	1	0	1
Tracheoesophageal fistula	1	0	1
Pneumothorax	0	3	3
Line infection	0	1	1
Atrial fibrillation	0	1	1
Deep venous thrombosis	0	1	1
Total	82	82	164

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reoperated on 75 patients, 16 failures of our own and 59 referred after failure elsewhere.[38] They had been managed with observation, dilation, tracheostomy, or T tube. Nineteen required laryngeal release and complications were more frequent. Results were surprisingly good in this group: 78.6% were good, 13.3% satisfactory, and 5.3% were failures. Two died (2.75%) after dehiscence.

Failure of tracheal reconstruction is best treated with a permanent T tube if there is insufficient tissue for a safe reconstruction. Although 16 of our own failures were reoperated upon, another 16 were not treated by re-resection.

Comment

The generally good and satisfactory results of surgical treatment of postintubation stenosis (93.7%), even when it involves the subglottic larynx (90.3%) and even in the presence of the rare TEF justify resection and reconstruction as treatments of choice.[32] Good results are confirmed in reports by and Maddaus and colleagues,[33] Pearson and Andrews,[35] Couraud and colleagues,[39] and Bisson and colleagues.[40] These excellent series are not further detailed here because they report findings very similar to those described. T tubes, inlying stents, and laser treatment may be applicable in a very limited spectrum of lesions and at a much lesser level of success. Laser treatment most often fails as a definitive treatment. The "thin, web-like stenosis," in which laser treatment might be expected to effect cure, is an extremely rare lesion (see Chapter 37, "Laser Therapy for Tracheobronchial Lesions"). Failure rate for resection was 3.9%, with the death rate at 2.4%, for all patients. For the more difficult laryngotracheal cases, these rates are 8.1% and 1.6%, respectively, counseling caution in these patients.

The lower surgical success rate in patients who had prior failure of reconstruction confirms the observation that the first operation is most likely to succeed and should ideally be performed by experienced hands. The more complex the prior treatment, the more likely eventual failure, even after reoperation. Finally, it must be emphasized that a permanent tracheal T tube may be the best solution for a patient with extensive tracheal damage that defies straightforward reconstruction.

References

1. Briggs BD. Prolonged endotracheal intubation. Anesthesiology 1950;11:129–31.
2. Lindholm C-E. Prolonged endotracheal intubation. Acta Anaesth Scand 1969;13 Suppl 33:1–131.
3. Whited RE. A prospective study of laryngotracheal sequelae in long-term intubation. Laryngoscope 1984;94: 367–77.
4. Donnelly WH. Histopathology of endotracheal intubation. An autopsy study of 99 cases. Arch Pathol 1969;88:511–20.
5. Bergström J, Moberg A, Orell SR. On the pathogenesis of laryngeal injuries following prolonged intubation. Acta Otolaryngol 1962;55:342–6.
6. Schmidt VW, Schaap RN, Mortensen JD. Immediate mucosal effects of short term, soft-cuff endotracheal intubation. Arch Pathol Lab Med 1979;103:516–21.
7. Hedden M, Ersoz CJ, Donnelly WH, Safar P. Laryngeal tracheal damage after prolonged use of orotracheal tubes in adults. JAMA 1969;207:703–8.
8. Grillo HC, Mathisen DJ, Wain JC. Laryngotracheal resection and reconstruction for subglottic stenosis. Ann Thorac Surg 1992;53:54–63.
9. Brantigan CO, Grow JB. Cricothyroidotomy: elective use in respiratory problems requiring tracheotomy. J Thorac Cardiovasc Surg 1976;171:72–81.
10. Kuriloff DB, Setzen M, Portnoy W, Gadaleta D. Laryngotracheal injury following cricothyroidotomy. Laryngoscope 1989;99:125–30.
11. Andrews MJ, Pearson FG. Incidence and pathogenesis of tracheal injury following cuffed tube tracheostomy with assisted ventilation. Analysis of a two year prospective study. Ann Surg 1971;173:249–63.
12. Cooper JD, Grillo HC. The evolution of tracheal injury due to ventilatory assistance through cuffed tubes. A pathologic study. Ann Surg 1969;169:334–8.
13. Florange W, Muller J, Forster E. Morphologie de la nécrose trachéale après trachéotomie et utilisation d'une prothèse respiratoire. Anesth Analg Réan 1965;22:693–703.
14. Grillo HC, Cooper JD, Geffin B, Pontoppidan H. A low-pressure cuff for tracheostomy tubes to minimize tracheal injury: a comparative clinical trial. J Thorac Cardiovasc Surg 1971;62:898–907.
15. Cooper JD, Grillo HC. Experimental production and prevention of injury due to cuffed tracheal tubes. Surg Gyn Obstet 1969;129:1235–41.
16. Grillo HC. The management of tracheal stenosis following assisted respiration. J Thorac Cardiovasc Surg 1969;57:52–71.
17. Stetson JB, Guess WL. Causes of damage to tissues by polymers and elastomers used in the fabrication of tracheal devices. Anesthesiology 1970;33:635–52.
18. Shelly WM, Dawson RC, May IA. Cuffed tubes as a cause of tracheal stenosis. J Thorac Cardiovasc Surg 1969;57:623–7.
19. Murphy DA, MacLean LD, Dobell ARC. Tracheal stenosis as a complication of tracheostomy. Ann Thorac Surg 1966;2:44–51.

20. Harley HRS. Laryngotracheal obstruction complicating tracheostomy or endotracheal intubation with assisted respiration. A critical review. Thorax 1971;26:493–533.
21. Tobin MJ. Advances in mechanical ventilation. N Engl J Med 2001;344:1986–96.
22. Adriani J, Phillips M. Use of the endotracheal cuff: some data pro and con. Anesthesiology 1957;18:1–14.
23. Knowlson GTG, Bassett HFM. The pressures exerted on the trachea by endotracheal inflatable cuffs. Br J Anaesth 1970;42:834–7.
24. Carroll R, Hedden M, Safar P. Intratracheal cuffs: performance characteristics. Anesthesiology 1969;31:275–81.
25. Lomholt N. A new tracheostomy tube. I. Cuff with controlled pressure on the tracheal mucous membrane. Acta Anaesth Scand 1967;11:311–8.
26. Geffin B, Pontoppidan H. Reduction of tracheal damage by the prestretching of inflatable cuffs. Anesthesiology 1969;31:462–3.
27. Ching NPH, Nealon TF. Clinical experience with new low-pressure high volume tracheostomy cuffs. N Y State J Med 1974;74:2379–84.
28. Ching NPH, Ayres SM, Paegle RP, et al. The contribution of cuff volume and pressure in tracheostomy tube damage. J Thorac Cardiovasc Surg 1971;62:402–10.
29. Kamen JM, Wilkinson CJ. A new low-pressure cuff for endotracheal tubes. Anesthesiology 1971;34:482–5.
30. Magovern GJ, Shively JG, Fecht D, Theroz F. The clinical and experimental evaluation of a controlled-pressure intratracheal cuff. J Thorac Cardiovasc Surg 1972; 64:747–56.
31. Arens JF, Ochsner JL, Gee G. Volume-limited intermittent cuff inflation for long-term respiratory assistance. J Thorac Cardiovasc Surg 1969;58:837–41.
32. Grillo HC, Donahue DM, Mathisen DJ, et al. Postintubation tracheal stenosis: treatment and results. J Thorac Cardiovasc Surg 1995;109:486–93.
33. Maddaus MA, Toth JLR, Gullane PJ, et al. Subglottic tracheal resection and synchronous laryngeal reconstruction. J Thorac Cardiovasc Surg 1992;104:1443–50.
34. Gaissert HA, Grillo HC, Wright CD, et al. Lengthening and complication of benign tracheobronchial strictures by self-expanding microinvasive ultraflex and wall stents. J Thorac Cardiovasc Surg 2003. [In press]
35. Pearson FG, Andrews MJ. Detection and management of tracheal stenosis following cuffed tube tracheostomy. Ann Thorac Surg 1971;12:359–74.
36. Dedo HH, Fishman NH. Laryngeal release and sleeve resection for tracheal stenosis. Ann Otol Rhinol Laryngol 1969;78:285–8.
37. Montgomery WW. Suprahyoid release for tracheal stenosis. Arch Otolaryngol 1974;99:255–60.
38. Donahue DM, Grillo HC, Wain JC, et al. Reoperative tracheal resection and reconstruction for unsuccessful repair of post intubation stenosis. J Thorac Cardiovasc Surg 1997;114:934–9.
39. Couraud L, Jongon JB, Velly JF. Surgical treatment of nontumoral stenosis of the upper airway. Ann Thorac Surg 1995;60:250–60.
40. Bisson A, Bonnette P, El Kadi B, et al. Tracheal sleeve resection for iatrogenic stenoses (subglottic laryngeal and tracheal). J Thorac Cardiovasc Surg 1992; 104:882–7.

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Acquired Tracheoesophageal and Bronchoesophageal Fistula

Hermes C. Grillo, MD

Acquired Tracheoesophageal Fistula
Bronchoesophageal Fistulae

Acquired Tracheoesophageal Fistula

Granulomatous infection, foreign bodies, and trauma used to be the most common causes of benign acquired tracheoesophageal fistula (TEF). Added to these were fistulae from complications of surgical procedures such as anterior cervical spine fusion and laryngectomy. With widespread use of cuffed tubes for ventilation, postintubation fistulae became predominant. In 1968, in a review of acquired nonmalignant esophagotracheal and esophagobronchial fistulae, Wesselhoeft and Keshishian reported no cases of tracheoesophageal fistula related to cuffs.[1] By 1973, Thomas collected 46 such cases (30 fully documented), including 7 of his own.[2] Although the use of low-pressure, large-volume cuffs has reduced the incidence, fistulae from this source remain the most common. Immunodeficiency syndromes may also result in fistulae.

In fistulae due to granuloma and foreign body, the pathology involves the membranous wall of the trachea and is often limited in extent.[3] Traumatic fistulae may be very extensive and be accompanied by mediastinal inflammation and infection. A postintubation fistula results from erosion of the membranous wall of the trachea and the adjacent esophageal wall, "the party wall," because of pressure from the ventilatory cuff usually exerted against a firm nasogastric tube lying in the esophagus (Figures 12-1, 12-2) (see Chapter 11, "Postintubation Stenosis"). Overinflation of a large-volume cuff by even a small, added volume of air converts it to a high-pressure cuff. The fistula may erode the entire width of the membranous wall; these are often termed "giant fistulae." Since the inflammatory process is progressive, there is never leakage into the mediastinum in the way there is in a traumatic fistula. Spontaneous healing of such fistulae has not been documented, although on rare occasion, a small recent traumatic fistula may close spontaneously. Circumferential injury to the trachea is almost always present concurrently with a postintubation TEF due to pressure necrosis caused by the cuff.

Fistula from the neoesophagus to trachea after esophagectomy is fortunately rare.[4] These may follow anastomotic leakage, dilation of stenosis, or tracheal or enteric ischemia as a result of surgical dissection.[5] Cervical anastomotic leakage is a principal cause. Symptoms range from cough associated with ingestion to life threatening aspiration pneumonia.

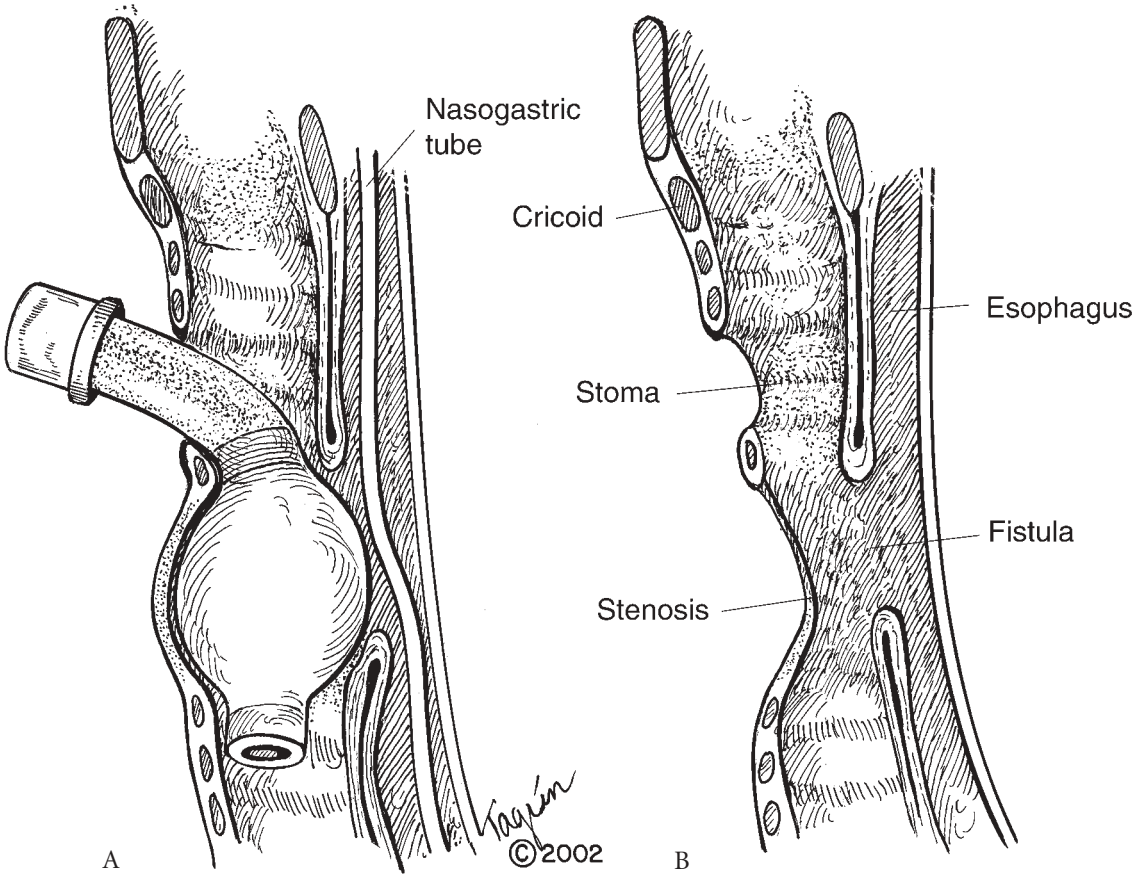


FIGURE 12-1 *Origin and anatomy of postintubation tracheoesophageal fistula. Lateral diagrams of the trachea and esophagus.* A, *The overdistended cuff has injured the trachea circumferentially. The "party wall" posteriorly has become devascularized and has necrosed by being pinched between the cuff and a firm nasogastric tube in the esophagus.* B, *The fistula is usually below the stoma, at the level of the balloon cuff.*

More recently, expandable stents have caused TEF spontaneously, after placement of an expandable coated esophageal stent, and as a consequence of difficult removal of a tracheal stent which has become severely obstructive.[6,7]

The rare but devastating occurrence of necrotizing esophagitis that occurs in immunodeficiency states produces both TEF and bronchoesophageal fistula (BEF), with a high mortality rate.[8]

Malignant TEF often results from carcinoma of the esophagus, and less so from carcinoma of the lung or lymphoma. Isolated instances have been associated with adenoid cystic carcinoma and even carcinoid tumor. Irradiation treatment of esophageal carcinoma that involves the tracheal wall may accelerate fistula formation.

The topic of congenital tracheoesophageal fistula and esophageal atresia has been extensively treated in many textbooks of surgery and pediatric surgery. It is not included in this book, especially since the reconstructive problem is largely esophageal. Rarely, a small recurrent fistula may become symptomatic many years after repair of a congenital TEF in infancy. Attention should be called, however, to the very rare and usually cervical congenital H-type fistula without esophageal atresia, which may give symptoms later in life (see Chapter 6, "Congenital and Acquired Tracheal Lesions in Children"). The signs are principally cough or choking episodes after ingestion of liquid or other food. The fistula may be very small and clini-

cal presentation not obvious. Repeated bouts of respiratory infection may occur. Suspicion leads to diagnosis by bronchoscopy and radiography.[9,10] A description is included at the end of this chapter of the rare entity of bronchoesophageal fistula, both congenital and acquired.

Clinical Presentation and Diagnosis

Benign Fistula. If a fistula develops in a patient on a respirator, a sudden increase in secretions is often noted as saliva enters the airway. It becomes difficult to maintain a seal with the cuff. Pulmonary infiltrates and pneumonia follow. Respiratory insufficiency may worsen. Cough follows swallowing. With ventilation, air may be heard escaping into the pharynx and the abdomen may become distended. Gastric feedings may appear on tracheal suctioning. Gastric reflux into the lungs can be disastrous and eventually fatal. If the patient is receiving oral feedings, these will appear in the tracheal suctioning.

Chest x-ray commonly shows the esophagus to be dilated distal to the fistula and the stomach may be dilated (Figure 12-3). A swallow of water stained with methylene blue will appear in the tracheostomy. This test is to be interpreted with caution since aspiration of dye into the larynx produces the same result. *Fluoroscopy* by an experienced radiologist, with ingestion of a small amount of barium, usually delineates the level and approximate size of the fistula (Figures 12-4, 12-5).

The fistula may be visible directly through a tracheostomy if it is present. *Bronchoscopy* should be done promptly if a fistula is suspected. In a patient who is on a respirator, a flexible bronchoscopy may be performed through an endotracheal tube, which is withdrawn just sufficiently to allow visualization of the

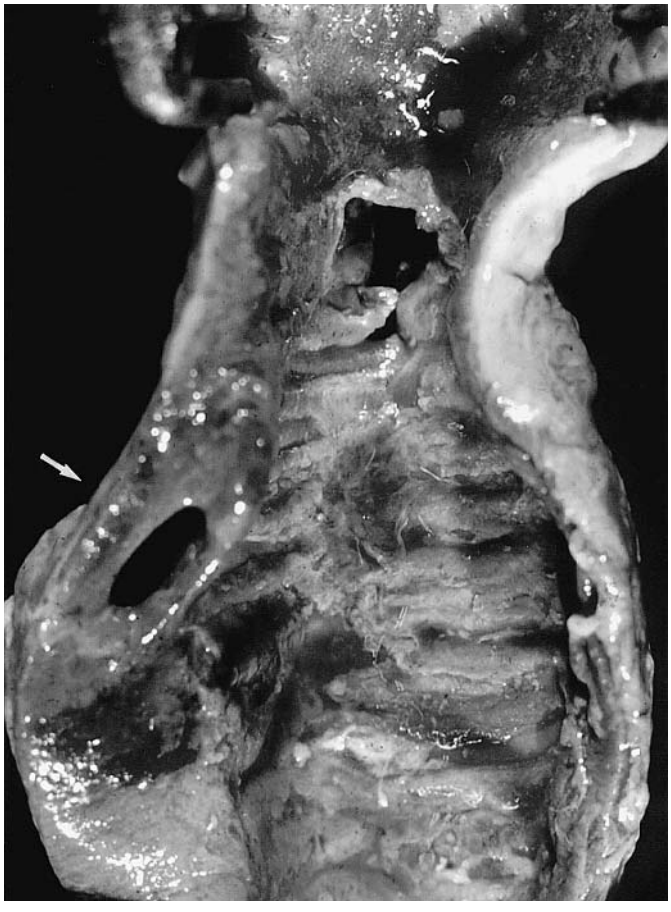


FIGURE **12-2** *Postmortem specimen of the trachea of a patient on long-term ventilation through a tracheostomy tube with a high-pressure cuff. A large membranous wall fistula has formed* (arrow). *The tracheal stoma is superior. Note the circumferential cuff damage.*

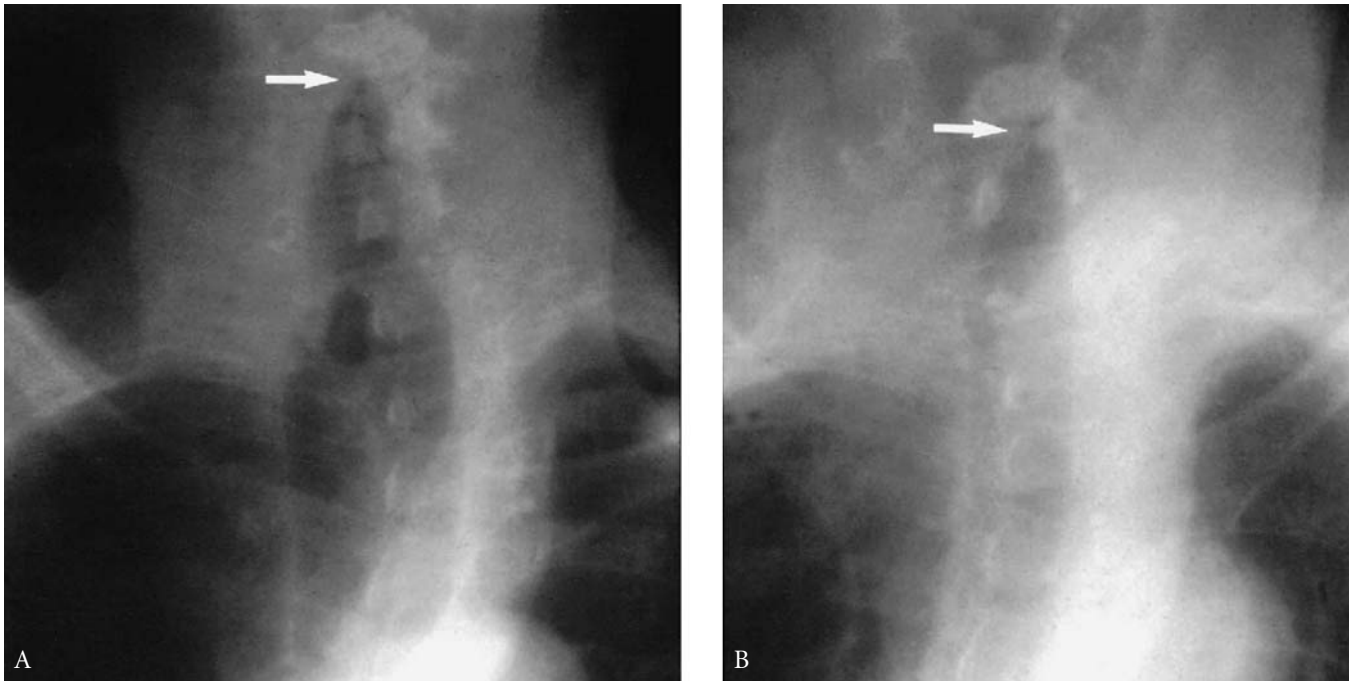


FIGURE **12-3** *Roentgenogram of a patient with postintubation tracheoesophageal fistula.* A, *Note the distended esophagus, typical of this condition. Fistula is visible as a radiolucent circle.* B, *After repair, a normal tracheal air column is seen.* Arrows *mark the glottic level in both roentgenograms.*

fistula. The same may be done through a tracheostomy tube while continuing ventilation in both cases. Passage of a rigid ventilating bronchoscope via the larynx allows the best assessment of the entire airway and locates the fistula relative to the cricoid and carina. The lengths of the fistula and of the normal airway are measured. A postintubation fistula is usually clearly identifiable (Figure 12-6). If not, methylene blue in saline may be instilled into the upper esophagus with the caution already noted to avoid overfilling and aspiration. Esophagoscopy is less likely to offer a good view, especially of smaller fistulae. A postintubation fistula usually lies a centimeter or two below the level of a tracheal stoma, since the fistula is located at the cuff site (see Figure 12-1).

Chronic fistulae from other causes present with cough on fluid or food ingestion, pulmonary infection, and occasional hemoptysis. Contrast images and bronchoscopy are diagnostic but most important is the clinician's suspicion of a fistula. This is even more critical after severe chest trauma since a tracheoesophageal rupture may go unrecognized, or a fistula may be delayed in its formation, until life threatening mediastinal sepsis is established.

Malignant Fistula. In an excellent review of 207 malignant esophagorespiratory fistulae, Burt and colleagues confirmed esophageal carcinoma as the primary neoplasm (78%).[11] The tumors were located principally in the upper thoracic esophagus and were principally squamous in histology. The incidence of fistulization in this series of esophageal carcinomas was 4.5%. Lung cancer accounted for 16% of the malignant fistulae, and in only 3 patients were tracheoesophageal fistulae related to primary tracheal neoplasms. Other neoplasms that were associated with a tracheoesophageal fistula were Hodgkin's disease, metastatic breast cancer, and laryngeal carcinoma.[12,13]

All patients with carcinoma of the upper- or midesophagus should undergo bronchoscopy in their initial work-up. If an abnormality is identified between the trachea and esophagus radiographically, on

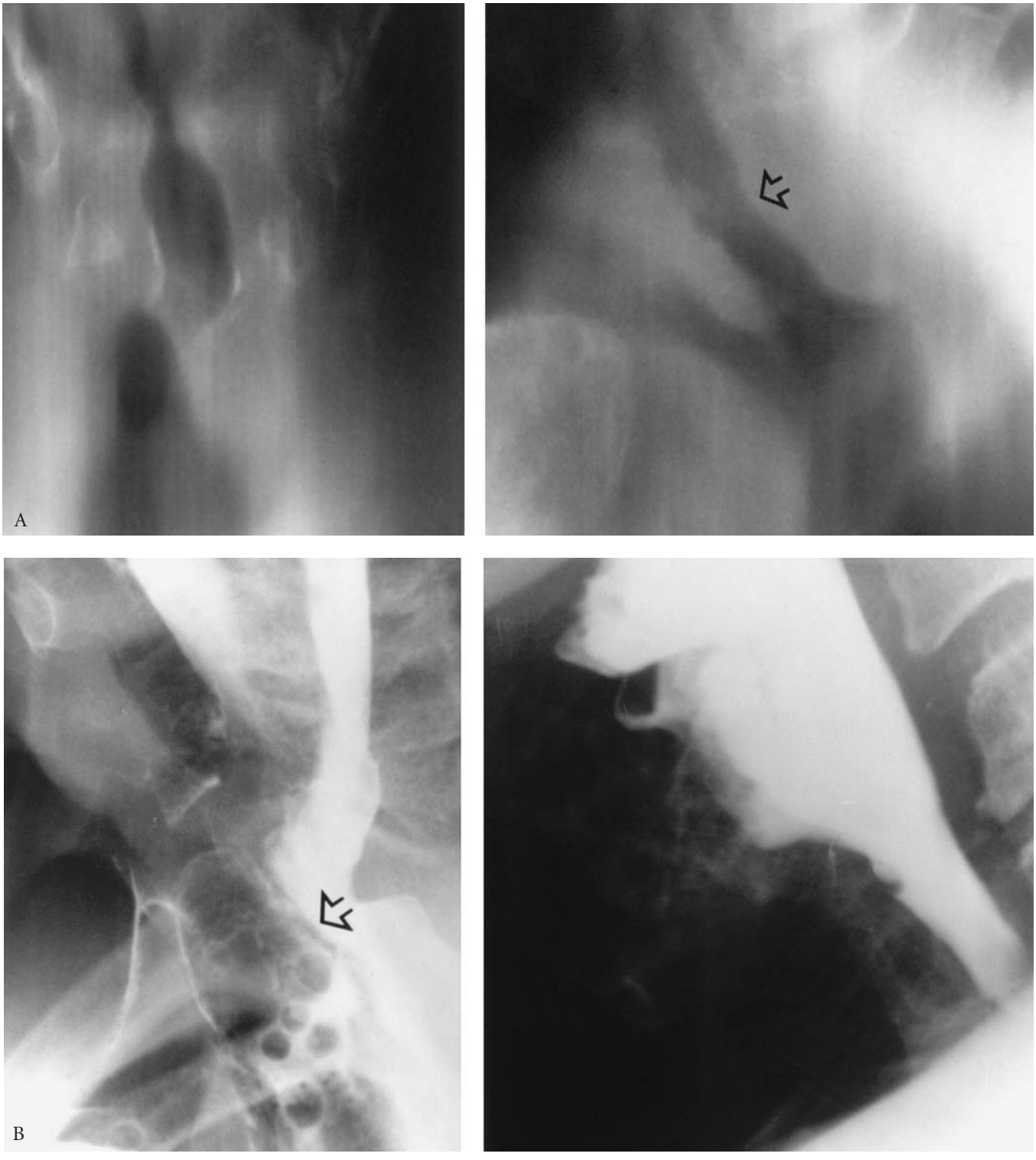


FIGURE **12-4** *Postintubation laryngotracheal stenosis with tracheoesophageal fistula (TEF) in a 56-year-old man. He had suffered cricoidostomy and failed TEF repair prior to referral.* A, *Tomograms of stenosis. Anteroposterior view on the* left *shows a deformed larynx with maximum stenosis at the laryngotracheal junction. The lateral view on the* right *demonstrates a narrowed subglottic larynx, with the most severe stenosis just below this* (arrow) *and above the stomal tract.* B, *Barium swallow in the same patient. The preoperative view on the* left *shows the fistula* (arrow). *On the* right *is a postoperative view of the repaired esophagus. Laryngotracheal resection of the stenosis and reconstruction were performed at the same time as closure of the fistula. There is no aspiration. Strap muscle was interposed between the two suture lines.*

computed tomography (CT) scan or, most definitively, by ultrasonography, an abnormality is likely to be seen bronchoscopically; that is, compression, induration, granularity, or infiltration. If tumor is identified or suspected, the possibility of a fistula may be anticipated. Cough, hemoptysis, fever, and aspiration all signal malignant fistulization. Bronchoscopy with the use of methylene blue and contrast esophagography will show the communication. Esophagoscopy is often less definitive because of the bulk of tumor adjacent to the fistula. The fistula may be tracheal (53%) or bronchial (38%), and a few are pulmonary (6%).[11] Frequently, the fistula follows prior radio- or chemotherapy, as might be expected, since treatment destroys tumor which had previously destroyed normal tissue. Although the disease may well be localized rather than disseminated at the time of manifestation of a fistula, progression of aspiration, pneumonia, lung abscess, and asphyxiation can be rapid. The clinical course typically is measured in weeks and months.

Management

It hardly needs to be said that major efforts must be made in all of these patients to clear up local and pulmonary sepses and to improve nutrition prior to surgical procedures. *Benign fistulae not related to ventilation* are individually managed depending on their cause, size, location, and degree of surrounding pathology. Cervical (with the possibility of partial upper sternal division) or, less frequently, right transthoracic approaches are used, depending upon the level of the fistula (Figure 12-7). Only a supracarinal fistula requires thoracotomy. Principles of closure of the TEF include complete dissection of the fistula, its division, effectively planned

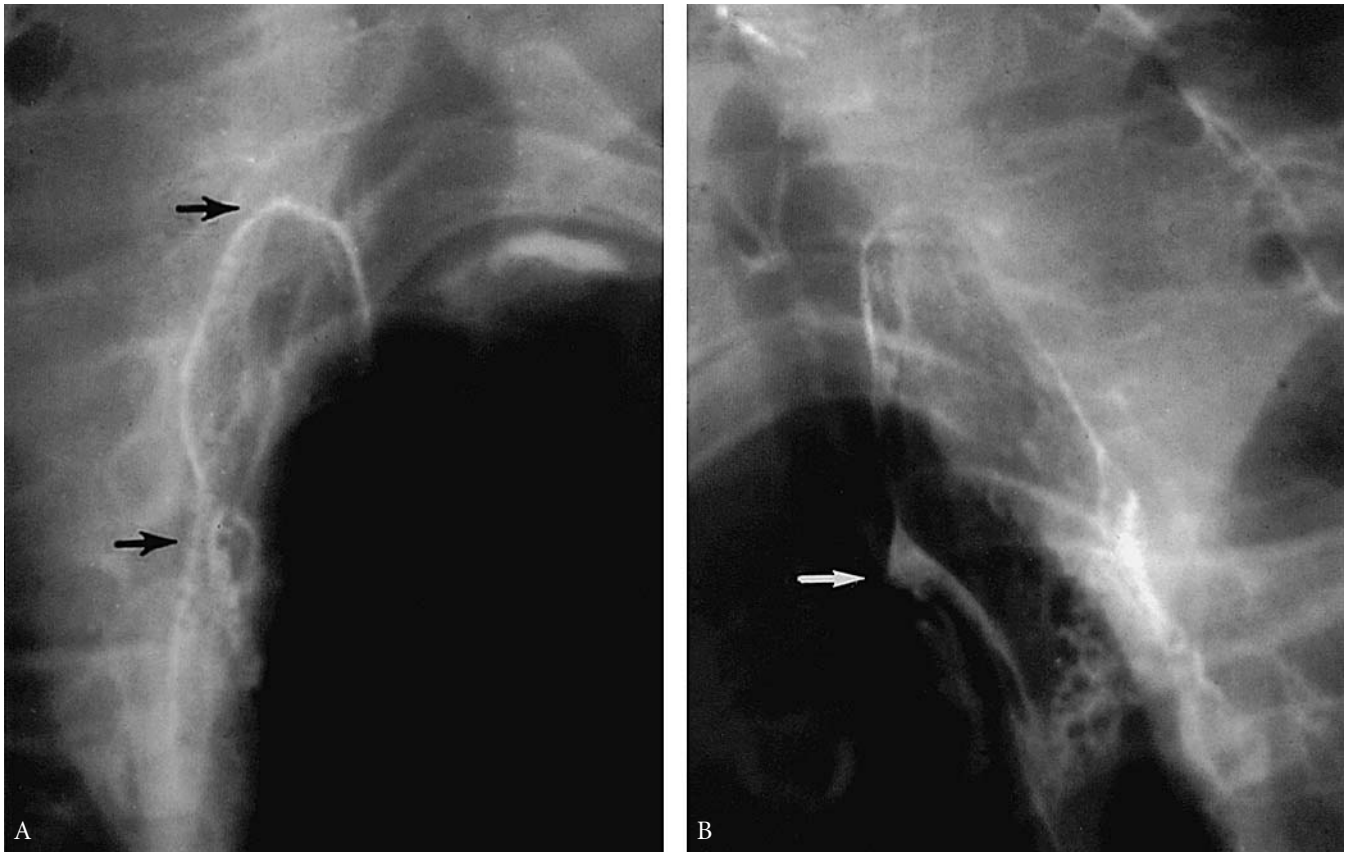


FIGURE 12-5 *Small chronic upper tracheoesophageal fistula in a 66-year-old woman resulting from foreign body ingestion at age 8, which when she was age 25 was endoscopically removed by Dr. Chevalier Jackson.* A, *The* upper arrow *in the contrast esophagogram indicates the cricopharyngeus, and the* lower arrow *indicates the fistula, in anterior view.* B, *Lateral view; fistula is indicated by the* arrow.

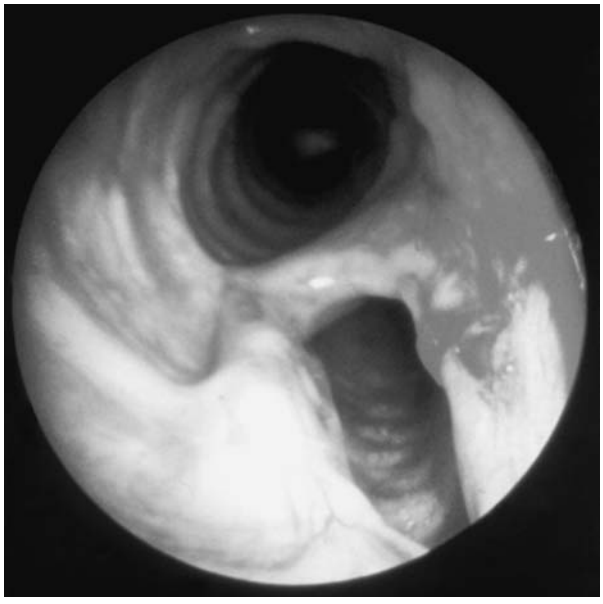


FIGURE **12-6** *Bronchoscopic view of postintubation tracheoesophageal fistula. Note the large size of the membranous wall defect and also that there is circumferential tracheal damage at the level of the fistula. Normal tracheal rings are visible distally. One-stage repair was done with esophageal closure and tracheal resection and reconstruction. Also, see Figures 31 and 32 (Color Plate 15).*

membranous wall suture closure which is tension-free, and two-layered esophageal closure (see Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula").[14] Recurrent fistulization is avoided by interposition of healthy pedicled tissue (such as a strap muscle in the neck or intercostal muscle in the chest) between the tracheal and esophageal suture lines. The technique of borrowing adjacent esophageal wall to facilitate closure without tension is also discussed in Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula." Recurrent laryngeal nerves must be carefully avoided. If the fistula is of any extent, the trachea may be best and most safely managed by resection of the segment containing the fistula, with end-to-end tracheal anastomosis after esophageal closure, even if circumferential tracheal damage is not present.

Post-traumatic fistulae may be extensive, accompanied by mediastinal injury and infection. Decision on therapy in these injuries must be individualized (see Chapter 9, "Tracheal and Bronchial Trauma"). In the most severe and delayed post-traumatic cases, esophageal exclusion may have to be considered, an alternative that is usually unnecessary in other types of TEF.[14] Surgical technique is described in Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula."

Postsurgical fistulae following esophagectomy are treated with respect to the location and size of the fistula, the presence or absence of necrosis in the neoesophagus, mediastinitis, and the severity of symptoms. Treatment may range from drainage with conservative management, local tissue excision with buttressed closures, to removal of the neoesophagus, and reconstruction of a new esophageal replacement, possibly in stages.[4]

Esophagorespiratory fistula due to *necrotizing esophagitis*, from infection in immunocompromised patients, requires esophagectomy.[8]

An attempt to close a *postintubation fistula* in a patient who is still on a respirator is almost certain to fail. Prolonged ventilation after tracheal reconstruction is likely to encourage dehiscence or stenosis. These patients are best managed conservatively, with every effort made to wean them from mechanical ventilation to permit later definitive surgical repair. If an esophageal tube is present, it is withdrawn. If possible, the tracheostomy cuff is situated just below the fistula, using as little pressure as possible to obtain a seal. A draining gastrostomy is positioned to avoid aspiration of gastric contents and a jejunostomy is placed for feeding. The head of the bed is kept in an elevated position. Vigorous efforts are made to clear any pulmonary infection. Under this regimen, the situation usually improves quite rapidly. The small amount of saliva that continues to trickle into the respiratory tree seems to be handled comparatively well with the help of frequent

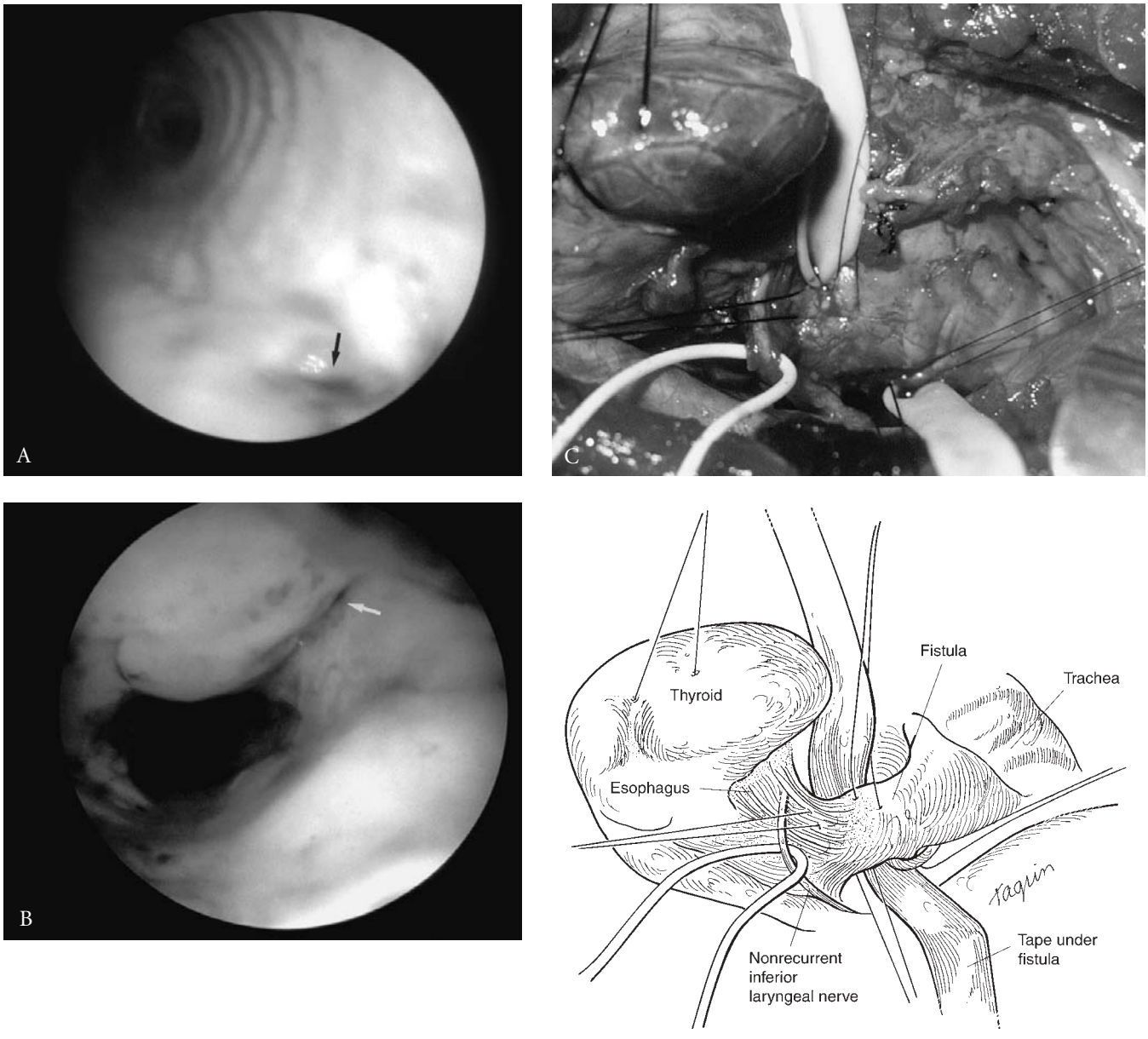


FIGURE 12-7 *Chronic fistula due to foreign body ingestion in childhood shown in Figure 12-5. Endoscopic findings and treatment.* A, *Bronchoscopic visualization of the small fistula* (arrow) *demonstrated in Figure 12-5, due to foreign body erosion. Compare this with the postintubation tracheoesophageal fistula in Figure 12-6.* B, *Esophagoscopic view of the same fistula* (arrow). *The small lesion is difficult to see in the esophageal folds.* C, *Right cervical operative exposure. The diagram clarifies the anatomy. A Penrose drain passes beneath the fistulous tract, which emerges from the posterolateral wall of the trachea on the right. Sutures have been placed in the tract closer to the esophagus* (on the left) *to leave more tissue for closure of the tracheal wall. The vascular loop passes beneath a nonrecurrent right inferior laryngeal nerve. This was critical since the left nerve had been injured in a prior failed attempt at another hospital to close the fistula from the left side. The right lobe of thyroid is retracted with a heavy suture at the left. Pedicled sternohyoid muscle was interposed between the tracheal and esophageal suture lines.*

tracheal suctioning. Esophageal diversion is almost never necessary. If it is required under highly unusual circumstances and is feasible, a disconnecting procedure is preferred to in-continuity esophagostomy. The proximal end of the esophagus is brought out laterally (left neck) as a salivary fistula and the distal end turned in with care. Since most of these fistulae are high in location, the point of division should be immediately above the fistula to simplify later reconstruction by leaving sufficient proximal esophagus. More often

than not, however, the fistula is located so close to the cricopharyngeus that exteriorized esophagostomy is impossible. The lower end of the esophagus at the gastric inlet should *not* be ligated, stapled, or divided. Continuous suction on the gastrostomy is usually sufficient to protect the trachea from reflux of gastric juices. The gastrostomy also serves to keep the stomach from becoming distended.

After weaning, surgical correction includes closure of the esophageal fistula in layers, resection of the circumferentially damaged tracheal segment and its reconstruction, plus interposition of viable tissue between the two suture lines. This is all performed in a single stage (Figure 12-8).[15,16] Even though the transverse tracheal anastomotic suture line and the vertical esophageal suture line may be at different levels, it always seems safer to use an interposition flap, as described. I have seen no difficulties arising from these flaps. The precise technique of repair and methods of dealing with special technical problems are detailed in Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula." In rare cases where the tracheal injury is too long to permit tracheal reanastomosis, the esophagus is closed nonetheless to eliminate the fistula and tracheal patency, and function is restored with a permanent T tube. If laryngotracheal stenosis is present, that is managed in the usual way for such lesions after closure of the esophagus (see Figure 12-4) (see Chapter 25, "Laryngotracheal Reconstruction").

In the past, it was recommended by some authors that the esophageal aperture be closed in an initial operation, and that any tracheal process be dealt with at a second procedure.[2] There is no justification for this approach.[14–16] Not only does the tracheal lesion require resection in any case, but both the proposed initial and later operations become much more difficult if staged.

Malignant fistula is most often best treated by palliative bypass intubation, given the patient's limited expectation for life. Exclusion of a segment of fistulized esophagus, with concomitant intestinal bypass of the esophagus, is only very rarely advisable either in a patient in very good condition or in

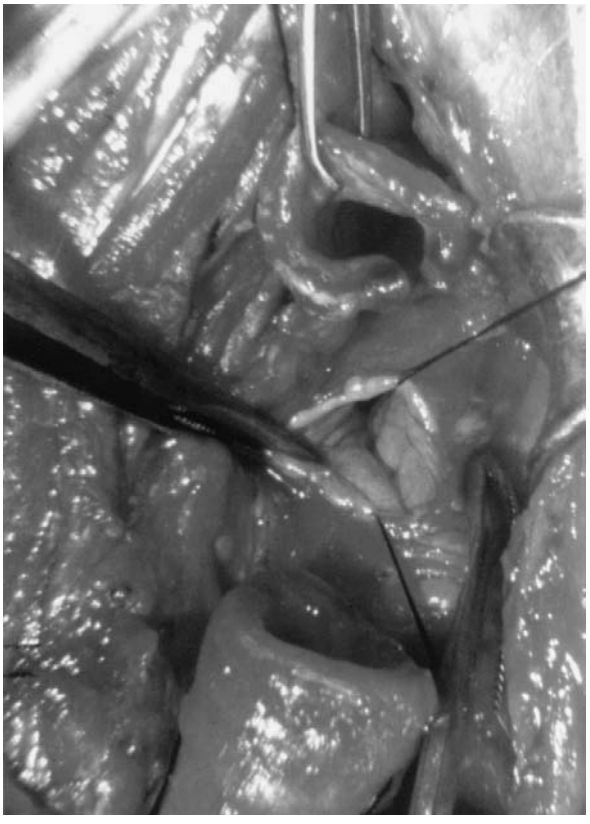


FIGURE **12-8** *Operative repair of postintubation tracheoesophageal fistula (TEF). At this stage, the circumferentially damaged segment of trachea wherein the TEF was located has been resected. An Allis forceps elevates the proximal end of trachea. The esophagus, held in forceps, is ready for meticulous closure. Sutures identify the proximal and distal margins of the defect. Tracheal anastomosis will then be done, after pedicled sternohyoid muscle interposition over the esophageal suture line. (See Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula," for operative details.)*

one with a rare slow-moving tumor of unusual type. An intermediate alternative of intubation with partial exclusion by esophagostomy and with gastrostomy plus jejunostomy (for prevention of aspiration and for nutrition) may be considered, with the possibility of a later restitution of gastrointestinal continuity by substernal gastric or colonic transposition. Duranceau and Jamieson carefully reviewed these options in 1984, and Burt and colleagues described their experiences in 1991.[11,17] In a terminal and debilitated patient, abstinence from any intervention may be the kindest therapeutic choice. If intubation through the tumor is elected, pulsion rather than traction (via a gastrostomy) seems to be the best palliative maneuver. Intubation techniques are not without morbidity and mortality. Tubes must be of impervious material or be coated if of an expandable type, in order to prevent prompt ingrowth of tumor through interstices.

In 1920, Kirschner used the stomach anastomosed to the cervical esophagus to bypass a malignant fistula.[18] Many variations have since been employed, using the stomach, jejunum, and colon for interposition as well as extracorporeal synthetic tubes. An esophagus excluded above the fistula, and below by occlusion of the gastroesophageal junction, has too often produced copious secretions, leaked, or ruptured. A residual esophagus should therefore be drained with a loop or arm of jejunum if left in situ. A preferable technique is to divide the esophagus just below the fistula as well as above, creating a smaller "diverticulum" at the fistulous site, and excise the distal esophagus (via a transhiatal approach). The stomach and colon are the favored gastrointestinal replacement conduits, and are placed substernally (see Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula").[11,17,19] Each patient must be carefully evaluated and treatment individualized, bearing in mind that *any* treatment will at best be palliative and usually for a short term. If radical treatment is to be offered, it should be instituted promptly.

Results

Benign Acquired Fistula. The difficult problem of treatment of a benign acquired tracheoesophageal fistula, and the particularly threatening one of fistula due to intubation and ventilation, may be successfully managed for the most part by attention to the principles stated and using the techniques detailed in Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula." The relatively few reports of any number of patients treated in accordance with these principles, which were enunciated by Grillo and colleagues in 1976, are all encouraging.[15] Couraud and colleagues, Dartevelle and Macchiarini, and Mathisen and colleagues, in a total of 78 patients, summarized by Dartevelle and Macchiarini, performed simple closure of fistula in 29, closure with tracheal resection in 44, and diversion in only 5 patients.[14,20,21] Recurrences of TEF were at the rate of 6.4 to 8.3% and mortality at 6.3 to 12.5% (Table 12-1). Macchiarini and colleagues commented on the superiority in their experience of

Table 12-1 Results of Operative Repair of Postintubation Tracheoesophageal Fistulae (TEF)

		Type of Operation				
Author	*Number of Patients*	*Simple Closure*	*TR + EC*	*ED*	*TEF Recurrence (%)*	*Mortality (%)*
Mathisen et al[14]	38	9	29	0	3 (7.9)	4 (10.5)
Couraud et al[20]	16	9	5	2	0	1 (6.3)
Dartevelle and Macchiarini[21]	24	11	10	3	2 (8.3)	3 (12.5)
Total	78	29	44	5	5 (6.4)	12 (10.3)

Data from Dartevelle P and Macchiarini P.[21]

EC = esophageal closure; ED = esophageal diversion; TR = tracheal resection.

the anterior approach as well as the definitive single-stage repair by our technique over other types of surgical repairs of varying complexity.[15,22]

In our series of 38 patients, 27 TEFs resulted from ventilation, 5 from laryngotracheal trauma, 2 from anterior spine fusions, and 2 from irradiation after prior laryngectomy for cancer.[14] Foreign body and possible congenital origin accounted for 2 more. Eight had one or more prior failed repairs, 7 had esophageal diversions, 3 had occlusion of the esophagogastric junction, and 1 had colon interposition. Approaches were by collar incision in 26, partial sternotomy in 10, and complete sternotomy or lateral thoracotomy in a few patients with special problems. Tracheal resection was done in 31 patients and laryngotracheal resection and reconstruction in 5. Laryngeal release was necessary in only 2 patients. Where there was insufficient tracheal mucosa for tracheal closure, adjacent esophageal mucosa was borrowed (see Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula"). In a patient where there was insufficient length of trachea for reconstruction, the TEF was closed, the repair buttressed, and the airway reestablished with a permanent T tube. Prior cervical esophagostomy necessitated end-to-end esophageal anastomosis in 5 patients.

Three deaths followed transthoracic repair of distal fistulae in the face of mediastinal sepsis (due to trauma), and 1 patient died from dehiscence after an extended tracheal resection. Two patients had successful reoperation for recurrent TEF and one healed a further small recurrence spontaneously. Two patients needed temporary tracheostomy. Postoperative aspiration in some patients gradually resolved. Thirty-three of 34 operative survivors were successful. Three of 4 with vocal cord problems had these conditions prior to operation.

These outcomes of success in patients, many made more complex by prior failed surgery, indicate that generally successful results can be obtained in this difficult group of patients. Clearly, esophageal diversion is almost never necessary and, equally clearly, a single-staged procedure is indicated where tracheal injury accompanies the TEF. Closure of a fistula should be accomplished after a patient has been weaned from the respirator.

Malignant Fistula. In their review, Duranceau and Jamieson cited complication and mortality rates in the order of 28 to 38% and 6 to 17%, respectively, for "push through" palliative intubation.[17] Control of aspiration pneumonia is not universal and does not last as the disease progresses. Early death still occurs in many patients, although with the best results, symptoms are controlled for a time and oral ingestion becomes possible. There is no clear-cut advantage to the older "pull through" method; it has the disadvantages of another procedure and probably more complications.

Burt and colleagues found a median survival of 5 weeks (35 days) in 207 patients receiving all modes of treatment.[11] The primary neoplasm and location of the fistula were not determinants of survival. Specific treatment extended the median survival from 22 to 47 days. Endoprosthesis did not alter the median survival. Supportive care included all minor methods but not functional exclusion of the fistula. Chemotherapy and radiotherapy alone led to slightly longer survival, probably by eliminating the negative effects of procedures. Esophageal exclusion alone was no better, but gastric or colonic bypass, done in a small number of patients, increased survival to a 77-day median, a significant but not overwhelming improvement in exchange for very extensive surgery. The overall 30-day mortality was 46%. Pulmonary sepsis was the principal cause of death in over 80%, with bleeding in 12%.

Bronchoesophageal Fistulae

Bronchoesophageal fistulae (BEF) are rare lesions, either congenital or acquired. The congenital fistulae can be insidious and may go unrecognized for years. They are more frequently recognized in the adult than in the child. Diagnosis of both congenital and acquired lesions depends upon a high index of suspicion.

Congenital Bronchoesophageal Fistula

Braimbridge and Keith classified BEF into four types (Figure 12-9).[23] Type I seems to result, at least sometimes, from inflammatory changes in an esophageal diverticulum, which then secondarily fistulizes to a bronchus. Although some diverticula are undoubtedly congenital, traction diverticula are also likely represented in this group of lesions and the fistula itself may not therefore always be of congenital origin. Nonetheless, I retain their classification of "congenital" BEF's. Type I presents with a small fistula, at times inflamed, at the tip of an esophageal diverticulum. Type II is a simple fistula extending from the esophagus at an upward angle to a bronchus. This is by far the most common type. Next in incidence is type III, where the fistula connects to a cyst in the lung and thence to bronchi. Type IV is a fistula to a sequestrated lung, which is supplied by a systemic artery. The tract runs most commonly from the middle third of the esophagus, and less often from the lower third to the right lower lobe (either segmental or lobar bronchus), while half as often to the bronchi of the left lower lobe and in diminishing frequency to the bronchus intermedius, left main bronchus, right middle lobe, and right upper lobe bronchi. In a review of 100 cases reported up to 1990, Risher and colleagues found only 5 cases of type III and 3 of type IV.[24] Congenital fistulae are characterized by lack of inflammation, absence of adherent or inflamed lymph nodes, and a tract lined with squamous or columnar mucosa with adjacent muscularis mucosa.

Clinical Presentation and Diagnosis. Distribution of patients is about equal for males and females, with 75% of patients over 17 years of age. Paroxysmal cough, cough on ingestion of food especially after liquids, frequent respiratory infections, hemoptysis, and hematemesis are found.[25–27] Symptoms may be largely of cough and respiratory infection for many years, leading to a delay in diagnosis from 5 to 30 years. Retrospectively, a long history is usually identified. Delay in appearance of severe symptoms has been attributed to the oblique course of the tract and to its possible initial obstruction either by a thin membrane, which later ruptures after inflammation, or to a mucosal flap valve in the tract. Marked delay in the discovery of a fistula can lead to death from recurrent pulmonary suppuration. Treatment is therefore urged as soon as diagnosis is made.

Diagnosis is made by contrast esophagography (Figure 12-10) and bronchoscopy, sometimes with methylene blue instillation into the esophagus. Esophagoscopy is less often definitive. Insufflation of gases into the trachea during esophagoscopy may aid in pinpointing a tiny fistula. Contrast should be introduced for esophagography with the patient in a position where cough ordinarily follows oral ingestion, rather than with the patient in a recumbent position. CT scan helps to assess pulmonary damage.

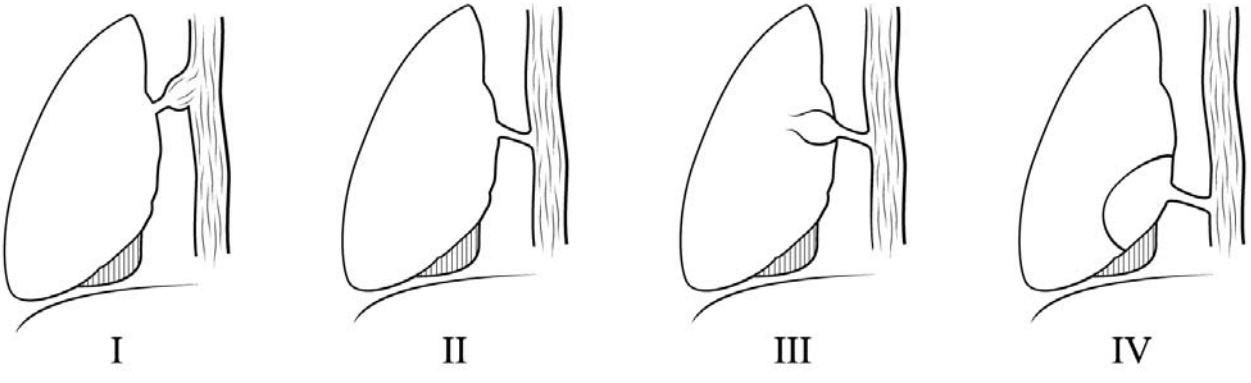


FIGURE **12-9** *Braimbridge and Keith's classification of congenital bronchoesophageal fistula: type I, wide neck diverticulum with inflammatory fistula at tip; type II, simple fistula, the most common type; type III, fistula with cyst; type IV, fistula with sequestration of lung.*[23]

Treatment. Despite a few reports of alternative treatments, surgical excision of the tract and closure of the fistula at either end is the best choice (Figure 12-11). Right or left thoracotomy is selected according to the location of the fistula. Two layers of 4-0 Vicryl sutures are advised for closure of the esophageal end, or one layer of staples with a sutured second layer. One layer of Vicryl is used on the bronchial side. A substantial flap of healthy tissue, such as a pericardial fat pad or intercostal muscle, is pedicled between the two suture lines. Simple stapling without division of the tract is likely to result in recurrent fistula. Any irremediably

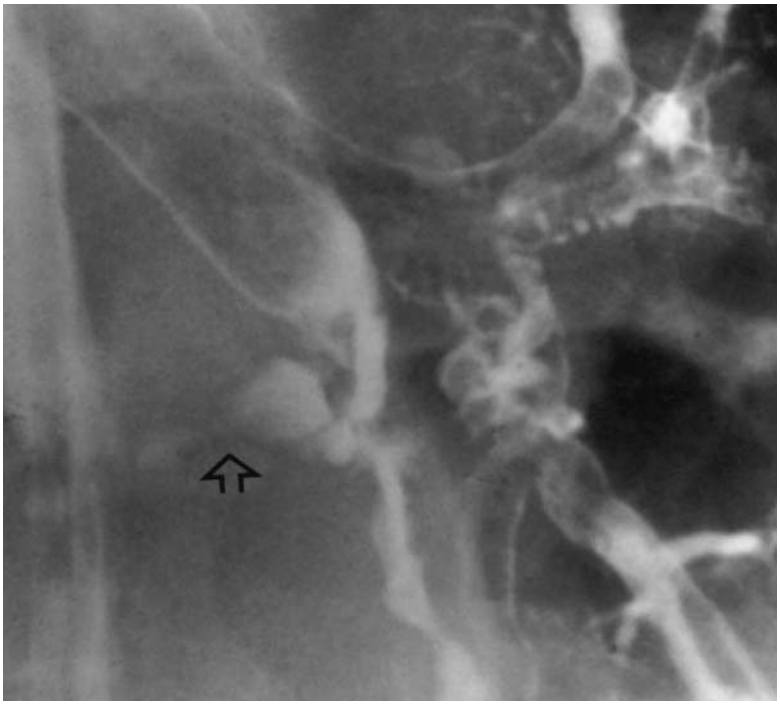


FIGURE **12-10** *Contrast esophagogram demonstrating congenital bronchoesophageal fistula* (arrow) *in a 65-year-old woman. The fistula opened into the left lower lobe bronchus just medial to the superior segmental bronchus.*

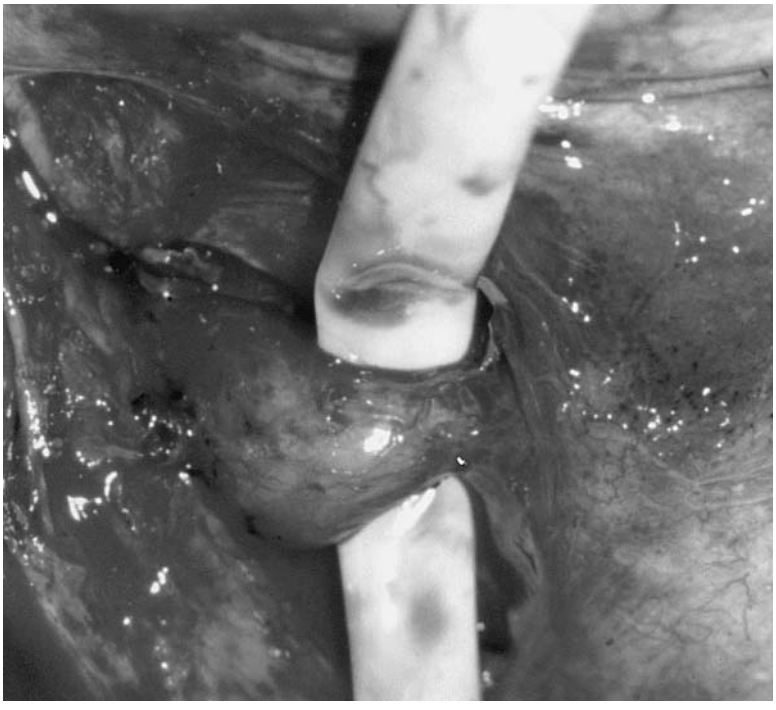


FIGURE **12-11** *Operative repair of type I congenital bronchoesophageal fistula via left thoracotomy. A Penrose drain encircles the dissected fistula and esophageal diverticulum. The esophagus is at the left, the lung at the right. The diverticulum was excised, both sides of the channel sutured, and intercostal muscle pedicled between.*

injured portion of lung is resected at the same time. This has been necessary in many cases. Results of treatment are generally excellent.

Acquired Bronchoesophageal Fistula

Bronchoesophageal fistula can result from trauma or infection or rare additional causes.[1,28–30] Blunt chest trauma has produced a BEF, although the cervical or supracarinal trachea is more likely to be a locus of posttraumatic esophagorespiratory fistula than a bronchus. Instrumentation (such as variceal sclerosis), chemical burns (lye ingestion), and foreign bodies have all been implicated. Infectious agents associated with fistula include tuberculosis, histoplasmosis (Figure 12-12), actinomycosis, and syphilis. Often, these are associated with inflammatory lymph nodes or broncholithiasis. Nontuberculous empyema, suppurative esophagitis, and infected bronchogenic cysts are other etiologies. Fistula has also been associated with traction diverticula related to lymph nodes. Fistulae have occurred due to necrotizing vasculitis and silicotic nodules.[1,28–30]

Clinical presentation, *diagnosis*, *and treatment* are much the same as described for congenital BEF. *Results* of surgical treatment are very satisfactory (Figure 12-13).[28–30] Since more inflammation may be encountered, and more dissection may be necessary to remove involved lymph nodes than in uninflamed congenital fistulae, I favor interposition of an intercostal muscle flap in these cases. The flap is raised at the time of initial thoracotomy. An associated esophageal diverticulum is resected and the esophagus repaired in two layers. Concomitant pulmonary resection is dictated by irretrievable lung damage from chronic infection.

In our series of 9 patients collected over 41 years, 4 followed thoracic surgery, 3 were due to histoplasmosis, and 1 each were due to silicosis, foreign body, lye ingestion, bronchogenic cyst, and esophageal diverticulum, respectively.[30] One BEF was congenital. The patient with lye ingestion succumbed. There were no recurrences after successful surgical closures, performed as described above. During this same period of time, 215 patients were recorded with BEF due to bronchogenic or esophageal malignancy.

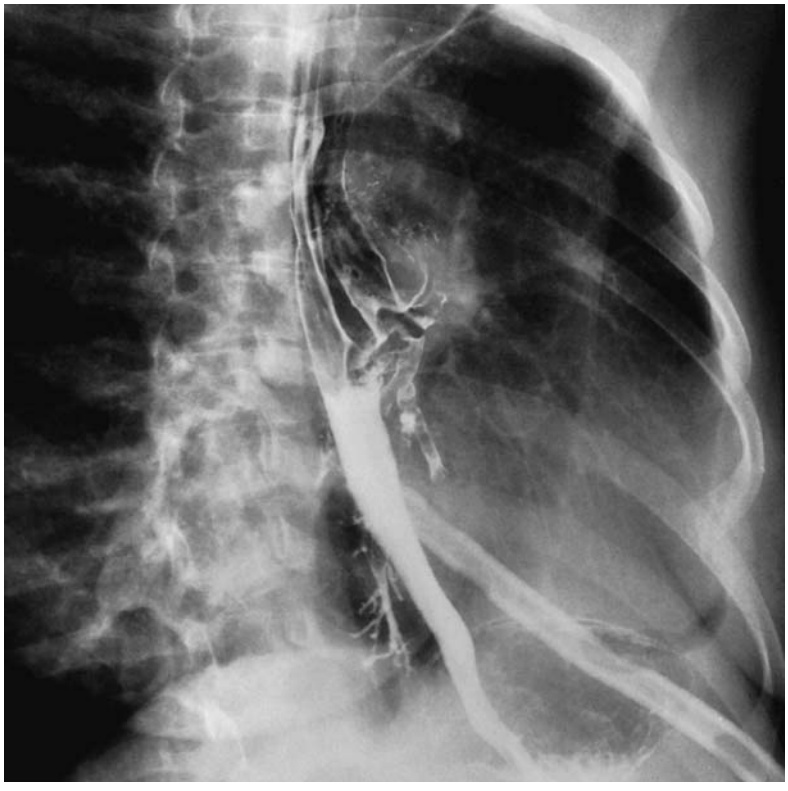
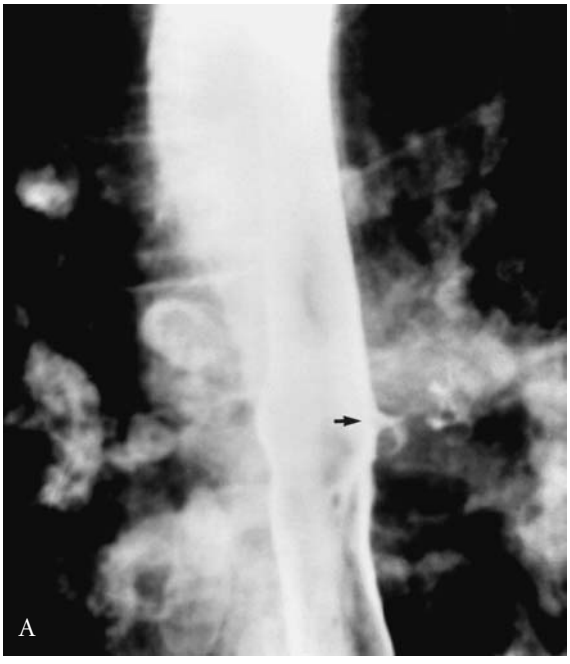


FIGURE **12-12** *Bronchoesophageal fistula due to histoplasmosis. (Courtesy of Dr. Delos M. Cosgrove III.)*



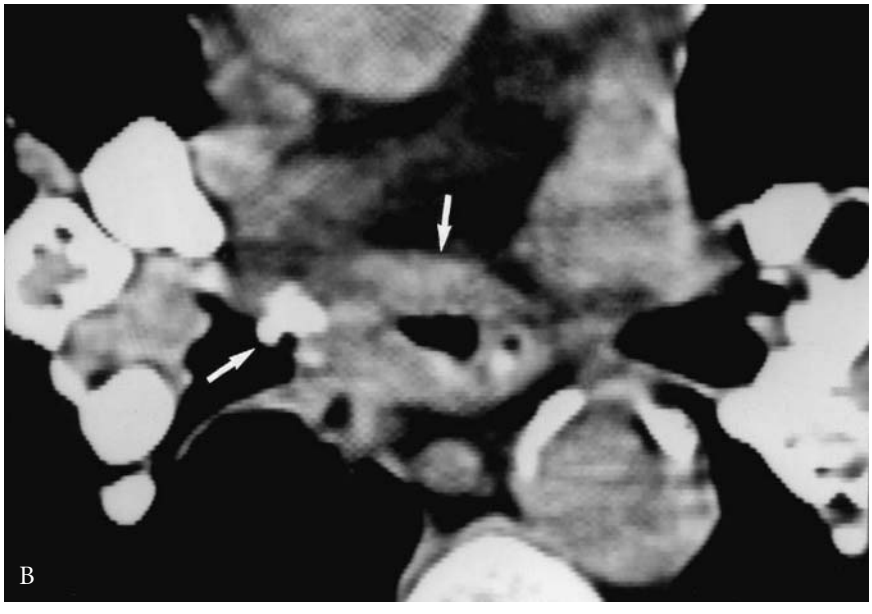


FIGURE **12-13** *Acquired bronchoesophageal fistula due to broncholithiasis from silicosis involving mediastinal lymph nodes.* A, *Barium swallow demonstrates a fistula* (arrow) *from the esophagus to the bronchus intermedius.* B, *Computed tomography section shows irregular thickening of the esophageal wall* (arrow) *and a calcified lymph node protruding into the bronchus intermedius* (arrow).

References

1. Wesselhoeft CW Jr, Keshishian JM. Acquired nonmalignant esophagotracheal and esophagobronchial fistula. Ann Thorac Surg 1968;6:187–95.
2. Thomas AN. The diagnosis and treatment of tracheoesophageal fistula caused by cuffed tracheal tubes. J Thorac Cardiovasc Surg 1973;65:612–9.
3. Macchiarini P, Delamore N, Beuzeboc P, et al. Tracheoesophageal fistula caused by mycobacterial tuberculosis adenopathy. Ann Thorac Surg 1993;55:1561–3.
4. Buskens CJ, Hulscher JBF, Fockens P, et al. Benign tracheo-neo-esophageal fistulas after subtotal esophagectomy. Ann Thorac Surg 2001;72:221–4.
5. Fujita H, Kawahara H, Hidaka M, et al. An experimental study on viability of the devascularized trachea. Jpn J Surg 1988;18:77–83.
6. Schawengerdt CG. Tracheoesopahgeal fistula caused by a self-expanding esophageal stent. Ann Thorac Surg 1999; 67:830–1.
7. Gaissert HA, Grillo HC, Wright CD, et al. Lengthening and complication of benign tracheobronchial strictures by self-expanding microinvasive ultraflex and wall stents. J Thorac Cardiovasc Surg 2003. [In press]
8. Gaissert HA, Roper CL, Patterson GA, Grillo HC. Infectious necrotizing esophagitis: outcome after medical

and surgical intervention. Ann Thorac Surg 2003; 75:342–7.
9. Acosta JL, Battersby JS. Congenital tracheoesophageal fistula in the adult. Ann Thorac Surg 1974;17:51–7.
10. Olivet RT, Payne WS. Congenital H-type tracheoesophageal fistula complicated by achalasia in an adult. Mayo Clin Proc 1975;50:464–8.
11. Burt M, Diehl W, Martini N, et al. Malignant esophagorespiratory fistula: management options and survival. Ann Thorac Surg 1991;52:1222–9.
12. Greven KM, Evans LS. The occurrence and management of esophageal fistulas resulting from Hodgkin's disease. Cancer 1992;69:1031–3.
13. Tse DG, Summers A, Sanger JR, Haasler GB. Surgical treatment of tracheomediastinal fistula from recurrent Hodgkin's lymphoma. Ann Thorac Surg 1999; 67:832–4.
14. Mathisen DJ, Grillo HC, Wain JC, Hilgenberg AD. Management of acquired nonmalignant tracheoesophageal fistula. Ann Thorac Surg 1991;52;759–63.
15. Grillo HC, Moncure AC, McEnany MT. Repair of inflammatory tracheoesophageal fistula. Ann Thorac Surg 1976;22:113–9.
16. Hilgenberg AD, Grillo HC. Acquired non-malignant tracheoesophageal fistula. J Thorac Cardiovasc Surg 1983;85:492–8.
17. Duranceau A, Jamieson GG. Malignant tracheoesophageal fistula. Ann Thorac Surg 1984;37:346–54.
18. Kirschner MB. Ein neues Verfohren der Oesophagoplastie. Arch Klin Chir 1920;114:606.
19. Symbas PN, McKeown PP, Hatcher CR Jr, Vlasis SE. Tracheoesophageal fistula from carcinoma of the esophagus. Ann Thorac Surg 1984;38:382–6.
20. Couraud L, Bercovici D, Zanotti L, et al. Traitement des fistules oesophago-trachéales de la réanimation. Ann Chir 1989;43:677–81.
21. Dartevelle P, Macchiarini P. Management of acquired tracheoesophageal fistula. Chest Surg Clin North Am 1996;6:819–36.
22. Macchiarini P, Verhoye JP, Chepelier A, et al. Evaluation and outcome of different surgical techniques for post-intubation tracheoesophageal fistulas. J Thorac Cardiovasc Surg 2000;119:268–76.
23. Braimbridge MV, Keith HZ. Oesophago-bronchial fistula in the adult. Thorax 1965;20:226–33.
24. Risher WH, Arensman RM, Ochsner JL. Congenital bronchoesophageal fistula. Ann Thorac Surg 1990;49:500–5.
25. Azoulay D, Regnard JF, Magdeleinat P, et al. Congenital respiratory esophageal fistula in the adult. Report of nine cases and review of the literature. J Thorac Cardiovasc Surg 1992;104:381–4.
26. Rämö OJ, Salo JA, Mattila SP. Congenital bronchoesophageal fistula in the adult. Ann Thorac Surg 1995;59:887–90.
27. Kim JH, Park K, Sung SW, Rho JR. Congenital bronchoesophageal fistula in adult patients. Ann Thorac Surg 1995;60:151–5.
28. Wychulis AR, Ellis FH Jr, Andersen HA. Acquired nonmalignant esophagotracheobronchial fistula. JAMA 1966;196:117–22.
29. Spalding AR, Burnery DP, Richie RE. Acquired benign bronchoesophageal fistulas in the adult. Ann Thorac Surg 1979;28:378–83.
30. Mangi AA, Gaissert HA, Wright CD, et al. Benign bronchoesophageal fistula in the adult. Ann Thorac Surg 2002;73:911–5.

Tracheal Fistula to Brachiocephalic Artery

Hermes C. Grillo, MD

A fistula between the trachea and brachiocephalic (innominate) artery is a rare complication either of tracheostomy, most often in conjunction with ventilatory support,[1,2] or of tracheal reconstructive surgery,[3,4] or exenteration. External trauma can rupture the artery, produce a false aneurysm or, less often, result in fistula to a lacerated trachea.

Post-Tracheostomy Fistula

Two types of tracheoarterial fistulae occur from tracheostomy. The first is due to erosion of the artery lying immediately beneath the curve of the tracheostomy tube. The second is caused by erosion of the anterior tracheal wall into the artery by either the cuff or the tip of the tracheostomy tube. The two lesions must be kept clearly in mind, since the emergency and definitive management of each is different (see Figures 27-1 through 27-3 in Chapter 27, "Repair of Tracheobrachiocephalic Artery Fistula"). Fortunately, the incidence of both of these potentially disastrous lesions has diminished, the first by avoiding errors in the technique of tracheostomy, and the second because of development of large-volume, low-pressure cuffs and their correct usage.

Erosion by a Tracheostomy Tube

In children and younger adults, hyperextension of the neck delivers half or more of the trachea into the neck, and the brachiocephalic artery also rises into the base of the neck. If tracheostomy is made with reference to the sternal notch, as was taught in the past, the stoma will be in the midtrachea and will lie just above the artery. The combined thrust of the respirator on the tube, failure to suspend the tube, and arterial pulsation can rapidly lead to arterial erosion by the elbow of the tube. The point of hemorrhage will be at the inferior margin of the stoma. In an emergency, digital pressure is applied at this point to control hemorrhage. Pressure is directed downward and forward against the sternum (see Chapter 27, "Repair of Tracheobrachiocephalic Artery Fistula"). The fistula may occur surprisingly soon after tracheostomy has been done, sometimes in days, although more often in weeks.

This catastrophe is *prevented* by correctly locating the tracheostomy at the level of the second and third tracheal rings, using the almost-always palpable cricoid as a landmark. It makes no sense to use the fixed point of the sternal notch with respect to a movable structure, the trachea. In a much less common situation, the uppermost trachea may be nearly contiguous with the brachiocephalic artery in an aged patient with marked kyphosis and very limited cervical extension.

Erosion by a Tracheostomy Tube Cuff or Tip

If a tracheostomy is correctly located at the level of the second and third rings, the ventilating cuff or tube tip very often lies behind the point where the brachiocephalic artery crosses the trachea, at about the ninth tracheal ring. If high-pressure cuffs are used, circumferential erosion is sometimes accompanied by sufficient anterior tracheal damage to produce a tracheoarterial fistula. Most often, these lesions follow prolonged ventilation. Less often, a tracheostomy tube that is angulated forward eccentrically, probably due to the distorted way in which a high-pressure cuff might expand, erodes the tracheal wall at this critical point (Figure 13-1). A 90° tracheostomy tube is more likely to be thus angulated. Although we continue to see concentric cuff injury by large-volume cuffs used in a high-pressure range (by overinflation), extreme anterior damage seems to occur only rarely nowadays. In these patients, hemorrhage occurs directly into the trachea at a point not accessible to the finger. Emergency control must be obtained by tamponade, by overinflating the tracheostomy tube cuff. An endotracheal tube with an overinflated cuff is more satisfactory since it can be positioned more easily. Management of a post-tracheostomy arterial fistula and of the trachea is detailed in Chapter 27, "Repair of Tracheobrachiocephalic Artery Fistula." Large-volume, low-pressure cuffs prevent most such injuries nowadays.

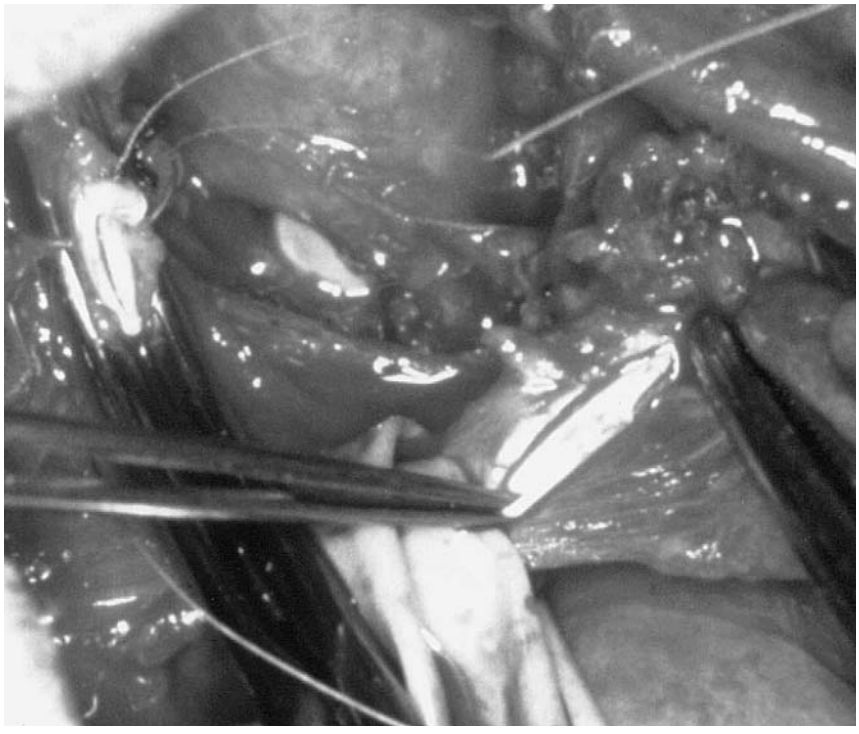
Postoperative Fistula

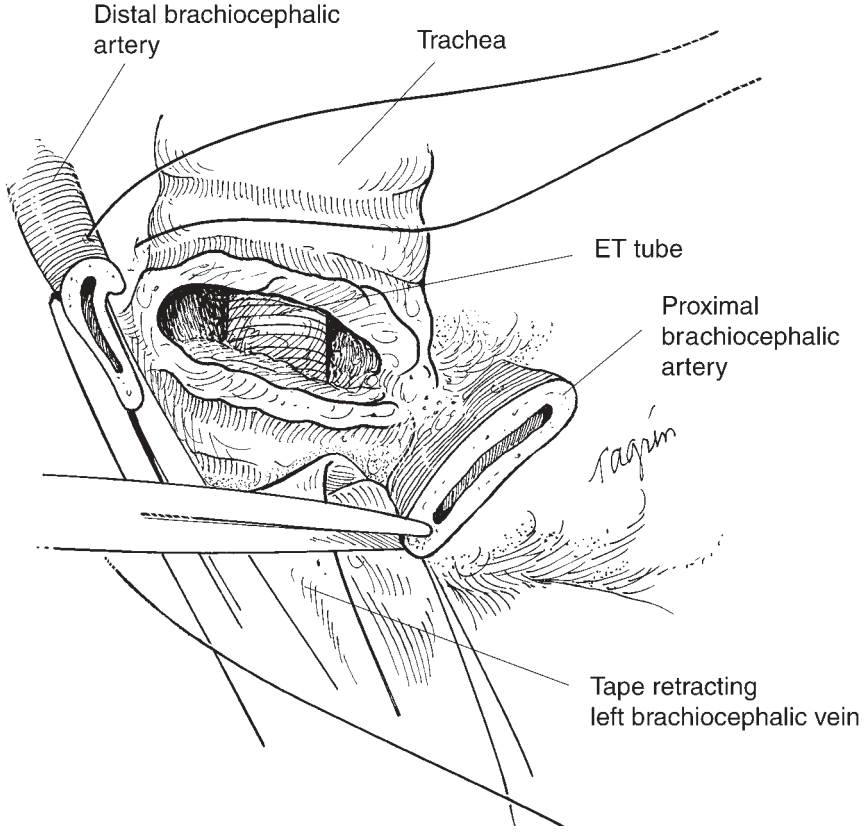
Early in the development of tracheal reconstruction, postoperative hemorrhage from the brachiocephalic artery occurred too frequently.[3,4] Since cuff stenosis often lies at the level of the artery, the vessel was frequently dissected free from scar and the tracheal anastomosis made immediately behind it. Local infection or erosion at this point of confluence could lead to bleeding. Suture material, a foreign body, probably contributes. One surgeon, who formerly used fine wire for an anterior tracheal anastomosis, attributed some fistulas to abrasion by this unyielding material. Anastomotic dehiscence after tracheal reconstruction, most often managed with subsequent intubation, may expose the artery in what is now an infected space.

If tracheal dissection is kept scrupulously close to the trachea and the artery is left undissected with its local tissue investment intact, hemorrhage will almost never follow. When the artery must be dissected because of adherence to tracheal scar, prior tracheal surgery, or in surgery for a neoplasm, it is advisable to place viable tissue, such as an inferiorly based pedicle of sternohyoid muscle or thymus, between the tracheal anastomosis and the overlying artery. The rarity of this complication is seen in its low incidence, occurring in only five (1%) of 503 patients who had tracheal resection and reconstruction for post-intubation lesions.[5] These instances occurred early in our experience.

Late brachiocephalic hemorrhage has long been the bane of mediastinal tracheostomy and exenteration. The artery would become exposed by failure of healing of the skin beneath the mediastinal tracheostomy, sometimes abetted by the effects of prior irradiation. This has largely been prevented by omental coverage or, where indicated, by prophylactic division of the brachiocephalic artery, after preoperative imaging and with intraoperative electroencephalographic monitoring (see Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy").[6]

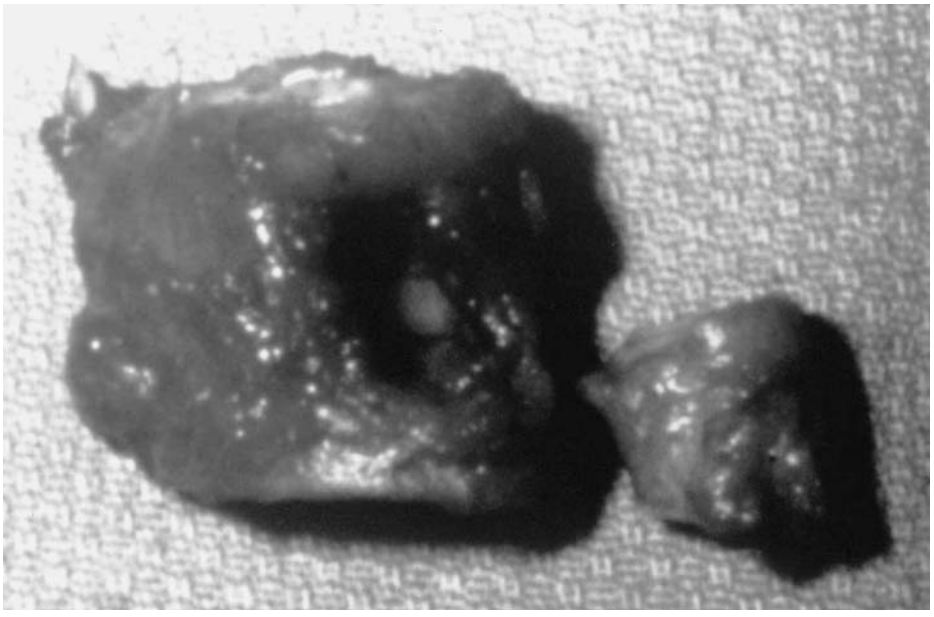
Innominate arteries or even aortic fistulae have resulted over the years from attempts to use tracheal prostheses made of various foreign materials.[3] Fixation points of Gianturco tracheobronchial stents have also produced arterial fistulae. Both demonstrate the all too obvious surgical principle of avoiding prolonged pressure on vascular structures by foreign material.





A

FIGURE 13-1 *Tracheobrachiocephalic artery fistula due to anterior erosion by tracheostomy tube cuff.* A, *Operative field after division and resection of a fistulous arterial segment. The forceps elevates the proximal brachiocephalic arterial stump. The distal artery is visible to the right of the trachea, which exhibits a sizeable anterior defect. The endotracheal tube is visible in the defect. The brachiocephalic vein is retracted caudad with a Penrose drain.*



B

FIGURE 13-1 (CONTNUED) B, *The resected segment of circumferentially damaged trachea is at the left. The anterior fistula is visible. The perforated section of the brachiocephalic artery is at the right. Both arterial ends were sutured closed and protected with thymus. A standard tracheal anastomosis was made.*

Diagnosis and Management

Bleeding immediately after a tracheostomy may result from incomplete hemostasis or from surgically injured branches of cervical vessels. Fresh bleeding thereafter, in any amount, from a tracheostomy or from a postoperative trachea should at once raise the question of innominate artery leakage. Bleeding from the innominate is often massive at the outset. Premonitory minor or limited bleeding may occur however. Only suspicion of the worst will lead to the prompt action necessary. Most instances of bleeding from a tracheostomy are due to granulation tissue, superficial ulceration, or tracheitis. This, however, must be established promptly by bronchoscopy. If bleeding ceases with overinflation of the cuff, a brachiocephalic artery fistula is most likely present. If the hemorrhage is of any moment, the examination is best done in the operating room, with personnel and equipment at hand for instant major intervention. Rigid bronchoscopy is advised, with the tracheostomy tube removed. If blood clot and anterior necrosis is observed, then operation is indicated even if bleeding has ceased at that moment. Examination by a flexible instrument through the tracheostomy tube is unlikely to be satisfactory. Angiography can be of value in showing false aneurysm or rupture but it is most often inapplicable in an urgent situation.

Both urgent and definitive surgical management of *post-tracheostomy arterial fistula* are detailed in Chapter 27, "Repair of Tracheobrachiocephalic Artery Fistula."[7] *Postoperative bleeding* demands immediate exploration with an endotracheal tube, placed so that the cuff may be tightly inflated at the anastomosis to provide tamponade. The collar incision is reopened and complete sternotomy is added so that adequate exposure is obtained for proximal and distal control of the brachiocephalic artery. Cooper preferred upper sternotomy angled off into the right third interspace.[8] This may help to avoid later sternal infection. Almost without exception, the artery is best excised, both arterial ends closed with running vascular sutures and covered with robust flaps of healthy tissue; that is, pedicled strap muscles, thymus, or omentum. If time permits, electroencephalographic monitoring can be helpful. Neurologic sequelae or subclavian "steal" syndrome becomes vanishingly rare after division of the brachiocephalic artery, as long as the carotid–subclavian

junction is intact.[2,9] We have preferred not to perform arterial reconstruction in these circumstances since the field is contaminated and often infected. In a single case, a small arterial perforation was successfully excised, arteriorrhaphy done, and the site sealed with muscle. In general, the danger of a repeat hemorrhage is high after arteriorrhaphy, grafting, or simple ligation (versus suture closure) in this situation. If anastomotic separation has occurred after some days, tracheal reanastomosis is unlikely to succeed. The tracheal defect is initially spanned with an endotracheal tube to be followed by a tailored, long T tube when ventilation is no longer necessary. Later reconstruction may be possible. The tube is compartmentalized from the sutured arterial stumps by the viable tissue placed over them.

The same principles of management, that is, arterial excision and tissue coverage, are applied to bleeding after cervicomediastinal exenteration, but the event is rare because of the prophylactic steps now advised (see Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy"). The most likely source of massive hemorrhage following a carinal reconstruction is the pulmonary artery. Routine interposition of tissue over airway anastomoses has largely prevented this disaster (see Chapter 29, "Carinal Reconstruction").

Results

Wright summarized the literature to show 70 survivors of operations for tracheo-innominate artery fistulae but with only 40 surviving more than 2 months.[10] Death is often related to the basic illness or its other complications. About 25% of patients who reach the operating room survive. As noted, neurologic problems are rare.

Prevention of a Tracheal-Arterial Fistula

Prevention of *hemorrhage after a tracheostomy* is accomplished by

1) correct placement of the tracheostomy at the level of the second or third cartilaginous rings;
2) avoidance of sharply angled (90˚) and excessively rigid tracheostomy tubes;
3) checking the alignment of the tube in the trachea with a flexible bronchoscope at the completion of tracheostomy. It is always advisable to have a variety of tracheostomy tubes available;
4) avoiding overinflation of tracheal cuffs. Check intracuff pressures routinely.

For prevention of *hemorrhage after tracheal resection and reconstruction*

1) do not dissect out the brachiocephalic artery unless necessary (adherence to trachea, prior surgery or dissection for tumor);
2) interpose robust, vascularized tissue between the dissected artery and tracheal anastomosis (sternohyoid muscle, thymus, omentum);
3) suture the sternohyoid muscle to the trachea over an artery if tracheostomy is anticipated, possibly later postoperatively;
4) do not use prosthetic materials, especially in contact with major vessels.

For a description of prevention of a tracheal-arterial fistula in *cervicomediastinal exenteration*, see Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy."

References

1. Gelman JJ, Aro M, Weiss SM. Tracheo-innominate artery fistula. J Am Coll Surg 1994;179:626–34.
2. Jones JW, Reynolds M, Hewitt RL, Drapanas T. Tracheo-innominate artery erosion: successful surgical management of a devastating complication. Ann Surg 1976;184:194–204.
3. Deslauriers J, Ginsberg RJ, Nelems JM, Pearson FG. Innominate artery rupture. A major complication of tracheal surgery. Ann Thorac Surg 1975;20:671–7.
4. Levasseur P, Rojas-Miranda A, Kulski M, et al. Les complications de la chirurgie des sténoses trachéales nontumorales. Ann Chir Thorac Cardiovasc 1971;10:393–8.
5. Grillo HC, Donahue DM, Mathisen DJ, et al. Postintubation tracheal stenosis. Treatment and results. J Thorac Cardiovasc Surg 1995;109:486–93.
6. Grillo HC, Mathisen DJ. Cervical exenteration. Ann Thorac Surg 1990;49:401–9.
7. Couraud L, Hafez A, Velly JF. Hémorrhages endotrachéales

par fissuration du tronc artériel brachio-céphalique au cours des canulations prolongées. Cure chirurgicale. Presse Med 1984;13:2577–9.
8. Cooper JD. Tracheo-innominate artery fistula: successful management of three consecutive patients. Ann Thorac Surg 1977;24:439–47.
9. Brewster DC, Moncure AC, Darling RC, et al. Innominate artery lesions: problems encountered and lessons learned. J Vasc Surg 1985;2:99–112.
10. Wright CD. Management of tracheoinnominate artery fistula. In: Mathisen DJ, editor. The trachea. Chest Surg Clin North Am 1996;6:865–73.

Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions

Hermes C. Grillo, MD

In addition to clearly defined and relatively more common diseases of the trachea (ie, primary and secondary neoplasms, postintubation lesions, congenital and traumatic lesions), a wide variety of uncommon conditions may be encountered. These lesions can be either intrinsic or extrinsic. *Intrinsic lesions* include the following: 1) those due to specific *infection*, such as tuberculosis and histoplasmosis; 2) defined conditions that affect other organs or tissues besides the trachea and bronchi, broadly characterized as *inflammatory* or *infiltrative*, but fundamentally of unknown etiology, including sarcoid, amyloid, Wegener's granulomatosis, and relapsing polychondritis; 3) *idiopathic* laryngotracheal stenosis; and 4) a miscellany of intrinsic conditions that present as *deformation* or *malacia* of the trachea. *Extrinsic lesions*, which cause tracheal obstruction by compression, include goiter, tumors and cysts, congenital and acquired vascular lesions, and mediastinal or chest wall displacement. Most of these two types of lesions are covered in this chapter, except for malacia and extrinsic compression, which are presented in Chapter 15, "Tracheobronchial Malacia and Compression."

Infection

Tuberculosis

Tracheobronchial tuberculous infections seem to involve principally the lower trachea and/or main bronchi (see Figure 37 [Color Plate 16]). Acute ulcerative tuberculous tracheitis is treated medically. Polypoid tissue and then cicatricial stenosis may result as the tracheitis or bronchitis heals.[1] This can occur despite adequate treatment of the tuberculosis.

Typically, the fibrosis that results is circumferential and submucosal (Figure 14-1). The airway may become extremely narrowed. Externally, the tracheal cartilages may appear to be intact, despite some peritracheal fibrosis. These days, cavitary disease is not often present with a tuberculous stenosis, as it once was. Primary parenchymal disease associated with endobronchial tuberculosis has been noted more in the lower lobes in the form of bronchiectasis. The length and severity of stenosis varies. The degree of resolution of

the frequently accompanying pulmonary lesions also varies, ranging from residual parenchymal scar to lobar fibrosis or destruction (Figure 14-2). Tuberculous broncholithiasis is not often seen anymore. Rarer still are tuberculous tracheoesophageal fistulae.[2]

If possible, active tuberculosis should be arrested before surgical resection and reconstruction is contemplated. The linear extent of mature fibrous stenosis of the trachea and bronchi may be such that excision and reconstruction is not presently possible. This leaves the possibility of dilation and stenting. When the stenosis is more limited in extent, surgical excision and reconstruction can be performed, with the likelihood of a good result. Such resections have included excision of the lower trachea and carina, isolated resection of the left main bronchus, as well as sleeve lobectomies.[3–5] Kato and colleagues, however, noted an increased frequency of postanastomotic stenosis.[5]

In 2 patients operated upon for acute and severe airway obstructions, the resection necessarily included a stenotic lower trachea, a stenotic right main bronchus, and a contracted and fibrotic right upper lobe. The right bronchus intermedius was anastomosed to the side of the trachea after the left main

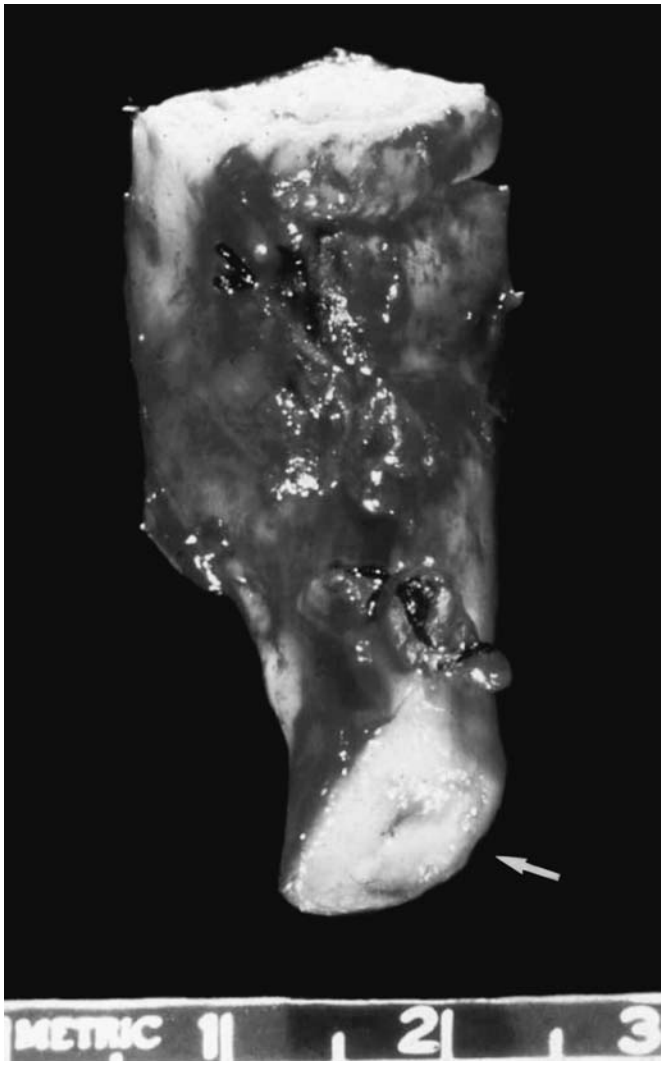


FIGURE **14-1** *Tuberculous stenosis of lower trachea and right main bronchus. The bronchial cross section* (arrow) *shows the lumen nearly obliterated by circumferential fibrosis.*

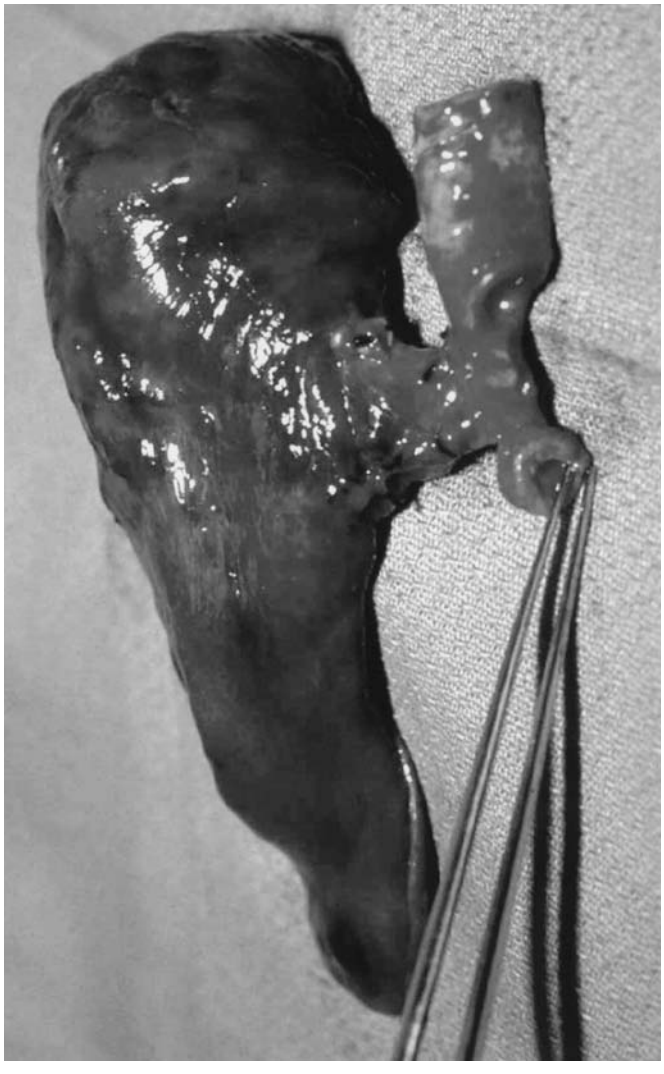


FIGURE **14-2** *Complete specimen from Figure 14-1, consisting of stenosed lower trachea and right main bronchus to the bronchus intermedius. The right upper lobe was also densely fibrosed and removed in continuity.*

bronchus had been joined end-to-end to the trachea (Figure 14-3). One patient with a fibrotic stenosis recovered well. In the second patient, with markedly active disease, the healing failed and the patient died. In the presence of active tuberculosis, obstruction must be managed other than by resection. This same distribution of disease and treatment has been described by others.[5,6]

Histoplasmosis

Histoplasmosis causes airway obstruction in several ways.[7] Large fibrotic or calcified lymph nodes may compress the main bronchi by forming what is well described as subcarinal histoplasmoma or mediastinal granuloma (Figure 14-4). The center of this mass usually contains necrotic material. The periphery is characterized by a considerable thickness of dense collagenous tissue. Enlarged lymph nodes and fibrosis may compress the right or left main bronchi, and, in particular, the bronchus intermedius, at the level of the large lymph node accumulation present around the middle lobe bronchus (Figure 14-5).[7,8] Calcified lymph nodes may also erode gradually into the carina, the right or left main bronchi, or the bronchus intermedius, causing obstruction and hemoptysis from granulation tissue, as well as eventual protrusion of the calcific node (Figure 14-6).

Histoplasmosis is now a principal cause of broncholithiasis, as tuberculous disease has receded (see Figure 36 [Color Plate 16]).[9] Intrinsic fibrosis of the wall of the lower trachea and one or both main bronchi or of the right bronchial tree and bronchus intermedius may also occur, with accompanying lymph node involvement (see Figure 35, Color Plate 16). Varying degrees of mediastinal fibrosis can present (Figure 14-7). Pulmonary infection and fibrosis may follow bronchial obstruction, and hemorrhage

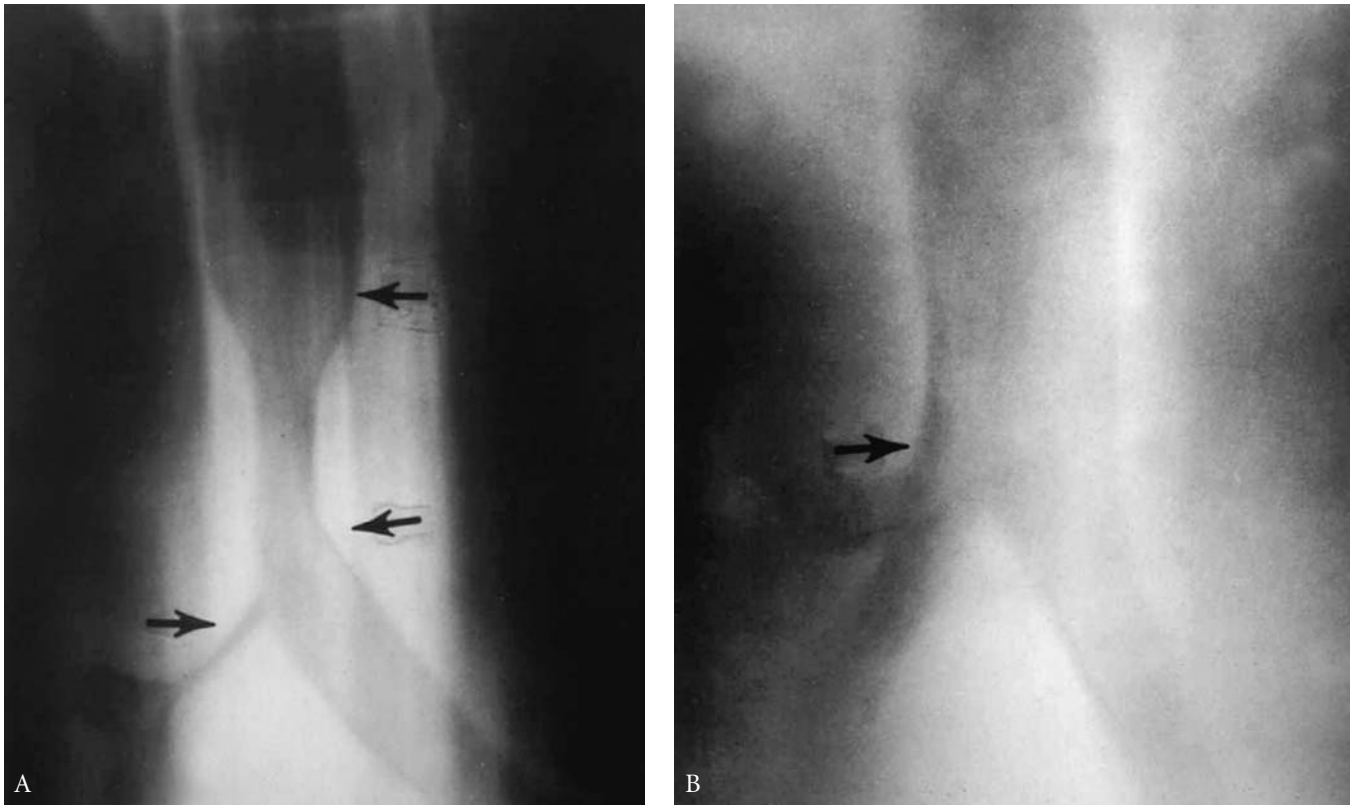


FIGURE **14-3** *Tomograms (retouched) of a 38-year-old woman with tuberculous stenosis seen in the surgical specimen in Figure 14-2.* A, *Lower tracheal and right main bronchial stenoses are indicated by* arrows. B, *Postoperative study shows anastomosis of bronchus intermedius* (arrow) *to side of trachea just above the end-to-end anastomosis of trachea to left main bronchus.*

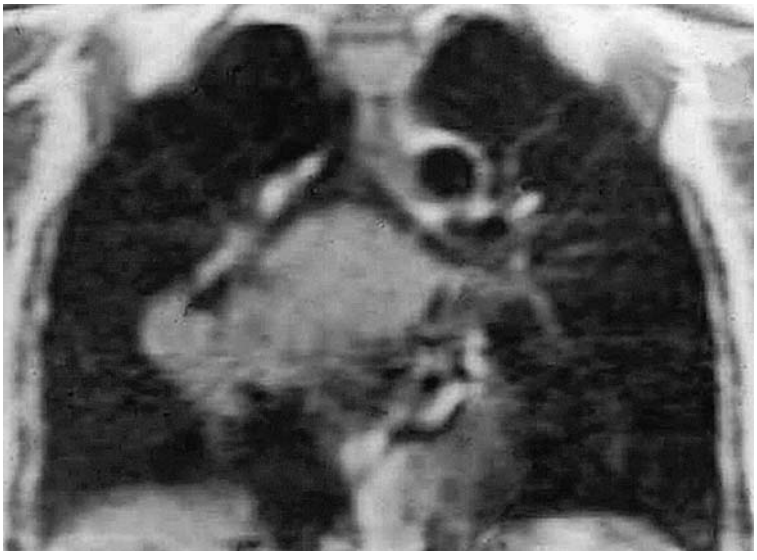


FIGURE **14-4** *Magnetic resonance image showing bilateral compression of main bronchi by a subcarinal mass due to histoplasmosis in a 35-year-old woman. Extensive disease also encircles the bronchus intermedius. The lesion developed radiologically in less than 5 years. This required removal of middle and lower lobes in continuity with the subcarinal mass. The main bronchi were intact.*

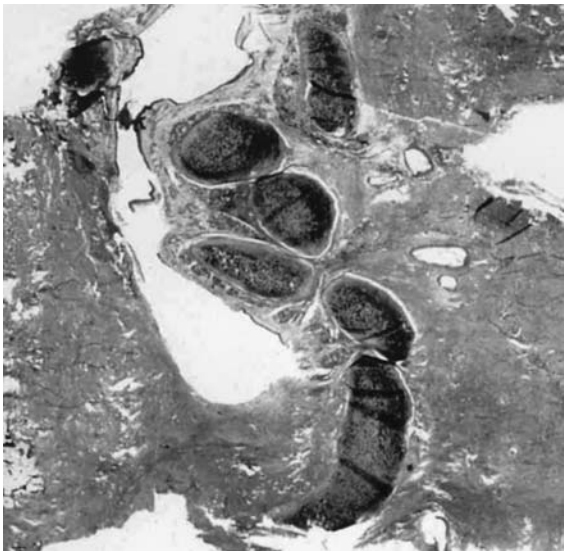


FIGURE **14-5** *Bronchus intermedius constricted by dense scar which envelopes cartilages, causing them to override. Nodes at the origin of this bronchus are frequently involved.*

may accompany broncholithiasis. The granulomatous process may compress the adjacent esophagus to some degree and even produce fistulae from the esophagus to the subcarinal or lobar lymph nodes or to the airway itself (see Figure 12-12 in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula").[7,10] Inability to identify *Histoplasma capsulatum* from biopsy specimens in many patients has led to conclusions that fibrosis is often not the result of active fungal proliferation but instead of hypersensitivity reaction to the healing infection.[11] Although not easily established, the interval between initial infection and these later presentations appears to be of several years.

Patients present with cough (41%), dyspnea (32%), hemoptysis (31%), or recurrent postobstructive pneumonia (23%). Pleuritic pain also occurs (23%).[11] Forty percent of patients may be asymptomatic. Superior vena cava syndrome is another presentation that may coexist with airway obstruction.

Computed tomography (CT) scanning with contrast has been of particular help in identifying the extent of involvement (see Figure 14-7*B*). Skin tests are of no use, in view of the widespread sensitization of the population in areas where the disease is endemic (up to 80%). Indeed, skin tests may be misleading, by causing conversion of serologic tests. Serial complement fixation titers may be of value and may help to determine the need for antifungal therapy in chronic states.[12] Antigenuria may be present in active disseminated infection. Organisms are most often demonstrated by silver methenamine stains in pathological material rather than found in aspirates, even in the presence of bronchial erosion. Although acute documented histoplasmosis is treated with amphotericin, the use of the drug has not proved to be of benefit in late cases of fibrosis without demonstration of active organisms. The finding of organisms may indicate the use of itraconazole or ketoconazole, which have less side effects than those seen with amphotericin.

Fibrosis may be so severe and so dense that dilation finally becomes impossible (see Figures 14-7, 14-8). There may be no useful medical treatment at this stage. Large, obstructing subcarinal masses may sometimes be removed by painstaking surgical excision, without the necessity of bronchial resection.

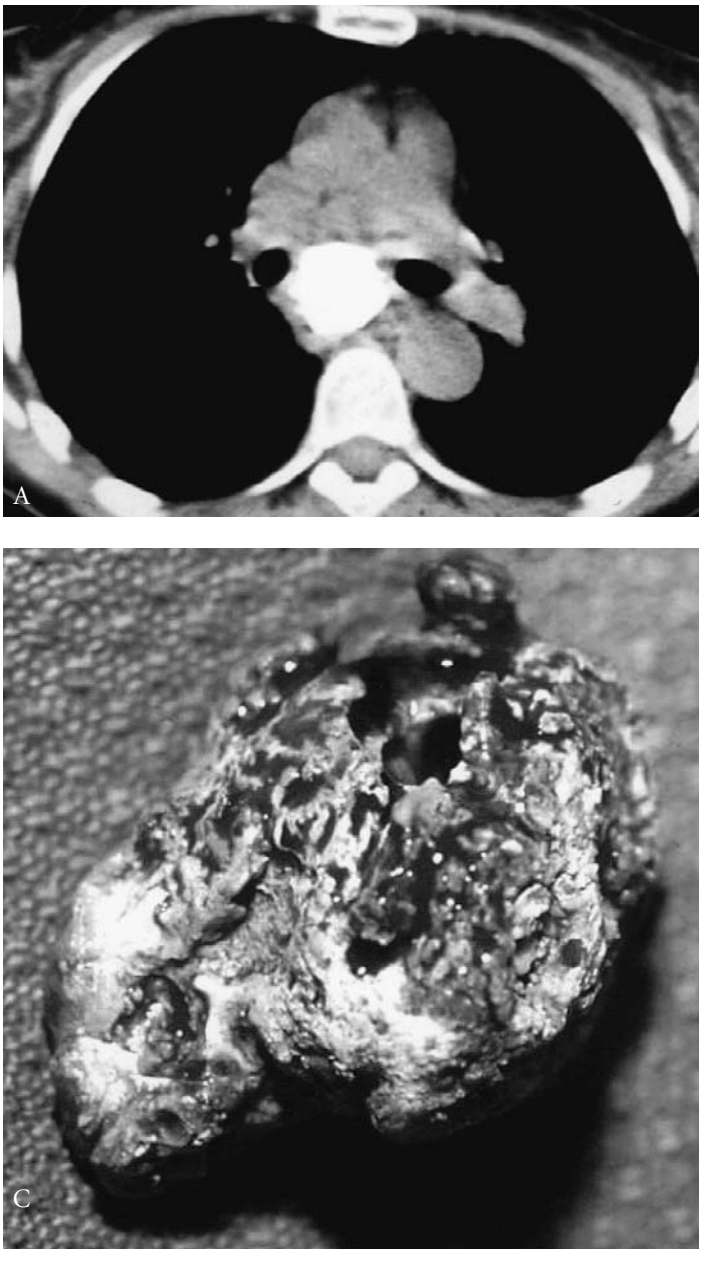
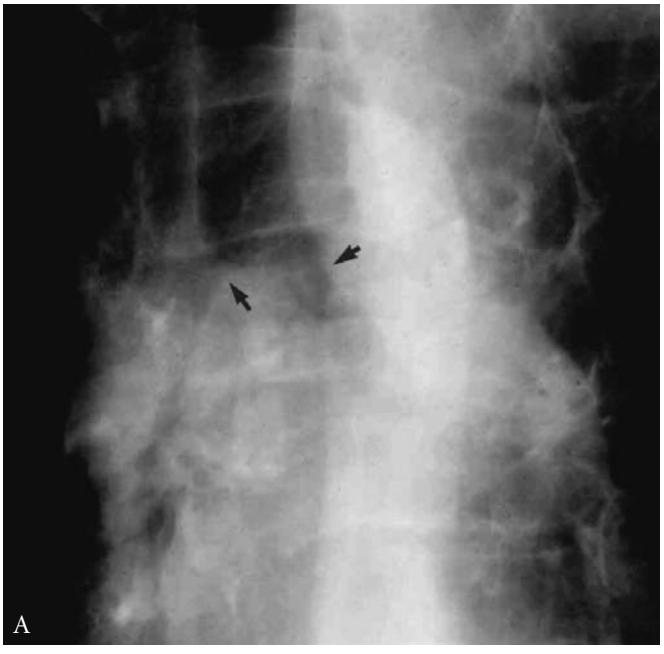


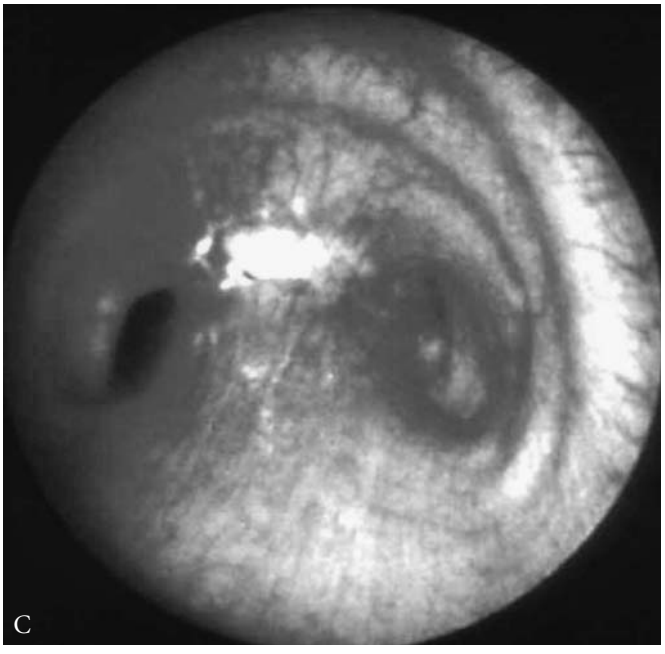
FIGURE **14-6** *Mass of calcified subcarinal lymph nodes due to histoplasmosis, with broncholithiasis of the left main bronchus.* A, *Computed tomography scan showing a calcified mass.* B, *Tomogram of the carina in the same patient. The* arrow *points to the broncholith protruding into the bronchial lumen.* C, *The mass excised with an attached intrabronchial extension. The bronchus was closed longitudinally in this particular patient, which is not always possible. Also, see Figure 36 (Color Plate 16).*

A resulting bronchial opening, if very limited, may indeed be closed with only slight ultimate narrowing of the bronchus. Small portions of the fibrotic wall of "histoplasmoma" may be left in place in the mediastinum rather than incurring serious technical problems or creating an unreconstructible situation.[7,8] James and colleagues essentially performed decortication of a severely stenotic trachea, which produced lasting relief despite necessarily incomplete removal of fibrosis.[13] Garrett and Roper noted relief of vascular and bronchial obstructions by unroofing soft nodes, leaving the adherent fibrous capsule behind.[14] In their series of 94 patients, 13 had respiratory symptoms, with major bronchial narrowing in 9 and compression in the other 4. Eleven patients had hemoptysis related to broncholithiasis. Postobstructive destruction of the middle and right lower lobe requires resection. *The technical procedure may be very difficult and often requires judicious placement of a proximal tourniquet on the pul-*

monary artery prior to proceeding with further dissection. The calcified lymph nodes often intrinsically involve the wall of the pulmonary artery.

We reported bronchoplastic procedures for airway involvement as follows: sleeve lobectomy in 3 patients, carinal resection in 1, carinal pneumonectomy in 4, and sleeve resection of the right main bronchus in 1.[7] The organism was identified in 9 of 20 patients by staining. Despite the adjacent fibrosis, the tracheobronchial anastomoses heal if there is not excessive tension. Dense cicatrization may involve a proximal pulmonary artery, necessitating intrapericardial control. Full thickness esophageal involvement or fistula is managed by careful layered esophageal closure, with firm buttressing of the suture line. Significant collateral circulation from the pleura to lung, sufficient to cause severe hemoptysis, may occur, especially where a pulmonary artery is occluded.[11] Superior vena cava obstruction is also well recognized in this process. Surgical intervention does seem to be advisable, even for asymptomatic mediastinal granulomas of large size. Dines and colleagues found that 34% of their cases of mediastinal granuloma progressed to





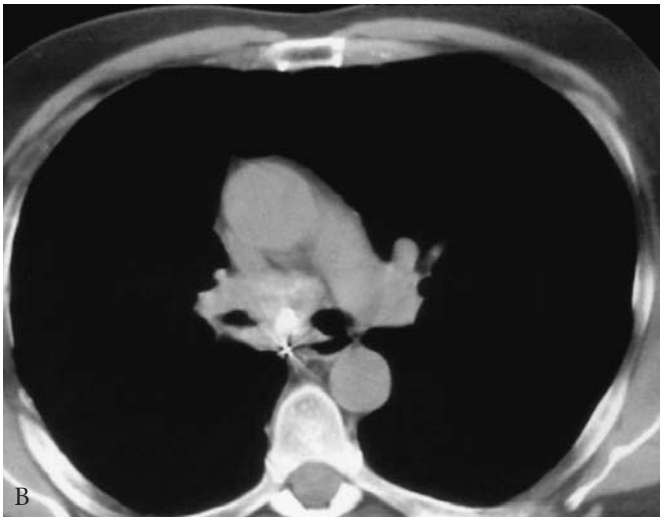
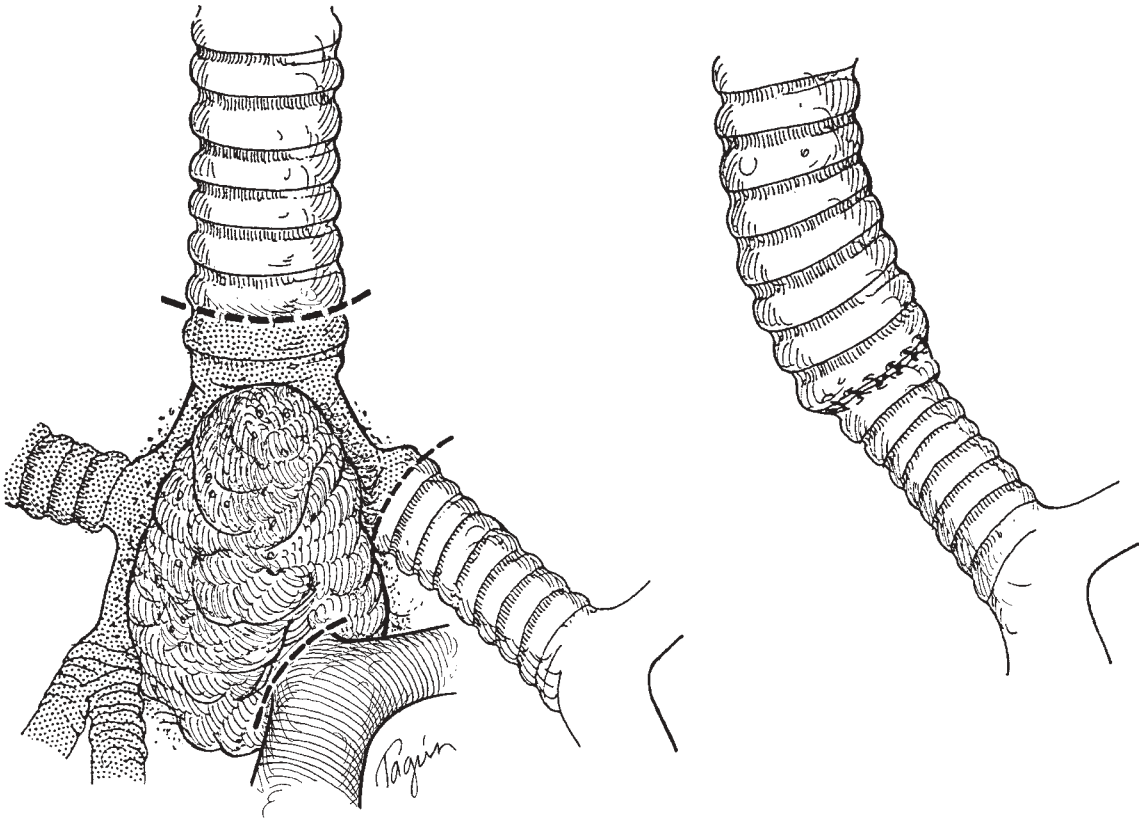


FIGURE **14-7** *Massive mediastinal fibrosis in a 57-year-old man who had reached an extreme of incapacitating dyspnea. Prior thoracotomy had failed and bronchial dilation proved impossible.* A, *Detail of chest roentgenogram showing critical stenosis of the right main bronchus* (right arrow) *and bronchus intermedius plus severe stenosis of the left main bronchus* (left arrow). B, *Computed tomography scan demonstrating a fibrocalcific mass at the carina in the same patient.* C, *Bronchoscopy reveals near total obstruction of the right main bronchus and a tiny opening on the left.*



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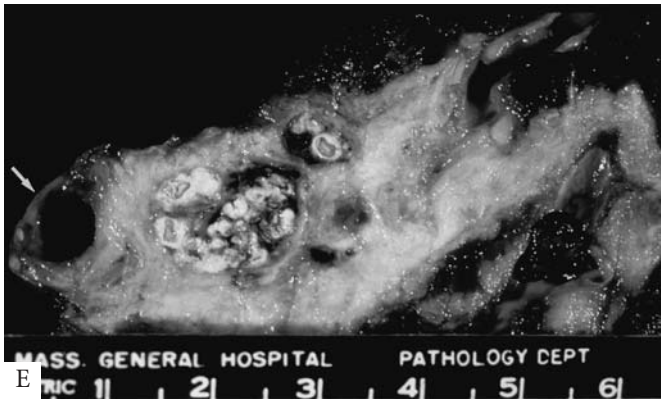

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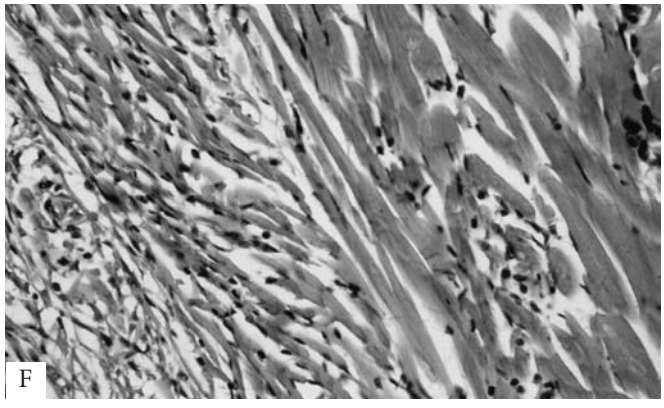

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FIGURE **14-7** (CONTINUED) D, *Operative treatment. Right carinal pneumonectomy was necessary, with intrapericardial division of the pulmonary artery, since calcification extended almost to the origin of the right pulmonary artery. Exposure was via a right thoracotomy. Recovery was uneventful.* E, *Specimen shows calcific and anthracotic lymph nodes encased by dense fibrosis. The left main bronchus is at the left* (arrow). F, *Photomicrograph of dense keloidal fibrosis (hematoxylin and eosin stain). Many fibroblasts are seen on the left and thick bundles of collagen on the right.* Histoplasma capsulatum *was identified on silver methenamine stain.*

fibrosing mediastinitis within 2 years, a result at variance with others.[10,11,14] In a study of 71 patients, Lloyd and colleagues found no support for evolution of a mediastinal granuloma into mediastinal fibrosis.[11]

Trastek and colleagues advised surgical removal of broncholiths rather than bronchoscopic attempts at removal, since pathologic involvement so often necessitated pulmonary resection.[9] We concur in this approach. The endoscopically visible broncholith is truly only the "tip of the iceberg."

Other Infections

Nocardiosis was reported as a mass in the right main bronchus in an isolated case.[15]

A necrotizing *mucormycosis* that involves the trachea, carina, or bronchi, as well as the lungs, is seen principally in diabetic patients and in patients who are immunosuppressed or undergoing chemotherapy, particularly for lymphoma. Chronic renal failure and a history of organ transplantation are factors.[16] Diagnosis is made on bronchial biopsy material by direct examination. Branching, nonseptate hyphae are noted with necrosis, fibrosis, and vessel thrombosis. Cultures are difficult to obtain. Progression of the infection from the bronchus may cause severe or fatal hemorrhage from pulmonary vessels.[17] Secondary bacterial infection also occurs in the lungs.

Prompt and very radical excision of the involved airway and lung, under the protection of vigorous and prolonged treatment with amphotericin or other drugs, may save some of these patients.[16,18] In a young

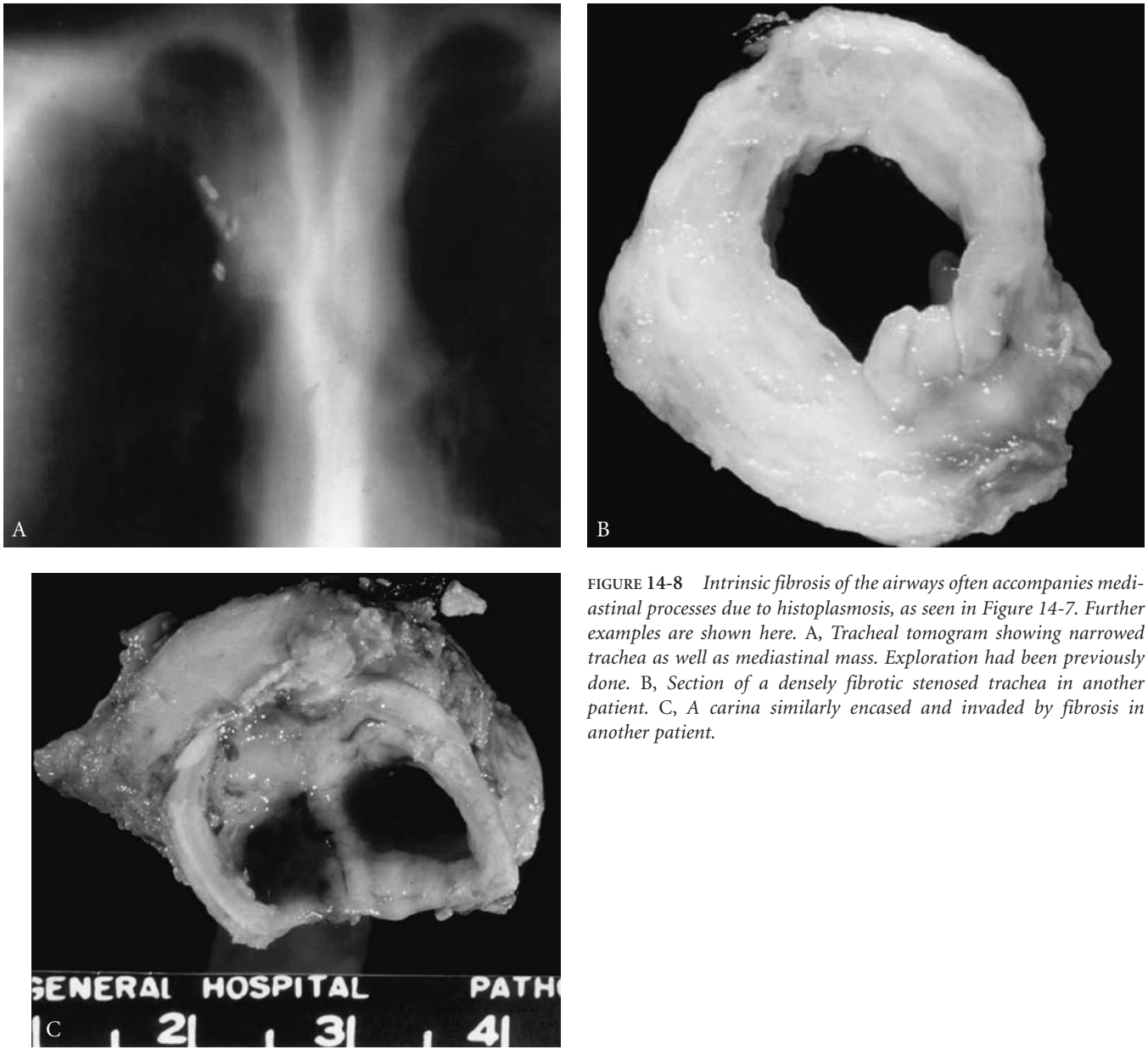


FIGURE **14-8** *Intrinsic fibrosis of the airways often accompanies mediastinal processes due to histoplasmosis, as seen in Figure 14-7. Further examples are shown here.* A, *Tracheal tomogram showing narrowed trachea as well as mediastinal mass. Exploration had been previously done.* B, *Section of a densely fibrotic stenosed trachea in another patient.* C, *A carina similarly encased and invaded by fibrosis in another patient.*

diabetic woman with destructive mucormycosis in the right lung, extensive disease involving the carina and lower third of the trachea, and with infiltrates in the opposite lung, who was deteriorating on drug treatment, an aggressive right carinal pneumonectomy and lower tracheal resection, combined with continued medical treatment, led to resolution and complete recovery. Only a few instances of tracheal bronchial mucormycosis are reported.[19] In a review of reported patients, Brown and colleagues confirmed the favorable results of early surgical treatment, against a high fatality with medical treatment alone.[17] Tedder and colleagues compared a mortality of 68% for medical treatment versus 11% for surgical and medical treatments.[16] Similar results were obtained by Donahue and Wain.[18]

Diphtheria in childhood may be followed many years later by tracheal stenosis or laryngotracheal stenosis; that is, involvement of the subglottic larynx as well as the upper trachea. Since most of the patients who had this disease in infancy or in early childhood were treated with one or more intubations and tracheostomies, it is difficult to determine whether the later stenosis is due to disease or to treatment. In several cases, the patient carried a tracheostomy for many years, later had it removed, and then appeared even later with severe stenosis. These late stenoses, which in my experience most frequently involve the subglottic larynx as well as the uppermost trachea and which lie in the region of prior tracheostomy, may be reconstructible. The anatomic and surgical considerations in such cases are the same as those for postintubation stenoses. Diminishing occurrence of these cases undoubtedly reflects the success of immunization programs.

Scleroma (rhinoscleroma) is a rare disease, which may cause fibrosing changes in the nasopharynx, larynx, and upper airways. In only 2% of cases does it occur in the trachea and bronchi.[20,21] Scleroma is related to infection by *Klebsiella rhinoscleromatis*. The organism is identified in biopsied material. The disease occurs in Mexico, Central and South America, Eastern Europe, the Middle East, India, and only occasionally in the United States.

Scleroma is most common in the first three decades of life and in patients with poor nutrition. As the disease appears and progresses, it is characterized by nasal obstruction, nasal deformity, hoarseness, epistaxis, sore throat, and lip swelling.[21] The tracheobronchial tree is kept open in these patients by repeated bronchoscopies, while prolonged and repeated antibiotic treatments (streptomycin and tetracycline) are given. A tracheostomy may become necessary. Glottic webs and subglottic scars may result as healing proceeds.

Although viral in origin, laryngotracheal *papillomatosis* is described in Chapter 7, "Primary Tracheal Neoplasms," because of its tumor-like appearance. Endobronchial *Kaposi's sarcoma* is also described in Chapter 7. Human immunodeficiency virus (HIV)-infected persons are also subject to endobronchial tuberculosis, aspergillosis, non-Hodgkin's lymphoma, and bacterial tracheitis.[22]

Idiopathic Stenosis

Cicatricial stenosis with a lesser inflammatory component, localized in the subglottic larynx and upper trachea, occurs without known cause.[23,24] This process is labelled *idiopathic laryngotracheal stenosis* (Figure 14-9). None of these patients have been intubated for ventilation or have suffered external or internal trauma to the trachea. The lesions are not congenital. There are no findings in this group of patients of associated mediastinal fibrosis or lymph node involvement by any pathologic process. Few patients have histories or findings suggesting esophageal reflux and aspiration. None have had specific or nonspecific tracheal infections, nor did they later develop manifestations of systemic disease such as polychondritis, or amyloid. A few with stenosis due to Wegener's granulomatosis confined to the upper airway were initially misdiagnosed as idiopathic stenosis, prior to routine screening with an antineutrophil cytoplasmic antibody (ANCA) test. In a series of 73 patients, 71 were female. Age distribution varied widely from 13 to 74 years, but the disease was seen chiefly in the third, fourth, and fifth decades of life.[24] Initial symptoms of dyspnea on effort (in 52%) progressed to dyspnea at rest, with noisy breathing, wheezing, or stridor (in 48%). The duration of symptoms prior to initial presentation varied between 4 months to over 30 years, with the greatest number reporting 1 to 4 years of symptoms. Careful history is necessary to identify the often subtle

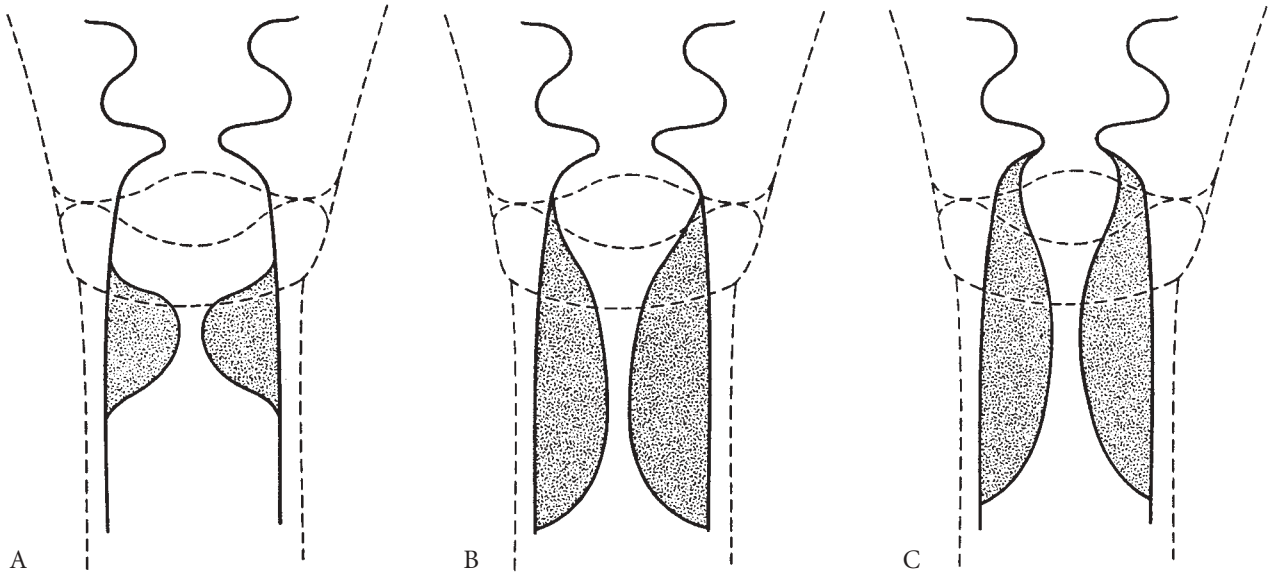
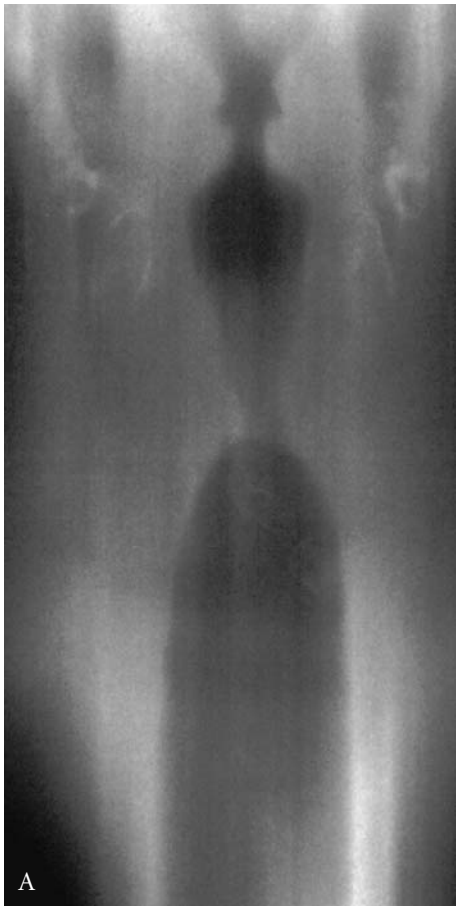


FIGURE **14-9** *Diagrams of typical distributions of idiopathic laryngotracheal stenosis.* A, *The lesion often impinges on the low subglottic larynx at the level of the cricoid cartilage. A resection need remove only a small margin of the lower cricoid.* B, *A lesion that begins in the subglottic larynx and extends to varying distances into upper trachea. Narrowing usually begins shortly below the vocal cords, but enough space remains below the glottis for laryngotracheal resection and adequate primary anastomosis.* C, *In this case, the stenosis is more severe immediately below the vocal cords, with the lumen, even at that level, being very narrow and possibly inadequate for anastomosis. Reproduced with permission from Grillo HC et al.*[23]

onset of the earliest symptoms. Patients were frequently misdiagnosed as having asthma. It is essential to obtain ANCA titer on every patient to rule out Wegener's disease.[25] Bronchoscopic biopsies are not diagnostic of either disease. Nasoseptal biopsies, on the other hand, may identify Wegener's granulomatosis histologically. Prior to the 1993 report by Grillo and colleagues, only limited or individual case reports were available.[23]

Koufman, in a detailed study of the laryngeal effects of gastroesophageal reflux, noted that 30% of otolaryngologic patients had pharyngeal reflux, as determined by a double pH probe technique.[26] Seventy-eight percent of patients with a laryngeal stenosis from any cause, but principally of postintubation origin, showed pharyngeal reflux. Maronian and colleagues, using three to four port pH probes, with one positioned proximal to the upper esophageal sphincter, recorded a pH of less than 4.0 in 5 of 7 patients with isolated idiopathic subglottic stenosis.[27] No control data were presented, however, as has most often been the case in assessing the significance of laryngeal gastroesophageal reflux. The significance of these findings as indicators of possible cause for idiopathic stenosis remains uncertain. Fifteen of our patients had a history or symptoms of gastroesophageal reflux. It must be noted that none of our patients who were operated upon for idiopathic stenosis suffered later progression of stenosis.[24] This would have been expected if untreated reflux was an important etiologic factor.

In most cases, *radiologic study* showed a circumferential lesion of varying length, most often between 1 to 3 cm, centered at the junction between the cricoid cartilage and trachea (Figure 14-10). In 4 cases, the subglottic larynx itself was not involved, but in another 69 cases, subglottic involvement was of varying degrees of severity. The narrowing most often began shortly below the vocal cords and rapidly became a more severe to maximal stenosis at approximately the cricoid level. Although the involvement might be eccentric, it was always circumferential. Vocal cord function appeared normal. Longitudinal roentgenograms brought out the extent of the lesion and its nature most clearly. Flow volume loops, as expected, demonstrated extrathoracic fixed obstruction. Cultures obtained from biopsies and from surgical specimens showed nothing but the usual upper respiratory flora. Skin tests and serologic tests, done



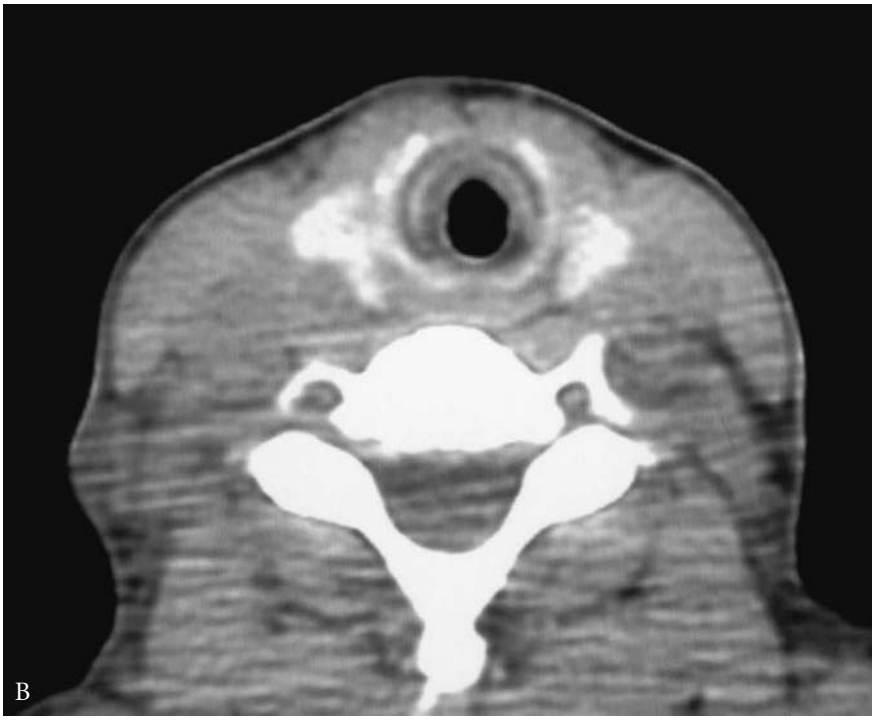


FIGURE **14-10** *Images of upper airway of a 50-year-old woman with typical idiopathic laryngotracheal stenosis.* A, *Tomogram of the larynx and upper trachea. The false and true vocal cords are well outlined at the top. The stenosis involves the subglottic larynx and uppermost trachea. Compare with Figure. 14-9B.* B, *Computed tomography scan of the neck. The circumferential disposition of the stenosis lies within the ring of cricoid cartilage. Occasionally, the lesion is eccentric, although still circumferential.*

especially in the earlier patients, were negative for tuberculosis, coccidioidomycosis, histoplasmosis, and blastomycosis. On *bronchoscopy*, the mucosa over the lesion appeared to be injected, and bled easily. Granulation tissue was uncommon (Figure 14-11). Ulceration was identified in only 1 patient. When these patients presented, the diameter of the aperture ranged from 3 to 10 mm, with most lying between 5 to 7 mm. The trachea distal to the stenosis appeared normal (see Figure 33 [Color Plate 15]).

Since the origin of a laryngotracheal and upper tracheal idiopathic stenosis is not understood, and since the entity had not been previously studied or followed over a long period of time, my initial approach was conservative. Some lesions remained stable over the period of observation, but in others, the obstruction worsened, requiring frequent dilations. Linear extension was not seen, however. Seventy-three patients have been subjected to surgical resection and reconstruction, with 4 cases involving the upper trachea, and in 10 cases, a rim of lower cricoid cartilage as well. In 59 cases with subglottic laryngeal stricture as well, the anterior portion of the subglottic larynx was removed and the posterior portion of the stenosis was managed by laryngotracheoplasty, as described in Chapter 25, "Laryngotracheal Reconstruction" (Figure 14-12). Thirty-six of these 59 patients required posterior laryngeal cricoid resurfacing with membranous tracheal wall. Initially, a protective complementary tracheostomy was performed in patients requiring laryngotracheoplasty. However, this was soon found to be unnecessary as a routine measure. Seven of 73 patients had temporary tracheostomies, but of the last 30 patients, only 1 required this. In 2 patients, in whom the stenosis was extremely severe and extended upward to the undersurface of the vocal cords, laryngofissure, excision of the scar, and resurfacing with buccal mucosa were performed. In 72 patients

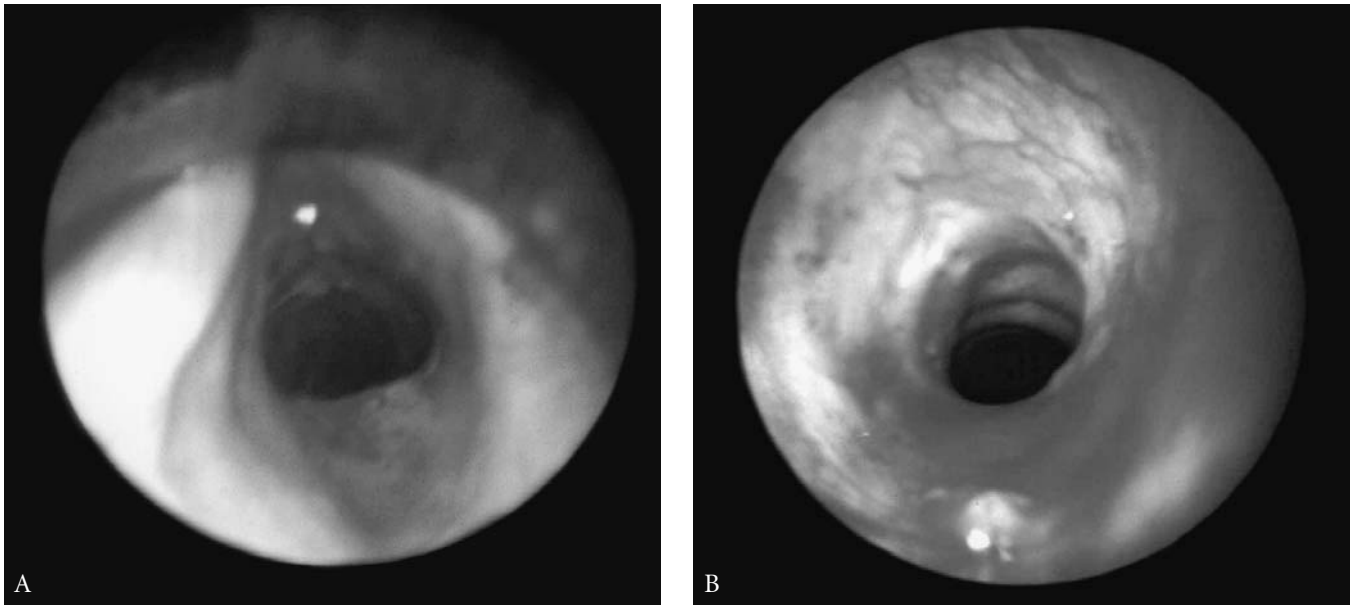


FIGURE **14-11** *Bronchoscopic findings.* A, *View through separated vocal cords, demonstrating the funnelled subglottic larynx, with the circumferential ring of a high idiopathic stenosis visible below. Correction required circumferential laryngotracheoplasty as described.* B, *Closer intralaryngeal view affirms the circular nature of the stenosis. Its short length and the normal tracheal rings below are well seen. No granulation tissue is present but the mucosa is easily abraded. Also, see Figure 33 (Color Plate 15).*

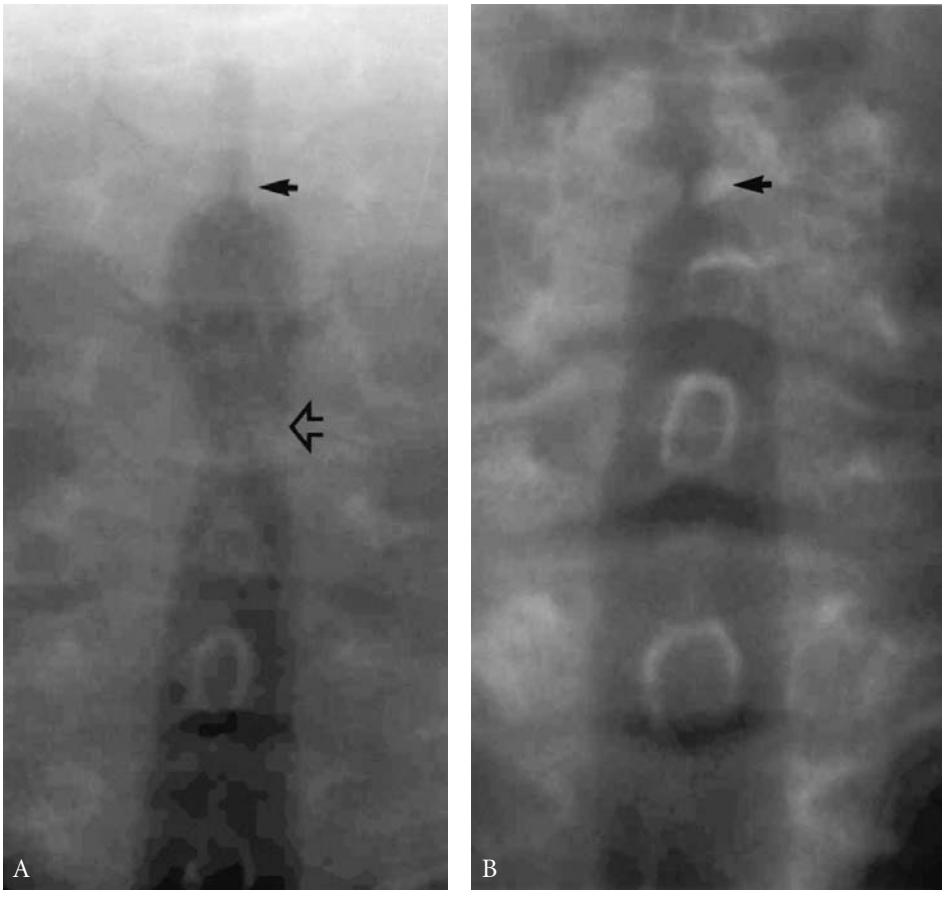


FIGURE **14-12** *Laryngotracheal resection and reconstruction for idiopathic stenosis, in a 55-year-old woman with a 3-year history of progressive symptoms.* A, *Preoperative tomogram. The* solid arrow *indicates the glottis; the* open arrow *indicates the stenosis.* B, *Postoperative tomogram. The* arrow *is at the glottis. Vocal cords are symmetrical. Note the slight indentation at the level of the anastomosis. The patient continued to do well 22 years after the operation.*

with proximal idiopathic laryngotracheal stenosis, who underwent operation and had long-term follow-up (median 8 years), 19 (26%) attained excellent results in respect to voice and airway, 47 (64%) had good results, and 5 had only fair results and required occasional dilation (Figure 14-13). One case with poor result required at least annual dilation. In only this patient was there evidence of extension of the inflammatory process beyond that originally seen, suggesting a different process. Of the patients followed conservatively, none demonstrated spontaneous regression or regression in response to other treatments, including systemic or local corticosteroids.

In light of our observations in 73 patients of 1) long-term good results from surgical treatment in most, and 2) failure of recurrence or progression of the disease, it is difficult to understand the contradictory results of Dedo and Catten, who reported relentlessly progressive disease and complete failure in their 7 patients treated surgically.[28]

Pathologically, fibrosis was generally circumferential and of even thickness. Preeminent was dense collagenous fibrosis of keloidal type, which thickened the lamina propria of the trachea (Figure 14-14).[23] Fibroblasts were relatively sparse. The surface epithelium usually showed squamous metaplasia. Inflammation was not prominent. Granulation tissue was also seen. Sometimes, this might well have been related to prior dilations, lasering, or other treatment. Cartilaginous rings were intact and essentially normal. There were no histologic characteristics to suggest a relapsing polychondritis, vasculitis, Wegener's granulomatosis, or amyloid, and calcification or stainable organisms were not seen. The location, configuration, gross appearance, and microscopic appearance of these upper airway stenoses are similar enough to suggest a definable disease entity. The predominance of the disease in females deserves notice.

In idiopathic lesions that involve the subglottic larynx (and these are in the majority), airway narrowing usually begins shortly below the vocal cords. It takes refined and experienced judgment to decide which patients should be operated upon. A reasonably sized "atrium" is needed below the vocal cords to provide an adequate luminal size for successful anastomosis. It should allow a luminal cross-sectional area of the airway, at least 50% of normal. The obliquity of the anastomosis helps to enlarge the new laryngotracheal junc-

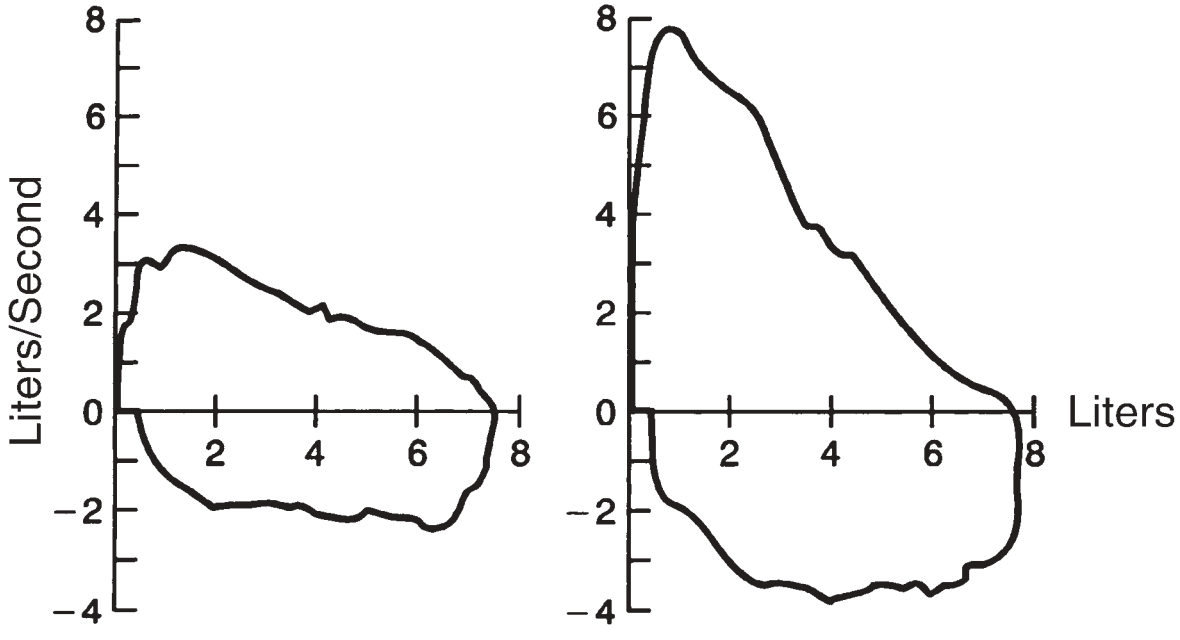


FIGURE 14-13 *Functional results of reconstruction. Flow volume loops before and after operation, showing marked improvement in inspiratory and expiratory flows. Reproduced with permission from Grillo HC et al.*[23]

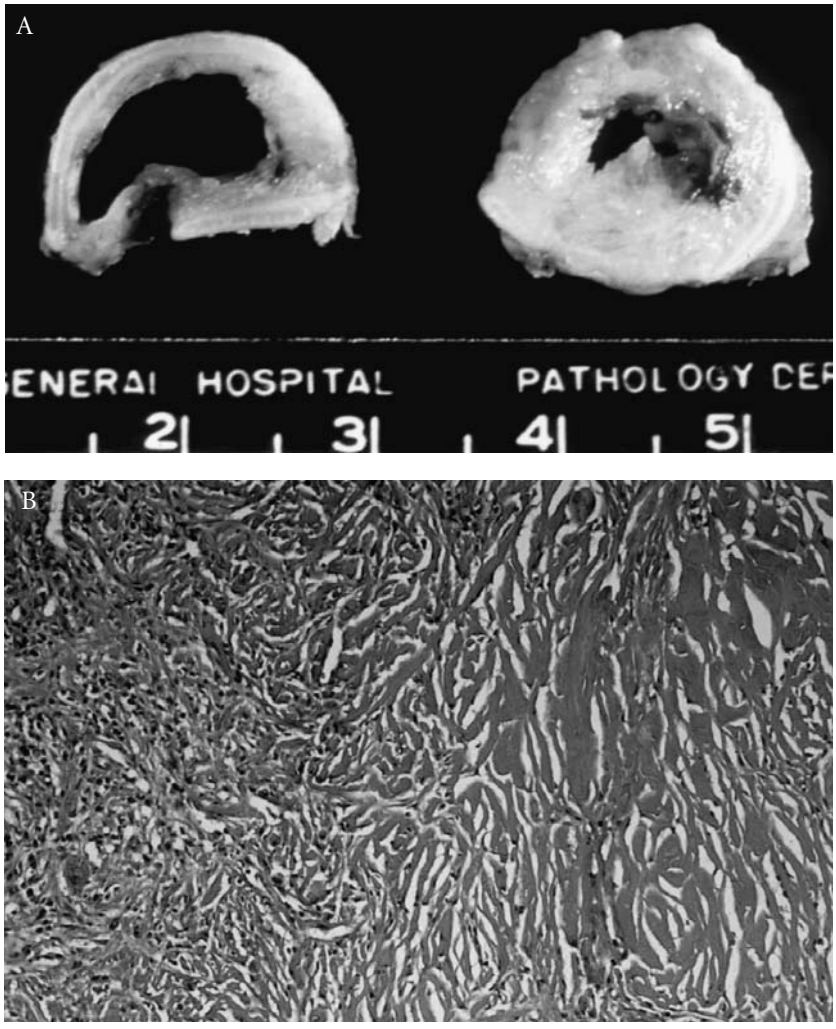
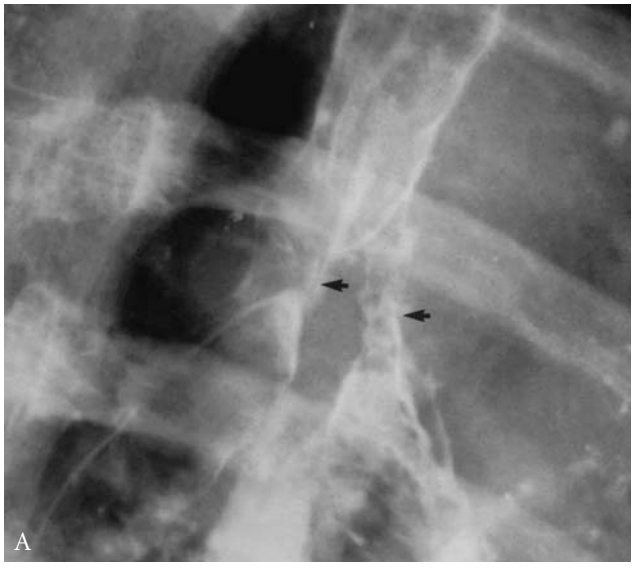


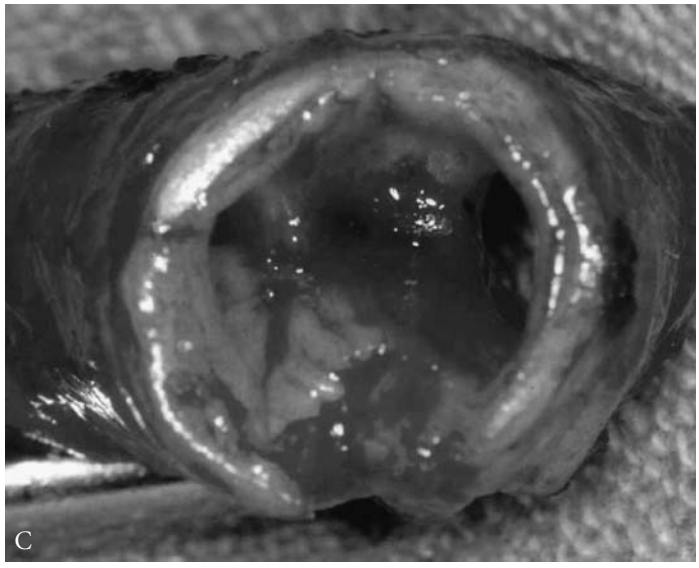
FIGURE **14-14** *Pathologic findings in idiopathic stenosis.* A, *Gross specimen, cross sections of trachea. The dense fibrosis lies inside of intact cartilaginous rings.* B, *Photomicrograph reveals keloidal fibrous tissue, which replaces the lamina propria of the tracheal mucosa. Inflammatory changes are found in many near the mucosal surface, and frank granulation tissue in a few (hematoxylin and eosin; ×125 original magnification).*

tion. Contraindications to operation are a significant stenosis that begins within 5 mm of the glottis and, less permanently, the presence of florid inflammation and/or granulation tissue in the stenosis. If the stenosis reaches or nearly reaches the undersurface of the vocal cords, then treatment by periodic dilation may be the best option. If florid inflammation and granulation tissue are present, the operation should be deferred. Periodic mechanical dilation (see Chapter 19, "Urgent Treatment of Tracheal Obstruction"), with or without local corticosteroid injection, permits temporizing for as long as is necessary.

Operative failure here would likely be complete and permanent, with scant opportunity for a second repair. Furthermore, the recurrent laryngeal nerves are at risk in all of these patients. Results are also limited in many patients by a permanent slight weakness in the ability to project voice and in a diminished ability to sing, which are common sequelae (42 of 67 patients with good to excellent surgical results) after surgical reconformation of the larynx.

In a much smaller number of patients, a *different stenotic process*, also idiopathic, was encountered at the *supracarinal and carinal levels*, which also involved the main bronchi (Figure 14-15). In another small number of patients, a more inflammatory stenosing process would usually (but not always) spread over time, to involve almost the entire trachea and main bronchi (Figure 14-16). In view of the persistently anatomically localized nature of the upper idiopathic tracheal stenoses, it seems likely that these few cases





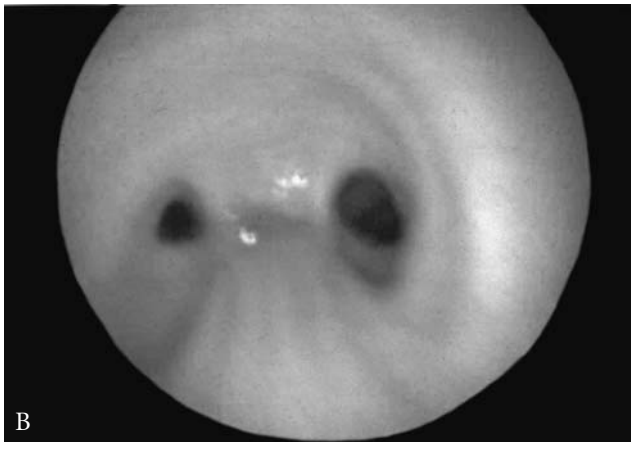


FIGURE **14-15** *Idiopathic stenosis of the distal trachea and carina in a 62-year-old woman with a 10-year history of "asthma," surgical correction of a high tracheal stenosis, with finding of carinal stenosis a year later, the duration of which is unknown.* A, *Tracheobronchogram showing stenosis of both main bronchi* (arrows). *Oblique view.* B, *Bronchoscopic view of the carina in this patient.* C, *Surgical specimen. Trachea was anastomosed end-to-end to the left main bronchus. The right main bronchial stump was implanted in the right wall of the trachea above the other anastomosis. The result remained good.*

of more diffuse and sometimes progressively longer stenoses are of other etiology or etiologies. These few patients did not show clinical or pathological characteristics of histoplasmosis.

Inflammatory or Infiltrative Lesions

A group of unrelated lesions are discussed here. Their only common feature is an alteration or infiltration of tracheal and bronchial walls by non-neoplastic processes that are clinically well characterized, but are of unknown cause. Idiopathic stenosis could well be included, in that it largely shows collagenous deposition in the larynx and trachea.

Relapsing Polychondritis

Relapsing polychondritis remains a disease of unknown origin, characterized principally by inflammatory degeneration of articular and extra-articular cartilages. It is believed to be an autoimmune disease, possibly a reaction to type II collagen, and has been reported in association with Wegener's granulomatosis, rheumatoid arthritis, vasculitis, and systemic lupus erythematosus.[29] The clinical course may be rapid or slow, and episodic or progressive.[30] Cardiovascular, renal, and neurologic manifestations occur later. A variety of other systemic diseases have been seen in association as well.[29] In those with a fully developed syndrome, the cartilage

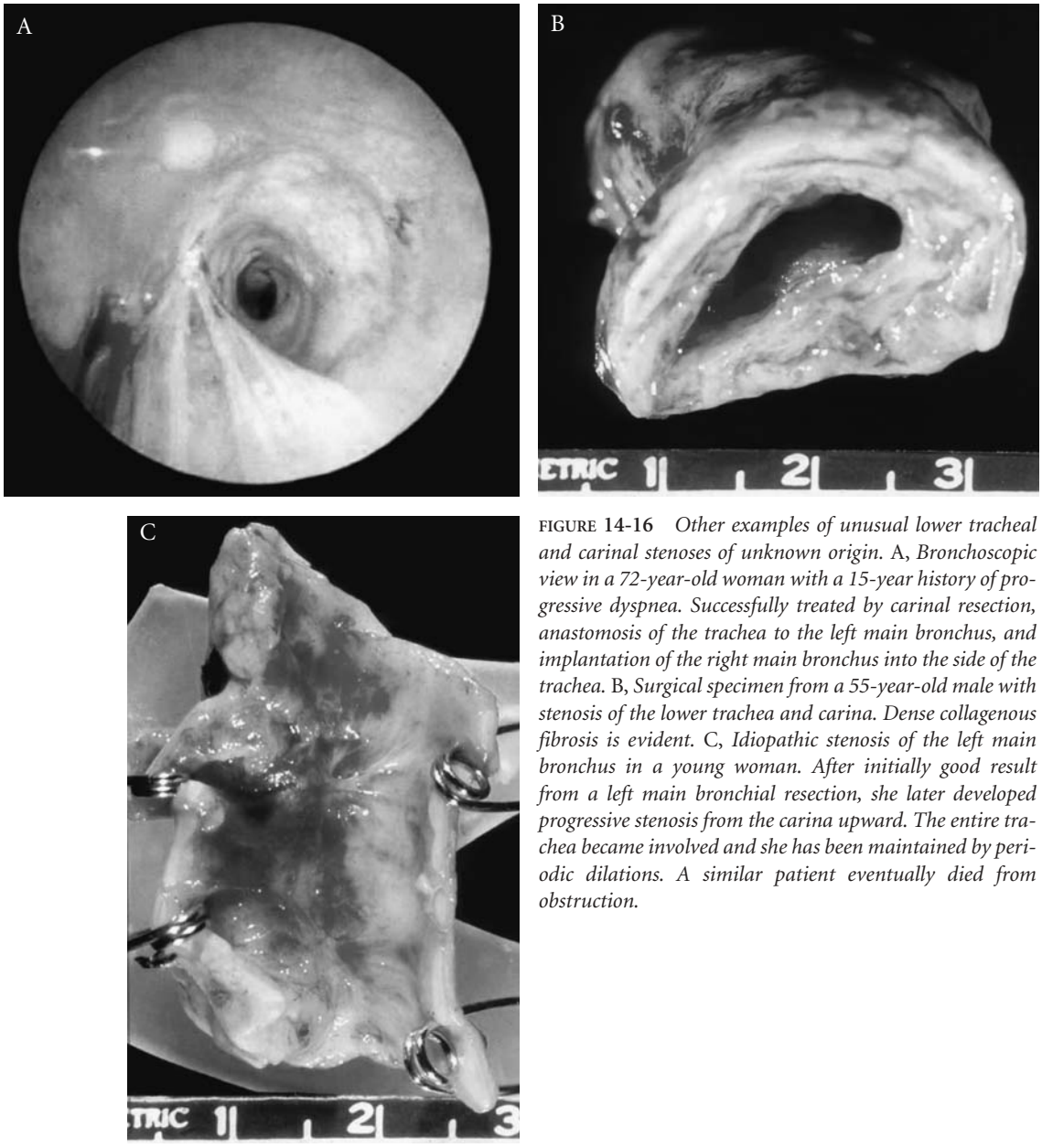


FIGURE **14-16** *Other examples of unusual lower tracheal and carinal stenoses of unknown origin.* A, *Bronchoscopic view in a 72-year-old woman with a 15-year history of progressive dyspnea. Successfully treated by carinal resection, anastomosis of the trachea to the left main bronchus, and implantation of the right main bronchus into the side of the trachea.* B, *Surgical specimen from a 55-year-old male with stenosis of the lower trachea and carina. Dense collagenous fibrosis is evident.* C, *Idiopathic stenosis of the left main bronchus in a young woman. After initially good result from a left main bronchial resection, she later developed progressive stenosis from the carina upward. The entire trachea became involved and she has been maintained by periodic dilations. A similar patient eventually died from obstruction.*

of the nose deteriorates, leading to a saddle nose, cartilages of the ears become thickened and inflamed, and cartilages of the airways from the larynx to the segmental bronchi may be involved. McAdam and colleagues collected reports of 159 patients with relapsing polychondritis, including 29 studied prospectively.[31] In addition to the elements noted, these patients had inflammatory polyarthritis, nasochondritis, ocular inflammation, and cochlear and vestibular ear injuries. As the cartilage is destroyed by the recurring inflammatory process, it is replaced by fibrous tissue. Initial airway obstruction is due to edema and inflammation before the cartilage is destroyed and before airway collapse occurs. Fever, weight loss, and lethargy accompany the illness. The onset of symptoms is most frequently in the fourth decade of life, but may be seen much earlier. Initial presentation may be with hoarseness, loss of voice, and tenderness over the larynx and trachea. The larynx and proximal trachea are most commonly affected.[32] The interval between first airway symptoms and

declaration of the disease's characteristics may be prolonged. Age range at diagnosis is from 13 to 84 years, but most are between 44 and 51 years of age. Men and women are affected equally.[29,31] Fifty-six percent of patients had evidence of respiratory tract involvement.[31] Eleven of 14 patients who presented initially with respiratory involvement required a tracheostomy. An additional 58 patients later developed respiratory problems, and 15 of them required a tracheostomy. Although the edematous and inflammatory processes may respond to cortisone treatment, cartilaginous destruction continues. In McAdam's series, 13 patients died of airway collapse or obstruction, and 4 more of pneumonia. The histologic picture is not absolutely characteristic of any disease but may be suggestive. Specific diagnostic tests are lacking.

In a review of experience with 36 patients and study of 30 additional patients in 1998, Trentham and Le found a similar prominence of auricular chondritis (92%), arthritis (48%), laryngotracheal symptoms (39%), nasochondritis (33%), and ocular inflammation (25%), but in greater percentages than in earlier series.[29] Audiovestibular problems occur in 6 to 9%. Delay between first symptoms and diagnosis averaged 2.9 years.

Radiography, including tomography, conventional and computed, show upper airway changes and the latter lower airway changes (Figure 14-17). Dynamic studies may demonstrate collapse more clearly. Pulmonary function tests, especially flow volume curves, offer a means of following the disease progression. Bronchoscopy, or any manipulation of the airway, must be done with a light touch to avoid inciting further edema, inflammation, and acute obstruction.

In addition to treatment with corticosteroids, cytotoxic agents (such as cyclophosphamide, methotrexate, and azathioprine) are used. Beyond tracheostomy, and a T tube for temporary relief in a few with upper airway disease, there is no standard surgical treatment. The disease is too extensive and progressive to be managed by resection or reconstruction. When the larynx is severely involved, a tracheostomy becomes necessary. If the disease progresses distally to involve the lobar and segmental bronchi, there is lit-

FIGURE **14-17** *Relapsing polychondritis. Tracheal tomogram in a 28-year-old male with progressive dyspnea on exertion. The trachea is diffusely narrowed to 12 mm from just below the cricoid to carina. The left main bronchus is also narrowed to 6 mm. Over 12 years, the narrowing worsened. Computed tomography scan confirmed circumferential narrowing, thickening, and calcification of tracheal or bronchial walls. Subsequently, ear changes occurred and biopsy of cartilage was consistent with relapsing polychondritis.*

tle that can be done therapeutically at present. Pulmonary sepsis may follow diffuse bronchial collapse. Survival with medical treatment has improved some over the years.[29]

Sarcoid

Sarcoid (or sarcoidosis) is a systemic disease of unknown origin or origins, possibly an autoimmune condition. It demonstrates racial proclivity for blacks. Many organ systems may be involved, but almost always the respiratory system. The condition is characterized pathologically by noncaseating granulomas. Sarcoid varies in manifestations, severity, and outcome.[33] In addition to nonspecific symptoms such as fatigue, anorexia, weight loss, and fever, respiratory complaints may include exertional dyspnea, retrosternal chest pain, and cough. Respiratory symptoms are prominent, but many patients are asymptomatic. The full spectrum of the disease will not be recounted here. The thoracic surgeon's encounter is often in reply to the need for mediastinal lymph node biopsy or bronchoscopic carinal biopsy, since both tissues are highly susceptible to sarcoidal granulomatous involvement. Nearly half of the bronchoscopic biopsies and more of the mediastinal node biopsies will be positive, but other causes of noncaseating granuloma must be ruled out.

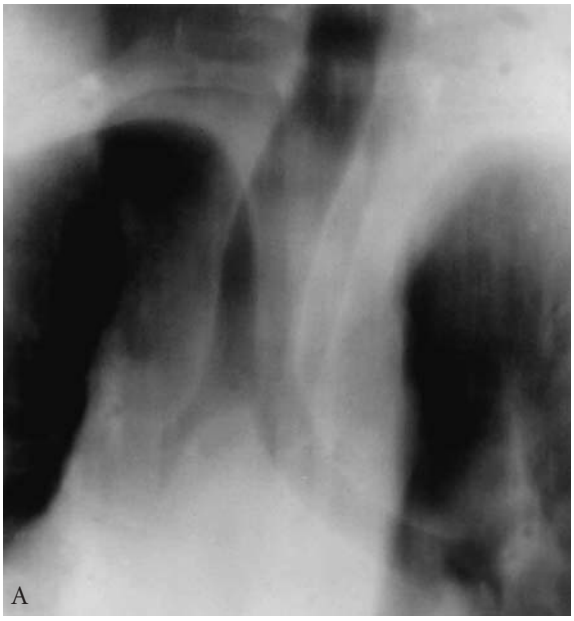
Sarcoid produces major airway obstruction in two ways: 1) by massive enlargement of mediastinal and hilar lymph nodes, with compression and distortion of the airway; and 2) by intrinsic fibrotic change in the wall of the trachea and bronchi (Figure 14-18).[34] Endobronchial nodules may be present with or without visible or symptomatic bronchostenosis.[35] Sarcoid may also cause hoarseness and, later, obstruction by laryngeal involvement. These changes are usually concomitant with parenchymal pulmonary changes. Airflow limitation, wheezing, and stridor may be present in 10% of patients, although the usual functional defect in pulmonary sarcoid is restrictive.[35] When the walls of the trachea or main bronchi or both of these structures are involved, the length of the stenotic segment may be long and the process progressive.

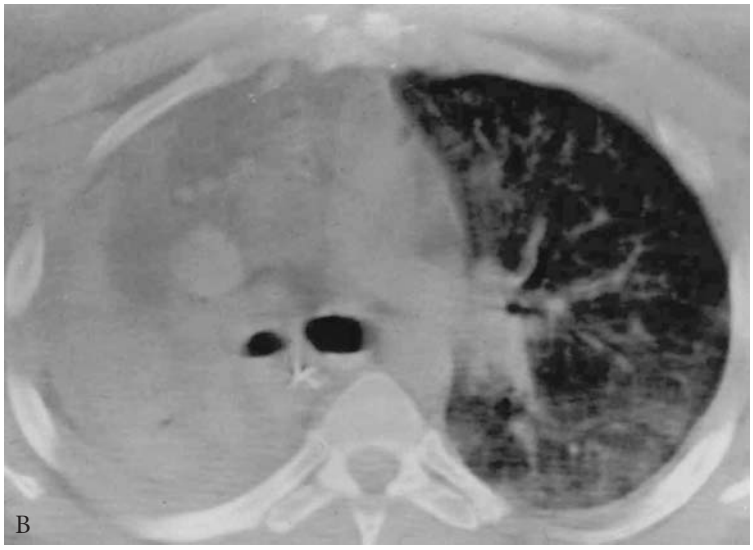
Because of the diffuseness of involvement and progression, these lesions are rarely amenable to resection and reconstruction. Periodic dilation will tide some patients over for a long time. As the tissues contract by cicatricial evolution of the scarring, the hilum may be pulled upward and the left main bronchus, in particular, takes on a sharp curvature beneath the aortic arch, making continued dilation increasingly difficult. If both main bronchi are stenotic, the patient's obstruction will mimic a fixed upper airway obstruction clinically and on functional study.[36] Sarcoidosis may also cause airway obstruction in the larynx by granulomas. This is usually associated with cutaneous disease, especially of the lupus pernio form.[37]

I prefer to use a rigid bronchoscope with Jackson bougies, or a small diameter Maloney bougie to dilate a markedly angulated bronchus. Dilation has also been done via a flexible bronchoscope.[38] There is no clear proof of the efficacy of inhaled corticosteroids, or, for that matter, systemic steroids for major tracheobronchial obstruction.

Wegener's Granulomatosis

Wegener's granulomatosis is a disease of unknown etiology characterized by granulomas, vasculitis, and necrosis, which involves the upper and lower respiratory tract, kidneys, central nervous system, and other organs. In 158 patients, 97% were white, the genders were equal in number, and 85% were over 19 years of age.[39] Typically, patients suffer serious consequences of the disease or its treatment (cyclophosphamide and glucocorticoids). In 99 patients followed longer than 5 years, 44% had prolonged remissions, but 13% died of the disease or treatment. Methotrexate has served as an alternative to cyclophosphamide and other drugs will undoubtedly be used in the future.[40] Sixteen percent of 158 patients treated at the National Institutes of Health (NIH) had subglottic stenosis, about double the previously reported incidence.[41] Subglottic stenosis is more common in Wegener's patients who are under 20 years of age. Some had limited disease, in that the kidneys were not involved. The larynx alone may be involved without evidence of vasculitis in other systems.[39,42] Patients with subglottic stenosis from Wegener's granulomatosis present with symptoms of effort





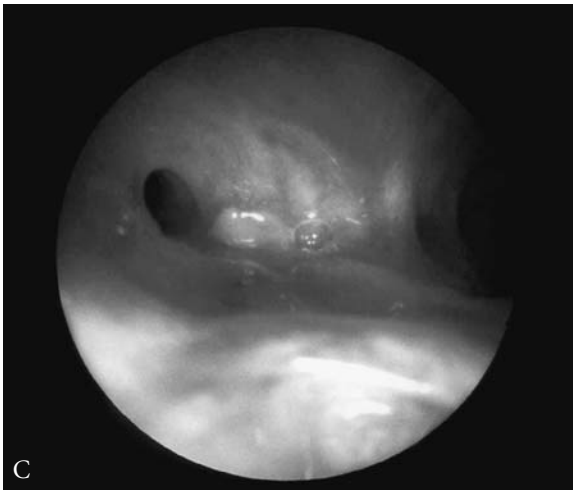
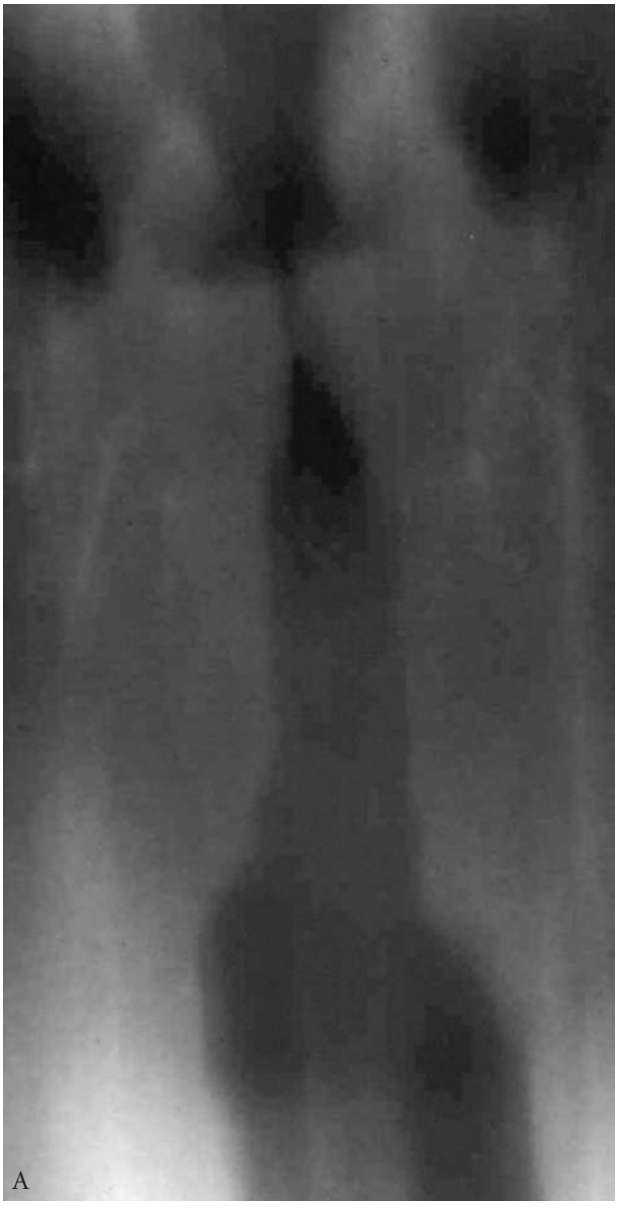
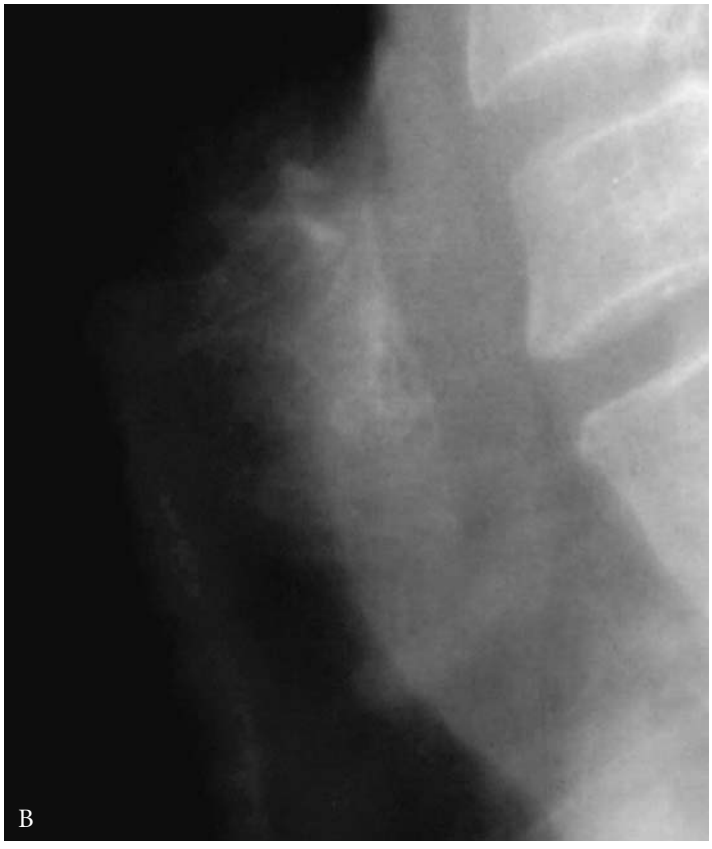


FIGURE **14-18** *Intrinsic bronchial stenosis due to sarcoidosis. A, Tomogram of carina in a 30-year-old man with a 3-year history of repeated and persistent pneumonia in the right lung, with collapse and fibrosis of middle and lower lobes and air trapping in the upper lobe. The right main bronchus is severely narrowed and cut off distally, approximately where the upper lobe bronchus and bronchus intermedius branch. This patient also had episodes of left-sided pneumonia.* B, *Computed tomography scan in the same patient as* A. *The carina is displaced to the right. Note the patchy infiltrates in the left lung. The entire right bronchial tree was stenosed, and involved nodes were prominent in right paratracheal and subcarinal distributions. Pneumonectomy was necessary.* C, *Bronchoscopic view in another patient, showing severe left main bronchial stenosis.*

dyspnea, hoarseness, cough, and discomfort in the throat. The local lesion is best defined by linear laryngotracheal x-rays supplemented by CT or magnetic resonance imaging and by bronchoscopy (Figure 14-19). Laryngeal biopsies may not be diagnostic. Adequate nasoseptal biopsies, however, are often helpful.[43] The ANCA is usually elevated and can be highly specific in diagnosis.[25] In isolated lesions, the differential from idiopathic stenosis and from early polychondritis may be unclear (see Figure 34 [Color Plate 15]).

Lebovics and colleagues found that 92% of 158 patients with Wegener's granulomatosis had otolaryngologic manifestations, 25 with subglottic stenosis.[41] Five responded to cytotoxic and steroid treatments alone. Sixteen of 20 who had fixed subglottic stenosis were treated by dilation, laser resection, and laryngotracheoplasty. Thirteen needed a temporary tracheostomy. Five underwent laryngotracheoplasty, 2 with microvascular reconstruction, using a rib graft with attached intercostal artery. An anterior and posterior cricoid split with cartilage augmentation was performed, with postoperative T tube stenting. Nonoperative treatment was applied first, including repeated intralesional injections of Depo-Medrol. Laser resection appeared to produce more scarring.





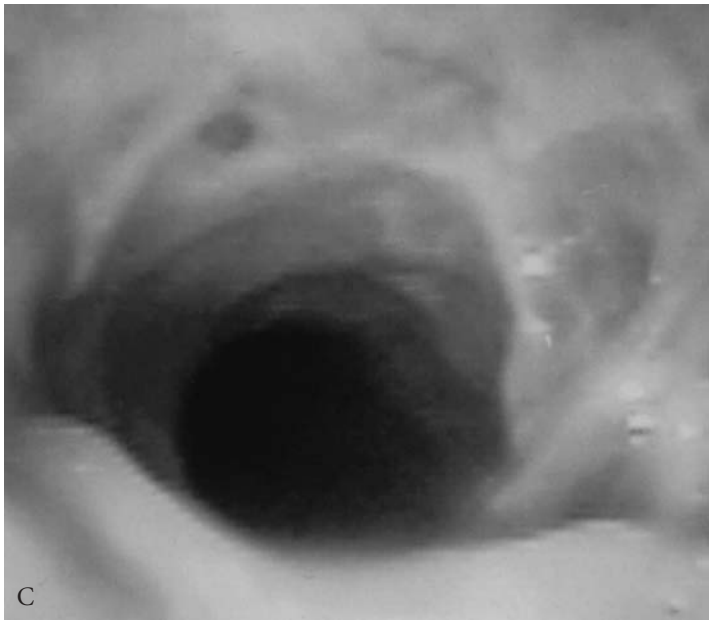


FIGURE **14-19** *Wegener's granulomatosis involving the upper airway.* A, *Anteroposterior tomogram of larynx and upper trachea. Note the severe narrowing of the subglottic larynx, beginning just below the glottis and extending into the proximal trachea.* B, *Lateral tomogram of the same lesion of* A. C, *Bronchoscopic view of the upper trachea in another patient. Irregular circumferential scarring is noted. In other cases, the gross appearance is similar to idiopathic stenosis. Bronchoscopic biopsy is not usually diagnostic. Also, see Figure 34 (Color Plate 15).*

Daum and colleagues from the Mayo Clinic found that 30 of 51 patients (59%) with proven Wegener's granulomatosis, who were followed bronchoscopically, had endobronchial abnormalities due to the granulomatous processes.[42] Five had subglottic stenosis, 18 had ulcerating tracheobronchitis (some with pseudotumors), 4 had tracheal or bronchial stenosis, and 2 had bleeding from unidentified sources. Seven

of 9 patients with ulceration progressed to stenosis. Treatment was by dilation, laser, and Silastic stent. No correlation was found between observed inflammation and ANCA titers.

Herridge and colleagues had success in 3 patients, who were in remission, treated by resection and thyrotracheal anastomosis, despite concurrent use of prednisone and cyclophosphamide.[44] Protective tracheostomy or a T tube was used in all 3. Stenosis did not recur in 2 with longer follow-up. We performed resection in 6 patients with Wegener's granulomatosis confined to the upper airway but misdiagnosed as idiopathic laryngotracheal stenosis. Two did well in long term, but 4 later restenosed.

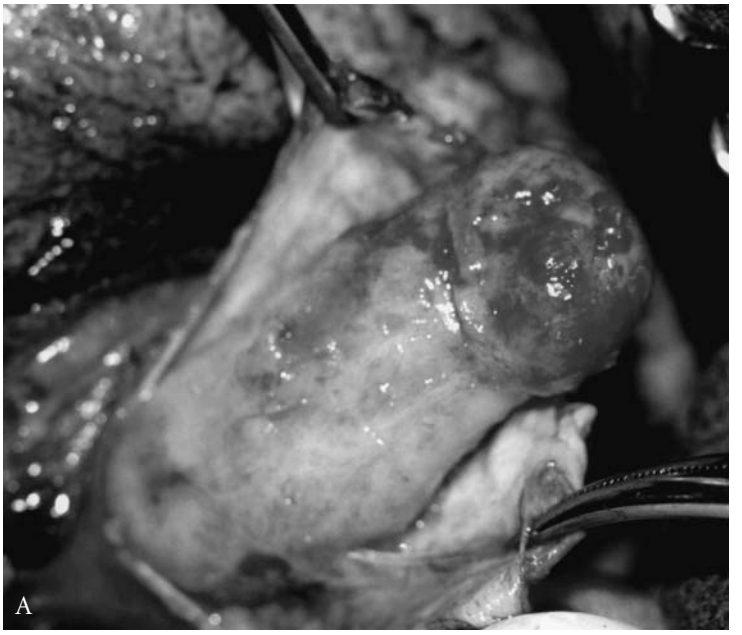
An NIH protocol from 1991 consisted of dilation and intralesional steroid injection at monthly intervals. The hope was that collagenous scar would be resorbed. Following dilation, the area of stenosis was injected with methylprednisolone acetate in four quadrants. One cc containing 40 mg of the drug was injected in each site. About half the volume leaked out. The patient was given Decadron briefly to minimize postoperative laryngeal swelling. Cyclophosphamide and glucocorticoids were continued only in those patients with other systems involved. Twenty patients underwent 113 procedures in total, with 14 requiring multiple treatments.[45] Six patients with tracheostomies were decannulated and none of the others needed a tracheostomy, in contrast to earlier experience, where over half of the patients required a tracheostomy. Given the unpredictable course of Wegener's granulomatosis, its varied manifestations, and response to treatment, conservative initial management of subglottic stenosis seems advisable. We presently employ repeated dilations with intralesional injection of methylprednisolone acetate (Depo-Medrol). Surgical resection is reserved for nonresponding patients who have a reasonably localized disease and who have been in prolonged systemic remission, and further, where the airway lesion does not evince florid inflammation. One-stage laryngotracheal resection is performed, when feasible (see Chapter 25, "Laryngotracheal Reconstruction"). Airway stenosis due to Wegener's granulomatosis should be approached surgically with the greatest caution, if at all.

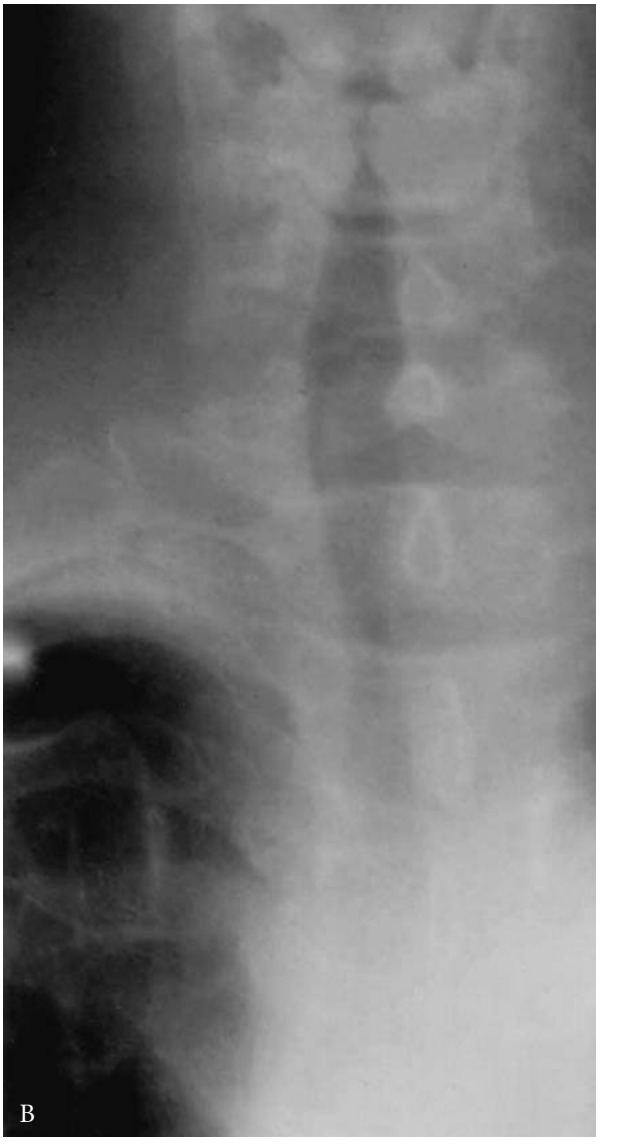
Amyloidosis

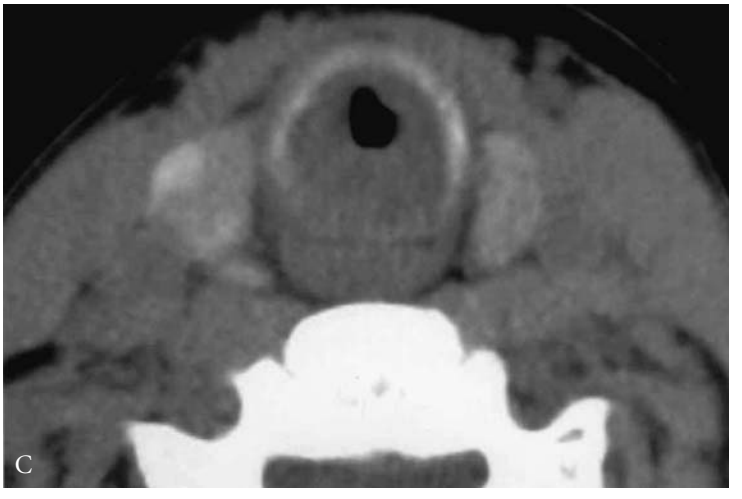
Falk and colleagues point out that "amyloidosis is not a single disease, but a term for diseases that share a common feature: the extracellular deposition of pathologic insoluble fibrillar proteins in organs and tissues."[46] Different proteins make up the amyloid fibrils in primary amyloidosis (AL), in reactive systemic (secondary) amyloidosis (AA), and in rare familial disease (ATTR most common). Secondary amyloid has diminished in frequency with a lower incidence of chronic infectious diseases. Clinical presentations vary, but the organs most often involved are the kidney, heart, and peripheral nervous system.

The disease is uncommon and airway involvement is more so. The now differentiated types of amyloid have not been identified in airway cases until recently. AL amyloidosis appears to have special affinity for lung tissue, presenting as nodular pulmonary and tracheobronchial amyloidoses.[47,48] The hyaline-eosinophilic material also deposits in the lamina propria of the bronchial mucosa of different parts of the bronchial tree. Multiple coalesced nodules can narrow the bronchial lumen, and involvement ranges from a localized segment to a large part of the bronchial tree (Figure 14-20). Systemic amyloidosis, which often involves the lungs, has a poor prognosis. The pulmonary involvement is usually diffuse and infiltrative in this case.

More localized, tumor-like amyloidoma, whether in the lung or tracheobronchial tree, appears to have a quite benign course, except for the consequences of airway obstruction, which may be fatal.[48,49] It is largely dissociated from systemic disease. Tracheobronchial lesions may be quite localized or infiltrate over long distances of the larynx or bronchi.[47,50,51] Multifocal submucosal plaques are more common than "amyloidomas," or tumor-like masses. Regional lymph nodes may exhibit amyloid.[48] In extensive tracheal involvement, amyloid material is also found, deposited peritracheally in the mediastinum and in the esophageal wall. Calcification may be noted in nodular amyloidomas on CT scan. In looking at 48 patients with lower respiratory tract amyloidosis, Hui and colleagues counted 28 with single or multiple nodules, 14 with tracheobronchial disease, of whom 4 were localized and 6 had diffuse interstitial pulmonary infiltrates.[51]







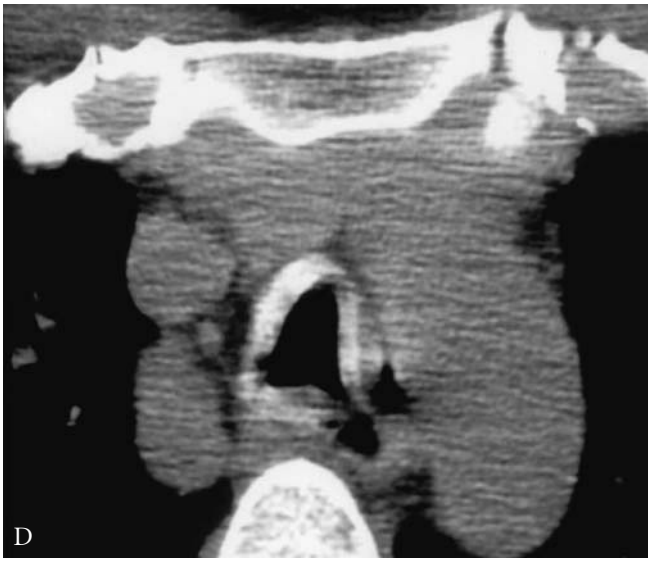
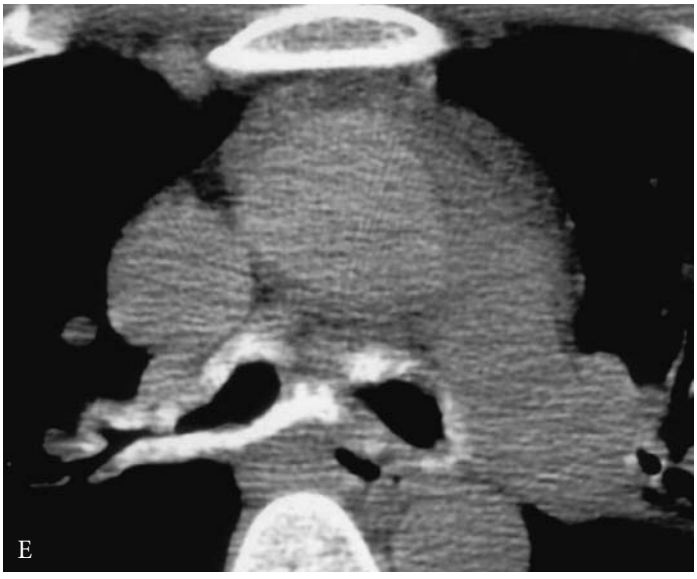
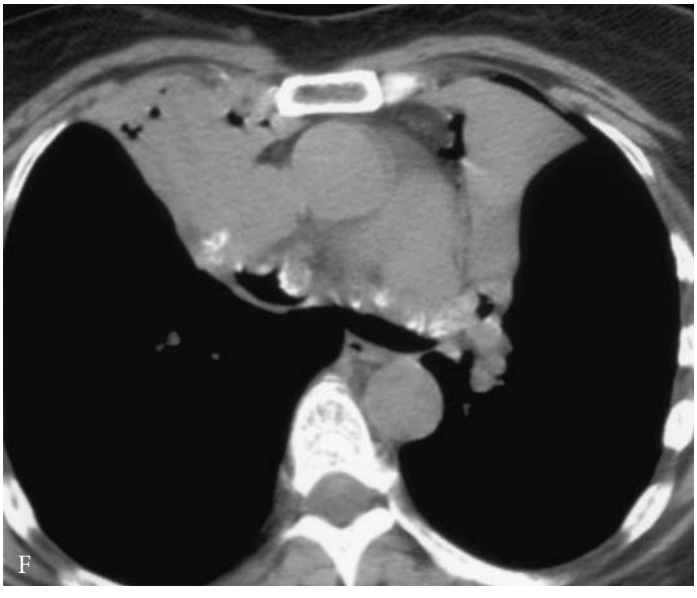


FIGURE **14-20** *Amyloidosis involving the airway.* A, *"Amyloidoma" localized to left main bronchus. The lesion fills the bronchus like a cork. A left main bronchial sleeve resection was performed, saving the lung.* B, *Anteroposterior view of the trachea, showing extensive linear involvement in a different patient.* C,D *Computed tomography scans of another patient, 54 years old, with tracheobronchial amyloidosis. The cervical trachea is markedly obstructed* (C). *The upper mediastinum and trachea in this patient are infiltrated by amyloid* (D). *Note the calcification.*





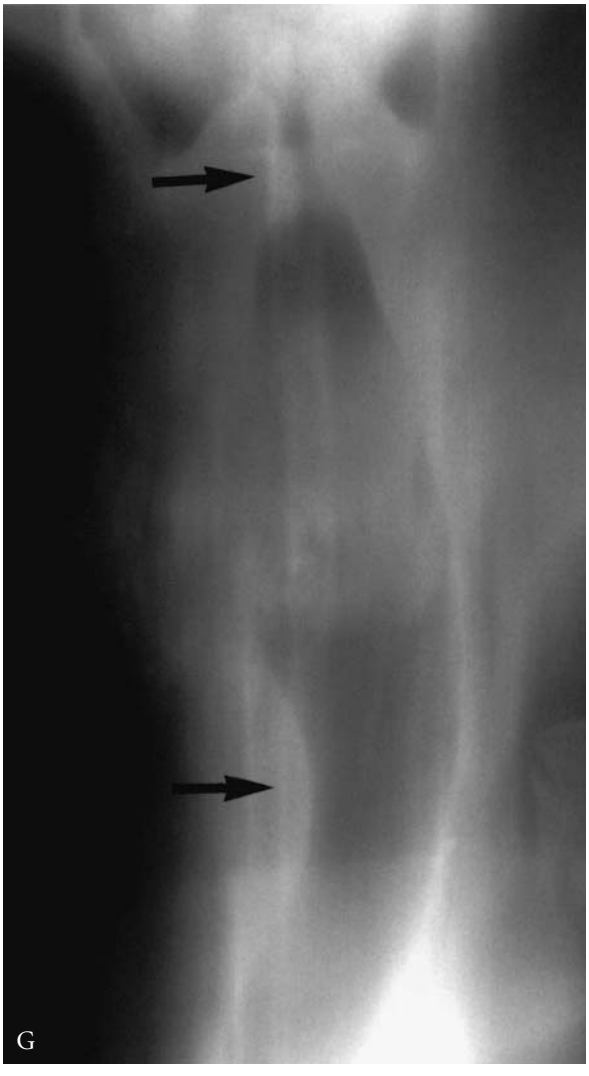


FIGURE **14-20** (CONTINUED) E, *Amyloid is present in the same patient's tissues at the carinal level. Bronchi are thickened and calcified.* F, *Bronchial and mediastinal infiltration and calcification of amyloid are particularly well seen in the same 54-year-old patient.* G, *Tomogram of the larynx and trachea in a 19-year-old male with at least 10 years of slowly progressive dyspnea, much worsened in the year and a half prior to study. The* upper arrow *indicates the glottis. Below this is the image of the amyloid with calcification invading the larynx and trachea. The* lower arrow *indicates deformation of the midtrachea by mediastinal amyloid accumulation.*

A literature review in 1972 produced 25 instances of primary localized tracheobronchial amyloidomas: trachea only in 2, bronchus only in 9, and trachea and bronchus in 14.[52] The genders were evenly spread, and the age range was 29 to 74 years (mean 53 years). Symptoms and signs were obstructive. Utz and colleagues described 4 patients with localized tracheobronchial amyloid in 55 patients with pulmonary amyloidosis (systemic and localized).[49]

Symptoms of nodular or tumorous tracheobronchial amyloidosis are cough, sputum, dyspnea, hemoptysis, and fever. The mucosa bleeds easily, but biopsy is necessary for diagnosis. Since it occurs so

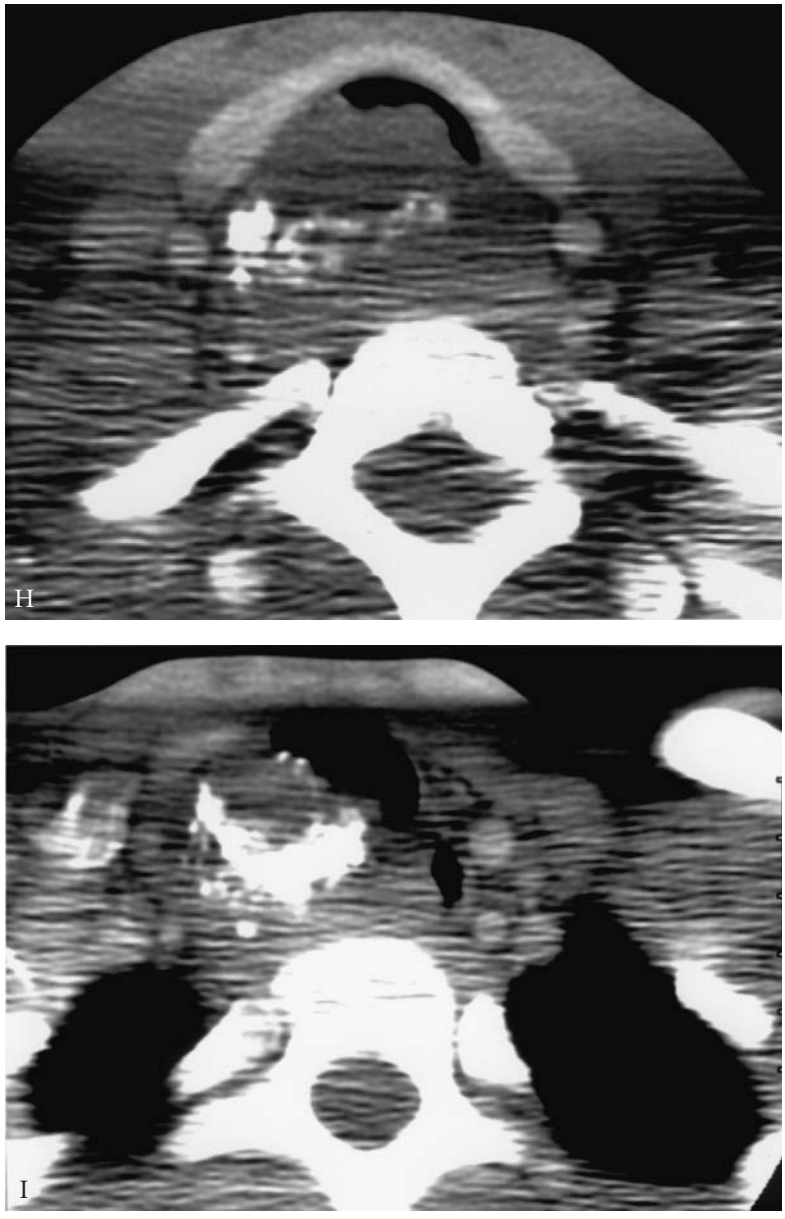


FIGURE **14-20** (CONTINUED) H, *Computed tomography image of the same patient in Figure 14-20*G, *showing severe tracheal compromise. Note the calcification in the amyloid mass.The splayed shadow anterior to the trachea is the thyroid.* I, *Upper thoracic trachea is also invaded. Amyloid infiltrates the esophageal wall and narrows its lumen. A 5 cm length of the trachea and the anterior subglottic larynx were resected, plus esophageal muscular coats. Putty-like amyloid infiltrated the mediastinum and behind the posterior cricoid. Good result was finally achieved but a re-resection of the anastomotic stenosis, due to failure of healing of the first anastomosis, was required.*

infrequently, it can easily be mistaken for a tumor, or even other infiltrative processes such as tracheopathia osteoplastica. Tracheobronchial amyloidosis is not usually associated with diffuse interstitial pulmonary amyloid deposition, as noted above. Classically, the protein stains with Congo red, but sophisticated techniques now permit precise determination of the amyloid type.[46]

Depending upon the location and extent of the process, surgical treatment may be possible.[48,50] A localized tracheal or bronchial deposit may simulate tumor. The lesion may enlarge slowly, in time producing obstructive recurrent pneumonia or atelectasis. Death occurs from recurrent pneumonia or respiratory failure under these circumstances. If possible, the involved segment of trachea or bronchus is resected (see Figure 14-20*A*). Residual amyloid may necessarily be left at resection margins, predicting possible slow future recurrence. The rate of clinical progression is not defined, due to lack of experience. In one of our patients, a pulmonary shadow had been present for 22 years.[48] In another very extensive lesion in a

young man, an extended laryngotracheal resection was necessary (see Figures 14-20*G*,*H*,*I*). A T tube was required after partial anastomotic separation had occurred, but eventually, a re-resection of the localized stenosis was successfully accomplished, resulting in an intact airway despite the extent of resection. Naef and colleagues operated on a similar patient and cited 22 prior reported cases with diffuse or localized pseudotumors of the tracheobronchial tree, usually treated at that time by bronchoscopic means.[50]

In a diffuse disease, we have used palliative bronchoscopic core-out treatment, whereas others have preferred laser therapy.[49] See also Figures 3-59, 3-60, and 3-61 (Color Plate 8).

Intrinsic Lesions Which Deform the Trachea

A number of conditions largely display gross deformation of the trachea in several characteristic patterns. These changes appear to be intrinsic to the trachea rather than being due to compression by adjacent structures or masses. Included are saber-sheath trachea, tracheopathia osteoplastica, and tracheobronchomegaly.

Saber-Sheath Trachea

A normal adult trachea has an essentially oval cross section, with the coronal diameter greater than that of the sagittal, although in many it is nearly circular (see Chapter 1, "Anatomy of the Trachea"). With increasing age, the configuration of the lower third of the trachea may be altered by the left lateral impression of an enlarging aorta. "Saber-sheath" trachea, however, describes a coronal narrowing of the entire *intrathoracic* trachea from both sides, with a corresponding increase in the sagittal diameter, usually affecting about the lower two-thirds of the trachea. The proximal cervical segment retains a normal shape (Figure 14-21). Saber-sheath trachea is identified most often in older patients, particularly in males over 50 years of age. Ninety-five percent of patients appear to suffer chronic obstructive pulmonary disease (COPD).[53,54] Not all patients with COPD, however, show saber-sheath deformity. In most patients, it is an incidental finding. However, with extreme progression of the deformity, a large part of the posterior lateral walls of the trachea approximate to one another on coughing and on increased respiratory effort, especially expiratory. As the cross-sectional area of the trachea is reduced, the rate of airflow is also reduced.[54] The patient finds it increasingly difficult to generate a cough sufficiently forceful enough to clear tenacious secretions, which are characteristic of the pulmonary disease.

Greene and Lechner described 13 patients with such narrowing, recalling the original use of the term in 1905.[53] Patients' ages ranged from 52 to 75 years, and clinical diagnoses were varied. All the patients had a history of heavy smoking, 10 had a diagnosis of chronic bronchitis, and 7 suffered from primary chronic obstructive pulmonary disease. The coronal intrathoracic tracheal diameters ranged from 7 to 13 mm with a mean of 10.5 mm; in contrast, the sagittal diameters showed a mean of 23.5 mm. The extrathoracic trachea was normal, with a coronal diameter of 20.6 mm. Ten of the 13 patients demonstrated calcification in the tracheal rings, which was probably age related.

The mechanical forces that lead to this deformation of the intrathoracic trachea are not understood. It should be emphasized that the cartilages are *not* malacic. It is the change in shape and approximation on respiratory and tussive efforts that can lead to clinical problems. In a very rare case, I found it necessary to splint the trachea externally (using specially made polypropylene rings), in order to allow the patient to clear secretions adequately. A large bore tracheal T tube or inlying stent could also provide a means for maintaining an open airway during expiration and cough in these few critical patients. Definition of the extent to which the airway is obstructed by the process is obtained by bronchoscopic examination in the awake patient, flow volume curves, and inspiratory and expiratory thin-section CT scans of the trachea. Patients who have this deformity, but who are not symptomatic from it, and who require tracheal resection between the differently shaped proximal and distal portions of the trachea, do not present any difficulties in anastomosis. Sutures are placed proportionally. The tracheal ends are sufficiently flexible to permit easy anastomotic accommodation.

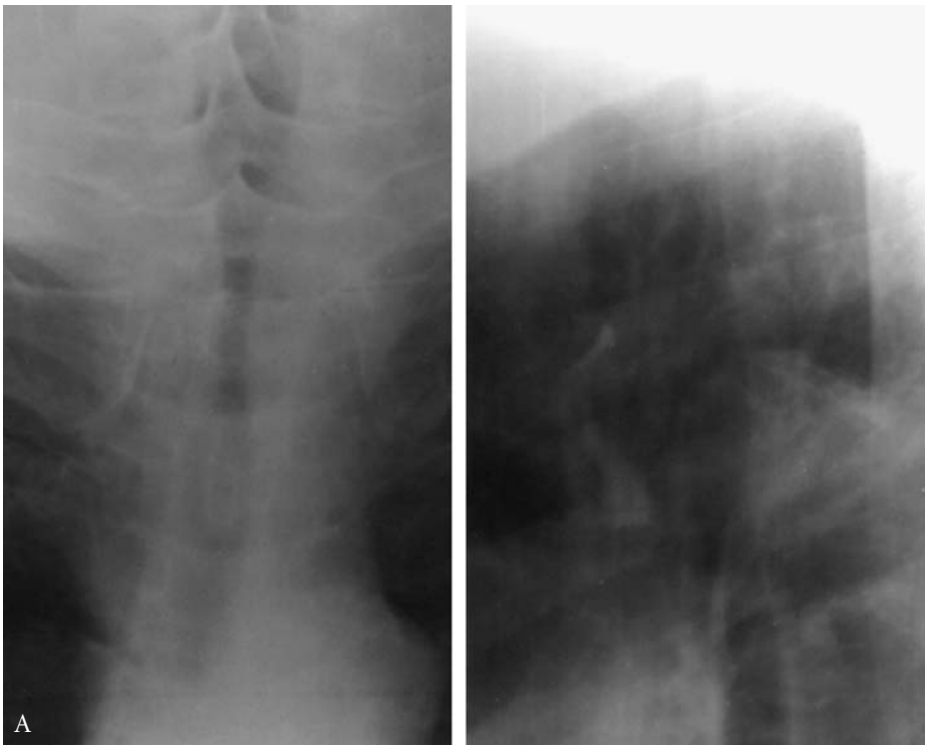


FIGURE **14-21** *Saber-sheath trachea.* A, *Anteroposterior* (left) *and lateral* (right) *roentgenograms, showing normal appearing cervical trachea with side-to-side narrowing of the intra-thoracic trachea. Near the carina, the trachea begins to widen again.*

Tracheopathia Osteoplastica

In tracheopathia osteoplastica (TPO), multiple submucosal osseocartilaginous nodules are present in the trachea, subglottic larynx, and bronchial tree (see Figures 42 and 43 [Color Plate 16]). An alternative name, tracheobronchopathia osteochondroplastica, is more completely descriptive, lacking only "laryngo"—but is excessively long and something of a tongue twister. The nodules are distributed only over underlying cartilages and not over the membranous wall. The most prominent nodules are usually seen in the trachea and the next most prominent in the main bronchi. Some are tiny and scattered like seeds. Others grow to considerable size (4 to 6 mm). The nodules are of hyaline cartilage with foci of ossification and lie submucosally, essentially forming a partial intratracheal osseocartilaginous cylinder (except over the membranous wall). Bridges of bone, cartilage, and collagen connect the inner tracheal cylinder formed by the pathologic bone and cartilage with the perichondrium of the regular tracheal cartilages. These findings led Young and colleagues to support ecchondrosis of the tracheal cartilages as the pathological process.[55] An often present saber-sheath configuration in these patients may commence just below the cricoid cartilage, rather than being confined to the intrathoracic trachea, as it is in isolated saber-sheath tracheal deformation. Portions of the trachea may be more severely affected by TPO than others, and occasionally involve only part of the trachea. The etiology is unknown. Earlier suggestions that TPO is an end stage of amyloidosis have not held up.[56] Other theories relate the calcifications to chronic purulent tracheitis or abnormalities in tracheal elastic tissue.[56,57] This rare condition is seen in older adults and is often asymptomatic. Fifteen patients were diagnosed with TPO over 36 years at the Mayo Clinic.[56] A number of cases reported were discovered incidentally at postmortem examination.[55] TPO may, however, progress insidiously to produce respiratory symptoms, including dyspnea, cough, sputum production and retention, wheezing, hoarseness, and hemoptysis.[56] A long history of exertional dyspnea and respiratory infection may be elicited. As obstruction worsens, the patient has progressive difficulty in raising viscid secretions.

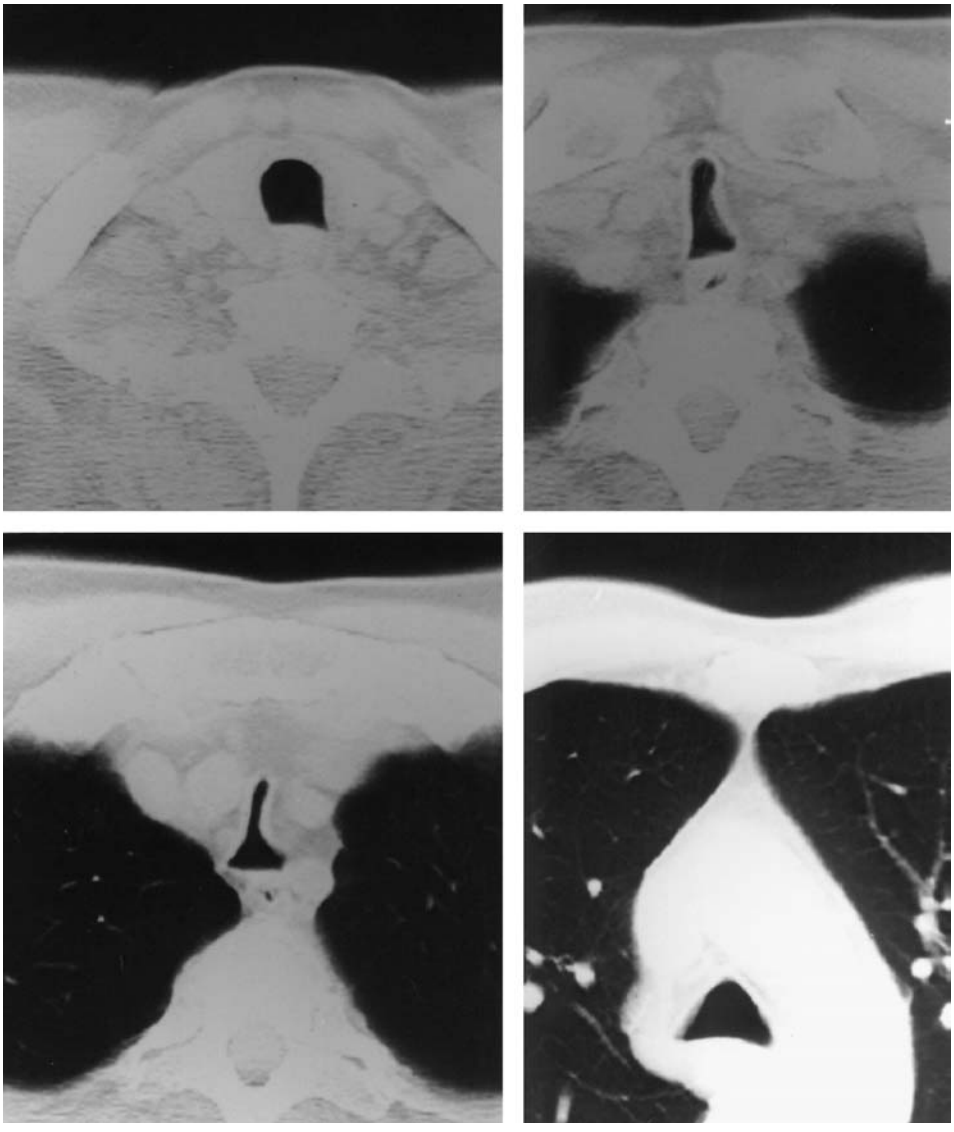


FIGURE **14-21** (CONTINUED) B, *Computed tomography scans showing changes in tracheal configuration with progression, from the cervical (*left upper image*), to the upper thoracic (*right upper and left lower image*), to the lower thoracic (*right lower image*). The shape of a saber sheath is best exemplified in the* right upper image. *As the carinal branching is reached, the airway becomes more triangular.*

When saber-sheath deformity is concurrent, as is often the case, the obstruction is worsened. Atrophic rhinitis and pharyngitis may be concurrent.[57]

Tomograms and CT scans show typical changes, with scalloped calcification in the nodules lining the lumen of the involved segments of the airway (Figure 14-22). These nodules are within the inner border of the cartilages, which are not themselves involved. Bronchoscopy is also diagnostic (Figure 14-23). The lesions may be seen extending from the subglottic larynx to distal bronchi. In advanced cases with marked airway constriction, it may not be possible to force an adult rigid bronchoscope very far into the trachea since the process is so rigid. Biopsy is extremely difficult due to the hardness of the nodules, and it is not usually productive or necessary.

Attempts to treat this condition by removal of the nodules either with bronchoscopic biopsy forceps or with laser have not been highly successful.[56] The trachea may be impossible to dilate, so that per oral stent placement is not feasible. Segmental stenosis of the trachea by TPO is infrequent. Localized disease, although rare, can be resected.[58,59] In 1 of our patients who had squamous cell carcinoma identified by biopsy in an obstructing localized segment of TPO, a resection gave complete relief. In 4 patients with severe and

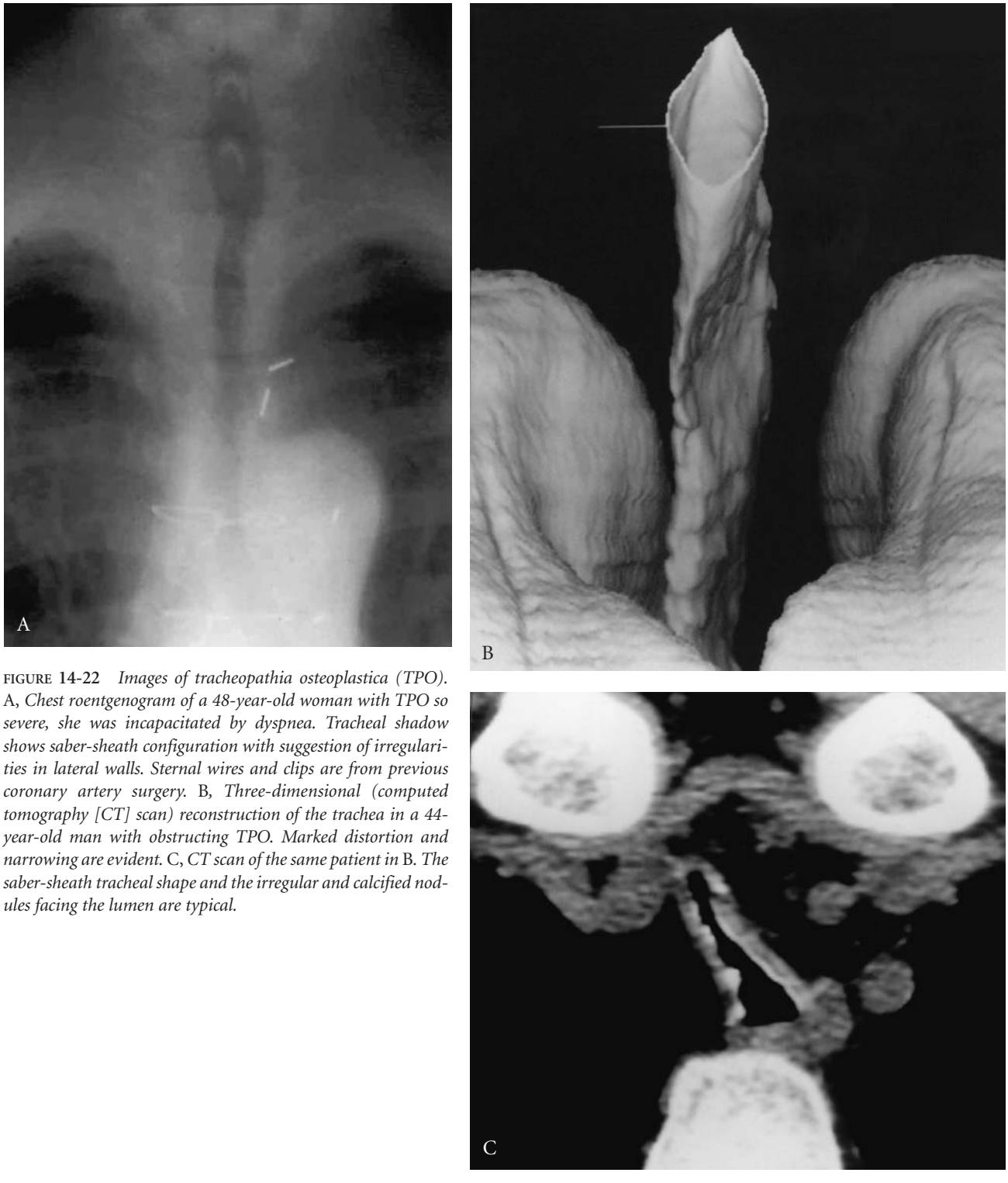
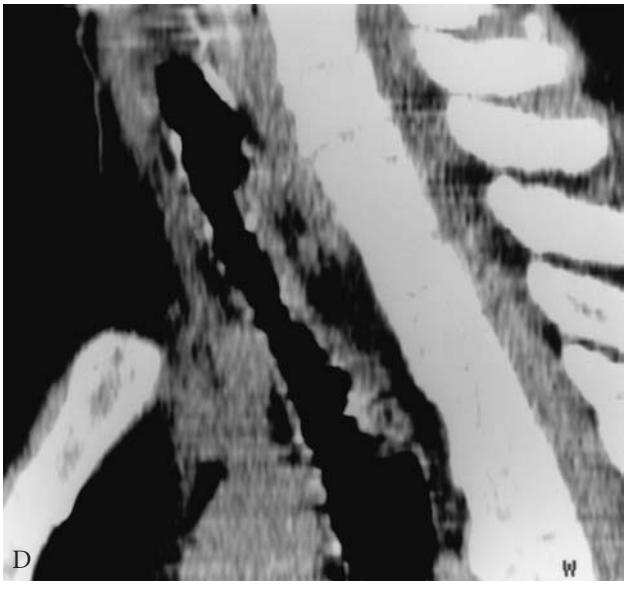


FIGURE **14-22** *Images of tracheopathia osteoplastica (TPO).* A, *Chest roentgenogram of a 48-year-old woman with TPO so severe, she was incapacitated by dyspnea. Tracheal shadow shows saber-sheath configuration with suggestion of irregularities in lateral walls. Sternal wires and clips are from previous coronary artery surgery.* B, *Three-dimensional (computed tomography [CT] scan) reconstruction of the trachea in a 44-year-old man with obstructing TPO. Marked distortion and narrowing are evident.* C, *CT scan of the same patient in* B. *The saber-sheath tracheal shape and the irregular and calcified nodules facing the lumen are typical.*

incapacitating airway obstruction due to disease involving the entire trachea (the more common presentation), relief was sought by reshaping the obstructed trachea over a silicone T or T-Y tube.[60,61] The entire length of the trachea was divided vertically in the anterior midline from the cricoid to the carina (see Chapter 32,



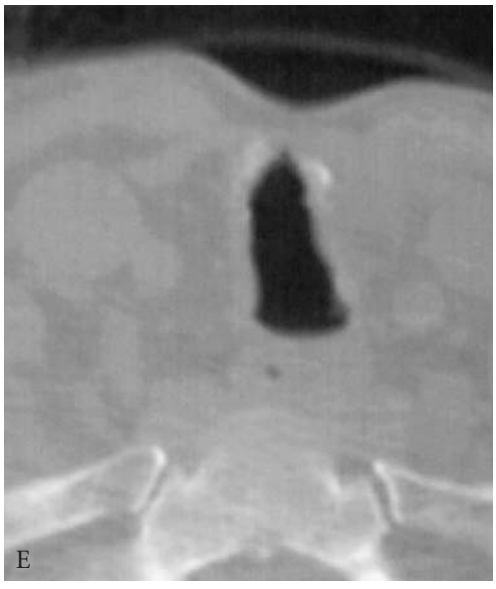


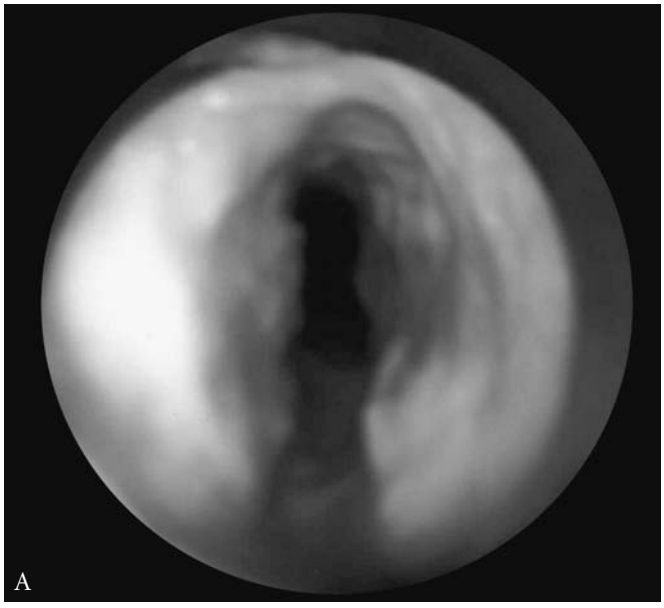
FIGURE **14-22** (CONTINUED) D, *Lateral longitudinal reconstruction. The apparently posterior nodules are in the distorted lateral wall (see Figure 14-22C), not the membranous wall, which is not involved.* E, *CT scan 4 years after T tube tracheoplasty in the patient in Figure 14-22C. The patient had returned to all activities, including small game hunting.*

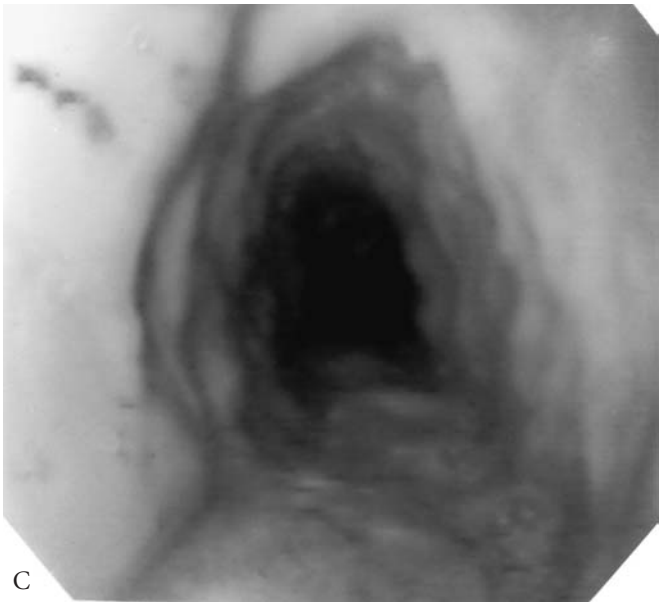
"Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea"). This permitted the rigid lateral walls to be hinged outward from the anterior midline incision, since the membranous wall is uninvolved. After placement of a T or T-Y tube, the anterior line of the incision was sutured closed. Pedicled strap muscles were interposed between the linear suture line and the brachiocephalic artery. After 4 to 6 months, when healing had occurred in the outwardly hinged position, the T tube was extracted. Airway patency was maintained in 3 patients long term (see Figures 14-22*E*, 14-23*C*). One patient continued to need a T tube, but was lost to follow-up. Viscid secretions, as expected, continue to be produced by the abnormal mucosa, but can be cleared through a now widely patent trachea. I have not, thus far, encountered a case where distal involvement also required surgical reshaping of the bronchi. This should be feasible with a custom-made T-Y tube. Kutlu and colleagues treated a single patient with narrowing of only the upper half of the trachea, by side to side slide tracheoplasty, with satisfactory follow-up at 15 months.[62] See also Figures 3-62, 3-63, and 3-64 (Color Plate 8).

Tracheobronchomegaly

Tracheobronchomegaly (TBM), the Mounier-Kuhn syndrome, is characterized by a hugely dilated trachea and central bronchi and frequently associated with recurrent respiratory infection. Widening of the trachea transversely may reach 35 to 45 mm or greater. Mounier-Kuhn described the radiographic and bronchoscopic presentations of a number of these patients and gave the syndrome its current name.[63] TBM is most likely of congenital origin.[64–67] It has been identified in the neonate, but usually becomes clinically manifest in adult life.[64] Katz and colleagues identified the condition in patients between the ages of 8 and 64 years, but found the majority to be in the fourth and fifth decades.[64] The patient often finds it difficult to date the origin of symptoms exactly, since these have appeared insidiously over many years, sometimes being retrospectively identifiable in childhood. Longitudinal elastic fibers of the central airways are atrophic or absent.

Symptoms are progressive dyspnea on exertion, ineffective cough, and difficulty in raising secretions. Emphysema, respiratory infections, and bronchiectasis follow.[64,65,67] The trachea appears hugely dilated on chest x-ray and on bronchoscopy. CT scan demonstrates the defects graphically (Figure 14-24). Cartilages





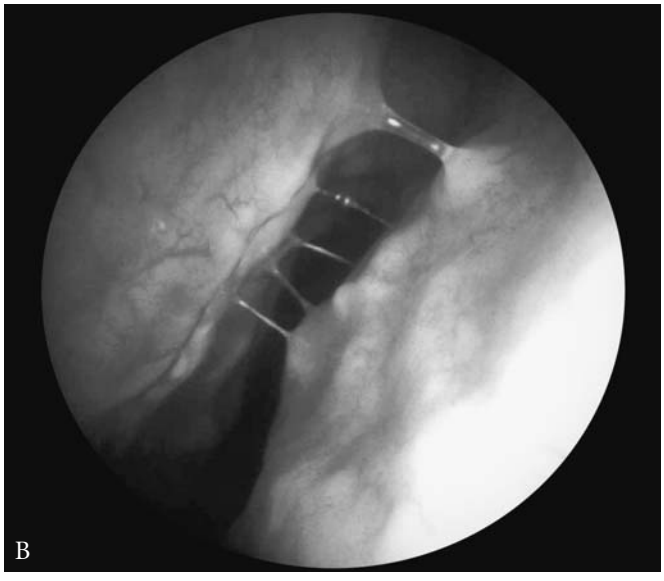


FIGURE **14-23** *Bronchoscopic views of tracheopathia osteoplastica.* A, *Patient is described in Figure 14-22*A, *having characteristic overall deformation of the trachea, with multiple submucosal nodules of varied size. There was marked luminal narrowing.* B, *Trachea of the patient described in Figure 14-22*B. *Note the extreme compromise of the airway and the viscid secretions.* C, *Postoperative view of the patient viewed in Figure 14-23*A, *9 years later, showing a completely adequate lumen. See also Figures 42 and 43 (Color Plate 16.)*

of the tracheal and central bronchial walls are markedly elongated and deformed, particularly in the mid and lower trachea.[67,68] Malacia is present, sometimes to an extreme degree. Dynamic change is observed, with collapse occurring at expiration. The trachea, right main bronchus, and left main bronchus greatly exceed normal maximum values for transverse diameters of 25, 23, and 20 mm, respectively (in men).[69] Peripheral airways are of normal caliber. The membranous wall is markedly widened. Tracheal sacculation can occur between the dysplastic rings and in the membranous wall and is bronchoscopically and radiologically visible. The curve of the cartilages becomes so distorted that there may be actual reversal of the anterior wall in some, so that it pushes in against the membranous wall. With expiration and with cough, the mucosa of the membranous wall approximates to a greater or lesser degree to the deformed anterior cartilaginous wall. High degrees of obstruction follow, particularly with respiratory or tussive effort.

In one patient, an attempt was made in several operations to reshape the anterolateral tracheal wall and the main bronchi, using polypropylene ring splints to support the cartilaginous wall, and

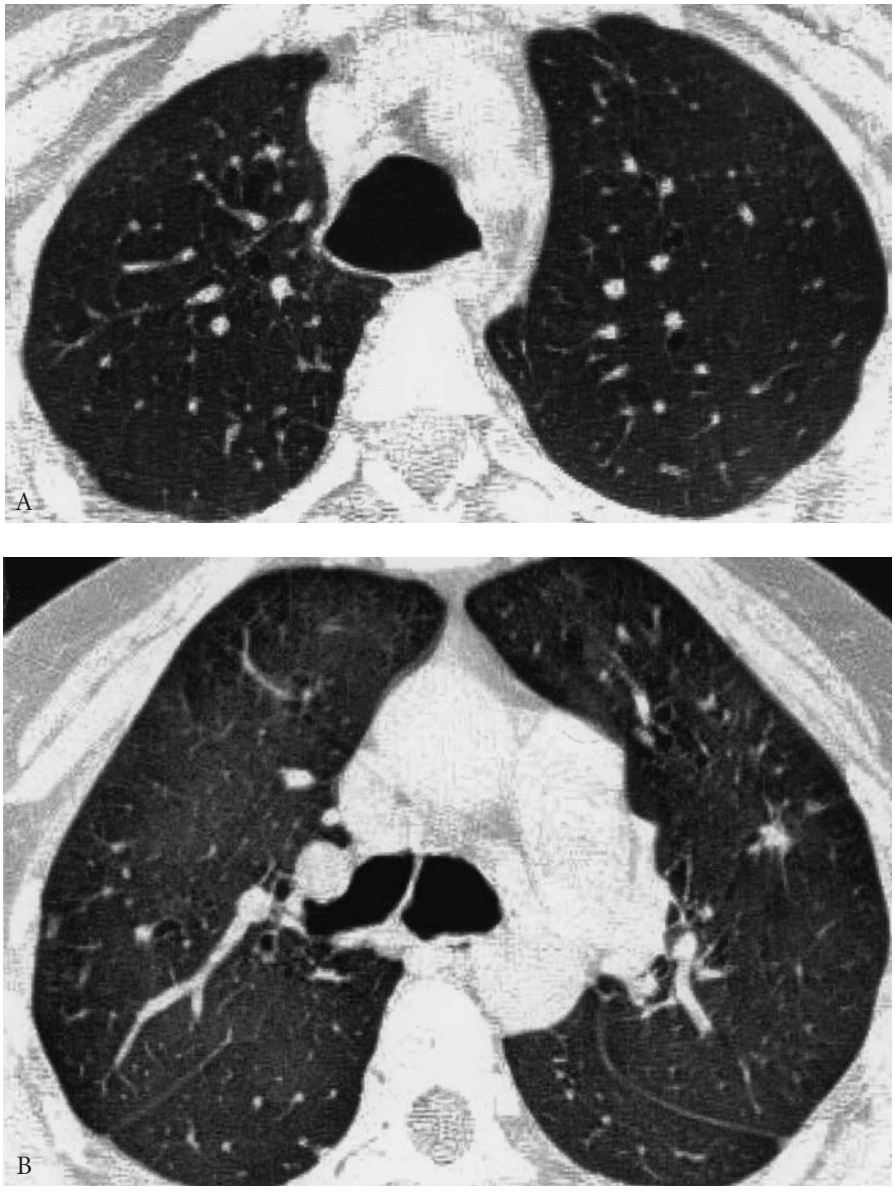


FIGURE **14-24** *Computed tomography scans showing tracheobronchomegaly in a 44-year-old male, with dyspnea for 10 years, much worse in the last year. There is massive dilation of the trachea (*A*) with elongated membranous wall and of main bronchi at the carina (*B*). More marked bronchiectatic changes were present at both lung bases.*

posterior splints to narrow the widened membranous wall. When these procedures failed, the patient was successfully managed over the long term with a large diameter silicone tracheal T tube. A T tube was used as primary treatment in subsequent patients, with successful results. A tracheal Y stent provides symptomatic relief if the bronchi also require support.[70] At the present time, placement of a silicone stent of large diameter appears to promise certain relief, and it is to be favored over extensive (and, therefore, more risky) surgical tracheobronchoplasty. The stent may have to be specifically made for the individual patient's tracheobronchial anatomy.

References

1. Smith LS, Schillaci RF, Sarlin RF. Endobronchial tuberculosis. Serial fiberoptic bronchoscopy and natural history. Chest 1987;91:644–7.

2. Macchiarini P, Delamare N, Beuzeboc P, et al. Tracheoesophageal fistula caused by mycobacterial tuberculosis adenopathy. Ann Thorac Surg 1993;55:1561–3.

3. Newton JR, Grillo HC, Mathisen DJ. Main bronchial sleeve resection with pulmonary conservation. Ann Thorac Surg 1991;52:1272–80.
4. Mitchell JD, Mathisen DJ, Donahue DM, et al. Clinical experience with carinal resection. J Thorac Cardiovasc Surg 1999;117:39–53.
5. Kato R, Kakizaki T, Hangai N, et al. Bronchoplastic procedures for tuberculous bronchial stenosis. J Thorac Cardiovasc Surg 1993;106:1118–21.
6. Natkunam R, Tse C, Ong BH, Sriragawan P. Carinal resection for stenotic tuberculous tracheitis. Thorax 1988;43:492–3.
7. Mathisen DJ, Grillo HC. Clinical manifestation of mediastinal fibrosis and histoplasmosis. Ann Thorac Surg 1992;54:1053–8.
8. Ferguson TB, Burford TH. Mediastinal granuloma. A 15-year experience. Ann Thorac Surg 1965;1:125–41.
9. Trastek VF, Pairolero PC, Ceithaml EL, et al. Surgical management of broncholithiasis. J Thorac Cardiovasc Surg 1985;90:842–8.
10. Dines DR, Bernatz PE, Pairolero PC. Mediastinal granuloma and fibrosing mediastinitis. Chest 1979;75:320–4.
11. Loyd JE, Tillman BF, Atkinson JB, DesPrez RM. Mediastinal fibrosis complicating histoplasmosis. Medicine 1988;67:295–310.
12. Urschel HC Jr, Razzuk MA, Netto GJ, et al. Sclerosing mediastinitis: improved management with histoplasmosis titer and ketoconazole. Ann Thorac Surg 1990;50:215–21.
13. James EC, Harris SS, Dillenburg CJ. Tracheal stenosis: an unusual presenting complication of idiopathic fibrosing mediastinitis. J Thorac Cardiovasc Surg 1980;80:410–3.
14. Garrett HE, Roper CL. Surgical intervention in histoplasmosis. Ann Thorac Surg 1986;42:711–22.
15. Brown A, Geyer S, Arbitman M, et al. Pulmonary nocardiosis presenting as a bronchogenic tumor. South Med J 1980;73:660–3.
16. Tedder M, Spratt JA, Anstadt MP, et al. Pulmonary mucormycosis: results of medical and surgical therapy. Ann Thorac Surg 1994;57:1044–50.
17. Brown RB, Johnson JH, Kessinger JM, Sealy WC. Bronchovascular mucormycosis in the diabetic: an urgent surgical problem. Ann Thorac Surg 1992;53:854–5.
18. Donahue DM, Wain JC. Mucormycosis of the respiratory tract: a critical assessment. Am Rev Resp Dis 1991;143:A495.
19. Andrews DR, Allan A, Larbalestier RI. Tracheal mucormycosis. Ann Thorac Surg 1997;63:230–2.
20. Acuna RT. Endoscopy of the air passages with special reference to scleroma. Ann Otol Rhinol Laryngol 1973;82:765–9.
21. Miller RH, Shulman JB, Canalis RF, Ward PH. *Klebsiella rhinoscleromatis:* a clinical and pathogenic enigma. Otol Head Neck Surg 1979;87:212–21.
22. Judson MA, Sahn SA. Endobronchial lesions in HIV-infected individuals. Chest 1994;105:1314–23.
23. Grillo HC, Mark EJ, Mathisen DJ, Wain, JC. Idiopathic laryngotracheal stenosis and its management. Ann Thorac Surg 1993;56:80–7.
24. Ashiku SK, Kuzucu A, Grillo HC, et al. Idiopathic laryngotracheal stenosis: effective definitive treatment by laryngotracheal resection. J Thorac Cardiovasc Surg 2003. [In press]
25. Merkel PA, Polisson RP, Chang YC, et al. Prevalence of antineutrophil cytoplasmic antibodies in a large inception cohort of patients with connective tissue disease. Ann Intern Med 1997;126:866–73.
26. Koufman JA. The otolaryngologic manifestations of gastroesophageal reflux disease (GERD). Laryngoscope 1991;101 Suppl 53:1–78.
27. Maronian NC, Azadeh H, Waugh P, Hillel A. Association of laryngopharyngeal reflux disease and subglottic stenosis. Ann Otol Rhinol Laryngol 2001; 110:606–12.
28. Dedo HH, Catten MD. Idiopathic progressive subglottic stenosis: findings and treatment in 52 patients. Ann Otol Rhinol Laryngol 2001;110:305–11.
29. Trentham DE, Le CH. Relapsing polychondritis. Ann Intern Med 1998;129:114–22.
30. Gilliland BC. Relapsing polychondritis and other disorders. In: Fauci AS, Braunwald E, Isselbacher KJ, et al, editors. Harrison's principles of internal medicine. New York: McGraw-Hill; 1998. p. 1951–3.
31. McAdam LP, O'Hanlan MA, Bluestone R, Pearson CM. Relapsing polychondritis: prospective study of 23 patients and a review of the literature. Medicine (Baltimore) 1976;55:193–215.
32. Eng J, Sahanathan S. Airway complications in relapsing polychondritis. Ann Thorac Surg 1991;51:686–92.
33. Newman LS, Rose CS, Maier LA. Sarcoidosis. N Engl J Med 1997;336:1224–34.
34. Brandstetter RD, Messina MS, Sprince NL, Grillo HC. Tracheal stenosis due to sarcoidosis. Chest 1981;80:656.
35. Stjernberg N, Thunell M. Pulmonary function in patients with endobronchial sarcoidosis. Acta Med Scand 1984;215:121–6.
36. Miller A, Brown LK, Teirstein AS. Stenosis of main bronchi mimicking fixed upper airway obstruction in sarcoidosis. Chest 1985;88:244–8.
37. Paller AS, Surek C, Silva-Walsh I, et al. Cutaneous sarcoidosis associated with sarcoidosis of the upper airway. Arch Dermatol 1983;119:592–6.
38. Fouty BW, Pomerantz M, Thigpen TP, Martin RJ. Dilatation of bronchial stenoses due to sarcoidosis using a flexible fibreoptic bronchoscope. Chest 1994;106:677–80.
39. Hoffman GS, Kerr GS, Leavitt RY, et al. Wegener's granulomatosis: an analysis of 158 patients. Ann Intern Med 1992;116:488–98.
40. Sneller MC, Hoffman GS, Talar-Williams C, et al. An analysis of forty-two Wegener's granulomatosis patients treated with methotrexate and prednisone. Arthritis Rheum 1995;38:608–15.
41. Lebovics RS, Hoffman GS, Leavitt RY, et al. The management of subglottic stenosis in patients with Wegener's granulomatosis. Laryngoscope 1992;102:1341–5.
42. Daum TE, Specks U, Colby TV, et al. Tracheobronchial involvement in Wegener's granulomatosis. Am J Respir Crit Care Med 1995;151:522–6.
43. Del Buono EA, Flint A. Diagnostic usefulness of nasal biopsy in Wegener's granulomatosis. Hum Pathol 1991;22:107–10.
44. Herridge MS, Pearson FG, Downey GP. Subglottic stenosis complicating Wegener's granulomatosis: surgical repair as a viable treatment option. J Thorac Cardiovasc Surg 1996;111:961–6.
45. Langford CA, Sneller MC, Hallahan CW, et al. Clinical features and therapeutic management of subglottic stenosis inpatients with Wegener's granulomatosis. Arthritis Rheum 1996;39:1754–60.
46. Falk RH, Comenzo RL, Skinner M. The systemic amyloidoses. N Engl J Med 1997;337:898–909.
47. Toyoda M, Ebihara Y, Kato H, Kita S. Tracheobronchial AL amyloidosis: histologic, immuno-histochemical, ultrastructural and immunoelectron microscopic observations. Hum Pathol 1993;24:970–6.
48. Mark EJ, LoCicero J, Wright CD, et al. Case records of the Massachusetts General Hospital – 1995 (tracheobronchial amyloidoma). N Engl J Med 1995; 332:110–5.

49. Utz JP, Swensen SJ, Gertz MA. Pulmonary amyloidosis. The Mayo Clinic experience from 1980 to 1993. Ann Intern Med 1996;124:407–13.
50. Naef AP, Savary M, Schmid de Grûneck JM, Baumann RP. Amyloid pseudotumor treated by tracheal resection. Ann Thorac Surg 1977;23:578–81.
51. Hui AN, Koss MN, Hochholzer L, Wehunt WD. Amyloidosis presenting in the lower respiratory tract: clinicopathologic, radiologic, immunohistochemical and histochemical studies on 48 cases. Arch Pathol Lab Med 1986;110:212–8.
52. Keen PE, Weitzner S. Primary localized amyloidosis of trachea. Arch Otolaryngol 1972;96:142–5.
53. Greene RE, Lechner GL. "Saber-sheath" trachea. A clinical and functional study of marked coronal narrowing of the intrathoracic trachea. Radiology 1975;115:265–8.
54. Greene RE. "Saber-sheath" trachea: relation to chronic obstructive pulmonary disease. Am J Roentgenol 1978;130:441–5.
55. Young RH, Sandstrom RE, Mark EJ. Tracheopathia osteoplastica: clinical, radiologic and pathological correlations. J Thorac Cardiovasc Surg 1980;79:537–41.
56. Nienhuis DMJ, Prakash UBS, Edell ES. Tracheobronchopathia osteochondroplastica. Ann Otol Rhinol Laryngol 1990;99:689–94.
57. Härmä RA, Suurkari S. Tracheopathia chondro-osteoplastica: a clinical study of thirty cases. Acta Otolaryngol 1977;84:118–23.
58. Besso J-C, Eschapasse H, Bullinelli M, et al. Trachéopathie ostéoplastique à forme sténosante et asphyxiante opérée. J Franc Med Chir Thor 1972;26:291–304.
59. O'Reilly RR, Marty AT. Tracheopathia osteoplastica: case report. Mil Med 1978;143:497–8.
60. Mark EJ, Patterson GA, Grillo HC. Case records of Massachusetts General Hospital 46 – 1992 (tracheopathia osteoplastica). N Engl J Med 1992;327:1512–8.
61. Braman SS, Mark EJ, Grillo HC. Case records of Massachusetts General Hospital 32 – 1999 (tracheopathia osteoplastica). N Engl J Med1999;341:1292–9.
62. Kutlu CA, Yeginsu A, Ozalp T, Baran R. Modified slide tracheoplasty for the management of tracheobronchopathia osteochrondroplastica. Eur J Cardiothorac Surg 2002;21:140–2.
63. Mounier-Kuhn P. Dilatation de la trachée, constatations radiographiques et bronchoscopiques. Lyon Méd 1932;150:106–9.
64. Katz I, LeVine M, Herman P. Tracheobronchomegaly. The Mounier-Kuhn syndrome. Am J Roentgenol Rad Ther Nucl Med 1962;88:1084–94.
65. Johnston RF, Green RA. Tracheobronchomegaly. Report of five cases and demonstration of familial occurrence. Am Rev Resp Dis 1965;91:35–50.
66. Engle WA, Cohen MD, McAlister WH, Griscom NT. Neonatal tracheobronchomegaly. Am J Perinatol 1987;4:81–5.
67. Woodring JH, Howard RS II, Rehm SR. Congenital tracheobronchomegaly (Mounier-Kuhn syndrome): a report of 10 cases and review of the literature. J Thorac Imaging 1991;6:1–10.
68. Shin MS, Jackson RM, Ho KJ. Tracheobronchomegaly (Mounier-Kuhn syndrome): CT diagnosis. Am J Roentgenol 1988;150:777–9.
69. Himalstein MR, Gallagher JC. Tracheobronchomegaly. Ann Otol Rhinol Laryngol 1973;82:223–7.
70. Collard P, Freitag L, Reynaert MS, et al. Respiratory failure due to tracheobronchomalacia. Thorax

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Tracheobronchial Malacia and Compression

Hermes C. Grillo, MD

Tracheomalacia and Bronchomalacia

Tracheomalacia describes softening of the tracheal cartilages, with consequent obstruction. The subject is itself "soft" and not a little confusing. Tracheomalacia is a consequence of multiple processes, some well defined clinically, but of poorly understood etiology. The lesions are rare and there are differences of opinion about their nature. For the most part, management ranges from difficult to experimental or currently impossible. The degree of loss of normal semirigidity of cartilages may be partial or complete, and may involve the entire trachea or a segment of it.

Congenital tracheomalacia is presented in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children" (see Figure 6-11). Not truly congenital, but of genetic origin, is tracheomalacia seen in patients with dwarfism due to *achondroplasia*. Three such patients had softened tracheal cartilages with easy collapse. In addition, the trachea had failed to grow in length or diameter as they became adults, so that the lumen was infantile and easily occluded. Each had already undergone a number of surgical procedures in childhood, including high tracheostomy with injury to the cricoid and to the subglottic larynx, tracheostomy with stenosis and/or granulomas, and failed tracheal resections. A prognathous jaw and limitation of cervical mobility in these patients can make rigid bronchoscopy (eg, for granuloma removal) impossible, unless there is an existing tracheostomy. Intubation, if necessary, can be done with some difficulty over a flexible bronchoscope. Because of the small diameter of the airway, a laryngeal mask airway (through which a flexible bronchoscope can be introduced) seems preferable. Each patient will present unique problems. If surgical reconstruction seems inadvisable in a given situation, recourse to a tracheostomy or T tube may be necessary. The latter may not be successful for the long term if the airway, and consequently the T tube, is too narrow (see Chapter 39, "Tracheal T Tubes").

Acquired segmental tracheomalacia ("focal" tracheomalacia) resulting from *postintubation injury* has been considered in Chapter 11, "Postintubation Stenosis" (Figure 15-1). Secondary malacia resulting from *extrinsic compression* by lesions such as goiter, tumors, and vascular structures is described subsequently in

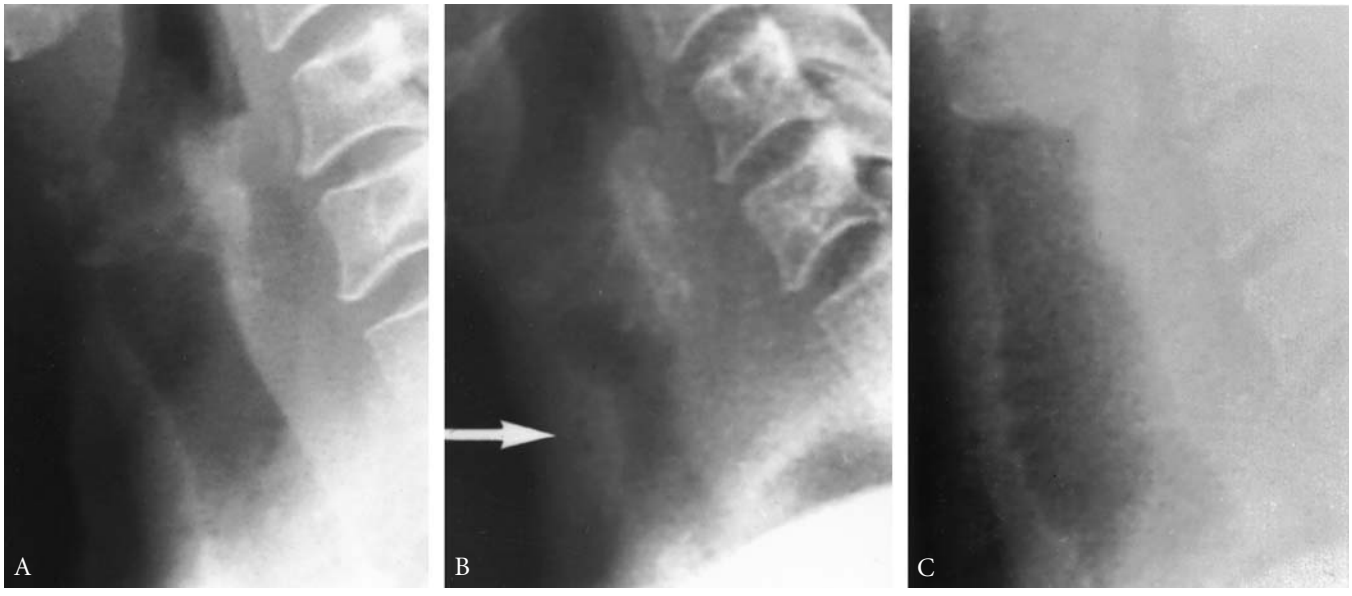


FIGURE **15-1** *Localized tracheomalacia in a 16-year-old male, resulting from prolonged ventilation for several months, via a tracheostomy, subsequent to severe head injury, coma, and multiple complications. In addition to a distal 2.5 cm long circumferential stenosis due to cuff injury 2 cm below a stoma, he had a 2 to 3 cm segment of malacia of the anterior tracheal wall above the stoma from prolonged pressure by the tracheostomy tube during ventilation.* A, *Lateral neck roentgenogram shows an apparently normal upper tracheal air column with the patient at rest. Stenosis is distal and not visualized in this roentgenogram. Calcification in larynx is visible superiorly.* B, *Forced inspiration shows collapse of the soft anterior tracheal wall* (arrow). C, *On expiration, the anterior tracheal wall is distended. After circumferential resection of the distal stenosis, obstruction was apparent due to the malacic trachea. Through a separate small incision, a perforated polypropylene ring was inserted to encircle and support the soft segment. Excision of both lesions in continuity would have been too long to allow a safe anastomosis. Two separate excisions would have endangered the blood supply of the short intervening segment of the trachea. Two-stage resection would also have been possible. This patient's airway remains excellent 32 years later.*

this chapter. Obstruction resulting from *relapsing polychondritis* was discussed in Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions." Two additional categories of intrinsic malacia are to be considered. The first is partial malacia, usually associated with chronic obstructive pulmonary disease (COPD), which can lead to severe expiratory collapse of the lower trachea and main bronchi. The second is the very rare occurrence of idiopathic complete malacia of tracheal cartilages.

Tracheal and bronchial malacia, most often associated with COPD, seems to occur infrequently. It varies in severity and may well be underdiagnosed, since COPD may seem to be sufficient cause for respiratory distress. It is also difficult to distinguish symptomatically from chronic asthmatic bronchitis. Patients complain of dyspnea, particularly in the expiratory phase, and this is especially manifest on effort. Respiratory crises that border on asphyxia may follow bouts of severe coughing. Seizures may occur, which in the past were termed "laryngeal epilepsy." Expiratory stridor is heard; cough may be intractable. The harder the patient works to breathe, the more difficult it becomes. Secretions are raised with difficulty, often a leading complaint, because the cough is weak. The cough is characterized by a "seal bark" quality. Recurrent respiratory infections ensue.

A history of pulmonary disease usually precedes the finding of tracheal disease. These changes are well identified by flexible bronchoscopy in a conscious patient, where dynamic changes with respiration and cough can be elicited and observed. This type of severe expiratory tracheobronchial collapse occurs most often in males over the age of 40 years. Diminution in elastic fibers of the membranous wall has been found in these patients, but may well be a secondary change.[1] The factors that lead to this deformity and its association with COPD remain unknown.

Bronchoscopic examination reveals a flattening of cartilages from a normal "C" configuration (Figure 15-2). The ends of the cartilages may curve slightly forward, so that the cartilages assume the configuration of an archer's bow. The membranous wall is elongated. On expiration, especially with forced respiration and cough, the cartilage flattens out even more and the membranous wall approximates toward the anterior wall. Complete or nearly complete tracheal occlusion results. The lower two-thirds of the trachea (the intrathoracic portion) and both main bronchi are most often involved. Sometimes, the malacia extends to more distal bronchi.

Fluoroscopy demonstrates anteroposterior tracheal occlusion, and inspiratory–expiratory computed tomography (CT) scan confirms this dynamic obstruction (Figure 15-3). Cross-sectional area normally reduces by 13 to 14% in the upper- and midtrachea on forced expiration. The degree of dynamic collapse in acquired tracheomalacia was measured by semiautomated CT cross-sectional area calculations by Aquino and colleagues.[2] For patients with tracheomalacia, the mean change in the upper trachea was 47% and was 54% for midtrachea. A greater than 30% change was 94% sensitive in detecting malacia. Most patients have advanced COPD with emphysematous change visible on images.

Herzog and colleagues studied in detail the pathophysiology of expiratory obstruction in chronic lung disease, in patients who demonstrated "dyskinesia" of the trachea and main bronchi.[3] They theorized that major intrathoracic airways were no longer held open in the expiratory phase of respiration due to loss of tissue elasticity. Expiratory airflow is consequently markedly reduced. Because of the collapse, a marked pressure gradient results between intra-airway pressure in the subglottic region and that at the carinal and main bronchial levels. Rate of progression of malacia is variable, but spontaneous improvement does not happen.

In an effort to improve this mechanical deficit, Herzog and Nissen narrowed and splinted the membranous wall of the lower trachea and the main bronchi, using various materials.[4] The ends of the cartilages were pulled to the edges of longitudinal splints fixed to the membranous wall, thus restoring a more normal horseshoe shape to the softened but still functional cartilage. Criteria for possible operative intervention, in addition to the clinical signs noted and bronchoscopic and imaging data, which were recommended by Herzog and colleagues, are 1) a forced expiratory volume in 1 second (FEV_1) of less than 40% of vital capacity indicating severe obstruction, and 2) an increase in central airway resistance over 20 cm H_2O per liter per second at an alveolar pressure of 60 cm H_2O (the pressure developed by moderately strong cough).[3] Herzog and colleagues further stated that "intractable cough due to (bronchoscopically observed) circumscribed contact of the membrane with the anterior wall of the trachea or main bronchi is an absolute indication for local stabilization of the membranous wall."[3] Inability to raise secretions because of collapse is a relative indication. Herzog and Rossetti reported success in treating 17 patients.[5] Respiratory function improved, as well as ease of expectoration, dyspneic crises were ameliorated, and patients were able to increase their physical activities. Although the basic pulmonary disease was not improved, some patients seemed to obtain significant palliation. Other reports are less encouraging.[6] Little systematic data has been reported. The long-term effectiveness of such procedures remains unclear. Complicating these issues are the progression of underlying COPD, presence of distal bronchial collapse, and also the variety of techniques of splinting employed.

Different materials have been used for the posterior splints: fascia lata, pericardium, perforated rigid plastic strips, lyophilized bone, and Goretex. Circumferential wrapping of the trachea and both main bronchi with heavy Marlex, using tissue adhesive, was reported to be successful, but raises concern about tracheal blood supply if more generally employed.[7] I was initially impressed with the ease of use and apparent effectiveness of Goretex strips for obtaining correction. However, since Goretex does not become incorporated by the tissues, late failure followed, due to partial separation of the Goretex from the membranous wall of the trachea. Sterile fluid collected between the Goretex and the membranous wall, producing obstruction. I subsequently used pericardium, but this appeared to remodel

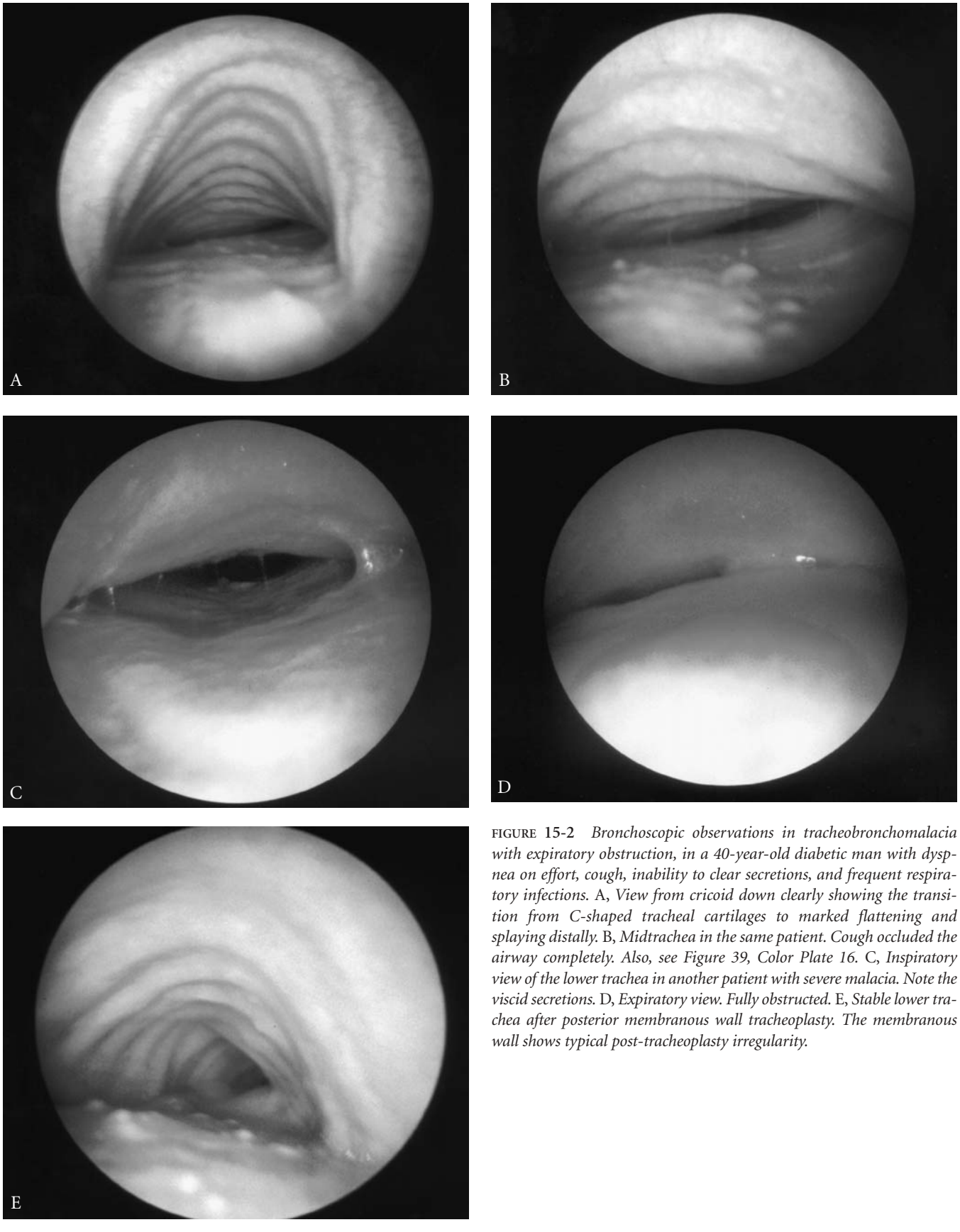


FIGURE **15-2** *Bronchoscopic observations in tracheobronchomalacia with expiratory obstruction, in a 40-year-old diabetic man with dyspnea on effort, cough, inability to clear secretions, and frequent respiratory infections.* A, *View from cricoid down clearly showing the transition from C-shaped tracheal cartilages to marked flattening and splaying distally.* B, *Midtrachea in the same patient. Cough occluded the airway completely. Also, see Figure 39, Color Plate 16.* C, *Inspiratory view of the lower trachea in another patient with severe malacia. Note the viscid secretions.* D, *Expiratory view. Fully obstructed.* E, *Stable lower trachea after posterior membranous wall tracheoplasty. The membranous wall shows typical post-tracheoplasty irregularity.*

with time, attenuate, and lose effectiveness. Marlex mesh has proved very satisfactory, since it causes sufficient inflammatory reaction, and is of porous structure which ensures permanent incorporation by fibrous tissue ingrowth (Figure 15-4) (see Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea").[8] Fifteen patients were treated by membranous wall tracheoplasty for severe symptoms of expiratory airway collapse, including dyspnea (15), inability to clear secretions (5), and intractable cough (10), using polypropylene mesh (Marlex). Thirteen had symptomatic improvement and most had marked bronchoscopic improvement. Pulmonary function tests improved in some (Table 15-1), as did flow volume curves (Figure 15-5). One failure needed a stent. Three required late reoperation, one due to fluid collection beneath a prior Goretex splint, as noted. All improved with reoperation.[8]

Idiopathic tracheomalacia has been seen in a very small number of patients. Cartilages throughout the trachea, and sometimes in the main bronchi, become softened. Generalized floppiness is visualized bronchoscopically, rather than the specific deformation just described. The extent of disease usually obviates resection. The few patients I have seen were adult males with no known disease, except for diabetes in 2. No history of intubation or other tracheal insult explained the lesion. Patients presented with expiratory obstruction and a rubbery, resonating, "seal bark" cough. A few patients were treated by splinting the trachea externally with polypropylene rings placed in individual channels, which were then imbedded in the tracheal wall by turning down the sternohyoid muscles over them and suturing these to the trachea in the intervals between the plastic rings (see Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea"). An alternative method of management is a long, large bore T or T-Y tube, as the anatomical defect requires, or an inlying stent, preferably of silicone.

Extrinsic Compressive Lesions

Parenchymal, cystic, and vascular lesions may obstruct the trachea by compression. Chronic compression may result in malacic changes in the deformed cartilages. The problem may, therefore, be dual. The lesion initially narrows the trachea by compression. If severe malacia results, the trachea may collapse following removal of the support which was provided by the mass lesion.

Goiter

A thyroid goiter may displace and deform the cervical or intrathoracic trachea markedly, over a short or long segment (Figure 15-6). The narrowing may be symmetrical, but more frequently is eccentric. In large goiters that have a major substernal or intrathoracic component, tracheal deformity and obstruction is more likely to be caused by those goiters that pass into the thorax laterally and posteriorly ("posterior descending goiter"), rather than by strictly anterior substernal goiters. The latter more often lie anterior to the great vessels and hence are less likely to compress the trachea seriously. Particularly in aged patients, where only a small portion of the trachea lies above the sternal notch, a goiter may be almost entirely intrathoracic and not be visible or palpable in the neck. In a series of 80 patients with intrathoracic goiters, reviewed by Katlic and colleagues, dysphagia was present in 33%, dyspnea in 28%, stridor in 16%, hoarseness in 13%, wheeze in 9%, and cough in 8%.[9] A cervical mass was the most common finding (69%). In one-third of the patients, the trachea was markedly deviated. Most lesions are multinodular goiters or follicular adenomas, with a rare incidence of Hashimoto's thyroiditis. Occult carcinoma is encountered in 2 to 3% of substernal goiters, at most.[10]

The treatment of obstructive goiter, whether cervical or intrathoracic, is surgical. There is little evidence that prolonged thyroid suppression (used in 59% of our series), radioiodine, or propylthiouracil effect regression of goiters and relief of compression. Since goiters may recur over a period of many years, complete removal of the affected lobe or lobes is advisable.

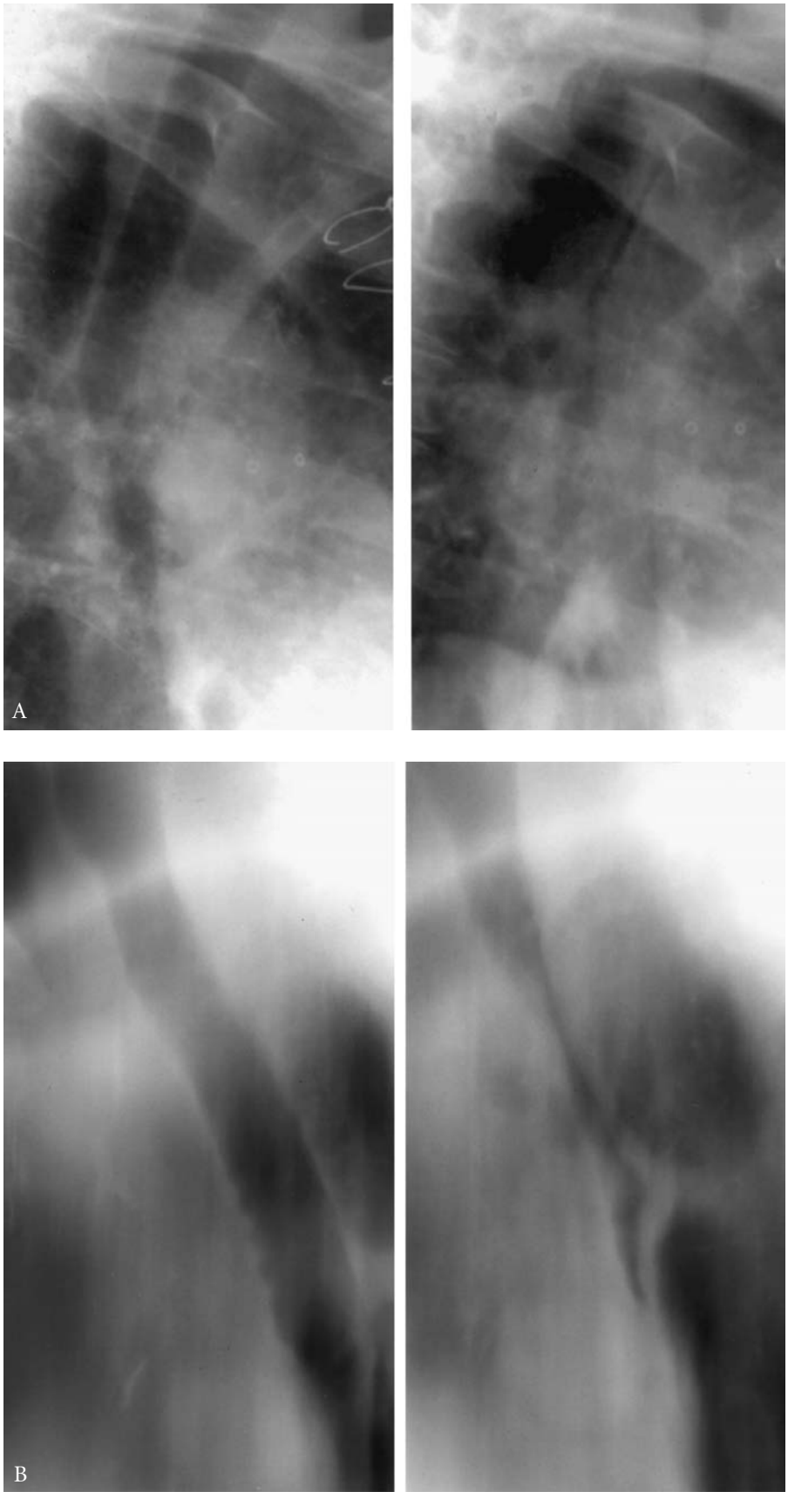


FIGURE **15-3** *Imaging in tracheomalacia.* A, *Fluoroscopy in a 57-year-old man with severely symptomatic malacia of the intrathoracic trachea and main bronchi. Oblique spot films of the trachea, inspiratory on* left *and expiratory on* right. *Note that the collapse is intrathoracic.* B, *Oblique tomograms, inspiratory on* left *and expiratory on* right, *in same patient of* A.

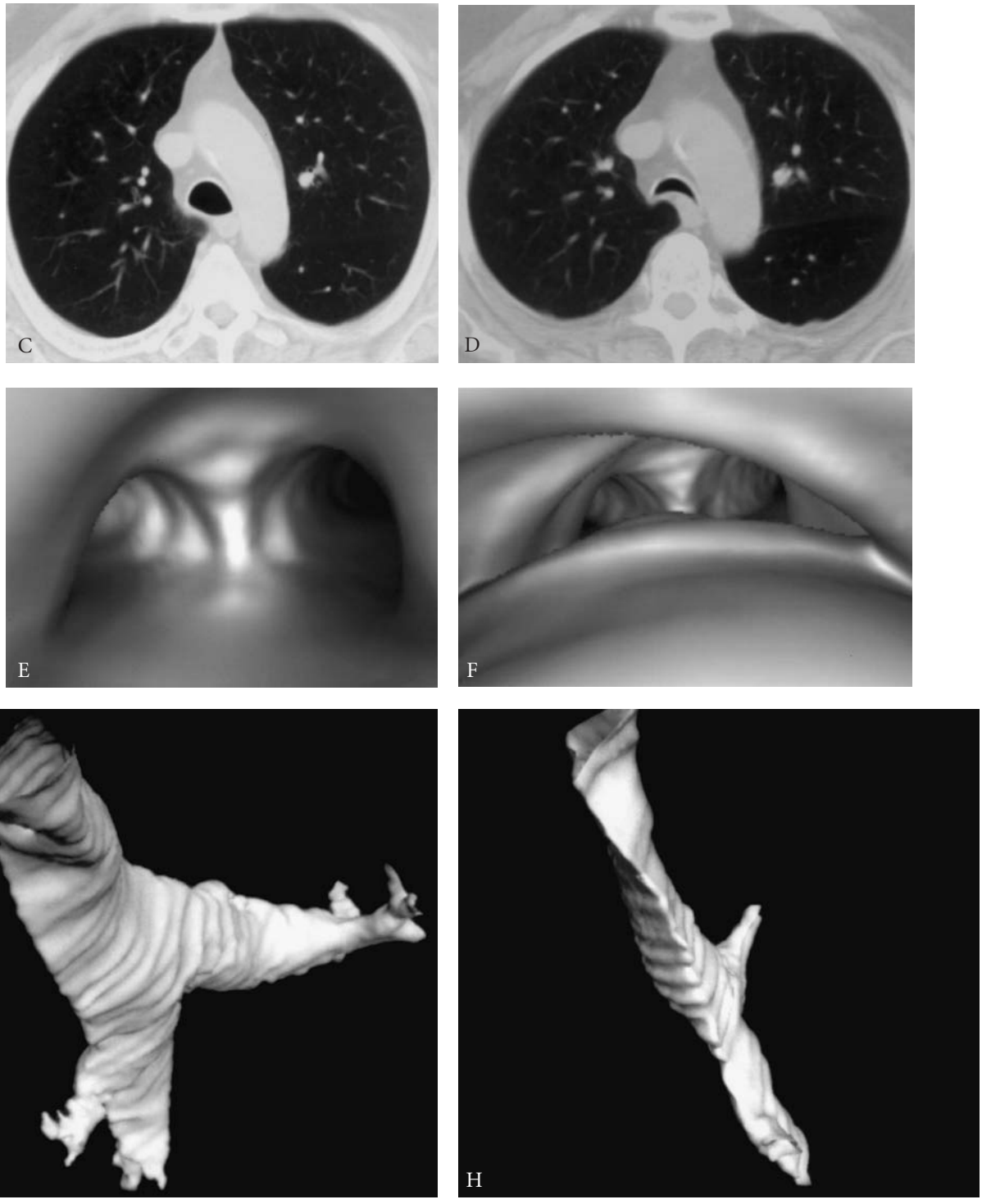
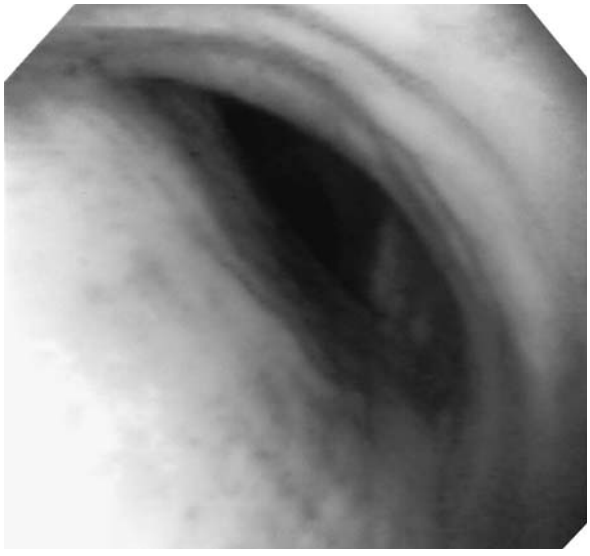


FIGURE **15-3** (CONTINUED) C, *Inspiratory–expiratory computed tomography (CT) scans demonstrate the degree of narrowing of the affected intrathoracic trachea. Inspiratory view.* D, *Expiration at the same level.* E, *Virtual bronchoscopy from a CT scan, showing the supracarinal trachea in inspiration in the same patient.* F, *View at the same level in expiration.* G, *Three-dimensional CT reconstructions of the trachea and main bronchi in another patient, a 57-year-old man with severely symptomatic obstructive malacia. Anterior view. Note the transition from a circular cervical configuration through a triangular shape to flattened cartilages distally.* H, *Oblique view showing ridging of the membranous wall. Corresponding bronchoscopic views are seen in Figures 15-4*C *and 15-4*D.



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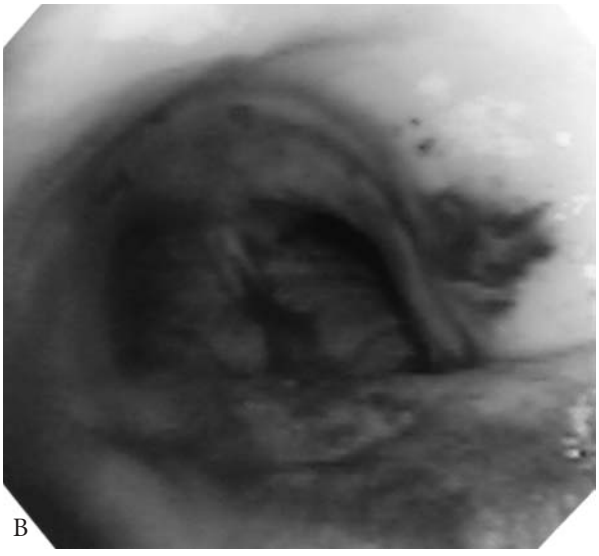

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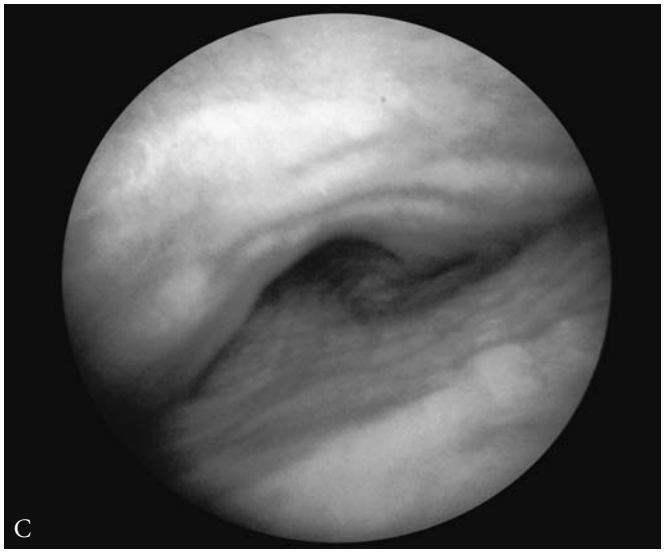

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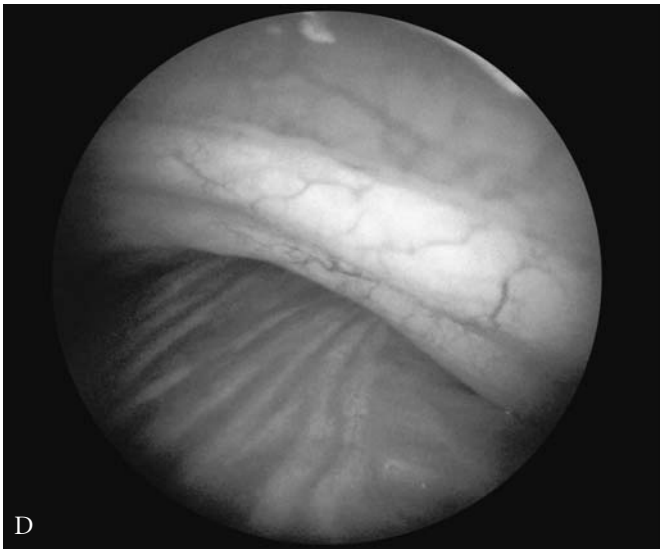

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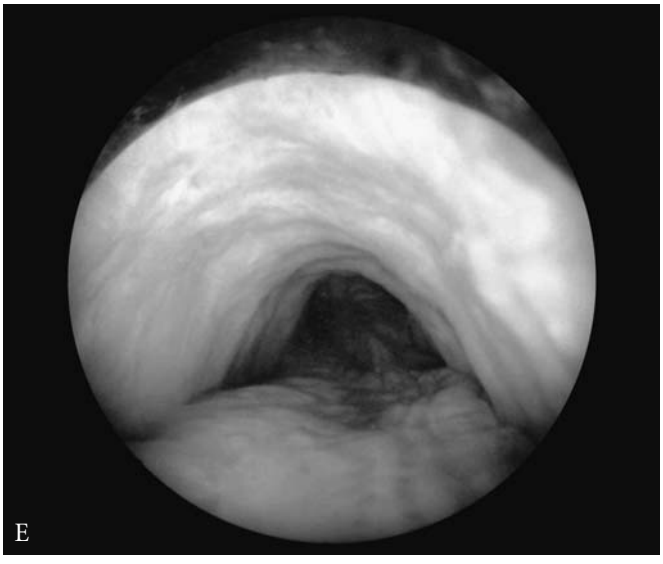


E

FIGURE **15-4** *Pre- and postoperative bronchoscopic observations.* A, *In a 79-year-old male with chronic obstructive pulmonary disease, bronchiectasis, and worsening dyspnea. Changes are evident in the lower trachea with characteristic splaying of malacic cartilages.* B, *Post-operative view at the same level demonstrates restoration of a D-shaped trachea. The carina is clearly visible. The membranous wall is puckered by posterior scar and sutures.* C, *Inspiratory view in a 57-year-old male, just above the carina. The right main bronchus remains nearly occluded.* D, *Expiration occludes the trachea and bronchi.* E, *Following posterior splinting procedure, a stable and open airway results. An open carina is below. The posterior wall is fixed to the Marlex backing.*

Table 15-1 Functional Results* of Membranous Wall Tracheoplasty for Malacia

	Preoperative		*Postoperative (2 months)*	
	Observed	*% Predicted*	*Observed*	*% Predicted*
FEV_1 (L)	1.39	39	2.96	72
FVC (L)	2.94	59	4.59	87
PEF (L/sec)	4.73	51	5.87	63
PIF (L/sec)	2.17	41	5.35	82
MBC (L/min)	63	47	104	72
FEV_1/FVC (%)	47	66	64	82

*In a 57-year-old male, presenting with 7 years of wheezing, dyspnea, cough, and respiratory infections.

FEV_1 = forced expiratory volume in 1 sec; FVC = forced vital capacity; MBC = maximum breathing capacity; PEF = peak expiratory flow; PIF = peak inspiratory flow.

(See flow volume loops in Figure 15-5.)

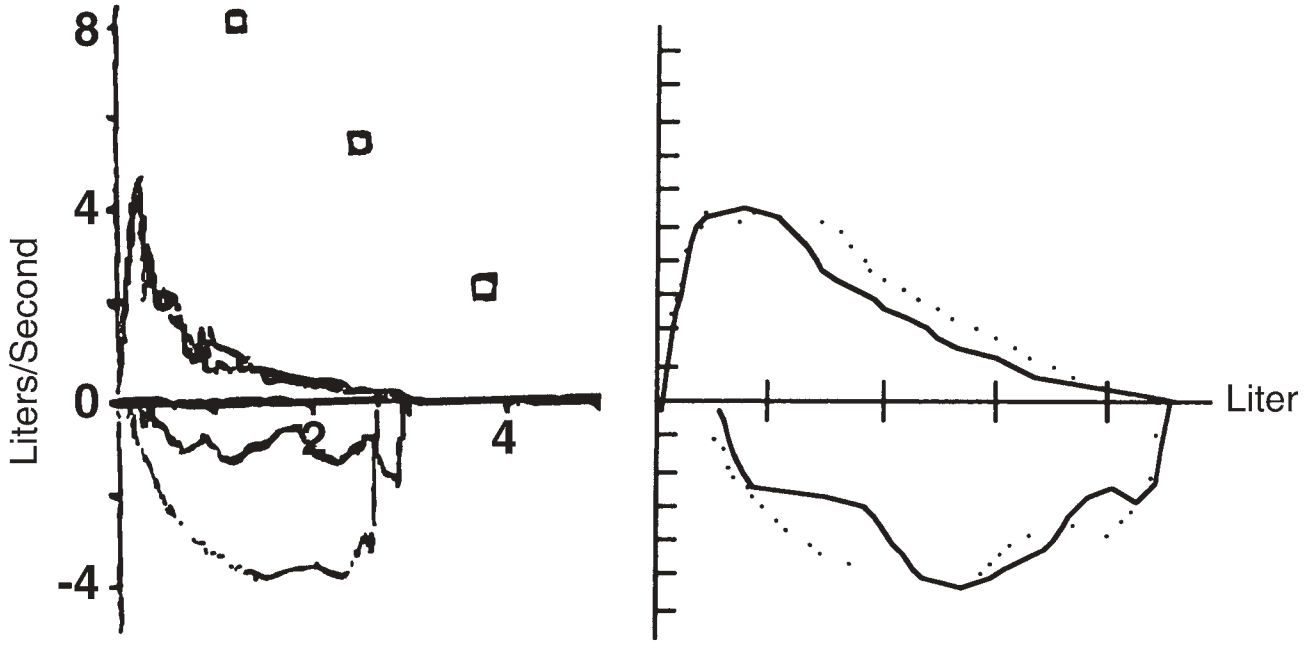


FIGURE **15-5** *Flow volume loops in a 57-year-old man with a 7-year history of progressive wheezing, dyspnea, cough, and respiratory infections. Preoperative on* left*; 2 months postoperative on* right*. Values for pulmonary function tests are in Table 15-1. The patient's bronchoscopic findings are displayed in Figures 15-4C,D,E.*

The goiter may be so large and of such long duration that unilateral vocal cord paralysis has resulted from pressure defunctioning of a recurrent laryngeal nerve. Nerve paralysis or malfunction may also be the result of prior thyroid surgery, which many of these patients have undergone. Thyroid function should always be measured, since a few patients may indeed be hyperthyroid. Isotopic thyroid scans are not of particular value since the goiters may well be nonfunctional. Pulmonary function tests can provide information about fixed upper airway obstruction and pulmonary parenchymal disease. However, even patients with very limited pulmonary function and other severe medical problems can usually safely undergo the requisite operation, with dramatic functional improvement. Cervical and thoracic CT scans demonstrate the distribution and extent of the intrathoracic goiter and are always advisable (Figure 15-7).

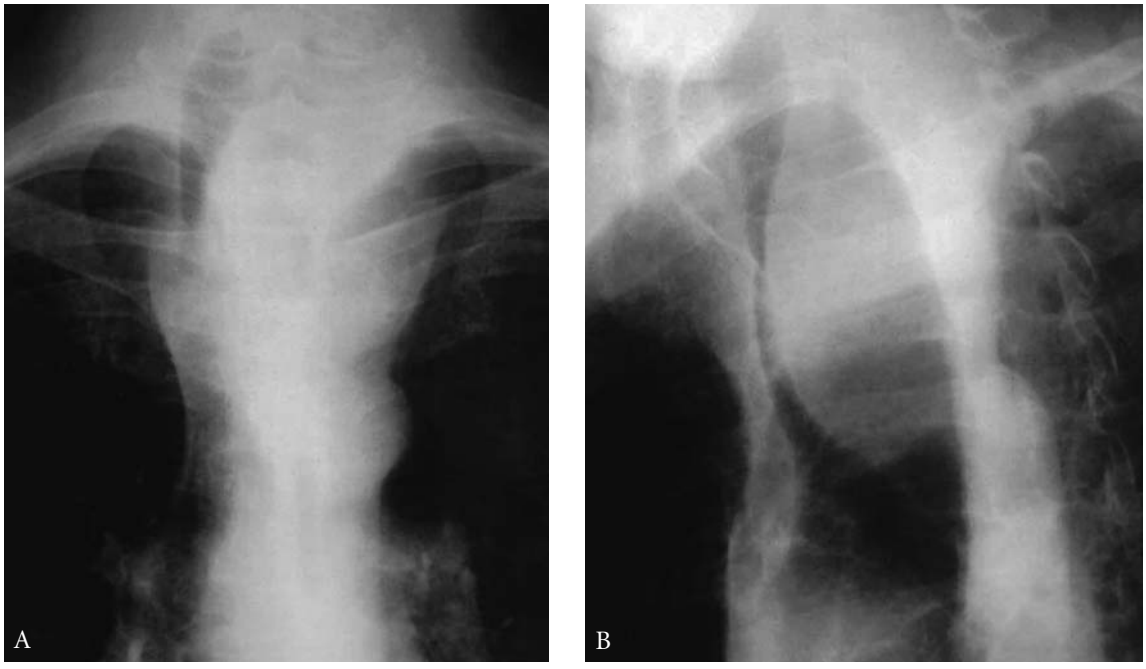


FIGURE **15-6** *Intrathoracic (substernal) goiter in a 72-year-old man with a history of dyspnea, dysphagia, and stridor for several years. Chest roentgenograms.* A, *Posteroanterior view showing marked displacement of the larynx and trachea to the right, with marked tracheal compression.* B, *Oblique view emphasizes severe tracheal deviation and compression, which reduced the airway cross section by over 80%. Complete relief followed removal of the bilateral adenomatous goiter by right total and left subtotal thyroidectomy via a collar incision and upper partial sternotomy.*

No matter how tortuous the airway appears on imaging studies, it has always been possible to pass a rigid bronchoscope through the trachea, straightening the airway out as the bronchoscope is advanced (Figure 15-8). An endotracheal tube can always be passed into the trachea, despite its tortuosity, and if need be, over a flexible bronchoscope or over a tube changer placed via a rigid bronchoscope. The difficulty that may be encountered is that of initiating bronchoscopy or laryngoscopy because of marked displacement and torsion of the larynx by the goiter. If difficulty in intubation is anticipated because of the size of the goiter, it is wise to have a rigid bronchoscope available. This can always be passed, even where it is difficult to identify a markedly distorted and deviated larynx with a flexible instrument. In an extremely difficult case, a laryngoscope is useful to help introduce the rigid bronchoscope into the glottis. If the trachea is markedly compressed, intubation is preferable to the compromise of laryngeal mask airway.

Operation is accomplished through a low collar incision, which may have to be longer than usual, depending upon the size of the goiter. In a number of patients, upper sternotomy becomes necessary in order to extract the intrathoracic component without undue bleeding. Only rarely, principally in patients who have had previous goiter surgery that extended intrathoracically, is a complete median sternotomy needed. In 2 unique cases of a very extensive goiter complicated by previous operations, further extension of the incision into the right hemithorax through the fourth interspace was also required. In huge goiters with posterior and inferior extensions, it is prudent to prepare and drape a wide operative field for possible incisional extension.

In most patients, removal of the goiter is all that is required to relieve tracheal and esophageal compression. On a rare occasion, cartilage thinning is so severe that the airway collapses and the patient becomes even more dyspneic following thyroidectomy. Usually, the trachea opens up when the mass is removed and the cartilages gradually return to a more normal shape and stabilize in the postoperative weeks. However, if

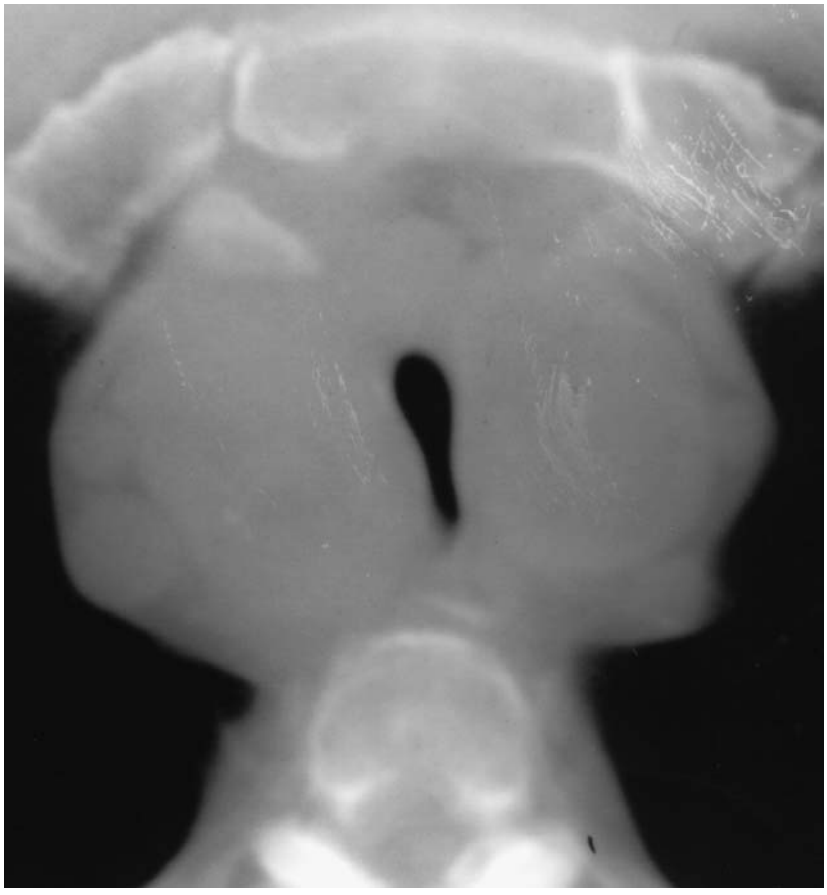


FIGURE **15-7** *Computed tomography scan reveals marked tracheal compression by a bilateral goiter.*

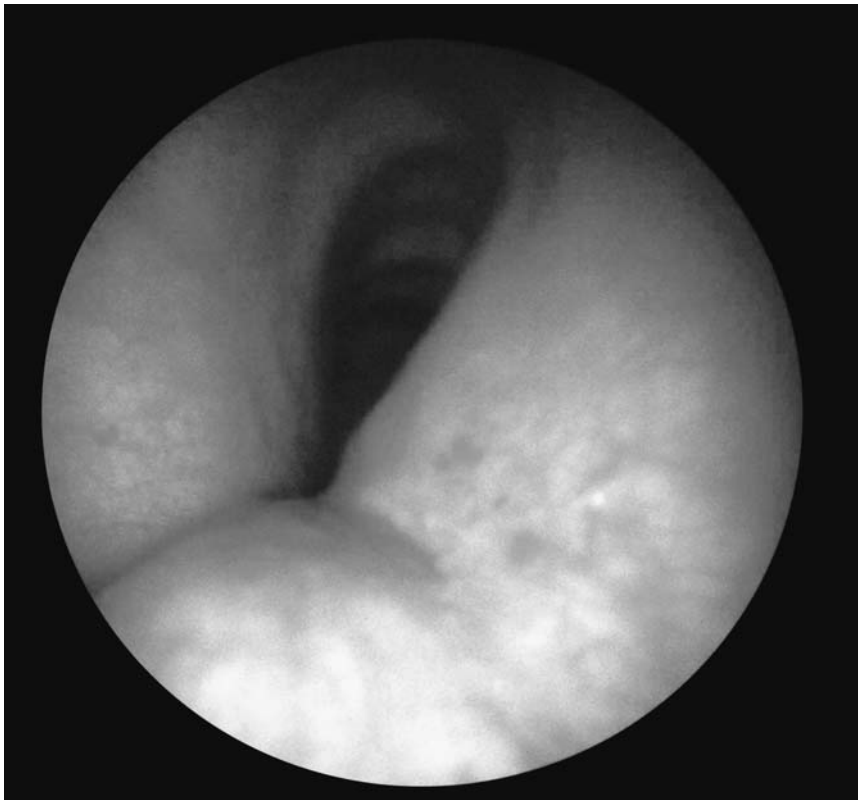


FIGURE **15-8** *Bronchoscopy showing marked bilateral tracheal compression by a goiter. Although the tracheal rings are difficult to see in the region of maximal deformation, enough substance remained to provide an airway of over 50%, immediately after thyroidectomy, without collapse on respiration. Also see Figure 41 (Color Plate 16).*

a now severely malacic trachea has lost the rigid splinting of the surrounding goiter, it can collapse more completely. The progression is from mass obstruction to that of malacic collapse. In order to recognize this possibility, the endotracheal tube is withdrawn intraoperatively, after thyroidectomy, to a point just below the glottis. The trachea is examined and palpated in the open operative field and the trachea is observed during respiration through a flexible bronchoscope passed through the endotracheal tube. It is helpful if the patient makes spontaneous respiratory efforts during examination. Usually, the airway appears adequate, although somewhat deformed, and no further reinforcing procedures are needed.

If this assessment proves incorrect and the patient becomes dyspneic following extubation at the close of operation, then the patient should promptly be relaryngoscoped and bronchoscoped. The possibility of recurrent laryngeal nerve damage causing glottic inadequacy must first be ruled out. The trachea is then examined carefully for evidence of malacic collapse and obstruction. For either problem, my preferred management is to pass an uncuffed endotracheal tube of smaller diameter to maintain a satisfactory postoperative airway. In a few days, after tissue planes are well coapted, the endotracheal tube may be removed over a flexible bronchoscope, preferably in the operating room, and the trachea and larynx reassessed. If obstruction persists, a tracheostomy may now be performed safely through the sealed tissue planes. It should be noted, however, that Lahey and Hoover did not observe infection in their patients after tracheostomy was done for tracheal collapse at the initial thyroid operation.[11] In the rare event of malacia, a T tube is preferable to a tracheostomy tube for both function and comfort. Over a period of weeks and months, the trachea usually becomes firm again, permitting removal of the T tube. Since the cartilages usually do regain stability, permanent expandable stents are inadvisable. Coated expandable stents are prone to producing rings of granulations at either end. As the incidence of massive goiter has diminished, critical tracheal collapse after a thyroidectomy is rarely encountered. In earlier days, failure of prompt recognition and treatment could even result in fatalities.[12]

If severe malacia is identified *during* surgery, then it may be managed safely, as just described. An alternative is to place splinting polypropylene rings in channels around the malacic segment of trachea, embedding them with strap muscles (see Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea"). I have done this in a few patients without long-term difficulty. Circumferential wrapping of the trachea with a heavy Marlex mesh attached with tissue adhesive has been reported, but I believe one must be concerned about possible devascularization.[13] Another procedure that was used in the past to solve this problem, in then areas of endemic goiter in Europe, was placement of traction sutures through the wall of the trachea, which were tied over buttons resting either on the surface of the sternocleidomastoid muscles on either side or on the skin. Externally tied traction sutures were removed after the trachea appeared to be stable. I have had no experience with such technique.

Special care must be taken to spare the recurrent laryngeal nerves and the parathyroid glands, since the bulk of large goiters often thwart their easy identification.

Cysts

Cysts that can compress the trachea are bronchogenic, thymic, and parathyroid.[14–19] Bronchogenic cysts are more likely to produce tracheobronchial symptoms in children, perhaps because the juvenile trachea is more malleable. Life-threatening distress can occur in infants.[20] Foregut enteric cysts are rare and may present as air-filled lesions adjacent to the trachea in its upper portion or at the thoracic inlet. If large enough, such cysts cause cough or an uncomfortable sensation of a mass in the neck. Esophageal compression may produce dysphagia. A cyst may be adherent to the tracheal wall, but communication to the trachea may be tiny or absent (Figure 15-9). Such cysts are presumed to be of congenital origin, but histology of the lining cells may not clearly differentiate between a tracheal or esophageal origin. The cyst must not be confused with herniation of the lung apex through Sibson's fascia or with apical bullae.

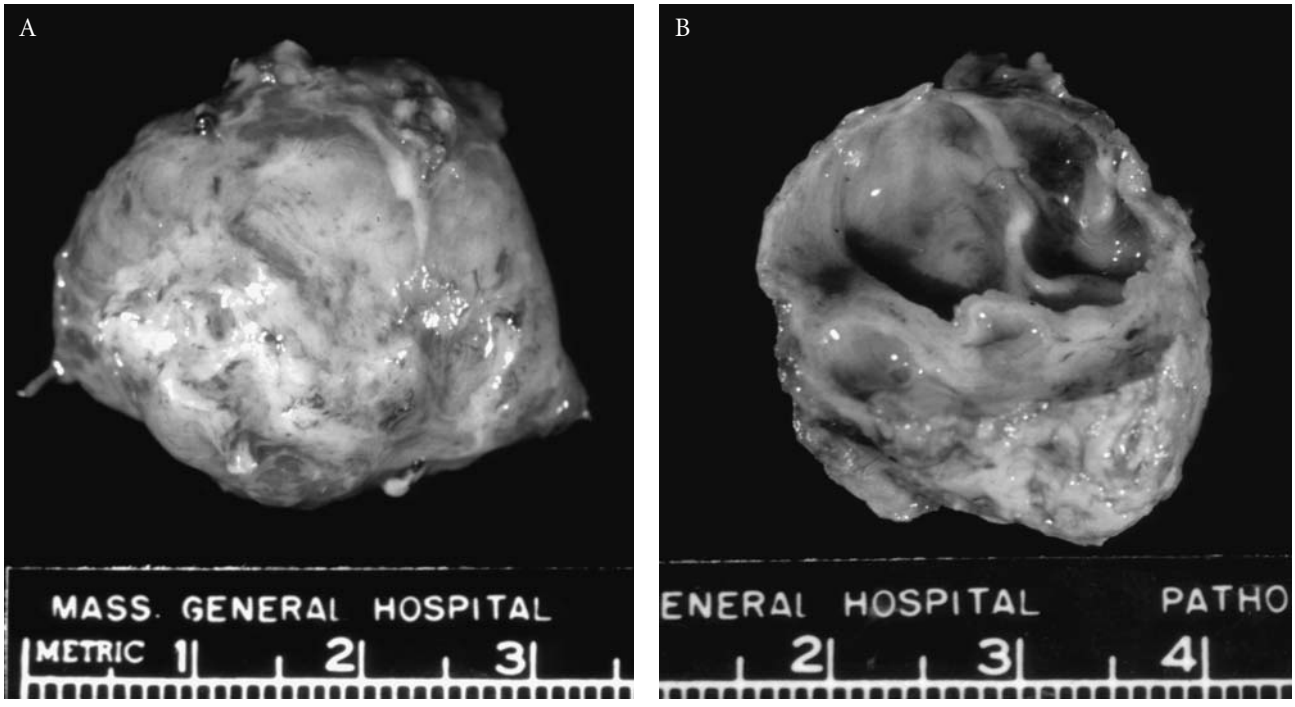


FIGURE **15-9** *Tracheal cyst. Dorsal enteric cyst, lined by respiratory-type epithelium, in a 61-year-old woman with dysphagia accompanied by gurgling, occasional dysphonia, and sense of a mass. She had no respiratory symptoms. The cyst was attached firmly to the tracheal wall at the right posterior cartilaginous margin, and lay at the level of the thoracic inlet. It was air filled, but communication with the trachea was too small to identify. The right recurrent laryngeal nerve was tightly adherent to the surface of the cyst.* A, *Exterior of cyst.* B, *Cyst on section.*

Suen and colleagues found mild dyspnea in only 1 of 42 patients with *bronchogenic cysts*, 9 of which were carinal and 9 peritracheal.[14] Respiratory distress occurred in 6 of 8 patients with cysts at the carina, with extrinsic airway compression found in all.[15] St. Georges and colleagues, in a multi-institutional study of 86 bronchogenic cysts, noted dyspnea in 16 which had mediastinal localization and in 8 with pulmonary cysts; specific airway compression was not mentioned.[21] Ribet and colleagues found dyspnea to be more common in 24 infants and children with bronchogenic cysts (25%) than in 45 adults (4 to 11%).[22] Compression was radiologically seen against the trachea in 5, against the left main bronchus in another 5, the right main bronchus in 2, and the intermediate bronchus in 3 patients.

Thoracotomy, most often posterolateral and on the side where the cyst presents, remains the favored surgical approach to bronchogenic cysts.[14,15,21,22] Median sternotomy is useful on occasion for a cyst deemed more accessible by this route, or was used in earlier cases prior to CT definition when a diagnosis of mediastinal tumor was entertained. Complete excision of a cyst prevents recurrence. Incomplete removal can result in infection, a chronic draining sinus, or even fistula to a bronchus or to esophagus. Secondary removal may prove technically very difficult (Figure 15-10).

Removal via cervicotomy or mediastinoscopy is applicable only in special circumstances. Communication from a cyst to a bronchus is usually managed by closure of a small opening, with second layer reinforcement. Bronchial sleeve resection and pericardial patch repair have been employed for larger defects.[15,23] In one 8½- year-old girl, a bronchogenic cyst in the carinal location, which had been excised elsewhere, proved to contain adenocarcinoma. Carinal resection and reconstruction was therefore subsequently performed, with apparent long-term cure.[14]

Thymic cysts are rare, but can occur in the neck and upper mediastinum, or, more often, entirely within the anterior mediastinum.[18] Thyroid and parathyroid tissues have also been found in these cysts. A true

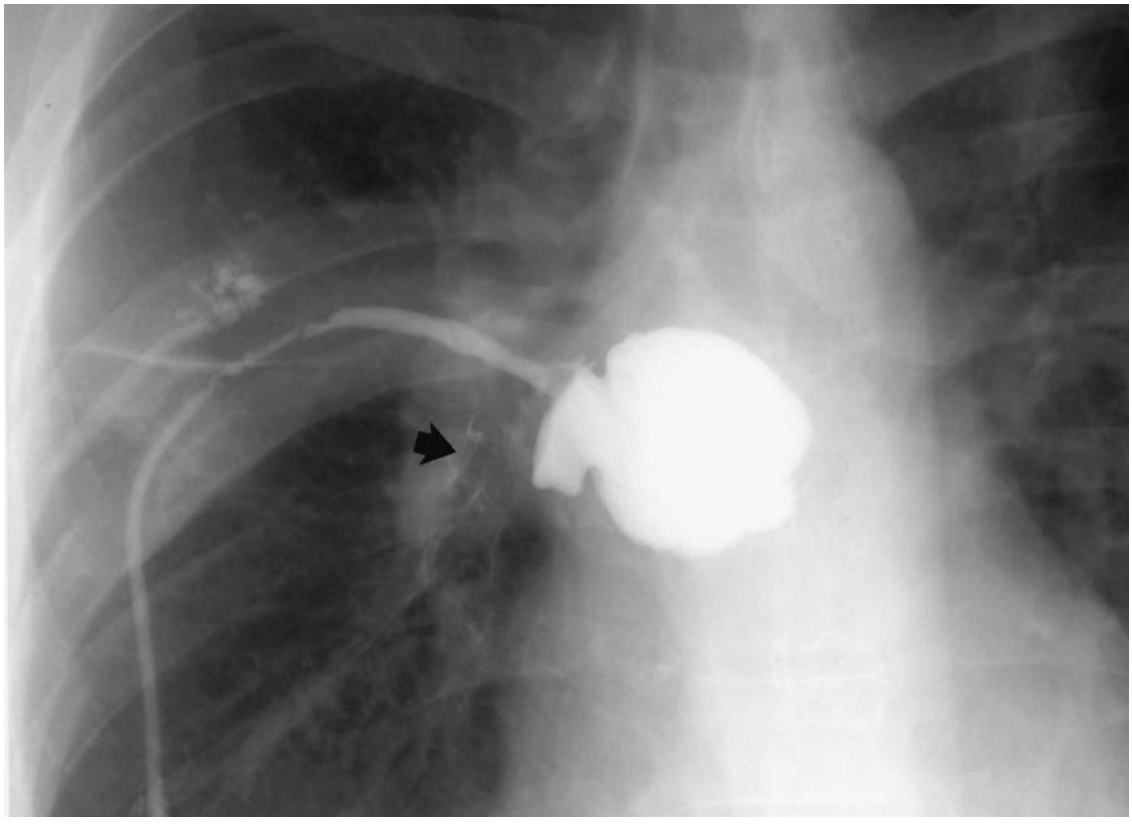


FIGURE 15-10 *Persistent bronchogenic cyst in subcarinal location in a 29-year-old male, 4 years after incomplete removal. Two years after the initial operation, following episodes of mediastinal infection and mediastinal drainage, a broncho-esophageal cystic fistula precipitated esophageal diversion. Intermittent signs of bronchial fistula, esophageal fistula, and sinus tract drainage led to referral. The sinogram shows a residual cyst with contrast entering the bronchus intermedius* (arrow). *The entire cyst was dissected out, the bronchial fistula closed, the esophagus repaired at the site of a densely scarred fistula, and the suture lines buttressed with healthy pericardial fat pad. Recovery was uneventful.*

thymic cyst is to be differentiated from a cystic thymoma, although a small thymoma or even carcinoma has been found in the cyst wall. In a review of 46 patients, Graeber and colleagues did not find tracheal compression, but rather dysphagia, hoarseness, and pain as manifestations.[17] This is probably due to the generally anterior location of cysts in front of the great vessels and pericardium. Cervical presentation in infancy or childhood appears more likely to cause tracheal obstruction.[16] In 1 child treated for severe tracheal compression by a thymic cyst, sufficient malacia proved to be present after cystectomy to require prolonged stenting with a tracheostomy tube until the cartilages became firmer (see Figure 6-16 in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children"). *Parathyroid cysts* are, on occasion, sufficiently large to narrow the trachea when they are in a cervical location or present as a cervical extension from the anterior–superior mediastinum.[18,19] A retrotracheal position is also described. The airway is not often affected symptomatically. In a few patients, hyperparathyroidism is present. The lesions are quite visible on roentgenograms and CT scans, and can easily be mistaken to be of thymic or thyroid origin. Surgical removal is likely to be effected through a cervical incision with, at most, an upper sternal extension.

Neoplasms

Airway invasion by secondary tumors (ie, carcinomas of the thyroid, lung, and esophagus) is discussed in Chapter 8, "Secondary Tracheal Neoplasms." Mediastinal tumors, including germ cell tumors,

hemangiopericytomas, thymomas, and lymphomas, are infrequent causes of tracheal obstruction. In the case of critical tracheal compression by a massive mediastinal tumor, a biopsy by core needle aspiration may be safer than subjecting the patient even to a brief general anesthesia for a diagnostic open biopsy. Cardiodynamic collapse has, on occasion, occurred under general anesthesia with absolutely massive tumors infiltrating the mediastinum, encasing the aortic root, and great vessels, despite a fully patent airway guaranteed with an endotracheal tube. The origin may be reflexive.

Hyalin-Vascular Lymph Node Hyperplasia (Angiofollicular Lymph Node Hyperplasia; Castleman's Disease)

A 69-year-old woman with a 4-month history of rapidly progressive dysphagia, dyspnea with stridor, and cervical swelling, presented with massive involvement of central mediastinal lymph nodes, shown by biopsy to be Castleman's disease. The lobulated disease narrowed and displaced the trachea to the right and posteriorly, impinging on the carina and narrowing main stem bronchi. The esophagus was displaced to the right. Aortography and venography showed brachiocephalic, left common carotid, and subclavian arteries displaced, and subclavian and brachiocephalic veins obstructed with thrombus. Extensive collateral venous flow in the neck and posterior mediastinum accompanied the superior vena cava syndrome.

The patient responded symptomatically to irradiation treatment (2,800 cGy), with improved breathing and swallowing, but died from bilateral pneumonia, likely associated with severe radiation pneumonitis, 2 months later.

Dr. Benjamin Castleman (personal communication) described 2 prior patients with bronchial compression from this disease, but without superior vena cava syndrome. One had responded to irradiation. Judging from other mediastinal masses that have been excised and proved to be Castleman's disease, surgical removal would have been the preferable treatment in the patients described above.

Tracheocele and Diverticula

Air-containing outpouchings adjacent to the trachea have been described by many terms, including tracheocele, tracheal diverticula, tracheal diverticulosis, trachiectasis, aerocele, and aerial goiter (Figure 15-11). These rare diverticula have been classified by Katz and colleagues as follows:[24]

1) Rudimentary bronchus. These rare and sometimes multiple diverticula involve all layers of the tracheal wall and occur at the junction of the membranous and cartilaginous wall, especially on the right. They are asymptomatic for the most part.
2) Ovoid diverticula, which appear pedunculated, are filled with viscous secretions and may be related to cystic dilation of mucus glands. They are asymptomatic.
3) A single air-filled sac, which usually has a wide mouth and may fill with enough air to produce symptoms, is seen rarely. It is of tracheal origin and differentiated from the slightly more common but still rare laryngocele. It may be associated with chronic cough.
4) Outpouchings of the thin tracheal wall, between deformed and partly malacic cartilaginous rings in tracheobronchomegaly (see Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions"), are seen as wide-mouthed diverticula on bronchoscopy and on imaging.

Symmetric Lipomatosis Colli

Symmetric lipomatosis colli (Lanois-Bensaude adenolipomatosis, multiple or benign symmetric lipomatosis, lipoma annulare colli, Madelung's disease), affecting the neck, supraclavicular regions, and upper back, very rarely is accompanied by respiratory distress due to laryngeal compression or infiltration of the false vocal cords and by tracheal compression from mediastinal fatty infiltration.[25,26] The fat may sometimes enlarge

rapidly, or it may remain unchanged for long periods. It is not encapsulated and penetrates surrounding structures deeply. Familial occurrence has been reported and mitochondrial DNA mutations have been implicated.[27] Multiple metabolic abnormalities and peripheral autonomic neuropathy have been found in association with the disease.[26]

The occurrence of airway difficulty is exceedingly rare in an already rare condition. The disease is observed in adult life and sometimes seems to be associated with large alcohol intake, but for the most part, it leads principally to disfigurement, limitation of movement of the neck and upper extremities, and difficulty in fitting clothing.[28] Other symmetric fat deposits occur in some patients in the breast, abdomen, lower back, and upper thighs.[26] Enzi found mediastinal involvement in 5 of 19 patients, confirmed by CT scans.[26] Four had clinical signs and symptoms of airway compression, and one who had severe narrowing of the trachea and superior vena cava obstruction as well, required a tracheostomy. A recurrent nerve was also paralyzed in this patient.

A collar of fatty tissue encircles the neck, lobular masses of fat fill the supraclavicular spaces, a "buffalo hump" is present in the back of the neck, and "football suit" shoulders are seen, summarized descriptively as a "horse collar" distribution (Figure 15-12). Parotid area deposits may produce a "chipmunk" appearance. Laryngeal involvement leads to decreased vocal pitch, progressive dyspnea, and stridor. Tracheal compression can contribute to dyspnea.[29] Laryngoscopy shows edema and bulging of the false cords, with marked narrowing of the airway at this level, and difficulty in visualizing the true vocal cords. Radiography of the larynx shows a slit-like upper air passage and reduced vocal cord movement. Cervical fatty accumulation occurs in the anterior neck, and preepiglottic fat lies anterior to the larynx.[30]

Neck dissection, with extensive removal of huge lipomas, failed to relieve one patient initially.[25] Laryngoscopic incision of a thin translucent false vocal cord mucosa released herniated fat, with slight improvement. A second open procedure via laryngofissure allowed excision of lipomatous tissue that bulged through the thyrohyoid membrane and dissected between thyroid laminae and laryngeal mucosa. Herniation of fat into the larynx extended from the anterior commissure to aryepiglottic folds posteriorly.

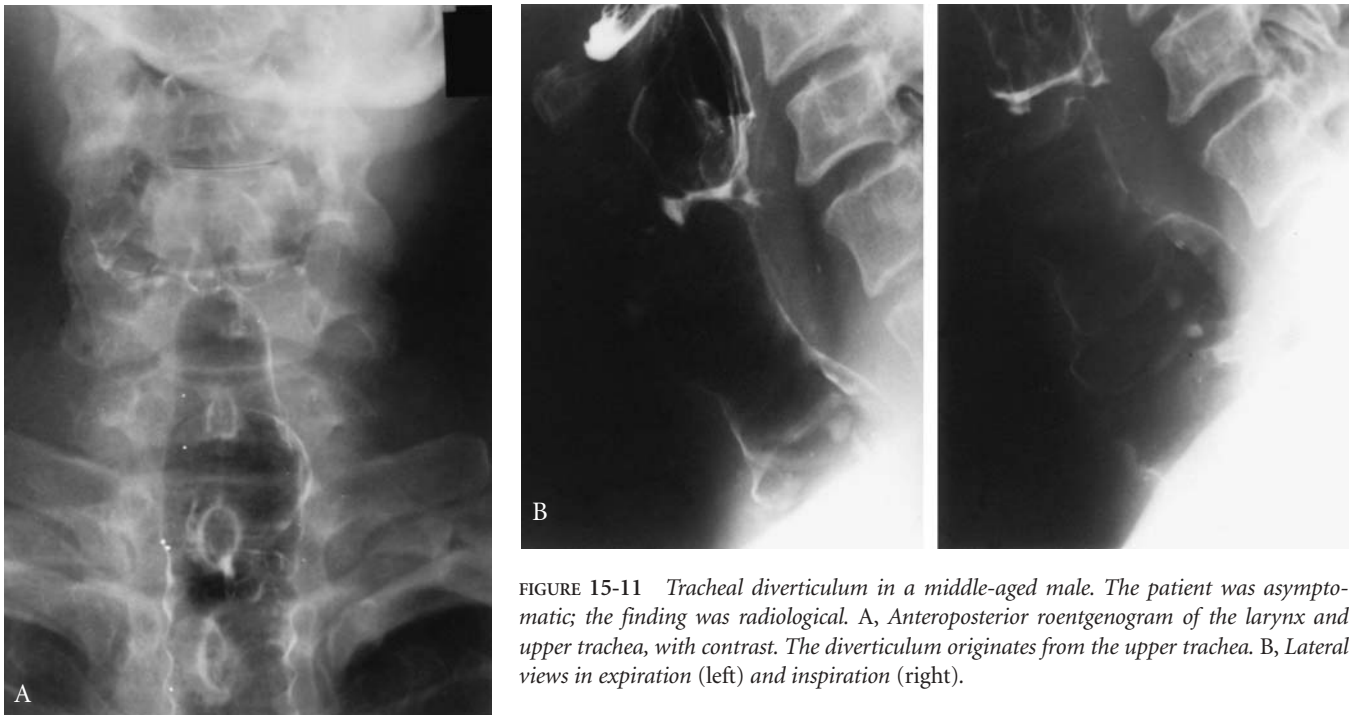


FIGURE **15-11** *Tracheal diverticulum in a middle-aged male. The patient was asymptomatic; the finding was radiological.* A, *Anteroposterior roentgenogram of the larynx and upper trachea, with contrast. The diverticulum originates from the upper trachea.* B, *Lateral views in expiration* (left) *and inspiration* (right).

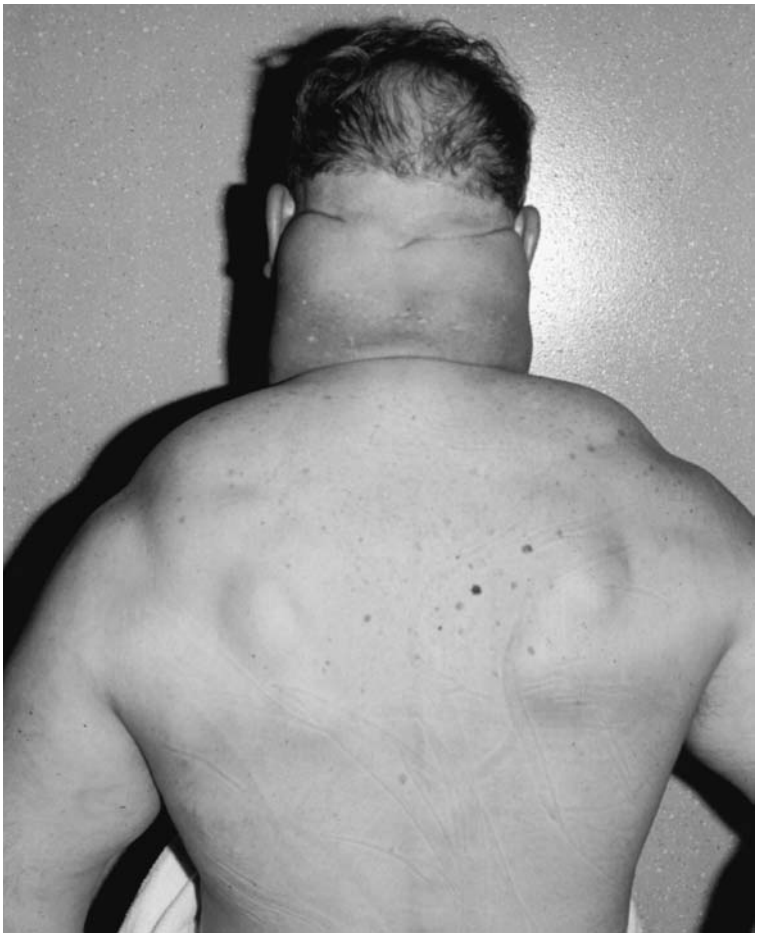


FIGURE **15-12** *Characteristic distribution of fat in symmetric lipomatosis colli. This patient underwent extensive neck dissection and also laryngofissure to relieve airway obstruction, as described in the text.*

The patient was fully relieved of respiratory and phonatory complaints and later underwent a further cosmetic procedure. The fat removed from these patients does not seem to regrow. Mounier-Kuhn and Haguenauer treated a similar patient by laryngoscopic excision of a posterior commissure lipoma.[31] On the basis of this very limited and scattered experience, the following approach seems prudent in case of upper airway obstruction in symmetric lipomatosis colli:

1) Careful assessment with CT, tomography, laryngoscopy, and bronchoscopy.
2) Cervical exploration for removal of lipomatous tissue.
3) Laryngofissure, if required, for excision of intralaryngeal submucosal lipoma causing obstruction at false cords or glottic structures.
4) Excision of mediastinal fat, if the trachea is compressed.

Intraoperative bronchoscopy should help to assess the extent of anatomic relief obtained at each step. Airway edema must be watched for postoperatively. The long-term prognosis should be satisfactory, given that lipomatous tissue does not seem to recur.

Vascular Compression

Note is made in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children," of the variety of *congenital vascular anomalies* that may cause airway obstruction. These include aortic vascular rings (double

aortic arch) with or without ligamentum arteriosum, pulmonary artery sling with or without congenital tracheal stenosis, and prominent innominate artery. An anomalous subclavian artery, which passes behind the esophagus and trachea, does not usually cause airway compression, except in conjunction with a right-sided and right descending aortic arch with Kommerell's diverticulum (See Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Recontruction of the Trachea.") *Aneurysm* of the thoracic aorta can obstruct the trachea and carina (Figure 15-13). A rare aneurysm of an anomalous subclavian artery, which was otherwise previously asymptomatic, also caused tracheal obstruction.

An enlarged right atrium has been reported to compress the airway, as has *massive dilation of pulmonary artery* due to congenital heart disease.

Postpneumonectomy Syndrome

In postpneumonectomy syndrome, a severely symptomatic airway compression is caused by extreme mediastinal shift and rotation, most frequently after a right pneumonectomy in the presence of a normal aortic arch.[32] Mirror image airway compression follows a left pneumonectomy in the presence of a right aortic arch. *After a right pneumonectomy*, the mediastinum moves to the right and posteriorly, as viewed on CT scans. Because of the attachments of the heart to the great vessels, the heart rotates counterclockwise with the great vessels, as viewed on CT scans (Figure 15-14). Herniation of the left lung and overdistension of the lung accompany this shift and rotation. In the most common situation, realignment of thoracic structures results in tracheal displacement to the right, and compression of the left main bronchus and sometimes the distal portion of the trachea, as the airway angles beneath the aorta and is flattened against the vertebral column or the descending aorta. The elongated residual pulmonary artery lies tightly against the anterior wall of the compressed bronchus (Figure 15-15). Such tracheobronchial compression has also been encountered in the analogous situation of displacement and rotation of the heart due to right lung agenesis.[33]

Similar anatomic distortion *after a left pneumonectomy* occurs in the presence of a right aortic arch.[32] In this case, a clockwise rotation of the mediastinum is seen, with the trachea pulled to the left and the right main bronchus compressed, overlying the vertebral column or aorta (Figures 15-16*A*,*B*). Because the right main bronchus is so much shorter than the left, it is not uncommon to find that the right upper lobe bronchus and the bronchus intermedius are also compressed against the vertebral column. Patients have also been observed with this syndrome after a left pneumonectomy with a normal left-sided aortic arch (Figures 15-16*C*,*D*).[34,35] A further variation was observed in a patient following a right pneumonectomy for infection in a congenitally nonfunctional lung. The left lower lobe bronchus alone was compressed over the aorta, perhaps due to limited left lung shift into a right hemithorax of chronically reduced volume (Figure 15-17).

Such an extreme shift was originally thought to occur principally or entirely in children. However, Grillo and colleagues presented 11 adults with severe symptoms, only 1 of whom had undergone pneumonectomy in childhood.[32] Seven were under the age of 30 years.

Radiographic demonstration of the intrathoracic realignment of the lung and mediastinum is an integral part of evaluation of this syndrome. Conventional radiography shows marked lateral and posterior displacement, but CT of the chest makes the rotation clear and shows the relationship of the airway to the great vessels and the spine.[36] Three-dimensional reconstruction adds further illumination. Multiplanar images obtained on magnetic resonance scanning may also be of help. With these adjuncts, angiography is no longer necessary for assessment. Bronchoscopy shows anterior–posterior or slightly oblique compression of the lower trachea and/or corresponding main bronchial origin (Figure 15-18). A small number of these patients also develop severe malacia of the cartilages of the compressed segment so that restoration of the mediastinal anatomy centrally fails to correct the airway obstruction. This is much more difficult to identify preoperatively, either by bronchoscopy or by radiography. Intraoperative bronchoscopy after initial surgical correction may reveal malacia.

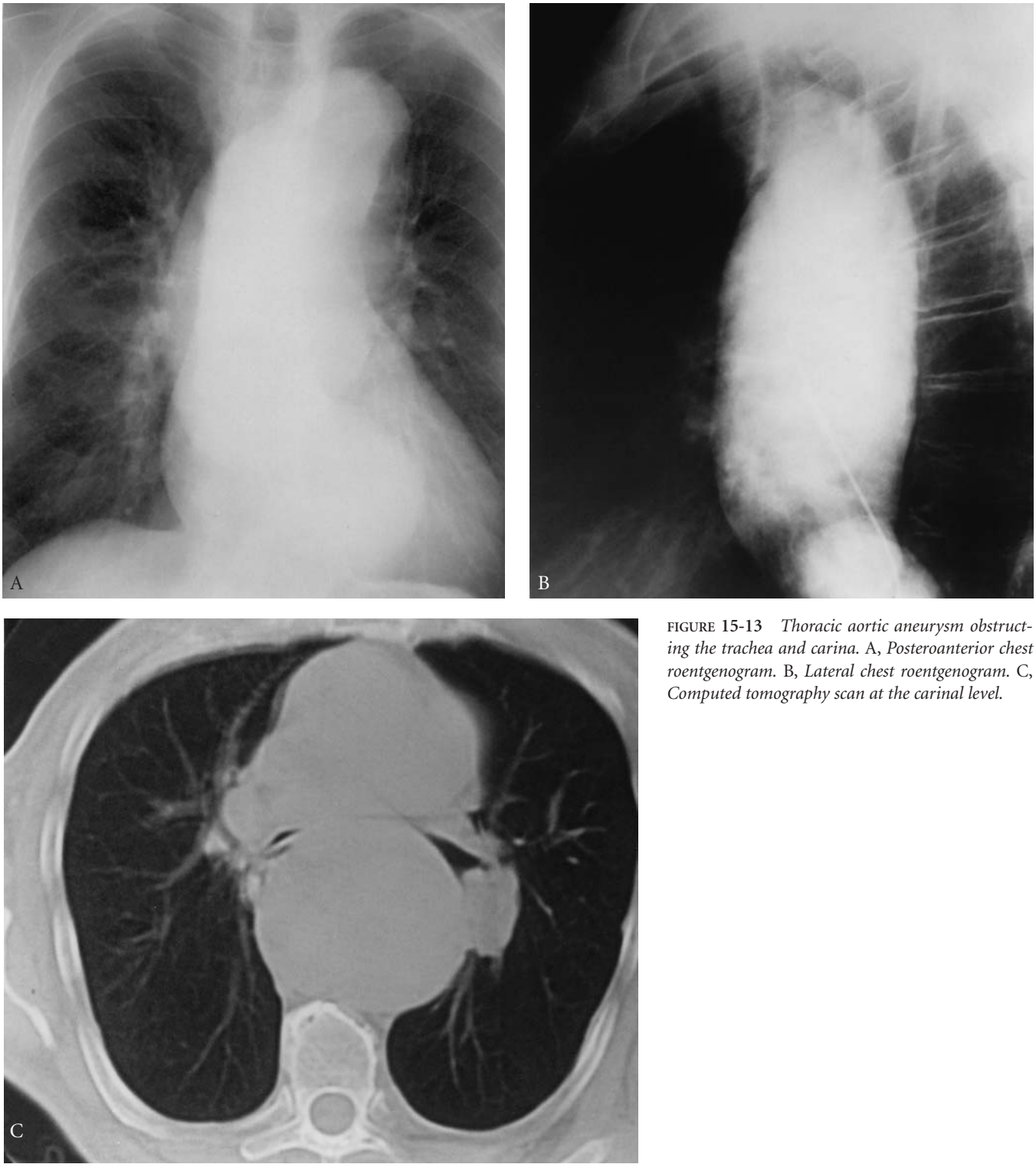


FIGURE **15-13** *Thoracic aortic aneurysm obstructing the trachea and carina.* A, *Posteroanterior chest roentgenogram.* B, *Lateral chest roentgenogram.* C, *Computed tomography scan at the carinal level.*

Kaunitz and Fisher proposed managing postpneumonectomy patients with indefinitely continued pneumothorax.[37] This seems impractical as a long-term solution for the problem. The available experience of attempts at definitive surgical correction of the syndrome formerly consisted mostly of case reports of

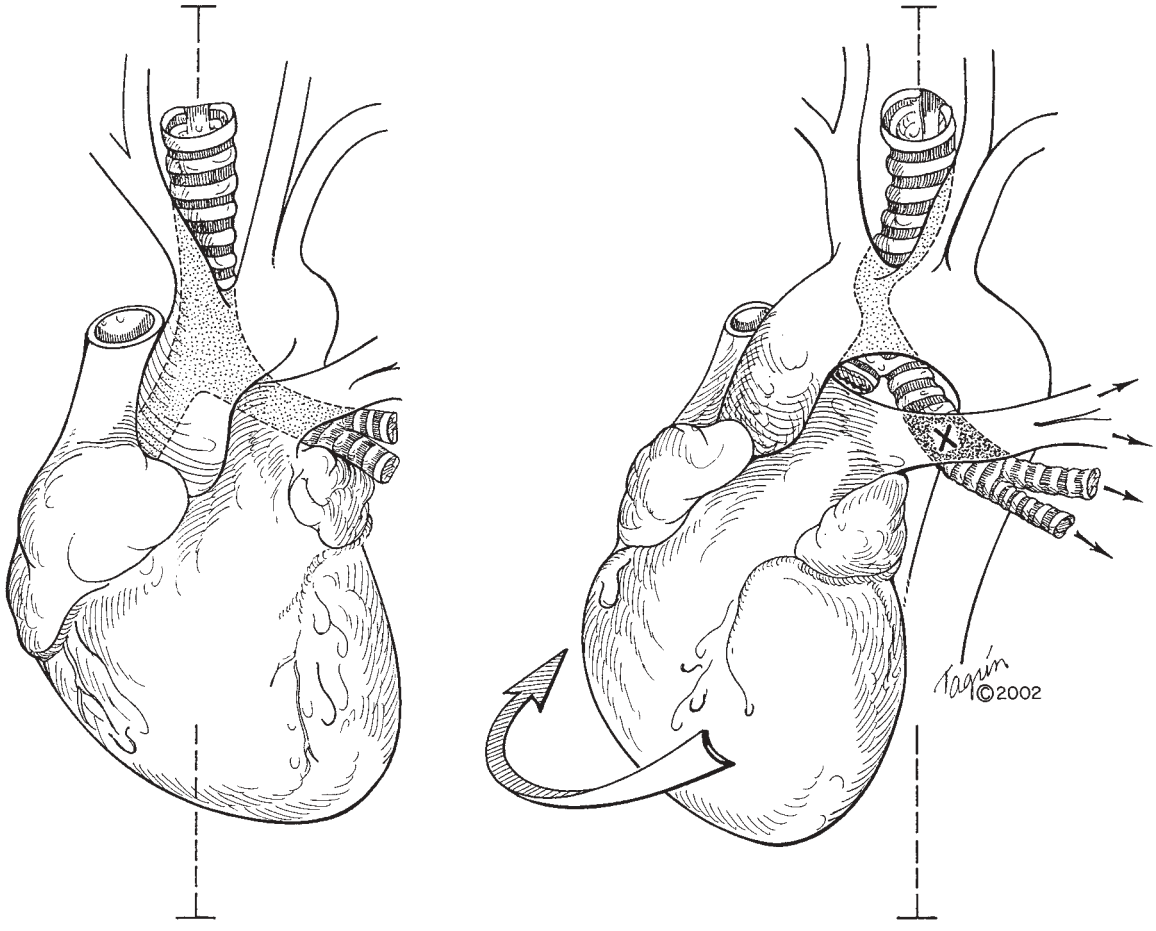


FIGURE **15-14** *After a right pneumonectomy with a left aortic arch, the heart and aortic arch are displaced to the right of the midline* (dashed vertical line) *and rotated* (arrow). *This results in displacement of the trachea and carina to the right and posteriorly. The left main bronchus is compressed between the left pulmonary artery and descending aorta or vertebral column. The mirror image is seen after a left pneumonectomy with a right aortic arch. Reproduced with permission from Grillo HC et al.*[32]

various techniques of treatment.[38–40] Surgical correction is best accomplished by replacement of the mediastinum to normal anatomic relationships, to allow the compromised airway to return to its normal position and patency (Figure 15-19).[32,39] Mediastinal repositioning relieves mechanical obstruction of the bronchial tree in those patients in whom malacia has not developed, and it also corrects overdistension of herniated and hyperexpanded lung. Our experience with later recurrences in two earlier patients indicated that simple replacement and suture fixation of the mediastinum alone is undependable, even though it may occasionally work in the long term. Therefore, the empty hemothorax must be filled to prevent recurrence of the disorder. We initially used silicone breast prostheses filled with silicone gel. However, after widespread concern about the possible untoward effects of leaking silicone arose, we used saline-filled prostheses instead.[32] This has seemed preferable to ping-pong ball plombage, in light of the very late complications seen with the latter in the treatment of tuberculosis. Expandable prostheses have been used and recommended for children and adolescents.[40] The use of breast prostheses has now been applied more generally, with success.[41,42] Stenting has also been applied as primary treatment for the syndrome.[43] Long-term results of stenting will be needed to clarify both its continuing efficacy and safety.

At corrective operation, the side of the original pneumonectomy is reentered and the scar and adhesions dissected sufficiently to permit repositioning of the heart and mediastinal structures to a normal central location. After scarification of the pericardium anteriorly and of the retrosternal fascia, the pericardium is fixed to the fascia behind the sternum with two rows of 0 Prolene sutures. Care is taken not to produce tamponade by reefing up too much pericardium. Careful cardiac monitoring is essential during this phase of the procedure (See Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea").

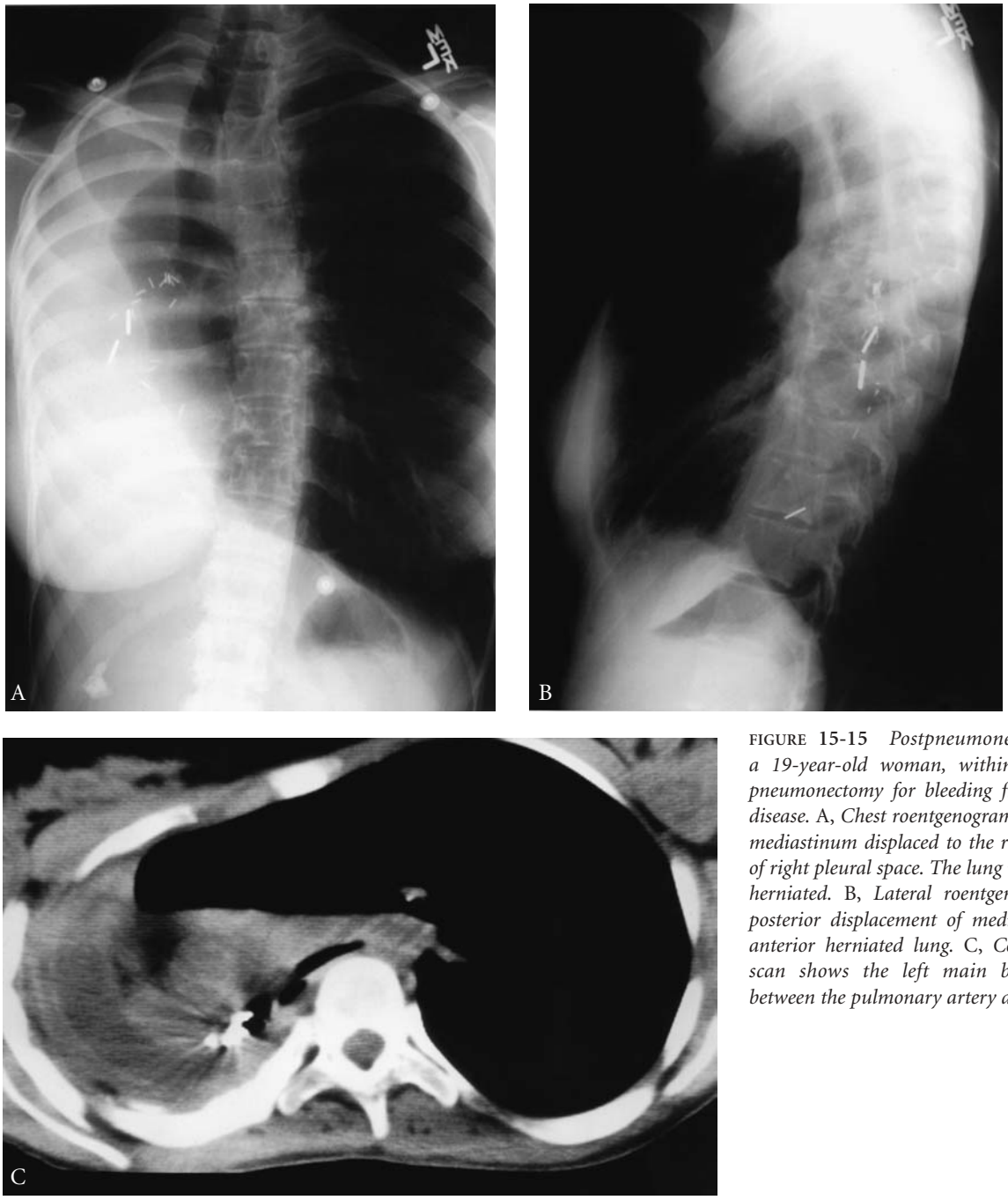
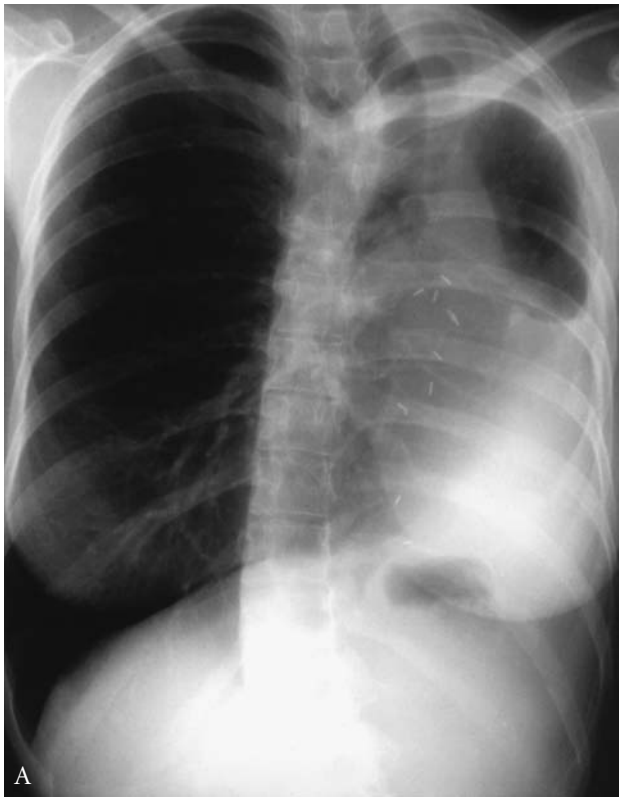


FIGURE **15-15** *Postpneumonectomy syndrome in a 19-year-old woman, within 1 year after right pneumonectomy for bleeding from congenital cystic disease.* A, *Chest roentgenogram shows the heart and mediastinum displaced to the right with obliteration of right pleural space. The lung is hyperexpanded and herniated.* B, *Lateral roentgenogram demonstrates posterior displacement of mediastinal contents and anterior herniated lung.* C, *Computed tomography scan shows the left main bronchus compressed between the pulmonary artery and spine.*



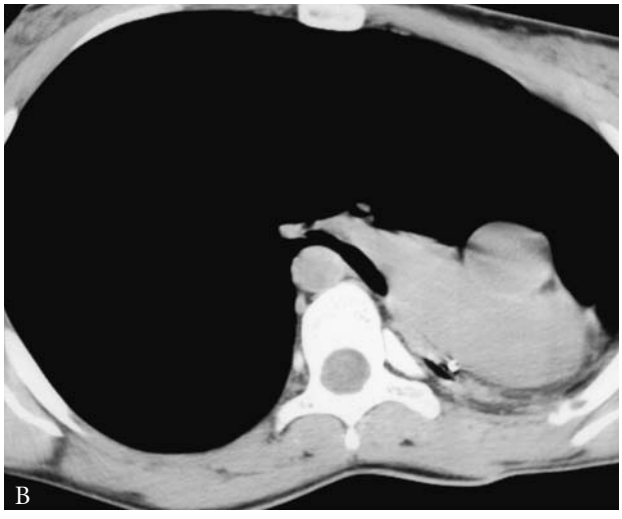
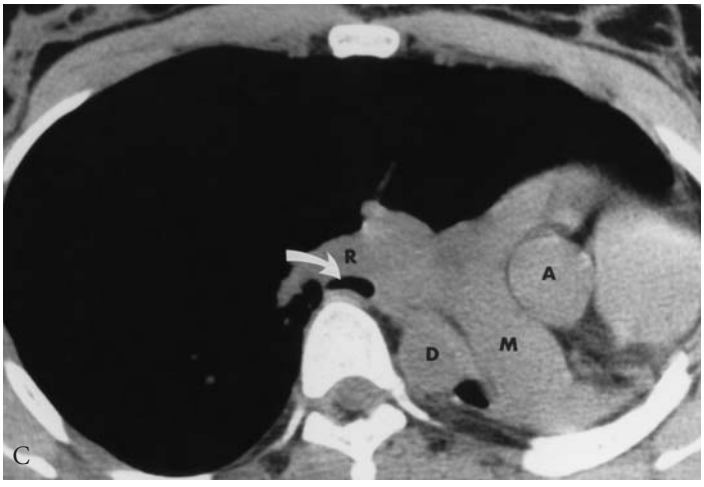


FIGURE **15-16** *Postpneumonectomy syndrome, following a left pneumonectomy with a right aortic arch, in a 29-year-old woman, 16 months after pneumonectomy for congenital hypoplasia with hemoptysis. She had dyspnea, cough, and increasing stridor.* A, *Chest roentgenogram is essentially a "mirror image" of Figure 15-15A. Lateral view corresponded.* B, *Computed tomography scan shows right upper and middle lobe bronchi squeezed between the pulmonary artery and aorta and spine.*



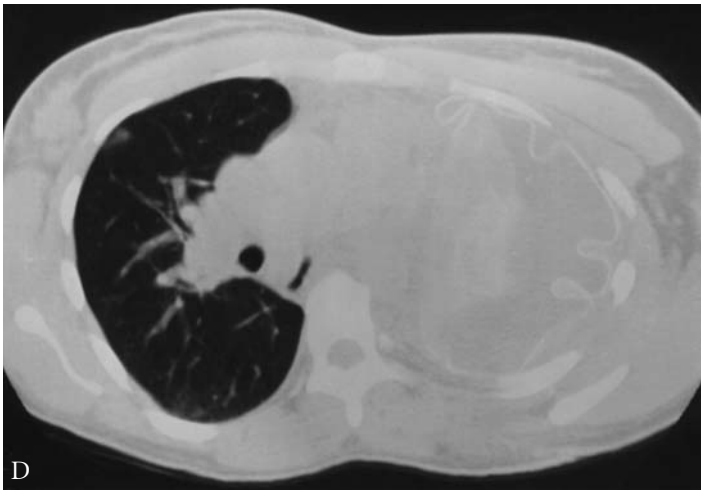


FIGURE **15-16** (CONTINUED) *Postpneumonectomy syndrome in a 26-year-old woman, 14 years after a left pneumonectomy in the presence of a normal left-sided aorta. Short carinal resection had also been necessary. Dyspnea was of 3 years duration.* C, *Computed tomography (CT) scan shows the bronchus intermedius* (arrow) *compressed against the vertebra. A = ascending aorta; D = descending aorta; M = main pulmonary artery; R = right pulmonary artery.* D, *CT scan after corrective surgery. Lung volume is returned to normal and the main bronchus is on the right of the spine.*

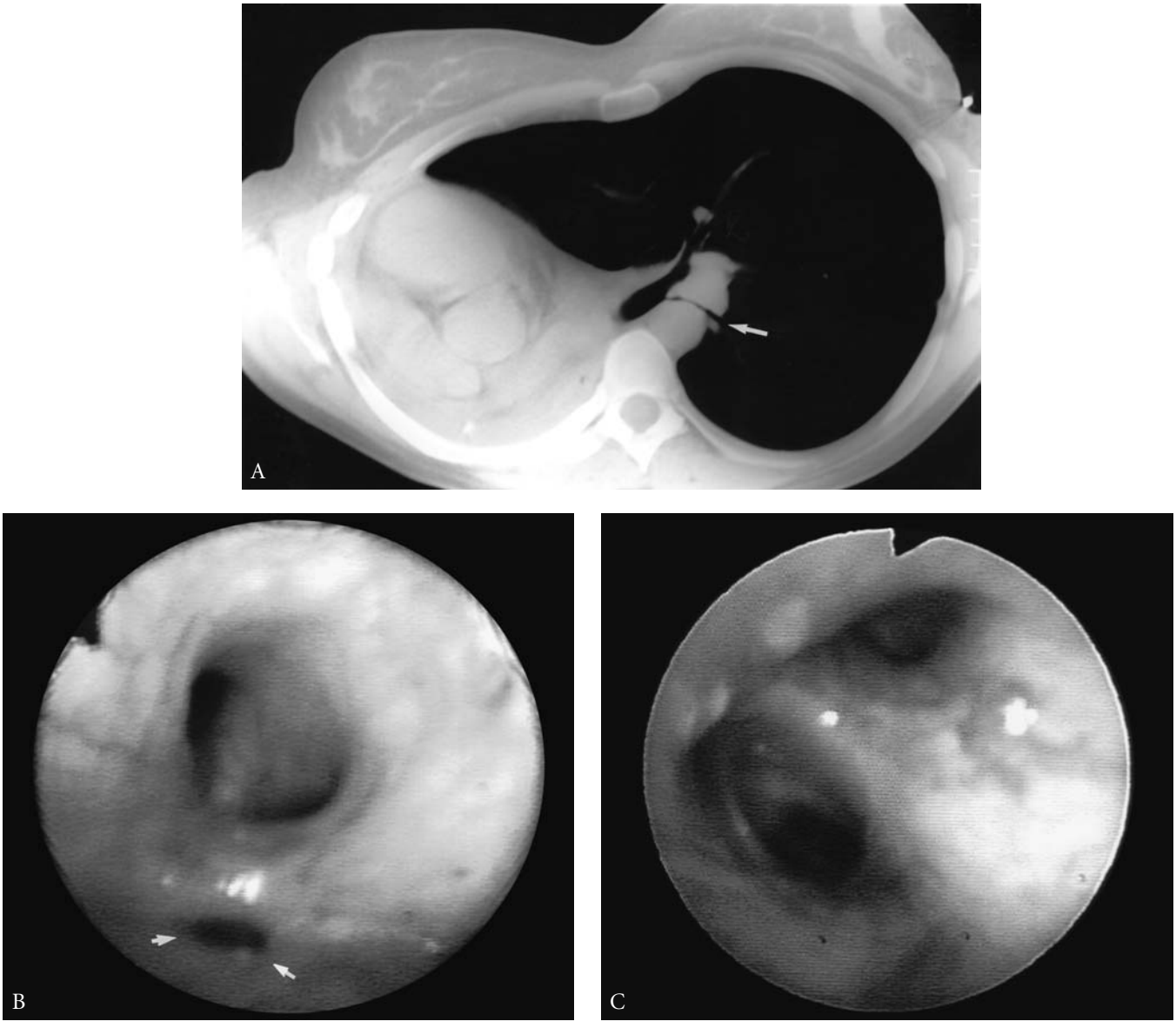


FIGURE **15-17** *Postpneumonectomy syndrome after a right pneumonectomy with obstruction of the left lower lobe bronchus alone, in a 39-year-old woman who had a right pneumonectomy 1 year before because of abscess, pneumonia, and hemoptysis in a congenitally cystic lung with ectatic bronchi and vascular anomalies. Six months later, she recognized progressive dyspnea, which became incapacitating.* A, *Computed tomography scan shows near complete obstruction of the left lower lobe bronchus* (arrow) *between the pulmonary artery and aorta. The upper lobe bronchus was rotated but open. Note the reduced volume of the right hemithorax related to the relatively nonfunctional right lung, which had resided there. Repositioning and insertion of saline-filled prostheses produced complete relief. Pre-* (B) *and postoperative* (C) *bronchoscopies. The lower lobe bronchus is a mere slit preoperatively* (arrows). *Both primary divisions of the upper lobe are seen. Both lobar orifices are widely patent postoperatively.*

Repositioning alone is insufficient, since the pericardium will eventually work its way free if the hemithorax is not filled. A prosthetic volume is selected which is sufficient to fill the space without compressing the heart and remaining lung, which have been returned to the heart's original position and to a more normal lung volume. Initially, I dropped four intercostal muscle bundles with their attached costal periosteum against the pericardium, leaving the muscles attached anteriorly and posteriorly in the manner of Kergin's modified thoracoplasty.[44] Periosteum was left on the lateral surfaces of the ribs to maintain bony

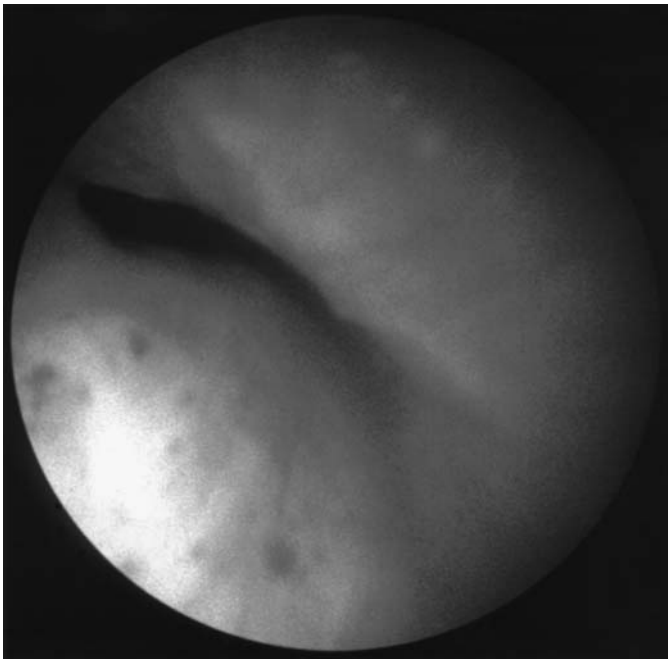


FIGURE 15-18 *Typical bronchoscopic appearance of the junction of the trachea and left main bronchus in postpneumonectomy syndrome, 2 years following a right pneumonectomy for trauma in an 18-year-old male. Also, see Figure 40 (Color Plate 16).*

integrity. The intent was to provide a firm partition, which would solidify as the periosteum calcified and so maintain the mediastinal repositioning, even if the prosthetic filler had to be removed later. This proved to be unnecessary, and we now simply fill the hemithorax with saline-containing prostheses of appropriate volume. The patency and stability of the airway is checked intraoperatively by flexible bronchoscopy after repositioning, and again after prosthetic placement. It is then rechecked when the patient is placed supine on the table after completion of the operation. The cardiopulmonary dynamics are also observed closely as the patient is placed on his/her back after the chest has been closed. Sometimes, it is found that as the thoracic incision is closed, the prosthetic volume proves to be too great and produces a tamponade effect. The partially closed incision is reopened and the volume adjusted.

In 10 patients who initially underwent mediastinal repositioning, including 2 who had recurrence prior to the use of prosthetics to maintain repositioning, 5 did well. One died a month later, presumably from pulmonary embolism. The seventh patient showed significant residual airway malacia, and the trachea was later reconstructed. Three other patients who had severe malacic obstruction of the airway after mediastinal repositioning underwent a variety of procedures, most of which involved aortic division to remove the compressive presence of the aorta, using bypass grafts, and in some, tracheobronchial reconstruction. Two patients died postoperatively, and 1 patient remains well many years later. It is clear that the problem of severe malacia in this syndrome has not yet been solved. Today, it is probably best to consider stenting the airway if such a case appears. Mediastinal repositioning generally seems prudent in order not to force a foreign body, such as a stent with semirigid components, into a bronchus that is pinched between the pulmonary artery and sometimes the aorta, where erosion would be disastrous. However, one frail, elderly patient, who had severe obstruction after a right pneumonectomy many years earlier for tuberculosis, was successfully palliated for some years by insertion of a specially constructed, extra-long silicone T tube. As noted, others have reported short-term success, at least, with stents.[43] We have not encountered severely malacic airways in 11 additional patients operated upon since the original report.

Pulmonary function studies demonstrated improvement in flow rates and a decrease in hyperinflation of the lung (Tables 15-2, 15-3; Figure 15-20).[32] Improved flow is reflected by an increase in the peak

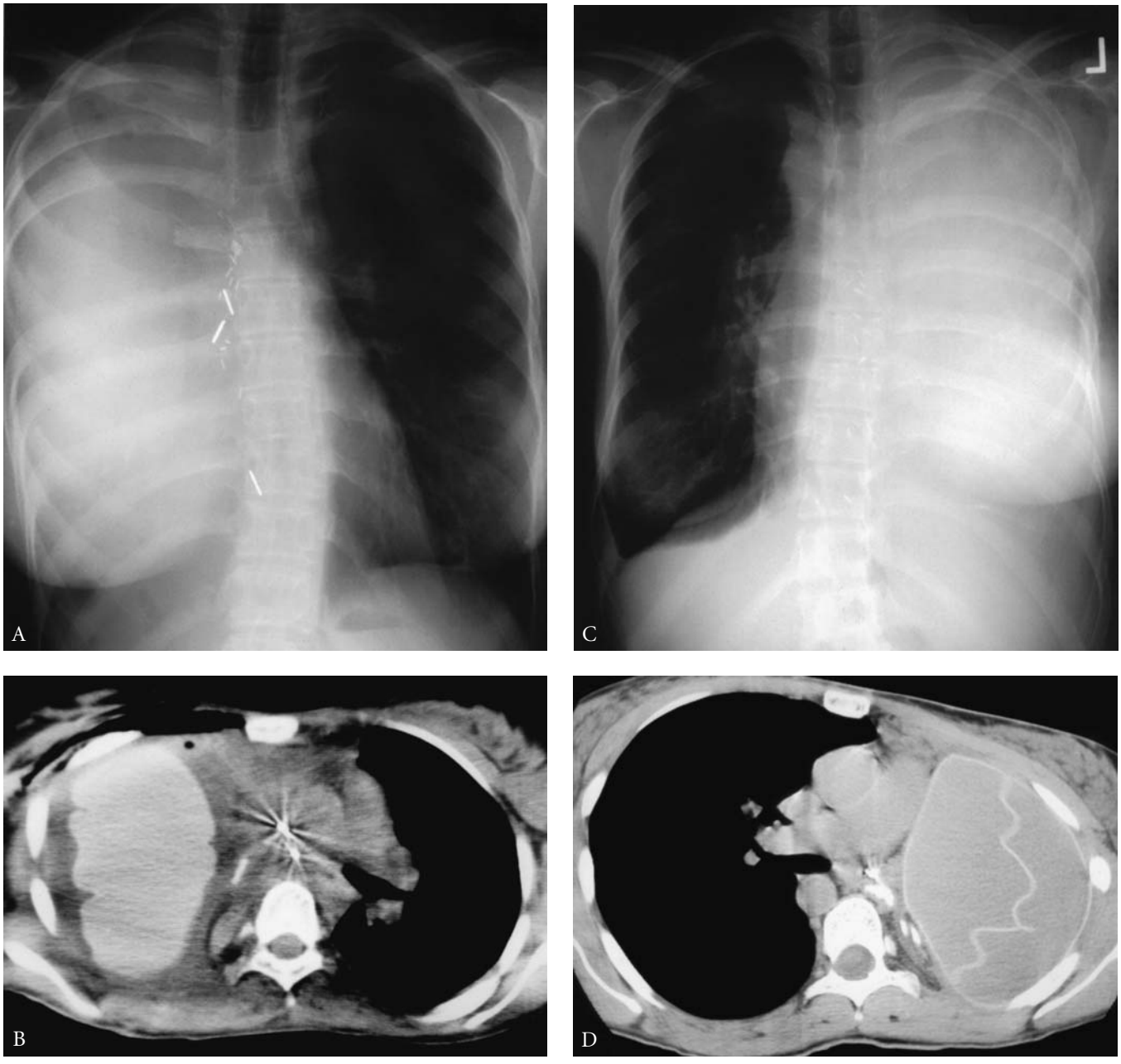


FIGURE **15-19** *Postoperative images after mediastinal repositioning with implantation of saline-filled breast prostheses.* A, *Chest x-ray film of the patient seen in Figure 15-15*A. *The trachea and mediastinum are returned to the midline and the lung to normal volume.* B, *Computed tomography scan of this patient shows the outline of the prostheses, and return of the left main and upper lobe bronchi to normal location and patency.* C, *Chest x-ray film of the patient from Figure 15-16*A. D, *Computed tomography scan shows right main and upper lobe bronchi widely open and to the right of the spine.*

expiratory flow rate and in the ratio of the forced expiratory volume in 1 second to the forced vital capacity (FEV_1/FVC). The increase in peak flow was associated with loss of an upper airway obstruction plateau and was primarily due to relief of tracheal compression. The improvement in the FEV_1/FVC ratio from a moderately obstructed value of 0.52 to a value in the normal range of 0.75 was due to several factors. First, the decrease in FVC due to relief of hyperinflation was always greater in absolute amount than the decrease in FEV_1. Second, upper airway obstruction was so severe in 3 patients that relief of tracheal compression

Table 15-2 Mediastinal Repositioning for Postpneumonectomy Syndrome: Preoperative and Postoperative Spirometry

	Preoperative Testing (% Predicted)				*Postoperative Testing (% Predicted)*			
	FEV_1 (L)	*FVC (L)*	*FEV_1/FVC*	*PEFR (L/sec)*	*FEV_1 (L)*	*FVC (L)*	*FEV_1/FVC*	*PEFR (L/sec)*
	1.48 (60%)	2.54 (77%)	0.58 (70%)	2.21 (37%)	1.31 (53%)	1.68 (53%)	0.78 (101%)	3.1 (52%)
	1.07 (29%)	1.66 (39%)	0.64 (73%)	1.97 (29%)	1.17 (35%)	1.52 (39%)	0.77 (80%)	2.5 (36%)
	0.35 (9%)	1.78 (40%)	0.19 (22%)	0.48 (7%)	1.85 (47%)	2.17 (47%)	0.85 (101%)	3.37 (46%)
	1.72 (61%)	2.44 (72%)	0.71 (87%)	2.31 (40%)	1.35 (52%)	1.78 (56%)	0.76 (93%)	2.63 (51%)
	0.81 (20%)	1.57 (31%)	0.52 (67%)	1.89 (20%)	1.15 (28%)	1.77 (34%)	0.65 (83%)	3.67 (39%)
	1.23 (26%)	3.08 (54%)	0.4 (48%)	2.88 (29%)	1.66 (35%)	2.66 (47%)	0.63 (76%)	4.5 (45%)
	1.67 (52%)	2.83 (75%)	0.59 (69%)	3.1 (48%)	1.41 (43%)	1.73 (46%)	0.81 (94%)	3.91 (61%)
Mean ± SE	1.19±0.19	2.27±0.23	0.52±0.07	2.12±0.32	1.41±0.10*	1.90±0.15*	0.75±0.03†	3.38±0.27†

Reproduced with permission from Grillo HC et al.[32]

FEV_1 = forced expiratory volume in 1 sec; FVC = forced vital capacity; PEFR = peak expiratory flow rate; SE = standard error.

*Not significantly different from the preoperative value.

†$p < .05$ preoperative value.

Table 15-3 Preoperative and Postoperative Plethysmography

	Preoperative Testing (% Predicted)			*Postoperative Testing (% Predicted)*		
	TLC (L)	*RV (L)*	*RV/TLC*	*TLC (L)*	*RV (L)*	*RV/TLC*
	3.5 (72%)	1.06 (64%)	0.3 (90%)	2.43 (51%)	0.63 (40%)	0.26 (76%)
	7.55 (105%)	5.98 (241%)	0.79 (229%)	3.62 (49%)	1.85 (73%)	0.51 (148%)
	5.69 (76%)	2.61 (141%)	0.46 (186%)	4.2 (56%)	1.54 (83%)	0.37 (150%)
	7.36 (144%)	4.53 (290%)	0.62 (203%)	3.36 (66%)	1.63 (104%)	0.49 (161%)
Mean ± SE	6.03±0.94	3.55±1.08	0.54±0.11	3.40±0.37*	1.41±0.27†	0.41±0.06†

Reproduced with permission from Grillo HC et al.[32]

RV = residual volume; SE = standard error; TLC = total lung capacity.

*$p < .05$ versus preoperative value.

†Not significantly different from preoperative value.

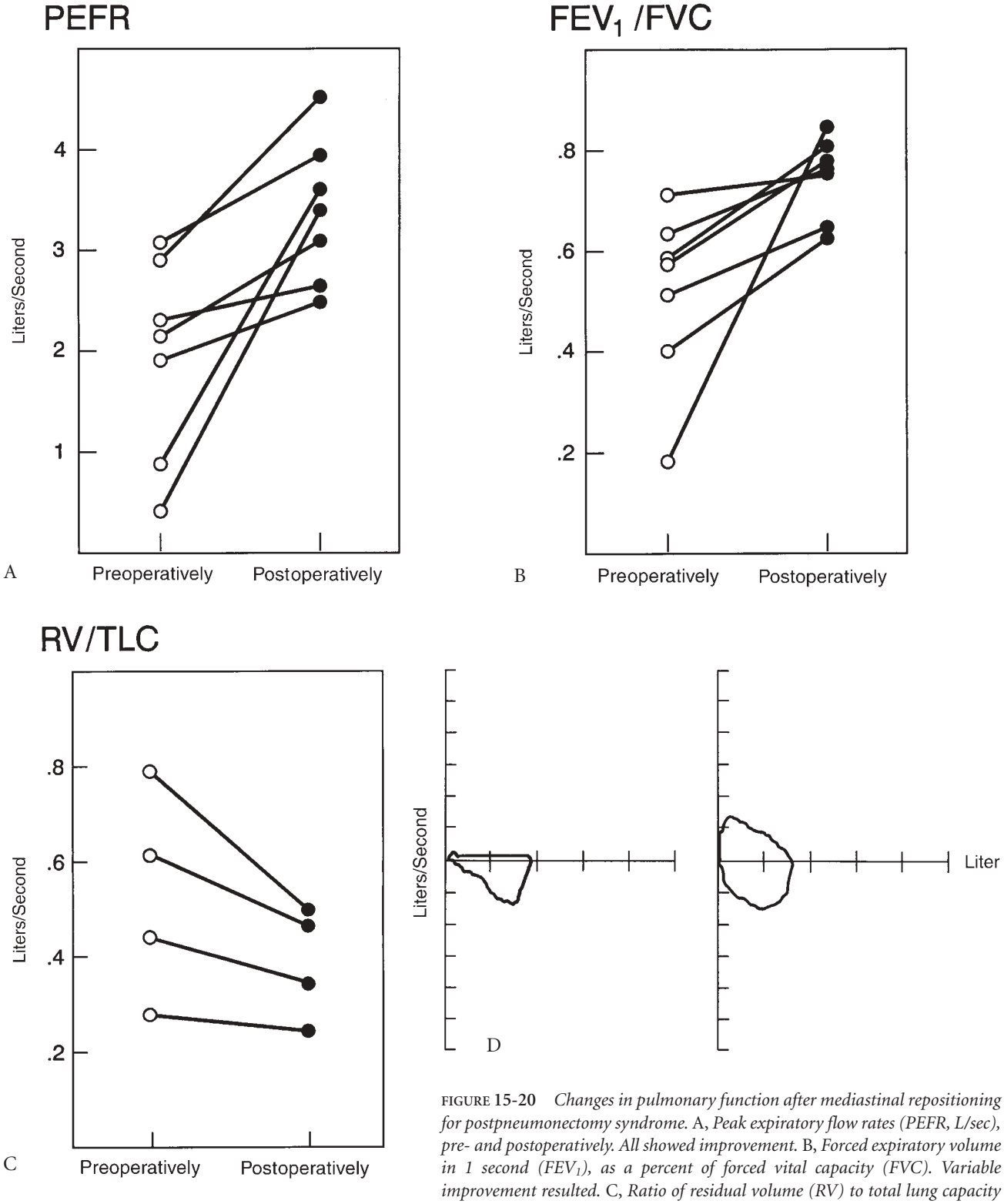


FIGURE 15-20 *Changes in pulmonary function after mediastinal repositioning for postpneumonectomy syndrome.* A, *Peak expiratory flow rates (PEFR, L/sec), pre- and postoperatively. All showed improvement.* B, *Forced expiratory volume in 1 second (FEV_1), as a percent of forced vital capacity (FVC). Variable improvement resulted.* C, *Ratio of residual volume (RV) to total lung capacity (TLC) measured pre- and postoperatively. RV decreased in all, as a percent of total lung capacity.* D, *Pre- and postoperative flow volume curves in a patient. FEV_1 rose from 0.35 L (9%) to 1.85 L (47%) and FVC from 1.78 L (40%) to 2.17 L (47%). Note the change in the shape of the curve. Reproduced with permission from Grillo HC et al.*[32]

resulted in an increased FEV_1. It is also possible that the severity of hyperinflation inhibited elastic recoil of the lung. There was marked decrease in all static lung volumes, total functional residual capacity, and residual volume. A decreased residual volume or total lung capacity resulted. Clinical symptomatic improvement following repositioning may be more significant than measured function.

There is no explanation from this study, subsequent patients, or other reports as to why such extreme displacement and rotation occurs in a small subset of patients who undergo pneumonectomy. The low incidence does not seem to justify attempts at prophylactic filling of the postpneumonectomy space in every patient undergoing pneumonectomy, if there is any risk. Prophylaxis might be advisable if a permanent and wholly innocuous method of filling the hemithorax were to be discovered.

Straight Back Syndrome

Straight back syndrome is a rare congenital presentation of a perfectly vertical cervicothoracic vertebral spine with absence of normal dorsal curvature. A degree of pectus excavatum is often present, and minimal space is noted between the posterior aspect of the upper manubrium and the vertebral spine (Figure 15-21).[45] Dyspnea may occur and restrictive ventilatory defect has been variably described, but some patients have no respiratory complaints. Palpitations and chest pain are other occasional complaints. Mitral valve prolapse is associated. Systolic murmur may be heard. The heart appears enlarged on a roentgenogram but is really displaced to the left by the bony deformities. Pulmonary hypertension during exercise, secondary to elevated pulmonary venous pressure, may be due to compression of the left inferior pulmonary vein against the aorta and the left atrium against the spine.

In a 20-year-old patient with dyspnea, which progressed over several years, the trachea was severely compressed against the vertebral column by the posterior ridge of the upper sternum, and was further indented by the brachiocephalic artery because of a limitation of mediastinal space due to a mild pectus excavatum deformity (Figure 15-22). The distance between the back of the upper sternum and vertebrae measured 1.5 cm. In addition, the lower trachea was splayed out against the vertebral column. Bronchoscopy clearly confirmed these three points of compression. Another patient presented to us with her trachea compressed between the back of the sternum, and a vertical spine, which had followed insertion of a rod for treatment of scoliosis in childhood. The trachea was also significantly indented by the brachiocephalic artery. Whereas this may be an example of an "acquired straight back syndrome," it might be noted that Winter and colleagues called attention to a significant diminution in pulmonary function in patients with idiopathic lordoscoliosis compared to those with kyphoscoliosis.[46] Operative correction (Harrington rods and vertebral fusion) usually improved pulmonary function in these patients and also enlarged the space between the sternum and vertebral column. Change in the thoracic inlet, however, was not commented upon.

Our first patient was transferred intubated for acute respiratory distress. Mild pectus excavatum was present. Its correction would not have relieved the tracheal obstruction. The upper point of compression was relieved by excising a plate of the upper sternum, removing the backward folding upper ridge of the manubrium, and thinning the residual plate of the sternum before replacing it. The brachiocephalic artery was transplanted more proximally and laterally on the ascending aorta to place it parallel to the trachea rather than across the trachea. The third point of narrowing, the distal trachea, was enlarged by fixing the cartilages and membranous wall to a vertical posterior Marlex splint, which returned the cartilages to a normal "C" shape (See Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea"). The result was excellent (Figure 15-23). The patient with the apparently acquired problem was treated in similar fashion for upper tracheal compression, since no distal tracheal problem existed. Continued intermittent obstruction, on rolling the clavicular heads forward and inward, necessitated their removal later. Professor Philippe Dartevelle and colleagues (personal communication, 1996) treated a patient with sternal tracheal compression by dividing the sternum and wedging a plate of methyl

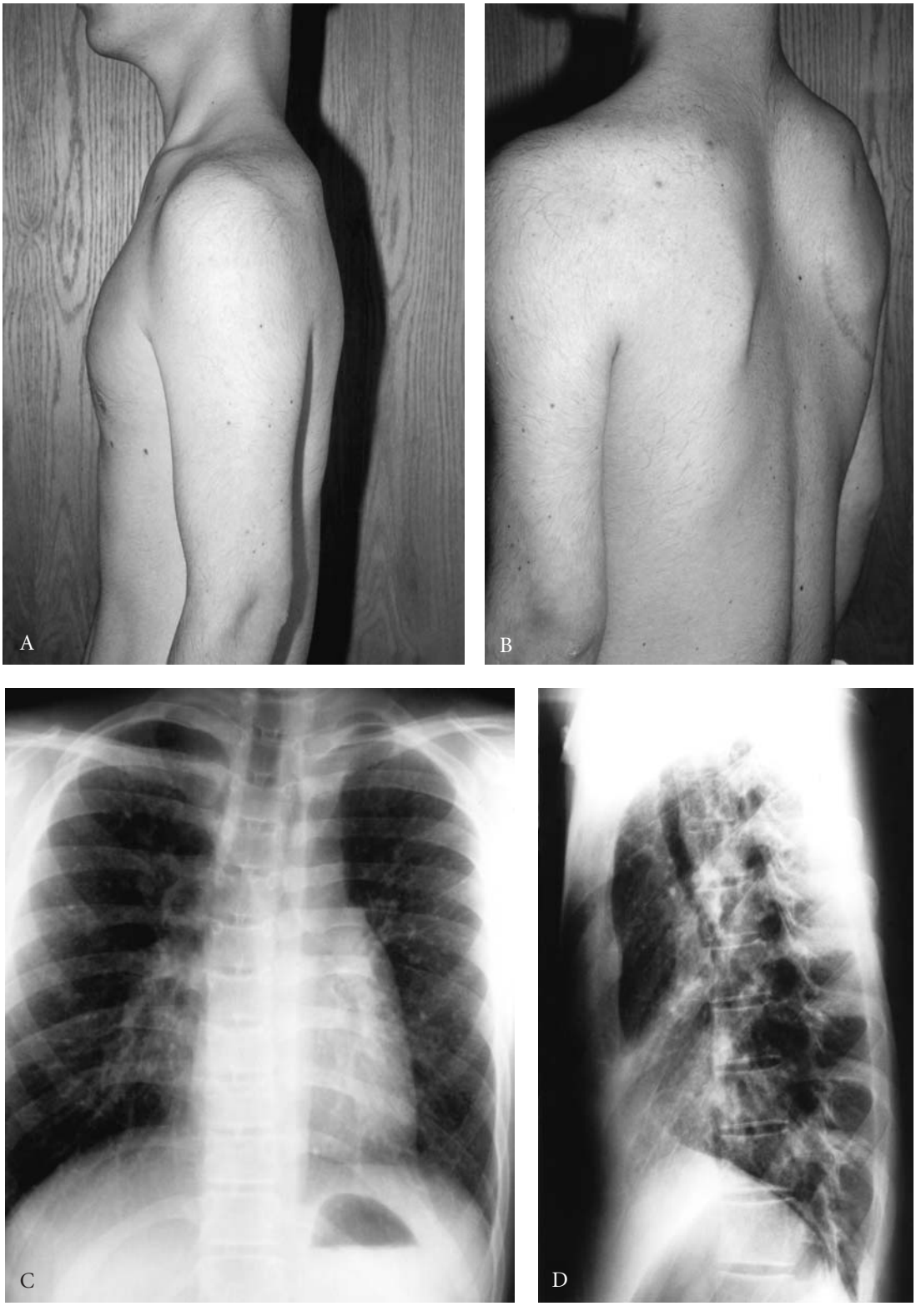


FIGURE **15-21** *Straight back syndrome.* A, *Lateral view shows the narrowness of the patient's chest, mild pectus excavatum, and a spine lacking normal curvatures.* B, *Posterior view. Absent curvature of the vertebral column is evident.* C, *Chest roentgenogram. The flattened trachea appears broad and widens even more distally.* D, *Lateral roentgenogram emphasizes the straight vertebral column. Note the narrowness of the thorax.*

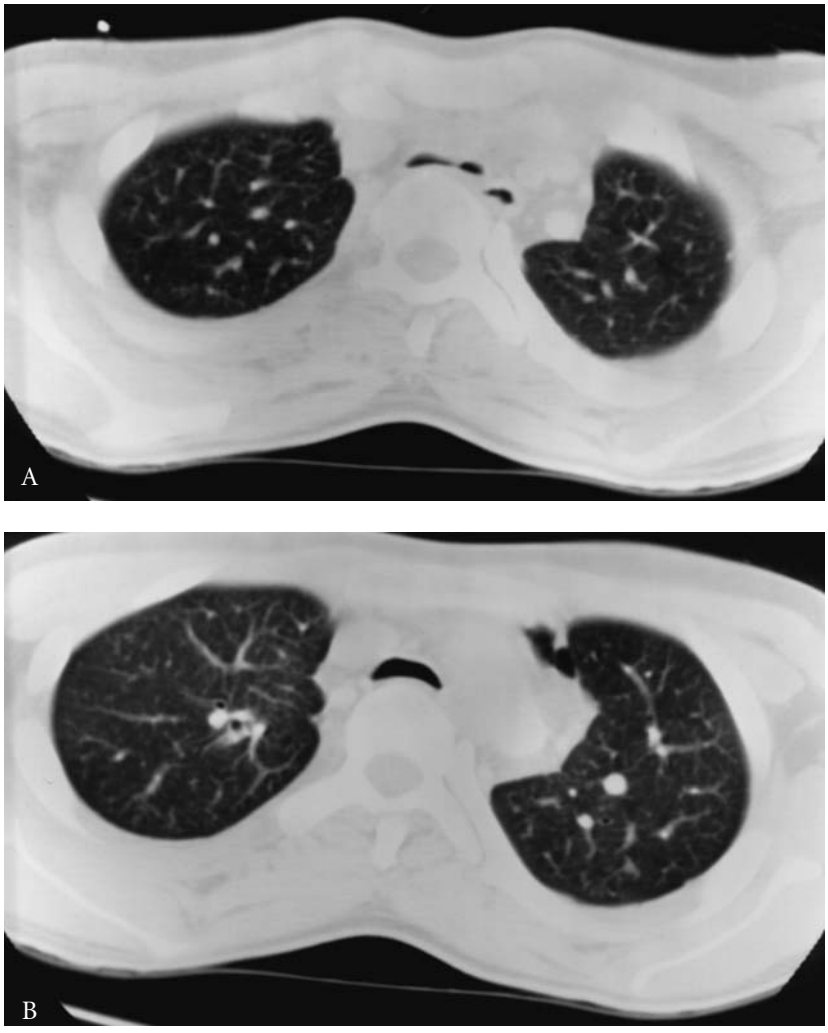


FIGURE **15-22** *Straight back syndrome in a 20-year-old male with extreme dyspnea, transferred intubated.* A, *Computed tomography (CT) scan at the thoracic inlet. The trachea is flattened to a slit between the manubrium sterni (and also by the brachiocephalic artery) and the vertebral column. Haller's "pectus index" (ratio of transverse internal diameter of the thorax to anteroposterior distance between sternum and vertebra, measured on CT scan) is 10.8:1 in this patient. Normal is < 3.25. In Haller's 13 to 18-year-old group of patients selected for pectus repair, the maximum indices were about 5:1 (Haller JA Jr, Kramer SS, Lietman SA. Use of CT scans in selection of patients for pectus excavatum surgery: a preliminary report. J Pediatr Surg 1987;22:904–6).* B, *The distal trachea is splayed against the vertebral column, with a widened membranous wall.*

methacrylate between the bony edges. Dr. John C. Wain, at Massachusetts General Hospital, managed a patient with straight back syndrome and pronounced pectus excavatum deformity, by performing a thorough correction of the pectus problem with osteotomies in both the manubrium and gladiolus. The correction was held in place by an Adkins strut. This successfully opened the trachea, which had been compressed to one-third of its normal cross-sectional area. Mori and colleagues corrected an asymptomatic but severe midtracheal stenosis caused by abnormal ossification behind the top of the manubrium, but in a patient with a vertebral column typical of straight back syndrome.[47] Andrews and colleagues described a narrowed anteroposterior thoracic dimension due to severe pectus excavatum and kyphoscoliosis, which produced "obstructive sleep apnea" in a 5-year-old, due to compression of the distal trachea and left main bronchus.[48] This problem was corrected by pectus repair and aortic suspension. A 15-year-old suffered progressive exercise dyspnea due to tracheal compression by the brachiocephalic artery because of a narrowed thoracic anteroposterior diameter. This was corrected by lateral transplantation of the artery. The curve of the spine was not described in this patient.

Tracheal compromise in similar situations has probably been overlooked in numerous instances. The severity and complexity of the defects vary in the cases cited, which thus far are the only reported instances

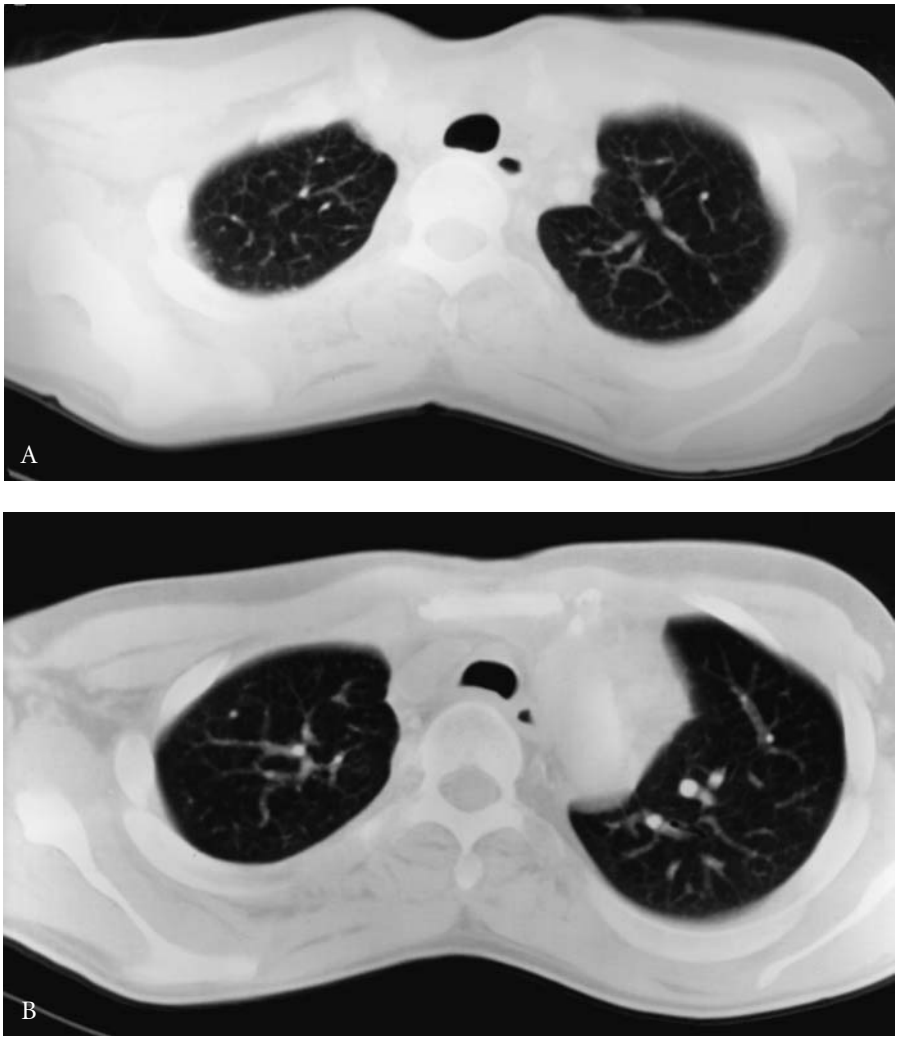


FIGURE **15-23** *Postoperative computed tomography scans in the patient seen in Figure 15-22, showing a very adequate lumen in both the upper* (A) *and lower* (B) *trachea. Note the sternal alteration in* A, *compared with Figure 15-22*A, *and the thickened membranous wall of the trachea in* B, *compared with Figure 15-22*B.

of which I am aware. Precise identification of the points of compression and their causes will guide the design of a corrective procedure along the lines illustrated in these necessarily anecdotal reports.

References

1. Nuutinen J. Acquired tracheobronchomalacia. Eur J Respir Dis 1982;63:380–7.
2. Aquino SL, Shepard J-A O, Ginns LC, et al. Acquired tracheomalacia: CT changes in tracheal lumen between inspiration and expiration measured by semiautomated cross-sectional area calculations. J Comput Assist Tomogr 2001;25:394–9.
3. Herzog H, Heitz M, Keller R, Graedel E. Surgical therapy for expiratory collapse of the trachea and large bronchi. In: Grillo HC, Eschapasse H, editors. International trends in general thoracic surgery. Vol 2. Philadelphia: WB Saunders; 1987. p. 74–90.
4. Herzog H, Nissen R. Erschlaffung und exspiratorische invagination des membranösen teils der intrathorakalen luftröhre und der hauptbrónchien als ursache der asphyktischen anfälle beim asthma bronchiale und bei der chronischen asthmoiden bronchitis des lungenemphysems. Schweiz Med Wochenschr 1954;84:217–9.
5. Herzog H, Rossetti M. Chirurgische möglichkeiten bei komplikationen des lungenemphysems. Thoraxchir 1968;16:99–116.
6. Rainer WG, Newby JP, Kelble DL. Long term results of tracheal support surgery for emphysema. Dis Chest 1968;53:765–72.
7. Halttunen P, Meurala H, Standertskjöld-Nordenstam C-G. New surgical correction of central airway collapse in an asthmatic patient. Scand J Thorac Cardiovasc Surg 1981;15:343–5.
8. Hammoud Z, Grillo HC, Wright CD, Mathisen DJ. Results of membranous wall tracheoplasty for large airway expiratory collapse. [In preparation]

9. Katlic MR, Grillo HC, Wang C-A. Substernal goiter. An analysis of 80 patients from Massachusetts General Hospital. Am J Surg 1985;149:283–7.
10. Katlic MR, Wang CA, Grillo HC. Substernal goiter: a collective review. Ann Thorac Surg 1985;39:391–9.
11. Lahey FH, Hoover WB. Tracheotomy after thyroidectomy. Ann Surg 1951;133:65–76.
12. Green WER, Shepperd HWH, Stevenson HM, Wilson W. Tracheal collapse after thyroidectomy. Br J Surg 1979;66:554–7.
13. Meurala H, Halttunen P, Standertskjöld-Nordenstam C-G, Keskitalo E. Surgical support of collapsing intrathoracic tracheomalacia after thyroidectomy. Acta Chir Scand 1982;148:127–9.
14. Suen HC, Mathisen DJ, Grillo HC, et al. Surgical management and radiological characteristics of bronchogenic cysts. Ann Thorac Surg 1993;55:476–81.
15. Zikri MA, Rice TW. Subcarinal foregut cysts. J Cardiovasc Surg 2000;41:137–41.
16. Fahmy S. Cervical thymic cysts: their pathogenesis and relationship to bronchial cysts. J Laryngol Otol 1974;88:47–60.
17. Graeber GM, Thompson LD, Ronnigen LD, Zajtchuk R. Cystic lesions of the thymus. An occasionally malignant cervical and/or anterior mediastinal mass. J Thorac Cardiovasc Surg 1984;87:295–300.
18. Shields TW. Mesothelial and other less common cysts of the mediastinum. In: Shields TW, editor. General thoracic surgery. 5th ed. Philadelphia: Lippincott, Williams and Wilkins; 2000. p. 2427–31.
19. Hauet EJ, Paul MA, Salm MKM. Compression of the trachea by a mediastinal parathyroid cyst. Ann Thorac Surg 1997;64:581–2.
20. Opsahl T, Berman EJ. Bronchogenic mediastinal cysts in infants: case report and review of the literature. Pediatrics 1962;30:372–7.
21. St. Georges R, Deslauriers J, Duranceau A, et al. Clinical spectrum of bronchogenic cysts of the mediastinum and lung in the adult. Ann Thorac Surg 1991;52:6–13.
22. Ribet ME, Copin MC, Gossselin B. Bronchogenic cysts of the mediastinum. J Thorac Cardiovasc Surg 1995;109:1003–10.
23. Pierson RN, Mathisen DJ. Pedicled pericardial patch repair of a carinal bronchogenic cyst. Ann Thorac Surg 1995;60:1419–21.
24. Katz I, LeVine M, Herman P. Tracheobronchomegaly. The Mounier-Kuhn syndrome. Am J Roentgenol Radium Thorac Nucl Med 1962;88:1084–94.
25. Moretti JA, Miller D. Laryngeal involvement in benign symmetrical lipomatosis. Arch Otolaryngol 1973;97:495–6.
26. Enzi G. Multiple symmetric lipomatosis: an updated clinical report. Medicine 1984;63:56–64.
27. Johns DR. Mitochondrial DNA and disease. N Engl J Med 1995;333:638–46.
28. Boozan JA, Maves MD, Schuller DE. Surgical management of massive benign symmetric lipomatosis. Laryngoscope 1992;102:94–9.
29. Taylor LM, Beahrs OH, Fontana RS. Benign symmetric lipomatosis. Mayo Clin Proc 1961;36:96–100.
30. Birnholz JC, MacMillan AS. Advanced laryngeal compression due to diffuse, symmetric lipomatosis (Madelung's disease). Br J Radiol 1973;46:245–9.
31. Mounier-Kuhn P, Haguenauer JP. Localization laryngée d'une maladie de Launois-Bensaude. J Fr Otorhinolaryngol 1967;16:603.
32. Grillo HC, Shepard J-A O, Mathisen DJ, Kanarek DJ. Postpneumonectomy syndrome: diagnosis, management and results. Ann Thorac Surg 1992;54:638–51.
33. Harrison MR, Hendren WH. Agenesis of the lung complicated by vascular compression and bronchomalacia. J Pediatr Surg 1975;10:813–7.
34. Shamji FM, Deslauriers J, Daniel TM, et al. Postpneumonectomy syndrome with an ipsilateral aortic arch after left pneumonectomy. Ann Thorac Surg 1996;62:1627–31.
35. Boiselle PM, Shepard JO, McLoud TC, et al. Post-pneumonectomy syndrome: another twist. J Thorac Imaging 1997;12:209–11.
36. Shepard JO, Grillo HC, McLoud TC, et al. Right postpneumonectomy syndrome: radiologic findings and CT correlation. Radiology 1986;161:661–4.
37. Kaunitz VH, Fisher FC. Continued pneumothorax in the management of the postpneumonectomy pleural space. Ann Thorac Surg 1966;2:455–63.
38. Powell RW, Luck SR, Raffensperger VG. Pneumonectomy in infants and children: the use of a prosthesis to prevent mediastinal shift and its complications. J Pediatr Surg 1979;14:231–7.
39. Wasserman K, Jamplis RW, Lash H, et al. Postpneumonectomy syndrome: surgical correction using Silastic implants. Chest 1979;75:78–80.
40. Rasch DK, Grover FL, Schnapf BM, et al. Right pneumonectomy syndrome in infancy treated with an expandable prosthesis. Ann Thorac 1990;50:127–9.
41. Jansen JP, Brutel de la Riviere A, Carpentier-Alting MP, et al. Postpneumonectomy syndrome in adulthood. Surgical correction using an expandable prosthesis. Chest 1992;101:1167–70.
42. Valji AM, Maziak DE, Shamji FM, Matzinger FR. Postpneumonectomy syndrome. Recognition and management. Chest 1998;114:1766–9.
43. Nakamura Y, Ohata M, Kawabe K, et al. Left pneumonectomy syndrome successfully treated with endobronchial stent. Intern Med 1998;37:880–3.
44. Kergin FG. An operation for chronic pleural empyema. J Thorac Surg 1953;26:430–4.
45. Davies MK, Mackintosh P, Cayton RM, et al. The straight back syndrome. Quart J Med 1980; 49:443–60.
46. Winter RB, Lovell WW, Moe JH. Excessive thoracic lordosis and loss of pulmonary function in patients with idiopathic scoliosis. J Bone Joint Surg 1975;57:972–7.
47. Mori M, Ikemune K, Hiasa H. Stenosis of the trachea caused by retrosternal ossification. Ann Thorac Surg 2001;71:1035–7.
48. Andrews TM, Myer CM, Gray SP. Abnormalities of the bony thorax causing tracheobronchial compression. Int J Pediatr Otorhinolaryngol 1990;19:139–44.

Bronchial Sleeve Resection

Henning A. Gaissert, MD

History

Surgeons began to develop an interest in preserving normal lung in an era when many parenchymal resections were done for tuberculosis or bronchiectasis and the complications and functional limitations of pneumonectomy became known. Griffith performed one of the first bronchial resections in 1947, for a post-traumatic stricture of the left mainstem bronchus.[1] In the discussion of a paper on experimental bronchial anastomosis, Jackson and colleagues recounted a patient in whom intraoperative separation of the left upper lobe bronchus from the mainstem required circumferential repair.[2]

Sir Clement Price Thomas gave a detailed account of sleeve lobectomy and the clinical observations that led to its development.[3] He entitled his observations "Conservative Resection of the Bronchial Tree" and found it important to introduce the topic with a description of segmental resection. The common denominator, of course, was not only the sparing of functioning normal lung but also the extent of hilar dissection and control required for both procedures. The first publications by Paulson and Shaw,[4,5] Price Thomas, and others described a range of bronchoplastic procedures, among them lateral resection of the bronchus, folding of bronchial flaps, and even dermal grafts.[6] Sleeve lobectomy has endured as a distinct procedure, because the underlying concept, excision of a tube of airway and attached parenchyma with reconstruction by anastomosis, is sound and has been applied successfully to every lobe.

The first sleeve lobectomy was done in 1947 for a "bronchial adenoma" of the right upper lobe in a Royal Air Force cadet. The patient went on to active flying duty, a status he could not have achieved had pneumonectomy been performed. Price Thomas reported the case 8 years later, an inconceivably long interval by today's standards. He and Johnston and Jones[7] gave credit to Philip Allison for the first sleeve lobectomy undertaken for bronchogenic carcinoma in 1952, which also entailed a pulmonary arterial resection yet was never published. Paulson and Shaw reported a variety of bronchoplastic procedures in 16 patients with inflammatory and neoplastic airway disease, without operative mortality; they performed 2 sleeve lobectomies for tuberculous bronchostenosis and 4 for cancer.[4] By 1959, Johnston and Jones had already collected a series of

98 sleeve lobectomies for bronchogenic carcinoma, with an operative mortality of 8%. Two deaths resulted from fistulae between the bronchus and pulmonary artery. In 1991, Newton and colleagues described 27 main bronchial sleeve resections for neoplastic and other lesions with complete pulmonary conservation.[8]

The early contributions on this subject are remarkable for the clarity with which the potential of the operation was recognized and, in retrospect, for how little has been added since. The use of sleeve lobectomy for cancer was encouraged by Nohl-Oser's then-recent studies of lymphatic spread, which established that lower and middle lobe nodal disease was uncommon in upper lobe tumors.[9] The procedure was at first considered solely for patients who, for lack of cardiopulmonary reserve, could not undergo pneumonectomy. However, by 1970, a majority of the sleeve lobectomies that Paulson and colleagues had performed for bronchogenic cancer were in candidates with adequate cardiopulmonary reserve, rather than just in compromised patients.[10] The concept of sleeve resection was also almost immediately applied to resections of the pulmonary artery. Johnston and Jones had 5 patients with combined bronchial and arterial resections.[7] The intervening four decades have added validity to the oncologic value of sleeve lobectomy, provided methods to secure and separate the bronchial anastomosis to prevent fistula formation, extended the concept to more peripheral pulmonary units, and advanced the functional studies of the reimplanted lobe. Time has not changed the basic concept or the indications for this procedure.

Indications

The operation is indicated in bronchogenic cancers and other primary benign or low-grade malignant tumors of the airway that extend to the lobar bronchus or into the adjacent mainstem bronchus. It is also indicated in certain cases of benign stenosis. The term "sleeve lobectomy" refers to resection of a circumferential sleeve of mainstem bronchus contiguous with a pulmonary lobe, whereas "bronchial sleeve resection" is used to describe excision of the airway with sparing of parenchyma. Bronchogenic carcinomas usually predominate when reported with other lesions in collective series of sleeve lobectomy. Lowe and colleagues found 20 patients with lung cancer and 7 patients with other airway tumors.[11] Among 104 patients treated by Watanabe and colleagues, there were 89 with lung cancer, 8 with benign or low-grade airway tumors, and 7 with inflammatory strictures.[12] Suen and colleagues noted 58 lung cancers and 19 low-grade bronchial malignancies among 77 patients undergoing sleeve resections.[13] In an earlier report from Massachusetts General Hospital (MGH), Frist and colleagues reported a more balanced distribution between bronchogenic carcinoma in 33 patients and other indications in 31 patients; among the latter were 24 other airway tumors and 7 benign strictures.[14]

Lung Cancer

Ever since its introduction, sleeve lobectomy has made an important impact on the surgical treatment of lung cancer. When considering surgical options and prognosis, it is useful to distinguish four anatomic situations (as detailed in Table 16-1) in two groups of patients. The first and classic anatomic situation is a tumor found on bronchoscopy to arise in a lobar bronchus so as to preclude standard lobectomy. The second situation is where a carcinoma extrinsic to the airway may extend to the lobar bronchus. In the third situation, the bronchial margin may be found on frozen section to contain tumor. Finally, the fourth is

Table 16-1 Relative Distribution of Anatomic Indications for Sleeve Lobectomy in Bronchogenic Carcinoma

First Author, Year	*Number of Patients*	*Endobronchial Tumor (%)*	*Extraluminal Extension (%)*	*Involved Margin (%)*	*Lymph Node Adherence (%)*
Gaissert,[15] 1996	72	75.0	8.3	5.5	11.1
Tronc,[16] 2000	175	38.6	13.6	26.6	13.0

where a lymph node with metastasis may adhere to the confluence of lobar and main bronchus and thus dictate resection of a sleeve of airway. Conversely, a metastatic node without adherence to the bronchus is not currently considered an indication for sleeve lobectomy. Table 16-1 lists the relative distribution of patients in two recent series among these anatomic situations. Among 72 patients in the MGH series, 6 patients had central tumors that extended into the bronchial wall and none survived for more than 2 years, suggesting in this limited experience that patients with adequate cardiopulmonary reserve and tumor permeation should undergo pneumonectomy.[15] Most patients require upper lobe sleeves and only a minority require lower lobe resections (Table 16-2).

At first conceived for patients unable to tolerate pneumonectomy, sleeve lobectomy has rapidly become an option for patients suitable for the more radical procedure. The former group of patients has been termed "compromised," whereas the latter group with adequate cardiopulmonary reserve is named "deliberate" or "elective." Table 16-2 lists the composition of recent surgical series according to the degree of cardiopulmonary reserve and tumor histology. The evidence for the oncologic equivalence of sleeve lobectomy and pneumonectomy has remained circumstantial and is based on retrospective comparison of heterogeneous groups.[13,15–21] However, there is now a broad clinical consensus to perform elective sleeve resection rather than pneumonectomy, provided that a complete resection is performed. According to Weisel and colleagues, in patients with uncompromised lung function, "…ipsilateral, intranodal metastases at the tracheobronchial angle are not a contraindication to upper sleeve lobectomy. However, nodal metastases in the major fissure constitute a contraindication to sleeve resection."[19] The success of elective sleeve lobectomy, and the prognosis of the individual patient, would seem to depend greatly on the intraoperative diligence of the surgeon in assessing the extent of disease.

Benign or Low-Grade Malignant Tumors of the Airway

A variety of primary airway tumors may necessitate sleeve lobectomy by their location within the airway. Table 16-3 lists the diagnosis of 99 benign or low-grade malignant airway lesions treated at the MGH.[22] Half of these required sleeve lobectomy, the other half needed bronchial sleeve resection. Patients ranged in age from 3 to 68 years, with a mean age of 42 years. Only 3 of 54 carcinoid tumors had lymph node involvement, involving a single node in each instance.

Table 16-2 Cardiopulmonary Reserve and Histology in Recent Surgical Series

First Author, Year	Number of Patients	Share (%) of all Resections for Lung Cancer	Cardiopulmonary Reserve		Histology			% Lower Lobe Resections
			Adequate (%)	Compromised (%)	Squamous (%)	Adeno (%)	Others (%)	
Watanabe,[12]* 1990	72	9.2			87.6	6.7	5.6	19
Gaissert,[15] 1996	72		51	49	68	26	6	14
Tronc,[16]* 2000	175				71.4	10.8	17.7	4.9
Okada,[17]* 2000	151	12.6			60	35	6	
Weisel,[19] 1979	70	7	61	39	88.5	11.4	0	11
Faber,[20] 1984	101				75	8.0	17	16
Icard,[21] 1999	110		59	41	86	9	4.5	13

Data in Okada and colleagues[17] were derived from 60 procedures paired with pneumonectomy. Icard and colleagues[21] performed a large wedge resection in 83% of their patients, leaving a small part of the bronchial wall intact.

References denoted with an asterisk included a small number of lesions other than bronchogenic carcinoma or procedures other than sleeve lobectomy.

Table 16-3 Pathology of 99 Low-Grade Malignant or Benign Bronchial Lesions Resected by Bronchial Sleeve Resection With (n = 49) or Without Lobectomy

Pathologic Type	*Number of Cases*
Low-Grade Malignancy (70%)	
Carcinoid	54
Mucoepidermoid carcinoma	7
Fibrous histiocytoma	5
Adenoid cystic carcinoma	2
Granular cell tumor	1
Hemangiopericytoma	1
Benign Masses (8%)	
Polypoid glandular neoplasm	1
Inflammatory pseudotumor	1
Lymphoid aggregate	1
Mucus gland cystadenoma	2
Hamartoma	3
Stenosis (22%)	
Histoplasmosis	4
Inflammatory	3
Idiopathic	2
Post-traumatic	6
Postoperative	7

Adapted from Bueno R et al.[22]

Benign Strictures

Inflammatory strictures requiring resection of the lung and adjacent main bronchus are rare and are almost always caused by tuberculosis (TB). Resection cannot be recommended in the presence of active TB or when active disease remains after resection. The adverse outcome under such circumstances is illustrated by Price Thomas's first patient to undergo a bronchoplastic procedure for benign disease.[3] This patient, who had a stricture of the distal mainstem bronchus and diffuse lobar disease, continued to have positive sputum and required a completion pneumonectomy 6 months later. Pharmacologic control has led to virtual disappearance of bronchial TB in the United States and Western Europe, and series of benign bronchial strictures now originate from regions where TB remains endemic. Kato and colleagues reported 36 patients with tuberculous airway stenosis over a 36-year period.[23] As shown in Figure 16-1, 13 patients had left upper sleeve lobectomy, 12 patients had sleeve resection of the left main bronchus (of whom 2 underwent concomitant left upper lobectomy), 5 patients had right upper sleeve lobectomy, 2 patients had sleeve resection of the right intermediate bronchus, and each of the remaining 4 patients had, respectively, right sleeve superior segmentectomy of the lower lobe, sleeve resection of the trachea with concomitant left pneumonectomy, carinal resection with right upper sleeve lobectomy and middle lobectomy, and dilatation of the left main bronchus with a free skin graft reinforced with a steel wire. In another Japanese series, Watanabe and colleagues saw 19 benign strictures over a 21-year period.[12] A histologic diagnosis of TB was made in 7, whereas the diagnosis was clinical in the others due to the typical location of the stricture. Of 12 patients undergoing surgical therapy, 4 underwent right upper and 3 had left upper sleeve lobectomy.

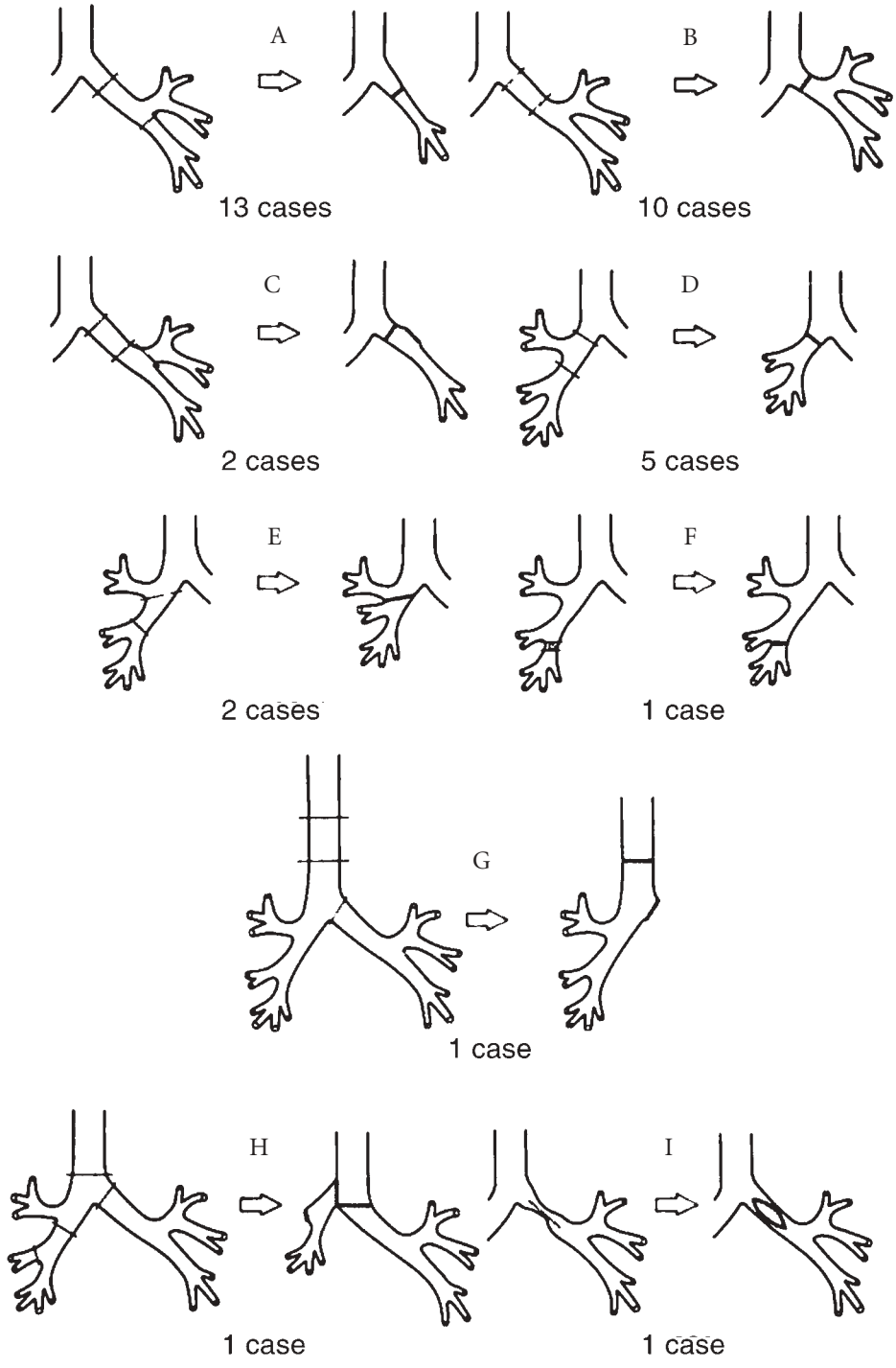


FIGURE **16-1** *Extent of airway resection and reconstruction in 36 patients with tuberculous bronchostenosis, as reported by Kato and colleagues.[23] Reprinted with permission from Kato R et al.[23]*

Bronchial disruptions may rarely require sleeve lobectomy.[24] Most injuries respond to repair by simple closure. Two of the 6 late post-traumatic stenoses reported by Bueno and colleagues (see Table 16-3) required bronchial sleeve resection with or without sleeve lobectomy after failed repair elsewhere, whereas

4 developed a delayed stenosis.[22] Benfield has emphasized the importance of involving trained thoracic surgeons in the care of these patients, and to recognize and repair the injury early with minimal loss of lung.[25]

Bronchial Resection in Children

Resection in children is performed for rare endobronchial tumors.[26] Six of 12 patients undergoing sleeve resection of the lower trachea or bronchi at MGH had main bronchial resections, with sleeve lobectomy in 3 of them.[27]

Preoperative Evaluation

As in other patients with lung cancer, standard evaluation begins with an accurate history and physical examination. Radiographic evaluation includes a chest radiograph and computerized tomography. Magnetic resonance imaging is not routinely obtained to determine extent of airway involvement, but may provide useful information on vascular invasion. Further radiographic staging examinations are obtained depending on individual circumstances. I perform cervical mediastinoscopy routinely for lung cancer, with selective addition of anterior mediastinotomy in patients with left-sided upper lobe or central tumors.

The functional evaluation is no different from other patients undergoing pulmonary resection. The predicted postoperative lung function may be calculated from preoperative pulmonary function studies and ventilation-perfusion scintigraphy to establish whether an individual patient would tolerate a lobectomy or a pneumonectomy (see "Postoperative Function below").[15,28,29]

In their report on tuberculous bronchostenosis, Kato and colleagues emphasized the importance of bronchoscopy to rule out active disease before a decision is made to perform resection.[23] They further recommended TB drug therapy for at least 6 months before resection, even when stenosis was only thought to be due to TB.

The need for resection of the pulmonary artery in carcinomas of the upper lobes is usually established at the time of thoracotomy, although a pulmonary angiogram[30] or magnetic resonance imaging may give information about arterial involvement.

Technique

The operative technique for bronchial and arterial sleeve resection is detailed in Chapter 30, "Main and Lobar Bronchoplasty." The standard approach is through a posterolateral thoracotomy. A lateral thoracotomy through a vertical incision along the anterior border of the latissimus dorsi muscle is the author's preference. The chest is explored and nodal sampling performed in lung cancer or carcinoid tumor to assess the extent of metastasis. If sleeve lobectomy is planned in a patient who would tolerate a pneumonectomy, a high level of confidence should exist about the absence of metastatic nodal disease. Such confidence requires sampling and intraoperative frozen section of abnormal and representative lymph nodes from the fissure and along the bronchus of the lobe to be preserved. To this point, the thoracic procedure is identical to a standard lobectomy.

The bronchus is isolated and divided. In tumors, this occurs with a single sharp incision through normal tissue. For tuberculous bronchostenosis, Kato and colleagues recommend transection close to or at the stenotic segment, with additional resection after examination of the bronchial lumen from the inside.[23] Kato and colleagues state that the affected bronchus should have no residual malacia and emphasizes that "excessive tension at the anastomosis caused by extensive resection would do more harm than would a slight remaining stenosis." In neoplastic disease, frozen section analysis is obtained by sending a thin ring of tissue from the two ends of airway to be anastomosed. No attempt is made to tailor either end of the bronchus. Figure 16-2 describes the extent of pulmonary arterial resection in 14 patients reported by Ricci and colleagues.[31]

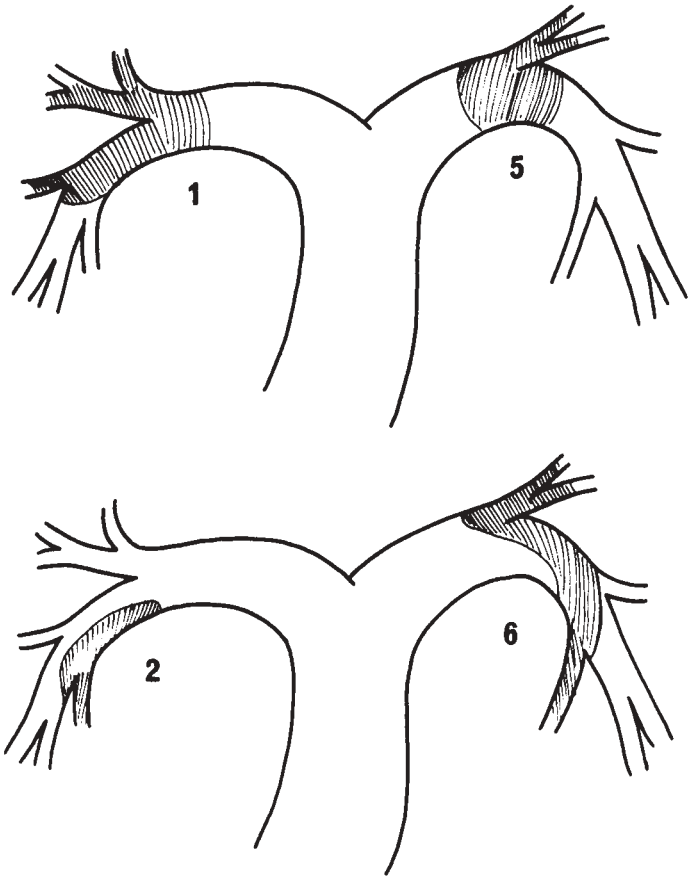


FIGURE **16-2** *Resection of the pulmonary artery in 14 patients with extrapericardial vascular involvement, as performed by Ricci and colleagues.[31] Panel* A *denotes sleeve resections, and panel* B *shows partial resections reconstructed with a pericardial patch. The numbers indicate the number of patients with each type of resection. Reprinted with permission from Ricci C et al.[31]*

Postoperative Care

The elements of care for patients after a sleeve lobectomy closely resemble that after standard lung resection. Careful attention should be given to the relief of postoperative pain, early ambulation, and relative fluid restriction supplement measures to encourage deep breathing and mobilization of respiratory secretions.

Postoperative attention to the anastomosis varies in the reported series according to the bias of the individual surgeon. It is good practice to view the anastomosis by bronchoscopy at the time of surgery, preferably before closure of the chest when corrective action can be taken, and again before discharge of the patient from the hospital. Such a liberal use of bronchoscopy identifies not only many normal anastomoses, but also the rare and unexpected problem that requires closer follow-up. Even when bronchoscopy is performed only selectively, it must be done when obvious problems are found, that is, atelectasis, pneumothorax, unexplained fever, or hypoxia, to rule out stricture and bronchial dehiscence.

A stricture may be observed when pneumonia and sepsis are absent. Early narrowing of the anastomosis may be caused by mucosal swelling, which should improve with time. Bronchial stenosis may not be obvious during the initial hospitalization, and continued attention to new symptoms should extend over the first 3 months after operation. New onset of wheezing, pneumonia, or atelectasis in the reimplanted lobe should prompt a bronchoscopic examination. Strictures at the anastomosis that arise beyond 3 months may represent a local recurrence; a biopsy should be performed. Dilatation of the anastomosis is best delayed for the first 3 weeks after the operation, but secretions should be removed aggressively and, if necessary, repeatedly. A stricture that recurs after dilatation may require placement of a bronchial stent or a surgical attempt to reconstruct the anastomosis.

When a dehiscence is discovered, its extent should be quantified. A partial dehiscence may be observed; however, an accompanying pneumothorax should be drained with a chest tube and every effort should be made to achieve complete expansion of the lung. If a fistula is large and is detected early, one may try to reconstruct the anastomosis. When discovered late or if the reimplanted lung is septic, completion pneumonectomy may need to be performed.

Pre- or postoperative steroids are generally regarded as contraindicated in resections of the airway, because of adverse effects of bronchial healing observed in tracheal surgery and lung transplantation. This reluctance to use steroids is not shared by Rendina and colleagues, who performed a randomized study of low-dose intra- and postoperative steroids in patients undergoing bronchial sleeve resection.[32] Their regimen consisted of methylprednisolone, given at 10 mg intravenously during surgery and daily for up to 10 days, intraoperative bronchial lavage with hydrocortisone succinate at 250 mg, and daily inhalations of 5 mg of methylprednisolone. They found that healing of the bronchial anastomosis in the low-dose steroid group occurred without edema or ulceration, although there was 1 dehiscence and 1 granuloma in the patients treated with placebo. Thus, healing was not impaired, and perhaps improved, by use of *low-dose* corticosteroids in airway anastomoses exposed to a *low* degree of tension.

Operative Results

The operative mortality of sleeve lobectomy consistently resembles that of standard lobectomy and is notably lower than that of pneumonectomy. This experience, repeated in virtually every retrospective comparison, supports a preference for bronchoplastic procedures over pneumonectomy. In their review of bronchoplastic operations reported over the 12-year period preceding 1990, Tedder and colleagues summarized results in 1,915 patients.[33] They found a 30-day mortality of 7.5%; the four most common causes of death were respiratory failure, cardiac events, pneumonia, and pulmonary embolus. Since then, the operative mortality at centers experienced in sleeve resections has declined further, along with that of standard resections, due to improvements in patient selection and perioperative care. Complications specific to the bronchial anastomosis are reduced due to the availability of absorbable suture material that causes minimal tissue reaction. The existing difference in the early results of patients with adequate and those with compromised lung function is expected to persist, although the mortality of patients with cardiopulmonary limitations has declined as well.

Table 16-4 summarizes operative mortality, morbidity, and bronchial complications in seven modern series. The reports by Weisel and Faber and their colleagues cover the early experience with sleeve lobectomy when silk, steel, Tevdek, or chromic catgut were used at the anastomosis.[19,20] Some of these patients also received preoperative radiotherapy with deleterious effects on bronchial healing. According to current knowledge, preoperative radiotherapy is only indicated for selected patients with confirmed ipsilateral mediastinal (N2) lymph node involvement. Few patients (less than 10% in most series) undergo sleeve lobectomy for stage IIIA disease. In these cases, careful protection of the bronchial anastomosis with an omental flap should be considered, both to separate the bronchus from the pulmonary artery and to promote capillary growth into the bronchus.

Bronchovascular and bronchopleural fistulae should be rare when using a circumferential wrap of pedicled pericardial fat or pleura around the anastomosis. Kutlu and Goldstraw reported on 100 consecutive tracheobronchial anastomoses performed with a continuous Prolene suture (Ethicon Inc., Somerville, NJ) without tissue coverage.[34] There were 3 partial anastomotic dehiscences, with death from a bronchoatrial fistula in 1 patient and completion pneumonectomy in another. Late bronchial stenosis occurred in 3 of 66 sleeve lobectomies. Thus, good anastomotic technique in the absence of tissue coverage may prevent the disaster of a communication between the airway and vascular structures in most patients.

Table 16-4 Operative Mortality and Morbidity with Rate of Complications Specific to Sleeve Lobectomy

First Author, Year	Operative Mortality (%)	Major Complications (%)	Bronchopleural Fistula (n / %)	Bronchovascular Fistula (n / %)	Bronchial Stenosis (n / %)
Watanabe,[12*] 1990	1.3	2.5	0	0	1 / 1.3
Gaissert,[15] 1996	4.2	11	1 / 1.4	0	2 / 2.8
Tronc,[16*] 2000	1.6	14.8	2 / 1.1	0	4 / 2.3
Okada,[17] 2000	0	13			2 / 3.0
Weisel,[19] 1979	11.4	11.4	3 / 4.3	5 / 7.1	
Faber,[20] 1984	2.0		6 / 5.9	1 / 0.99	18 / 17.8
Icard,[21] 1999	2.7	14.5	4 / 3.6	1 / 0.9	4 / 3.6

Data in Okada and colleagues[17] were derived from 60 procedures paired with pneumonectomy.

References denoted with an asterisk included a small number of lesions other than bronchogenic carcinoma or procedures other than sleeve lobectomy.

Postoperative Function

Experimental Data

The early clinical experience had not provided sufficient information as to how much the reimplanted lung contributes to overall lung function, particularly in the early postoperative period. Yet, sleeve lobectomy is frequently used in patients with a marginal lung reserve, who are sensitive to even temporary loss of function or an important pulmonary arteriovenous shunt. Wood and colleagues compared the standard right upper lobectomy to a sleeve lobectomy in dogs, using differential bronchospirometry, and found that oxygen uptake dropped by an additional 22% immediately after sleeve lobectomy and was maximally depressed after 3 days.[35] This difference from standard lobectomy resolved 4 weeks after the procedure. Postoperative ventilation scintigraphy showed a comparable decrease for standard and sleeve resections, but the perfusion scans suggested that the impaired oxygen uptake of reimplanted lobes was due to a transient abnormality in perfusion. The authors concluded that temporary dysfunction after sleeve lobectomy places patients with impaired lung function at increased risk of respiratory failure compared to simple lobectomy. These findings may also explain the unilateral pulmonary edema occasionally observed in the reimplanted lobe.

Sputum retention and atelectasis are not uncommon after sleeve lobectomy. Paul and colleagues found no difference of mucociliary drainage or bronchial mucus consistency in dogs after sleeve lobectomy, whereas auto- and allotransplantation of the lung delayed clearance and increased mucus viscosity.[36] Light microscopy showed relative disappearance of bronchial glands and squamous metaplasia peripheral to the anastomosis in all three groups. These findings support the general attitude that patients who undergo sleeve resection should not require any additional preventive measures for sputum retention.

Clinical Data

Observation of intermediate results after the procedure has confirmed the experimental data. Deslauriers and colleagues confirmed in 19 patients that the reimplanted lung contributed substantially to overall lung function.[37] In 15 patients after right bronchoplasty, the right lung received 41% of perfusion, whereas the left lung in 4 patients after left bronchoplasty captured 29% of perfusion. Figures 16-3 and 16-4 describe the forced vital capacity (FVC) and postoperative scintigraphy in their patients. Brusasco and colleagues also studied patients with spirometry and ventilation-perfusion scintigraphy.[38] Two weeks after sleeve lobectomy, they found a slightly, but significantly higher forced expiratory volume in 1 second (FEV_1) compared with standard lobectomy. Over 3 to 12 months, they found significant improvements of

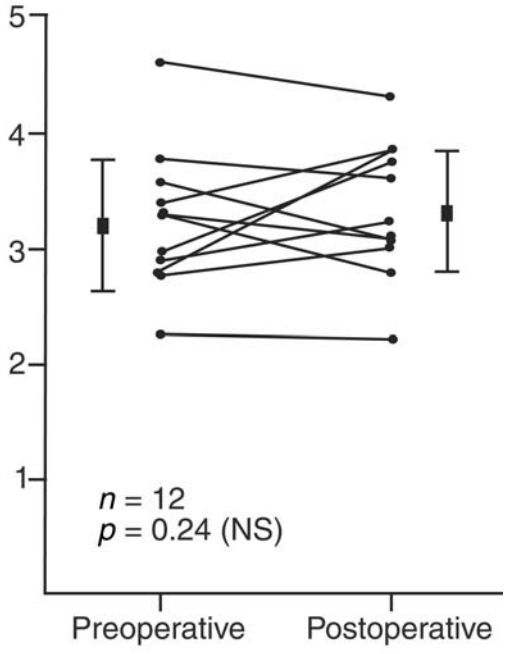


FIGURE **16-3** *Individual and mean (± standard deviation) forced vital capacity (FVC) before and after sleeve lobectomy in 12 patients with pre- and postoperative spirometry, as reported by Deslauriers and colleagues.[37] NS = not significant. Reprinted with permission from Deslauriers J et al.[37]*

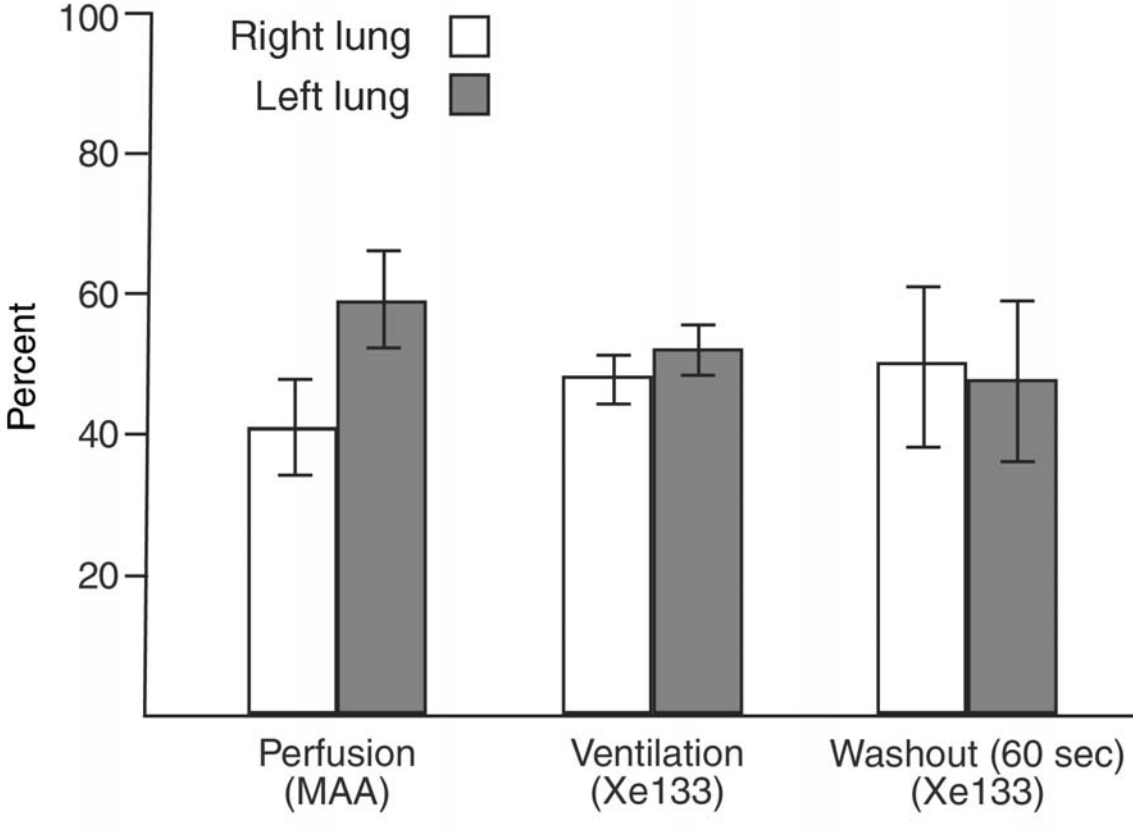


FIGURE **16-4** *Results (mean ± standard deviation) for perfusion, ventilation, and washout curves for all 15 patients after right lung sleeve lobectomy, as reported by Deslauriers and colleagues.[37] MAA = macroaggregated albumin; Xe133 = ^{133}Xenon. Reprinted with permission from Deslauriers J et al.[37]*

regional ventilation and perfusion in both types of lobectomy, with a higher regional ventilation after sleeve lobectomy.

Postoperative lung function may be predicted by calculation of the expected functional loss using the following equation:

$$\text{preoperative } FEV_1 \times \frac{\text{(number of functional segments in the lobe to be resected)}}{\text{(total number of segments in both lungs)}}$$

Khargi and colleagues noted a good correlation between predicted postoperative FEV_1 and measured postoperative FEV_1 in 109 patients who had spirometry 25 to 342 days after sleeve lobectomy.[29] Thus, postoperative FEV_1, although not necessarily the functional outcome, may be reliably predicted in every patient from preoperative pulmonary function tests and from ventilation and perfusion scintigraphy.

In a group of 52 patients with resection of the pulmonary artery, as reported by Rendina and colleagues, the mean FEV and FVC were, respectively, 72% and 80% before operation and 65% and 76% at 1 month after surgery, and then reached their plateau at 70% and 78% after 6 months.[39] Echocardiography showed patterns in the normal range and normal estimates of pulmonary artery (PA) pressures in all but 2 patients. These values indicate that lobectomy with PA resection may be followed by limited loss of spirometric lung function.

Long-Term Results

Lung Cancer

For patients with a nonsmall cell carcinoma surviving resection, long-term survival appears to be related to the disease stage but not to the type of resection. In retrospective comparisons adjusted for stage, there is no important difference in survival between sleeve lobectomy and pneumonectomy. In a paired comparison between the two procedures, by nearest available matching of patients, Okada and colleagues found significantly longer survival at 3-, 5-, and 10-year intervals after sleeve lobectomy, with no difference in local recurrence.[17] They pursue bronchoplastic procedures aggressively and advocate extended sleeve resections to avoid pneumonectomy.[40] Their comparison, although carefully performed, points to a weakness of any retrospective attempt to demonstrate superior results of sleeve lobectomy. In favoring sleeve resection, their pool of pneumonectomy patients contains fewer and presumably more advanced tumors.

Following sleeve lobectomy, surgical resection results in 5-year survival of 36 to 79% in stage I, and 23 to 55% in stage II (Table 16-5).[41] In the MGH series, a few patients had chest wall involvement.[15] However, because T3 or T4 tumors are rarely treated with bronchial sleeve resection (as opposed to carinal resection), some authors distinguish between N0 and N1 disease, with comparable results, as noted in Table 16-5. The numbers for patients with stage IIIA or N2 disease are too small to draw conclusions about the value of sleeve resection or the effectiveness of adjuvant therapy. The MGH series showed no difference in survival at 5 years between 48 patients undergoing upper lobectomy (46 ± 8.5%) and in 13 patients after middle and lower lobectomy (52 ± 14%).

Okada and colleagues performed extended resections in 15 of 157 patients undergoing sleeve lobectomy, ranging from resection of the upper lobe and superior segment to resection of the lower lobe and lingula (Figure 16-5).[40] Eight of their 15 patients underwent an arterial resection. After 12 to 106 months, all the patients with stage IIB disease and half of the patients with stage IIIA disease were alive without recurrence, although the other half of stage IIIA patients died of distant metastases within 1 year. The reimplanted lung contributed 26 ± 18% of overall function.

Table 16-5 Late Results and Local Recurrence after Sleeve Lobectomy for Lung Cancer

First Author, Year	*% Patients undergoing Mediastinoscopy*	*Overall 5-Year Survival (%)*	*5-Year Survival by Nodal Status (%)*			*5-Year Survival by Stage (%)*			*Anastomotic Recurrence (%)*	*Local Recurrence (%)*	*Second Primary Lung Cancer (%)*
			N0	N1	N2	I	II	IIIA			
Watanabe,[12]* 1990					17	79	55	30			2.5
Gaissert,[15] 1996	89	42	57	38					1.4	14	
Tronc,[16]* 2000	87	52	63	48	8.0					22	12‡
Okada,[18] 2000		48			21					8.0	
Weisel,[19] 1979	94	37 / 20				43	31	22			11
Faber,[20] 1984		30				36†	23		8.9		
Icard,[21] 1999		39	57	29	33	60	30	27		34	12

Data in Okada and colleagues[17] were derived from 60 procedures paired with pneumonectomy.

References denoted with an asterisk included a small number of lesions other than bronchogenic carcinoma or procedures other than sleeve lobectomy.

†Includes 10 patients with T1N1 which are now grouped in stage II.

‡Quoted in earlier report from the same group.[41]

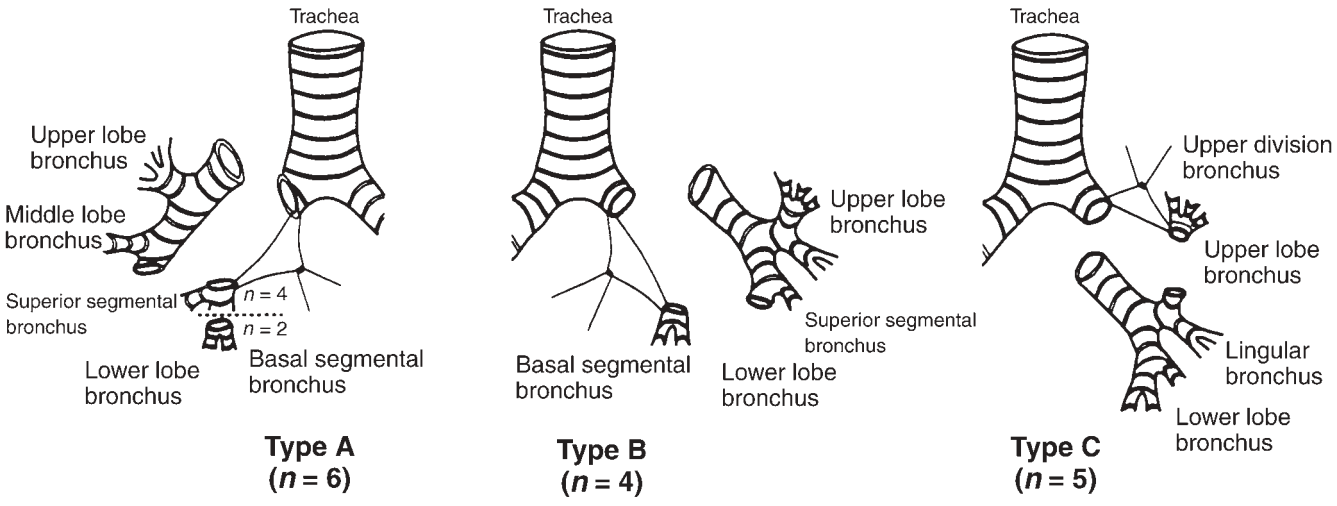


FIGURE **16-5** *Reconstruction in 15 patients undergoing atypical extended sleeve lobectomy, classified by Okada and colleagues[40] into types A to C according to the mode of reconstruction. Reprinted with permission from Okada M et al.[40]*

Pulmonary Arterial Resection

Late results after resection of the pulmonary artery alone or in combination with a bronchial sleeve in some series have been disappointing and fostered criticism regarding the oncologic value of arterial resection. In 108 patients with stages I and II undergoing broncho- and angioplastic operations, Vogt-Moykopf and colleagues noted a 5-year survival of 35%.[42] In their most recent report, Rendina and colleagues noted a 5-year survival of 38.3% for the entire group, 64.4% for stages I and II, and 18.6% for stages IIIA and IIIB.[39] These latter outcomes are comparable to survival in patients without PA resection, and are expected to support a more widespread use of arterial sleeve resection.

Low-Grade Bronchial Malignancy

Complete resection of carcinoid tumors by sleeve resection, as in conventional resection, is followed by excellent long-term survival.[22] In the quoted report, one patient had recurrent adenoid cystic carcinoma, whereas none of the other patients developed recurrence.[22]

Benign Strictures

Long-term survival after resection of a benign stenosis depends on control of the underlying disease, and is in general excellent. Kato and colleagues achieved an operative mortality of 5.6% (2 of 36 cases) after resection of tuberculous bronchostenosis.[23] They used an anti-TB regimen for 6 to 12 months after resection and found restenosis in 2 patients after right upper sleeve lobectomy and in another patient after left upper resection. A total of 7 of their 36 patients had postoperative narrowing and 1 underwent a reoperation.

Among the 22 benign stenoses reported in the series of Bueno and colleagues, 1 patient died of progressive idiopathic airway stenosis and there was no recurrence in the others.[22]

References

1. Griffith JL. Fracture of the bronchus. Thorax 1949;4:105–9.
2. Jackson TL, Lefkin P, Tuttle W, Hampton F. An experimental study in bronchial anastomosis. J Thorac Surg 1949;18:630–41.
3. Price Thomas C. Conservative resection of the bronchial tree. J R Coll Surg Edin 1956;1:169–86.
4. Paulson DL, Shaw RR. Bronchial anastomosis and bronchoplastic procedures in the interest of preservation of lung tissue. J Thorac Surg 1955;29:238–59.
5. Paulson DL, Shaw RR. Preservation of lung tissue by means of bronchoplastic procedures. Am J Surg 1955;89:347–55.

6. Gebauer P. Pulmonary surgery salvage by bronchial resection. Surg Gynec Obstet 1952;94:347–57.
7. Johnston JB, Jones PH. The treatment of bronchial carcinoma by lobectomy and sleeve resection of the main bronchus. Thorax 1959;14:48–54.
8. Newton JR, Grillo HC, Mathisen DJ. Main bronchial sleeve resection with pulmonary conservation. Ann Thorac Surg 1991;52:1272–80.
9. Nohl-Oser HC. An investigation into the lymphatic and vascular spread of carcinoma of the bronchus. Thorax 1956;11:172–85.
10. Paulson DL, Urschel HC, McNamara JJ, Shaw RR. Bronchoplastic procedures for bronchogenic carcinoma. J Thorac Cardiovasc Surg 1970;59:38–48.
11. Lowe JE, Bridgman AH, Sabiston DC. The role of bronchoplastic procedures in the surgical management of benign and malignant pulmonary lesions. J Thorac Cardiovasc Surg 1982;83:227–34.
12. Watanabe Y, Shimizu J, Oda M, et al. Results in 104 patients undergoing bronchoplastic procedures for bronchial lesions. Ann Thorac Surg 1990;50:607–14.
13. Suen HC, Meyers BF, Guthrie T, et al. Favorable results after sleeve lobectomy or bronchoplasty for bronchial malignancies. Ann Thorac Surg 1999;67:1557–62.
14. Frist WH, Mathisen DJ, Hilgenberg AD, Grillo HC. Bronchial sleeve resection with and without pulmonary resection. J Thorac Cardiovasc Surg 1987;93:350–7.
15. Gaissert HA, Mathisen DJ, Moncure AC, et al. Survival and function after sleeve lobectomy for lung cancer. J Thorac Cardiovasc Surg 1996;111:948–53.
16. Tronc F, Grégoire J, Rouleau J, Deslauriers J. Long-term results of sleeve lobectomy for lung cancer. Eur J Cardiothorac Surg 2000;17:550–6.
17. Okada M, Yamagishi H, Satake H, et al. Survival related to lymph node involvement in lung cancer after sleeve lobectomy compared with pneumonectomy. J Thorac Cardiovasc Surg 2000;119:814–9.
18. Yoshino I, Yokoyama H, Ueda T, et al. Comparison of the surgical results of lobectomy with bronchoplasty and pneumonectomy for lung cancer. J Surg Oncol 1997;64:32–5.
19. Weisel RD, Cooper JD, Delarue NC, et al. Sleeve lobectomy for carcinoma of the lung. J Thorac Cardiovasc Surg 1979;78:839–49.
20. Faber LP, Jensik RJ, Kittle CF. Results of sleeve lobectomy for bronchogenic carcinoma in 101 patients. Ann Thorac Surg 1984;37:279–85.
21. Icard P, Regnard JF, Guibert L, et al. Survival and prognostic factors in patients undergoing parenchymal saving bronchoplastic operations for primary lung cancer: a series of 110 consecutive cases. Eur J Cardiothorac Surg 1999;15:426–32.
22. Bueno R, Wain JC, Wright CD, et al. Bronchoplasty in the management of low-grade airway neoplasms and benign bronchial stenosis. Ann Thorac Surg 1996;62:824–9.
23. Kato R, Kakizaki T, Hangai N, et al. Bronchoplastic procedures for tuberculous bronchial stenosis. J Thorac Cardiovasc Surg 1993;106:1118–21.
24. Baumgartner F, Sheppard B, de Virgilio C, et al. Tracheal and main bronchial disruptions after blunt chest trauma: presentation and management. Ann Thorac Surg 1990;50:569–74.
25. Benfield JR. Traumatic bronchial rupture and other major thoracic injuries [editorial]. Ann Thorac Surg 1990;50:523.
26. Black CT, Luck SR, Raffensperger JG. Bronchoplastic techniques for pediatric lung salvage. J Pediatr Surg 1988;23:653–6.
27. Gaissert HA, Mathisen DJ, Grillo HC, et al. Tracheobronchial sleeve resection in children and adolescents. J Pediatr Surg 1994;29:192–8.
28. Bria WF, Kanarek DJ, Kazemi H. Prediction of postoperative pulmonary function following thoracic operations. Value of ventilation-perfusion scanning. J Thorac Cardiovasc Surg 1983;86:186–92.
29. Khargi K, Duurkens VA, Verzijlbergen FF, et al. Pulmonary function after sleeve lobectomy. Ann Thorac Surg 1994;57:1302–4.
30. Suri RM, Keshavjee SH, Herman S, et al. Pulmonary arteriography for the assessment of technical feasibility of sleeve resection in lung cancer. Ann Thorac Surg 1997;63:800–5.
31. Ricci C, Rendina EA, Venuta F, et al. Reconstruction of the pulmonary artery in patients with lung cancer. Ann Thorac Surg 1994;57:627–33.
32. Rendina E, Venuta F, Ricci C. Effects of low-dose steroids on bronchial healing after sleeve resection. J Thorac Cardiovasc Surg 1992;104:888–91.
33. Tedder M, Anstadt MP, Tedder SD, Lowe JE. Current morbidity, mortality, and survival after bronchoplastic procedures for malignancy. Ann Thorac Surg 1992;54:387–91.
34. Kutlu CA, Goldstraw P. Tracheobronchial sleeve resection with the use of a continuous anastomosis: results of one hundred consecutive cases. J Thorac Cardiovasc Surg 1999;117:1112–7.
35. Wood PB, Gilday D, Ilves R, et al. A comparison of gas exchange after simple lobectomy and lobectomy with sleeve lobectomy in dogs. J Thorac Cardiovasc Surg 1974;68:646–53.
36. Paul A, Marelli D, Shennib H, et al. Mucociliary function in autotransplanted, allotransplanted, and sleeve resected lungs. J Thorac Cardiovasc Surg 1989; 98:523–8.
37. Deslauriers J, Gaulin P, Beaulieu M, et al. Long-term clinical and functional results of sleeve lobectomy for primary lung cancer. J Thorac Cardiovasc Surg 1986; 92:871–9.
38. Brusasco V, Ratto GB, Crimi P, et al. Lung function following upper sleeve lobectomy for bronchogenic carcinoma. Scand J Thorac Cardiovasc Surg 1988;22:73–8.
39. Rendina EA, Venuta F, De Giacomo T, et al. Sleeve resection and prosthetic reconstruction of the pulmonary artery for lung cancer. Ann Thorac Surg 1999;68:995–1002.
40. Okada M, Tsubota N, Yoshimura M, et al. Extended sleeve lobectomy for lung cancer: the avoidance of pneumonectomy. J Thorac Cardiovasc Surg 1999;118:710–4.
41. Mehran RJ, Deslauriers J, Piraux M, et al. Survival related to nodal status after sleeve resection for lung cancer. J Thorac Cardiovasc Surg 1994;107:576–82.
42. Vogt-Moykopf I, Fritz TH, Meier G, et al. Bronchoplastic and angioplastic operation in bronchial carcinoma: long-term results of a retrospective analysis from 1973 to 1983. Int Surg 1986;71:211–20.

Part 2

Therapeutic Techniques and Management

ANESTHESIA, PRE- AND POSTOPERATIVE CONSIDERATIONS AND COMPLICATIONS

SURGICAL TECHNIQUES

SPECIAL PROBLEMS AND MODES OF TREATMENT

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Preoperative Considerations

Hermes C. Grillo, MD

The best opportunity to correct a tracheal lesion is at the initial operation. Given the short length of the trachea, the opportunity for successful reconstruction may be lost if the first operation is poorly conceived, poorly executed, or is followed by serious complications. Although a successful reconstruction may be accomplished despite an initial failed operation, it often becomes increasingly difficult and is sometimes less likely to succeed.[1] The surgeon must exercise mature and informed judgement in deciding whether to operate and when to operate, and should not assume that a failed operation can be successfully revised. The tracheal tailor quickly runs out of cloth, and in the absence of development of a truly dependable prosthetic replacement, which I believe is unlikely to ever fully substitute for the native trachea, an operation may be peculiarly fateful (see Chapter 45, "Tracheal Replacement").

It is therefore often judicious to delay the operation, in order to investigate local and systemic conditions, to improve these with treatment or time, and to obtain sophisticated consultation if the problem is a knotty one.

Urgency of Tracheal Surgery

When I began to perform tracheal surgery and encountered a patient in severe respiratory distress, gasping for breath, stridorous, and with an airway measuring 3 to 4 mm in diameter, I would proceed to emergency resection after dilating the obstruction. An emergency operation, performed with limited opportunity for complete assessment of the lesion or the patient, and sometimes operating at an inappropriate time, might set a scene for complications and failure. *Emergency resection of tracheal stenosis is rarely, if ever, needed.* It is generally possible to establish an adequate airway safely, either for a short or long term, without immediate reconstruction. This can usually be accomplished without compromising subsequent early or late elective reconstruction.

Urgent management of tracheal obstruction is discussed in Chapter 19, "Urgent Treatment of Tracheal Obstruction" and Chapter 31, "Repair of Tracheobronchial Trauma." The most common causes of acute tracheal obstruction are postintubation stenosis, primary or secondary tumor, and airway trauma. Successful management demands prompt and precise treatment, with close cooperation between the anesthetist and surgeon. Tracheostomy is often inadvisable and, if necessary, must be correctly located in order as not to

compromise future repair. In particular, there is no justification for a tracheostomy to provide "safe access" for repeated laser treatments. In specific instances of trauma, however, emergency tracheostomy becomes necessary (see Chapter 9, "Tracheal and Bronchial Trauma" and Chapter 31, "Repair of Tracheobronchial Trauma"). One of the few tracheal lesions that requires immediate definitive reconstructive surgery is the very rare tracheoarterial fistula (see Chapter 27, "Repair of Tracheobrachiocephalic Artery Fistula").

Preoperative Assessment

The Tracheal Lesion

Important historical facts that should alert the surgeon to potential complications include multiple intubations and tracheostomies, including successive stomas at different levels; treatments, such as laser, cryotherapy, coagulation, repeated dilations, and local injection of steroids; and other maneuvers which may have compounded the problem. Particularly important are data on prior open operative procedures, including resection and reconstruction, laryngofissure, plastic procedures, cartilage grafts, cutaneous troughs or tubes, stenting with silicone stents or expandable devices, buccal or cutaneous grafts, and the implantation of prosthetic meshes or materials.

Assessment of the local lesion is best begun with a complete radiological study (see Chapter 4, "Imaging the Larynx and Trachea"). A tracheostomy tube should be removed intermittently during these studies. A surgeon should be at hand during the studies to remove and replace the tube as necessary. Gloves, suction catheter and apparatus, tracheostomy tubes of the same and smaller sizes with obturators should all be available for the surgeon's use. Replacement can very quickly become difficult. T tubes should not be removed during a radiologic study, although these are sometimes removed and replaced with a tracheostomy at bronchoscopy, in anticipation of a subsequent tracheal reconstruction. Endoscopy is the next critical method of examination. Bronchoscopy is often scheduled at the time of operative repair (see Chapter 5, "Diagnostic Endoscopy"). However, if for historical reasons, on the basis of external examination of the trachea or for systemic consideration, it is likely that the operation will be deferred or impossible, then a bronchoscopy should be done early to plan future treatment. The bronchoscopy may reveal the need for preliminary therapeutic procedures, such as dilation of a stenosis, coring out of tumor, removal of granulations, removal of a stent, change of a tracheal tube or placement of a T tube, with postponement or abandonment of surgical reconstruction.

How much trachea can be removed and anastomosis safely accomplished? This is the key question that is always asked about tracheal resection with primary anastomosis. For any given clinical problem, the question becomes, "Will resection and reconstruction likely exceed safe limits?" Various anastomotic tensions, within which safe anastomosis could be performed, have been *experimentally* determined over the years. Cantrell and Folse observed the disruption of anastomoses in dogs between 1,700 and 3,100 g of tension, at which 46 to 63% of tracheal length had been removed.[2] Flexion and extension of the neck made a great difference in the permissible length of resection. Maeda and Grillo observed no deaths from anastomosis in adult dogs following resections that demanded up to 1,500 g of tension.[3] Puppies, on the other hand, showed fatal stenosis in one-half of anastomoses performed at 1,250 g, and in all done at 1,500 g. With variable lengths of resection calculated to result in 750 g of anastomotic tension in puppies, stenosis could be further reduced by the use of anastomotic "tension" or "stay" sutures.[4] Ferguson and colleagues observed in dogs that about one-third of the trachea could be removed with 450 g of tension.[5] Michelson and colleagues freed the right main bronchus in dogs, divided the left main bronchus, and reanastomosed it to the bronchus intermedius.[6] This allowed the resection of 12 rings.

Efforts to determine acceptable limits of resection in humans were made in *cadaver studies* using various methods to mobilize the trachea. Ferguson and colleagues found the extensibility of the trachea in

cadavers to range from 35% at 29 years of age to 17% at 76 years, with the most stretch obtained at 200 g of tension.[5] Michelson and colleagues observed in cadavers that the human trachea could be stretched 4 to 6 cm by mobilization, and an added 1.5 to 5 cm could be obtained by the maneuver described earlier in dogs.[6] In fresh cadavers, using 450 g tension, the division of the pulmonary ligament allowed 2.5 to 3 cm of tracheal elevation. Freeing the left main bronchus permitted 5 to 6 cm in subjects under 50 years of age. Between 50 to 75 years of age, these distances were only 1 to 1.5 cm and 2 to 3 cm. In contrast to their observations in dogs, Cantrell and Folse observed that a resection greater than 2 cm in cadavers over 80 years of age produced unacceptable tension.[2] Grillo and colleagues concluded that over one-half of the trachea could be removed in human cadavers with stepwise mobilization.[7] These steps included 1) dissection of the right hilum with division of the pulmonary ligament (3 cm, 3 to 8 rings), 2) division of the left main bronchus (2.7 cm, 3 to 12 rings), and 3) freeing of pulmonary vessels from the pericardium (0.9 cm, 0.5 to 3 rings), for a total of 6.4 cm (11 to 18 rings). Tensions for anastomoses rose sharply, as 1 cm segments were successively excised, from 25 g at 1 cm to 675 g at 7 cm. Age was not a factor. Mulliken and Grillo found that 4.5 cm of the upper trachea (7.2 rings) could be resected and anastomosis performed at 1,000 to 1,200 g, with pretracheal mobilization and cervical flexion of 15 to 35 degrees.[8] Right hilar dissection allowed a further 1.4 cm (2.5 rings) to be removed, for a total of 5.9 cm, whereas cervical flexion allowed a gain of 1.3 cm (2.5 rings) over the neutral position.

Efforts to measure anastomotic tensions intraoperatively were not systematically successful. The figures obtained in cadaver investigations provide only general guidelines. Permissible lengths vary individually with a patient's age, body build, height, pathology, and prior surgery. A young adult with a long, supple neck may permit resection of 60% or more with only pretracheal mobilization and cervical flexion, whereas a heavyset, kyphotic, aged person may tolerate a maximum of 30%. The length of resection likely to be required clinically is based on careful study of linear radiologic studies, bronchoscopic examination and measurement, and sometimes with further information provided by biopsies and, uncommonly, by exploration.

Clinical mobilization maneuvers, described in subsequent chapters, must be applied thoughtfully with regard to surgical accessibility, pulmonary reserve, degree of added trauma, and increase in surgical risk. The surgeon's experience and judgment make final determination of what is likely to be safely possible. It must be remembered that excessive anastomotic tension is a principal root of surgical disaster, and of possible fatality. A *general rule* that approximately one-half of the adult trachea and one-third of the juvenile trachea may be removed,[9] and reconstruction safely made, seems to be the best answer to the question at present.

Delay of Resection

Excessive inflammation characterized by florid granulation tissue, easy bleeding, inflamed mucosa, and an inability to determine a boundary between quiescent and inflamed tissues is a warning sign against immediate resection and reconstruction. Although operation has been successfully carried out in the face of inflammation, postoperative complications and restenosis appear to occur more often.

Invasive infection and cellulitis, although surprisingly rare, should be treated before the operation. Chronic infection in a cervical or sternal wound adjacent to the operative field or within the operative field also signals caution. However, a ring of stomal granulation tissue and exudate, which often shows *Staphylococcus aureus* or *Pseudomonas aeruginosa* on culture, are not contraindications to reconstruction. Treatment of invasive infection may require removal of sternal wires, debridement of the bone, and local wound treatment as well as appropriate antibiotics. For long-term treatment, a tracheostomy or T tube may be necessary, if not already in place.

If a patient has had a *recent tracheostomy* for treatment of a stenosis, it is best to wait a period of time for regression of inflammation and healing of the local wound. Four weeks is usually adequate for this. Less delay may be accepted if there is a truly pressing need to get on with the operation. If the tracheostomy has

been placed *below* a cervical stenosis, it may be necessary to transfer the stoma to the damaged cervical segment of the trachea and allow the fresh stoma to heal to recover tracheal length. A removable stent is best withdrawn to allow regression of inflammation and granulations. A T tube may be exchanged for a small tracheostomy tube to allow regression of subglottic inflammation instigated by the upper limb of the T tube. Expandable stents produce difficult problems, which may require unique open surgical procedures (see Chapter 40, "Tracheal and Bronchial Stenting").

Reoperation after *failure of tracheal reconstruction* should be delayed for at least 4 months, and preferably for 6. Inflammatory changes are always huge in an operative field. Tissue planes are obliterated by edema, inflammation, tissue proliferation, and easy bleeding. Although scar becomes more dense with time, the remodelling process in a wound lessens the inflammatory changes progressively. A mature cicatrix is more easily dealt with surgically than induration and inflammation. If reoperation is done too soon, the chance of further failure of reconstruction or of damage to recurrent laryngeal nerves increases. If a patient is referred for treatment soon after failure of reconstruction, a T tube may stabilize the airway during the 4- to 6-month healing period.

A pressing reason for deferring immediate surgery is to define the full extent of airway injury and, in particular, *laryngeal function*. Fluoroscopy may reveal glottic dysfunction and also the presence of tracheomalacia. Laryngeal function must always be assessed thoroughly (see Chapter 35, "Laryngologic Problems Related to Tracheal Surgery"). Direct or indirect laryngoscopy is best done by a consulting laryngologist. Few thoracic surgeons have acquired special competence in laryngology. Laryngeal x-rays, including tomography, which provide information about subglottic narrowing and stenosis (see Chapter 4, "Imaging the Larynx and Trachea"), may reveal more than a computed tomography scan. Tracheal surgery performed unknowingly with a malfunctioning larynx can result in disaster. If the larynx is found to be incompetent only after resection, then a tracheostomy may become necessary before subsequent correction of the laryngeal defect. If the larynx is now at or below the sternal notch, then the brachiocephalic artery may be at risk. The fresh anastomosis may also be damaged. *Laryngeal lesions should be recognized and treated prior to, or in some cases concomitant with, tracheal surgery*.

If there is any question about the patient's ability to swallow or the occurrence of *aspiration*, then a careful barium study of deglutition is essential, preferably a provocative study with food. If aspiration results from neurological deficit, then tracheal resection and reconstruction is inadvisable, since a tracheostomy would then be required for protection.

Systemic Considerations

Generally speaking, few patients should be rejected for correction of a postintubation tracheal stenosis because of age, suboptimal pulmonary or myocardial function, or associated diseases. Resection and reconstruction of the trachea for postoperative stenosis can usually be conducted so that insult to the patient's general physiology is minimal. With skillfully given anesthesia, so that the patient continues to breath spontaneously throughout the operation, and with immediate postoperative extubation, little hazard is incurred. Great care is taken to avoid impairment of respiration. The airways are kept clear of blood and secretions by attentive suctioning, pneumothorax is avoided, and the field is confined to a cervical exposure and added upper mediastinal exposure in some. Even stenotic lesions at the supracarinal level can be repaired through this exposure. Partial sternotomy avoids the pain and impairment of respiration occasioned by a full sternotomy or by thoracotomy. If body cavities are not violated and surgery is performed gently and with precision, then systemic impact is small. I have operated on many patients with borderline pulmonary function due to chronic pulmonary disease that had caused respiratory failure, ventilation, and stenosis in the first place. These patients have most often subsequently remained free from tracheostomy. Their medical conditions are not often a justification for alternative use of laser treatment.

Respirator support should be avoided, if possible, following tracheal reconstruction. The possibility is judged by careful assessment of preoperative respiratory function. Poor function may militate against a transthoracic approach to a tracheal tumor, for example. Complete median sternotomy might be preferable. A fuller discussion is found in Chapter 23, "Surgical Approaches" and Chapter 28, "Reconstruction of the Lower Trachea (Transthoracic) and Procedures for Extended Resection." Management of a patient who requires respirator support following tracheal resection at different levels is discussed in Chapter 20, "Postoperative Management." Tracheal resection and reconstruction is not advised in a patient *already* on a respirator. If a patient has developed stenosis below a ventilating tracheal tube, then it is better to dilate the stenosis and place a tube of length sufficient to pass the obstruction. At the carinal level, granulations and stenosis must be dealt with repeatedly, and an attempt must be made to wean the patient from the respirator. Some of the small number of patients who died following resection of a tracheal stenosis were referred to us on respirators after failed operations and were reoperated on in our early experience. The necessarily shortened trachea obliges the cuff of the endotracheal tube to lie adjacent to the area of reconstruction. Even with a low-pressure cuff, this causes inflammation and dehiscence in time.

As much as possible, *pulmonary infection* should be treated preoperatively with antibiotics, chest physiotherapy, bronchoscopy, and tracheal dilation. However, surgical relief of the obstruction may be necessary to clear pneumonia.

A recent *myocardial infarction* provides the same warning against immediate airway reconstruction as it does for other surgery. Appropriate time should be allowed for myocardial recovery and for careful assessment of myocardial function before tracheal reconstruction. A benign stricture may be managed by dilation, and a tumor by coring out, to gain time for improvement.

If tracheal stenosis resulted after *multiple injuries*, then all other predictable surgery should be completed before a tracheal repair is done. This will avoid the hazard of repeated postoperative intubation and ventilation, which could damage the recently repaired trachea. Wounds or infection elsewhere in the body should be cleared up prior to correcting the tracheal lesion.

Neurologic injuries raise concerns. The patient must be cooperative, and will need to be able to accept chest physiotherapy, controlled coughing, and suctioning. A guardian chin suture must be tolerated and head tossing avoided. If neurologic improvement seems to be occurring, surgical delay is advisable, if necessary with placement of a tracheostomy or T tube. Neurologic defects in deglutition, which cause aspiration, must be ruled out. If it seems that a tracheostomy will be permanently needed to control secretions, then there is no point in repairing the trachea. If speech is impaired by a stenosis, then appropriate placement of a tracheostomy tube or T tube may restore communication and provide a port for suctioning. Usually, paraplegia does not present difficulties in clearance of secretions. The quadriplegic patient must be assessed very carefully, however, to determine how much chest wall respiratory function is preserved, whether the phrenic nerves are functional, and whether the patient could potentially clear secretions without a tracheostomy. Candidates for reconstruction must be selected with great care. Surgeons must be aware of pressures placed upon them to repair a stenotic trachea in a patient with central neurologic defects. Families may feel great urgency to correct whatever is correctable in an effort to bring a young patient especially, who has suffered such injury in a motor vehicle accident, back to normal function. Since the trachea is reparable, families focus upon this step toward recovery, even though this is not the patient's major problem and may be unlikely to provide general improvement.

Although the trachea may be repaired successfully in an *obese patient*, pulmonary hazards are increased, including the possibility of postoperative ventilation. In benign stenosis, I feel it to be the patients' obligation to participate in their own care by losing weight. The prospect of getting rid of a tracheostomy can be nearly as great a stimulus to reduction in weight as is closure of a colostomy.

Tracheal repair is inadvisable in a few diseases. If a patient with myasthenia gravis gives a history of frequent exacerbations, which led to respiratory failure and need for ventilation, then it makes little sense to reconstruct the trachea. Such a patient is better managed with a tracheostomy or T tube, so that when the next and predictable episode occurs, a tracheostomy tube can be replaced for ventilation without difficulty. If, on the other hand, the disease seems to be well controlled, then reconstruction is appropriate, with cautionary statements to the patient about what might occur in the event of respiratory failure. A similar approach is taken to patients whose stenosis was due basically to chronic pulmonary disease.

Drugs can adversely affect tracheal reconstruction. Regular aspirin dosage causes, at most, a brief delay in operation. Many patients with tracheal stenosis or tumor are misdiagnosed and treated for adult-onset asthma with steroids at increasing dosage. Operative failure in 2 patients receiving 40 to 50 mg/day of prednisone was characterized not by acute anastomotic separation but by slow restenosis. Re-resection was successfully accomplished, long after prednisone was discontinued. The findings suggested a gradual distraction and elongation of the anastomosis, which might be expected to result from the usual anastomotic tension acting in the absence of a prompt and vigorous healing response. Patients should be weaned from steroids prior to tracheal reconstruction, but they should usually receive short-term stress doses of steroid perioperatively. The patient's airway is maintained by repeated dilations or by a tracheostomy or T tube during weaning from corticosteroids. Tracheal tumors are cored out when surgery is delayed. I prefer not to use irradiation during this period of time prior to surgery.

Preoperative Interview

It is important that the patient and the patient's family have a clear understanding of the possibilities for restoration of normal function as well as the hazards of operation. It can be difficult to discuss the many options that may arise during a procedure. The surgeon must have a series of alternatives in mind and should review these with the patient. In particular, when the patient will undergo bronchoscopy just prior to operation (a generally preferable way to proceed), alternatives should be discussed fully. It is often possible to predict with accuracy from radiologic studies whether reconstruction will be possible. All too many patients, both with stenosis and with tumors, are on a borderline. Bronchoscopy may make clearly apparent that a lesion cannot be resected with safe primary reconstruction. In benign stenosis, insertion of a T tube may be appropriate. This possibility must be discussed in advance. If tumor is irresectable, then coring out may be done and the patient returned home for irradiation. At times, exploration is necessary to determine if correction is possible.

Even in more usual cases, patients and families must recognize the possibilities of failure, of injury to recurrent laryngeal nerves, of temporary tracheostomy, of periods of hoarseness, of possible complications of laryngeal release if required, and of the need for temporary gastrostomy. The hazards of carinal reconstruction are not to be underemphasized.

The Surgeon and Tracheal Reconstruction

This book represents an effort to codify information that has been gained over nearly 40 years of development of tracheal surgery. Conceptually, the operation of tracheal resection and reconstruction is simplicity itself—resection of a segment of airway and end-to-end anastomosis. Experience shows that this can be illusory. This text hopes to outline the numerous complexities. Carinal resection and reconstruction is recognized as being fraught with hazards and possibilities of disaster. However, I continue to be appalled by the number of patients, with what seem to be simple stenotic problems, who have experienced surgical failure and are referred for reoperation. Often, it is not possible to determine where the errors were. At other times, it seems likely that excessive amounts of trachea were circumferentially dissected and devascularized, that heavy suture materials indicative of crude technique were used, and that the operation was performed without proper attention to detail and precision.

Tracheal surgery has now been standardized and most complications are avoidable in straightforward cases. The burden is on the occasional tracheal surgeon to think carefully before embarking upon a procedure. Tracheal cases are few in number and, therefore, it is difficult for any large number of surgeons to obtain profound experience in the many variations which present. The question may properly be posed as to whether patients would benefit if tracheal surgery were concentrated in a number of geographically distributed centers, all of which remained in close contact to review experiences. The rarity of tracheal lesions makes them attractive and challenging to a surgeon. Surgeons should ask themselves scrupulously whether the operation should indeed be performed. I have never seen a patient or a family who have faulted a surgeon for referring a patient elsewhere for initial tracheal surgery. I have, however, encountered many patients and families who have deeply resented receiving what they later recognized to be inexperienced or inappropriate surgery.

References

1. Donahue DM, Grillo HC, Wain JC, et al. Reoperative tracheal resection and reconstruction for failed repair of post-intubation stenosis. J Thorac Cardiovasc Surg 1997;114:934–9.
2. Cantrell JR, Folse JR. The repair of circumferential defects of the trachea by direct anastomosis: experimental evaluation. J Thorac Cardiovasc Surg 1961;42: 589–98.
3. Maeda M, Grillo HC. Effect of tension on tracheal growth after resection and anastomosis in puppies. J Thorac Cardiovasc Surg 1973;65:658–68.
4. Kotake Y, Grillo HC. Reduction of tension at the anastomosis following tracheal resection in puppies. J Thorac Cardiovasc Surg 1976;71:600–4.
5. Ferguson DJ, Wild JJ, Wangensteen OH. Experimental resection of the trachea. Surgery 1950;28:597–619.
6. Michelson E, Solomon R, Maun L, Ramirez J. Experiments in tracheal reconstruction. J Thorac Cardiovasc Surg 1961;41:748–59.
7. Grillo HC, Dignan EF, Miura T. Extensive resection and reconstruction of mediastinal trachea without prosthesis or graft. An anatomical study in man. J Thorac Cardiovasc Surg 1964;48:741–9.
8. Mulliken JB, Grillo HC. The limits of tracheal resection with primary anastomosis: further anatomical studies in man. J Thorac Cardiovasc Surg 1968;55:418–21.
9. Wright CD, Graham BB, Grillo HC, et al. Pediatric tracheal surgery. Ann Thorac Surg 2002;74:308–14.

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Anesthesia for Tracheal Surgery

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The treatment of tracheal disease, specifically by resection and reconstruction, comes from the systematic work of Grillo and others exploring methods of tracheal repair. It is no accident that the early surgical papers included descriptions of the anesthetic approach as well. Successful tracheal surgery requires skilled coordination between the surgeon and anesthesiologist.

Much of the early need for tracheal surgery was a sequel to the success of anesthesia and intensive care. As intubation and ventilation became a common means for supporting critically ill patients, tracheal injury from endotracheal tubes and tracheostomy tubes required treatment. In turn, we now have better designed endotracheal tubes and a better understanding of the proper way to handle patients with airway appliances.

Tracheal surgery spans from simple fiber-optic examinations to complex resection and reconstruction. The more complex cases should be sent to centers with extensive experience and an institutional commitment to the specialty. All anesthesiologists, however, will benefit from an understanding of the principles and practical approaches to lower airway lesions.

The American Society of Anesthesiology has promulgated a clear algorithm for the difficult airway.[1] That algorithm is really for patients with difficult oral and facial anatomy. Tracheal anatomy is not addressed, and without careful thought, using its methods in these patients may actually be harmful. We will review the special considerations for tracheal lesions and anesthetic approaches to surgical procedures.

Patient Considerations

Fixed Stenosis

There are many diseases and injuries that can cause a fixed tracheal stenosis. This is characterized by a region of the trachea that is narrowed throughout the respiratory cycle, as contrasted with the dynamic obstruction caused by a weakened tracheal wall. On examination, there will be limitation of flow to both inspiration and expiration, with stridor. Flow volume loops will show attenuation of both phases of the flow curve. Plain chest radiographs, and certainly tomograms, will show a stenotic segment.

Patients commonly present with increasing dyspnea. They may note exacerbation with respiratory infections, and certainly decreased exercise capacity. Other presenting symptoms may be hemoptysis, persistent bronchitis, goiter or neck masses, or superior vena cava (SVC) syndrome. Patients may also be identified after difficulty with routine intubation (an endotracheal tube would not pass), or when persistent "asthma" finally precipitates bronchoscopy or pulmonary function tests for assessment. It is distressingly common for patients with tracheal lesions to have been mistakenly treated for the much more common bronchospastic disease.

Airflow in the respiratory tract usually shows laminar characteristics—smooth flow patterns with a parabolic velocity profile. Because of the rapid branching of the tracheobronchial tree, the majority of airflow resistance is in the major conducting airways, so stenosis in this region will be clinically relevant. In laminar flow, the Hagen-Poiseuille relationship holds:

$$Resistance \quad \alpha \quad \frac{viscosity \times length}{diameter^4}$$

Note the profound effect of diameter, which corresponds to the clinical observation that patients note few symptoms until their stenosis reaches a certain threshold (on the order of 5 to 6 mm in adults), when dyspnea without exertion first appears. Once symptomatic, small changes in diameter, from secretions, infection, trauma, or progression of disease, can greatly exacerbate the obstruction.

Airflow does not remain laminar in severe stenosis. Irregularities in the profile of the tracheal lumen can create disturbed flow, and even flow in smoothly contoured tubes will become turbulent when the inertial forces overwhelm viscous damping. Reynolds' number is a nondimensional parameter balancing viscous and inertial forces. Above a geometry-dependent threshold (2,100 in cylindrical conduits), turbulent flow predominates. Reynolds' number is defined as:

$$R_e \quad \equiv \quad \frac{density \times diameter \times velocity}{viscosity}$$

In turbulent and orifice flow, gas density is a determinant of airway resistance. Furthermore, airflow resistance is higher in turbulent flow than in equivalent laminar flow. Dilating the airway, reducing peak flows (by calming and sedation), and reducing gas density all can restore laminar flow. Helium is less dense, so helium–oxygen mixtures can improve flow characteristics in severely stenotic airways.[2] Helium can be used as a temporizing measure while the patient is transported and the surgical team assembled.[3,4] Anesthetic management is typically performed with a high O_2 concentration and potent inhalation agent. Whether the flow advantages of added helium would offset the lowered oxygen reserve from the lowered fraction of inspired oxygen (FiO_2) is an open question.

It is helpful to know the location and cause of the stenosis before planning the anesthetic. Typical locations are the midtrachea for cuff stenoses and the site of a previous tracheostomy if scarring from that procedure has caused the injury.

Dynamic Obstruction

Flow-dependent obstruction is caused by a weakened tracheal wall, or by an intraluminal mass that moves with respiration. Patients will have symptoms that are manifest primarily during inspiration or expiration. Position may be an important factor in their airway patency and every effort should be made to mimic their optimal position during induction.

Normal pressure relationships in the tracheobronchial tree show a gradient that directs airflow. During inspiration, the pleural and alveolar pressures are subambient, the tracheal and conducting airway

intermediate, and the mouth at ambient pressure. In the extrathoracic region, the tracheal lumen will be at a lower pressure than the surrounding tissue, and would collapse were it not for the resiliency of the cartilage rings that nearly encircle the trachea. If the rings have been damaged or eroded, the trachea will tend to collapse on inspiration, worse when a higher gradient is attempted. Thus, attempting to compensate for the increased resistance of a tracheal lesion by more respiratory effort will be counterproductive. On flow volume loops, there will be a plateau in the inspiratory phase, where optimum flow is achieved by a balance of the pressure gradient and lumen size. Positive pressure ventilation should counteract this tracheal collapse, although more complex injuries make the balance unpredictable.

On expiration, the thorax is at a higher pressure than the tracheal lumen, which in turn is at a higher pressure than the mouth. Intrathoracic lesions will be manifest in expiration. There will be the same plateau in the flow volume loop, but now in the expiratory phase. Controlled ventilation should have no advantage, although the application of positive end-expiratory pressure (PEEP) may increase the intraluminal pressure. Indeed, patients may use pursed lip breathing on expiration to induce this phenomenon. This suggests that the transluminal pressure seen in the trachea is not just the pulmonary pressure, which would be similarly raised by PEEP.

Loss of integrity of the cartilage skeleton of the trachea is seen after trauma, tumor, and some congenital abnormalities. Any lesion in the trachea will worsen the effect by increasing the needed pressure gradients. Large external masses in the anterior mediastinum can compress and distort, and should trigger concern.[5,6] To some extent, the tone of the muscles of respiration seems to play a role, with worsened obstruction seen after the administration of muscle relaxants. In these patients, positional symptoms may be very important. Mediastinal masses may also encroach on vascular structures, with SVC syndrome and airway swelling, worsening the symptoms. In experimental studies, the cardiovascular effect of an anterior mediastinal mass was right heart failure due to pulmonary vessel obstruction, which was independent of mode of ventilation.[7]

A careful history should identify those patients at risk for problems with airway obstruction, at induction of anesthesia. The safest approach in patients at significant risk is to maintain spontaneous ventilation until the obstruction can be assessed and the airway secured. A potent inhalation agent is employed until loss of consciousness. At this stage, it is possible to attempt control of ventilation, although the patient will not be ready for instrumentation. If it is possible to control ventilation, then we know that positive airway pressure is advantageous, and we will be able to speed the arrival at a surgical anesthetic plane as well as consider the use of muscle relaxants. If controlled ventilation is not possible (after suitable methods of relieving upper airway obstruction), then muscle relaxants should not be used.

It is possible to perform rigid bronchoscopy and intubation solely on deep inhalation anesthesia with spontaneous ventilation. The onset will be slow since ventilation will decrease as the anesthetic depth increases, especially in the presence of airway obstruction; the drive to ventilation in the presence of a respiratory load is attenuated by potent inhalation as well as intravenous agents. Sevoflurane is currently the best choice for inhalation induction; it is potent and surprisingly nonirritating to the respiratory tract.[8]

If ventilation can be successfully controlled, then the use of neuromuscular blockers can be considered. The advantages of relaxants are better intubating conditions and less chance of movement during rigid bronchoscopy. The disadvantages are that spontaneous ventilation will not be possible and that muscle tone will be lost. Being able to recover spontaneous ventilation can be important for diagnostic reasons, to assess if there is a dynamic component to the tracheal obstruction, and for therapeutic reasons, to illustrate by the movement of air where the true lumen is located in a badly diseased trachea. As the bronchoscopy proceeds, an initially adequate airway can become bloody and swollen and no longer safely controlled. Being able to fall back on spontaneous ventilation in this circumstance may be essential, and cannot be assured after paralysis.[9] In the end, the point in the procedure when muscle relaxants can be safely used

depends on the experience and judgment of the anesthesiologist and surgeon. Some centers have published good results using intravenous techniques alone.[10]

Tracheoesophageal Fistula

A tracheoesophageal fistula (TEF) creates unique challenges to the anesthesiologist. Until the trachea is intubated below the fistula, there is no way of preventing refluxed gastric contents from entering the lungs, or of assuring that controlled breaths will enter the lungs instead of the gastrointestinal (GI) tract. Excluding congenital abnormalities in neonates, the common causes of TEF are postintubation fistulae, as well as traumatic tears, and erosion from tumor, infection, or radiation. In the worst cases, large holes allow free and chronic aspiration with severe lung injury.

Aspiration risk can be minimized by elevating the head of the bed (reverse Trendelenburg position), gastric drainage, and withholding food. Nasogastric tubes or gastrostomy tubes can be suctioned to reduce gastric contents and to vent gas from mask ventilation. Cricoid pressure will obviously be ineffective. If one lung has been more severely affected by aspiration, then that side should be dependent to prevent gastric contents from injuring the better side.

Spontaneous ventilation has strong advantages over controlled ventilation for the unsecured airway in TEF. The descending diaphragm draws gas into the lungs. All that is required is a patent airway above the lesion, which can be obtained with a standard jaw thrust, oral or nasal airways, or a laryngeal mask airway (LMA). Anesthetic induction methods that maintain spontaneous ventilation, like potent vapors, allow ventilation while the airway is assessed and secured. In contrast, controlled ventilation requires that the compliance of the lungs be lower than that of the GI tract. Even if a sufficient fraction of each delivered breath enters the lungs, there will be progressive abdominal distention since the esphagogastric junction acts as a one-way valve. Eventually, the distended abdomen will compromise ventilation. Despite the advantages of spontaneous ventilation, controlled ventilation and muscle relaxation is often employed in neonates, with subsequent occlusion of the TEF by a balloon until the repair is complete.[11]

Patients with very small fistulae present a different challenge: finding the tract. Controlled ventilation is not a problem in these patients. The fistula may be found with contrast studies in the radiology suite, or intensive bronchoscopic examination of the trachea, possibly while dye is introduced into the esophagus.

Endotracheal tubes should be positioned precisely under direct fiber-optic guidance in patients with tracheoesophageal fistulae. The tube must be distal to the fistula, and it is preferable to have the cuff distal to the lesion, so that it does not enlarge the hole. Fistulae near the carina may be better managed with double lumen tubes or endobronchial intubation.

Airway Trauma

Anesthetic considerations in traumatic injuries to the airway revolve around identifying those patients at risk, and choosing techniques that do not cause a tenuous airway to be lost.[12–14] Traumatic injuries to the airway are usually classified by their mode of injury. Penetrating injuries are the result of gunshots and knives. Blunt trauma can be from direct blows, or secondary to rapid deceleration.

Penetrating Trauma. The presence of a penetrating airway injury in the neck is usually unsubtle. An open airway, with gas exchange from the wound, will be present. The optimum approach in these cases is to directly intubate the exposed distal tracheal end, perform any other resuscitative care, and then surgically address the trachea.[15,16] There is the risk that placing a tube in the distal trachea will disrupt the mucosa, pushing it with the end of the tube to fold and obstruct the airway. If possible, the mucosa should be grasped with a surgical instrument and the tube gently slid in. The patency of the tube can then be assessed by conventional means as well as by fiber-optic examination.

Other injuries that should be considered include trauma to the innervation of the vocal cords, pneumothorax, and injury to the major vessels. The surgical approach to the trachea can be a primary repair in auspicious circumstances. Induction of anesthesia is obviously easy, and the tube can be switched to an orotracheal tube under controlled conditions, or with retrograde methods described later.

Penetrating injury within the chest may not initially be recognized. Pneumothorax and major vascular injury will be the initial focus. Hemoptysis, pneumomediastinum, and pneumothorax can be caused by lung injury and well as tracheal injury.

Blunt Trauma. The mechanism of blunt airway injury is still a debatable issue. Most blunt traumas are seen in motor vehicle accidents or other high-impact encounters. Direct injuries to the larynx and cervical trachea are caused by a violent blow by the steering wheel in automobile drivers and by branches or chains in motorcyclists or bicyclists, otherwise known as "clothesline injuries." In these cases, the fractured tracheal ring can be dislocated, thereby obstructing the trachea.[17,18]

Deceleration injuries are where portions of the trachea, hilum and lungs, and chest wall slow at different speeds, producing ruptures of the trachea at various levels, or disruption at the mainstem bronchi. Injuries can occur at all levels but are common in midtrachea, where the trachea passes behind the subclavian vessels and at the hilum, and in the right mainstem bronchus. Alternative hypotheses are that the acute lateral deformation of the thoracic cages causes traction injuries, or that high airway pressures from the sudden compression of the chest cause blowout fractures of the trachea at places where the wall stresses are concentrated.

Tracheal injuries should be suspected in all patients involved in high-speed motor vehicle accidents. Symptoms include hoarseness, voice change, or pain. Air in the neck is a sensitive sign, and can be seen on an x-ray or a computed tomography scan, which will be obtained in any case to evaluate the cervical spine. In grosser cases, subcutaneous emphysema will be present. Hemoptysis, stridor, and respiratory distress can all be signs of airway injury, as is the dropped lung sign seen in complete disruption of a mainstem bronchus. A few patients will only be recognized later in their hospital course, when tracheal stenosis is seen.[19]

These patients require careful airway management. A partially disrupted trachea can be completely disrupted by the passage of an endotracheal tube. Cricoid pressure can dislocate a fractured cricoid or thyroid cartilage. Positive pressure ventilation may either put sufficient stress on a partially ruptured segment to complete the disruption, or be ineffective as gas escapes through a ruptured wall. The safest course is to maintain spontaneous ventilation, either with deep inhalation agents or topical anesthesia and fiber-optic intubation.

Bronchopleural Fistula

Bronchial disruption can occur at any level of the tracheobronchial tree. Peripherally, it is usually managed conservatively. The pleural space must be vented to prevent a tension pneumothorax, and adequate ventilation must be assured. Spontaneous ventilation will work well, as long as the lung stays inflated. If controlled ventilation is required, methods of minimizing airway pressures should be employed, including high-frequency jet ventilation. Placing a double-lumen tube and ventilating the two lungs independently allows the lung with the fistula to not steal all the tidal volume.

Disruption of the major airways, including lobar bronchi after lobectomy and the mainstem bronchi after pneumonectomy, requires surgical intervention. Lung isolation will be required, both to allow surgical exposure and to prevent spillage of pleural contents, blood and pus, into the open airway and to the other side. Prior to induction, patients should be placed with the affected side down. A tube should be placed endobronchially on the side opposite the lesion under fiber-optic guidance.

Procedures

Rigid Bronchoscopy

The rigid bronchoscope finds particular application in tracheal lesions and tracheal surgery. Unfortunately, at many institutions, its use is rare because flexible bronchoscopy is more common for routine examination. The unique feature about the rigid bronchoscope to the anesthesiologist is that it takes the place of an endotracheal tube, serving as an airway as well as a surgical tool.

Rigid bronchoscopes come in various sizes, but share common features: a hollow central lumen open at each end, a side opening for gases, and some small channels along the side for light and jet ventilation. The upper end is opened to allow instruments to be passed, or closed to allow gas delivery.

In the anesthetized patient, the bronchoscope is introduced either directly, or with the assistance of a laryngoscope. Once in the trachea, the neck is extended and the bronchoscope attached to the anesthesia circuit. A variable leak will be present, worsened with proximal position and smaller bronchoscopes, and lessened with distal passage, or passage though a narrowed tracheal region.

Ventilation in the presence of a substantial leak can be problematic. First of all, end-tidal gas tension measurements are useless, as all the returning gas leaks out. Second, the attempted tidal volume is largely lost due to leakage, and only a small and variable fraction is actually delivered to the patient. Observation of chest excursion is the best way to assess ventilation. Blood gas sampling can describe the patient's ventilation at one point in time, but an indwelling continuous arterial gas catheter would be required to provide adequate feedback to guide therapy.

Closing the mouth and nose can lessen the leak (although gas can easily enter the stomach as well). Using high gas flows can also help, but special modifications of modern anesthesia machines are needed since most machines currently on the market do not allow O_2 flows above 12 L/min, whereas flows of 30 L/min may be necessary.

Another method of ventilation with a rigid bronchoscope is to jet gas down the side port.[20] This method requires an open system to prevent severe barotrauma. The gas is delivered at up to 50 psi (300 kPa) at the source, and without a channel for egress, pressure would be transmitted to the entire respiratory organ.[21] The effectiveness of ventilation again has to be assessed by chest excursion. Anesthetic agents must be administered by the intravenous route during jet ventilation.

The rigid bronchoscope can be used to slice off fragments of granulation tissue or tumor in the airway. The anesthesiologist must be aware of this possibility and refrain from ventilation until the loose tissue is retrieved. The anesthesiologist and the operator of the bronchoscope must also negotiate adequate time to ventilate in between apneic periods required for instrumentation. Examination through the rigid bronchoscope is accomplished with prismatic telescopes that have superior optical properties. Ventilation is maintained in the annular space around the telescope.

The rigid bronchoscope is well suited to examination and interventions in the trachea and main bronchi. A flexible fiber-optic bronchoscope is needed for more distal work. The flexible instrument can be passed through the rigid bronchoscope, allowing ventilation and distal observation.

Anesthesia can be maintained by either the inhalation or intravenous route. Neuromuscular blockade is advantageous if the airway can be secured, since vigorous movement with a rigid bronchoscope in place could be dangerous. Because of the large leak with the uncuffed rigid bronchoscope, inhalation agents are less desirable. The quantity of agent used will be uneconomical with the high flows required, and the contamination of the operating room environment is considerable. Levels of inhalation agent in the 200 ppm range have been published, well above the US National Institute for Occupational Safety and Health guidelines of 2 ppm for a potent inhalation agent used without N_2O.[22] Intravenous agents should be selected for their short duration of activity. Remifentanil and propofol are ideal. Awake rigid bronchoscopy in topically anesthetized patients is only of historical relevance.

The rigid bronchoscope can be used as a platform for laser surgery of the airway. The major risk is airway fire. Ventilation at low FiO_2 must be employed during laser segments, and N_2O is not a safe diluting gas since it too supports combustion. Even though the bronchoscope is inert, and no combustible endotracheal tube is used, dessicated tissue can be ignited. The treatment for airway fire has been well described, including cessation of O_2, removal of the fuel, saline quenching, reestablishing an airway, and supportive care.

Flexible Bronchoscopy

Flexible bronchoscopy allows inspection of the major airways as well as several generations of the tracheobronchial tree. Snares, brushes, and fine forceps can be passed through its channel to allow instrumentation of the airway. The flexible bronchoscope is well tolerated by awake patients if adequately topicalized, and so allows inspection of the extent of functional tracheal damage or recovery after surgery. The flexible bronchoscope is also used for placement of endotracheal or endobronchial tubes, and for pulmonary toilet.

In the presence of severe tracheal stenosis, the flexible bronchoscope is less useful. It is difficult to ventilate through the bronchoscope (although jet ventilation through the suction channel has been described) and so the tube will further obstruct the airway. A rigid bronchoscope allows ventilation through its generous internal lumen, and can be used to directly dilate stenotic regions. There is a system for using a flexible bronchoscope to dilate the airway, first by passing a wire guide, followed by a balloon dilator. Ventilation will have to be through the stenosis until the dilation is complete. General anesthesia may not be required for balloon dilation with a flexible bronchoscope (a supposed advantage).[23]

There are several methods for managing the airway during flexible bronchoscopy. Awake patients with adequate topical anesthesia can breathe around the bronchoscope that is passed through the mouth or nose. Under general anesthesia, an endotracheal tube can be introduced and the bronchoscope passed through the tube. Finally, an LMA will sit above the larynx, allowing a bronchoscope to be passed through the LMA, the cords, and into the trachea. The LMA is particularly advantageous in very proximal lesions and in cases where muscle relaxants are otherwise not needed.

Tracheal Resection and Reconstruction

Initial Phase. At the simplest level, anesthesia for tracheal reconstruction is an exercise in sharing the airway. After evaluation, induction, and possibly bronchoscopy, the airway is secured with a tube distal to the lesion. Part of the value of the initial bronchoscopy is to assist planning of the means of securing the airway. The anesthesiologist should view the airway with the surgeon, and get a sense of the lumen size and course. Knowing the airway is bloody or friable will help in the management of a sudden obstruction during the operation.

There are three good reasons to start with rigid bronchoscopy: 1) examination of the trachea and assessing resectability, 2) dilating tight stenoses or removing some endoluminal tumor to permit passage of an endotracheal tube, and 3) in the worst cases, providing the airway used for the procedure. Rigid bronchoscopy is essential when the airway lumen is significantly compromised; that is, less than 5 or 6 mm in diameter. Unless the airway is dilated, the initial operative course will feature hypoventilation with attendant hypoxia, hypercarbia, and arrhythmias.

Induction can be either inhalation or intravenous depending on the underlying pathology and the experience of the practitioners. Long-acting agents are unwise since the initial bronchoscopic evaluation may find that a resection should be postponed or cancelled.

Anesthetic maintenance can be achieved in several ways.[24,25] Inhalation agents, which blunt airway reflexes well, are inexpensive and are relatively quickly dissipated. The disadvantage is that the airway will be opened intermittently during the procedure, so that much of the agent will contaminate the operating room environment. Also, during those periods, no anesthetic will be administered to the patient, requiring compensatory deeper levels before and after.

Total intravenous anesthesia is well suited to tracheal surgery. The processes of ventilation and anesthesia delivery are decoupled, and the operating room air is not contaminated. Remifentanil and propofol delivered by infusion are an excellent choice. Airway reflexes are well blunted, and the effects wear off quickly at the conclusion. Other intravenous regimens such as ketamine hydrochloride infusions, sufentanil citrate or alfentanil hydrochloride infusions, and barbiturates are certainly possible, but run the risk of producing lingering postoperative sedation, which is undesirable.

Regional techniques are used for simple tracheal procedures, like flexible bronchoscopy and occasionally tracheostomy. In theory, a cervical tracheal resection could also be performed under block, but the level of cooperation required, particularly if rigid bronchoscopy is contemplated, makes it impracticable.

The monitoring required for tracheal surgery concentrates on the assessment of respiration. CO_2 measurements in the end-tidal gas and arterial blood are helpful to assess the adequacy of ventilation. Oxygenation is confirmed by pulse oximetry. An arterial catheter is helpful, especially in the postoperative period. It is possible to compress the innominate artery, which crosses the trachea at the sternal notch. Such compression will impair blood flow to the right arm and right carotid. Either an arterial line or pulse oximeter on the right arm will provide warning. More extensive hemodynamic monitoring should be dictated by other coexisting conditions. Intravenous access will be needed, but major volume requirements are rare.

Resection. The preferred position of the endotracheal tube at the start of the resection is distal to the lesion (Figure 18-1*A*). If the tube is proximal, the surgical manipulation may provoke airway obstruction in some types of tracheal lesions. In some situations, distal intubation will not be feasible. Once the airway is secured, the patient is positioned for the tracheal resection with the neck extended to deliver the trachea out of the thorax. Space will be tight around the patient's head, but access must be preserved to allow the anesthesiologist to manipulate the endotracheal tube, an essential part of the procedure.

Surgical dissection is performed to expose the affected region, and the endotracheal tube is withdrawn sufficiently to allow severing of the trachea (Figure 18-1*B*). Ventilation is then accomplished with a tube placed in the surgical field into the distal trachea, attached to a circuit that is accessible by the anesthesiologist. If possible, the circuit should include a sampling port to allow assessment of end-tidal CO_2.

The tube placed on the field will lie in a tightly curved position, so a nonkinking flexible armored tube is preferable. The tube is also placed into a rather short distal tracheal segment, so that it will easily be advanced into a mainstem bronchus. For all these reasons, as well as the potential that blood and clots can run into the trachea from the surgical field, the anesthesiologist must maintain close vigilance over the tube position and pulmonary compliance.

An alternative to distal intubation with an endotracheal tube is the use of jet ventilation with a jet catheter.[26,27] The jet catheter is of smaller caliber and does not require a seal to deliver tidal volume. There are some drawbacks to jet ventilation, including more difficult assessment of the adequacy of ventilation, unless a separate distal sampling catheter is used. Barotrauma is a constant risk if the catheter is advanced into too small a segment of the pulmonary tree. Jetted gases are rarely humidified, so the respiratory tract will become dehydrated, and secretions will be more difficult to mobilize. It is hard to deliver gaseous anesthetics via jet ventilation, and there is the aesthetic problem of aerosolized blood and secretions from the jetting process. Finally, the jet catheter will tend to recoil from the airway, so it must be held in place by a member of the surgical team.

One other proposed method for oxygenation and ventilation is the use of cardiopulmonary bypass (CPB). No airway appliance would be required with this technique. There are substantial disadvantages to CPB, including the need for anticoagulation, microemboli, and more invasive access. More difficult reconstructions, where CPB might be contemplated, usually are intrathoracic, and the substantial lung manipulation required for surgical access would be damaging in the anticoagulated state. Indeed, in experienced centers, CPB is never needed unless major vascular or cardiac work is also required (eg, vascular rings).

Reconstruction. The necessary resection is done and the reconstruction prepared by placing sutures loosely spanning the resected area. Intermittent removal of the endotracheal tube facilitates placement of sutures in the distal margin. When it is time to bring the two tracheal ends together, the surgical tube is withdrawn and the orotracheal tube is carefully advanced into the distal trachea (Figure 18-1*C*).

Flexion of the neck shortens the distance from the trachea to the carina, so the neck is now flexed to allow the tracheal ends to be reapproximated without tension. This neck flexion will be maintained for the balance of the procedure and throughout the postoperative recovery period.

In some instances, the orotracheal tube will have been withdrawn entirely from the trachea during the reconstruction to allow better exposure of the subglottic larynx for repairs at that level. Direct laryngoscopy to replace it would be difficult under the surgical drapes, but fiber-optic intubation would certainly be possible. Fortunately, there is a clever alternative method of intubation. A small stiff catheter is passed retrograde from the surgical field, and fished out of the mouth. An endotracheal tube can then be sutured to the end of the catheter, and the whole assembly pulled into the trachea. This maneuver will be necessary if the orotracheal tube is pulled out, if the patient had a preexisting tracheostomy that was used initially, if the rigid bronchoscope served as the airway during the initial phase of the resection, or if the endotracheal tube was damaged when the trachea was entered. For proximal lesions, where the tube will likely need to be pulled out of the trachea, suturing a catheter to the orotracheal tube before it is fully withdrawn may simplify the reintubation.

Emergence. At the end of the procedure, the goal is to have an extubated patient with a patent airway. There are several reasons to prefer extubation. An appliance in the trachea will irritate the tracheal anastomosis, especially if the end of the tube or the cuff is at the suture line. Positive pressure ventilation will also put strain on the suture line, and tend to push air into the tissues until the mucosa has sealed. A tracheostomy distal to the repair is possible, but will injure some of the remaining good trachea, so it is avoided. If the airway needs to be secured in the postoperative period, either because of transient swelling or injury to the innervation to the cords, then a small uncuffed tube is preferred. The reintubation can be achieved either with direct laryngoscopy while maintaining strict neck flexion (a straight laryngoscope blade works best) or by fiber-optic intubation.

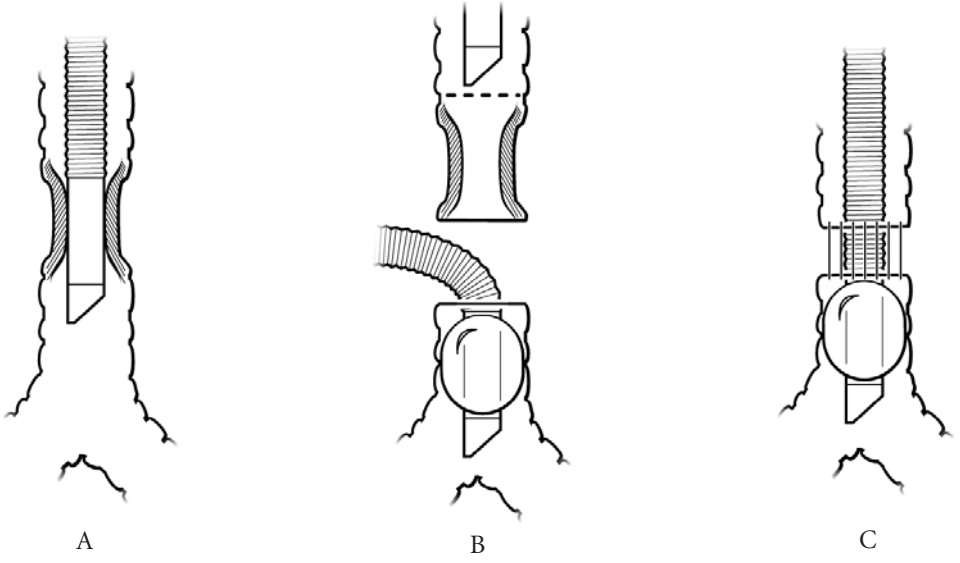

FIGURE **18-1** *Ventilation for tracheal resection.* A, *The endotracheal tube (ET) is passed beyond the obstructing lesion. In this case a tight stenosis provides a seal without inflating the cuff.* B, *The tube is retracted and the trachea is divided below the lesion. Cross-field intubation is performed. If there is insufficient distal trachea, the cuff is seated in the left main bronchus.* C, *After all sutures are placed, the proximal ET is passed distally and the cuff is inflated while the anastomosis is completed.*

Neck flexion must be rigorously maintained. Even one episode of extension in the emergence, period may disrupt the anastomosis. This will be a disaster, both acutely (bleeding, subcutaneous emphysema, and loss of airway) and longer term (leak, scarring, and restenosis). To prevent neck extension, a suture is placed from the chin to the chest. This will prevent an awake patient from unconsciously extending the neck, but it will not stop the movements of a partially conscious patient in the midst of emergence. A prudent anesthesiologist will keep a hand on the occiput throughout the emergence and transport process, forcing the head to follow the torso during patient movement. All other matters of organization and equipment can be delegated. To the extent possible, gentle emergence, avoiding violent coughing and movements, is preferable.

Opiates should be used sparingly. Pain is usually minor; small amounts of narcotic and nonnarcotic analgesics are quite sufficient. Furthermore, we desire an awake, cooperative patient who is able to maintain upper airway patency.

A large part of the art of anesthetizing patients for tracheal reconstruction occurs in the immediate postoperative period, when the adequacy of the airway must be evaluated. Good tidal volumes and a strong voice are signs of a successful outcome. If the patient does not appear to be moving air well, several alternatives must be quickly assessed. Absent or inadequate respiratory effort is a result of inadequate respiratory drive or muscle strength. Both scenarios should be treated symptomatically with support of ventilation and reversal of neuromuscular block or respiratory depression. Again, since these patients have less tolerance of respiratory compromise, a properly planned anesthetic will tend toward light levels of block and sedation.

Patients with respiratory obstruction will show vigorous effort, but inadequate air movement. Sternal and intercostal retraction, and discoordinated chest and abdominal excursion are all signs of obstruction. In the more awake patients, anxiety and air hunger will be manifest. The clinical question is then whether the obstruction is in the upper airway (above the vocal cords), or in the (more worrying) lower airway. Upper airway obstruction can be treated by conventional methods: oral suctioning, jaw thrust, oral or nasal airways, or even an LMA. The only caveat is to avoid neck extension.

Lower airway obstruction can also be due to edema of the trachea or larynx. This is particularly likely if extensive manipulation was required, if the disease process affected nonresected areas, or if infection is present. Severe cases will require a stenting tube; that is, a small caliber uncuffed endotracheal tube. In less severe situations, nebulized epinephrine, upright posture, and a short course of steroids will be sufficient.

Cord function can be impaired from the preexisting disease, or by the surgical dissection. Even though the nerves may be anatomically intact, stretch or trauma may cause transient spasm that adducts the cords. Fiber-optic examination or direct laryngoscopy will show tightly adducted vocal cords, and passage of a small endotracheal tube, or temporary tracheostomy, will be necessary.

Finally, technical problems with the anastomosis are always possible. It is wise to inspect a problematic reconstruction fiber-optically, before the end of the procedure, since unanticipated problems can occur. In the extreme situation, the operation will have to be resumed, and the reconstruction revised—not a pleasant occurrence.

In the first day or two after the procedure, intensive nursing care with close observation is needed. Problems include difficulty in clearing secretions, and bleeding in the neck, compromising the airway. Serial bronchoscopy at the bedside is common, both to assess the surgical repair and to assist with pulmonary toilet.

Patients with previous tracheal reconstruction presenting for unrelated anesthesia are not particularly difficult to manage, although fiber-optic intubation or examination has some merits. The larynx may be relatively distal if the resection was extensive or the tracheal mobility limited (heavy set and older patients). A successfully reconstructed trachea should have smooth walls without stenotic segments; however, even clinically satisfactory repairs can have mild narrowing at the site of the anastomosis. Furthermore, there is risk of inadvertent endobronchial placement of an endotracheal tube, since the tracheal length may be shortened after resection. Effort to be gentle in introducing the endotracheal tube and inflating the cuff is desir-

able. If possible, it would be better not to have the cuff sit across the anastomosis. Techniques that avoid intubation, such as regional techniques, mask ventilation, or the LMA, also are appropriate.

It is possible that there is residual disease or stenosis at the site of the surgery that is not yet symptomatic. If the patient has not been routinely or recently evaluated, a prudent approach to these patients is to have the thoracic surgeon perform bronchoscopy prior to the surgery.

Carinal Resection and Reconstruction

Lesions near the carina add some new challenges to the process of the tracheal resection and reconstruction discussed above.[28,29] The procedure is intrathoracic, usually approached from a thoracotomy, and the lungs cannot be treated as a single entity. As in all thoracotomies, arterial monitoring is prudent and methods of postoperative analgesia, such as thoracic epidural catheters, are needed.

The considerations for induction of anesthesia are similar to those for surgery for higher lesions, although it is even more clear that surgical access to the airway, in the case of a complete obstruction, is not an option. Some authors suggest veno-venous bypass as a fallback technique.[30] Severely obstructing tumor at the carina may be cored out bronchoscopically to provide an adequate initial airway (see Chapter 19, "Urgent Treatment of Tracheal Obstruction"). Once the bronchoscopic examination is completed, it is time to intubate the trachea. There are several options. If the airway is not too compromised, then the tube can sit above the lesion. The trachea will often be approached via thoracotomy, and the airway entered. Exposure cannot be assisted by collapse of the lung unless a blocker is passed into the surgical side. If the bronchus is already damaged, a blocker would be unwise, so small tidal volumes and some effort from the surgeon's assistants are required. Once the trachea is entered, a tube is passed on the field into the opposite mainstem bronchus, and single-lung ventilation is employed.

Some lesions are better addressed by a distal intubation from the start. Since the lesion is at or beyond the carina, the only option is endobronchial intubation (Figure 18-2). An endobronchial tube is chosen instead of a standard double-lumen tube, because the double-lumen tube is too bulky to permit tracheal surgery. Long flexible tubes of small diameter but sufficient length (>31 cm) to reach the bronchus are not currently widely available. It is possible to easily construct them by combining two tubes. An example is shown in Figure 18-3, using a Phycon, a silicone armored cuffed tube with its integral collar, and a length of standard PVC tubing placed in the collar with a friction fit. The lumen stays a constant diameter and the tube has the desirable properties of being more stiff in the upper portion and flexible and nonkinking in the lower portion. Note that the tip design in endobronchial tubes is important. There is not a long segment of bronchus in which the cuff and distal portion may sit, so a shorter cuff-to-end design is preferable. Trimming the end of an endotracheal tube will make the cuff incompetent since the cuff air channel runs beyond the cuff. The endobronchial tube is positioned under fiber-optic guidance. The jury-rigged nature of these tubes, as well as the extensive surgical manipulation of the region, causes frequent tube malpositioning. A fiberscope should be constantly at hand, as the anesthesiologist will be required to make frequent corrections as the operation proceeds.

Other creative endobronchial tubes have been fashioned by trimming the distal portion of the tracheal lumen off a double lumen (destroying the tracheal cuff), as shown in Figure 18-4. This leaves a single lumen in the distal trachea that ends in the bronchus. The endobronchial lumen is well designed for the bronchus, especially the right mainstem bronchus. The abbreviated tracheal lumen is in fact quite useful, allowing introduction of a fiber-optic bronchoscope, jet catheter, bronchial blocker, or oxygen insufflation. This modified double-lumen tube is less satisfactory, however, in greatly distorted airways, since the contour is designed for normal anatomy and has no protection against kinking once warmed and bent.

With endobronchial intubation, one lung will not be ventilated. As in all thoracotomies, the level of shunt and desaturation is variable and unpredictable. Standard maneuvers include suctioning, confirming

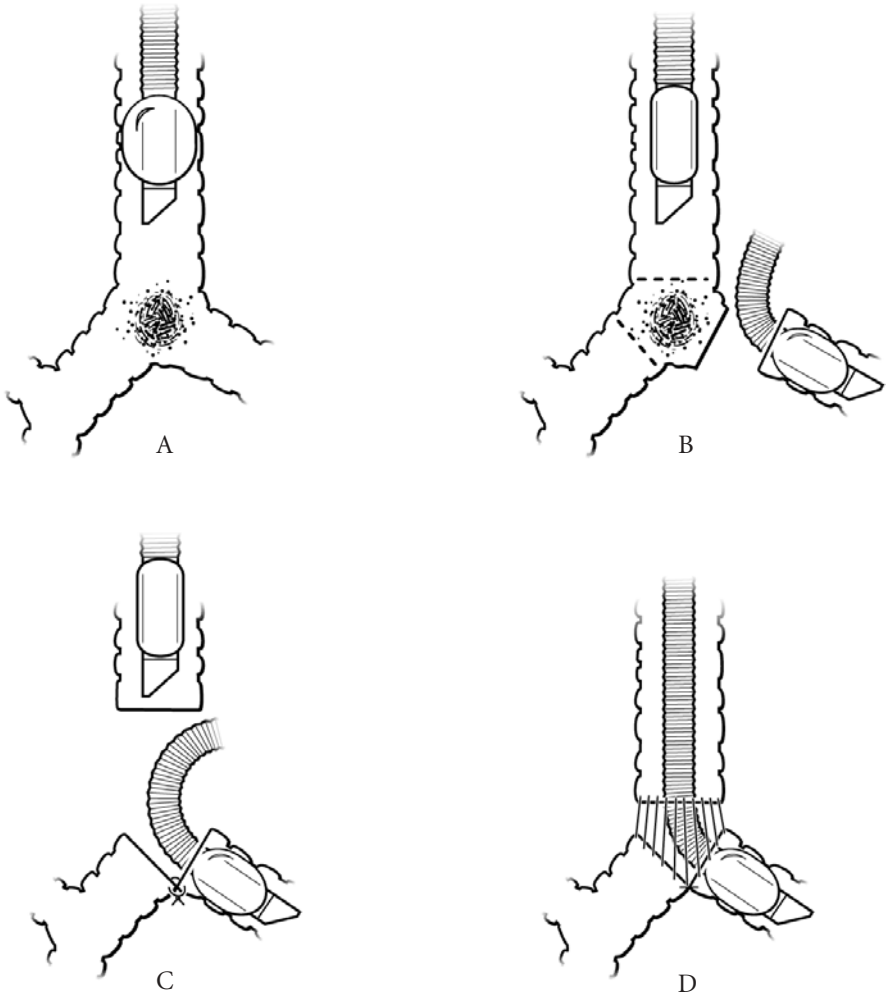

FIGURE 18-2A *Intubation and ventilation for carinal resection and reconstruction. Lesion limited to carina.* A, *A long endotracheal tube (ET) is proximal to the lesion, but is preferable passed into the left main bronchus.* B, *The ET is retracted, the left main bronchus is divided and intubated across the operative field.* C, *Main bronchi are joined medially to form a neocarina. The ET in the left main bronchus is removed intermittently to facilitate suturing.* D, *After all anastomatic sutures placed between the trachea and the joined main bronchi, the proximal ET is advanced into the left main bronchus and anastomosis is completed.*

position, increasing FiO_2, and varying the ventilatory patterns. Unlike many thoracotomies, it is not as easy to administer ventilation to the deflated lung. While the airway is intact, deflating the endobronchial cuff, blocking the mouth and nose, and delivering longer and larger tidal volumes can help. Alternatively, placing another endotracheal tube high in the trachea (ie, two endotracheal tubes, of small diameter) can allow differential ventilation, or at least constant positive airway pressure (CPAP). Another approach is to place an LMA after the endobronchial tube, and if it seals sufficiently, CPAP can then be administered. Finally, a jet catheter can be placed in the trachea. Indeed, a technique using two jet catheters has been described.[31] The jet catheter has the advantage of not requiring a seal, but may not be effective if there is substantial distal obstruction. In extreme circumstances, blood flow to the pulmonary artery can be restricted, lessening shunt.

Once the airway is open, CPAP cannot be administered from above. A second tube can be placed from the field into the deflated lung, or a jet catheter can be used (see Figure 18-2*C*). The jet catheter has the advantage of being small enough to allow surgery to proceed, of not requiring an entire circuit, and that the length that needs to be placed in the trachea is very small. Since large tidal volumes are not needed on the surgical side, the force and mess of jetting is less.

The conduct of one-lung ventilation for carinal surgery requires all the considerations given to conventional pulmonary resection. Care with airway pressures and avoidance of overdistention are

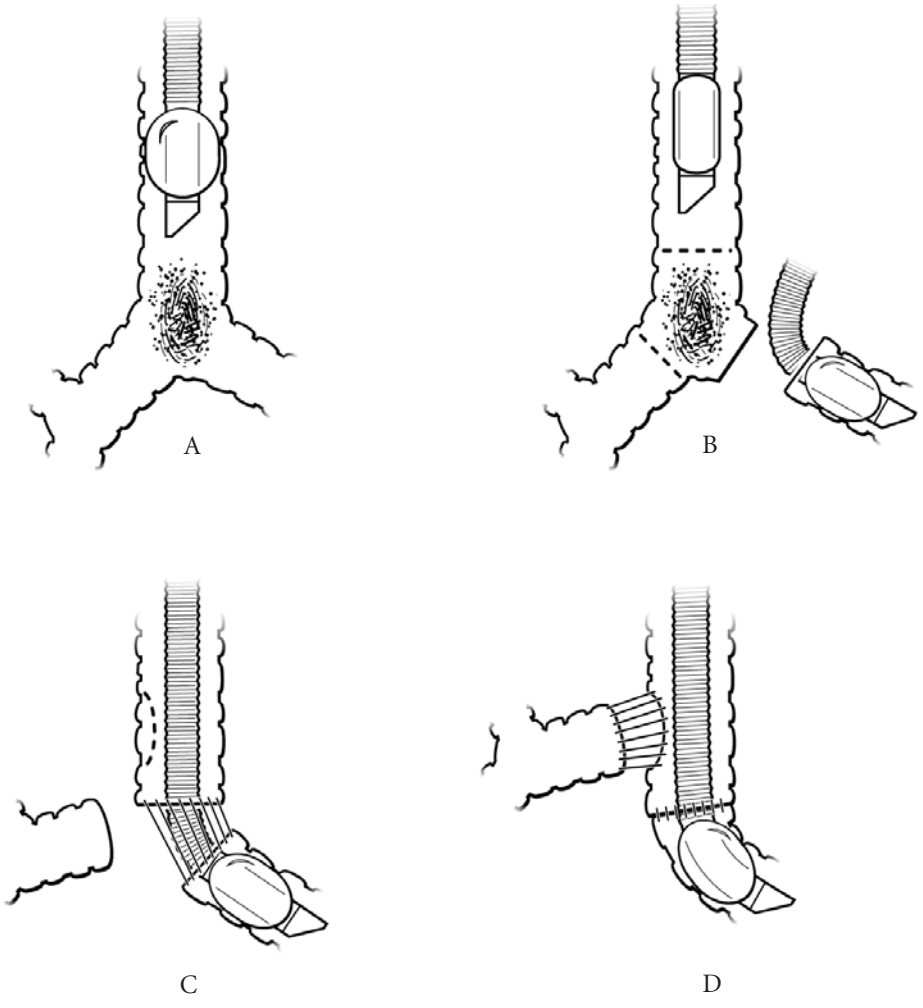

FIGURE **18-2B** *Intubation and ventilation for carinal resection and reconstruction (continued). Lesion involving carina and less than 4 cm of trachea.* A, *A long endotracheal tube (ET) is used as in 18-2A.* B, *Intubation across the operative field into left main bronchus.* C, *The proximal long ET is advanced for completion of anastomosis between trachea and left main bronchus. The* dotted line *in the lower trachea indicates site of orifice to be made for the elevated right main bronchus.* D, *Completion of the end to side anastomosis of right main bronchus to trachea.*

important. In cases where a pneumonectomy is performed, there are also the issues of thoracic volume and shift of the mediastinum, as well as the need for careful fluid management, lower FiO_2, and gentle surgical manipulation.[32,33]

There is little difference in the management of emergence from anesthesia in carinal surgery as opposed to tracheal surgery. Cord swelling is less likely, but obstruction from blood and secretions is more common. Pain control will be a bigger factor; the selection of methods that do not suppress respiratory drive is preferable.

Patients with previous carinal surgery who present for unrelated surgery should not, in general, present particular anesthetic problems. It would be prudent to avoid pushing an endotracheal tube too distally and risk injuring an anastomosis. Most anesthesiologists at centers with experience with these patients would confirm good tube position and an undamaged distal airway with bronchoscopy after intubation. If lung isolation is required for the surgical procedure, the considerations are more involved. As much as possible, it is wise to avoid instrumenting and especially inflating a cuff in a repaired region. The carinal anatomy will be abnormal, with possibly shorter bronchial lengths and different angles of departure for the bronchi. The use of an endobronchial tube may be necessary. In any case, all tube positionings should be done under fiber-optic guidance to avoid the risks of malpositioning.

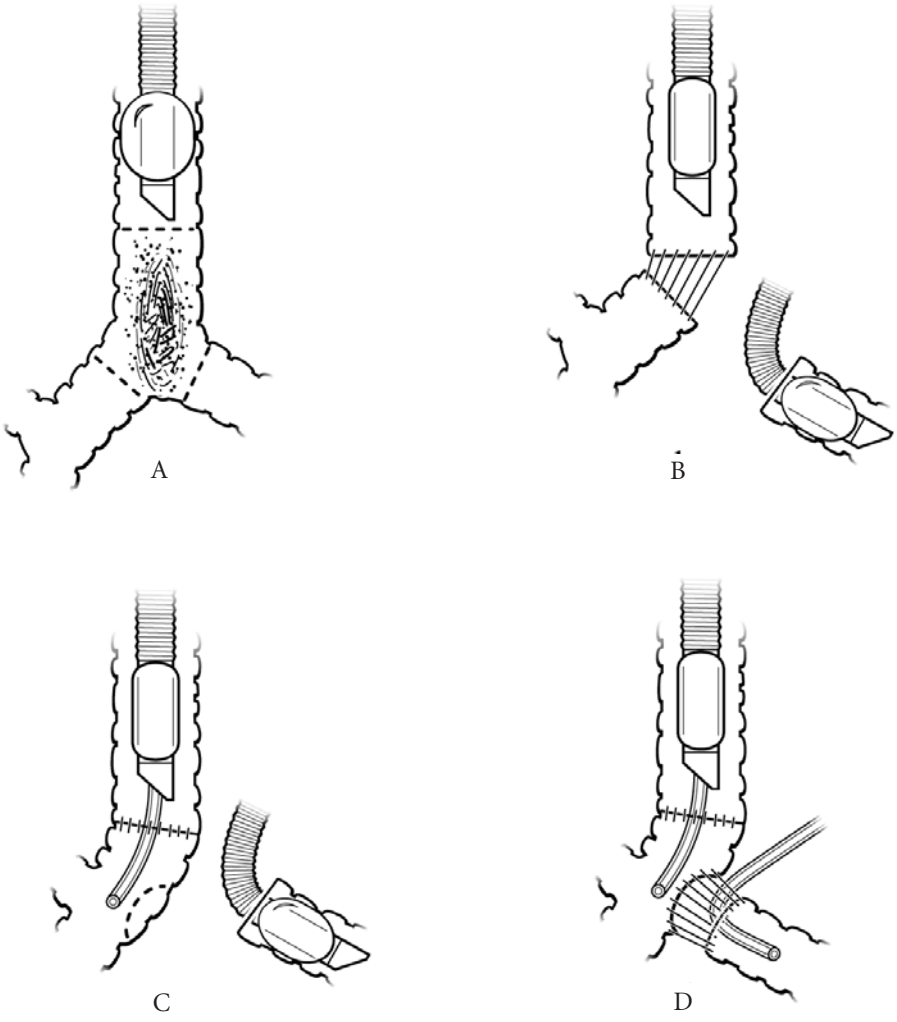

FIGURE **18-2C** *Intubation and ventilation for carinal resection and reconstruction (continued). Lesion involving carina and an extensive segment of trachea.* A, *Ventilation as previously indicated.* B, *Mobilization and anastomosis of right main bronchus to proximal trachea is performed first while ventilation is effected across the operative field in the left main bronchus.* C, *After completion of the first anastomosis, a jet catheter may be passed to provide oxygenation in the right lung.* Dotted line *indicates location of orifice in bronchus intermedius for implantation of left main bronchus.* D, *Endotracheal tube across the field in the left main bronchus may be intermittently removed as sutures are placed. As anastomosis is being completed, a jet catheter may be substituted to provide high frequency ventilation of the left lung.*

Stents

If primary removal or correction of airway lesions is not possible, other means of assuring an airway must be found. These include tracheostomy tubes, T tubes, and stents. [34,35]

Tracheostomy. The standard tracheostomy should be familiar to all practitioners. It can be performed under topical anesthesia in impending airway obstruction, or on a patient with an endotracheal tube in place. The management is similar to reconstructive surgery, except that extubation, neck flexion, and concerns about upper airway obstruction are not present.

The indications for tracheostomy include facilitating controlled ventilation, protection of the airway against aspiration and bleeding, and allowing airway access distal to an obstructed or damaged portion. Tracheostomy is also commonly performed in patients requiring prolonged intubation since it is more comfortable and less injurious to vocal cords than orotracheal or nasotracheal intubation. There has to be special consideration of gas humidification since the function of the nasopharynx is bypassed. If no humidification and cleaning is done, then the tube will eventually become obstructed with secretions. Voice can be preserved via a number of tube modifications, including choosing not to use a cuff or choosing a tube with a special fenestration.

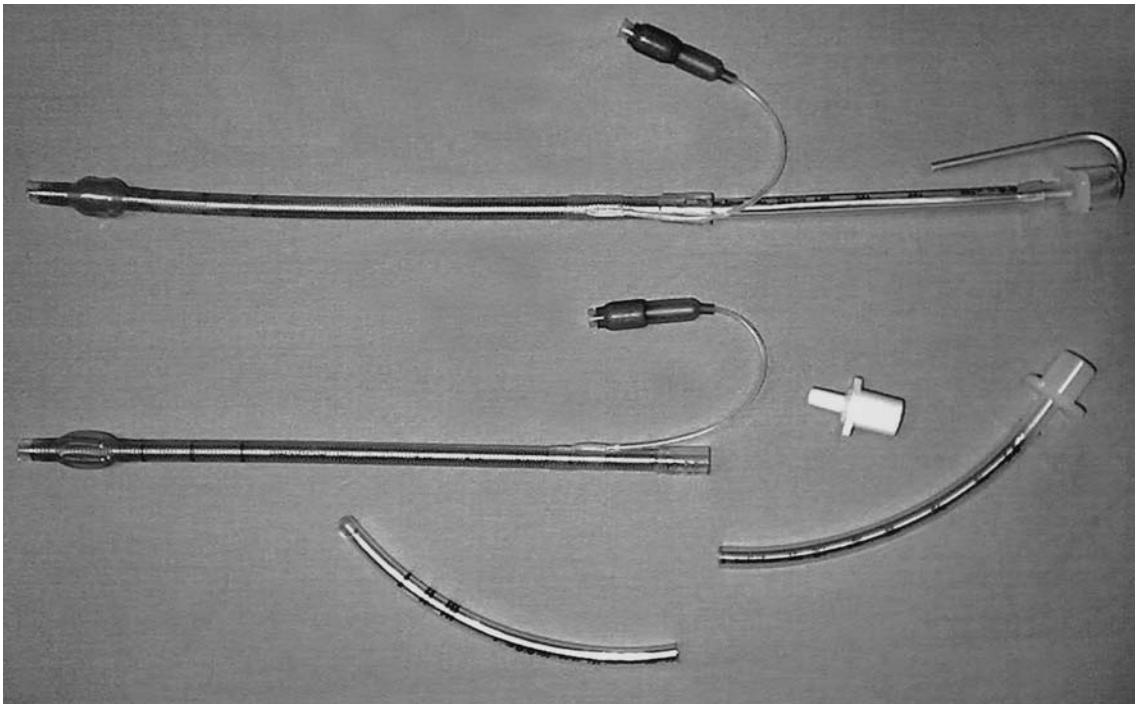

FIGURE **18-3** *Endobronchial tube made from an extended armored endotracheal tube. This technique allows considerable flexibility in length, caliber, and use. Cuff design is not optimal for the right bronchus.*

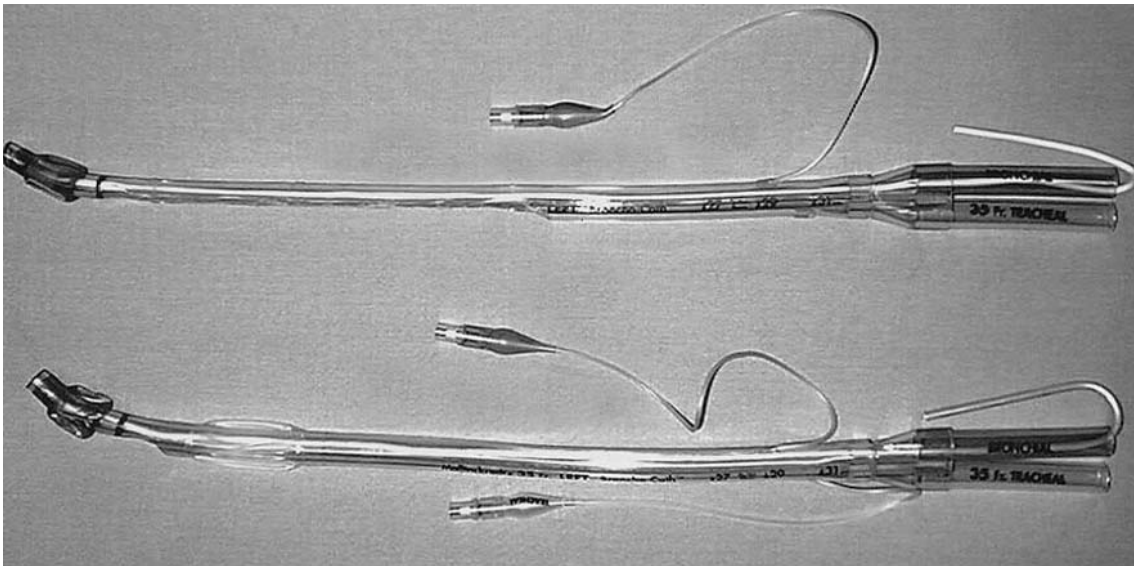

FIGURE **18-4** *Endobronchial tube made from a modified endotracheal tube. The tracheal lumen is removed from the distal portion of the tube. The endobronchial design is excellent, but different for right and left bronchi.*

When presenting for another procedure, a patient with a tracheostomy tube can either be ventilated through the existing tube, or the tracheostomy tube can be exchanged for a cuffed endotracheal tube passed through the tracheostomy stoma. It is unwise to remove a fresh tracheostomy tube since the stoma has not yet healed and the replacement tube can easily enter another tissue plane. If a tube is placed, attention must

be paid to the depth of insertion, since the length to the carinal bifurcation can be deceptively short. The effectiveness of ventilation will also depend on whether there is a cuff in place, or whether the inner cannula that covers the fenestration of a fenestrated tube is in place.

T Tube. The T tube is an uncuffed tube that sits in the trachea, and is held in place by the sidearm through a tracheostomy stoma. It is designed to bridge any diseased portion of the trachea, allowing full voice and mouth breathing. The tracheostomy segment is typically capped, but can be opened if there is a proximal obstruction, and for suctioning (see Chapter 39, "Tracheal T Tubes").

Placement is done in an anesthetized patient, with the folded tube placed through the stoma and springing into proper position. Evaluation via flexible bronchoscopy through the sidearm, and rigid bronchoscopy from above, will also show adjustments that need to be made. The procedure can take many iterations to find the optimal size and length for each segment. At times, the tube may lie in a folded state in the distal trachea, completely obstructing the airway. The anesthesiologist needs to recognize this promptly and provoke correction or removal. There will be extensive apneic periods during placement and manipulation, requiring coordination between the anesthesiologist and the surgeon. In the interlude, a small endotracheal tube can be placed in the stoma, or the stoma can be covered and ventilation accomplished through the rigid bronchoscope.

At the end of the procedure, the patient should be ready to resume spontaneous ventilation. Blood and secretions can precipitate an acute obstruction, so close observation, suctioning, and even removal of the tube may be needed. Although the goal is to cap the sidearm, there may be enough swelling of the larynx after all this manipulation to delay capping for a few hours. Humidified oxygen applied to the stoma will be needed.

If a patient with a T tube in place comes for unrelated surgery, there are several options. Regional techniques, where appropriate, will avoid airway manipulation. An LMA can be placed, and the sidearm left capped. The tube will then perform its function as a tracheal stent. The sidearm can be accessed by removing its cap and placing the adapter to an appropriately sized endotracheal tube. In this case, the trachea is open above, and ventilation will depend on the relative resistance of the two arms. The upper arm can be obstructed by a blocker, placed via laryngoscopy or placed via the sidearm, and cajoled into sitting in the proximal lumen. Finally, the T tube can be entirely removed and replaced with a tube through the tracheostomy. The choice of method should depend on the nature of the procedure and the predicted postoperative course.

Endoluminal Stents. Endoscopically placed stents can be silicone tubes or expanding wire devices (see Chapter 40, "Tracheal and Bronchial Stenting"). The stents are placed directly via a rigid bronchoscope, or using a flexible guidewire placed with a flexible bronchoscope. Rigid bronchoscopy allows ventilation through the bronchoscope, whereas flexible bronchoscopic techniques require intermittent ventilation with a mask, tube, or jet catheter.[36] The major anesthetic considerations, besides the underlying tracheal pathology, is the chance that a poorly positioned stent will completely obstruct the airway. As with T tubes, the stent, once deployed, may need minor corrections to improve its function, so an anesthetic of unpredictable duration ensues. On emergence, patients tend to find the stent irritating, and can cough it out of position. The patient will need to be reinduced and the process restarted. Assessment of the adequacy of air movement, and maneuvers to improve the airway, such as nebulized epinephrine and a short course of steroids, may be needed.

When a patient with a stent presents for unrelated surgery, many of the considerations for patients with T tubes pertain. Regional techniques and the LMA will avoid tracheal manipulation. If it is necessary to secure the airway, all the information available on the stent and the underlying tracheal lesion should be

collected (probably a wise precaution even if intubation is not planned). The location of wire stents can be seen on an x-ray. If the stent is relatively distal, then a tube placed in the proximal trachea may not impinge. The tube should be placed under fiber-optic guidance, and care must be taken not to allow it to move during patient positioning. Although it is possible to intubate into or beyond the stent, there is the real risk of dislodging the stent, either by pushing it distally and causing obstruction, or by inadvertently removing it on extubation and leaving a vulnerable trachea. With time, wire stents become embedded into the tracheal wall, and potentially become more secure, but the extent and safety of this is not yet known.

Conclusion

The anesthetic management of patients is entwined with the surgical management in tracheal surgery. Anesthesia can not be planned in isolation. Nevertheless, a deep understanding of the anatomy of the lesions, and the physics of airflow in the airways, guides our approach. The art of anesthetic management is in knowing when spontaneous versus controlled ventilation is preferable, and in recognizing and responding to an obstructed airway. The focus is much more on ventilation, with hemodynamics, fluid management, and pain control secondary.

What will the future bring? Some changes in anesthetic agents, surely. Fast acting narcotics and sedatives are here; we now need a muscle relaxant with sufficiently transient action to allow recovery of spontaneous respiration when control is unsuccessful. New surgical techniques are also inevitable. We can look forward to continued improvements in diagnostic imaging techniques, stents, and fiber-optic instruments. Perhaps tracheal replacement will be possible. The anesthetic management will need to evolve in step.

References

1. The American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway. A report. Anesthesiology 1993;78:597–602.
2. Eisenkraft JB, Barker SJ. Helium and gas flow. Anesth Analg 1993;76:452–3.
3. Polaner DM. The use of heliox and the laryngeal mask airway in a child with an anterior mediastinal mass. Anesth Analg 1996;82:208–10.
4. Chen K, Varon J, Wenker OC. Malignant airway obstruction: recognition and management. J Emerg Med 1998;16:83–92.
5. Prakash UB, Abel MD, Hubmayr RD. Mediastinal mass and tracheal obstruction during general anesthesia. Mayo Clin Proc 1998;63:1004–11.
6. Ferrari LR, Bedford RF. General anesthesia prior to treatment of anterior mediastinal masses in pediatric cancer patients. Anesthesiology 1990;72:991–5.
7. Johnson D, Hurst T, Cujec B, Mayers I. Cardiopulmonary effects of an anterior mediastinal mass in dogs anesthetized with halothane. Anesthesiology 1991;74: 725–36.
8. Mutoh T, Kanamaru A, Tsubone H, et al. Respiratory reflexes in response to upper-airway administration of sevoflurane and isoflurane in anesthetized, spontaneously breathing dogs. Vet Surg 2001;30:87–96.
9. Heier T, Feiner JR, Lin J, et al. Hemoglobin desaturation after succinylcholine-induced apnea: a study of the recovery of spontaneous ventilation in healthy volunteers. Anesthesiology 2000;94:754–9.
10. Perrin G, Colt HG, Martin C, et al. Safety of interventional rigid bronchoscopy using intravenous anesthesia and spontaneous assisted ventilation. A prospective study. Chest 1992;102:1526–30.
11. Andropoulos DB, Rowe RW, Betts JM. Anaesthetic and surgical airway management during tracheo-esophageal fistula repair. Paediatr Anaesth 1998;8:313–9.
12. Cicala RS, Kudsk KA, Butts A, et al. Initial evaluation and management of upper airway injuries in trauma patients. J Clin Anesth 1991;3:91–8.
13. Alfille PH, Hurford WE. Upper airway injuries [comment]. J Clin Anesth 1993;3:88–90.
14. Devitt JH, Boulanger BR. Lower airway injuries and anaesthesia. Can J Anaesth 1996;43:148–59.
15. Rossbach MM, Johnson SB, Gomez MA, et al. Management of major tracheobronchial injuries: a 28-year experience. Ann Thorac Surg 1998;65:182–6.
16. Huh J, Milliken JC, Chen JC. Management of tracheobronchial injuries following blunt and penetrating trauma. Am Surg 1997;63:896–9.
17. Shorten GD, Alfille PH, Gliklich RE. Airway obstruction following application of cricoid pressure. J Clin Anesth 1991;3:403–5.
18. Flood ME, Astley B. Anaesthetic management of acute laryngeal trauma. Br J Anaesth 1982;54:1339–43.
19. Dowd NP, Clarkson K, Walsh MA, Cunningham AJ. Delayed bronchial stenosis after blunt chest trauma. Anesth Analg 1996;82:1078–81.
20. Godden DJ, Willey RF, Fergusson RJ, et al. Rigid bronchoscopy under intravenous general anaesthesia with oxygen Venturi ventilation. Thorax 1982;Jul:532–4.
21. Ostfeld E, Ovadia L. Bilateral pneumothorax during pediatric bronchoscopy (high-frequency jet injection ventilation). Int J Pediatr Otorhinolaryngol 1984; 7:301–4.
22. Occupational Safety and Health Administration Fact Sheets. Waste anesthetic gases. 1991; Fact sheet no. 91-38. Available at: http://www.osha-slc.gov:80/OshDoc/Fact data/FSN091-38.html.
23. Sheski FD, Mathur PN. Long-term results of fiberoptic bronchoscopic balloon dilation in the management of benign tracheobronchial stenosis. Chest 1998;114: 796–800.
24. Sandberg W. Anesthesia and airway management for tra-

cheal resection and reconstruction. Int Anesthesiol Clin 2000;38:55–75.
25. Pinsonneault C, Fortier J, Donati F. Tracheal resection and reconstruction. Can J Anaesth 1999;46:439–55.
26. Cay DL. Venturi for tracheal reconstruction. Anaesth Intensive Care 1978;6:171.
27. Giunta F, Chiaranda M, Manani G, Giron GP. Clinical uses of high frequency jet ventilation in anaesthesia. Br J Anaesth 1989;63(7 Suppl 1):102S–6S.
28. Mathisen DJ. Carinal reconstruction: techniques and problems. Semin Thorac Cardiovasc Surg 1996;8:403–13.
29. Grillo HC. Carinal reconstruction. Ann Thorac Surg 1982;34:356–73.
30. Horita K, Itoh T, Furukawa K, et al. Carinal reconstruction under veno-venous bypass using a percutaneous cardiopulmonary bypass system. Thorac Cardiovasc Surg 1996;44:46–9.
31. Perera ER, Vidic DM, Zivot J. Carinal resection with two high-frequency jet ventilation delivery systems. Can J Anaesth 1993;40:59–63.
32. Deslauriers J, Aucoin A, Gregoire J. Postpneumonectomy pulmonary edema. Chest Surg Clin North Am 1998;8:611–31.
33. Jordan S, Mitchell JA, Quinlan GJ, et al. The pathogenesis of lung injury following pulmonary resection. Eur Respir J 2000;15:790–9
34. Jones LM, Mair EA, Fitzpatrick TM, et al. Multidisciplinary airway stent team: a comprehensive approach and protocol for tracheobronchial stent treatment. Ann Otol Rhinol Laryngol 2000;109:889–98.
35. Cavaliere S, Venuta F, Foccoli P, et al. Endoscopic treatment of malignant airway obstructions in 2,008 patients. Chest 1996;110:1536–42.
36. Hautmann H, Gamarra F, Henke M, et al. High frequency jet ventilation in interventional fiberoptic bronchoscopy. Anesth Analg 2000;90:1436–40.

Urgent Treatment of Tracheal Obstruction

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A frequent complication and presentation of tracheal disease is obstruction. Failure to recognize critical degrees of obstruction can lead to death. As noted elsewhere, a tracheal obstruction due either to benign stenosis or neoplasia, in patients who have radiologically normal lung fields, is frequently diagnosed as "adult onset asthma," allowing obstruction to reach a critical level. In an emergent state, a small mucous plug or bleeding can easily cause fatal obstruction. Airway obstruction may produce unyielding pneumonia. Prompt treatment is necessary. Acute obstruction due to trauma is dealt with in Chapter 9, "Tracheal and Bronchial Trauma." Obstructions from benign stenosis or neoplasia are each managed differently.

Cicatricial Stenosis

Postintubation stenosis, either stomal or cuff induced, is the most common non-neoplastic cause of severe obstruction. Other causes are idiopathic, post-traumatic, postoperative, postirradiation, post-infectious stenosis, and those due to diseases such as sarcoid, amyloid, and Wegener's granulomatosis. The security of an intensive care unit, good humidification, face mask oxygen or heliox, gentle chest physiotherapy, and mild sedation usually comforts the patient enough to permit radiologic studies. If urgency is very acute, the patient is intubated, suctioned, and ventilated on the Venturi principle with an Ambu bag. This degree of urgency is exceedingly rare. Usually, the patient is simply moved expeditiously to the operating room.

The goals of emergency treatment must be kept clearly in mind. Emergency resection of a lesion is almost never required, although it may be the correct choice if patients' problems are well defined, their physical state is otherwise optimal, no complicating factors are present, and the operating room circumstances are ideal. Even so, a tight stricture should be dilated prior to resection. As a rule, if tracheal stenosis shows a 5 to 6 mm diameter or less, dilation should be done immediately after induction of anesthesia. This permits passage of a small endotracheal tube through the stenosis for safe ventilation. If this is not done, then the patient may well accumulate CO_2, which can precipitate arrhythmia during exposure of the lesion and then require urgent measures. This is completely avoided by insisting on an adequate airway prior to commencing operation.

A second goal of dilation is to allow safe deferral of operation. Reasons for delay are to complete the work-up, to treat concurrent medical problems, to optimize operating conditions and staff, to clear obstructive pneumonia, and to wean from high doses of corticosteroids. The last can severely impair healing of an anastomosis. Patients may be maintained with periodic dilations, if necessary. If dilation is not necessary too frequently, then it may be a preferred treatment in such cases as severe idiopathic stenosis extending to the vocal cords, or severe late irradiation stenosis of the subglottic larynx and upper trachea.

Anesthesia

Emergency tracheal dilation must be done under general anesthesia. Most essential is the complete confidence of the anesthetist in the surgeon's ability to obtain an airway immediately, if demanded. Conversely, the surgeon must have full confidence in the anesthesiologist's ability to handle a difficult airway before and during dilation. Basic monitoring includes electrocardiography, oximetry, and an atrial line. The procedure is carried out under deep inhalation anesthesia (see Chapter 18, "Anesthesia for Tracheal Surgery"). The advantage of inhalation anesthesia is that the patient maintains respiration, thus avoiding dangerous periods of apnea. Furthermore, the patient will breath spontaneously immediately after the procedure. An alternative is the use of the Sanders' apparatus, which provides "Venturi"-type ventilation. Drugs and relaxants that depress respiration are avoided. The surgeon must be present when anesthesia is induced, with all the necessary bronchoscopic equipment at hand. Induction with a 2 to 3 mm airway may take a prolonged time due to slow gas exchange, thus patience is required. At any moment, if the obstruction worsens, the surgeon proceeds immediately to bronchoscopy and dilation, as described below.

Technique of Dilation

The instruments that should be available include rigid bronchoscopes, ranging as follows: 3.5, 4, 5, 6, 7, 8, and 9 mm sizes (Figure 19-1). Jackson bronchoscopes with a ventilating sidearm are preferred. These bronchoscopes have the advantage of a somewhat rounded character to their bevelled tip. This contrasts with the rather sharp, spade-like lip of Storz bronchoscopes (see Figure 5-3 in Chapter 5, "Diagnostic Endoscopy"). The latter, in pediatric sizes, are superb for diagnostic examination in infants and children but are less advantageous for dilation of tight stenoses. Small-sized Jackson-Pilling esophageal bougies should be available. These may be difficult to obtain. Standard bronchoscopic biopsy forceps, both straight and angled, should also be available. A wide suction cannula is essential. A zero degree Hopkins telescope is used.

The lesion is visualized through an adult bronchoscope and secretions are cleared. Dilation is commenced with successively larger bougies. The full length of the bougie is not passed, to avoid distal injury of the membranous wall. At this point, it is sometimes possible to advance the adult bronchoscope over a larger bougie, with a firm but gentle corkscrewing motion, after engaging the bronchoscope's tip in the stenotic orifice. Excessive force should not be used, nor should this be done blindly. The stricture may be far firmer than normal tracheal tissue. If excessive force is used prior to adequate dilation, the bronchoscope could perforate the softer membranous wall just above the stricture. For the same reason, an endotracheal tube must not be forced through a stenosis over a stylet. If there is firm resistance, or if it appears nonfeasible, then the patient should be ventilated briefly, with the proximal end of the bronchoscope occluded either with the thumb or an occlusive window. At any point during these maneuvers, should the anesthetist request that manipulations cease in order to ventilate the patient through the undilated stricture, the surgeon must comply immediately.

Jackson pediatric bronchoscopes are used next. The starting size is chosen after visualizing the stenosis. The smaller pediatric bronchoscopes (3.5 to 5 mm) are passed through the vocal cords with a laryngoscope. A Jackson laryngoscope with a removable slide is ideal for this purpose (Figure 19-2). Other laryngoscopes

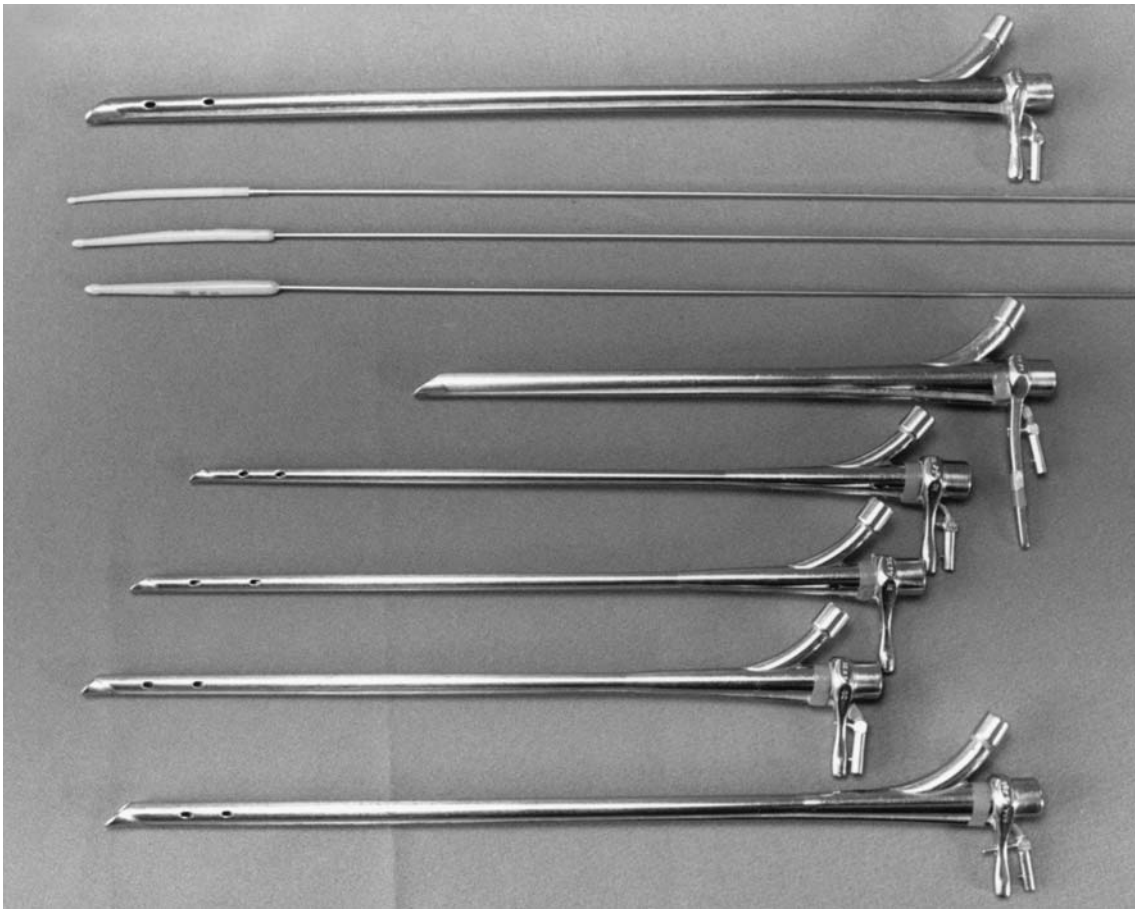


FIGURE **19-1** *Instruments for tracheal dilations. From* top *to* bottom*: adult Jackson ventilating bronchoscope (7, 8, and 9 mm); range of small diameter semirigid plastic esophageal bougies which will pass through a 7 mm bronchoscope; 9 mm by 25 cm ventilating bronchoscope, useful but not essential for treating subglottic stenosis; pediatric ventilating Jackson bronchoscopes (3.5, 4, 5, and 6 mm).*

may be used, with a straight Miller blade being preferred. The slide must be removed from the Jackson intubating laryngoscope so that the laryngoscope can be removed after a short bronchoscope has been inserted. The bronchoscope is introduced through the stenosis with a firm but gentle, slightly rotary movement. Secretions are aspirated as necessary. The patient is immediately ventilated vigorously, with the proximal end of the bronchoscope occluded. With even a 3.5 mm bronchoscope, it is possible to attain entirely satisfactory levels of oxygenation and CO_2 removal. When the anesthesiologist agrees that it is safe to proceed, the next sized bronchoscope is passed. Serial dilations continue until adult-sized bronchoscopes are passed. The surgeon must decide how large a bronchoscope to pass without danger of splitting the trachea. It is almost always possible in adults to carry dilation through a 7 or 8 mm bronchoscope, effectively a 9 to 10 mm diameter. After the airway has been thoroughly cleansed, and the patient is well oxygenated and stable, the last bronchoscope is withdrawn and the patient awakened.

A stenosis, particularly when inflamed, may be divulsed by dilation. Bits of granulation tissue and torn fragments of scar that remain on the tracheal wall are removed patiently and conservatively with biopsy forceps before withdrawing the bronchoscope. I have never encountered excessive bleeding with these maneuvers. Minimal bleeding from biopsy can be easily tamponaded with the bronchoscope, and soon stops. If spontaneous ventilation has been used, it is usually unnecessary to intubate the patient following the procedure. If undue secretions

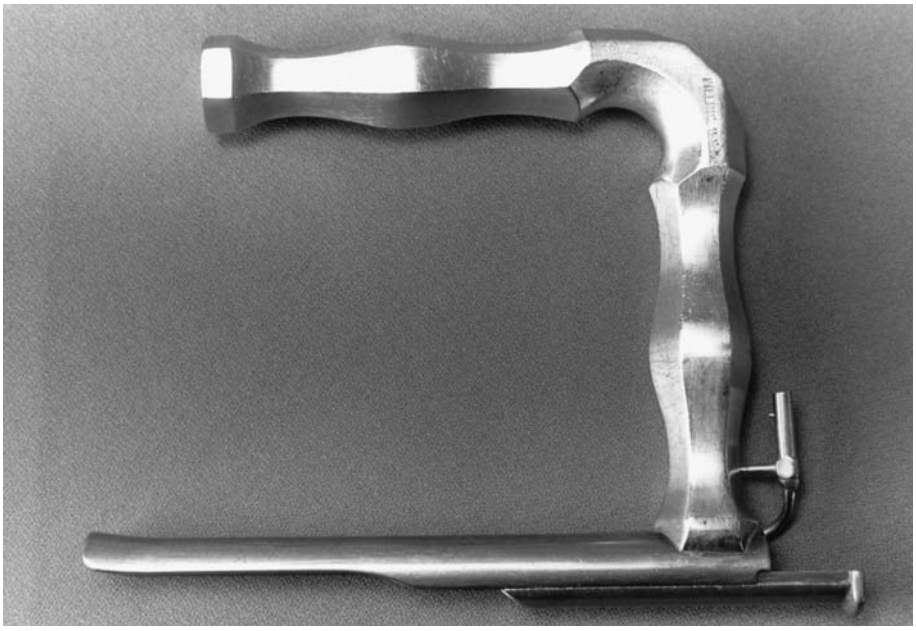


FIGURE **19-2** *Jackson laryngoscope to facilitate rigid bronchoscopy. The slide attached to the blade is removed prior to the introduction of a pediatric bronchoscope, since the latter is too short to allow the slide (and hence the bronchoscope) to be removed after passing the bronchoscope. Because of the small diameter of a 3.5 or 4 mm bronchoscope, the intubating laryngoscope greatly facilitates their passage.*

or blood are present or if the patient breathes poorly, an endotracheal tube is passed through the dilated area for adequate toilet until the patient is fully awake. Ventilation is seldom necessary.

Dilation performed precisely in this stepwise fashion has been completely safe in our hands, in hundreds of patients. Deviations or loss of control could easily lead to neurologic damage or death. The airway that is attained may remain satisfactory in caliber for days to months (Figure 19-3). The length of palliation is highly unpredictable. Subsequent management depends on the therapeutic plan for the basic disease. If patients are expected to need frequent dilations, require a long wait before definitive surgery, will be returning to uncertain medical supervision, or are unsuitable for repair and hence require permanent splinting, then a T tube should be considered.

Stenosis due to postintubation cuff injury responds well in the short term to dilation since the pathology consists principally of a circumferential cicatrix. In contrast, stomal stenosis is the result of contraction of an anterior defect, which pulls the tracheal walls together. The dilating bronchoscope is therefore easily passed through a stomal stenosis, but the walls of the trachea snap back together again as soon as the dilating instrument is withdrawn. The bronchoscope will remove granulomas that may be present on the anterior scar, producing some immediate relief.

A *laser* can be used to trim part of a stenosis adjacent to the tracheal lumen. Granulations can also be removed. Laser excision to any depth risks perforation of an hourglass stenosis into the mediastinum, esophagus, or brachiocephalic artery. In stomal stenosis, lasering to any depth will destroy the contracted tracheal wall. Radial laser incisions followed by dilation have been recommended. There is little to indicate that the laser improves upon direct bronchoscopic dilation for acute obstruction. Repeated laser treatment produces more scarring and can lengthen a previously short lesion. It is not very satisfactorily curative. Experienced practitioners hence advise use of laser definitively only in thin, "web-like" benign stenosis.[1,2] Such lesions are vanishingly rare and also respond to simple dilation urgently and to surgical resection definitively (see Chapter 37 "Laser Therapy for Tracheobronchial Lesions"). Institution of a tracheostomy below a stenotic lesion to permit repeated laser treatments is to be deplored, since it compounds the lesion by damaging a greater length of trachea (see Figure 10-2*A* in Chapter 10, "Tracheostomy: Users, Varieties, Complications").

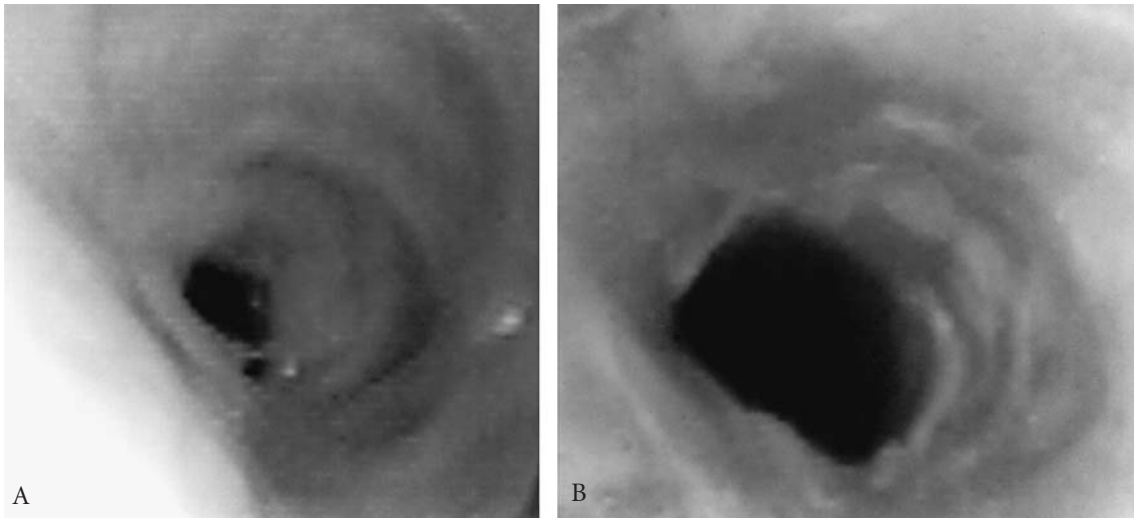


FIGURE **19-3** *Postintubation tracheal stenosis before dilation* (A) *and after bronchoscopic dilation* (B). *Circumferential stenosis (of cuff origin) usually responds to dilation in this way, since a ring of scar tissue is stretched. Stomal stenosis, which results from anterior loss of tracheal substance, is easily dilated, but it usually snaps back when the bronchoscope is withdrawn.*

Tracheostomy should generally not be used to manage acute obstruction. Dilation, performed as described, is preferred, since it does not injure more trachea, which makes future resection more complicated. If tracheostomy must be used, either acutely or to temporize after initial dilation, it should ideally be placed through the stenotic segment if this is accessible in the neck. Future resection will remove both stenosis and stoma without loss of more trachea. If the stenosis is below the sternal notch, and tracheostomy is necessary for some reason, it should be placed at the level of the second ring, the distal stenosis dilated, and a tube introduced that is long enough to pass *through* the dilated stricture.

Neoplastic Obstruction

The problems and management of acute tracheal obstruction due to neoplasia are different from those of benign stenosis. Blockage by a tumor must be removed rather than dilated. Therapeutic goals are l) to facilitate safe anesthesia for resection; 2) to permit delay of resection for study, to clear obstructive pneumonia, to wean from high doses of steroids, and to stabilize medical conditions; and 3) to allow palliative irradiation, external or brachytherapy. If obstruction is due to extrinsic compression by a tumor, an internal stent or T tube stent can provide temporary relief.

Anesthesia

The same considerations apply as for benign stenosis. Communication between the endoscopist and the anesthetist must be complete. For example, momentary interruption of ventilation avoids driving loosened bits of tumor and blood into distal bronchi.

Technique

An airway can always be established promptly in a patient with neoplastic obstruction of the trachea or of the carina. Bleeding studies should be done. If any portion of the tracheal circumference is free of tumor, a rigid bronchoscope will pass beyond the obstruction. Cartilages and tumor yield to the bronchoscope. In the uncommon situation where tumor is completely circumferential, a bronchoscope may be insinuated through the residual tiny lumen with a corkscrewing motion. Blood or tumor debris is removed with a wide-

bore suction tip and the patient stabilized with respect to oxygenation and partial pressure of carbon dioxide (PCO_2). The tumor is initially assessed with a Hopkins magnifying telescope to be certain it is not unduly vascular. Excessively vascular lesions, such as a hemangiomatous malformation, are very rare. Initial minimal biopsy can test vascularity if there is concern. Biopsy in any case is needed if a diagnosis has not previously been established. Excessively vascular lesions or the rare arteriovenous malformations should not be biopsied. Limited tumors can be resected and diagnosis established by frozen section from the specimen.

A coring technique is used to remove obstructing tumor of the trachea, carina, or main bronchus.[3] A rigid bronchoscope of adult size (7 to 9 mm) is passed through the tumor with a rotary notion. The bevelled tip of the Jackson bronchoscope is favored. The axis of the airway must be kept in mind to avoid penetrating the tracheal wall, which may be completely replaced by tumor. Special care must be taken at the carinal spur. Potential exists, as with a laser, for injury to the pulmonary artery or other major structures. Detached pieces of tumor are quickly removed with suction. Coring is continued until a satisfactory airway is obtained (Figure 19-4). The bronchoscope is periodically occluded for ventilation between coring and suctioning. After a major amount of tumor has been removed, the biopsy forceps are used to further trim the tumor, to establish a satisfactory airway. If a very large chunk of tumor is detached, it can be held against the tip of the bronchoscope with the suction tip, and the bronchoscope withdrawn along with the suction tube and tumor. The bronchoscope is reinserted. Bleeding is not often a problem, but it can usually be controlled by bronchoscopic pressure. Saline irrigation clears the field as necessary. If bleeding seems excessive, the following may also be useful: epinephrine-soaked pledgets (0.l mg/mL) on long applicators, to stop oozing. Coagulation with long insulated electrodes or a laser has not been necessary, nor has an endotracheal tube with a tamponading balloon. These have not been required in our practice. A Foley venous occlusion catheter can also be used for bleeding distally in the bronchial tree. Coring would be inadvisable in an anticoagulated patient. Insertion of an endotracheal tube would be appropriate until coagulation levels are controlled.

At conclusion, the airway is carefully aspirated, checked for bleeding, and the patient is awakened. If anesthesia is profound, the patient should awaken with an endotracheal tube in place. The airway can be easily suctioned or irrigated. The tube is removed when the patient is sufficiently awake to demonstrate a normal protective cough reflex. The patient is best observed in a respiratory care unit for a few hours.

Clinical Experience

We have used this simple, direct, efficient, safe, and low-cost procedure for over 35 years. In more recent years, a large literature has grown recommending use of the laser to treat obstructed airway tumors. It is based on the argument that bleeding will be excessive without use of the laser. It has even been argued that it is impossible to clear obstruction of the airway without a laser. We therefore examined a consecutive series of 56 patients with tumor treated by the coring technique.[3] All were symptomatic, with shortness of breath or dyspnea on exertion (88%), hemoptysis (45%), or obstructive pneumonia (in 18 patients). In 23%, coring was done emergently. In 15%, it was performed urgently because of obstructing pneumonia, and in 62%, it was carried out electively. Tumors were distributed widely: 16 occurred in the trachea, 24 at the carina, 8 in main bronchi, and 8 in lobar or segmental bronchi. Squamous cell carcinoma was most common at all levels, adenoid cystic was next in frequency, and the others were a variety of primary and secondary tumors including thyroid carcinomas, carcinoids, mucoepidermoid carcinomas, sarcomas, lymphomas, and metastatic lesions. Twenty-nine percent of patients ultimately went on to surgical resection. Sixty-one percent had unresectable disease that was later treated with radiotherapy, chemotherapy, and combined modalities. Six patients, following failure of prior radiotherapy and chemotherapy, had no further adjunctive therapy. Two refused additional therapy.

Complications were as follows: Five developed pneumonia in previously unaffected pulmonary parenchyma, after relief of postobstructive pneumonia. All responded to chest physiotherapy and antibiotics.

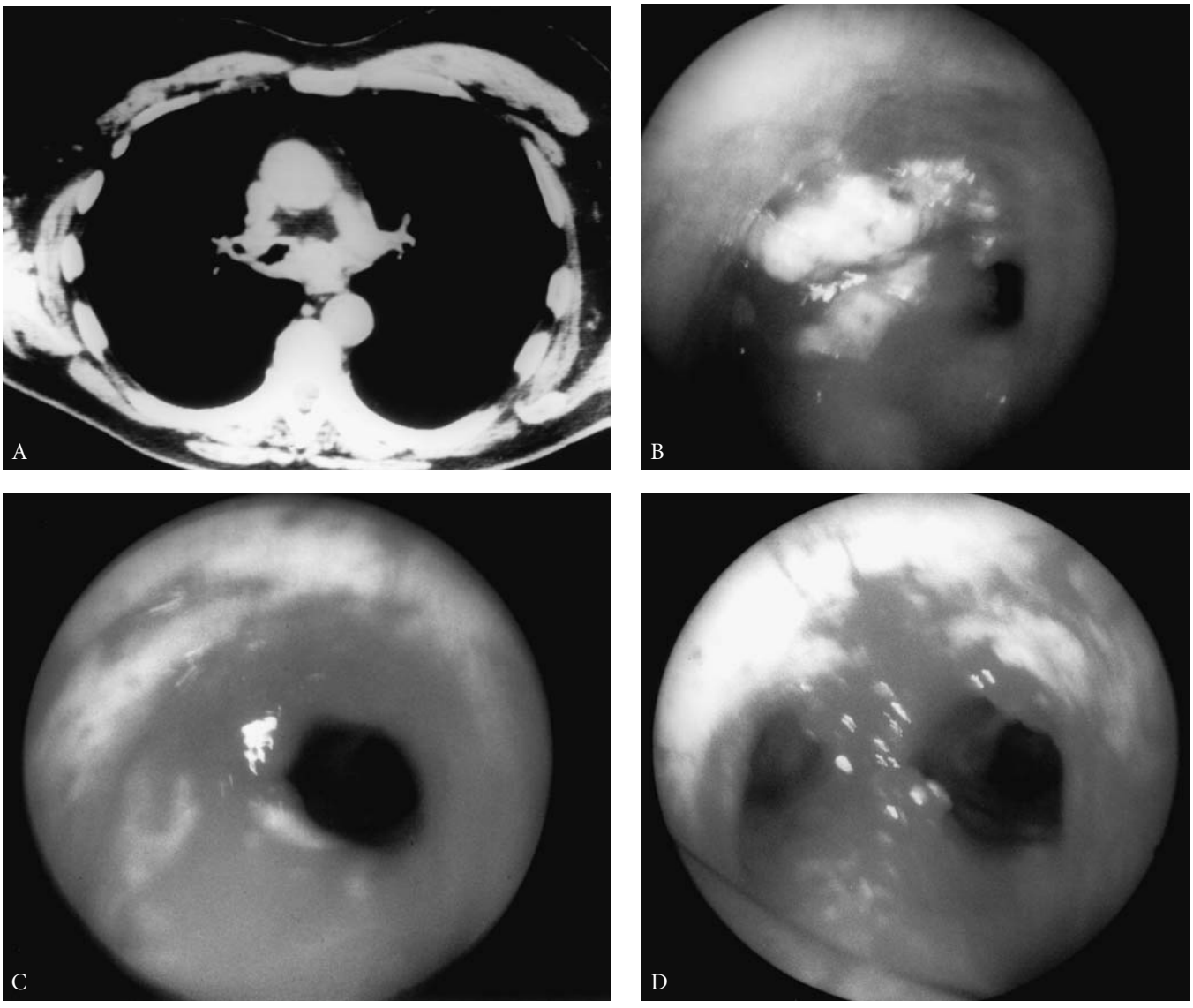


FIGURE **19-4** *Relief of carinal obstruction due to tumor.* A, *Computed tomography scan demonstrating complete obstruction of the left main bronchus and partial obstruction of the right by squamous cell carcinoma.* B, *Bronchoscopic view showing the severity of blockage.* C, *Right main bronchus opened by bronchoscopic core-out.* D, *After both main bronchi have been opened. Tumor remains at the bifurcation to avoid the danger of perforation into the mediastinum. Carinal resection and reconstruction followed.*

Three had bleeding of slightly greater amounts than usual, but none in excess of 500 mL. Bleeding was controlled conservatively in all. Pneumothorax was seen in 2 patients and 1 patient required a chest tube. Two developed hypercarbia and hypoxia and needed brief intubation (< 24 hours). Minor arrhythmia occurred during the procedure in some patients. Six required pharmacological treatment. One patient developed laryngeal edema requiring racemic epinephrine and a brief dosage of steroids. None required tracheostomy. Long-term results depended, of course, on the individual's basic disease and not on the method of unobstructing the airway.

The use of the laser to relieve airway obstruction due to neoplasm has progressed from initial tedious, repeated sessions of charring the tumor followed by scraping away of the char, to use of the rigid bronchoscope

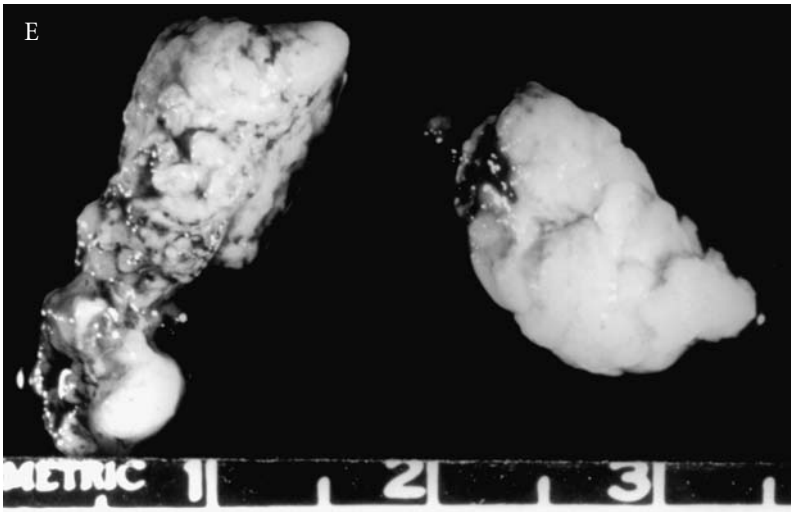
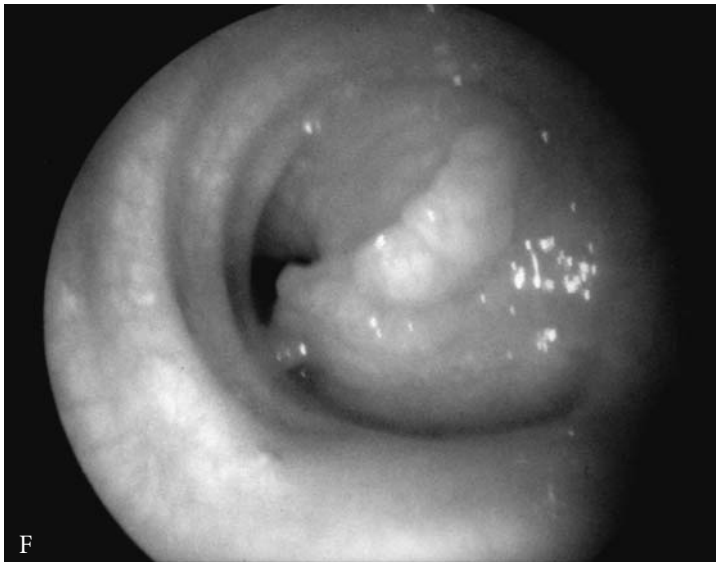
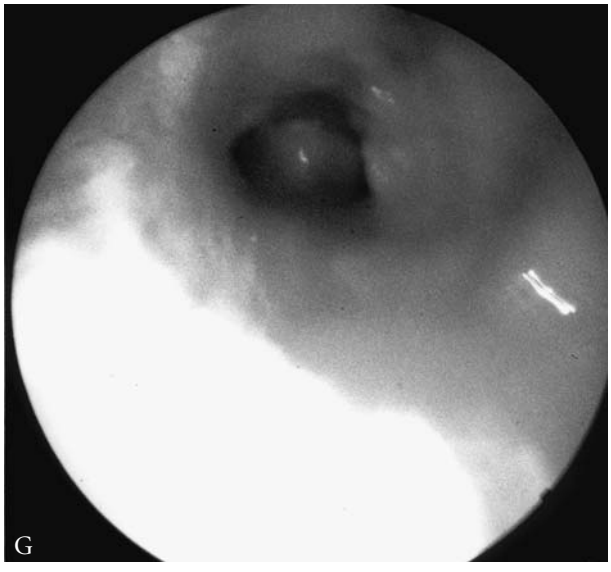


FIGURE **19-4** (CONTINUED) E, *Principal cores of tumor removed from the carina and main bronchi.* F, *A second patient with adenoid cystic carcinoma at the carina. The patient was on high doses of prednisone following misdiagnosis as "asthma."* G, *Following bronchoscopic coring, both main bronchi are open. A period of rapid weaning from the prednisone followed, prior to carinal resection and reconstruction, which was accompanied by stress doses of corticosteroids.*





to core out the tumor, much as described above. The base from which the tumor has been cored is coagulated with the laser. Experience suggests that the results would be equally as good without the final coagulation!

A further concern must be voiced about the consequences of using laser therapy for clearing airway obstructing tumors. Treatment is frequently given by pulmonologists, otolaryngologists, and even thoracic surgeons, who are unaware of the possibilities of curative surgical therapy. Undue delay in the definitive surgical treatment of these patients has followed. In some, whatever opportunity might have existed for cure has been lost. Also evident in the literature is the misconception about the curative potential of the laser for tracheal tumors. With few exceptions, pathologic study of primary tracheal tumors, and certainly of any with malignant potential, makes it clear that endoscopic cure would require removal of the entire thickness of the tracheal wall. Infatuation with technology sometimes obscures basic science and common sense.

References

1. Dumon VF. YAG laser bronchoscopy. New York: Praeger; 1985. p 71–6.
2. Toty L, Personne C, Colchen A, et al. Laser treatment of postintubation lesions. In: Grillo HC, Eschapasse H, editors. International trends in general thoracic surgery. Vol. 2. Philadelphia: WB Saunders; 1987. p. 31–8.
3. Mathisen DJ, Grillo HC. Endoscopic relief of malignant airway obstruction. Ann Thorac Surg 1989;48:469–75.

Postoperative Management

Hermes C. Grillo, MD

In the Operating Room

With proper conduct of anesthesia and of the operation, most patients who undergo cervical or cervicomediastinal approach to the trachea may be extubated in the operating room at the end of the procedure. This requires that patients maintain spontaneous respiration throughout the procedure or, if assisted during portions, that they return to spontaneous respiration as the procedure draws to a close. Frequent suctioning is necessary throughout the operation and just prior to completing the anastomosis. At the end, thorough suctioning is done through the endotracheal tube and particularly after carinal resection by flexible bronchoscopy. It is preferable also that all patients who undergo airway reconstruction, and especially carinal resection, should be run on the dry side with respect to intravenous fluids. The patient is observed in the operating room until ready for extubation. Extubation is usually possible except in some patients who have undergone carinal resection and reconstruction. This is indeed the best time to observe for the absence of airway obstruction. Flexible bronchoscopy is performed to check the anastomosis and to look for laryngeal edema. Patients who are most likely to demonstrate a degree of airway obstruction at this point are those who have undergone a complex laryngotracheal reconstruction for inflammatory stenosis. In some patients, the remaining subglottic larynx is abnormally narrowed, and manipulation may result in laryngeal edema. Decadron is given to these patients in anticipation of edema, even where there is no significant obstruction. If the airway is insufficient, then the patient is reintubated with a small-bore endotracheal tube, preferably uncuffed or with the cuff deflated. The smallest diameter is selected, through which the patient may breathe safely, to avoid pressure trauma to the swollen glottis. The place of tracheostomy is discussed in Chapter 25, "Laryngotracheal Reconstruction."

Ventilation for 12 to 24 hours postoperatively may be necessary after prolonged transthoracic operations. The endotracheal tube is positioned with care so that the cuff or tube tip does not rest against the anastomosis. After carinal resection, the tube is seated in the proximal trachea. The position of the tube is checked with a flexible bronchoscope, the tube taped in place, and note made of the length of tube that must be inserted if it needs to be replaced. If ventilation is required after upper- or midtracheal reconstruction, then the

cuff is seated either above or below the anastomosis. The cuff is inflated with the minimum volume of air needed to obtain a seal. An inflated cuff adjacent to a fresh anastomosis may induce inflammation.

Initial Intensive Care

Most patients who undergo anterior approaches for tracheal resection do not require high levels of intensive care, but they must be observed in a setting where the staff understand the problems of airway reconstruction. Problems may occur quickly and require immediate action to clear an obstructed airway or intubate expeditiously. In our hospital, patients move from the operating room to a respiratory intensive care unit, and finally to a thoracic surgical nursing unit.

The patient is provided humidity and oxygen by a face mask, or if a T tube or tracheostomy tube is present, via a tracheostomy mask. Secretions must not be allowed to dry in an uncapped T tube. Frequent and vigorous chest physiotherapy provided by intensive care unit nurses and chest physiotherapists keeps the airway clear. Patients receive preoperative instructions in these techniques and in the expectations of performance. The trachea is suctioned when necessary using gentle technique. After carinal resection, a firm catheter should not be thrust into the trachea. Flexible bronchoscopy is performed whenever necessary to clear stubborn secretions. Segmental or lobar collapse on an x-ray is indication for aspiration with a flexible bronchoscope. Bronchoscopy is not often required, except after carinal reconstruction. Generally, the lower the anastomosis, the more difficult it is for a patient to clear secretions spontaneously in the first days postoperatively. Aspirates are cultured.

Decadron is often, but not routinely, given for the first 24 to 48 hours after laryngotracheal reconstruction. Further administration is less likely to be helpful and can interfere with healing. Racemic epinepherine is also used if laryngeal edema is present.

The patient is extubated as soon as the respiratory mechanics and blood gas levels are suitable. Extubation is better done early in the day, so that the patient's response may be observed closely, especially if intubated for edema. If extubation is done in the postoperative unit, provision is made for prompt and expert reintubation, should the airway be inadequate. If a patient fails an extubation that had been necessary for glottic or subglottic edema, it is best to wait 4 or 5 days for a further trial. If extubation is then successful, the patient is returned to the care unit. If unsuccessful, the patient is reintubated, anesthetized, and tracheostomized, usually in a preselected location (see Chapter 25, "Laryngotracheal Reconstruction"). Brief general anesthesia is advised because tracheostomy may be difficult in this 5-day-old operative field, even when the site for stoma has been marked in advance.

Patients are monitored in the usual fashion. Oxygen saturation is observed. An arterial line, placed at the time of surgery, allows blood gases to be checked regularly. Additional monitoring is determined by the condition of the particular patient. A chest x-ray is at first obtained daily, particularly in transthoracic cases. If the initial chest x-ray is clear after an uneventful upper tracheal resection, it is repeated only on indication. The chest x-ray is examined for areas of pulmonary collapse or infiltration and for pneumothorax, which although rare, can occur even after an anterior approach.

The neck is maintained in a position of slight flexion by the guardian chin suture and supporting pillows. The goal is to prevent hyperextension rather than to seek hyperflexion. The suture remains in place for about a week on a purely empirical basis.

Antibiotics, begun when the patient is placed on call for the operating room or as anesthesia is started, are continued perioperatively or longer if the wounds were exposed to contaminants from a long-standing tracheal stoma or granulation tissue. Infection has been surprisingly rare.

Continuing Postoperative Care

Feeding is withheld for the first 24 to 48 hours. Liquids are then begun with caution. A number of factors may encourage aspiration on deglutition. A markedly shortened trachea can interfere with tracheal elevation

and epiglottic closure during swallowing, particularly in older patients. Surgery close to the subglottic level of the larynx may also make swallowing difficult for a time. Glottic function may be abnormal preoperatively in some patients, and certainly postoperatively. Vocal cord paralysis, due to sacrifice of the recurrent nerve because of invasion by tumor, to preoperative inflammatory change, or because of operative injury, hinders the protective reflex of glottic closure during swallowing. Laryngeal release, even of the suprahyoid type, may also lead to aspiration despite care to avoid trauma to the superior laryngeal nerves. Since this procedure is useful in patients undergoing extensive tracheal resections, the factors may be additive.

If the patient aspirates clear liquids, then it is better to wait several days before trying again. If aspiration still occurs, then gelatin is fed. This does not cause much irritation if aspirated, and is often more easily swallowed than thin liquids. The problem is seen especially after laryngeal release. Patients are managed by slow progression of feeding beginning with gelatin, then custard and soft solids, followed by thick liquids, eventually solids and, finally, thin liquids such as water or wine. Recovery, most probably due to cicatricial reattachment of muscles to the hyoid bone, may take weeks or months. In some patients, temporary feeding via gastrostomy is necessary.

Ambulation is begun at once with all patients. Chest physiotherapy is continued as necessary throughout hospitalization.

If a change in respiratory pattern occurs, such as dyspnea, wheezing, stridor, or cough productive of unusual sputum or blood, then a prompt flexible bronchoscopy is in order. If recovery is uneventful, bronchoscopy is performed on the sixth postoperative day. It can usually be accomplished under local anesthesia, with sedation if need be. A few patients will require a brief general anesthesia. Very brief visualization of the anastomosis should be sufficient to establish whether healing is proceeding satisfactorily and if hospital discharge is appropriate. Formerly, I obtained a limited series of tracheal x-rays or carinal tomograms to assess healing. I found that a patient can suffer limited anastomotic separation that is not detectable on x-ray because minimal granulation tissue forms in the defect very early and contraction occurs only later. If bronchoscopy is satisfactory, there is no need for radiologic imaging at this time. I prefer that patients who are discharged after a major tracheal reconstruction this early remain in the local area for a few days. They are examined in the office before departing for home. The signs, symptoms, and management of postoperative complications are outlined in Chapter 21, "Complications of Tracheal Reconstruction."

Patients are instructed to move their heads and necks normally but to avoid active extension of the neck for another week. Thereafter, they can return as quickly as they feel able to normal activities. Strenuous exercise is limited for 1 month.

Follow-Up Care

The patient is seen 1 month and again 2 months following discharge, either by the surgeon or by the referring doctor. If the patient has no indication of airway difficulty, then x-rays of the trachea are obtained 2 months following surgery. If the lesion was a benign stenosis with a good mechanical result from surgery, and all is well clinically and radiologically, then the patient may be discharged, but is cautioned to report at once if there is any suggestion of airway difficulty. If there is any question of incomplete healing or stenosis, then bronchoscopy should be repeated.

In patients with benign tumors, even where margins were clear, it seems wisest to follow them in 6 months and in 1 year with additional x-rays and clinical review. At the 1-year visit, a bronchoscopy is in order. In patients who have had either frankly malignant tumors or tumors of low-grade invasiveness, a bronchoscopy should be done at 6 months and again at 1 year. Bronchoscopy should continue annually thereafter. Patients with adenoid cystic carcinoma should have bronchoscopy done indefinitely. The first patient for whom I performed extended carinal resection for adenoid cystic carcinoma was followed for 10 years with no evidence of recurrence. Her initial specimen had shown negative microscopic margins and

lymph nodes. Seventeen years following the resection, she returned with a large local recurrence at the suture line. Earlier detection might have improved her treatment.

Patients who have had squamous cell carcinoma of the trachea should be followed in a similar fashion. Their recurrence pattern is similar to that of squamous lung cancer; if the lesion has not recurred within 5 years, it is unlikely to do so. However, these patients are at risk for the development of other squamous carcinomas of the respiratory and upper digestive systems. Annual bronchoscopy should include panendoscopy of all susceptible areas, and not of the trachea alone.

I advise postoperative irradiation, commencing 1 month after resection of a malignant tracheal tumor. X-rays or a bronchoscopy should be obtained prior to the irradiation to exclude anastomotic stenosis. Anastomotic revision is preferably done prior to the irradiation.

Complications of Tracheal Reconstruction

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The complications of tracheal reconstruction may be many, they may be severe, and they may be fatal. As tracheal surgery has developed over the years, the causes of many complications have been identified, so that it is now possible to eliminate some altogether and to minimize the occurrence of others.[1,2] Complications follow this type of surgery too frequently when operation is performed by an inexperienced surgeon, particularly if the surgeon has not gone through the trouble to become thoroughly informed. Inexperience may produce technical failures, but it also brings failure from inadequate appraisal of the extent or nature of a lesion or of the presence of accompanying pathology, such as laryngeal inadequacy. An individual's surgical "learning curve" should not recapitulate that of the development of tracheal surgery. Division of a series of the first 279 tracheal reconstructions that I performed for postintubation lesions between 1962 and 1982 into consecutive halves shows 4 deaths in the first half compared with 1 in the second, 13 failures in the first group compared with 7 in the second, and a drop in complications from 42 to 30 in the respective halves.[2] The lessons learned are transferable. Needless to say, complications occur in any type of surgery, even with mature judgment and the most meticulous attention to technique, but should be reduced in number and kind. Nonetheless, the small volume of tracheal operations overall does make it difficult for the occasional operator to recognize subtle variations in problems.

In addition to correct appraisal of the extent of a lesion, refined judgment in selecting the operative procedure, meticulous surgical technique, and the pathology of the lesion itself may affect the surgical result.[3,4] Tracheal tissue beyond the margins of a tumor is usually normal, and heals predictably, as long as anastomotic tension is not excessive. With postintubation lesions, however, considerable inflammation is often present proximal to a stenotic lesion. The possibility of microscopic extension of a malignant neoplasm may prompt the surgeon to perform an excessively long resection. Although a more aggressive approach is justified for malignant neoplasms, surgical limits must be carefully assessed. Anastomosis in wholly normal trachea may be impossible. A surgeon must always keep in mind that most postintubation or inflammatory lesions can be managed successfully and permanently, if necessary, by dilation and insertion of a silicone T tube. Serious complications or fatality are unacceptable in these patients.

Complications and Their Causes

The incidence and distribution of complications were reviewed in our first 365 patients with either primary ($n = 56$) or secondary neoplasms ($n = 30$) or with postintubation lesions ($n = 279$) of the trachea, who underwent resection (Table 21-1).[2] As earlier pointed out, this included a "learning curve" from the first patients of the series, when surgical techniques were not developed, which was reflected in decline in mortality, failure, and number of complications found in later patients of the series. The complications of tracheal reconstruction were grouped into three categories: 1) complications due to failure of diagnosis; 2) complications due to failure of technique; and 3) additional miscellaneous complications. These are discussed in this order below.

Postoperative complications have been further analyzed in reports of surgical series, classified by disease and also by type of operation, as our experience grew. By 1995, a total of 503 patients had undergone reconstruction for *postintubation lesions*, including those previously reported.[3] Five required immediate

Table 21-1 Complications of Tracheal Resection

	Failure of Diagnosis	
	Number	
Glottic incompetence	2	
Residual malacia	3	
Extent of stenosis	—	
	Failure of Technique	
	Postintubation	*Neoplasms*
Granulations	28	10
Separation	4	6
Air leakage only	—	1
Stenosis		
Partial	6	3
Complete	15	—
Hemorrhage	2	1
Tracheoesophageal fistula	1	—
Esophagocutaneous fistula	—	1
Cord dysfunction	5	3
Aspiration	1	—
Hypoxemia	—	1
Necrosis	—	—
	Other Complications	
	Postintubation	*Neoplasms*
Wound infection	6	—
Laryngeal edema	1	—
Respiratory failure	—	2
Pneumonia	—	2
Persistent stoma	5	—

Data from Grillo HC et al.[2]

Indications for resection: primary tumors 56, secondary tumors 30, postintubation lesions 279.

reoperation because of residual malacia. Thirteen restenosed and underwent subsequent re-resection. Anterior cricoid resection was necessary in 62 patients (13%) for subglottic extension of stenosis. In 117 patients, anastomosis was made to the cricoid cartilage. Minor complications increased with higher levels of anastomosis (from 16 to 21%) but major complications remained in the same range. The complications noted in this grouping are listed in Table 21-2. In a later study of reconstructions performed for prior unsuccessful repair of postintubation stenosis in 75 patients (59 referred patients), 39% suffered major complications compared with 15% after primary tracheal resection and reconstruction.[5] Eleven were due to nonabsorbable sutures, which have since been abandoned. Laryngeal release, more frequently needed in these patients because of a shortened trachea, increased the incidence of complications, including vocal cord dysfunction and aspiration. Despite these, good and satisfactory results were achieved in almost 92%.

In 82 resections performed for *primary tracheal tumors*, excluding carinal lesions, 2 patients required late reoperation for separation and stenosis, 2 had unpredicted vocal cord paralysis, several had granulation tissue (before absorbable Vicryl was used), 1 died from pneumonia (the only death), and 1 had an esophagocutaneous fistula.[4]

In contrast, *carinal resection and reconstruction* is fraught with serious complications and mortality (Table 21-3).[4,6] In 134 resections by a variety of techniques, with and without pulmonary resection (lobar or total) for primary and secondary tumors (bronchogenic carcinoma), infections, and inflammatory and idiopathic lesions, the mortality rate fell from 16% to 9% from the first to second halves of the series. Early deaths were due to acute respiratory distress syndrome (ARDS), particularly after carinal pneumonectomy. Late deaths were due principally to anastomotic complications where attempt was made to salvage a lobe, or after extensive tracheal resection with carinal pneumonectomy.

Table 21-2 Complications of Operations for Postintubation Stenosis

	Major	*Minor*	*Total*
Granulations			
Before 1978	10	34	44
After 1978	1	4	5
Dehiscence	28	1	29
Laryngeal dysfunction	11	14	25
Malacia	10	0	10
Hemorrhage	5	0	5
Edema (anastomotic)	3	1	4
Infection			
Wound	7	8	15
Pulmonary	5	14	19
Myocardial infarction	1	0	1
TEF	1	0	1
Pneumothorax	0	3	3
Line infection	0	1	1
Atrial fibrillation	0	1	1
DVT	0	1	1
Totals	82	82	164

Reproduced with permission from Grillo HC et al.[3]

DVT = deep venous thrombosis; TEF = tracheoesophageal fistula.

Table 21-3 Complications in Primary Carinal Resection and Reconstruction

Complication	N	*% of 134*
Anastomotic *†	23	17.2
Atrial arrhythmias	20	14.9
Pneumonia	11	8.2
Acute respiratory distress syndrome	10	7.5
Reintubation ‡	3	2.2
Vocal cord paresis	3	2.2
Gastrointestinal bleeding	3	2.2
Empyema	3	2.2
Lobar collapse	2	1.5
Fever	2	1.5
Cardiac herniation	1	0.8
Phrenic nerve paresis	1	0.8
Candidal esophagitis	1	0.8
Arterial embolus	1	0.8
Aspiration	1	0.8
Quadriplegia	1	0.8

Reproduced with permission from Mitchell JD et al.[6]

* Necrosis, separation, stenosis, bronchial mucosal slough, excessive granulation tissue formation.

† Includes early and late morbidity.

‡ After respiratory failure, not secondary to pneumonia or acute respiratory distress syndrome.

Failure of Diagnosis

If a preexisting *glottic incompetence* is not recognized by careful preoperative examination, the patient may present postoperatively either with airway obstruction from adducted vocal cords or, in some cases, with aspiration due to a paralyzed cord, possibly compounded by a lengthy tracheal resection, laryngeal release, and the advanced age of the patient. It must be remembered that patients who acquired tracheal stenosis from intubation through a tracheostomy would usually have also been intubated previously with an endotracheal tube. Glottic injury may have resulted. Also, with or without prior tracheal resection, the recurrent laryngeal nerve may have been injured. Anterior or posterior commissural glottic stenosis, or vocal cord granulomas or polyps, can also be problems if discovered postoperatively. *Aspiration* may also preexist and its origin may be neurological. This possibility must be explored in patients with known neurological damage resulting from head trauma or stroke. Prior extensive tracheal surgery, particularly with vocal cord dysfunction, may favor aspiration. If a patient has suffered failure in repair of a tracheoesophageal fistula, he/she may well have an enteral feeding tube, which will mask aspiration.

Tracheomalacia may be present, proximal to a cuff stenosis. If not identified by fluoroscopy or bronchoscopy, and not accounted for in the surgical procedure, the residual malacia may cause obstruction following resection of the stenosis alone.

Particularly grievous is the failure to identify the actual *extent of stenosis* or tumor. Pitfalls in preoperative appraisal have been discussed in chapters on radiologic and endoscopic diagnosis. Inaccurate assessment of the length of a lesion may force the surgeon in either one of two undesired directions: 1) excessive resection, which leads to tension and separation; or 2) inadequate resection, which results in restenosis or recurrent tumor.

Failure of Technique

Granulation tissue can form at the anastomosis. This was most commonly seen previously around unabsorbable suture material of various types. My early experience included use of Tevdek, Dacron, Mersilene, Prolene, and nylon. The sutures, even though knotted outside of the tracheal wall, would work their way into the lumen over time. Sometimes, a complete ring of granulation tissue would follow, most likely in patients who showed florid inflammation adjacent to the stenosis at operation. This ring could progress to stenosis. Suture line granulomas have largely disappeared since the introduction of Vicryl polyglactin 910, a fine, synthetic, absorbable suture. After Vicryl was adopted for anastomosis (4-0 in adults, 5-0 in infants and children), the incidence of granulations fell from 23.6% to 1.6%. Granulations understandably are more likely to form with the more complicated laryngotracheal reconstruction, but have become more easily managed bronchoscopically since nonabsorbable sutures were abandoned. The monofilament polydioxanone suture (PDS) was briefly tried and discarded because it has no true biological advantage and is more difficult to use. Although experiment has described less early histologic inflammation with PDS than with Vicryl, in practice the latter has produced nearly perfect final results, which is ultimately what matters.[3] Granulation tissue may also occur uncommonly at the site of a tracheal stoma that was left to close postoperatively, or was closed with a muscle flap (where the cutaneous inversion technique was not applicable).

Anastomotic separation rarely occurs acutely after resection. It represents technical failure most likely due to excessive anastomotic tension. Manifestations are coughing with blood staining, airway obstruction with stridor and dyspnea, and subcutaneous or mediastinal air, depending on the level of separation. If separation occurs later in the patient's course, within the first week, peritracheal inflammation and healing may be sufficient to wall off the trachea so that air leakage is not evident. Initially, there may be no symptoms or a mild cough and slight wheeze that will worsen. Major separation can result in obstruction, pneumonia, mediastinitis, and brachiocephalic artery erosion. Prompt recognition is necessary for effective treatment. Flexible bronchoscopy is in order if any suspicion of separation arises. Partial separation of an anastomosis along a segment of the suture line has less grave consequences, but must be treated and followed to be sure late stenosis does not occur.

Air leakage alone may occur without a major anastomotic separation. An opening of millimeters at the site of one or another suture may lead to tissue emphysema. This is a rare complication as an isolated finding if the anastomosis was airtight when completed.

Anastomotic stenosis may occur quite early in the postoperative course, or later. Early occurrence most often results from cicatrization after partial or complete anastomotic separation. As healing progresses, the stenosis becomes more symptomatic and may close the airway completely if not detected and treated. In most cases, if the anastomosis is intact when examined bronchoscopically at 6 or 7 days postoperatively, then it will remain so. Infrequently, the bronchoscopic appearance of the stenosis may be identical with that seen in experimental animals, in which ever greater lengths of trachea were resected in order to test the effect of increasing anastomotic tension. The stenosis in this case may be more evident at the sides of the anastomosis than anteroposteriorly. This evolution was clearly seen in 2 patients who underwent what were expected to be acceptable lengths of resection, 1 for benign tumor and 1 for postintubation stenosis. They had been treated with steroids for months and were still on high doses of prednisone. Healing appeared satisfactory initially. Presumably, insufficient collagen had been laid down prior to the loss of suture strength, and a gradual distraction followed under high anastomotic tension. Both patients were later re-resected successfully, after being fully weaned from steroids. Some stenoses produce partial narrowing of the anastomosis, to degrees which are clinically acceptable but which limit the patient's activities.

Tracheal necrosis adjacent to an anastomosis is attributable to excessive circumferential dissection of that portion of the trachea that is not resected. Since the blood supply of the trachea is segmental, unnecessary dissection of the trachea circumferentially damages its blood supply. Since cartilages hold sutures for

quite some time, disaster does not become evident until much later. As the trachea necroses, mucosa sloughs and pieces of cartilage are discharged into the lumen. Surrounding inflammatory reaction usually prevents an air leak. In the course of healing, granulations form and cicatrize circumferentially. I became aware of this potential problem early in my experience with staged repair of the trachea by fashioning of a cutaneous tube in stages. I observed approximately 1 cm of necrosis of the trachea distal to the interposed cutaneous trough. The segment was resected and the viable trachea healed to the skin. Extensive circumferential resection was never done again. Definition of the segmental nature of the tracheal blood supply subsequently further reinforced this conclusion. Although we have not since encountered this problem in our own operative experience, I am sure that devascularization has produced some postoperative disasters referred for secondary repair. Necrosis after resection and anastomosis due to prior irradiation is discussed in Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation."

Hemorrhage of significance may occur from two sources. After cervicomediastinal tracheal resection, the anastomosis often lies directly behind the *brachiocephalic artery*, and erosion may occur. Premonitory hemorrhage in a smaller significant amount may manifest as hemoptysis. The initial event may also be a massive and potentially fatal hemorrhage. This indeed was a too common complication in a number of early reports of tracheal reconstruction. I believe that the principal cause of this disaster lies in the technique of surgical dissection. If the innominate artery is dissected out and retracted away from the trachea, the arterial wall is bared. The adventitia then lies in direct contact with the sutures of the tracheal anastomosis. Even if these sutures are of flexible material, they may produce a nidus of inflammation and then of vascular erosion.

If the artery is adherent to the stenosis, it must be dissected free in order to correct the lesion, leaving the vessel without its normal protective investing tissues. Also, in reoperative tracheal resection, the artery may be adherent to the anastomosis. The vessel wall is necessarily thinned and bared during dissection. A further insult may be from inadvertent intraoperative trauma from needles, instruments, or retractors.

A second source of hemorrhage after carinal reconstruction is from the *pulmonary artery*. Following tracheobronchial reconstructive procedures, the pulmonary artery or one of its branches usually lies adjacent to the anastomosis. A tracheal or bronchial suture line immediately against the arterial wall is a source of danger.

Tracheoesophageal fistula is an extraordinarily rare complication of tracheal resection and is only likely to be seen where the full thickness of the esophageal wall has also been resected, as for a tumor. If the esophageal suture line is contiguous with the tracheal suture line, a fistula may result. The problem is analogous to the recurrence of a tracheoesophageal fistula after its surgical correction. One esophagocutaneous fistula resulted in an unusual case of a very extensive intrathoracic resection of the trachea for recurrent plexiform neurofibroma, which included part of the esophageal wall. The small sinus healed spontaneously.

Vocal cord dysfunction may follow injury to recurrent laryngeal nerves during dissection. The risk to these nerves is discussed in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection," Chapter 25, "Laryngotracheal Reconstruction," and Chapter 29, "Carinal Reconstruction," describing techniques of resection. Injury may be temporary or permanent. The risk of nerve injury is greater when dissection is in dense scar near the cricoid, in laryngotracheal resection, in carinal resection and reconstruction, and where operation is to excise a malignant neoplasm. If the patient presents with a paralyzed vocal cord, sometimes without prior surgery or after tracheostomy alone, or if the cord had been paralyzed from prior surgery, then even greater care must be taken to avoid injuring the remaining functional nerve.

Vocal cord dysfunction and *aspiration* may follow adjunctive laryngeal release procedures. As noted elsewhere, deglutitional difficulty seems more likely to follow thyrohyoid release than suprahyoid release, and it is particularly associated with prior or concomitant recurrent laryngeal nerve injury, extended tracheal resection, old age, or neurologic disability.

Other Complications

It seems surprising that *wound infection* does not occur more often. Cultures from patients with an open tracheostoma or granulation tissue at a stenosis produce *Staphylococcus aureus*, often resistant, and/or *Pseudomonas aeruginosa*, as well as a variety of other pathogens, including *Streptococcus* and *Escherichia coli*. A moderate incidence of wound infection might be expected because of the open nature of the surgery with the passage of tubes and instruments through the larynx and pharynx, concurrent exposure of the mediastinum, and, frequently, of the partially or completely divided sternum. This is not the case. Techniques of surgery and perioperative management may well play important roles. Since I have no intention of performing randomized series to explore this surprisingly benign situation, clarification may not be possible.

Laryngeal edema is rare, but is somewhat more common when surgery involves the larynx directly, in resection of laryngotracheal stenosis and in laryngoplasty for tumors of the upper airway. Edema is manifested by wheezing or stridor upon extubation at the conclusion of the operation or during the next 24 hours. Direct examination of the vocal cords in the patient who is still lightly anesthetized or awakening usually allows differentiation of edema from vocal cord paralysis.

Major pulmonary complications are very rare after cervical or cervicomediastinal tracheal resection, despite the fact that many patients have limited pulmonary reserve due to chronic obstructive pulmonary disease or emphysema. Postoperative pneumonia is more common after carinal resection and reconstruction. This can be minimized by careful postoperative care. However, postoperative ARDS may occur without warning, despite attempted prophylaxis, particularly after a right carinal pneumonectomy. The operation may have been uneventful. The immediate postoperative course appears to be similarly uneventful. Most frequently, patients have been extubated promptly and do not then require respiratory assistance.

In approximately 36 hours, however, a light infiltrate appears, often in midfield of the remaining lung. This worsens steadily over the next few days (Figure 21-1). The patient does not produce significant sputum, and bronchoscopy reveals only scant secretions which culture normal flora. As the radiologic picture worsens, the patient goes into florid ARDS with widespread pulmonary infiltrate, and finally whiteout of the remaining lung. Intubation, maximum conventional respiratory support, aspiration bronchoscopy, adjuvants including wide-spectrum antibiotics, diuretics, and corticosteroids all usually fail to salvage the patient. At autopsy, the lungs are found to be very heavy with interstitial fluid. Cultures do not reveal significant pathogens, although a few colonies may appear, consistent with intubation and ventilation. These patients were diagnosed as having died from "bronchopneumonia." This was more a diagnosis of convention. The clinical picture is that of noncardiogenic "postpneumonectomy pulmonary edema," which was described by Zeldin and Peters and colleagues after routine pneumonectomy.[7] Peters' thesis was that this represents pulmonary edema due to the inability of the lung to rid itself of interstitial water from perioperative fluid overload, perhaps compounded by a decrease in lymphatic capability. This was not confirmed in other studies and ARDS was also seen to follow a lobectomy occasionally.[8,9] Although we had scrupulously managed such patients, ever since our initial encounters, by rigorous fluid restriction perioperatively and with the other measures noted, the syndrome nonetheless occurred unpredictably. A subsequent reduction in the incidence of this dire complication may be attributed to the adjustment of the intraoperative airway ventilatory pressure and tidal volumes to reduce and avoid barotrauma to the lungs. Additional complications that are common to any kind of surgery, and some more common after intrathoracic procedures, have been few in number. These include pneumothorax, line infection, atrial fibrillation, myocardial infarction, and deep venous thrombosis. Special postresectional problems, such as those inherent in airway surgery after irradiation, and those following unusual procedures for cervicomediastinal exenteration, posterior wall splinting, correction of postpneumonectomy syndrome, tracheoplasty for congenital stenosis, and resections of rare inflammatory lesions, are discussed in the chapters devoted to these diseases and techniques.

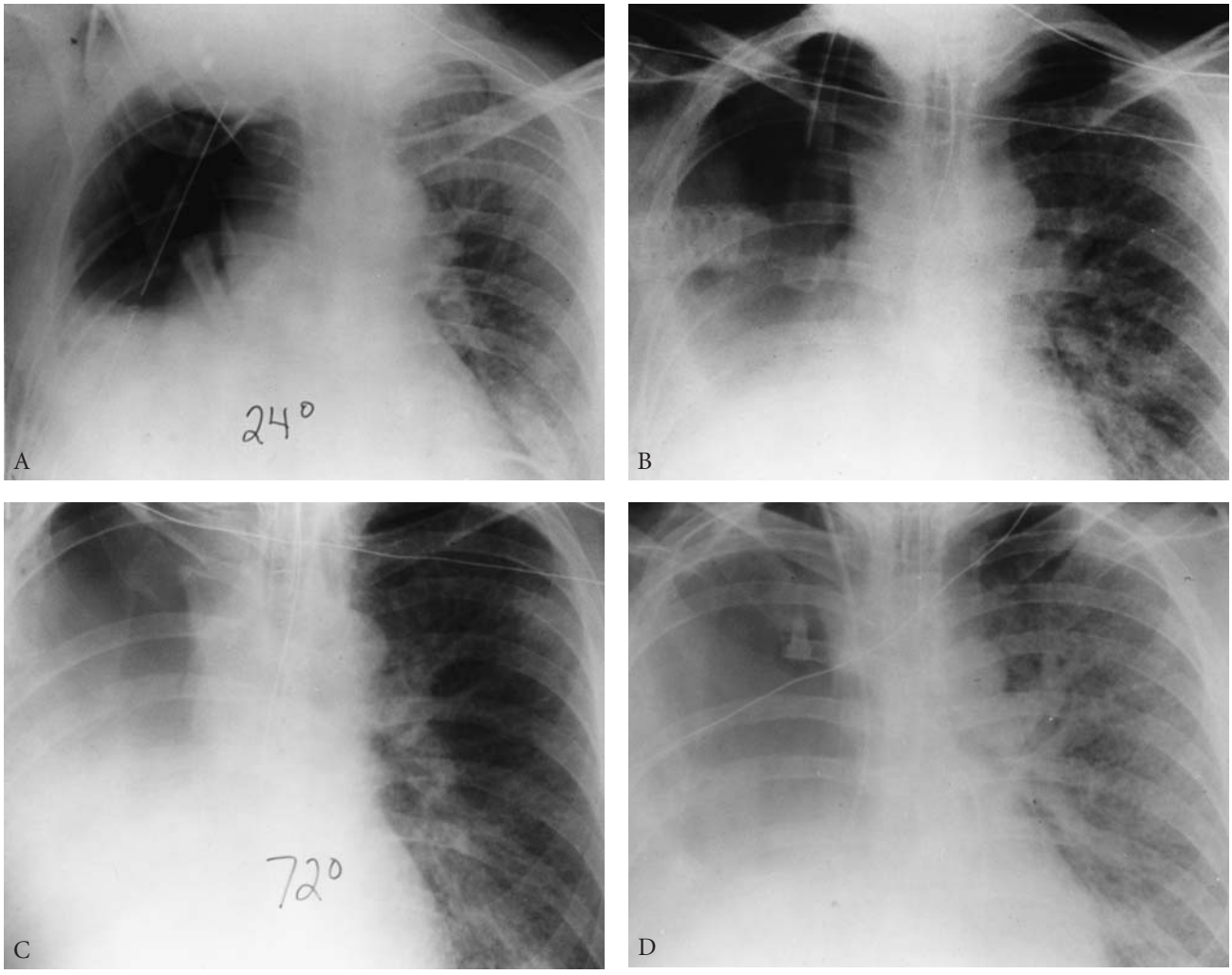

FIGURE **21-1** *Development and progression of "postpneumonectomy pulmonary edema" to fatal acute respiratory distress syndrome. Uneventful right carinal pneumonectomy was performed for a squamous cell bronchogenic carcinoma invading the carina.* A, *The patient was extubated and clinically doing very well. At 24 hours, dyspnea appeared with diffuse left lung infiltrate.* B, *At 48 hours, the infiltrate had worsened. The patient was reintubated and ventilated.* C, *Progression at 72 hours.* D, *Chest roentgenogram at 120 hours, with marked worsening of the parenchymal infiltration. The patient continued to deteriorate and died 12 days after operation. This predated our use of nitric oxide treatment.*

Quadriplegia can occur infrequently as a devastating and irreversible consequence after tracheal resection. I have personally never encountered it, although one such event occurred in our thoracic surgical unit. It has happened in young, otherwise healthy patients who had no hypotension or untoward events during anesthesia and operation. Suspicion has focused on cervical hyperextension during surgery, and perhaps extreme flexion postoperatively, in an effort to lessen anastomotic tension. Extremes of extension and flexion were not used in our one patient.

Midcervical quadriplegia has followed neurosurgical procedures with patients operated upon in a sitting position with accompanying cervical flexion. Spinal cord ischemia is therefore suspect. Anesthesiologists caution that the chin and the chest should not be closer than one inch in this position.[10] Dominguez and colleagues reported this complication immediately *after* tracheal resection, upon assumption of a sitting position plus "extreme" cervical flexion.[11] Magnetic resonance imaging 19 days later revealed findings

consistent with cervical spinal cord ischemia. Two other instances of quadriplegia after tracheal resection were cited. I have also been told of a fifth patient elsewhere.

It must be emphasized that the "guardian suture" is placed to prevent postoperative hyperextension, *not* to gain extreme cervical flexion. Given the uncertainties noted, it is also best to avoid extreme cervical hyperextension in positioning the patient for anterior tracheal resection.

Late coronary artery disease has been noted in some patients who received mediastinal irradiation following tracheal resection for a malignant tumor (in doses often exceeding 5,000 cGy), most likely as a result of irradiation.

Treatment and Prevention of Complications

Complications Due to Failure of Diagnosis

Precise *assessment of the extent of pathology*, and also of the proportion of trachea that remains uninvolved, guides preoperative judgment about the possibility of resection. Radiologic and endoscopic studies are the keys. The surgeon must not hesitate to cancel plans for tracheal resection, even after exploration of the trachea, should unexpectedly extensive disease be identified. A T tube that provides a satisfactory airway is preferable to an operation that risks life. Also, primary palliative radiotherapy may be the only appropriate treatment for a very extensive neoplasm. Adjunctive procedures at various times in the clinical course are core-out, T tube, or stent and brachytherapy.

The importance of careful functional and anatomic assessment of the larynx and, in particular, *evaluation of glottic function* has been emphasized. If the glottic aperture is insufficient, a preliminary procedure for correction is required. Skilled otolaryngological consultation is advisable. The glottis must be visualized in an awake patient. Fluoroscopy may also identify asymmetric or inadequate vocal cord function.

If glottic inadequacy is not identified preoperatively, management may demand transient intubation, followed by careful tracheostomy, placed to avoid injury to a fresh anastomosis and to the brachiocephalic artery. Vocal cord medialization or lateralization may be needed electively, with eventual decannulation. Commissural stenosis or vocal cord lesions must be treated, if discovered late, with consideration of a temporary tracheostomy or T tube.

If there is any reason to suspect that *aspiration* occurs during deglutition, or that it might occur if the patient were permitted to swallow (as in the case of a patient who is nil per os [NPO] due to a tracheoesophageal fistula), the danger should be revealed by a meticulously performed barium swallow, with close attention paid to the pharyngeal, cricopharyngeal, and upper esophageal phases of swallowing. A small amount of aspiration is quite common in elderly patients and should not be confused with abnormal aspiration.

If aspiration is severe and cannot be improved in preparation for tracheal surgery, then a permanent tracheostomy may be the appropriate alternative.

Postoperative aspiration may improve with time, especially with the assistance of speech pathology training. In the worst case, temporary feeding tubes may become permanent and tracheostomy may be necessary for protection and clearance of the airway.

Fluoroscopy of the trachea by a radiologist familiar with normal tracheal dynamics, and an awake flexible bronchoscopy, will not only identify vocal cord malfunction but also areas of *tracheomalacia* and their severity. Whereas short areas of severe collapse may be resected, more diffuse areas require stents or special procedures. Malacia present in addition to postintubation stenosis must be accounted for in the operative plan. My early hope that resection of a stenosis with anastomosis of a softened adjacent tracheal segment to a "normal" distal trachea would provide sufficient support proved wrong. Although the use of external splinting polypropylene rings has worked in a few cases, failure has also occurred due to loss of blood supply and infection. Dissection and foreign body placement in a bacterially contaminated zone is best avoided entirely. Dilation with a T tube or stent seems a better solution.

Complications from Failure of Technique

The problem of *granulation tissue* at the suture line has been almost eliminated by the use of 4-0 Vicryl for anastomosis. All nonabsorbable sutures often produced granulomas. On two occasions, when a single reinforcing suture of Tevdek was placed anteriorly, a granuloma appeared at that site only! Since then, 3-0 Vicryl sutures have been used in circumstances that require reinforcement.

Granulations are removed with biopsy forceps through a rigid bronchoscope. An offending suture at the base of a granulation is also plucked. Upper tracheal manipulations like this are facilitated by using a 25 cm × 9 mm bronchoscope. When extensive granulations were encountered in the past, Aristocort (triamcinolone acetonide) or Depo-Medrol (methylprednisolone acetate) were injected through extra-long needles with a 25 gauge tip specially made for this purpose, using a tuberculin syringe to provide sufficient pressure (see Figure 5-5 in Chapter 5, "Diagnostic Endoscopy"). Evidence is not clear that these steroids inhibited recurrence of granulations. Nowadays, only a rare, usually single, small granulation seems to occur.

Mitomycin-C, an antineoplastic alkylating agent, which inhibits DNA synthesis and hence fibroblast proliferation, has been used topically to try to prevent or treat scarring following laryngotracheal reconstruction.[12,13] Thus far, results remain equivocal.

If *partial or complete separation of the anastomosis* occurs postoperatively within the first week following surgery, reexploration may be in order to debride necrotic tissue and to place a T tube across the area of separation. Advancement of an inferiorly pedicled sternohyoid muscle flap, or of an omental pedicle (substernally), may be advisable if the brachiocephalic artery is nearby, and certainly if its wall is exposed. If the separation seems well contained and appears like a cuff or sleeve or granulation tissue, then it may simply be debrided bronchoscopically (rigid instrument) and a T tube placed to span the separation. If the separation is accessible in the neck, the sidearm of the T is placed through the damaged area to preserve the maximum of undamaged trachea for later re-reconstruction. A limited separation of part of the circumference of anastomosis is also best treated conservatively with a T tube. If the patient is sick enough to require ventilation, a (cuffed) tracheostomy tube is initially used, to be replaced with a T tube later.

A small very *early leak in an anastomosis* (not frank separation) is rare but may be repaired and buttressed by immediate reoperation. A tiny air leak after carinal reconstruction, manifest only by continuing air leakage without pneumothorax, and where bronchoscopy reveals no separation, should respond in 24 to 48 hours to continued gentle chest suction.

Limited intrathoracic separation can sometimes be spanned, initially with an endotracheal tube carefully positioned by a flexible bronchoscopy. Limited carinal separation without air leakage or patient distress can be bridged with a silicone Y stent. A specially proportioned and angled stent may have to be fabricated for the patient, with side openings cut and sanded or alterations made in standard T-Y or Y stents. Some prefer expandable coated stents, but the fringed edges may cause granulations. Uncoated expandable stents should *never* be placed across granulating tissue. The granulations will grow through the lattice.

After apparent healing of a stented complete separation, removal of the supporting stent is usually followed by tracheal (or bronchial) stenosis. If re-resection is planned, it should be done no sooner than 4 months after the separation, and preferably 6 months, to allow subsidence of inflammatory changes. On two occasions, patients have healed short but completely circumferential carinal separations under a silicone Y stent, and not required re-resection. Presumably, scar contraction pulled the ends together (over a short distance) but did not contract centrally, as usually occurs.

Separation of a part of the anastomotic circumference may heal without significant narrowing, or to a minor degree without functional importance. Repeated bronchoscopy should be done until stability is certain. If dilation fails to give a lasting result, and significant stenosis persists, then a reoperation is indicated. Rarely, a narrow shelf will respond to lasering.

Elsewhere, note has been made of the need to avoid placing an endotracheal tube cuff against a freshly completed tracheal anastomosis if a patient requires postoperative ventilation. Even a low-pressure cuff can endanger the repair in time. Patients who present with tracheal lesions, who are already being ventilated, are not appropriate candidates for elective reconstruction

Later *restenosis* presents with the same clinical findings as an initial stenosis. Treatment within 6 months of the original surgery should be by careful dilation under general anesthesia and insertion of a T tube or silicone stent. A stomal site for the T tube must be selected carefully, preferably through the damaged trachea, with a plan for future resection and reconstruction of the stenotic area. If the stenosis is deep in the mediastinum, the tracheostomy port should be high enough so that a good segment of normal trachea lies between it and the zone to be resected. Bronchoscopic examination of all anastomoses just prior to hospital discharge should avoid late discovery of anastomotic failure. Once satisfactory healing is observed, a late stenosis is unlikely unless irradiation is a factor.

In 503 patients who underwent *reconstruction for postintubation lesions*, 29 dehisced or restenosed.[3] Seven died, 2 with erosion of the brachiocephalic artery. Eight were successfully reconstructed later, 4 required permanent tracheostomy, and 5 required a T tube, 3 of which were temporary. Three with partial separations were treated by primary closure (2) and cervical drainage (1).

An excessive number of anastomotic complications followed *carinal resection*, and from these experiences some cautionary principles have been learned.[6] Twenty-three of 134 patients (17%) had anastomotic complications. Eight died early postoperatively with necrosis, separation, or mucosal slough. Two with bronchovascular fistulae have been described. Seven had re-resection and 2 had completion pneumonectomy. Two were treated for stenosis by dilation and stenting. It is clear that a residual right lower lobe should not be anastomosed to the trachea after carinal resection and anastomosis of trachea to left main bronchus. The tension is excessive. Right pneumonectomy is a safer alternative; if pulmonary function does not permit pneumonectomy, then the right lower lobe bronchus must be anastomosed to the medial side of the left main bronchus. Furthermore, if right carinal pneumonectomy appears to require resection of more than about 4 cm of trachea, postoperative tension will be excessive and resection is usually contraindicated. In contrast, the stump of the right main bronchus can be safely elevated quite high for anastomosis to the residual trachea after left carinal pneumonectomy plus excision of a length of trachea (see Chapter 29, "Carinal Reconstruction").

Prevention of separation and stenosis of tracheal anastomoses lies in the avoidance of anastomosis under excessive tension. Devascularization of the trachea must also be avoided. Avoiding tension depends on careful assessment of the extent of the lesion and of the nature of the patient. Patients vary in the amount of trachea that may be safely resected, which depends on body build, age, and prior surgery. In children, the trachea tolerates tension less well than in the adult. It is possible, but difficult, to measure the tension required for tracheal approximation in the operating room. The experienced surgeon can judge quite accurately whether the tension seems excessive. At this point, the die is cast. Adjunctive release maneuvers may be required. Since laryngeal release, in particular, carries with it the potential for complications, it must be used with caution and never routinely. On occasion, it is better to retreat after exploration than to perform too lengthy a resection. Devascularization of trachea remaining in the patient is avoided by carefully sparing the blood supply in the lateral pedicles. Ideally, no more than 1 cm of trachea should be free of lateral blood supply.

Caution should be repeated against operating prematurely on a massively inflamed trachea, where these changes extend beyond the stenotic segment, usually proximally. First, it may be difficult to define the exact extent of the pathology, and second, anastomosis in floridly inflamed tissue may result in granulation tissue and restenosis.

Major hemorrhage is managed by prompt reexploration after emergency intubation, with a tamponading balloon cuff placed at the level of the anastomotic–arterial fistula. Operative management for

brachiocephalic arterial injury is the same as that for tracheoarterial fistula due to erosion by a high-pressure cuff or tube tip (see Chapter 27, "Repair of Tracheobrachiocephalic Artery Fistula"). Proximal and distal control must be obtained and the area of leakage carefully assessed. In a very rare case, it is possible to excise a small area of damage and perform arteriorrhaphy. Patches of foreign material are to be avoided. More often, it is advisable to divide the artery, excise the damaged segment, and to close the arterial ends with running vascular sutures. Ligation should not be done. If possible, the operation should be done with electroencephalographic monitoring. Neurologic sequelae are rare (see Chapter 13, "Tracheal Fistula to Brachiocephalic Artery"). The ends of the artery are buried in healthy tissue, such as pedicled strap muscle, thymus, or omentum. The tracheal lesion is managed in accordance with the approach described for tracheal separation. Either an endotracheal tube or a T tube will span the area of injury. The damaged trachea may be debrided and patched if separation is only partial, or it may be spanned with a tube. In 2 patients suffering brachiocephalic artery bleeding following tracheal resection, a diagnosis was made promptly and the patients salvaged, one by arterial excision and one by arterial repair of a tiny defect. Both occurred within the first postoperative week, while in hospital.

Two patients died from massive hemorrhage after anastomoses at the carinal level. In both, the presumed source of bleeding was the *pulmonary artery*. One occurred on the ninth postoperative day, and the other several months later while the patient was being bronchoscoped for a stenotic anastomosis at the site of implantation of bronchus intermedius into the side of the trachea. No tissue had been interposed between the intrathoracic anastomosis and pulmonary artery in the first patient. This was the only patient with carinal reconstruction who was ever so managed. In the second case, the responsible factors were probably local separation due to tension, with infection, stenosis, and perianastomotic inflammation.

Brachiocephalic artery injury is avoided by keeping dissection close to the trachea and the stenotic lesion. The artery is not exposed at all and is retracted along with the balance of the tissues anterior to the trachea. A protective layer of connective tissue remains. If the artery must be dissected, either because of prior surgery or because of inflammatory adherence to the stenosis, then it is essential that well vascularized tissue be interposed between the tracheal anastomosis and the dissected artery. A strap muscle may be detached superiorly and pedicled downward for this purpose, dividing only those attachments that are necessary to allow the muscle to reach the anastomosis, thus preserving its blood supply as much as possible. Occasionally, thymic tissue or omentum may be used.

Intrathoracic tracheal or bronchial anastomosis is always wrapped. Well-vascularized pericardial fat pad is excellent for this purpose. Less satisfactory, but usually adequate, is a broad-based pleural flap (see Chapter 29, "Carinal Reconstruction").

If the rare complication of *tracheoesophageal fistula occurs*, it should be repaired. Adequate tissue must be interposed between the esophageal and tracheal suture lines to prevent reestablishment of the fistula. Gastrostomy and jejunostomy tubes to avoid aspiration from reflux and to insure nutrition during healing are advisable (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula," and Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula"). Prevention lies in buttressing any esophageal suture lines at the initial procedure. This interposes healthy tissue between tracheal and esophageal suture lines.

Management of postoperative *vocal cord dysfunction* or paralysis varies. Watchful waiting appears to be the best policy for single cord paralysis. Over a period of 6 to 12 months, voice may return to an adequate level, even if not wholly normal. If recovery fails, then injection or medialization of the paralyzed cord may improve function. Temporary injection of the cord with absorbable material may provide palliation in the intervening period if the patient is much discomforted by the injury, and it may be important if aspiration is a problem. Division of a recurrent laryngeal nerve usually leads to vocal cord abduction, whereas lesser trauma may lead to paresis in a more median position. The first is more likely to follow intrathoracic oper-

ations and require intervention to prevent aspiration.[14,15] Early otolaryngologic consultation is advisable. Henderson and colleagues warned of concomitant pharyngoesophageal dysphagia and recurrent laryngeal nerve palsy, recommending myotomy for its treatment.[16] Mom and colleagues proposed thyroplasty at once, following certainty of laryngeal nerve severance.[17] Obstruction from bilateral cord paralysis requires urgent endotracheal intubation and probably later tracheostomy followed by a cord lateralization procedure or arytenoidectomy. A T tube placed through the vocal cords is less satisfactory because of the inhibition of normal voice (see Chapter 35, "Laryngologic Problems Related to Tracheal Surgery").

Tracheal surgery and, especially, laryngotracheal and carinal procedures demand constant awareness and concern about the recurrent laryngeal nerves. The techniques of dissection described in Chapters 24 to 29 should minimize the frequency of injury. Dissection, especially in laryngotracheal reconstruction, can damage the nerves, usually transiently, even with meticulous care, just by proximity of trauma.

If *aspiration* follows tracheal surgery, an attempt must be made to analyze the causative factors. Such factors have already been itemized to be lengthy tracheal resection, injury to recurrent laryngeal nerves, and dysfunction due to laryngeal release. Age and neurologic difficulties are other factors. The larynx is normally protected by elevation of the epiglottis of the larynx during deglutition, which is inhibited by tracheal shortening (by resection) and by hyoid detachment (by laryngeal release). Reflex glottic closure during swallowing is impaired by vocal cord paralysis.

Assessment is best made by careful radiologic studies, including fluoroscopy of the larynx and trachea, and modified barium swallow, with special attention paid to the phases of deglutition. Otolaryngologic examination of glottic function is essential. Endoscopy may be of value. It is important not to temporize too long during assessment. An atraumatic pediatric feeding tube may be useful. It is often better to proceed early to a gastrostomy for drainage in order to avoid reflux and pulmonary injury from gastric contents. A jejunostomy tube is simultaneously placed for prolonged nutritional support. Temporary tracheostomy with, if necessary, a cuffed tube to protect against aspiration may be necessary. Instruction in swallowing techniques to avoid minor aspiration can be of great help and requires the skills of a trained speech pathologist. Tracheal shortening may be compensated by flexing the neck and by "tucking" the chin during a swallow, thus enhancing epiglottic closure. Each case tends to be highly individual and should be carefully analyzed with the assistance of an otolaryngological colleague with expertise in such problems. Thus, vocal cord medialization may lessen aspiration through a glottis widened by paralysis and inhibited from normal reflex closure during swallowing. Patients usually recover from swallowing deficits due to laryngeal release, although it may take 6 months or longer. I believe this occurs as the divided suprahyoid muscles gradually reattach by scarring and restore laryngeal elevation during deglutition. Characteristically, the patient will first tolerate soft solids, then thick liquids, and finally, thin liquids (water, apple juice, and wine).

Prevention of these problems to the extent possible lies in scrupulously avoiding damage to recurrent laryngeal nerves, avoiding excessive tracheal resection, and using laryngeal release procedures only where absolutely required. It has been our qualitative observation and that of F.G. Pearson (personal communication, 1979) that suprahyoid release results in fewer problems and of less duration than thyrohyoid release. This may be because the latter puts greater traction on the superior laryngeal nerves. Injury to these nerves must be scrupulously avoided, since this can lead to irreversible loss of laryngeal sensation, and hence of protective reflexes on swallowing. Indeed, Dedo subsequently recommended avoidance of the thyrohyoid laryngeal release altogether.[18] It must be emphasized again that glottic function and the presence of possible neurologic deficits in deglutition must be identified prior to surgical repair.

Other Complications

Laryngeal edema, which may follow laryngotracheal procedures especially, is treated immediately with Decadron systemically for a short period of time (24 to 48 hours). This brief treatment will not adversely

affect healing. Racemic epinephrine is also administered by nebulization. If the airway is severely obstructed, an uncuffed small-bore endotracheal tube is placed. After a few days, the tube is removed in the operating room. If the airway is still unsatisfactory, the tube is replaced and a small tracheostomy tube is located judiciously away from the suture line and the brachiocephalic artery. In such cases (see Chapter 25, "Laryngotracheal Reconstruction"), the site of tracheostomy would best have been previously identified at the time of the original reconstruction, and protective tissue barriers sutured over the brachiocephalic artery and the anastomosis. A tracheostomy tube is never placed through a fresh anastomosis or immediately adjacent to it, since this may well result in stenosis at the anastomosis.

There is no completely effective way to avoid laryngeal edema in patients undergoing procedures involving the larynx itself. In addition to local trauma, interruption in lymphatic drainage may be presumed. However, it is important to observe the usual rules of instrumentation and surgery, namely, gentleness, use of equipment of necessary size and not larger, and application of meticulous technique. If there is any question of laryngeal adequacy after removal of the endotracheal tube, it is far better to perform a tracheostomy than to run a risk of sudden airway obstruction.

Wound infection is fortunately rare and treated in the usual manner. More commonly, minor cellulitis and induration may occur in a cervical incision in an area of old scarring. This is treated by continuing with antibiotics. Patients receive antibiotics immediately preoperatively, intraoperatively, and for 24 to 48 hours postoperatively. Even where tracheostomy and granulations are absent, an operation with an open airway and cross-field intubation appears to justify prophylaxis.

Pneumonia and lung infection is treated in the usual manner. Every effort is made during surgery to avoid allowing large amounts of blood to collect distally. The airway is suctioned repeatedly during operation, and especially thoroughly immediately prior to anastomosis. Postoperative flexible bronchoscopy is performed prior to awakening the patient. Postoperative bedside bronchoscopy is performed frequently, particularly in patients who have undergone carinal reconstruction. We have not hesitated to use a mini-tracheostomy after carinal reconstructions if the patient has difficulty in raising secretions.

Prevention of postpneumonectomy pulmonary edema (ARDS) is hampered by the failure thus far to discover its cause. A step forward in the management of this dire occurrence may be the finding by Mathisen and colleagues, that nitric oxide administration at inception of the syndrome seemed to benefit patients remarkably.[9] The complication seems to occur less often nowadays, probably due to lowered ventilatory pressures.

General Precepts

E. Stanley Crawford, the renowned vascular surgeon, was fond of the homespun comment, "Most of your complications begin in the operating room." The remarks here on the prevention of complications in tracheal surgery show that many are preventable. The most difficult to eliminate are those that are based on initial diagnosis, assessment of the patient, and selection of the operation, since these are based on experience. Technical problems are largely prevented by precise and meticulous technique, guided by past observations.

Certain general precepts emerge, which may significantly reduce the number of complications.

1) The surgeon undertaking tracheal surgery should, at the very least, have spent time and scholarly effort to become familiar with currently available information about tracheal surgery. If the surgeon has not received training in this specialty, then ideally, such surgeon should observe work done by surgeons skilled in these techniques. The surgeon must make a willing commitment of time, effort, and patience in performing this surgery and not undertake it as a casual effort.
2) Considerable thought must be given to the preoperative diagnosis of the lesion and to its extent, and careful search must be made for any complicating additional airway pathology that might seriously affect the outcome.

3) Surgical approach must be carefully planned in a step-wise fashion. No irrevocable moves should be made until the surgeon is certain that it is appropriate to proceed to resection. The surgeon should attempt resection only if it appears reasonably possible, and a clear picture must be had of all alternative modes of therapy available.
4) Techniques can never be meticulous enough. Great care must be taken to avoid devascularization and anastomotic tension. Improvisation should be avoided until the surgeon has obtained experience in the field.
5) Thoracic surgeons should not undertake tracheal surgery unless they have access to expert help in radiology of the airways, thoracic anesthesia with knowledge of the unique problems of airway reconstruction, expert intraoperative pathology, experienced nursing care, and consultation, particularly with otolaryngologists who have interest and experience in airway problems.

References

1. Couraud L, Bruneteau A, Martigne C, Meriot C. Prevention and treatment of complications and sequelae of tracheal resection and anastomosis. Int Surg 1982; 67:235–9.
2. Grillo HC, Zannini P, Michelassi F. Complications of tracheal reconstruction: incidence, treatment and prevention. J Thorac Cardiovasc Surg 1986;91:322–8.
3. Grillo HC, Donahue DM, Mathisen DJ, et al. Postintubation tracheal stenosis: results and surgical treatment. J Thorac Cardiovasc Surg 1995;109:486–93.
4. Grillo HC, Mathisen DJ. Primary tracheal tumors: treatment and results. Ann Thorac Surg 1990;49:69–77.
5. Donahue DM, Grillo HC, Wain JC, et al. Reoperative tracheal resection and reconstruction for unsuccessful repair of postintubation stenosis. J Thorac Cardiovasc Surg 1997;114:934–9.
6. Mitchell JD, Mathisen DJ, Wright CD, et al. Clinical experience with carinal resection. J Thorac Cardiovasc Surg 1999;117:39–53.
7. Zeldin RA, Normandin D, Landtwing D, Peters RM. Postpneumonectomy pulmonary edema. J Thorac Cardiovasc Surg 1984;87:359–65.
8. Waller DA, Gebitekin C, Saunders NR, Walker DR. Noncardiogenic pulmonary edema complicating lung resection. Ann Thorac Surg 1993;55:140–3.
9. Mathisen DJ, Kuo EY, Hahn C, et al. Inhaled nitric oxide for adult respiratory distress syndrome after pulmonary resection. Ann Thorac Surg 1998;65: 1894–902.
10. Alexander CM, Vandam LD. Positioning of patients for operation. In: Longnecker DE, Tinker JH, Morgan GE Jr, editors. Principles and practice of anesthesiology. 2nd ed. St. Louis: Mosby; 1998. p. 680–99.
11. Dominguez J, Rivas JJ, Lobato RD, et al. Irreversible tetraplegia after tracheal resection. Ann Thorac Surg 1996;62:278–80.
12. Rehbar R, Valdez TA, Shapshay SM. Preliminary results of intraoperative mitomycin-C in the treatment and prevention of glottic and subglottic stenosis. J Voice 2000;14:282–6.
13. Hartnick CJ, Hartley BEJ, Lacy PD, et al. Topical mitomycin application after laryngotracheal reconstruction: a randomized, double-blind, placebo-controlled trial. Arch Otolaryngol Head Neck Surg 2001; 127:1250–4.
14. Kraus DH, Ali MK, Ginsberg RJ, et al. Vocal cord medialization for unilateral paralysis associated with intrathoracic malignancies. J Thorac Cardiovasc Surg 1996;111:34–41.
15. Filiare M, Mom T, Laurent S, et al. Vocal cord dysfunction after left lung resection for cancer. Eur J Cardiothorac Surg 2001;20:705–11.
16. Henderson RD, Boszko A, Van Nostrand AWP. Pharyngoesophageal dysphagia and recurrent laryngeal nerve palsy. J Thorac Cardiovasc Surg 1974;68:507–12.
17. Mom T, Filaire M, Advemier D, et al. Concomitant type I thyroplasty and thoracic operations for lung cancer: preventing respiratory complications associated with vagus or recurrent laryngeal nerve injury. J Thorac Cardiovasc Surg 2001;121:642–8.
18. Dedo HH. Surgery of the larynx and trachea. Philadelphia: BC Decker; 1990. p. 182.

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Tracheostomy, Minitracheostomy, and Closure of Persistent Stoma

Hermes C. Grillo, MD

Tracheostomy

Endotracheal intubation rather than tracheostomy is now used to establish an emergency airway. Even in difficult anatomic situations, intubation can usually be accomplished over a flexible intubating laryngoscope or bronchoscope. Failing this, a rigid bronchoscope is introduced. A ventilating bronchoscope may serve as an airway during tracheostomy, if necessary. A laryngeal mask airway may be considered. Tracheostomy is preferably done in an operating room with an airway already established by one of the methods noted. The patient is supine on the table with the neck moderately extended (see Figure 24-3*B* in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). An inflatable bag placed beneath the patient's shoulders is helpful. The table is flexed at hip level to elevate the head and neck, thus reducing pressure in the cervical veins. Brief general anesthesia is preferred, but local anesthesia may be used.

The incision is located with reference to the cricoid cartilage and *not* the sternal notch, since the trachea and larynx move independently of the sternum with flexion and extension of the neck (see Figure 1-4, in Chapter 1, "Anatomy of the Trachea"). A more cosmetic transverse incision is used since urgency is moderated by the inlying airway. A short incision is made 1 cm below the cricoid cartilage, which can usually be palpated, even in obese patients (Figure 22-1*A*). It is carried through the platysma, the flaps elevated, and the strap muscles are separated in the midline. The thyroid isthmus overlies the second tracheal cartilage in most patients.

Dissection is begun at the lower border of the cricoid cartilage and the trachea is identified above and below the isthmus. Clamps are slipped beneath the isthmus, which is divided and suture ligated, providing a clean exposure of the upper trachea from the lower border of the cricoid cartilage down to the fourth ring or lower. The trachea is cleared of adventitial tissue and the thyroid isthmus is dissected laterally a few millimeters on either side to improve tracheal exposure. Occasionally, the pyramidal lobe must also be divided to improve access.

A vertical midline incision is made through the second and third rings, which are identified precisely (Figure 22-1*B*). The first ring is always left intact. In children, the third and fourth rings are selected. If rings are heavily calcified, straight scissors will crunch through them after an initial incision in the interannular space. Bleeding points may be touched with a cautery, but this must not contact an inlying plastic endotracheal tube or cuff since these can ignite and cause disastrous airway burns. The tracheal lumen is exposed. The endotracheal tube is withdrawn proximally to a point just above the incision in the trachea, but is not yet removed. Thyroid pole retractors are slipped into the tracheal lumen and the stomal edges retracted laterally (Figure 22-1*C*). If the opening is not large enough, part or all of the fourth ring may also be divided vertically.

The tracheostomy tube, with its previously tested cuff deflated, is slipped into the trachea using a small amount of water-soluble lubricant. With the tube in place, the cuff is inflated just enough to provide a seal and the volume noted. The tracheostomy tube should be no larger in diameter than is needed. Rarely does any patient require a tube larger than no. 7. A patient with small airways should not have a tube fitted snugly into the trachea. A swivel adaptor is attached to the tube and ventilation continued through the tracheostomy tube. Only when the tracheostomy tube is functioning satisfactorily, is the endotracheal tube removed. Traction sutures in the cartilaginous margins of the stoma are acceptable, but unnecessary. The skin is closed loosely with 3-0 nylon monofilament vertical mattress sutures. The flange of the tracheostomy tube is sutured to the skin with four additional sutures to prevent inadvertent extubation postoperatively. A tracheostomy tape is also tied, flexing the neck slightly as this is done so that the tape will be snug postoperatively.

Tracheostomy performed in this deliberate, unhurried way avoids postoperative hemorrhage from anterior cervical veins and the brachiocephalic vein or artery. It avoids pneumothorax, which, in the past, complicated tracheostomy, especially in children. Placement of the stoma with relation to the cricoid avoids an excessively low stoma that may later lead to erosion of the brachiocephalic artery. Division of the thyroid isthmus provides access to the proper level of the trachea and allows for enumeration of the rings so that there is no question about where the stoma is placed. Although there is no harm in excising a small amount of anterior tracheal wall for the placement of a tracheostomy tube, there is little to be gained by it. There is some hazard in teaching removal of any tracheal wall or the creation of flaps, since, if these are made excessively large, they may result in a large stoma, which tends to stenose as it heals by cicatrization. Interannular horizontal tracheostomy may lead to excessive lateral widening of the stoma by erosive pressure. In children, it has been found that vertical incision is safest since it destroys the smallest amount of tracheal wall. Pediatric tracheostomy tubes are also uniquely designed. Some tracheal wall will be damaged by necrosis due to pressure by the tube, regardless of incision. Sutures in the flange of the tube will prevent the tube from slipping out in the early period of healing before the stomal tract is well established. Sutures are removed when it is necessary to change the tube. By this time, the tract is well established. Loose closure of the incision around the tube will avoid subcutaneous emphysema.

Tracheostomy performed with such care avoids most early complications. Appropriate care of the tube from this point on avoids most later complications including stomal and cuff stenoses. Stomal stenosis is avoided by suspending the connecting tubing, thus eliminating leverage of the tube against the tracheal wall and its erosion. Cuff stenosis is avoided by scrupulously preventing overinflation of the large-volume cuff (see Chapter 11, "Postintubation Stenosis"). Semirigid esophagogastric tubes should be removed or replaced to avoid posterior erosion between the tube and the cuff, which can lead to tracheoesophageal fistula (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula"). See Chapter 10, "Tracheostomy: Uses, Varieties, Complications," Chapter 38, "Tracheal Appliances," and Chapter 39, "Tracheal T Tubes" for further considerations regarding tracheostomy and tracheostomy devices. Cricothyroidostomy is not an acceptable alternative to tracheostomy for most purposes (see Chapter 10, "Tracheostomy: Uses, Varieties, Complications").

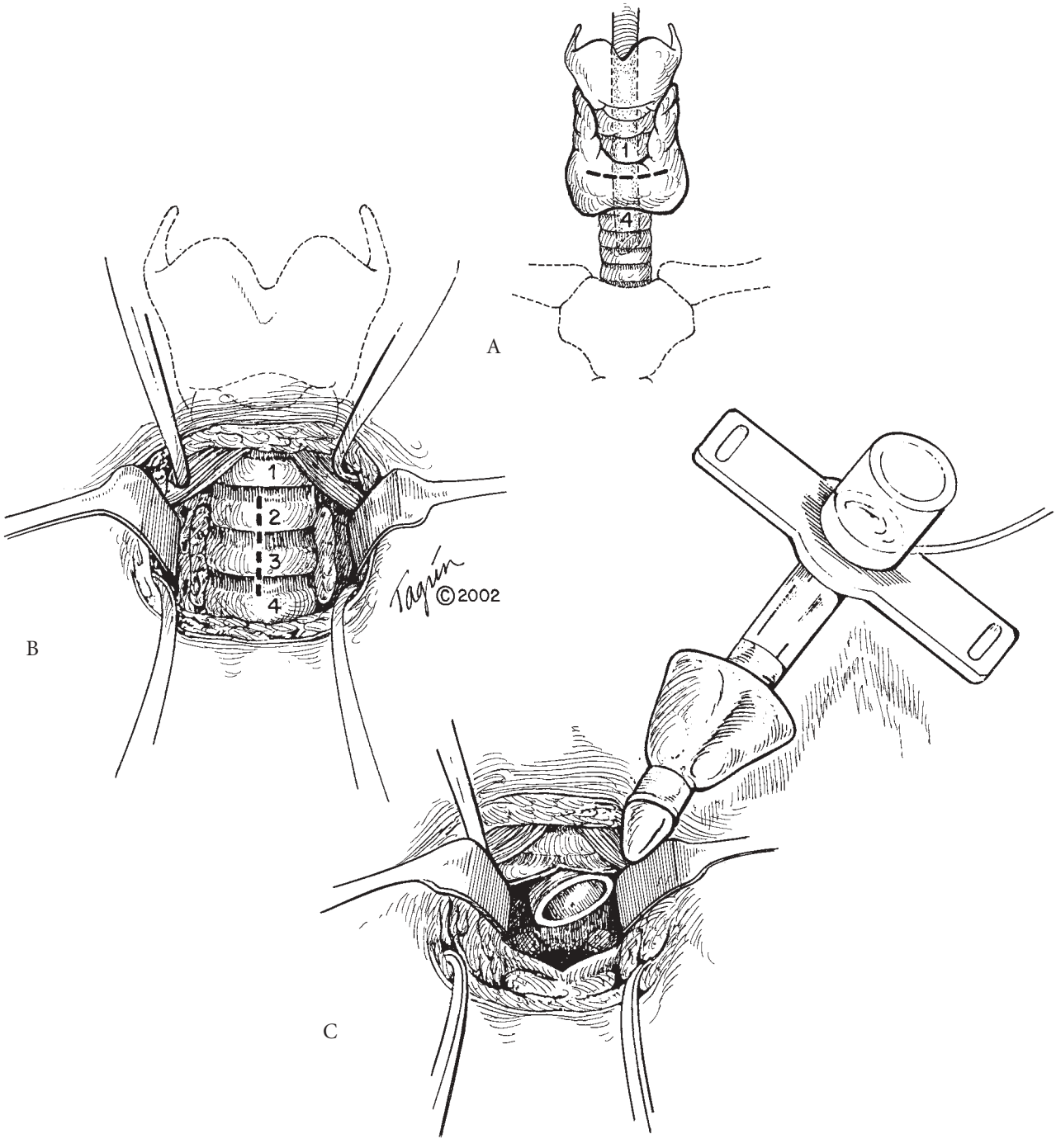


FIGURE **22-1** *Technique of tracheostomy.* A, *The patient is intubated so that tracheostomy, even if urgent, now becomes "elective." Incision (about 3 to 4 cm) is transverse and located 1 cm below the lower border of the cricoid cartilage, which is usually easily palpable. Skin and platysma are divided transversely, and upper and lower flaps are elevated to expose sternohyoid muscles. The incision frequently overlies the thyroid isthmus.* B, *The midline is opened between the sternohyoid and sternothyroid muscles. Their medial edges are elevated sufficiently to expose the lower border of cricoid, upper trachea, and thyroid isthmus. The isthmus is divided between clamps and sutures, and elevated sufficiently to expose the anterior surface of the trachea from just below the cricoid to approximately the fourth ring.* C, *The second and third rings are divided vertically, hemostasis obtained, and the tracheal walls retracted to expose the lumen. The endotracheal tube is retracted to a point just above the tracheotomy and the tracheostomy tube is inserted. In some, the fourth ring may also have to be divided. The balloon cuff of the tracheostomy tube is stripped back from the tube tip as shown, as the cuff is deflated. This facilitates passing the tube. This minor point of technique is generally useful for inserting cuffed tubes into tight stomas.*

Repeat Tracheostomy

If tracheostomy must be re-instituted in a patient who had had one previously, it should generally be located precisely at the same location as the prior tracheostomy—unless there are pressing reasons not to do so. This is especially important if there is any stenosis at the prior site, and is an absolute requirement if there is significant stenosis at the site. This avoids injury to additional trachea and simplifies the operation (see Chapter 10, "Tracheostomy: Uses, Varieties, Complications").

In these patients, only a very short transverse incision is necessary; often about l cm in length. Since prior healing has scarred all tissue layers together at this point, the tiny incision is easily carried down into the tracheal lumen. If stenosis is present, dilation may be necessary, if not already performed bronchoscopically. For dilation, serially larger tracheostomy tubes (with obturators) or uterine dilators are useful.

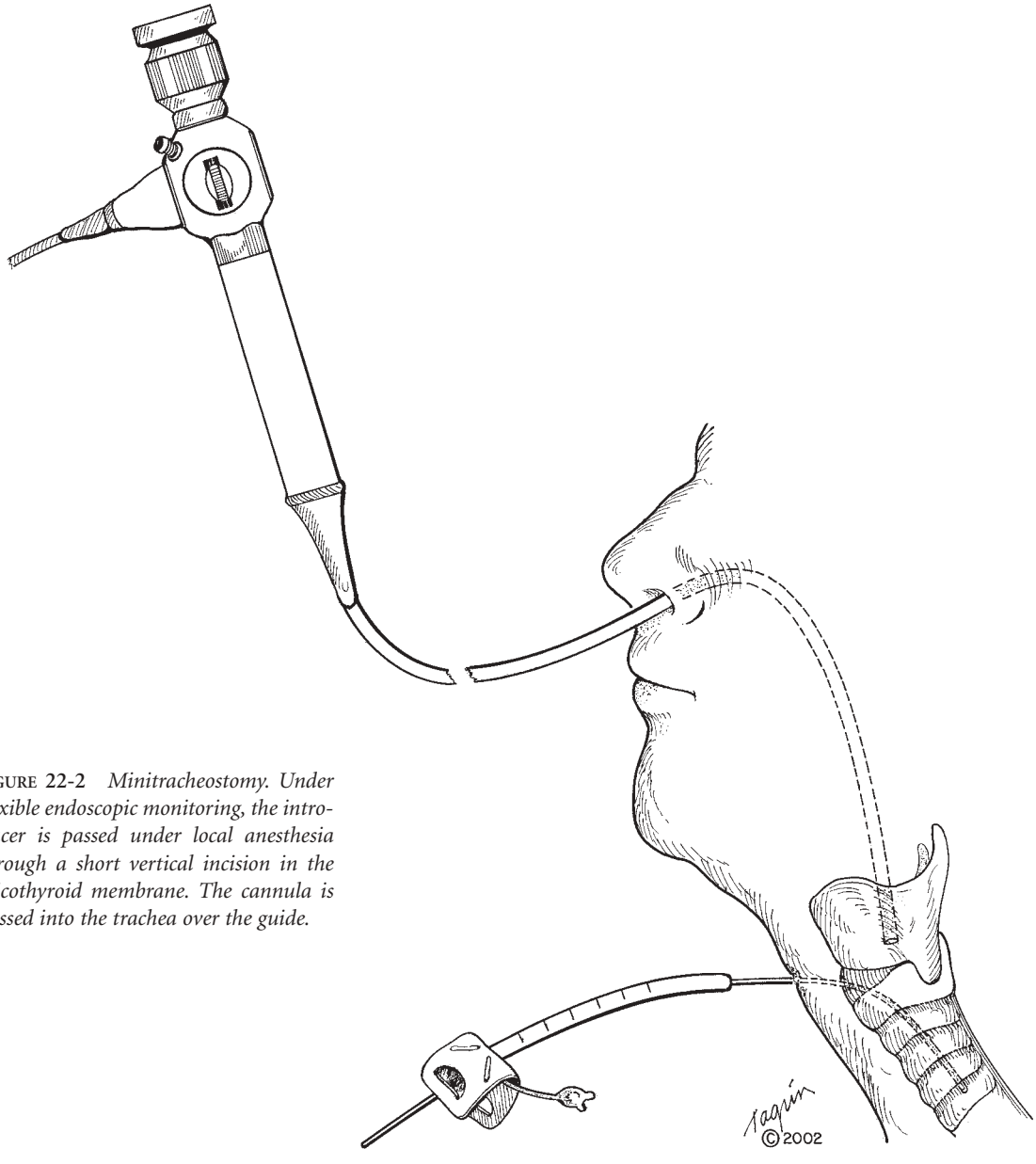


FIGURE 22-2 *Minitracheostomy. Under flexible endoscopic monitoring, the introducer is passed under local anesthesia through a short vertical incision in the cricothyroid membrane. The cannula is passed into the trachea over the guide.*

Minitracheostomy

Minitracheostomy is a technique used to assist in removal of airway secretions while maintaining glottic function, by placing an inlying small bore catheter in the trachea through the cricothyroid membrane.[1] With the neck in extension, anatomic landmarks are precisely identified: the thyroid notch, the cricoid cartilage, and the cricothyroid membrane. Five cc of 2% lidocaine hydrochloride with epinephrine is infiltrated over a site of incision in the midline of the cricothyroid membrane. The cannula is placed over a guide through a vertical incision that is 3 to 5 mm in length (Figure 22-2). A kit for the procedure contains a reclosable flanged cannula, obturator, bevelled knife blade, 15 mm adaptor, no. 10 French suction catheter, and tracheostomy tape (Figure 22-3). We have not used the Seldinger method.

Successful placement of the catheter is confirmed by listening for air exchange through the proximal end of the cannula, by aspiration of tracheobronchial secretions, by stimulation of cough with the catheter, and *routinely* by flexible bronchoscopy to observe the position of the catheter directly and rule out intratracheal bleeding. Aspiration of secretions is performed with the suction catheter. Complications following careful placement are few, but include hematoma, subcutaneous emphysema, and hoarseness. Hemorrhage is rare but potentially serious. It is best managed by immediate intubation and an inflated cuff to guarantee an airway and to tamponade further bleeding. With routine bronchoscopy, bleeding will not go unnoticed.

Percutaneous tracheostomy is discussed in Chapter 10, "Tracheostomy: Uses, Varieties, Complications."

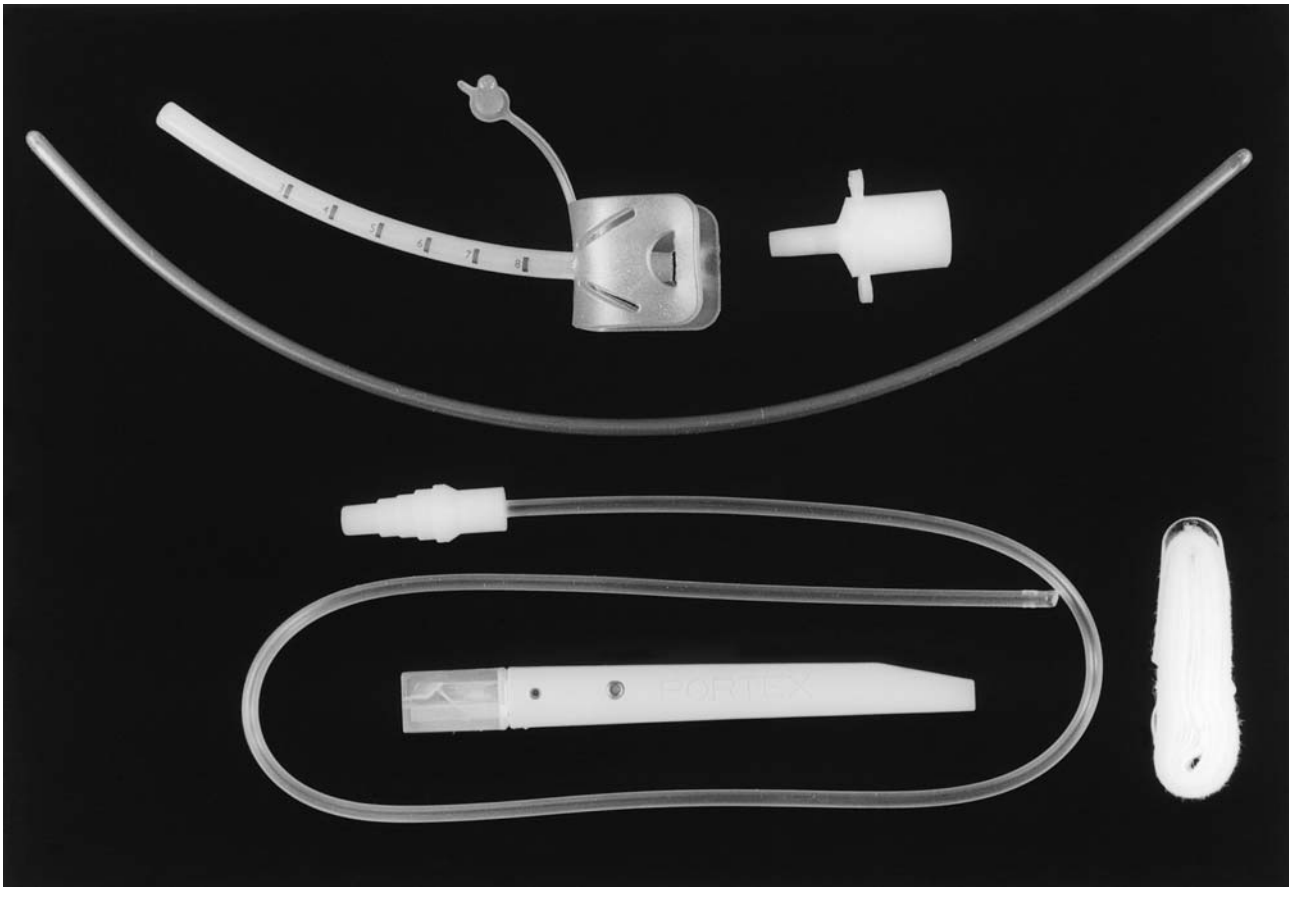


FIGURE **22-3** *Minitracheostomy kit (Portex, Keene, NH). From the* top*: cannula, with connector at* right*; introducer; suction catheter. Tracheostomy tape and disposable scalpel are also included.*

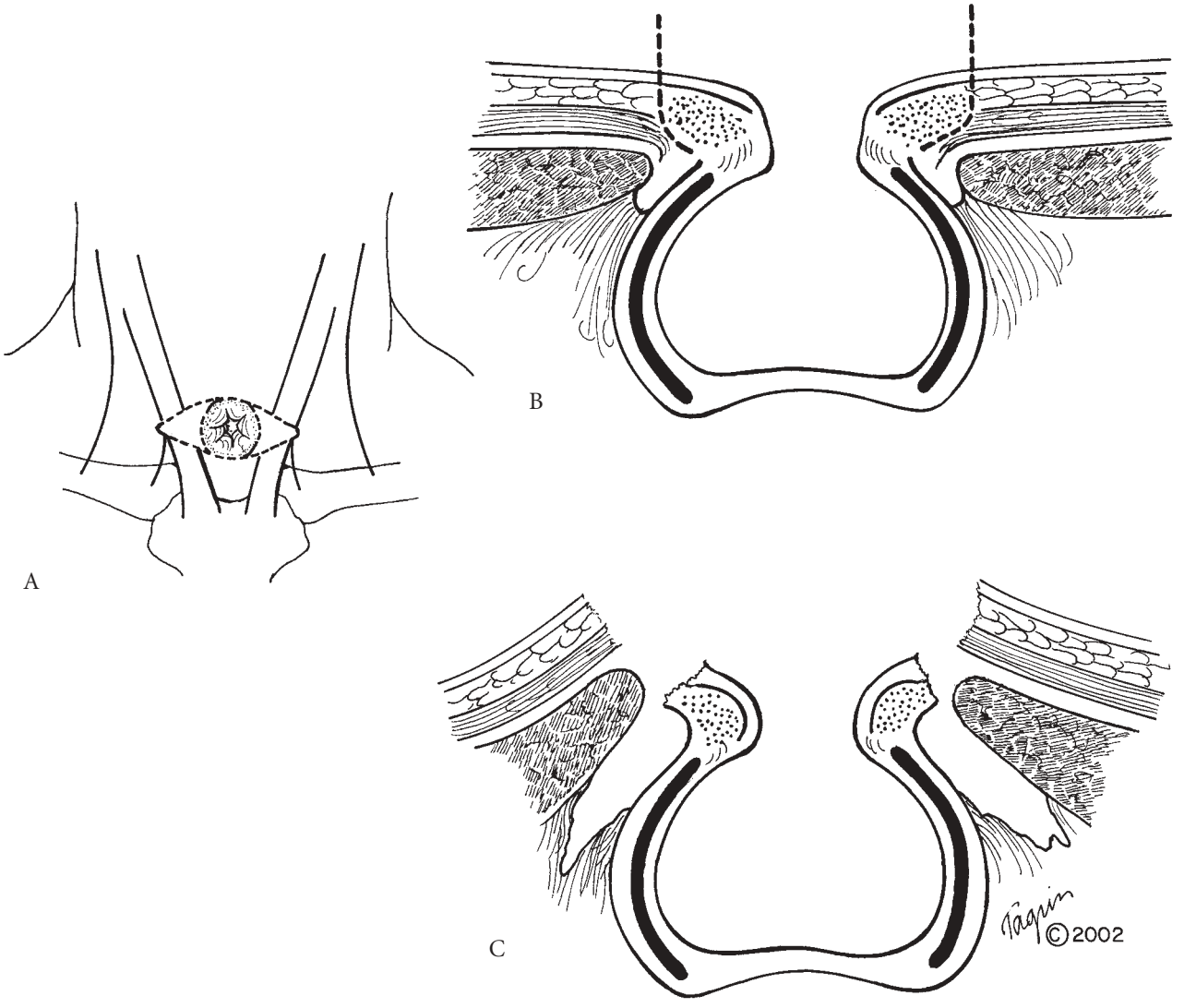


FIGURE 22-4 *Closure of persistent tracheostomy.* A, *The chronic stoma is circumscribed, leaving sufficient surrounding skin to effect closure. The tapered oval incision around the circular incision will become a transverse incision. Incision includes platysma. Superior and inferior flaps are raised over the sternohyoid muscle surfaces. Both lateral cutaneous triangles of the incision are excised.* B, *Cross section indicating cutaneous incision around stoma, carefully dissected to preserve the central blood supply of the circular flap, which arises through marginal stomal scar.* C, *The peristomal flap is elevated. The medial margins of sternohyoid muscle are dissected and elevated sufficiently to allow later midline apposition without tension.*

Closure of Persistent Tracheal Stoma

A persistent tracheal stoma most often has epithelial union of tracheal mucosa and cutaneous epithelium. Usually, a patient has had a stoma for a long time, frequently with an extended period of ventilation. Not infrequently, the patient is older, debilitated, or has been chronically on steroids. If the stoma fails to close spontaneously within 6 months following decannulation, it should be closed electively. A stoma may be closed by drawing muscle over the aperture, but this may lead to granuloma formation. I, therefore, close persistent stomas of any size with an epithelized flap using the adjacent skin, which has healed to the margin of the stoma.[2] Tracheal x-rays including fluoroscopy and bronchoscopy are advisable to rule out other lesions such as tracheomalacia, stenosis, or granuloma.

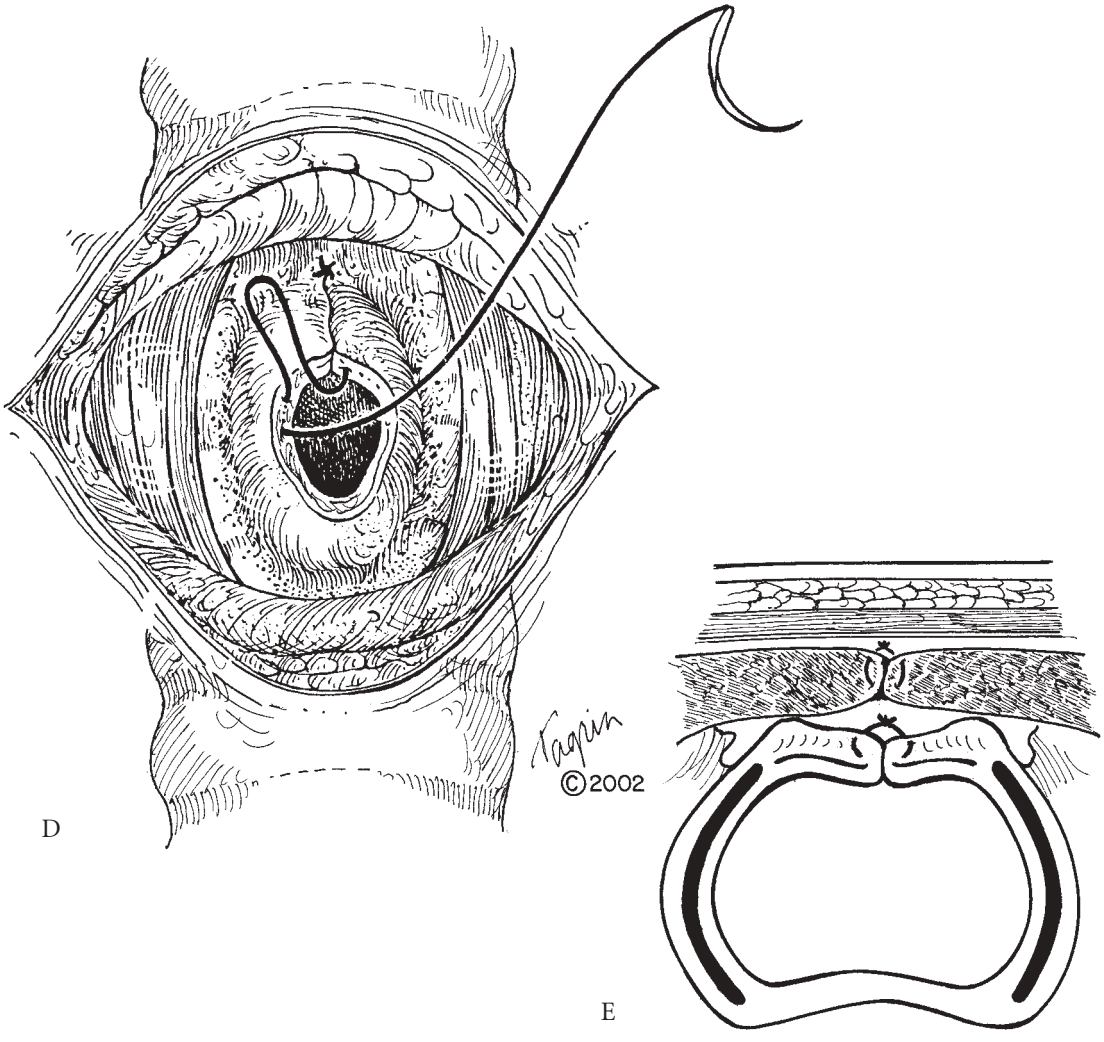


FIGURE **22-4** (CONTINUED) D, *After trimming excess skin from the margin of the flap, it is inverted and closed with running subcuticular 4-0 Dexon, presenting an epithelized surface to the tracheal lumen.* E, *The strap muscles are approximated in the midline, after which platysma and skin are sutured to close the transverse incision. A small drain is advisable for 24 hours.*

A sufficiently generous circular incision is made around the stoma to define the skin, which will be preserved and used for closure (Figure 22-4*A*). The circular incision is encompassed by a horizontal tapered ellipse and the cutaneous triangles on either side are excised. This will provide linear closure. The upper and lower skin flaps with platysma attached are elevated a few centimeters over the midline scar and trachea and over strap muscles laterally. The edge of the circle of skin is dissected up from its margins toward the center, taking care to leave an adequate breadth of circumferential attachment to the trachea (Figures 22-4*B*,*C*) where the parasitic blood supply of the flap originates. Excessive skin may be trimmed from the margin of the circular flap. The flap is inverted on itself using a subcuticular suture of absorbable material such as 4-0 Dexon (Figure 22-4*D*). The stoma is thus sealed with full thickness skin, with an epithelial surface presenting inside the lumen of the trachea. Granulomas do not form. The strap muscles are dissected from peristomal scar and elevated sufficiently so that they are easily approximated in the midline without tension. Normal tissue fullness is restored, eliminating the

unsightly pit so often seen at a tracheostomy site (Figure 22-4*E*). The mobilized flap of skin and platysma are closed transversely in layers, the skin with a subcuticular suture to provide a cosmetic scar. A small drain for 24 hours is advisable. The single complication I encountered after this procedure was a hematoma in an undrained patient.

The method is not applicable where the stomal margin is still surrounded by granulation tissue. This closure may also be used during tracheal resections for another lesion such as an inferior stenosis, where stomal closure is desired. Otherwise, muscle flap closure is advisable, after debridement of marginal granulations.

References

1. Matthews HR, Hopkins RB. Treatment of sputum retention by minitracheostomy. Br J Surg 1984;71:147–50.
2. Lawson DW, Grillo HC. Closure of a persistent tracheal stoma. Surg Gynecol Obstet 1970;130:995–6.

Surgical Approaches

Hermes C. Grillo, MD

The choice of surgical approach to a specific tracheal lesion is based principally on diagnosis and on observations of the extent and location of the lesion, made from imaging studies and rigid bronchoscopy, using magnifying telescopes. Also important in the equation are age, body build, and prior treatment, especially operative procedures and irradiation. Specific diagnostic factors that are assessed include 1) whether the lesion is benign or malignant; 2) the exact location of the lesion; 3) the extent of the lesion, both longitudinally and laterally (including possible involvement of other organs, such as the larynx or esophagus); 4) the state of the glottic aperture and recurrent laryngeal nerve function; and 5) the length of the normal trachea that may predictably be left, after adequate excision of the lesion.

Since detailed endoscopy is often deferred until the time of the proposed operation, it may be advisable, in the case of extensive lesions, to complete a bronchoscopy before the anesthesiologist inserts arterial or other monitoring lines, both to determine operability and also to place lines in the appropriate arm. Specific diagnosis is of great importance. A well-defined inflammatory stenosis can be delimited both radiologically and endoscopically. On occasion, there may be more unexpected inflammatory injuries, especially proximal to a stenosis. A benign tumor is easily delimited visually. However, malignant tumors or those of borderline malignancy often extend further than is grossly evident. This is particularly true with adenoid cystic carcinoma. Squamous cell carcinoma is also sometimes poorly definable visually. Therefore, it may be strategically necessary, particularly with tumors, to position and drape a patient so that the initial incision may be extended in a planned fashion, as additional access becomes needed.

Approach to the trachea should be determined by the characteristics of the lesion and patient and not in accord with prejudices related to the surgeon's training or specialty. I have equal impatience with a thoracic surgeon who insists on approaching a lesion transthoracically, which would be better and more simply managed through an anterior approach, as I do with an otolaryngologist who poses the question, "How much trachea can be removed through the neck alone?" If a surgeon is to perform effective tracheal resection and reconstruction, then that surgeon must become familiarized with the anatomy of the larynx, the trachea, the neck, the mediastinum, and the thorax. The range of potentially necessary techniques should be available to the surgeon,

or there should be cooperation with colleagues who can compensate for the surgeon's deficits. The alternative is referral to a center where such work is done regularly. I hope it is superfluous to point out that the question to be asked at initial evaluation is not "What service am I equipped to provide this patient?" but "What service does this patient need?"

This chapter discusses approaches to tracheal lesions of different types and at different levels.[1,2] Detailed descriptions of resection and reconstruction are found in subsequent chapters.

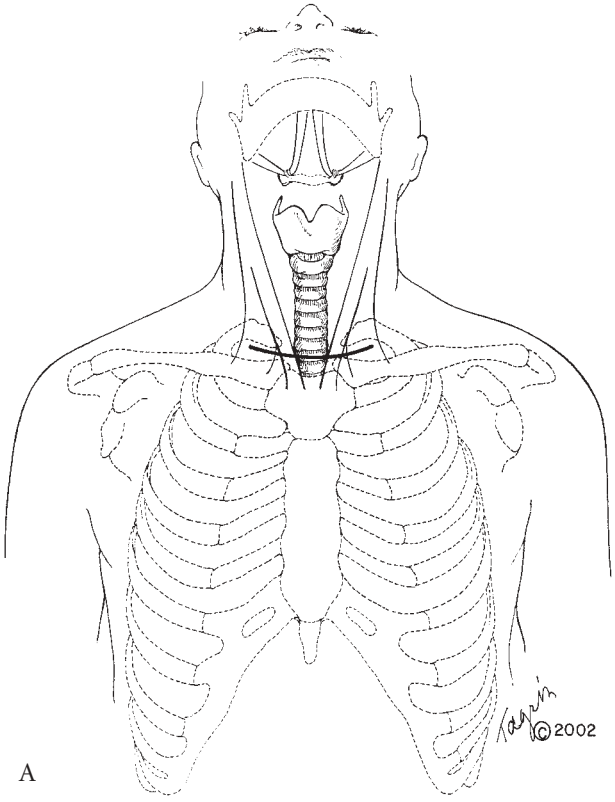
Upper Tracheal Lesions

Most benign stenoses can and should be resected through an anterior approach, even when located at the supracarinal level. Stenosis involving the uppermost trachea, or the lower larynx and upper trachea, will almost uniformly be resectable and reparable through a low collar incision alone (Figure 23-1*A*). A low incision results in more cosmetic scar than in one placed higher across the neck. The length of incision will depend upon the vertical spread of incision needed in each patient, usually that which is sufficient to reach the cricoid above and the sternal notch below. A stenosis that extends further into the mediastinum requires additional exposure, which is obtained by dividing the upper sternum just through the sternal angle (Figure 23-1*B*), the junction of manubrium and corpus sterni. In short-necked, older patients, with a low-lying larynx, and a trachea that fails to rise into the neck on cervical extension, it is advisable to proceed to an upper sternal division early in the operation, even where the lesion is high in the trachea, since it is clear that even if resection can be accomplished through a collar incision, the anastomosis would be unnecessarily difficult. Sternotomy should be added as soon as it becomes evident that it will be necessary, since the added exposure greatly facilitates dissection of the trachea low in the neck. A vertical limb is most often added to the cutaneous incision to form a "T" with the collar incision. A low "apron" incision is also effective if the need for upper sternotomy is unquestionable from the outset (Figure 23-1*C*). In upper sternotomy, it is not necessary to angle the bony division laterally to an interspace, since the sternum will fracture appropriately as the retractor is opened. Morbidity from an upper sternal division is minimal, so it may be used quite freely, even in patients with poor lung function. Complete sternal division is unnecessary and adds nothing to the ease of exposure. Full sternotomy in such cases contributes only increased postoperative pain and potential complications.

The pretracheal plane is dissected and the great vessels are retracted forward. For very distal benign lesions, described later, lateral traction sutures are placed at the tracheobronchial junction, on either side, through this cervicomediastinal approach. It is not necessary in these cases to dissect the lower trachea transpericardially between the superior vena cava and aortic arch beneath the brachiocephalic artery. I have used this anterior approach successfully, even in patients who have had prior transthoracic low tracheal resection for stenosis (postintubation and traumatic).

In resections for postintubation lesions, an existing stoma, frequently present, should not unduly influence placement of the collar incision. The incision is electively placed within 1 cm of the level of the clavicular heads (see Figure 23-1*A*). If a stoma is only a bit superior, then the collar incision may be made higher, excising the stoma elliptically (Figure 23-2*A*). However, if the stoma is still higher, then the stoma may be separately excised (Figure 23-2*B*). Thus, if an inferior vertical limb of incision becomes necessary in order to divide the upper sternum, then a long vertical tethering scar will not result in the neck.

When a malignant tumor of the upper trachea is approached, it is prudent to prepare and drape the patient so that extensions can be made through the entire length of the sternum (Figure 23-3*A*) or into the fourth right interspace (Figure 23-3*B*), should gross findings on frozen sections indicate a need for extended resection. Positioning is described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection." The cutaneous incision in this case lies just below the inframammary crease, but entry into the chest is in the fourth interspace, just over the fifth rib. On occasion, a left fourth interspace extension is also useful for access to the left hilum (Figure 23-3*C*).



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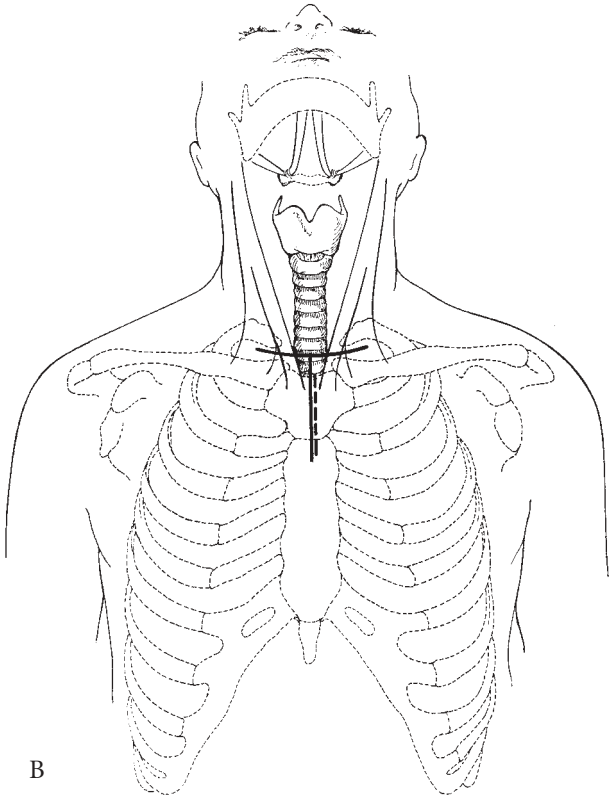

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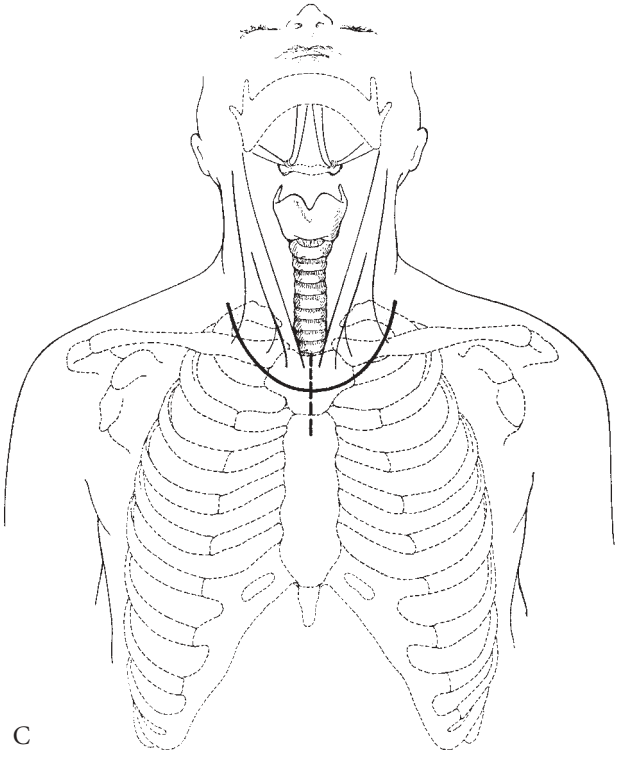


C

FIGURE **23-1** *Incisions for approach to the upper trachea.* A, *A collar incision is electively placed 1 cm above the clavicular heads. Length is determined by the extent of superior exposure required.* B, *Extension for upper mediastinal access (cervicomediastinal resection). The vertical arm of the cutaneous incision reaches just below the sternal angle. The sternum is also divided just beyond the angle (*dashed line*). It is unnecessary to divide the bone laterally at the distal end since it will fracture appropriately as the retractor is opened.* C, *An apron incision allows the same access but removes most of the incision from the neck.*

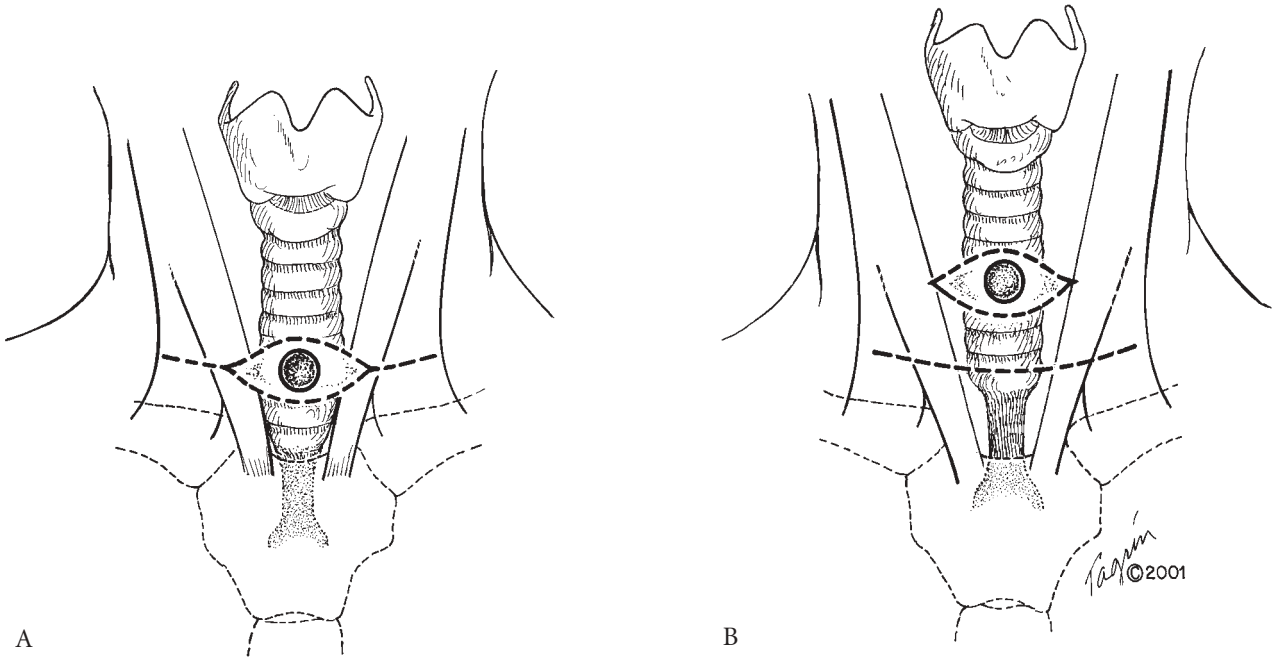
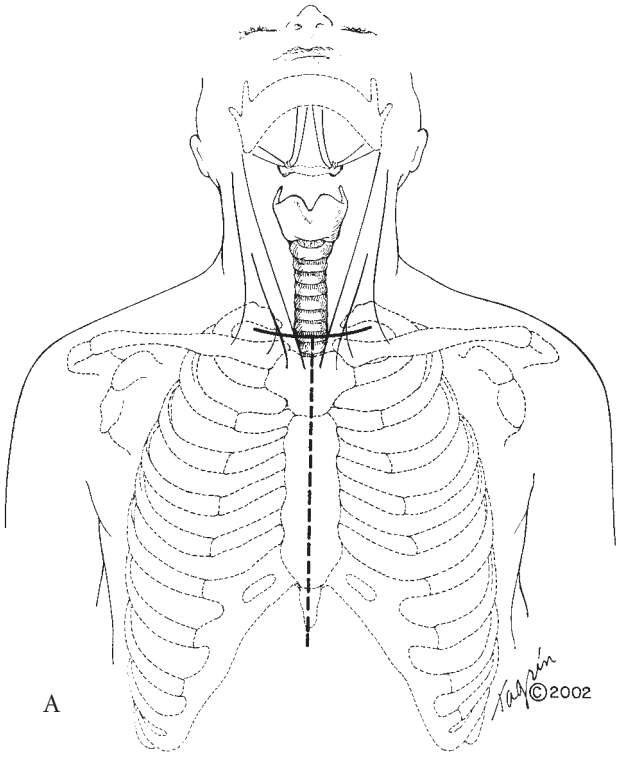


FIGURE 23-2 *Placement of a cervical incision with a preexisting stoma.* A, *A fairly low stoma is elliptically excised with a collar incision.* B, *If the stoma is high, then the collar incision is placed at the elective level and the stoma separately excised as the superior flap is raised.*

In anterior approaches, either cervical or cervicomediastinal laryngeal release, if necessary, may be performed best through a short transverse incision over the hyoid bone (Figure 23-4). The long U-shaped flaps favored by many otolaryngologists add nothing to the exposure and leave an unsightly scar, compared with two transverse "ladder" incisions. In a short-necked, older individual, with the larynx fixed at the suprasternal notch even despite cervical extension, a single collar incision carried a little further laterally on both sides may be elevated to provide access to the hyoid region for laryngeal release. The possibility of a high incision over the hyoid bone must be recalled when the field is draped.

On occasion, in order to preserve the skin of the upper chest below the sternal notch, I have used a long, U-shaped incision that ends laterally out on each clavicle, with the midpoint of the incision just above the level of the sternal angle (Figure 23-5*A*). This flap has also been used for cosmetic reasons, to move the incision below the neckline. When the superior flap is elevated, access to the entire trachea and lower larynx is easily obtained. In a rare case where it may be thought that the skin will have to be preserved in connection with a complex cutaneous reconstruction or for mediastinal tracheostomy, a similar approach may be used, if necessary in combination with a vertical sternotomy or a "J" incision, which provides access to the right thorax as well (see Figure 23-5*A*). Access to the entire neck and to the upper sternum as well is also provided through a long supraclavicular incision (Figure 23-5*B*). This is useful for initial dissection, where cervicomediastinal exenteration is a possibility (see Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy").

Postintubation tracheoesophageal fistula is usually reparable through a collar incision, but it occasionally requires an upper sternal division. Urgent surgical management of a fistula into the brachiocephalic artery is expedited by a collar incision plus complete vertical sternotomy (see Figure 23-3*A*) or partial sternotomy angled into the right third interspace. These provide additional lateral exposure of the arteries.



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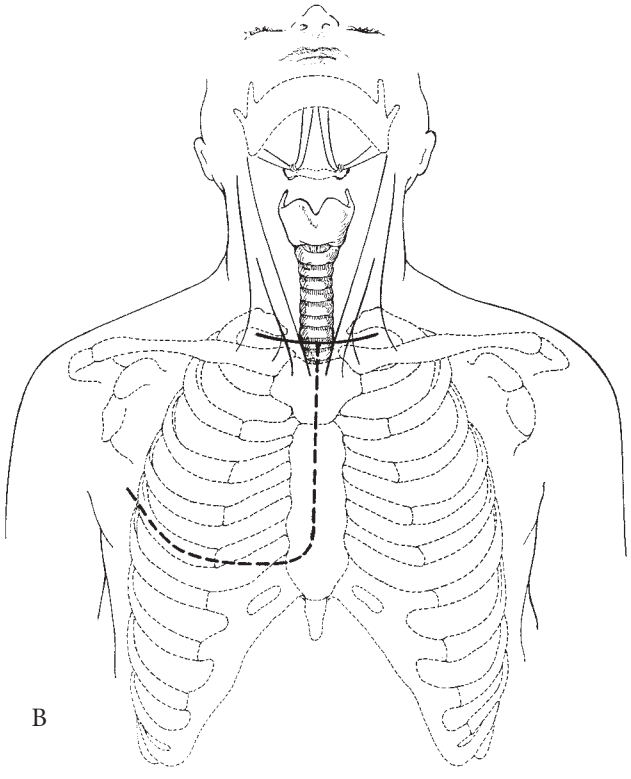

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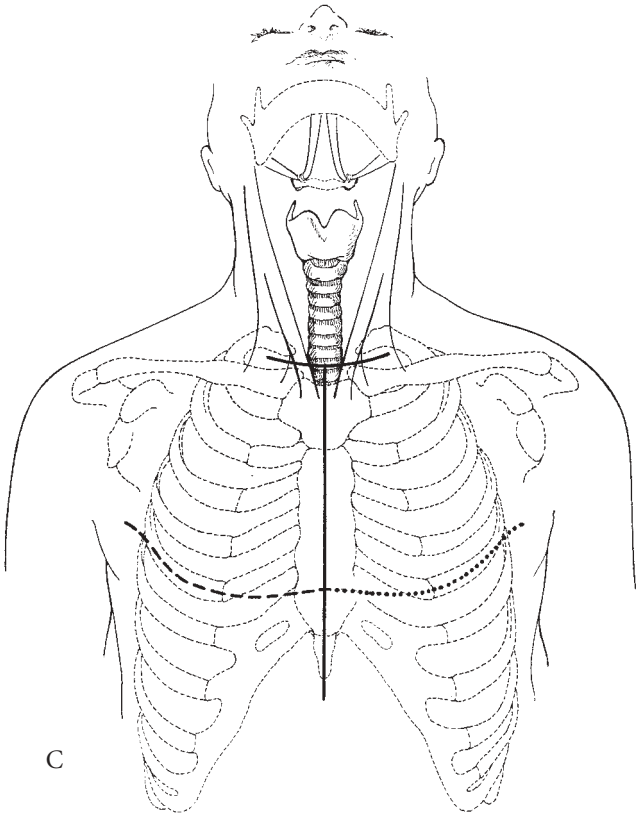


C

FIGURE **23-3** *Extended tracheal resection.* A, *Complete sternotomy. The cervical incision is still needed for exposure of the upper trachea and larynx. Access to the lower trachea and carina is made possible.* B, *Incision permitting access to the entire trachea from the larynx to the carina. The cutaneous incision (*dashed line*) is submammary and the thoracic wall incision lies in the fourth interspace. The pectoralis muscle is elevated with the cutaneous flap.* C, *Further variations in access. Either right or left hemithoraces may be exposed by lateral extensions into fourth interspaces, if sternotomy is inadequate.*

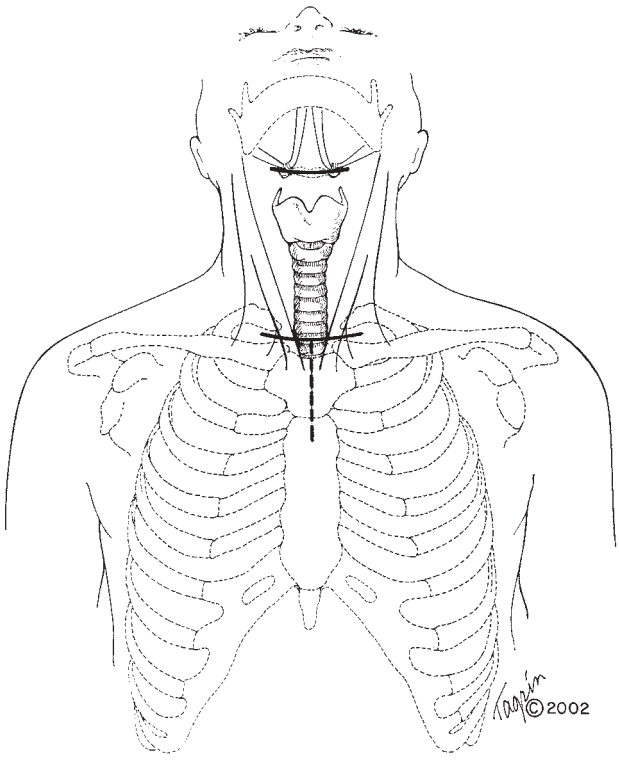
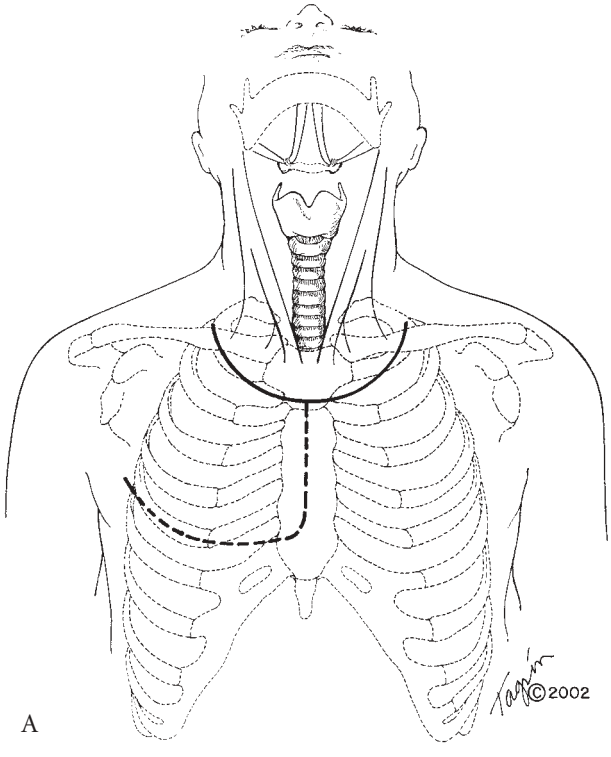


FIGURE **23-4** *The incision for the suprahyoid laryngeal release lies over the hyoid bone. The skin flap between "laddered" incisions does not have to be dissected free, except as needed for exposure. This is preferable to a long "U" incision.*



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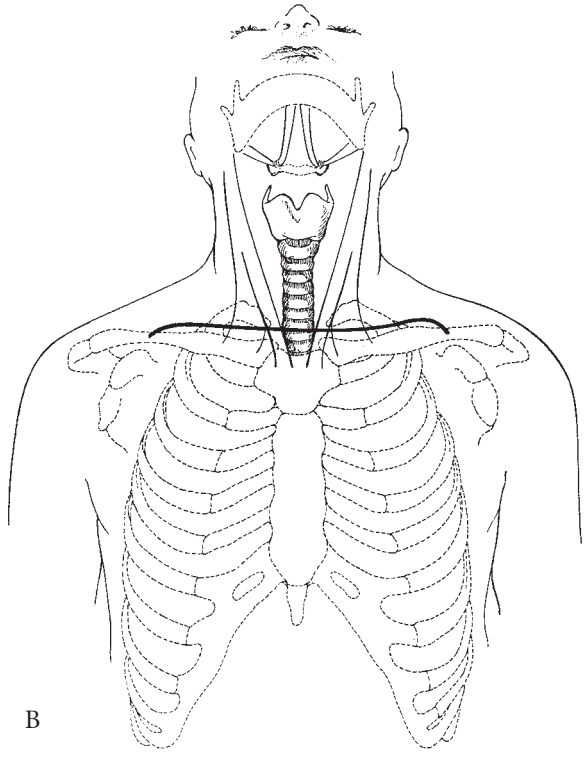


B

FIGURE **23-5** *Incisions for special circumstances.* A, *The "apron" flap preserves the site for possible mediastinal tracheostomy. It also cosmetically removes incision from the lower neck. The* dashed line *indicates the possible extension of the field for access to the lower trachea and carina.* B, *Supraclavicular incision, which allows for complete exploration of the neck and mediastinum (via upper sternotomy under the lower flap). For use where cervicomediastinal exenteration is likely.*

Midtracheal Lesions

A lengthy benign midtracheal lesion or a malignant lesion of midtrachea, which proves microscopically to be of greater length than anticipated, will not necessarily be most accessible through posterolateral thoracotomy, either for resection or for reconstruction. Several approaches are possible in this potentially difficult situation. The patient should be positioned so that possibilities for successively wider accesses are available. A supporting roll may be placed vertically beneath the chest of the supine patient, to the right of the midline, with the patient's arm abducted to a point where access to the right chest is available, as far as the anterior border of the latissimus dorsi muscle. A lateral tilt table permits levelling of the anterior chest wall so that the position during initial incision remains horizontal. Exploration may be commenced through a collar incision with upper sternal division. If further access is needed, a complete median sternotomy may be added (see Figure 23-3*A*) or an extension made into the right chest (see Figure 23-3*B*). The cutaneous incision angles out from the sternotomy beneath the mammary crease to the posterior axillary line, elevating the skin and pectoralis muscle so that the thorax is entered through the fourth interspace. It is also possible to "T" a full sternotomy to the right or left (see Figure 23-3*C*).

These approaches permit the use of mobilization maneuvers at both ends of the trachea, both of which may be required for reconstruction after removal of a lengthy midtracheal lesion. Laryngeal release may be done in the usual manner through an additional short transverse incision over the hyoid bone (see Figure 23-4). Mobilization of the right hilum by intrapericardial release can be accomplished either through the fourth interspace incision, or less easily through a full median sternotomy after opening the pleura. Median sternotomy does not easily permit transpleural left-sided intrapericardial release, since the required degree of traction on the heart is not well tolerated. A "T" into the fourth left interspace facilitates left hilar release (see Figure 23-3*C*). A preferable option for left hilar release via a complete sternotomy was devised by Dr. Cameron D. Wright at Massachusetts General Hospital. Through the open pericardium, with gentle retraction of the heart, a U-shaped incision is made in the pericardium, beneath and around the inferior pulmonary vein. The right-sided release is better accomplished transpleurally because intrapericardial access to the right inferior pulmonary vein is made difficult by the right atrium and vena cava. Bilateral intrapericardial release may also be accomplished via bilateral submammary thoracotomy, but access to the upper trachea is limited unless the sternum is also split vertically.

Complete median sternotomy allows entry into a quadrilateral space framed by the medial border of the superior vena cava, the medial border of the ascending aorta, the inferior margin of the brachiocephalic artery and, below, the right pulmonary artery. The pericardium is opened in the front and in the back, exposing the lower trachea and carina (Figure 23-6*A,B*). This approach was described by Abruzzini and amplified by Perelman and colleagues, who advised opening the pericardium anteriorly and posteriorly.[3,4] Access is deep and restricted, making major dissection and complex carinal reconstruction difficult.

Lower Tracheal and Carinal Lesions

Tumors of the lower trachea and carina are best approached through a high right posterolateral thoracotomy (Figure 23-7). This is particularly true for tumors of the carina and for more extensive tumors of the lower trachea. I prefer this approach for tumors of the lower trachea of any complexity, although benign stenoses and simple tumors at this level are approached anteriorly. Pearson and colleagues prefer a median sternotomy,[5] whereas Perelman,[6] who initially favored the transsternal approach to mainstem bronchial fistulae, prefers right thoracotomy for lower tracheal and carinal tumors. Perelman has also largely abandoned the transsternal approach for fistulae. For special cases, one must keep in mind both the J-shaped trap door incision (see Figure 23-3*B*), a "T" extension from median sternotomy (see Figure 23-3*C*), and bilateral thoracotomy (Figure 23-8).

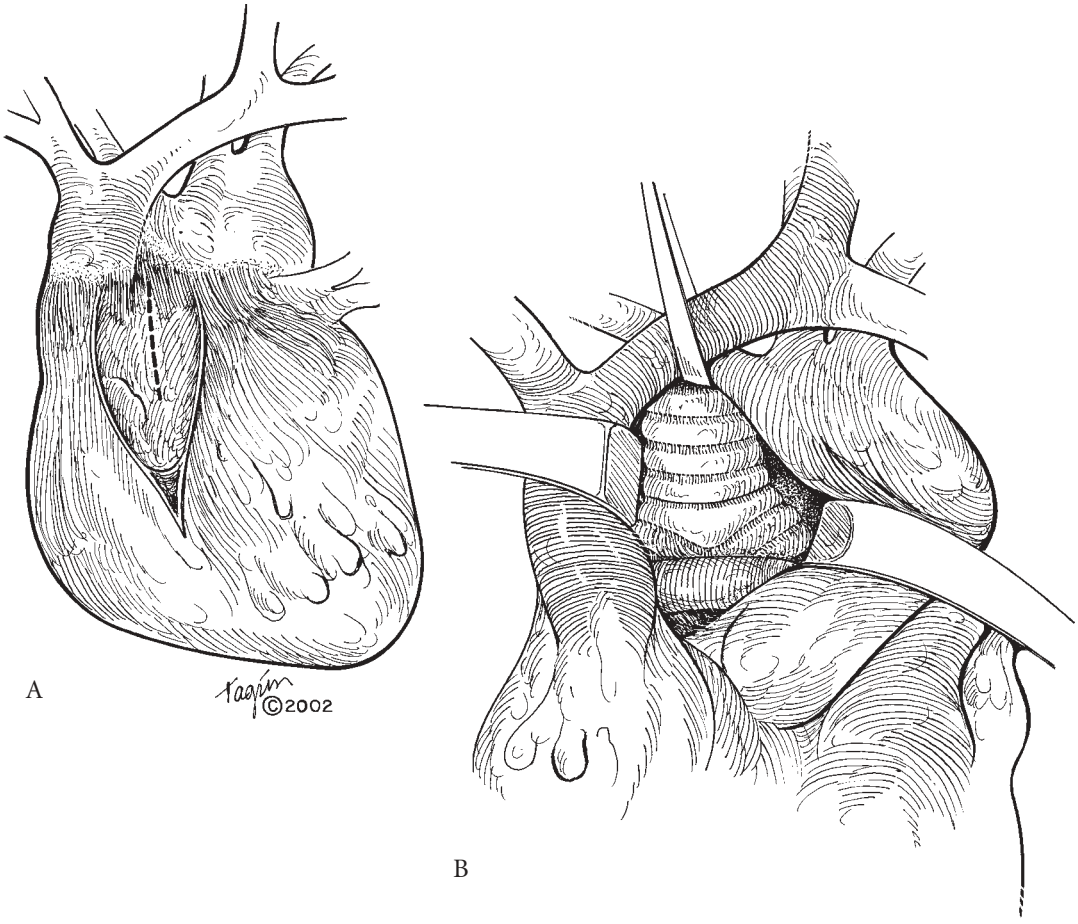


FIGURE **23-6** *Transmediastinal approach to the entire trachea. The sternum is fully divided, as shown in Figure 23-3*C. A, *The anterior pericardium is opened vertically between the superior vena cava and the aorta. The posterior pericardium is similarly opened (*dashed line*).* B, *Retraction of the vena cava and aorta exposes a quadrilateral space in which the lower trachea and carina are seen. A tape around the brachiocephalic artery and vein helps exposure. The right pulmonary artery lies just below the carina.*

Formerly, when a right thoracotomy was elected for resection of a tumor of greater length in the lower trachea, the patient's right arm was draped into the field and the neck also prepared. This permits access, with some difficulty, for laryngeal release, by swinging the arm to the side and laterally tilting the operating table to a more horizontal position. Laryngeal release is now used only where the lesion reaches up to midtrachea, since it has *not* been found to provide increased mobility for reconstruction of the lower trachea or carina (see Chapter 29, "Carinal Reconstruction").

Left posterolateral thoracotomy may be used for treating a tumor that involves the carina as well as the left main bronchus to such extent that the left lung cannot be salvaged. It is quite possible to perform left pneumonectomy, excise the carina (see Chapter 29, "Carinal Reconstruction"), and perform end-to-end anastomosis between the trachea and the right main bronchus beneath the aortic arch from the left side. Tracheal excision must be very limited when this approach is used. Swinging the arch of the aorta forward from the left after dividing four upper intercostal arteries, as described by Björk,[7] does not provide good access for tracheal procedures.[8]

If tumor at the carina involves any significant length of trachea, and also enough of the left main bronchus, to force consideration of a left pneumonectomy as well as tracheal and carinal resection, then the

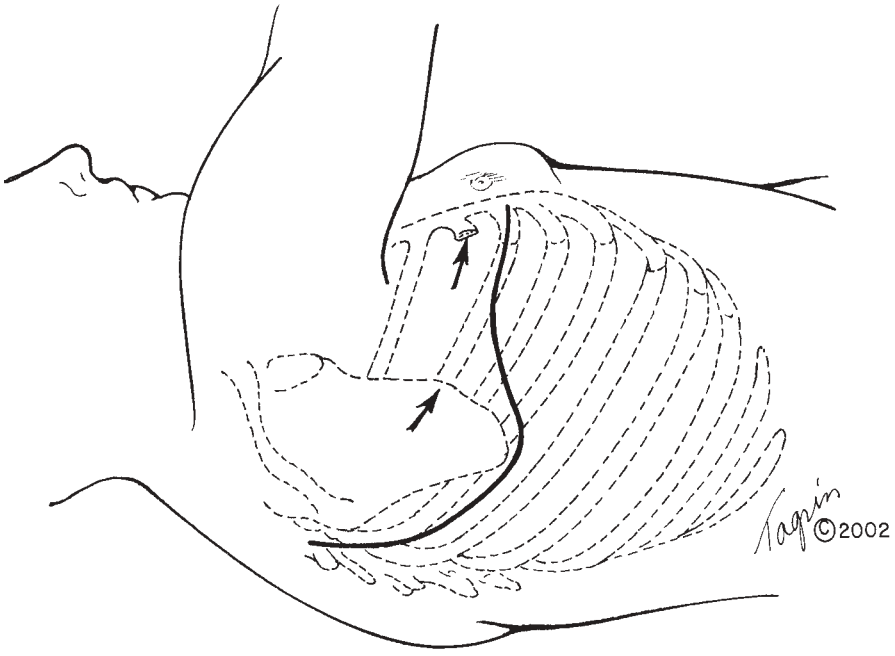


FIGURE **23-7** *Transthoracic approach provides excellent access to the lower trachea and carina, including the left main bronchus. The posterior cutaneous incision lies midway between the vertebral spine and posterior scapular border. The incision curves beneath the scapular tip, is directed transversely forward, and then curves inferiorly in its anterior portion, in order to overlie the anterior portion of the fourth and fifth ribs; hence, the "Lazy S" configuration of the incision. Individuals vary, but resection of the fourth rib is usually optimal (*anterior arrow*). For airway dissection of any complexity, a posterolateral thoracotomy has been found to be more advantageous than a limited thoracotomy.*

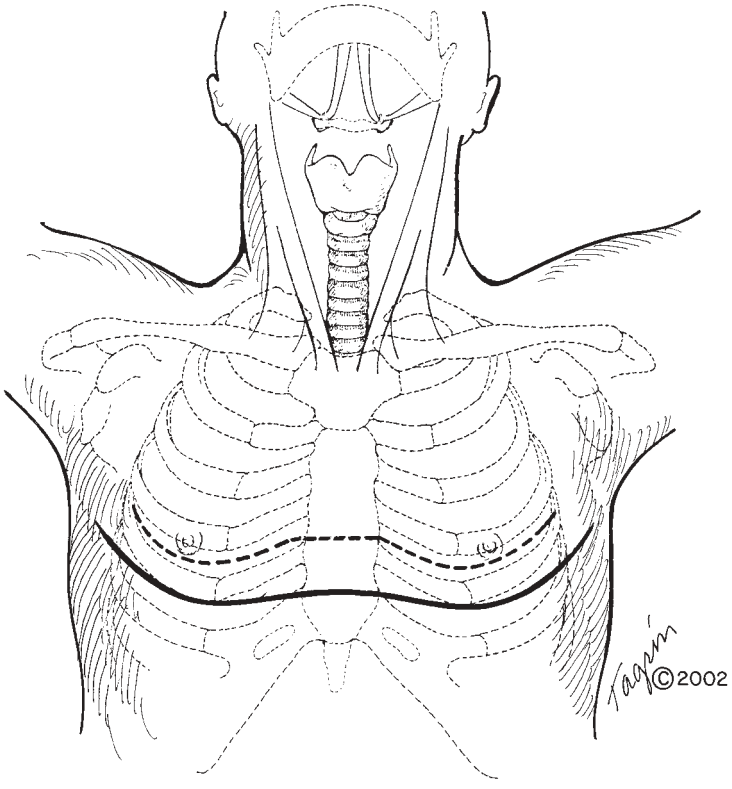


FIGURE **23-8** *Bilateral thoracotomy incision. The skin incision (solid line) is submammary. The thorax is entered via the fourth interspaces with transverse sternotomy (*dashed line*). For initial exploration, the skin incision may be limited and only one hemithorax opened, adding transverse sternal division to widen the initial thoracotomy exposure. Pectoralis muscles are elevated with the skin flap to expose intercostal muscles.*

incision providing the best exposure is a bilateral thoracotomy extending through the fourth interspace from one posterior axillary line to the other across the sternum, accomplished through a submammary incision (see Figure 23-8). In some cases where the feasibility of resection must be determined operatively, the right-sided portion of the incision is made initially, with transverse division of the sternum, but without opening the left pleura. This provides excellent exposure. The incision is completed when resectability is assured. The use of epidural analgesia postoperatively has minimized the significant impact of bilateral thoracotomy on respiration. Caution is still recommended. Bilateral thoracotomy was earlier used for cardiac surgery. We modified it for the purposes described. It was later applied to double lung transplantation.

An alternative approach to carinal resection with accompanying left pneumonectomy is median sternotomy (with right pleurotomy) plus a left-sided "T" (see Figure 23-3*C*). Earlier solutions to this problem included staged right and left thoracotomies, still occasionally useful in unusual circumstances. Perelman performed a right thoracotomy for excision of the carina and anastomosis, leaving the left lung in situ, with the bronchus closed.[6] It became necessary to ligate the left pulmonary artery to avoid symptomatic shunting. The bronchial arteries nourished the remaining defunctioned left lung. This approach is no longer used.

Approach to *cervicomediastinal exenteration* of the larynx, trachea, and esophagus is described in Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy."

References

1. Grillo HC. Surgical approaches to the trachea. Surg Gynecol Obstet 1969;129:347–52.
2. Grillo HC. Surgery of the trachea. In: Current problems in surgery. Chicago: Yearbook Medical Publisher, Inc.; 1970 (July).
3. Abruzzini P. Trattamento chirurgico delle fistole del bronco principale consecutive a pneumonectomia per tuberculosi. Chir Torac 1961;14:165.
4. Perelman MI, Rymko LP, Ambatiello GP. Bronchopleural fistula: surgery after pneumonectomy. Int Trends Gen Thorac Surg 1987;2:407–12.
5. Pearson FG, Cardoso P, Keshavjee S. Upper airway tumors. In: Pearson FG, Deslauriers J, Ginsberg RJ, et al, editors. Thoracic surgery. New York: Churchill Livingstone; 1995. p. 285–99.
6. Perelman MI. Surgery of the trachea. Moscow: Mir; 1976.
7. Björk VO. Left-sided bronchotracheal anastomosis. J Thorac Surg 1955;30:492–8.
8. Maeda M, Nakamoto MD, Tsubata N, et al. Operative approaches for left-sided carinoplasty. Ann Thorac Surg 1993;56:441–6.

Tracheal Reconstruction:
Anterior Approach and Extended Resection

Hermes C. Grillo, MD

Anterior Tracheal Reconstruction
Extended Resection

The cervical and cervicomediastinal approach is used for upper tracheal tumors, for limited tumors of the mid and even lower trachea, and for almost all benign strictures of the trachea at any level. Although more than half of the trachea may be removed through such an approach in young patients of appropriate body habitus, and approximation gained by cervical flexion alone, much less may be resected in the older, kyphotic, heavy-set patient, or in a patient who has undergone extensive prior tracheal surgery. Pretracheal mobilization and cervical flexion are key in this approach. Laryngeal release is the adjunctive procedure most easily added in this approach if further relaxation of anastomotic tension is needed.

I use the term "cervicomediastinal" to indicate the addition of upper sternotomy to the cervical approach. The uses of complete sternotomy are discussed in Chapter 28, "Reconstruction of the Lower Trachea (Transthoracic) and Procedures for Extended Resection." The upper mediastinal approach is easily converted to full sternotomy for transmediastinal exposure and may be further widened with a thoracic component if needed. Such potential extensions should be planned for preoperatively.

Anterior Tracheal Reconstruction

Anesthesia, Bronchoscopy, Intubation

The patient is usually induced with inhalation anesthesia and a rigid bronchoscope passed. Even if a tumor nearly occludes the trachea, it usually arises only from a portion of the tracheal circumference. The bronchoscope may be passed beyond the tumor beside that portion of the trachea not involved by tumor base, even against cartilaginous wall. In the case of the rare circumferentially based tumor, the rigid bronchoscope may be inserted through the residual central lumen initially, using the dilation technique described in Chapter 19, "Urgent Treatment of Tracheal Obstruction." If the airway in a patient with benign stenosis measures less than 6 mm in diameter, then it is routinely dilated under direct vision at this point, using the technique described. The airway must be adequate from the start to avoid retention of CO_2 and possible arrhythmia during early phases of operation. An endotracheal tube is passed either beside a high

or obstructing tumor, through the tumor or, in the case of stenosis, through the lesion. Apparent near-occlusion of the trachea is *never* an indication for cardiopulmonary bypass. If the lesion is lower and the obstruction is not critical or has been previously dilated, then the endotracheal tube may initially reside above the lesion. Management of an obstructing tumor that is very low in the trachea or at the carina is discussed in Chapter 19, "Urgent Treatment of Tracheal Obstruction." A critically obstructed airway must be opened in a rapid but wholly systematic way to avoid risking fatality. The special problem of critical subglottic stenosis in the larynx is also discussed in Chapter 25, "Laryngotracheal Reconstruction."

If the lesion is high or in midtrachea, then a standard full-length endotracheal tube is used. If the lesion lies low in the trachea, then the patient is intubated with a proximally extended tube with an armored flexible distal component (Figure 24-1). This allows the tube to be advanced into the left main bronchus, if needed. If a stoma is present, then intubation is still often carried out perorally as the most convenient technique for managing a tracheal lesion. In cases of tight stenoses that involve the subglottic intralaryngeal airway in addition to the uppermost trachea, and in patients in whom there is discontinuity of the airway due to stenosis between the larynx and the trachea, and where the stoma resides below the obstruction, intubation is done with a standard flexible armored tube through the stoma and induction carried out in this way. The tube is removed to permit rigid bronchoscopic examination of the distal trachea and then replaced. The operative field is prepared around such a tube, which is held vertically so that it will not contaminate the field, and the field is draped. A sterile endotracheal tube and necessary equipment are made ready at the operative field. The inlying tube is then removed, the sterile adhesive drape applied, and then the fresh sterile tube with sterile connecting tubing is placed through the sterile adhesive drape and the ends of the anesthesia tubing are handed off the field to the anesthetist (Figure 24-2). This anticipates the same arrangement of cross-field ventilatory tubing, which ordinarily follows tracheal division intraoperatively.

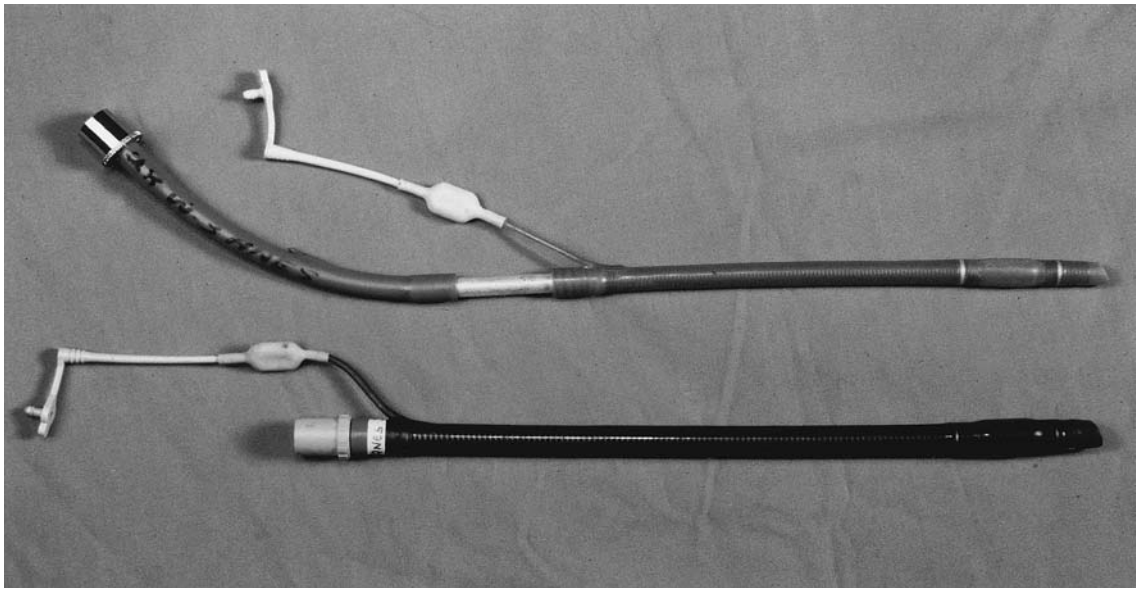


FIGURE **24-1** *Top of photograph is an extended endotracheal tube designed by Dr. Roger Wilson, which reaches from the mouth into the left main bronchus, if necessary. Its components are 1) a segment of Rusch tube for added length, 2) a metal sleeve connector, which hardly diminishes the lumen, and 3) a Tovell flexible armored tube, which has the advantages of a short balloon cuff. An extension is usually added to the sidearm for ease in cuff inflation and deflation. The tube is positioned with a flexible bronchoscope, sometimes aided intraoperatively by the surgeon. Other versions of an elongated tube are pictured in Figures 18-3 and 18-4 in Chapter 18, "Anesthesia for Tracheal Surgery." Pictured below is a flexible armored tube used for intubation of the distal trachea across the operative field.*

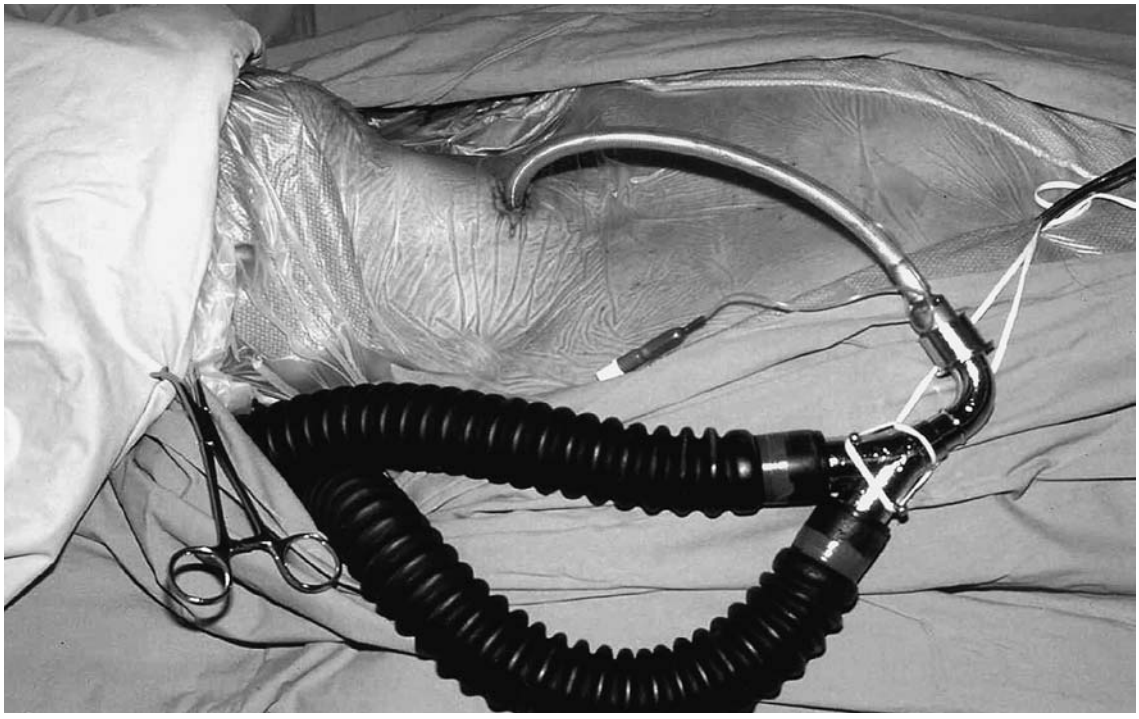


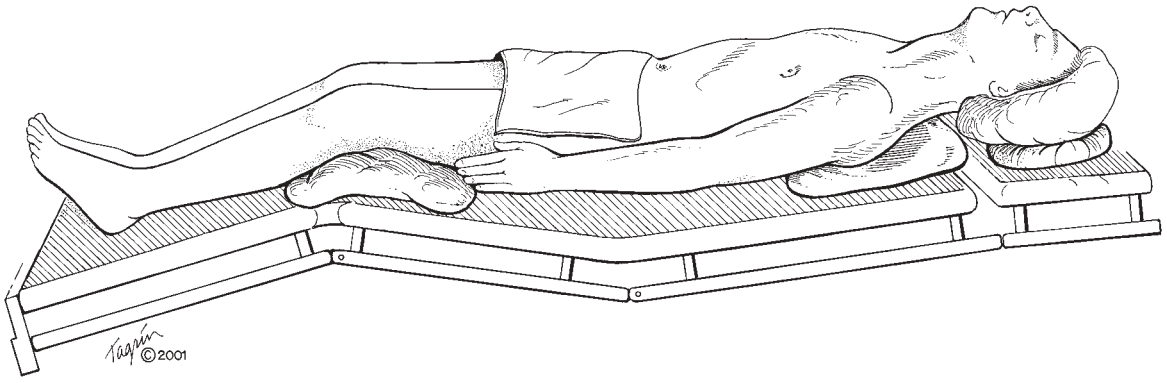
FIGURE **24-2** *Intubation for operation through existing stoma. Note the sterile anesthesia tubing. The field is prepared for upper tracheal resection. Access is available to the hyoid bone above, and for partial or complete sternotomy below, if any of these extensions are necessary.*

The anterior approach has limited physiologic impact on the patient, but pneumothorax occurs rarely intraoperatively. It must be considered if a patient abruptly develops unexplained physiologic problems during operation, such as hypotension, diminished compliance, or falling oxygen saturation.

Position

The majority of patients are positioned supine with an inflatable bag beneath the shoulders (Figures 24-3*A*,*B*). This permits extension of the neck in a controlled fashion, but allows the extension to be removed easily during tracheal anastomosis, when cervical flexion is desired. Extreme cervical extension is to be strictly avoided. The patient is usually positioned with slight flexion at the hips and at the knees so that positioning the neck and upper sternum appropriately for the surgeon, often with the sternum approximately horizontal, will not result in a reverse Trendelenburg position with blood pooling in the lower extremities. If access to both arms is desired for intravenous and arterial lines, then these may be abducted on arm boards at an approximately 30° angle. The arm board is placed carefully so that the surgeon may stand against the table above the arm without encountering the board itself (Figure 24-3*C*). With intravenous support poles placed at the ends of either one or both of the arm boards, as well as poles in the usual position at the head of the table, it is possible to drape the patient so that access may be had by the surgical team, above and below the arm board, but yet leave the arm accessible to the anesthesia team. Limitation of abduction to 30° also protects the brachial plexus.

In a patient with an upper- or midtracheal tumor of uncertain extent, or in a patient with an extensive stenosis that may have been complicated by prior unsuccessful surgical procedures, intrathoracic mobilization may be required. It is occasionally judicious to position such a patient as described with the addition of a roll support, placed longitudinally beneath the upper back to the right of the midline (Figures 24-4*A*,*B*). With the right shoulder partly abducted and the elbow partly flexed, the right chest is



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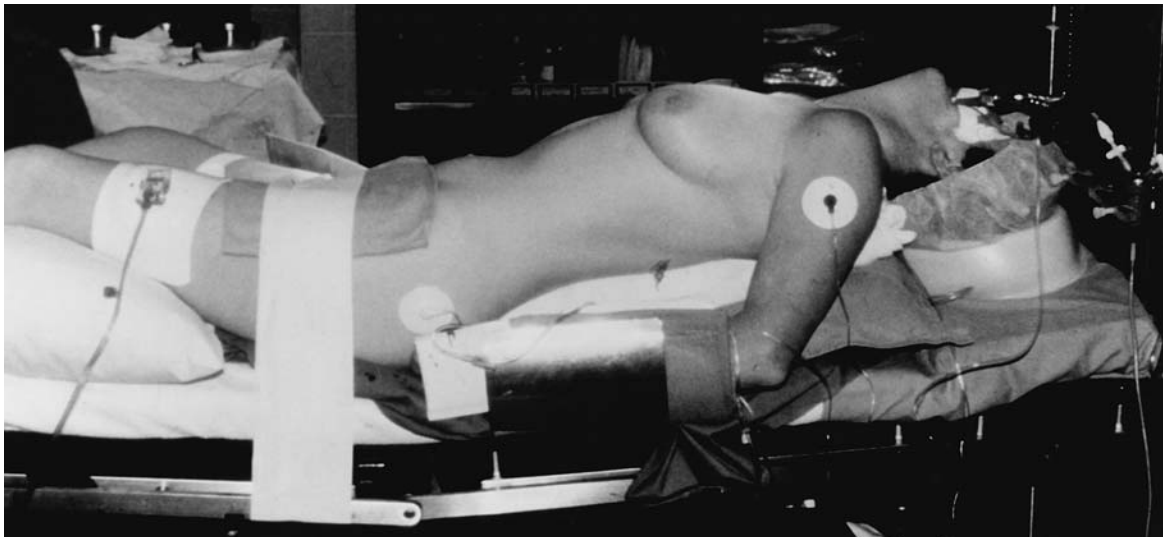

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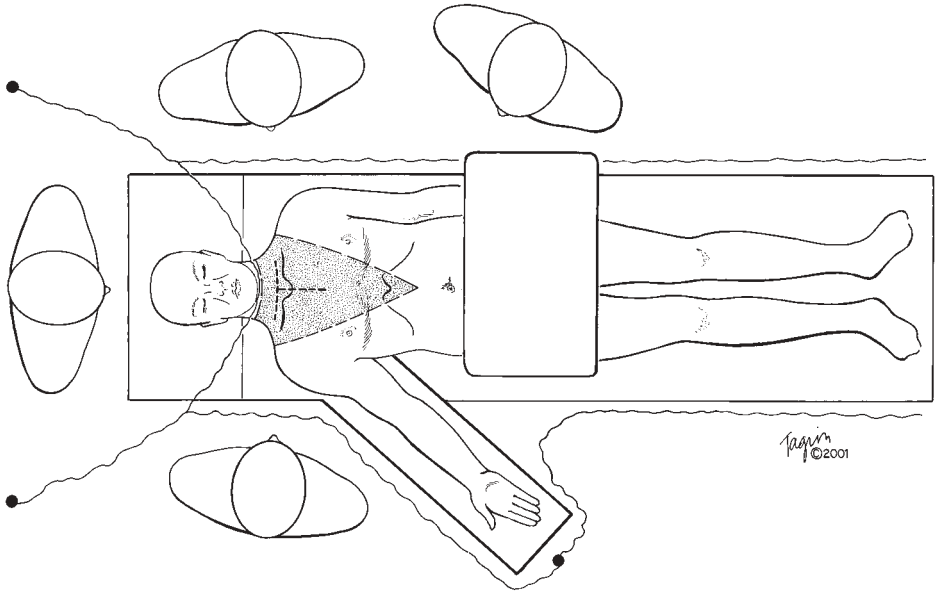


C

FIGURE **24-3** A, *Position of patient for anterior tracheal resection. Note the flexed table, inflated bag beneath upper shoulders, horizontal neck and sternum, extended neck, and head support. Details are in the text.* B, *Photograph of a patient in the position described in* A. C, *View of the operative arrangement from above. The* wavy lines *indicate the borders of sterile draping, supported by poles (*black dots*) at head of the table and for arm board access. The* stippled area *designates the usual operative field. The acute angle of the arm board allows easy access to the field by the surgeon.*

included in the operative field to the level of the posterior axillary line. A midline sternal incision may be extended laterally, if necessary, beneath the right breast, elevating the pectoral muscles and entering the right hemithorax through the fourth interspace (see Chapter 23, "Surgical Approaches"). The resultant "trapdoor" offers exposure to the entire trachea from cricoid to carina and even provides access to the posterior aspect of the carina. After positioning and draping the patient for this approach, the operating table may be side-tilted to level the sternum so that the initial part of the operation is done as if the patient were simply supine on the table (see Figure 24-4*B*). A midline posterior support provides a possibility of opening the left side as well (with suitable draping in advance) for left hilar mobilization, if that possibility is anticipated (Figure 24-4*C*).

Incision and Management of an Existing Stoma

The initial incision for the anterior approach is a low collar incision. This is usually relatively short since the lateral extent need only be sufficient to permit elevation of skin flaps to the level of the cricoid cartilage above and to the sternal notch below. If a previous cervical incision does not lie too high, it is reopened. As much as possible, unsightly scars from a prior surgery or tracheostomy are excised with the incision. Most often, an existing stoma is circumcised by the incision, which extends laterally on either side at the level of the stoma (Figure 24-5*A*). In a patient with a long neck, in whom the stoma is unusually high, the incision is best placed low in the neck and, if necessary, the stoma is circumcised as the flap is elevated past the level of the stoma (Figure 24-5*B*).

Rarely, in a patient with a short stenosis that is well below a high stoma, it is possible to expose the stenosis and perform the resection, leaving the stoma in situ for later spontaneous closure. If the stomal fixation in any way inhibits the operation, or if it pulls the attached skin too far into the upper mediastinum, it is preferable to separate the skin and platysma from the trachea and then deal with the stoma, when it is not excised in continuity with the principal lesion. Ideally, if the cutaneous epithelium has healed to the tracheal epithelium, as happens in some chronic stomas, then the stoma may be closed by the method described for closure of a long-persistent stoma (see Chapter 22, "Tracheostomy, Minitracheostomy, and Closure of Persistent Stoma"). In other patients, the stoma may be reestablished at a new level by making a small nick in the skin and suturing the skin to the trachea at this level, at the conclusion of the procedure. A preferred method, if cutaneous closure is inappropriate, is to cover the unresected stoma carefully with strap muscle. This is obligatory if the stoma lies below the sternal notch after the trachea is reconstructed. Should a granuloma later form at the site of the stoma, then it can easily be removed bronchoscopically, as required.

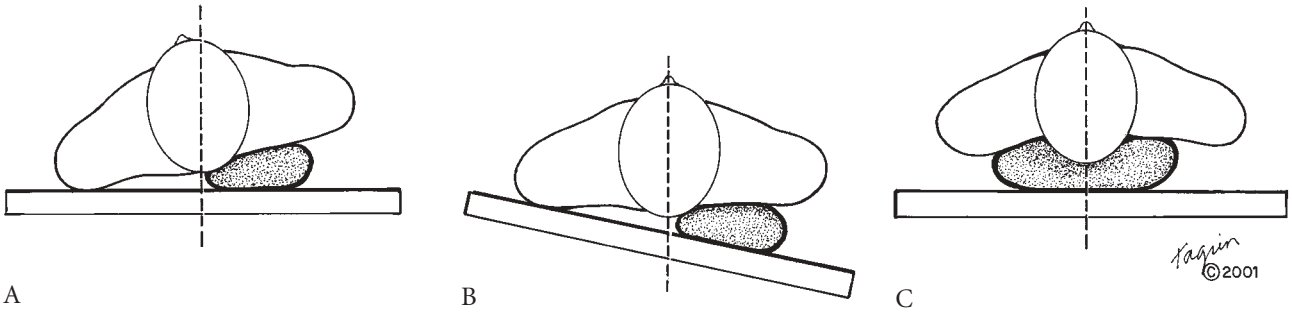


FIGURE **24-4** *Modifications of the patient's position to facilitate additional access to one hemithorax or both. View from the head of the table.* A, *Longitudinal roll to the right of midline with partial abduction of the arm allows access to the right hemithorax.* B, *Lateral tilt of the table levels the cervicomediastinal field for initial anterior access.* C, *Midline roll and appropriate arm positioning allows access to either or both hemithoraces, with side tilt of the table.*

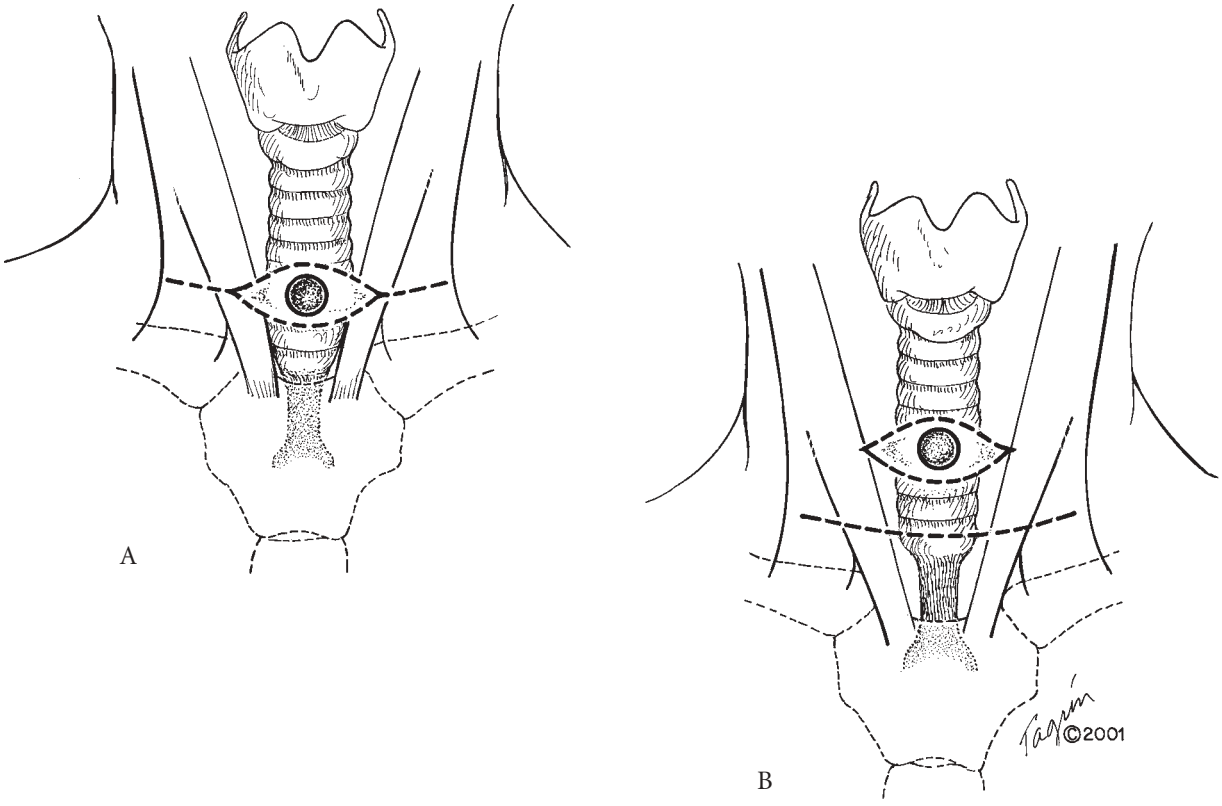


FIGURE **24-5** *Management of an existing tracheostomy.* A, *A low stoma is included in the collar incision.* B, *A high stoma is usually separately exposed by a short incision parallel to the low collar incision. If the collar incision was placed high in the neck and a vertical extension was later necessary for upper mediastinal exposure, then the resulting scar would likely form a cervical tether.*

Dissection

In many patients, dense scarring is found at the level of the stoma as well as at the site of postintubation stenosis. Scarring is heightened by prior surgical procedures. Normal subplatysmal anatomy is therefore identified first at the lateral ends of the transverse incision and dissection carried along the surface and border of the sternocleidomastoid muscles, both above and below on either side. The surgeon then works toward the midline and gradually elevates the skin and what is left of the platysma from the strap muscles and the midline points of adherence to the trachea. Initial dissection in almost every case is carried up to the level of the cricoid cartilage. A long-necked patient requires a longer horizontal incision so that the upper flap may be raised higher. I have avoided U-shaped incisions, which are less cosmetic and offer no better exposure. Inferiorly, the cutaneous and platysmal flaps are raised to the sternal notch. The anterior borders of the sternocleidomastoid muscles are followed to their attachments to the sternum. The anterior jugular veins are divided and raised with the flap superiorly. Inferiorly, they are usually left behind as the flap is elevated, or are redivided if the scarring is dense. The flaps are spread vertically with Gelpi retractors, exposing a field from cricoid cartilage to sternal notch (Figure 24-6).

The midline is identified above and below an existing stoma or a point of dense adherence to a prior stoma. The medial margins of the sternohyoid muscles are identified and elevated laterally for a short distance, followed by the sternothyroid muscles. The pad of fat, and often scar, which lies suprasternally is divided by dissecting beneath it, just above the sternal notch between the heads of the sternocleidomastoid

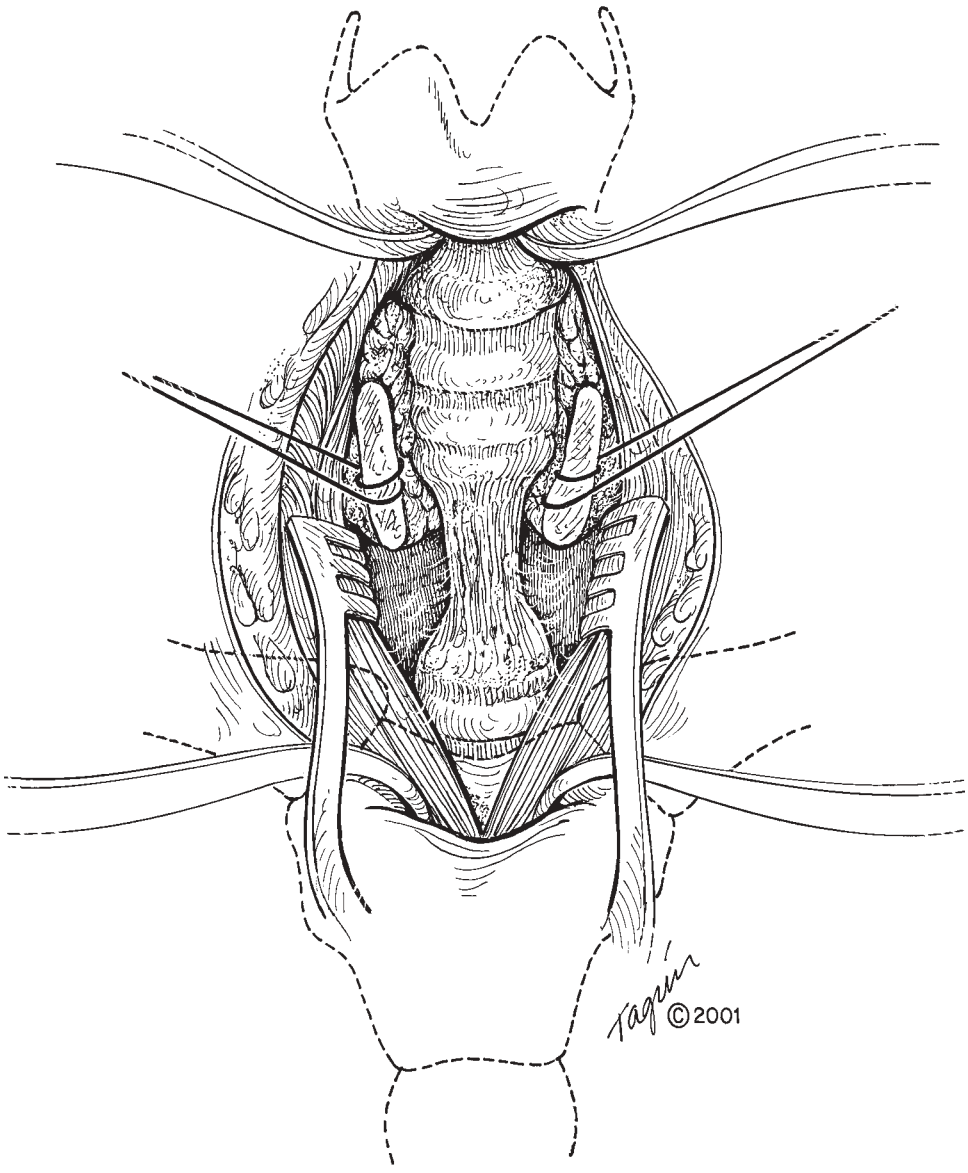


FIGURE **24-6** *Exposure of the cervical tracheal stenosis after spreading the collar incision vertically. Strap muscles are reflected laterally and held by a self-retaining retractor. The thyroid isthmus or its remnants are divided, suture-ligated, and retracted with traction sutures. Only thyroid adherent to anterolateral trachea is dissected at this point. The anterior surface of the trachea is dissected sharply from the cricoid to the sternal notch, and bluntly to the carina inferiorly. At this stage, only the anterior surface of the stenosis has been dissected.*

muscles and superficial to the level of the strap muscles. This bundle of tissue may be divided between the clamps rather than by tedious dissection. Adherent strap muscles are dissected sharply from a stomal site. When a stoma is present, it is helpful to place hemostats for traction on either side of the ellipse of skin that has been left around the stoma. The anterior surface of the trachea must now be dissected. Above, the cricoid cartilage is usually identified in the midline and dissection carried onto the anterior tracheal wall immediately below it. It is usually possible to dissect beneath the remnants of the thyroid isthmus at the stomal area. If still intact, then the isthmus is sharply divided and suture-ligated on either side. The thyroid is dissected away from the tracheal wall on either side, but at this stage, only over the anterior portion of the tracheal surface. In order to reflect the isthmus completely, the pyramidal lobe is often divided just above the isthmus. Inferior to the point of maximal adherence, the anterior surface of the trachea is discovered. This is sometimes difficult, particularly where the trachea has been previously exposed surgically well into the mediastinum.

Decision is next made on whether or not an upper sternal division will be needed to enlarge surgical access. Even if a stenosis can be dissected without dividing the upper sternum, greater access will be needed for the anastomosis. Reconstruction will be completed with the neck in a slightly flexed position, with the chin brought toward the sternum. If it appears that the sternum will be better divided, then this should be done promptly in order to facilitate dissection. A vertical cutaneous incision is made from the midpoint of the collar incision down over the midline of the sternum to a point 2 cm below the sternal angle (Figure 24-7*A*). The substernal plane is bluntly dissected and the sternum divided vertically through the sternal angle. A small, pediatric-type chest retractor serves to spread the upper sternum several centimeters. One side or other of the divided portion of the sternum usually fractures at its lower end. No advantage is gained by deliberately dividing across one of the limbs of the partially divided upper sternum. Since all that is needed is access to the trachea behind the great vessels, complete sternotomy only extends the operative field without contributing to required exposure. Surface projection of the carina lies at the level of the sternal angle, but the carina rests posteriorly nearly against the vertebral bodies. After partial sternotomy has been added, a single Gelpi retractor is placed with one point against either clavicular head and the other at the midpoint of the upper incisional flap (Figure 24-7*B*). Since a significant number of patients with tracheal stenosis acquired their lesions during treatment for complications of cardiac surgery, the hazards of reoperative sternotomy must be remembered. Careful technique, plus use of a Lebsche sternal knife and mallet instead of mechanical saws, will minimize these problems. Once in my experience, a cemented brachiocephalic vein was entered in such a case. Bleeding was controlled with Fogarty catheters while the vein was dissected free for definitive control.

The anterior tracheal surface is dissected completely from the cricoid cartilage to the carina, in most cases. The more normal segments of trachea above and below the stenosis and stoma are dissected first, often bluntly. Freeing the pretracheal plane will allow the trachea to slide more easily for anastomotic approximation. In contrast, a later freeing of the membranous wall of trachea from the esophagus will not greatly increase mobility, and hence it is done only to the limited extent needed to permit anastomosis. Due to the dense adhesions and unpredictable deformities seen with postintubation stenosis, dissection (especially after prior failed surgery) is guided by a combination of direct vision, palpation, and judgment from experience about where scar ends and the trachea or its stenotic remnants begin. Dissection proceeds from more normal trachea to scarred trachea. In the area of stenosis, dissection is kept very close to the scarred portion of the trachea and elsewhere against the trachea itself, in order not to damage recurrent laryngeal nerves. A stenosis may be indented in hourglass fashion. There may be no rings left to act as guides, and remnants of cartilages may be disordered. Traction sutures in the divided thyroid isthmus are useful as the gland is dissected from the lateral tracheal wall. Dissection of the normal distal trachea is done only on the anterior surface, once past the level of the lesion. Distal pretracheal dissection is done bluntly, as in mediastinoscopy, if possible.

Great care must be taken not to injure the lateral blood supply of the portion of trachea that is to remain after resection of the lesion. Circumferential dissection of the trachea should be made *only* at the level of the lesion that is to be excised, and for no more than 1 to 2 cm above and below that level. Injury to blood supply may result in a later tracheal necrosis and severe restenosis, which may no longer be reconstructible. If a stenosis lies in the upper trachea, initial dissection is usually made circumferentially, immediately distal to the area of stenosis. A figure of 8 suture may be placed in the stenotic segment to facilitate retraction of the trachea, as dissection proceeds laterally and posteriorly on the trachea. Since the tissue below the stenosis is relatively normal, it is more easily dissected than that adjacent to the stenosis itself, particularly posteriorly. Circumferential dissection is done just below the lower border of the lesion. If the membranous wall is damaged during dissection, the perforation will be adjacent to the pathology and may be resected with the specimen. A lower or longer injury must be repaired. With delicacy and care, this will seldom occur.

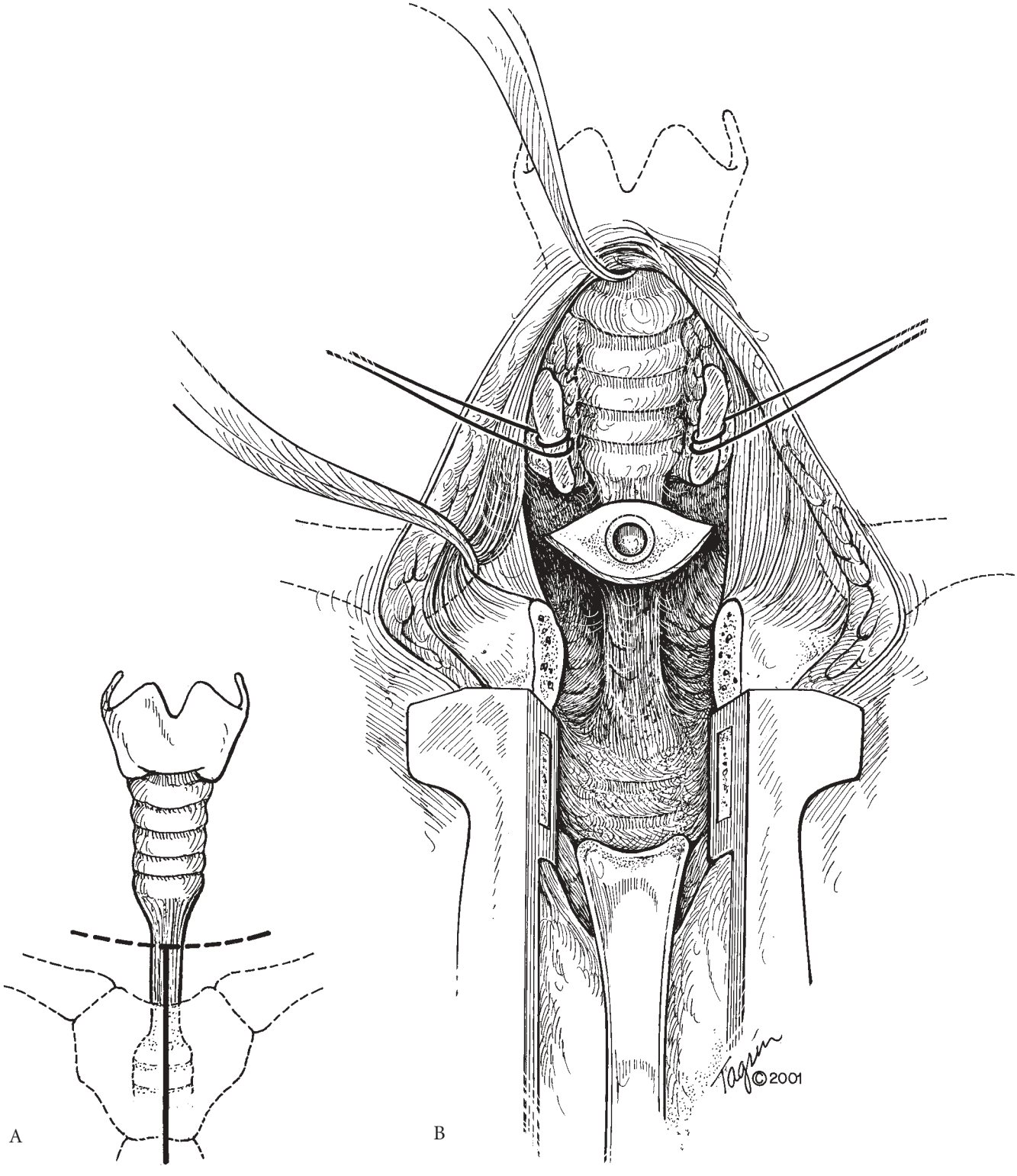


FIGURE **24-7** *Cervicomediastinal exposure of a longer stenosis.* A, *The dashed line indicates the exploratory cervical incision. The vertical extension carried just past the sternal angle (*solid line*) provides the access to the mediastinum needed in this patient.* B, *A single Gelpi retractor exposes the cervical field. A pediatric chest wall retractor or a Tuffier retractor holds the sternal edges apart. In this patient, a stoma was present in the upper stenosis. Hemostats on the tips of the cutaneous oval around the stoma provide excellent traction later to rotate the trachea for dissection. The inferior Richardson retractor holds the undissected upper mediastinal vessels back to simplify lower pretracheal dissection. The distal trachea has not yet been exposed.*

Esophageal injury is even less likely. If dissection proceeds easily around a circumferential stenosis itself, much or all of it may be completed before dividing the trachea. Otherwise, I prefer to divide the trachea just below the lesion, and elevate the specimen to facilitate completion of dissection (Figure 24-8*A*). In a supracarinal lesion, circumferential dissection is better done first just above the stenosis, and the trachea is divided proximal to the lesion (Figure 24-8*B*). Cross-field intubation in this case is done through the lesion, providing a good handle to facilitate distal dissection.

It is critically important to dissect close to the tracheal wall and to the lesion in cases of stenosis in order to avoid injury to the recurrent laryngeal nerves. Particularly in the upper trachea, these nerves lie in the tracheoesophageal groove. Dense peritracheal fibrosis may make it hazardous to try to identify the nerves or to isolate them in the vicinity of the lesion. To minimize the incidence of nerve injury, it is far better to dissect meticulously and patiently against the trachea without trying to visualize the nerves. The nerves remain in the scarred lateral tissues, which fall to either side. Elevation of the specimen with traction further protects the nerves during dissection, especially as the critical zone near the cricoid cartilage is approached.

Two major hazards of this dissection—devascularization of the trachea and injury to recurrent laryngeal nerves—have been emphasized. A third potential major hazard is injury to the brachiocephalic artery, which may result in postoperative hemorrhage. In many patients with a properly placed tracheostomy, the cuff will lie at the level of the innominate artery. Dense peritracheal fibrosis may form, where a subsequent stenosis lies directly behind the artery. In addition, if tracheal reconstruction has been attempted previously, the artery may have been dissected free, and may therefore be adherent to the trachea. In a number of earlier reports of upper tracheal resections, postoperative hemorrhage from the brachiocephalic artery occurred much too frequently. This complication is usually avoidable. The key principle is to dissect "hard against" the trachea, keeping the dissection on the tracheal surface or against the stenosis. No attempt should be made to dissect out the artery itself or to place loops around it for retraction. Dissection in this manner leaves a protective barrier of the artery's normal investments of connective tissue as well as scar tissue. If this technique is followed, then the artery is not likely to leak postoperatively, even when it lies against the anastomosis. In case of justifiable concern that the artery may be at risk, usually because of prior surgery, a strap muscle is pedicled and sutured between the artery and tracheal anastomosis to provide a buttress of healthy tissue. Less often, a lobe of thymus may be interposed. If the artery is fused to the trachea (due to prior surgery), proximal and distal control must be obtained before freeing it. In such cases, tissue interposition becomes essential.

Resection

A Penrose drain is passed around the trachea where circumferential dissection has been done just below the lower end of the stenosis. The anesthetist is asked to deflate the cuff on the endotracheal tube, and two sutures of 2-0 Vicryl are placed vertically in the *midlateral line* of the distal trachea on either side, through the full thickness of the tracheal wall, usually encircling one ring (Figure 24-9). These are placed at a point that is estimated to lie one or two rings distal to what will be the line of tracheal division. This is not critical since the traction sutures may be easily replaced after tracheal division. Indeed, it is sometimes convenient to place the traction sutures after tracheal transection via the open lumen. In many cases, the border of the lesion is clearly visible from outside of the trachea. A tentative transverse incision is next made in the trachea, at the lower end of identifiable pathology. One can always remove more trachea, but one cannot replace it. If there is more disease distally, then successive partial transverse incisions are made until a cartilaginous ring of acceptable quality is encountered. In many cases, some inflammation continues distally and, more frequently, is noted proximal to the stenotic lesion. Indeed, sometimes, no absolutely normal trachea or larynx will be found proximally. All severe diseases must be removed to avoid restenosis, but unneeded resection increases the risk of complications from anastomotic tension. Some fibrosis or inflammation can be accepted if healthy cartilage is visible on transection.

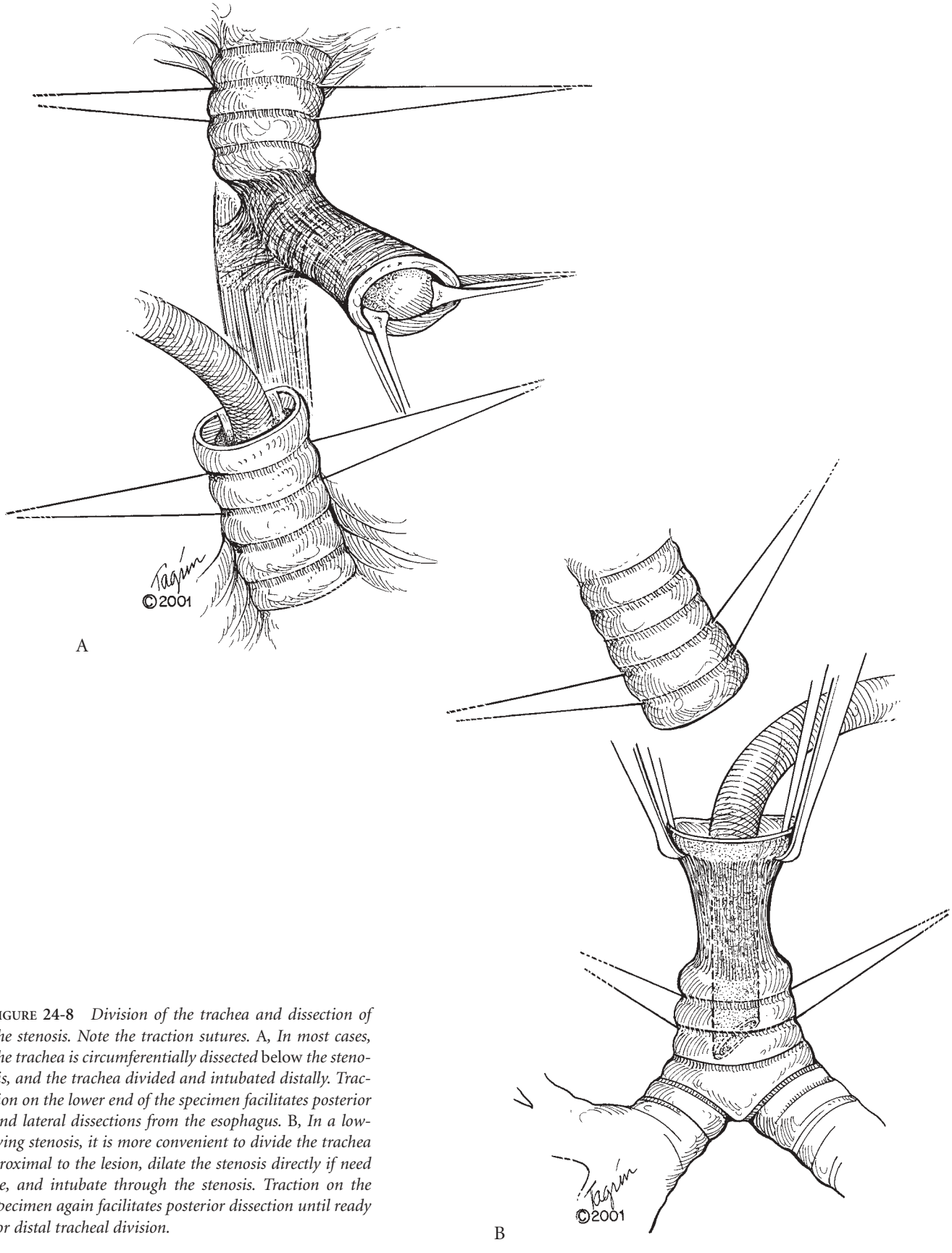
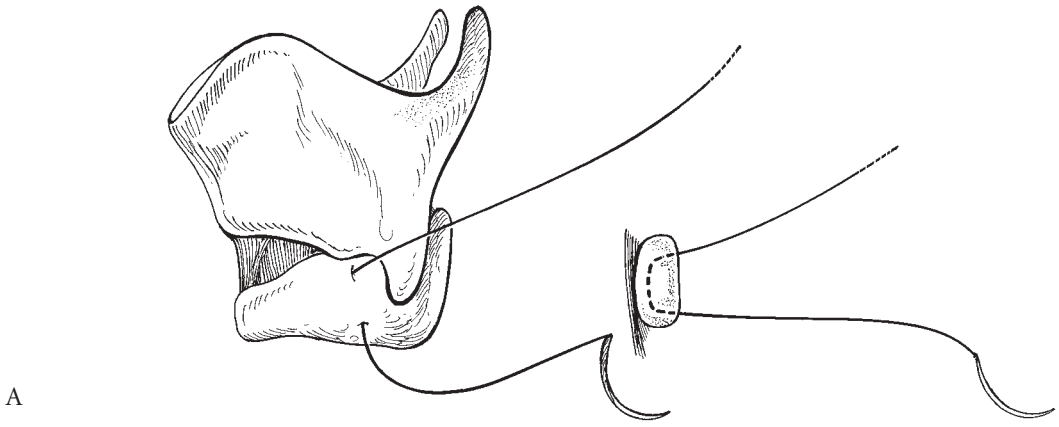


FIGURE **24-8** *Division of the trachea and dissection of the stenosis. Note the traction sutures.* A, *In most cases, the trachea is circumferentially dissected* below *the stenosis, and the trachea divided and intubated distally. Traction on the lower end of the specimen facilitates posterior and lateral dissections from the esophagus.* B, *In a low-lying stenosis, it is more convenient to divide the trachea proximal to the lesion, dilate the stenosis directly if need be, and intubate through the stenosis. Traction on the specimen again facilitates posterior dissection until ready for distal tracheal division.*



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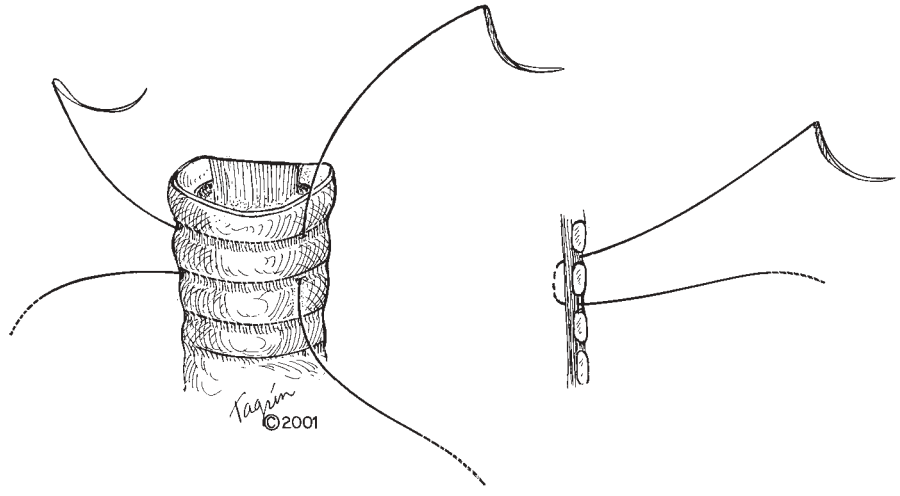


B

FIGURE **24-9** *Placement of traction sutures.* A, *When transection is just below the cricoid cartilage, the proximal traction sutures are placed in the midlateral line in the lateral cricoid laminae. The suture grasps a sturdy amount of cartilage but does not enter the lumen of the larynx (see detail).* B, *In the trachea, distal or proximal, the midlateral sutures pass around a cartilage, about one ring below the point of transection, and pass in and out of the tracheal lumen, as shown in detail.*

In patients who particularly have stenosis related to the cuff of an endotracheal tube and who have not had a tracheal stoma, the external surface of the trachea may appear to be relatively normal, except for mild peritracheal fibrosis. The exact level of stenosis must be precisely localized intraoperatively, and not from earlier bronchoscopic measurement alone or from radiographs. A flexible bronchoscope is passed intraoperatively through the endotracheal tube, and the tube is withdrawn slowly under direct vision to a subglottic position. The bronchoscopist identifies the precise level of the lower end of the stenosis. With the operating field lights deflected, the area of tracheal transillumination is clearly seen. A no. 25 hypodermic needle is passed through the tracheal midline from the operative field, at what appears to be the distal margin of the stenosis. The bronchoscopist checks the position of the needle until it is precisely located (Figure 24-10). The outer tracheal wall is marked with a suture at this exact level. The upper end of the stricture may be similarly identified, although it will be easier to identify intraluminally once the trachea has been divided distally and the specimen elevated.

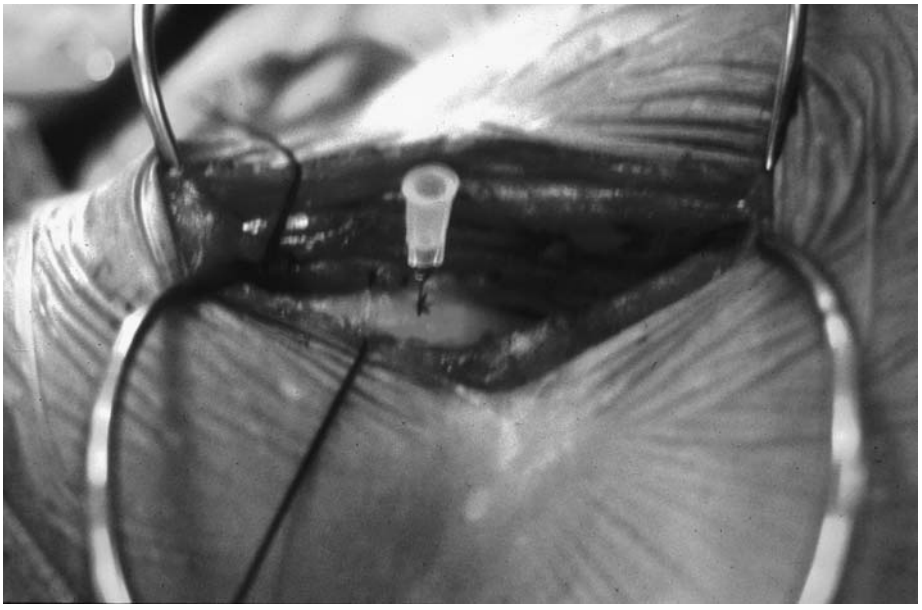


FIGURE **24-10** *Flexible bronchoscopic transillumination of the trachea to identify the borders of a stenosis not clearly identifiable externally. A fine needle is inserted and placed precisely at a margin of the lesion under visual bronchoscopic guidance, and the point marked by suture.*

A flexible endotracheal tube with its connectors and sterile anesthesia tubing is clipped into place at the level of the incision, and the proximal anesthesia tubes are passed through the drapes to the anesthetist. Our staff dubbed these "elephant trunks." The peroral endotracheal tube is partly withdrawn and taped. The trachea is divided transversely. Care is taken to make this a clean cut, preferably but not necessarily, between cartilages. The distal trachea is intubated across the operative field, inflating the cuff just enough to obtain a seal. The traction sutures may be elevated and crossed over the tube where it lies, and fixed to the drapes or held by a second assistant. This keeps the tube in place without the need for tying or suturing it in place. The endotracheal tube and its attached "trunks" may be flipped up or down to improve access at different stages of operation, or the tube may be removed intermittently during anastomosis. Perelman suggested placing the anesthesia tube through a temporary and more distal vertical tracheostomy,[1] but I have usually avoided this. Even when the cuff of the tube is inflated, the operative field and the distal trachea are suctioned frequently to minimize seepage of blood into the tracheobronchial tree. This is especially important in patients with marginal pulmonary function.

The specimen is grasped with Allis forceps on either side of its distal margin and elevated upward or to the side, as necessary (see Figure 24-8). The paired Allis forceps expose the plane of dissection more clearly. Dissection is completed with this exposure in areas of difficult scarring, until the proximal end of the area of stenosis is reached. Great care must be exercised proximally if the cricoid cartilage is approached. The recurrent laryngeal nerves enter the larynx, medial to the inferior cornua of the thyroid gland, against the broadened portion of the reverse signet ring of the posterolateral cricoid cartilage. Dissection is carried up to the palpable inferior margin of the cricoid cartilage where stenosis extends that high. As one elevates the specimen with traction, the esophagus may be dissected away from the trachea without injury. Care must be taken not to dissect posteriorly superior to the inferior margin of the posterior cricoid plate, since this is the point where the larynx and esophagus are attached, and also where the recurrent nerves enter the larynx posterolaterally. It is also important to have the anesthetist withdraw the endotracheal tube in high lesions so that one does not palpate the tip of the tube and mistake this for the cricoid plate. Indeed, for higher lesions, the bulk of the endotracheal tube may be very much in the way of the operator. I, therefore, usually suture a catheter into the leading tip of the endotracheal tube and ask the anesthetist to withdraw the

endotracheal tube from the larynx altogether (Figure 24-11). The catheter later serves as a guide to bring the endotracheal tube back down through the larynx, just prior to completion of the anastomosis. The narrow diameter of the catheter does not interfere with visualization of the interior of the airway.

When the proximal end of a tracheal stenosis has been reached, dissection is carried a little further in order to make place for lateral traction sutures and to prepare a margin for suturing. Obviously, if this is the cricoid cartilage itself, then minimal further dissection is performed. Dissection is often required for a short distance laterally between cricothyroid muscles and the upper pole of the thyroid gland. More posterior dissection is not useful and can endanger the recurrent laryngeal nerves. In this case, the midlateral traction sutures (2-0 Vicryl) are placed in the larynx itself, obtaining a good firm bite in the lateral cricoid cartilage laminae, since purely muscular sutures will pull out. Sutures thus placed in the larynx do not enter the lumen (see Figure. 24-9*A*).

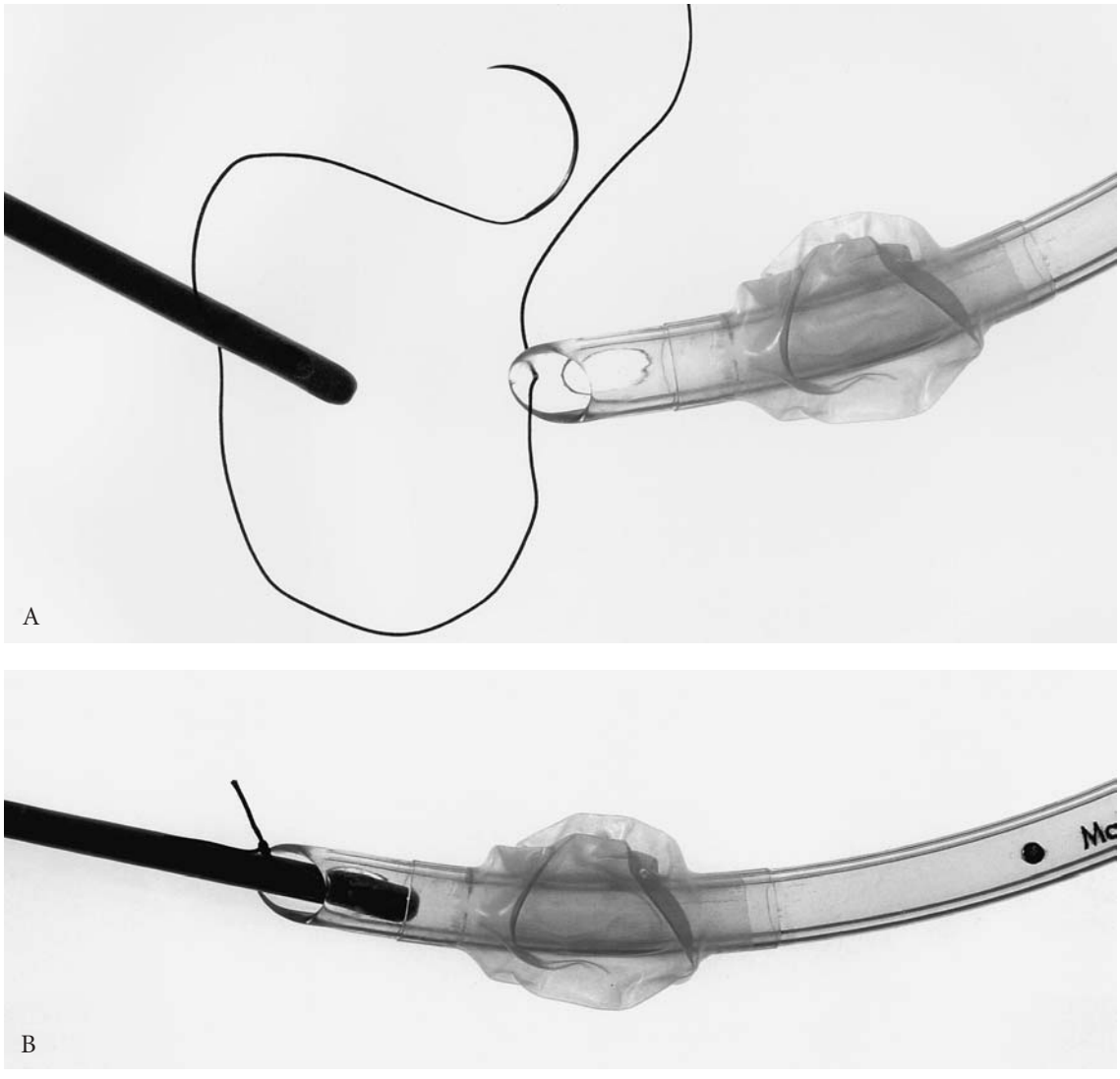


FIGURE **24-11** *Use of a catheter as a "leader," which will be used to draw the endotracheal tube back through the larynx.* A, *Heavy suture passed through the tip of the endotracheal (ET) tube and through the catheter, 1 to 2 cm from its tip.* B, *The suture fixes the "leader" to the leading tip of the ET tube so that it will not be caught on the vocal cord as it is pulled distally through the larynx.*

If the stenosis is low in the trachea, it is more convenient initially to dissect circumferentially around the trachea, *proximal* to the lesion. This permits division of the trachea, where it is easily accessible. The endotracheal tube across the operative field passes through the stenosis into the distal trachea (see Figure 24-8*B*). The stenosis is dilated, if necessary. Uterine or common duct dilators serve well. Allis forceps placed on either side provide good traction to complete the distal dissection. With the brachiocephalic vessels gently retracted forward, a benign stenosis located even at the supracarinal level may be resected by this approach. With very distal stenoses, lateral traction sutures may be placed with minimal difficulty at the tracheobronchial angles and, later, the anastomotic sutures. In such cases, intubation is usually made into the longer left main bronchus after the specimen is removed, ventilating one lung only. High frequency ventilation may be used, either with a single or bifid catheter. This approach is worthwhile, in order to avoid the discomfort of a thoracotomy, and it is essential in patients with a low tracheal stenosis, who suffer from severe chronic obstructive pulmonary disease and who could not safely undergo thoracotomy. A complete sternotomy does not provide better exposure than a partial one, since dissection lies behind the great vessels. A transpericardial approach is not necessary here.

With careful dissection, it is extraordinarily rare to enter the esophagus. However, in patients with circumferential cuff lesions, the stenosis may be densely adherent to the esophagus and dissection must be precise. If there was any likelihood of injury, then methylene blue in a large volume of saline should be instilled into the esophagus through a high nasogastric tube for direct inspection. An esophageal injury is meticulously closed in two layers (see Chapter 26, "Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula") with an overlying buttress of pedicled strap muscle.

Two Concurrent Tracheal Stenoses

A few times, I have encountered a patient operated upon for a cuff stenosis, in whom a stomal stenosis of significance later develops. If the length of normal trachea permits, resection of the subsequent stricture is to be done. Even more rarely, a patient may present with concurrent clinically important stenoses at stomal and cuff sites, with a segment of normal trachea between the lesions. Dual resections have been reported but I believe it safer to stage the procedures. The distal (cuff) stenosis is resected and a short T tube is placed in the proximal (stomal) stenosis, with the stoma located in the damaged segment (Figure 24-12). The T tube stops short of the fresh anastomosis to avoid irritating it. In dissecting the stenoses, particular care is given to avoiding injury to the blood supply of the short intervening segment. The second resection is delayed for 4 months.

Neoplasms

Dissection for *tumors* follows a slightly different course. Dissection is begun at a distance away from the tumor, in order not to break into it. The recurrent laryngeal nerves, if they are likely to be involved or are involved, are identified (as in surgery for thyroid neoplasm) at a distance from the tumor and followed up to the area of the tumor. If one of the nerves is clearly involved by tumor, or if the vocal cord is already paralyzed by tumor, it will have to be sacrificed. Dissection is done with great delicacy to avoid bruising injury to a nerve, even if it is not divided. Extensive lymph node dissection is generally not possible, except in the area adjacent to the tumor, since excessive dissection of the paratracheal nodes may injure the blood supply of the trachea. If tracheal tumor invades the thyroid gland, the lobe or a portion of it may be resected en bloc with the trachea. If the tumor involves the anterior esophageal wall, it may be necessary to remove a portion of the muscular wall or, less commonly, a full thickness portion of the wall. In the case of muscular removal, muscular edges are reapproximated with fine interrupted 4-0 silk or Vicryl sutures. Following full thickness removal, the esophagus is closed in two layers, using Sweet's technique of inverting the esophageal mucosa with 4-0 sutures, followed by Lembert sutures of the muscularis.[2] A flap of strap muscle should be sutured over the closure so that muscle will be interposed between the esophageal suture

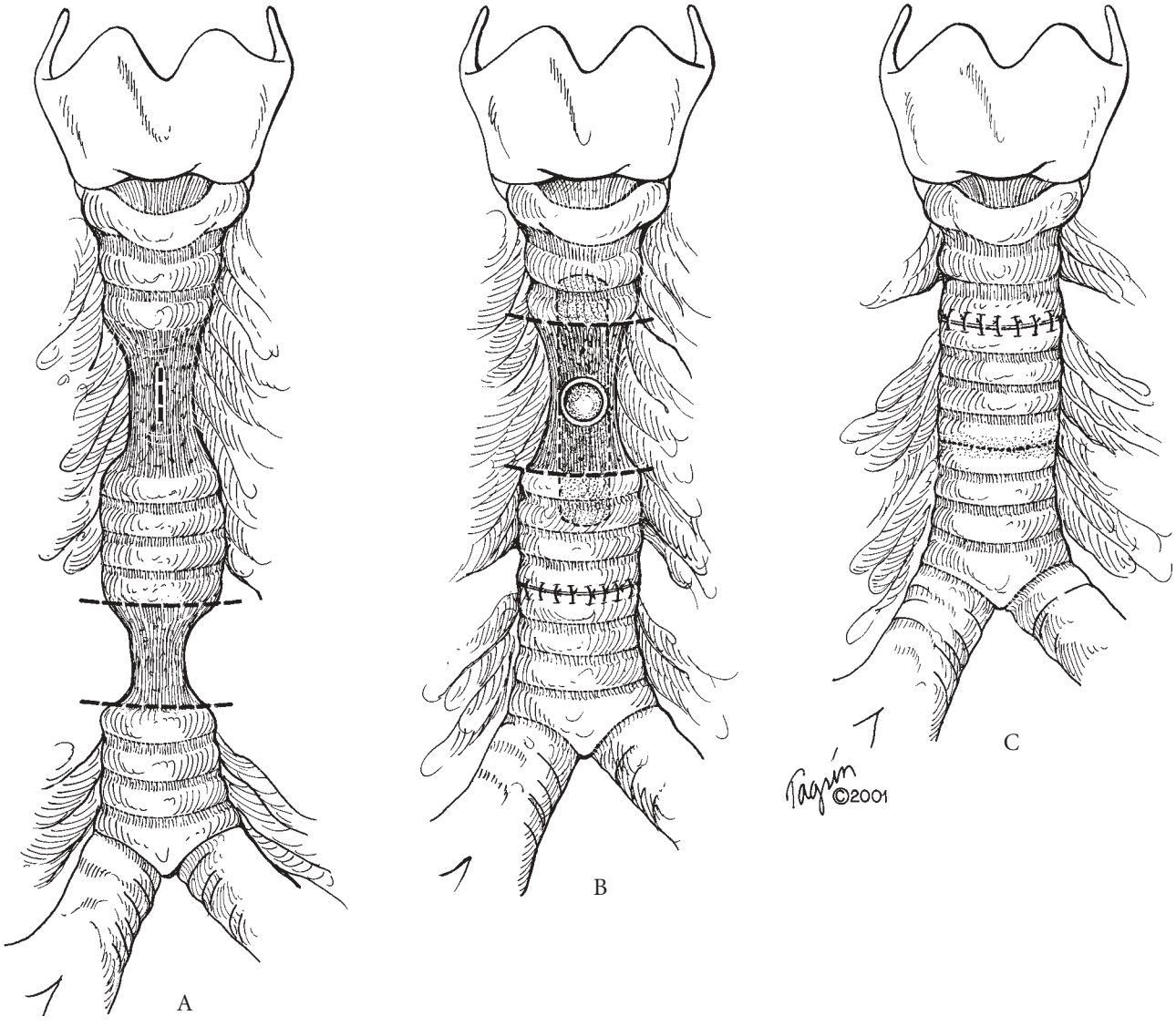


FIGURE **24-12** *Management of two separate tracheal stenoses. The proximal lesion is usually due to a stoma and the distal lesion to a cuff.* A, *The distal stenosis is resected and tracheostomy placed in the proximal lesion. Great care is taken to preserve blood supply, especially of the intervening segment.* B, *A short T tube provides an airway during healing of the distal anastomosis.* C, *Final result. The previous anastomosis is indicated by a* dotted line.

line and the tracheal suture line to minimize the possibility of a fistula. An esophagus so narrowed usually does not require subsequent dilations.

Frozen sections from margins of resection are essential in all malignant or indeterminate neoplasms and also in case of close resection of a benign neoplasm. Specific tumors present special problems. Thyroid carcinoma, which involves the lower portion of the larynx as well as upper trachea, is dealt with subsequently (see Chapter 25, "Laryngotracheal Reconstruction"). Since adenoid cystic carcinoma often extends for long distances beyond visible pathology, the approach for resection and for appropriate release maneuvers must be carefully planned. It may become necessary to accept microscopic tumor at the margins of resection, in order not to compromise the possibility of healing by excessive anastomotic tension following an extended resection.

Reconstruction

After resection of the specimen, and after examination of the proximal and distal ends of the remaining trachea to be certain that the tissue is of good enough quality to promise healing without stenosis, the ease of approximation is determined. The anesthetist or an assistant is asked to put the patient's neck in flexion with a hand beneath the occiput. The chin must approach the upper sternum. This should not be done by raising the headpiece of the operating table, as this tends to thrust the chin forward rather than to provide flexion of the neck. The surgeon and the surgical assistant grasp the upper and lower tracheal traction sutures on their respective sides and draw these together without excessive tension (Figure 24-13). The anesthesia tube on the field is usually removed transiently to clarify this observation. Since the patient is fully oxygenated, there is no urgency for continuous ventilation. Initially, I measured the tension required for approximation in the operating room, using spring tensiometers. This was mechanically difficult and proved to be unnecessary. It becomes clear to the experienced surgeon's judgment as to whether the tension required for approximation is appropriate. Excessive tension must be avoided. Initial assessment of the lesion and its length by radiology, by bronchoscopy, and finally (in a very few) by external exploration should eliminate most patients in whom tension would be excessive. In borderline cases, however, adjunctive measures such as laryngeal release are available. These methods are described later.

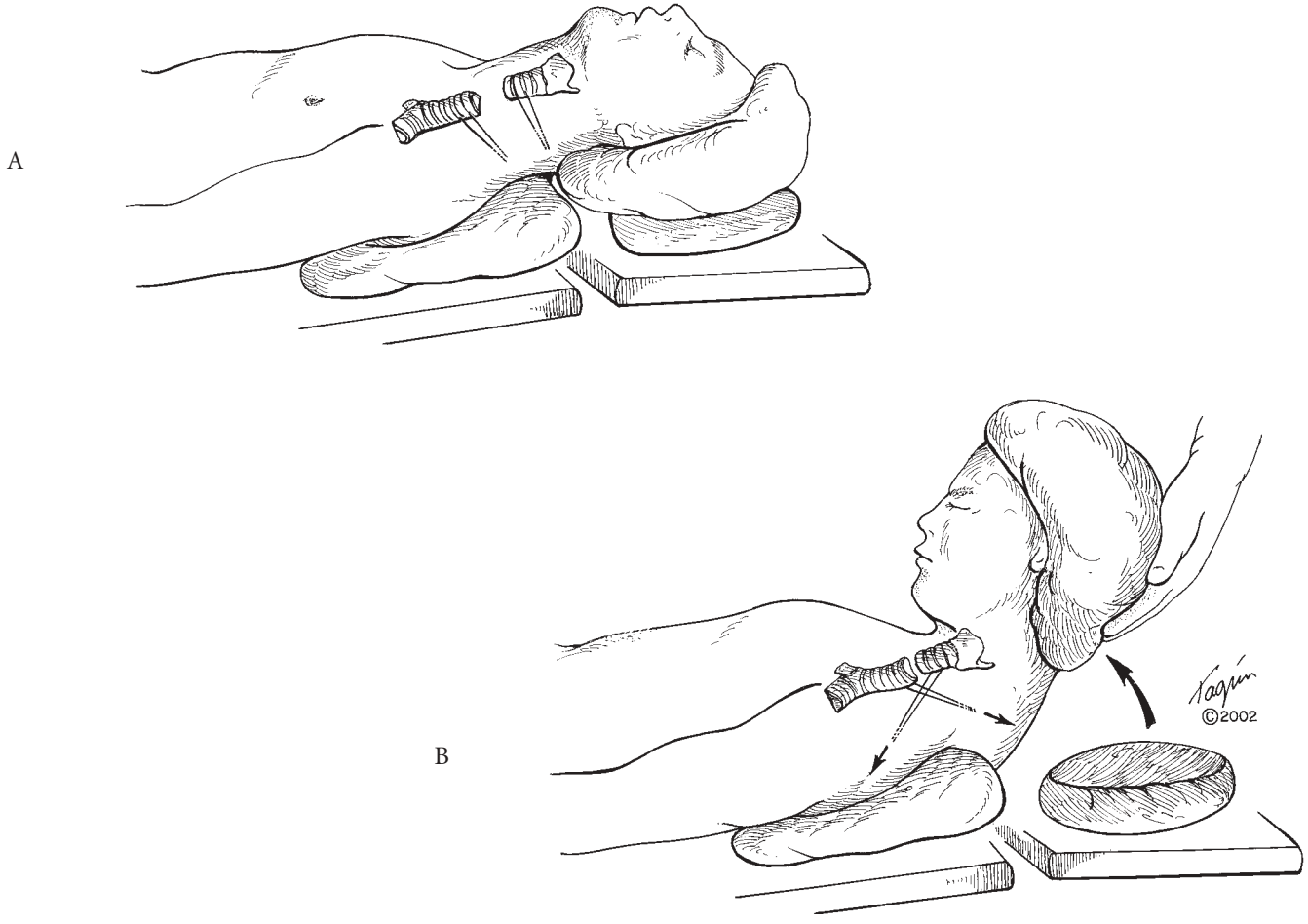


FIGURE **24-13** A, *Testing for ease of tracheal approximation after resection.* B, *The patient's head is tentatively elevated so that the chin approaches the chest wall. Forceful or extreme flexion is to be avoided. The surgeon and an assistant pull the tracheal ends together using the lateral traction sutures. Approximation must be feasible without excessive tension.*

After demonstration that the ends do approximate without excessive tension, the patient's neck is again extended and anastomotic sutures are placed. After trials with various suture materials (including Mersilene, Tevdek, Prolene, and polydioxanone suture [PDS]), I settled on 4-0, absorbable but long lasting synthetic polyglycolic acid sutures (Vicryl, coated). 5-0 Vicryl is used in infants and small children. This material has provided the necessary strength, flexibility, ease of use, and duration, essentially without complications. Use of Vicryl has almost wholly eliminated suture line granulomas in tracheal anastomosis. Monofilament PDS has proven more difficult to use in comparison, and it has no practical advantages over Vicryl. A lesser degree of early histologic reaction to PDS, over that seen with Vicryl,[3] seems to have no real importance with respect to final results, as we have demonstrated.[4]

Anastomosis is commenced with an initial suture posteriorly in the midline of the membranous wall, which passes from outside into the lumen, in either the upper or the lower segment of the trachea, and then

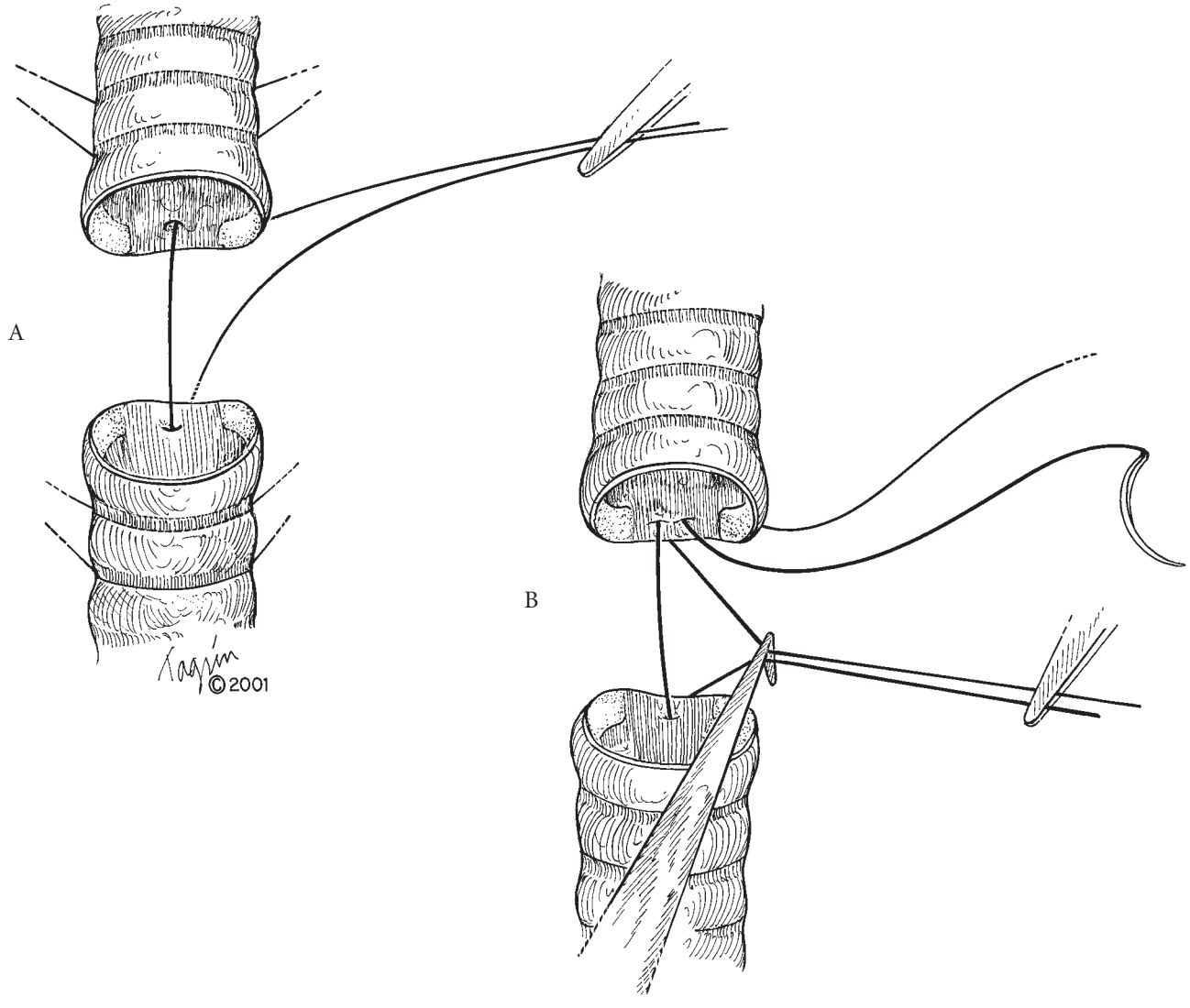


FIGURE **24-14** *Commencement of tracheal anastomosis.* A, *Lateral stay sutures are in place. The initial anastomotic suture is located in the posterior midline so that the knot will be extraluminal. A hemostat holds the suture and this in turn is clipped to the drapes laterally.* B, *The next suture is placed 4 mm lateral to the first. The previous suture is pulled out of the way with a Blalock hook so that the subsequent suture will be anterior to the first. Each is clipped to the drapes caudad to the previous suture.*

from inside to outside in the opposite segment (Figure 24-14*A*). The suture is clipped with a hemostat and this in turn is clipped to the drapes with a curved hemostat at the cephalad end of the field, but no higher than the level of the base of the patient's neck. Sutures clipped to head drapes will fall against one another and possibly become snarled when the neck is later placed in flexion. Each successive suture is placed anterior to the previous one in the tracheal wall, progressing laterally (Figure 24-14*B*). Since the sutures will be tied in the reverse order from which they are placed, it is important that these be laid down precisely. In this way, they will not cross one another as they are successively tied (Figure 24-14*C*). Each is clipped to the drapes progressively from cephalad to caudad, with the most posterior suture being most cephalad. Placement of these sutures is facilitated if the surgical assistant uses a Blalock hook to pull the previous sutures out of the way as each succeeding suture is passed through the tracheal wall (see Figure 24-14*B*). Sutures are placed approximately 4 mm apart and 3 to 4 mm distant from the cut edge of the trachea. No particular effort is made to pass around the rings; indeed, this should be avoided in small children, since the rings may be flexible enough so that they can be folded together and the anastomosis narrowed. Suturing thus progresses from the midline of the posterior wall of the trachea to a point just behind a midlateral traction suture (Figure 24-15*A*). The traction sutures should lie exactly in the midline laterally, so that superior and inferior traction sutures will correspond when the trachea is pulled together. A second group of sutures is placed from the posterior midline to the opposite lateral traction suture and clipped to that side of the table (Figure 24-15*B*). I prefer to place each "set" of sutures from the corresponding side of the table. Traction sutures are aligned so that the upper ones stretch out in the superior end of the field and the lower ones in the inferior end of the field. Placement of anastomotic sutures is facilitated by the assistant's pull on the traction sutures in appropriate directions.

The balance of the sutures are now placed anteriorly between the two sets of traction sutures and clipped in a fan-like arrangement to the drapes over the lower anterior chest wall below the inferior por-

C

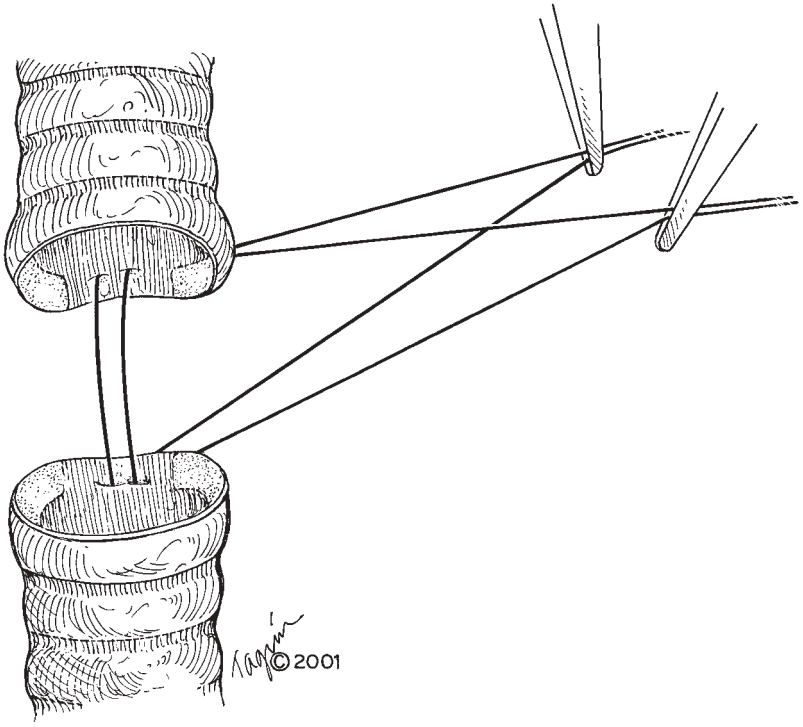


FIGURE **24-14** (CONTINUED) *Tracheal anastomosis.* C, *Each succeeding suture will follow this pattern, clipped to the drapes caudad to the prior suture but lying anterior to it.*

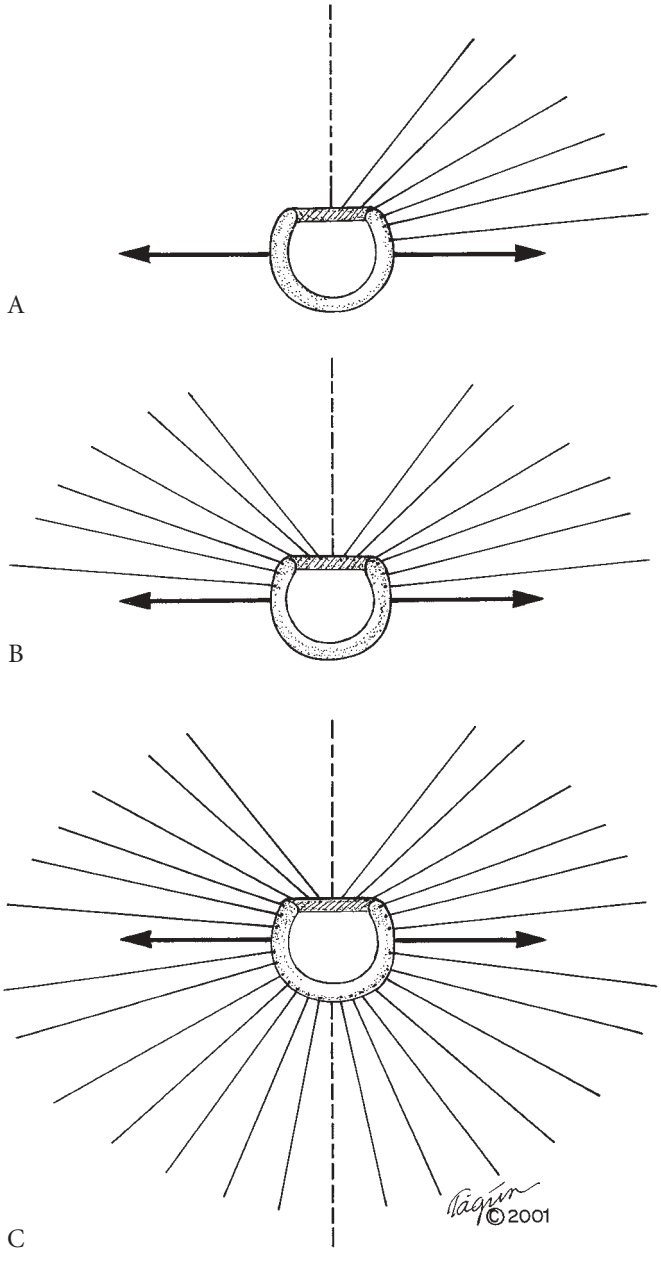


FIGURE **24-15** *Placement of the three groups of anastomotic sutures.* Dashed line*: midline.* A, *Initial posterolateral group running from the posterior midline of the trachea to a lateral midline stay suture (*arrow*).* B, *Second posterolateral group similarly placed on the opposite side of the trachea.* C, *Anterior and anterolateral sutures are finally placed, running from one lateral stay suture to the opposite lateral stay suture.*

tion of the incision (Figure 24-15*C*). I usually place bracketing sutures, one on either side, just anterior to the lateral traction sutures, and then fill in appropriately between them. Discrepancy in the size of the proximal and distal trachea can be corrected by eye, with proportional placement of sutures. The tracheal ends are *never* tailored with wedge resections to correct discrepancies in size, even in a marked "saber sheath" trachea. The endotracheal tube in the distal trachea may be periodically removed if it interferes with suture placement. Monitoring oxygen saturation helps to guide intermittent removal of the tube.

When all anastomotic sutures are placed, the endotracheal tube across the field is removed. The peroral proximal tube is drawn into the field and, if a guiding catheter has been placed, it is removed. The distal airway is carefully suctioned. The proximal endotracheal tube is then guided further down into the dis-

tal segment of the trachea using blunt forceps. It is important not to pass it too far distally, since when the neck is flexed the tube will be driven into the right main bronchus or against the carina, making approximation difficult. The anastomotic sutures should be pulled taut before advancing the proximal endotracheal tube, and the tube advanced with care to avoid looping a suture over the tube. If a suture is inadvertently tied over the tube, it may be impossible to extubate at the end of the operation without breaking the suture forcefully.

The inflatable bag beneath the patient's shoulders ("thyroid bag") is deflated, giving a measure of cervical flexion. With the surgeon and the surgical assistant on each side gently drawing together the 2-0 lateral Vicryl traction sutures, one against the other, the anesthetist or the anesthetist's assistant places the patient's neck in flexion. Folded blankets are placed beneath the occiput to hold the neck firmly in flexion. This directs the chin down toward the sternal notch, lowering the larynx. Flexion is *never* extreme or forced. The endotracheal tube is adjusted in case it has slipped too far distally, and the cuff is inflated to provide a seal for ventilation. Lateral traction sutures are tied on either side to provide approximation without significant intussusception, using the double strands tied in a surgeon's knot. If due to pathology or anatomic discrepancy a degree of intussusception occurs, it is acceptable. Smooth healing will follow. The ends of these tied traction sutures are clipped with a larger clamp on either side.

The anterior anastomotic sutures are tied first. If the tracheal wall or cricoid cartilage is rigidified due to calcification, then the sutures may be tied more easily by approaching the midline from each side. This avoids initial excessive tension on the central suture. Sometimes, tentative approximation can be encouraged using a clamp-held "peanut" to push the ends together while initial tying is done. A more effective maneuver is for the assistant to pull the ends together by crossing the next suture beyond the one being tied by the surgeon. In cases where there is more discrepancy than desirable, I use one or more heavier sutures in the midline (3-0 or, on rare occasions, a 2-0 midline "keystone" suture). After each suture is tied with five knots, the excess suture is cut. This frees the field of dangling sutures.

On occasion, a considerable degree of calcification, particularly in the cricoid and sometimes in the trachea, resists placement of sutures. By taking a short hold on the anastomotic suture needle and driving it in at a right angle, a spot is often found where the needle will penetrate. If this is not possible, then I use either a more rugged needle (a hypodermic needle held in a hemostat) to force a channel for sutures, or occasionally a fine dental drill to produce a hole through which the suture needle can be passed.

After the anterior sutures are tied, a retractor is slipped in behind the lateral traction and posterior anastomotic sutures on one side (but in front of the thyroid isthmic traction suture). The assistant gently retracts the tied lateral tracheal traction suture of that side, and the surgeon continues to tie the lateral and posterior groups of anastomotic sutures, beginning with the one just behind the traction suture and progressing toward the posterior midline. The long ends of each suture are cut after the knot has been tied. The most posterior sutures may have to be tied by palpation rather than by direct vision. Each knot is carefully set to eliminate slack. After completing the first set of lateral sutures, the surgeon goes to the other side of the table and repeats the process on the remaining posterolateral sutures. The anastomosis is now complete (Figure 24-16). To test the integrity of a high tracheal anastomosis, the cuff on the endotracheal tube is deflated, the wound is flooded with saline, and the anesthetist transiently produces 30 cm of water pressure by continuous forced ventilation. Air is heard passing out through the larynx and pharynx. If there is a leak, air bubbles will appear in the saline. Usually, there is not. A low anastomosis is tested with similar ventilatory pressure, but the inflated cuff lies above the anastomosis. Since it is contaminated, the wound is thoroughly irrigated.

Posterior anastomotic sutures could be placed and tied prior to placing the anterior sutures. This, however, creates difficulty in patients who undergo lengthy resections of the proximal trachea, especially when close to the larynx. The cervical flexion that is required to bring the patient's trachea together allows only poor access for placing anterior sutures, if the posterior sutures are tied first. Although the described

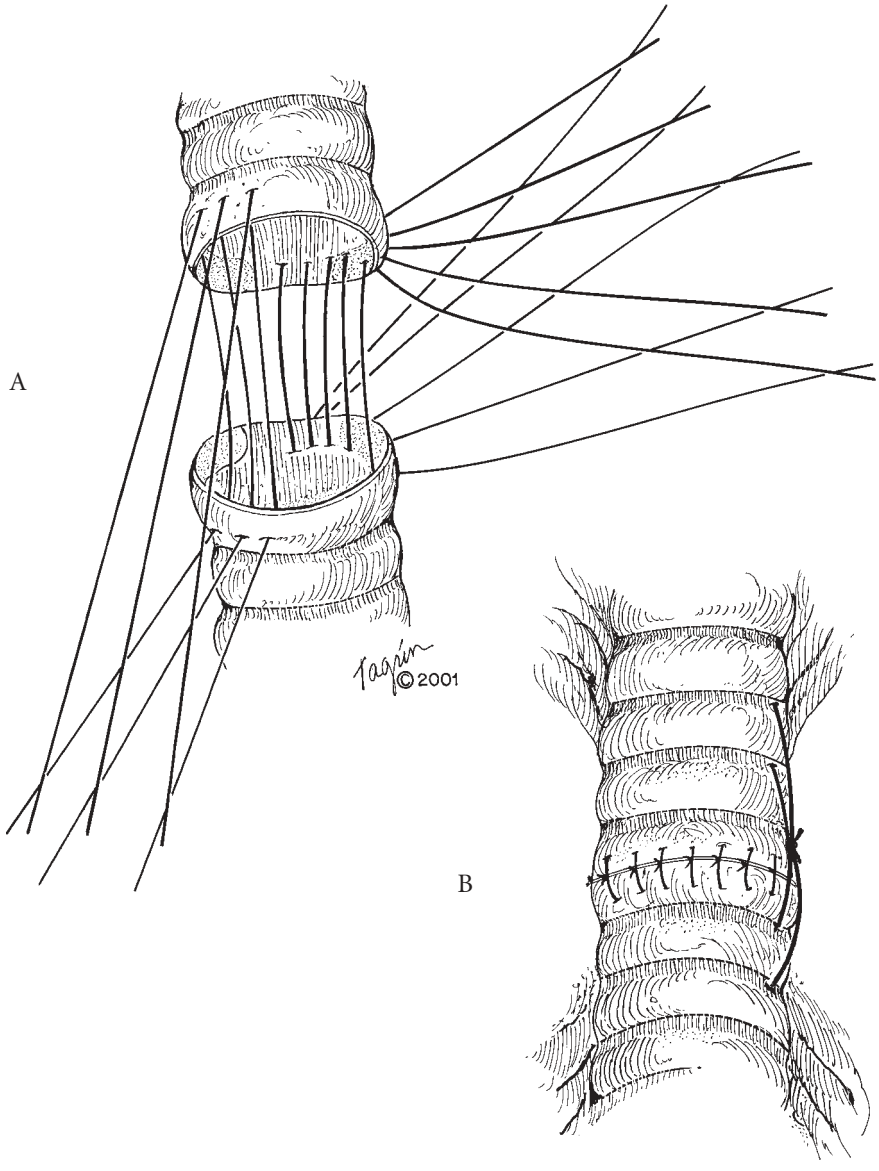


FIGURE **24-16** *Completion of anastomosis.* A, *Diagram to indicate how left posterolateral sutures lie, with the most anterior of the group being in front of the previous suture. For clarity, the right posterolateral as well as the stay sutures are omitted. A few anterolateral sutures are also shown. See text for the order of placement.* B, *The finished anastomosis. The heavy lateral stay sutures are tied first, taking tension off the anastomotic sutures. Only the left-sided stay suture is visible. Sutures have been tied in the order described in the text: anterior sutures first, followed by each posterolateral group, from lateral to medial.*

system of placing all sutures prior to final anastomosis may seem to be complex at first, it becomes easy with consistent use. Its dependability is gratifying. I use this same method for carinal and for bronchial sleeve anastomoses and find it equally satisfactory in all cases.

The thyroid isthmus is frequently reapproximated over a high tracheal anastomosis. If for any reason a further seal is desired over an upper tracheal anastomosis, the strap muscles may be sutured above and below the anastomosis to trachea and in the midline to provide additional buttressing. A second layer is not routinely necessary, except intrapleurally. The use of a muscle pedicle to protect the brachiocephalic artery from the anastomosis has already been noted. The divided upper sternum is sutured with heavy sternal wires, using two or three sutures. Suction drains, which are placed through small stab wounds lateral to each end of the collar incision, lie in the substernal and pretracheal spaces. Strap muscles are sutured in the midline. The platysma is closed and the skin sutured with subcuticular stitches.

After the incision has been closed, the drapes over the neck and head are removed. Often, it is noted at this point that the patient's head and neck are not actually in as marked a flexion as was thought. A heavy "guardian" suture (no. 2) is placed to prevent excessive extension of the neck in the immediate postoperative period. This suture passes transversely through a generous bite of skin in the submental crease and then through the presternal skin (Figure 24-17). Where there is a midline vertical incision, I use two sutures from the chin to either side of the presternal incision. These sutures should be fixed in skin that has not been dissected up from the chest wall. Obviously, it is important to warn the patient and the patient's family about these sutures in advance! The guardian sutures remain for 6 or 7 days following operation. It must be emphasized that the purpose of the guardian suture is to *prevent inadvertent hyperextension postoperatively*, as during sleep. *Extreme approximation of the chin to chest is to be avoided*. Permanent quadriplegia has been reported after extreme positioning intra- and postoperatively, possibly analogous to similar disasters related to prolonged extreme cervical flexion during certain neurosurgical procedures.[5] Extreme *extension* of the neck intraoperatively is also to be avoided. The origin of this disastrous complication is not clear but it may be due to vascular deprivation of the spinal cord. I have not personally encountered this disaster.

Since the patient has been carried on spontaneous or assisted ventilation without paralyzing agents, extubation can usually be carried out as the patient awakens. The airway should be satisfactory. Rarely, laryngeal edema may cause transient difficulty, particularly in procedures that involve the larynx itself. In these cases, a small endotracheal tube may be left in position for a few days, preferably without a cuff or with a deflated cuff. Tracheostomy is to be avoided initially, particularly in cases of lengthy resection. A tracheostomy could lie close to the anastomosis and produce a real risk of injury that could cause restenosis. Tracheostomy is *never* performed through a fresh anastomosis. If anastomosis of the trachea has been done properly, internal stenting, as with a T tube or endotracheal tube, is not required. Further management of a patient with persistent postoperative airway obstruction is discussed in Chapter 20, "Postoperative Management," Chapter 21, "Complications of Tracheal Reconstruction," and Chapter 25, "Laryngotracheal Reconstruction."

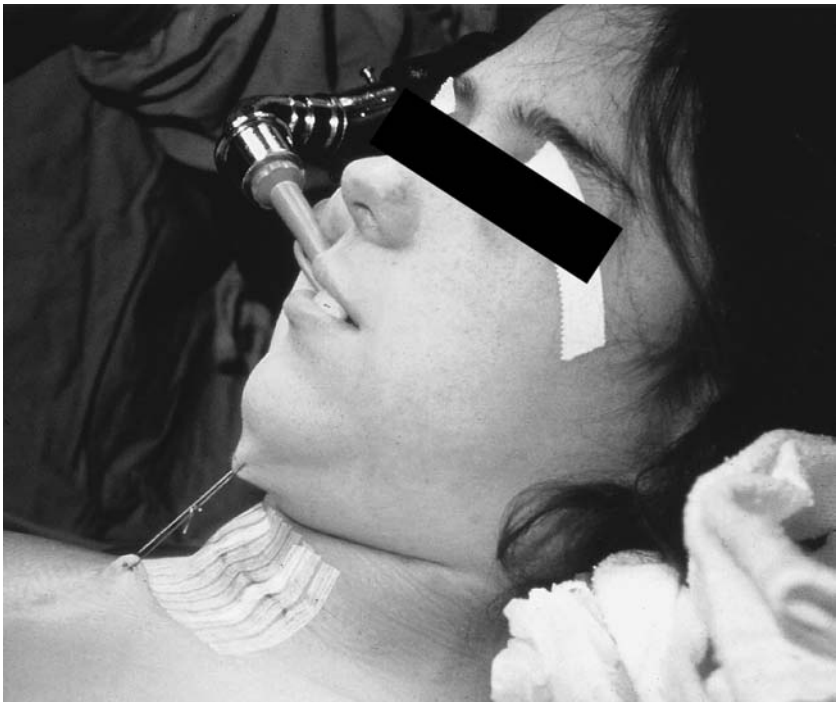


FIGURE **24-17** *The "guardian" chin stitch from the submental crease to presternal skin. The stitch is to prevent hyperextension postoperatively and not to effect extreme hyperflexion.*

Extended Resection

Laryngeal Release

When the length of resection of upper- or midtrachea is so great that anastomosis cannot be completed without excessive tension, anatomic release of the larynx to allow it to devolve distally may provide the necessary additional relaxation. Ogura and Roper described cutting the "ribbon muscles" of the larynx to achieve more mobility after a partial cricoid resection.[6] Two principal methods described were *thyrohyoid release*, proposed by Dedo and Fishman,[7] and *suprahyoid release*, developed by Montgomery.[8] The length of release which results varies individually, but is not great. In practice, between 1 to 2 cm may be expected from this maneuver alone. However, even 1 cm represents nearly 10% of the trachea's length.

Zitsch and colleagues more optimistically measured anatomically a tension-free drop of 1.5 cm with suprahyoid release and a total of 2.0 cm with additional division of the inferior constrictor muscle from the thyroid cartilage.[9] When a tension of less than 1,700 g was applied, a gain in length of 3.5 cm and 4 cm, respectively, was found. Clinical application was not described. The effect on deglutition is hence unknown. Other combinations of release have also been proposed, including infrahyoid and inferior constrictor release[10] and intralaryngeal division of the thyroid cartilage.[11] These last two methods were described in small numbers of patients. I have had no experience with these three techniques.

Laryngeal release is a useful procedure for extended upper tracheal resection and it may also contribute in a lengthy resection of the midtrachea or, on rare occasions, in a lower tracheal and carinal resection which extends to the midtrachea. However, I learned from experience that laryngeal release contributes nothing to low tracheal resection or carinal resection. The relaxation afforded by laryngeal release simply does not transfer all the way to the lower trachea. This was confirmed experimentally by Valesky and colleagues.[12]

Laryngeal release is *not* necessary routinely in tracheal reconstruction, as was advocated.[7] Indeed, in 521 operations for postintubation stenosis, including 53 re-resections, laryngeal release was employed in 49 patients.[4] Laryngeal release was understandably more frequently necessary in secondary operations (29% versus 6.4% in primary operations). In 80 laryngotracheoplastic resections for stenoses of varying etiology, only 7 releases were necessary.[13] Seven laryngeal releases were performed in 89 tracheal resections and 19 laryngotracheoplastic resections for primary tracheal and secondary tumors.[14]

Thyrohyoid Laryngeal Release. I initially used the "Dedo" release technique for thyrohyoid laryngeal release.[7] In this technique, the thyrohyoid muscles and the thyrohyoid membrane are divided above the thyroid cartilage, after retracting the sternohyoid and omohyoid muscles, and the superior cornua of the thyroid cartilage are detached to permit the larynx to drop (Figure 24-18). Great care is taken not to injure the internal branches of superior laryngeal nerves, which lie just behind and medial to the superior cornua of the thyroid cartilage. In a limited personal experience, most patients had significant postoperative difficulty in swallowing, some with aspiration. Patients gradually overcome this disability. F. G. Pearson (personal communication) reported a similar experience. I then moved to the "Montgomery" release technique.[8] Fewer patients have had difficulty with aspiration following this method of release. Those who do are often older and, of course, have needed extended resections. These are precisely the patients most likely to need release. Nearly all patients overcome their aspiration problem with time and with assistance (see Chapter 21, "Complications of Tracheal Reconstruction").

In *Surgery of the Larynx and Trachea* published in 1990, Dedo no longer described the thyrohyoid release, instead presenting the suprahyoid technique which "releases the larynx just as well as the thyrohyoid membrane technique and probably minimizes the risk of injury to the superior laryngeal artery and vein and the internal nerve branch."[15] His technique is therefore presented here for completeness only.

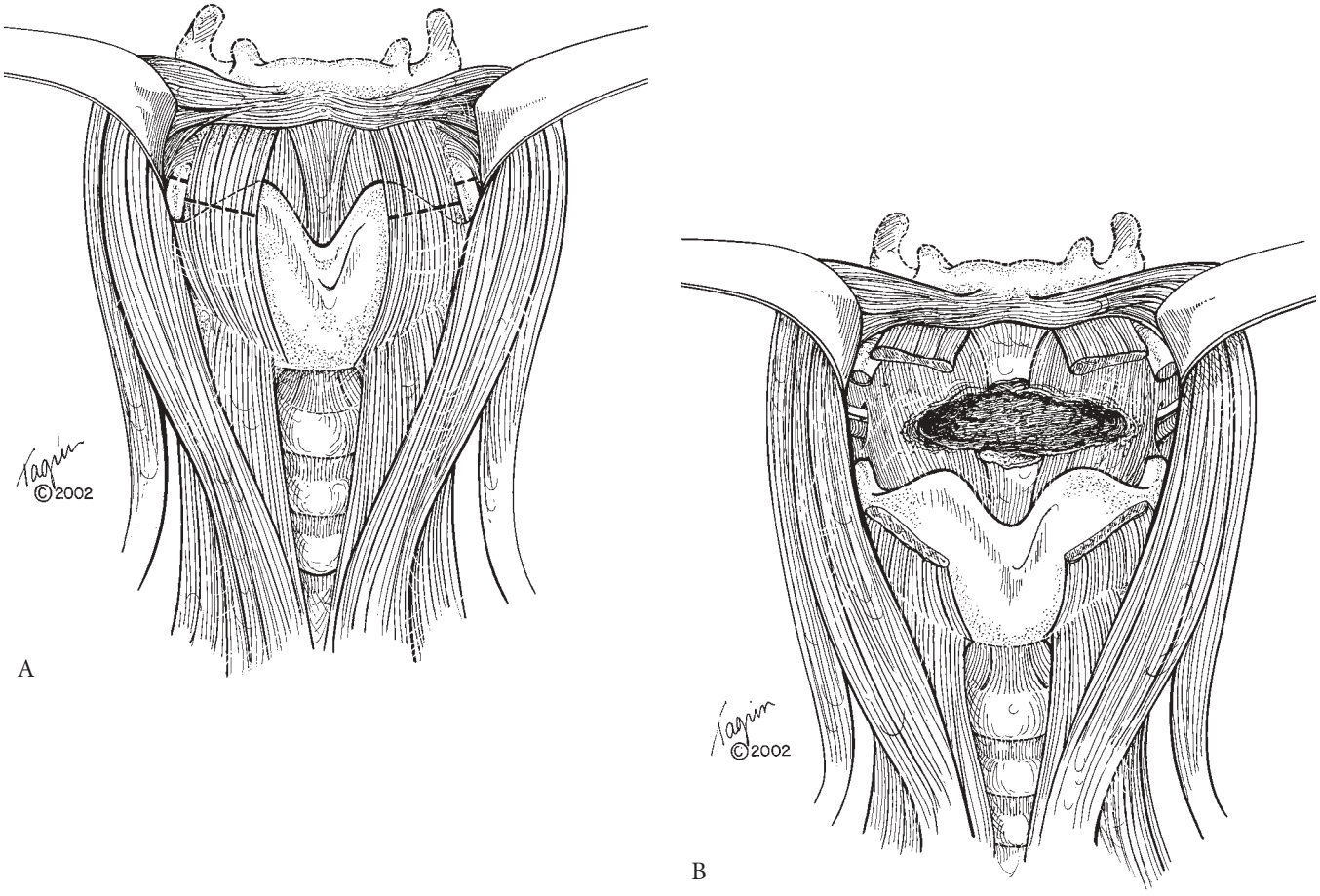


FIGURE **24-18** *Thyrohyoid laryngeal release procedure. Technique of Dedo and Fishman.*[7] A, *Exposure of the larynx between retracted sternohyoid muscles. The thyrohyoid muscles are divided at the upper edge of the thyroid cartilage, and the superior cornua of the thyroid cartilage are divided. The internal branch of the superior laryngeal nerve and the superior laryngeal artery are medial to the cornu and must be carefully avoided.* B, *The thyrohyoid ligament and membrane are opened transversely. Note the artery and nerve described in the legend for* A.

Suprahyoid Laryngeal Release. Since I prefer to use a low collar incision for tracheal resection and reconstruction, rather than a higher incision or a U-shaped flap, I make a second short transverse incision directly over the hyoid bone (Figure 24-19*A*). This combination of incisions affords good access for the release and the reconstruction, and at the same time is more cosmetic. The incision for release is carried directly down to the hyoid bone. The superior surface of the central hyoid is exposed and dissection is carried laterally (Figure 24-19*B*). The tendons of the stylohyoid muscles attaching just below the sling for the digastric muscle are divided on either side. The digastric sling is left intact. All muscles attached to the hyoid between the two digastric slings, including the tendons of chondroglossus muscles to the lesser cornua of the hyoid bone, are detached. These are the mylohyoid, geniohyoid, and genioglossus (Figure 24-19*C*). I have found it convenient to do this with cautery, although this makes definition of the individual muscles less clear. The hyoid bone itself is divided lateral to the lesser cornu and medial to the digastric sling on either side (see Figure 24-19*C*). The preepiglottic space is widely opened (Figure 24-19*D*). Dissection, subcutaneously on the suprafascial plane to join the two cutaneous incisions, adds nothing to the release. A flat suction drain is placed in the preepiglottic space and the incision is closed in two layers. Immediate closure of the incision is important because it becomes inaccessible after completion of the tracheal anastomosis, when the neck is placed in flexion.

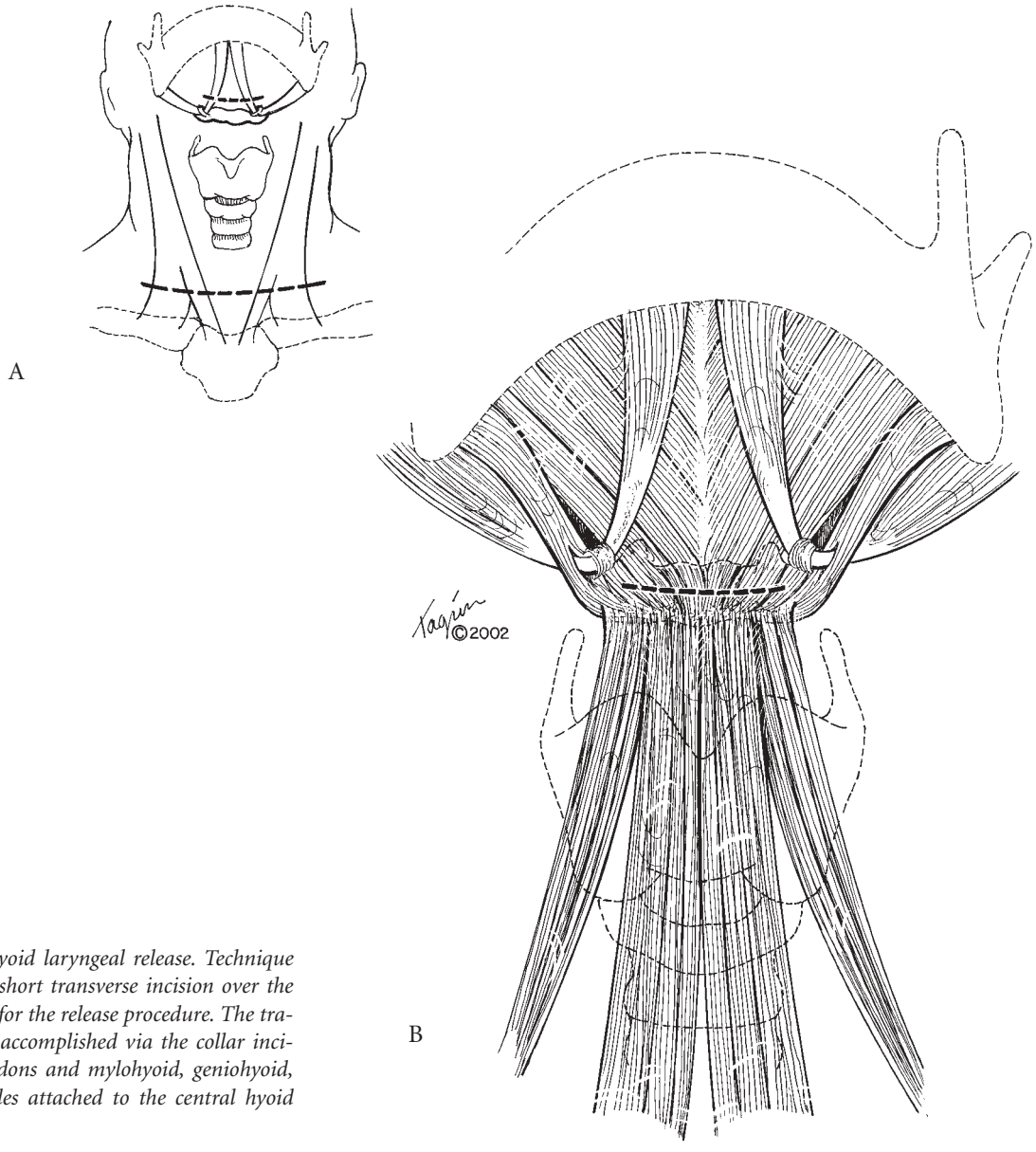
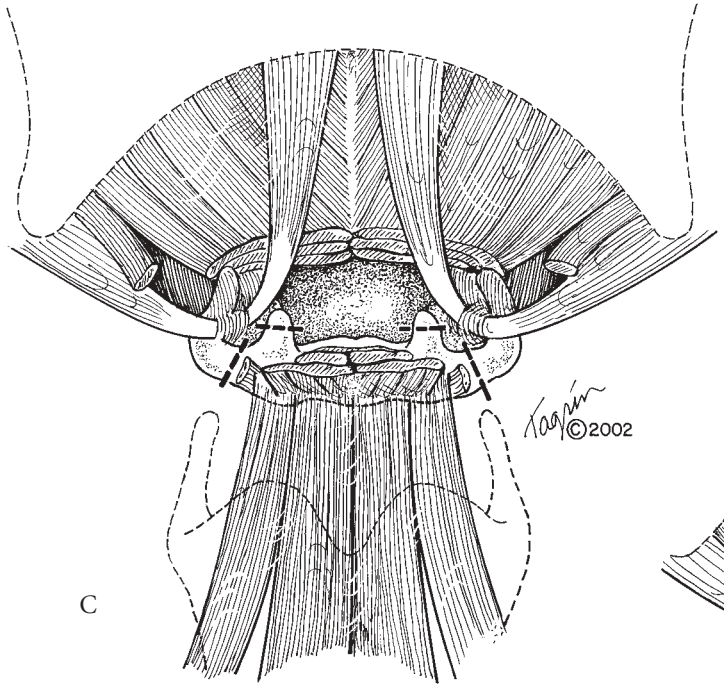


FIGURE **24-19** *Suprahyoid laryngeal release. Technique of Montgomery.*[8] A, *A short transverse incision over the hyoid bone is sufficient for the release procedure. The tracheal reconstruction is accomplished via the collar incision.* B, *Stylohyoid tendons and mylohyoid, geniohyoid, and genioglossus muscles attached to the central hyoid bone are divided.*

Recapture of Tracheal Length

In some patients, a tracheostomy has been made just above or below a lengthy segment of tracheal stenosis, as emergency treatment for the stenosis, unfortunately even where the stenotic segment was accessible in the neck. In some, a stoma has been located below a stenosis as a safeguard for repeated laser treatments of the stenosis. These compound the length of lesions, which might otherwise have been easily corrected by resection. The stoma may lie so close to the area of stenosis that there is not a bridge of normal enough trachea left between the stoma and stenosis to permit dependable anastomosis. Resection of the new tracheal stoma, along with the long adjacent stenosis, may result in excessive anastomotic tension. In such a patient, the recent tracheostomy may be abandoned and allowed to heal (Figure 24-20). This may recapture 1.0 to 2.0 cm of trachea, adequate for reconstruction.[16] The patient is observed in hospital without a tracheostomy



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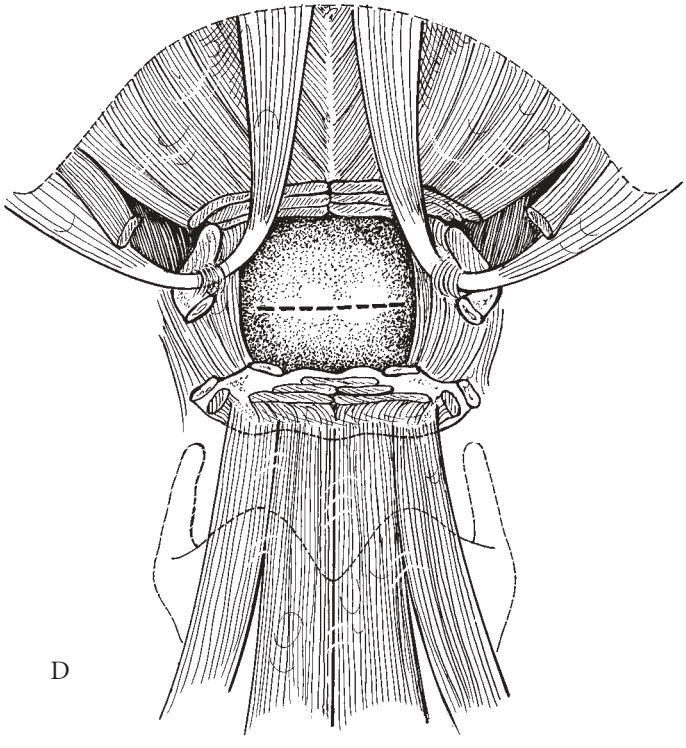

D

FIGURE **24-19** (CONTINUED) *Suprahyoid laryngeal release.* C, *The lesser cornua of the hyoid with the chondroglossus muscles are transected. The digastric muscle slings attaching to the hyoid are left intact. The hyoid itself is divided on both sides anterior to the digastric attachments and lateral to the lesser cornua.* D, *The suprahyoid membrane is opened and the preepiglottic space entered.*

during stomal healing, with periodic dilations as needed to maintain the airway. This is usually preferable to inserting an endotracheal tube, which causes inflammation, or to locating another tracheostomy at a remote distance since this may limit mobility. If any inlying stent is used to temporize, it should be of silicone so that it will be easily removable with minimal injury to the trachea. If the stenosis is accessible in the neck, the tracheostomy is replaced in the damaged segment, which is ultimately to be resected (Figure 24-21) while waiting for the prior stoma to heal. After the offending stoma has sealed sufficiently (usually within 2 weeks) and some regression of inflammation has occurred, then resection of the stenotic segment with primary anastomosis is accomplished. These considerations do not apply where the existing stoma is remote from the lesion or where its inclusion in resection will not unduly lengthen the resection (Figure 24-22).

Hilar Release

A hilar release has only rarely been performed for benign stenosis of the upper or midtrachea, since it hugely increases the extent of surgery otherwise planned as a cervicomediastinal procedure. The gain in length

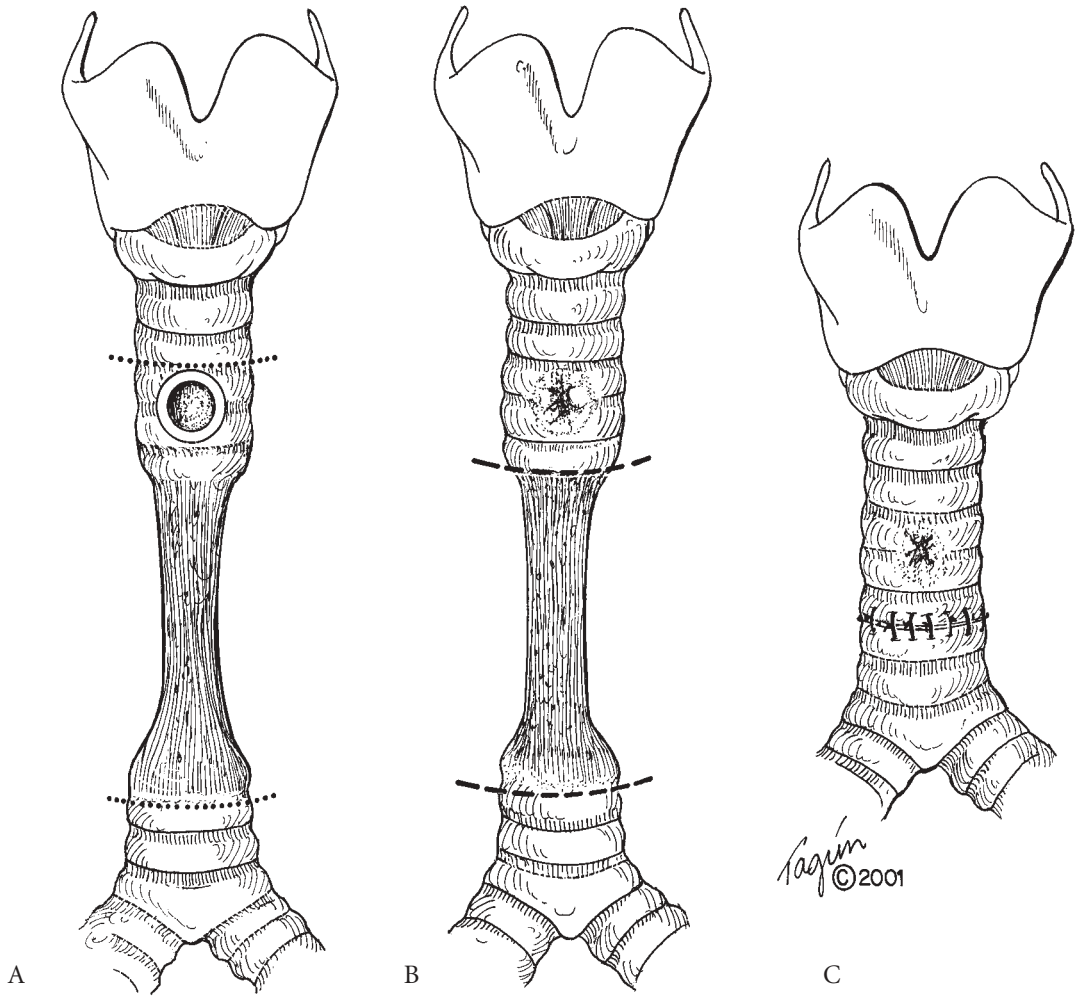

FIGURE **24-20** *Recapture of tracheal length for recontruction where a stoma has been made just proximal to a lengthy stenosis.* A, Dotted lines *indicate the excessively long resection that would be required.* B, *The stoma is allowed to heal prior to resection* (C).

over mobilization of the pretracheal plane, cervical flexion, and laryngeal release is not great, even when release is bilateral. Accomplishing bilateral hilar release is difficult, requiring in such a patient a right pleurotomy via complete sternotomy and a T incision into the left fourth interspace or intrapericardial exposure on the left (see Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). Although this may be justified for neoplasm in a patient who can tolerate the procedure, acceptance of T tube restoration of an airway may be more judicious for a lengthy benign stenosis, except in a young and fit patient. In such an extreme circumstance, laryngeal release is often also useful if the lesion involves the midtrachea.

Management of Malacic Segments

A segment of trachea may rarely and inexplicably become malacic rather than stenotic from the effect of a cuff injury. Such a lesion is best treated by resection and anastomosis. A small number of patients with a well defined cuff stenosis may also have a malacic segment between the site of the original tracheal stoma (or an existing stoma) and the stenosis. When the distance between the stoma and the stenosis is short, and removal will not result in too extensive a resection, it is preferable to resect the entire area of damaged

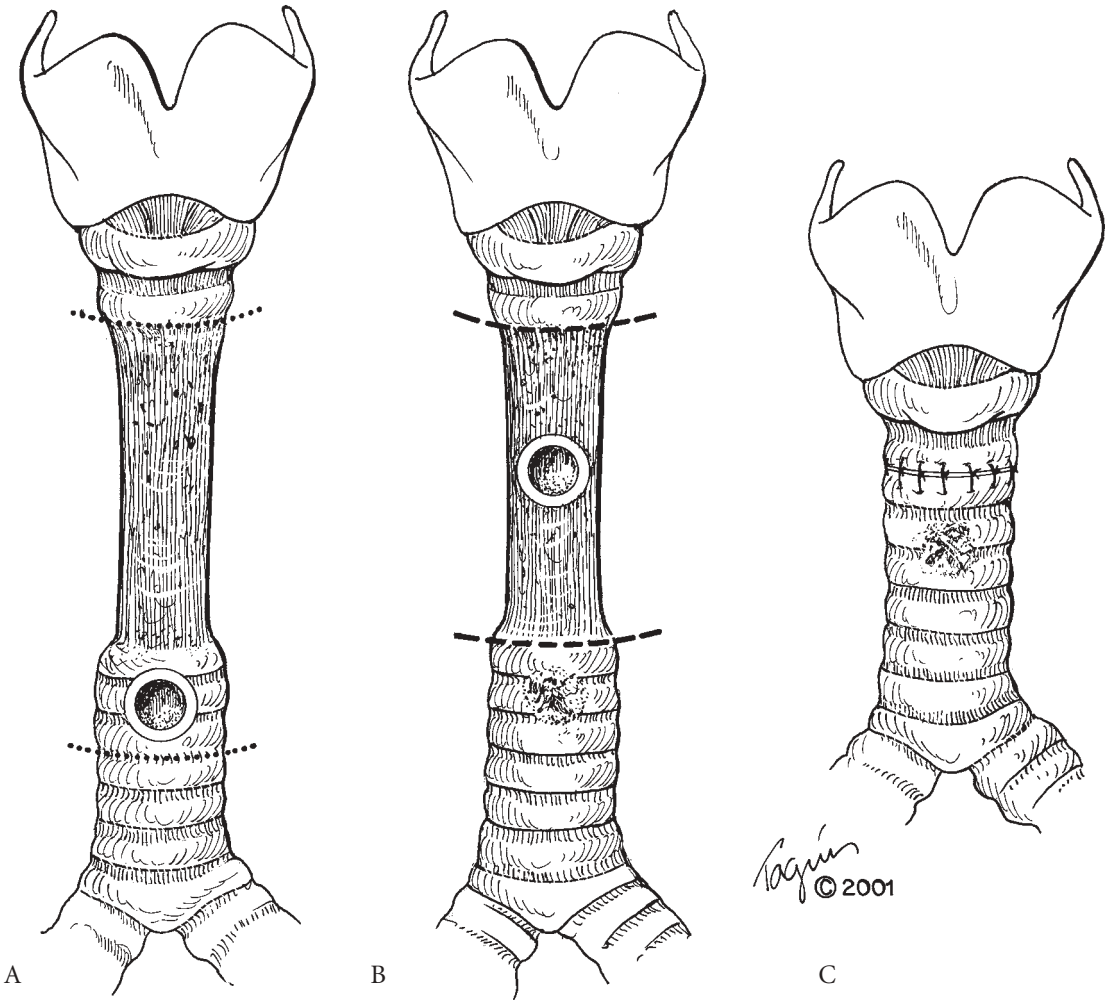

FIGURE **24-21** *In this case, a stoma was made below a lengthy stenosis* (A). *It is transferred into the stenotic trachea* (B) *and resection performed* (C) *after stomal healing.*

trachea. External cartilage grafts have not been very successful in malacia. The grafts survive, but most often are not incorporated into the tracheal wall to provide stability.

A small number of patients, in whom complete resection of both lesions would have been impossible, were managed by resecting the stenosis and by external splinting the malacic segment with specially designed polypropylene rings.[16] Splinting was limited to the use of one or two rings. These rings were developed originally for construction of cervical cutaneous tubes. Their use in tracheal splinting is described in more detail in Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea." Splinting a malacic segment of the trachea in this way may be hazardous. Care must be taken not to free up such a thin and altered segment circumferentially throughout its entire length or it is likely to necrose, as occurred in one patient. Tracheomalacia resulting from softening or loss only of cartilages differs from malacia due to what is essentially total loss of the tracheal wall and scar replacement. The latter necroses easily when freed from its connection to surrounding tissue from which blood supply derives. It is necessary to create an individual tunnel for each ring around softened trachea, so that the blood supply will remain. A ring must not be in contact with either a stoma or the anastomosis to avoid infection. Rings must be thoroughly imbedded in tissues using strap muscles. These foreign bodies

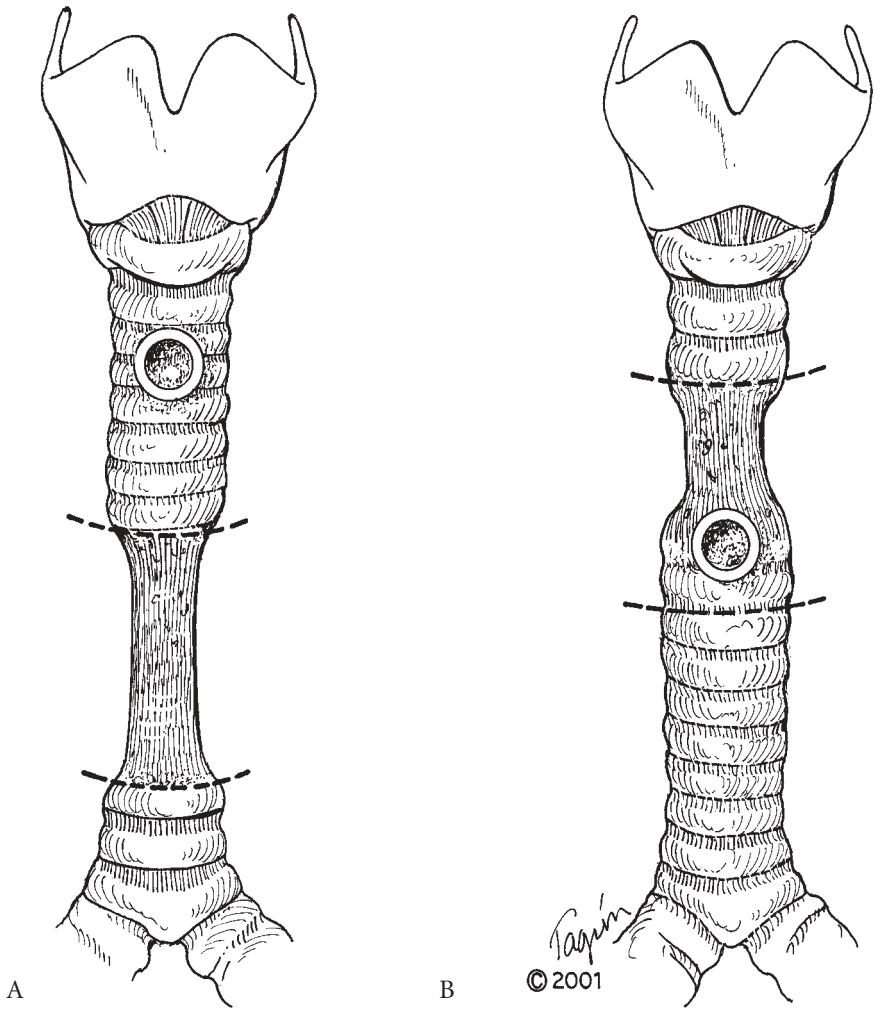

FIGURE **24-22** A, *A stoma sufficiently remote from a lesion so that anastomosis can be made in the healthy trachea above and below the stenosis. The stoma is left in place, replanted, or surgically closed, depending upon circumstances.* B, *The stoma and stenosis may be excised in continuity here since the total length is not great enough to produce excessive anastomotic tension.*

may become infected from the contaminated operative field and require later removal. The general rule, that it is best to avoid foreign material in tracheal reconstruction, remains sound. No consistent success in the treatment of tracheomalacia has been reported with the use of other types of external splints, either of synthetic or cartilaginous autografts. Expandable internal stents are not safely used adjacent to tracheal anastomoses. A safer alternative, generally, for management of an otherwise unreconstructible trachea, is a permanent T tube. Long-term results of the primary use of inlying stents remain uncertain (see Chapter 40, "Tracheal and Bronchial Stenting").

References

1. Perelman MI. Surgery of the trachea. Moscow: Mir; 1976. p. 197.
2. Sweet RH. Thoracic surgery. Philadelphia: WB Saunders; 1950. p. 232.
3. Friedman E, Perez-Atayde AR, Silvera M, Jonas RA. Growth of tracheal anastomoses in lambs. Comparison of PDS and Vicryl suture material and interrupted and continuous techniques. J Thorac Cardiovasc Surg 1990;100:188–93.
4. Grillo HC, Donahue DM, Mathisen DJ, et al. Postintubation tracheal stenosis. Treatment and results. J Thorac Cardiovasc Surg 1995;109:486–93.
5. Dominguez J, Rivas JJ, Lobato RD, et al. Irreversible tetraplegia after tracheal resection. Ann Thorac Surg 1996;62:278–80.
6. Ogura JH, Roper CL. Surgical correction of traumatic stenosis of larynx and pharynx. Laryngoscope 1962;72:468–80.

7. Dedo HH, Fishman NH. Laryngeal release and sleeve resection for tracheal stenosis. Ann Otol Rhinol Laryngol 1969;78:285–96.
8. Montgomery WW. Suprahyoid release for tracheal anastomosis. Arch Otolaryngol 1974;99:255–60.
9. Zitsch RP III, Mullins JB, Tampler J, Davis WE. Suprahyoid and inferior constrictor release for laryngeal lowering. Arch Otolaryngol Head Neck Surg 1995;121:1310–3.
10. Biller HF, Munier MA. Combined infrahyoid and inferior constrictor muscle release for tension-free anastomosis during primary tracheal repair. Otolaryngol Head Neck Surg 1992;107:430–3.
11. Sachs ME, Conley J, Rabuzzi DD. Intralaryngeal release for tracheal anastomosis. Ann Otol Rhinol Laryngol 1983;92:482–4.
12. Valesky A, Hohlbach G, Schildberg FW. Wertigkeit unterschiedlicher Mafsnahmen zur Minderung der Anastomosenspannung nach Kontinuitätsresektion der trachea. Langenbecks Arch Chir 1983; 360:59–69.
13. Grillo HC, Mathisen DJ, Wain JC. Laryngotracheal resection and reconstruction for subglottic stenosis. Ann Thorac Surg 1992;53:54–63.
14. Grillo HC, Mathisen DJ. Primary tracheal tumors: treatment and results. Ann Thorac Surg 1990;49:69–77.
15. Dedo HH. Surgery of the larynx and trachea. Philadelphia: BC Decker; 1990.
16. Grillo HC. Surgical treatment of postintubation tracheal injuries. J Thorac Cardiovasc Surg 1979;78:860–75.

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Laryngotracheal Reconstruction

Hermes C. Grillo, MD

Inflammatory Subglottic Stenosis
Neoplasms Involving the Lower Larynx and the Trachea

Postintubation stenosis often involves both the subglottic larynx and the upper trachea. Stenoses from other causes, such as idiopathic stenosis, trauma, or Wegener's granulomatosis, may involve the same portions of the upper airway. Tumors may also extend across the laryngotracheal junction, and, in particular, invasive differentiated thyroid carcinomas. Simple circumferential segmental resection of these lesions would fail to preserve recurrent laryngeal nerve function. However, a single-stage resection and reconstruction of the airway can be performed with salvage of a functional larynx. Since pathology and its management differ between inflammatory stenosis and neoplastic lesions of this region, they are considered separately in this chapter.

Inflammatory Subglottic Stenosis

Combined subglottic laryngeal and upper tracheal stenosis following intubation may result from tissue damage by an endotracheal tube, from cricothyroidostomy, or from a high stoma which was misplaced or eroded superiorly (see Chapter 11, "Postintubation Stenosis"). If the tracheal stenosis abuts the cricoid cartilage without intralaryngeal extension, circumferential tracheal resection is performed just below the cricoid (Figure 25-1*A*). If, however, there is a small "atrium" or space immediately below the vocal cords, even though not of normal caliber, a tight stenosis below this level, which begins within the subglottic larynx, may be treated by the procedures described below (Figures 25-1*B*,*C*,*D*). Procedures for primary reconstruction of the airway after resection of lesions involving the subglottic larynx and upper trachea, with preservation of recurrent laryngeal nerves, evolved from work by Conley in 1953, Ogura and colleagues in 1964 and 1971, and notably by Gerwat and Bryce, and Pearson and colleagues in 1974 and 1975, respectively.[1–5] Couraud and colleagues applied the procedure successfully in 1979, Grillo described a modified operation in 1982 and 1992, and Monnier and colleagues reported successful reconstruction and subsequent growth in children using such a technique.[6–11] These procedures are very different from simple segmental tracheal resection, are difficult to accomplish, and require meticulous attention to details. The immediately subglottic laryngeal airway to which trachea is anastomosed is almost always narrowed, and inflammation or scar is usually present. Procedures described here are applicable to idiopathic stenosis and, less commonly, to other stenotic lesions

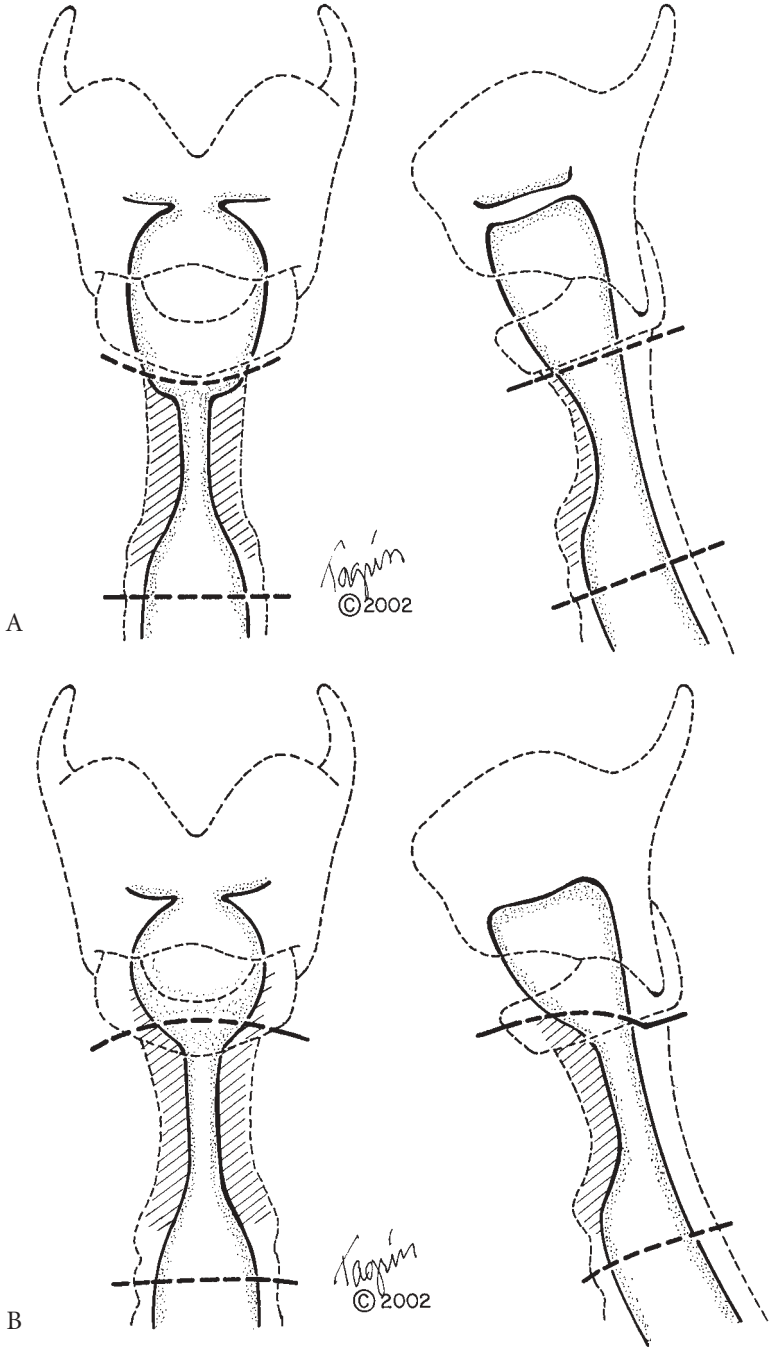
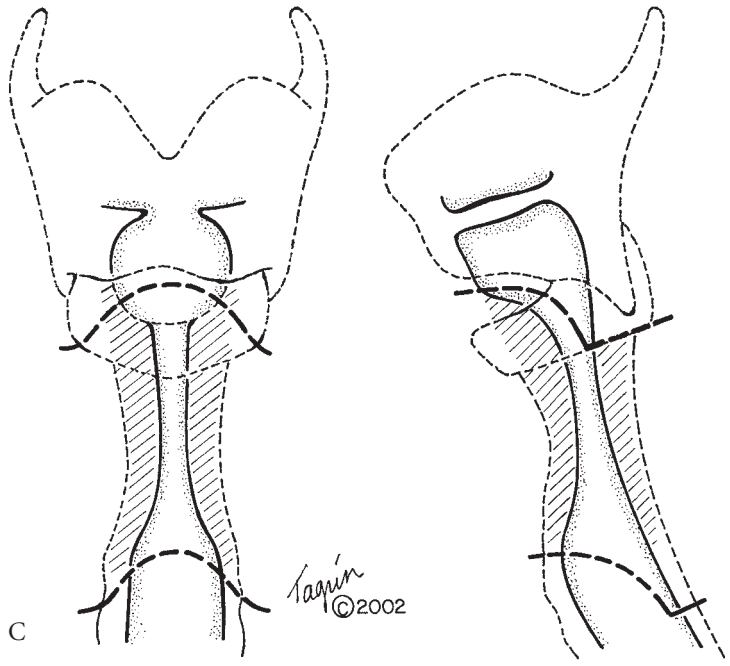


FIGURE **25-1** *Spectrum of high tracheal and laryngotracheal stenosis. The precise extent of stenosis, both linear and anteroposterior (AP), determines the type of resection and reconstruction applicable. In these diagrams, AP projection is at the* left, *lateral at the* right. *Cricoid and thyroid cartilages are outlined with* dotted lines. *The vocal cords and the borders of the subglottic laryngeal and tracheal airway are demarcated by* solid lines. *The extent of the stenosis is* shaded. A, *High tracheal stenosis, in this case anterior, without extension into the subglottic larynx. Simple circumferential tracheal resection suffices. The proximal line of resection is at the inferior border of the cricoid. Circumferential stenosis at this level is identically resected. The trachea is anastomosed to the cricoid cartilage.* B, *Anterior stenosis extending into the lower subglottic larynx, but involving only the inferior portion of the cricoid cartilage anterolaterally. The line of resection bevels the anterolateral cricoid at a point where the lumen becomes acceptable.*

(see Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions"). Reconstructive procedures must be applied cautiously in certain diseases of unknown origin, such as Wegener's granulomatosis, which may have a progressive course despite surgery.

If the stenosis abuts the vocal cords without any free space in the immediate subglottic larynx (Figure 25-1*E*), it is not possible to perform a single-stage operation for correction. One must then resort to older, multistaged procedures which use the following elements in various combinations: laryngofissure, excision of scar tissue, placement of skin graft or buccal mucosal graft, insertion of a



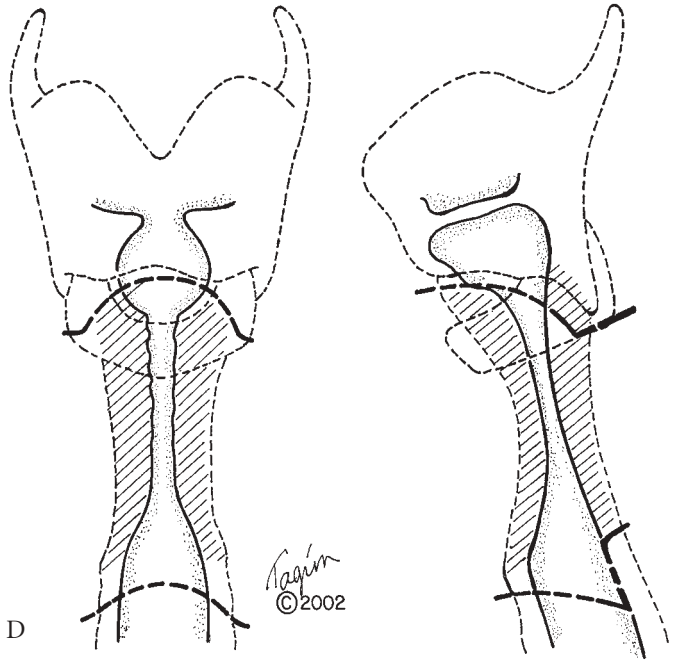
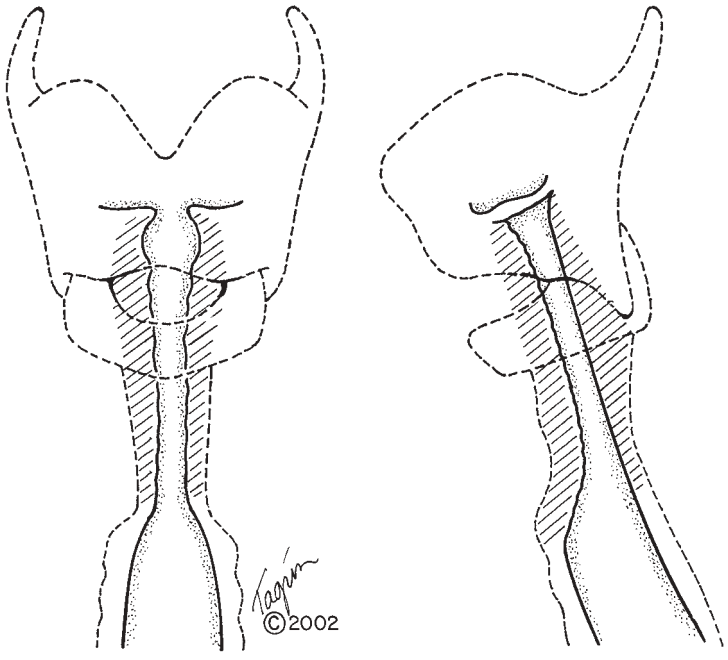


FIGURE **25-1** (CONTINUED) C, *When subglottic damage involves the entire extent of the anterior cricoid, this portion of cricoid arch and lateral laminae must be excised. The distal trachea is tailored to repair the anterior arcuate defect. There is minimal or no involvement of the posterior cricoid. The membranous wall of the trachea may or may not be damaged.* D, *Circumferential stenosis involving the lower subglottic larynx posteriorly as well as anteriorly. Proximal extent of the stenosis varies widely and determines the feasibility of single-stage repair. The anterior portion of the stenosis is excised with the anterior cricoid arch. The posterior cricoid lamina is preserved in order to protect the recurrent laryngeal nerves. The internal ring of scar is excised from the posterior cartilaginous plate. The distal line of tracheal division fashions an anterior "prow" of cartilage, but preserves a broad-based flap of posterior membranous wall to resurface the bared posterior cricoid plate.*

stent, anterior or posterior cricoid division, costal cartilage or hyoid bone grafts (pedicled or not).[8,12] Such otolaryngological procedures are not within the scope of this discussion.

Otolaryngological consultation should be sought to assess subglottic strictures that involve the larynx. Complete and accurate assessment of vocal cord function and glottic adequacy must be made preoperatively. In addition, it is important to identify and assess the severity of additional lesions that may be present, such as posterior commissural interarytenoid stenosis, and to appraise the size and quality of the subglottic space. If procedures are required to assure glottic competence (eg, vocal cord lateralization,



E

FIGURE 25-1 (CONTINUED) E, *Extreme subglottic stenosis that extends circumferentially up to the vocal cords. No residual "atrium" remains beneath the glottis to which the distal airway can be satisfactorily anastomosed. Such lesions are treated surgically by laryngofissure, excision of scar, and resurfacing with buccal mucosa or skin, held with a temporary stent. Tracheal repair and reconnection usually is best done in a second procedure.*

arytenoidectomy, lysis of commissural stenosis, rotation of a posterior commissural esophageal mucosal flap), I prefer to see these accomplished first and be assured of their success before embarking on subglottic resection and reconstruction. Many of these patients already have a tracheostomy, which provides an airway while any necessary glottic repair is healing. In some cases, glottic procedures and laryngotracheal reconstruction have been combined. Maddaus and colleagues reported combined repairs, which required postoperative tracheostomy and splinting with T tubes.[13] If the glottic defect is corrected first, then a tracheostomy is most often unnecessary following repair of a subglottic stenosis.

Sufficient space must be present below the cords where the anastomosis may be fashioned, in order for a single-staged procedure to succeed. A rough rule to follow is that the cross-sectional area of the aperture below the vocal cords in this uppermost segment should be at least half of normal and, further, should not be excessively inflamed, ulcerated, or granulomatous. If the process involves the glottis or extends uninterruptedly to the vocal cords from below, then it is clearly in the province of the otolaryngological surgeon initially. In every case, patients should be warned that their voice may well be hoarse for a time postoperatively, and that quality and volume may well always differ from normal.[9]

Two situations are noted. In the first and simpler case, the stenosis lies in the *anterior* subglottic larynx, exemplified by the lesions that occur following erosion into the cricoid by a high stoma or cicatrization due to cricothyroidostomy (see Figures 25-1*B*, *C*). The second and more difficult problem is that of *circumferential* stenosis, where the lesion includes the anterior surface of the posterior cricoid plate below the arytenoid cartilages (see Figure 25-1*D*). Circular stenosis of this type most often follows circumferential damage from prolonged endotracheal tube intubation or is seen in idiopathic stenosis.

Intubation for Anesthesia

Many patients will already have a tracheostomy tube in place either in the damaged segment or below it. This stoma is used for induction and maintenance of anesthesia by placing a flexible endotracheal tube through it, as described in Chapter 18, "Anesthesia for Tracheal Surgery." In the absence of a preexisting tracheostomy, I prefer not to dilate a tightly stenotic laryngeal airway any more than is absolutely necessary, in order to

avoid producing sufficient trauma in the larynx to make repair difficult and, also, to contribute to postoperative laryngeal edema. After minimal dilation, an uncuffed endotracheal tube of small diameter (5.5 or 6 mm ID) is passed to provide safe ventilation in the early phases of dissection. The tightness of stricture provides a seal for the tube. Tight stenosis is encountered more often with circumferential subglottic stenosis than with anterolateral stenosis. Once the trachea is transected below the lesion and a distal airway is established, the proximal endotracheal tube is withdrawn. In these patients, where repair will be intralaryngeal, I prefer to withdraw the translaryngeal tube completely, rather than suture a catheter to it. Even a small catheter crowds the tiny operative field. An endotracheal tube is replaced later, as described below.

Resection and Reconstruction: Anterior Subglottic Stenosis

Basic dissection is entirely similar to anterior upper tracheal resection (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). Often, little pretracheal mobilization is necessary. This approach is applicable where the entire extent of the *anterior* cricoid is involved and, also, where there may be circumferential damage in the proximal trachea, but where the posterior cricoid plate is not significantly involved (Figures 25-2*A*,*B*). If only the lower border of the anterior cricoid cartilage is involved, then this technique is not needed. In such a case, a simple segmental resection of the upper tracheal stenosis is accomplished, additionally bevelling off a portion of the lower margin of the anterior cricoid cartilage (see Figure 25-1*B*). If the full vertical length of the anterior cricoid is involved (see Figure 25-1*C*), then it is resected. The cricothyroid muscle on either side is elevated sharply with a scalpel, carrying the dissection sufficiently posterior for division of lateral cricoid laminae. The proximal line of resection encompasses the anterior arch of the cricoid cartilage, including as much as is necessary of the cricothyroid membrane, usually more rather than less (see Figures 25-2*A*,*B*), thus bringing the resection nearly to the level of the thyroid cartilage. The resection line bevels laterally through the lateral laminae of the cricoid cartilage, usually more than halfway to a midlateral line. The balance of the line of resection posteriorly is at the level of the inferior margin of the cricoid cartilage (see Figures 25-2*B*,*C*). The surface of the vocal cords may be seen just a few millimeters above the anterior inferior midline border of the thyroid cartilage. If there is any question about the extent of resection of the anterior cricoid arch that will be needed, the initial proximal line of resection can be made just beneath the cricoid circumferentially. The arcuate excision is then made under direct vision from below.

The distal line of resection of the lesion is placed below the level of stenosis, just above the first uninvolved tracheal ring or a ring that has good cartilaginous structure, even if slightly involved by inflammation or scar. This ring is bevelled backward from a high point in the anterior midline to the lower margin of that ring posteriorly on either side (see Figures 25-2*A*–*D*). The membranous wall is cut straight across. Only one ring is bevelled in this way, even though the inverted "U" of the superior anterior laryngeal resection line may seem to be a much sharper angle than that below. This avoids creating a floppy flap of cartilage anteriorly, which might occur if two rings were so trimmed. As a consequence, the trachea may arch forward slightly in the subsequent anastomosis. This may provide a better lumen at the anastomotic level.

Initial dissection is similar to that described in the anterior approach to the trachea (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). The upper incisional flap must be elevated higher on the surface of the thyroid cartilage, usually to the superior notch of the thyroid cartilage. Dissection of the anterior surface of the airway is superficial to the cricothyroid muscle and cricothyroid membrane. The thyroid isthmus or its remnant is divided and retracted laterally. The pyramidal lobe of the thyroid is divided or removed if it lies in the way. It is necessary to detach the often scarred medial attachments of the upper poles of the thyroid gland from the cricothyroid muscle. Dissection is not, however, carried any further laterally than is necessary to expose the midportion of the lateral laminae of the cricoid cartilage. Pretracheal dissection down to the carina is often done, but may be omitted in those patients in

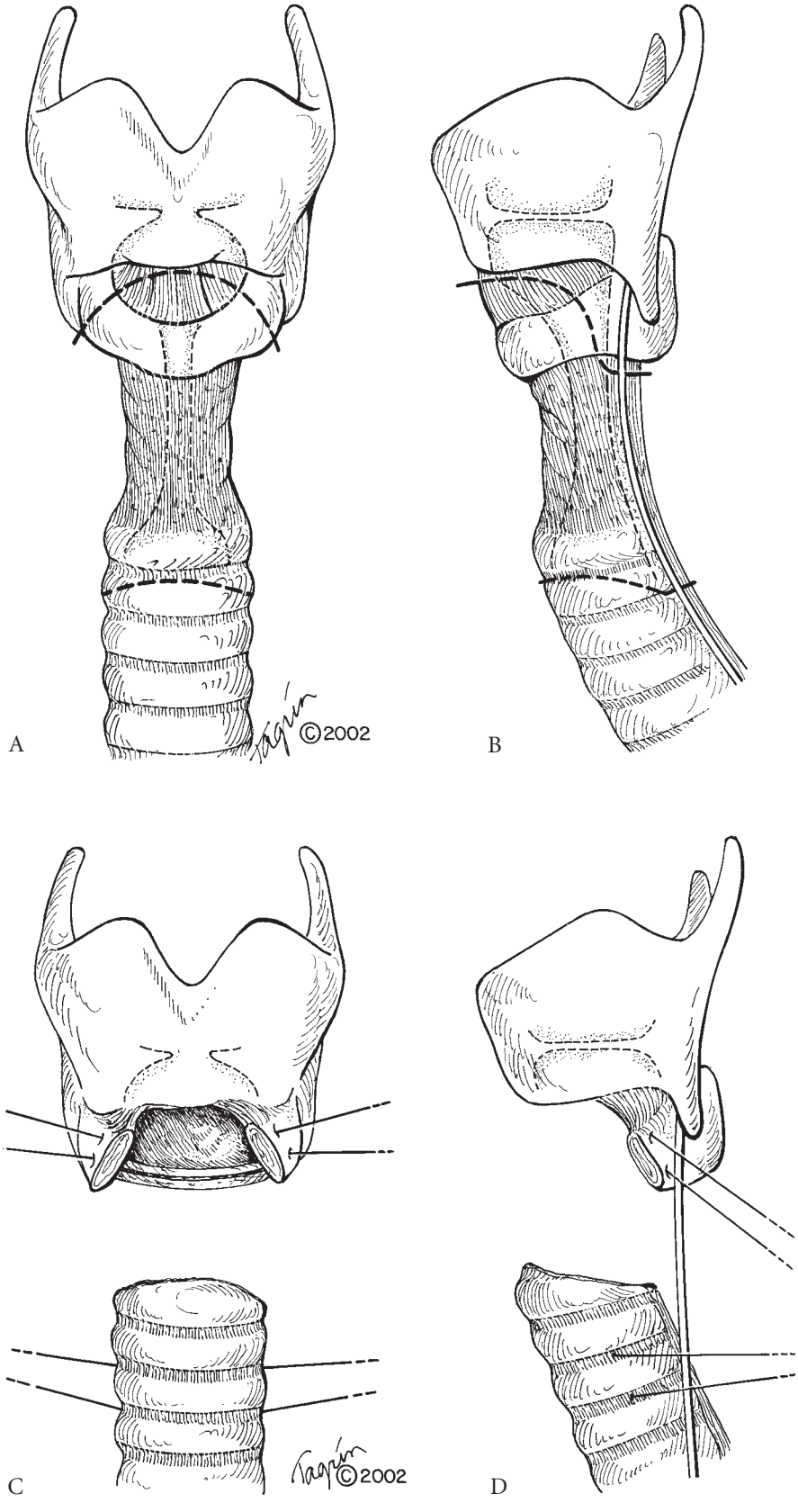


FIGURE 25-2 *Anterolateral stenosis involving the subglottic larynx most often results from high or erosive tracheostomy or from cricothyroidostomy.* A, *The anterior cricoid arch and cricothyroid membrane, which overlies the stenosis, is excised with the stenosis in an arcuate line. The first intact cartilage below the stenosis is trimmed in similar contour.* B, *Lateral lines of resection. The posterior plate of the cricoid and its posterior perichondrium is preserved. The recurrent inferior laryngeal nerves* (illustrated) *are not dissected, but allowed to fall laterally. Only the inferior rim of the posterior cricoid cartilage is dissected.* C, D, *Stay sutures (2-0 Vicryl) are placed laterally in the substance of the remaining lateral laminae of cricoid, but do not penetrate the laryngeal mucosa. Corresponding tracheal stay sutures encircle a ring at least one cartilage distal to the most proximal complete ring, and do include mucosa. Cricoid and tracheal stay sutures are located at corresponding points in the circumference of the airway.* Dotted lines *outline vocal cords. For clarity, surrounding tissues are not illustrated, but minimal trachea is dissected circumferentially in order to preserve blood supply. Often, only a small space is opened in the lateral attachments of the trachea in order to place the stay sutures.*

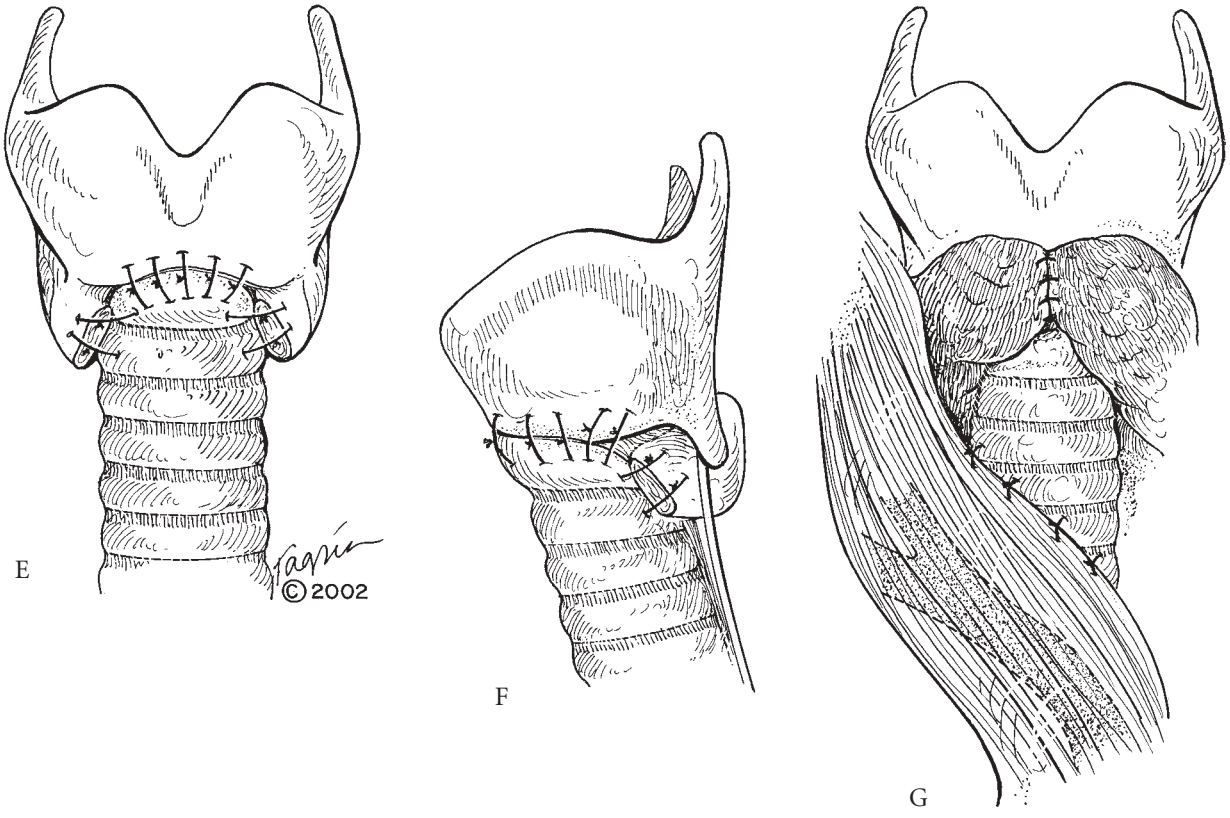


FIGURE **25-2** (CONTINUED) E, F, *Completed anastomosis. Anastomotic sutures are placed as described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection," for tracheotracheal anastomosis. Posteriorly, because of the thickness of the cricoid plate, sutures often traverse only part of the cartilage's thickness, but enter the airway lumen through full thickness of mucous membrane. Stay sutures are approximated first, as described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection." They are omitted in the illustration for clarity. Some overriding of the cut edges of lateral cricoid laminae and trachea often occurs.* G, *The anastomosis is usually covered with reapproximated thyroid isthmus or strap muscles, which are sutured together over it and to the larynx and trachea above and below the anastomosis. The right sternohyoid muscle may be sutured to the trachea over the brachiocephalic artery, as shown, for its protection. The site of a potential tracheostomy in the triangle between the covered anastomosis and the artery will be marked with a single fine suture.*

whom only a short length of resection is required. Where a longer segment is to be resected, the surgeon must remember that nearly a centimeter of trachea will be needed to repair the defect in the anterior larynx, in addition to the length of trachea resected. Infrequently, a laryngeal release may be required, with its potential attendant difficulties.

Circumferential dissection is effected just below the lesion in the usual manner and tracheal transection is performed. The trachea is initially divided transversely, with oblique bevelling of the cartilage done later. Midlateral traction sutures of 2-0 Vicryl are placed in the distal segment and the distal trachea is then intubated (see Figures 25-2*C*, *D*).

The stenotic segment is grasped laterally with two Allis forceps, as in upper tracheal resection (see Figure 24-8*A* in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"), and the trachea is carefully dissected cephalad to the inferior margin of the cricoid cartilage laterally and posteriorly. This dissection must be done painstakingly, adjacent to the specimen. The surgical scissors will be working within millimeters of the recurrent laryngeal nerves. No attempt should be made to identify or dissect out the nerves. If the principles of dissection described are rigorously followed, damage to recurrent laryngeal nerves

will be very rare and is usually transient if it should occur. It is important during dissection to palpate frequently and not carry posterior dissection above the lower border of the posterior cricoid plate. Such dissection could endanger the recurrent laryngeal nerves at their point of entry into the larynx posteriorly.

In commencing the anterior cricoid resection, the cricothyroid muscles are dissected off their attachment to the anterior and lateral cricoid cartilage. The line of resection of the lower larynx commences in the midline anteriorly, usually just below the thyroid cartilage. It sweeps laterally beneath the border of the thyroid cartilage, through the cricothyroid membrane obliquely inferiorly and posteriorly, dividing the lateral laminae of the cricoid cartilage (see Figures 25-2*C*,*D*). If the lateral laminae of the cricoid cartilage are severely deformed by the pathological process, the division will be more posterior, closer to the posterior cricoid plate. Great care is necessary at this point, because of the proximity of the recurrent laryngeal nerves. The posterior line of division of the larynx is at the inferior margin of the posterior cricoid plate.

Should the proximal airway appear smaller than expected, a small right-angle clamp is inserted into the residual lower larynx beneath the vocal cords and the tip drawn downward along the anterior wall of the larynx to discover whether a "shelf" is present. At times, a very small amount of additional excision of a rim of anterior tissue will very much enlarge the airway, at what will become the anastomotic level. Lateral midline traction sutures (2-0 Vicryl) are placed in the larynx, being certain that these pass through cartilage substantially, so that they will not pull out during approximation of the airway (see Figures 25-2*C*,*D*). The sutures usually lie in the residual lateral cricoid laminae just behind the line of their transection, but they may sometimes be partly in the thyroid cartilage. They do not enter the laryngeal lumen (see Figure 24-9 in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). In placing these sutures, it must be noted that the direction of the needle cannot be changed once it enters the cartilage. Therefore, the angle of placement must be carefully directed initially.

The distal trachea is tailored next, as previously described, preparing an anterior "prow" of tracheal cartilage, which will be drawn into the laryngeal defect to restore this portion of the airway wall (see Figures 25-2*A*–*D*). In case of a lengthy resection, it may be necessary to accept some slight degree of scarring or inflammation in the midline of the tracheal ring, which will be shaped for anastomosis, but its cartilaginous structure must be acceptably intact. Preparation of the trachea for anastomosis is deferred until after laryngeal division, in order to establish with certainty that a posterior flap of membranous trachea will not be needed to resurface the posterior cricoid plate.

Tentative approximation of the airway (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection") demonstrates the degree of tension that will be placed on the anastomosis. The anastomosis is done with great care, using the basic technique described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection" and the same 4-0 Vicryl sutures. All sutures are placed before any are tied. Since both laryngeal and tracheal ends are obliquely divided and disparate in size, sutures must be placed proportionately, using the midlateral sutures as guide points as well as anterior and posterior midpoints. Since mucosal approximation is sought, sutures may in some cases pass through a partial thickness of laryngeal cartilage above, but it must always pass through mucosa of the larynx, into or from the lumen, in order to assure approximation with tracheal mucosa. The purchase on cartilage must be secure, however. In the presence of dense scarring or calcification, one or more anterior "keystone" sutures of heavier Vicryl (usually 3-0 and rarely 2-0) may help the anastomotic approximation. Dense calcification is dealt with as described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection," and may necessitate use of a fine dental drill. In placing the anterior sutures, it helps to place the midline suture first, halfway between the lateral traction sutures. This makes it easier to space the remaining anterior sutures proportionally on each side.

After placement of all sutures and prior to final flexion of the neck, a catheter is passed from the operative field into the pharynx. It is retrieved by the anesthetist, who draws it out through the mouth and sutures

to it an endotracheal tube of a caliber that will pass atraumatically through the smaller than normal airway (see Figure 24-11 in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). This tube is pulled into the field and passed into the distal trachea where it replaces the cross-field tube. The patient's neck is placed in flexion, the lateral traction sutures are tied, followed by the anastomotic sutures (Figures 25-2*E*,*F*). Because of the disparity in tissues and, frequently, their rigidity, the anterior sutures are often best tied by starting first with the most lateral sutures on either side, and then gradually working toward the center where the greatest tension is. The use of heavier central sutures helps in such cases. The posterior line of sutures is tied last, with half being tied from one side and half from the other side (see Figure 24-15 in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection").

Since some of these patients have, at best, 50% of normal subglottic laryngeal cross-sectional area just below the vocal cords, and since some intraoperative laryngeal trauma is unavoidable, the possibility of early postoperative laryngeal edema is real. Initially, I routinely placed a small tracheostomy tube distal to the anastomosis, but it became clear that few patients required this.[9] The anastomosis is covered with tissue to seal it from a potential later tracheostomy. The thyroid isthmus may serve this purpose in some cases. More often, the strap muscles are sutured together in the midline and to the airway above and below the anastomosis, sealing it off. Since the trachea is much shortened in many patients, the site of a potential tracheostomy, which should be placed at least two rings below the laryngotracheal anastomosis, might lie dangerously close to the brachiocephalic artery below. The brachiocephalic artery is therefore compartmentalized, by suturing the right sternothyroid muscle obliquely across the trachea, placing the sutures just behind the artery to the tracheal wall. The muscle will conform appropriately without being pedicled. This leaves a triangle of anterior tracheal wall (Figure 25-2*G*), which lies below the protected anastomosis and above the obliquity of the course of the protected brachiocephalic artery. A 4-0 silk suture is placed to mark the point of election for tracheostomy, should it become necessary later.

The endotracheal tube is removed in the operating room as the patient awakens. If the airway appears adequate, the patient is returned to the intensive care unit breathing spontaneously. If the patient is stridorous, the glottis is examined directly, using either a rigid laryngoscope (without extending the neck) or a flexible bronchoscope. The glottis can usually be examined quite satisfactorily without extending the neck, even by laryngoscopy. If vocal cord paralysis is seen (a rare occurrence unless present preoperatively), or if laryngeal edema is present, a small bore endotracheal tube, usually uncuffed, is passed, either nasotracheally or orotracheally. The nasotracheal route is preferable, if it can be done without excessive extension and traction on the suture line (most easily over a pediatric flexible bronchoscope). Patients are informed preoperatively of this possibility. The tube is removed 4 or 5 days later in the operating room under general anesthesia, and if the airway is still not adequate, a small sized tracheostomy tube is inserted at the premarked site. Tissue planes over the anastomosis and artery are well sealed by this time. The possibility of damage to either of these critical areas is minimized.

Circumferential Subglottic Stenosis

In these more difficult cases, the stenosis is circumferential and overlies the posterior cricoid plate as well (Figure 25-3*A*). The mucosa is involved with inflammation and scar or it has been destroyed. Idiopathic stenoses are always circumferential, as are most postintubation stenoses from endotracheal tubes as well as those from certain miscellaneous causes such as Wegener's disease or trauma.[9,14,15] Exposure, dissection, and initial tracheal division are performed exactly as described for anterolateral subglottic lesions. The anterior and lateral laryngeal division is also executed in an identical fashion. Most often, the tracheal specimen is initially resected by dividing posteriorly along the lower border of the posterior cricoid plate (see Figure 25-3*A*). Alternatively, the specimen remains attached posteriorly, and the final separation of the stenosis is commenced higher on the posterior cricoid plate, as described below.

We are left at this point with the anterior cricoid arch (and cricothyroid membrane) removed, the lateral cricoid laminae obliquely divided, and the stenotic distal tracheal segment removed, but with a ring of posterior shelf-like scar remaining on the anterior surface of the posterior cricoid plate. This stenosis is excised by making a transverse cut against the posterior plate of the cricoid cartilage inferior to the vocal cords and arytenoid cartilages, at a level dictated by the stenotic process (Figure 25-3*B*). The scarred mucosa and ridge of stenotic scar are excised sharply from the anterior surface of the posterior cricoid plate using a scalpel with a no. 15 blade or a small Beaver blade, leaving the denuded posterior plate of the cartilage as intact as possible. There is usually no reason to remove the posterior cricoid plate completely. Removal could endanger the recurrent nerves. Varying degrees of deformity and destruction of the posterior cartilage will be encountered, requiring surgical ingenuity. An absolute requirement is preservation of the posterior perichondrium.

The distal trachea is prepared differently from the case of anterior stenosis. The tracheal cartilage anteriorly and laterally is trimmed as before, sloping the line of division backward on either side over the width of only one ring. The membranous wall, however, is formed into a broad-based flap with the superior corners slightly curved. This full thickness flap of healthy tissue has excellent blood supply (see Figure 25-3*B*).

The anastomosis is more complicated, as might be expected. The goal is to resurface the bared posterior cricoid plate with the tracheal membranous wall flap, uniting it with the mucosa of the posterior larynx above (Figures 25-3*C*,*D*). The "prow" of cartilage will be sutured into the oblique defect in the anterior and lateral larynx, as previously described. To insure apposition of the membranous tracheal flap to the posterior plate of larynx, four nonabsorbable 4-0 sutures (usually Tevdek) are first placed from the inferior margin of the posterior cricoid plate to a line that runs across the *back* of the membranous wall from the lowest level of the flap on either side (Figures 25-4*A*,*B*). Two inner sutures are placed first, neatly dividing the posterior plate of cricoid into thirds. The two lateral sutures are next placed from the lateral corners of the posterior plate of cartilage to the lateral corners of the tissue just below the origin of the membranous wall flap, close to the cartilaginous junction of the trachea. These sutures do not penetrate the mucosa. The inner two Tevdek sutures are clipped to the drapes on either side, and the lateral sutures are clipped to the drapes caudad to the first. Since these are of different material, they are easily distinguishable from later anastomotic sutures of Vicryl. They will be tied in the same order as described for tracheal anastomosis, namely, the most lateral sutures will be tied first and the medial ones tied second (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection").

A series of 4-0 Vicryl mucosal sutures are next placed from the posterior laryngeal mucosa and submucosa to the membranous wall flap of trachea. These sutures are placed from within the larynx, although they are passed from the posterior wall of the laryngeal or tracheal mucosa into the lumen, and then from the lumen through the opposite mucosa to emerge posteriorly (see Figures 25-4*B*,*C*). The central suture is most conveniently placed first, then working laterally to either side. All sutures are clipped serially to the drapes over the patient's face. The endotracheal tube in the distal trachea is intermittently removed to facilitate placement and tying of sutures.

After placing the necessary number of sutures to anastomose the posterior flap to the mucosa within the larynx, the first of the "standard" anastomotic sutures are placed on either side, moving from posterior to anterior, and pausing at the level of the lateral traction sutures. This usually requires two or three sutures on each side. Each suture is passed through cartilage of the lateral laminae of cricoid or of the cut edge of the lateral lamina and edge of laryngeal mucosa above, and thence through the full thickness of mucosa and cartilage of the trachea below. These sutures are placed from posterior to anterior and tagged in the usual manner from cephalad (most posterior suture) to caudad (anterior sutures) on the drapes, usually above the Tevdek fixation sutures, which were initially placed. The next one or two sutures anterior to the traction sutures are also placed on each side at this time, since it will be more difficult to insert these after airway approximation.

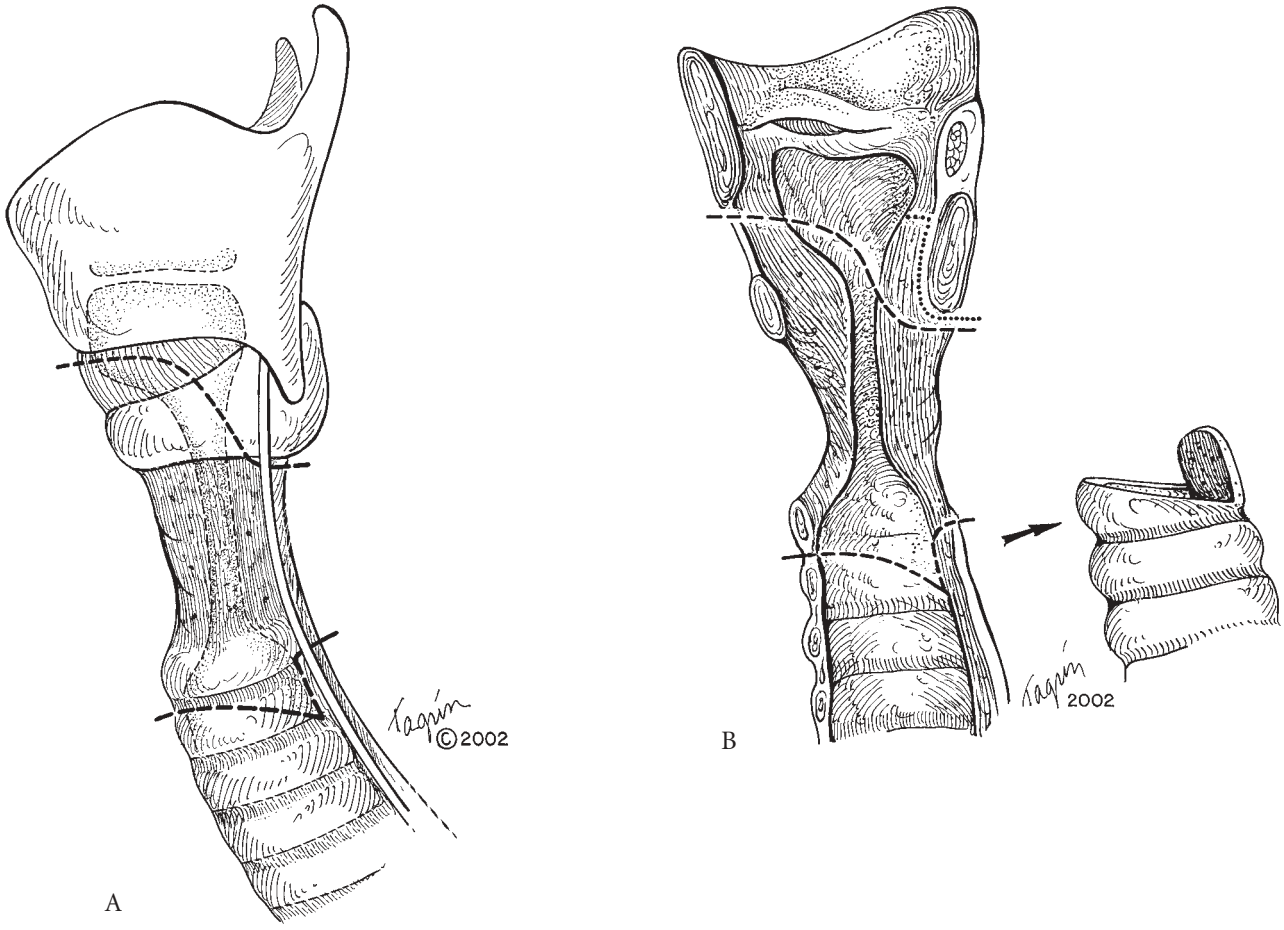


FIGURE **25-3** *Circumferential stenosis involving subglottic larynx results from endotracheal tube injury, idiopathic stenosis, trauma, and other causes.* A, *The initial* (dashed) *line of inferior laryngeal resection is the same as for anterior stenosis. The line* (dashed) *of tracheal division is also the same anterolaterally, but posteriorly, a flap of membranous wall is preserved. If a longer posterior flap is necessary, then the line of cartilage division may be dropped one ring. The "prow" of tracheal cartilage is limited to one ring, even if the membranous flap is longer, in order to avoid flaccidity after anastomosis. The* dotted lines *indicate residual lumen and position of vocal cords. Note the circumferential involvement and extent of the stenosis over the anterior surface of the posterior cricoid plate.* B, *Internal view of the resection.* Dashed lines *show the level of the laryngeal and tracheal transection.* Dotted line *indicates the line of resection of the stenotic scar from the anterior surface of the cricoid plate. Irregular, damaged cartilage is also carved away, preserving a stable shell of cartilage with its posterior perichondrium. A posterior flap of membranous tracheal wall is preserved distally. Its corners are carefully rounded.*

The inflatable "thyroid bag" beneath the patient's shoulders is deflated, the neck propped in partial flexion, and the lateral traction sutures are tied to remove tension from the anastomosis about to be commenced. The four posterior Tevdek sutures are next tied, to fix the membranous flap to the inferior margin of the cricoid cartilage plate posteriorly. The long ends of each suture are cut after tying. The internal sutures, which will anastomose the membranous flap to the posterior mucosa of the larynx below the arytenoids, are next tied with great care, to avoid tearing sutures out of the laryngeal mucosa. The tip of the finger may usually be inserted easily into the still open anterior larynx to tie these sutures. The endotracheal tube is removed when each suture is tied. If there is a gap or if one of the sutures should cut out of the mucosa of the larynx, because of inflammatory changes or excessive tension, another suture may be carefully placed. The integrity of this suture line is inspected directly before proceeding. Lateral anastomotic sutures posterior to the traction sutures are tied next. The one or two sutures already placed anterior to the traction sutures are not yet tied.

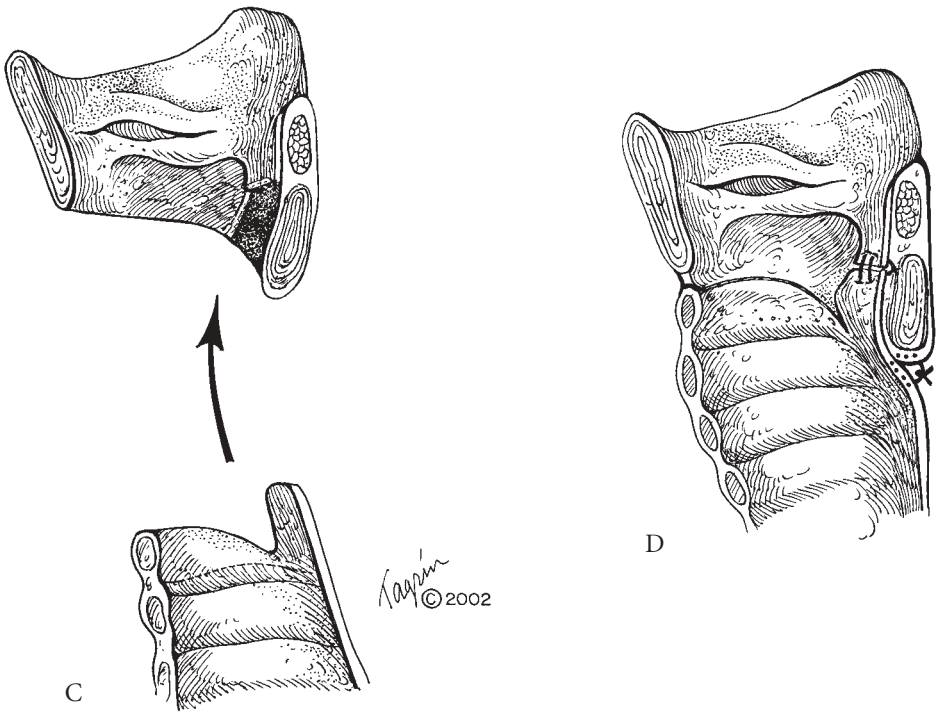
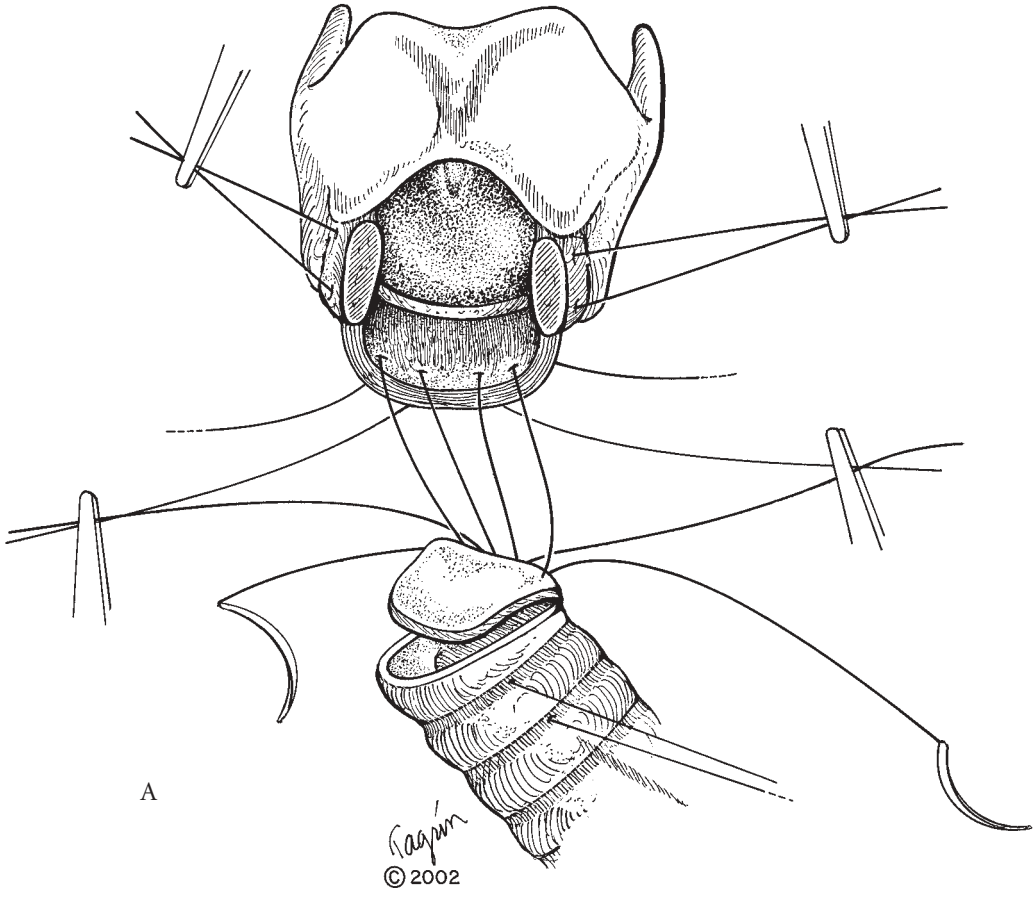


FIGURE 25-3 (CONTINUED) C, *The larynx and trachea are prepared for anastomosis. The bared inner surface of the cricoid will be covered by advancement of the posterior tracheal flap. The curved tracheal cartilage will repair the anterior cricoid gap.* D, *The reconstituted upper airway. For clarity, only two posterior mucosal sutures are shown. Their knots lie behind the mucosa. One "fixing" suture is also shown between the outer layer of the base of the membranous wall flap and the inferior margin of the posterior cricoid.*

The balance of the anterior anastomotic sutures are placed next (Figure 25-4*D*). It is helpful to place an anterior midline suture initially, in order to simplify proportional placement of the lateral anterior sutures. Also, the midline suture may be of a size 3-0, or even 2-0, in order to facilitate approximation, when structures are rigid. The central suture often passes through the inferior margin of the thyroid cartilage in the midline. A catheter is passed upward, as previously described, and an appropriate endotracheal tube is slipped distally to replace the cross-field tube. The remaining "standard" anastomotic sutures are tied from lateral to anterior on each side. The order of anastomotic steps is summarized in Table 25-1.

Repair of laryngotracheal lesions is demanding and should not be undertaken until the surgeon has extensive and successful experience in tracheal resection and reconstruction as well as knowledge of laryngeal anatomy. Results justify appropriate application of these methods, despite some variations in technique (see Chapter 9, "Tracheal and Bronchial Trauma," Chapter 11, "Postintubation Stenosis," and Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions").[7,9,11,13] Pearson and colleagues alternatively curetted a groove in the inferior portion of the posterior cricoid plate.[5] The distal trachea was transected horizontally and the two cartilaginous ends sutured together, imbricating the membranous wall. This narrowed opening was fitted into the groove created in the posterior laryngeal cartilage. Pearson, together with Maddaus and colleagues, subsequently adopted the posterior flap for lesions extending far up on the posterior cricoid plate.[13] Anastomoses were splinted postoperatively with a T tube. Their results have been good, as have been those by Couraud and colleagues using the same procedure, including postoperative stenting.[7,13] As might be anticipated, however, results for laryngeal anastomoses generally do not wholly equal those obtained in the simpler reconstructions for tracheal stenosis.



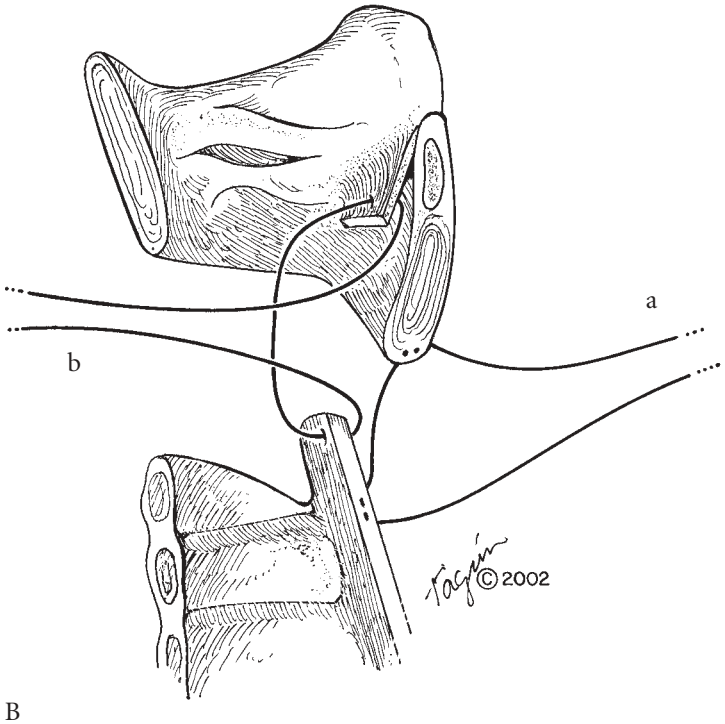
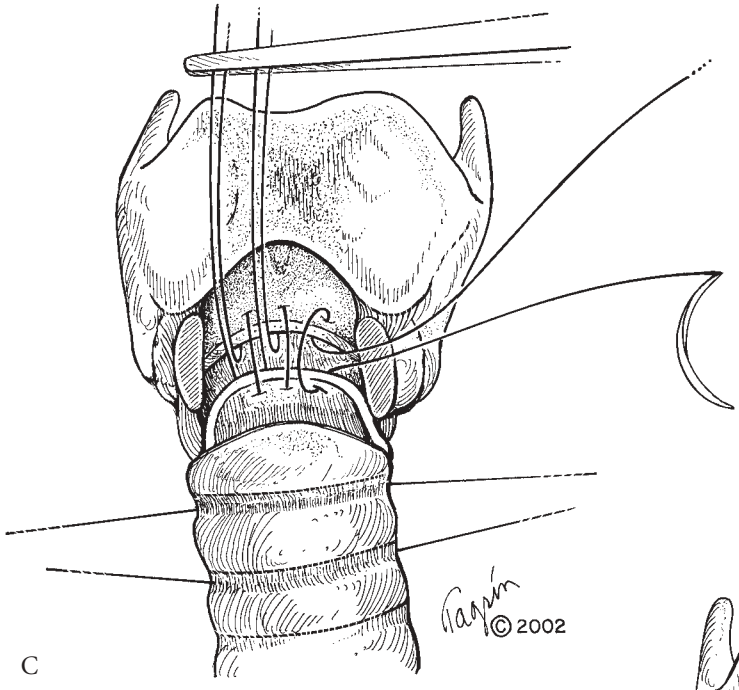


FIGURE **25-4** *Laryngotracheal anastomosis after resection of circumferential stenosis, which involved the subglottic larynx.* A, *The base of the membranous wall flap is fixed to the inferior margin of the posterior cricoid plate with four sutures of nonabsorbable 4-0 material (such as Tevdek). These sutures do not penetrate the mucosa. Vicryl (2-0) stay sutures have been placed in the cricoid and trachea, located to correspond to each other when the larynx and trachea are drawn together. Scar and damaged mucosa have been excised from the posterior cricoid. The margin of remaining laryngeal mucosa is visible.* B, *Detail of suture placement.* (a) *Suture fixing base of the posterior flap to the inferior cricoid margin.* (b) *Posterior mucosal anastomotic suture placed so that the knot will lie beneath the mucosa when tied.*



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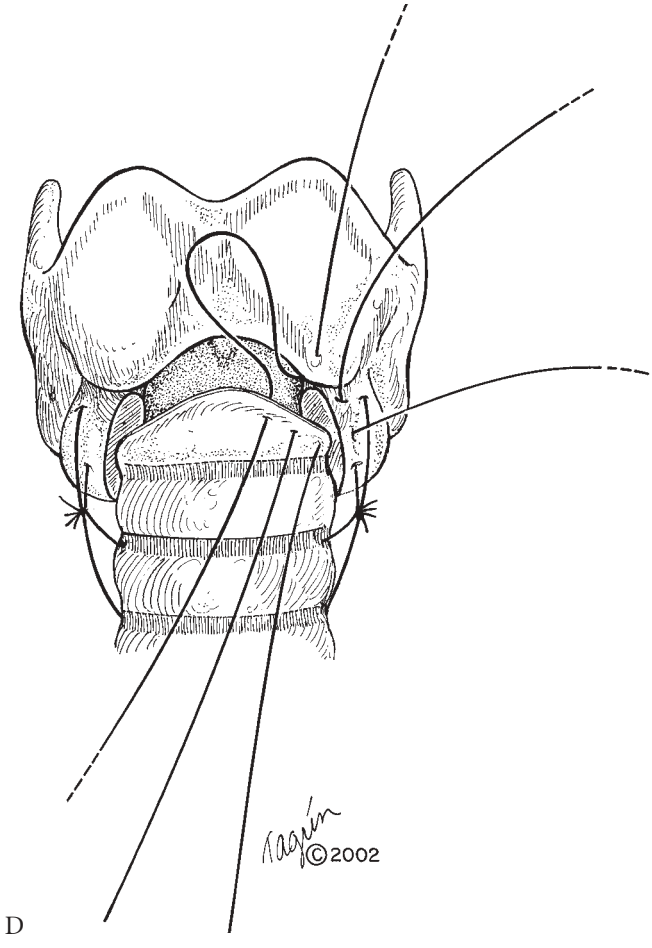

D

FIGURE **25-4** (CONTINUED) C, *Posterior mucosal anastomotic sutures are placed serially from both sides of the midline up to the edges of the flap. The first "conventional" anastomotic sutures, placed as previously described for resection of an anterior stenosis, are also placed on both sides between the cricoid and cartilaginous tracheal rings up to one to two sutures anterior to the traction sutures.* D, *Lateral traction sutures have been tied, holding the larynx and trachea in approximation. The four posterior "fixing" sutures are tied next, followed by the posterior mucosal sutures. The remainder of the anterior anastomotic sutures are now placed. The patient is reintubated translaryngeally and the anastomosis completed, tying the anterior sutures from both sides gradually toward the anterior midpoint.*

Neoplasms Involving the Lower Larynx and the Trachea

When tumors occur at the junction between the lower larynx and the trachea and involve both organs, a conservative laryngeal sparing approach seems appropriate. Benign neoplasms such as granular cell tumor, chondroma, and paraganglioma can be effectively removed in conservative fashion, with a portion of larynx, accepting a narrow margin of normal tissue around the tumor. Some malignant tumors, especially of limited aggressiveness, may be similarly removed, but patients must be closely followed. Reconstruction varies uniquely with size and location of the tumor.[16]

Table 25-1 Laryngotracheal Anastomosis with Posterior Mucosal Flap: Order of Anastomosis

1.	Place midlateral traction sutures (2-0 Vicryl) deeply in the lamina of the cricoid, above and through the wall of the trachea below (see Figure 25-4*A*).
2.	Place four fixation sutures (4-0 Tevdek) from the inferior margin of the posterior cricoid plate to the base of the membranous tracheal flap. Sutures do not penetrate the mucosa (see Figures 25-4*A*,*B*).
3.	Place anastomotic sutures (4-0 Vicryl) between the posterior mucosa of the larynx (over the upper posterior cricoid plate) and the membranous wall flap of the trachea. Knots will lie beneath the mucosa (see Figures 24-4*B*,*C*).
4.	Place anastomotic sutures (4-0 Vicryl) from the lateral lamina of the cricoid and laryngeal mucosa to the full thickness of the lateral tracheal wall, *posterior* to the midlateral traction sutures.
5.	Place one or two additional anastomotic sutures on both sides, *anterior* to the midlateral traction sutures.
6.	Flex the neck as necessary. Tie traction sutures, approximating the airway posteriorly and laterally.
7.	Tie Tevdek fixation sutures posteriorly. Cut off excess of each suture as tied.
8.	Tie Vicryl posterior mucosal flap anastomotic sutures inside the larynx.
9.	Tie lateral anastomotic sutures *posterior* to the traction sutures.
10.	Place anterior anastomotic sutures (4-0 Vicryl, with occasional midanterior 3-0 or 2-0 Vicryl suture, as needed) (see Figure 25-4*D*).
11.	Advance the endotracheal tube from the larynx into the trachea.
12.	Tie all sutures anterior to the lateral traction sutures, working from both sides toward the center. The surgical assistant crosses the next suture to assist in the approximation.

An *anteriorly located tumor* that involves the cricoid is removed by excision of the anterior subglottic larynx, employing individualized modifications of the technique of laryngotracheal resection described for resection of benign anterolateral subglottic stenosis (see Figure 25-2). For *posterior midline tumors*, a membranous tracheal wall flap is advanced for reconstruction. In some cases, the mucosa and submucosa overlying the posterior plate of the cricoid alone need to be resected (see Figure 7-14 in Chapter 7, "Primary Tracheal Neoplasms"). In others, various amounts and depths of cricoid must be resected to obtain suitable margins. If the tumor is limited enough to permit a larynx conserving operation, usually enough posterior cricoid cartilage remains below and between the arytenoids to serve as a bridge, which preserves laryngeal stability. When a posterior laryngeal resection alone is necessary, adequate exposure for posterior mucosal anastomosis is obtained by vertical midline division of the cricoid, essentially a limited laryngofissure (Figure 25-5). An example of excision of a posterior centrally located tumor is shown in Figures 25-5*A*–*F*. The technical steps are explained in the legends.

In other cases with *unilateral involvement*, an appropriate margin is obtained by resection of portions of the lower larynx, tailoring the trachea to fit the laryngeal gap created.[16,17] A major application of such tailoring procedures, following partial resection of the lower larynx for the tumor, has been for differentiated carcinomas of the thyroid (follicular, papillary, and mixed types).[17,18] Undifferentiated thyroid cancer most often, but not universally, invades too aggressively for conservative surgery to be applied. If any surgical treatment is applicable in these patients, en bloc resection of larynx and trachea may be required (see Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy"). For differentiated thyroid cancer, for benign tumors, or for tumors of lesser malignancy, the objective is to remove the local tumor and prevent airway obstruction, while preserving laryngeal function. This is achieved by carefully circumscribing the tumor,

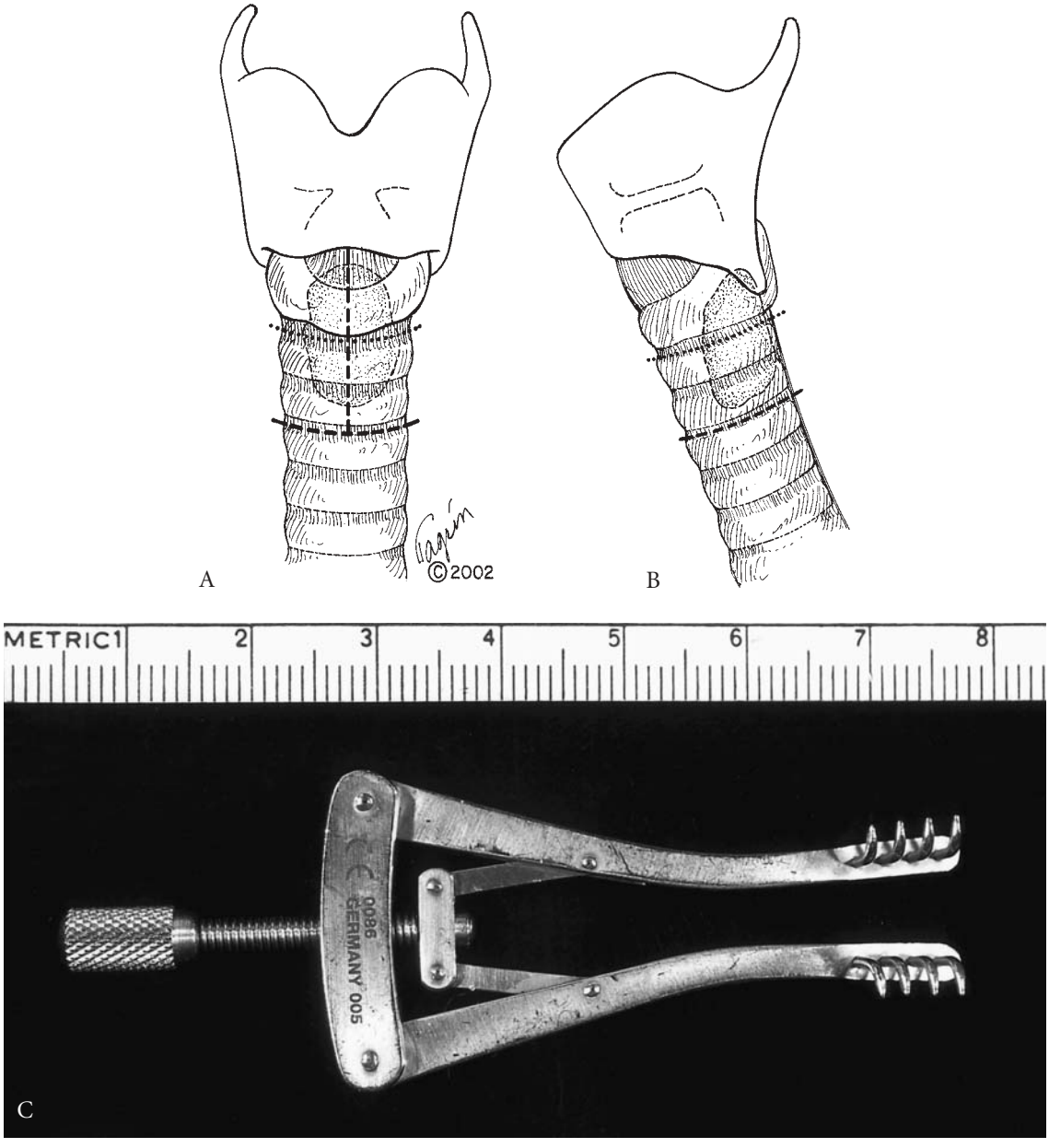

FIGURE **25-5** *Excision of a central posterior tumor involving the subglottic larynx and trachea.* A, *The trachea is transected at an appropriate level below the tumor* (horizontal dashed line). *A midline vertical incision divides the anterior wall of the subcricoid segment of the trachea and continues up as a limited laryngofissure across the midpoint of the cricoid cartilage and the cricothyroid membrane to the thyroid cartilage. Exposure is further improved by dividing the trachea from beneath the cricoid horizontally on either side* (dotted line)*, working posteriorly toward the tumor. The glottic level is also indicated.* B, *Lateral view showing the level of tumor and lines of incision. The recurrent nerves are carefully spared by keeping dissection close to the tracheal wall and not dissecting out the nerves.* C, *The Alm expandable retractor facilitates anterior exposure through the laryngofissure.*

with a sufficient margin of subglottic larynx so that the tumor is locally excised (Figure 25-6). The larynx on the side opposite from the tumor is transected horizontally beneath the lower border of the remaining cricoid cartilage, very carefully preserving that recurrent laryngeal nerve. If a recurrent laryngeal nerve must be excised, it is usually one that has already been paralyzed by tumor invasion or is inseparable from the tumor. Distally,

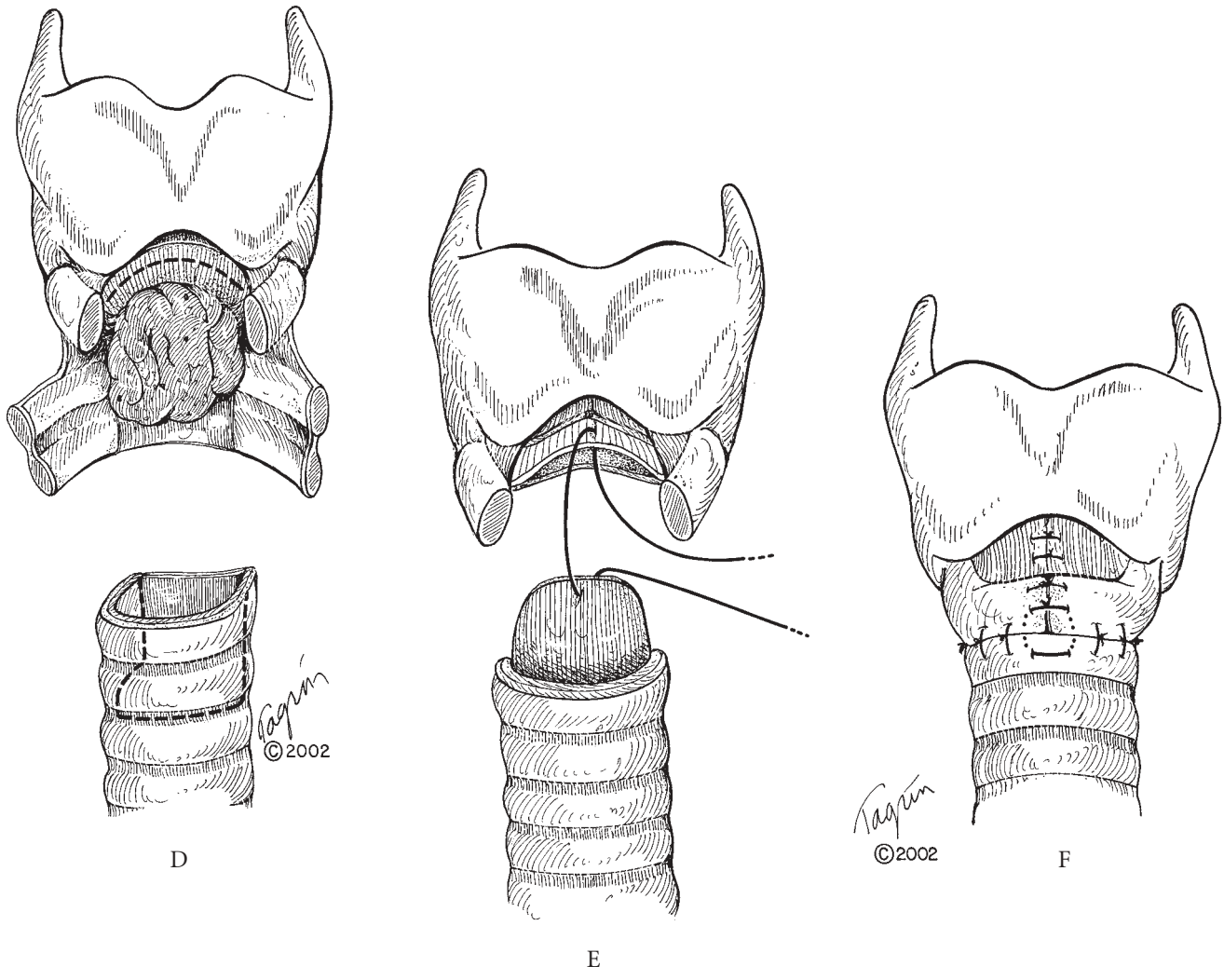

FIGURE **25-5** (CONTINUED) D, *The tumor and posterior subglottic larynx are exposed, permitting resection to continue. The posterior mucosal division superior to the tumor is indicated by the* dashed line. *This will be connected laterally to the line of resection on either side just below the cricoid. A portion of posterior cricoid cartilage is excised, if involved by tumor. Laryngeal stability will be maintained if a bridge of posterior cricoid remains above the level of cartilage excision. In order to fashion a posterior membranous tracheal flap for reconstruction of the defect, even if resection included cartilage, additional tracheal rings are resected, as outlined in the trachea below* (dashed line). *An estimate of the total number of rings that will be removed should be made at the outset. Resection of cartilages should be performed conservatively and, if necessary, in stages in order not to shorten the trachea unnecessarily. The corners of the flap are shaped as needed.* E, *Reconstruction is commenced by anastomosis of the posterior mucosa and submucosa of the larynx to the posterior tracheal flap. Sutures are placed so that knots will lie submucosally. If a wide expanse of cartilage of the posterior cricoid plate is exposed below the cut edge of the posterior laryngeal mucosa and submucosa, it is sometimes advisable to fix the back of the flap to the lower edge of the cartilage, as shown in Figures 25-4A and 25-4B. The balance of the anastomosis will be of trachea (cartilage and mucosa together) to residual anterior and lateral cricoid, with its underlying laryngeal mucosa.* F, *After placement of all circumferential anastomotic sutures, the vertical cricothyroid membrane incision is sutured closed. A mattress suture is placed at the junction of the inverted T suture lines. The anastomosis is then completed. The usual midlateral Vicryl stay sutures (in the lateral cricoid laminae and in the trachea below) are omitted in the drawing for clarity, but are necessary.*

the trachea is shaped after laryngeal excision, in such fashion that it will mortise into the defect created by the excision (see Figure 25-6). Suture lines are irregular and unique to each case, and often "bayonet" shaped. On occasion, a partial or even full thickness of esophageal wall must be resected to obtain a posterior margin (see Figure 25-6*E*). Generally, the length of resection is not so great that a problem of anastomotic tension results.

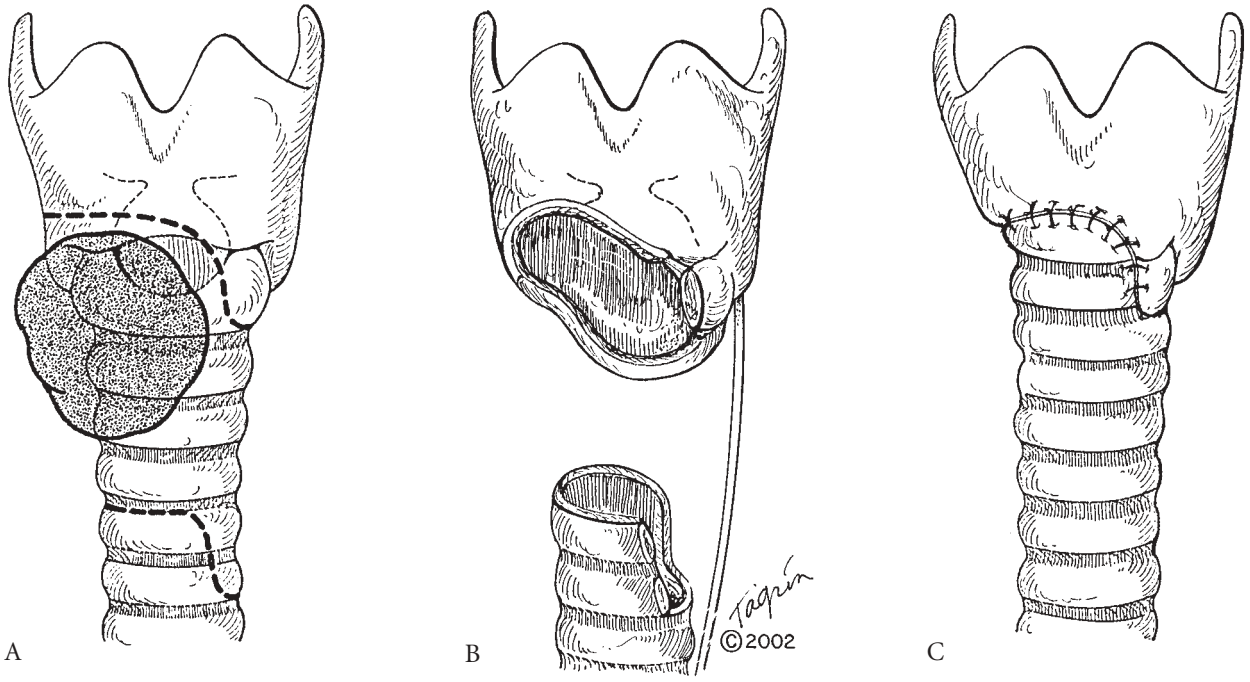

FIGURE 25-6 *Lateral laryngotracheal excision for tumor, such as differentiated thyroid carcinoma or low-grade chondrosarcoma.* A, *Initial tracheal division is made horizontally at an appropriate level below the tumor. The extent of tailoring which is necessary for reconstruction is not known at the outset. The* dashed line *represents the final tracheoplasty incision, which is completed later. Transection is made at the level of the horizontal tracheal incision line closest to the tumor. The* dashed line *of laryngeal excision above circumscribes the portion of larynx invaded by tumor. The laryngeal excision is commenced by incising horizontally beneath the cricoid, opposite to the side of tumor, to visualize the edge of the tumor intralaryngeally. If this does not provide clear visualization, then the tracheal segment, already divided transversely below the tumor, may be opened vertically up to the uninvolved cricoid, and the extent of the tumor in the larynx can then be observed directly. As the line of excision advances, the extent of tumor becomes increasingly evident.* B, *The anterolateral laryngeal defect will be repaired with trachea, fashioned by removing segments of tracheal rings on the side opposite to the laryngeal excision. Since the circumference of laryngeal defect is likely to be greater in length, only a lesser segment of trachea is removed, allowing cartilage to spread somewhat. Slight rotation of the trachea prior to anastomosis is permissible, further sparing cartilage in order to provide stability. The recurrent nerve has been carefully spared on the uninvolved side. The nerve is not dissected out. Instead, dissection is kept close to the larynx and trachea on this uninvolved side, with no more exposure of the inferior cricoid margin than is absolutely necessary.* C, *Anastomosis is accomplished in the usual way, using 4-0 Vicryl sutures and 2-0 lateral stay sutures.*

Particular aspects of the technique applicable to resection of laterally located tumors are noted in the legends for Figure 25-6. Since the tissue beyond the tumor is normal, the difficulty caused by residual inflammatory thickening and narrowing, which is seen in similar surgery for inflammatory strictures, is not a factor. Repair following laryngotracheoplasty for tumor is therefore more likely to succeed without danger of a stenosis. Results of laryngotracheal resections for tumor and for inflammatory lesions are not comparable.

A conservative approach is applicable in a small number of patients with squamous cell carcinoma or adenoid cystic carcinoma of the uppermost trachea with impingement on the larynx.[19] On occasion, a microscopically positive margin is accepted in such cases, just as in the case of an invasive thyroid carcinoma, in order to save the larynx. Full-dose irradiation follows. In thyroid carcinoma, radioiodine may be indicated if uptake occurs. Chondroma or low-grade chondrosarcoma, most often arising from the posterior cricoid plate, may be similarly treated, conserving a functional larynx.[16,20] Other patients treated by conservative resection had mucoepidermoid and spindle cell tumors and pseudotumors. These patients must be followed in the long term for possible recurrence. Laryngotrachiectomy may later be possible for local recurrence, despite irradiation received (see Chapter 34, "Cervicomediastinal Exenteration and Mediastinal

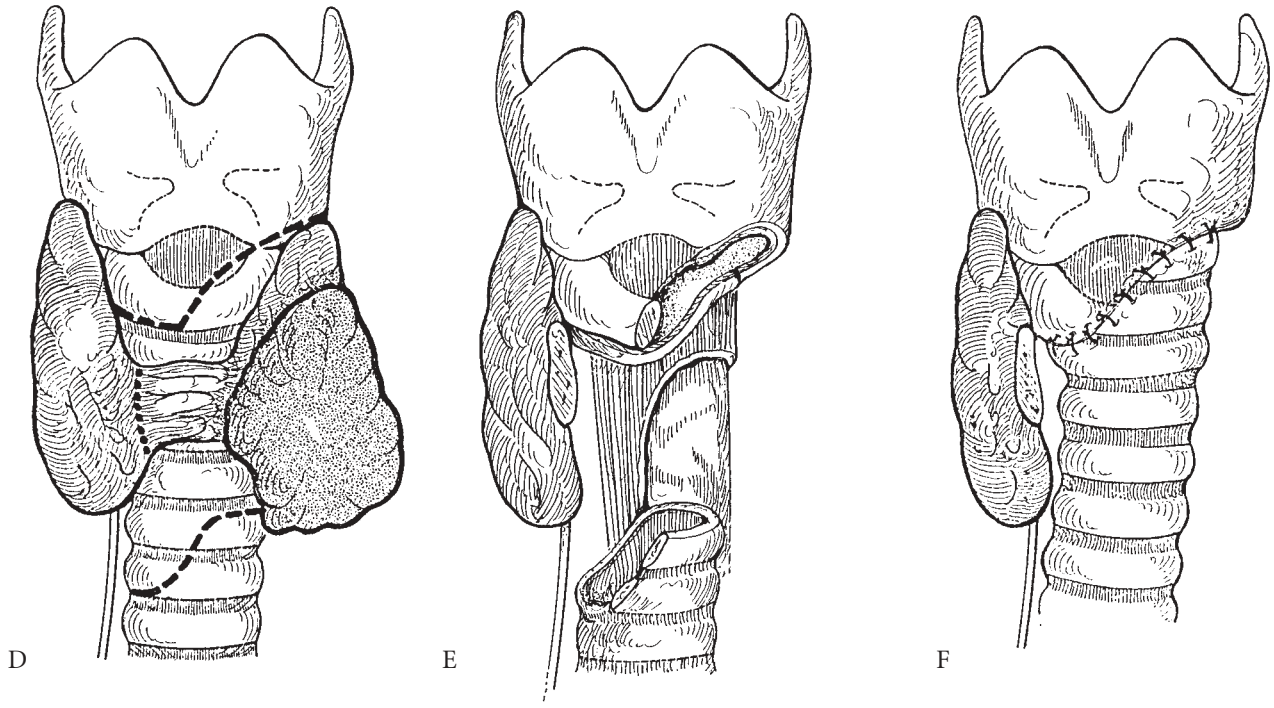

FIGURE **25-6** (CONTINUED) D, *In this patient, a differentiated thyroid carcinoma invades the larynx, trachea, and esophagus. The left recurrent nerve is paralyzed. A similar "bayonet" excision of airway is performed as in the previous illustrated case. I elected to save the uninvolved right hemithyroid, further ensuring safety of the right recurrent laryngeal nerve. The lobe, however, is dissected free from the trachea and larynx medially.* E, *Invasion of the esophagus was limited to the muscularis, and the mucosa was saved. The esophageal muscle is sutured with a single layer of interrupted 4-0 Vicryl or silk Lembert sutures. If the esophageal mucosa must also be resected due to invasion, it is closed with interrupted inverting sutures of 4-0 Vicryl or silk, and the muscularis is closed in a second layer, as described. A pedicled sternohyoid muscle is sutured over the esophageal repair prior to airway reconstruction, in order to prevent fistulization. At most, the esophagus may require one or more dilations postoperatively. In many cases, no dilation is needed.* F, *The completed laryngotracheal reconstruction.*

Tracheostomy"). Indeed, primary irradiation alone has been elected by some patients, where larynx sparing surgery was impossible. The use of the omentum in concert with resectional or reconstructive airway surgery is discussed in Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation." Patients often prefer to take risks in order to salvage laryngeal function for a considerable period of time, if not permanently. There is no way to estimate the hazard of metastasis from a residual but irradiated disease, in the interval.

References

1. Conley JJ. Reconstruction of the subglottic air passage. Ann Otol Rhinol Laryngol 1953;62:477–95.
2. Ogura JH, Powers WE. Functional restitution of traumatic stenosis of the larynx and pharynx. Laryngoscope 1964;74:1081–110.
3. Ogura JH, Biller HF. Reconstruction of the larynx following blunt trauma. Ann Otol Rhinol Laryngol 1971;80:492–506.
4. Gerwat J, Bryce DP. The management of subglottic laryngeal stenosis by resection and direct anastomosis. Laryngoscope 1974;84:940–57.
5. Pearson FG, Cooper JD, Nelems JM, Van Nostrand AWP. Primary tracheal anastomosis after resection of cricoid cartilage with preservation of recurrent laryngeal nerves. J Thorac Cardiovasc Surg 1975;70:806–15.
6. Couraud L, Martigue C, Hondelette P, et al. Intérét de la résection cricoidienne dans le traitement des sténoses crico-trachéales apres intubation. Ann Chir Thorac Cardiovasc 1979;33:242–6.
7. Couraud L, Hafez A, Velly JF, et al. Current reconstructive management of subglottic stenosis of the larynx with reference to sixty consecutively treated cases. J Thorac Cardiovasc Surg 1985;33:263–7.
8. Grillo HC. Reconstruction of airway after resection of subglottic laryngeal and upper tracheal stenosis. Ann Thorac Surg 1982;33:3–18.
9. Grillo HC, Mathisen DJ, Wain JC. Laryngotracheal resection and reconstruction for subglottic stenosis. Ann Thorac Surg 1992;53:54–63.
10. Monnier P, Savary M, Chapuis G. Partial cricoid resection

with primary tracheal anastomosis for subglottic stenosis in infants and children. Laryngoscope 1993;103:1273–83.
11. Monnier P, Savary M, Chapuis G. Cricotracheal resection for pediatric subglottic stenosis: update of the Lausanne experience. Acta Oto-rhino-laryngol Belg 1995; 49:373–82.
12. Cotton RT. The problem of pediatric laryngotracheal stenosis. Laryngoscope 1991;101 Suppl 56:1–34.
13. Maddaus MA, Toth JLR, Gullane PJ, Pearson FG. Subglottic tracheal resection and synchronous laryngeal reconstruction. J Thorac Cardiovasc Surg 1992;104: 1443–50.
14. Grillo HC, Mark EJ, Mathisen DJ, Wain JC. Idiopathic laryngotracheal stenosis: treatment and results. J Thorac Cardiovasc Surg 1995;109:486–93.
15. Herridge MS, Pearson FG, Downey GP. Subglottic stenosis complicating Wegener's granulomatosis: surgical repair as a viable treatment option. J Thorac Cardiovasc Surg 1996;111:961–6.
16. Gaissert HA, Grillo HC, Shadmehr MB, et al. Laryngotracheoplastic resection for primary tumors of the proximal airway. [In preparation]
17. Grillo HC, Zannini P. Resectional management of airway invasion by thyroid carcinoma. Ann Thorac Surg 1986;42:287–98.
18. Grillo HC, Suen HC, Mathisen DJ, Wain JC. Resectional management of thyroid carcinoma invading the airway. Ann Thorac Surg 1992;54:3–10.
19. Gaissert HA, Grillo HC, Shadmehr MB, et al. Comparative long term survival after resection of adenoid cystic and squamous cell carcinoma of trachea and carina. [In preparation]
20. Bogdan CJ, Maniglia AJ, Eliachar I, Katz, RL. Chondrosarcoma of the larynx: challenges in diagnosis and management. Head Neck 1994;16:127–34.

Repair of Acquired Tracheoesophageal and Bronchoesophageal Fistula

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The characteristics of tracheoesophageal fistulae and their surgical management differ from benign to malignant, congenital to acquired, acute to chronic, and cervical to thoracic fistulae, and from those with or without concurrent tracheal stenosis. These categories are described in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula." Techniques for surgical repair are described here. Congenital tracheoesophageal fistula is not presented. That problem is primarily esophageal and has been exhaustively expounded in many textbooks since Haight and Towsley performed the first successful primary anastomosis for congenital atresia and fistula.[1]

Benign Tracheoesophageal Fistula

Closure of a Fistula with Tracheal Resection

Postintubation tracheoesophageal fistulae usually result from erosive injury by a cuff, which concurrently causes circumferential tracheal injury (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula") (Figure 26-1). Fistulae very rarely can also result from erosion by the tip of a posteriorly angulated tracheostomy tube, without circumferential injury. Tracheal injuries, when present, should be corrected at the same time as closure of the fistula. An attempt to do this in stages demands two difficult operations, the second compounded by surgical reaction to the first intervention. Furthermore, resection of a tracheal stenosis provides incomparable access to an esophageal fistula. If a patient is still on a respirator when the fistula is discovered, the patient should be weaned *prior to repair*. Attempts to seal a fistula in a ventilated patient with muscle flaps or the defunctioned esophagus are likely to fail. Any tube in the esophagus is removed and ventilation is continued with the lowest cuff pressure that will provide a gentle seal of the trachea. If the cuff can be placed below the fistula, so much the better. A gastrostomy tube drains the stomach to prevent reflux and aspiration, and a jejunostomy tube is used for feeding. After weaning from the respirator, single-stage repair is done. Esophageal diversion by cervical esophagostomy or exclusion is almost never necessary. In the case of high fistulae close to the cricopharyngeus (as many of these are), diversion is impossible anyway. Elec-

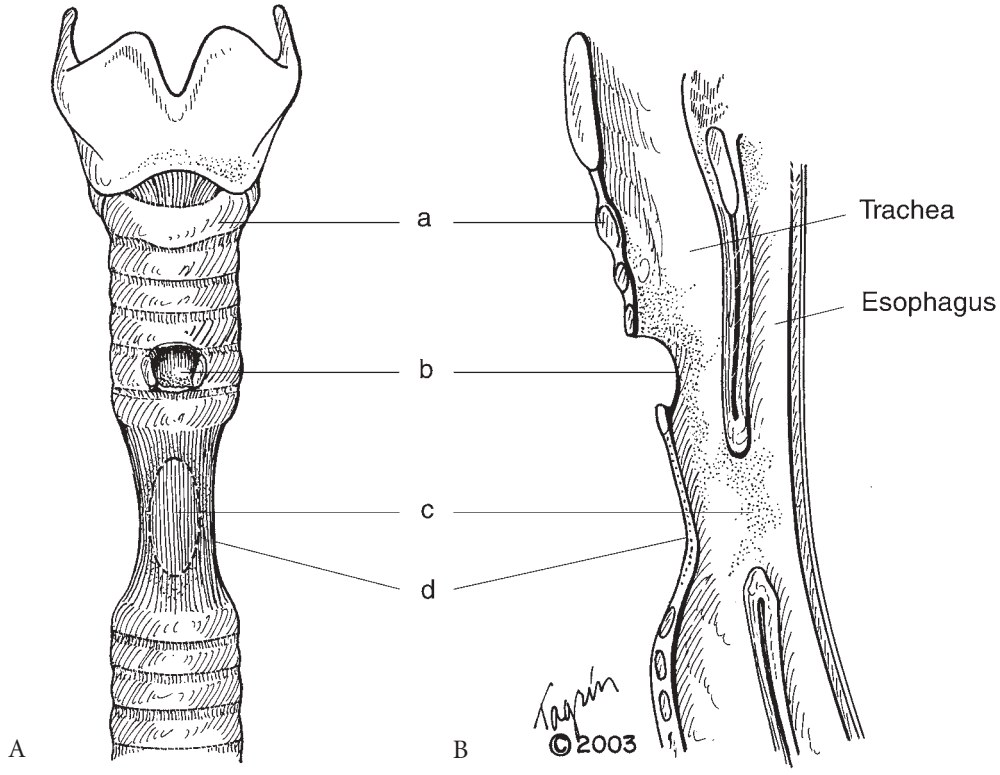


FIGURE **26-1** *Postintubation tracheoesophageal fistula.* A, *Fistula resulting from pressure damage to the trachea by the sealing cuff on a tracheostomy tube used for ventilatory support.* a = *cricoid cartilage. The stoma* (b) *lies above the area of circumferential injury* (d) *caused by the cuff. The fistula* (c) *is located posteriorly in this segment. The length of relatively normal trachea between the stoma and segmental injury varies but is most often short.* B, *Diagram of the sagittal section of* A. *Note the relative levels of stoma and fistula, and also the damage to the cartilaginous tracheal wall at the same level as the fistula. Due to the gradual and inflammatory nature of the fistulization, the membranous wall of trachea is fused to the anterior esophageal wall and the margin of the fistula is epithelized.*

tive diversion is to be avoided, since it complicates treatment and may be difficult to reverse regardless of whether the esophagus is transected proximally or exteriorized in continuity.

Anesthesia for repair may be initiated through an existing tracheostomy, which is usually present. The gastrostomy tube is put on suction. If neither is present, the stomach is aspirated, placed on suction, and endotracheal intubation is accomplished rapidly. Bronchoscopy and esophagoscopy are performed, if not previously done by the surgeon, using rigid endoscopes. An endotracheal tube is then positioned, preferably perorally, so that its cuff seals the fistula during initial dissection. The endotracheal tube must not be allowed to slip into the esophagus via the fistula. Gastrostomy tube or nasogastric tube suction is continued to control leakage of gas through the fistula. A nasogastric tube is also useful as a guide for esophageal dissection. Most postintubation fistulae are high since they result from erosion related to the cuff on a tracheostomy tube (see Figure 26-1).[2] They are approachable, therefore, through a collar incision, and rarely require even upper sternal division except in the aged and kyphotic patient.

The collar incision usually circumcises the stoma. Initial dissection, as described for anterior tracheal resection, is kept very close to the trachea at the level of the lesion (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). The recurrent laryngeal nerves will fall away with the lateral tissues and must not be individually dissected or identified. The trachea is dissected circumferentially, just inferior to the fistula. This plane can usually be established with gentle and persistent dissection

despite inflammatory adherence of the posterior wall of the trachea to the esophagus, which may extend for varying distances below the fistula (Figure 26-2*A*). Only 1 or 2 cm of trachea that will remain should be dissected circumferentially. The esophagus does not have to be circumferentially dissected, but it is necessary to free enough esophageal wall lateral to the fistula on both sides so that esophageal closure can be done in two layers without tension on the suture line. As much dissection as can be accomplished superiorly is also performed prior to division of the trachea. Proximity to the cricoid often inhibits posterior superior dissection at this stage. The thyroid gland is separated from the trachea only to the extent needed for tracheal resection and excision of fistula. This dissection is described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection." The recurrent nerves are not visualized but are displaced laterally, because the plane of dissection is immediately on the trachea and then the esophagus.

The trachea is transected below the distal margin of the stenotic segment. The proximal specimen is elevated with two laterally placed Allis forceps and dissection continued until the fistulous connection is completely freed circumferentially, preferably before it is opened into (Figure 26-2*B*). Occasionally, the proximal end of the fistula commences immediately below the cricoid cartilage, making it impossible or unwise to dissect over the top of the fistula until it is incised from below on each side. Otherwise, recurrent nerve injury could result near their entry points into the larynx posterolaterally. In many cases, a tracheal stoma is so close to the injured segment of trachea that it is most conveniently included in the resection of the tracheal segment. If, however, there is a segment of normal trachea between the stoma and the stenotic segment, the stoma may be left in place to avoid excessively lengthy resection. If the lower border of the fistula is coincidental with the lower margin of the tracheal stenosis, the fistula is entered at the point where it joins the esophagus, and it is excised upward in an elliptical fashion, saving all esophageal wall that is of good quality (see Figure 26-2*B*). The trachea is divided above and the excision of the fistula completed.

Postintubation fistulae are sometimes described as "giant" fistulae. They may involve the entire width of the membranous tracheal wall. A large fistula may also involve the membranous wall to a level considerably below the level of circumferential damage to the cartilaginous wall of trachea. In such a case, it is often best to preserve normal cartilages rather than extend the tracheal resection to a length that might produce hazardous anastomotic tension (Figure 26-3*A*). With a giant fistula, the remaining membranous wall will be insufficient at the level of the fistula to allow simple closure of the posterior wall of the trachea. The distal line of excision of the fistula, at the level of the tracheal cartilages that are to be salvaged, is therefore tailored into the esophagus, "stealing" portions of esophageal wall for reconstruction of a membranous trachea (Figures 26-3*B*,*C*). These flaps are dissected very carefully in order not to injure their effectively parasitic blood supply from the trachea. The "U" gap in the posterior membranous wall is closed by suturing the attached segments of esophageal wall vertically together in the midline using 4-0 Vicryl sutures (Figures 26-3*D–F*). The knots are placed posteriorly rather than in the lumen. There is more than adequate esophageal circumference, so that loss of this tissue will not adversely affect esophageal closure or function (see Figure 26-3*E*). It is a mistake to pull the trachea together under tension in an attempt to close a wide defect in the membranous wall.

The esophagus is closed longitudinally with two layers of 4-0 silk or Vicryl, using the technique of Sweet.[3] The inner layer of sutures is placed so that the knots will lie inside the esophageal lumen (Figure 26-4*A*). These sutures include only the esophageal mucosa, which is of strong consistency. They are started from above, then from below, inverting each successive suture. The final suture is placed in the middle of the esophageal closure and inverted with a Connell suture. The second layer closure of Lembert sutures includes the esophageal muscularis (Figure 26-4*B*). Enough esophageal circumference should be dissected so that closure is without tension, but no more than that.

If the superior margin of the esophageal fistula is close to the lower border of the cricoid, closure may be difficult. Dissection may be carefully made a few millimeters cephalad behind the posterior plate of the cricoid.

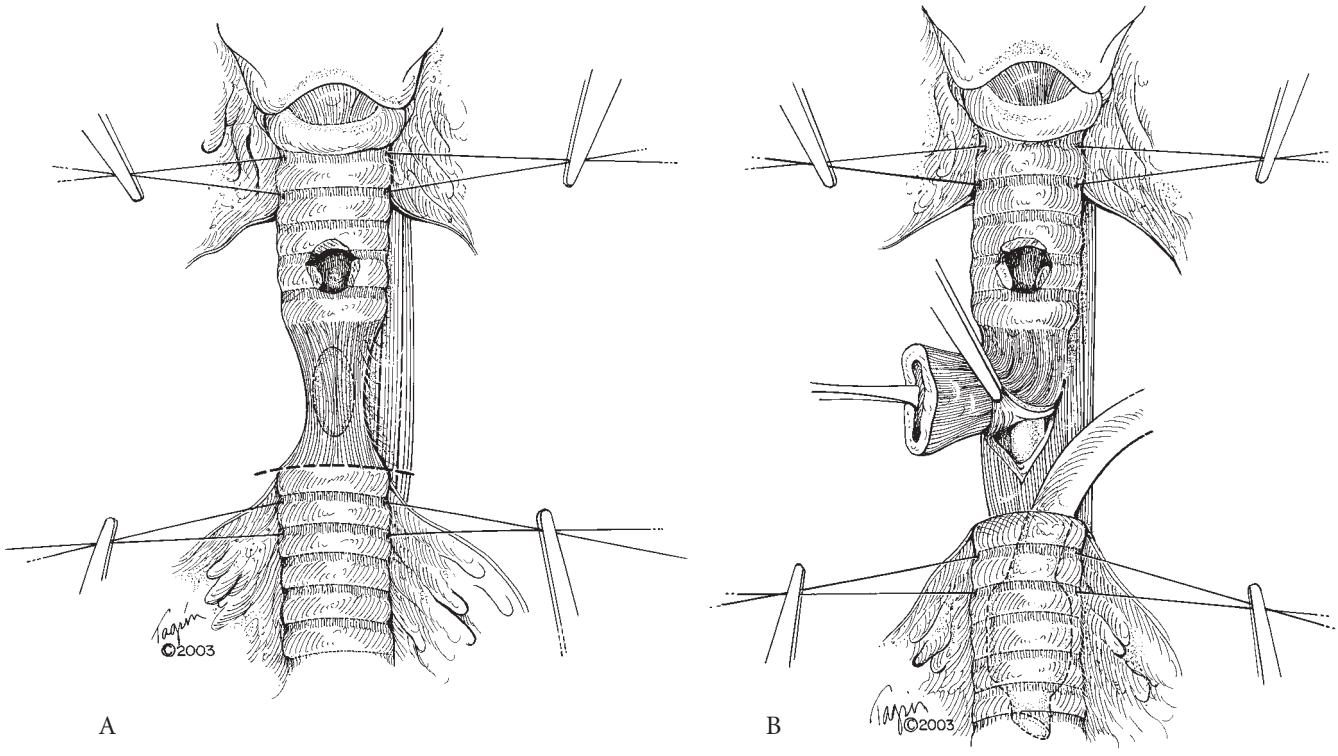


FIGURE **26-2** *Resection of tracheoesophageal fistula. Since tracheostomy is usually high in the trachea, the fistula resulting from cuff injury is not far below and approach is most often by collar incision only. In older patients or where a stoma was placed too low in a young patient, upper sternotomy may be required. Usually, the collar incision circumcises the stoma in its midpoint. If the stoma is very high, the collar incision is placed low in the neck (see Figure 23-2 in Chapter 23, "Surgical Approaches"), and the stoma is excised later through a separate short incision, as the upper flap of skin and platysma are elevated. Dissection of the trachea is initially done as described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection." Recurrent laryngeal nerves are not dissected out.* A, *Circumferential dissection is accomplished just below the stenotic segment, freeing no more than two intact tracheal rings. Note preserved lateral attachments which carry tracheal blood supply. Proximal circumferential dissection is also limited if a tracheal segment below the stoma is to be preserved. In this case, the "isthmus" between the stoma and stenosis is too limited and involved by stomal granulations and inflammation to be useful in reconstruction. The total length of resection when stenosis, stoma, and residual segment are added together must not prohibit safe reconstruction. If more than one satisfactory cartilaginous ring is present below the stoma, this segment should be saved. Remember, the surgeon can always remove more trachea later in the operation, so initial resection should be conservative. Often, circumferential dissection above the fistula is very difficult due to inflammatory adhesion or proximity to cricoid. In such case, it should be deferred and accomplished later as the fistula is excised and the divided trachea is retracted upward. The esophagus is freed bilaterally enough to provide for tension-free closure. It is not usually necessary to dissect it circumferentially. If this is required, short segment esophageal mobilization will not injure its blood supply. Note the lateral traction sutures (2-0 Vicryl) placed one or two rings away from the anticipated level of tracheal transection. Proximal sutures may be placed in lateral cricoid laminae, if the stoma is just below the cricoid or has injured it (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection" and Chapter 25, "Laryngotracheal Reconstruction").* B, *The trachea has been transected and intubated distally. The damaged trachea is elevated by placing Allis forceps on either side (only one is shown for clarity) of the specimen. The fistula is circumcised following its margins on either side. This dissection allows easier development of the plane above the fistula between trachea and intact esophagus. (See text for description of dissection when superior margin of fistula is adjacent to the posterior cricoid lamina.) Resection is completed by proximal division of trachea (see Figure 12-8 in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula").*

Posterolateral dissection at this level is inhibited by concern for recurrent laryngeal nerves. This limited dissection should provide sufficient mobility to commence apical closure. In a single patient with laryngotracheal stenosis and what appeared to be iatrogenic fistulization through the posterior cricoid plate, closure finally required elevation of laryngeal mucosa over the plate and removal of some posterior cricoid cartilage to give access for pharyngoesophageal closure. Strap muscle was interposed and a posterior membranous tracheal wall flap was anastomosed to the laryngeal mucosa (see Chapter 25, "Laryngotracheal Reconstruction").

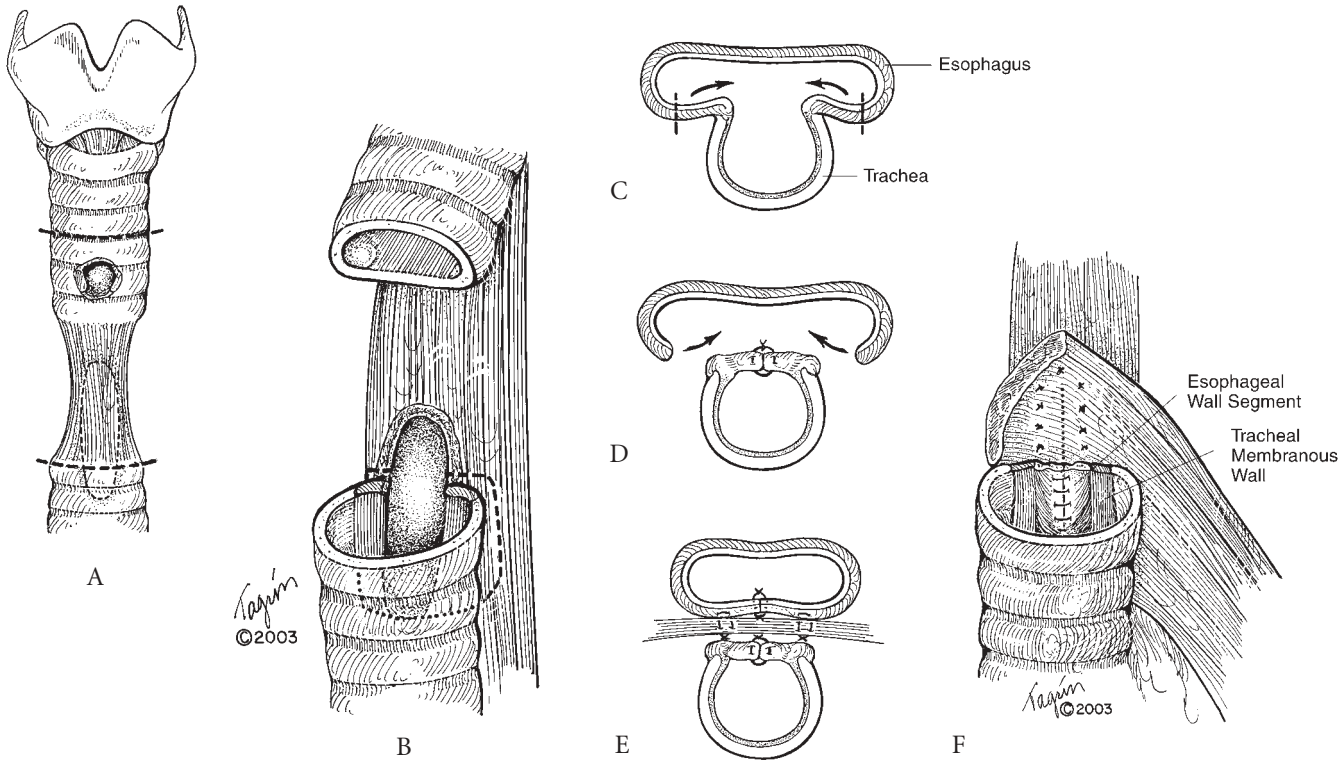


FIGURE **26-3** *Preservation of tracheal length where giant fistula extends distal to the level of cartilaginous injury, and where extension of tracheal resection would increase the prospect of excessive anastomotic tension.* A, *Here, the distal extremity of a giant fistula is below the level of distal intact tracheal rings. Since the proximal stoma must also be resected because of proximity to the stenosis, further removal of two distal tracheal rings to encompass the fistula would result in possibly excessive anastomotic tension.* B, *Stenotic, severely damaged tracheal segment has been excised, saving distal anterolateral undamaged trachea despite extension of a giant posterior tracheoesophageal fistula below this level. In order to reconstruct the membranous wall and so salvage a functional length of trachea, full thickness flaps of esophageal wall are created bilaterally* (dashed lines), *extending from the level of tracheal division to the bottom of the membranous wall defect. In the initial dissection, and in raising these flaps, care is taken not to thin the tissues at the junction of the fistulous margin between trachea and esophagus, since this will be the source of blood supply for the flaps. A minimal amount of distal dissection opens the plane between intact membranous tracheal wall and esophagus to allow linear closure of the esophagus. Two-layered closure is done, as shown in Figure 26-4.* C, *Cross-sectional diagram of membranous wall reconstruction at the level of the fistula. Flaps of esophageal wall are outlined on each side, with care to preserve blood supply at the junction of esophageal and tracheal walls.* D, *Defect repaired by inversion of esophageal wall flaps. The esophageal defect is repaired in its entire length. The slight esophageal narrowing is of no consequence.* E, *After layered esophageal closure, the suture line is covered with a flap of strap muscle.* F, *Esophageal closure has been completed* (dotted line) *and covered with a sternohyoid muscle flap. The posterior tracheal wall is reconstructed by turning the bilateral esophageal flaps medially, approximated with interrupted 4-0 Vicryl sutures placed with knots outside the tracheal lumen. The tracheal anastomosis completes the repair, performed as described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection." A mattress suture (4-0 Vicryl) is used at the junction point of the circumferential tracheal anastomosis and the vertical suture line of the membranous wall repair.*

A sternohyoid muscle or, less commonly, a sternothyroid muscle, is divided high and rotated to provide a flap over the esophageal suture line, to interpose healthy tissue between esophageal and tracheal suture lines (Figure 26-4*C*). As little muscle is dissected free as is needed, in order to protect blood supply. Even slightly dusky muscle survives. It is sutured with interrupted 4-0 silk or Vicryl around the entire length of the esophageal closure as if it were an anastomosis. The tracheal anastomosis is next completed as previously described. If a vertical closure of "membranous wall" of the trachea has been necessary, as shown in Figure 26-3*F*, special care is taken at the meeting point of that suture line with the tracheal anastomotic line, using a mattress suture in this case. The gastrostomy and jejunostomy are left in place until healing

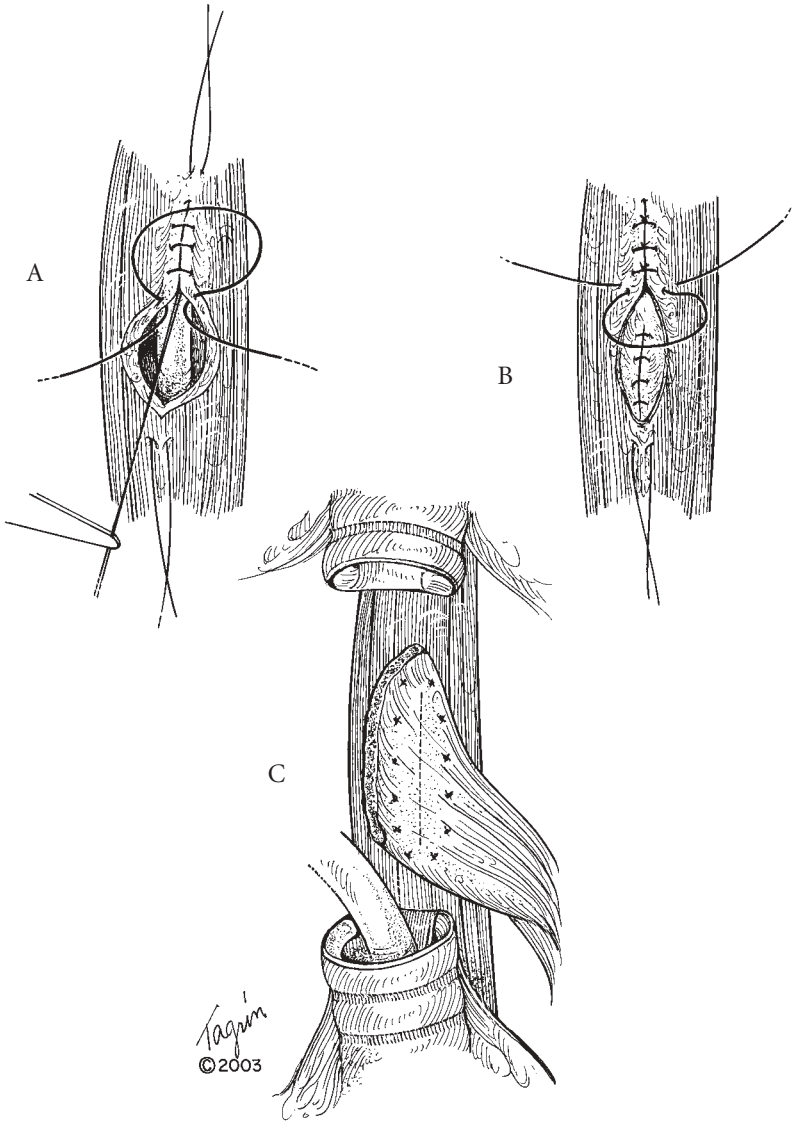


FIGURE **26-4** *Esophageal closure.* A, *Sutures (4-0 silk or Vicryl) mark the proximal and distal limits of the defect, which will be closed vertically. Mucosal closure (same sutures) is made with inverting interrupted sutures, placed as shown. As each mucosal suture is tied, the prior suture is held on slight tension, thus inverting the mucosa. The excess suture is then cut, preserving the last suture's length for successive inversion of the next. All knots lie inside the lumen. Suturing is begun from both ends of the defect with a final Connell suture placed at the midpoint.* B, *A second layer of interrupted Lembert sutures approximates the esophageal muscle coats.* C, *Pedicle of sternohyoid muscle, detached from the hyoid bone, is sutured over the esophageal closure* (dotted line) *with multiple fine interrupted 4-0 sutures placed with the proximity of anastomotic sutures. Reconstruction is completed with end-to-end tracheal anastomosis.*

has occurred. If these adjunctive tubes are not present, they are inserted at completion of the operation. This approach has been successfully applied by a number of surgeons.[2,4–7]

Closure of a Fistula without Tracheal Resection

In the case where there is no tracheal injury except at the site of a fistula to the esophagus, repair does not require tracheal resection.[6] Dissection differs at critical points from that just described. A collar incision will provide as good exposure as an oblique incision anterior to the sternocleidomastoid muscle and a better cosmetic result. The collar approach also facilitates bilateral dissection should this become necessary. This can be important if prior attempt at closure has been made. A unilateral approach is used initially in order to minimize the possibility of injury to recurrent laryngeal nerves. If the fistula is midline but low in the neck, the left side is preferably selected. The left recurrent laryngeal nerve enters the neck lying in the tracheoesophageal groove rather than crossing the lower neck obliquely to reach the groove, as on the right. The collar incision is therefore slightly eccentric, with a greater length to the left than to the right. The cuta-

neous and platysmal flaps are elevated above and below to expose the anterior border of the left sternocleidomastoid muscle, thus providing the same exposure as an oblique incision.

Dissection is made in the classic avascular plane medial to the carotid artery and internal jugular vein until the esophagus is reached. The trachea is *not* exposed at this point. The esophagus is dissected at the location of the fistula, working carefully above and below it behind the trachea, with the plane of dissection *on* the esophageal wall. The membranous wall of the trachea is exposed above and below the fistula (Figure 26-5*A*). The fistula is usually encircled unless scar from a prior failed procedure is too dense. In this dissection, the recurrent laryngeal nerve remains with the trachea. The nerve is not deliberately identified, unless required. An alternative is to dissect from the anterior trachea laterally at the level of the fistula and then to the esophagus, displacing the nerve laterally. The surgeon must have a clear concept of the anatomic pathway being taken. If dissection must be carried out from the right side, it will be necessary to approach the fistula from this latter path since the recurrent nerve arrives at the larynx and trachea higher and at a more acute angle to lie in the tracheoesophageal groove superiorly. Great care must be taken to avoid injury to the nerve. In this case, it may even be necessary to dissect the nerve with delicacy. The rare anomaly of a nonrecurrent right laryngeal nerve, which crosses the neck transversely from the vagus nerve, must be kept in mind (see Figure 12-7 in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula"). This occurs in patients with an anomalous right subclavian artery arising on the left and passing behind the esophagus (see Chapter 1, "Anatomy of the Trachea"). Even less often encountered is a nonrecurrent nerve on the left, which occurs with right aortic arch and an anomalous left subclavian artery. Chest computed tomography scan often alerts the surgeon now to these possibilities. Exposure of these fistulae, even though they are usually smaller than postintubation fistulae, may be considerably more difficult, since the trachea is not divided, and there is little space between the trachea and esophagus, especially when the fistula is just below the posterior cricoid.

The fistula is divided at a distance farther from the membranous wall of the trachea to facilitate closure on that side, where tissue is shorter in supply than on the esophageal side (see Figure 26-5*A*). If the fistula is of any size, some esophageal wall is included to facilitate closure of the membranous wall of the trachea without tension. After division of the fistula, the esophagus is closed in layers using the technique described (Figure 26-5*B*). The tracheal defect is repaired next with 4-0 Vicryl sutures. The trachea is usually, but not necessarily, sutured longitudinally. A flap of strap muscle is interposed and sutured over the esophagus to prevent reestablishment of the fistula. If access is difficult, the trachea may be circumferentially dissected above and below the fistula and retracted with tape (see Figure 26-5*A*). Care must be taken not to injure the recurrent laryngeal nerves. Bilateral access through a collar incision may make this dissection safer in a difficult case.

If a fistula is of large size, segmental tracheal resection becomes advisable, even though the trachea is not circumferentially damaged. In the case of a larger fistula where limited circumferential tracheal resection is likely to be desirable, approach should be anterior and dissection directed at first to the trachea, as described for postintubation tracheoesophageal fistulae with tracheal damage. In this situation, the recurrent nerves are allowed to fall laterally and remain undissected.

The approach described is applicable to benign upper tracheoesophageal fistula without extensive tracheal damage regardless of etiology; that is, foreign body, postoperative, instrumental, post-traumatic, or a high congenital H-type fistula (see Figure 6-3 in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children").

Benign Intrathoracic Fistula

Benign acquired fistula is not often seen in the thorax, but inflammatory diseases, including tuberculosis, histoplasmosis, and silicosis, produce tracheoesophageal fistulae just above the carina or broncho-

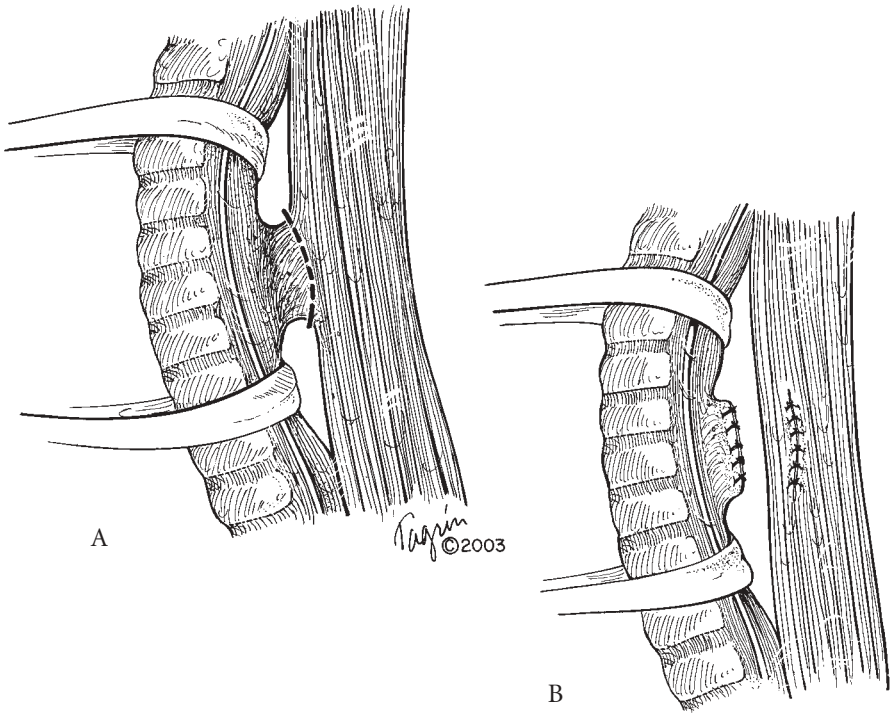


FIGURE **26-5** *Closure of tracheoesophageal fistula in the absence of a circumferential tracheal lesion.* A, *A small fistula may be approached initially laterally at esophageal depth with the trachea drawn anteriorly. The left recurrent nerve can remain with the trachea, depending on the size and location of the fistula. The thyroid lobe may have to be elevated to provide access. Alternatively, it may sometimes be preferable to identify the tracheal wall and follow this plane carefully to the esophagus, displacing the undissected nerve laterally. A clear plan of approach is essential. Although tapes are shown encircling the trachea, circumferential tracheal dissection is not always necessary. The fistula is isolated. In reoperation after prior exploration, a bilateral approach may be preferable.* B, *Division of the fistula is made close to the esophagus to preserve enough tissue for easy closure of the membranous wall of trachea, where there is no excess of tissue. Closure is most often, although not necessarily, vertical, using interrupted 4-0 Vicryl sutures. The esophagus is repaired longitudinally in two layers, as previously described. A large posterior fistula is in some instances best managed by short tracheal segmental resection and esophageal closure.*

esophageal fistula (see Figures 12-12, 12-13 in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula"). If a fistula has been previously approached transthoracically with failure, it is usually better to reoperate transthoracically. Furthermore, with the diseases listed above, the dense scar that is likely to be encountered is best dealt with by this route, at a distal level through the wide exposure it offers. Post-traumatic fistula is discussed in Chapter 31, "Repair of Tracheobronchial Trauma."

Exposure is usually through a right thoracotomy, at the fourth interspace or fifth rib bed. A long, flexible single-lumen tube is placed with endoscopic guidance into the left main bronchus, permitting easier retraction and manipulation of the trachea and carina than does a double-lumen tube. If left thoracotomy is performed to deal with a left main bronchial fistula, the tube is positioned in the right main bronchus. A bronchial blocker may also be useful. High-frequency ventilation is not appropriate for esophageal fistula repair. The azygos vein is divided and the trachea dissected and retracted above the fistula. The esophagus is dissected and retracted above and below the fistula and the fistula isolated. The extent of bronchial and carinal dissection depends upon the unique pathology. The principles of fistula closure were outlined above, including preservation of sufficient tissue for closure of the membranous tracheal wall and a two-layer esophageal repair. The trachea is closed with interrupted 4-0 Vicryl sutures. Tracheal resection is unlikely to be necessary.

In these cases, I prefer to elevate a long posteriorly-based intercostal muscle pedicled flap as the thoracotomy incision is made (see Figure 31-6 in Chapter 31, "Repair of Tracheobronchial Trauma"). The flap is sutured in linear fashion over the esophageal closure, using multiple 4-0 silk or Vicryl mattress sutures, placed closely enough so that a complete tissue layer seal is obtained. The muscle will also lie against the membranous tracheal wall. Additional sutures may be placed from the trachea to the muscle flap, if thought to be necessary to secure the tracheal closure. In this situation, where the intercostal flap is used as an onlay buttress, and *not* circumferentially, it is not necessary to remove the costal periosteum from the flap. A *circumferential* wrap of intercostal muscle is avoided, since it can result later in an obstructive "grommet" of new bone formation from the attached periosteum. If there is any question about the integrity of the esophageal closure, it is tested by injecting a large volume of methylene blue-colored saline into the esophagus at the level of repair via a nasogastric tube prior to applying the intercostal muscle flap. The tracheal closure is tested by applying 30 cm of water ventilatory pressure through the endotracheal tube, with the cuff transiently deflated, while the hemithorax is filled with saline.

If prior irradiation is a factor, the pedicled omentum is advanced and wrapped around the esophagus and trachea and interposed between them to facilitate healing. The omentum may be prepared through an upper midline incision. If an omentum is likely to be needed, the patient should be positioned for lateral thoracotomy with hips slightly angled, so that abdominal access is facilitated (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation").

Malignant Tracheoesophageal Fistula

Acquired tracheoesophageal fistula due to carcinoma of the esophagus, or much less often of lung, usually predicts brief survival (see Chapter 8, "Secondary Tracheal Neoplasms"). Palliative management with endoesophageal tubes or coated stents may be indicated. Alternatively, a tracheal or carinal stent may be helpful, although usually less so. Rarely is operative treatment justified (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula"). A few patients with fistula due to less common lesions such as adenoid cystic carcinoma, carcinoid, and lymphoma (usually after irradiation) may possibly look forward to prolonged survival. Palliative surgical treatment may selectively be considered for them. The technique must be individualized. The general principles are 1) to isolate the fistula from the alimentary system, and 2) to leave as little esophagus as possible attached to the airway as a "diverticulum," in order to minimize mucus production, pooling, and aspiration. An older alternative is to divide the esophagus above the fistula and drain the distal esophagus by a Roux-Y loop, using the stomach for substernal esophageal bypass (Kirschner procedure).[8] If the colon is used for esophageal bypass, the distal esophagus may be left to drain into the stomach, but a hazard of reflux into the respiratory tree remains. "Bipolar exclusion" of the esophagus with a retained distal segment is ill-advised, since pooled secretions will discharge into the trachea and distal leakage may occur. Jejunostomy for postoperative alimentation is advisable with all of these procedures. Alimentary tract restoration provides both oral feeding and ability to swallow saliva. Generally, the fistula and tumor cannot be successfully resected nor the airway reconstructed.[9–11]

A collar incision plus upper sternal division provides access for dissection of the esophagus under direct vision down to the area of tumor, followed by esophageal division with precise closure above the tumor (Figures 26-6*A*,*B*). The esophagus is stapled and divided and a reinforcing layer of interrupted fine sutures inverts the stapled closure. Any excess of proximal esophagus will be removed at the time of enteric anastomosis. Complete sternotomy adds very little exposure. Laparotomy permits transhiatal resection of the esophagus below the tumor and fistula, leaving as small a residual pouch of esophagus around the fistula as possible (Figure 26-6*C*). Transhiatal removal is facilitated by using Pinotti's maneuver of dividing the diaphragm from xiphoid to hiatus, which allows dissection under direct vision.[12] Gastric or left colonic mobilization is accomplished as well as omental mobilization to the degree necessary (see Figure 26-6*B*).

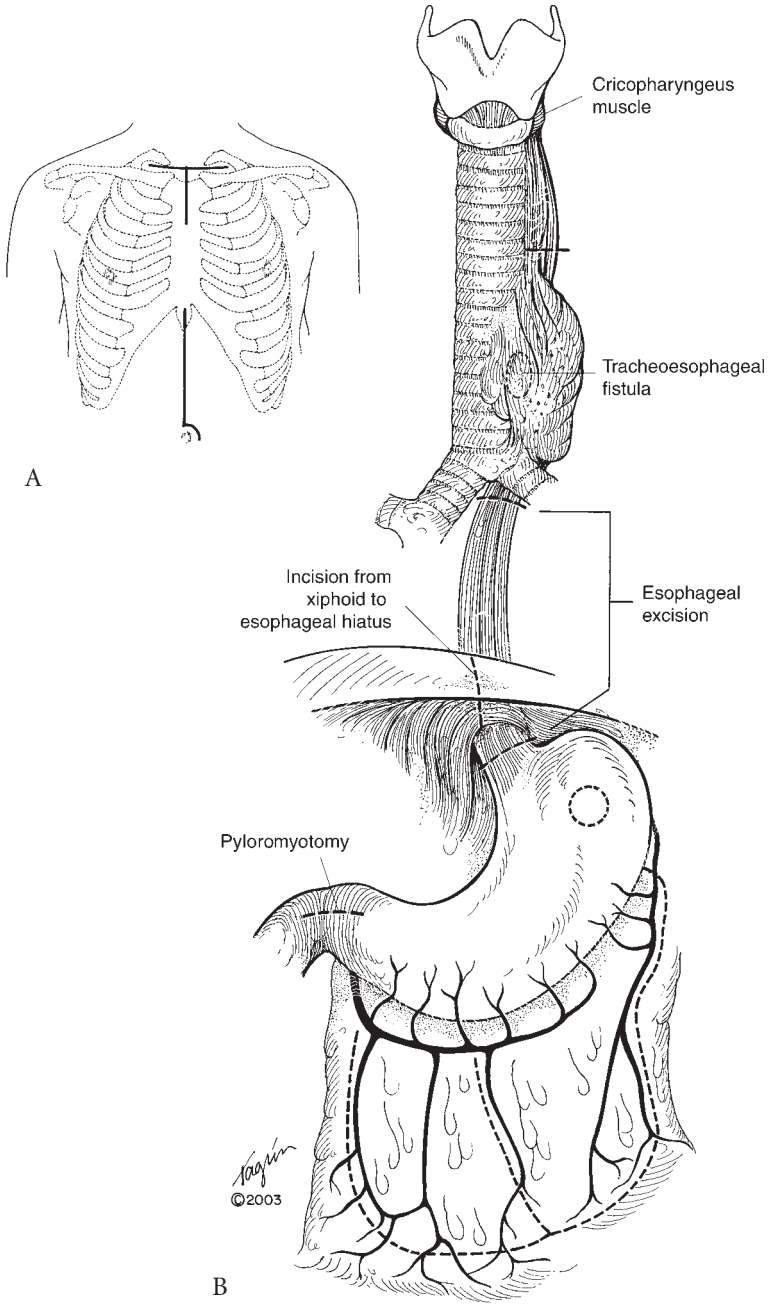


FIGURE **26-6** *Palliative treatment of malignant tracheoesophageal fistula by exclusion and esophageal bypass. The text notes variations of this procedure. It may be justifiable in a few patients, whose prospects for longevity are unusually encouraging.* A, *Collar incision with upper sternotomy provides access to the upper esophagus to a point just above the tumor, and later for esophagogastric anastomosis. Laparotomy allows omental mobilization, gastric mobilization, pyloromyotomy, and intrathoracic excision of the lower esophagus up to the tumor (with additional anterior diaphragmatic incision).* B, *Diagram of components of procedure. Esophageal division above and below the tumor and fistula converts this to a "diverticulum" of the trachea. The lower esophagus is removed. The stomach is fully mobilized with omentum remaining attached, and both are nourished by the right gastroepiploic artery. Pyloromyotomy or pyloroplasty is necessary. The stomach is advanced through a substernal tunnel made from above and below. The omentum is divided into two aprons, one inserted into the posterior esophageal bed and the other pedicled into the neck to reinforce the esophagogastric anastomosis. Cervicomediastinal and lower mediastinal drains are inserted and jejunostomy added.*

Esophageal bypass is constructed by cervical anastomosis of the proximal esophagus to a substernally placed stomach or colonic segment (Figure 26-6*D*). Blunt dissection of the substernal tunnel is usually not much impeded by prior mediastinal irradiation. The omentum is tucked into the esophageal bed to avoid leakage from the often previously irradiated residual esophageal "diverticulum." The omental pedicle may be split and one limb brought up anteriorly beside the stomach to reinforce the anastomosis. Obviously, if stomach is used for enteric reconstruction, the omentum remains attached. If colon is used, the omentum is pedicled on the mobilized right gastroepiploic artery (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation"). The superior and inferior mediastinum is drained.

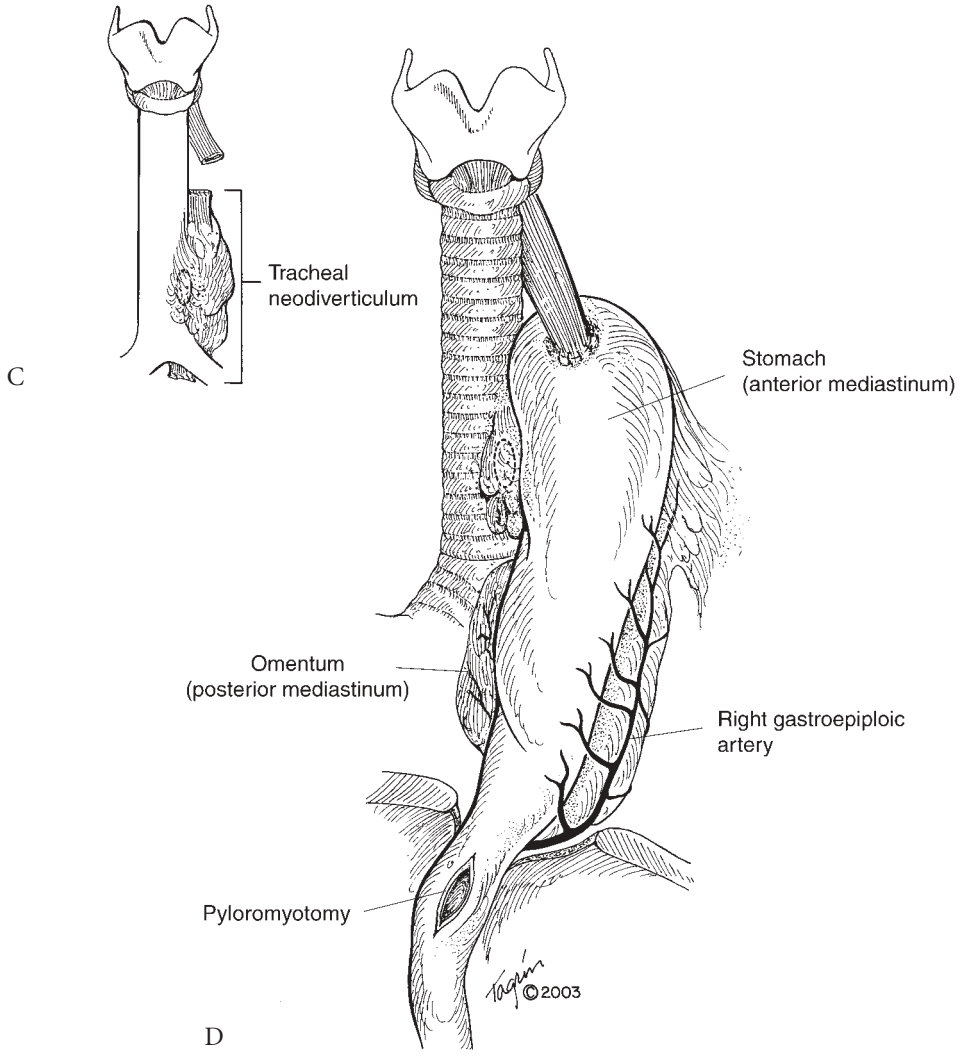


FIGURE **26-6** (CONTINUED) C, *The tumor and fistula are converted to a neodiverticulum of the trachea. Proximal and distal esophageal ends of the "diverticulum" have been stapled and, if possible, reinforced with sutures.* D, *Completed exclusion of the fistula and anterior mediastinal bypass. The esophagogastric anastomosis will be reinforced by the upper half of the omental pedicle. The lower omental flap has been tucked into the mediastinum posteriorly, in the esophageal bed.*

Pyloromyotomy is added because of the necessary bilateral vagectomy. A temporary feeding jejunostomy is judicious. As the disease progresses, a tracheal or tracheobronchial stent may become necessary later.

Bronchoesophageal Fistula

Benign bronchoesophageal fistula is quite rare.[13] Types of congenital fistulae are described in Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula" (see Figures 12-9, 12-10). The fistula is dissected out via a thoracotomy, and both esophageal and bronchial ends are carefully closed by suture. Even where there is sufficient pulmonary infection to require a lobectomy, dissection of a congenital fistula is not difficult. Healthy tissue is sutured over the esophageal closure. A Brewer pericardial fat pad will usually suffice.

Fistula of inflammatory origin, that is, tuberculosis, histoplasmosis, silicosis, instrumentation, surgery, or trauma, present a somewhat greater technical challenge (see Chapter 12, "Acquired Tracheoesophageal and Bronchoesophageal Fistula"). Fibrotic lymph nodes and local scar are encountered. The principles of management are the same. If the fistula is large, an intercostal muscle pedicle interposition may be prudent. In recent years, fistulae associated with tuberculosis and fungi have been seen in patients with acquired immunodeficiency syndrome.

References

1. Haight C, Towsley HA. Congenital atresia of the esophagus with tracheoesophageal fistula. Surg Gynecol Obstet 1943;76:672–88.
2. Grillo HC, Moncure AC, McEnany MT. Repair of inflammatory tracheoesophageal fistula. Ann Thorac Surg 1976;22:113–9.
3. Sweet RH. Thoracic surgery. Philadelphia (PA): WB Saunders; 1950.
4. Couraud L, Bercovici D, Zanotti L, et al. Traitment des fistules oesophago-trachéales de la réanimation. Ann Chir Thorac Cardiovasc 1989;43:677–81.
5. Marcelle J, Dartevelle P, Khalife J, et al. Surgical management of acquired postintubation tracheoesophageal fistulas: 27 patients. Eur J Cardiothorac Surg 1989;3:499–503.
6. Mathisen DJ, Grillo HC, Wain JC, et al. Management of acquired nonmalignant tracheoesophageal fistula. Ann Thorac Surg 1991;52:759–63.
7. Macchiarini P, Verhoye J-P, Chapelier A, et al. Evaluation and outcome of different surgical techniques for postintubation tracheoesophageal fistulas. J Thorac Cardiovasc Surg 2000;119:268–76.
8. Kirschner MB. Ein neues Verfahren der Oesophagoplastik. Arch Klin Chir 1920;114:606–63.
9. Duranceau A, Jamieson GG. Malignant tracheoesophageal fistula. Ann Thorac Surg 1984;37:346–54.
10. Burt M, Diehl W, Martini N, et al. Malignant esophageorespiratory fistula: management options and survival. Ann Thorac Surg 1991;52:1222–9.
11. Couraud L, Ballester MJ, Delaisement C. Acquired tracheoesophageal fistula and its management. Semin Thorac Cardiovasc Surg 1996;8:392–9.
12. Pinotti HW. Acesso extrapleural ao esôfago por frenolaparotomia. Rev Assoc Med Bras 1976;22: 57–60.
13. Mangi AA, Gaissert HA, Wright CD, et al. Benign broncho-esophageal fistula in the adult. Ann Thorac Surg 2002;73:911–5.

Repair of Tracheobrachiocephalic Artery Fistula

Hermes C. Grillo, MD

Post-Tracheostomy Fistula
Postoperative Fistula

Fistula between the trachea and the innominate or brachiocephalic artery is a rare but potentially lethal lesion. It may result from tracheostomy used for ventilatory support and, also, as a complication of tracheal reconstruction. Tracheal stents may also erode into the artery. External traumatic injuries are basically vascular problems and are not presented here. Improved technique for tracheostomy, proper use of large-volume, low-pressure cuffs, and correct conduct of anterior tracheal reconstruction have markedly lessened the occurrence of fistulae between the trachea and brachiocephalic artery.

Post-Tracheostomy Fistula

The two types of tracheoarterial fistula following tracheostomy and ventilation are 1) those due to erosion by the tracheostomy tube itself, and 2) those due to erosion by the tracheostomy tube cuff or tip (Figures 27-1*A*,*B*). These are described in Chapter 13, "Tracheal Fistula to Brachiocephalic Artery." It is important that the two lesions be kept clearly in mind, since both emergent and definitive treatments differ.[1,2]

Erosion by Tracheostomy Tube

In the event of a massive hemorrhage from erosion of the brachiocephalic artery by an adjacent tracheostomy tube (Figure 27-2), control is obtained by firm finger compression downward at the site of bleeding and forward against the sternum (Figure 27-2*A*). An endotracheal tube is slipped into the trachea via the stoma, and the cuff is firmly inflated to minimize blood running into the tracheobronchial tree. The surgeon's finger or that of an assistant is held in place while the patient is expeditiously moved to the operating room, since cuff inflation alone may not control hemorrhage in this location. Exposure is best obtained through a collar incision at the level of the stoma plus a vertical sternotomy. Complete sternotomy at the outset will usually facilitate exposure and dissection under emergency circumstances (Figure 27-3*A*). Since sternal infection is a hazard, partial sternotomy through the manubrium angled into the third right interspace has been recommended.[3] The upper abdomen is included in the potential operative field, in case an

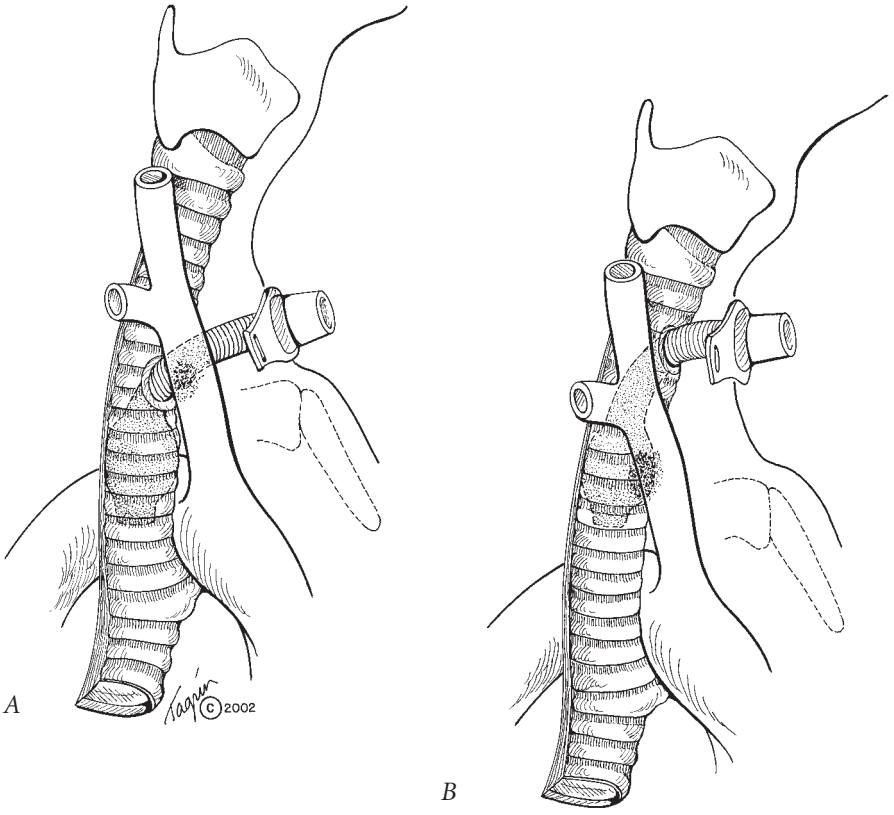


FIGURE 27-1 *Etiology of a fistula between the trachea and brachiocephalic artery. Oblique views.* A, *Erosion of the artery, lying adjacent to a tracheal stoma, by the tube resting against the artery. The stoma has been made lower in the trachea than ordinarily. In younger patients, both the trachea and the artery rise into the neck with cervical extension.* B, *Fistula caused by tracheoarterial erosion by a high-pressure tracheostomy tube cuff or by an angulated tube tip. In this case, tracheostomy lies in the conventional site (second or third ring).*

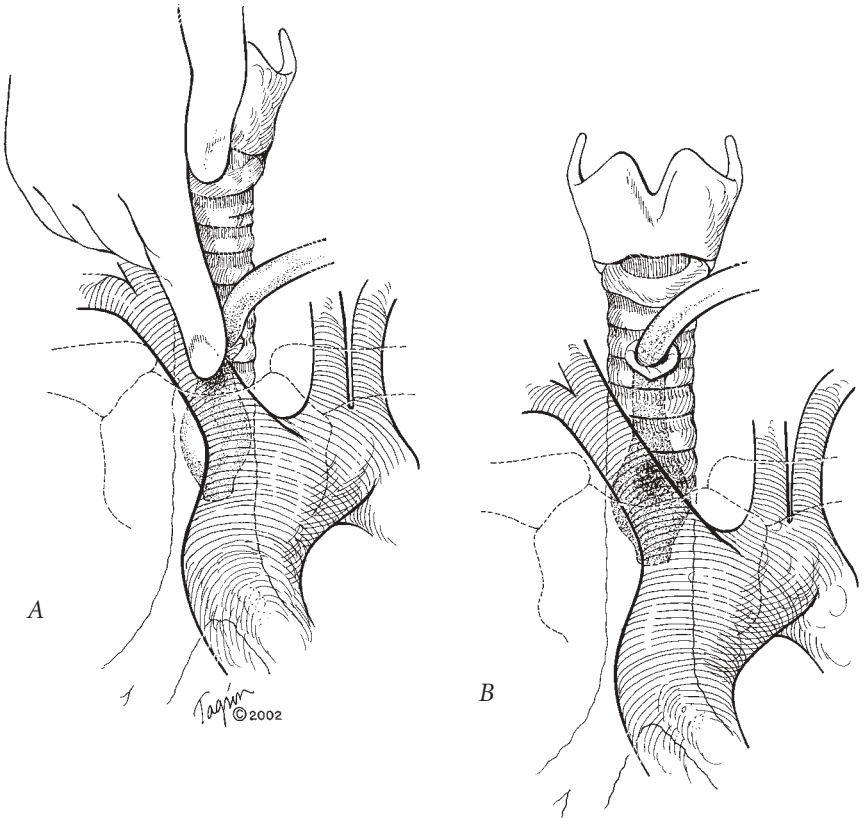


FIGURE 27-2 *Emergent control of a hemorrhage.* A, *When the hemorrhage is from direct erosion by the side of a tracheostomy tube, the bleeding point is at the lower margin of the stoma. Digital pressure on this spot, pressing down and also forward toward the sternum, provides control. Inflation of the cuff alone is unlikely to check bleeding, although it will help to keep the distal airway free of blood. Digital pressure must be maintained into the operating room.* B, *Where the fistula is the result of erosion through the trachea by a cuff or tube, an endotracheal tube with the cuff located at the site of the fistula and hyperinflated will tamponade the hemorrhage.*

omental pedicle is desirable. Control of the hemorrhage is maintained with digital pressure during the initial dissection. Proximal dissection of the brachiocephalic artery should be done with the greatest of care and patience, since there may be only a very short segment of uninvolved artery between the aortic arch and the fistula. If the left carotid artery arises from the aorta as a common trunk with the brachiocephalic artery, care must be taken to clamp and divide the brachiocephalic artery distal to the carotid origin. Distal arterial control is usually obtained just proximal to the bifurcation of the brachiocephalic artery into the right carotid and subclavian arteries. In the rare case where both carotids arise from a single trunk from the aorta, a carotid reconstruction is mandatory (see Chapter 1, "Anatomy of the Trachea"). The left brachiocephalic vein may be divided to enlarge the exposure, if retraction is not enough. This possibility negates placement of a central venous line on the left preoperatively. Erosion into the artery is usually of sufficient size and with enough adjacent damage so that primary repair of the artery is unwise and resection is elected (Figure 27-3*B*). Placement of a graft in this contaminated and potentially infected field seems unwise, although it has been done successfully. A graft can be placed from low on the aortic arch, passing to the right side of the field, if restoration is essential. If this is too close to the contaminated area, then a crossing graft can be placed from the left subclavian artery, above the area of the fistula.

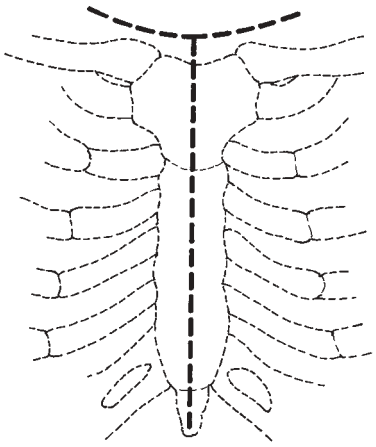
Neurologic sequelae due to division of the innominate artery, in contrast to division of the common carotid artery, are extremely rare.[4] Elective division of the brachiocephalic artery during mediastinal exenteration under controlled circumstances has proved safe.[5] Our preferred method of management is to resect the damaged artery and oversew both ends with double layers of fine vascular suture material (Figure 27-3*C*). Both arterial stumps are buried in substantial healthy tissue, using either thymus or strap muscle as available. Simple double ligation of the artery, even with division, is dangerous, since later erosion of these large ligatures may occur with secondary hemorrhage. If an arterial graft is used, then it is probably wise to advance an omental pedicle substernally to protect the graft.

Tracheal resection is usually unnecessary if the trachea is otherwise normal, since the erosion occurs at the stomal margin. A new stoma is made proximally at an appropriate level (second or third tracheal rings), since the patient usually still requires ventilation (see Figure 27-3*C*). A tube that is long enough to pass *beyond* the previous stoma is placed. It may be necessary to fashion a tube using an endotracheal tube *with a large-volume cuff* and a movable tracheostomy flange to obtain sufficient length. The margins of the old stoma are debrided conservatively and the stoma closed with pedicled or medialized strap muscle. If for any reason there is concern about placing a new tracheostomy tube, then the patient may be managed with an endotracheal tube for a time. If needed, a second tracheostomy may be established later, when the operative field is well sealed. In this case, the site for the possible later tracheostomy insertion should be marked with a single silk suture on the tracheal wall.

Erosion from Tracheostomy Tube Cuff

Erosion of the anterior tracheal wall overlying the brachiocephalic artery, either due to necrosis from a high-pressure cuff or ulceration by an angulated tip of a tracheostomy tube, is now rarely seen (see Figure 27-1*B*). In these cases, the stoma is usually at a more nearly correct level, high in the trachea. In emergency, it is not possible or advisable to expose the fistula by finger dissection or by cutting down to it in order to place a tamponading finger on the artery, since the fistula usually occurs 1 to 4 weeks after tracheostomy. The pathology lies entirely within the mediastinum and *not* at the stomal level. This is also the case in stent induced hemorrhage. Bleeding is controlled initially by overinflating the tracheostomy cuff and then by inserting an endotracheal tube and inflating the cuff with high pressure to tamponade the fistula (see Figure 27-2*B*).

Exposure is obtained as described previously. If the lesion has resulted from erosion by a high-pressure cuff, then there is usually circumferential damage to the trachea at the cuff level (Figure 27-4*A*). Control of the artery is achieved as previously described and the damaged segment of artery is excised.



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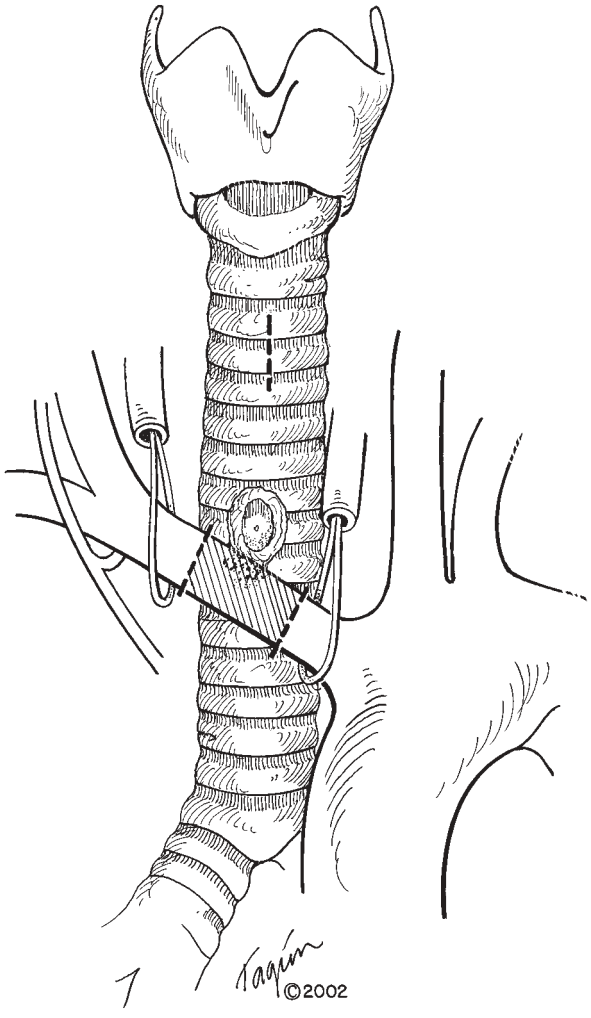

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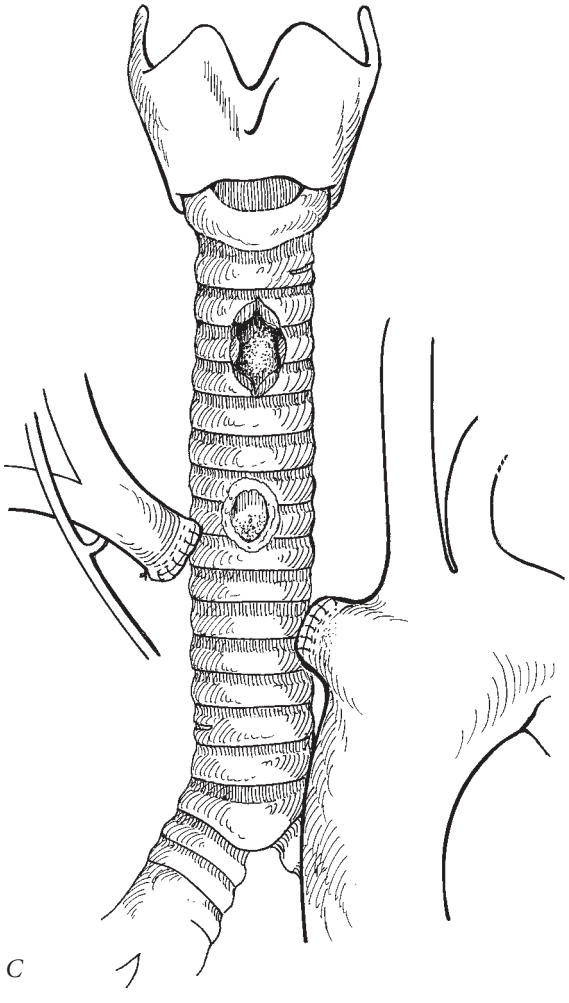

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FIGURE **27-3** *Surgical management of erosion by tube at the stoma.* A, *Incision for exploration includes collar incision and sternotomy. Sternotomy should either be complete, as shown, or angle into the right third interspace. Either will provide the necessary exposure for arterial control.* B, *If a tube is still present in the stoma, it is replaced by an oral endotracheal tube. The artery is carefully dissected proximal and distal to the lesion and controlled with tourniquets or vascular clamps. Only then is the injured segment dissected from the trachea and stomal margin. The damage is resected. If ventilation will be needed postoperatively, then a new stoma is made proximally* (dashed line) *or an endotracheal tube is used.* C, *The proximal and distal arterial stumps are sutured closed. The arterial ends are carefully covered with well vascularized tissue, such as strap muscle or thymic lobes. If there is no other tracheal injury, the offending stoma is debrided and closed with a pedicled sternohyoid muscle flap. In this drawing, a new stoma has been placed at a conventional site. The cuff will preferably not rest directly against the old stomal closure.*

Here, however, the circumferentially damaged segment of the trachea is also excised (Figure 27-4*B*). Healthy tissue is placed over the sutured arterial stumps and the tracheal suture line. If the patient is still on a ventilator, as is likely, the tube cuff for continued ventilation should be positioned either well above or, more likely, well below the anastomotic line, in order to avoid either pressure or inflammation at the level of the tracheal anastomosis. Muscle is pedicled over the tracheal anastomosis to seal it from the tracheostomy. If necessary, the stoma may be sealed with a pedicled strap muscle, and an endotracheal tube employed with proper positioning of the cuff remote from the anastomosis. Tube position is checked bronchoscopically.

Postoperative Fistula

Prevention of postoperative hemorrhage from the innominate artery following tracheal resection and reconstruction has been discussed in Chapter 13, "Tracheal Fistula to Brachiocephalic Artery." Should a

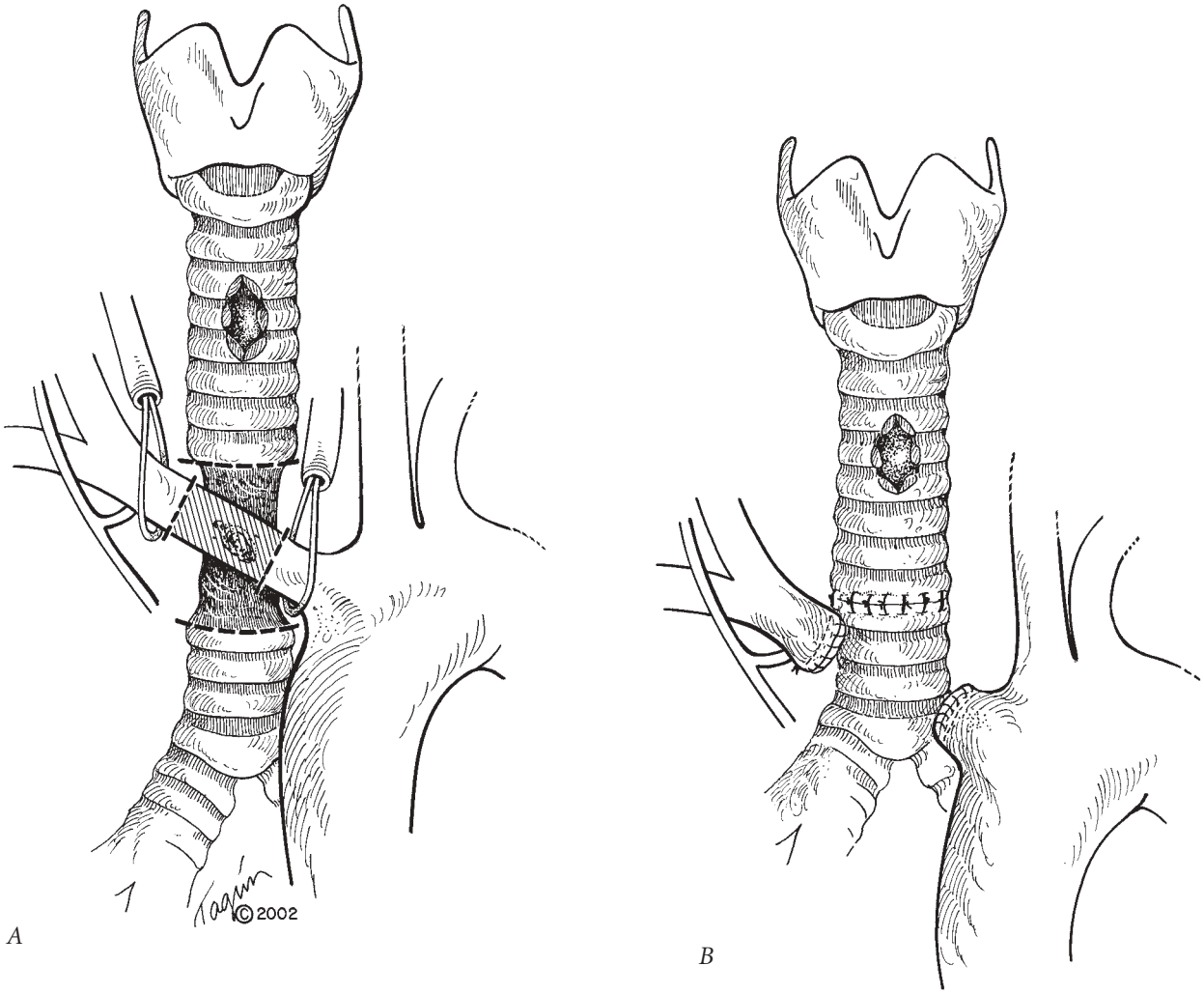

FIGURE **27-4** *Surgical management of a fistula due to tracheal cuff injury. Exposure is the same as previously described.* A, *Arterial control is obtained, the artery divided above and below the fistula, and proximal and distal stumps sutured closed. The circumferentially damaged segment of trachea is dissected and excised along with the damaged vessel.* B, *End-to-end anastomosis of the trachea is completed. Arterial stumps are buried in healthy tissues, and tissues are arranged to fill dead space. The prior stoma remains available for the ventilating tube, if necessary. The cuff must not be placed in direct contact with the suture line.*

hemorrhage occur after tracheal resection and reconstruction by the anterior approach, then an endotracheal tube with the cuff tightly inflated is used to occlude the fistula. Exposure must be obtained promptly as described, and control established. In most cases, the damaged artery is best resected and managed as advised earlier. In a rare case, a tiny fistula due to erosion by an adjacent suture may be cleanly debrided and the artery closed with vascular suture material. If this is to be done, the artery must otherwise be in excellent condition and, following arteriorrhaphy, a second layer buttress with a pedicled strap muscle is advisable. This not only helps to buttress the repaired artery but it also interposes healthy muscle between the site of arterial injury and the site of repair of the tracheal anastomosis. Any defect in the anastomosis must also be primarily repaired. The muscle flap also serves to seal this repair.

Even more disastrous, and likely to be promptly fatal, is a late hemorrhage from an artery that has been exposed after separation of a tracheal anastomosis, with placement of a tracheostomy tube or a T tube. If possible, the injured arterial segment is excised, and the sutured stumps are protected by flaps of healthy tissue. This situation may well present an indication for use of pedicled omentum. Most anastomotic separations are due either to devascularization or excessive tension. Further tracheal repair may not be possible. The airway may be managed initially by a properly positioned endotracheal tube that spans the defect, since ventilatory support will likely be needed. Later, the airway is maintained with an extra-long T tube.

Hemorrhage from erosion into the adjacent brachiocephalic artery, at the margin of a mediastinal tracheostomy, requires excision of the injured segment of artery. It seems unwise to attempt to reconstruct the artery in this essentially septic circumstance. A pedicled omentum will be useful. Such surgical catastrophes have been largely eliminated by the precautions described in Chapter 13, "Tracheal Fistula to Brachiocephalic Artery."[5]

References

1. Grillo HC. Congenital lesions, neoplasms and injuries of the trachea. In: Sabiston DC, Spencer FC, editors. Gibbons's surgery of the chest. 4th ed. Philadelphia: WB Saunders; 1983. p. 274–6.
2. Wright CD. Management of tracheoinnominate artery fistula. Chest Surg Clin North Am 1996;6:865–73.
3. Cooper JD. Tracheo-innominate artery fistula: successful management of 3 consecutive cases. Ann Thorac Surg 1977;24:439–47.
4. Brewster DC, Moncure AC, Darling RC, et al. Innominate artery lesions: problems encountered and lessons learned. J Vasc Surg 1985;2:99–112.
5. Grillo HC, Mathisen DJ. Cervical exenteration. Ann Thorac Surg 1990;49:401–9.

Reconstruction of the Lower Trachea (Transthoracic) and Procedures for Extended Resection

Hermes C. Grillo, MD

In describing the anterior approach to the trachea (Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"), I noted that the majority of benign strictures of the trachea at any level can be resected through a cervicomediastinal incision, using only partial division of the sternum and working behind the great vessels. Midtracheal tumors that are largely intraluminal, especially if benign or without esophageal or mediastinal invasion and not of extended length, may be dealt with similarly by that approach. For tumors of the lower third of the trachea where it is advisable to dissect a wider lateral margin, where a greater extent of trachea may have to be resected, or where hilar mobilization may be required, I prefer a transthoracic approach. This is described in detail in the first part of this chapter. An additional section expands upon the use of alternative approaches to the lower trachea, including median sternotomy and cervicomediastinothoracic or "trapdoor" incision, indicating their applications and limitations. Basic incisions for these approaches are described in Chapter 23, "Surgical Approaches."

Transthoracic Tracheal Resection

Incision

Right posterolateral thoracotomy in the fourth interspace or through the bed of the resected fifth rib provides wide access to the lower two-thirds of the trachea, the carina, the right hilum, and the esophagus (see Figure 23-8 in Chapter 23, "Surgical Approaches"). Options for this incision can be broadened if the right arm is kept free of arterial or venous lines and is prepared and draped into the field so that it may be swung backward and forward. The anterior chest wall, which may include the neck, can thus also be prepared and draped. However, since we learned that laryngeal release does not transmit relaxation for lower tracheal reconstruction, this is now rarely done.

Intubation

The endotracheal tube preferred in this approach is an extra-long one, initially devised by Dr. Roger Wilson (see Figure 24-1 in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended

Resection"). It consists of a flexible armored endotracheal tube, with its adaptor removed, connected by a short length of metal tubing to a proximal generous segment of Rusch tube or other endotracheal tube. The thin metal sleeve avoids significant reduction of the lumen. Dr. Paul Alfille describes a similar alternative tube (see Chapter 18, "Anesthesia for Tracheal Surgery"). The length of the tube is such that it easily reaches from the mouth into the left main bronchus. The flexible portion conforms to angulation of the bronchus. The tube diameter is selected in accord with the size of the bronchus. An extension to the sidearm attached to the cuff facilitates inflation and deflation. Double-lumen tubes are cumbersome and interfere with this type of surgery. The endotracheal tube may be placed in the left main bronchus with the aid of a flexible bronchoscope prior to thoracotomy, to facilitate collapse of the right lung. Because of its flexibility, the tube may slip out of the left main bronchus when the patient is positioned laterally; therefore, the tube position should be rechecked bronchoscopically just prior to thoracotomy. A flexible pediatric bronchoscope should be available since the tube may not accept an adult bronchoscope. Should it seem impossible to place the tube in the left main bronchus, initial intrathoracic dissection may be carried out with gentle compression of the right lung. The tube can then be coaxed into the left main bronchus while a finger compresses the membranous wall of the right main bronchus.

Mediastinoscopy

Mediastinoscopy is not necessary for benign tumors or tumors of low malignancy. The pretracheal plane can be adequately freed to the neck by intrathoracic blunt dissection. Mediastinoscopy is also not routinely necessary or advantageous for all malignant tumors, since the finding of paratracheal nodal metastases will not preclude resection of a tracheal tumor. In the case of a tracheal tumor, mediastinal nodes may be considered as N1 nodes. A principal value of mediastinoscopy is in additional assessment of a neoplasm of questionable resectability. Mural involvement and invasion of mediastinal structures may often be better determined in this way than by imaging alone. If question remains, thoracotomy is the final mode of evaluation.

Dissection

After assessment of the extent of tumor by palpation and inspection of the trachea, lung, pleura, and lymph nodes, the pleura is opened over the trachea (Figure 28-1). The pleura is included in the specimen if adherent to tumor. The azygos vein is doubly ligated and divided. An adherent segment of vein is left attached to tumor. The trachea is dissected circumferentially below the tumor if there is room, and sometimes above it, although the upper portion of the tracheal dissection may be deferred until the trachea is divided below the tumor. Completion of as much peritracheal dissection as can be accomplished with ease prior to tracheal division simplifies the operation. The pretracheal avascular plane of the proximal uninvolved trachea is bluntly dissected from below, well into the neck, to facilitate tracheal devolution into the thorax, if this has not already been effected by prior mediastinoscopy. If it is clear that hilar mobilization and intrapericardial release will be needed, these are more conveniently completed prior to dividing the airway. These techniques are described later.

Resection and Reconstruction

In preparation for distal tracheal transection, lateral traction sutures (2-0 Vicryl) are bilaterally placed, vertically in the midlateral tracheal wall, about one ring below the anticipated level of division (Figure 28-2). The sutures pass into the lumen and surround one ring. If a smaller bite of tissue is taken, the suture may subsequently tear out as traction is applied. The endotracheal tube cuff is deflated transiently while placing the sutures. The endotracheal tube is next withdrawn to a point above the lesion. The trachea is opened transversely, usually in an annulus on the side of the trachea away from the tumor base. A nerve hook is useful to expose the lumen through the partial transection, to see whether the level is distant enough from

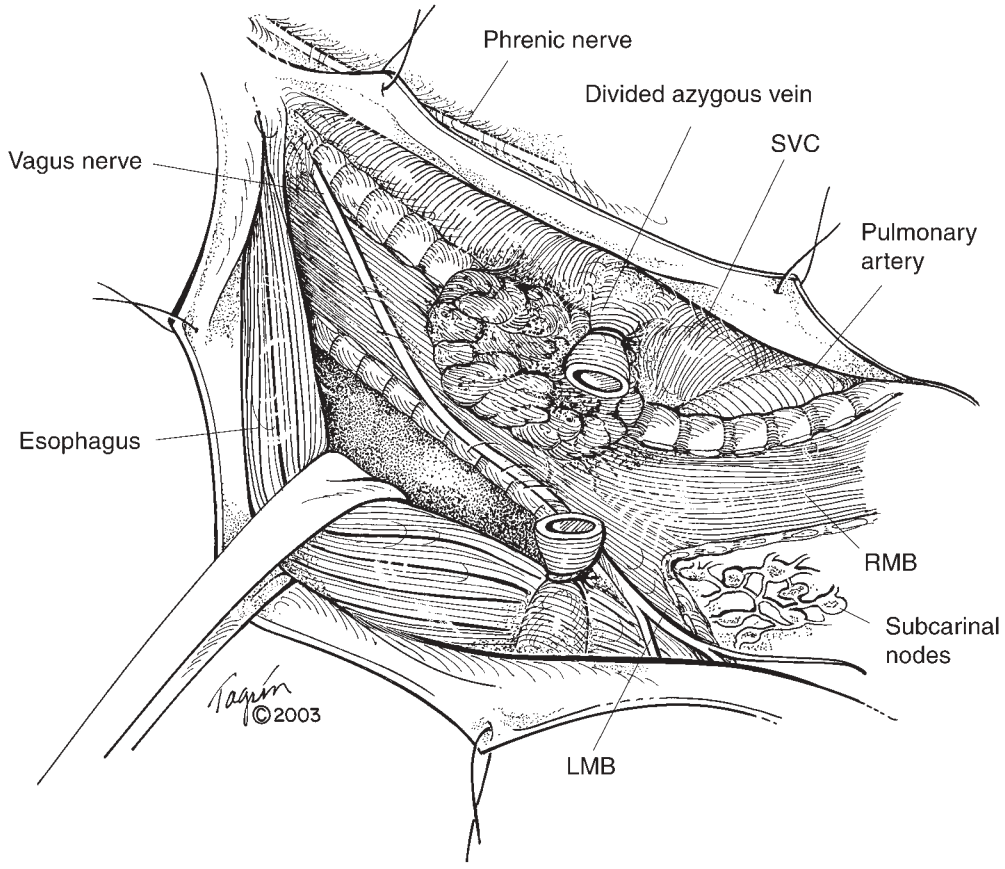

FIGURE **28-1** *Exposure of the mid and lower trachea via right thoracotomy. A standard posterolateral incision is used. The pleura is incised over the lateral tracheal wall from the apex of the hemithorax to a point below the carina. The azygos is doubly ligated and divided. A segment of vein may be left attached to a tumor that seems to involve it. The phrenic nerve lies on the superior vena cava, which is contiguous with the trachea anteriorly. The esophagus is contiguous posteriorly. The extent of dissection is determined by the location and extent of the lesion. Some right lateral tracheoesophageal arterial branches are divided as trachea is dissected free. The right vagus nerve travels distally obliquely over the trachea to assume a paraesophageal course below the carina. Numerous branches are encountered to the trachea, carina, and esophagus (and to the cardiac plexus). Bronchial arteries, lymphatics, and lymph nodes are encountered at the carina. The superior bronchial artery crosses the esophagus to reach the carina and right main bronchus. Paratracheal lymph nodes and those at the right tracheobronchial angle are not shown, for clarity. The apical segmental ramus of the principal upper lobe arterial trunk is adjacent to the upper lobe bronchus. The left main bronchus is easily accessible in this exposure. The arch of aorta arcs over the left main bronchus, and the left recurrent laryngeal nerve loops up beneath the arch in just this location to assume a course in the groove between the left posterior tracheal wall and esophagus. The right recurrent nerve branches from the right vagus nerve high in the thorax to travel beneath the subclavian artery. The right vagus nerve is often divided to provide improved surgical access and to facilitate later tracheal reapproximation. LMB, RMB = left, right main bronchus; SVC = superior vena cava.*

tumor. If not, the level is moved distally, replacing the traction sutures, if necessary. If the transection is to be close to the carina, lateral traction sutures are placed in the midlateral proximal walls of the right and left main bronchi.

The transected trachea is intubated across the operative field with a flexible armored tube. Usually, the tube is advanced into the left main bronchus to collapse the right lung, to provide optimal exposure during the remainder of dissection and anastomosis. Oxygen saturation is monitored throughout, and if saturation falls, the right pulmonary artery may be gently clamped to eliminate the shunt through the collapsed right lung. This is very rarely needed. The tube is held in place either by arranging the lateral traction sutures appropriately over the tube or by having a second assistant hold these sutures in one hand while steadying

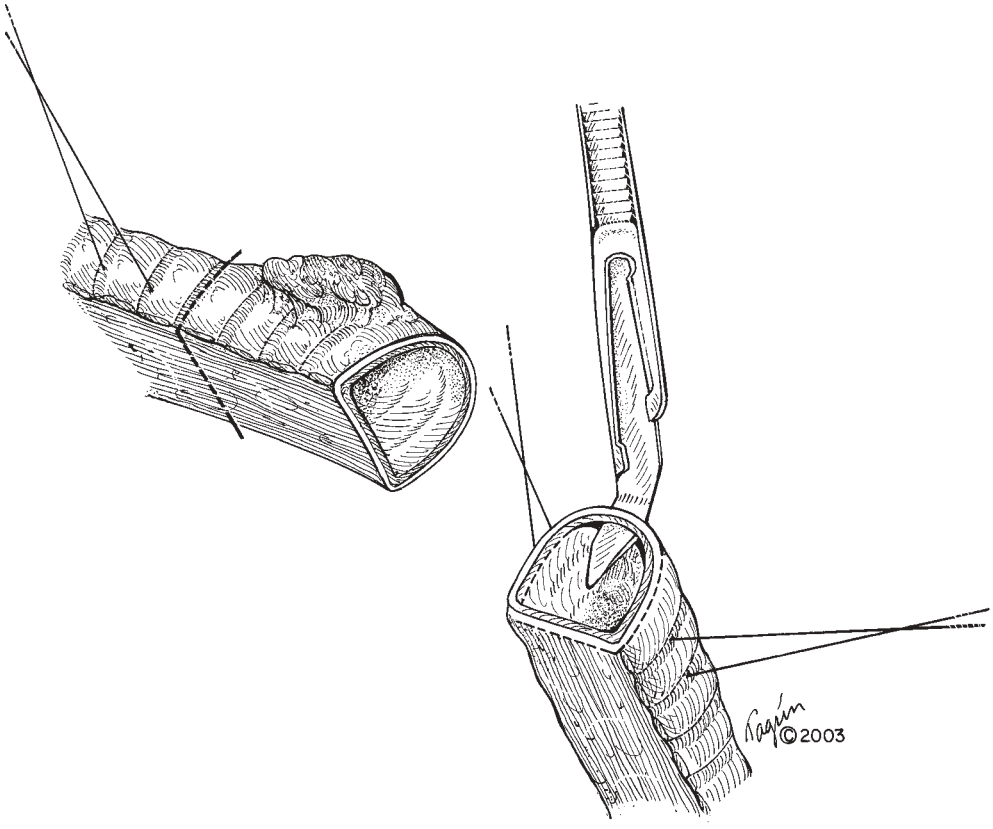

FIGURE **28-2** *Resection of a tumor low in the trachea. The trachea has been divided distal to the tumor. Since it lies close to the carina, intubation across the operative field distally was made into the left main bronchus. This also collapses the right lung to give the best exposure. For this reason, the endotracheal tube is preferably placed in the bronchus rather than distal trachea, even if a greater length of distal trachea is available. The endotracheal tube is intermittently removed to simplify surgical maneuvers. A thin sliver of distal tracheal margin is taken for histologic frozen sections. To eliminate any possible question about the significance of positive findings, the specimen is preferably excised from the margin of trachea that is to remain. The part of the ring specimen that lies closest to tumor is marked with a fine suture.*

the tube with long forceps. Since the endotracheal tube and connecting anesthesia tubing lie at a margin of the incision, they do not interfere with dissection. A tube may be sutured into place or inserted via bronchotomy, but I prefer not to further injure the trachea or bronchus. Also, moving the endotracheal tube about, as dissection and reconstruction proceed, is helpful. High-frequency ventilation may be used with a catheter placed in the left main bronchus, or, if insufficient, with a bifid catheter into the right and left sides. With high-frequency ventilation, the right lung is not fully inflated and so it hardly impedes the surgical progress. The technique is a bit noisy, blows blood about, and, by itself, occasionally does not maintain satisfactory oxygenation. Since the method has not shown enough advantages, and because of our wholly satisfactory experience with cross-table ventilation, we use high-frequency systems only occasionally for augmentation or for special problems of carinal reconstruction, rather than by preference.

Division of the trachea distal to the tumor allows the specimen to be elevated, using Allis forceps, facilitating completion of dissection. Care must be taken not to injure the left recurrent laryngeal nerve as it lies on the aortic arch just beyond the left posterolateral tracheal wall. If a portion of esophageal wall is resected, repair of that structure is done as soon as the tracheal specimen is removed. Midlateral traction sutures are placed in the upper tracheal segment just prior to proximal division. After removal of tumor,

horizontal slivers of tissue from proximal and distal ends of the remaining trachea, taken from the side closest to the tumor, are sampled for frozen sections (see Figure 28-2). If positive margins indicate the need for further resection, the surgeon must carefully judge the limit of tension to be accepted as being safe for the anastomosis. On occasion, a microscopically positive margin must be accepted as preferable to risking anastomotic dehiscence. Judgment of ease of approximation may be made by having the anesthesiologist flex the patient's neck (remembering the patient's orientation in the lateral thoracotomy position), while the surgeon and the surgeon's assistant each draw together the lateral traction sutures in proximal and distal tracheal segments on both sides of the trachea. It may be easily forgotten that cervical flexion (chin toward sternum) devolves the trachea into the mediastinum, even in the lateral thoracotomy position. If tension for approximation seems excessive, intrapericardial release may then be added, if not already performed. Limited blunt dissection over the anterior surface of the left main bronchus may also be helpful. The blood supply of the bronchi and carina must be respected (see Chapter 1, "Anatomy of the Trachea"). With tumors that are seen bronchoscopically to lie just above the carina, the surgeon must be prepared for the possibility that frozen sections will impel the need for carinal resection and reconstruction, a procedure of considerably greater complexity.

Reconstruction is performed in a manner almost identical to that described for the upper trachea (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). For anastomosis in adults, 4-0 coated Vicryl sutures are used, 5-0 in infants. I place all anastomotic sutures before tying any. The first suture placed, however, is the most "posterior" *with respect to the operative field*. Thus, in right thoracotomy, the suture actually lies in the left lateral tracheal wall, usually just anterior to the level of the left-sided lateral traction suture. The true anterolateral sutures (cartilaginous wall) are placed first, progressing anteriorly from that initial suture in the lateral wall (Figure 28-3). The sutures are clipped to the drapes on the anterior portion of the incision, almost as described for upper tracheal reconstruction, but instead ranging from caudad for the most "posterior" suture to cephalad for the last true anterior wall suture (cartilaginous wall), just in front of the *right* lateral wall traction suture. This reversal simply allows the successive sutures to be placed and to lie more easily. The surgeon's assistant exposes the trachea for placement of each successive suture, holding the previously placed sutures out of the way with the nerve hook. Tension on the trac-

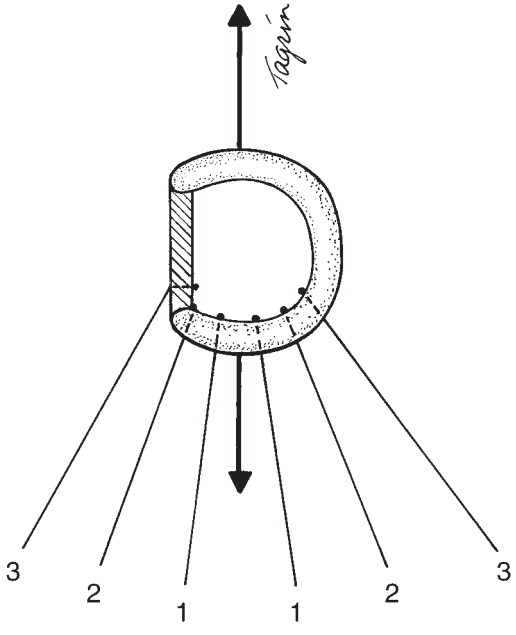

FIGURE **28-3** *Order of suture placement for intrathoracic tracheal anastomosis. The diagram represents the proximal end of the divided trachea. The distal end is a mirror image. The* heavy lines *indicate lateral traction sutures, with the right side of trachea upper and the left side lower (located deepest in the wound). Anterolateral sutures are placed first, beginning anterior to the left-sided traction suture and progressing as shown. After all these sutures have been placed up to the right-sided traction sutures, the posterior sutures are placed similarly, from the deepest to the most superficial.*

tion sutures may help exposure. The process is now repeated for the "posterior" sutures, commencing just behind the left lateral traction suture (the deepest part of the wound) and progressing across the membranous tracheal wall to that part of the right lateral cartilaginous wall behind the right-sided traction suture. These sutures are clipped in order to the drapes at the posterior portion of the incision.

The cross-table endotracheal tube may be removed intermittently during placement of sutures. After all sutures are in place, the tracheobronchial tree is suctioned thoroughly and the long endotracheal tube re-advanced from above into the left main bronchus. The patient's neck is flexed and held in position by the anesthesiologist's assistant, propping it in position with folded blankets or pillows. Extreme flexion is always to be avoided and certainly not maintained after anastomosis is completed. The surgeon and assistant each simultaneously draw together the lateral traction sutures on their respective sides of the trachea until the ends of the trachea are in contact. This is accomplished by throwing the first two loops of a surgeon's knot (with the double strands) and pulling the ends together, taking care not to lock the suture against either end of the trachea. The traction sutures are tied, one set at a time, while the other set is held firmly approximated. They are tied as double strands. Their bulk has not led to complications, although the sutures pass around a ring and through the tracheal lumen. Left in place, these sutures lessen tension on the anastomosis, not only during its completion but during early healing as well. The ends of the tied traction sutures are held with clamps until the anastomosis is completed, tested, and tissue for second-layer closure has been drawn beneath the anastomosis. They remain useful for slight rotation and elevation of the trachea up to that point. As in the anastomosis of the upper trachea, the sutures are tied in reverse order of their placement; namely, from the most superficial to the most posterior (or deepest), with respect to the incision. Excess suture material is cut after each knot is tied.

A second layer of tissue is routinely placed over all intrathoracic anastomoses, not only to assist in sealing the anastomosis but also to interpose tissue between the suture line and major vessels, which often lie adjacent to the anastomosis. I believe that this provides protection against bronchovascular fistula, a potentially lethal complication. For this purpose, I prefer a *pedicled pericardial fat pad*. If this is not anatomically available, a pedicled pleural flap is used. Brewer and Bai popularized the use of the pericardial fat pad to buttress bronchial closures, pedicling it on the usually prominent middle pericardial branch of the internal thoracic artery (Figure 28-4*A*).[1] If needed, greater length may be obtained by pedicling the pad superiorly on a branch of pericardiacophrenic artery, elevating just enough length to obtain the needed circumferential wrap. Excessive dissection risks losing its vascularity. Elevation of the flap is commenced at the diaphragm, using cautery and very carefully preserving proximal blood supply. The phrenic nerve must be protected from injury. The flap is drawn under the anastomosis (Figure 28-4*B*) but is not immediately sutured. Anastomotic integrity is checked by retracting the endotracheal tube into the trachea, proximal to the anastomosis, and expanding the lung after the thoracic cavity has been filled with warm saline solution. The anastomosis should easily accept 30 to 35 cm H_2O ventilatory pressure without leakage. The anastomosis should also be visualized with a flexible bronchoscope passed through the endotracheal tube. The second-layer wrap is now meticulously sutured to the trachea and to itself, completely covering the anastomosis and with the tissues closely approximated. The chest is closed in routine fashion, leaving two thoracotomy tubes in place for pleural drainage.

Pedicled omentum is used in patients who have the added hazard of prior significant irradiation. The technique of advancement is described in Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation." Since lateral tracheal and bronchial pedicles are carefully preserved to maintain blood supply, the space remaining around the completed anastomosis may be minimal. An omental pedicle, even when thinned down, may be difficult to pass around the anastomosis. In order to facilitate passage of the omental pedicle through this limited space, the pedicle is first introduced into a short length of broad Penrose drain and fixed at its tip with a suture. The drain is then pulled gently through the narrow aperture, the suture cut, and the drain slid off the now properly positioned pedicle.

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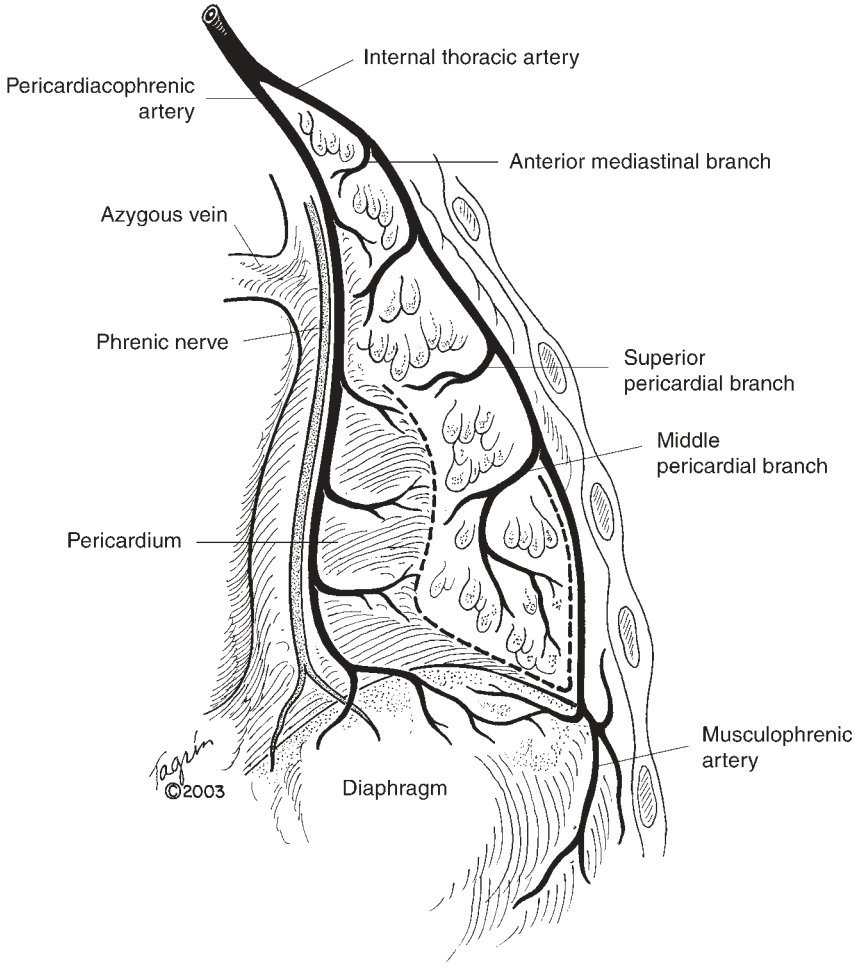


B

FIGURE **28-4** *Buttressing a tracheobronchial anastomosis with pedicled pericardial fat pad.* A, *The line of dissection* (dotted line) *preferably starts below the large middle pericardial branch of the internal thoracic artery, follows the anterior fold of mediastinal pleura into the costophrenic sinus, and then back and up on the pericardium along the border of the subpleural fat pad. The pad is raised from the pericardial surface, dividing small branches of musculophrenic and pericardiacophrenic arteries in this course.* B, *The elevated pedicle of pericardial fat is passed beneath the anastomosis. The fragile tissue must be handled with delicacy and without tension on the pedicle. It is sutured with fine (4-0) sutures to the airway proximal and distal to the anastomotic line and to itself. The fat pad is thus packed against the suture line. It must be viable tissue.*

Pedicled intercostal muscle should not be used to wrap anastomoses circumferentially. Not only is this flap bulky and hence awkward to use, but if the costal periosteum is not meticulously removed from the superior and inferior margins of the flap, a circle of bone—a veritable obstructing grommet—can form around the anastomosis. However, periosteal removal without injury to the flap's blood supply may be difficult. In one early case where periosteum was not removed, a reoperation was required months later to excise obstructing bone formation. This unique problem only follows *circumferential* wrapping.[2] The intercostal pedicle flap has many excellent applications, such as buttressing the main bronchial closure or esophageal closure after perforations. The periosteum need not be removed in such cases since the wrap is not circumferential.

If the patient requires ventilation following intrathoracic reconstruction of the trachea, the endotracheal tube is changed in the operating room to a conventional one, and the cuff is carefully positioned with a flexible bronchoscope to lie above the anastomosis (if possible) and not in contact with it. A well-constructed anastomosis should tolerate pressures necessary for ventilation, but the inflammation caused by a cuff lying in contact with an anastomosis should be avoided, even though a low-pressure cuff is used.

Alternative Approaches

Cervicomediastinothoracic Resection

A cervicomediastinothoracic incision allows wide access to the entire upper airway from the hyoid bone to the carina, if necessary.[3] This consists of a collar incision and a vertical sternotomy which angles into the right fourth interspace (see Chapter 23, "Surgical Approaches"). The cutaneous component sweeps beneath the breast, but the breast and the underlying pectoralis muscle are elevated as a single flap up to the fourth interspace. The interspace is entered over the top of the fifth rib (see Figure 23-3*B* in Chapter 23, "Surgical Approaches"). Initially, I used this approach electively for lower tracheal tumors, but it proved to be unnecessary as a routine approach to the lower trachea. It has occasional usefulness for special problems. If such access might be needed, the patient is placed on the table with a long roll beneath the patient's back just to the right side of the midline. The patient's neck is extended with an inflatable bag beneath the shoulders. The right shoulder is abducted, with the elbow flexed and resting at the side, but supported to avoid brachial plexus or ulnar nerve injury. The right chest is accessible as far as the posterior axillary fold. The right arm may be draped into the field so that it can be swung back and forth, but this is usually not necessary. Exploration may commence with a cervical incision, enlarged by partial sternal division. When the extent of the pathology is determined, and if wide access to the lower trachea becomes necessary, the thoracic portion of the incision is completed. I have found this approach useful on rare occasions for a very long midtracheal tumor that required extensive tracheal dissection, laryngeal release, and right intrapericardial mobilization. It was also used, for example, in a patient who had undergone elsewhere two previous failed attempts at tracheal reconstruction, including one operation under cardiopulmonary bypass, and who also had scarring from mediastinal sepsis. Furthermore, in the same patient, the brachiocephalic artery had common origin with the left common carotid and was fused to the trachea by scar.

Transmediastinal Resection

A combination of a collar incision, for the best access to the cervical trachea, with a full sternotomy has merits (see Figure 23-3*A* in Chapter 23, "Surgical Approaches"). It does not provide any better access to the upper trachea than partial sternotomy but gives much improved access to the lower trachea and carina.[4–6] The pericardium is opened widely anteriorly and posteriorly between the superior vena cava and the aortic arch (see Figure 23-7 in Chapter 23, "Surgical Approaches"). Working beneath the brachiocephalic artery and above the right pulmonary artery, a deep quadrilateral space is opened, which permits access to

the lower trachea and carina. Exposure may be difficult and does require vigorous retraction of vessels. Intraluminal tracheal tumors without invasion into adjacent structures, such as the esophagus, may be removed through this approach without undue difficulty. Reconstruction may be difficult, however, if a complex carinal repair is necessary. The principal advantage of the approach is avoidance of the morbidity of thoracotomy. Right intrapericardial hilar mobilization may be added to gain length (see below). Cardiac retraction, necessary for transpleural exposure of the left hilum, is usually not tolerated. If left hilar mobilization is also necessary, a "T" into the left fourth interspace may be added. However, Cameron D. Wright (personal communication, 2001) found that intrapericardial release of the left hilum, by incision of the pericardium beneath and around the inferior pulmonary vein, can be effectively and preferably accomplished through the widely opened anterior pericardium with gentle cardiac retraction (see "Intrapericardial Release," below). Right-sided hilar mobilization is better done transpleurally, since access to the inferior vein intrapericardially is limited by the right atrium and vena cava. The patient is also in optimal position for laryngeal release, potentially useful if the resection begins high on the trachea. The principal use of this incision, in my experience, has been for removal of a lengthy tumor of the midtrachea or of the lower trachea, where it is desirable to avoid lateral thoracotomy. Effective control of thoracotomy incisional pain by epidural analgesia makes this a less important consideration. I do not usually consider median sternotomy to be the incision of choice for lower tracheal or carinal resection, although some surgeons prefer its use. Median sternotomy can be extended by opening the fourth right interspace across the right side of the sternum, if sternotomy is found to be insufficient. Each extension adds to morbidity. Bilateral transverse thoracotomy has been confined to special cases of carinal resection and reconstruction (see Chapter 29, "Carinal Reconstruction").

Extended Resection of Lower Trachea

Cervical flexion and blunt dissection of the avascular pretracheal plane are routine components of lower tracheal resection and reconstruction, for all but the most minimal lengths of excision. *Flexion of the neck* to permit the cervical trachea to descend into the mediastinum, thus lessening tension on the anastomosis, is basic to accomplishing lower tracheal resection and reconstruction. This may easily be forgotten in a transthoracic approach. The length gained may not be as great as that obtained for upper tracheal resection, but is always significant. *Freeing the pretracheal plane* by blunt dissection appears to facilitate this descent. The carina may also be pulled up to a small degree, minimally improved by blunt dissection of analogous planes anterior to the main bronchi. The subaortic location of the left main bronchus prevents more than a small degree of carinal ascent. The proportional length of resection permitted by these basic steps varies, as in upper trachea, with age, body build, disease, and prior treatment. Between one-third to one-half of trachea is usually, but not always, resectable without producing excessive tension on the anastomosis. Judgment and experience appear to be the only final guides to avoiding the potentially fatal consequences of excessive resection.

Laryngeal release (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection") can be a useful adjunct in lengthy upper tracheal reconstruction, providing a small but sometimes critical increment. It will also provide some length for lower resection, if the resection extends above the midtrachea. However, laryngeal release does not translate to the lower trachea or carina. This fact became evident with experience and was supported by experiment.[7] The lateral attachments of the trachea, including critical vascular supply, prevent this. What may be done additionally to extend resection?

Right hilar release by dissection of vascular structures, with *division of the inferior pulmonary ligament*, assisted by blunt dissection of the avascular plane anterior to the left main bronchus, provides some mobility.[8] Bronchial blood supply must be preserved. The carina ascends, rocking somewhat to the right. The ascent of the right hilum is checked principally by the pericardial attachments of the inferior

pulmonary vein. Much more is accomplished by intrapericardial release. Where anastomotic tension is likely to result, I proceed *directly* to this step with minimal dissection of hilar structures before tracheal resection. Division of the pulmonary ligament provides access to the pericardium, although it adds little mobility by itself.

Intrapericardial release, especially release of the lowest part of the hilum, is widely applicable for extended lower tracheal resection, carinal resection, and also for left main bronchial sleeve resection.[8] It may be done on the right, left, or bilaterally, depending on need and access. In resection of a lower tracheal tumor, which is performed most frequently through right thoracotomy, right-sided release is often used. The pericardium is cleared anterior to the pulmonary vessels behind the phrenic nerve, with care to avoid injuring the nerve. The (inferior) pulmonary ligament is divided. A U-shaped incision is made in the pericardium in front, beneath, and behind the inferior pulmonary vein (Figure 28-5*A*). The longitudinal frenum, which attaches the pericardium to epicardium, is freed above to the entry point of the inferior pulmonary vein into the atrium, and below for a centimeter or two. Hilar mobility can be increased by carrying the pericardial incision up to the top of the hilum, anteriorly and posteriorly (Figure 28-5*B*). In effect, the pericardium is completely circumcised. Between 1 to 2 cm of hilar elevation are obtained. In opening the pericardium posteriorly, I endeavor to save a pedicle of mediastinal tissue to the hilum, which lies posterior to the pericardium and below the main bronchus. A small Penrose rubber drain is passed around this pedicle for retraction and the pericardial incision is extended upwards beneath the pedicle. Although this maneuver may not indeed be critical, the effort is directed to conserving bronchial blood supply and pulmonary lymphatics. This may be important in carinal resection. Pericardial closure is not necessary after hilar release.

Left-sided pericardial release finds special value in complete or subtotal left main bronchial resection —the only isolated *bronchial* sleeve resection that may produce anastomotic tension. Bilateral intrapericardial release is more effective in some cases of extended tracheal or carinal resection, but bilateral access can be difficult, as noted. Cardiac retraction, necessary via sternotomy for left-sided access to the lower hilum, is not tolerated by most patients.

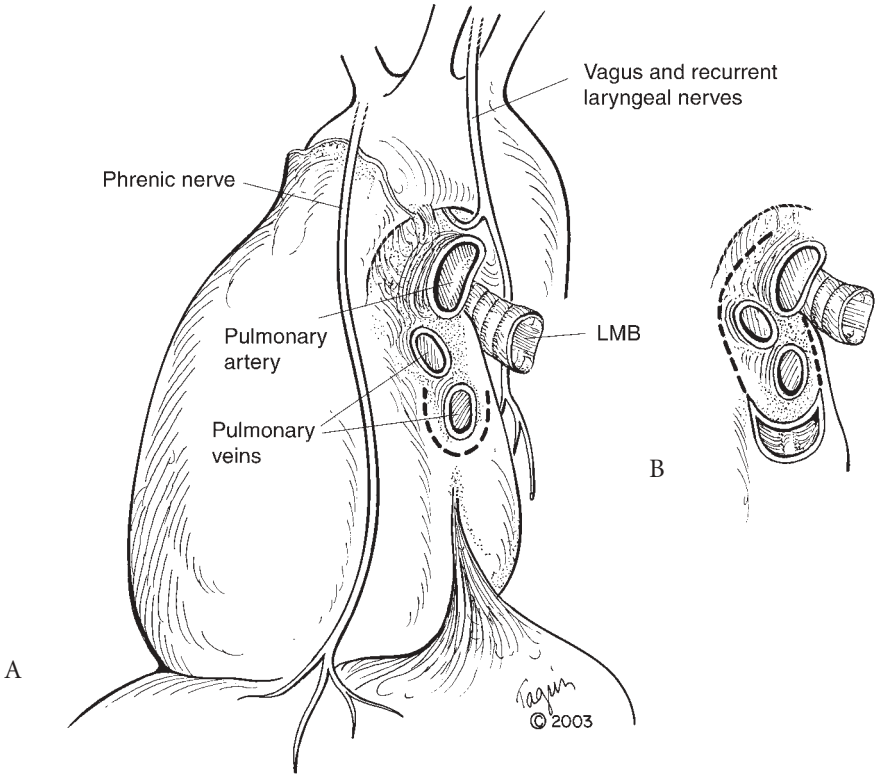
Left hilar mobilization may be accomplished transsternally, directly via wide pericardiotomy, as discovered by Dr. Cameron D. Wright at Massachusetts General Hospital (Figure 28-5*C*). Incision is made below and beside the inferior pulmonary vein *inside* the opened pericardium, and the frenum from the inside of the pericardium to epicardium is also divided. The incision through the pericardium adjacent to the vein must be very close to the vein, since the phrenic nerve lies just anterior to the vein outside of the pericardium. The nerve, of course, can not be visualized from within. If required, the phrenic nerve can be visualized by dissecting between the outer surface of pericardium and the left mediastinal pleura.

Posterior opening of the left pleura from right thoracotomy has been suggested but provides unsafe access. A left-sided "T" from sternotomy is feasible. Bilateral thoracotomy, elected for this purpose alone, seems excessive. Furthermore, the aorta continues to tether the left main bronchus to a degree in any case.

Other Techniques

In the anatomy, laboratory radical techniques for extreme anatomic mobilization were explored early. These included 1) division of the left main bronchus to permit elevation of the carina and right main bronchus high up into the right hemithorax to bridge a long tracheal gap; and 2) devolution of a cervical segment of trachea with intact lateral blood supply into the mediastinum to effect primary anastomosis there, leaving a cervical gap to be reconstructed later.[9] Exchange of tracheal segments had earlier been tried in dogs.[10] A "trough" technique was developed to reconstruct the cervical trachea in stages, using a bipedicled full thickness cervical skin flap with attached platysma, supported by inlying polypropylene rings.[11]

These potential techniques were explored before the simpler maneuvers of cervical flexion and laryngeal release were introduced. These two easily employed maneuvers, together with pretracheal dis-



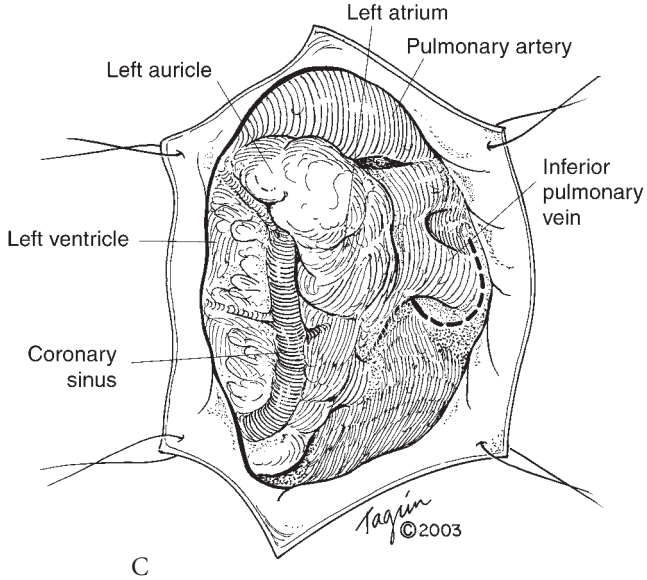


FIGURE **28-5** *Intrapericardial hilar release, from outside of the pericardium,* A *and* B. A, *A U-shaped incision* (dashed line) *is made around the inferior pulmonary vein (IPV) after dividing the (inferior) pulmonary ligament. The phrenic nerve must not be injured.* B, *The frenum between the pericardium and epicardium beneath the IPV has been divided, allowing elevation of the hilum, as shown. The* dashed line *indicates extension of the pericardial incision around the hilum to achieve further mobility.* C, *Release of the IPV via wide pericardiotomy, accomplished through complete median sternotomy from inside the pericardium. Technique of Cameron D. Wright. The line of pericardial incision* (dashed line) *anterior to the pulmonary vein must be close to the vein in order to avoid injury to the phrenic nerve, which lies quite close to the vein outside of the pericardium. LMB = left main bronchus.*

section and intrapericardial release, allowed most resections to be accomplished without extreme measures. The first "extreme" technique noted above was not used as described, both because of its complexity and the likelihood of loss of carinal blood supply. However, the concept proves invaluable for the resection of carina and a long length of trachea. As described in Chapter 29, "Carinal Reconstruction," reimplantation of the left main bronchus into the bronchus intermedius permitted high elevation of the right main bronchus to a tracheal remnant.

The second technique, devolution of cervical trachea to effect safe intrathoracic primary anastomosis, was employed in a small number of patients who had extreme resections in very complex situations. In some of these patients, a splinted cutaneous trachea was constructed successfully, primary reanastomosis became possible at a later date in one patient, and reconstruction was not completed for various reasons in several other patients. This record makes it obvious why, in addition to the complexity of the technique and potential for damage to recurrent laryngeal nerves, the method was abandoned.

The key issue, however, is that extremely few lesions that should be resected can not be resected and reconstructed by the presently devised surgical techniques.

References

1. Brewer LA III, Bai AF. Surgery of the bronchi and trachea. Am J Surg 1955;89:331–46.
2. Prommegger R, Salzer GM. Heterotopic ossification in pedicled intercostal muscle flaps causing clinical problems. J Thorac Cardiovasc Surg 1998;115:466–7.
3. Grillo HC, Bendixen HH, Gephart T. Resection of the carina and lower trachea. Ann Surg 1963;158: 889–93.
4. Abruzzini P. Trattamento chirurgico delle fistole del bronco principale consecutive a pneumonectomia per tubercolosi. Chir Torac 1961;3:165–71.
5. Perelman MI. Resection and plastic surgery of bronchi. Moscow: Mir; 1968. p. 202–13.
6. Ginsburg RJ. Median sternotomy for repair of post-pneumonectomy fistulae. In: Martini N, Vogt-Moykopf I, editors. Thoracic surgery: frontiers and uncommon problems. St. Louis: Mosby; 1989. p. 149–52.
7. Valesky A, Hohlback G, Schildberg FW. Wertigkeit unterchiedlicher Matsnahmen zur Minderung der Anastomosenspannung nach Kontinuitätsresektion der Trachea. Langenbecks Arch Chir 1983;360:59–69.
8. Grillo HC. Surgery of the trachea. In: Current problems in surgery. Chicago (IL): Year Book Medical Publishers; July 1970.
9. Grillo HC, Dignan EF, Miura T. Extensive resection and reconstruction of mediastinal trachea without prosthesis or grafts: an anatomical study in man. J Thorac Cardiovasc Surg 1964;48:741–9.
10. Assouad M, Merlier M, Forster E, et al. Échange sans dévascularisation de segments trachéaux chez le chien. Poumon Coeur 1960;16:121–31.
11. Grillo HC. Circumferential resection and reconstruction of the mediastinal and cervical trachea. Ann Surg 1965;162:374–88.

Carinal Reconstruction

Hermes C. Grillo, MD

Carinal resection and reconstruction remains one of the most challenging areas of airway reconstruction. There is no single ideal technique for reconstruction. The type of resection and reconstruction varies with the location and extent of the lesion.[1] The first decision to be made by the surgeon is whether a tumor or inflammatory process is indeed amenable to resection and primary reconstruction. Except for instances of dense inflammation or of extensively invasive tumor, resection is most often possible. Reconstruction, however, lacking an acceptable tracheal replacement, quickly becomes impossible as the length of tracheal involvement increases. The management of extensive carinal lesions has been fraught with so many complications in the use of staged reconstruction, or failures in the use of prosthetics, that I presently believe that radiotherapy is preferable for a susceptible tumor, if direct anastomosis does not seem safely possible by one of the techniques that have evolved.

Surgery for carinal reconstruction is technically difficult and shadowed with the possibility of complications such as devascularization, separation, and stenosis. Ventilation under anesthesia can still present difficulties in complex cases. Postoperative difficulties include management of secretions, which may sometimes be troublesome for many days until healing has progressed sufficiently. Gradually, a spectrum of techniques has evolved for the reconstruction of the carina, with and without the resection of varying amounts of lung tissue, depending upon the nature and extent of the lesions.

Approach

Approach is made primarily through the right hemithorax, but certain lesions are approached from the left side, and a few are best managed through bilateral "clamshell" thoracotomy. The simplest carinal reconstruction—restitution of the carina after limited resection for a small tumor—can be accomplished through complete sternotomy with pericardiotomy. More complex reconstructions become more difficult by this route. See Chapter 23, "Surgical Approaches."

Anesthesia

I prefer inhalation anesthesia, managed as previously described. An extra-long single lumen endotracheal tube (ET) with a flexible distal part (Wilson tube) (see Figure 24-1 in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection") is placed orally and seated with the help of a flexible bronchoscope. The "down lung" is ventilated right or left with a standard flexible ET across the operative field during most of the resection and the reconstruction.[2,3] An alternative is a single lumen tube in the trachea with a bronchial blocker placed in the "up lung." High frequency jet ventilation has proved useful, particularly in certain complex reconstructions, during the phase just prior to completion of anastomoses.[4] It is sometimes a useful adjunct to cross-field ventilation by an ET. Chapter 18, "Anesthesia for Tracheal Surgery," expands on these general statements, and Figure 18-2 illustrates the management of intubation and ventilation for different schemes of carinal resection and reconstruction.

Successful use of cardiopulmonary bypass has been reported in a few cases of simple tracheal resection, where it was not necessary. Bypass has no place in most carinal reconstructions. Manipulation of the lung, which is necessary in the most difficult cases, where bypass might really be thought to be of help, prohibits its use. In such cases where it has been tried, anticoagulation has led to massive bleeding within the parenchyma of the manipulated lung and dependence upon whatever residual function there was in the portion of opposite lung that remained. If this is insufficient, the patient will die. Therefore, *when cardiopulmonary bypass might really be useful in airway reconstruction, it is usually unsafe to use it*. I employed a bypass only in unique situations for brief portions of operations, such as reconstruction of the main pulmonary artery because of localized tumor invasion or for reimplantation of a pulmonary artery "sling" that accompanies congenital stenosis.

Resection

Approach is most often through the right chest (see Figure 23-7 in Chapter 23, "Surgical Approaches") as described for transthoracic tracheal resection (see Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). The azygos vein is divided, the pleura opened, and the carina dissected (see Figure 28-1 in Chapter 28). Penrose drains are usually passed around the trachea, the right main bronchus, and the left main bronchus, if the lesion permits. Principles of oncologic dissection are followed insofar as it is possible without destruction of tracheobronchial blood supply. A Penrose drain is often placed around the esophagus at the level of the carina to retract the esophagus gently. The left main bronchus is often transected first, after placing lateral traction sutures on either side of that bronchus. Intubation across the operative field into the left main bronchus allows the right lung to collapse fully, facilitating further dissection. Either the trachea or right main bronchus may next be transected, depending upon the particular problem at hand. One or both lateral traction sutures are placed in the trachea or right main bronchus before division. With traction, the carinal specimen may now be separated with ease from any remaining attachments to mediastinal structures. Particular caution is used in dissecting it away from the aortic arch, which lies immediately deep to the junction of the trachea and left main bronchus. The left recurrent laryngeal nerve passes under the arch at this point on its way toward the left tracheoesophageal groove.

The pretracheal plane is dissected bluntly upward to the thyroid isthmus. If intrapericardial hilar mobilization is clearly going to be required, it is more easily done before airway division. Pericardial mobilization, especially the "U" incision beneath the inferior pulmonary vein, is so simple, consumes so little time, and carries so little hazard of complications, that I lean readily toward doing it. Intrathoracic anastomoses are *always* covered with a pedicled second layer of tissue. The pericardial fat pad (elevated as described in Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extend-

ed Resection") is preferred, since its bulk interposes substantial tissue between pulmonary vessels and tracheobronchial sutures lines, minimizing the possibility of bronchovascular fistula (see Figure 28-4*B* in Chapter 28). The omentum is preferred if irradiation has been given previously (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation").

Reconstruction

Without Pulmonary Resection

Techniques for carinal resection and reconstruction are described below, in order of increasing complexity.[1,5–8]

Lesion Confined to the Carina. Restitution of the carina by suturing the right and left main bronchi together and approximating these to the end of the trachea is an attractive concept, but is frequently impossible without tension (Figures 29-1*A–D*). The reason is that once the right and left main bronchi are sutured together, the left bronchus is held by the halter of the aortic arch, and most of the length for reapproximation must come from devolution of the trachea to the newly formed bifurcation. This length is obviously limited since most of the mobility is obtained by flexion of the neck. This type of reconstruction is, therefore, safely possible only for a small tumor or lesion located precisely at the carina. As previously noted, laryngeal release does not translate to relaxation at the carina (see Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection").

Lateral traction sutures are placed in the midlateral line of the trachea on either side and in the midlateral lines of the right and left main bronchi. The medial walls of the right and left main bronchi are sutured together with a series of interrupted 4-0 Vicryl sutures, placed so that the knots are tied outside of the lumen (see Figures 29-1*B*,*C*). Anastomosis of the trachea to the new bronchial bifurcation is done in

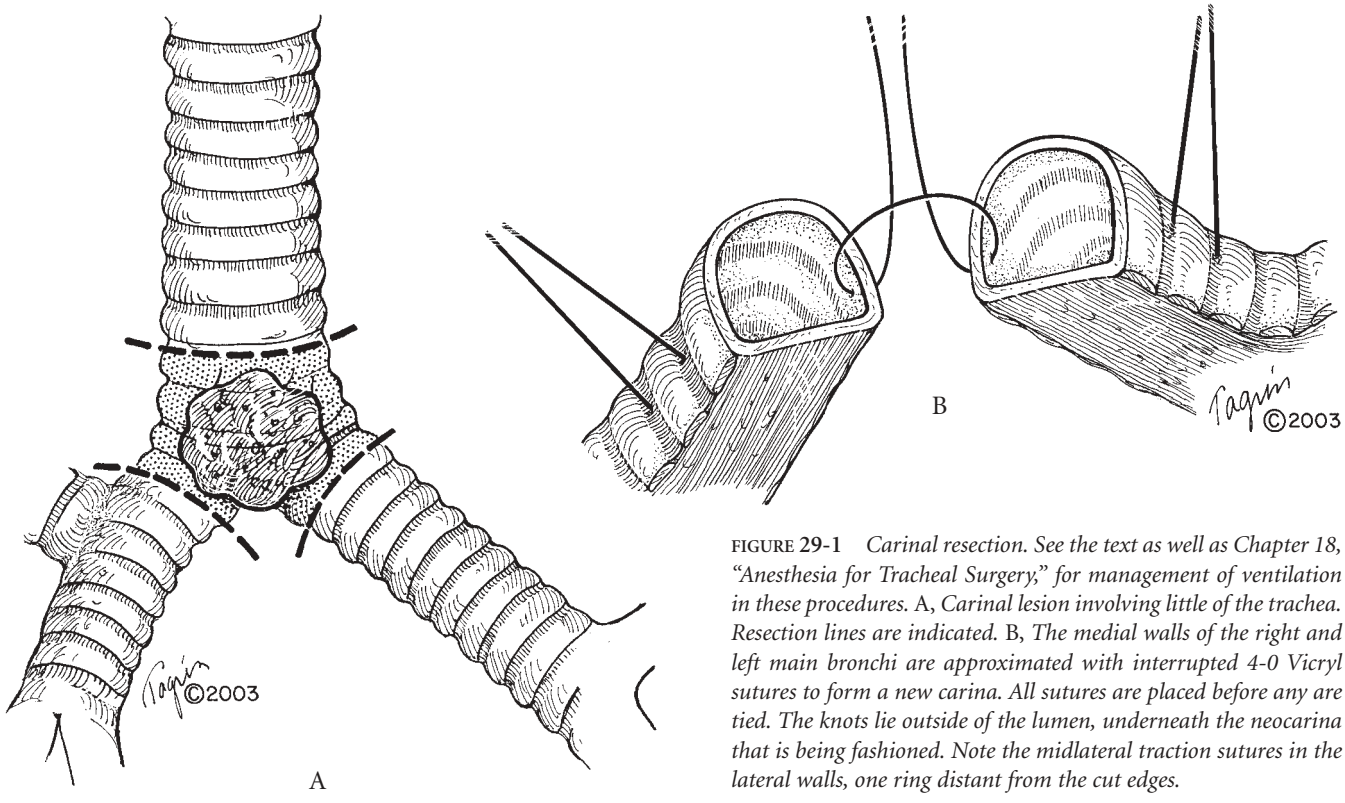
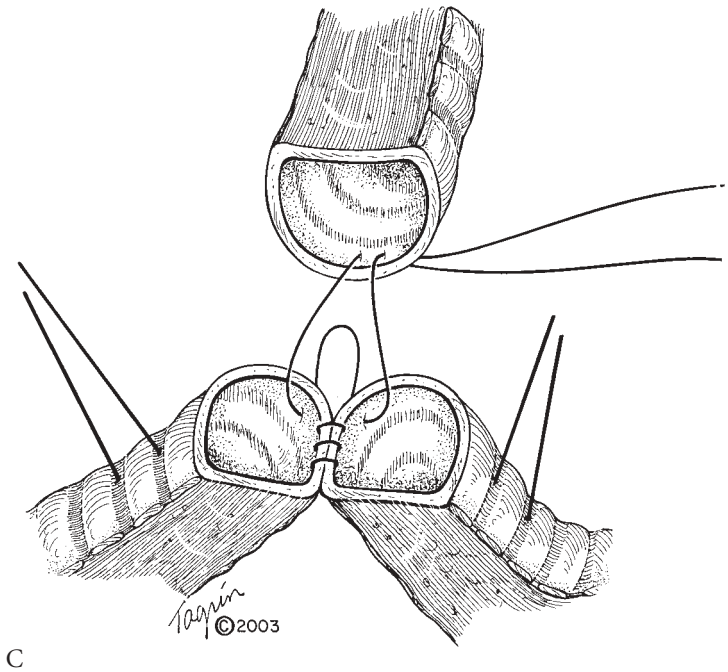


FIGURE **29-1** *Carinal resection. See the text as well as Chapter 18, "Anesthesia for Tracheal Surgery," for management of ventilation in these procedures.* A, *Carinal lesion involving little of the trachea. Resection lines are indicated.* B, *The medial walls of the right and left main bronchi are approximated with interrupted 4-0 Vicryl sutures to form a new carina. All sutures are placed before any are tied. The knots lie outside of the lumen, underneath the neocarina that is being fashioned. Note the midlateral traction sutures in the lateral walls, one ring distant from the cut edges.*



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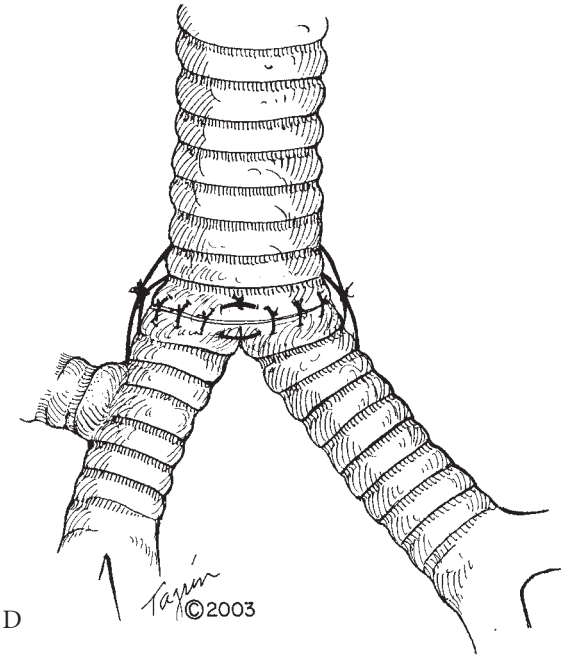


D

FIGURE **29-1** (CONTINUED) C, *After the neocarina is completed, sutures for anastomosis between the trachea and the bronchial circumference are placed. The joined main bronchi are treated as a single unit. Sutures will be closer together on the tracheal side since the bronchial circumference is longer. Sutures are placed proportionally; the tracheal and bronchial edges are not tailored. The anterior cartilaginous wall sutures are placed first. The anterior mattress suture at the confluence of the trachea and both bronchi is shown. The balance of the anterior anastomotic sutures is serially placed on both sides, from the midpoint to the lateral traction sutures, arrayed on each side in the order of placement. These will be tied in reverse order of placement. Membranous wall sutures are placed next. A mattress suture is also used at the posterior confluence. Midlateral stay sutures in the trachea are not shown. If this anastomosis is performed through a median sternotomy (with partial pericardiotomy), the membranous wall sutures are placed first, with half ranged to either side. The anterior sutures are then placed. Traction sutures are tied before the anastomotic sutures.* D, *After all anastomotic sutures are placed, the paired lateral traction sutures are tied on each side simultaneously. Cervical flexion is maintained during this maneuver. Anastomotic sutures are tied, beginning with the most accessible ones in the cartilaginous wall posterior to the traction sutures. This will further reduce any residual tension as the next sutures are tied, since the membranous wall tears more easily. As each suture is tied, excess suture is removed. Membranous wall sutures are tied next, followed by anterior sutures. Note the mattress suture in the anterior midpoint.*

the usual manner, with care taken to place the sutures proportionally in view of the discrepancy in shape and size of proximal and distal "ends." I use a mattress suture both anteriorly and posteriorly at the point of junction of the tracheal anastomosis with the bronchial anastomosis (see Figure 29-1*C*). This pulls the bronchial suture lines together at the corners (see Figure 29-1*D*), obtaining an airtight seal. A second layer flap is routinely used (see Figure 28-4 in Chapter 28, Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection).

Carinal Lesion with Involvement of the Trachea. If a carinal tumor or stenosis involves more than a minimal amount of trachea, the remaining length of trachea becomes insufficient to approximate it without tension to a neocarina formed by suturing right and left main bronchi together (Figure 29-2*A*).[1] In this case, the trachea is most often sutured end-to-end to the left main bronchus, and the right main bronchus is implanted into the side of the trachea (Figure 29-2*B*), usually after right hilar intrapericardial release. Ease of approximation of the trachea to the left main bronchus is tested, with the neck in tentative flexion,

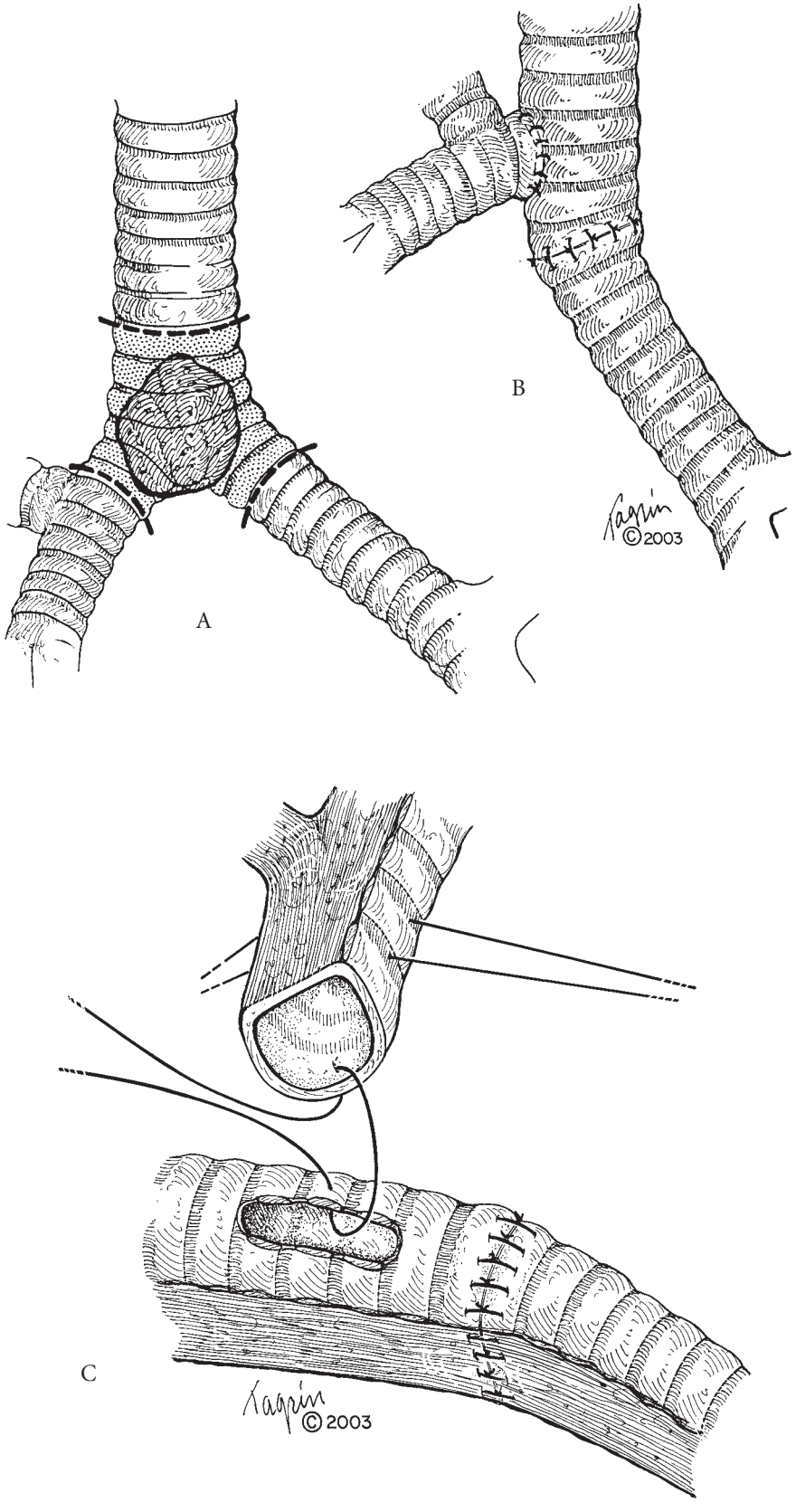


FIGURE **29-2** *Resection of the carina with involvement of a longer segment of trachea.* A, *The gap after the resection, indicated by the* dashed lines, *is sufficiently long so that excessive anastomotic tension would result if the bronchi were first sutured together, or anastomosis might be impossible. This is because the mobility of the left main bronchus is checked by the aortic arch. The gap between the trachea and left main bronchus should be no greater than 4 cm.* B, *Anastomosis is first completed between the trachea and left main bronchus. The long proximal endotracheal tube is then advanced into the left main bronchus. The right main bronchus, which has been freed by intrapericardial hilar mobilization, is anastomosed to an orifice in the lateral wall of the trachea. On rare occasions, it may be preferable to implant the right main bronchus into the medial wall of the left main bronchus. The decision is based on evaluation of relative tensions intraoperatively.* C, *Anastomosis of the right main bronchus to the trachea. The oval orifice in the trachea is located entirely within the cartilaginous wall. The lung is retracted anteriorly. The opening is as long as the bronchus is wide, but the width of the aperture is somewhat less than the anteroposterior diameter of the bronchus. Note the location of the orifice about two rings above the prior anastomosis. The initial anastomotic suture is shown. Stay sutures in the trachea on either side of the aperture, which are sometimes omitted, are placed closer to the ends of the aperture, if used. An alternative is to use an anastomotic suture of 3-0 Vicryl at each end of the oval to help in initial approximation and to reduce tension on the other 4-0 anastomotic sutures. See the text for details of technique.*

by pulling together the paired traction sutures of the trachea and left main bronchus on both sides. In general, anastomosis of the trachea to the left main bronchus may be safely accomplished in the adult without dangerous tension, if the initial gap is no greater than 4 cm. This varies with individual patients. If tension seems too great, the technique described in the next section should be employed instead.

The end-to-end anastomosis between the trachea and left main bronchus is accomplished in the usual manner, even though there is discrepancy between the diameter of the trachea and that of the left main bronchus. Ordinarily, I do not make any effort to bevel the main bronchus or to reduce the circumference of the trachea. The anastomotic sutures are placed proportionally between the trachea and bronchus (see Figure 30-1 in Chapter 30, "Main and Lobar Bronchoplasty"). When the ends are drawn together, there may be a small degree of intussusception. Although one might expect that the resulting irregularity inside the lumen would produce granulation tissue at the point of healing, this is almost never the case, probably because epithelization proceeds rapidly. The lateral traction sutures are tied first, followed by the anastomotic sutures. Placement of sutures and their temporary alignment on the drapes is done in a manner analogous to that described for transthoracic tracheal reconstruction (see Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection").

The special long ET is now passed from above, through the anastomosis between the trachea and left main bronchus into the left main bronchus (see Figure 18-2*B* in Chapter 18, "Anesthesia for Tracheal Surgery"). Care is taken not to advance it so far that the left upper lobe orifice becomes obstructed. Inhalation anesthesia is continued using the left lung only. The right lung remains collapsed. An ovoid opening is made in the right lateral wall of the lower trachea, approximately 1 cm proximal to the anastomosis just performed between the trachea and left main bronchus (Figure 29-2*C*). This is done in order to maintain blood supply in this isthmus of cartilage between the two anastomoses and to separate them by a bridge of healthy tissue. The aperture lies entirely within the cartilaginous lateral wall of the trachea to help assure patency (see Figure 29-2*C*). It does not extend into the membranous wall. The right main bronchus should be easily elevated to this level, facilitated by prior inferior intrapericardial release (see Figures 28-5*A*,*B* in Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). If there appears to be tension when the right main bronchus is advanced to the new aperture in the lower trachea, it may be necessary to complete the intrapericardial release circumferentially. Cervical flexion is maintained during completion of the anastomosis. Anastomoses are tested for air tightness under saline after retracting the ET proximally to a point above the anastomoses.

Anastomosis between the trachea and right main bronchus, which in some cases is essentially at the bifurcation into the upper lobe bronchus and bronchus intermedius, must be made with great care. 4-0 Vicryl sutures from the anterior wall of the right main bronchus to the anterior margin of the ovoid opening are placed carefully so that the knots will lie on the outside of the lumen (see Figure 29-2*C*). Lateral traction sutures may be helpful. These are placed in the usual orientation in the right main bronchus or sometimes in the lateral walls of the right upper lobe bronchus and bronchus intermedius if much of the right main bronchus has been resected. Traction sutures at the superior and inferior margins of the oval opening in the trachea, if used, must be placed with unusual care, not too close or too far from the new stoma, and possibly transversely or obliquely. 3-0 Vicryl is preferred for traction sutures in these locations to avoid injury to the thinner cartilage of the bronchus.

One or two anastomotic sutures may be placed just anterior to the midlateral traction suture in the inferior margin of the right main bronchus. The balance of sutures, beginning just beyond this point, is serially placed anteriorly, ranging to the superior margin of the right main bronchus. When the bronchus is approximated to the tracheal aperture, fingertip access for tying the sutures is better from cephalad to caudad, except for the first few sutures placed, which are accessible inferior to the bronchus. The posterior wall sutures are placed after the anterior ones, retracting the lung anteriorly.

It often is not necessary to place traction sutures in the trachea for approximation to the traction sutures in the main bronchus, since the approximation is usually made without tension by simply sliding the collapsed lobe and its bronchus very carefully toward the point of anastomosis. The traction sutures on the bronchial side are helpful in thus positioning the bronchus for anastomosis. An alternative is to use two slightly heavier anastomotic sutures (3-0) at either end of the ovoid anastomosis. The anterior and superior cartilage-to-cartilage sutures are tied, beginning superiorly and progressing in a caudad direction. The few caudad sutures are tied with a little more difficulty because of the presence of the apical segmental branch of the upper division of the right pulmonary artery. The artery is retracted gently. The posterior anastomotic sutures are then tied. Special care must be taken not to pull sharply on these sutures in the membranous wall, since they can easily cut through the thinner membranous wall of the bronchus, causing troublesome leaks.

An alternative method, less often used, is to anastomose the right main bronchus to the trachea and implant the left main bronchus into the left side of the trachea. There is no special reason to recommend this scheme.

A second layer wrap is interposed between the anastomoses and pulmonary artery. The pericardial fat pad is preferred (see Figure 28-4 in Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"), but is somewhat more difficult to use than a broad-based pleural flap because of the bulkiness of the fat and the small space between the anastomosis of the right main bronchus to the trachea and the pulmonary artery ramus. A fat pad flap is employed by splitting it into two tails in its distal portion so that one may be carried around the anastomosis of trachea to the left main bronchus and the other circumferentially around the right main bronchial anastomosis to the trachea. To simplify passing these flaps in these tight spaces, they may first be carefully drawn into a Penrose drain (using three hemostats to spread the drain lumen). The sheathed and moistened flap is then pulled gently into place around the anastomosis, and the drain slipped off.

Before suturing the second layer flaps, the ET is drawn up into the trachea (with cuff deflated) to a point above the proximal anastomosis and the cuff is reinflated. The anastomoses are checked for leaks under saline. The ET is guided back into the left main bronchus in order to collapse the right lung again. After suturing the pericardial fat pad over the anastomoses and to itself, the ET is repositioned in the proximal trachea and the right lung fully expanded before closing the chest.

Extended Resection of Carina and Trachea. Where even a greater length of trachea must be removed, the technique just described will not suffice (Figure 29-3*A*). The length of tracheal resection will not permit safe approximation of the trachea to the left main bronchus, even with maximum mobilization. The left main bronchus remains tethered by the aortic arch. Access is not regularly available to perform intrapericardial mobilization of the left hilum, and even this would often not provide sufficient mobility. In contrast, full intrapericardial mobilization of the right hilum permits the right main bronchus to be elevated high in the chest for end-to-end anastomosis to the remaining trachea (Figure 29-3*B*). Ventilation is maintained with a tube across the operative field in the left main bronchus during this phase (see Figure 18-2*C* in Chapter 18, "Anesthesia for Tracheal Surgery").

After anastomosis of the trachea to the right main bronchus, an ovoid opening is made in the medial wall of the bronchus intermedius, and the left main bronchus is anastomosed to this opening (see Figure 29-3*B*). This technique was first described in patients by Barclay and colleagues,[5] who reported 2 cases in 1957, and then by Eschapasse and colleagues[7] and Grillo.[1] The bronchotomy is made with care not to remove excessive cartilage and so narrow the bronchus intermedius. On the other hand, a simple slit will not remain sufficiently patent. A slim oval of bronchial wall is excised, leaving a margin of cartilage all around the aperture. The anteroposterior diameter of the left main bronchus is somewhat greater than the

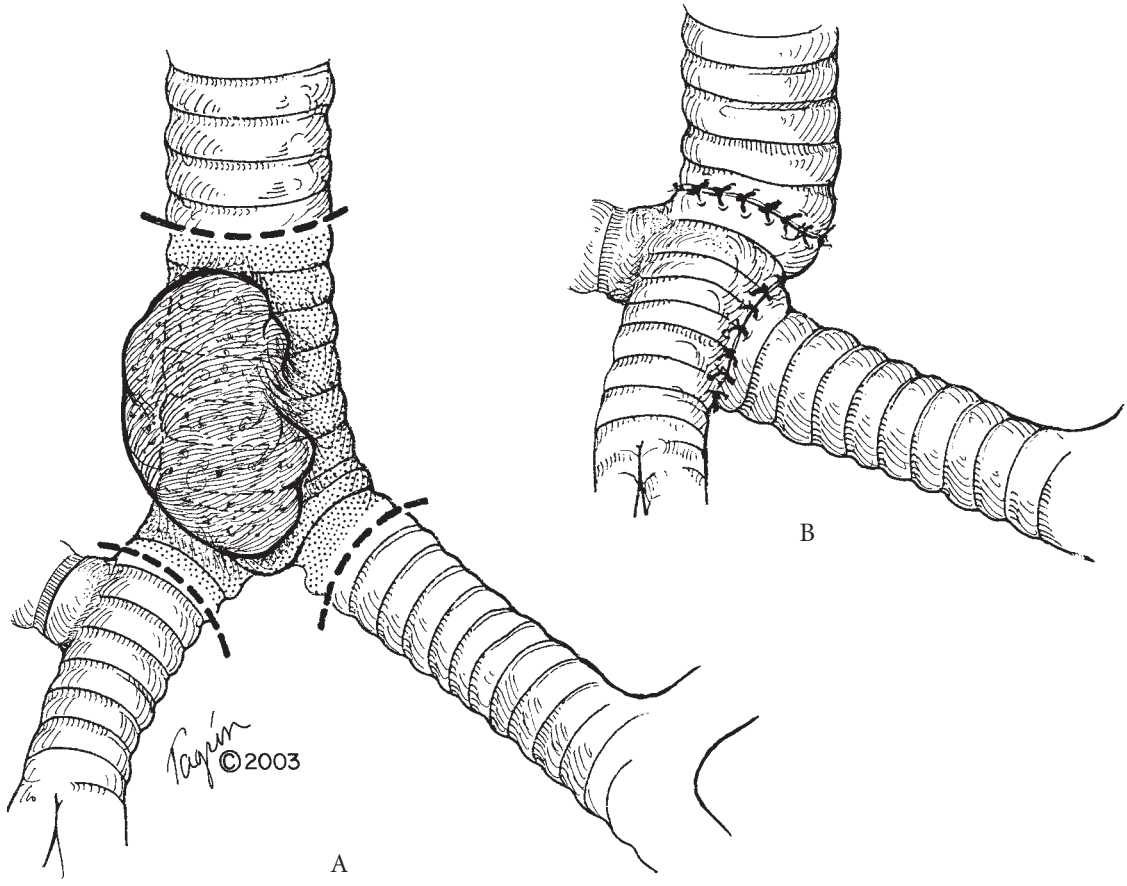


FIGURE **29-3** *Resection of the carina and a significant length of the trachea.* A, *The length of trachea resected* (dotted lines) *exceeds 4 cm. The trachea and left main bronchus will not approximate safely.* B, *The elevated right main bronchus is anastomosed to the trachea after intrapericardial hilar mobilization. The left main bronchus is then anastomosed to the medial wall of the bronchus intermedius, in similar fashion to Figure 29-2*C.

width of the ovoid opening. When the anastomosis is completed, the neo-orifice is held more widely open and the circumference of intermediate bronchus is slightly increased at this point.

Exposure is more difficult in this repair than in the two previously described. Sutures from the anterior cartilaginous wall of the left main bronchus to the anterior margin of the stoma in the bronchus intermedius are placed with half of the sutures ranged cephalad and half caudad. Posterior sutures are then placed from the membranous wall of the left main bronchus to the posterior margin of the ovoid opening in the bronchus intermedius. All sutures are placed before any are tied. The same considerations apply here to use of traction sutures as in the anastomosis earlier described between the end of the right main bronchus and the side of the trachea (see Figure 29-2*C*).

It is difficult to perform the second anastomosis using cross-field anesthesia. The flexible armored tube must be removed intermittently for placement of the sutures. The right lung should remain collapsed for access. Following placement of all the sutures, a small flexible tube may sometimes be passed from above, through the new opening into the left main bronchus, while the sutures are tied. This may be cumbersome or not be feasible. I, therefore, prefer to do this part of the anastomosis using high-frequency jet ventilation of the left lung, usually with a catheter passed from above, through the ET that lies in the trachea or across the operative field, and which is removed as the last sutures are tied (see Figure 18-2*C* in Chapter 18, "Anesthesia for Tracheal Surgery"). In using high-frequency ventilation in this situation, both

the surgeon and anesthetist must be continuously alert to the hazard of inadvertent occlusion of the route of egress of the gases that are being insufflated. Although, mechanically, one would expect that there would be free flow, this is not always the case. Development of sudden high pressure could rupture the left lung. If oxygenation is unsatisfactory, supplementary high-frequency ventilation may be added with a catheter in the right main bronchus or a bifid catheter in the right upper lobe bronchus and in the bronchus intermedius. Since the right lung does not fully expand, gentle retraction allows satisfactory surgical exposure.

With Pulmonary Resection

No resection of pulmonary tissue accompanies the three techniques described above. The following sections describe either concurrent or prior removal of lung tissue in addition to resection and reconstruction of the carina.

Carinal Resection with Right Upper Lobectomy. Tracheal or carinal tumors or fibrotic processes easily extend down the short right main bronchus sufficiently far to involve the right upper lobe bronchus. This may necessitate removal of the upper lobe in addition to the carina. Bronchogenic carcinoma in this location may also involve the main bronchus and carina (see Chapter 8, "Secondary Tracheal Neoplasms"). If carinal pneumonectomy is not elected, or is functionally impossible, the carina, right main bronchus, and right upper lobe may be removed, sometimes with the middle lobe as well (Figure 29-4*A*). The middle and lower lobes or the lower lobe alone may be salvaged and reimplanted into the bronchial tree, usually into the left main bronchus (Figure 29-4*B*). Elevation of the bronchus intermedius, and even more so of the right lower lobe bronchus, to the side of the trachea very often creates excessive tension, risking separation or stenosis. This will occur despite intrapericardial mobilization (which should always be done), because of the absent span of the right main bronchus. Furthermore, since reconstruction requires end-to-end anastomosis of the trachea to the left main bronchus, the excision must not leave a gap greater than 4 cm between the trachea and the left main bronchus, or excessive tension on this anastomosis will result.

Dissection is commenced as previously described. The pulmonary artery and superior pulmonary vein are dissected in anticipation of the lobectomy or lobectomies required and also to determine that there is no tumor involvement, either of the arteries or of adjacent lymph nodes, which would make it unwise or impossible to attempt a conservative lung sparing procedure. Hilar dissection is completed anteriorly and posteriorly, tapes are placed around the trachea above the lesion and around the left main bronchus, and the bronchus intermedius is cleared appropriately. The requisite branches of the pulmonary vein and artery to the upper lobe or upper and middle lobes are doubly ligated and divided. If the middle lobe is also to be removed, the middle lobe bronchus is divided and closed with 4-0 Vicryl sutures. Care is taken not to strip the bronchus intermedius of its overlying vasculature and connective tissue. The bronchus intermedius is divided below the upper lobe bronchus. If the tumor extends to the middle lobe bronchus, it is usually not possible to salvage the lower lobe, since the superior segmental orifice lies opposite the middle lobe bronchus. Rarely, oblique bronchial resection can be done, but this presents potentially greater difficulties for anastomosis.

In cases of lobar reimplantation after carinal resection, the hilum must be mobilized intrapericardially to obtain enough length to reattach the lower lobe with minimal tension. It is usually easier to divide the left main bronchus first, after placement of lateral traction sutures, and institute cross-field intubation and ventilation of the left lung. This allows the right lung to collapse, facilitating completion of dissection. After excision of the specimen, which includes the upper lobe (or lobes) and the carina in continuity, end-to-end anastomosis is performed between the trachea and the left main bronchus. Anastomotic integrity is checked under saline. The bronchus intermedius or lower lobe bronchus is drawn upward with its 3-0 traction sutures to determine whether it can be implanted into the side of the left main bronchus without excessive tension. Initially, I implanted the bronchus intermedius or right lower lobe bronchus into the side

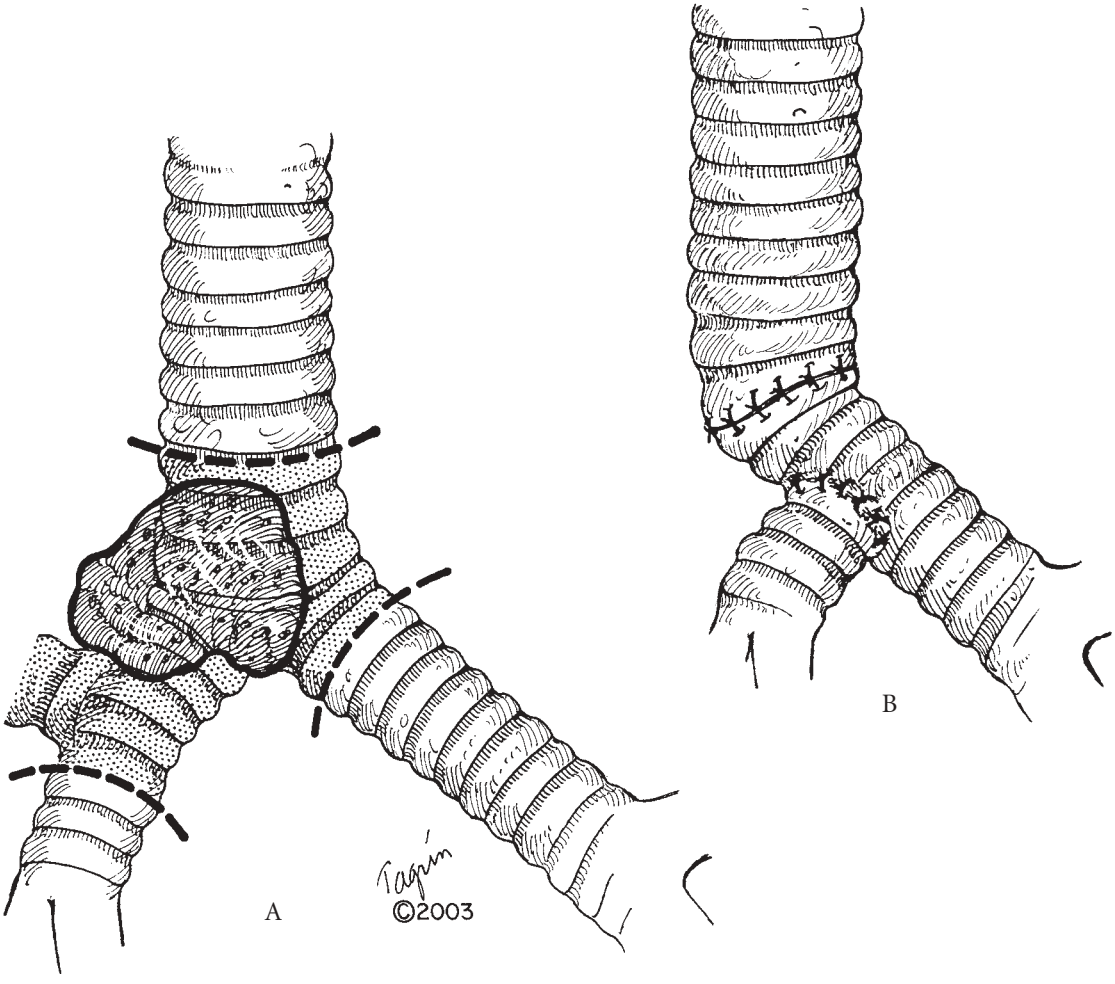


FIGURE **29-4** *Resection of the carina and right upper lobectomy or bilobectomy (right upper and middle lobes). The gap between the trachea and left main bronchus should be less than 4 cm.* A, *Lines of resection are indicated. If the middle lobe parenchyma is involved by contiguity, it is also resected. The middle lobe bronchus is closed, allowing salvage of the bronchus intermedius above the superior segmental bronchus.* B, *The trachea is anastomosed end-to-end to the left main bronchus. The bronchus intermedius is anastomosed as shown to the medial wall of the left main bronchus, with the aid of right hilar mobilization. Although the bronchus intermedius sometimes will reach the side of the trachea, danger from excessive tension makes the anastomosis shown to be preferable, even though its execution is more difficult. If an anastomosis to the left main bronchus also appears questionable, right pneumonectomy is preferable, assuming the patient can tolerate loss of the right lung.*

of the trachea. Although this succeeded in a number of patients, it also led to serious or lethal complications in too many others, due to excessive tension. These complications included obstruction of the implanted right bronchus, stenosis, separation, and hemorrhage. Anastomotic tension, in this case, is by its nature much greater than that developed by anastomosis of the right main bronchus to the trachea.

Implantation of the smaller bronchus intermedius into the relatively narrow left main bronchus, while maintaining anesthesia via an ET in the left main bronchus, can be difficult. This is another situation in which high-frequency ventilation is useful.

Tissue for the second layer is prepared and brought in beneath the anastomoses, but it is not sutured in place until after testing the anastomoses under saline.

If the patient can tolerate a pneumonectomy, it may be the safer course for many.

Carinal Resection and Right Pneumonectomy. Resection of the carina with the right lung is most frequently done for bronchogenic carcinomas involving the origin of the main bronchus or the carina.[9–11]

If the tumor does not extend very far up the trachea, the operation is conceptually and mechanically relatively easy to perform (Figure 29–5). While physiologically, the operation should be the equivalent of a right pneumonectomy, it has been general experience that mortality was much higher for this operation than for a simple pneumonectomy, largely due to "postpneumonectomy pulmonary edema." Its etiology remains unclear, although attention to minimizing barotrauma appears to be helpful.[12]

The patient is selectively intubated into the left main bronchus with an extra-long flexible-tipped ET. Mediastinoscopy should be performed prior to embarking upon such a resection to be certain that the patient is potentially curable. This is best done under the same anesthesia as that for resection. When lymph nodes are not involved and the only limitation to resection is involvement of the origin of the main bronchus or carina, these patients should really be classified as stage IIIA carcinoma of the lung rather than stage IIIB. If lymph nodes are involved, the procedure is only justified in the setting of a study protocol.

Most of the technical points have already been discussed. Resectability is determined by careful exploration of the mediastinum and examination early in the operation of the origin of the pulmonary vessels, if necessary, intrapericardially. If more than 4 cm of trachea will have to be resected, the surgeon must be wary of excessive anastomotic tension, and question the feasibility of surgery. I try to avoid radical mediastinal lymph node dissection to minimize possible injury to the blood supply of the trachea or to lymphatic drainage from the remaining lung. When it is clear that resection can be done from the vascular standpoint, the trachea is circumferentially dissected at the level of the expected division and the left main bronchus at its origin. The left main bronchus is usually divided first after placement of traction sutures, so that intubation may be carried out and ventilation continued in the left lung. Tracheal division is sometimes accomplished before mediastinal dissection has been completed. Traction on the divided bronchus and tracheal end of the specimen facilitates residual dissection. Dissection must be done carefully to avoid injury to the left recurrent

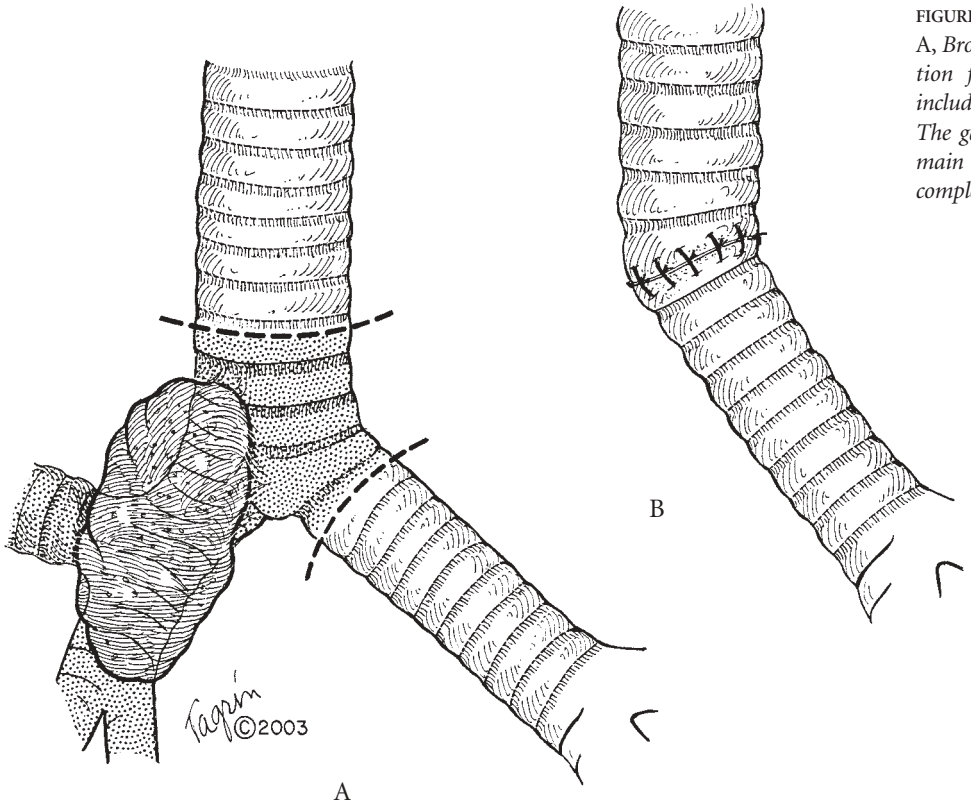


FIGURE **29-5** *Right carinal pneumonectomy.* A, *Bronchogenic carcinoma is the usual indication for this procedure. Complete staging, including intraoperative assessment, is required. The gap between the divided trachea and left main bronchus should not exceed 4 cm.* B, *The completed anastomosis.*

laryngeal nerve where it lies beneath and on the aortic arch. Anastomosis is performed as described earlier and a second layer closure is always applied. As always, approximation is facilitated by cervical flexion.

Carinal Resection and Left Pneumonectomy. Carcinoma of the left lung only rarely extends to the carina, given the length of the left main bronchus. Infrequently, other primary tumors such as adenoid cystic carcinoma are encountered, which involve the left main bronchus and the carina. The entire left main bronchus may be resected separately (see Chapter 30, "Main and Lobar Bronchoplasty") and the lobar bronchial bifurcation of the left lung reattached to the base of the carina[13]; however, if the carina also must be resected with a large part of the left main bronchus, there is currently no feasible technique to salvage the left lung. Under these circumstances, a left pneumonectomy and carinal resection may be performed through a *left* thoracotomy (Figure 29-6*A*).[1] This approach is safely feasible only where a limited amount of trachea is involved; that is to say, where no more than 1 cm of trachea and of right main bronchus will have to be resected above and below the margins of the left main bronchus.[1,14,15]

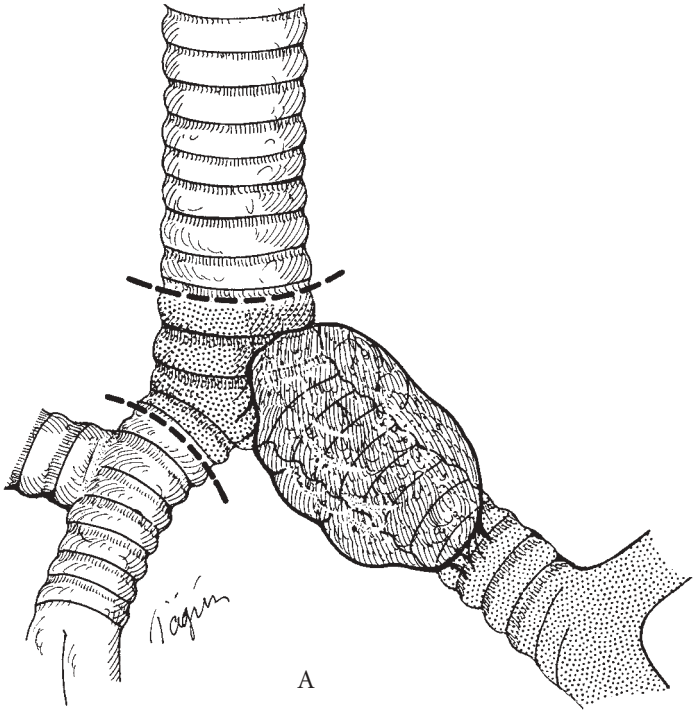
Access to the carina from the left is constricted, but adequate for such limited resection (Figure 29-6*B*). The right main bronchus is selectively intubated with a Wilson tube. After determination of resectability, dissection is carried up beneath the aortic arch, taking great care to identify and preserve the recurrent laryngeal nerve. The left vagus nerve will be divided well below the point of origin of the recurrent nerve. The ligamentum arteriosum is divided to improve exposure. The aortic arch is dissected circumferentially and tapes placed around it for retraction. With the aid of these tapes and retractors, and with cervical flexion to devolve the trachea, dissection is carried around the lowermost end of the trachea and a Penrose drain placed around the trachea. The pretracheal plane is dissected bluntly into the neck. The carina is identified, the right main bronchus is dissected, and a Penrose drain placed around it. Once it is absolutely certain that resection and reconstruction will be possible, the pulmonary vessels are divided.

The neck is held in flexion, the aorta retracted, and traction placed on the peritracheal and peribronchial tapes, pulling the carina into the field. Then, 2-0 Vicryl traction sutures are placed in the left lateral wall of the trachea, proximal to the projected point of transection, and in the medial wall of the right main bronchus, distal to the point of bronchial division. The endotracheal tube is withdrawn into the proximal trachea. The right main bronchus is transected and a cross-field flexible ET is passed into the most proximal portion of the right main bronchus. The trachea is transected and the specimen extracted. With traction on the initial stay sutures, additional 2-0 Vicryl traction sutures are now placed in the right side of the lower trachea and in the right side of the right main bronchus. Blunt dissection should not be carried very far down the right main bronchus, because of the proximity of the pulmonary artery and the short length of the bronchus.

Using the tapes around the aorta for intermittent retraction of the aortic arch, anastomotic sutures are placed in the usual manner, commencing just anterior to the right lateral traction sutures of trachea and main bronchus and arranging these sutures forward on the anterior cartilaginous wall until the left-sided traction sutures are reached. A similar group of sutures is placed posteriorly, including the membranous wall and the two segments of cartilaginous wall posterior to the traction sutures on either side. When all of these sutures have been placed, the cross-field ET is removed, the tube from above is once again passed just into the right main bronchus, and, with cervical flexion, both sets of lateral traction sutures are tied. The ET must not be pushed into the right main bronchus or it will hinder approximation or obstruct the upper lobe orifice.

The anastomotic sutures in the cartilaginous parts of the tracheobronchial walls, posterior to the traction sutures, are tied first on both sides. With this added security, the membranous wall sutures are tied gently to avoid cutting through the soft wall. Excess suture is cut after each is tied. The sutures in the cartilaginous walls, anterior to the stay sutures, are then tied, completing the anastomosis (Figure 29-6*C*).

The anastomosis is tested for air tightness under saline and, following this, a second layer pedicled flap is passed around the anastomosis and sutured to the airway and to itself. The long ends of the tied



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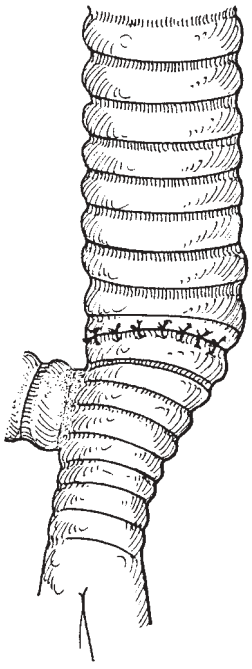

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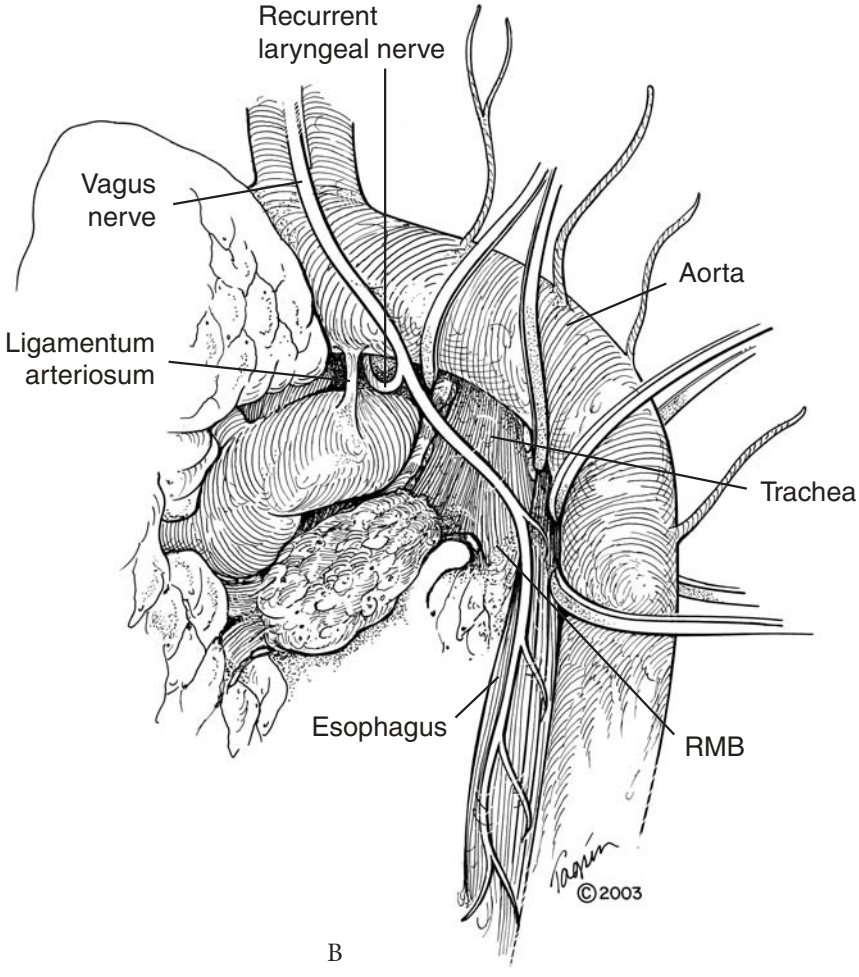


B

FIGURE **29-6** *Left carinal pneumonectomy.* A, *The procedure is feasible via a left thoracotomy with subaortic approach, but only when a short length of carina and trachea is involved.* B, *Exposure is facilitated by retraction of the aortic arch using tapes. The left pulmonary artery is also gently retracted, with care not to injure the first branch. The ligamentum arteriosum is usually divided. The recurrent laryngeal nerve must be meticulously identified and preserved. The vagus nerve is usually well divided, distal to the recurrent nerve, in order to improve exposure. After pretracheal blunt dissection and with cervical flexion, traction is placed on the trachea just above the carina and on the right main bronchus at its origin, using tapes. Traction sutures are placed in the trachea and right main bronchus before the airway is divided.* C, *Completed anastomosis. It is wrapped with a second layer. RMB = right main bronchus.*

traction sutures are left in place until the second layer flap is passed beneath the anastomosis in order to facilitate this maneuver. The excess length is cut off, but the knotted traction sutures remain to minimize tension on the anastomotic sutures during early healing.

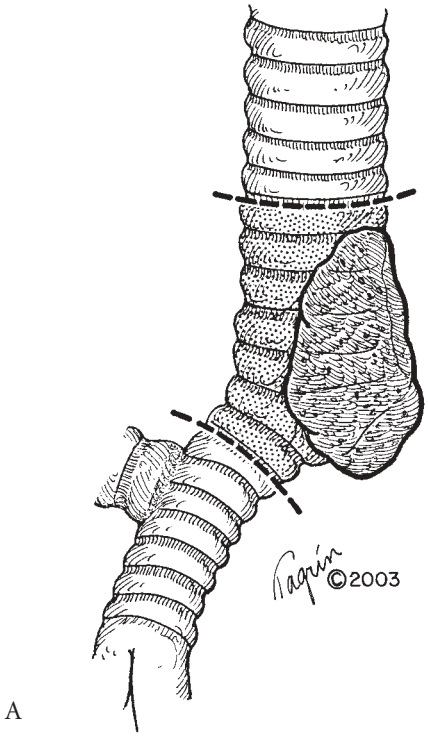
Carinal Resection for Recurrent Tumor or Stenosis after Pneumonectomy. I have treated a number of patients with tumor either residually present following a left pneumonectomy, as in a case with adenoid cystic carcinoma where concurrent carinal resection should have been done initially, or where later recurrence of tumor such as adenoid cystic carcinoma or carcinoid had followed a left pneumonectomy (Figures 29-7*A*,*B*). These patients and others with stenosis of an anastomosis between the trachea and the right main bronchus following prior left carinal pneumonectomy have been approached through the *right* chest with gentle retraction of a ventilated right lung. This can be done conventionally using hand ventilation or with high-frequency ventilation.

The area of the lesion is carefully dissected out, first encircling the trachea above the stump of the left main bronchus and then the right main bronchus just below the carina. With traction on both of these loops, the specimen is gradually dissected out, with meticulous care being taken to avoid any possible injury to the stump of the left pulmonary artery. The pretracheal plane is bluntly dissected. Once the specimen is resected (Figure 29-7*C*), end-to-end anastomosis is done in the manner already described and a second layer closure applied (Figure 29-7*D*). Intrapericardial hilar mobilization is very likely to be necessary and is best done before resection. On a rare occasion, anastomotic stenosis between the trachea and left main bronchus after a right pneumonectomy has been managed by repeat right thoracotomy and excision of the stenotic area deeply in the mediastinum.

In one patient who underwent extensive tracheal resection for adenoid cystic carcinoma that also required removal of the carina and left lung, anastomotic stenosis occurred after postoperative irradiation. The anastomotic stenosis was successfully resected through a cervical incision, for it rose that high in the mediastinum upon cervical extension. The long-term result was excellent.

Carinal Resection for Lesions Involving Major Portions of the Trachea and Left Main Bronchus. Special difficulties are presented by a lesion that involves a considerable length of trachea, carina, and enough of the left main bronchus so that the left lung cannot be salvaged (Figure 29-8*A*). When it seemed feasible that the right main bronchus could be elevated sufficiently to meet the length of trachea that would remain after resection of the lesion, several approaches for resection were employed. Perelman initially advocated approach through the right chest, stapling the left main bronchus distal to the lesion, leaving the left lung in situ.[8] However, this resulted in postoperative physiological difficulties, due to a large shunt through the nonventilated residual lung, which later required left pneumonectomy.[1] He, therefore, later advocated ligating the left pulmonary artery via the right chest through an opening in the pericardium. Another option was to perform concomitant or delayed left thoracotomy for left pneumonectomy, after the lesion had been removed and the right-sided anastomosis completed.

The approach that I favor for this rare and difficult problem is single-stage resection and reconstruction via transverse bilateral thoracotomy through the fourth interspace across the sternum (see Figure 23-8 in Chapter 23, "Surgical Approaches"). The thoracotomy extends to the posterior axillary line on both sides and provides generous access for carinal resection, left pneumonectomy, and anastomosis of the trachea to the right main bronchus. The postoperative physiological burden of the incision must be considered, but epidural analgesia for chest wall pain has made this approach safely applicable. Prolonged postoperative ventilatory support is no longer routinely expected. Alternate exposure via median sternotomy and left anterior thoracotomy is less satisfactory and presents technical difficulties for dissection of an extensive tumor, bilateral hilar dissection, and reconstruction.



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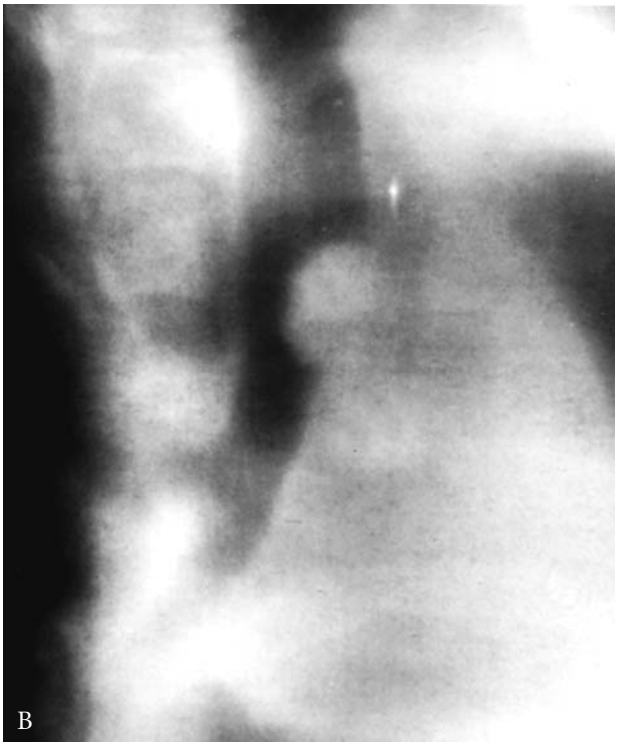

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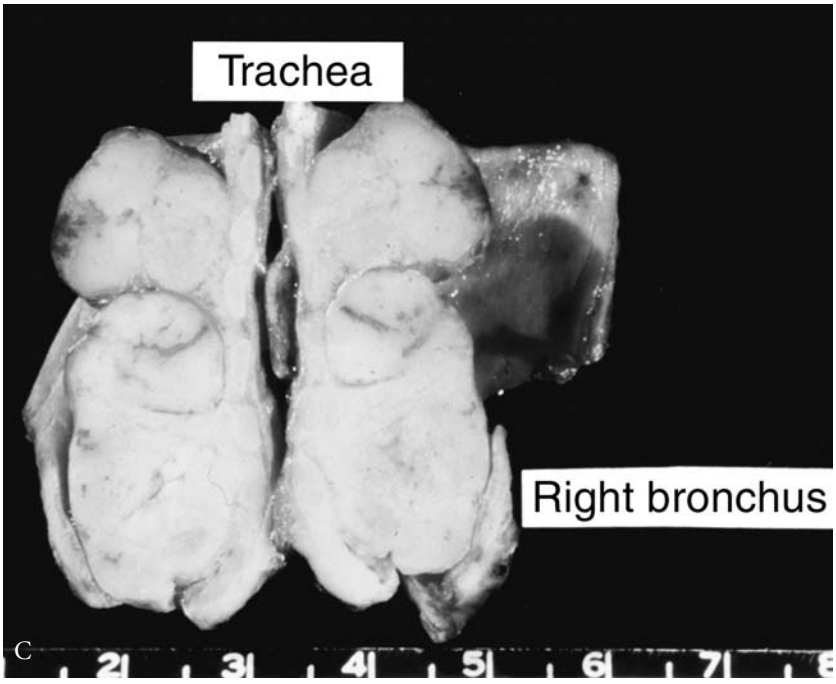

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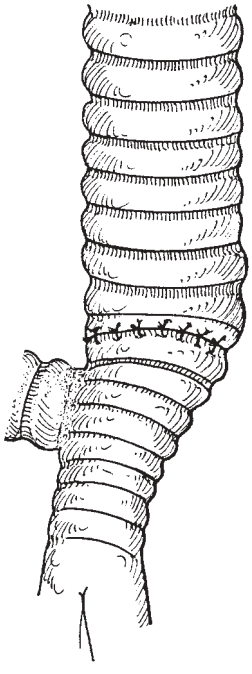

D

FIGURE **29-7** *Carinal resection for recurrent or residual tumor or for stenosis following prior left pneumonectomy.* A, *Recurrent tumor of some bulk, after left pneumonectomy, is shown. Resection is done via a right thoracotomy, with right hilar intrapericardial mobilization. The right lung is ventilated gently throughout the procedure. High-frequency ventilation may be preferred. A smaller recurrent tumor or residual tumor at the carina, following left pneumonectomy, is resected from the left (see Figure 29-6). Residual tumor on the right after right pneumonectomy is, of course, approached from the right.* B, *Tomogram showing recurrent carcinoid at the location of the left main bronchial stump in a 28-year-old woman who had undergone left pneumonectomy 14 years previously.* C, *The tumor seen in Figure 29-7*B *was resected through a right thoracotomy. There was no recurrence in 29 years of follow-up.* D, *The completed anastomosis.*

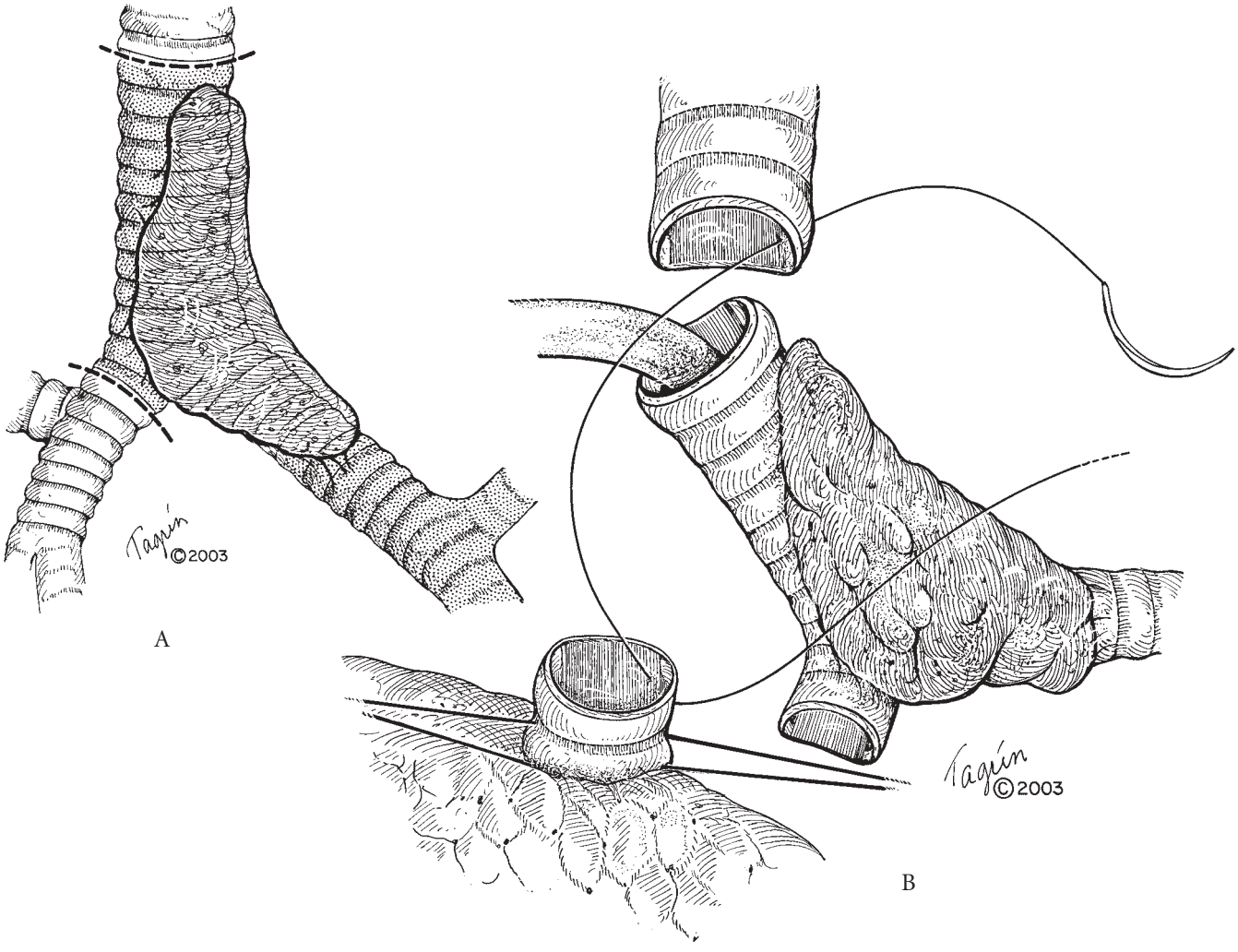

FIGURE **29-8** A, *Carinal resection for tumor that involves a long segment of trachea and extends so far into the left main bronchus that salvage of the left lung is impossible by present anastomotic techniques. See text for details. Reconstruction is done by end-to-end anastomosis of the right main bronchus to residual trachea, high in the thorax. Approach is via bilateral thoracotomy (see Figure 23-8).* B, *Ventilation is maintained initially in the left lung through an endotracheal tube (ET), which was bronchoscopically guided past the tumor into the left main bronchus. Dissection is commenced. Right hilar intrapericardial mobilization is completed. The trachea is next divided above the tumor, and left lung ventilation is continued via an ET placed across the operative field, into the trachea, and thence into the left main bronchus. The right lung remains collapsed. The right main bronchus is then divided below the tumor and dissection completed, as fully as possible from the right. Anastomosis between the trachea and the elevated right main bronchus is performed anterior to the ET and the fully-mobilized specimen.*

The right side of the incision *including transverse sternotomy* is completed initially for exploration. If the lesion can be removed and is not so extensive that reconstruction will be unsafe, the left side of the incision is completed. After mobilizing the tumor, the trachea is divided above the tumor and a flexible ET is threaded distally through the divided end of the trachea, past the tumor into the left main bronchus, and is sutured into position to ventilate the left lung (Figure 29-8*B*). The left main bronchus, in many such cases, cannot be conveniently transected, since there is insufficient bronchus distal to the tumor to accept the seating of an ET. If need be, some tumor may be cored out at initial rigid bronchoscopy to provide a channel for an ET. The right main bronchus is transected next and the specimen dissected as completely as is possible from the right. The dissected but attached specimen, with its inlying ET, is dropped posteriorly

FIGURE **29-8** (CONTINUED) C, *Anastomosis in progress. All sutures have been placed between the trachea* (white arrow) *and a short stump of right main bronchus* (black arrow). *The left-sided tracheal stay suture is clearly visible, lying on the ventilated left lung. The cross-field endotracheal tube enters the distal transected trachea near proximal tumor and lies on the collapsed right lung parenchyma, which is behind the anastomosis. After completion of the anastomosis, the right lung is ventilated, the left lung is collapsed, the hilar structures on the left are divided, and the left lung is extracted with the attached tracheal and carinal specimen. The final anastomosis is as shown in Figure 29-7*D.

(see Figure 29-8*B*). Intrapericardial hilar mobilization of the right lung is accomplished, and anastomosis performed between the trachea and the right main bronchus (Figure 29-8*C*). The right lung is expanded and ventilation commenced on that side. The ET into the left lung is extracted, and the dissection and left pneumonectomy completed from the left side.

Individualized Reconstruction after Carinal Resection. Unique problems present that demand individualized solutions. In devising bronchoplastic procedures, the surgeon must be careful not to revert inadvertently to an earlier procedure that might be likely to fail. Complex tracheo- or bronchoplasties may seem attractive, but can fail easily due to 1) deficits in healing of complex suture lines, with leak, mediastinitis, and death, or tracheal or bronchial stenosis; or 2) early recurrence of inadequately excised neoplasm. Wedge resections at the carina usually have little to recommend them, since they are more likely to effect incomplete resection, can easily produce extra tension on a suture line, or cause lumenal buckling.

A solution used for a patient with adenoid cystic carcinoma, involving a long length of trachea, the right main bronchus, and the right upper lobe bronchus, is illustrated (Figure 29-9*A*) as an example. Circumferential resection of that length of trachea, carina, and right main bronchus would have made reconstruction impossible, since neither the left main bronchus nor the bronchus intermedius could have reached the proximal trachea. Fortunately, the tumor was limited to the right tracheal wall. The diagrammed procedure was done after careful appraisal of the extent of circumferential tracheal involvement (Figures 29-9*B*,*C*). The patient healed per primam, received routine postoperative irradiation, and remained tumor free for 32 years.

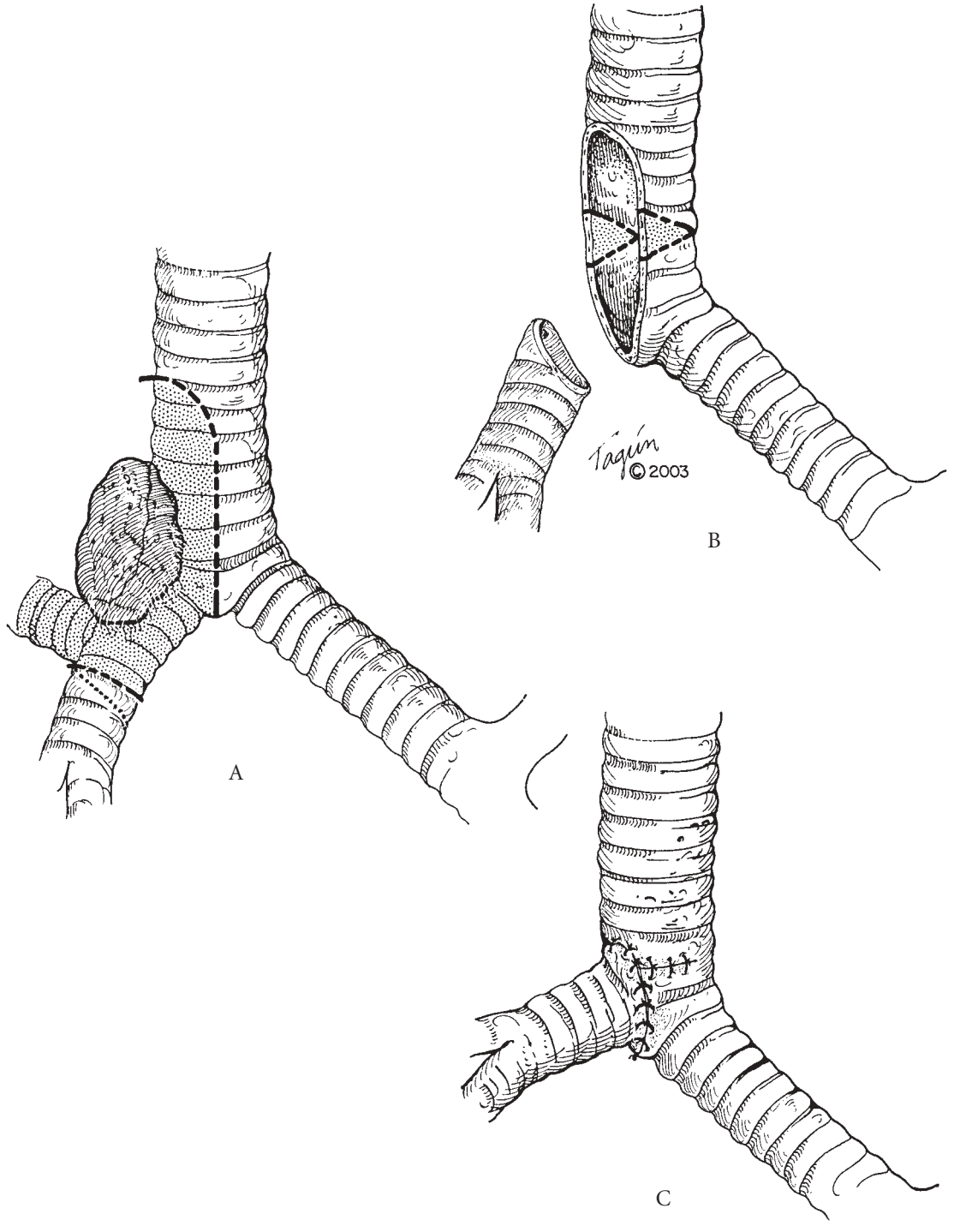

FIGURE **29-9** *Individualized solutions to unique problems.* A, *This 34-year-old woman had recurrent and persistent middle lobe pneumonitis, but a normal chest x-ray. Her adenoid cystic carcinoma involved a considerable length of the right lateral wall of the trachea, the carina on the right side, the right main bronchus, and the right upper lobe bronchus. The necessary length of circumferential tracheal resection would have obviated tracheal anastomosis to the left main bronchus. The bronchus intermedius would not have reached the shortened trachea. A compromise approach was designed as diagrammed, resecting half the circumference of the trachea.* B, *The lateral defect was reduced markedly by resection of lateral wedges of trachea, as diagrammed. The transected bronchus intermedius was trimmed obliquely* (dotted line in A) *to enlarge its circumference to fit the sizable defect that still remained after tailoring the trachea.* C, *The completed anastomosis. The woman enjoyed a good result and remains disease free after 32 years.*

References

1. Grillo HC. Carinal reconstruction. Ann Thorac Surg 1982;34:356–73.
2. Geffin B, Bland J, Grillo HC. Anesthesia management of tracheal resection and reconstruction. Anesth Analg 1969;48:884–94.
3. Wilson RS. Anesthetic management for tracheal reconstruction. In: Grillo HC, Eschapasse H, editors. International trends in general thoracic surgery. Vol 2. Philadelphia: WB Saunders; 1987. p. 3–12.
4. El Baz N, Ganzouri AE, Gottschalk W, Jensik RJ. One lung high frequency positive pressure ventilation for sleeve pneumonectomy. Anesth Analg 1981;60:683–6.
5. Barclay RS, McSwann N, Welsh TM. Tracheal reconstruction without the use of grafts. Thorax 1957;12: 177–80.
6. Grillo HC, Bendixen HH, Gephardt T. Resection of the carina and lower trachea. Ann Surg 1963;158:889–93.
7. Eschapasse H, Vahdat F, Gaillard J, Besso JC. Réflexions sur la résection de la tracheé inférieure et de le bifurcation bronchique. Ann Chir Thorac Cardiovasc 1967;6:63–70.
8. Perelman M. Surgery of the trachea. Moscow: Mir; 1976.
9. Jensik RJ, Faber P, Milloy FJ, Goldin MD. Tracheal sleeve pneumonectomy for advanced carcinoma of the lung. Surg Gynecol Obstet 1972;134:231–6.
10. Mathisen DJ, Grillo HC. Carinal resection for bronchogenic carcinoma. J Thorac Cardiovasc Surg 1991; 102:16–23.
11. Mitchell JD, Mathisen DJ, Wright CD, et al. Clinical experience with carinal resection. J Thorac Cardiovasc Surg 1999;117:39–53.
12. Mathisen DJ, Kuo EY, Hahn C, et al. Inhaled nitric oxide for adult respiratory distress syndrome following pulmonary resection. Ann Thorac Surg 1998;65: 1894–902.
13. Newton JR Jr, Grillo HC, Mathisen DJ. Main bronchial sleeve resection with pulmonary conservation. Ann Thorac Surg 1991;52:1272–80.
14. Salzer GM, Muller IC, Kroesen G. Resection of tracheal bifurcation through a left thoracotomy. Eur J Cardiothorac Surg 1987;1:125–8.
15. Maeda M, Nakamoto K, Tsubota N, et al. Operative approaches for left-sided carinoplasty. Ann Thorac Surg 1993;56:441–6.

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Main and Lobar Bronchoplasty

Douglas J. Mathisen, MD

All general thoracic surgeons should be familiar with bronchoplastic techniques. Every pulmonary surgeon should at least be familiar with the therapeutic possibilities of "sleeve resection," so that bronchoplastic surgery is considered for appropriate lesions. The need for bronchoplastic procedure may be anticipated in most patients, allowing preoperative planning, but some situations may not be evident until the surgical procedure is being performed. Bronchoplastic procedures can be performed on any lobe of either lung or mainstem bronchi. Although somewhat different techniques have been described by different authors, all have more in common than they have differences. We employ the same principles and techniques at every level for airway reconstruction, tracheal or bronchial.

The major goal of bronchoplastic procedures is preservation of viable lung to ensure a better functional result. The preserved lung functions physiologically.[1,2] At no time, however, should the desire to preserve lung function interfere with sound oncologic principles. Some patients may be unable to tolerate loss of an entire lung because of limited pulmonary reserve. In this circumstance, a bronchoplastic procedure may be the only surgical option, and hence be "obligatory." Careful preoperative evaluation of function and assessment of the extent of disease are essential to avoid unnecessary thoracotomy for pathology that is not amenable to bronchoplastic procedures and when pneumonectomy is unacceptable.

Indications

The main indication for bronchoplasty is an anatomically suitable lesion confined to a bronchus or lobe.[1–5] The ideal circumstance is a benign or low-grade lesion anatomically situated in the main bronchus or at the origin of a lobar bronchus.[4,5] Anatomically suitable malignant neoplasms are also amenable to bronchoplastic procedures.[1–3] Decision is more complicated for malignant neoplasms, since clear margins are mandatory and pathologic involvement of lymph nodes must be considered. Sleeve resection in the presence of positive mediastinal lymph nodes is debatable. If a complete in-continuity lymph node dissection can be performed, I believe a bronchoplastic procedure is acceptable. This is obviously most applicable for

right and left upper lobe lesions and, to a lesser extent, for left lower lobe lesions with only positive subcarinal lymph nodes. Many patients in this era are treated with neoadjuvant chemotherapy and radiotherapy when mediastinal nodes are involved. Concern about the effects of radiotherapy and lymph node dissection on bronchial healing has not been borne out in most series.[1–3] We wrap all anastomoses with a viable tissue flap, such as a pedicled pericardial fat pad. If concern exists about bronchial healing, it is best to use pedicled omentum.[6] Use of a circumferential intercostal muscle flap without *complete* removal of periosteum can produce a bony ring that causes bronchial stenosis and is difficult to correct.

Contraindications

Three main relative contraindications to bronchoplasty are high-dose corticosteroids, active bronchial inflammation, and remote (greater than 1 year prior) high-dose irradiation. Lung transplantation is done with success while patients are on steroids. The difference from bronchoplastic procedures is the degree of tension that exists after bronchoplasty. Low-dose steroids (5 to 10 mg of prednisone daily) are probably safe, but the anastomosis is best buttressed with robust tissue other than pleura. It is always important to identify active inflammation at the intended point of bronchial anastomosis, especially if the underlying disease is tuberculosis. Airway reconstruction in the presence of active inflammation is likely to lead to stenosis. Radiation in excess of 5,000 cGy, completed over a year prior to planned bronchoplasty, must be viewed with circumspection. The higher the dose and the longer the interval, the greater is the concern. When bronchoplasty is contemplated in such patients, it is prudent to wrap the anastomosis with pedicled omentum.[6,7] Omentum appears to be the most reliable anastomotic buttress in high-risk circumstances.[4] It reaches either hilum easily when pedicled on the right gastroepiploic arterial arcade and tunnelled substernally.

Postobstructive pneumonia is *not* a contraindication to bronchoplasty, provided that the lung is not destroyed by infection. A few days' delay for treatment with antibiotics, aspiration bronchoscopy, and possibly core-out of tumor to clear obstruction are prudent. Chronically atelectatic lung can be reinflated, with expectation of useful function. Every attempt should be made to clear the lung of inspissated secretions. Bedside flexible bronchoscopy is invaluable to help clear secretions. The lung may be manually reinflated at thoracotomy by inserting a sterile endotracheal tube into the affected lobar bronchus and passing sterile connecting tubing to the anesthesiologist.

Preoperative Evaluation

Patients must be screened from a medical standpoint to be certain that they can tolerate thoracotomy. Standard pulmonary function testing is imperative. Pneumonectomy is always possible, either for unexpected extent of tumor or technical difficulties. Quantitative ventilation-perfusion scans should be obtained if there is any doubt about the patient's ability to withstand a pneumonectomy, and the potential postoperative function should be calculated. Metastatic work-up is advisable for malignant neoplasms.

Radiologic evaluation helps to determine the extent of involvement by neoplasms and possible lymph node involvement. Computerized axial tomography (CT) of the chest with contrast is obtained in all patients. Magnetic resonance imaging is helpful in evaluating vascular involvement. Spiral CT scans and virtual bronchoscopy are increasingly being used and may have a greater role in evaluating patients.

Bronchoscopy before or at the time of surgery is imperative to evaluate the extent of bronchial involvement, quality of the mucosa, and condition of the lung to be spared. Rigid bronchoscopy with magnifying telescopes is superior for this purpose. Bronchoscopy should be done by the surgeon performing the procedure rather than relying on the observations of others. Mediastinoscopy is important for malignant neoplasms. Under ideal circumstances, it is best performed at the time of planned resection. Although

not an insurmountable problem, the resulting scarring can make node dissection more difficult and distinction between scar and neoplasm more troublesome.

Anesthesia

Anesthetic considerations are discussed in Chapter 18, "Anesthesia for Tracheal Surgery." A technique that allows the patient to be extubated at the conclusion of the procedure is ideal. This avoids risks of endotracheal tubes and positive pressure ventilation on the anastomotic suture line. Double-lumen tubes are suitable for almost all bronchoplastic techniques for the main bronchi and lobes. One exception might be the situation where the entire left mainstem bronchus is to be resected. It may be desirable, in this case, to use a long single-lumen tube to ventilate the right lung. This allows the carina to be encircled from a left thoracotomy. The aortic arch is mobilized to allow delivery of the carina into the operative field for ease of anastomosis (see Figure 29-6*B* in Chapter 29, "Carinal Reconstruction"). Double-lumen tubes are somewhat larger and more rigid, making this maneuver more difficult.

Surgical Technique

General Principles

Certain principles are common to all bronchoplastic techniques. The importance of careful, meticulous surgical technique cannot be stated strongly enough. Delicacy in handling of tissues is imperative. Unnecessary dissection that may devascularize the bronchial blood supply is to be avoided. This is especially an issue when lymph node dissection is performed. Balance must be struck between the desire to remove every last node and the need to preserve crucial blood supply. Mature surgical judgment must guide these decisions. Clean, sharp lines of bronchial transection are important. Jagged, devascularized edges must be avoided. Special concern should attend reoperation on a bronchus (as when prior lobectomy has been done), since previous dissection and scar may damage bronchial blood supply.

Bronchial margins must be examined microscopically intraoperatively to ensure the greatest chance for cure. Frozen sections are imperative. It is often helpful after removing the pathologic specimen to take separate margins from the remaining ends of the airway, properly labeled, to avoid confusion for the pathologist (see Figure 28-2 in Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). Once again, a balance must be struck between the need for clear surgical margins and concern about reconstructing the airway.

A variety of suture materials have been used for bronchial anastomosis. For many years, we have used absorbable sutures to avoid development of suture line granulomas. Suture line granulomas are very rarely encountered with absorbable sutures. Our preference has been 4-0 Vicryl sutures for anastomosis in the adult and 3-0 Vicryl sutures for bronchial traction sutures (instead of the 2-0 sutures used for the larger structure of the trachea). These sutures are strong, slide easily, and do not stretch while tying, giving the surgeon an excellent feel.

Excessive tension is the enemy of successful bronchoplastic procedures. It is less of a concern for most bronchial and lobar bronchoplasties than it is in tracheal anastomosis, but must be avoided when suspected. The gap, after sleeve resection of most lobes, is small and anastomosis produces negligible tension. Resection of the entire left main bronchus, however, plus or minus the upper lobe, is likely to produce excessive tension. The same is true for the right main bronchus when the upper lobe and bronchus intermedius are included. When tension is a concern, division of the inferior pulmonary ligament and a U-shaped pericardial incision just below the inferior pulmonary vein will give added mobility to the bronchus (see Figure 28-5 in Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). It is requisite after total or subtotal excision

of the long left main bronchus. Complete pericardial release is unlikely to be necessary. Hilar release maneuvers are discussed in Chapter 28.

Anastomosis

Open anastomotic technique is preferred, using interrupted sutures, placed to allow the knots to be tied outside of the bronchial lumen. Each suture is precisely placed through the full thickness of the bronchial wall, with minimal handling of the mucosa. Sutures are spaced proportionally with regard to different diameters of the two bronchial margins being approximated. Traction sutures placed in the midlateral wall of both ends of the airway allow for easy manipulation of the airway (see Figure 24-8 in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"), assessment of tension, and, when tied together, reduce tension on the individual anastomotic sutures (see Figure 24-16*B* in Chapter 24). In most bronchoplasties, the length of resection is insufficient to produce significant anastomotic tension, so that traction sutures are chiefly for alignment. The traction sutures are placed full-thickness around a cartilaginous ring, at least one ring distant from the terminal ring where anastomotic sutures are placed. The traction sutures are used to test approximation of the two ends to determine if any release maneuvers might be necessary. They are secured with clamps that are different from the clamps placed on the anastomotic sutures for ease of identification. Traction sutures should not be placed in the membranous wall, since they may tear as they are tied.

We use essentially the same anastomotic technique for bronchi as for the trachea. The sequences of suture placement and the system for keeping the sutures in sequential order for tying are detailed and illustrated in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection" (see Figures 24-14 through 24-16). The following briefly summarizes the steps in the anastomosis. If the two ends of the bronchi are thought of as faces of a clock, the first anastomotic suture is placed at 6 o'clock (see Figure 24-14*A*). Sutures are placed about 3 mm from the cut end of the bronchus and 3 to 4 mm apart. A straight clamp is placed across the suture at a slight angle to prevent the suture from slipping through the clamp. This clamp is secured to the surgical drapes at the head of the table with another clamp. Each subsequent suture is placed in similar fashion in front of the preceding suture (see Figure 24-14*B*). As each successive suture is placed, the surgical assistant retracts the preceding suture with a nerve hook. Sutures are clipped to the drapes sequentially caudad (see Figure 24-14*C*). The sutures will eventually be tied in the reverse order in which they were placed. Back row sutures are placed until the lateral traction suture is reached. The surgeon changes sides of the table to place the remaining back row of sutures on the opposite side in similar fashion, starting again at 6 o'clock and working laterally to the opposite midlateral traction suture. When the back row has been completed, the front row sutures are placed (see Figures 24-15, 24-16). Proper spacing of these sutures is very important. The sutures are clipped to the drapes caudad between the two rows of back wall sutures that lie on either side of the field. These sutures are clipped to the drapes with some slack so as not to hinder the two ends from being brought together when the traction sutures are tied.

After traction sutures are tied, the anterior sutures are tied and cut after each suture. It is important to "slide" each suture to remove any slack that might be present. After the last suture has been tied, the field is flooded with saline and the lung inflated to check for air leaks. The anastomosis should be airtight to a pressure of 35 to 40 cm of water. If any leaks are found, they must be repaired. The anastomosis is inspected with a flexible bronchoscope to identify any problems that might not be apparent from the outside, so that corrections can be made. A pedicled flap of pleura or pericardial fat is then passed around the anastomosis as a buttress and to separate the suture line from nearby vascular structures (see Figure 28-4*B* in Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). Patients should be bronchoscoped before discharge to assess viability, impending fistula, or dehiscence. There is no contraindication to passing a bronchoscope across the anastomosis at this time or irrigating with saline to remove secretions.

Special Circumstances

Size Discrepancies

There often are size discrepancies between the two ends of the airway to be joined. We believe that this is best managed by careful spacing of sutures. Sutures should be placed closer together in the smaller end and farther apart in the larger end. This may lead to telescoping of the two ends, but it has not produced problems and, in fact, might be desirable (Figure 30-1). It is our feeling that removing wedges, creating "pleats," or incising a bronchus are to be avoided. Complex bronchial suture lines can result in problematic healing. There are, of course, exceptions. The most common, albeit rare, situation to arise is when the proximal line of transection is flush with the trachea, creating a considerably larger proximal opening. Partial lateral closure, creating a T-junction of the anastomosis, may be required (Figure 30-2). A pedicled intercostal muscle is preferred to buttress a potential weak spot in the anastomosis. The pedicled muscle is preferably laid on the critical area rather than applied circumferentially and other tissue is used to complete the anastomotic wrap.

Postoperative Pitfalls

Routine postoperative bronchoscopy is invaluable in identifying potential anastomotic complications. A small necrotic area may be managed expectantly if a fistula has not actually developed. The pedicled tissue wrap may seal the area. If a small fistula does develop, proper dependent drainage and cautious irrigation may lead to closure. If major or total dehiscence of the entire anastomosis seems imminent or has occurred, immediate completion pneumonectomy is advised. Because the resulting pneumonectomy stump may be short, under tension, or of poor quality, it is advisable to cover it with a pedicled intercostal muscle flap or omentum. It is better in this circumstance not to attempt reanastomosis unless

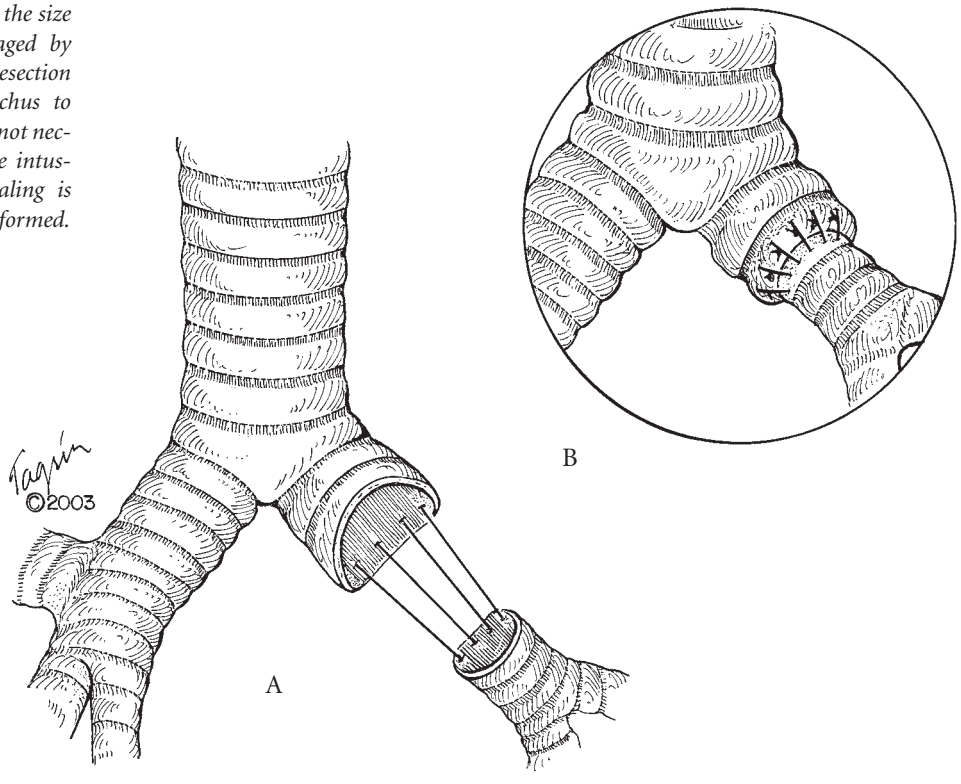

FIGURE **30-1** A, *Discrepancies in the size of the bronchial ends are managed by appropriate spacing of sutures. Resection of wedges from the larger bronchus to reduce its circumference is usually not necessary and is ill-advised.* B, *Some intussusception inevitably occurs. Healing is not impeded, nor are granulomas formed.*

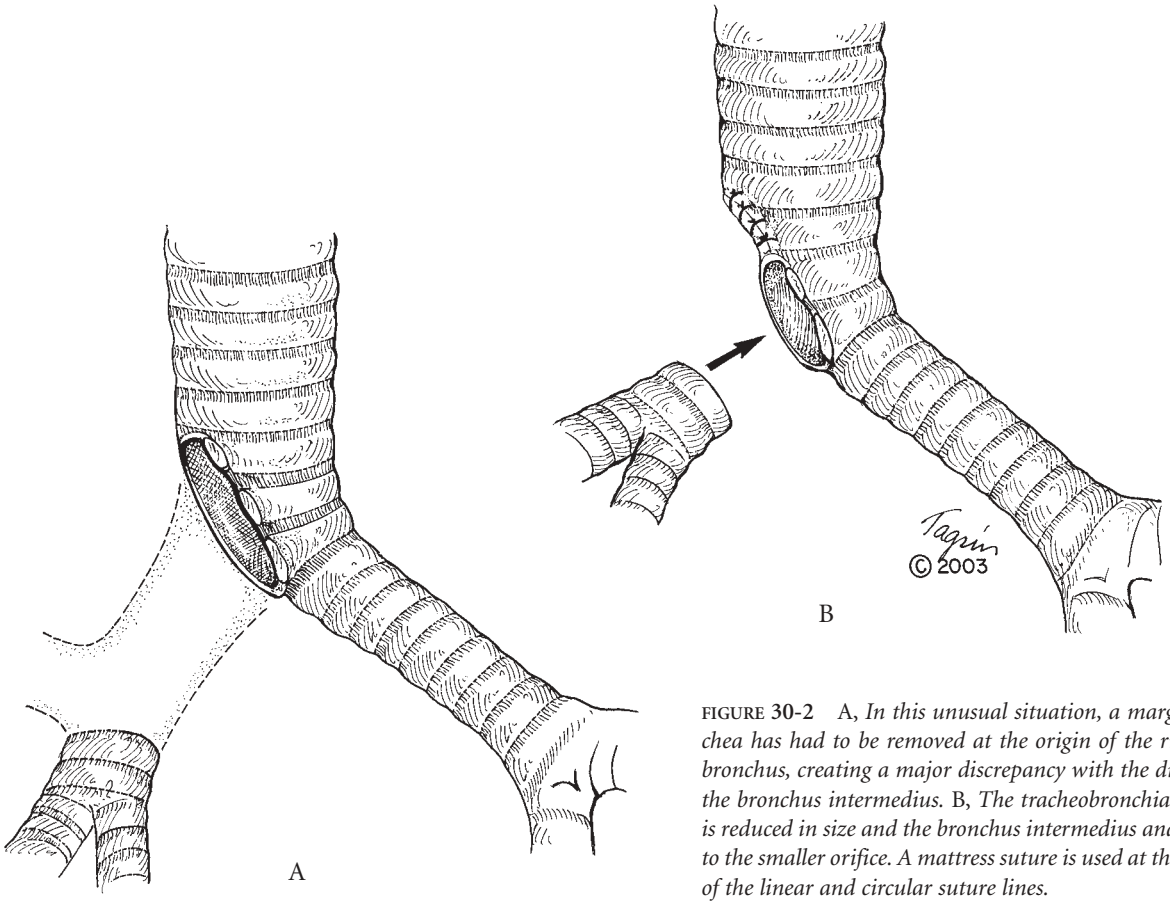

FIGURE **30-2** A, *In this unusual situation, a margin of trachea has had to be removed at the origin of the right main bronchus, creating a major discrepancy with the diameter of the bronchus intermedius.* B, *The tracheobronchial aperture is reduced in size and the bronchus intermedius anastomosed to the smaller orifice. A mattress suture is used at the junction of the linear and circular suture lines.*

there is *no* chance for survival of the patient with a calculated residual postpneumonectomy lung function. Bronchoscopy may reveal an ischemic, but not necrotic ring of bronchus. Bronchoscopy should be performed often until the situation declares itself. If early stenosis occurs, it should be cautiously dilated to maintain patency. Small balloon dilators may be least traumatic. Sometimes, enough of an opening may be maintained that nothing further is required. If stenosis is progressive and symptomatic, reresection may be attempted, but at least 3 months should pass before doing so. Even then, it may be impossible to reconstruct the airway and completion pneumonectomy may be required. A bronchial stent may also be considered.

Specific Bronchoplasties

Right Upper Lobe Bronchoplasty

Right upper lobe bronchoplasty (right upper lobe "sleeve resection") is the most common of all bronchoplasties (Figure 30-3*A*). Initial hilar dissection proceeds as in any right upper lobectomy until the bronchus is reached. The subcarinal space is dissected, and the bronchus encircled by surgical tape at the levels of the right mainstem bronchus and bronchus intermedius. Proximal transection of the main bronchus is usually done first, any incomplete bronchial dissection completed, and the intermedius divided. Specimens for frozen sections are taken, as previously described. As noted, judgment must be exercised as to the extent of lymph node dissection.

Right Upper and Middle Lobe Bronchoplasty

A right upper lobe tumor that involves the middle lobe parenchyma but spares the bronchus intermedius is best managed by right upper lobe bronchoplasty and standard in-continuity middle lobectomy (Figure 30-3*B*). This avoids a major size discrepancy between the right main bronchus and the right lower lobe bronchus. It is important to preserve the peribronchial tissue around the bronchus intermedius and middle lobe bronchus.

If it is necessary to remove the bronchus intermedius up to the right lower lobe bronchus, it is important to preserve as much of the right main bronchus as possible. Oblique transection of the right main bronchus may sometimes facilitate anastomosis, with the obliquity of division of the lower lobe bronchus, which is made necessary when the bronchus intermedius and middle lobe bronchus are removed (Figure 30-3*C*). This is necessarily done at an angle to preserve the superior segmental bronchus of the lower lobe. These steps will minimize size discrepancy and reduce tension as well. When the entire right main stem and bronchus intermedius are removed, it may be necessary to incise the pericardium beneath the inferior pulmonary vein to release the hilar structures and reduce anastomotic tension. The anastomosis is done as described for right upper lobe bronchoplasty.

Right Lower and Middle Lobe Bronchoplasty

When a tumor of the right lower lobe extends proximally along the bronchus intermedius to compromise the point of the bronchial transection, it is possible to preserve the upper lobe (Figure 30-3*D*). The lower and middle lobectomy proceeds as usual until only the bronchus remains. The bronchus is transected at the level of the right main bronchus and the origin of the right upper lobe bronchus. The upper lobe bronchus is properly aligned with the right mainstem bronchus. Size discrepancy is usually managed with careful placement and spacing of anastomotic sutures. Even though the right upper lobe originates perpendicular to the right main bronchus, rotating it 90 degrees aligns the bronchi without causing torsion or kinking of the major vessels. Alternatively, judicious trimming of both bronchial stumps, slightly on the oblique, can simplify alignment. If mediastinal lymph nodes are involved by tumor, this operation may not be oncologically sound. Right pneumonectomy may then be preferable.

Left Upper Lobe Bronchoplasty

Left upper lobe bronchoplasty is very similar to right upper lobe bronchoplasty (Figure 30-3*E*). The dissection is identical to a standard left upper lobectomy until the bronchus is reached. The pulmonary artery is retracted, the mainstem bronchus is divided, and then the bronchus to the left lower lobe. The bronchial ends are naturally in alignment.

Left Lower Lobe Bronchoplasty

This procedure is very similar to the procedure for right lower and middle lobe bronchoplasty (Figure 30-3*F*). The lower lobe resection is carried out in standard fashion until the bronchus is reached. The bronchus to the left upper lobe is divided at its origin, and the left main bronchus just proximal to the takeoff of the left upper lobe bronchus. Traction sutures are placed to ensure proper alignment. The remainder of the anastomosis is carried out as described previously.

I have occasionally had difficulty with left-sided bronchoplastic procedures using standard techniques. The problem has usually been related to airway alignment causing kinking or twisting of the anastomosis. It is difficult to know if this is related to the presence of the aorta or pulmonary artery or both. Sometimes, the problem can be anticipated beforehand because the airway does not appear to line up properly. At other times, it is only determined by bronchoscopy, after completion of the anastomosis. I have dealt with this sit-

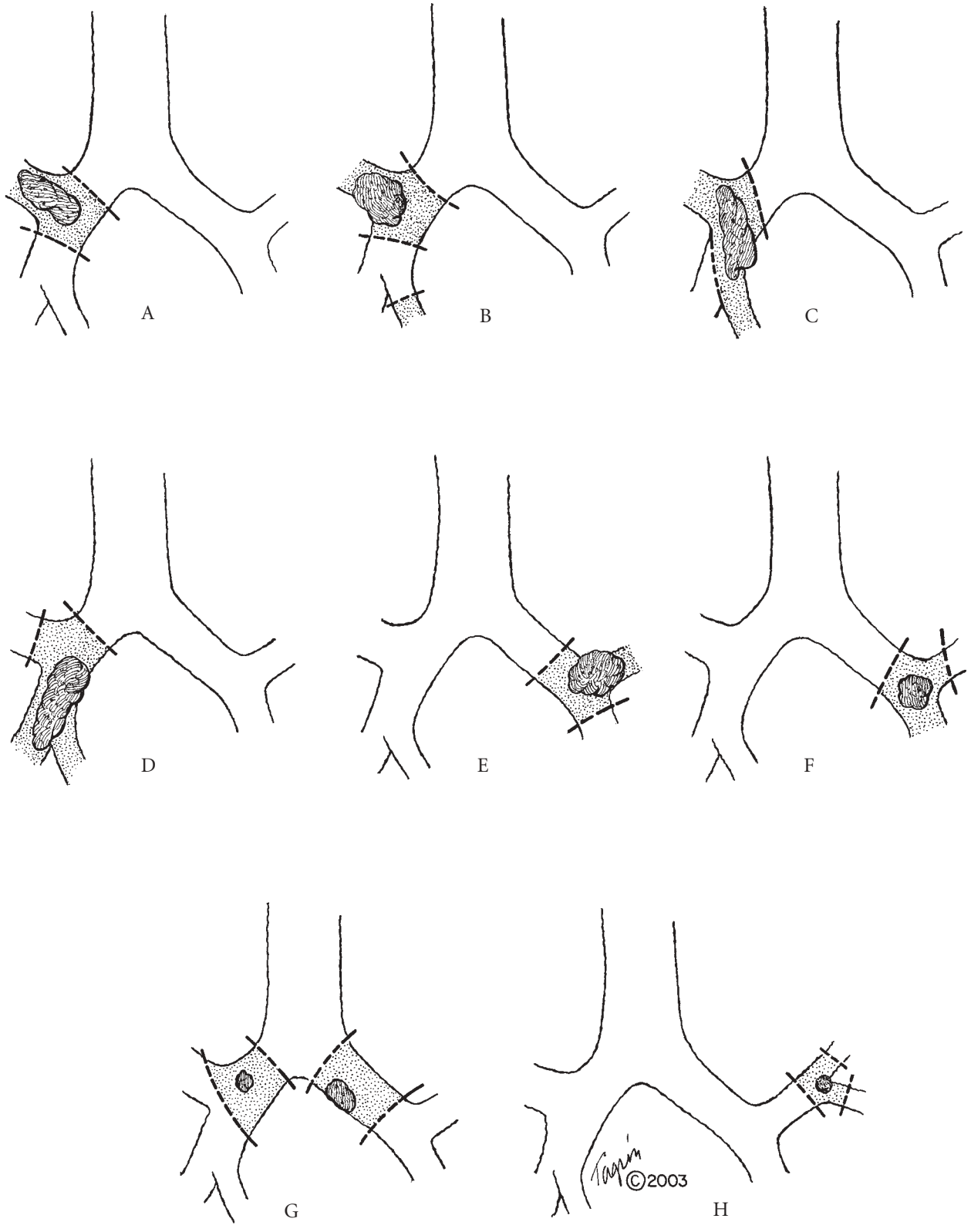

FIGURE **30-3** *Diagrams of specific bronchoplasties.* A, *Right upper lobe.* B, *Right upper lobe and middle lobe.* C, *Right upper and middle lobe with resection of the bronchus intermedius.* D, *Right lower and middle lobe.* E, *Left upper lobe.* F, *Left lower lobe.* G, *Main bronchial resection.* H, *Segmental bronchoplasty.*

uation by modifying the standard technique as follows. Traction sutures still help with alignment, reduction of anastomotic tension, and bringing the ends of the airway together. The modification consists of placing and tying individual sutures serially. The traction sutures are held together to reduce tension on the first two sutures, and ultimately are tied at the completion of the anastomosis. As each subsequent anastomotic suture is carefully placed and tied, the surgical assistant helps to "roll" the distal bronchus into perfect alignment. Alternatively, beveling the bronchial ends can assist in alignment for anastomosis.

Right and Left Mainstem Bronchi

Resection of the mainstem bronchi encompasses the same principles as the previously described procedures (Figure 30-3*G*). The single most important difference is exposure of the left mainstem bronchus. If the resection requires removal of the proximal left mainstem bronchus, it may be necessary to mobilize the aortic arch for adequate exposure. The aorta is dissected circumferentially to allow passage of tapes for retraction, in order to expose the bronchus fully (see Figure 29-6*B* in Chapter 29, "Carinal Reconstruction").

Care must be taken to avoid injury to intercostal vessels originating from the aorta. These can be difficult to repair. The recurrent laryngeal nerve must be protected from direct traction or injury, since it passes beneath the aorta. It may be necessary to encircle the distal trachea and right mainstem bronchus with tapes for retraction. With additional cervical flexion, this allows the carina and the proximal left main bronchus to be delivered into the operative field, simplifying anastomosis. Resection of most or all of the left mainstem bronchus produces tension, which will require intrapericardial mobilization. Angulation of the pulmonary artery has not been a problem.

On rare occasions, a limited lesion involving only the very proximal left mainstem bronchus may be approached from the right side. If more than just the very proximal left main stem is involved, it would be very difficult to do the operation through a right thoracotomy. Airway management is more complicated in this case. Since exposure is through the right chest, it is desirable to deflate the right lung. This is accomplished with an extra-long single-lumen endotracheal tube, positioned in the left mainstem bronchus at the beginning of the operation. The right lung remains deflated during dissection of the left main bronchus. After the left mainstem bronchus is transected and the specimen removed, ventilation is carried out across the operative field with a sterile endotracheal tube inserted into the open left mainstem bronchus. The tube can be safely removed for brief periods of apnea to allow suture placement. Traction sutures and anastomotic sutures are placed exactly as described previously. Once all anastomotic sutures have been placed, a decision must be made on how to ventilate the patient while tying the anastomotic sutures. The long endotracheal tube may be advanced back into the left main bronchus and the left lung ventilated. This is the most convenient option for tying the sutures, but it may make approximation of the ends of the airway difficult. The right lung may be ventilated intermittently and retracted while sutures are tied. This method is somewhat awkward. A high-frequency ventilation catheter can be advanced from above to ventilate one or both lungs and is an effective method of maintaining ventilation. The final method is to use the endotracheal tube (in the trachea) to ventilate the patient, accepting some air leak until the anastomosis has been completed. A finger can be used to temporarily occlude the opening in the anastomosis if more efficient ventilation is required.

Segmental Bronchoplasty

We have performed a number of sublobar sleeve resections. Very precise anastomosis of smaller segmental bronchi is required. In some cases, such as excision of the carina between the upper and lower divisions of the left upper lobe bronchus for a tiny carcinoid tumor, without pulmonary resection, exposure is difficult because all segmental vessels must be preserved (Figure 30-3*H*). With patient dissection and meticulous reconstruction, results have been good in these cases. Repeated flexible bronchoscopic aspiration may be necessary postoperatively to prevent early atelectasis.

Bronchoplastic and Angioplastic Procedures

When the tumor involves both the bronchus and pulmonary artery, consideration is given to sleeve resections of both the bronchus and pulmonary artery in order to avoid pneumonectomy. Judgment must be exercised to determine whether such a procedure can be done without violating oncologic principles.

Combined angioplastic and bronchoplastic procedures have historically been associated with higher morbidity and mortality rates than bronchoplasty procedures alone.[8] Morbidity and mortality have decreased, and survival increased, as more experience has been gained. Major complication has been related to the demands of the pulmonary artery anastomosis. Just the right amount of tension is required to avoid kinking, twisting, or narrowing of the artery. It is imperative to interpose a viable tissue pedicle between the two suture lines to minimize the risk of bronchovascular fistula. When the point in the operation is reached for division of the artery and airway, proximal and distal control of the pulmonary artery must be secured. Intrapericardial control of the artery may be advisable. Proximal occlusion requires a vascular clamp or a tourniquet, whereas distal control is achieved either with the use of vessel loops around the lower lobe pulmonary arterial trunk and its branches, or by occlusion of the pulmonary veins. It is preferable to perform the arterial anastomosis first. Heparin 5,000 U is administered intravenously prior to clamping. The pulmonary artery is either divided to conduct a sleeve resection or part of the circumference is resected. I prefer 4-0 or 5-0 continuous Prolene sutures for the arterial anastomosis. Two sutures are used to avoid the risk of "purse stringing" the anastomosis. Sleeve resection shortens the artery, avoids angulation, and is preferred to near-circumferential patching, particularly if a bronchial sleeve resection is combined with arterial resection. After lateral resection, transverse closure is considered to prevent kinking. For patch repair, autologous or bovine pericardium is available. Rendina and colleagues have reported the use of a conduit made of autologous pericardium,[9] but prefer bovine pericardium for patch repair because the material is stiffer and easier to handle.[10] Before tying the last suture, the proximal clamp is removed to flush any clots. The same is done with the distal clamp. If concern arises about patency of the arterial anastomosis, pressures taken on either side of the anastomosis will show if a significant gradient exists. The bronchoplasty is performed in exactly the same manner as earlier described. After completion, the reconstruction needs to be carefully inspected during inflation of the lung for angulation of the artery. While in hospital, subcutaneous heparin is administered at doses for prophylaxis of deep vein thrombosis. No additional anticoagulation is administered.

Editor's Note

"Sleeve lobectomy"—bronchoplastic resection—was slow to be adopted by thoracic surgeons. Earlier concerns about oncologic validity have been allayed by data that support its use in appropriately selected patients (see Chapter 16, "Bronchial Sleeve Resection"). Two feared complications—bronchostenosis and bronchovascular fistula—show a vanishing incidence when the procedure is done in accord with the principles that Dr. Mathisen elucidates. These are precise dissection, preservation of blood supply, meticulous anastomosis, elimination of anastomotic tension, anastomotic buttressing, and interposition of viable tissue between bronchial and vascular structures.

Hermes C. Grillo

References

1. Gaissert HA, Mathisen DJ, Moncure AC, et al. Survival and function after sleeve lobectomy for lung cancer. Thorac Cardiovasc Surg 1996;111:948–53.
2. Deslauriers J, Gaulin P, Beaulieu M, et al. Long-term clinical and functional results of sleeve lobectomy for primary lung cancer. J Thorac Cardiovasc Surg 1986; 92:871–9.
3. Faber LP, Jensik RJ, Kittle CF. Results of sleeve lobectomy for bronchogenic carcinoma in 101 patients. Ann Thorac Surg 1984;37:279–85.
4. Newton JR, Grillo HC, Mathisen DJ. Main bronchial sleeve resection with pulmonary conservation. Ann Thorac Surg 1991;52:1272–80.
5. Bueno R, Wain JC, Wright CD, et al. Bronchoplasty in the

management of low-grade airway neoplasms and benign bronchial stenoses. Ann Thorac Surg 1996; 62:824–9.
6. Mathisen DJ, Grillo HC, Vlahakes G, Daggett WM. The omentum in the management of complicated cardiothoracic problems. J Thorac Cardiovasc Surg 1988; 95:677–84.
7. Muehrcke DD, Grillo HC, Mathisen DJ. Reconstructive airway surgery after irradiation. Ann Thorac Surg 1995;59:14–8.
8. Schirren J, Muley T, Vogt-Moykopf I. Sleeve lobectomy. In: Shields TW, LoCicero J III, Ponn JB, editors. General thoracic surgery. Philadelphia: Lippincott Williams & Wilkins; 2000. p. 399–409.
9. Rendina EA, Venuta F, DeGiacomo T, et al. Reconstruction of the pulmonary artery by a conduit of autologous pericardium. J Thorac Cardiovasc Surg 1995; 110:867–8.
10. Rendina EA, Venuta F, DeGiacomo T, et al. Sleeve resection and prosthetic reconstruction of the pulmonary artery for lung cancer. Ann Thorac Surg 1999; 68:995–1002.

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Repair of Tracheobronchial Trauma

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Acute Trauma

The diversity of tracheal trauma in location, extent, complexity, and possible involvement of the adjacent larynx, esophagus, and major vessels demands a variety of approaches, techniques, and ingenuity for repair. These aspects are reviewed in Chapter 9, "Tracheal and Bronchial Trauma." Principles of repair are as follows:

1) Immediate establishment of a secure airway;
2) Complete assessment of the extent of injuries including those of the larynx, the functional state of vocal cords, possible esophageal laceration, damage to major vessels, and spinal injury;
3) Optimal exposure for repair;
4) Conservative débridement of damaged tracheobronchial tissue;
5) Meticulous reconstruction of all injured structures;
6) Internal laryngeal splinting after a crushing injury to the larynx;
7) Provision of an adequate postoperative airway, usually by tracheostomy, if glottic paralysis or swelling exists;
8) Gastrostomy and jejunostomy, if needed, to prevent aspiration and to provide alimentation due to compromised laryngeal function or significant esophageal injury.

Typical injuries are illustrated in this chapter. The surgeon must use initiative and ingenuity in dealing with the multiple variations that may present, while following established principles of tracheal reconstruction.

Cervical Trauma

Airway Management. The first priority in management of upper airway injury is establishment of a dependable airway. Even the patient with little or no sign of airway obstruction, but with suspected or demonstrated airway separation may decompensate rapidly. Equipment for intubation, flexible bronchoscopy, rigid bronchoscopy, and tracheostomy should be immediately available. An anesthesiologist should

be in attendance. The surgeon should attempt to pass an endotracheal tube, preferably over a flexible bronchoscope (see Figure 10-1*A* in Chapter 10, "Tracheostomy: Uses, Varieties, Complications"). Blind intubation is better avoided, although it may succeed. The ends of the separated trachea may be offset, and the appearance of the gap on bronchoscopy may be confusing, with torn tissue and blood. The fact that the patient can breathe, however poorly, proves that there is an air channel through the disruption. Usually, a flexible bronchoscope can be threaded into the distal trachea and the endotracheal tube slipped in over it. Some assessment of the larynx also is made in passing, but it may not be entirely satisfactory. Rigid bronchoscopy is considerably more difficult, unless the patient is less responsive. It should not be used if there is any hint of cervical spine injury. Drugs that depress respiration are to be avoided. If an attempt to insert a flexible bronchoscope fails, the surgeon should proceed directly to emergency tracheostomy. This is one of the few remaining indications for emergency tracheostomy. Since all maneuvers may have to be telescoped into a few minutes, it is essential that necessary instruments be at hand at the outset. Upon entering the neck, the surgeon may encounter a mass of lacerated, swollen, and contused tissues and blood clots. The trachea will not be seen if it has retracted into the mediastinum. The simplest way to find the distal trachea in these circumstances is to insert a finger into the mediastinum to seek the lumen. The edge of the trachea is grasped with an instrument such as Allis forceps and drawn upward into the base of the neck. The torn distal end is intubated directly across the field.

After suctioning and stabilization of the patient, two courses of action are open. Examination of extent of injury may be completed and the airway injury repaired. If more pressing concerns demand attention, such as major vascular injury, intracranial damage, or intra-abdominal hemorrhage, or if the attending surgeon is not versed in the techniques of airway reconstruction, it may be preferable to settle for the security of a tracheostomy. The distal end of the severed trachea is fixed to tissues at the base of the neck with a few sutures, and a cuffed tracheostomy tube is placed directly into the distal end. Minimal or no débridement is done. The neck is drained. Damage to the esophagus or pharynx must be repaired at once and suitably reinforced to prevent secondary leakage. If tracheostomy alone is elected for interim management of the injury, the tube should be placed in the already open end of the distal trachea and *not* in a new opening made in the tracheal wall below the rupture. The latter would further damage the trachea, accomplish nothing of value, and complicate future definitive repair. The proximal airway opening is exteriorized or drained.

Approach. The cervical trachea is approached through a low-collar incision. Extension is not often needed, but the field should be prepared to permit vertical partial or complete sternotomy.

Tracheal Laceration. A partial injury confined to the anterior tracheal wall alone is débrided conservatively if the injury is blunt, and repaired directly, if possible, with interrupted 4-0 Vicryl sutures. A penetrating injury caused by a sharp instrument such as a knife is more simply repaired (Figure 31-1*A*). Loss of a piece of anterior tracheal wall, as occurs due to a missile, whether a flying industrial fragment or a bullet, on rare occasions may be débrided and repaired. However, if the damaged segment is of any great length, it is best managed by resection of the involved segment of trachea and end-to-end anastomosis (Figure 31-1*B*). The surgeon must be wary of wide wedge resections, which can result in obstructive kinking of the trachea. In these missile-induced injuries, the airway is established easily by endotracheal intubation because the trachea remains in continuity. Tracheostomy is not routinely necessary. Its use depends on the adequacy of glottic function. If tracheostomy is performed distal to the level of injury, it is sealed from the anastomosis by suturing strap muscles or the thyroid isthmus over the anastomosis and to the trachea. If the level of the injury pushes the tracheostomy into potential proximity to the brachiocephalic artery, the artery is protected from the tracheostomy site by suturing a strap muscle obliquely across the anterior surface of the trachea beneath the tracheostomy and above the artery (Figure 31-1*C*).

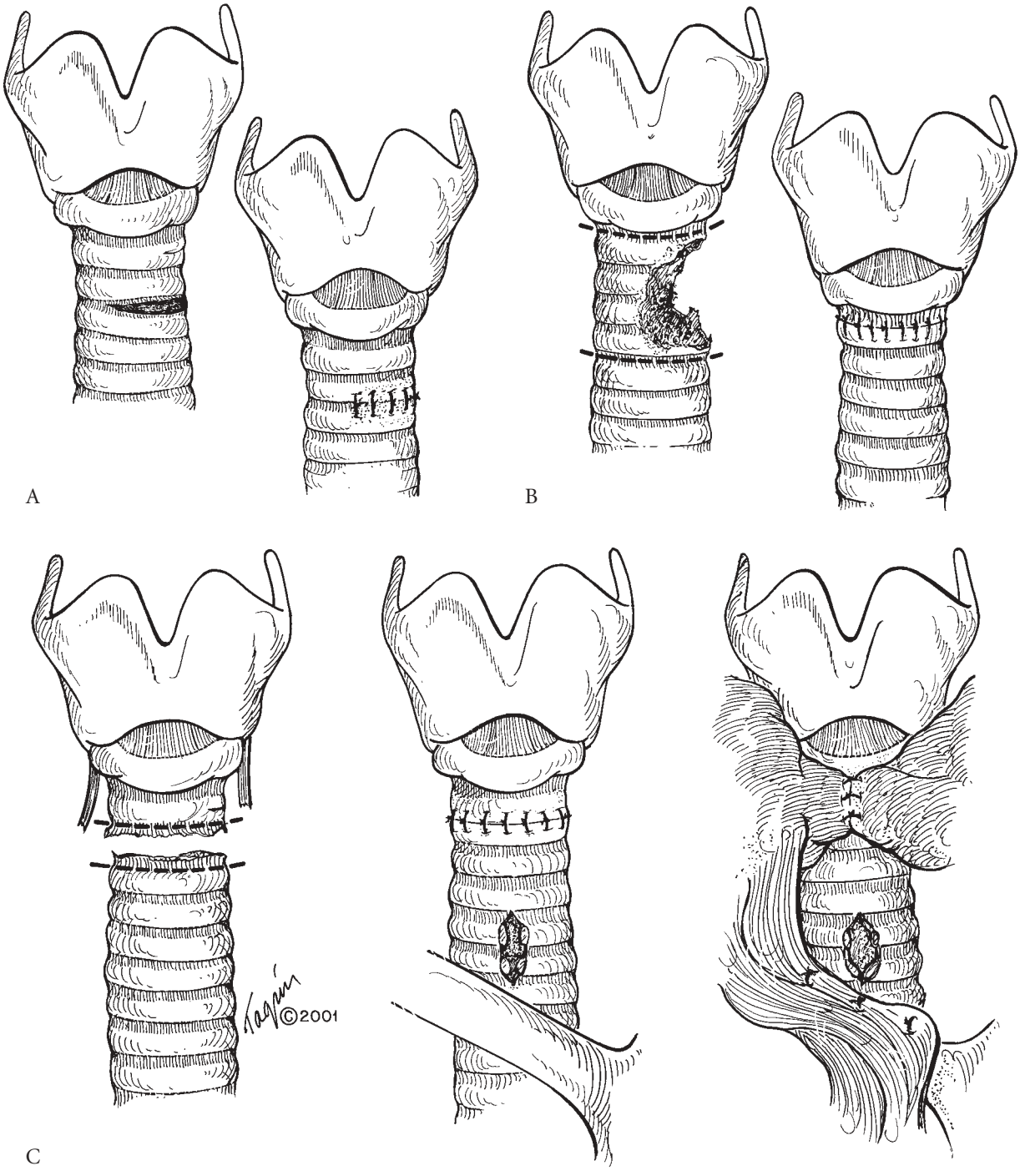


FIGURE **31-1** *Acute cervical tracheal injuries.* A, *A sharp wound such as that caused by a knife is cleansed and sutured with interrupted 4-0 Vicryl sutures, after inspection of the surrounding structures (esophagus, carotid artery, jugular vein). A recurrent laryngeal nerve may be traumatized.* B, *Irregular penetrating injury caused by a bullet or a military or industrial fragment requires débridement at minimum. Usually, limited circumferential resection is preferable to angulated closure after wedge excision, and to local tissue patches, which are subject to leakage, accumulation of granulation tissue, and cicatricial stenosis.* C, *Tracheal separation is most often due to blunt cervical trauma ("clothes line" or "dashboard" injuries). One or both recurrent laryngeal nerves may be injured. Limited débridement and a direct anastomosis is performed. Hence, tracheostomy distal to injury and repair is needed. If the tracheostomy is adjacent to the brachiocephalic artery, sternohyoid muscle is sutured to the trachea beneath the stoma to protect the artery from erosion. The anastomosis is covered by suturing strap muscles together or by suturing the thyroid isthmus over the anastomosis (and to the trachea). If endotracheal intubation is selected but tracheostomy is likely to be needed after a few days, coverage of the anastomosis and the artery is done, and the site of the future tracheostomy is marked with a single silk suture.*

Tracheal Separation. A patent airway must be ensured urgently after acute tracheal separation. Cervical tracheal separation due to blunt injury often occurs just beneath or a short distance below the cricoid cartilage (see Figure 31-1*C*). Serrated tracheal margins are conservatively débrided. Careful determination is made by esophagoscopy and intraoperative examination that the esophagus is not lacerated. Instillation of dye high into the esophagus may be difficult to interpret since dye may flood up through the cricopharyngeus and down through the glottis, and appear in the operative field. Following débridement, the trachea is precisely anastomosed with 4-0 Vicryl sutures, using the technique described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection." Since negligible loss of trachea has occurred, approximation is without tension, despite the apparently wide separation initially observed of the tracheal ends. In complete tracheal separation, recurrent laryngeal nerves are either temporarily or, more often, permanently injured unilaterally or bilaterally. Tracheostomy is usually needed distal to the line of repair. If tracheostomy is clearly necessary, the endotracheal tube for intraoperative ventilation may be placed at the outset through a vertical tracheostomy incision in an elective location below the laceration, thus simplifying débridement and anastomosis. Tracheostomy should lie at least 1 to 1.5 cm away from the anastomosis and not directly adjacent to the brachiocephalic artery. If these conditions cannot be met, it may be safer to manage the patient with endotracheal intubation for 4 or 5 days until the area has sealed, and then to establish a tracheostomy in a previously marked site.

Recurrent laryngeal nerves are not dissected since there are presently no reliable methods for reanastomosis. The larynx is reassessed by a skilled otolaryngologist after initial healing to determine what further procedures may be necessary to improve function, thereby permitting eventual closure of the tracheostomy.

Tracheal Separation with Laryngeal Damage. It is not the purpose of this section to describe all intricacies of blunt or sharp trauma to the larynx. However, the larynx may incur diverse injuries due to the level and force of the trauma in addition to avulsion of trachea from larynx (see Figure 9-1 in Chapter 9, "Tracheal and Bronchial Trauma"). Conservative débridement is the rule. Assessment of laryngeal injury is best performed with the cooperation of an experienced otolaryngologist. Early repair of complex laryngeal injuries should be made by an otolaryngologist. Therefore, only a brief statement about these injuries follows. Laryngeal structures such as a fractured or separated thyroid or cricoid cartilages should be repaired carefully with Vicryl sutures. Arytenoid and vocal cord injuries require repair. A trimmed tracheal edge is precisely anastomosed to the inferior margin of a repaired cricoid cartilage (Figure 31-2*A*). If laryngeal injuries are significant, internal splinting is advisable. Stents have been devised for this purpose, such as the molded silicone stents devised by Montgomery.[1] The tracheostomy is placed distal to a laryngotracheal anastomosis (Figure 31-2*B*). In some cases, a T tube passing through the vocal cords may serve as an appropriate splint (Figure 31-2*C*). Eliachar and Nguyen devised a hollow-shaped laryngeal stent that rests on a tracheostomy tube lying just below it (Figures 31-3*A,B*).[2]

Separation of the airway at the cricotracheal junction may cause avulsion of posterior subglottic laryngeal mucosa from the cricoid plate. If this occurs, only limited débridement of the irregular edges of the flap attached to the trachea should be done. The preserved flap is used to resurface the bared posterior cricoid cartilaginous plate (Figure 31-4*A*). All bared cartilage should be covered with mucosa. If the flap is of insufficient length to surface the cartilage easily, slight further removal of anterior tracheal cartilage, especially on the lateral sides, will adequately lengthen the posterior flap. This flap is similar to that prepared surgically for primary repair of laryngotracheal stenosis (see Chapter 25, Laryngotracheal Reconstruction"). If the cricoid cartilage has not also been divided anteriorly by the injury, exposure for repair is facilitated by a limited anterior midline cricoid fissure (Figure 31-4*B*).

Tracheal and Esophageal Separation. Blunt injuries that lead to laryngotracheal separation or upper tracheal rupture also may produce laceration or separation of the upper esophagus, or pharyngoesophageal separation.

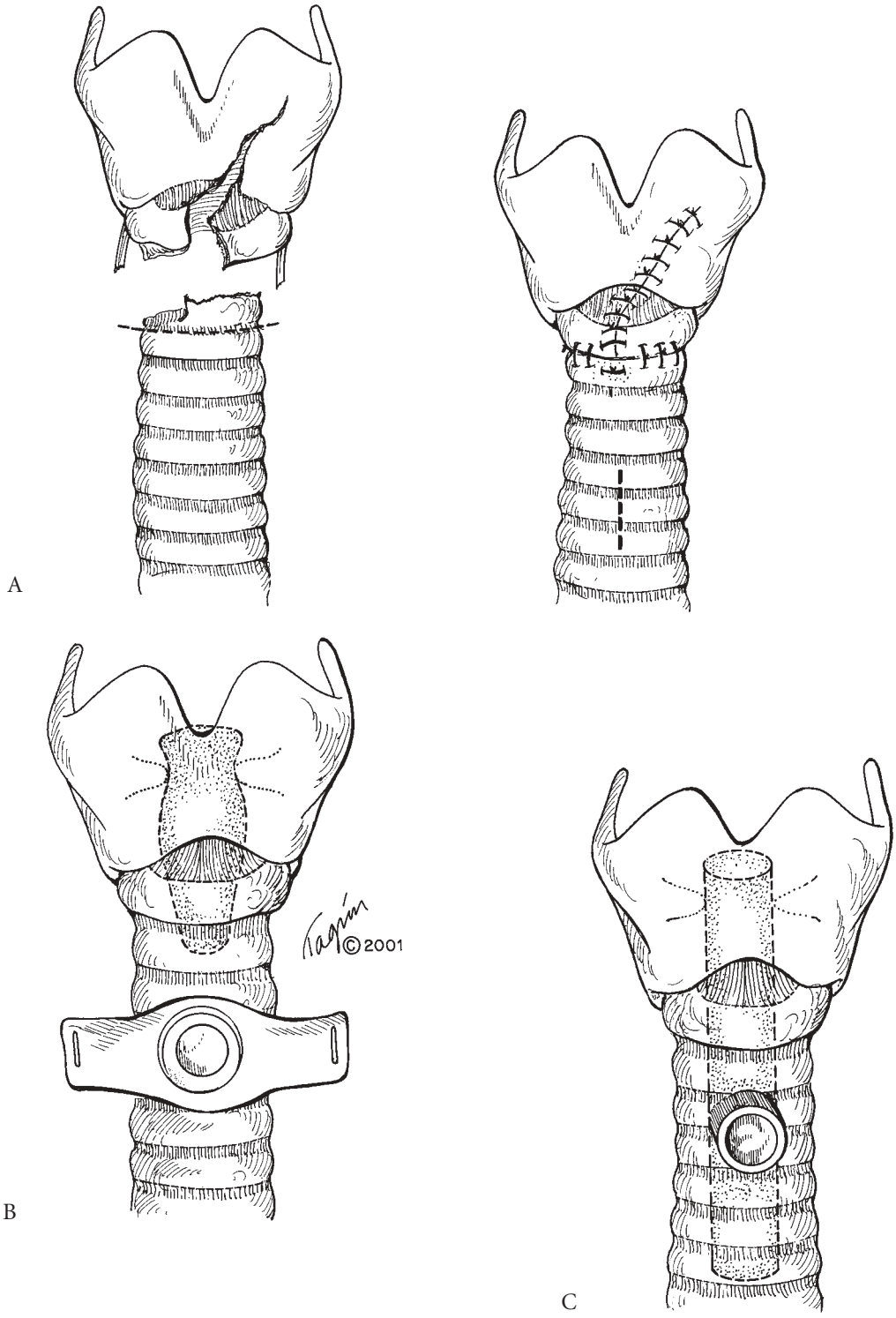


FIGURE **31-2** *Blunt trauma resulting in laryngotracheal separation and laryngeal fracture.* A, *Many patterns of injury may occur, including fractures of supra- and infraglottic larynx, vocal cord injury, arytenoid dislocation, and mucosal laceration. Very conservative laryngeal and conservative tracheal débridement precedes repair. The larynx and glottis are meticulously repaired, and laryngotracheal anastomosis is performed. A mattress suture can be employed at the "T" meeting of laryngeal and laryngotracheal suture lines. Distal tracheostomy is used for intraoperative anesthesia, since it is required postoperatively due to recurrent nerve injuries.* B, *If the larynx is crushed or deformed by the injury, it is reconstructed around a Montgomery laryngeal molded stent. The tracheostomy tube is in place.* C, *Silicone T tube employed as a stent. The proximal end lies in the laryngeal ventricle, below the false vocal cords.*

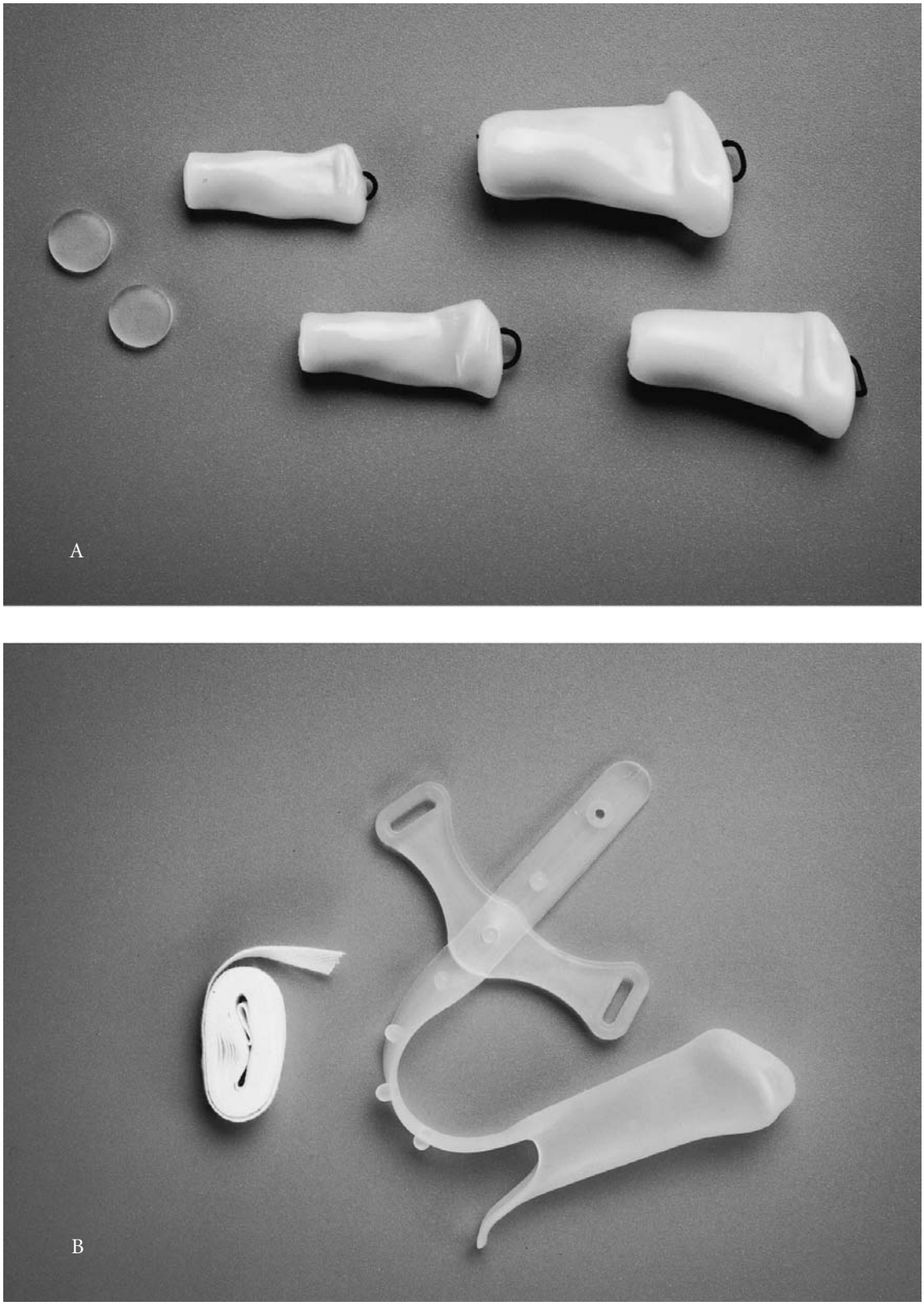


FIGURE 31-3 A, *Montgomery molded silicone laryngeal stent designed to conform to the endolaryngeal surface. It is available in three sizes: adult male, adult female or adolescent, and child. The stent is held in place with sutures.* B, *Eliachar laryngeal stent. It is held in place against a tracheostomy tube and by contour, with an added strap tied to the neck.*

Complete traumatic separation of the airway provides easy access to the esophagus (Figure 31-5*A*). However, anatomic planes may be confused by the trauma. Access is improved by placing traction sutures of 2-0 Vicryl in the lateral laminae of the cricoid cartilage. These must be firmly anchored in cartilage, but they do not enter the lumen of the larynx. Elevation of the larynx by traction on these sutures improves access to the retracted proximal pharyngoesophageal or esophageal tissues. Identifying sutures are placed at critical points in the esophageal mucosa proximally. The distal end is similarly identified. Very conservative débridement is accomplished. Precise two-layer anastomosis of the distal to the proximal esophageal segments or of the distal esophagus to the proximal pharyngeal segment is accomplished (Figure 31-5*B*). The author prefers 4-0 Vicryl sutures. In circumferential separation, the first layer consists of interrupted mattress sutures from the posterior muscularis of the distal esophagus to the proximal posterior esophageal or cricopharyngeal musculature. Next, the posterior mucosa is approximated with interrupted sutures, placed so that the knots lie inside the lumen. After completing the back wall of the mucosal anastomosis, these sutures are continued anteriorly, inverting the mucosa as they are tied. Anastomosis is difficult when avulsion is at the cricopharyngeus. Minimal dissection may be required anteriorly to free up an edge of tissue between the pharyngeal mucosa and the back of the pos-

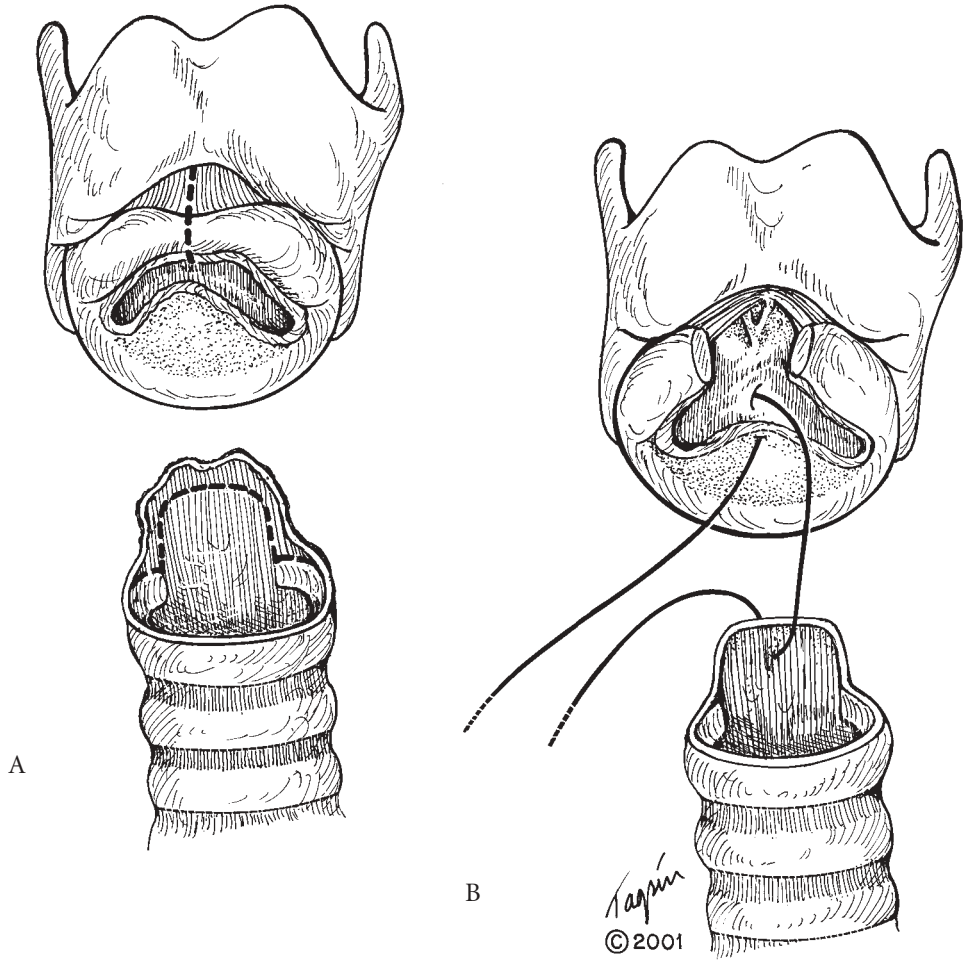


FIGURE **31-4** *Blunt laryngotracheal rupture with avulsion of posterior laryngeal mucosa.* A, *Anterior fissure of cricoid and cricothyroid membrane provides access to posterior subglottic larynx. Mucosal edges are very conservatively trimmed. The posterior mucosal flap avulsed from over the posterior plate of cricoid is conservatively débrided, as is the trachea anteriorly and laterally.* B, *The avulsed posterior flap is used to cover the bared posterior plate of cricoid. Interrupted 4-0 Vicryl sutures are placed with knots outside of the mucosa. The technique is as described for laryngotracheal reconstruction in Chapter 25, "Laryngotracheal Reconstruction." Tracheostomy is used for intraoperative anesthesia as in Figure 31-2*A.

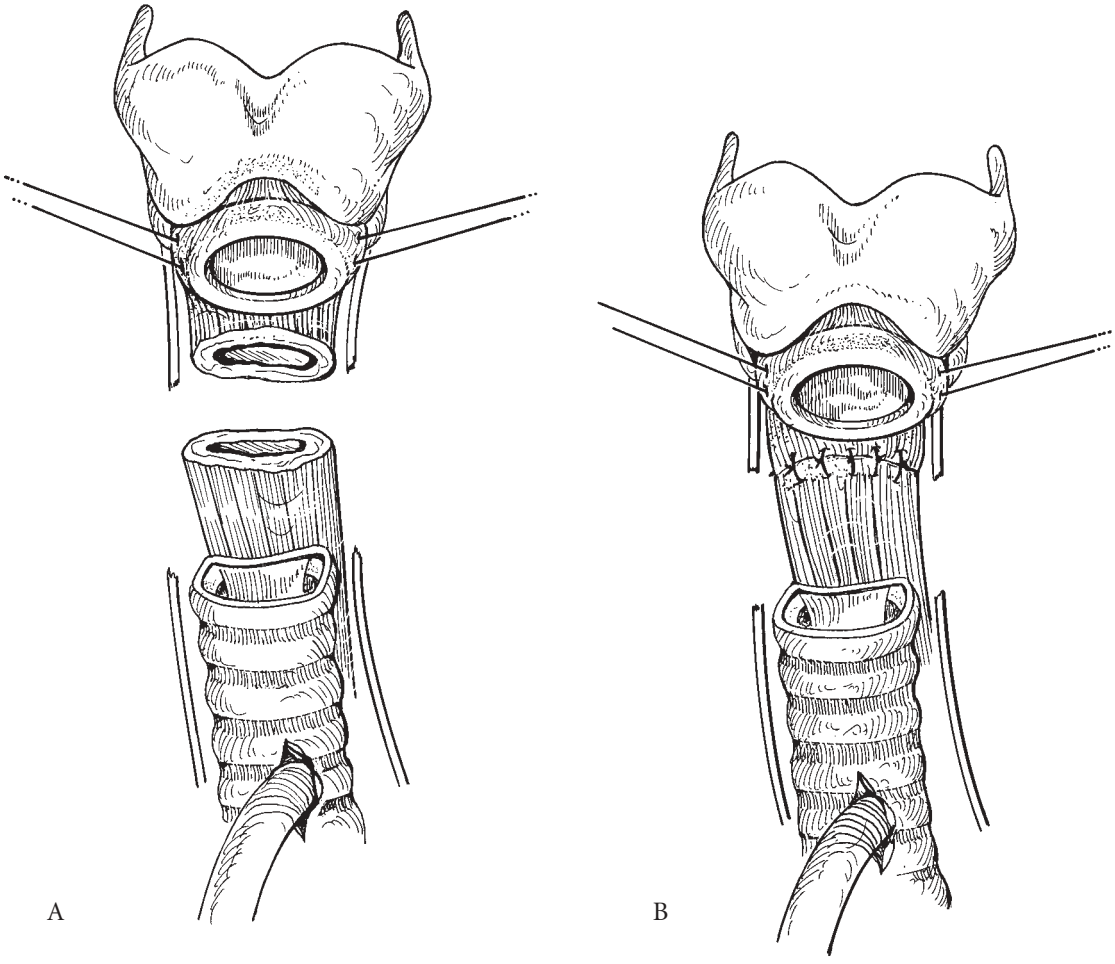


FIGURE 31-5 *Combined cervical tracheal and esophageal rupture due to blunt injury.* A, *The esophageal injury may be anterior or circumferential. Access is improved with traction sutures of 2-0 Vicryl firmly placed extraluminally in the lateral cricoid laminae. Irregular edges are débrided. Note the endotracheal tube in the elective tracheostomy incision. The laryngeal and tracheal margins also are conservatively débrided.* B, *Esophageal or pharyngoesophageal reconstruction is performed with two layers of interrupted 4-0 Vicryl (or silk) sutures, according to Richard Sweet's technique (see text). The mucosal layer is inverted. Limited dissection behind the posterior margin of posterior cricoid provides sufficient mucosa and tissue for a layered anastomosis. Special care is used in dissection, even though recurrent laryngeal nerves are likely to have been divided in such an injury if esophageal division is complete.*

terior cricoid plate. After the mucosal closure has been completed, it is usually possible to suture the anterior esophageal musculature either to muscle or to connective tissue lying just behind the lower edge of posterior cricoid. With lower lacerations, suturing is correspondingly easier. Extreme care and precision is necessary to obtain satisfactory closure. A pedicled strap muscle is sutured transversely over the esophageal closure prior to restoring the airway (Figure 31-5*C*). This interposition prevents later tracheoesophageal fistula. Tracheostomy is almost always needed (Figure 31-5*D*). Linear esophageal lacerations are minimally débrided, closed in two layers with interrupted sutures, and covered with a pedicled strap muscle.

Iatrogenic Tracheal Laceration. Lacerations of the trachea caused by endotracheal intubation are linear and occur in the membranous wall of the distal trachea centrally or at the junction with the cartilaginous wall. These may extend into the main bronchus, principally the right side (see Chapter 9, "Tracheal and Bronchial Trauma"). Upper lacerations are approached cervically. Lower injuries are usually repaired

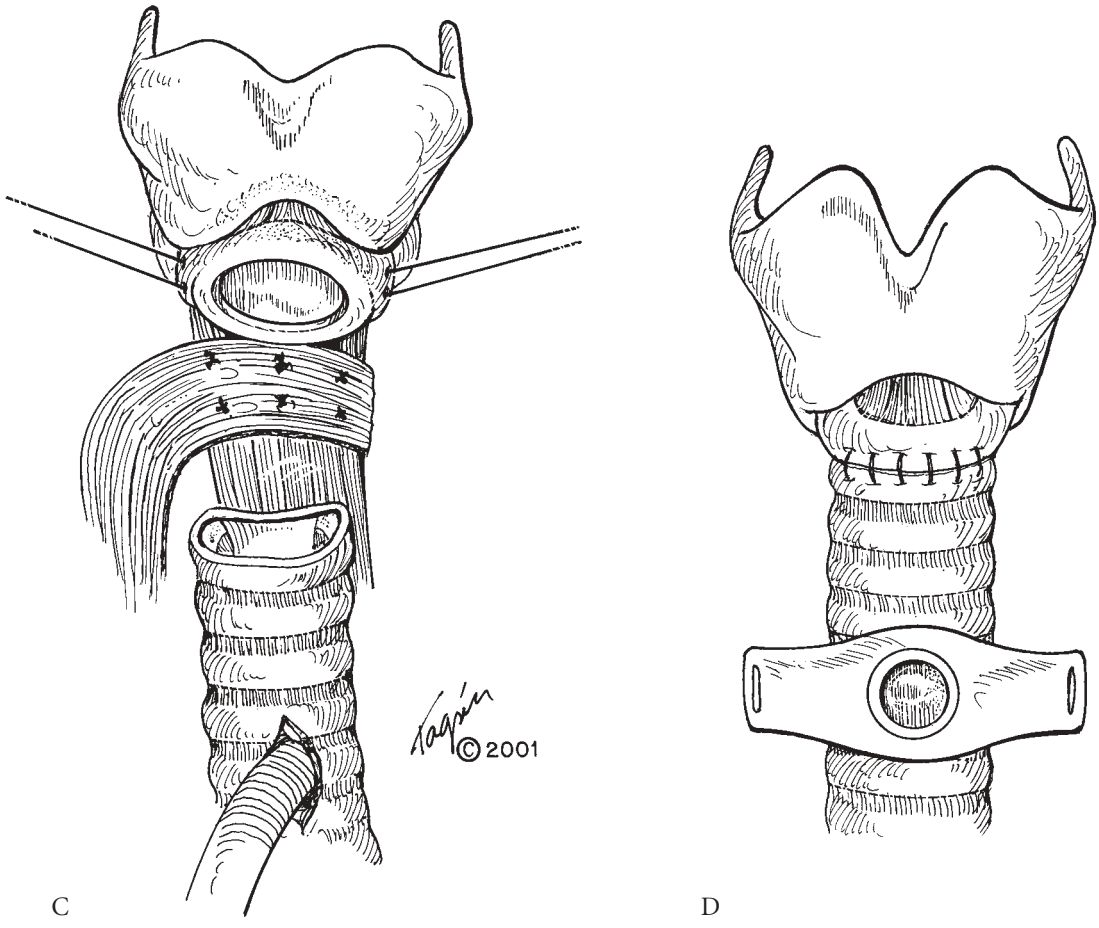


FIGURE **31-5** (CONTINUED) C, *A pedicled sternohyoid muscle is sutured over the enteric anastomosis. Its bulk does not interfere with laryngotracheal anastomosis.* D, *Laryngotracheal (or tracheotracheal) anastomosis is completed in the usual fashion using 4-0 Vicryl sutures. A tracheostomy tube is placed at the conclusion of the operation.*

transthoracically. Angelillo-Mackinlay has described access to a midtracheal membranous tear via anterior linear tracheotomy.[3] A laceration near the carina, especially if it extends into the main bronchus, is approached most readily by thoracotomy, usually on the right side. In the unlikely case in which the entire trachea must be exposed, a cervicomediastinothoracic or "trapdoor" incision extending to the right side may be used. Although concomitant esophageal injury is rare in these patients, it should be ruled out.

Tracheal laceration generally is best repaired when recognized. In selected cases, conservative treatment may be successful but only with careful observation.[4] The laceration should be linear and short in length, clinical manifestations should be minor, and the situation should be stable and without progression of signs or symptoms. When these are in question, prompt and skillful repair is more appropriate.

Thoracic Trachea and Bronchi

Crushing chest injuries can result in trauma to the mid- and lower trachea. Partial or complete transverse tracheal division may follow sternal fracture and other chest wall injuries (see Chapter 9, "Tracheal and Bronchial Trauma"). A spiraling rupture from the carina, either through the cartilaginous or membranous tracheal wall, may occur alone or in conjunction with main bronchial injuries (see Chapter 9, "Tracheal and Bronchial Trauma").

Precise bronchoscopic identification of the injury and its level, as well as priorities of concomitant injuries, determine the approach. The rare injuries confined to the midtrachea, particularly if they are anterior or transecting, are managed by upper sternal division with retraction of the great vessels. Débridement and repair follow the principles of tracheal reconstruction. Transpericardial exposure is a further option, but it is unlikely to be necessary (see Chapter 23, "Surgical Approaches").

Injuries to the lower portion of the trachea, those to the carina, and those that involve the main bronchi are best approached transthoracically for adequate exposure. A right posterolateral thoracotomy is usually the most satisfactory overall incision. It allows access to the trachea, carina, and esophagus, and also to both right and left main bronchi. If the left main bronchus alone is ruptured, a left posterolateral thoracotomy (fourth interspace or fifth rib) is preferred. The aortic arch may be retracted; care must be taken not to injure the left recurrent laryngeal nerve. The carina may be brought into view with tapes around the trachea and the right main bronchus. A flexible endotracheal tube permits this angulation (see Chapter 29, "Carinal Reconstruction"). If a cervical spine fracture complicates intrathoracic tracheal, carinal, or bronchial injuries, the spine may be stabilized with cranial tongs. A tracheal approach is then accomplished through a median sternotomy or a trapdoor exposure, which does not require a lateral thoracotomy position. The trapdoor incision allows easier access to the posterior wall of the trachea, bronchi, and esophagus than does a sternotomy alone.

Lobar and segmental bronchi also can be repaired if pulmonary damage is not too severe. A contused and hemorrhagic lung is often salvageable despite an initially discouraging appearance. An extra-long endotracheal tube with a flexible armored tip (a Wilson tube) is placed in the intact bronchus with the aid of a flexible bronchoscope (see Chapter 18, "Anesthesia for Tracheal Surgery"). The bulk and relative rigidity of double-lumen tubes can make tracheobronchial repair difficult. The endotracheal tube can be further adjusted or guided by finger pressure intraoperatively. Intubation across the operative field is not usually needed, but tubing and equipment for cross-field ventilation should be available since findings may be unpredictable, despite preoperative evaluation. Jet ventilation is unlikely to be needed, but it should be available for injuries that prove to be complex, such as a tracheal rupture at the carina combined with bilateral bronchial lacerations.

Cardiopulmonary bypass should be avoided because the heparinization necessary may produce bleeding at the sites of trauma and operation. On an exceedingly rare occasion, such as the finding of a concurrent false aneurysm of the pulmonary artery at the origin of the main pulmonary artery in conjunction with bronchial rupture, bypass may be needed.[5]

After limited débridement, more or less linear tracheal and bronchial lacerations are repaired directly with interrupted 4-0 Vicryl sutures (Figures 31-6*A,B*). Transections and near transections are conservatively trimmed to intact tissue and anastomosed. Less often, limited resection and reanastomosis is indicated, particularly with missile wounds. Lacerations may be irregular. Débridement of the tracheal wall in linear or spiral injuries is necessarily limited. For this reason, as well as on general principle, the author believes it advisable to buttress intrathoracic repairs. The pericardial fat pad elevated carefully to preserve blood supply offers a good second layer, particularly for circumferential tracheal or bronchial suture lines. It serves to seal the repair, and it provides important tissue interposition between suture line and adjacent major blood vessels. An intercostal muscle pedicle flap is excellent to buttress a long membranous wall injury or a linear esophageal repair, interposed between the esophagus and the trachea, where suture lines would otherwise be adjacent (Figure 31-6*C*). It should *not* be used for circumferential wrapping (see Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). Other tissues that may be used for second layers, although less effectively, are the pericardium and the pleura.

Esophageal perforation or laceration must be sought, both by direct inspection and by instillation of a bolus of methylene blue-stained saline into the esophagus through a nasogastric tube. The esophagus is repaired in two layers with interrupted sutures.

The tracheobronchial repair should be visualized via flexible bronchoscopy at the close of the procedure. Prompt or early extubation is preferred. If postoperative ventilation is required, an effort is made to avoid placing the cuff of the endotracheal tube against the suture line. Gas pressure of ventilation is less inimical to healing than is the irritating contact of an inflated cuff. The lowest effective pressures should be employed for ventilation and for cuff inflation.

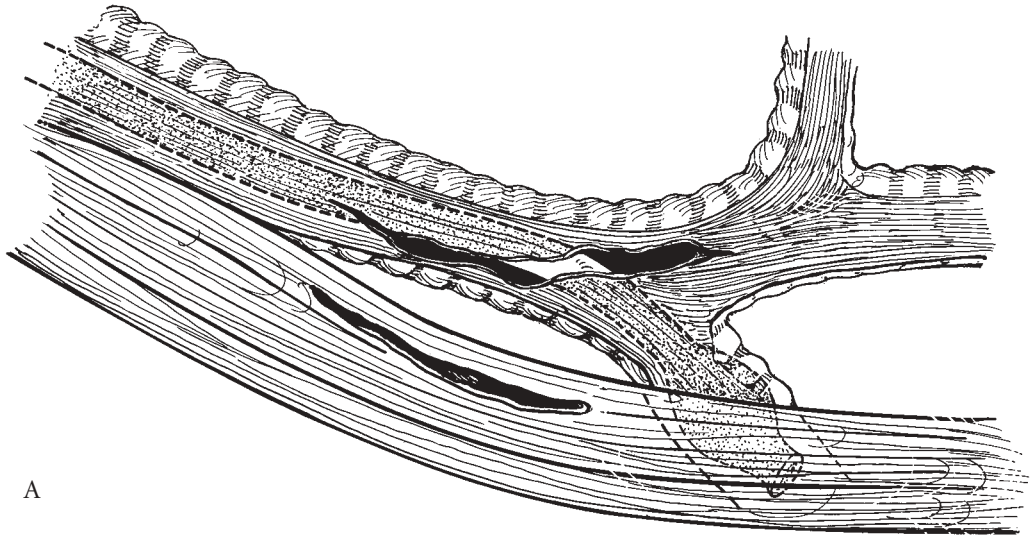
Chronic Injuries

Trachea

A tracheostomy is usually present in patients who present for delayed repair of tracheal separation. Unilateral or bilateral vocal cord paralysis is likely to be present. Several of our patients have also had acquired tracheoesophageal fistulae.[6] A patient must no longer require ventilation assistance to be a suitable candidate for an operation. An interval of 4 to 6 months should elapse after injury or previous tracheal surgery to allow for subsidence of inflammation and maturation of a scar. If further procedures are anticipated to correct or ameliorate other systems (such as orthopedic operations), these are best completed before the tracheal repair to avoid risk of repeated ventilation after tracheal reconstruction.

Complete evaluation of laryngeal structure and function is essential and is preferably done by an experienced otolaryngologist. This author prefers to see any laryngeal deficits corrected while the preexisting tracheostomy is still in place, to be certain that correction is achieved prior to the tracheal repair. If simultaneous repair of the glottis and trachea is done, a new tracheostomy or splinting T tube is usually necessary. The procedure becomes more complicated if the trachea must be markedly shortened in the repair. As emphasized in Chapter 9, "Tracheal and Bronchial Trauma," a completely paralyzed larynx can be made functional and will subsequently justify reconnection of a chronically separated trachea.

After complete radiologic and endoscopic examination, repair is undertaken as described in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection," and Chapter 25, "Laryngotracheal



A

FIGURE **31-6** A, *Intrathoracic lower tracheal laceration with accompanying esophageal tear. Exposure is via a right posterolateral thoracotomy after guided intubation of the left main bronchus with an extra-long endotracheal tube. Only enough dissection is done to expose the injuries fully. The closeness of the left recurrent laryngeal nerve in the left tracheoesophageal groove is kept in mind. Tracheal lacerations may vary in extent, location, and spiral course, and may involve the main bronchi. Little débridement is necessary or permissible.*

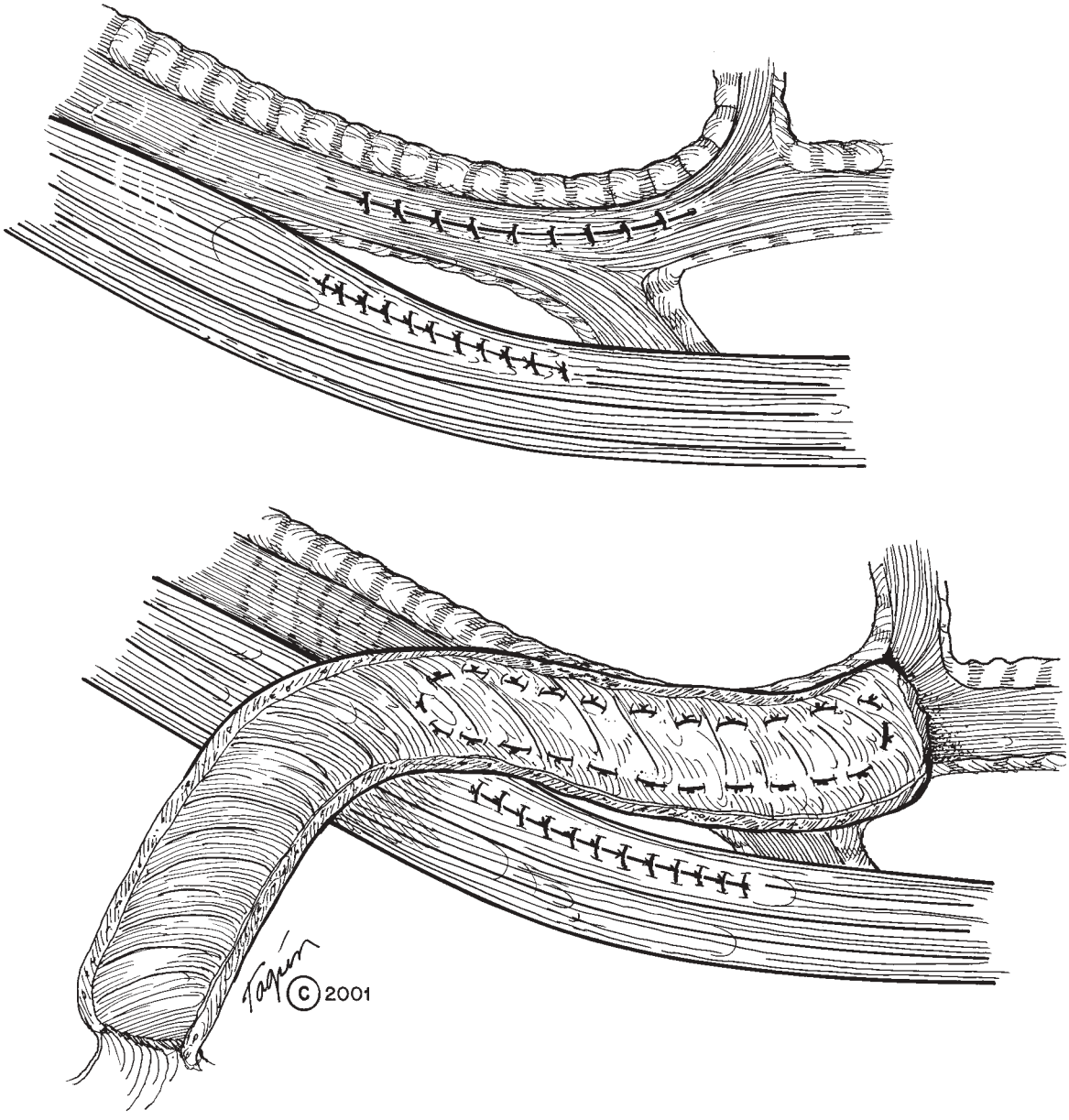


FIGURE **31-6** (CONTINUED) B, *The tracheal laceration is repaired with interrupted 4-0 Vicryl sutures. The esophagus is closed with two layers of interrupted 4-0 sutures. The mucosa is inverted, and the muscularis is closed with mattress sutures.* C, *A long intercostal muscle pedicle has been raised, preferably during the thoracotomy, on the basis of identification of the tracheal laceration on preoperative bronchoscopy. The pedicle is sutured over the line of tracheobronchial repair with multiple fine sutures—not merely "tacked" in place. There should be no air leak on a test at this point. The esophageal closure usually falls on the pedicle. Additional sutures may be used to buttress the esophageal suture line against the intercostal flap.*

Reconstruction." A dense post-traumatic scar is to be anticipated. Since a severed trachea descends into the mediastinum, the gap between proximal and distal airway segments may not indicate the true length of destroyed trachea. Dissection of the anterior surface of distal trachea may initially be difficult, and care must be taken to protect the brachiocephalic artery. If a tracheoesophageal fistula is present, it is repaired and buttressed with a pedicled strap muscle prior to completing the airway anastomosis. Since laryngeal adequacy is established prior to late tracheal reconstruction, there should be no need for a complementary tracheostomy.

Mid- or lower tracheal fracture may initially go unrecognized and present as a stricture. Such a patient usually has not undergone prior tracheal surgery and is a candidate for early repair. The anterior approach described in Chapter 23, "Surgical Approaches," is used.

Bronchi

A delay in the diagnosis of bronchial rupture is too common. Resection of the resultant stricture with primary reconstruction must be done as soon as it is diagnosed. A right or left posterolateral thoracotomy is elected as is appropriate. A single-lumen Wilson tube is placed in the opposite main bronchus for anesthesia. The bronchus is trimmed to healthy tissue on either side, without stripping its blood supply. The distal bronchial tree is suctioned, irrigated, and cultured. Gentle insufflation prior to anastomosis will demonstrate the expandability of the atelectatic obstructed lung. Even long-collapsed lungs may regain significant function. Precise anastomosis is done with 4-0 Vicryl interrupted sutures. On rare occasions, intrapericardial hilar mobilization may be a useful adjunct. The anastomosis is wrapped as previously noted. Bronchoscopic examination is performed before chest closure.

References

1. Montgomery WW. Surgery of the upper respiratory system. Vol 2. 2nd ed. Philadelphia: Lea and Febiger; 1989. p. 683.
2. Eliachar I, Nguyen D. Laryngotracheal stent for internal support and control of aspiration without loss of phonation. Otolaryngol Head Neck Surg 1990;103:837–40.
3. Angelillo-Mackinlay T. Transcervical repair of distal membranous tracheal laceration. Ann Thorac Surg 1995;59:531–2.
4. Jougon J, Ballester M, Choukron E, et al. Conservative treatment for postintubation tracheobronchial rupture. Ann Thorac Surg 2000;69:216–20.
5. Symbas PN, Justicz AG, Ricketts RR. Rupture of the airways from blunt trauma: treatment of complex injuries. Ann Thorac Surg 1992;54:177–83.
6. Mathisen DJ, Grillo HC. Laryngotracheal trauma. Ann Thorac Surg 1989;43:254–62.

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Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea

Hermes C. Grillo, MD

Tracheomalacia
Tracheopathia Osteoplastica
Postpneumonectomy Syndrome
Straight Back Syndrome
"Hairpin" Aorta
Cutaneous Tube Reconstruction of the Trachea

In this chapter, techniques are described that are used to correct airway obstruction due to the following conditions:

1) Acquired tracheomalacia
2) Tracheopathia osteoplastica
3) Postpneumonectomy syndrome
4) Straight back syndrome
5) "Hairpin" aorta

Treatment of tracheal compression due to two unique conditions—*postpneumonectomy syndrome* and *straight back syndrome*—are described below. Compression due to an unusual variant of a vascular ring, which I term "*hairpin*" *aorta*, is also noted. Obstructive compression by *extrinsic masses* is discussed in Chapter 15, "Tracheobronchial Malacia and Compression," but requires no special surgical techniques for correction, other than added treatment of the infrequent circumstance of residual malacia after removal of the compressing mass. Since clinical experience in treating the special conditions listed here is limited, comment is made on whether each procedure should be considered as proven effective treatment or still remains an exploratory trial.

A method is also described for staged reconstruction of a tracheal gap with a splinted cutaneous tube. This principally provides a historical example of an earlier type of "trough" procedure, made generally obsolete by the techniques developed for tracheal resection and reconstruction. It is not regularly used or recommended. Remote circumstances, where it might possibly be useful, are however noted.

Tracheomalacia

Expiratory Collapse with Chronic Obstructive Pulmonary Disease

The type of tracheomalacia that often accompanies chronic obstructive pulmonary disease (COPD) is manifest as expiratory collapse. The curve of the intrathoracic tracheal cartilages is flattened, the extreme tips of the

cartilages sometimes curve slightly forward, the cartilages are thinned and softened, and the membranous wall is markedly widened. The membranous wall approximates to the flattened cartilages on expiration and cough (see Figures 15-2, 15-3, in Chapter 15, "Tracheobronchial Malacia and Compression," and Figure 39 [Color Plate 16]). Since the cartilages still retain considerable substance, correction is obtained by restoring them to a C shape, by pulling the tips closer together, while shortening the membranous wall to a more normal width. This is accomplished by attaching longitudinal strips of splinting material along the membranous walls of the intrathoracic trachea, the right and left main bronchi, and the bronchus intermedius. Redundant membranous wall is quilted to the splint to prevent its infolding into the lumen of the airway.

I early abandoned using strips of fascia lata as a material for splints, since an additional incision was required. Pericardium was used only briefly, for it seemed to attenuate with time. Goretex failed because it could not become firmly incorporated into the tracheal wall by ingrowth of scar tissue. Obstruction recurred in some. Numerous splinting materials have been used by others, including perforated solid plastic strips, lyophilized bone, and absorbable synthetic mesh. I found Marlex (monofilament knitted polypropylene mesh; Davol Inc., Cranston, RI) to be very satisfactory. It is easily sutured into place and holds sutures well. Tissue ingrowth into its interstices fully and permanently incorporates the splint into the tracheobronchial walls, thus maintaining the restored curvature of the cartilages and preventing fluid from accumulating between the membranous wall and prosthetic strip as occurred with Goretex.

Preoperative bronchoscopy is repeated for careful assessment of the extent of malacia. Left lung ventilation is established with a long single lumen endotracheal tube bronchoscopically placed in the left main bronchus. The posterior wall of the entire thoracic trachea is exposed transpleurally through a right posterolateral fifth rib (or intercostal space) incision (see Figure 28-1 in Chapter 28, "Reconstruction of the Lower Trachea [Transthoracic] and Procedures for Extended Resection"). The azygos vein is divided. The posterior trachea is dissected to the apex of the chest. The right vagus nerve and its branches to the hilum are divided for exposure. Only the edges of the cartilages on either side along the margins of the membranous wall are freed for suturing, thus preserving tracheal blood supply. The membranous walls of both main bronchi and the bronchus intermedius are similarly exposed, if the malacic process extends this far, as usually it does. It is possible to expose even the distal end of the left main bronchus from this approach. As with the trachea, the bronchi are *not* dissected circumferentially, but only sufficiently to free up 3 to 5 mm of cartilaginous edges along the margins where the cartilages meet the membranous wall.

A strip of Marlex is cut approximately 2 to 2.5 cm wide (depending on the size of the trachea and the width of the membranous wall) and long enough to extend from the apex of the trachea which is accessible in the thorax, to a few centimeters beyond the carina or longer as noted below. Four rows of interrupted sutures are placed across the width of the trachea from the Marlex strip to the tracheal wall, spaced 6 to 8 mm apart (Figure 32-1*A*). Suturing is commenced at the top of the accessible trachea. One longitudinal row of sutures will extend along each edge of the membranous wall and fixes the borders of the Marlex strip to the tips of the cartilages on either side. The two other longitudinal central rows fix Marlex to the membranous wall (see Figure 32-1*A*). These rows are spaced by eye across the membranous wall, dividing it into approximately even thirds. Each succeeding row of four sutures placed transversely is about 6 to 8 mm below the previous one. Suturing progresses from the apex of the chest to the carina.

I have used both 4-0 Tevdek and 4-0 Vicryl for suture material. Effort is made to avoid penetrating the tracheal mucosa. Where this has occurred on rare occasion, even a nonabsorbable suture such as Tevdek has not, thus far, caused infection or other late problems. Vicryl, although ultimately absorbable, has such a long life that fibrosis and Marlex incorporation should occur long before the suture is absorbed. I therefore used Vicryl in later patients and have recognized no problems to date. The marginal sutures are placed through the edge of the Marlex from posterior to anterior, then through a cartilage end from lateral to posterior—a firm purchase being taken—and thence back through the Marlex (see Figures 32-1*A*,*B*). This is

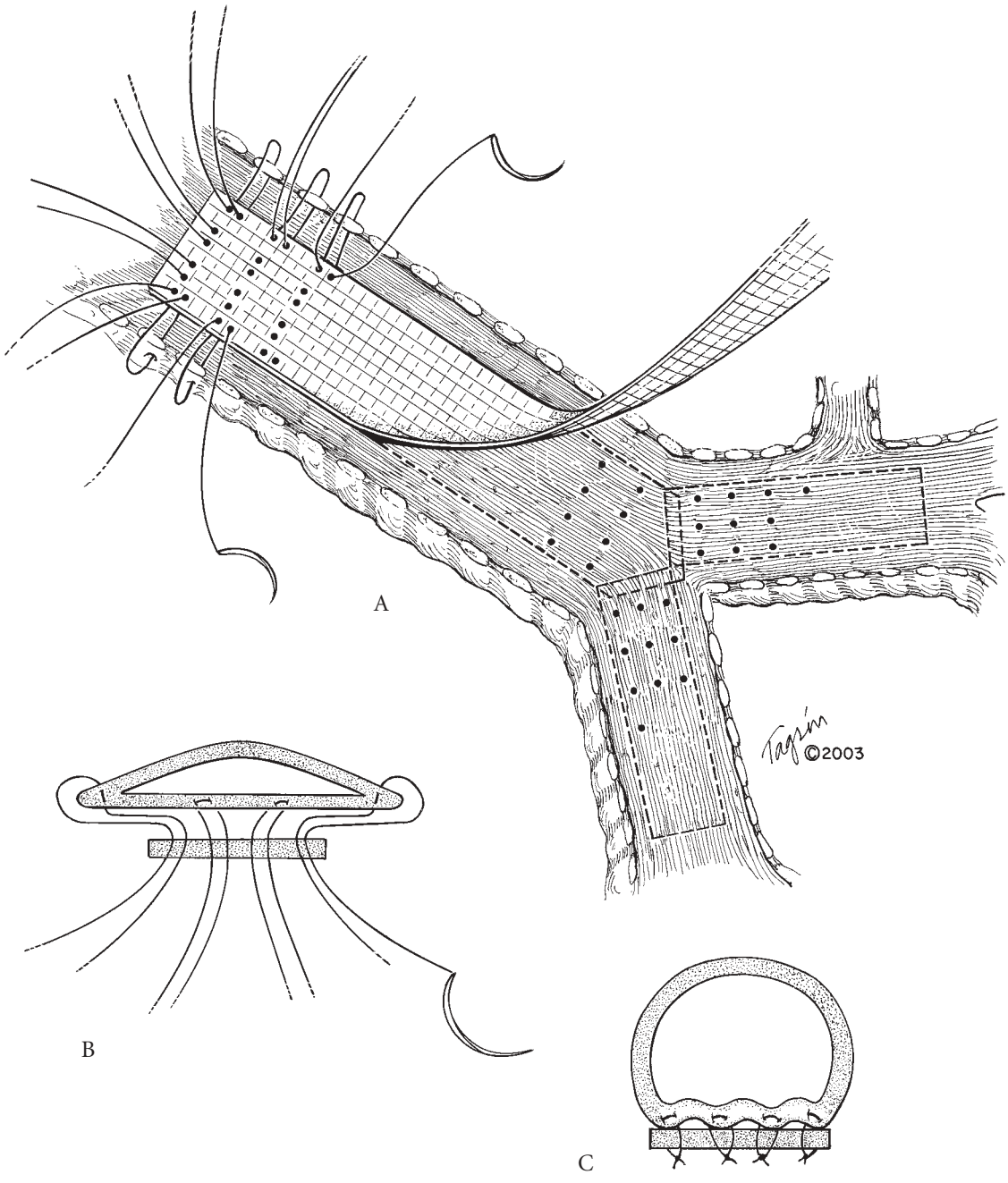

FIGURE **32-1** *Posterior splinting procedure for expiratory tracheobronchial collapse.* A, *Marlex strip of desired width is sutured to the posterior tracheal wall from the apex of the chest to the carina, or in some, to the distal bronchus intermedius. Successive registers of four sutures (4-0 Vicryl) are placed across the Marlex as shown. The outer sutures grasp the lateral tips of the cartilages; the inner two fix the redundant membranous wall in equal thirds. Each row is about 6 to 8 mm distal to the prior row. Lateral sutures of each row are placed first. After three rows of sutures are placed, the first row may be tied. One previous row is left untied as you progress distally, to facilitate placement of the next row.* Dots *in the proximal Marlex indicate sites for following sutures. The distal* dashed lines *indicate where the Marlex will be applied to the lower trachea and to the bronchi. Three strips of Marlex are usually used, but occasionally, a narrowed extension of the tracheal strip may be carried onto the right main bronchus and bronchus intermedius.* Dots *indicate placement of distal sutures in the Marlex. Note three longitudinal rows in the bronchial strips versus four in the tracheal.* B, *Cross-sectional diagram showing spacing of sutures. The lateral ones will restore the "C" curve of the flattened cartilage. The central ones fix the elongated membranous wall to the Marlex. These sutures do not penetrate the membranous wall.* C, *The completed restoration of airway configuration. Scar will firmly fix the membranous wall and lateral tracheal corners to the Marlex splints. The correction is checked bronchoscopically after completion.*

not a true mattress suture, but serves well to pull the cartilage to the Marlex when tied. The two median rows of sutures are true mattress sutures.

I prefer to place the lateral sutures of the apical row first, then the two apical central membranous wall sutures—each of these spaced equally across one-third of the membranous wall. Thus, an initial horizontal row of four sutures is placed. Each suture is clipped with a hemostat and arranged systematically, two on either side of the operative field. Successive rows of four similar sutures are next placed, proceeding distally toward the carina. When all tracheal sutures are in place, they are tied—lateral sutures of each horizontal group of four initially, followed by the central two of each group of four, the quilting sutures (Figure 32-1*C*). When the carina is reached, the Marlex strip is trimmed, usually perpendicular to the axis of the bronchus on either side (see Figure 32-1*A*). Sometimes, it is convenient to tie the rows successively to anchor the strip as it is sutured. If this is done, it is best not to tie down an immediately preceding row, since this narrows the exposure for placement of the next row of sutures.

A strip of Marlex approximately 1.5 cm wide is next applied to the left main bronchus using only three longitudinal rows of sutures, commencing distally as near to the bronchial bifurcation into lobar bronchi as possible. When the proximal end of the bronchus is reached at the carina, the Marlex strip is trimmed. It is cut to fit to the tracheal Marlex strip with slight overlap (see Figure 32-1*A*). The final row of sutures at the origin of the bronchus includes both tracheal and bronchial walls and both the tracheal and bronchial strips of Marlex, thus fastening all of these together. The same is done for the right main bronchus and the bronchus intermedius—again with only three longitudinal rows of sutures (see Figures 32-1*A*, 32-2), using a single strip of Marlex. Sometimes, a single long strip of Marlex has been carried from the tracheal wall to extend over the right main bronchus and bronchus intermedius, but narrowed over the bronchi.

After all sutures are tied, the correction achieved is examined by flexible bronchoscopy, prior to closing the thoracotomy. The correction should be substantial but will not, of course, appear like a wholly normal trachea or bronchus (see Figure 15-4 in Chapter 15, "Tracheobronchial Malacia and Compression"). The field is thoroughly irrigated and the mediastinal pleura is closed loosely. Patients are usually extubated at the conclusion of the operation. If short-term ventilation is necessary, the endotracheal tube cuff pressure should be minimal.

Success in use of posterior surgical splinting procedures is conditional. Patients continue to suffer from their underlying COPD and may have residual obstruction in the more distal bronchi. Secretions may continue to be a problem but can usually be raised by a now more effective cough, since the major airways

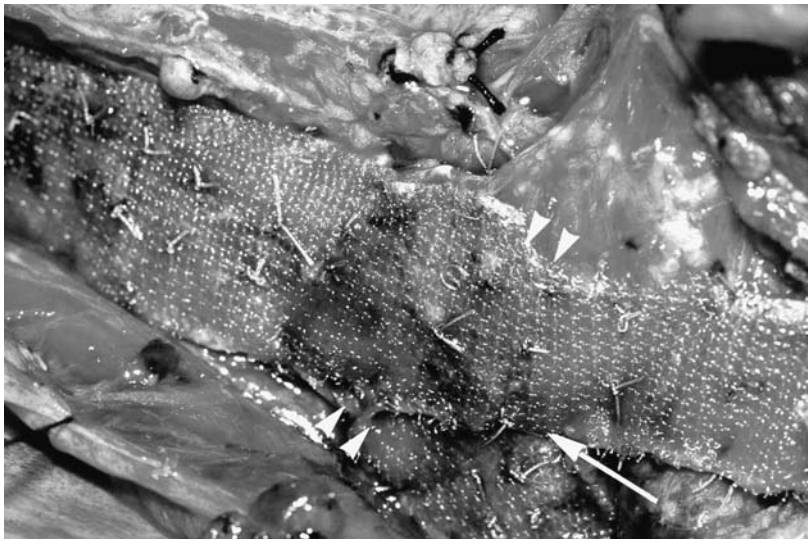

FIGURE 32-2 *Marlex splints in place. The* single arrow *locates the carina. The* double arrows *indicate the origin of the right and left main bronchi.*

no longer collapse with cough. Extensive case series with prolonged follow-up of these procedures are lacking (see Chapter 15, "Tracheobronchial Malacia and Compression").

Although experience with this procedure is small, it appears to be effective for the purposes stated. It is recommended for use after very careful assessment of the patient and with awareness that underlying COPD will not improve.

Alternatively, an individually fashioned Y silicone prosthesis of Dumon type (ie, with "studs" to hold it in place) may be employed. The proximal end must be long enough to splint the relatively long length of collapsing intrathoracic trachea, and the diameters for trachea and bronchi must be adequately large. Tracheobronchial angles vary individually. Some patients do not tolerate such stents and dislodge them with vigorous cough. It seems unwise to deploy expandable stents in these patients, even if large enough sizes were available, because of the hazard of irreversible injury in patients with benign disease (see Chapter 40, "Tracheal and Bronchial Stenting").

Malacia of Other Types

Short segment tracheomalacia, which occasionally results from postintubation injury instead of stenosis, is best treated by segmental resection and primary anastomosis (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). Tracheomalacia in children is discussed in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children."

Rarely, long segment or subtotal tracheomalacia has been encountered with no residual rings identifiable. In a very few patients in this category, the trachea has been approached anteriorly via cervicotomy and complete sternotomy. If the lower trachea cannot be exposed adequately from above behind the brachiocephalic vessels, inferior exposure is made anteriorly by dividing the anterior and posterior pericardium between the superior vena cava and aorta, beneath the brachiocephalic vessels (see Figure 23-7 in Chapter 23, "Surgical Approaches").

Three, four or more separate channels are bluntly dissected circumferentially around the malacic trachea one at a time, taking care to preserve tracheal blood supply between channels and to avoid injuring the trachea. Specially-made perforated polypropylene rings (Figure 32-3), discontinuous at one point, are passed through the channels, sometimes facilitated by a suture passed initially through a distal perforation in the ring (Figure 32-4*A*). The open side of the ring will ultimately lie anteriorly in the tracheal midline. Approximately three 4-0 Tevdek sutures are placed horizontally on each side of the trachea through adjacent perforations in the rings into tracheal tissue, with care to avoid the lumen. Account must be made of the spacing of perforations in the ring in placing these sutures, and also of the length of the ring circumference relative to the final tracheal circumference. Excessive ring length is removed with straight scissors.

FIGURE **32-3** *Polypropylene rings used on rare occasions for splinting of wholly malacic tracheal segments, where resection is not possible. These were originally devised for tracheal replacement with a tube of full-thickness cervical skin and underlying platysma. The perforations are for suture placement. The rings may be shortened as necessary. They are not routinely available.*

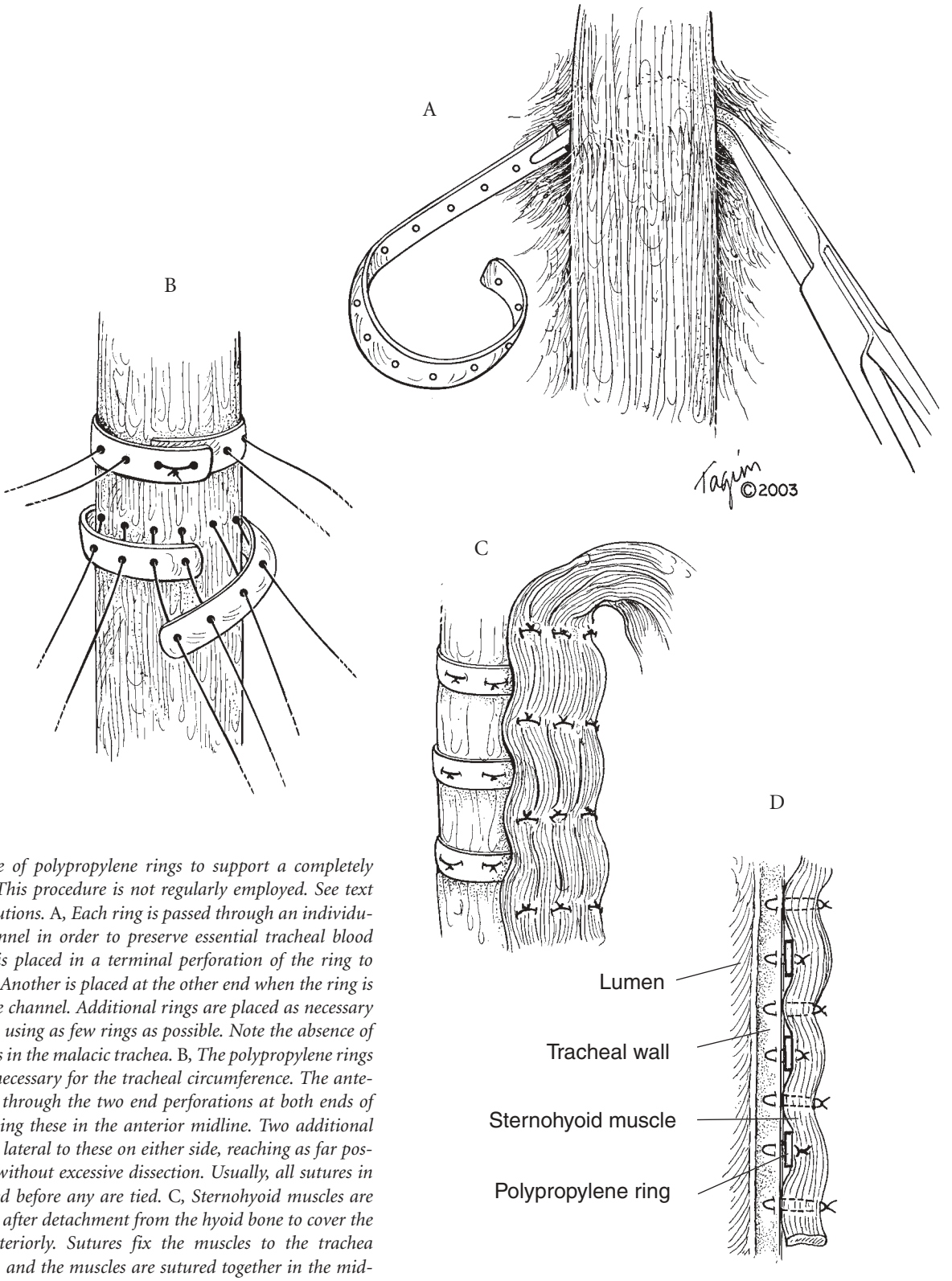

FIGURE **32-4** *Use of polypropylene rings to support a completely malacic trachea. This procedure is not regularly employed. See text for alternative solutions.* A, *Each ring is passed through an individually dissected channel in order to preserve essential tracheal blood supply. A suture is placed in a terminal perforation of the ring to maintain control. Another is placed at the other end when the ring is pulled through the channel. Additional rings are placed as necessary about 2 cm apart, using as few rings as possible. Note the absence of cartilaginous rings in the malacic trachea.* B, *The polypropylene rings are shortened as necessary for the tracheal circumference. The anterior suture passes through the two end perforations at both ends of the ring, overlapping these in the anterior midline. Two additional sutures are placed lateral to these on either side, reaching as far posterior as possible without excessive dissection. Usually, all sutures in all rings are placed before any are tied.* C, *Sternohyoid muscles are pedicled as shown after detachment from the hyoid bone to cover the exposed rings anteriorly. Sutures fix the muscles to the trachea between the rings, and the muscles are sutured together in the midline. This serves to embed the rings for permanent support and also to protect against erosion of the brachiocephalic artery.* D, *Longitudinal section of the tracheal wall showing sternohyoid investment of polypropylene rings.*

The two perforations at each end of a ring will overlap each other anteriorly (Figure 32-4*B*). The initial suture passes through the perforations at both ends of the ring and grasps tracheal tissue in the midline (see Figure 32-4*B*). The overlap of polypropylene rings anteriorly also provides more rigidity. The two or three lateral sutures on each side start as far posteriorly as possible without excessive dissection, which might threaten devascularization.

When all the necessary rings and sutures are placed, the sutures are tied. Bronchoscopy monitors the correction achieved. Both sternohyoid muscles are detached from the hyoid bone and turned down over the reconstruction (Figure 32-4*C*). The muscles easily reach the carina. Fine mattress sutures are passed through the muscle on each side into anterolateral exposed tracheal wall between the polypropylene rings. Finally, the muscles are sutured together in the midline and to the trachea. This thoroughly embeds the plastic rings (Figure 32-4*D*), to prevent later separation of the rings from the tracheal wall, and provides a secure buttress between the foreign material of the rings and the great vessels that lie just anterior to the trachea.

The polypropylene rings were originally designed for cervical cutaneous staged tracheal reconstruction (see below), but were found to be occasionally useful as described. These are not commercially available. The surgeon must avoid circumferential dissection of an *entire* malacic segment prior to ring placement. Complete dissection of a short segment was done in 1 patient many years ago to manage malacia where a long stenosis was also resected. Necrosis resulted, as might well have been predicted.

This procedure has been applied in only a small number of patients, most often with success. A few have been followed for many years without subsequent difficulty. The rings have generally not eroded or required late removal. However, since experience is so limited and the rings described are not regularly available, the operation must be considered as a trial only. It is presented as a possible basis for future development, although indications now are few.

Other open techniques for tracheal stabilization have been reported, usually in single cases, including Marlex wrapping with tissue adhesives and dental arch bars fitted as supports. Free cartilage grafts often fail to provide tracheal rigidity. Even when the grafts survive as adjacent tissue implants, they usually do not bind to the tracheal wall and hence fail in their purpose. T tubes offer an alternative method of treatment with less surgical impact on the patient (see Chapter 39, "Tracheal T Tubes"). Dumon-type silicone stents are another solution but are subject to migration and can incite granulations, especially in the subglottic larynx. Expandable stents should not be used for benign disease since they may lead to obstructive granulations and become irremoveable (see Chapter 40, "Tracheal and Bronchial Stenting").

Tracheopathia Osteoplastica

Tracheopathia osteoplastica (TPO) is rarely encountered, and when it is, is often asymptomatic or only mildly symptomatic (see Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions"). In a few patients, however, obstruction becomes so severe that surgical relief is necessary (see Figures 14-22*A–D*, 14-23*A*,*B* in Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions," and Figures 42 and 43 [Color Plate 16]). In these patients, the entire trachea has been involved by the process, as seems usually to be the case. Bronchoscopic removal of nodules with biopsy forceps or by laser is difficult to accomplish, especially since saber-sheath deformity is often also present. TPO is so rigid that stents or T tubes can not be forced into the undilatable lumen. However, since the process only involves that part of the tracheal wall that contains cartilages, the membranous wall is spared. By incising the anterior wall of trachea from cricoid to carina, the two halves of the rigid anterolateral walls can be hinged apart, stretching the membranous wall to normal width, and in this way, opening the tracheal lumen despite persistent presence of the nodules (Figure 32-5).

Assessment of the diseased airway is necessary both bronchoscopically and radiographically, before corrective surgery. Imaging includes precise anteroposterior tomography, and now may include three-

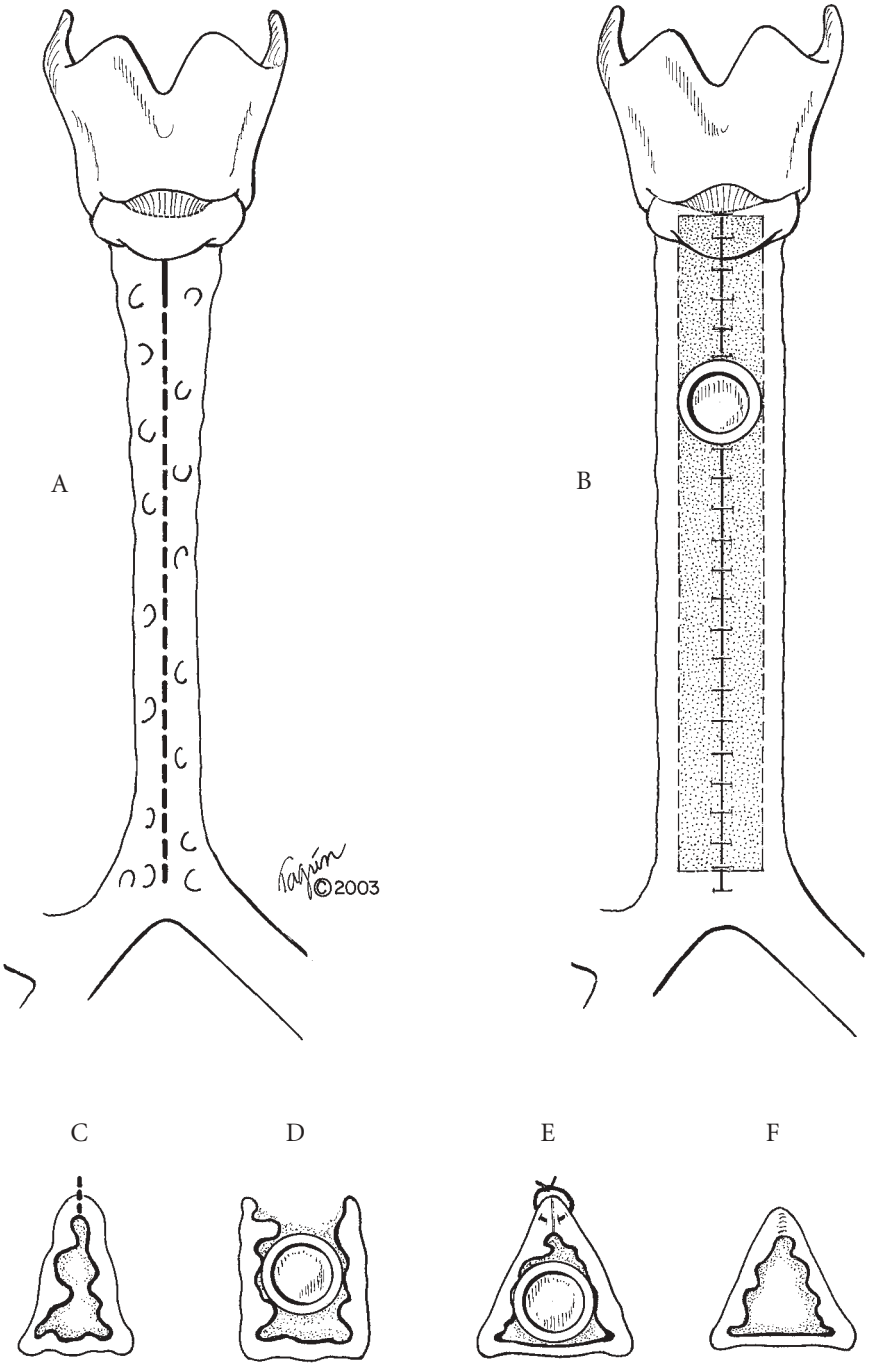

FIGURE 32-5 *Tracheoplasty for obstructive tracheopathia osteoplastica.* A, *The obstructed trachea is opened from just below the cricoid to the carina. A small endotracheal tube may be passed into the left main bronchus at this point. See text for further details about maintenance of ventilation. In a patient with severe main bronchial obstruction, both main bronchi were also opened anteriorly, and a T-Y tube was placed.* B, *A preselected or prepared long T tube is inserted, spanning the entire length of the trachea. In many patients, a T-Y tube is necessary, with limbs extending into the bronchi. This must be planned in advance of operation.* C, *Cross-sectional diagram of a trachea obstructed by tracheopathia osteoplastica. The saber-sheath configuration seems to be common.* Dashed line *indicates the linear tracheotomy. Note the uninvolved but foreshortened membranous wall.* D, *The rigid lateral walls are hinged on the normal, soft membranous wall, to allow insertion of the silicone tube.* E, *The cartilaginous walls are easily resutured anteriorly as the membranous wall is spread.* F, *After firm healing has occurred in this open position, the T tube is extracted. The correction holds for the long term.*

dimensional computed tomography reconstruction. T or T-Y tubes are specially fabricated in advance in accord with precise measurements derived from these examinations (see Chapter 39, "Tracheal T Tubes"). Tubes of at least two diameters (most often 12 and 14 mm) are made, to be certain of a fit at operation. The T sidearm must be at an appropriate height in the trachea and at a proper distance from the carina if a T-Y tube is used. The angles of bronchial take-off must be correct. Proximal and distal ends can be shortened. As described in Chapter 39, "Tracheal T Tubes," these cut ends are carefully sanded smooth.

The long anterior tracheal incision is closed over the tube. After 4 to 6 months, during which firm healing of the wall takes place in its new position, the stent is extracted, leaving a permanently enlarged lumen.

The trachea cannot be dilated with a rigid bronchoscope, so firm is the pathologic process. Subglottic intralaryngeal nodules seem to remain small, allowing a small endotracheal tube to be inserted at least sufficiently to provide a satisfactory initial airway for anesthesia. A laryngeal mask airway may be considered in an extreme case where intubation seems tenuous or impossible.

Approach is through a cervical incision, with dissection high enough to allow later division of sternohyoid muscles at the hyoid bone, and by complete vertical sternotomy. The thyroid isthmus is divided and reflected. The anterior surface of the trachea is dissected to the carina. Access to the lowermost trachea can be made, if necessary, by dividing the anterior and posterior pericardium between the superior vena cava and aorta beneath the brachiocephalic vessels and above the pulmonary artery (see Figure 23-7 in Chapter 23, "Surgical Approaches").

After opening the anterior midline of the trachea (see Figure 32-5*A*), a suitably-sized endotracheal tube is inserted across the operative field, either into the most distal trachea or into the left main bronchus, to continue ventilation. If urgency demanded, two small-bore tubes could be placed, one right and one left, using two anesthesia machines. This seems unlikely. A bifid high-frequency catheter is another option, but care needs to be taken that the nodularity in the bronchi does not hinder escape of gases around the catheters.

A previously prepared or selected extra-long silicone T tube (or more often, a T-Y tube) is trimmed and fitted exactly to the airway, extending from just above the cricoid (but not impinging on the conus elasticus below the glottis), either to the lowermost trachea or into main bronchi if there is serious bronchial involvement (see Figure 32-5*B*). Bronchial disease is common but is not uniformly present or necessarily severe.

Ventilation is continued by tucking the tip of a properly sized endotracheal tube into the proximal end of the T tube in the subglottic larynx and capping the sidearm. Alternatively, ventilation is carried out across the operative field via the T sidearm, using an endotracheal adapter and a light tracheostomy swivel connector. The proximal vertical arm of the T tube above the sidearm in this case is occluded with a small inflatable balloon with a small access catheter (eg, Pruitt catheter), which emerges through the T tube sidearm beside the endotracheal tube adapter. A Fogarty catheter can also be inserted translaryngoscopically to occlude the proximal T tube.

The tracheal wall is so rigid that a small amount of tissue usually must be excised to allow space for emergence of the T tube sidearm. This tissue provides a specimen for histologic examination. In one patient, a few large nodules were removed from the opened trachea with a pituitary rongeur. The long linear tracheotomy is easily approximated with interrupted 4-0 Vicryl sutures, as the lateral walls hinge outward like a book's covers (see Figures 32-5*C–E*).

A sternohyoid muscle pedicle, inferiorly based, is turned caudad and sutured over the tracheal incision inferior to the T tube sidearm (see Figure 32-4*C*). Both sternohyoid muscles may be required for this length of incision. The sternothyroid muscles may be approximated to cover the upper tracheal incision superior to the T tube sidearm.

The splinting T or T-Y tube is left in place for 4 to 6 months, with usual T tube care. The silicone rubber tube is then extracted under general anesthesia, with a ventilating rigid bronchoscope positioned through the vocal cords just above the tube. The airway is carefully inspected with a Hopkins telescope. I prefer to leave a capped stomal cannula (Montgomery) in place to maintain a tracheostomy channel until it is certain that the patient continues to enjoy a good airway. After about a month, during which the patient has been able to tolerate the cannula capped, it is removed and the stoma allowed to heal. Periodic bronchoscopic follow-up is advised, at least for a number of years. Patients with TPO produce quite an amount of mucoid secretions, which continues after airway obstruction is relieved. They are better able to clear secretions, however, because effective cough is possible. Only 4 patients have been seen with sufficiently severe

obstruction to require reconstruction. In 3 patients, the procedure was successful. In 2 patients, follow-up has been over a period of many years (see Figures 14-22*E*, 14-23*C* in Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions") and over 3 years in the third patient. The fourth patient, who had difficulty after extraction of the T tube, disappeared from follow-up. Although experience is limited, I believe that the operation can be recommended.

We encountered TPO in the main bronchi in 1 patient, severe enough to require bronchoplasty as well as tracheoplasty. Both main bronchi were incised and a previously prepared T-Y tube was inserted. Access to the entire left main bronchus was obtained by opening the pericardium anteriorly and then posteriorly lateral to the aorta, after division of the ligamentum arteriosum. The recurrent laryngeal nerve was carefully preserved. No difficulty was encountered in suturing the bronchi closed over a T-Y tube. Thymic lobes were used to cover bronchial suture lines. In a very rare case of tracheal TPO with only segmental tracheal involvement, limited tracheal resection and reconstruction has been done. References to stented tracheoplasty and to segmental resection for TPO are noted in Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions." Modified lateral slide tracheoplasty has also been employed in an instance of segmental narrowing, but T tube tracheoplasty as I have described seems a simpler solution.[1]

Postpneumonectomy Syndrome

The keys to successful surgical treatment of postpneumonectomy syndrome are 1) restoration of the mediastinum to a normal central position; 2) implantation of filler to prevent recurrence of mediastinal displacement; and 3) correction of severe residual malacia, when present. Herniated lung is also reduced (see Chapter 15, "Tracheobronchial Malacia and Compression," and Figure 40 [Color Plate 16]).

The prior thoracotomy incision is carefully reopened since there may be essentially no residual pleural space, and postsurgical scar is present in variable intensity. The pericardium and heart may lie just beneath the chest wall. Dissection is not usually very difficult and it is pursued until the heart and mediastinum can be gently moved to a normal midline position, and until the herniated lung is sufficiently free to be returned to the opposite hemithorax. Intraoperative flexible bronchoscopy through the endotracheal tube will assess the correction attained at this point.

Although it may not be essential, I prefer to abrade the endothoracic fascia behind the sternum anterior to the pericardium and also the opposing pericardium. Two vertical rows of 0 Prolene sutures are placed to fix the pericardium to the retrosternum, replacing the mediastinum to a central position. Blood pressure, pulse rate, and pulse pressure are monitored during this part of the operation, since reefing up too much pericardium can quite easily produce tamponade. The herniated upper lung is replaced across the midline, and upper mediastinal tissues are sutured together to contain the herniation. These anatomic corrections will not, however, be maintained unless the hemithorax is filled. The volume of the reconstituted postpneumonectomy pleural cavity is measured with breast prosthesis "sizers." One or more breast prostheses are filled with the appropriate total volume of sterile saline and inserted into the space (see Figures 15-19*A–D* in Chapter 15, "Tracheobronchial Malacia and Compression"). Bronchoscopic monitoring is repeated at this point.

The costal and intercostal chest wall is now reapproximated and the patient once again observed for signs of cardiac tamponade. If there is evidence of such, the volume of saline is reduced in decrements by aspiration, until physiologic stability is attained. The airway is rebronchoscoped. If the result is satisfactory both anatomically and physiologically, the incision is completely closed and the patient is placed supine. Cardiac physiology and tracheobronchial anatomy are rechecked finally in this position. On at least one occasion, it was necessary to reopen the hemithorax to reduce filler volume further. Use of a tissue expander with a subcutaneous port could simplify volume adjustment and also offers advantages in a child, where volume can be adjusted as the child grows. However, tissue expanders are not meant for permanent implantation, whereas breast prostheses are. We therefore have preferred to use the latter in our adult practice.

Long-term results over many years have been very satisfactory. Multiple reports of the use of similar surgical techniques confirm its effectiveness.

In a few earlier cases, with symptoms of long duration, severe segmental tracheomalacia continued to produce obstruction despite mediastinal repositioning. These patients were managed variously by tracheal resection and reconstruction or by relief of vascular compression by aortic grafting and division. The problem of residual malacia has not been encountered since, however, and repositioning with prosthetic filler alone in numerous additional patients has proved successful. The alternative of primary insertion of stents has been offered, but raises concern about long-term pressure against the aorta and pulmonary artery by any type of stent, if the basic anatomic deformity remains uncorrected. If residual obstructive malacia were to present again *after* successful repositioning, silicone stent placement might indeed provide a simpler solution to stabilize the malacic segment. The long-term hazards of expandable stents for benign disease must be remembered (see Chapter 40, "Trachial and Bronchial Stenting").

Straight Back Syndrome

The components of this rare syndrome are described in Chapter 15, "Tracheobronchial Malacia and Compression." The principal factor affecting the trachea is the sharply-reduced distance between the abnormally vertical dorsal vertebral column and the sternum. The posterior superior infolding of the top of the manubrium compresses the trachea at the thoracic inlet (see Figure 15-22*A* in Chapter 15, "Tracheobronchial Malacia and Compression"). The degree of pectus excavatum deformity usually present in these patients can cause a second point of compression where the brachiocephalic artery crosses the trachea (Figure 32-6*A*).

The very few patients reported with such problems have been managed variously. In one, conventional pectus excavatum repair with double sternal osteotomies and an Adkins bar to secure the repair produced relief. In an extremely severe case with both points of obstruction, relief was achieved as follows. The upper third of the manubrium was removed to eliminate the "bar" of bone that projects posteriorly at the top of the manubrium. The balance of the manubrium was excised, split into anterior and posterior layers, the posterior plate removed, and the thinned anterior plate then replaced (see Figures 32-6*A*,*B*). The obstructing brachiocephalic artery was lengthened with a graft and reimplanted in the proximal aorta to the right of the trachea (Figures 32-6*C*,*D*). In this patient, a third area of lower tracheal obstruction, due to splaying of the trachea over the vertebral column, was corrected by posterior membranous wall splinting with a Marlex strip, as described in this chapter for correction of acquired tracheomalacia with COPD, after removal of bony prominences by shallow osteotomy. This procedure was performed through additional right thoracotomy at the same operative session. Another patient with only tracheal compression by upper sternum was treated elsewhere by sternotomy and insertion of a wedge of methylmethacrylate (P. Dartevelle, personal communication, 1996).

Obviously, since individual pathology varies so much, no standard surgical procedure is applicable.

"Hairpin" Aorta

Division of the more often-identified vascular rings that cause airway obstruction has been described elsewhere, often and in detail. These anomalies are listed in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children." Also noted there is the less-common occurrence of "circumflex aorta." Brief technical note will be made here, however, of the highly unusual compression, described in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children," which is due to right aortic arch, right descending aorta, ligamentum arteriosum, Kommerell's diverticulum, and aberrant left subclavian artery (Figure 32-7A). The aortic arch is usually high, and bends sharply before descending in a "hairpin" configuration on the right side, directly behind the ascending arch. The distance between the sternum and vertebral column is diminished, sometimes with mild pectus excavatum deformity. This leaves little space between the ascending and

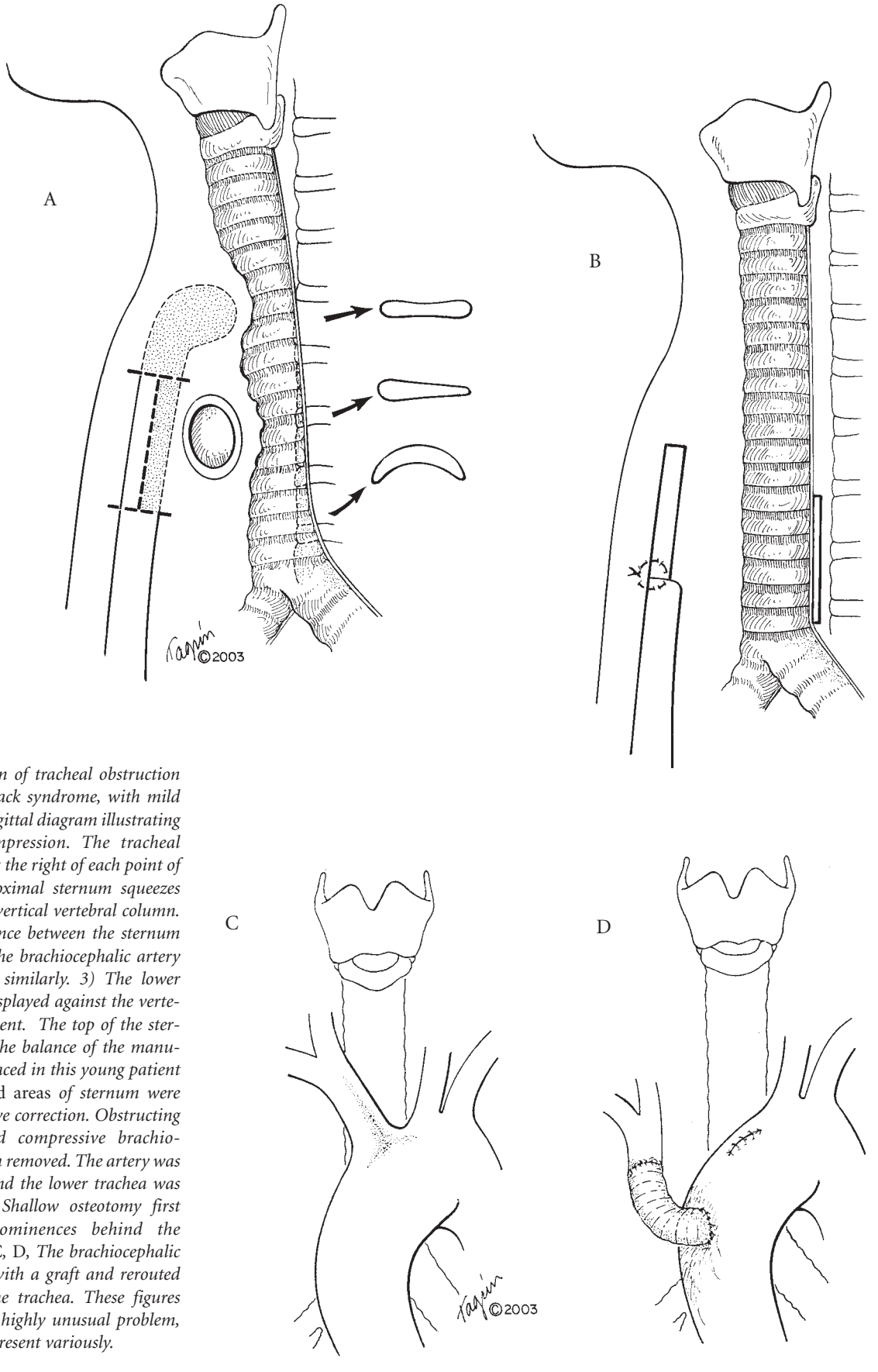

FIGURE **32-6** *Correction of tracheal obstruction due to severe straight back syndrome, with mild pectus excavatum.* A, *Sagittal diagram illustrating points of tracheal compression. The tracheal lumen is diagrammed at the right of each point of obstruction. 1) The proximal sternum squeezes the trachea against the vertical vertebral column. Note the minimal distance between the sternum and the vertebrae. 2) The brachiocephalic artery compresses the trachea similarly. 3) The lower intrathoracic trachea is splayed against the vertebral column in this patient. The top of the sternum was resected and the balance of the manubrium thinned and replaced in this young patient* (dashed lines). Stippled areas *of sternum were removed.* B, *Postoperative correction. Obstructing proximal sternum and compressive brachiocephalic artery have been removed. The artery was transplanted laterally and the lower trachea was splinted with Marlex. Shallow osteotomy first removed vertebral prominences behind the splayed lower trachea.* C, D, *The brachiocephalic artery was lengthened with a graft and rerouted lower and lateral to the trachea. These figures describe correction of a highly unusual problem, which apparently may present variously.*

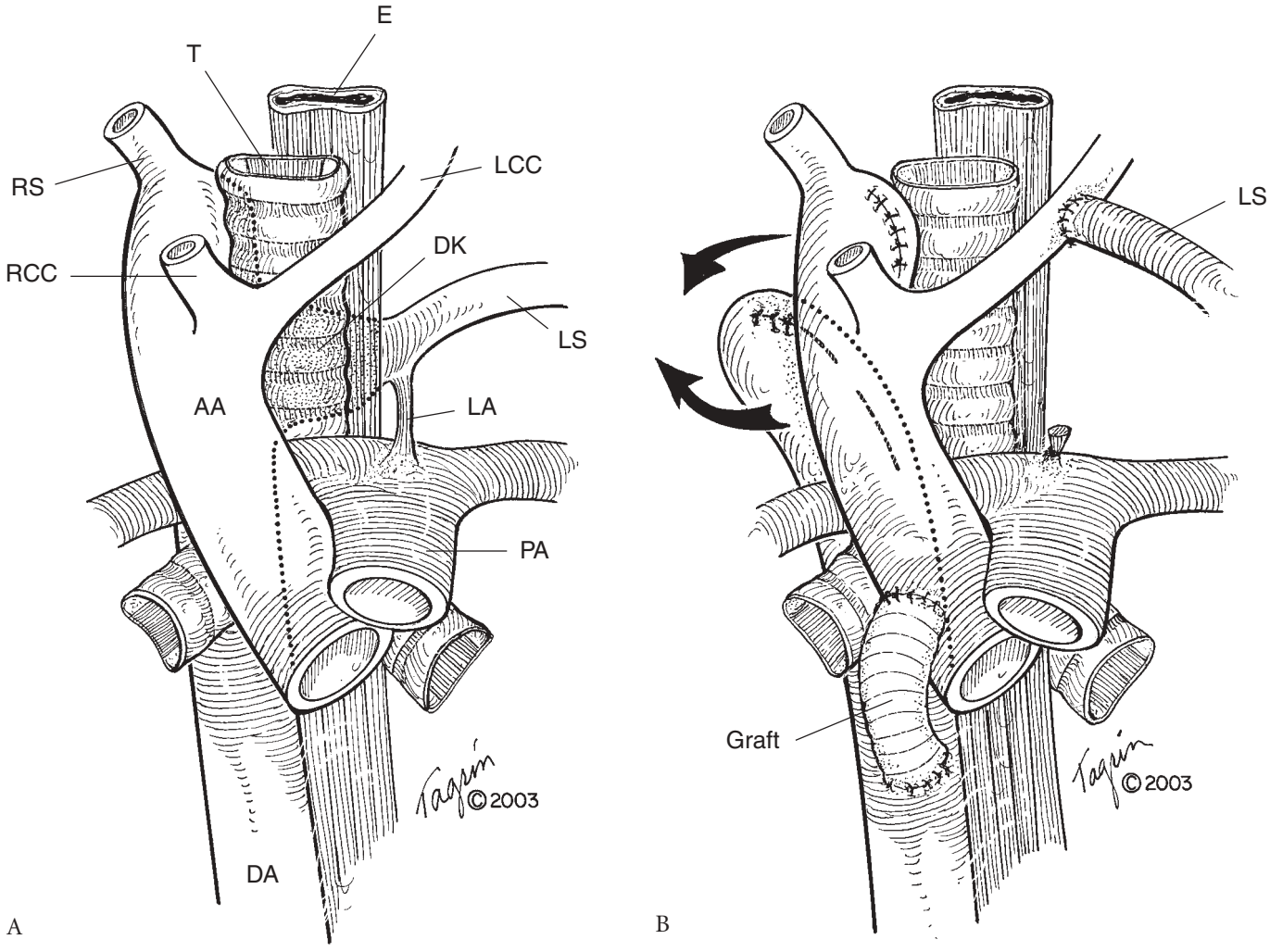

FIGURE **32-7** *Correction of obstruction from an unusual vascular ring, termed "hairpin" aorta, seen in patients with diminished distance between the sternum and vertebrae.* A, *The ascending aorta (AA) is on the right and also descends (DA) on the right, leaving only a narrow space underneath the arch between the ascending and descending limbs. Left subclavian (LS) artery is anomalous, arising from a Kommerell's diverticulum (DK) and encircles trachea (T) and esophagus (E). Ligamentum arteriosum (LA) completes the ring to the pulmonary artery (PA). Right (RCC) and left (LCC) carotid arteries branch anteriorly and the right subclavian (RS) arises from the arch. Our patients variously had undergone division of LA, LS, and excision of DK without relief of tracheal obstruction.* B, *Correction required removal of a Kommerell's diverticulum, division of the ligamentum arteriosum and left subclavian (LS), and reimplantation of the LS into the left carotid artery. If aortopexy (of one or both limbs of the arch) gave insufficient relief (observed bronchoscopically), aorto–aortic graft was performed below the hilum and the aorta divided, as shown. The limbs were pexed to upper ribs anteriorly and posteriorly.*

descending aorta. The airway is compressed in this vise, with the contributing factor of Kommerell's diverticulum and an anomalous left subclavian artery.

A small number of patients have been seen with this problem and treated. Prior division of the ligamentum, division of the anomalous subclavian artery, and excision of the diverticulum in several patients had failed to relieve the severely symptomatic tracheal compression. Variations in the small group of patients seen makes it difficult to recommend a categorical procedure. However, opening the narrow space between the aortic limbs has proved effective, when excision of the aortic diverticulum, reimplantation of the retroesophageal subclavian artery, and division of the ligamentum arteriosum failed to provide relief of the airway compression.

The anatomy must be carefully delineated by imaging (see Figure 6-15 in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children") and account is taken of any prior surgical treatment. Through right thoracotomy, the aberrant left subclavian artery is divided and tentatively closed. The Kommerell's diverticulum is excised and the ligamentum is divided. Then, the degree of airway relief obtained is observed by flexible bronchoscopy via the endotracheal tube. The aortic arch is next tentatively retracted with slings to determine (by bronchoscopy) whether aortopexy alone will be sufficient to relieve the obstruction completely. If not, a synthetic graft is placed between the ascending and descending aorta, beneath the right hilum. The aortic arch is divided at its apex, and each limb is pexed as necessary to an adjacent upper rib anteriorly and posteriorly (Figure 32-7*B*). If retracting the aorta alone is sufficient, the arch is pexed with slings to maintain the space so enlarged. Broad slings of Goretex are used to avoid a potential cutting effect on the aorta. It is important to assess the effect on airway compression bronchoscopically as each step is completed. The anomalous subclavian artery is finally anastomosed to the left carotid artery through a cervical incision (see Figure 32-7*B*).

Cutaneous Tube Reconstruction of the Trachea

Prior to recognizing the extent of tracheal resection, which is made feasible by cervical flexion, anatomic mobilization, and other maneuvers, a "trough" method for staged reconstruction of the cervical trachea was developed, using cervical skin and underlying platysma. The specific technique described was just one of several such procedures described by various surgeons. Patency was assured here by implantation of several polypropylene rings (see Figure 32-3) in laddered fashion between the dermis and the platysma, carefully avoiding epithelial communication and its consequences of probable infection.[2,3] The technique offered the possibility of transferring a segment of cervical trachea into the mediastinum, with its lateral blood supply intact, in order to effect safe intrathoracic anastomosis where complete anatomic tracheal reconstruction seemed to be impossible. The gap in the cervical trachea was then to be filled with the cutaneous conduit in a later procedure. The procedure is detailed here largely for historic reasons. It must *not* be considered as a substitute for the now-standard reconstructive techniques described in earlier chapters.

At initial operation (Figure 32-8), a very low cervical collar incision is made, which later becomes the inferior margin of a horizontal bipedicled full-thickness flap of lower cervical skin and platysma that will ultimately form the cutaneous conduit (see Figure 32-8*A*). The tracheal resection is performed through this incision, if necessary with a vertical inferior extension over part or all of the sternum. The thyroid isthmus is divided and the thyroid dissected free from the trachea. The cervical tracheal segment is divided between first and second cartilaginous rings and is dissected anteriorly and posteriorly, carefully sparing the blood supply pedicles on both sides. The recurrent laryngeal nerves are very carefully preserved.

The precise location of the initial incision must be selected with consideration of the parallel incision to be made later at the lower edge of the beard line, to form the upper margin of the skin flap (see Figure 32-8*A*).

A bipedicled flap is elevated beneath the platysma, long enough to provide slack for infolding the flap into a trough to form the tracheal replacement (see Figures 32-8*B*,*C*). Blood supply arising from both lateral bases of the flap is adequate. Through short carefully placed incisions, a series of perforated polypropylene rings is introduced between the undersurface of the dermis and the platysma, using specially-made needles (Figure 32-9) that emerge through other short incisions at a point equidistant from the midline of the neck from the line of incisions made for introduction of the needles (see Figure 32-8*B*). The rings are drawn through the flap until a suture fixing the ring to the hub of the needle is seen and cut. Long sutures are attached to the last perforation of the free ends of the rings, and these are used to adjust the rings precisely in their subcutaneous location. If there is excess length of plastic ring, it is cut off with heavy, straight scissors and the corners trimmed. The mini-incisions for ring introduction on either side are closed with

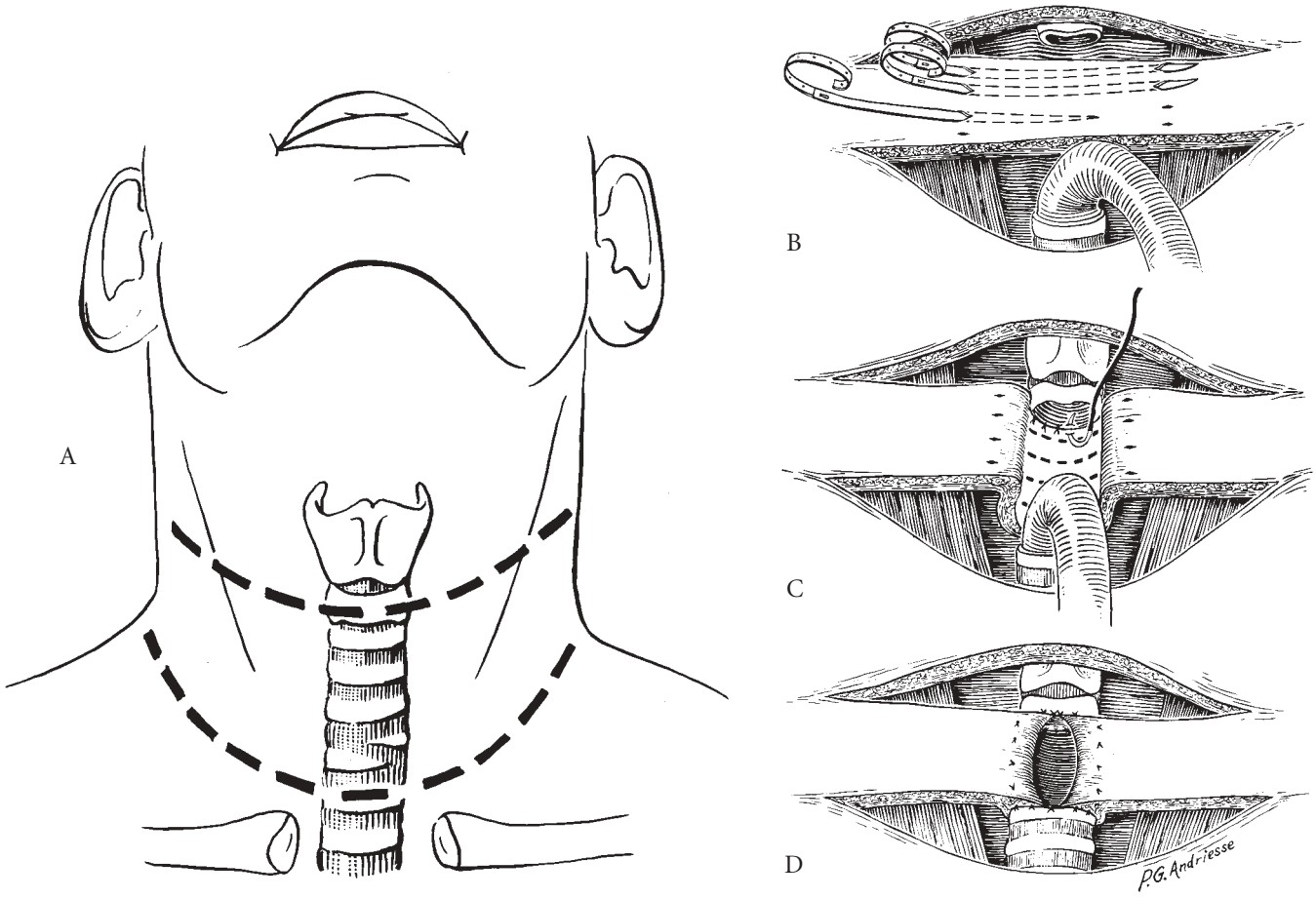

FIGURE **32-8** *Staged reconstruction of the cervical trachea by construction of a cutaneous-platysmal tube.* A, *Two long horizontal parallel cervical incisions demarcate the vertical length of the tracheal replacement. In a male, the upper incision is placed just below the beard line. A bipedicled flap of skin with underlying platysma is elevated, with blood supply at both ends. The lower incision is the primary incision for cervical tracheal exploration, mobilization, resection, and devolvement, as is necessary.* B, *Perforated polypropylene rings of open circular form (see Figure 32-3) are introduced in separate channels between the undersurface of dermis and the platysma, using specially-made needles, and introduced and exited through lines of cutaneous nicks (as shown) far enough apart to provide tissue for a major part of the trough's circumference. The needle entry and exit points are sutured.* C, *Anastomosis is made to a residual tracheal ring proximally, or to the cricoid, using individual 4-0 Vicryl sutures, and to the trachea distally.* D, *In the midline superiorly and inferiorly, the infolded skin flap may not easily meet and the borders of the superior and inferior horizontal incisional edges may serve to complete the closure of the tracheal tissues. The horizontal incisions are closed lateral to the completed trough. Reproduced with permission from Grillo HC.*[2]

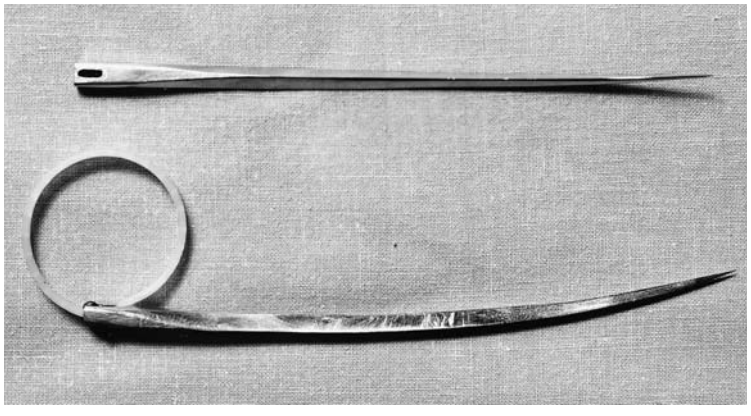

FIGURE **32-9** *Special needles for subdermal introduction of polypropylene rings. An end of a ring is transiently sutured into the hub of the needle.*

sutures, burying the rings completely in mesenchymal tissue. If the rings are left too long, they may erode to the skin surfaces, allowing infection to occur and forcing removal of the infected ring.

The inserted rings serve to shape the trough as part of a circular tube (see Figure 32-8*C*). Anastomosis is performed with 4-0 Vicryl between the upper and lower margins of the flap forming the cutaneous trough, to the remaining ring of trachea superiorly and to the tracheal end presenting inferiorly (see Figure 32-8*C*). Anteriorly, some of the tracheal margin may be sutured to the cutaneous margins of the flaps above and below, completing circumferential closure (Figure 32-8*D*).

During a healing interval, any hairs noted in the central portion of the bipedicled flap of skin are depilated by electrolysis. This is necessary in some male patients. Approximately 2 months are allowed for firm healing and acquisition of parasitic blood supply by the tissues of the trough.

At a second stage, an incision is circumscribed about the stoma that has been created, placed just far enough from the aperture to provide sufficient skin and platysma to infold for precise closure of the tube (Figure 32-10*A*). The platysma is elevated with the skin (Figure 32-10*B*). Only enough dissection of this

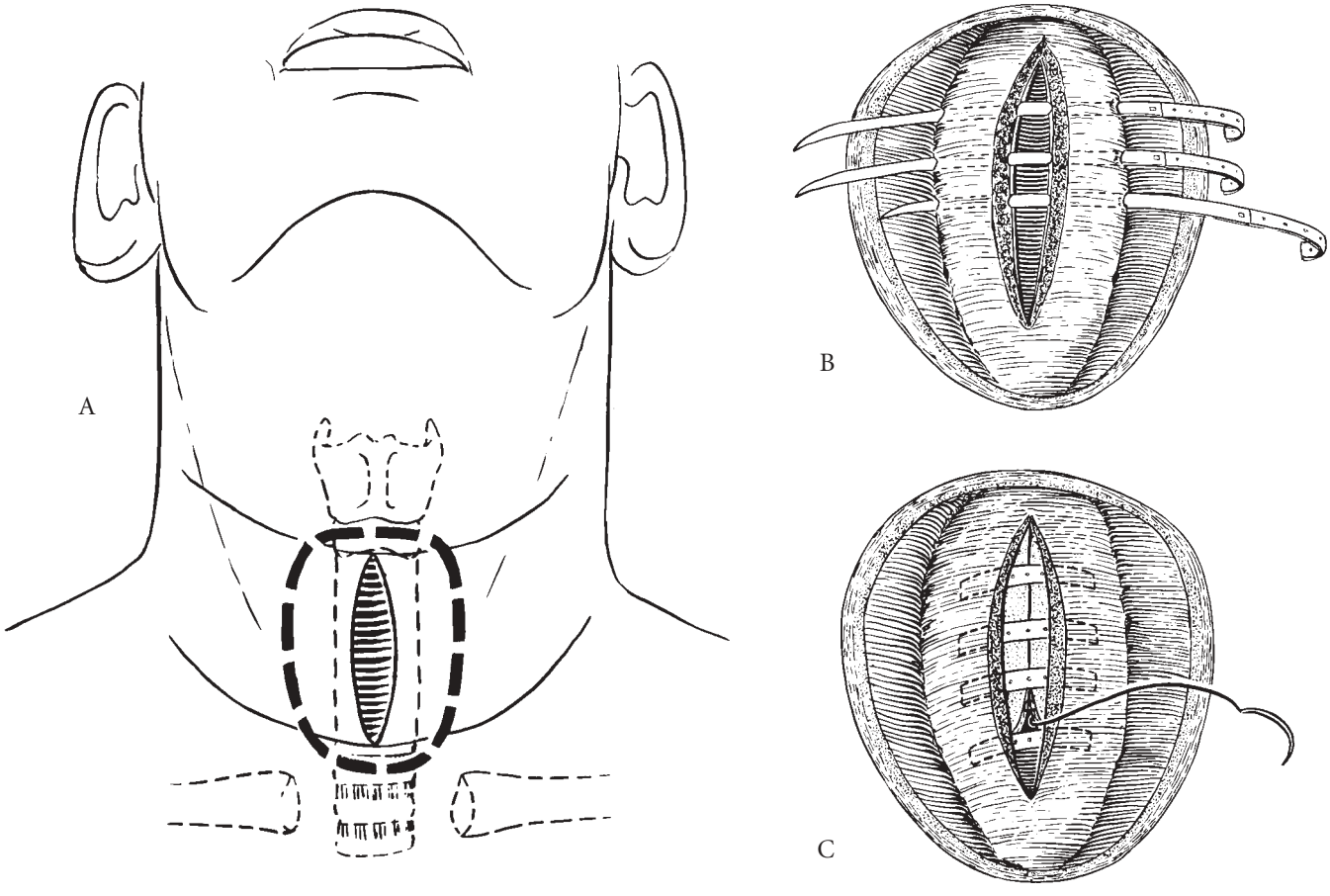

FIGURE **32-10** *Second-stage completion of cutaneous tracheal replacement. This is performed several months after complete healing to allow time for the trough to acquire a posterior blood supply.* A, *The vertical stoma is circumscribed as shown (*dashed line*), more generously laterally to provide tissue for construction of the anterior wall of the tracheal reconstruction.* B, *Placement of additional segments of polypropylene rings anteriorly may be necessary, since a full-length ring can erode the skin by pressure against the anterior skin tube margins in the interval between the stages. These additional segments are also placed between the dermis and platysma. In turning these flaps, care is taken not to risk losing the posterior parasitic blood supply acquired after the first stage.* C, *The inverted skin incision is closed with a subcuticular absorbable suture. The platysma is next closed over the prosthetic rings. Reproduced with permission from Grillo HC.*[2]

central circular flap necessary to allow tension-free closure is done, in order to preserve the secondary blood supply of the final cutaneous flap, which enters posteriorly only. The tube is closed to itself with a fine subcuticular running suture (Figure 32-10*C*).

The tips of the plastic rings may sometimes be sutured together in the midline. More often, short additional segments of polypropylene rings must be added, between the undersurface of the dermis and platysma, after elevating the flap circumferentially (see Figures 32-10*B*,*C*). On occasion, sufficient stability is evident without either joining the previously placed rings or adding segments anteriorly.

The inverted skin edge is closed with a fine running subcuticular absorbable suture (see Figure 32-10*C*). The platysma of the flap is next closed to itself over added rings with interrupted fine sutures (Figure 32-11*A*).

With cutaneous undermining and use of relaxing incisions laterally on both sides of the neck, the skin and platysma on either side of the incisional defect can be closed in layers in the midline. The gaps that present where relaxing incisions were made are grafted with split-thickness skin grafts, well away from the area of tracheal reconstruction (Figure 32-11*B*). The "surgical embryology" of the replacement segment is diagrammed in Figure 32-12.

Patients clear sputum through a cutaneous tracheal reconstruction with vigorous cough. The absence of cilia does not seem to be a major problem. A patient with procedure completed is shown in Figure 32-13.

Relatively few of these procedures were done and the operation described is largely of historic interest. The reasons for infrequent use are 1) mobilization techniques now available for tracheal resection rarely require such added length; 2) the complexity of reconstruction and the multiple stages needed caused excessive complications, delays, and led to failure to complete the tubes in some patients; 3) for benign

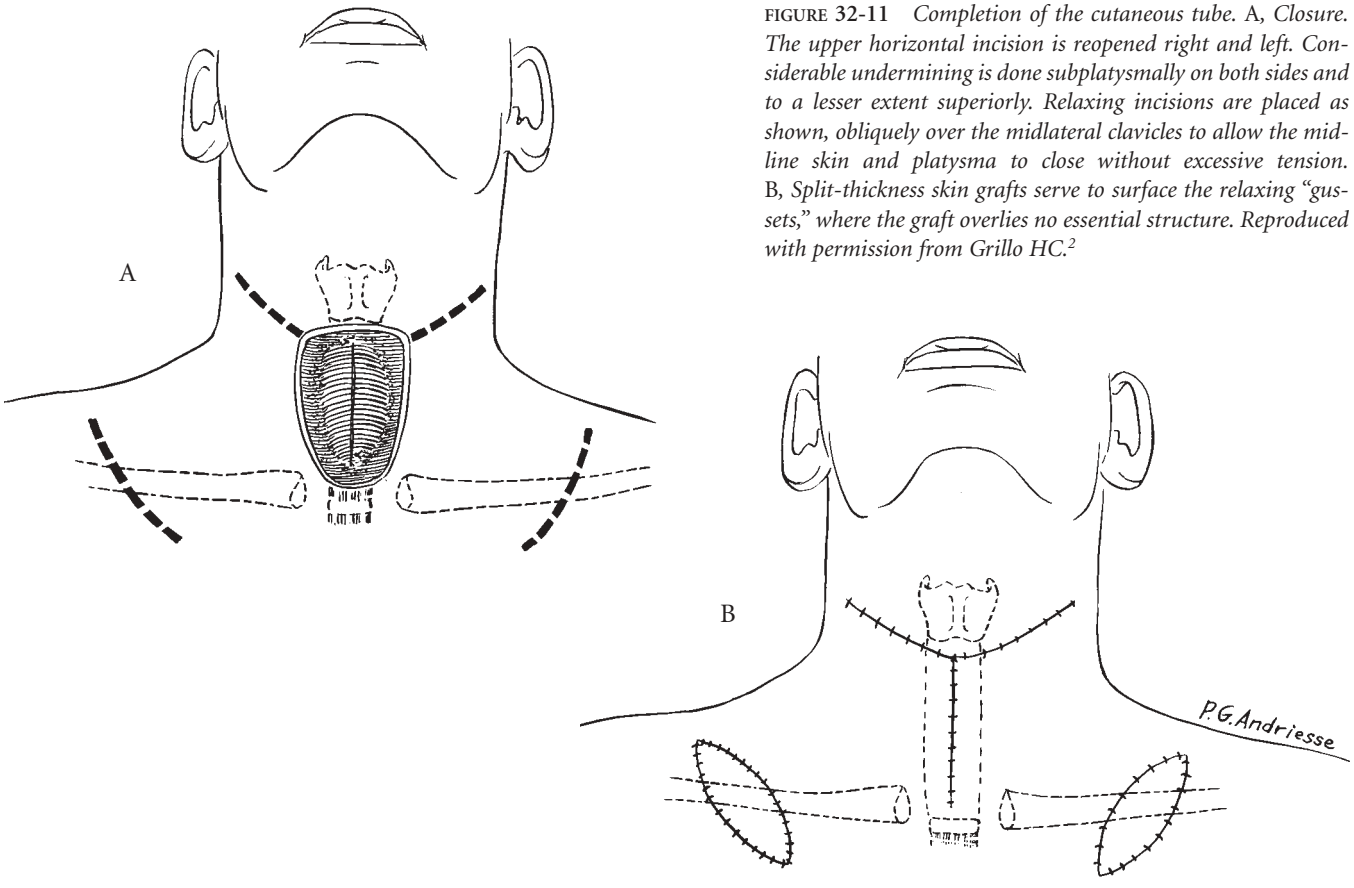

FIGURE **32-11** *Completion of the cutaneous tube.* A, *Closure. The upper horizontal incision is reopened right and left. Considerable undermining is done subplatysmally on both sides and to a lesser extent superiorly. Relaxing incisions are placed as shown, obliquely over the midlateral clavicles to allow the midline skin and platysma to close without excessive tension.* B, *Split-thickness skin grafts serve to surface the relaxing "gussets," where the graft overlies no essential structure. Reproduced with permission from Grillo HC.*[2]

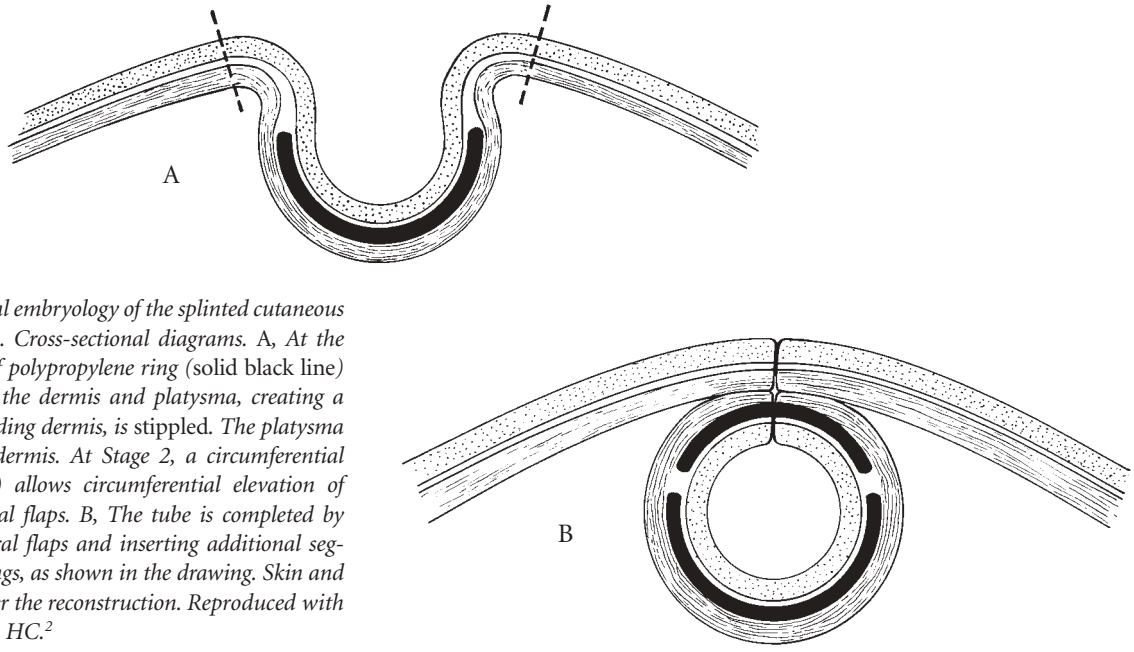

FIGURE **32-12** *Surgical embryology of the splinted cutaneous tracheal reconstruction. Cross-sectional diagrams.* A, *At the first stage, a segment of polypropylene ring (*solid black line*) is introduced between the dermis and platysma, creating a trough. The skin, including dermis, is* stippled. *The platysma (*lined*) underlies the dermis. At Stage 2, a circumferential incision (*dashed line*) allows circumferential elevation of the cutaneous platysmal flaps. B, The tube is completed by turning the anterolateral flaps and inserting additional segments of supporting rings, as shown in the drawing. Skin and platysma are closed over the reconstruction. Reproduced with permission from Grillo HC.*[2]

disease, a T tube is a simpler, safer, yet generally satisfactory solution for managing a nonreconstructible trachea; and 4) huge tumors often also involve the larynx, and reconstruction is pointless if a functional larynx is not salvageable. In such cases, mediastinal tracheostomy may be in order.

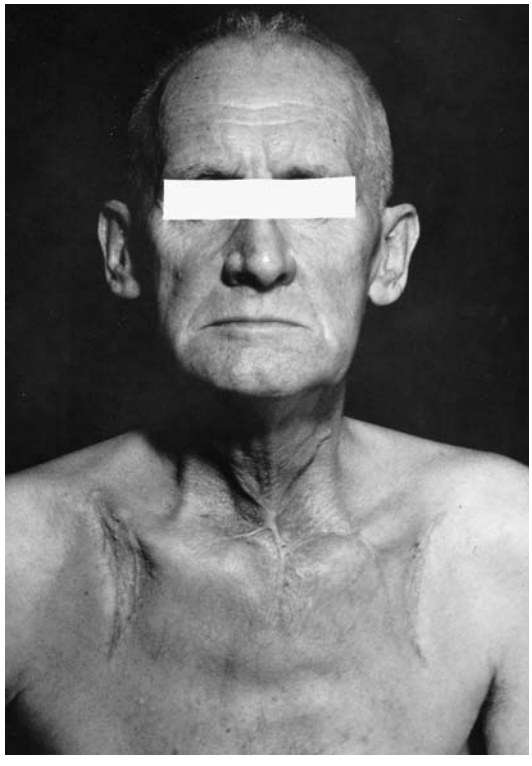

FIGURE **32-13** *A completed cutaneous tube reconstruction in a patient who had extensive obstruction and invasion of the trachea by recurrent papillary carcinoma of the thyroid.*

Although the operation is not advised, for the reasons given, and has largely been abandoned, it found use in a very few patients to provide continuity where this was demanded and seemed appropriate, and where no other reconstructive possibility was dependably available. In my longest follow-up, a cutaneous tube of this construction functioned well for 30 years. Its original indication was to restore a trachea following catastrophic failure of inauspicious tracheal resection for a supracarinal granular cell tumor performed elsewhere. Invasive papillary thyroid carcinoma required resection of the tube after 30 years. Tissue remodelling then permitted successful primary anastomosis.

References

1. Kutlu CA, Yeginsu A, Ozalp T, Baran R. Modified slide tracheoplasty for the management of tracheobronchopathia osteochrondroplastica. Eur J Cardio-Thorac Surg 2002;21:140–2.
2. Grillo HC. Circumferential resection and reconstruction of mediastinal and cervical trachea. Ann Surg 1965; 162:374–88.
3. Grillo HC, Dignan EF, Miura T. Experimental reconstruction of cervical trachea after circumferential excision. Surg Gynecol Obstet 1966;122:733–8.

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Repair of Congenital Tracheal Lesions

33A Tracheoplasty for Congenital Tracheal Stenosis

Hermes C. Grillo, MD

Congenital stenosis involving long segments of the trachea has presented particular difficulties for reconstruction. Congenital stenosis varies in location, severity, and length (see Chapter 6, "Congenital and Acquired Tracheal Lesions in Children"). The stenotic segment is usually characterized by completely circular "O" rings of cartilage. Dilation is therefore of no value and incurs the distinct possibility of splitting the trachea if force is used. When the stenosis is of limited extent, it may be resected and meticulous end to end reconstruction performed. When the extent is too great to permit safe anastomotic reconstruction other techniques have been applied. No single group has accumulated a large experience, particularly with prolonged follow-up. Two techniques—*patch tracheoplasty* and *slide tracheoplasty*—have been used successfully for long stenoses.

Resection and Reconstruction

The length of resection that allows reconstruction by standard anastomotic techniques is limited.[1,2] Experimental and clinical experience indicates that the limits of safe resection in children are lower than in adults (see Chapter 6, "Congenital and Acquired Tracheal Lesions in Children"). Anastomotic tension appears to be tolerated less well in the small child or infant.[3] Circumferential growth, on the other hand, has been shown to occur both experimentally and clinically. Anastomotic technique is the same as in adults except for the use of 5-0 Vicryl sutures in small children and 6-0 in infants (Figure 33-1).

Anterior Patch Tracheoplasty

The initial solution to the problem of long congenital tracheal stenosis, devised by Kimura and colleagues, was vertical incision of the entire length of the stenotic segment with insertion of a patch or "gusset" to widen the lumen.[4,5] Costal cartilage or pericardium are principally used for the patch.[4–8]

Proponents of these techniques favor use of cardiopulmonary bypass, although the procedure could be performed without bypass in the absence of associated vascular anomalies.[6–8] Approach is via complete

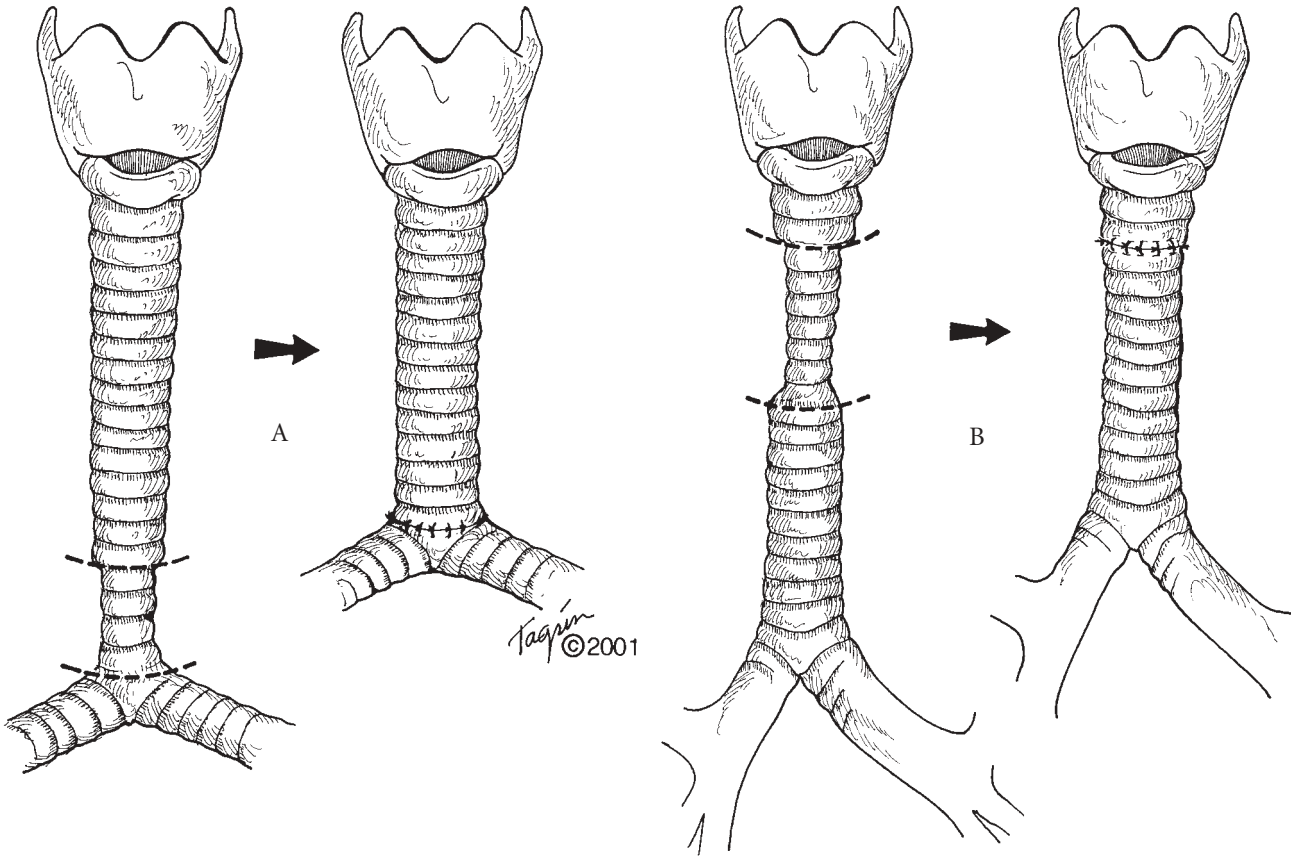


FIGURE **33-1** *Examples of congenital tracheal stenosis short enough to be treated by resection and reconstruction.* A, *Three-month-old boy with a stenotic segment less than ¼ of the length of his trachea. In addition, there was malacia present, which was best treated by resection.* B, *Followed from infancy with a noncritical stenosis, resection was done at the age of 23 years to improve exercise performance. With over 30% of his trachea involved, slide tracheoplasty would now be elected in infancy, but this length of resection was easily tolerated in a young adult. Reprinted with permission from Grillo HC et al.*[12]

median sternotomy. The thymic lobes are separated, the pericardium is opened, and the aortic arch retracted, permitting exposure of the lower trachea and carina (Figure 33-2). The entire trachea is visualized anteriorly. If partial cardiopulmonary bypass is to be used, cannulas are then placed in the atrium and aorta. If there is difficulty in identifying the proximal end of the stenosis, the patient is bronchoscoped and the point marked with a suture.

An anterior midline vertical incision is made in the trachea through the stenosis from its upper end to its lowermost point. This may be determined by direct inspection, as the incision is carried inferiorly. If pericardium is used for repair, a rectangular patch is outlined and excised from the readily available anterior pericardium.[6,7] It is trimmed and sutured into the tracheal defect. Interrupted 5-0 Dexon sutures or continuous running 6-0 polydioxanone suture, which do not penetrate tracheal mucosa, have been recommended.[6,7] Several partial thickness sutures are used to suspend a pericardial patch to mediastinal structures. Added sutures are placed to suspend the tracheal margins similarly. The innominate artery is suspended to the sternum in closing. I believe that the patch could be safely sutured with interrupted 4-0 or 5-0 Vicryl sutures passing through full thickness of the tracheal wall.

The patient remains intubated for a week or longer, as necessary, to permit the patch to stiffen so that the airway will not collapse. The upper and lower ends of the tracheoplasty are marked with hemoclips. The

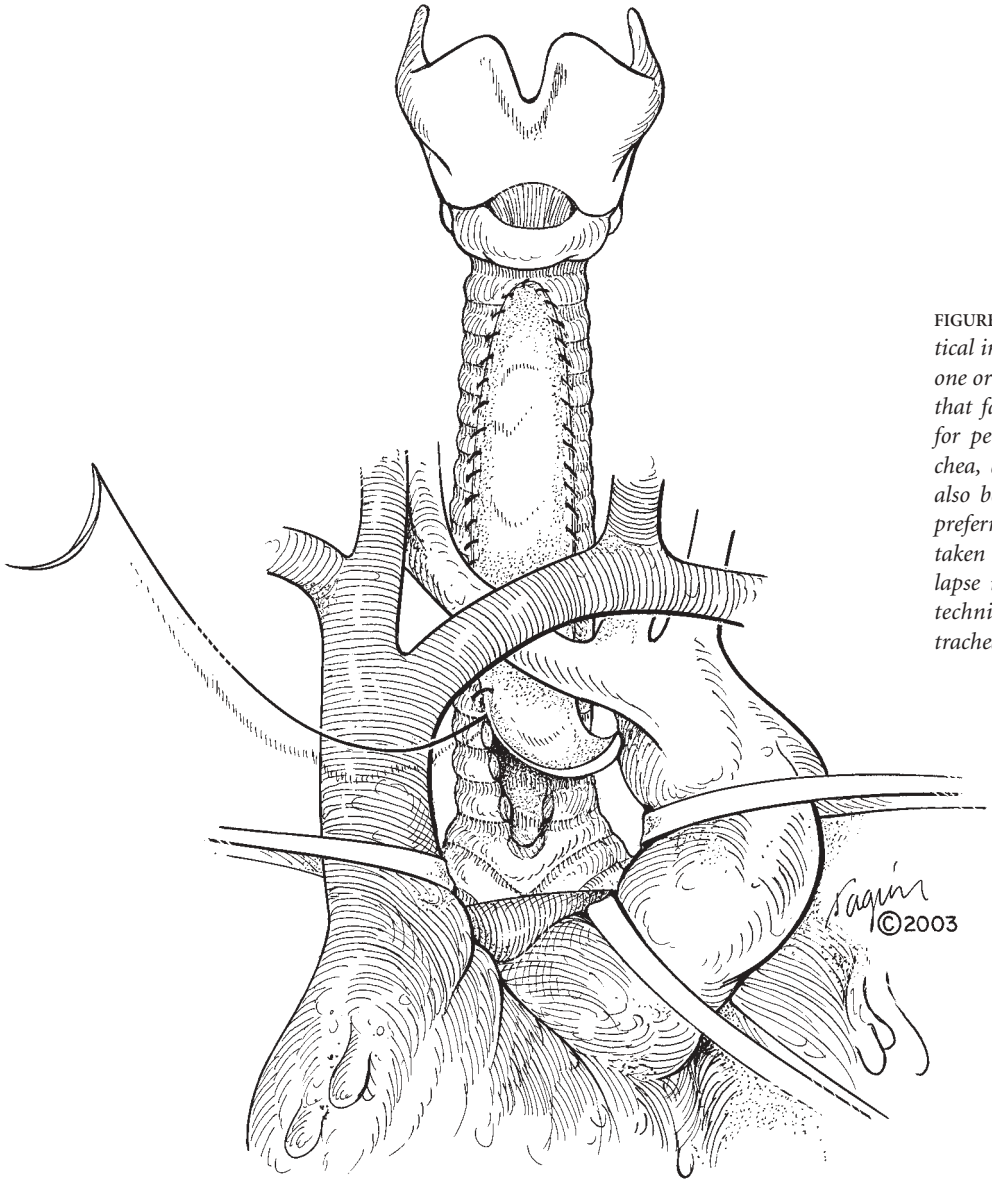


FIGURE **33-2** *Patch tracheoplasty. The vertical incision in trachea can be carried onto one or both main bronchi if stenosis extends that far. Running suture has been advised for pericardial repair. The patch, the trachea, and the brachiocephalic artery must also be suspended. Interrupted sutures are preferred for a cartilage patch, and care is taken to fashion it so that it does not prolapse into the lumen of the trachea. Both techniques require postoperative endotracheal tube splinting.*

repair is examined with a flexible pediatric bronchoscope and an endotracheal tube is positioned so that its tip lies just distal to the lower end of the pericardial patch. The integrity of the tracheoplasty is checked under saline. In reports of this technique, second-layer buttressing has not been thought necessary. Granulation tissue formation, which requires repetitive bronchoscopic removal, occurs frequently.[9] Contraction of the pericardial patch and complete epithelization both appear to occur in the long run. Tracheal growth has been adequate in follow-up.

Other native or preserved tissues have been used for augmentation of the trachea, but Kimura and colleagues' original use of cartilage has been successfully employed.[4,5,8] Cartilage is harvested from the costal margin through the lower part of the sternotomy incision. After the length and shape of the tracheal defect is determined, the cartilage graft is carefully shaped, preserving perichondrium. The cartilage is precisely sutured into the defect with perichondrium facing the lumen, using interrupted sutures. Seven to 10 days of intubation and ventilation are followed by bronchoscopy to determine adequacy and stability of the repair

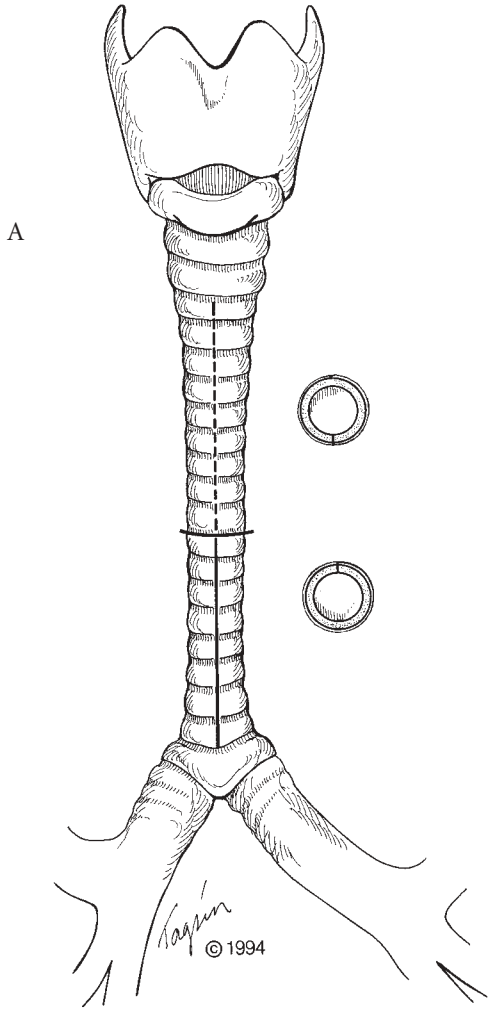


FIGURE **33-3** *Technique of slide tracheoplasty.* A, *The extent of stenosis is precisely defined by intraoperative examination and bronchoscopy, if necessary. The stenotic segment is divided at its midpoint. Only that point is circumferentially dissected, in order to preserve as much lateral blood supply as possible. The lower stenotic segment will be divided anteriorly and the upper posteriorly for the full length of the stenosis.*

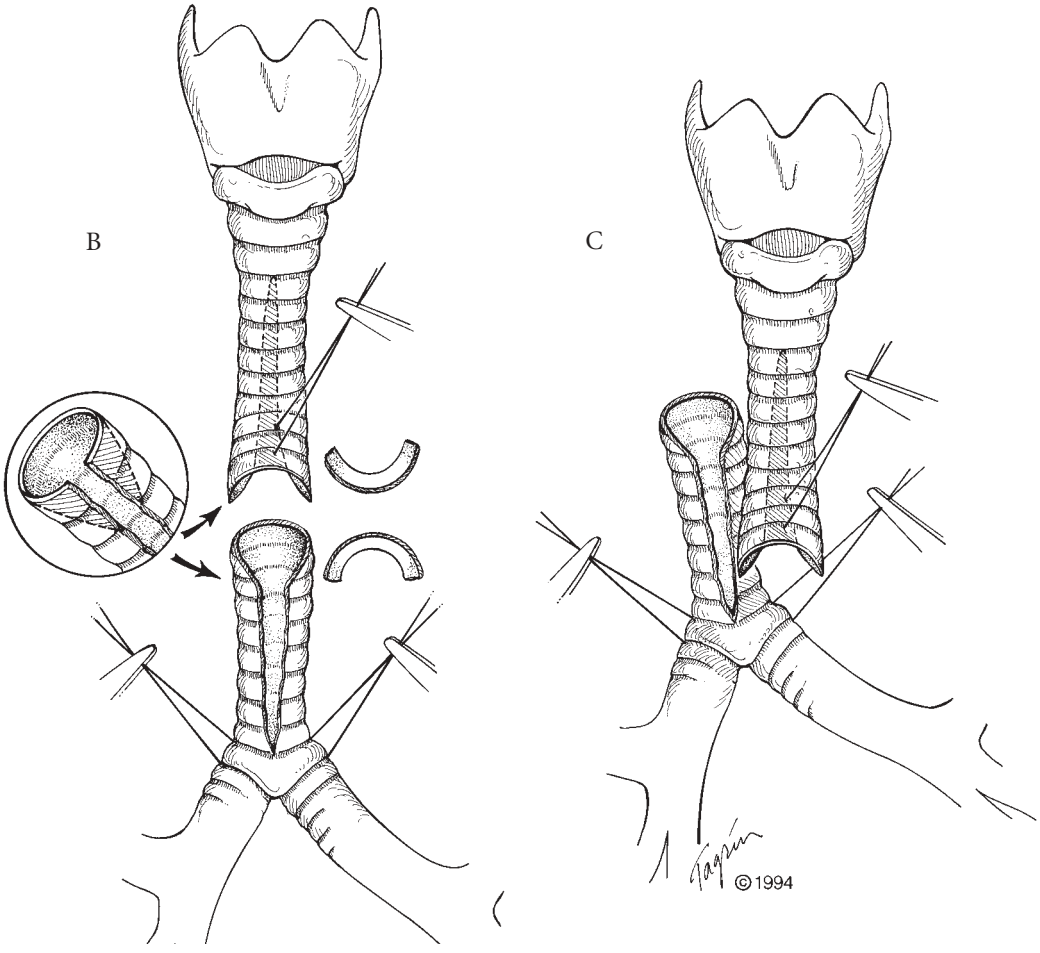
and to remove granulation tissue. Rigidity of cartilage compared to pericardium obviates the need for mediastinal suspension and allows the endotracheal tube to be positioned more proximally.

I prefer to use slide tracheoplasty rather than patch tracheoplasty.

Slide Tracheoplasty

Tsang, Goldstraw and colleagues proposed doubling the circumference of the stenotic trachea by dividing the stenotic segment horizontally at its midpoint, slitting the upper and lower stenotic segments respectively anteriorly and posteriorly, and sliding the two segments together.[10] The trachea is thus effectively shortened by half of the length of the stenotic segment, which is usually an acceptable length of shortening. The circumference is doubled and the cross-sectional area of the trachea is quadrupled. Grillo adapted the technique with some modifications (Figure 33-3).[11]

The procedure is performed under ventilatory anesthesia in the absence of associated vascular or cardiac anomalies. It is preferable if a small bore endotracheal tube can be slipped through the stenosis, but it is possible with delicate dissection to perform the first part of the exposure with a tube positioned in the proximal airway above the stenosis (Figure 33-4). This is usually the case in infants and small children. It has been possible to expose the trachea sufficiently in these young patients through a collar incision alone. If additional exposure is required, the upper sternum or even the entire sternum may be divided. In most



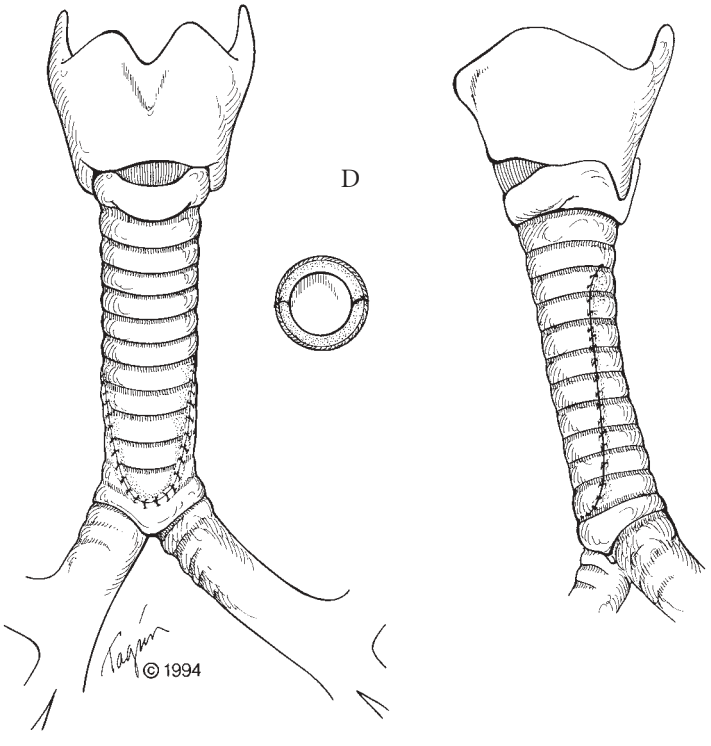


FIGURE **33-3** (CONTINUED) B, *The right-angled corners at the midpoint tracheal division are trimmed above and below. A single traction suture is placed near the tip of the upper segment and two lateral sutures below the stenosis inferiorly, hence often at the tracheobronchial angles. Only absolutely necessary dissection is done, in order to conserve tracheal blood supply. Tracheal circumference will be doubled.* C, *After all anastomotic sutures have been placed and marshaled in orderly array, the segments are slid together, with the help of the stay sutures, and the anastomotic sutures are tied.* D, *An oblique suture line results, with quadrupling of the cross-sectional area, since the circumference of the trachea has been doubled. Reproduced with permission from Grillo HC.*[11]

patients, the trachea can be exposed satisfactorily behind the aortic arch and brachiocephalic artery, but in some, the lower trachea is better seen by dividing the pericardium between the retracted vena cava and aorta. If a pulmonary artery sling is present, complete sternotomy becomes necessary. In this case, partial cardiopulmonary bypass is necessary for reimplantation of the left pulmonary artery, and it is then convenient to perform tracheoplasty also under bypass. All dissection prior to these steps is done under ventilatory anesthesia to minimize bypass time.

The exact proximal end of the stenosis is identified with a bronchoscope, usually flexible. With transillumination of the trachea, at the point of stenosis, a fine needle is used to locate it precisely, marking the point with a fine suture. The lower end of the stenosis is generally evident. The trachea is divided transversely at the midpoint of stenosis after dissecting circumferentially *only* at this level (see Figure 33-3*A*). Ventilation is continued across the operative field, most easily after the lower stenotic segment has been incised vertically anteriorly. The endotracheal tube is usually passed into the proximal left main bronchus, which provides very adequate left lung ventilation (see Figure 33-4*B*). It may be episodically removed for brief periods to facilitate suture placement and then easily replaced. Tsang and Goldstraw and their colleagues' original technique incised the upper segment anteriorly and lower segment posteriorly.[10] I prefer to open the lower segment anteriorly, allowing easy insertion of the endotracheal tube .[11] The lateral attachments of the segment remain undissected to guarantee blood supply. The upper stenotic segment is dissected posteriorly, and to a lesser extent laterally, only until posterior vertical incision of the stenosis can be

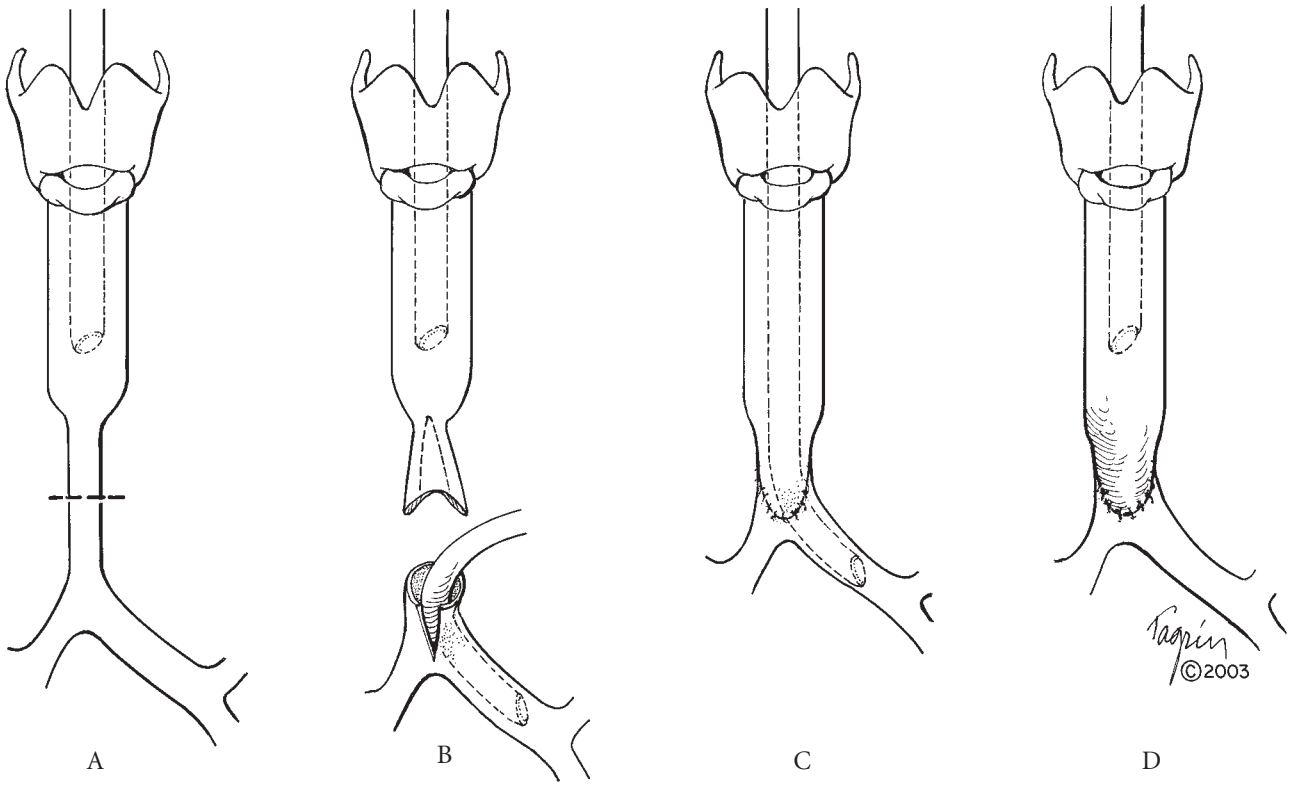


FIGURE **33-4** *Management of ventilation during repair of long congenital stenosis, when cardiopulmonary bypass is not needed.* A, *Tightness of stenosis most often requires a small bore endotracheal tube (ET) to reside above the narrowing.* B, *After division of trachea, the distal segment is opened anteriorly and a second ET passed into the left main bronchus (LMB). Another small bore tube can be directed into the right if necessary, but it rarely is.* C, *After all sutures have been placed, the proximal ET is directed into the LMB and anastomosis performed.* D, *When the anastomosis is completed, the ET tube is retracted proximally to ventilate both lungs.*

clearly performed. It is essential that the entire length of stenosis be incised. Inferiorly, this frequently requires that the tracheal incision reach the carina. At the point of intersection of the vertical incisions with the transverse tracheal division, the corners are trimmed conservatively to round the respective ends of the trachea for insertion at the apices of the vertical incisions during reconstruction (see Figure 33-3*B*). The originators of the technique dissected the entire stenotic segment circumferentially and noted no devascularization in a limited experience.[10] I have preferred to conserve as much blood supply as possible. No devascularization problems have occurred in 8 patients aged 10 days to 19 years.[12]

Traction sutures help during suturing and in approximating the tracheal ends. In a low stenosis, I place one suture at each tracheobronchial angle, using 4-0 Vicryl in infants, and a single suture near the tip of the superior flap which lies anteriorly (see Figure 33-3*B*). Tentative approximation may be tested by drawing these sutures together while the patient's neck is gently flexed (see Figure 33-3*C*). Even with very long stenosis, approximation has been easily effected without clinically evident tension. In one patient who had undergone previous extensive mediastinal dissection, including anastomosis of a posterior pulmonary artery to superior vena cava, tracheal mobility was somewhat limited, but still feasible.[12]

Anastomosis is made with 4-0, 5-0, or 6-0 Vicryl sutures depending upon the patient's age and tracheal dimensions. I prefer interrupted sutures. The first is placed as a mattress suture from the apex of the "tongue" of the inferior tracheal segment to the top of the posterior vertical incision in the proximal segment. All sutures are placed so that the knots will lie outside of the tracheal lumen. Sutures are placed successively on both sides, working down from posterior to anterior. Attention is paid to even spacing about 3 mm apart. Sutures pass through the full thickness of the tracheal wall. Each suture is clipped with a hemostat and fastened serially to the drapes with a second hemostat. All anastomotic sutures are placed before any are tied. The endotracheal tube can be removed at intervals briefly for suture placement if necessary, since the patient is kept fully oxygenated. Once all sutures have been placed, an endotracheal tube is passed down from the proximal trachea into the left main bronchus (see Figure 33-4*C*). Generally, sutures are tied in reverse order of placement, commencing in anterior midline inferiorly and progressing up on either side to the apical posterior suture. The stay sutures and cervical flexion minimize tension during anastomosis. The endotracheal tube is retracted into the upper trachea, and the anastomosis checked for integrity under saline (see Figure 33-4*D*). The anastomosis is examined via endotracheal tube with a flexible pediatric bronchoscope. Stay sutures are removed (see Figure 33-3*D*).

The thymic lobes, divided in the midline during initial exposure, provide convenient tissue for a "second-layer" closure. Since the trachea has been reconstructed with tracheal tissue, an endotracheal tube is not necessary as a stent postoperatively. Most often, the endotracheal tube can be removed at the conclusion of the operation or the next day, if it is initially needed for suctioning. Guardian chin sutures to restrain hyperextension have been used. Drastic flexion is avoided. Laryngeal release has not been required in our experience.

Slide tracheoplasty may well be the procedure of choice for long congenital tracheal stenosis. The advantages are numerous. The trachea is repaired with native trachea, which is lined with normal tracheal epithelium and has firm cartilaginous structure. The trachea grows with the child.[12] Intraoperative suspension and postoperative intubation for splinting, with many days of ventilation, are not required. Bypass is unnecessary except where there is a concomitant lesion such as a pulmonary artery sling that needs reimplantation. Because of the immediate approximation of epithelium to epithelium, granulation tissue formation is very rare and minimal, obviating the need for multiple therapeutic bronchoscopies postoperatively.

Other Techniques

With complex anomalies, ingenuity may be required. Congenital stenosis may be associated with anomalous right upper lobe bronchus (*bronchus suis*), bridging bronchi, stenotic main bronchi, degrees of

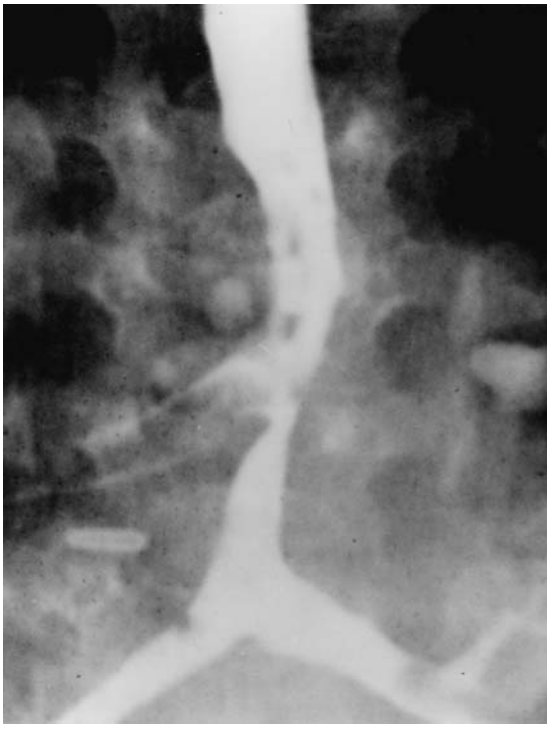


FIGURE **33-5** *Tracheobronchogram showing a complex stenosis. A segment of relatively mild stenosis lies proximal to an anomalous right upper lobe bronchus. The stenotic "bridge bronchus" below shows maximal narrowing in its most proximal portion. The main bronchi are adequate in diameter.*

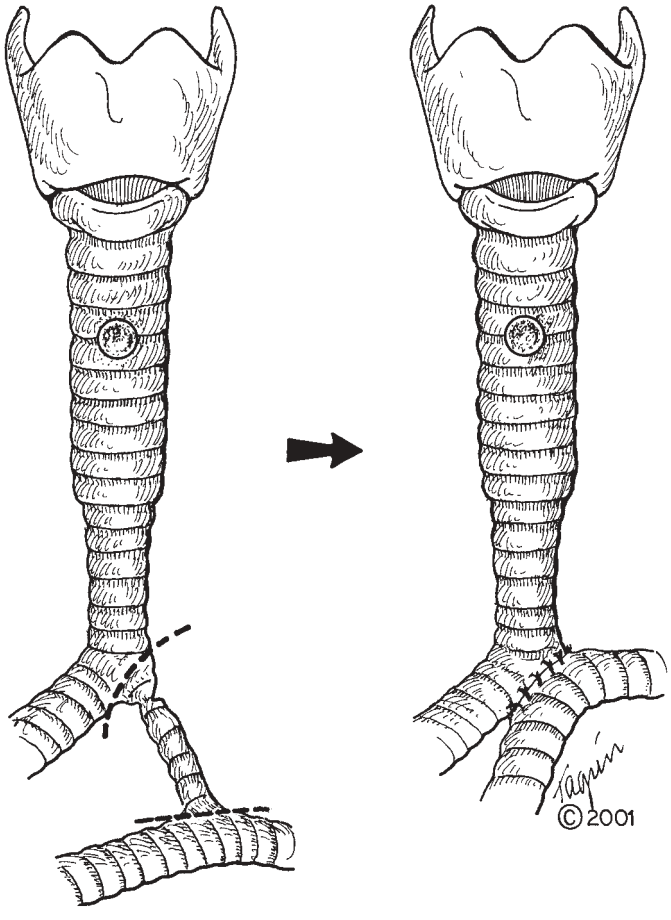


FIGURE **33-6** *Resection of "bridge bronchus" and anastomosis shown in Figure 33-5. The apertures must be carefully designed to avoid anastomotic kinking. The patient had a tracheostomy on arrival. Reproduced with permission from Grillo HC et al.*[12]

bronchomalacia, and, as noted, pulmonary artery sling. I have used Cantrell and Guild's technique for management of a "bridging" bronchus that was extremely narrow and funneled to a tiny diameter at its proximal end (Figure 33-5).[13] Neither patch nor slide tracheoplasty would have sufficed. The bronchus was short enough so that it could be resected, and the junction of trachea and right upper lobe bronchus could be anastomosed to the residual "carina" between right and left "main" bronchi (Figure 33-6). Slide tracheoplasty was successfully used in another bridging bronchial stenosis (Figure 33-7). It should be possible to modify the slide technique to widen proximal main bronchi if necessary (Figure 33-8), although I have not yet encountered this situation. Where I encountered circular tracheal rings in the main bronchi, the narrowing was not severe enough to require bronchial enlargement.

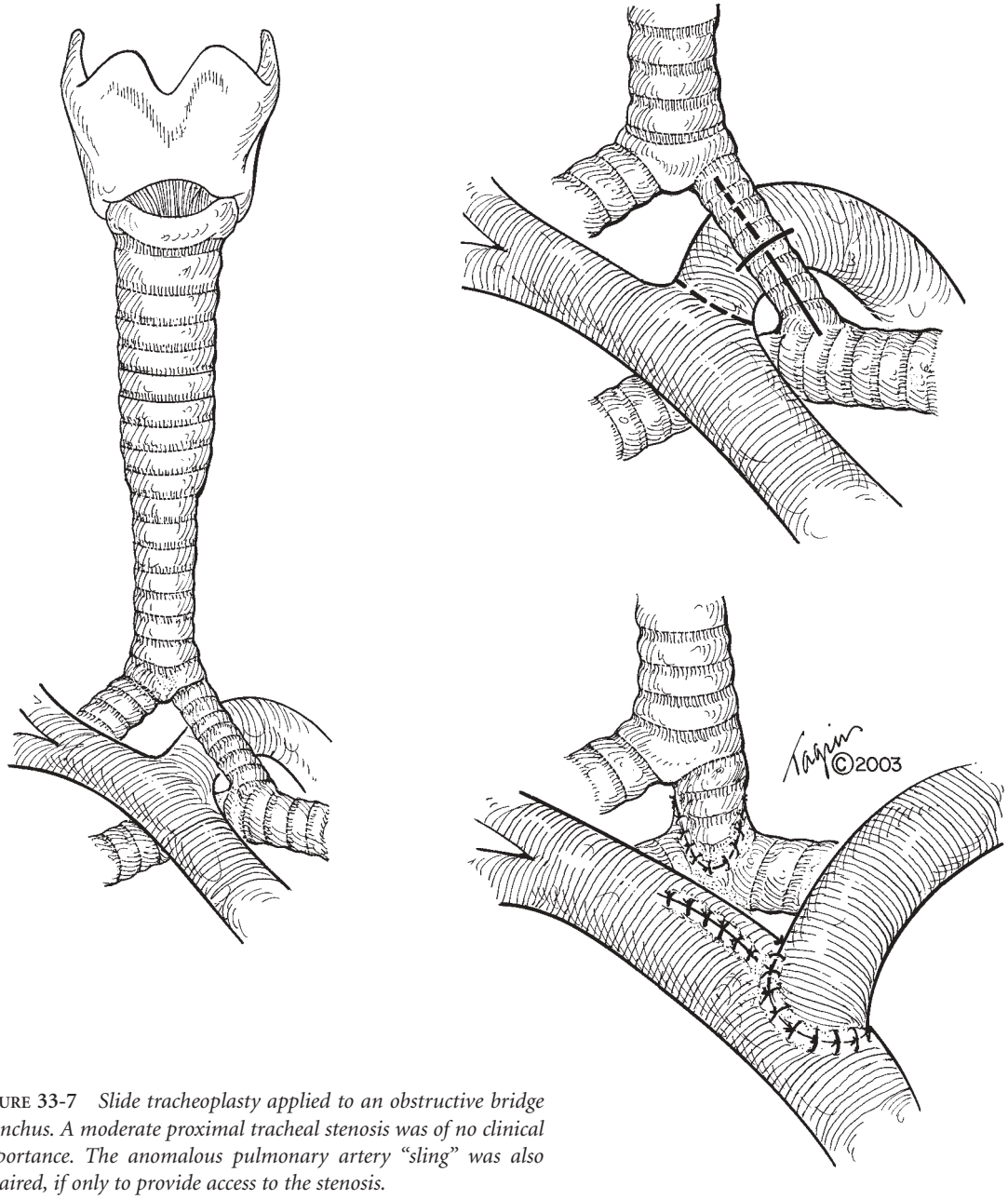


FIGURE **33-7** *Slide tracheoplasty applied to an obstructive bridge bronchus. A moderate proximal tracheal stenosis was of no clinical importance. The anomalous pulmonary artery "sling" was also repaired, if only to provide access to the stenosis.*

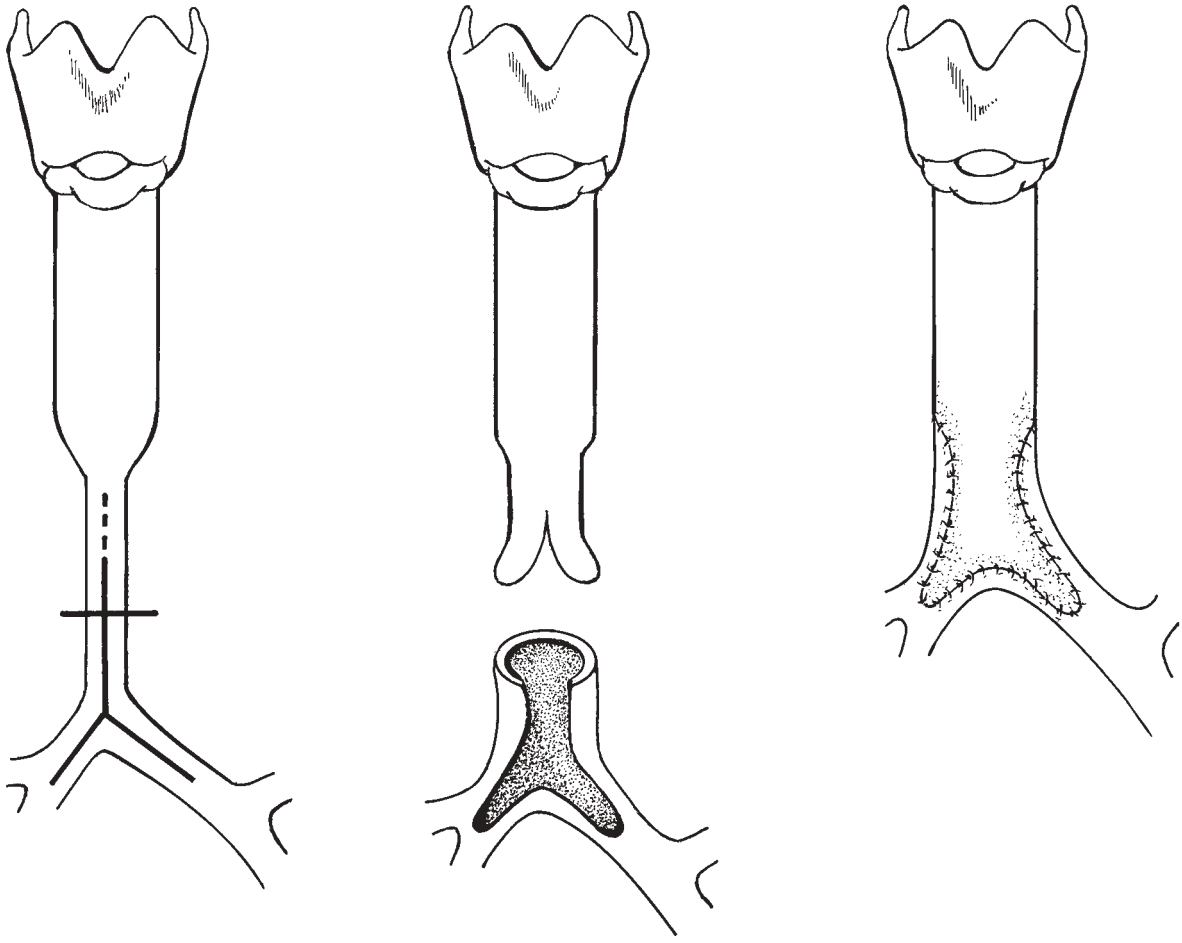


FIGURE **33-8** *Proposed procedure for slide tracheobronchoplasty where significant stenosis is also present in proximal main bronchi. The point of transverse division of the stenotic trachea is below the midpoint of tracheal stenosis to provide necessary length for slide bronchoplasties also. The* dashed line *indicates proximal extent of posterior incision of upper portion of stenosis. We have not yet encountered this situation and must emphasize that this technique is untested.*

References

1. DeLorimier AA, Harrison MR, Karen H, et al. Tracheobronchial obstruction in infants and children: experience with 45 cases. Ann Surg 1990;212:277–89.
2. Grillo HC, Zannini P. Management of obstructive tracheal disease in children. J. Pediatr Surg 1984; 19:414–6.
3. Wright CD, Graham BB, Grillo HC, et al. Pediatric tracheal surgery. Ann Thorac Surg 2002;74:308–14.
4. Kimura K, Mukohara N, Tsugawa C, et al. Tracheoplasty for congenital stenosis of entire trachea. J Pediatr Surg 1982;17:869–71.
5. Tsugawa C, Kimura K, Muraji T, et al. Congenital stenosis involving a long segment of the trachea: further experience in reconstructive surgery. J Pediatr Surg 1988;23:471–5.
6. Idriss FS, DeLeon SY, Ibani MN, et al. Tracheoplasty with pericardial patch for extensive tracheal stenosis in infants and children. J Thorac Cardiovasc Surg 1984; 88:527–36.
7. Bando K, Turrentine MW, Sun K, et al. Intermediate to long-term outcomes after anterior pericardial tracheoplasty for congenital tracheal stenosis. Ann Thorac Surg 1996;62:981–9.
8. Jaquiss RDB, Lusk PR, Spray TL, et al. Repair of long-segment tracheal stenosis in infancy. J Thorac Cardiovasc Surg 1995;110:1504–12.
9. Dunham ME, Backer CL, Holinger LD, et al. Management of severe congenital tracheal stenosis. Ann Otol Rhinol Laryngol 1994;103:351–6.
10. Tsang V, Murday A, Gilbe C, et al. Slide tracheoplasty for congenital funnel-shaped tracheal stenosis. Ann Thorac Surg 1989;48:632–5.
11. Grillo HC. Slide tracheoplasty for long segment congenital stenosis. Ann Thorac Surg 1994;58:613–21.
12. Grillo HC, Wright CD, Vlahakes GJ, MacGillivray TE. Management of congenital tracheal stenosis by means of slide tracheoplasty or resection and reconstruction with long term follow-up of growth after slide tracheoplasty. J Thorac Cardiovasc Surg 2002;123: 145–52.
13. Cantrell JR, Guild HC. Congenital stenosis of the trachea. Am J Surg 1964;108:297–305.

33B Laryngotracheoesophageal Cleft Repair

Daniel P. Ryan, MD

Laryngotracheoesophageal cleft is a rare, congenital anomaly that results from failure of separation of the trachea and esophagus (see Figure 6-4 in Chapter 6, "Congenital and Acquired Tracheal Lesions in Children").[1,2] Early manifestations are related to the airway and feeding difficulties. Associated malformations should also be recognized. Many of these patients have foreshortened tracheas and most also have microgastria with severe gastroesophageal reflux. This description of technique for repair is limited to the most extensive clefts: type III, which extends to the carina; and type IV, in which the cleft extends into one or both mainstem bronchi.

Patients present with respiratory distress related to the floppy airway or with aspiration with feeding. They are often difficult to ventilate after intubation because of the connection between the trachea and esophagus, and may require cuffed endotracheal tubes to maintain airway pressure. There also seems to be an association with esophageal atresia and often the cleft is not identified until after repair of esophageal atresia.

When suspected, diagnosis is best confirmed by rigid bronchoscopy. Identification of the cleft is usually obvious for the most extensive lesions (Figure 33-9). However, due to floppiness of the tracheal rings, examination can sometimes be confusing. During rigid bronchoscopy, the bronchoscope "drops" posteriorly through the larynx and into the esophageal space. Maintenance of ventilation during bronchoscopy can be difficult. The anatomy must be carefully assessed, in order to adequately plan repair and stabilize the infant. The length of the trachea needs to be very precisely evaluated. It is very easy to be fooled into thinking that the trachea looks normal when, indeed, the long left mainstem bronchus is being visualized, with the right bronchial origin hidden in folds of the esophagus.

After securing the airway, the initial step in caring for these patients is to do a laparotomy and divide the stomach, placing a draining tube in the proximal aspect and a feeding tube in the distal part of the stomach. Because of the associated microgastria and the concurrent massive gastroesophageal reflux that these patients have, this is the safest way to protect the airway and the lungs during the recovery period and allow enteral feedings, thus avoiding the risk of long-term parenteral nutrition. Intestinal continuity can be reconstructed at a later date.

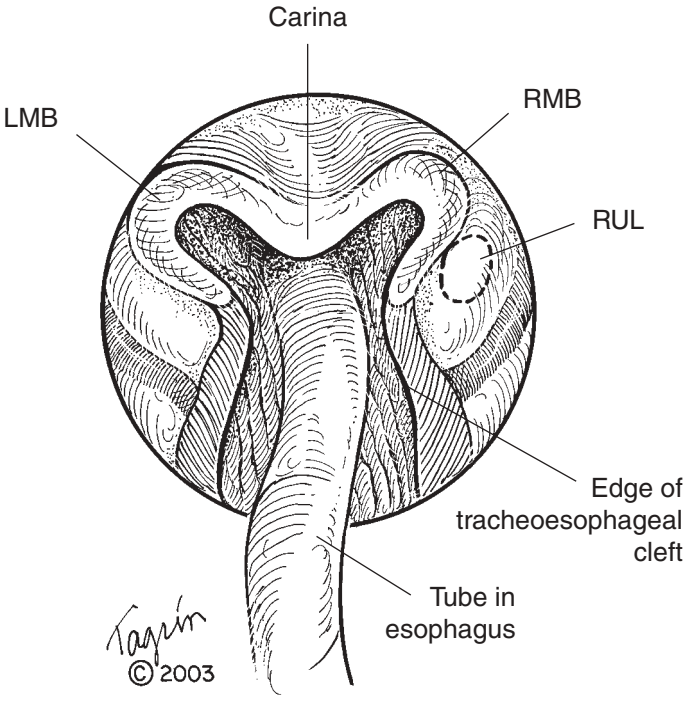


FIGURE **33-9** *Bronchoscopic view at carina of laryngo tracheoesophageal cleft type IV. The carina is flattened. The right upper lobe bronchial orifice is out of view (indicated by* dotted circle*). A nasogastric tube is in the esophagus. LMB, RLM = left, right main bronchus.*

The patient can often be cared for without cardiopulmonary bypass. However, there is no direct contraindication to bypass for this procedure. If it is elected, extrathoracic cannulation is usually easiest and should only be necessary for the duration of the procedure, since the airway will be secure at the completion of the repair. We have not used cardiopulmonary bypass for cases like this, but have used it in other patients to stabilize their respiratory situation while evaluation was proceeding.

Often, the most difficult aspect is securing the airway prior to operation. Under endoscopic guidance, we have introduced custom-made bifurcated endotracheal tubes into the respective mainstem bronchi in order to secure the airway and avoid cardiopulmonary bypass. Because of the small size of the tubes, it is sometimes difficult to keep them in place and to secure them. We have used a tracheostomy incision high up in the airway, with a catheter looped around the endotracheal tube, in order to secure it anteriorly and maintain ventilation (Figure 33-10*A*).

After the airway is secure, the operative approach can be planned. In patients who have a normal length of trachea, a combination of a right cervical incision and right thoracotomy provides the necessary exposure. In those with a foreshortened trachea, a cervical incision with perhaps a minor extension into the sternum is all that is necessary.

After the common tracheoesophagus is exposed, we prefer to make an incision on the right side along the junction between the tracheal cartilages and the wall of the esophagus (see Figures 33-10*A*,*B*). At this point, the endotracheal tube can be fitted into its best position under direct vision. The incision is started at the midpoint of the trachea and carried down to the distal extent of the cleft. One then needs to fashion a second incision on the wall of the esophagus, which will then be brought around posteriorly to create the "trachealis muscle" in the airway (Figures 33-11*A*,*B*). At the distal ends, the incision into the esophagus must be tailored in order to provide flaps of tissue to close the cleft into each mainstem bronchus (see Figures 33-10, 33-11). Care must be taken to avoid taking too much tissue, causing tracheobronchomalacia, which can be very problematic in the long-term. Usually, there is sufficient esophageal tissue so that esophageal narrowing has not been a long-term issue.

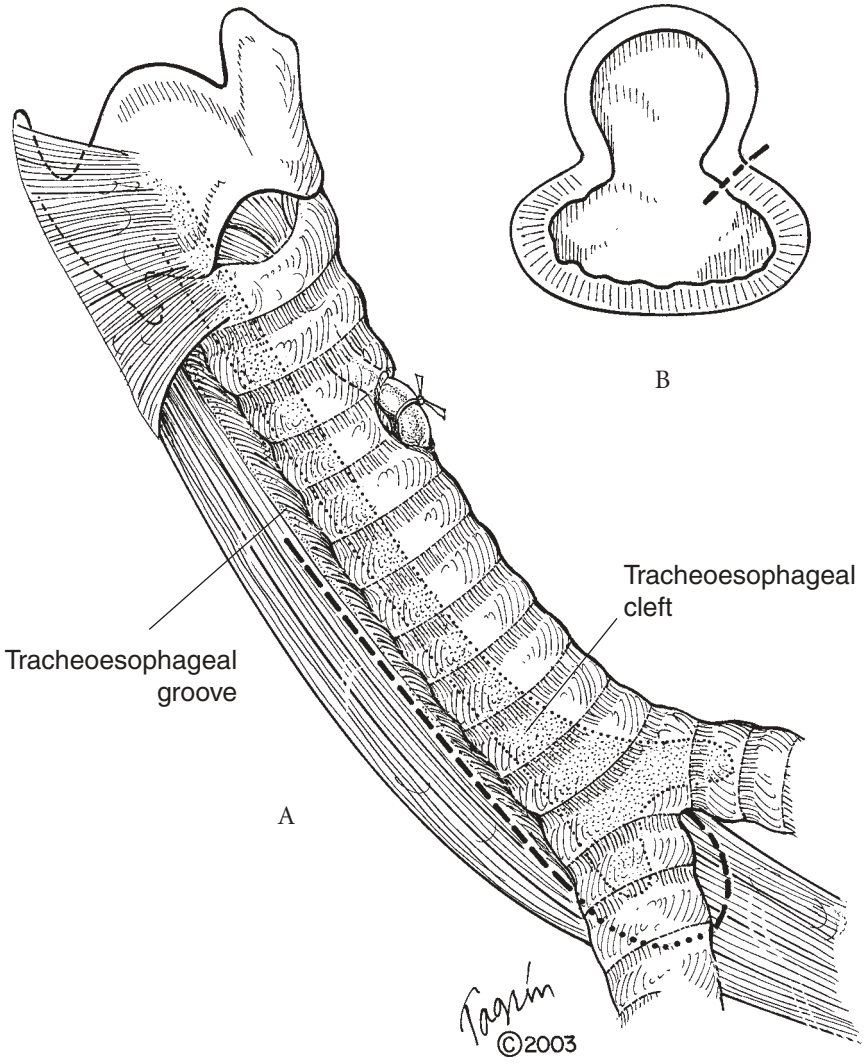
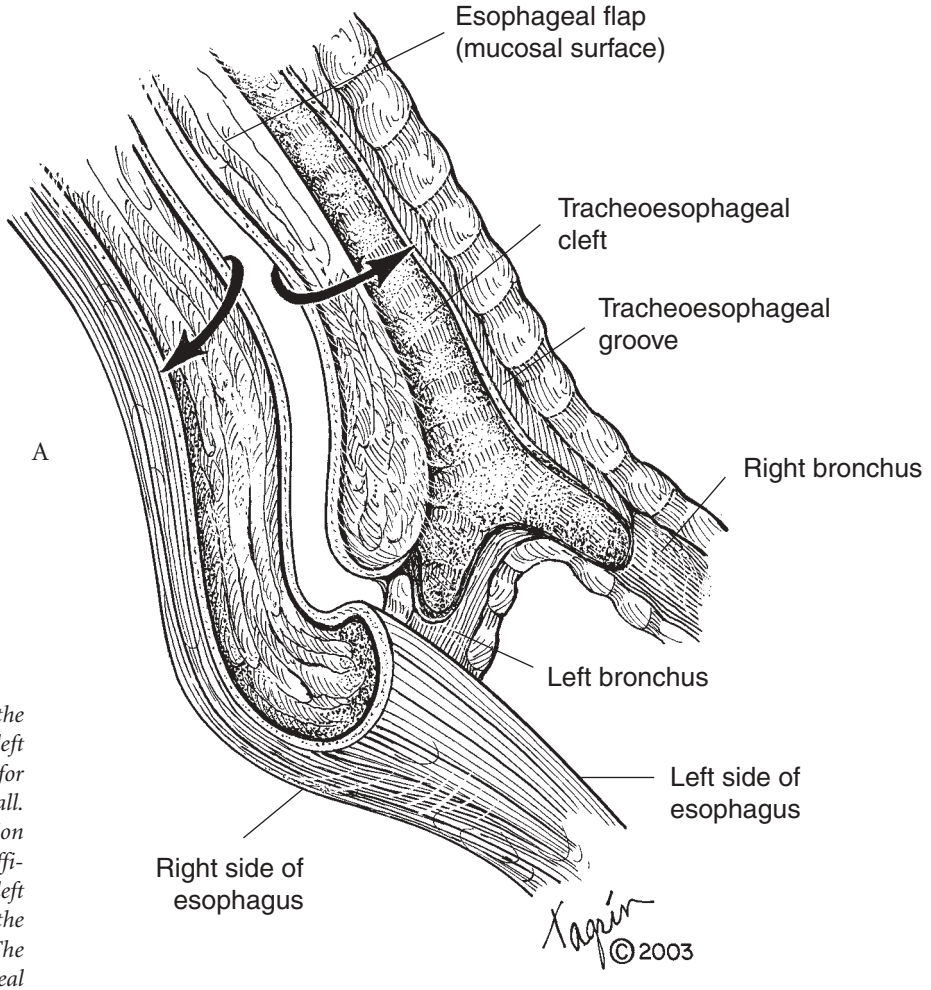


FIGURE **33-10** *Repair of type IV cleft with extension into both main bronchi* (dotted line). A, *A longitudinal incision is made in the right tracheoesophageal groove* (dashed line) *beginning at the midtrachea. At the distal extent, the esophageal incision is carried beneath the mainstem bronchus on the anterior esophageal wall to create a "U" flap. Note the loop in the tracheotomy, which holds the endotracheal tube anteriorly in the trachea.* B, *Cross-sectional diagram of the trachea and esophagus showing the location of the longitudinal incision.*

We usually begin the repair distally in the mainstem bronchi, suturing the "esophageal muscle flap" to the cut edge along the tracheal cartilages (see Figures 33-11*A*,*C*). We have used 5-0 Vicryl or polydioxanone sutures in either running or interrupted fashion. This gives an airtight closure and, starting distally, helps to prevent dislodgment of the endotracheal tube.

After the distal trachea and bronchi are closed, and before the repair is completed above, the bifurcated endotracheal tube is removed and a tracheostomy tube is placed under direct vision and connected to sterile ventilator tubing on the field. Alternatively, an orotracheal tube can be positioned just above the carina to maintain ventilation. We have preferred to use a custom-made "right-angled" tracheostomy tube. The patients often have concurrent tracheobronchomalacia and require positive pressure ventilation for some time after repair. The "right-angled" tube has shorter distance between the tube and the neck flanges that help prevent posterior pressure on the fresh suture lines.

Closure of the larynx and separation from the pharynx can be confusing, because the anatomy is seen from an unusual position on the right side. We have found that the best way to line up the structures is to open the pharynx laterally as a continuation of the incision between the tracheal cartilages and the esophagus below (Figure 33-12*A*). At the level of the arytenoid cartilages, an incision is made to separate



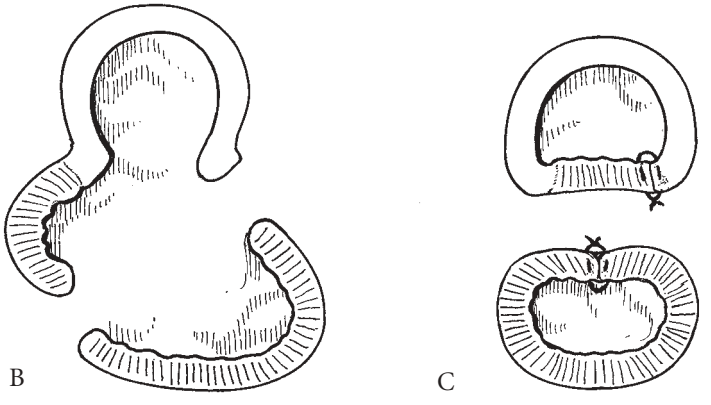


FIGURE **33-11** A, *Incision is continued in the left wall of the esophagus, 1 cm from the left tracheoesophageal groove, to create a flap for reconstruction of a membranous tracheal wall. Enough tissue is provided at the carinal region to allow closure of the bronchial clefts. A sufficiently broad flap is created to preserve the left recurrent laryngeal nerve, which travels in the left-sided tracheoesophageal groove.* B, *The point of division of the left-sided esophageal wall is indicated. Esophageal mucosa thus becomes the mucosa of the membranous wall of the trachea.* C, *The posterior tracheal wall is repaired, using interrupted sutures. A continuous suture is used to reconstruct the esophagus to the level of the inferior cornu of the thyroid cartilage. Care is taken to avoid redundant esophageal wall in repair of the trachea in order to prevent intraluminal prolapse.*

the larynx from the esophagus and pharynx. We start a mucosal closure at that level and proceed inferiorly. The landmarks of the superior and inferior edges of the thyroid cartilage approximate the larynx and the area of the cricoid cartilage (Figure 33-12*B*). One needs to be careful not to put too much tissue from the esophagus in this area. There usually is room to place a second layer of suture in the muscles of the

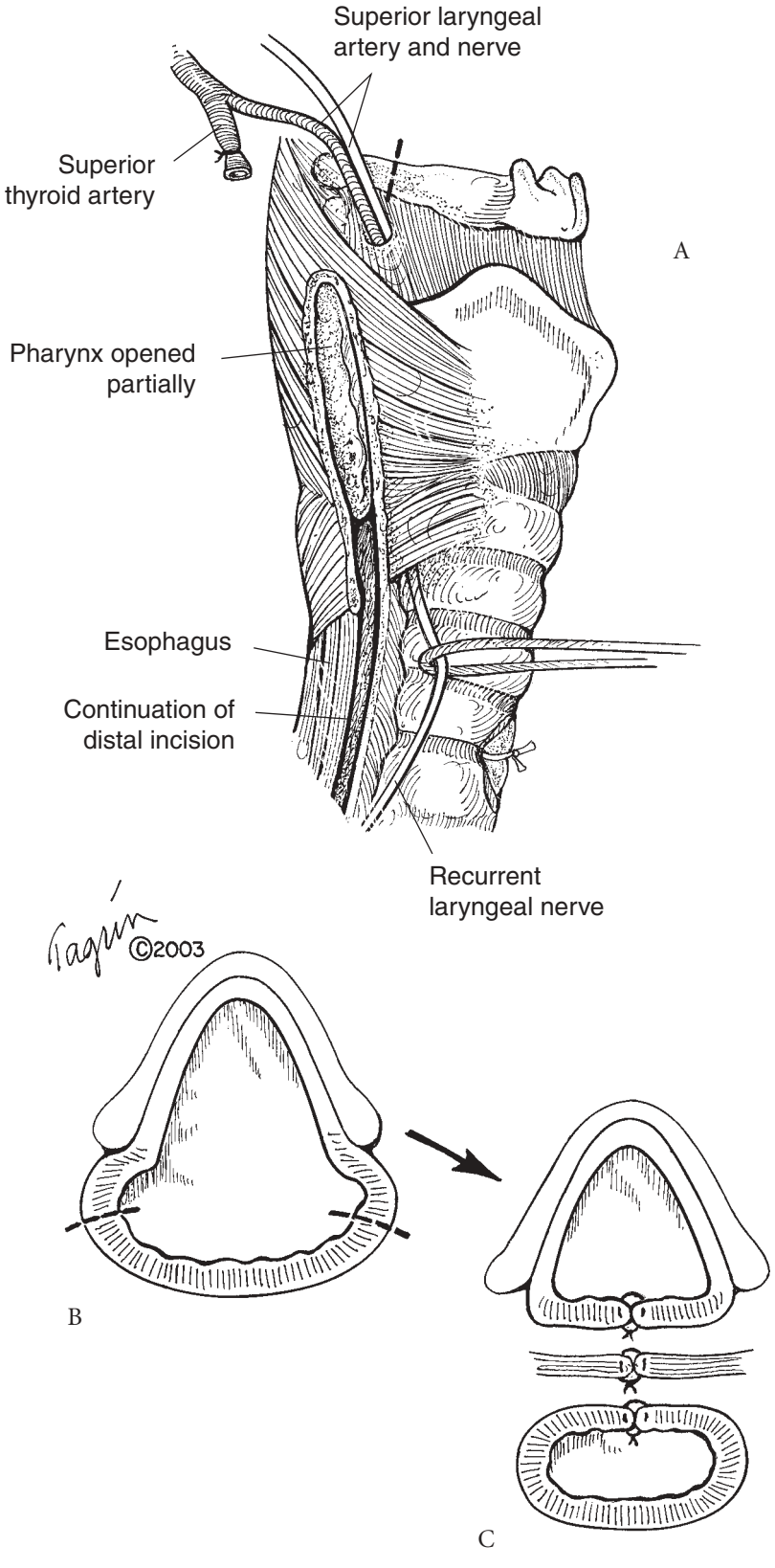


FIGURE **33-12** *Laryngotracheoesophageal cleft repair. Approach to the proximal segment.* A, *Removal of a segment of hyoid bone will expose the right superior laryngeal nerve and artery. This is not always necessary. The recurrent laryngeal nerve is identified below. The pharyngeal wall is exposed through the inferior pharyngeal constrictor muscle.* B, *After the pharynx is opened laterally, the cleft larynx is incised from inferior to superior cornu of thyroid cartilage on each side, in preparation for layered closure.* C, *The intralaryngeal mucosa is closed, connective tissue and laryngeal muscle is used to create a layer beneath this, and the mucosa of the pharynx bordering the laryngeal cleft is closed behind the laryngeal repair. The intralaryngeal suture line meets the membranous wall reconstruction inferiorly. Finally, the pharyngeal incision is closed.*

larynx posterior to the airway mucosa (Figure 33-12*C*). The pharynx can then be reconstructed with a mucosal layer, starting superiorly and coming down inferiorly to meet with the esophageal closure.

If the approach is through a right thoracotomy, we have positioned a pleural flap between the suture lines. In the neck, the sternal half of the right sternocleidomastoid muscle is brought behind the tracheostomy tube to separate the suture lines. Combination of these maneuvers has minimized recurrent fistula formation.

After closing the incisions, we position the patients in a posterior padded splint in order to prevent movement of the head and neck, avoiding pressure on these new suture lines. The patient is kept sedated and ventilated for 1 week or more in order to allow the tissues to begin healing before the posterior splint is removed. We inspect the airway frequently by bronchoscopy after the first week in order to identify granulation tissue formation and evaluate the degree of tracheobronchomalacia.

The patients can be weaned from muscle relaxants and sedatives and be maintained with continuous positive airway pressure as they continue to grow, until weaned from positive pressure altogether. In the long term, some patients have been decannulated, but others, due to the tracheobronchomalacia, require prolonged internal support with a tracheostomy tube.

In patients with severe bronchomalacia who cannot be maintained with positive airway pressure alone, we have used a custom-made bifurcated tracheostomy tube. This tube is designed to extend into the left mainstem bronchus and into the right mainstem orifice to stent these airways open and prevent the collapse and "death spells" that occur in patients with severe bronchomalacia. As the airway grows along with the children, these tubes are changed to conventional tracheostomy tubes for the long term.

References

1. Ryan DP, Muehrcke DD, Doody DP, et al. Laryngotracheoesophageal cleft (type IV): management and repair of lesions beyond the carina. J Pediatr Surg 1991;26:962–9.

2. Simpson BB, Ryan DP, Doody DP, et al. Type IV laryngeal tracheoesophageal clefts: surgical management for long-term survival. J Pediatr Surg 1996; 31:1128–33.

Cervicomediastinal Exenteration and Mediastinal Tracheostomy

Hermes C. Grillo, MD

A lesion, most often neoplastic, may involve the upper trachea and larynx to such an extent that its removal is impossible without sacrificing the entire larynx, although it may nonetheless be so localized that extirpation might lead to cure. Occasionally, even palliative excision is justified, if the lesion is slowly progressive and would otherwise cause a miserable death by airway obstruction. Lesions that produce these circumstances include adenoid cystic carcinoma of the larynx and trachea (see Figure 7-20 in Chapter 7, "Primary Tracheal Neoplasms"), differentiated carcinoma of the thyroid (papillary, follicular, and mixed variants) (see Figure 8-9 in Chapter 8, "Secondary Tracheal Neoplasms"), a rare poorly-differentiated yet localized carcinoma of the thyroid, recurrent carcinoma of the larynx at the stomal site, postcricoidal squamous carcinoma of the esophagus, and uncommonly, postirradiation lesions of the larynx, trachea, pharynx, and esophagus (see Figure 42-4 in Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation").

General Considerations

If the lesion involves the trachea so extensively that a low cervical tracheostomy is impossible, a mediastinal stoma must be established. If the mediastinal end of the trachea is pulled up to the surface of the chest wall, then tension is produced, with likelihood of anastomotic separation, mediastinal sepsis, and death from erosion of major mediastinal vessels. Numerous solutions were attempted, including the ingenious formation of a tubular conduit into the mediastinum using crossed flaps of anterior chest wall skin.[1] Complex repairs such as this are still likely to result in failure of primary healing. Sisson and colleagues performed mediastinal dissection for recurrent laryngeal carcinoma, which required rotation of cutaneous and pectoralis muscle flaps.[2] I proposed as a solution that the skin surface be brought down to the trachea remaining in the mediastinum, eliminating tension, and further, making a simple stomal anastomosis that would be more likely to heal.[3]

This is accomplished by removing the bony chest wall over the upper mediastinum, including bilaterally the medial heads of the clavicles and the first two costal cartilages, plus a plaque of sternum down through the second interspace. The anterior chest wall skin, mobilized as a broad-based bipedicled flap

with excellent blood supply, falls into the mediastinum where it is sutured to the end of the trachea without anastomotic tension. Initially, this appeared to be a successful solution, but when the trachea was cut very short, nonhealing of the suture line at the point where it overlay the innominate artery still occasionally occurred, with the possibility of hemorrhage from the brachiocephalic artery. Modifications were introduced that seemed to have solved the problem: elective division of the brachiocephalic artery, monitored by electroencephalogram, and/or rotation of omental pedicle flaps.[4–6] Waddell and Cannon suggested transposition of the distal tracheal stump to the right side of the junction of the brachiocephalic artery and aorta to reduce tension on the cutaneous anastomosis, but their maneuver did not solve the problem. This maneuver was also proposed by Orringer, who also concluded that the single transverse skin incision allowed sufficient cutaneous mobility without creating a bipedicled flap, when rotational flaps were not needed.[7]

The complexity of operation depends upon the pathology. Cervical exenteration for postcricoidal carcinoma of the esophagus requires laryngectomy, upper trachiectomy, pharyngectomy, and esophagectomy. Esophageal reconstruction is done by bringing up the stomach or the left colon, either in the bed of the resected esophagus or substernally, with anastomosis to the pharynx and establishment of a tracheostomy low in the neck rather than in the upper mediastinum. Cervicomediastinal removal of extensive adenoid cystic carcinoma may well not require esophagectomy. The pharyngeal defect is closed, as in total laryngectomy, and mediastinal tracheostomy established. However, if the mobilized stomach is to be used for gastrointestinal continuity rather than the colon, the residual esophagus is best removed by the transhiatal technique. The gastric tube is usually placed substernally. If only a portion of the anterior esophageal wall is involved, as occurs in some thyroid cancers, this is excised and the esophagus repaired longitudinally in layers. When major invasion of the esophagus has occurred, either by extensive recurrent thyroid carcinoma or recurrent laryngeal carcinoma at the stoma, complete exenteration is necessary, with mediastinal tracheostomy and esophageal substitution to the pharynx. Cutaneous invasion requires design of unique chest wall flaps, usually myocutaneous.[2,7] Irradiation adds further difficulty both in dissection and in its effect on potential for local tissue healing. Since there is such variation in the extent of pathology, each procedure must be planned individually. Preoperative studies should include endoscopy of both the airway and upper digestive tract. Barium swallow helps to delineate esophageal involvement prior to esophagoscopy. A computed tomography scan is essential to detect the extent of invasion into cervical and mediastinal structures; the contrast helps to define invasion of the carotid or brachiocephalic arteries and the jugular veins. For precise definition, an aortic arch angiogram may be useful. In patients in whom the tracheal stoma is likely to be very low, an aortic arch angiogram may be needed to define the patency of the carotid and vertebral vessels and anastomoses of the circle of Willis. This information will be important if it becomes necessary to divide the brachiocephalic artery. If the colon is likely to be used for reconstruction of the esophagus, angiography of the superior and inferior mesenteric arteries demonstrates their patency, adequacy of marginal arteries, and communication between middle and left colic arteries. Although stomach used as esophageal replacement will usually reach the cervical esophagus at the base of the neck, it may *not* easily reach the partly resected pharynx. The Kocher maneuver may be helpful to mobilize the stomach. Resection of the lesser curvature in successive segments serves to elongate the lesser curvature and the gastric tube so created. A left colon replacement is divided in the neighborhood of the hepatic flexure to guarantee sufficient length. Since a left colic artery pedicle is generally most appropriate, the middle colic arcade must show good communication. This usually requires division of the middle colic artery at its origin to use the right and left branches as an arcade. The vascular anatomy of the right colon is notoriously variable and, often, has poor intercommunications. An additional option, rarely necessary, is free jejunal interposition with vascular anastomosis. Prior cervicomediastinal irradiation makes this inadvisable because of the danger of anastomotic thrombosis of the irradiated recipient arteries.

I routinely prefer to transfer the omentum into the neck and mediastinum in these patients. It is either mobilized with the stomach, leaving the right gastroepiploic artery intact to supply both the stomach and omentum, or pedicled separately on the right gastroepiploic artery if the colon is advanced. It provides an excellent buttress for esophageal and pharyngeal suture lines, a secure seal between the trachea and skin stoma, and covering for mediastinal vessels and vascular suture lines. It is especially valuable where the field has previously been irradiated.

If the operation is accomplished successfully, then despite its magnitude, the patient will be left with the anatomic defect of a "simple" laryngectomy. Many patients learned to use an electrolarynx or other device quite successfully. A few developed an "esophageal voice," even with a colonic or gastric bypass. Thyroid and parathyroid replacement therapies were necessary in many. There are the usual difficulties attendant to gastric or colonic replacement of the esophagus. Late development of thoracic outlet symptoms, from sagging of the shoulder girdle due to loss of clavicular attachments, has also occurred. If stomal stenosis occurs, it is directly managed by dilation and placement of a small silicone prosthesis. Excision of scar and reanastomosis or local Z-plasties at the stoma produced variable results.

Operation

Preparation

The patient is positioned supine with an inflatable bag behind the shoulders to produce cervical extension. The hips and knees are slightly flexed so that the upper cervicosternal field is horizontal without having the legs dependent during the long procedure. Dependable arterial and venous access to the arms is obtained in the same manner as described for anterior approach to the trachea, remembering that interruption of the brachiocephalic artery and left brachiocephalic vein is possible. A bladder catheter is necessary for such a prolonged procedure. Electroencephalographic leads are usually placed when there is any likelihood of division of the brachiocephalic artery. The operative field includes the entire neck, from the chin superiorly to the trapezius muscles and the lateral ends of the clavicles laterally. The entire anterior chest wall and abdomen are prepared, well laterally to the midaxillary line. The anterior surface of one thigh is prepared for possible harvesting of a split thickness skin graft and, less likely, saphenous vein excision for arterial replacement.

Exploration

The cervical incision is made first to determine whether the lesion can be removed and reconstruction accomplished. The initial incision is transverse and follows the line of the clavicles at the base of the neck (Figure 34-1). If resection is to be performed, the incision is extended laterally and turned downward slightly at either end as the shoulder is reached in order to allow the flap to be moved caudad and into the mediastinum more easily. The upper skin flap is elevated superiorly, with platysma attached to a point above the hyoid bone. If skin must be removed with the specimen due to invasion by tumor, this area is outlined during initial elevation of the flaps and remains with the specimen. In such case, the incision will require individual planning. Inferiorly, the skin flap is initially raised to the sternal notch. The mode of exploration will necessarily vary with the lesion. Strap muscles are usually left attached to a bulky tumor in the case of thyroid carcinoma. If not involved, the strap muscles are separated in the midline, elevated, and eventually divided inferiorly at the sternal level. Often, to determine resectability, it is necessary to develop the plane medial to the sternocleidomastoid muscles and to the internal jugular veins and carotid arteries to discover whether tumor has invaded these vessels. If there is no involvement of the thyroid gland on one side or both, the isthmus is divided in the midline and the lobes turned laterally, with the superior thyroid blood supply intact. This will permit continued thyroid and parathyroid function, if not already destroyed by irradiation.

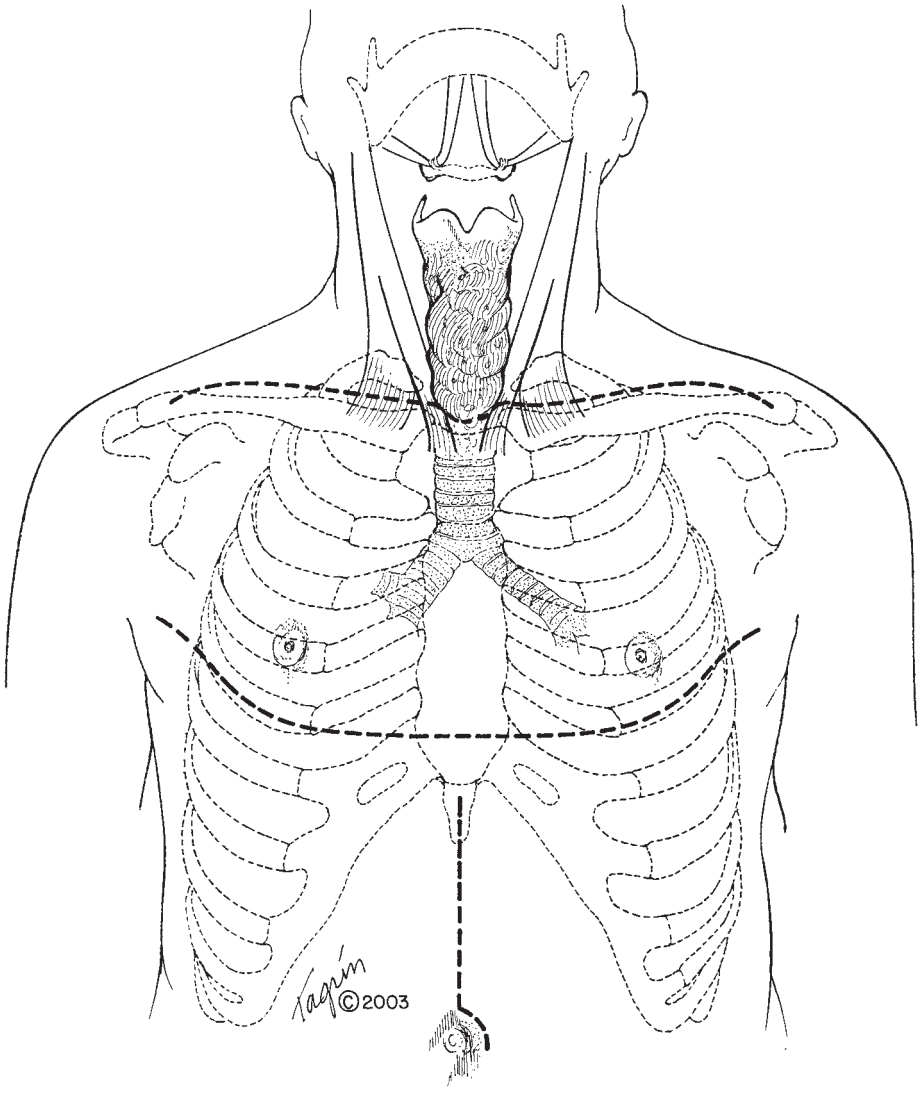


FIGURE **34-1** *Incisions for cervicomediastinal exenteration and mediastinal tracheostomy. The neck and mediastinum are explored through the upper horizontal incision. The lower parallel horizontal incision is made later when the decision has been made to proceed with resection. The upper midline laparotomy incision provides for mobilization and transfer of the pedicled omental flap and esophageal substitute, if the procedure is to be completed.*

However, if tumor invades the thyroid gland, it is removed en bloc with the specimen. No irrevocable moves, such as division of functioning recurrent laryngeal nerves, are made until a final decision has been made that the lesion is indeed removable either for satisfactory palliation or possible cure. I have been willing to sacrifice one internal jugular vein, if necessary, but have generally preferred not to add carotid artery replacement to this extensive oncologic procedure, unless the problem is uniquely promising.

The inferior extent of a tumor and its possible invasion of mediastinal structures must be assessed to determine resectability. The inferior skin flap is elevated from the pectoral fascia with cautery, raising it from medial to lateral in order to follow the natural lines of the pectoral muscle fibers. The flap is retracted and elevation carried broadly to a point below the second interspace over the sternum. The sternoclei-

domastoid muscles are detached from the sternum and clavicles bilaterally (Figure 34-2*A*). The pectoral muscles are elevated on either side from the midline of sternum to expose the cartilages of the first and second ribs and medial portions of the first and second intercostal muscles (see Figure 34-2*A*). Most of this dissection is done with cautery. In baring the heads of the clavicles, which are likely to be resected later, the periosteum is left on the bone. If periosteum remains in the tissues, bone spurs are likely to form. The margins of the sternum in the second interspace are developed bilaterally, below the angle of Louis, with care taken to avoid the internal mammary vessels. The mediastinum is dissected bluntly away from the undersurface of the bone. The sternum is divided transversely, using either a Gigli saw, a Lebsche knife, or a sternal saw. The undersurface of the sternum is bluntly dissected from the sternal notch, and the proximal sternum is divided from the notch to the transverse sternal division (see Figure 34-2*A*). The margins of the divided sternum are spread with a sternal retractor, permitting definitive assessment of the extent of mediastinal tumor. This access also facilitates later removal of the anterior chest wall segments.

Resection

In the cervicomediastinal field, the anterior bony plaque is removed (Figure 34-2*B*). This commences by dissecting circumferentially beneath the clavicle, on either side, at a point approximately 4 cm from the medial end of the bones, with care taken to avoid injury to the subclavian vein. A Gigli saw is passed beneath the bone, and with pressure on the bone to prevent binding of the saw, both clavicles are divided. The intercostal muscles are carefully separated from the lateral margins of the sternum and the second costal cartilages are divided. The divided sternal segments are elevated on either side from the underlying mediastinal tissues. After dissecting beneath the first cartilages, these are divided and, keeping dissection close to the junction of clavicles and sternum, the segments of bony plaque on each side are resected (Figure 34-2*C*).

Tactics for removal of the specimen depend upon what structures are involved. In general, lateral dissections are completed, exposing the medial margins of the carotid arteries and jugular veins. The dissection extends posteriorly toward either the esophagus, if it will remain, or to the anterior surface of the vertebral bodies, if laryngotracheoesophagectomy is necessary. Handling of the thyroid gland and strap muscles has been discussed.

The trachea is dissected circumferentially at the level of proposed division, taking care not to destroy blood supply to the distal trachea. Lateral traction sutures of 2-0 Vicryl are placed on either side through the full thickness of the tracheal wall, one ring below the anticipated level of division, and the trachea is then divided. Intubation is carried out across the operative field with a flexible armored tube. The distal margin is examined by frozen section. The proximal end of the trachea is retracted with Allis forceps to elevate the esophagus. If anterior exenteration only is being done, resection is carried upward on the preesophageal plane to remove the specimen en bloc. Localized invasion of the esophageal wall may be excised, either with muscularis alone or full thickness, as indicated. If the upper esophagus only is to be removed with the specimen, dissection is carried distally into the mediastinum, as far as is desired, before esophageal transection and closure. If the colon is to be used for replacement of the esophagus, there is no reason for a total esophagectomy. The entire specimen, including the trachea and esophagus, is elevated from the prevertebral fascia and dissected to its cervical attachments (see Figure 8-9 in Chapter 8, "Secondary Tracheal Neoplasms"). If the stomach is used for reconstruction, the entire esophagus is resected by completing the dissection transhiatally from below, facilitated by Pinotti's maneuver of dividing the diaphragm from the hiatus forward to the sternum.

Proximally, the hyoid bone and epiglottis are usually removed with the larynx. A residual epiglottis can interfere with swallowing. Muscles attaching to the superior margin of the hyoid bone are divided, as described for the laryngeal release procedure (see Figure 24-19 in Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). The posterior cornua of the hyoid bone may be divided and

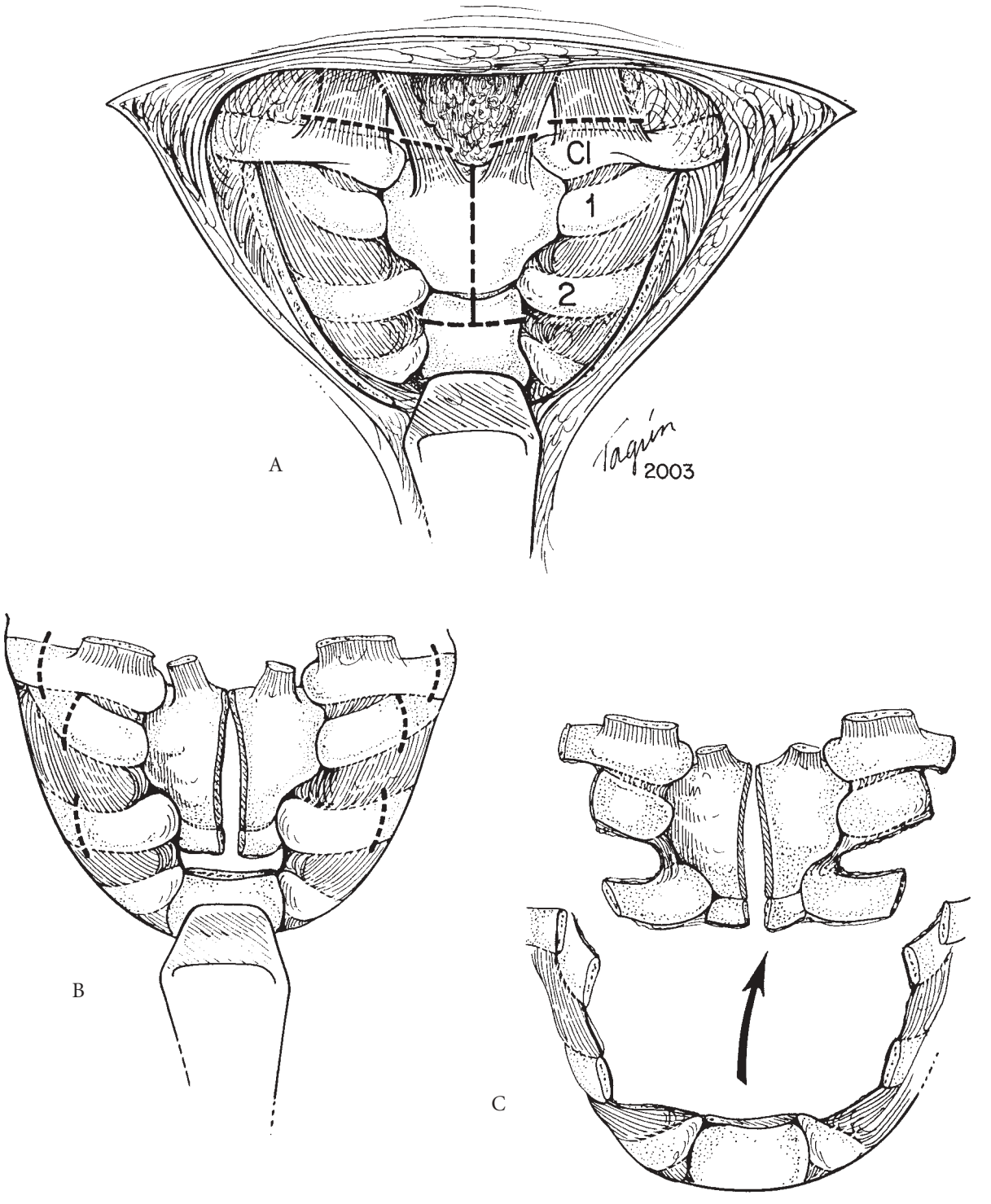
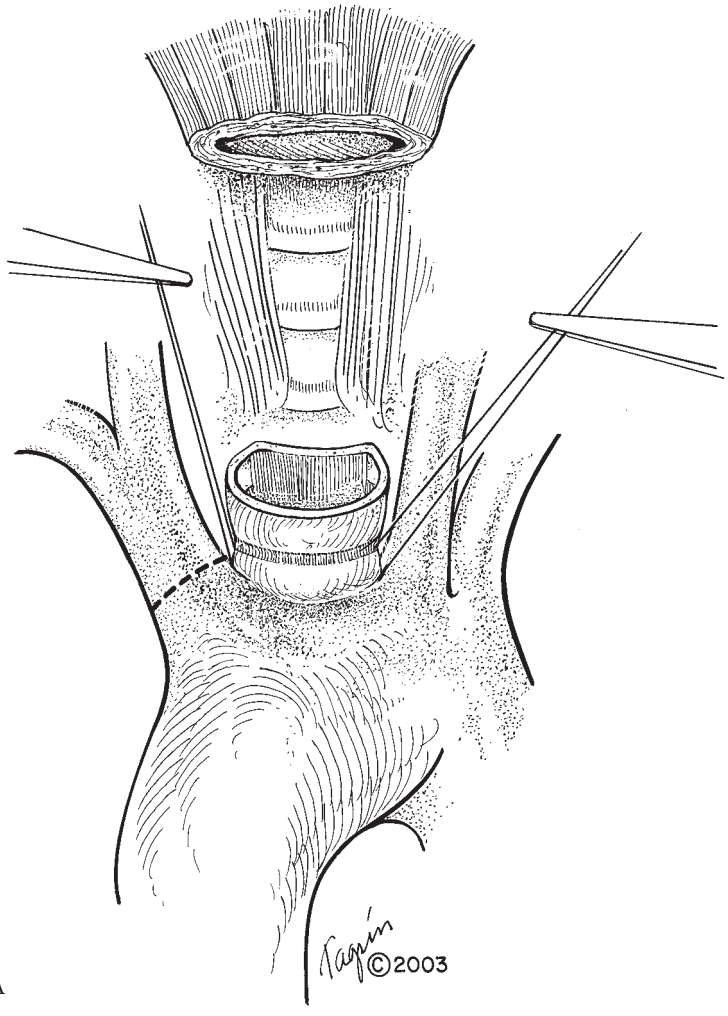


FIGURE **34-2** *Exposure of the operative field and exploration.* A, *The upper skin flap has been elevated to the hyoid bone. Cervical exploration is completed first to determine operability in that region. The lower flap is then elevated to below the sternal angle. The pectoralis muscles are reflected as shown.* B, *The sternum is divided vertically through the manubrium and across the second interspace, allowing full exploration of the mediastinal trachea and extent of involvement. For succeeding steps, the sternocleidomastoid muscles are divided from the clavicles and medial heads from the sternum.* C, *The plaque of sternum, which was divided for exploration, is removed with the medial heads of the clavicles and the first two costal cartilages on either side. The already divided sternum facilitates removal of bony segments.*

left in place. The lateral musculature of the larynx is divided and the pharynx entered just above the epiglottis and on either side above the hyoid bone. Sutures are placed to mark the mucosa—in the anterior midline, laterally, and, finally, posteriorly above the cricopharyngeus (Figure 34-3*A*). The proximal extent of tumor sometimes may be visualized directly through the opened pharynx.

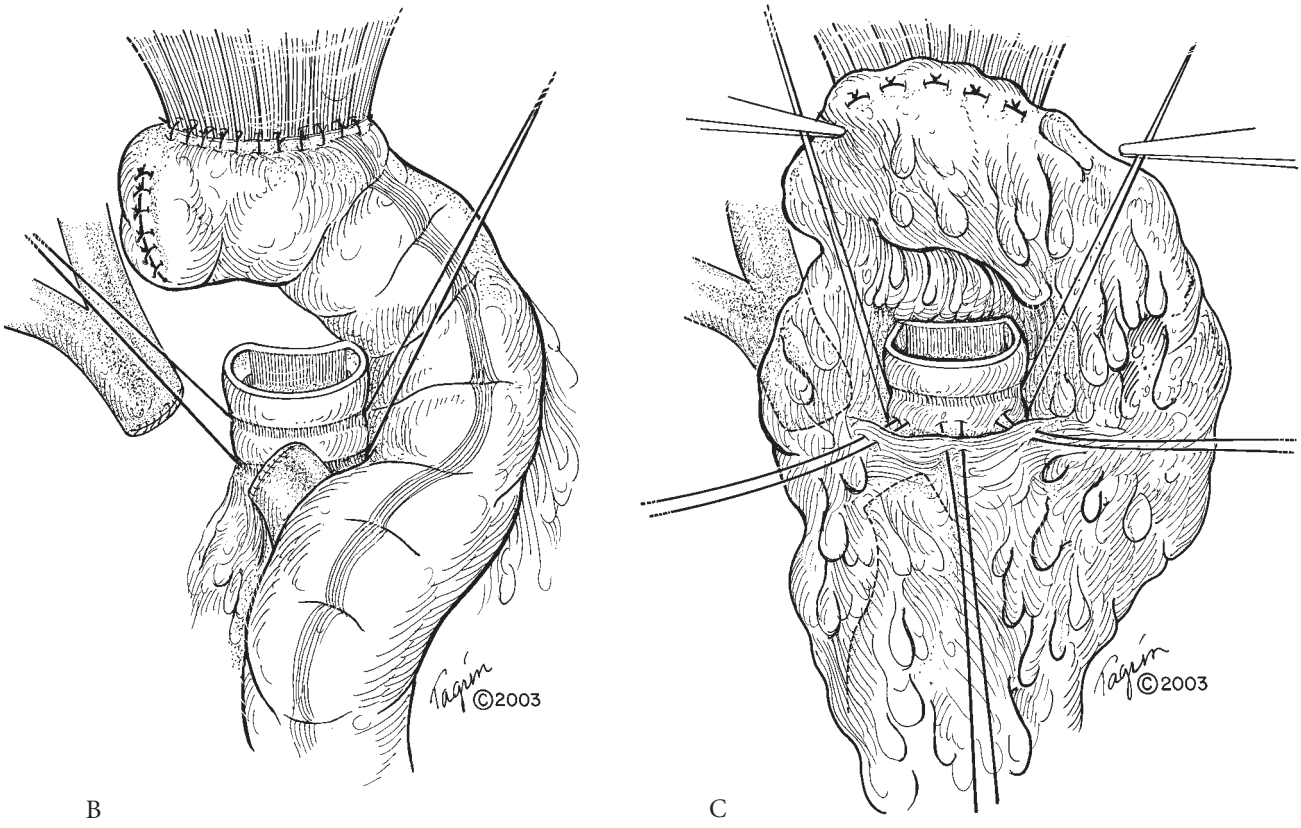
A substernal tunnel must be made wide enough to permit passage of the esophageal substitute and omentum. In making the tunnel, it is better to keep the hand to the left of the midline over the heart. This helps to avoid herniation of the colon into the right chest and possibly redundance of the colon bypass later. It also minimizes the occurrence of a right pneumothorax during dissection.

Once it is clear that the operation can proceed, a second team commences the abdominal portion of the procedure. The omentum is mobilized from the transverse colon, preserving all of its components, since it will be transferred into the neck. If the colon is to be used for reconstruction, the omentum is usually pedicled on the right gastroepiploic artery in order to reach the pharyngeal level (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation"). If the stomach is used for reconstruction of the esophagus, the omentum remains attached to the greater curvature and will be transferred upward with the stomach. The abdominal team is charged with providing an adequate length of omentum and an adequate segment of well-vascularized gut for reconstruction of the esophagus. Even



A

FIGURE **34-3** *Reconstruction of the gastrointestinal tract and fashioning of mediastinal tracheostomy.* A, *The operative field after resection of the larynx, trachea, thyroid gland, tumor, and involved lower-most pharynx and upper esophagus. Contents of the neck between the carotid sheaths down to the prevertebral fascia have been removed en bloc. The lateral traction sutures in the distal residual trachea are shown. Intubation is done across the operative field. For a very short tracheal stump, the innominate artery is electively divided and sutured closed following appropriate pre-operative studies and with electroencephalogram monitoring. Four sutures are placed in the pharyngeal end for alignment of anastomosis.*



B C

FIGURE 34-3 (CONTINUED) B, *Reconstruction beginning with the left colonic segment anastomosed widely to the pharynx. The curve of the colon is such that anastomosis is usually made end-to-side, although end-to-end is occasionally possible. The stomach is used frequently. Either is advanced substernally.* C, *The omentum has been advanced substernally and arranged to cover pharyngogastric or colonic anastomosis, to protect sutured ends of the innominate artery or that artery in continuity, and is also sutured to the emerging stump of trachea one ring below transection. The tracheal stump is passed through an aperture made in the omentum. Traction sutures are in place for ease of manipulating the tracheal stump. See the text for description of advancement of the omentum.*

where the entire esophagus is removed transhiatally, I prefer to advance the esophageal replacement substernally. It is a shorter route for the colon and, in the case of the stomach, permits the bulky omentum to be advanced easily with the stomach.

Reconstruction

The level of the mediastinal tracheostomy must be determined. The end of the distal trachea is gently drawn upward in the mediastinum, using the lateral traction sutures. If it is not well above the brachiocephalic artery, elective division of the artery will have to be considered (see Figures 34-3*A*,*B*). This is not done lightly, but the hazards of erosion of the artery are great if the stoma lies at the last few centimeters of trachea above the carina. The surgeon should have obtained information about the patency of the carotid and vertebral arteries preoperatively. If it appears that the brachiocephalic artery might be endangered by proximity of the stoma, it is preferable to divide the artery. The brachiocephalic artery is temporarily occluded atraumatically and the electroencephalogram (EEG) is carefully observed over a period of 20 minutes. If there is no change in EEG pattern, it is usually safe to divide the artery. The artery is never ligated, but rather is divided, and the proximal and distal ends oversewn with two layers of vascular suture

material. It is essential that the carotid–subclavian communication be intact on the distal side. In numerous cases of emergency and elective division of the artery, we have not seen neurological damage.[8,9] One patient suffered from faintness following division when he stood up quickly, but this passed with time. Another patient, after division, complained of transient weakness in the right arm, when used for heavy work. F. G. Pearson (personal communication) reported a young patient who had transient neurological sequelae following brachiocephalic artery division.

The substernal colon or stomach is delivered to the left of the tracheal stump and pharyngoenteric anastomosis is performed (Figure 34-3*B*). In the stomach, an appropriate linear incision is made, which is adequate for a broad anastomosis. The end of a colon segment may be used, but if it does not lie in easy apposition to the pharynx, the end is closed with staples, oversewn with interrupted 4-0 silk sutures, and the pharynx anastomosed to the side of the colon. Anastomosis is done with two layers of 4-0 Vicryl or silk sutures, using great care and precision in the placement of each suture. A posterior layer of sutures is placed first, prior to opening the replacement organ. After all posterior seromuscular wall sutures have been placed and tied, the replacement organ is opened and the full thickness of the mucosa and muscular wall of either the colon or stomach is sutured to the mucosal layer only of the pharynx. This inner layer is completed anteriorly with inverting sutures and then the anterior seromuscular outer layer of sutures is placed. With precise anastomotic technique and omental buttressing, leakage is unlikely, even where the proximal end has received high-dose irradiation in the remote past. The key here is that the distal side of the anastomosis and the omentum are unirradiated.

The omentum is delivered from beneath the sternum and spread out. If the stomach or colon and omentum are first placed in a flexible plastic bag (such as that used for sterilization of laser equipment), it greatly facilitates their delivery through the substernal tunnel. The omentum is divided into two tongues, taking care not to injure its blood supply. One is sutured against the pharyngoesophageal closure, or around the pharyngocolonic or pharyngogastric anastomosis, to buttress these sutures lines. The other wraps the trachea circumferentially beneath the stoma (Figure 34-3*C*). The omentum is carefully interposed between the trachea and innominate artery or its divided ends. It is essential that any vascular ends be buried in healthy tissue (see Figure 34-3*C*). The omentum serves several functions in respect to the trachea: it provides buffering between the trachea and the artery or its sutured ends; it provides healthy and unirradiated tissue around the trachea, if the trachea has been irradiated; and it provides a seal, if the tracheal end separates from the skin in whole or in part postoperatively. This has effectively prevented brachiocephalic arterial hemorrhage. The omentum is sutured to the tracheal wall subterminally (see Figure 34-3*C*), leaving enough length for anastomosis to the skin and subcutaneous tissues.

The lower incision for creation of the bipedicled anterior cutaneous flap is made. It runs from the right to the left anterior axillary lines beneath the breasts (see Figure 34-1). The flap is raised from the sternum and the pectoralis muscles laterally, meeting the dissection plane from above. Retraction on either side permits lateral access. The surgical plane lies on the pectoral fascia to avoid injuring the blood supply to the flap. Perforating vessels are encountered and controlled. The fully dissected flap is slid upward and deeply into the mediastinal defect. A point in its midline is selected for emergence of the tracheal stoma. A circle of skin is excised, about equal to the size of the trachea (Figure 34-4*A*).

The subcutaneous tissues of the flap at the margin of the stoma are sutured to the tracheal wall, just below the cut end of the trachea and above the ring of omentum. These may be placed from beneath the flap and tied by palpation. The lateral traction sutures draw the tracheal stoma through the cutaneous opening and the subcutaneous sutures are tied. The endotracheal tube may be successively removed for convenience as the cutaneous anastomotic sutures are next placed. 4-0 Vicryl sutures pass through the skin margin and then through the full thickness of tracheal wall. Four sutures—one anterior, one posterior, and one at each lateral margin—are initially placed to align the anastomosis. Three to four sutures are placed

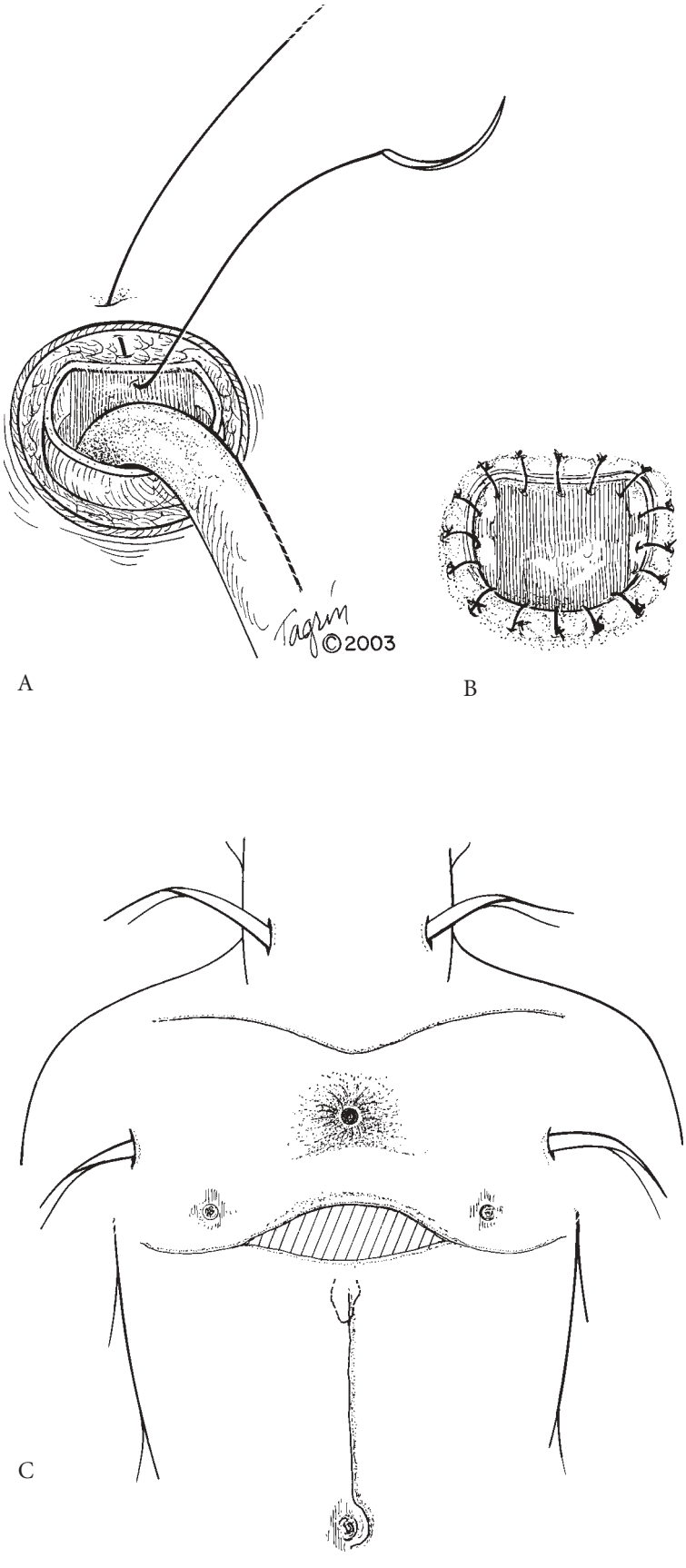


FIGURE **34-4** *Completion of tracheostomy.* A, *Tracheostomy is implanted in a carefully-made circular excision in the skin flap. Anastomotic sutures are placed circumferentially between the skin and mucosa of the trachea, as described in the text. Subcutaneous sutures to the trachea are sometimes also used. The lateral tracheal stay sutures are removed as the tracheocutaneous sutures are placed.* B, *The completed stoma.* C, *The completed reconstruction. The central defect below the flap is covered with a split-thickness skin graft overlying intact chest wall. An attempt to close the skin primarily is likely to place undue tension on the margins of the mediastinal tracheostomy. Subcutaneous, cervical, and mediastinal drains are shown.*

between each of these four sutures to surround the stoma. The mucosa is accurately approximated to the skin (Figure 34-4*B*).

Multiple flat suction drains are placed through lateral stab wounds to drain the neck, mediastinum, and subcutaneous spaces. The upper incision is closed using subcutaneous and subcuticular sutures or skin sutures. Usually, the upper incision can be closed without tension. Slight duskiness may be seen in the skin around the stoma at this time. It will almost invariably recover, provided there is no tension.

The lateral parts of the inferior flap incision may be sutured closed, but the central defect should be allowed to remain unstretched even if it seems possible to pull the gap together. This could place too much tension on the mobilized flap. The cutaneous gap overlies healthy lower anterior chest wall, well below the sternal defect. It is closed with a split-thickness skin graft (Figures 34-4*C*, 34-5).

If the vagus nerves are divided, pyloroplasty or pyloromyotomy is added. A feeding jejunostomy is placed. If colon bypass has been used and the stomach remains in the abdomen, a draining gastrostomy is also placed. If transhiatal esophagectomy has been done, a posterior mediastinal drain is brought out through the abdomen.

If the patient requires ventilation postoperatively, a flexible armored tube is placed in the trachea and carefully taped to the skin. Cuff pressure is minimal. The angle of a tracheostomy tube is inappropriate for a mediastinal tracheal stoma. Every effort should be made to discontinue ventilation as soon as possible, since the pressure from the cuff and the presence of the tube may interfere with healing of the proximal trachea. Humidity via a tracheotomy mask is necessary.

If a significant amount of anterior chest wall skin has necessarily been excised, as in the case of stomal recurrence of laryngeal carcinoma, either rotational or island musculocutaneous flaps must be planned. These are individually designed for each patient. The mediastinal tracheostomy will emerge through the flap.

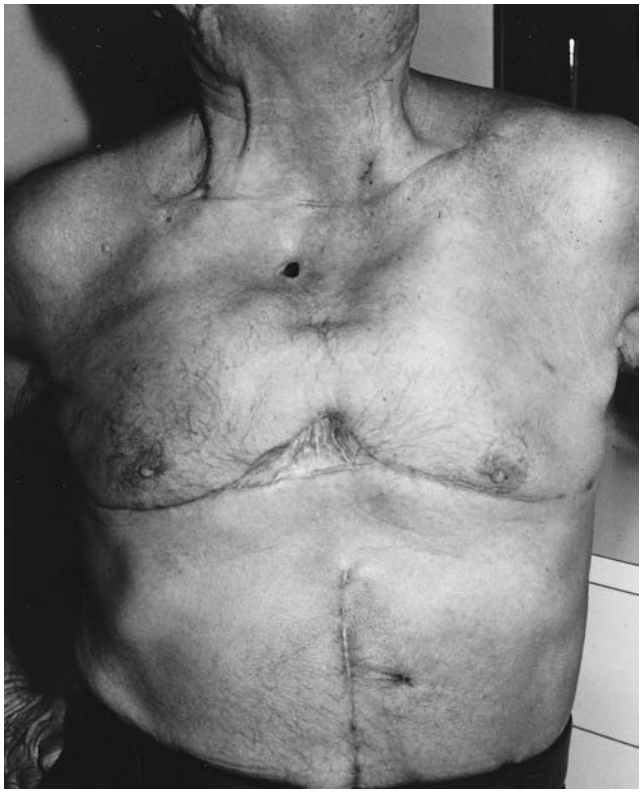


FIGURE **34-5** *Final result after resection of the larynx, trachea, esophagus, thyroid, plus left cervical node dissection in a 69-year-old man. The brachiocephalic artery was divided, the left colon was used for esophageal reconstruction, an omental pedicle was advanced, and mediastinal tracheostomy was then established. The tumor was an unusual squamous cell carcinoma of the thyroid, with negative lymph nodes. The defect from upper sternectomy, resection of clavicular heads, and the first two cartilages is visible, with the end tracheostomy to the right of the midline. The skin graft has contracted. The patient learned effective speech with an electrolarynx, and continued to serve as a moderator in town meetings. He died 3 years later from coronary artery disease, without recurrence of tumor.*

Results

Since cervicomediastinal exenteration and mediastinal tracheostomy describe procedures rather than disease entities, a brief review of results of the operation is in order. Of 18 patients reviewed, who required mediastinal tracheostomy, 5 had prior failed operations and 11 had received irradiation of between 5 and 6,000 cGy.[9] Seven had elective division of brachiocephalic artery (under EEG monitoring). Colon bypass was used in 10, the stomach in 3, and a reverse gastric tube in 1 patient. In 4 patients, portions of the esophageal wall were removed. Omentum was used in 11 individuals.

In 10 cases, there were no complications; 2 esophageal leaks healed on drainage. Stomal separation was managed conservatively in one and in a second by pectoral flap. Hemiplegia occurred only where the carotid subclavian bifurcation was resected and this was promptly managed successfully by vascular bypass. One patient died from necrosis of the reversed gastric tube. Five required later stomal revision. Many of the earlier patients did not have circular excision of skin for their stomas. Survival was disease dependent. Palliation of esophageal and tracheal obstruction was achieved in all.[9]

Despite the magnitude of the procedure, cervicomediastinal exenteration does provide gratifying palliation in carefully selected patients. Meticulous attention to the host of details recorded above is essential in order to obtain satisfactory results and to avoid disastrous complications.

References

1. Waddell WR, Cannon B. A technique for subtotal excision of the trachea and establishment of a sternal tracheostomy. Ann Surg 1959;149:1–8.
2. Sisson GA, Straehley CJ Jr, Johnson NE. Mediastinal dissection for recurrent cancer after laryngectomy. Laryngoscope 1962;72:1064–77.
3. Grillo HC. Terminal or mural tracheostomy in the anterior mediastinum. J Thorac Cardiovasc Surg 1966;51:422–7.
4. Grillo HC, Mathisen DJ. Cervical exenteration. Ann Thorac Surg 1990;49:401–9.
5. Mathisen DJ, Grillo HC, Vlahakes GJ, Daggett WM. The omentum in the management of complicated cardiothoracic problems. J Thorac Cardiovasc Surg 1988; 95:677–84.
6. Shrager JB, Wain JC, Wright CD, et al. Omentum is highly effective in the management of complex cardiothoracic surgical problems. J Thorac Cardiovasc Surg 2003; 125:526–32.
7. Orringer MB. Anterior mediastinal tracheostomy with and without cervical exenteration. Ann Thorac Surg 1992;54;628–37.
8. Brewster DC, Moncure AC, Darling RC, et al. Innominate artery lesions: problems encountered and lessons learned. J Vasc Surg 1985;2:99–112.
9. Madsen JC, Mathisen DJ, Grillo HC. Cervical exenteration. Semin Thorac Cardiovasc Surg 1992; 4:292–9.

Laryngologic Problems Related to Tracheal Surgery

Robert H. Lofgren, MD

Anatomy and Function

The larynx extends from the epiglottis to the lower margin of the cricoid cartilage, and includes the epiglottis, the thyroid cartilage, the cricoid cartilage, and the arytenoids. The thyroid cartilage sits on top of the cricoid cartilage, which is signet ring-shaped with the broader margin posteriorly. The arytenoids sit on either side in the superior border of the posterior cricoid lamina, forming the cricoarytenoid joints. From the anterior medial border of each of the arytenoids arises the vocal process, the point of insertion of the vocalis muscle, which forms the muscular substance of the vocal cords. The glottis (also known as "glottic chink") refers to the space between the vocal cords.

The intrinsic muscles of the larynx basically function as antagonists to each other (Figure 35-1). The cricothyroid muscle lengthens and tenses the vocal cord, whereas the internal thyroarytenoid muscle shortens and abducts the vocal cord. The external thyroarytenoid muscle shortens and adducts the vocal cord. The posterior cricoarytenoid muscle abducts the vocal cord, whereas the lateral cricoarytenoid muscle adducts the cord. The arytenoid muscle, which lies in the space between the arytenoids posteriorly, closes the posterior commissure and thus adducts the cords. All muscles are innervated by the recurrent laryngeal nerve, except for the cricothyroid muscle, which is innervated by the external branch of the superior laryngeal nerve. The internal branch of the superior laryngeal nerve provides sensory innervation for the larynx.[1]

The larynx has three main functions; the most basic is respiration. The posterior cricoarytenoid muscle abducts the vocal cords, allowing a clear flow of air through the larynx. This is the "normal" resting position of the vocal cords. The second important function of the larynx is protection of the airway, with adduction of both the true and false vocal cords acting as a laryngeal sphincter closing the airway while swallowing. In addition, with swallowing, the larynx rises up under the epiglottis which is deflected downward to deflect food and liquid off into the pyriform sinuses away from the laryngeal introitus. The third function, which is not nearly so basic but important for humans, is phonation, the production of sound. Phonation is produced by exhaled air interacting with the two nearly opposed vocal cords, whose tension is

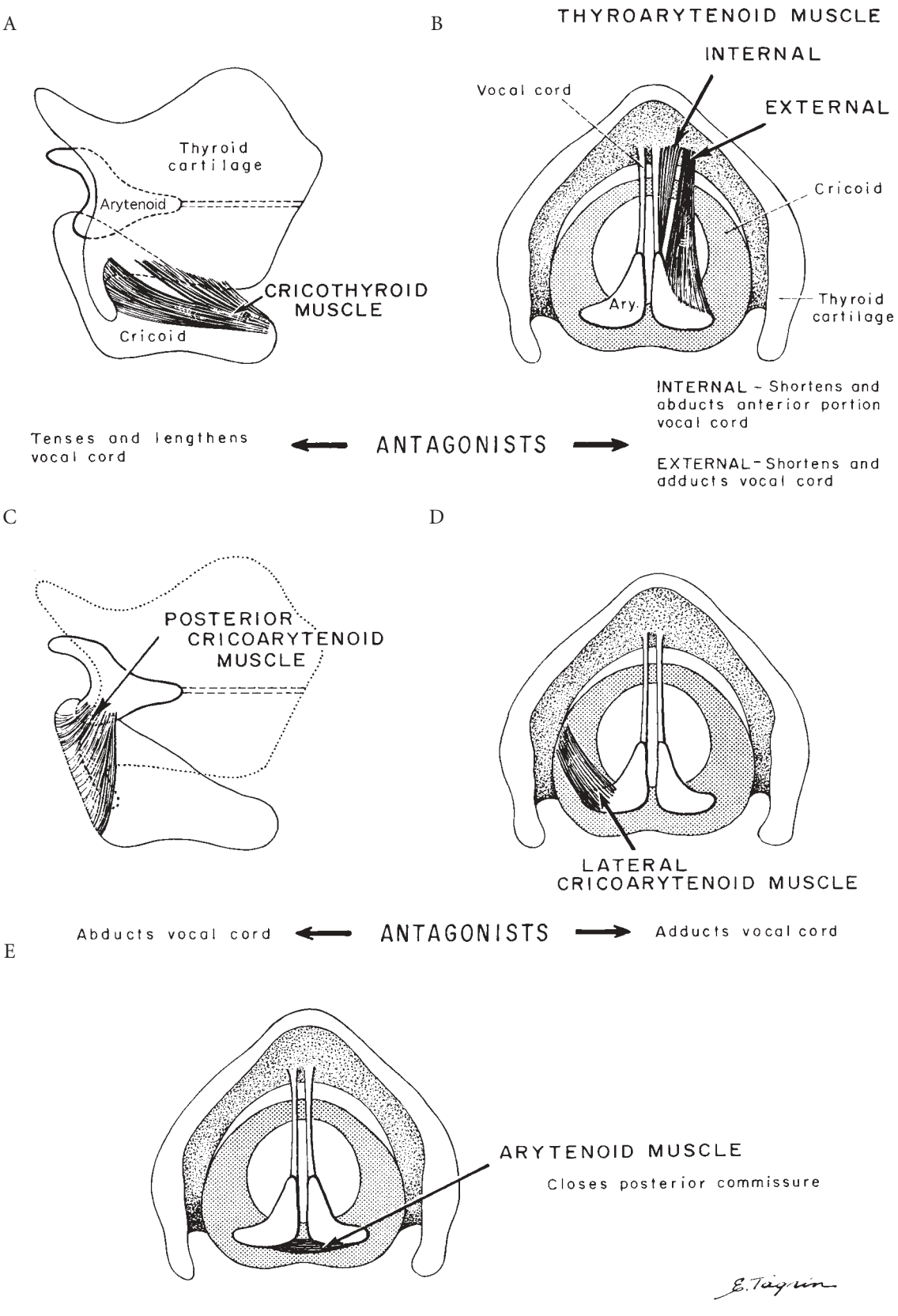


FIGURE 35-1 *The intrinsic muscles of the larynx function as antagonists to each other.*

controlled by the laryngeal intrinsic muscles. Speech itself is articulated by the mouth, tongue, lips, and teeth, and resonates in the throat, nose, and mouth.

Assessment

It is very important that the larynx be functioning adequately before tracheal reconstruction surgery is attempted. Tracheal stenosis resection and primary reanastomosis can be accomplished without a tracheostomy in a setting of an adequate laryngeal airway. Such complex tracheal surgery can only be successful in the setting of a patent larynx. Adequate evaluation of the larynx for stenosis and/or paralysis and correction of any problem is essential if subsequent tracheal surgery is to be performed successfully.

Evaluation of the larynx should be done prior to any procedure that might involve damage to recurrent laryngeal nerves. The patient should always be questioned as to hoarseness after previous surgery, especially thyroid surgery or cardiothoracic surgery. A normal voice at the time of admission does not preclude a vocal cord paralysis on one side. An otolaryngologist should evaluate any patient who has had previous thyroid surgery, tracheal injury, or upper mediastinal, thoracic, or cardiac surgery before any procedure is done where damage to a recurrent laryngeal nerve is possible. It is quite common to find a paralyzed cord in a person with a normal voice who has had a thyroidectomy years before and who, on close questioning, does remember hoarseness for 4 to 6 weeks after the surgery, but then the voice recovered. If only one recurrent laryngeal nerve is functioning, then damage during subsequent surgery to the remaining nerve will result in bilaterial vocal cord paralysis, causing glottic level obstruction. Laryngeal examination is usually performed quite easily by indirect mirror examination of the vocal cords. In patients with a strong gag reflex, anatomic variation, or when a prolonged view is necessary, a fiber-optic laryngoscopy is performed. This preoperative, awake laryngeal examination provides an assessment of vocal cord function, information which is of extreme importance in surgical planning but is not available during endoscopic evaluation under anesthesia.

Unilateral recurrent laryngeal nerve injury typically causes the affected vocal cord to rest in the paramedian position (not fully abducted or fully adducted) although the exact position varies. In this position, the glottic airway is not significantly narrowed, and airway complaints are rare; however, the opposing vocal cord may not be able to provide adequate glottic closure during phonation. This small remaining glottic gap during phonation results in air escape and a weak and breathy voice. The vocal surfaces are not irregular and the voice is not truly hoarse. Effective cough, which requires transient, tight glottic closure, can not be mounted. The degree of vocal change depends upon the exact vocal cord position and the degree of opposite vocal cord compensation. Unilateral vocal cord paralysis can be associated with significant aspiration depending upon the exact vocal cord position, as the paralytic cord is unable to protect its half of the glottis from saliva or ingested material, especially liquids. The degree of symptoms (breathy voice, ineffective cough, and aspiration) varies with the degree of injury (paresis vs complete paralysis), exact position of the paralyzed vocal cord, and the degree of compensation from the opposite vocal cord. Bilateral vocal paralysis causes a very different constellation of symptoms as compared with unilateral paralysis.

The bilaterally denervated vocal cords typically come to rest in the midline, demonstrating very little abduction ability. Voice is usually good with this glottic configuration, but respiratory function is compromised. Such patients can present with respiratory distress in the recovery room after bilateral thyroidectomy and may require urgent tracheostomy. Unrecognized respiratory distress without change in voice, in a patient with bilateral vocal cord paralysis, can result in hypoxia, respiratory arrest, brain anoxia, and death. Bilateral vocal cord paralysis is usually caused by bilateral thyroid surgery but it can also occur as a result of neurologic events or neck trauma. Intraoperative recurrent laryngeal nerve electromyography (EMG), during a thyroidectomy and other surgeries that have risk to the recurrent laryngeal or vagus nerves, aides the surgeon in locating the nerve and allows the surgeon to assess the functional integrity of

the nerve at the end of surgery.[2] This type of monitoring allows the surgeon to defer contralateral surgery if there is loss of significant EMG activity during evoked stimulation of the operated nerve on the first side.

In problem cases where a laryngeal abnormality is suspected, or a high tracheal lesion exists, it is usually helpful to do direct laryngoscopy at the same time that the thoracic surgeon is performing a diagnostic bronchoscopy. At this time, the arytenoids can be palpated to see if they are fixed or if there is actually cord paralysis. Both the thoracic surgeon and the laryngologist can also more carefully evaluate the subglottic region. For this procedure, we usually use the Holinger laryngoscope with the Lewy suspension. The subglottic area can be evaluated by passing a zero degree or angled telescope through the laryngoscope, through the cords into the subglottic larynx and upper trachea, providing a high-resolution image of this area. This procedure is performed under general anesthesia, administered through the tracheostomy tube if one is in place, or through a small (5 or 5.5 mm) endotracheal tube that will lie in the posterior commissure and not obstruct vision. A quick look can be obtained without intubation if the anesthetized patient is breathing spontaneously. For further evaluation of the larynx, radiography, using the techniques discussed in Chapter 4, "Imaging the Larynx and Trachea," is very helpful. The laryngologist and the thoracic surgeon should always personally review the x-rays preoperatively, and not just depend on the report. This is essential in putting the whole picture together.

The most important decision for the laryngologist to make preoperatively is the adequacy of the larynx. In my experience, a 4 mm glottic chink is about the minimum that can safely support survival. This is measured posteriorly and represents the widest aperture between the cords. One can get by temporarily with a 2 to 3 mm glottis, but this does not allow for the development of any laryngitis or edema with upper respiratory tract infection. Even a 4 mm glottis will often become inadequate if an endotracheal tube is passed and left in place for more than a very brief period of time, or if the surgery is in the upper trachea or subglottic area where there can be interference with the lymphatic drainage of the vocal cords. High tracheal anastomosis or tracheal-thyroid cartilage anastomosis is more likely than lower tracheal work to induce unforgiving laryngeal edema. Even in the setting of a preoperatively adequate laryngeal airway, other problems to be found are local laryngeal lesions such as large polyps, tumors of the larynx, acute laryngitis, or edema as seen in inhalation burn injuries. When one sees an apparent cord paralysis (ie, better described as vocal cord immobility), one has to look carefully to decide if this is indeed paralysis or rather vocal cord fixation from, for example, cricoarytenoid arthritis or some other cricoarytenoid process. In both (paralysis and fixation), the arytenoids do not move. It is important to distinguish which is the underlying true cause, as treatment differs significantly. Cricoarytenoid arthritis may be a form of osteoarthritis from trauma related to either external injury or internal injury from intubation.[3] It may also be part of the generalized picture of rheumatoid arthritis.[4] Occasionally, it is seen as the result of long standing vocal cord paralysis. Other forms of cricoarytenoid dysfunction, such as joint ankylosis secondary to trauma, may frequently occur in a patient with airway stenosis. If the patient will tolerate a careful examination, then with arytenoid fixation, one can usually see motion of the midportion of the vocal cord in the appropriate direction; that is, laterally on inspiration and medially on phonation. With true paralysis, motion of the cord is paradoxical, with the cord drawn medially on inspiration and pushed laterally on expiration and phonation. Videostroboscopy may also be helpful in this evaluation. Usually, an indirect examination is inadequate to give a definite diagnosis, and one must do palpation at direct laryngoscopy to be sure. Bilateral vocal cord paralysis often coexists with other laryngeal pathologies. The injury that gave rise to the paralysis may also give rise to glottic level stenosis. Anterior or posterior commissure stenosis may be seen. Generally, in adults, glottic and subglottic stenoses result from traumatic insult to the airway, often from endotracheal tube intubation. Thus, the cartilaginous dimension of the cricoid and thyroid cartilage are often not the source of the stenosis; rather, the stenosis arises secondary to submucosal scarring, in distinction to pediatric glottic and subglottic stenoses.

Anterior Glottic Stenosis

Anterior glottic stenosis is usually secondary to laryngeal injury anteriorly, or laryngeal surgery where the surgeon has denuded the mucosa from both the right and left vocal cords into the anterior commissure in removal of polyps. The cords will then heal together, forming a web (Figure 35-2*A*). Anterior glottic webs, which are thin and short, can often be treated with laser division of the web. More significant webs, and those that recur after laser division, are best treated by doing a laryngofissure procedure, dividing the web and inserting a laryngeal keel (Figure 35-2*B*), a T-shaped piece of silicone that is sutured in place so that the long arm of the "T" keeps the two raw edges of the vocal cords separated until they heal. The keel is removed after a period of approximately 2 weeks, by which time the cord should have epithelialized.[5]

Posterior Glottic Stenosis

Posterior glottic stenosis is usually due to intubation, when an endotracheal tube lies in place too long or is too large a caliber, especially if the patient is awake and swallowing, because the natural point of maximum pressure of an oral endotracheal tube is at the posterior commissure of the larynx. Often, this problem is combined with a subglottic stenosis due to insertion of the too large endotracheal tube. From actual measurements done in anatomy laboratories, it has been shown that one should never use a larger than 6 mm endotracheal tube in a female patient. Many women can take larger than 6 mm, but a significant number have a cricoid lumen that will not permit larger than a 6 mm tube to pass atraumatically through it. Patients who have a prolonged intubation should be placed on medication to minimize gastric acid secretion, in case of gastroesophageal reflux. Reflux can lead to inflammatory changes that would add to those of intubation and help generate stenosis in the glottis and subglottis.

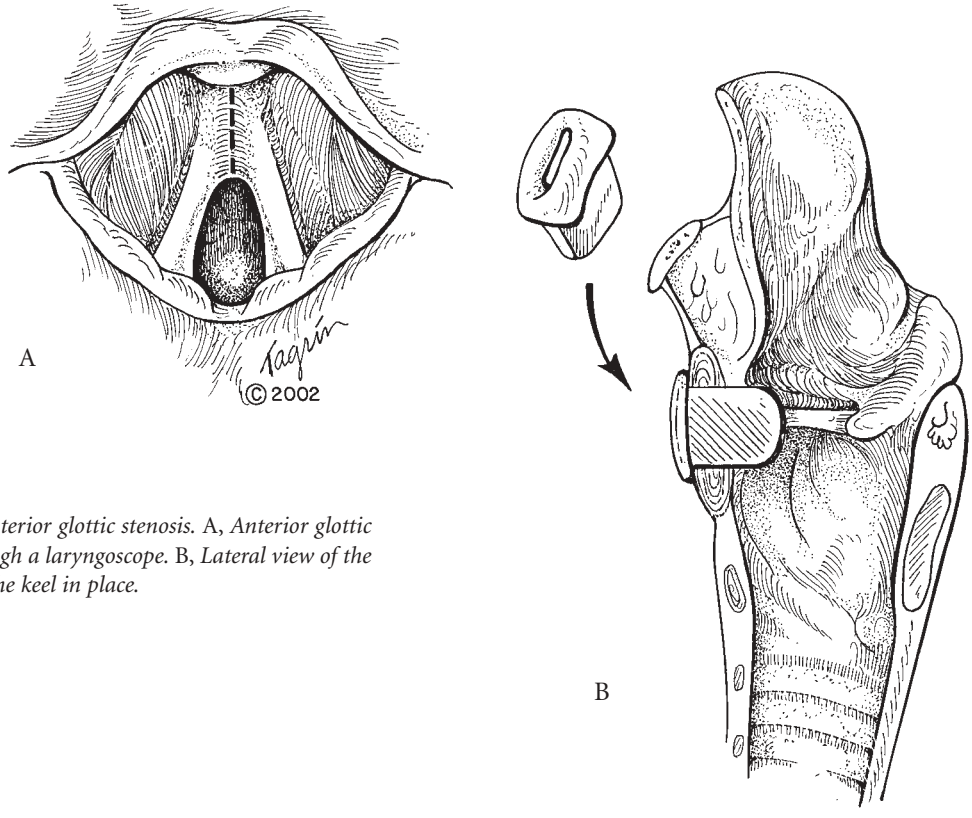


FIGURE **35-2** *Anterior glottic stenosis.* A, *Anterior glottic web as seen through a laryngoscope.* B, *Lateral view of the larynx showing the keel in place.*

Other causes of stenosis are direct external trauma to the cricoid and/or thyroid cartilage and cricothyrotomy. Many advocate doing a cricothyrotomy as an emergency tracheostomy. It is a quicker way into the trachea with less bleeding, as it is closer to the surface, and one does not have to worry about the thyroid isthmus. A cricothyrotomy should always be converted to a standard tracheostomy as soon as possible. If left in place, the tube will erode the cricoid, and often the thyroid cartilage as well, leading to stenosis.

A posterior glottic stenosis is a band of scar tissue between the two arytenoids involving the mucosa and extending into the arytenoid muscle, preventing the vocal cords from abducting normally. It is often difficult to diagnose a posterior glottic stenosis with a mirror or a fiber-optic endoscope. One can be suspicious when the cords move briskly but not widely, as if tethered on a short cord. Usually, on direct laryngoscopy, when the cords are parted with the anterior tip of the laryngoscope, the stenosis can be readily seen as a thickened linear band of tissue between the arytenoids (Figure 35-3*A*). Laser division of this band is occasionally helpful, but usually open surgery is necessary. If a tracheostomy is not already present, one must be done. A laryngofissure is then performed through a horizontal incision over the midportion of the thyroid cartilage. The dissection is carried down to the strap muscles, which are separated in the midline. The perichondrium is incised in the midline. The cartilage is incised in the midline with a knife blade. If the cartilage is ossified, then it is cut with a Stryker saw (Figure 35-3*B*). A button knife is inserted through a small incision in the cricothyroid membrane, dividing the mucosa in the midline. The thyroid laminae are held apart with a self-retaining retractor or with hand retractors held by an assistant. The web is identified visually, and to assess its extent, it is palpated in the area of the posterior commissure and is incised in the midline down through the scar into the interarytenoid muscle until all the fibrotic muscle has been divided (Figures 35-3*C*,*D*). A mucosal flap is then elevated from posteriorly over the arytenoid and the esophageal introitus, and is advanced to cover the raw surface in the interarytenoid space, and then sutured in place with a 4-0 chromic suture on a small half-round needle (Figures 35-3*E*,*F*). This work can be performed with otologic instruments such as a Rosen or McHugh knife and canal wall or drum elevator. A stent is then inserted and the wound is closed. The thyroid laminae are closed by suturing the perichondrium edges together. A drain is inserted and the skin and subcutaneous tissues are closed in the usual fashion. Care is taken throughout this procedure to avoid connecting the laryngofissure dissection with the tracheostomy.

If the patient has a normal subglottis and trachea, then a solid Montgomery conforming laryngeal stent is used.[6] The stent is held in place with a 2-0 nylon suture, placed through and through from externally and tied over plastic buttons (Figure 35-4). Several different size stents are available. This stent is left in place for 4 to 8 weeks and is then removed via a laryngoscope after first cutting and removing the nylon suture. It has the advantage of being a conforming stent and the disadvantage that the patient is totally aphonic while it is in place and totally dependent of the tracheostomy for airway. As the stent is solid, aspiration is prevented, which makes it more useful in patients who would be apt to aspirate, such as older patients and patients who have had a previous superior laryngeal release procedure. If the patient has subglottic disease that is going to be repaired later, or possibly was repaired at the same time, then a Montgomery T tube is inserted, with the upper end coming up through the vocal cords to the level of the laryngeal ventricle. The tube is cut to length and smoothed with a sander or a sterile emery board before it is inserted. The upper end should lie at the level of the laryngeal ventricle. This allows the false cords to close over the tube, preventing aspiration, and the patient is able to talk in a somewhat hoarse voice, phonating with the false vocal cords. In patients who have chondromalacia, granulation tissue, or some condition in the upper trachea that one wants to stent with a larger tube than will fit through the vocal cords, a tapered T tube works well (Figure 35-5). The lower limb is 13 mm in diameter to fit into the trachea, and the upper limb tapers to 10 mm at the level of the vocal cords.

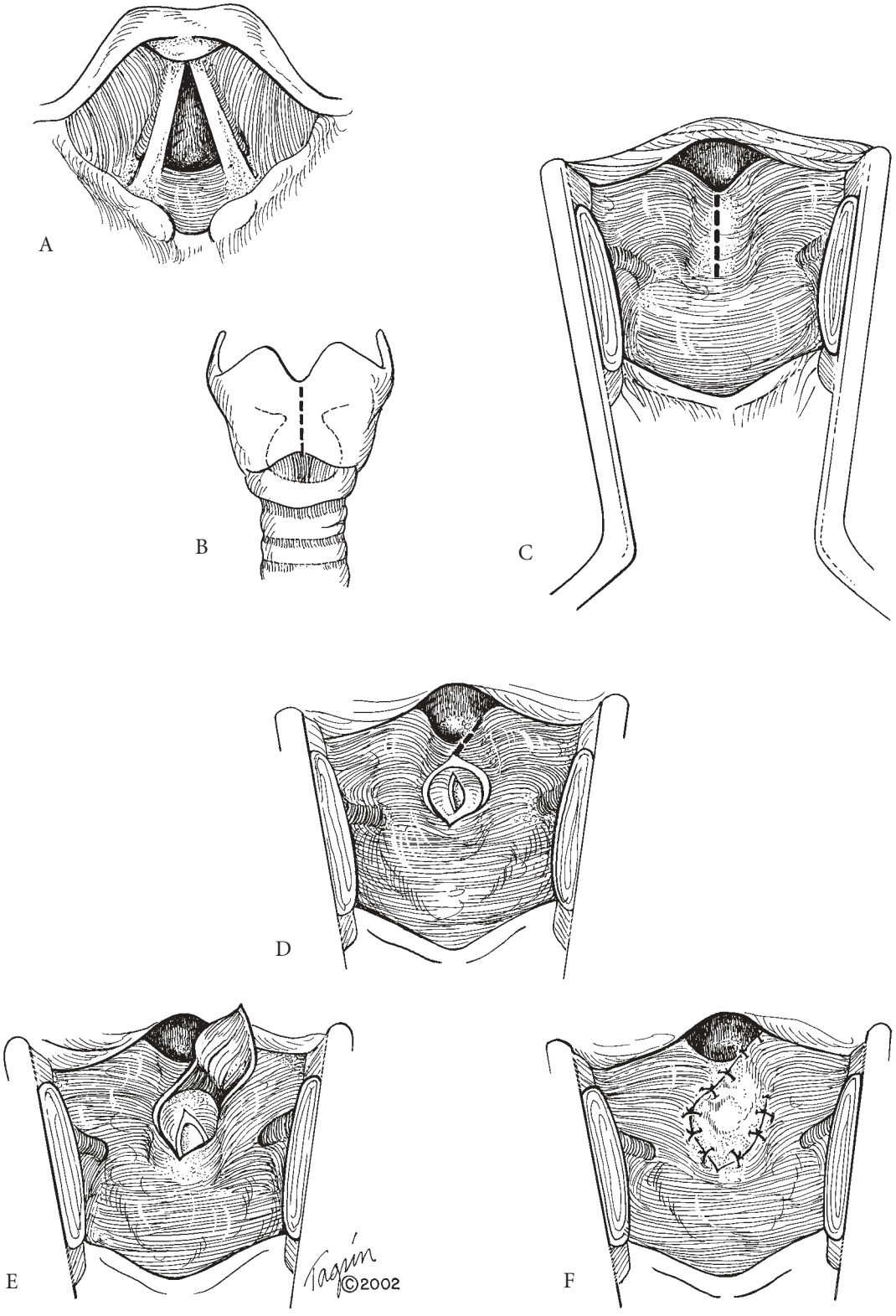
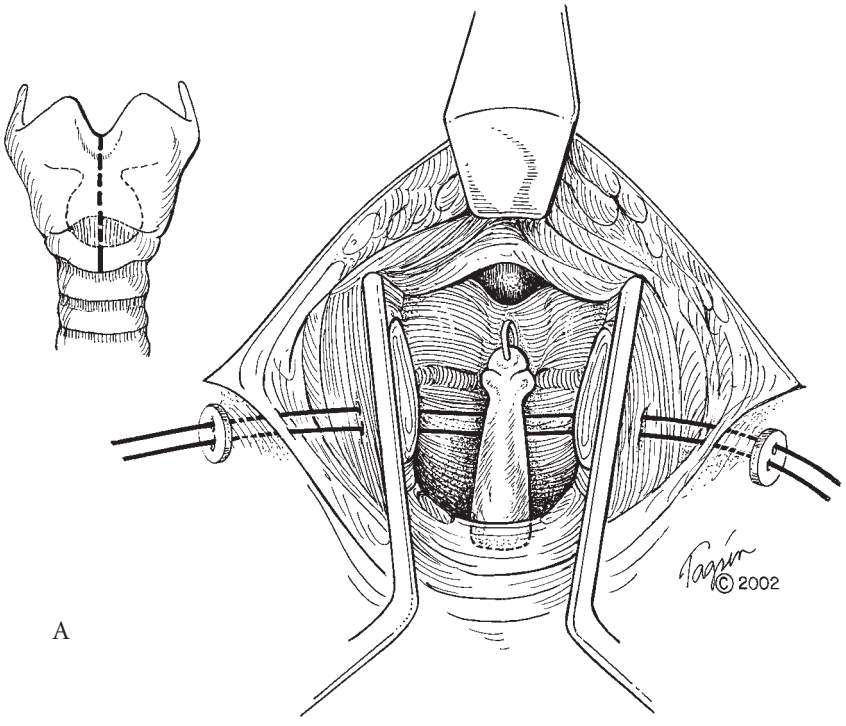


FIGURE **35-3** *Posterior glottic stenosis.* A, *Laryngoscopic view of a posterior band between the arytenoids.* B, *Midline incision in the thyroid cartilage.* C, *Incision of web mucosa.* D, *Incision of scarred interarytenoid muscle. Incision of mucosa for advancement flap.* E, *Mucosal flap elevated.* F, *Mucosal flap advanced and sutured in place.*



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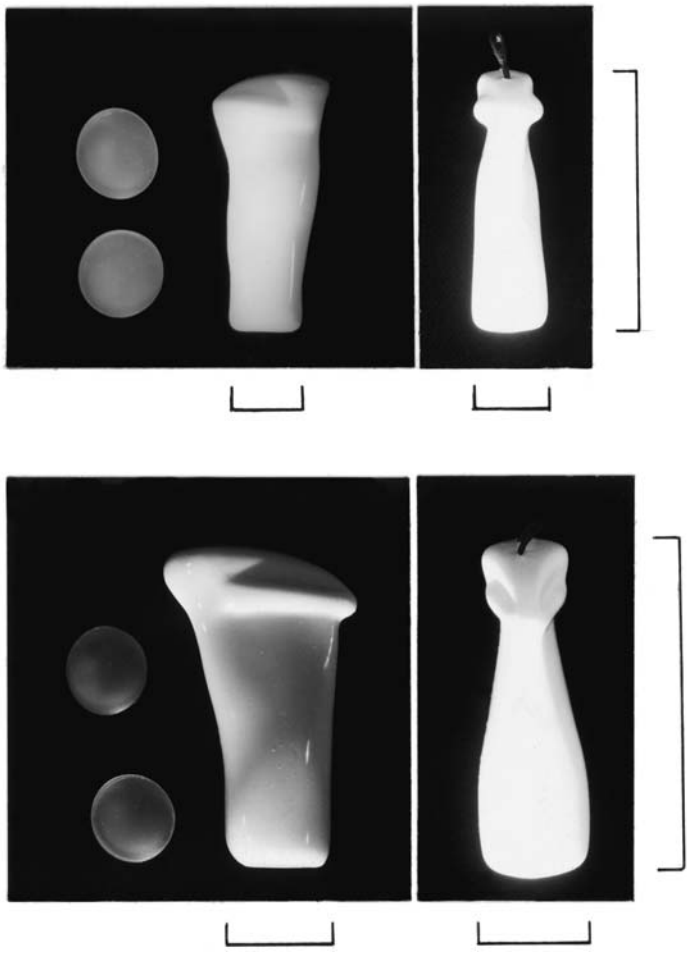

B

FIGURE **35-4** A, *Conforming stent in place with the small bulge into the ventricle between the true and false vocal cords, and the wider lower end through the cricoid ring into the upper trachea.* B, *Small adult (female) and large adult (male) stents. Also available are adolescent and child size stents.*

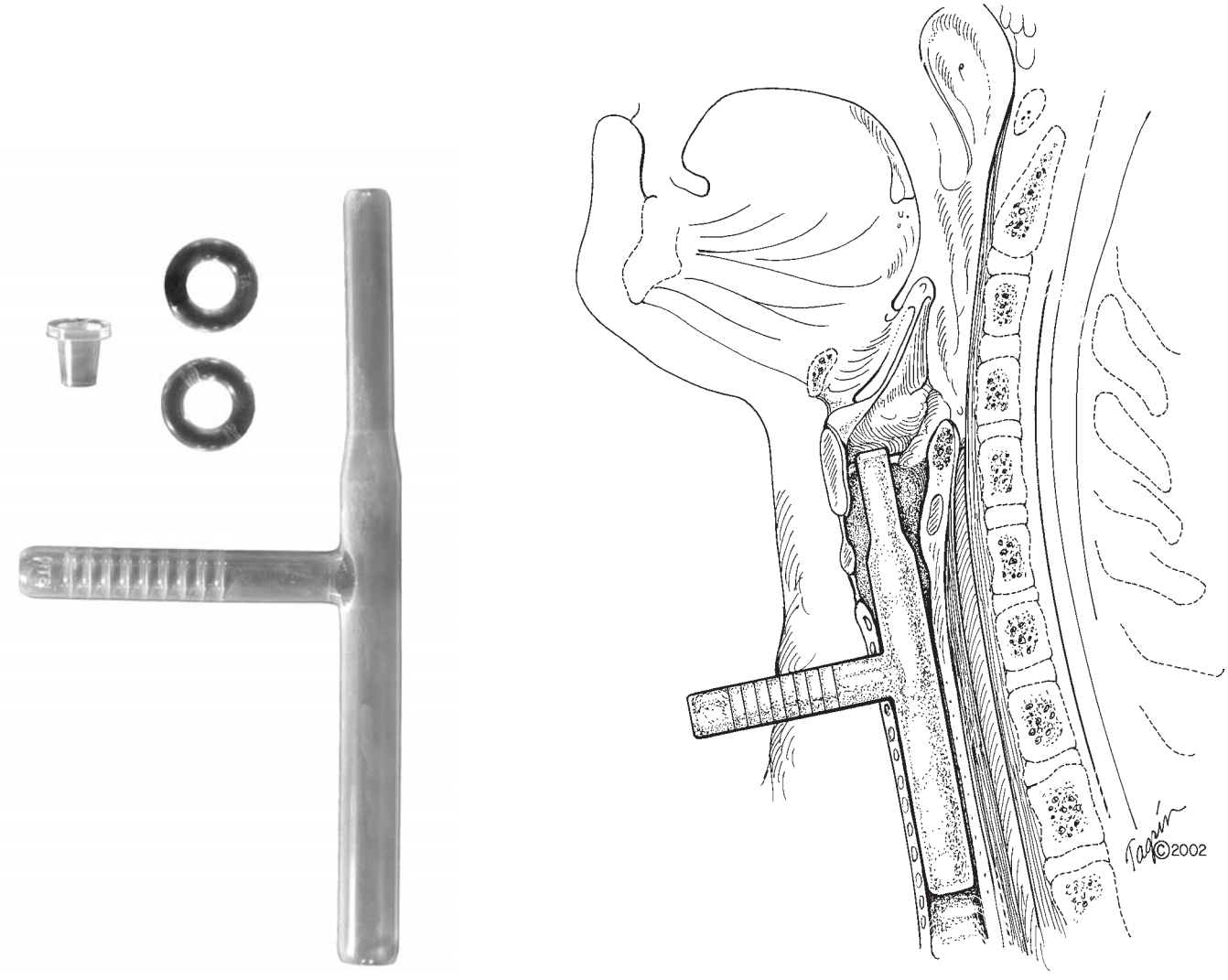

FIGURE **35-5** *Tapered T tube in place. Note the upper end is at the level of the laryngeal ventricle.*

Subglottic Stenosis

Subglottic stenosis due to a fractured cricoid or extensive scarring, often with a small cricoid ring, is treated with a laryngofissure plus cutting the cricoid cartilage anteriorly. A thin and short web-like scar may be treated with a laryngoscopic laser procedure. This rarely works in the typical thick, broad scar where an open procedure is necessary. If the cricoid ring is large, then an incision can be made in the mucosa, the local mucosal flaps raised, and the scar tissue removed submucosally. If the cricoid has thickened calcified cartilage that is obstructive, then this area can be curetted with mastoid and stapes curettes or drilled down with a rotating burr. The mucosal flaps are then replaced and sutured and a stent is inserted. If the cricoid ring is too small, then a second incision is made in the cricoid posteriorly to break the spring of the cricoid cartilage. If additional cricoid diameter is needed, a graft can be placed anteriorly, and if necessary, a second graft can be placed posteriorly. A piece of cartilage can be harvested from the thyroid lamina and inserted between the cut edges of the cricoid, or the center portion of the hyoid bone can be removed with at least one of the sternohyoid muscles still attached for a blood supply and brought down to fit into the anterior cricoid defect. If the posterior periosteum

is left on the hyoid bone, a mucosal graft is not necessary in this area. The hyoid bone has a curved shape that fits naturally into the anterior cricoid arch and works well. Other donor sites for cartilage are the nasal septum and the concha of the ear. Rib cartilage grafts have also been used. A solid stent or a T tube is inserted and the graft is sutured in place. After subglottic cricoid stenosis repair, even with an otherwise normal larynx, a T tube, if used, should be placed with the superior limb up to the level of the laryngeal ventricle. The T tube, if left subglottic, would be too close to the vocal cords, causing irritation with granulation formation and scarring. The upper end of the T tube should never be closer than 6 mm to the undersurface of the vocal cord, and preferably 10 mm below it. If it needs to be higher than that, it should go through the cords to the level of the ventricle, if granulations are to be prevented. In patients undergoing subglottic stenosis and stent placement, a tracheostomy is necessary. All such tracheostomy tubes should be placed through any existing tracheal stenosis area, rather than through a normal segment of nonstenotic trachea.

For small mucosal defects, local flaps are preferable, but when there is a large area of denuded mucosa, especially if it is circumferential, a graft is necessary. Although one may use a split thickness skin graft, buccal mucosa taken from the inside of the cheek, or septal mucosa from one side of the nasal septum, typically performs better. These can be easily harvested with minimal problems to the patient. The graft is sewn in directly or wrapped around the conforming stent and sutured in place with the mucosal surface against the stent (Figure 35-6). When the stent is positioned within the laryngeal lumen, the graft's submucosal undersurface will adhere to the patient. Topical application of a 1% solution of mitomycin-C for 3 minutes to mucosal incisions is reported to markedly reduce granulation and subsequent scarring.[7]

Bilateral Vocal Cord Paralysis

In patients with bilateral vocal cord paralysis, the classic treatment has been an arytenoidectomy, either performed endoscopically or from the laryngofissure approach.[8,9] If a patient has arytenoid fixation, either traumatic or from rheumatoid arthritis, this is still the procedure of choice. The arytenoid is removed and the posterior cord is sutured laterally to the thyroid cartilage. If the arytenoids are mobile, a simpler technique is to endoscopically lateralize the vocal cord.[10–12] A no. 20 spinal needle is passed through the skin under binocular vision, using the laryngoscope with the Lewy suspension attached. With one eye looking through the laryngoscope and the other eye on the needle, one can quite accurately aim the needle, so that

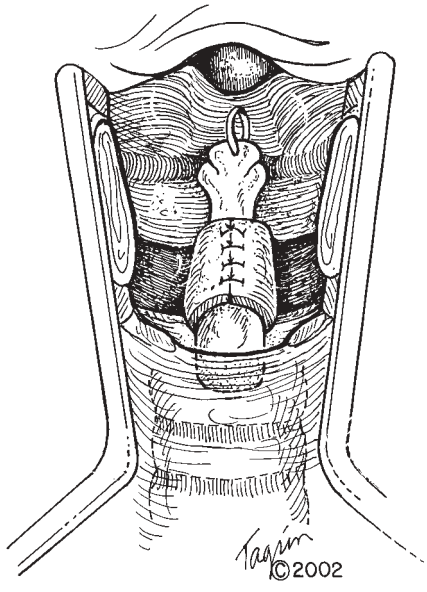

FIGURE **35-6** *Conforming stent in place with the mucosal graft over the stent at the level of the denuded mucosa.*

when it penetrates the thyroid cartilage, it will come out at the level of the vocal process. One needle is placed below the cord and the other into the ventricle so that it comes out above the cord. The obturators are removed and a no. 0 or 2-0 nylon suture is threaded down the needle and into the larynx where it is grasped with a small forceps and brought out through the mouth (Figure 35-7*A*). This is done with both sutures and a square knot is tied (Figure 35-7*B*). An incision is made externally between the two needles down to the cartilage. One of the needles is then removed and that suture drawn back until the knot is pulled through the cartilage. The other needle is left in place until the knot has been pulled through. With nylon, even a square knot may untie during the pull through. By leaving one needle in place, it is much easier to reinsert the other needle into the proper place. Once the knot is pulled through, the second needle is removed, the cord pulled laterally, and the suture is tied down so that the knot is on the thyroid cartilage (Figures 35-7*C*,*D*). If one is quite certain that recovery will not take place, then prior to pulling the cord over, scarring is created lateral to the vocalis muscle, either by using a laser or a long laryngeal electrocautery needle. This tends to hold the cord lateral if the suture later cuts through the tissue or otherwise becomes loosened. Examining through the laryngoscope, one can immediately ascertain as to whether enough airway has been obtained. If not, a second suture is placed more anteriorly in the midvocal cord. On occasion, both cords can be lateralized, but that is rarely necessary. An ideal result is an angulated cord, where the two cords approximate in the anterior portion for vocalization and are separated posteriorly for respiration. With arytenoidectomy, a temporary tracheostomy must be performed; most patients will already have one. An endoscopic lateralization procedure can often be done without a tracheostomy if the patient is covered with steroids during surgery, though they must always be warned that a tracheostomy may become necessary if edema develops.

In addition to the fact that this procedure is simple and usually successful, another advantage is that it is easily reversible if function recovers. Guided by the scar, the skin is incised under local anesthesia and the suture is cut at the knot on the lateral surface of the thyroid cartilage and removed. Many paralyses do recover, especially the idiopathic ones and those due to retractor pressure or stretching of the recurrent laryngeal nerve in thyroid or cardiac surgery. In the pediatric age group, recovery is even more frequent, but it may take up to 5 years.[13] Others have advocated partial excision of one or both vocal cords, using either knife or laser.[14,15] These do restore an airway and preserve some voice, but they are not reversible and so are not recommended unless one is certain the recurrent laryngeal nerve has been severed with no attempted grafting.

One must always discuss preoperatively with patients the fact that the better the airway, the worse the voice will be postoperatively, no matter which procedure is used. A patient with bilateral cord paralysis will usually come in with a good voice and a poor airway. Indeed, some patients, when presented with the alternatives, will prefer a permanent tracheostomy and simply plug the tube with a finger when talking, or have a cannula with a speaking valve.

Unilateral Vocal Cord Paralysis

In unilateral paralysis, the usual problem is one of poor voice rather than airway difficulty. A unilateral paralysis is usually of little permanent significance, as 80% of patients will recover a normal speaking voice within 3 months without any treatment whatsoever. This recovery is of just normal use of the voice; they usually do not get a good singing voice back. Of the other 20% of patients, most can be rehabilitated by a voice therapist, using exercises to strengthen the functioning cord to compensate for the paralyzed one. A small percentage will need surgical help to obtain an adequate voice and to prevent aspiration. There is also the rare patient in whom the cord is paralyzed in abduction. Paralysis in abduction is usually due to a more central injury, and these patients have typically a very breathy voice and aspirate freely. They are best treated by a thyroplasty to medialize the vocal cord.[16–18] This can be accomplished by inserting a small rectan-

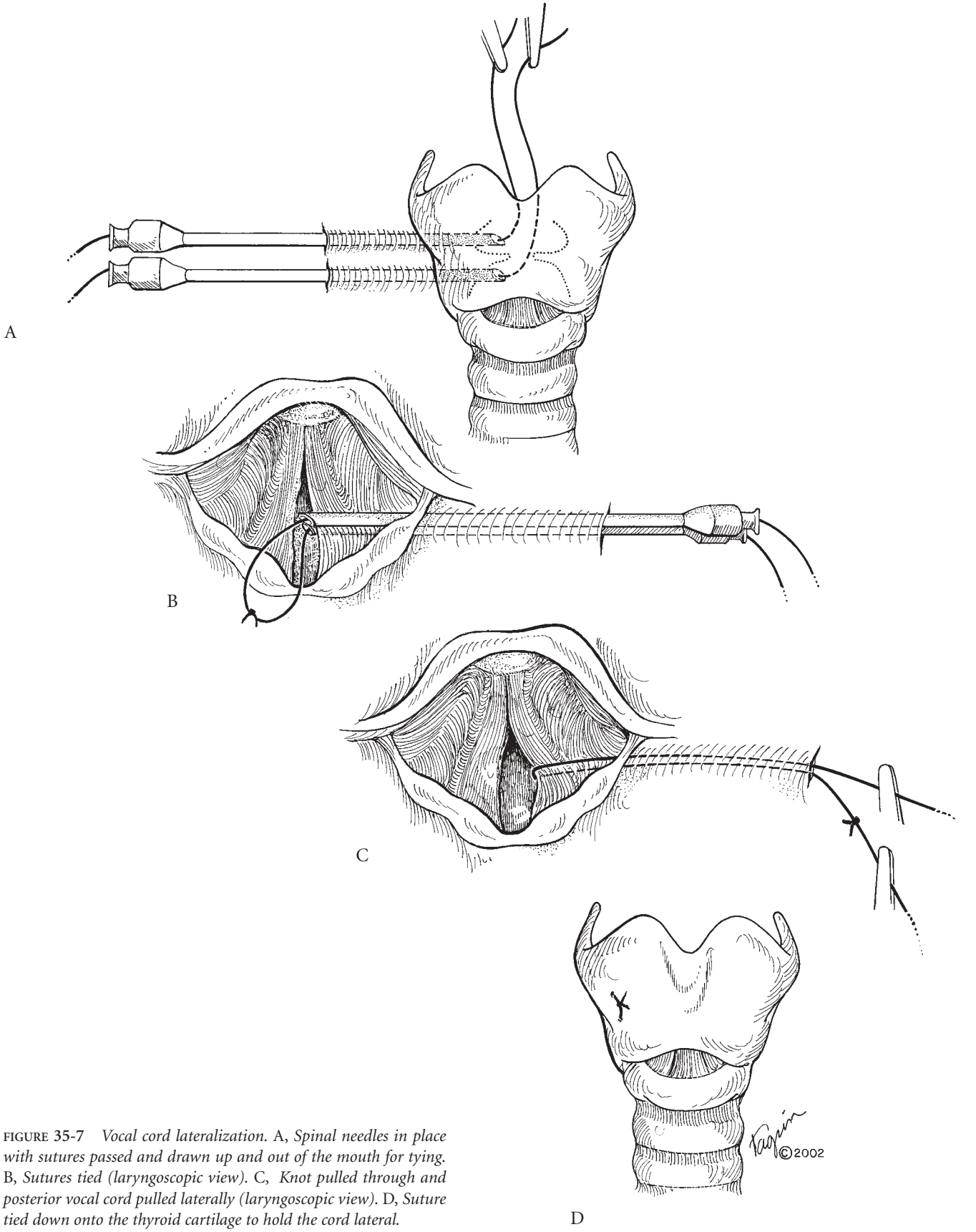

FIGURE 35-7 *Vocal cord lateralization.* A, *Spinal needles in place with sutures passed and drawn up and out of the mouth for tying.* B, *Sutures tied (laryngoscopic view).* C, *Knot pulled through and posterior vocal cord pulled laterally (laryngoscopic view).* D, *Suture tied down onto the thyroid cartilage to hold the cord lateral.*

gular piece of cartilage, taken either from elsewhere on the thyroid cartilage or from the nasal septum, and placed lateral to the vocalis muscle. Even better is a silicone implant such as the Montgomery implant (Boston Medical Products, Inc., Westborough, MA), which has a triangular shape, giving more medialization posteriorly where it is needed (Figure 35-8).[19] This gives an improvement in voice and decreases aspiration. Other authors have used titanium,[20] Vitallium, ceramic materials,[21] Goretex,[22] and hydroxylapatite[23] implants to medialize the vocal cord.

When the cords nearly approximate, but the voice is still not good, then Teflon, fat,[24,25] Gelfoam, or collagen[26] can be injected into the cord lateral to the vocalis muscle. Teflon was widely used from the 1960s to the 1980s but is no longer available due to legal difficulties from use in breast augmentation. When properly placed in the vocal cord, migration and granuloma formation were uncommon. However, if the Teflon was injected in the superficial areas of the cord, then granulomas were a problem; if placed too deeply in the cord, the Teflon could migrate subglottically. Currently, fat, Gelfoam, and collagen are most commonly used, with several small injections made along the length of the cord. This is usually done under local anesthesia so the patient can speak, saying "E" to give the surgeon an idea of how much injection is necessary to produce a good voice, but not so much so as to compromise the airway. Typically, these procedures give the patient a good voice, but as these materials tend to be absorbed over time, the benefits may fade.

Aspiration

Aspiration can be a very vexing problem for both the surgeon and the patient, and can lead to failure and significant patient morbidity even when the underlying stenosis has been corrected. Besides unilateral and bilateral vocal cord paralysis, all of the following may contribute to aspiration: loss of sensation from superior laryngeal nerve injury, old age, superior laryngeal release procedures, and cricopharyngeal muscle spasm from denervation, chronic tracheostomy tube use ("trach dominance"), and a fixed partially stenotic glottis. As we age, we lose laryngeal sensation and do not feel aspirated liquids in the larynx and trachea as well. Injury to the superior laryngeal nerve also decreases sensation. After a superior laryngeal release, the larynx is dropped down away from the epiglottis, so the epiglottis does not protect the larynx to the same extent. With cricopharyngeal spasm, swallowing is more difficult, and food and liquid tend to spill over into the larynx. Cricopharyngeal muscle spasm can be improved by inferior constrictor myotomy. Vocal cord paralysis implies that a segment of the laryngeal introitus does not move normally and to some

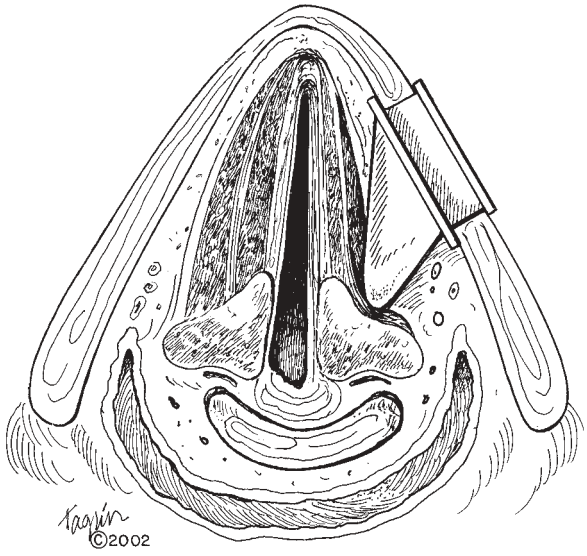

FIGURE **35-8** *Montgomery thyroplasty implant in place, medializing the vocal cord.*

degree predisposes to aspiration. This can become manifest after a chronic obstructive stenosis has been surgically corrected. A preoperative barium swallow can help detect occult preoperative aspiration so as to forewarn the surgeon that postoperative aspiration may become a problem. Unilateral vocal cord paralysis with associated aspiration can be improved with vocal cord medialization, as noted above. Swallowing training and rehabilitation by a speech pathologist can often overcome the aspiration problems of the aging and those with loss of laryngeal sensation. The patient is taught techniques of swallowing, following each swallow with a little cough to clear any aspirated material. Trach dominance refers to patients who lose laryngeal function as a result of chronic diversion of airflow away from the laryngeal lumen, as in a patient who has an unvalved tracheostomy chronically. Laryngeal function, including the protection from aspiration, slowly degrades in the setting of a chronic tracheostomy tube. Similarly, patients with a chronic, significant glottic level stenosis also are deprived of normal laryngeal airflow and can develop a type of disuse atrophy which may require aggressive speech and swallowing therapy post corrective stenosis surgery. All these problems are obviously much more difficult when dealing with infirm, aged, and mentally slow patients in whom gastrostomy may be necessary to provide ongoing nutrition. Vocal cord paralysis, due to neurologic problems such as stroke or amyotrophic lateral sclerosis, often requires a tracheostomy with a cuffed tube. Over the long term, glottic closure by bilateral medialization thyroplasty or a laryngofissure procedure with suturing together of the denuded vocal cords can be necessary. This, of course, requires a permanent tracheostomy and eliminates normal speech; loss of speech is often of little importance in these patients, who are typically aphonic.

References

1. Montgomery W. Surgery of the upper respiratory system. Vol II. Philadelphia (PA): Lea and Febiger; 1973. p. 574–96.
2. Randolph G. Thyroid and parathyroids. In: Lee KJ, editor. Essential otolaryngology. 7th ed. Stanford (CT): Appleton and Lang; 1999. p. 573–643.
3. Montgomery W, Lofgren R. Usual and unusual causes of laryngeal arthritis. Arch Otolaryngol 1963;77:29–33.
4. Lofgren R, Montgomery W. Incidence of laryngeal involvement in rheumatoid arthritis. N Engl J Med 1962;267:193–5.
5. Montgomery W. Anterior glottic stenosis; experiment and clinical management. Arch Otolaryngol 1970;92:560–7.
6. Montgomery W. Posterior and complete laryngeal (glottic) stenosis. Arch Otolaryngol 1973;98:170–5.
7. Spector J, Werkhaven J, Spector N, et al. Preservation of function and histologic appearance in the injured glottis with topical mitomycin-C. Laryngoscope 1999;109:1125–9.
8. Thornell W. Introlaryngeal approach for arytenoidectomy in bilateral abductor vocal cord paralysis. Arch Otolaryngol 1948;47:505–8.
9. Woodman D. A modification of the extralaryngeal approach to arytenoidectomy for bilateral abductor paralysis. Arch Otolaryngol 1946;43:63–5.
10. Kirchner J. Endoscopic lateralization of the vocal cord in abductor paralysis of the larynx. Laryngoscope 1979;89:1779–82.
11. Lewy R, Borkenhagan R, Hinojosa J. Validation of a vocal cord lateralization procedure. Laryngoscope 1982; 92:697–9.
12. Ejnell H, Mansson I, Hallen D, et al. A simple operation for bilateral vocal cord paralysis. Laryngoscope 1984; 94:954–8.
13. Daya H, Hosni A, Bejar-Solar I, et al. Pediatric vocal fold paralysis. Arch Otolaryngol Head Neck Surg 2000; 126:21–5.
14. Ossoff R, Sisson G, Duncavage J, et al. Endoscopic laser arytenoidectomy for the treatment of bilateral vocal cord paralysis. Laryngoscope 1984;94:1293–7.
15. Prasad U. CO_2 surgical laser in the management of bilateral vocal cord paralysis. J Laryngol Otol 1985;99:891–4.
16. Isshiki N, Morita H, Okamura H, Hiramoto M. Thyroplasty as a new phonosurgical technique. Acta Otolaryngol (Stockh) 1974;78:451–7.
17. Isshiki N, Okamura H, Ishikawa T. Thyroplasty type I (lateral compression) for dysphagia due to vocal cord paralysis or atrophy. Acta Otolaryngol (Stockh) 1975;80:465–73.
18. Isshiki N, Kojima H, Taira T, Shoji K. Recent modifications in thyroplasty type I. Ann Otol Rhinol Laryngol 1989;98:777–9.
19. Montgomery W, Blaugrund S, Varvaras M. Thyroplasty: a new approach. Ann Otol Rhinol Laryngol 1993; 102:571–9.
20. Frederick G. Titanium vocal fold medializing implant: introducing a novel implant system for external vocal fold medialization. Ann Otol Rhinol Laryngol 1999;108:79–86.
21. Sakai N, Nishizawa N, Matsushima J. Thyroplasty type I with ceramic shim. J Artif Organs 1996;20:951–4.
22. McCulloch T, Hoffman H. Medialization laryngoplasty with expanded polytetrafluoroethylene. Surgical technique and preliminary results. Ann Otol Rhinol Laryngol 1998;107:427–32.
23. Cummings C, Purcell L, Flint P. Hydroxylapatite laryngeal implants for medialization. Preliminary report. Ann Otol Rhinol Laryngol 1993;102:843–51.
24. Brandenberg J. Vocal cord augmentation with autogenous fat. Laryngoscope 1992;102:495–500.
25. Woo P, Rahbar R, Wang Z. Fat implantation into Reinke's space: a histologic and stroboscopic study in the canine. Ann Otol Rhinol Laryngol 1999;108:738–44.
26. Remade M, Delos M, Lawson G, et al. Correcting vocal fold immobility by autologous collagen injection for voice rehabilitation, a short term study. Ann Otol Rhinol Laryngol 1999;108:788–93.

Foreign Body Aspiration

Daniel P. Doody, MD

Aspiration of foreign material remains a common cause of accidental death in the United States. Over 3,000 people annually die with unintentional aspiration identified as the primary cause. It is the fourth leading cause of accidental death in children under the age of 5 years and the leading cause of accidental death in children under the age of 1 year. Besides these deaths, an uncertain number of patients survive with variable sequelae, including chronic pulmonary injury and anoxic brain damage.

Historical Overview

Aspiration as a cause of death was recognized as early as the mid-1600s when Bradwell reported on objects that could suddenly endanger breathing, and noted that he heard of a child who had been " . . .strangled with a Grape."[1] Muys in 1690 reported a 7-year-old who died from "suffocation three weeks after aspirating a bean."[2] In 1854, Samuel D. Gross, then Professor of Surgery at the University of Louisville, wrote a treatise on foreign body aspirations, reporting over 200 cases collected from the world literature or cases of which he was personally aware.[2] It was in that monograph that foreign body aspiration in children was found to be a common problem and appeared to occur as frequently in that younger age group as it did in adults.[2]

In 1895, Killian performed purposeful bronchoscopy, and 2 years later in 1897, successfully carried out the endoscopic removal of a foreign body, a piece of bone.[3] The first successful bronchoscopy and extraction of a foreign body in the United States is credited to Algernon Coolidge Jr at the Massachusetts General Hospital. Using a headlight and female urethral speculum in 1899, he performed bronchoscopy through a tracheostoma and removed a disconnected portion of the tracheal cannula from the bronchus of a 23-year-old male.[4]

With these seminal successes, the work of Chevalier Jackson in the early 1900s led to the development of increasingly sophisticated instrumentation for the safe practice of airway endoscopy.[5–7] With improved magnification in the 1940s and a fiber-optic illuminator introduced in the 1960s, the extraction of most foreign bodies can be performed endoscopically, and the current mortality rate, if the victim can be brought to medical care, is less than 1%.

Epidemiology

Although 2,000 to 3,000 children annually will suffer some type of foreign body aspiration, the annual mortality rate for foreign body aspiration in the pediatric age group has decreased over the last two decades. In 1998, the last time frame for which data are available, 184 children in the United States under the age of 15 years died as a result of aspiration and occlusion of the airway. Fortunately, with increased public awareness of aspiration as a critical problem in young children, the death rate in the pediatric age group has decreased from approximately 450 deaths annually in 1979 to the current low of 184 deaths in 1998.

In the pediatric age group, the highest incidence of foreign body aspiration occurs in children under 4 years of age, with the maximal frequency occurring between 1 and 2 years of age. Infants and toddlers, the pediatric age group at greatest risk, are prone to aspiration for a variety of reasons: oropharyngeal coordination is not fully mature, mastication of hard food objects (ie, candy, nuts) is not thorough, curiosity leads small children to place objects in their mouths, and finally, children are easily distracted, further interrupting the normal chewing and swallowing mechanism. Boys are at greater risk for aspiration, with most series reporting a male to female ratio of 2:1. The most common aspirated foreign materials include nuts, grapes, seeds, other vegetable material, and small hard candies. Fatal aspirations in this age group are frequently associated with meat and meat products. Hotdogs, because of their size, compressibility, and widespread availability are one of the leading food products associated with fatal aspiration.[8]

Death from foreign body aspiration remains a major problem in the adult population. Over 3,000 adult victims were identified in 1998, with the primary cause of death being ascribed to inhalation of foreign object leading to airway obstruction or suffocation. The "café coronary" syndrome, first so termed by Haugen in 1963, is sudden collapse and death while eating, secondary to acute obstruction of the airway by food.[9] Initially ascribed to an acute "coronary" event, the problem was later found to be associated with the impaction of food (most typically meat) in the major airway (larynx or trachea). In that initial report, predisposing factors to fatal aspiration included alcohol ingestion and poor dentition. Other factors subsequently identified include institutionalization in long-term care facilities, sedative agents, and Parkinson's disease.[10,11] This risk in the general population is 0.66 per 100,000 population and has remained relatively constant since it was first described. Epidemiologically, the highest incidence and death rate from foreign body aspiration occurs in the senior age group (age greater than 74 years), and these rates have risen in the last several years as the elderly population expands. With age, dentition is typically poorer, oropharyngeal coordination lessens, and confusion can lead to accidental aspiration, these risks being very similar to the younger pediatric population.

Clinical Presentation

In most cases, the clinical presentation will be determined by the size of the foreign body, its location in the respiratory tract, the type of material aspirated, and the time from the aspiration event until presentation for care. The classically described triad of foreign body aspiration is paroxysmal coughing, wheezing, and diminished breath sounds on the affected side. At least one of these three symptoms is reported in 75 to 97% of children with proven foreign body aspiration.[12–14] The classic triad itself is found in a much lower percentage, approximately one-third of all patients with foreign bodies in the airway. A history of witnessed choking is an additional important feature in the history, and in some series, it has the highest sensitivity in correctly identifying foreign body aspiration as the basis of the patient's respiratory symptoms.[14,15] If the material is large, then the patient may present with signs of severe respiratory distress. This is particularly true with laryngotracheal foreign bodies where dyspnea, stridor, and cyanosis are noted, and respiratory arrest occurs in the most severe cases.

Fortunately, most cases of foreign body aspiration will not result in complete and catastrophic airway occlusion, but instead present with lesser signs and symptoms. Almost all patients will experience some

symptoms at the time of the aspiration episode. Paroxysmal coughing is the primary sign, noted by parents or caregivers, in the pediatric population. With time, however, these characteristic features may abate. With delayed presentation, distal airway foreign bodies may present without symptoms or with nonspecific symptoms such as a nonproductive cough, the new onset of inspiratory and/or expiratory wheezing (often assumed to be newly diagnosed asthma), and complaints of mild shortness of breath. Unlike aspirated foreign material in the laryngotracheal location, dyspnea is less frequently reported as a symptom of foreign bodies in the distal airway compared with those centrally located in the airway.

Unsuspected foreign bodies may be found in a small number of patients who present with nonspecific pulmonary diseases. In one large pediatric series, over 1,000 patients underwent flexible bronchoscopy, and in slightly less than 1% of those patients, an unanticipated foreign body was identified.[16] The authors noted that there were no significant differences in clinical signs and symptoms in a child who presented with an unsuspected foreign body than in those children who had a more typical pulmonary pathology.

Physical Examination

Physical examination may or may not substantiate the symptoms related in the history. With large foreign bodies located in the upper airway, stridor, sternal retraction, use of ancillary muscles, central cyanosis, and dyspnea are obvious. With bronchial obstruction, auscultation may reveal inspiratory and expiratory wheezing and decreased or absent breath sounds on the side of obstruction. However, one of the most important features of this clinical problem is that, in 20 to 40% of cases, the examination will be totally normal, despite the later discovery of a foreign body in the tracheobronchial tree.

Radiographic Evaluation

Radiographic evaluation of foreign body aspiration is helpful, but a normal radiographic examination does not exclude the possibility of an airway foreign body. Various series have reported normal radiographs in 6 to 80% of children with proven foreign bodies in the tracheobronchial tree. Although some foreign bodies are easily seen on standard chest radiographs (Figure 36-1), 80 to 90% of aspirated foreign bodies are vegetative material, thus radiolucent and not visualized. The most common abnormal finding in the pediatric patient with aspiration is hyperexpansion of the affected side, which occurs in approximately 60% of cases (Figure 36-2), although atelectasis or an infiltrate may be noted in a lesser number of cases. In the adult population, atelectasis and loss of lung volume is more common on the affected side (Figure 36-3). Not unexpectedly, the more dangerous laryngotracheal foreign bodies are likely to have normal x-rays. However, this subset of patients, usually accounting for 5 to 15% of all foreign body aspirations, is more likely to present with severe clinical symptoms (dyspnea, sternal retraction, cyanosis). If there is a significant delay in presentation, as occurs in up to 50% of cases of foreign body aspiration in the pediatric age group, then there is higher likelihood that the initial chest x-rays will be abnormal as secondary features become apparent in the obstructed lung.

If the chest radiographs are normal, then inspiratory and expiratory films or films in both the left and right lateral decubitus positions may be helpful. In a child with partial bronchial obstruction, the expiratory film will demonstrate hyperinflation secondary to check-valve effect from the obstructing foreign body (Figures 36-4*A*–*C*). Some authors promote the expiratory phase by placing gentle pressure over the child's epigastrium and timing exposure of the film with a natural expiration. Even with these measures, due to an inability to cooperate, inspiratory and expiratory films in most young pediatric patients are inconclusive. In the younger patient, the use of right and left lateral decubitus views of the chest may be helpful. The lung in the dependent position would typically appear compacted. In a child with a foreign body aspiration and partial obstruction, the dependent lung would not appear compressed and may even appear slightly hyperinflated.

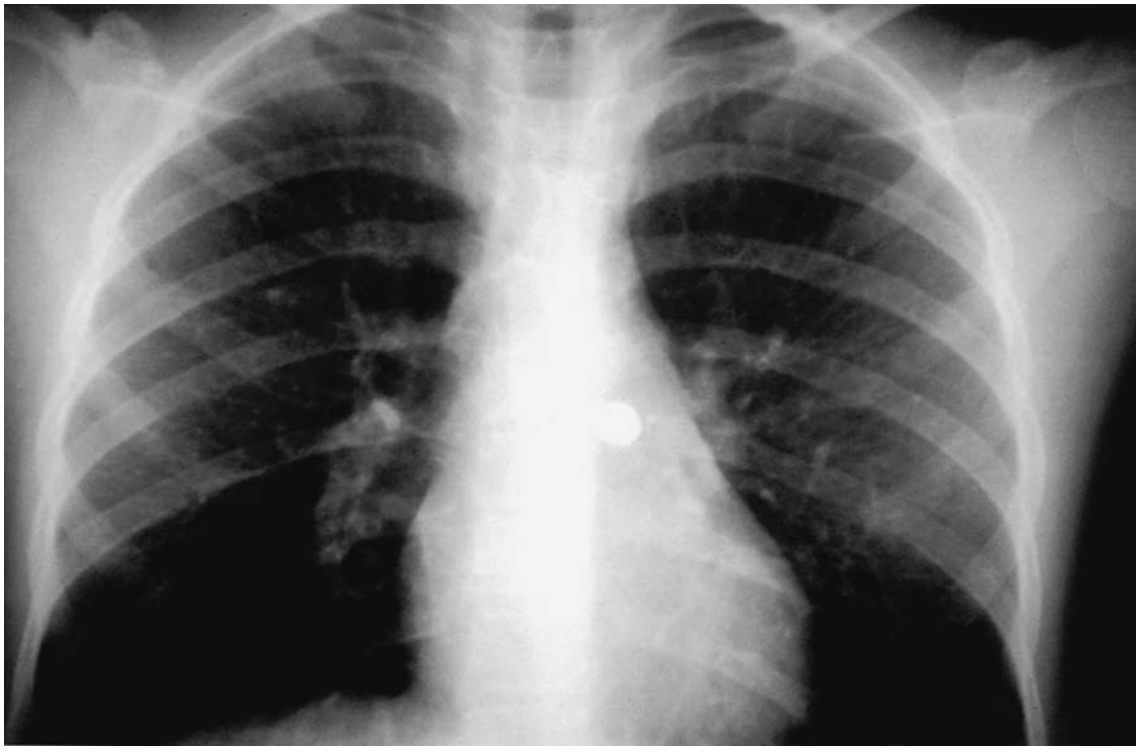

FIGURE **36-1** *Ten-year-old with a radiopaque thumbtack located in the left mainstem bronchus.*

False-positive findings on a chest radiograph will occur in about 12% of children who are not found to have an aspirated foreign body on tracheobronchoscopy. Conversely, up to 65% of the children who had normal chest x-rays, but a history consistent with aspiration, were found to have a foreign body on endoscopy.[15]

In an older patient, fluoroscopy of the chest may be helpful in establishing the diagnosis of aspirated foreign body. With laryngotracheal foreign bodies, paradoxical mediastinal movement and an increase in the size of the mediastinal structures may be noted during inspiration. However, abnormal fluoroscopic findings are less frequently seen with bronchial foreign bodies.[17] Occasionally, with foreign bodies in a bronchial location, a shift of the mediastinal structures toward the normal airway may be noted during expiration, because of the check-valve effect of the partially obstructing lesion and entrapment of air in the obstructed lung.

Although authors have reported the use of ventilation-perfusion scanning, it is infrequently used in the emergent evaluation of a possible foreign body obstruction. Similarly, computed tomography (CT) scans have been reported to be helpful, particularly in a patient who may have a delayed presentation and an atypical history. However, findings on CT imaging usually show the secondary effects of the foreign body aspiration (Figure 36-5) and may or may not demonstrate the foreign body itself.[18] In the acute setting, there appears to be little role for this imaging study.

Treatment

As radiographic studies cannot exclude foreign body aspiration, children and adults who present acutely with a history consistent with foreign body aspiration (choking, sudden paroxysmal coughing) and any abnormal physical findings (wheezing, diminished breath sounds) should undergo bronchoscopy for removal of the presumed foreign body. With that presentation, there is no indication for additional studies. In the hands of experienced bronchoscopists, a tracheobronchoscopy has minimal risk and is associated with few complications.

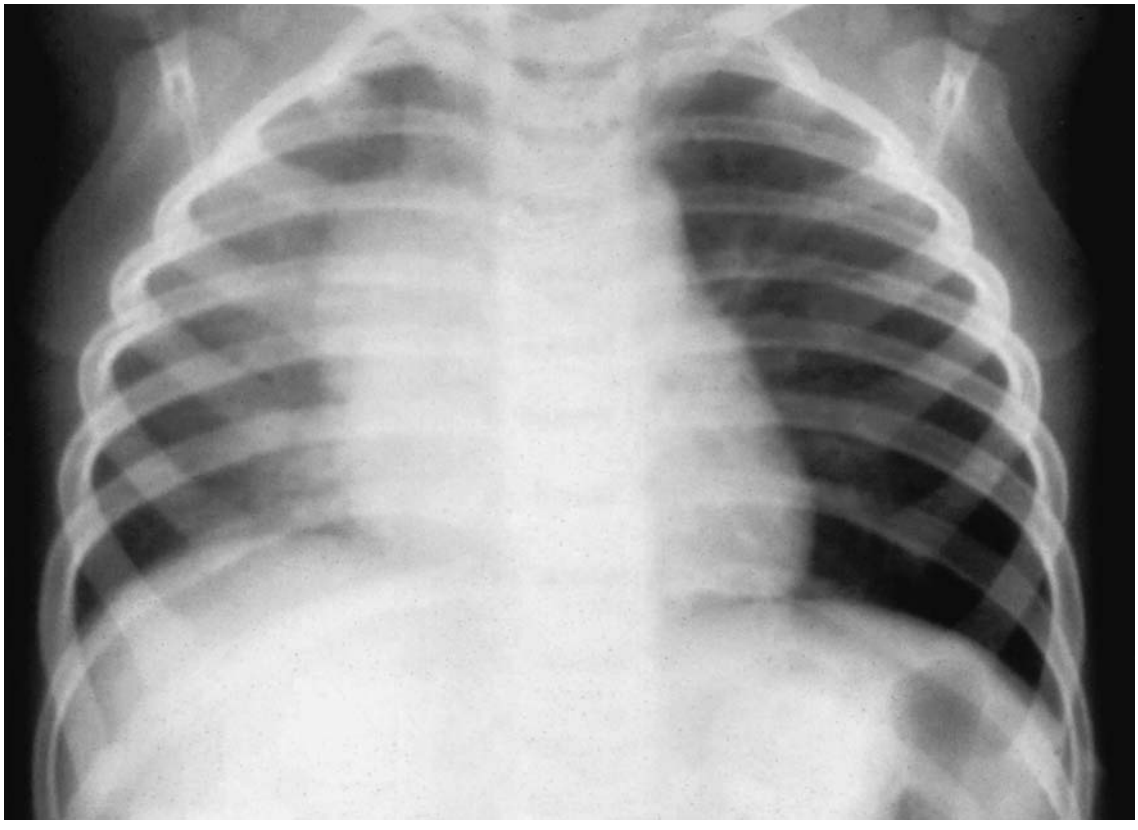

FIGURE **36-2** *Carrot in left mainstem bronchus demonstrating the effect of a check-valve obstruction. There is hyperexpansion of the left lung and right-sided volume collapse with mediastinal shift.*

At this time, most bronchoscopies for a suspected foreign body aspiration are performed as a rigid (open tube) bronchoscopy, so that the airway can be controlled and the patient ventilated as the foreign body is removed. The use of sophisticated optical grasping forceps, snares, Dormia stone basket,[19–22] or Fogarty balloon[23–27] allows removal in 97 to 99% of foreign bodies identified.

Some clinicians have used flexible bronchoscopy as an initial diagnostic technique in children who have a history suspicious for foreign body aspiration but do not have conclusive evidence of aspiration (ie, no findings on physical examination and normal or near-normal radiographs).[16,28] Performed with local anesthesia, this flexible examination of the airway might obviate the need for general anesthesia and rigid bronchoscopy if the airway is normal. If a foreign body is identified, then rigid bronchoscopy is performed to remove the material. Others have disagreed with these recommendations, noting that extraction of the foreign body can be performed successfully with the flexible bronchoscope in up to 80% of the cases.[29] However, these reported series are small and the authors, experienced in both flexible and rigid tracheobronchoscopy, were capable of performing rigid bronchoscopy if problems arose during attempts at flexible bronchoscopic extraction. At the present, in most centers, open tube bronchoscopy appears safer than the flexible bronchoscopy and is the preferred method for extraction of foreign bodies in the pediatric population.

In adults, both flexible bronchoscopy and rigid bronchoscopy have been used successfully to remove foreign bodies.[30–33] The flexible bronchoscope may mitigate the need for general anesthesia for extraction of the foreign body, but like the pediatric patient, there is greater risk with the use of this technique if the foreign body is lost. Several groups, most notably the Mayo group, have used flexible bronchoscopy in adults and children with success.

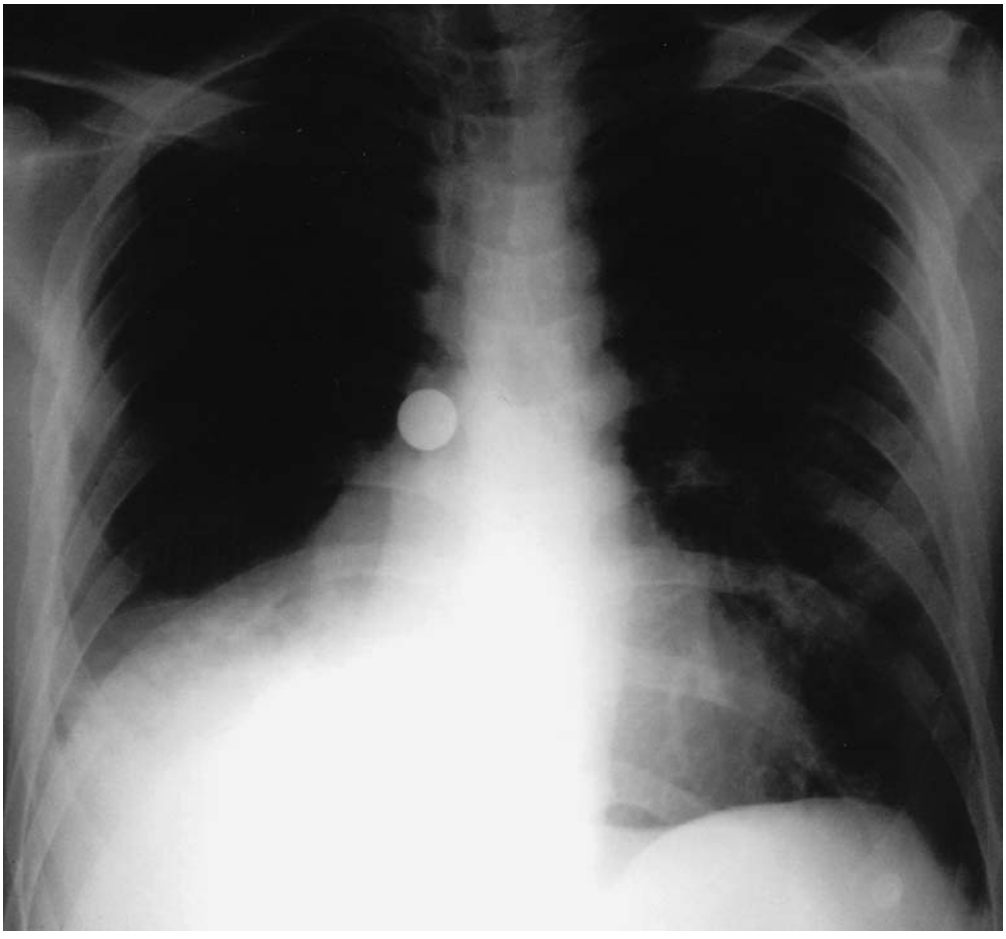

FIGURE **36-3** *Metal bearing in the bronchus intermedius, in a young adult with a history of pica. Distal atelectasis of the right middle and lower lobes is appreciated.*

Currently, the flexible bronchoscope allows a more limited choice of instrumentation, its diameter is more likely to obstruct the airway (particularly in pediatric patients), and it may not be as well suited as the rigid bronchoscope for retrieval and extraction of foreign bodies.[34] We feel that the rigid bronchoscope allows a greater margin of safety and prefer this technique to the use of flexible bronchoscopy in children and adults. Flexible bronchoscopy has some advantage over rigid bronchoscopy in its ability to retrieve more distal tracheobronchial foreign bodies. Flexible bronchoscopy should also be considered in those patients who have cervical or maxillofacial trauma, who are intubated and in whom there is concern for aspiration.[35]

In the rare instances where the foreign body is too large to pass retrograde through the glottis, a tracheostomy should be performed, the foreign body extracted, and a tracheostomy tube placed. Once the patient is stable, the tracheostomy tube can be removed and allowed to close secondarily, usually within 48 to 72 hours.

Occasionally, foreign bodies will move to a remote position in the bronchial tree or there may be a marked delay in presentation for care. Either of these features may lead to secondary pulmonary complications, including bronchiectasis, localized pneumonia, and lung abscess formation (Figure 36-6). In such instances, if the foreign body cannot be reached by endoscopic means (flexible or rigid), then a thoracotomy with bronchotomy and extraction of the foreign body and/or resection of the affected lung segment is appropriate. The subset of patients who require this more extensive surgery is small and in the current

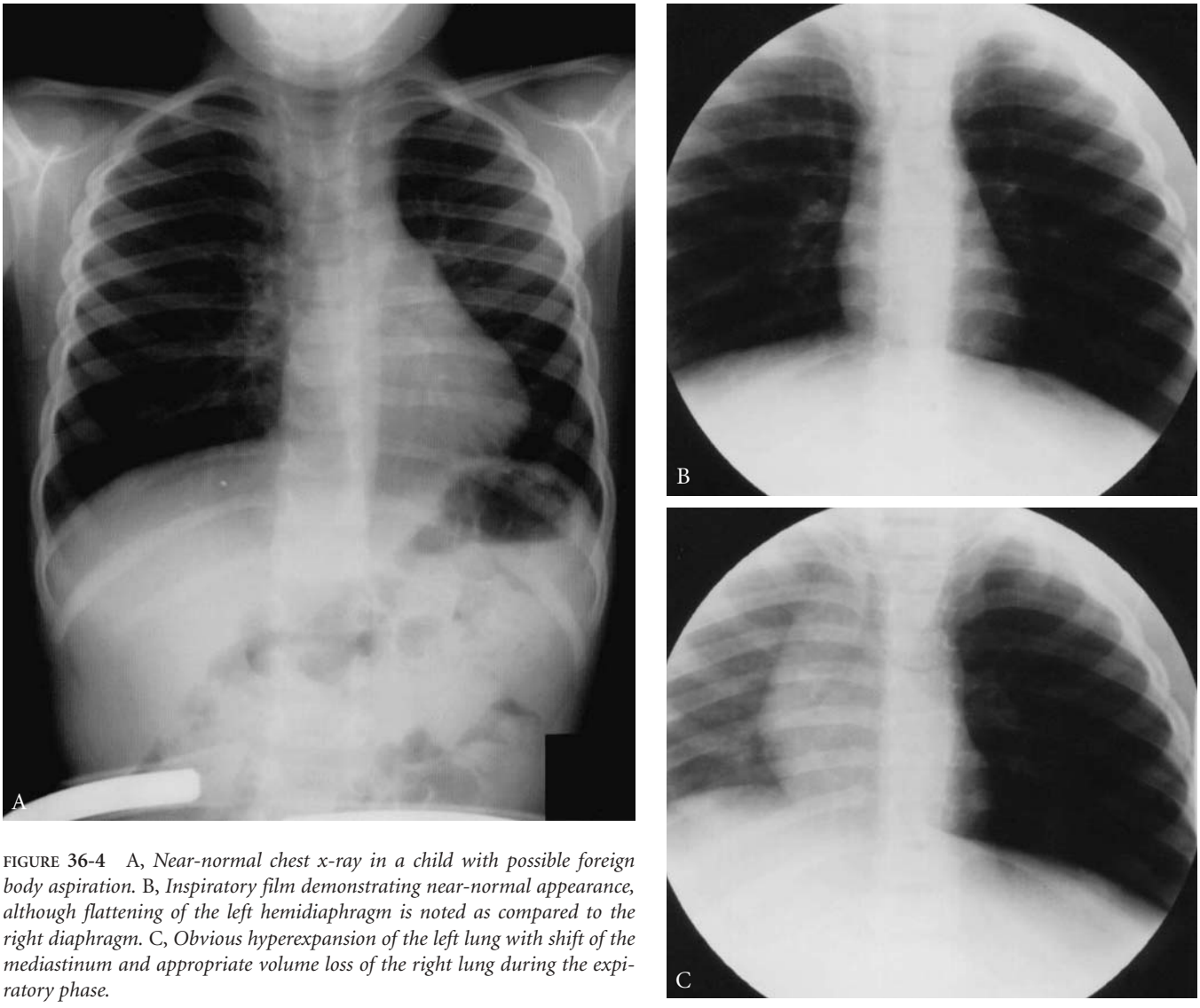

FIGURE **36-4** A, *Near-normal chest x-ray in a child with possible foreign body aspiration.* B, *Inspiratory film demonstrating near-normal appearance, although flattening of the left hemidiaphragm is noted as compared to the right diaphragm.* C, *Obvious hyperexpansion of the left lung with shift of the mediastinum and appropriate volume loss of the right lung during the expiratory phase.*

era will account for only 1 to 3% of cases of aspiration. Most patients requiring a thoracotomy for extraction have a delayed presentation, often found to be months or years after the aspiration event.

The use of chest physical therapy, postural drainage, and inhaled bronchodilator therapy was briefly recommended as an alternative to bronchoscopy in the treatment of foreign body aspiration, as it appeared to be "safe" and was effective in 85% (24 of 28) of the children treated.[36,37] However, even in those initial reports, 2 infants were noted to have "serious" episodes of respiratory or cardiorespiratory arrest. A later and larger study from the same institution found a much lower success rate with this conservative management (25% vs 85%) and acknowledged that there is a definite risk to this management.[38] They recommended conservative therapy in an intensive care unit setting only for a 24-hour period and concluded that undertaking this therapy was inappropriate for foreign bodies present for greater than 4 weeks.[38] By 1980, with continued improvement in endoscopic instrumentation, coupled with a low success rate and a small but potentially life-threatening risk with conservative management, inhalation therapy and postural drainage was considered contraindicated as treatment for foreign body aspiration.[39]

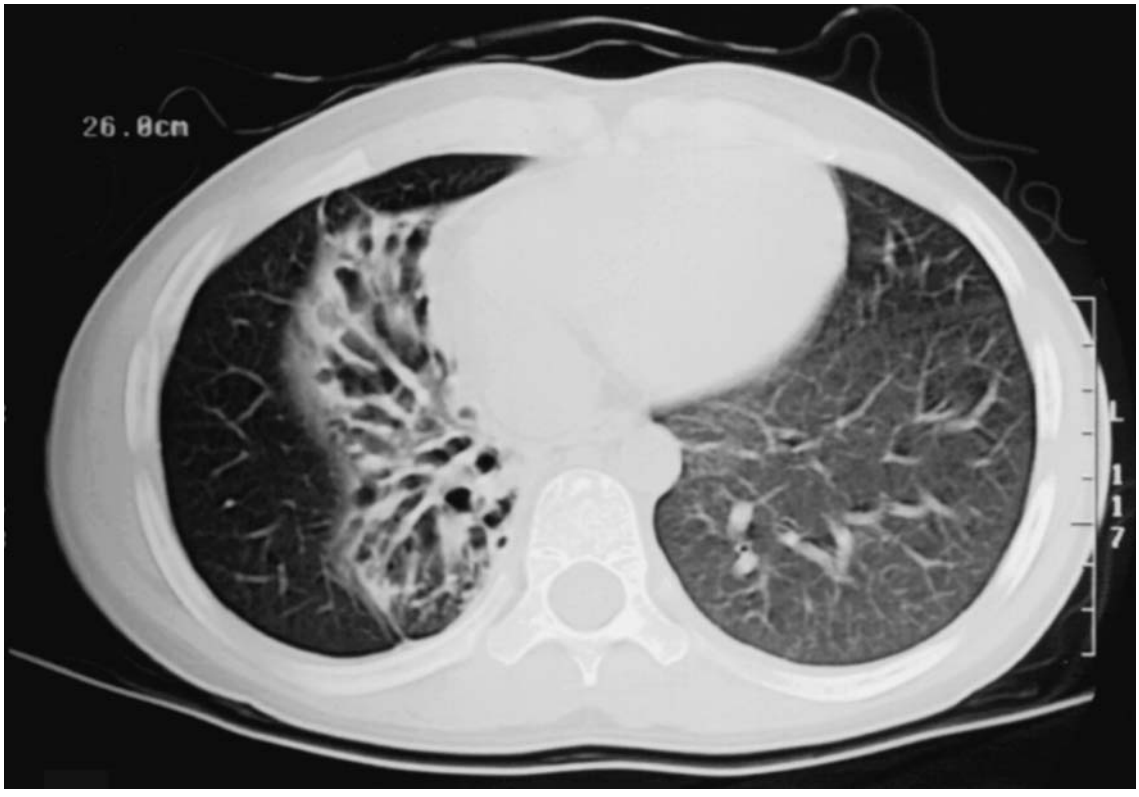

FIGURE **36-5** *Bronchiectasis of the right lower and middle lobes complicating radiolucent foreign body aspiration. The child had a history of recurrent right-sided pneumonia for over 2 years prior to evaluation and surgical treatment.*

Physical therapy with percussion and dependent drainage may help to mobilize distal secretions in those patients with atelectatic lung segments complicating foreign body obstruction, but only after the foreign body has been removed endoscopically.

With an acute aspiration event, antibiotics and steroids are not routinely given. If there is evidence of an infection distal to the impacted foreign body, then broadspectrum antibiotics (second generation cephalosporin plus clindamycin, ampicillin-sulbactam) may be appropriate. Steroids may be appropriate in those instances where the foreign body has resulted in airway narrowing secondary to inflammation. In most instances, even in the smaller airway with young children, steroids are not indicated and have not been proven to be beneficial. As a result, antibiotics and/or steroids should be considered on a case-by-case basis and their use dependent on bronchoscopic findings.

Complications

Complications secondary to foreign body aspiration can be identified as 1) *emergent* secondary to asphyxiation with airway obstruction, 2) *immediate* with the presence of the foreign body or secondary to surgery to treat it, and 3) *delayed* with secondary lung injury due to the foreign body itself or inflammatory changes in the endobronchial tree secondary to its presence.

In the operating room, the foreign body itself can be difficult to manipulate. This is particularly true with round, smooth-surfaced metallic or glass bodies. These foreign objects may fall into the normal airway, obstructing it while releasing distal secretions from the previously obstructed lung. Either of these events can lead to acute airway obstruction and death. Spherical foreign bodies need to be controlled, and

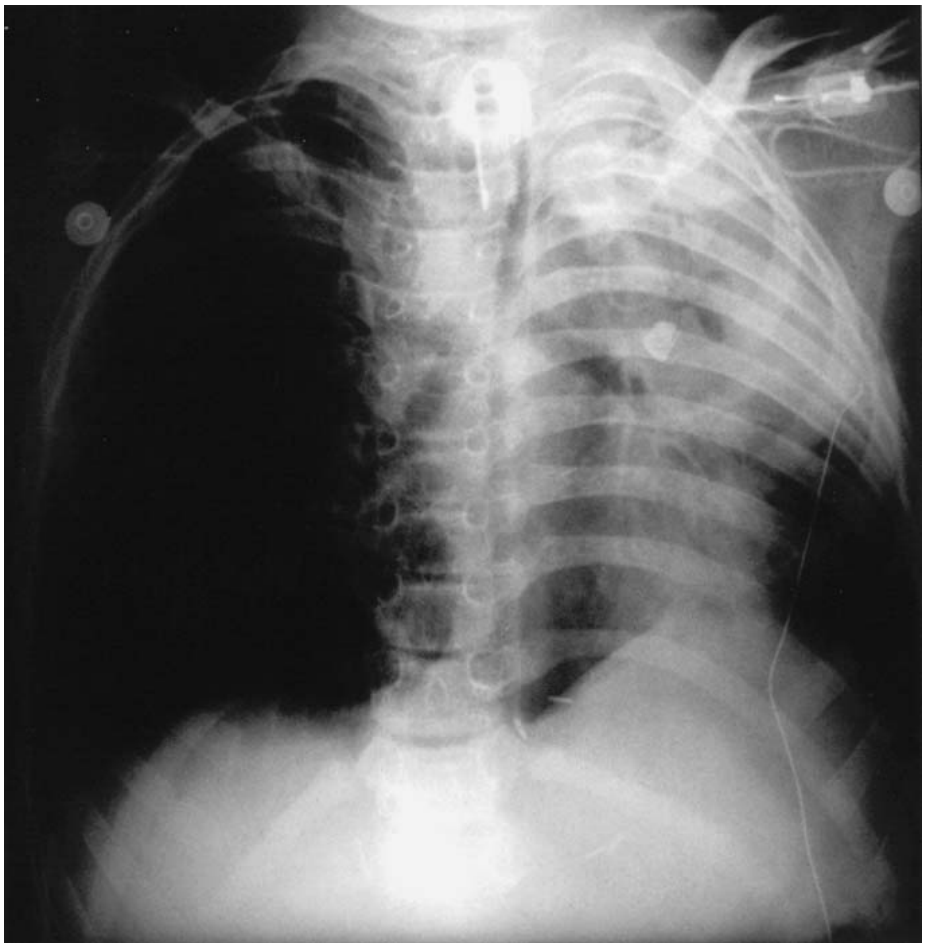

FIGURE **36-6** *Neurologically impaired child with a history of pneumonia and coughing 2 months prior to this evaluation. Calcific density in the left upper lobe is appreciated, which proved to be an aspirated tooth. It could not be extracted endoscopically, and required thoracotomy with bronchotomy for extraction.*

in that instance, the Fogarty catheter balloon technique is inappropriate. Control of the spherical foreign body with a Dormia stone basket during extraction is the safest course. The endoscopist should also have a well-functioning suction device immediately available to aspirate any overflow of secretions.

If there is a delay in diagnosis, when the aspiration event is not witnessed, it is not unusual for the initial evaluation to occur weeks to years after the aspiration event. Organic foreign material, in particular nuts, typically cause an intense inflammatory response in the tracheobronchial mucosa (Figure 36-7). This may lead to secondary granuloma formation and a localized tracheobronchitis, and in the most severe cases, bronchial stenosis. As the obstruction worsens, the distal lung segment will collapse. With time, lung abscess formation and bronchiectasis will occur. Rarely, erosion of the foreign body may lead to the development of a bronchopleural fistula with the appearance of pneumothorax and/or pneumomediastinum. Bronchiectasis with hemoptysis has been reported and deaths secondary to massive hemoptysis have been noted.[40,41]

Bronchiectasis deserves special mention as a known complication of a retained endobronchial foreign body.[42] However, foreign body aspiration as the cause of bronchiectasis is only a minor subset of all patients with bronchiectasis. In the large series from Edinburgh, 8 of over 1,000 patients with known bronchiectasis were found to have an aspirated foreign body.[43] Four of the 8 patients required bronchial resection because of the severity of the disease, but 4 improved without requiring additional intervention.

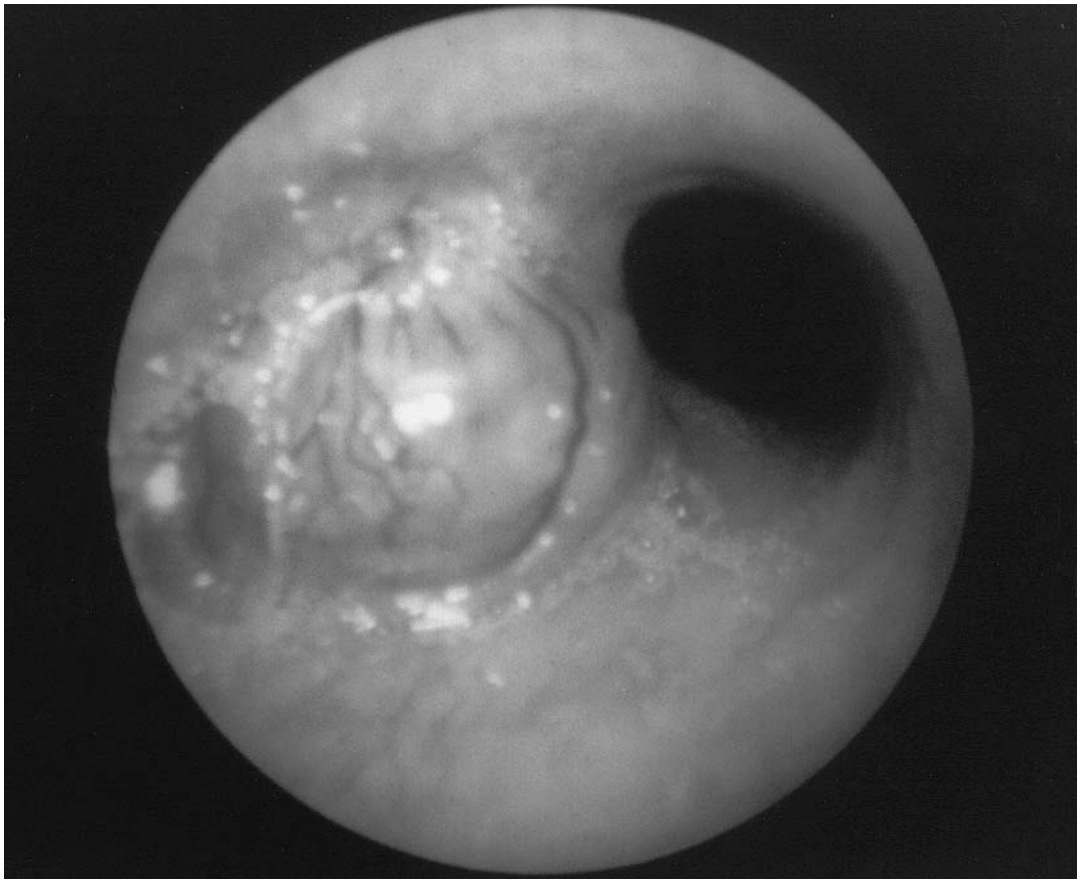

FIGURE 36-7 *Peanut in the left mainstem bronchus with early inflammatory changes.*

If the foreign body can be extricated, then a 4 to 6 week period of observation is indicated to see if the pulmonary changes are reversible.

Timothy grass, a prevalent grass on most continents, as well as some grains, are common causes of bronchiectasis complicating aspiration. This form of aspiration often leads to a progressive, insidious, and eventually debilitating respiratory process. Unlike larger foreign materials, the flowering heads of the grasses and grains can propel the aspirated material into the more distal airway, leading to these parenchymal changes. Clinical symptoms in those instances often are mild chronic cough that may or may not be productive. Occasionally, as the disease progresses, other symptoms of infection such as fever, lethargy, and failure to thrive may occur. An alert clinician must consider this type of aspiration in a patient whose pulmonary symptoms persist or whose pneumonia recurs following adequate treatment. Endoscopic extraction of the grass or grain approaches only 30%, as opposed to the larger more proximal foreign bodies.

Conclusions

Foreign body aspiration can be a difficult diagnosis to establish. However, in a patient, particularly a child, who presents with an episode of sudden choking or spasmodic coughing, an aspiration event should be considered as a primary diagnosis. Radiographic studies may be helpful, but no test is sufficiently sensitive and specific to exclude the diagnosis of foreign body aspiration. A good history and examination is of greater import than a confirmatory x-ray study. Most importantly, the diagnosis of an airway foreign body cannot be excluded without bronchoscopy.

In the patient who presents with an episode of choking or coughing, and who has physical findings on pulmonary examination to suggest the presence of a foreign body (ie, wheezing, rhonchi, decreased breath sounds), a bronchoscopy should be performed. There is no need for additional radiographic studies. In the patient who has a history consistent with possible aspiration but a normal examination, x-ray studies including inspiratory and expiratory films, bilateral decubitus films, or fluoroscopy of the chest may be helpful. If there is a good history for aspiration, the examination is normal, and the radiographic studies are normal, then careful follow-up with repeat x-rays within 1 week of the event would be appropriate.

It is important to remember that early identification of an aspirated foreign body will lead to fewer long-term problems. Therefore, the endoscopist should expect that a certain percentage of patients (generally accepted at 10 to 20%) will not have a foreign body found on bronchoscopy. Otherwise, some foreign bodies will be missed, and complications from foreign body aspiration are almost always secondary to a delayed diagnosis.

There is an increasing place for fiber-optic bronchoscopy in evaluation for possible foreign body aspiration. In patients who have a chronic respiratory process even without a history of aspiration, one must consider foreign body aspiration as a cause of the pulmonary symptoms. Flexible bronchoscopy is a good way to survey the tracheobronchial tree. If a foreign body is found, then it is reasonable in adults and generally recommended in children to remove the flexible bronchoscope and perform a rigid bronchoscopy for extraction of the foreign material. As experience accrues and instrumentation improves, flexible bronchoscopy and extraction may become the major treatment option for this problem.

References

1. Bradwell S. Helps for Suddain accidents endangering life. London: Thomas Purfoot; 1633.
2. Gross S. A practical treatise on foreign bodies in the air-passages. Philadelphia: Blanchard and Lea; 1854.
3. Killian G. Removal of a bone splinter from the right main bronchus by direct laryngoscopy. Munch Med Wsch 1897;44:86.
4. Clerf LH. Historical aspects of foreign bodies in the air and food passages. S Med J 1975;68:1449–54.
5. Jackson C. Foreign bodies in the trachea, bronchi, and esophagus—the aid of esophagoscopy, bronchoscopy, and magnetism in their extraction. Laryngoscope 1905;15:257–81.
6. Jackson C. Observations on the pathology of foreign bodies in the air and food passages, based on the analysis of 628 cases. Surg Gynecol Obstet 1919;28:201–24.
7. Jackson C. Bones as overlooked foreign bodies in the lung. Arch Otolaryngol 1930;12:499–507.
8. Harris CS, Baker SP, Smith GA, Harris RM. Childhood asphyxiation by food. A national analysis and overview. JAMA 1984;251:2231–5.
9. Haugen R. The café coronary: sudden deaths in restaurants. JAMA 1963;186:142–3.
10. Mittleman RE, Wetli CV. The fatal cafe coronary: foreign-body airway obstruction. JAMA 1982;247:1285–8.
11. Whitlock WL, Brown CR, Young MB. Tracheobronchial foreign bodies. Ann Intern Med 1990;113:482–3.
12. Wiseman NE. The diagnosis of foreign body aspiration in childhood. J Pediatr Surg 1984;19:531–5.
13. Black RE, Johnson DG, Matlak ME. Bronchoscopic removal of aspirated foreign bodies in children. J Pediatr Surg 1994;29:682–4.
14. Barrios Fontoba JE, Gutierrez C, Lluna J, et al. Bronchial foreign body: should bronchoscopy be performed in all patients with a choking crisis? Pediatr Surg Int 1997;12:118–20.
15. Metrangelo S, Monetti C, Meneghini L, et al. Eight years' experience with foreign-body aspiration in children: what is really important for a timely diagnosis? J Pediatr Surg 1999;34:1229–31.
16. Wood RE, Gauderer MW. Flexible fiberoptic bronchoscopy in the management of tracheobronchial foreign bodies in children: the value of a combined approach with open tube bronchoscopy. J Pediatr Surg 1984;19:693–8.
17. Baharloo F, Veyckemans F, Francis C, et al. Tracheobronchial foreign bodies: presentation and management in children and adults. Chest 1999;115:1357–62.
18. Zissin R, Shapiro-Feinberg M, Rozenman J, et al. CT findings of the chest in adults with aspirated foreign bodies. Eur Radiol 2001;11:606–11.
19. Dajani AM. Bronchial foreign-body removed with a Dormia basket. Lancet 1971;1:1076–7.
20. Tsueda K, Sjogren S, Debrand M, Pulito AR. Wire basket extraction of foreign bodies from the tracheobronchial tree of small children. J Ky Med Assoc 1981;79:13–5.
21. McCullough P. Wire basket removal of a large endobronchial foreign body. Chest 1985;87:270–1.
22. Moss R, Kanchanapoon V. Stone basket extraction of a bronchial foreign body. Arch Surg 1986;121:975.
23. Ullyot DG, Norman JC. The Fogarty catheter: an aid to bronchoscopic removal of foreign bodies. Ann Thorac Surg 1968;6:185–6.
24. Hunsicker RC, Gartner WS Jr. Fogarty catheter technique for removal of endobronchial foreign body. Arch Otolaryngol 1977;103:103–4.
25. Saw HS, Ganendran A, Somasundaram K. Fogarty catheter extraction of foreign bodies from tracheobronchial trees of small children. J Thorac Cardiovasc Surg 1979;77:240–2.
26. Kosloske AM. The Fogarty balloon technique for the removal of foreign bodies from the tracheobronchial tree. Surg Gynecol Obstet 1982;155:72–3.

27. Wiesel JM, Chisin R, Feinmesser R, Gay I. Use of a Fogarty catheter for bronchoscopic removal of a foreign body. Chest 1982;81:524.
28. Martinot A, Marquette CH, Ramon P, Leclerc F. Foreign body aspiration in childhood: management algorithm. Eur J Emerg Med 2000;7:77–8.
29. Prakash UB, Midthun DE, Edell ES. Indications for flexible versus rigid bronchoscopy in children with suspected foreign-body aspiration. Am J Respir Crit Care Med 1997;156:1017–9.
30. Lan RS, Lee CH, Chiang YC, Wang WJ. Use of fiberoptic bronchoscopy to retrieve bronchial foreign bodies in adults. Am Rev Respir Dis 1989;140:1734–7.
31. Limper AH, Prakash UB. Tracheobronchial foreign bodies in adults. Ann Intern Med 1990;112:604–9.
32. Lan RS. Non-asphyxiating tracheobronchial foreign bodies in adults. Eur Respir J 1994;7:510–4.
33. Debeljak A, Sorli J, Music E, Kecelj P. Bronchoscopic removal of foreign bodies in adults: experience with 62 patients from 1974–1998. Eur Respir J 1999;14:792–5.
34. Weissberg D, Schwartz I. Foreign bodies in the tracheobronchial tree. Chest 1987;91:730–3.
35. Fieselmann JF, Zavala DC, Keim LW. Removal of foreign bodies (two teeth) by fiberoptic bronchoscopy. Chest 1977;72:241–3.
36. Burrington JD, Cotton EK. Removal of foreign bodies from the tracheobronchial tree. J Pediatr Surg 1972; 7:119–22.
37. Cotton EK, Abrams G, Van Houtte J, Burrington JD. Removal of aspirated foreign bodies by inhalation and postural drainage: a survey of 24 cases. Clin Pediatr (Phila) 1973;12:270–6.
38. Law D, Kosloske AM. Management of tracheobronchial foreign bodies in children: a reevaluation of postural drainage and bronchoscopy. Pediatrics 1976;58:362–7.
39. Kosloske AM. Tracheobronchial foreign bodies: back to the bronchoscope and a balloon. Pediatrics 1980; 66:321–3.
40. Slim MS, Yacoubian HD. Complications of foreign bodies in the tracheobronchial tree. Arch Surg 1966;92:388–93.
41. Dudgeon DL, Parker FB Jr, Frittelli G, Rabuzzi DD. Bronchiectasis in pediatric patients resulting from aspirated grass inflorescences. Arch Surg 1980;115:979–83.
42. Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 31-1998. An eight-year-old boy with bronchiectasis. N Engl J Med 1998;339:1144–51.
43. Kurklu EU, Williams MA, Le Roux BT. Bronchiectasis consequent upon foreign body retention. Thorax 1973;28:601–2.

Laser Therapy for Tracheobronchial Lesions

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Lasers have been available for the treatment of diseases affecting the upper and lower airway since the early l970s.[1] Refinements in the technology and in the delivery systems have taken place over the past 30 years, extending the usefulness of lasers in laryngology and bronchology. It is extremely important, however, to appreciate the indications, contraindications, and precautions when using this technology. Lasers are powerful surgical tools which, when used appropriately, can be extremely useful. Conversely, there is always the potential for disastrous complications when safety and suitable indications are not guiding principles. Since there are several lasers to choose from, it is extremely important for the clinician to understand the basic laser–soft tissue interaction for each wavelength and the unique safety precautions necessary for each laser type. This understanding permits application of laser technology to provide optimal results for the appropriate clinical situation. Lack of understanding often leads to misuse and abuse of lasers, causing complications and potential disasters such as tracheobronchial perforation, massive hemorrhage, airway obstruction, and damage from fire. This chapter reviews the basic soft tissue interaction of commonly used lasers in the airway and presents the authors' recommendations for the use of lasers in the tracheobronchial airway based upon the senior author's (SMS) 25-year experience.

The lasers most commonly used in the airway are the CO_2 laser, neodymium:yttrium-aluminum-garnet (Nd:YAG) laser, potassium titanyl phosphate (KTP) laser, argon laser, holmium:YAG laser, diode laser, and the pulsed dye laser (PDL) (Table 37-1). For practical purposes, only the CO_2 and the Nd:YAG lasers will be emphasized since they are the most important lasers for clinical application. Since these lasers are worthless unless they can be delivered to treat the pathology with precision and clarity, special mention of delivery systems will be made as well as ideal ventilatory techniques of anesthesia.

Historical Review and Soft Tissue Effect of Lasers in the Airway

CO_2 Laser

The carbon dioxide laser is the mainstay of lasers for the treatment of conditions involving the larynx and upper trachea. The 10.6 μm wavelength produced is well absorbed by water, and since 90% of soft tissue is composed of water, the laser is ideal for incision, excision, and vaporization of soft tissue. The development

Table 37-1 Different Laser Absorbers and Laser Wavelengths

Laser	*Wavelength*	*Absorption*
Argon	514 nm	Hemoglobin
KTP 532	532 nm	Hemoglobin
Diode laser	810 nm	Pigment (indocyanine green)
Nd:YAG	1.06 μm	Pigmented tissue
Ho:YAG	2.1 μm	Water ($\frac{1}{6} < CO_2$)
CO_2	10.6 μm	Water
PDL	585 nm	Hemoglobin

Ho:YAG = holmium:yttrium-aluminum-garnet; KTP 532 = potassium titanyl phosphate at 532 nm;
Nd:YAG = neodymium:yttrium-aluminum-garnet; PDL = pulsed dye laser.

of the micromanipulator, an optic system coupling the laser source to the operating microscope, was key in providing microprecision and the ability to achieve small vessel (< 0.5 mm) hemostasis.

The CO_2 laser was first clinically applied in the airway by Strong and Jako in 1972 for a variety of laryngeal lesions which included laryngeal papillomas, carcinoma in situ, and vocal cord nodules, among others.[1] Over time, the number of applications and techniques using the CO_2 laser has multiplied, and today, it is considered the mainstay of treatment for laryngeal hemangiomas, recurrent laryngeal papillomatosis, and for properly selected cases of laryngeal cancer and laryngeal stenosis.[2–5]

CO_2 Laser–Soft Tissue Interaction. The 10.6 μm infrared wavelength of the carbon dioxide laser undergoes a 90% absorption by water contained in soft tissue. This laser light is then transformed into heat within tissue, raising the tissue temperature to 100°C and vaporizing the water content. The remaining 10% is carbon residue, which should be removed manually to avoid inflammatory tissue reaction. The steam of vaporization needs to be removed with an efficient suction device to avoid collateral damage from the hot vapors.

There are three important parameters that should be kept in mind when delivering laser energy. These are power (w), exposure time (sec), and power density (spot size). Of these basic parameters, time exposure is the most important. Limiting the time of laser exposure with short applications minimizes heat conduction within tissue, thus limiting collateral damage. Basic studies of soft tissue interaction in canine trachea showed excellent depth of penetration with minimal damage to the adjacent tissue.[6] The energy curve of the focused CO_2 laser (micromanipulator) is in the shape of an inverse gaussian curve; that is, the maximal power is at the center of the beam, fading at the margins.

A major disadvantage of the CO_2 laser is that it can not be transmitted through flexible fibers, which limits its usefulness in the trachea and bronchi. The CO_2 laser must be delivered from the source, using a somewhat awkward articulated mirror system. Other delivery systems such as a semiflexible hollow waveguide can also be used, however, this does not provide a focused, "clean" beam and suffers from imprecise soft tissue interaction. The CO_2 laser is usually and best used in a focused fashion through a microspot micromanipulator (250 micron spot size) for applications in the larynx and subglottis down to the first tracheal ring, using a laryngoscope placed on a suspension system, thus freeing up the surgeon's hands. We prefer a standard diagnostic laryngoscope such as the Dedo design (Pilling Co., Philadelphia, PA), inserting the laryngoscope between the vocal cords and exposing the subglottic larynx up to the first tracheal ring.

Nd:YAG Laser

The Nd:YAG laser has a wavelength of 1.06 microns, which is poorly absorbed by water and, therefore, penetrates tissue deeply. It is well absorbed by pigment (deep purple), vascular tissue, and the char of carbon

deposits (see Table 37-1). The energy is not dissipated at the surface, as is the case with the CO_2 laser; it scatters within the tissue depending on the degree of tissue pigmentation for absorption. The YAG laser in a noncontact mode is a "cooker" or coagulator of tissue as opposed to the more precise cutting effect of the CO_2 laser. In the less common contact mode, the YAG laser is somewhat wavelength independent and the energy concentrates at the tip of the fiber and causes limited vaporization of tissue and little damage to the surrounding tissue. The contact mode is good for coagulating blood vessels less than 1 mm in diameter; its effect on soft tissue is similar to the CO_2 laser.[6]

The YAG laser can be transmitted through commonly available flexible quartz fibers that make possible its use in the tracheobronchial tree. Using the nonfocused fiber, the laser beam diverges approximately 10° as it leaves the fiber; the closer the fiber is to the tissue, the smaller the spot size. The fiber is normally used at 0.5 to 1 cm from the target. The fiber can be connected to a series of contact tips of differing geometric shapes for contact mode use for the purpose of cutting, coagulating, and ablating. Care must be taken to deliver the beam in short exposures of 1 sec or less at a power setting at 40 W or less. Continuous exposure of the Nd:YAG laser at a high power setting (above 40 W) may result in an explosion of tissue, caused by concentration of high energy below the tissue surface that creates an expanding cavity.

The Nd:YAG laser wavelength has little visible effect on colorless tissue; the laser beam readily traverses it, causing thermal damage to the more pigmented underlying tissue or structures, such as blood vessels. It is important to note that the white color of the normal tracheal cartilage will not absorb the laser energy unless it becomes dehydrated from the continuous heat and char to form an absorbing element. The laser will otherwise pass through the unpigmented wall and may be absorbed by the underlying vascular structure and lung tissues. It is well known that the thermal effects of the Nd:YAG laser go well beyond its immediate area of visible impact; what is seen is not necessarily what you get.[6] A study by Shapshay, comparing the CO_2 laser with different Nd:YAG delivery systems, points out the pitfalls in using the Nd:YAG laser.[6] Thermal damage from using the YAG laser in the normal tracheobronchial tree delays healing and reepithelialization, and may cause scarring. Mucosal charring and blood deposition on the tracheal wall enhance the absorption of the Nd:YAG laser beam. Rapid propagation of thermal energy can ensue, causing tracheal perforation.

KTP and Argon Lasers

The KTP (wavelength 532 nm) and the argon (wavelength 518 nm) lasers have similar characteristics. Both lasers operate in the visible region of the spectrum and can be delivered through flexible quartz fibers. These lasers are well absorbed by pigmented tissue and hemoglobin, and only weakly absorbed by pale, poorly pigmented tissue. These characteristics make them good superficial coagulators, with a fairly good ability to ablate pigmented tissue. Compared with the Nd:YAG laser, the depth of penetration and scatter in soft tissue is limited, making the Nd:YAG laser a better coagulator with better hemostasis for vascular tumors. Very small spot sizes can be achieved with the KTP or argon lasers depending on the optics used, creating high-power densities capable of cutting and ablating tissue, independent of the wavelength of absorption. The ability of the KTP laser to pass through flexible fiber-optic fibers of less than a millimeter makes this laser a good choice for applications in the pediatric airway.[7]

Studies of the effect of a high-powered argon laser on the tracheobronchial tree showed extensive subepithelial damage, with a somewhat variable delayed reaction between the time of application of the laser and the detectable break in the epithelium.[8] Postoperative edema and acute inflammation lasted for 3 and 7 days, respectively. The extent of injury was 30% greater than the original defect. The unpredictable effects of the argon laser on soft tissue limit its application in the airway.[8]

Other Lasers: Diode, Holmium, 585 nm Pulsed Dye

Diode Laser. The near infrared wavelength (810 nm) of the diode laser allows a relatively deep penetration of soft tissue. Its soft tissue effects are somewhat similar to those of the Nd:YAG. We have used this diode laser for mucosal graft soldering in the treatment of posterior glottic stenosis, combining indocyanine dye with fibrin glue.[9] Ongoing research is focused on creating a higher power diode laser, which may be a cheaper, more portable laser alternative in the future.

Holmium:YAG Laser. The holmium:YAG laser provides a pulsed beam with an infrared wavelength of 2.1 μm. Studies indicate that it is best used on bone, since it can provide relatively high energy (up to 2.0 J/pulse) in a short period of time (250 μsec), which is less than the thermal relaxation time of tissue. The senior author (SMS) has limited experience in using the holmium:YAG laser at 1.2 to 2.0 J/pulse at 3 Hz for cases of tracheopathia osteoplastica.[10] However, the holmium:YAG laser has an explosive effect on tissue absorption and would not be suitable for treatment of other conditions in the tracheobronchial wall.

Pulsed Dye Laser. The pulsed dye laser (PDL) at 585 nm has excellent (perhaps the best) absorption for hemoglobin of all the lasers previously mentioned. This laser has been used most recently in the larynx for treatment of recurrent respiratory papillomatosis.[11,12] Its high affinity for oxyhemoglobin allows microvascular targeting of the papillomatous lesions, leaving the surface mucosa intact. This results in a minimally invasive procedure, with fewer soft tissue complications than other commonly used techniques. Although this laser can be delivered through a flexible fiber, it may have very limited use in the trachea, perhaps for sessile papillomas to control growth and for superficial vascular malformations.

Laser Applications in the Subglottic Larynx and Cervical Trachea

The CO_2 laser has remained the laser of choice for precise excision of scar tissue and treatment of benign tumors in the subglottic area. However, this laser possesses poor hemostatic properties and is unable to deliver focused energy through a flexible fiber, resulting in severe limitations for treatment of hypervascular tumors and lesions located in the distal airway.

The Nd:YAG laser has become the laser of choice for vascular tumors such as venous malformations and obstructing tumors of the airway. Diverse conditions involving the subglottic larynx and cervical trachea require different lasers. A positive treatment outcome depends on the adequate selection of the laser to be used, according to its soft tissue interactions, emphasizing the histologic properties of the pathological condition to be treated. The use of lasers in the tracheobronchial tree is appropriate for a number of conditions, both congenital and acquired, involving patients in every age group. Some of these lesions include vascular malformations, systemic conditions, viral infections, and malignant conditions (Table 37-2).

Exposure and Technique

Adequate exposure is extremely important to avoid complications from the use of the CO_2 laser. We recommend the use of a "subglottic laryngoscope" with an attached Venturi Jet ventilation system when using a micromanipulator to deliver the CO_2 laser energy. Ventilation is usually maintained with a Venturi apparatus and anesthesia is provided using intravenous compounds with muscle relaxation techniques. Since the use of intravenous anesthesia and muscle relaxation can severely compromise the patient's ventilation, spontaneous respiration with topical anesthesia is not recommended for cases with airway obstruction, until an adequate airway is established.

A tracheoscope is preferred to the long standard ventilating bronchoscope when dealing with lesions of the subglottis or upper trachea. Ventilating bronchoscopes are awkward to use in the upper airway and

Table 37-2 Choice of Laser for Various Conditions

Airway Lesions	*Choice of Laser*
Vascular Lesions	
Subglottic hemangioma	CO_2 laser
Venous malformation	Nd:YAG
Benign Tumors	CO_2 laser
Vocal Cord Lesions	
Laryngeal papillomas	CO_2 laser or PDL 585 nm
Chondroma	Nd:YAG
Tracheal papillomas	Nd:YAG or KTP
Metabolic/Idiopathic	
Sarcoid	Larynx: CO_2 laser; Trachea: Nd:YAG
Amyloidosis	Larynx: CO_2 laser; Trachea: Nd:YAG
Locally Invasive Tumors	
Thyroid	Nd:YAG
Metastatic Tumors	
Breast, Colon, Kidney	Nd:YAG
Primary Malignant Tumors	
Squamous cell	Larynx: CO_2 laser; Trachea: Nd:YAG
Carcinoid	Nd:YAG
Adenoid cystic	Nd:YAG
Lymphoma	Nd:YAG
Melanoma	Nd:YAG

have the disadvantage of allowing loss of ventilation through the ventilating openings at the glottis. On the other hand, for lesions involving the distal trachea, main bronchi, and bronchus intermedius, ventilating bronchoscopes are the preferred method for laser application. We prefer to use a ventilating Storz tracheoscope (Karl Storz Endoscopy, Culver City, CA) with telescopic imaging through the video (Figure 37-1). This bronchoscope incorporates an internal light source system, which offers the possibility of use without a telescope, thus allowing the use of larger instruments for tumor removal. Air leakage from the open end of the bronchoscope does not constitute a problem since most lesions are treated using a Venturi jet ventilation technique. Rigid bronchoscopy should always be performed in an operating room, with all necessary precautions to guarantee safe use of lasers.

The average laser power used with the Nd:YAG laser varies with the pathology. Vascular tumors with good laser absorption and respiratory papillomas with good vascular flow require 20 to 30 W, whereas for malignant tumors, in which the goal is to obtain good coagulation, 30 to 40 W are normally used. When delivering the laser energy, it is critical to maintain both adequate visualization and a clean and dry surgical field. For this purpose, we use a 0° telescope with one or two semiflexible suctions to remove steam caused by vaporization and simultaneously keep the field free of blood. The fiber can be guided with a steering device or an optical bridge but we prefer free use through an open system for ease of application and rapid removal of secretions, blood, or tumor fragments.

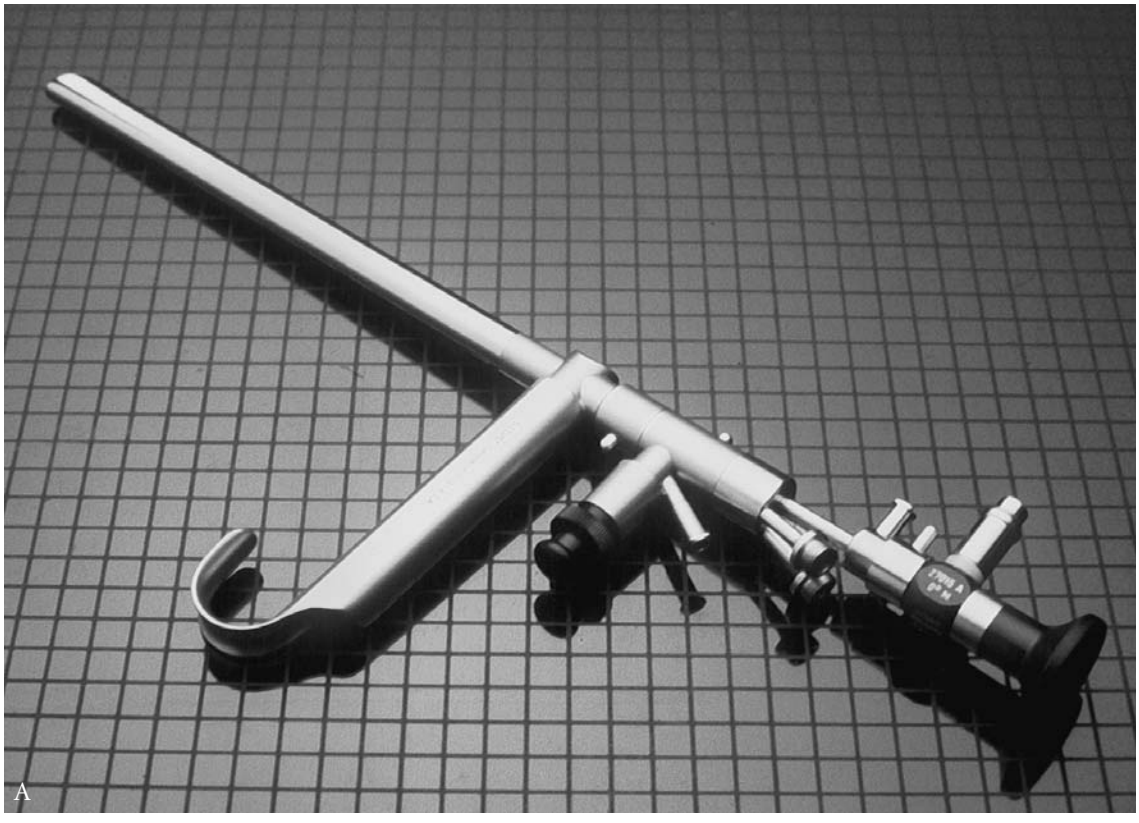


FIGURE 37-1 A, *Storz rigid bronchoscope.* B, *Distal end of the bronchoscope.*

Specific Applications

Subglottic Stenosis

Subglottic stenosis has multiple causes, but it is most commonly associated with mechanical trauma secondary to endotracheal intubation. Adequate treatment requires a complete preoperative work-up, which should include a thin slice computed tomography (CT) scan or magnetic resonance imaging (MRI) and a flow volume loop. However, a reliable diagnosis with mapping of the lesion can only be established by direct endoscopy, performed under general anesthesia. Using telescopic optics and palpation, the endoscopist should be able to establish the length of the lesion, the extent of cartilage damage, and the presence of vocal cord fixation or paresis. All these factors combined with the general status of the patient should be taken into account when deciding on the best possible treatment.

Endoscopic management of subglottic stenosis with the CO_2 laser was first described by Strong and colleagues in 1979.[5] Today, endoscopic techniques employing the CO_2 laser continue to be used successfully in properly selected cases. Adequate patient selection is extremely important. A group of factors have been identified which can influence the success or failure of endoscopic laser procedures. Best results are achieved with endoscopic laser treatment when the length of the lesion is less than 1 cm, the stenosis is limited, comprising mainly soft tissue, with minimal cartilage involvement, and good vocal cord function is maintained.[13,14] Cases in which a bilateral fixation or a paresis coexists with the subglottic stenosis must be addressed as well. Some factors that may play a deleterious role in the final treatment outcome are the presence of active inflammation of the airway (eg, bacterial, fungal, preexisting tracheostomy, or active Wegener's vasculitis), and the presence of conditions that may impair wound healing such as diabetes, hypothyroidism, and chronic steroid use.[15–19]

Since 1987, we have employed laser incision and dilation technique to treat selected cases of subglottic and tracheal stenosis (Figure 37-2).[19] For this procedure, we prefer the microspot delivered CO_2 laser with a 0.25 mm spot size, using a 16× magnification in the microscope. The laser power is set at superpulse, usually at 5 W and 30 pulses/sec.

Under adequate exposure, three to four radial incisions are made through the scar tissue without causing a circumferential defect, since this is likely to induce rescarring. Care is taken to use short pulses of energy exposure of less than 1 sec; usually 0.2 to 0.5 sec to avoid transmission of heat, which could result in further scarring. Islands of mucosa are preserved to assist with reepithelialization. The treated area experiences a race between scar formation and reepithelialization. The incised scar is further stretched or dilated with the use of sequential sizes of ventilating bronchoscopes, used in a "corkscrew" fashion as atraumatically as possible, until reaching an 8.5 mm ventilating bronchoscope. The CO_2 microspot delivery system is preferred, due to the precise cutting nature of the focused beam, rather than a waveguide delivery system, which is nonfocused and subsequently less precise. The CO_2 laser is preferred to the YAG and other lasers, due to both its superior precision and better control of the depth of penetration.

Recent evidence shows that scar formation is retarded with the topical application of mitomycin-C.[20–22] Mitomycin-C modulates the wound healing response by inhibiting proliferation of fibroblasts. Our experience with endoscopic laser surgery, followed by intraoperative application of 0.4 mL of mitomycin-C in 11 patients, has shown improvement both in the size of the airway and in the symptoms.[22] However, despite significant decrease in scar formation, some scarring does occur, and patients may require more than one procedure to further improve symptoms.

Previously, employment of intralesional or systemic steroids has had poor results, since these not only modulate the inflammatory response of fibroblasts but also delay the reepithelialization process. Postoperatively, it is important to treat the patient with cool mist inhalation for several days and to use perioperative systemic steroids (48 hours), antireflux medication (30 days), and antibiotics (7 days) to prevent

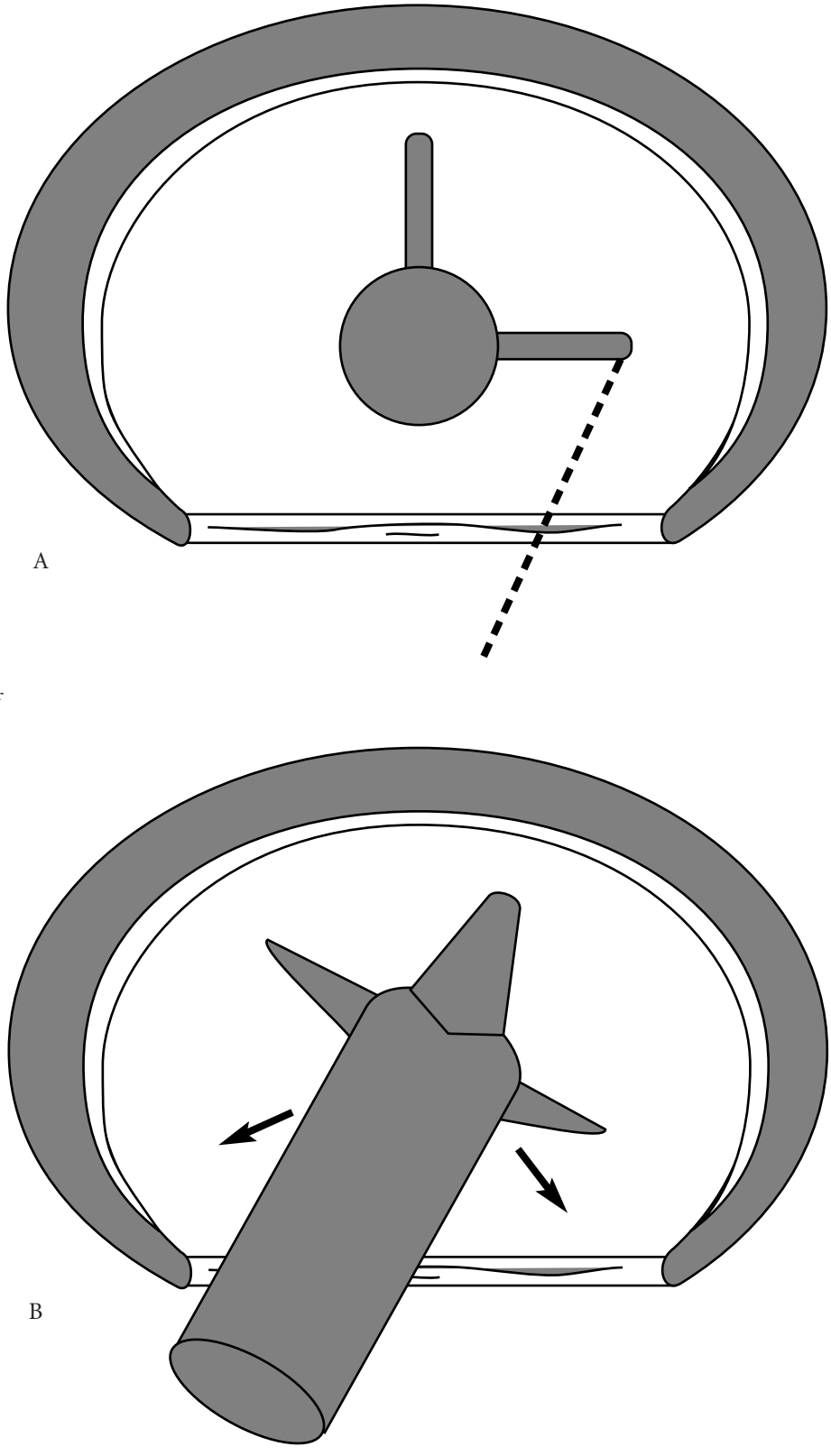


FIGURE 37-2 A, *Treatment of web-like stenosis with laser radial incisions.* B, *Dilation of the stenotic area with a bronchoscope.*

infection or crusting, which can lead to airway obstruction. We avoid tracheostomy in the acute presentation since this would damage the normal trachea and add a risk of infection. In our 15-year experience, this technique has had a success rate of approximately 70%. However, some patients require up to three times of endoscopic laser treatment before an adequate airway is achieved. In cases in which there is no improvement, or recurrent stenosis, we recommend open resection and tracheothyroid anastomosis rather than rib graft laryngotracheoplasty. The technique is described in Chapter 25, "Laryngotracheal Reconstruction."

Other techniques employing the laser to treat subglottic stenosis have been described. In 1984, Dedo and Sooy introduced the micro-trapdoor flap technique.[23] This technique is performed by making a crescent-shaped incision in the mucosa overlying the stenosis. A microelevator is used to elevate a mucosal flap from the superior edge of the stenosis to its inferior aspect. The laser is used to vaporize the scar tissue causing the stenosis. Once the scar tissue has been vaporized, the flap is carefully repositioned. The laser beam is used in a defocused mode to weld the edges of the incision back together or the flap is simply allowed to re-adhere. A success rate of 90% has been reported in lesions of less than 10 mm in length. Our success rate has been poor since it is technically difficult to preserve the elevated mucosal flap.

Other techniques using laser to treat subglottic and posterior stenoses involve the use of the diode laser and exogenous dyes for graft soldering.[9] In this technique, a buccal mucosal graft, slightly larger than the size of the surgical wound from the scar resection, is harvested. Submucosal fat is removed and the graft tailored to the size and shape desired. The graft is placed on the wound, which has been covered with indocyanine green mixed with autologous fibrin glue, and soldered using the 810 nm diode laser in a noncontact mode with a 1 mm spot size at 400 to 800 nW power. Experience with this technique has been limited and a larger series of patients would have to be studied to prove its effectiveness and safety.

The CO_2 laser waveguide or bronchoscopic coupler system can be used for lesions located in the mid to distal trachea, where laser energy can not be delivered with precision using a micromanipulator. Alternatively, the KTP laser or contact Nd:YAG laser could also be used to deliver energy through flexible fibers.

Subglottic Hemangioma and Other Vascular Lesions

The CO_2 laser has been the laser and treatment of choice for over 15 years for hemangiomas with limited involvement of the subglottic airway.[24,25] The CO_2 laser provides an excellent choice of treatment because of its soft tissue interaction and the ability to achieve microhemostasis for capillary sized blood vessels. Most importantly, it offers the advantage of avoiding tracheostomy during the proliferative phase of the hemangioma. Approximately 80% of subglottic hemangiomas can be eradicated with one or two laser treatments. CO_2 laser surgery is often combined with steroids and interferon for management of subglottic hemangiomas.[26] The main complication following CO_2 laser treatment for subglottic hemangioma is subglottic stenosis, which can occur in up to 20% of patients.[25]

Low-flow venous malformations, on the other hand, are best treated with the Nd:YAG laser, used in a noncontact fashion.[27,28] These lesions normally appear in the supraglottic larynx, but may be found in the trachea as well. In the majority of adult laryngeal venous malformations, a policy of nonintervention combined with annual observation and photo documentation is recommended unless there is airway compromise, dysphagia, bleeding, or severe voice impairment. Since these lesions are usually not discrete and well defined by a capsule, an external surgical approach for removal is extremely difficult to perform and therefore not recommended. For the most part, we have used endoscopic laser photocoagulation to reduce the size of these malformations. Good results with minimal bleeding are achieved with low power (15 to 20 W of nonfocused energy and less than 0.5 sec exposures). Treatment of large venous malformations should be performed in stages with 4- to 6-month intervals between procedures. Recently, interstitial application of the Nd:YAG laser monitored with MRI or ultrasound has been reported to achieve promising results in the treatment of laryngeal vascular malformations.[29–31]

Respiratory Papillomatosis

Viral papillomas are the most common benign laryngeal tumors in the pediatric population. The disease tends to be more aggressive in children, usually presenting with symptoms of airway obstruction and a higher number of recurrences, whereas in adults it tends to be milder, with hoarseness as the main complaint.

The disease is associated with the human papillomavirus types 6, 11, and occasionally 16.[32] These lesions tend to affect areas of junction between squamous and respiratory epithelium, with the vocal cords being the most commonly affected site.[33] However, these lesions can also occur in the subglottic and tracheal airway. There is no cure for recurrent respiratory papillomatosis, and treatment is directed to the control of its symptoms. CO_2 laser has become the standard treatment since first introduced by Strong and Jako in 1972.[1]

The CO_2 laser treatment of laryngeal papillomas is best delivered with a microspot micromanipulator. Tracheal papillomas, in which the CO_2 laser can not be precisely delivered with a micromanipulator, are best treated with a KTP laser in pediatric cases and a Nd:YAG laser in the adult patient.[34, 35] Treatment of airway papillomas often combines laser resection with the use of systemic drugs and nutritional supplements such as interferon and indole-3-carbinol (I3C).[36] I3C, found in cruciferous vegetables, has the ability to arrest the proliferative effect of estrogen in laryngeal papillomas by altering its metabolic pathway in the cytochrome P_{450}. However, unlike interferon, I3C can not induce regression of papillomas that are already developed.

Our experience with laryngeal papillomas has been chiefly in adult patients. These patients tend to have more sessile lesions, which are well suited for treatment with the CO_2 laser using a "laser painting technique" (Figure 37-3 [Color Plate 9]). This technique relies on delicately ablating the papilloma 50 microns at a time, using the laser in a sweeping fashion, at longer exposure settings such as 0.5 to 1 sec at 2 to 3 W of power. Gentle suctioning and cleaning of the treated area, with a small cottonoid moistened in a 1/1,000 adrenaline solution, is performed to remove char and control bleeding after each laser sweep. As the papilloma base is approached, the laser is intermittently applied using 2 W at 0.5 sec, at 25× magnification in the operating microscope. We recommend using a micro-whistle-tip suction close to the laser site of impact to remove hot steam of vaporization, which can be injurious to the normal epithelium. Papillomas involving the anterior commissure should be managed with special care, leaving at least 1 mm of mucosa untreated to avoid web formation during healing. Interferon-α at 10 units/mL can be injected intralesionally in the anterior commissure, to better control scarring from the laser.

When treating tracheal papillomas, the YAG laser photocoagulates the tumors, using 30 to 40 W for protuberant and partially obstructing lesions, which are then mechanically débrided with the ventilating bronchoscope and optical forceps. If the lesions are sessile and nonobstructing, then low powers of 20 to 30 W are used intermittently to photocoagulate and allow spontaneous sloughing to occur.

Amyloidosis

Amyloidosis is an infiltrative process, wherein a group of fibrous proteins accumulates in the submucosal space and can cause obstruction in time. The condition rarely affects the head, neck, and respiratory tract. The larynx is the most common site of amyloid deposition in the airway, followed by the trachea and bronchus.[37] Since amyloid lesions bleed easily, the Nd:YAG laser is favored because of its hemostatic ability.[38] Palliation is the goal and laser bronchoscopy may be repeated as needed.

Tracheopathia Osteoplastica

This is a rare lesion, where calcified nodules of cartilage appear adjacent to tracheal cartilages (see Chapter 4, "Imaging the Larynx and Trachea," and Chapter 14, "Infectious, Inflammatory, Infiltrative, Idiopathic, and Miscellaneous Tracheal Lesions"). The holmium laser can be used to remove the dense deposits to achieve palliation of symptoms. More than one procedure may be required during the course of the disease.

Obstructing Tracheobronchial Tumors

The application of laser technology to the endoscopic treatment of patients with tracheobronchial disorders was first introduced by Strong and colleagues in 1973, who used the CO_2 laser to ablate peristomal papillomas.[39] The effectiveness and safety of laser application in bronchology were enhanced after the introduction of the Nd:YAG laser in the l980s.[40–42] The special hemostatic qualities of this laser energy make it the most suitable laser for endoscopic removal of malignant tracheobronchial lesions with a propensity for hemorrhage into the airway (Table 37-3). Today, tracheobronchial obstruction secondary to primary bronchogenic carcinoma is the most common indication for Nd:YAG laser bronchoscopy, accounting for 51 to 75% of all cases.[43–46]

The majority of patients presenting with lesions that obstruct the central airways all have tumors that are inoperable, because of mediastinal or lymph node involvement, or lesions that are too central for surgical resection. The most common primary lung tumor treated with the Nd:YAG laser is squamous cell carcinoma, followed by adenocarcinoma (Figure 37-4 [Color Plate 9]).[43] Many of these patients have failed radiation therapy and/or chemotherapy (Table 37-4). Unlike ionizing radiation therapy, the laser may be used repeatedly for the palliation of malignant tracheobronchial obstruction.

Although the CO_2 laser was the initial laser used for tracheobronchial laser therapy (Figure 37-5), its use in the treatment of patients with malignant obstruction is severely limited by poor hemostatic properties.[47,48] The hypervascularity of many malignant endobronchial tumors, such as carcinoids, adenoid cystic carcinomas, and metastasis from renal cell, thyroid, breast, and esophageal carcinomas, is best treated with the Nd:YAG laser because of its excellent coagulation properties (Figures 37-6, 37-7 [Color Plate 9]). Tracheobronchial obstruction caused by endobronchial metastasis constitutes the second most common indication for Nd:YAG laser bronchoscopy, accounting for 10 to 18% of laser bronchoscopies. The Nd:YAG laser has also been used successfully in the treatment of benign conditions causing tracheobronchial obstruction, such as amyloidomas, lipomas, fibromas, and hamartomas.[49,50] Some cases of tracheal stenoses are also amenable to treatment with the Nd:YAG laser. However, attention must be paid to the type of stenosis, since laser treatment of lesions secondary to tracheal collapse without an intraluminal component is contraindicated.

We use the rigid bronchoscope in the majority of procedures because this instrument offers a number of advantages over the flexible bronchoscope. Rigid bronchoscopes offer better control of hemorrhage by use of large suction catheters, assure ventilation with the Venturi jet. They also provide excellent visualization with use of the telescope, and facilitate rapid removal of tumor with large biopsy forceps. The flexible bronchoscope is often a helpful tool, when used through the rigid bronchoscope, for treatment of more distal or upper lobe tumors and for tracheobronchial toilet. In general, however, the exposure to the upper lobes is somewhat difficult and fraught with dangers of hemorrhage from pulmonary artery and vein branches.

Flexible bronchoscopy is most useful for delivery of the YAG laser in the outpatient bronchoscopy suite for treatment of patients with small, noncritically obstructing (less than 50% lumen), distal tumors that require photocoagulation for control of hemoptysis, or for benign lesions such as granulation tissue and papillomas.[51] One advantage of using flexible bronchoscopy is the avoidance of general anesthesia and the inherent risks associated with it. However, this bronchoscopy technique has its limitations, especially

Table 37-3 Indications of Laser Bronchoscopy

Reestablish airway patency (malignant tumor: palliation; benign tumor: resection)
Hemoptysis
Dilation of stenosis
Stent insertion

Table 37-4 Advantages and Disadvantages of Laser Bronchoscopy

Advantages
Use of flexible or rigid bronchoscope
Hemostatic ability
Repeatability of treatment
Rapid relief of symptoms
Prepares airway for stent insertion
Disadvantages
Severe complications*
Special training
Expensive equipment

*See Table 37-5.

when dealing with bulky lesions that may require more than one procedure and when treating highly vascular lesions where an inability to control hemorrhage can lead to severe complications.

Anesthetic Technique

Laser bronchoscopy can be challenging to the anesthesiologist and the endoscopist.[52] The anesthetic method used depends on the type of bronchoscopic technique. Most of the patients are high-risk, with concomitant chronic obstructive pulmonary disease and cardiovascular disease. In these patients, topical anesthesia offers the possibility of avoiding potential complications of general anesthesia, such as arrhythmia, myocardial infarction, CO_2 retention, neuromuscular weakness, and hemodynamic instability. In these cases, topical lidocaine combined with intravenous sedation/anesthesia and assisted ventilation are used. With this topical technique used in high-risk patients, the control of cough is often difficult when working near the carina. If possible, complete control of the airway with relaxation techniques and Venturi jet ventilation is desired, allowing use of the open bronchoscope for rapid application of the laser and removal of obstructing tumor

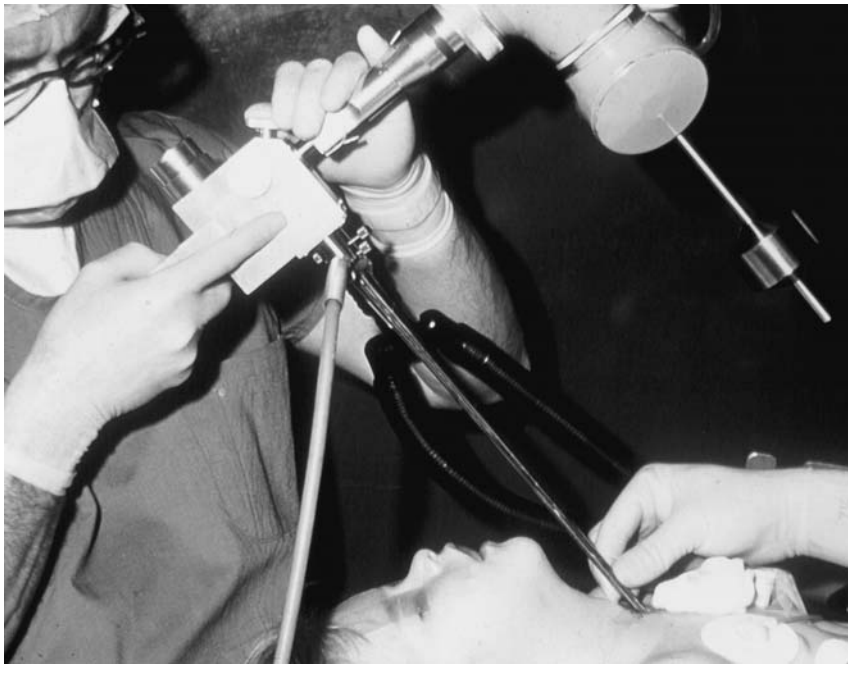


FIGURE 37-5 Early application of laser technology in the trachea. Treatment of peristomal papillomas with the CO_2 laser.

particles, blood, and secretions. Pulse oximetry is recommended to monitor for desaturation. The mean operating time is approximately 30 to 40 minutes and the procedure is usually well tolerated.

Operative Technique

The most common rigid laser bronchoscopes available are the Storz and the Dumon-Harrell. The latter offers separate channels for the telescope, laser fiber, and suction catheters. We prefer to use the Storz ventilation brochoscope with Venturi jet adaptor (7.5 or 8.5) with an open proximal window to introduce the 0° telescope, laser fiber, and one or two semiflexible heat resistant suction catheters. The Dumon-Harrell bronchoscope is commonly used for stent placement following a laser procedure.

The actual technique depends on the site of the tumor and the degree of obstruction. For tracheal tumors, the YAG laser is used to photocoagulate the tumor prior to manipulation with 30 to 40 W at 0.5 to 1 sec. After thorough coagulation, the tumor is mechanically débrided by a shearing, corkscrew action of the rigid bronchoscope and the tumor removed with optical forceps or open rigid suction. The bronchoscope is then inserted distally to suction secretions and to obtain good pulmonary toilet. The major risks to the patient are hypoxemia from accumulation of blood and secretions in the distal airway.[53,54] The bronchoscope is also used to tamponade the tumor base for further hemostasis for about 3 to 5 min while ventilating the patient. Pledgets of cotton soaked with 1/1,000 epinephrine can also be used for hemostasis. The laser can then be used to vaporize tumor at the base of resection to further enlarge the tracheal lumen. It must be stressed that palliation is the goal and no attempt is made to resect the tumor completely since it most likely involves the tracheal wall and, in some cases, extends into the mediastinum.

Several studies have demonstrated the benefits of laser bronchoscopy in palliating dyspnea and hemoptysis.[44,46] Both symptomatic improvement and improvement in spirometry and flow volume loops have been well documented. Waller and colleagues reported an overall improvement in the forced expiratory volume (FEV_1) of 27% and relief of symptoms in 103 of 116 patients.[55] When compared with other palliative treatments, such as radiation therapy, the laser offers rapid resolution of symptoms and the possibility to avoid mechanical ventilation in patients with severe endotracheal obstruction.[56] Photocoagulation with the Nd:YAG laser is also effective in controlling hemoptysis, with a response rate of approximately 60%. Laser bronchoscopy can also be complementary to other types of therapy, such as radiation, chemotherapy, stenting, or surgery.

There is controversy about the best palliative treatment for obstructing airway lesions. From our perspective, no technique is overwhelmingly superior to the others and different factors must be weighed when making a decision. When laser bronchoscopy is compared with bronchoscopy and mechanical débridement, both offer similar advantages in rapid relief of symptoms and repeatability. Laser bronchoscopy may provide better hemostasis and a flexible bronchoscope may gain access to areas not reachable with the rigid bronchoscope. On the negative side, laser bronchoscopy requires expensive instruments and special training. We would like to emphasize this last issue since a well-trained team is mandatory to obtain optimal results and to swiftly manage complications. Nowadays, when "cost effectiveness" in medical procedures is under great scrutiny, more economical techniques such as bronchoscopic mechanical débridement and electrocautery have an advantage. The most important factor in the decision should be the experience and the treating physician's level of comfort with the technique.

Complications and How to Avoid Them

Tracheal Fire

This is a disastrous complication, usually secondary to ignition of the endotracheal tube or a flexible bronchoscope used through the endotracheal tube. If the laser is used (CO_2 or KTP) in the larynx, then a non-

flammable endotracheal tube is mandatory. We favor a metallic tube such as the Mallinkrodt semiflexible type. Never use flammable polyvinyl chloride (PVC) since these tubes can ignite despite being coated with metallic tape.[57,58] An endotracheal tube should be avoided, if at all possible, when using a laser through a flexible bronchoscope. A spark resulting from explosion of a carbon particle can ignite the endotracheal tube, particularly in a high oxygen environment. The PVC tube, when ignited, will serve as a blowtorch, depositing hot ignition debris down the tracheobronchial tree.[57] Immediate response to laser induced fire is removal of the endotracheal tube and rapid establishment of the airway with either a rigid bronchoscope or by reintubation. This should be followed by bronchoscopy to assess the degree of the damage. Antibiotics, steroids, and possibly stenting of the airway with an endotracheal tube or a T tube may be required, depending on the extent of the injury.

Tracheal Perforation

The laser should never be fired perpendicular to the airway wall. The energy levels must be kept down to recommended levels of power and always used in intermittent short exposures.

Hypoxemia

Hypoxemia is the most common intraoperative and postoperative complication (Table 37-5). It is usually the result of accumulation of distal secretions or blood during the procedure. Respiratory depression induced by anesthesia and also persistent hemorrhage can result in hypoxemia. Vigilant tracheobronchial toilet should be maintained throughout the procedure.

Hemorrhage

Hemorrhage is usually persistent rather than massive. It is normally controlled with the laser or by tamponade with the bronchoscope and application of adrenaline pledgets. Tracheal hemorrhage is rare and better prevented than treated. The main priority is maintaining tracheobronchial patency; for this purpose, the rigid bronchoscope is passed through the area of hemorrhage. The hemorrhage is controlled by tamponade with the bronchoscope allowing ventilation of the patient and removal of distal accumulation of blood. Once ventilation is secured, coagulation should be carried out from distal to proximal. Massive hemorrhage from a major blood vessel is an unusual complication, which may require emergency thoracotomy and vessel repair (usually too late).

Infection

Infection may occur secondary to spillage of pus from an obstructed airway, which can contaminate other bronchi. The pus is drained by bronchoscopy after placing the patient in a lateral position with the affected bronchi dependent. Antibiotic coverage should be based on the results of culture.

Table 37-5 Complications of Laser Bronchoscopy

Hypoxia
Hemorrhage
Tracheal perforation
Infection
Pneumothorax
Tracheal fire
Pneumomediastinum

Summary

Laser applications in the airway are well defined for the treatment of stenosis and malignancy as well as of selected benign tumors such as papillomatosis and unusual conditions such as amyloidosis. Most important is knowing when to apply the laser and how to avoid complications. It is essential for the endoscopist to understand the basic soft tissue interaction of each laser wavelength and its associated safety concerns. Laser technology can be an elegant addition to the bronchoscopist's armamentarium when used properly for appropriate indications or it can be a very dangerous instrument when these dicta are not adhered to.

References

1. Strong MS, Jako GJ. Laser surgery in the larynx: early clinical experience with continuous CO_2 laser. Ann Otol Rhinol Laryngol 1972;81:791–8.
2. Healy GB, McGill T, Simpson GT, et al. The use of the carbon dioxide laser in the pediatric airway. J Pediatr Surg 1979;14:735–40.
3. Simpson GT, Strong MS. Recurrent respiratory papillomatosis: the role of the CO_2 laser. Otolaryngol Clin North Am 1983;16:887–94.
4. Shapshay SM, Hybels RL, Bohigian RK. Laser excision of early vocal cord carcinoma: indications, limitations, and precautions. Ann Otol Rhinol Laryngol 1990;99:46–50.
5. Strong MS, Healy GB, Vaughan CW, et al. Endoscopic management of laryngeal stenosis. Otolaryngol Clin North Am 1979;12:797–805.
6. Shapshay SM. Laser applications in the trachea and bronchi: a comparative study of the soft tissue effects using contact and noncontact delivery systems. Laryngoscope 1987;97(7 Pt 2 Suppl 41):1–26.
7. Ward RF. Treatment of tracheal and endobronchial lesions with the potassium titanyl phosphate laser. Ann Otol Rhinol Laryngol 1992;101:205–8.
8. Gillis TM, Strong MS, Shapshay SM, et al. Argon laser and soft tissue interaction. Otolaryngol Head Neck Surg 1984;92:7–12.
9. Wang Z, Pankratov MM, Gleich LL, et al. New technique for laryngotracheal mucosa transplantation. 'Stamp' welding using indocyanine green dye and albumin interaction with diode laser. Arch Otolaryngol Head Neck Surg 1995;121:773–7.
10. Shapshay S, Rebeiz E, Wang Z. Application of the neodymium:yttrium aluminium garnet, and other solid-state lasers in the larynx and trachea. In: Fried M, editor. The larynx: a multidisciplinary approach. St Louis: Mosby; 1996. p. 413–21.
11. McMillan K, Shapshay SM, McGilligan JA, et al. A 585-nanometer pulsed dye laser treatment of laryngeal papillomas: preliminary report. Laryngoscope 1998; 108:968–72.
12. Valdez TA, McMillan K, Shapshay SM. A new laser treatment for vocal cord papilloma—585 nm pulsed dye. Otolaryngol Head Neck Surg 2001;124:421–5.
13. Simpson GT, Strong MS, Healy GB, et al. Predictive factors of success or failure in the endoscopic management of laryngeal and tracheal stenosis. Ann Otol Rhinol Laryngol 1982;91:384–8.
14. Duncavage JA, Ossoff RH, Toohill RJ. Carbon dioxide laser management of laryngeal stenosis. Ann Otol Rhinol Laryngol 1985;94:565–9.
15. Lebovics RS, Hoffman GS, Leavitt RY, et al. The management of subglottic stenosis in patients with Wegener's granulomatosis. Laryngoscope 1992;102:1341–5.
16. Sasaki CT, Horiuchi M, Koss N. Tracheostomy-related subglottic stenosis: bacteriologic pathogenesis. Laryngoscope 1979;6:857–65.
17. Healy GB. An experimental model for the endoscopic correction of subglottic stenosis with clinical applications. Laryngoscope 1982;92:1103–15.
18. Olson NR. Wound healing by primary intention in the larynx. Otolaryngol Clin North Am 1979;12:735–40.
19. Shapshay SM, Beamis JF Jr, Hybels RL, et al. Endoscopic treatment of subglottic and tracheal stenosis by radial laser incision and dilation. Ann Otol Rhinol Laryngol 1987;96:661–4.
20. Correa AJ, Reinisch L, Sanders DL, et al. Inhibition of subglottic stenosis with mitomycin-C in the canine model. Ann Otol Rhinol Laryngol 1999;108:1053–60.
21. Spector JE, Werkhaven JA, Spector NC, et al. Preservation of function and histologic appearance in the injured glottis with topical mitomycin-C. Laryngoscope 1999;109:1125–9.
22. Rahbar R, Valdez TA, Shapshay SM. Preliminary results of operative mitomycin C in the treatment and prevention of glottic and subglottic stenosis. J Voice 2000; 14:282–6.
23. Dedo HH, Sooy CD. Endoscopic laser repair of posterior glottic, subglottic and tracheal stenosis by division or micro-trapdoor flap. Laryngoscope 1984;94:445–50.
24. Healy GB, Fearon B, French R. Treatment of subglottic hemangioma with the carbon dioxide laser. Laryngoscope 1980;90:809–13.
25. Sie KC, McGill T, Healy GB. Subglottic hemangioma: ten years' experience with the carbon dioxide laser. Ann Otol Rhinol Laryngol 1994;103:167–72.
26. Ohlms LA, Jones DT, McGill TJ, et al. Interferon alfa-2a therapy for airway hemangiomas. Ann Otol Rhinol Laryngol 1994;103:1–8.
27. Rebeiz E, April MM, Bohigian RK, et al. Nd-YAG laser treatment of venous malformations of the head and neck: an update. Otolaryngol Head Neck Surg 1991;105:655–61.
28. Yellin SA, LaBruna A, Anand VK. Nd:YAG laser treatment for laryngeal and hypopharyngeal hemangiomas: a new technique. Ann Otol Rhinol Laryngol 1996;105:510–5.
29. Clymer MA, Fortune DS, Reinisch L. Interstitial Nd:YAG photocoagulation for vascular malformations and hemangiomas in childhood. Arch Otolaryngol Head Neck Surg 1998;124:431–6.
30. Cholewa D, Waldschmidt J. Laser treatment of hemangiomas of the larynx and trachea. Lasers Surg Med 1998;23:221–32.
31. Cholewa D, Wacker F, Roggan A, et al. Magnetic imaging controlled interstitial laser therapy in children with vascular malformations. Lasers Surg Med 1998;23:250–7.
32. Terry RM, Lewis FA, Griffiths S, et al. Demonstration of human papillomavirus types 6 and 11 in juvenile laryngeal papillomatosis by in situ hybridization. J Pathol 1987;153:245–8.
33. Kashima H, Mounts P, Leventhal B, et al. Sites of predilection in recurrent respiratory papillomatosis. Ann Otol Rhinol Laryngol 1993;102:580–3.

34. Rimell FL, Shapiro AM, Mitskavich MT. Pediatric fiberoptic laser rigid bronchoscopy. Otolaryngol Head Neck Surg 1996;114:413–7.
35. Shapshay SM, Ruah CB, Bohigian RK, et al. Obstructing tumors of the subglottic larynx and cervical trachea: airway management and treatment. Ann Otol Rhinol Laryngol 1988;97:487–92.
36. Rosen CA, Woodson GE, Thompson JW, et al. Preliminary results of the use of indole-3-carbinol for recurrent respiratory papillomatosis. Otolaryngol Head Neck Surg 1998;118:810–5.
37. Lewis JE, Olsen KD, Kurtin PJ. Laryngeal amyloidosis: a clinicopathologic and immunohistochemical review. Otolaryngol Head Neck Surg 1992;106:372–7.
38. Talbot AR. Laryngeal amyloidosis. J Laryngol Otol 1990;104:147–9.
39. Strong MS, Jako GJ, Polanyi T, et al. Laser surgery in the aerodigestive tract. Am J Surg 1973;126:529–33.
40. Dumon JF, Reboud E, Garbe L, et al. Treatment of tracheobronchial lesions by laser photoresection. Chest 1982;81:278–84.
41. Toty L, Personne C, Colchen A. Bronchoscopic management of tracheal lesions using the neodymium yttrium aluminium garnet laser. Thorax 1981;36:175–8.
42. Shapshay SM, Simpson GT II. Lasers in bronchology. Otolaryngol Clin North Am 1983;16:879–86.
43. Cavaliere S, Foccoli P, Tonielli C, et al. Nd:YAG laser therapy in lung cancer: an 11-year experience with 2,253 applications in 1,585 patients. J Bronchol 1994;1:105–11.
44. Beamis JF Jr, Vergos K, Rebeiz EE, et al. Endoscopic laser therapy for obstructing tracheobronchial lesions. Ann Otol Rhinol Laryngol 1991;100:413–9.
45. Personne C, Colchen A, Leroy M, et al. Indications and techniques for endoscopic laser resections in bronchology: a critical analysis based upon 2,284 resections. J Thorac Cardiovasc Surg 1986;91:710–5.
46. Brutinel WM, Cortese DA, McDougall JC, et al. A two-year experience with neodymium-YAG laser in endobronchial obstruction. Chest 1987;91:159–65.
47. Laforet EG, Berger RL, Vaughan CW. Carcinoma obstructing the trachea. Treatment by laser resection. N Engl J Med 1976;294:941.
48. Shapshay SM, Davis RK, Vaughan CW, et al. Palliation of airway obstruction from tracheobronchial malignancy: use of the CO_2 laser bronchoscope. Otolaryngol Head Neck Surg 1983;91:615–9.
49. Shah H, Garbe L, Nussbaum E. Benign tumors of the tracheobronchial tree. Endoscopic characteristics and role of laser resection. Chest 1995;107:1744–51.
50. Turner JF Jr, Wang KP. Endobronchial laser therapy. Clin Chest Med 1999;20:107–22.
51. Chan AL, Tharatt RS, Siefkin AD. Nd:YAG laser bronchoscopy: rigid or flexible fiberoptic mode? Chest 1990;98:271–5.
52. Perrin G, Colt HG, Martin C, et al. Safety of interventional rigid bronchoscopy using intravenous anesthesia and spontaneous assisted ventilation: a prospective study. Chest 1992;103:1526–30.
53. Dumon JF, Shapshay S, Bourcereau J, et al. Principles for safety in application of neodymium-YAG laser in bronchology. Chest 1984;86:163–8.
54. Shapshay SM, Beamis JF Jr. Safety precautions for bronchoscopic Nd-YAG laser surgery. Otolaryngol Head Neck Surg 1986;94:175–80.
55. Waller DA, Gower A, Kashyap AP, et al. Carbon dioxide laser bronchoscopy. A review of its use in the treatment of malignant tracheobronchial tumours in 142 patients. Respir Med 1994;88:737–41.
56. George PJ, Garrett CP, Hetzel MR. Role of the neodymium YAG laser in the management of tracheal tumours. Thorax 1987;42:440–4.
57. Hayes DM, Gaba DM, Goode RL. Incendiary characteristics of a new laser resistant endotracheal tube. Otolaryngol Head Neck Surg 1986;95:37–40.
58. Ossoff RH, Aly A, Gonzales D, et al. A new endotracheal tube for carbon dioxide and KTP laser surgery of the aerodigestive tract. Otolaryngol Head Neck Surg 1993;108:96–9.

Editor's Note

This survey by Drs. Shapshay and Valdez of the possible uses of lasers in treating airway problems is admirably thorough. As an experienced otolaryngologist, Dr. Shapshay brings balance to his appraisal rather than uncritical advocacy of a technique. I wish to emphasize, however, that most obstructive lesions of the trachea, such as postintubation stenosis, are safely and expeditiously corrected by one well-designed operation, which is well tolerated by almost all patients. It is my opinion that this should therefore be the initial treatment. Preliminary trials of laser treatment, often repetitive and too often accompanied by tracheostomy, only delay definitive treatment, frequently compound the tracheal damage, make later operation more difficult, and add greatly to cost. (See Chapter 11, "Postintubation Stenosis.")

Airway obstruction by neoplasm can also be relieved more simply by the well-established technique of coring out the tumor. Lasers have not been needed to prevent or control bleeding (see Chapter 19, "Urgent Treatment of Tracheal Obstruction"). Indeed, I have seen instances where bleeding incited by lasering has complicated and made resectional treatment of bronchial neoplasm urgent—which would have been ultimately necessary in any case.

Hermes C. Grillo

Tracheal Appliances

Donna J. Wilson, RN, MSN, RRT

Although the importance of airway management has been recognized for decades, many practitioners are unfamiliar with the design and use of many commercially available airway appliances. A tracheostomy tube is often the airway appliance of choice for long-term airway management for upper airway obstruction, and for access for pulmonary toilet, positive pressure ventilation, and airway protection from aspiration of gastric and pharyngeal contents. The benefits of using a tracheostomy tube instead of an endotracheal tube include 1) a reduced likelihood of injury to laryngeal structures, 2) ease of deep tracheal suctioning, 3) tube stability, 4) overall patient comfort, 5) ability to provide a means for verbal communication, and 6) ease of swallowing.

Placement of a tracheostomy tube usually requires a surgical procedure, or percutaneous technique, and therefore poses risks of complications. When performed electively, under controlled conditions, by appropriately trained physicians, the incidence of complications including bleeding, hypoxia, pneumothorax, subcutaneous and mediastinal emphysema, and tracheal injury should be minimal. The skill and experience of those providing postoperative management is paramount to minimize long-term complications.

This chapter describes the design characteristics and clinical use of tracheostomy tubes. It also reviews several special airway devices that are used in acute and chronic airway management.

Components of a Tracheostomy Tube

Tracheostomy tubes, although designed to perform a few specific functions, are available in a variety of configurations. Components of a modern tracheostomy tube include the 1) tube shaft, 2) neck flange, 3) cuff, 4) obturator, and 5) swivel adapter. Each tracheostomy tube is packaged individually with an obturator, a tracheostomy tie, and product information. The obturator is used for insertion unless the tracheostomy tube has a Magill tip. A swivel adapter is attached between the tracheostomy tube cannula and the respiratory equipment to minimize torque on the airway. Each tracheostomy tube differs with respect to composition, degree of flexibility or rigidity, neck flange configuration, diameter, length, and curvature. Cuff design, including shape, compliance, and material, is another important variable.

Composition

Most contemporary tracheostomy appliances are manufactured using polyvinyl chloride (PVC). Metal tubes, made of silver and stainless steel, were commonly used in the past because of their durability and ease of maintenance. Disadvantages of metal tubes include cost associated with short-term use and their rigid structure. Increased rigidity may heighten the risk of mucosal trauma or tracheal wall injury during tube insertion and maintenance. Cases have been reported of corrosion and separation of silver tracheostomy tubes at the junction between the neck plate and the outer tube.[1] PVC is the material of choice since it provides varying degrees of flexibility based on composition and the possibility to design tubes to conform to varied anatomic configuration. Other materials including Silastic, silicone, and Teflon are used for specialty applications. Silastic tracheostomy tubes and stoma stents are soft, flexible, and become more pliable at body temperature. The use of these materials is considered later in this chapter.

Neck Flange

The neck flange or plate serves as a support between the tube and cervical tissues. Its primary purpose is to stabilize the tube position. Multiple styles of neck flanges are available. The neck flange can be designed as a solid plate, or in a soft and flexible configuration. Although most flanges are fixed to the tube at a designated location, others incorporate an adjustable design allowing for variation in effective tube length. There are two swivel flange designs to allow the angle of the tube to change. The double cannula cuffed and cuffless tubes, made by Shiley Medical of Mallinckrodt Inc. (St. Louis, MO), have a neck flange that moves horizontally in one direction. The other swivel neck flange design allows movement on two axes, horizontally and vertically, to conform to the patient's airway (Tracoe Flex tracheostomy tube, Boston Medical Products, Inc., Westborough, MA).

The Bivona hyperflex tube (flexible wire-reinforced silicone shaft; Bivona Medical Technologies, Gary, IN) has an adjustable flange to provide for change of usable shaft length. Practical uses of this tube include situations following total laryngectomy with an end tracheal stoma when positive pressure ventilation is required, patients with a mediastinal stoma, and patients with short, fat, or "bull" necks having an increased transmural distance.

The neck flange should be securely tied around the neck or sutured to the skin to avoid excessive tube movement and allow easy visualization of the area surrounding the stoma. Although many types of tapes and ties have been used, a cost effective and secure tie is manufactured from velcro.[2]

Size

Tubes are manufactured in a wide variety of sizes. PVC tracheostomy tubes are sized according to their inner diameter (ID) and outer diameter (OD), expressed in millimeters. The size is denoted on the neck flange of the tube. The metal Jackson and Shiley cannula tubes are sized according to an older system, the Jackson equivalent number. There exists a conversion between sizes; for example, a Shiley no. 6 equals a no. 7.0 mm internal diameter. To eliminate confusion, one should select tubes according to the internal diameter. In general, with plastic materials, there are approximately 3 mm of wall thickness between the inner and outer cannula (Table 38-1). Adult tracheostomy tubes range in size from 4.0 mm ID to 12.0 mm ID.

Length

The length of a tracheostomy tube is defined as the distance from the neck flange to the tip of the tube. To insure the proper fit of a tracheostomy tube, it is crucial to understand the relationship between tube size and length. The length varies in proportion to the size of the tube. As the inner diameter of the tube increases there is a corresponding increase in the length (see Table 38-1). A common problem occurring in tube

Table 38-1 Size and Length of Tracheostomy Tubes

Manufacturer	*Size (mm)*	*ID (mm)*	*OD (mm)*	*Length (mm)*
Bivona Fome-Cuf	5	5.0	7.3	60
	6	6.0	8.7	70
	7	7.0	10.0	80
	8	8.0	11.0	88
	9	9.0	12.3	98
	9.5	9.5	13.3	98
Portex Low Profile	6	6.0	8.5	64
Disposable	7	7.0	9.9	70
Inner Cannula	8	8.0	11.3	74
	9	9.0	12.6	80
	10	10.0	14.0	80
Shiley Reusable Cannula	4	5.0	9.4	65
Low-Pressure Cuffed	6	6.4	10.8	76
	8	7.6	12.2	81
	10	8.9	13.8	81
Tracoe Flex	4	4.0	7.2	65
	6	6.0	9.2	73
	8	8.0	11.5	76
	10	10.0	13.5	80
	12	12.0	15.9	83

ID = inner diameter; OD = outer diameter.

size selection is failure to realize that smaller ID tubes are shorter and therefore have greater potential for malposition due to inadequate length. The length of a tracheostomy tube also varies among different manufacturers. Standard tubes are generally designed to accommodate patients with normal neck and airway anatomy. If a longer tube is necessary, one can use a tube with a larger inner diameter in order to gain needed length or use extra-long tubes available from several companies (Bivona Medical Technologies; Portex, Inc. [Keene, NH]; Shiley Medical of Mallinckrodt Inc.). These are especially useful for obese patients, patients with short bull necks or masses in the neck. The extra-length tracheostomy tubes are manufactured in size ranges from 5.0 to 10 mm ID. Shiley makes a single cannula tube that is longer than their standard double cannula tube. This has a relatively short distance from the flange to its circular bend. Portex designed a tracheostomy tube with extra horizontal length that has an increased distance from the neck flange to its bend. It then drops vertically. This helps to insure proper fit in a patient with a thick neck. Bivona manufactures an adjustable neck flange, hyperflex (wire-reinforced silicone shaft), and extra-long tracheostomy tube that may be instantly customized. The maximum usable length for a 7 mm ID tube is 120 mm, and the no. 9 mm ID tube has 140 mm of usable length. This tube is available in two cuff designs, Aire-Cuf and tight-to-shaft cuff. These tubes can be used for a wide variety of anatomical variations or pathology. Most manufacturers offer a custom tracheostomy tube service for unusual specifications. Bivona offers a tracheostomy tube template so that all aspects of a tracheostomy tube, including tube shaft style, curvature, length, diameter, cuff design, cuff position, and neck flange, can be customized.

Currently, two designs of extra-long, double cannula cuffless tracheostomy tubes are available; the Moore tracheostomy tube and Tracoe comfort tracheostomy tubes (Boston Medical Products, Inc.). The

Moore cuffless tube is a size 6, of which the inner diameter with the inner cannula in place is 6.4 mm, and the outer diameter is 11 mm. This tube is flexible and made of radiopaque silicone. The extra-long length is 115 mm and can be trimmed for individual specifications. This tube can be cut to patient specification with a scalpel and a steady hand or with the available customizing kit that includes tube cutter and grinding stones to smooth edges (Boston Medical Products, Inc.). The Moore tube comes with an obturator, two inner cannulas, a tracheostomy tie, and a 15 mm connector to attach a speaking valve or ventilator equipment. The Tracoe comfort tracheostomy tube is available in a wide range of sizes (5.0 to 14.0 mm ID), and in standard and extra-long length. An extra-length size 7.0 mm ID tracheostomy tube is 100 mm long, and with each increasing size, the length is 5 mm longer. A custom length can be ordered. This tube is flexible and becomes more pliable at body temperature. Other design features of the Tracoe tube are that it can be ordered with or without fenestrations and/or a speaking valve and an inner cannula with a 15 mm adapter. Paraffin oil is used as a lubricant for placement of the inner cannula.

Cuff Design

Currently, all tracheostomy tubes are produced with large-volume, low-pressure cuffs which are soft, thin walled, and compliant.[3] Cuffs are designed to be cylindrical, round, or pear-shaped. The cuff should conform to the cross-sectional shape of the trachea, achieving a seal with minimal cuff to lateral wall pressure. It must be noted that a low-pressure, high-volume cuff can produce tracheal mucosal ischemia if it is overinflated, creating increased pressure on the tracheal wall.[4] Measurement of cuff pressure is an important step whenever the cuff is inflated and on a regularly scheduled basis. It is important to maintain the lowest possible cuff pressure at the lowest possible cuff volume required. The ideal cuff pressure should be maintained at a value less than 25 mm Hg to ensure that blood flow is maintained in the tracheal mucosa. In a normotensive patient, the estimated tracheal arterial tissue perfusion pressure is 30 mm Hg. Cuff pressure monitoring should be performed daily, or more frequently, if necessary. The cuff pressure and cuff volume should be recorded and observed for changes.

Two techniques of cuff inflation that are commonly in use are the minimal leak volume technique (MLT) and minimum occlusion volume (MOV). MLT is the smallest volume of air in the cuff that allows for a small leak on inspiration associated with a positive pressure breath. MOV is the smallest volume of air needed in the cuff to prevent an air leak on inspiration with a positive pressure breath. It is uncertain, based on the published literature, if one technique is superior to the other with respect to complication rate.[5,6] However, the MOV technique may be the method of choice in patients with increased risk for aspiration, and in the ventilator-dependent patient in order to assure the set ventilator volume is delivered.[7,8] The MLT is designed to minimize the potential for tracheal trauma by providing minimal cuff to tracheal wall contact. When using this technique with patients on mechanical ventilation, the delivered tidal volume may need to be increased to compensate for a leak and to achieve adequate ventilation. Potential problems with the MLT include tube movement, possible tracheal mucosal drying from airflow leaks, aspiration of upper airway secretions, and loss of set ventilator volume.[9] Routine cuff deflation and reinflation, although once common practice, is now no longer indicated. Cuff deflation increases the risk of aspiration and hypoxemia.[10] There is a potential for overinflation of the cuff. If a previously determined volume of air is replaced in a not fully deflated cuff or if a larger volume is used, then the cuff may be overinflated. If this volume exceeds maximal resting volume of the fully inflated cuff, then the intracuff pressure and consequent pressure on the tracheal wall may increase sharply because of lack of elasticity of plastics, with conversion effectively to a high-pressure cuff. If cuff deflation is performed, suctioning the oropharynx and applying positive pressure during cuff deflation to decrease the risk of aspiration of pharyngeal secretions is recommended. Cuff deflation is indicated in selected situations such as evaluation of a cuff leak, to clear upper airway secretions, to allow the patient to vocalize, and after surgery to reevaluate the volume of air

required for proper cuff inflation. This volume should be recorded on an appropriate record. If an increasing volume of air is required in the cuff to maintain a seal, then one must consider the potential for iatrogenic tracheal dilation. Tracheal injury can be assessed by evaluating the cuff diameter to tracheal diameter ratio on a chest radiograph, with a range higher than 1.5:1.0 being predictive of damage.[5]

As an alternative to air, the cuff design may incorporate material such as foam. Although such a special design can be used on a routine basis, it is more often used when the cuff pressure and volume with a standard air-filled cuff is excessive, usually in the setting of tracheomalacia.[11] The "Fome-Cuf" tracheostomy tube is manufactured from silicone (Bivona Medical Technologies) with a large diameter, high residual volume cuff filled with plastic foam with a silicone covering. This cuff is unique since it is self-inflating. The cuff inflates in a passive manner when the pilot balloon is opened to atmospheric pressure and the intracuff pressure is zero. Deflation of the cuff is accomplished by withdrawal of air from the cuff using a 60 cc syringe. Prior to insertion, one must insure that all air is removed from the foam cuff. The foam cuff should hug the tube shaft tightly to create the smallest cuff diameter and to decrease the risk of tearing the cuff on insertion. With the design of this large, foam-filled cuff, even in the deflated state, the excess material increases the diameter at the cuff site, making passage through the stoma difficult. After placement of the tube, the pilot port is open to the atmosphere and the cuff is self-inflating. Patients requiring positive pressure ventilation with airway pressure greater than 45 cm H_2O will often develop a leak around the inflated cuff. Air can be added (1 cc increments) to create an adequate seal, usually at the cost of increased cuff pressure. The pilot port is then occluded to maintain the additional air in the cuff. The cuff pressure should be monitored carefully if this approach is used.

Another approach to maintain a cuff seal in the setting of positive pressure ventilation is the use of the side port auto control airway connector that fits between the end of the tracheostomy tube and the ventilator tubing with a side port. The pilot port tip fits onto the side port, and with each positive pressure breath, air inflates the cuff to create an adequate seal at peak airway pressures. Thus, the foam cuff inflates and deflates with the cycling of each breath from the ventilator. A tear in the silicone covering creates special problems. The cuff remains in the inflated state but the inability to produce a negative intracuff pressure limits cuff deflation. In such a circumstance, the tracheostomy tube must be removed with the cuff in the inflated state. This can lead to patient discomfort and release of foam into the airway.

When no longer needed, the cuff is deflated and the patient progresses to an uncuffed tracheostomy tube.

Single and Double Cannulas

Tracheostomy tubes are available with single or double cannulas. A removable inner cannula is either reusable or disposable. The inner cannula can be removed for cleaning while the outer cannula remains in place to provide airway continuity. This is particularly useful for patients in a home setting or where medical care providers are not trained in tracheostomy tube removal and reinsertion. The reusable inner cannulas cannot be used interchangeably between tubes of the same size. The disposable inner cannula is commonly changed daily, although research suggests this routine practice of changing the inner cannulas may be unnecessary.[12] Standard practice is to check all inner cannulas (reusable and disposable) daily for obstruction. Notable disadvantages of the double cannula tracheostomy tube are reduction in airway cross section, increased resistance, and potential for increased work of breathing.[13] This may be significant in patients with marginal respiratory function.

Specialized Tracheal Appliances

Several specialized tracheal appliances are commercially available. These are generally used to provide some form of vocalization in chronically ventilated patients. These tracheal appliances, such as "talking" and

"fenestrated tracheostomy tubes," have been available for many years. These tubes give patients a sense of comfort and decrease in frustration and anxiety by providing them with the ability to communicate using normal glottic function.

Talking Tracheostomy Tubes

Talking tracheostomy tubes permit vocalization with the cuff inflated (Figure 38-1).[14–16] Positive airway pressure and airway protection are maintained. Examples are Bivona's tracheostomy tube with talk attachment, Portex's "Trach-Talk" tracheostomy tube, and Implant Technologies' "Communi-Trach I." These have a cuff inflation line and speaking port. The speaking port is a small bore tube, set into the curvature of the tracheostomy tube and stopping just above the cuff. The external end has a two-way connector. One end is connected to compressed gas at a flow rate of 4 to 8 L/min. Air flows into the trachea above the cuff and retrogrades through the vocal cords, allowing vocalization. During vocalization, the other end of the connector is occluded. The liter flow required for an audible voice may be different for each patient. Patients must understand that the quality will be different from their normal voice, usually at a lower pitch. The patient must be instructed to speak in short sentences; with long sentences, the voice will drift off to a whisper, due to the continuous flow of gas through the vocal cords. Common problems with these appliances are poor voice quality and occlusion of the talk port with secretions. The talk port can be cleared with a 50%–50% solution of saline and acetylcysteine (Mucomyst). Another complication is the potential for the compressed airflow to escape

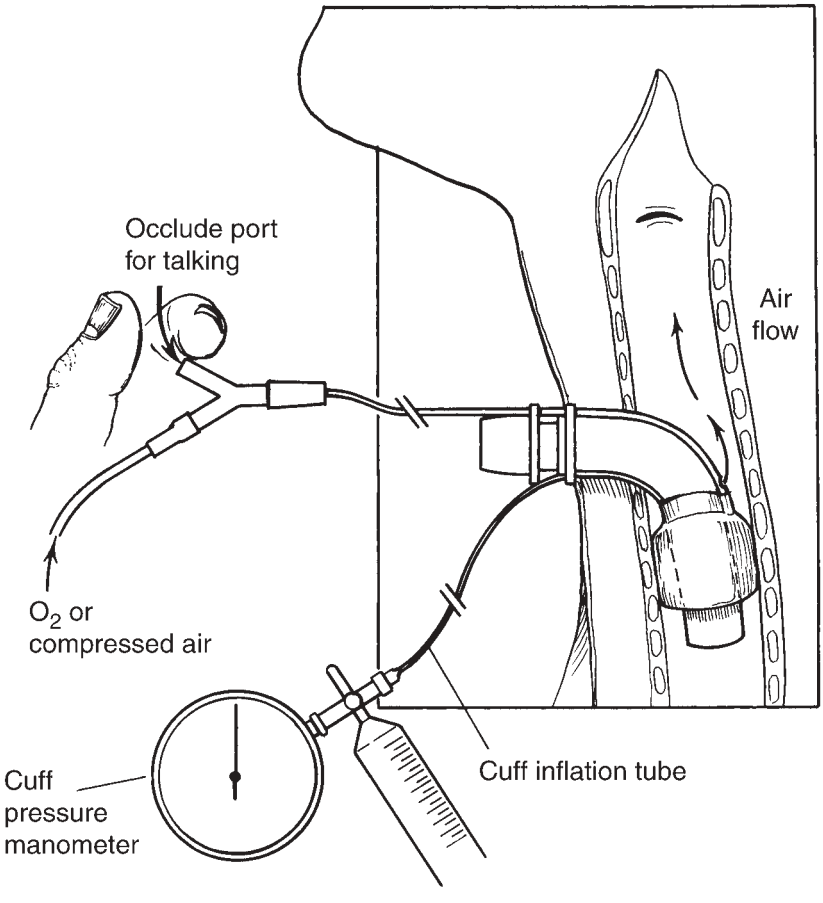


FIGURE **38-1** *Talking tracheostomy tube.*

through the tracheal stoma or into the pretracheal tissue. Thus, it is advisable to wait one or more days after tube placement to permit the stoma to close down before the talking option is used. Secretions that are pooled above the cuff can be removed by the application of a suction to the "talk port." It is important to monitor the potential for aspiration. This can be accomplished by the administration of blue-dyed ice chips via the oral route with suctioning through the talk port. This is only a rough guide to the degree of aspiration; the swallowing function must be evaluated more fully.

Fenestrated Tracheostomy Tubes

Several types of fenestrated tubes are available: "Lo-Profile" tracheostomy tube (Portex Inc.), Low-pressure cuffed tracheostomy tube (Shiley Medical, Mallinckrodt Inc.), and Tracoe flex tracheostomy tubes (Boston Medical Products) are examples of cuffed or cuffless precut fenestrated tracheostomy tubes. These are available in a variety of sizes (Figure 38-2).[16] The fenestration is a hole in the outer cannula. When the inner cannula is removed, the fenestration is open, providing airflow through the vocal cords, thus allowing speech. These are double cannula tubes, which permit speech when the cuff is deflated, the inner cannula is removed, and the tube is plugged. When the cuff is inflated and the inner cannula is in place, positive

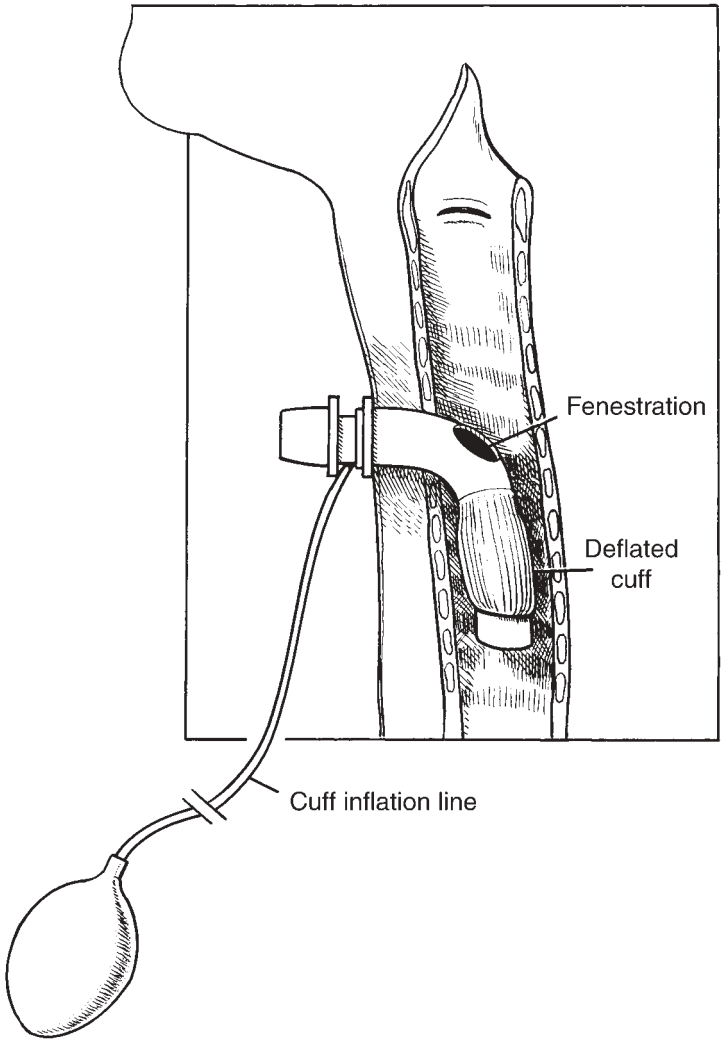


FIGURE **38-2** *Proper placement of a cuffed fenestrated tracheostomy tube in the trachea. The cuff is deflated, the fenestration opened, and the tube plugged to allow vocalization.*

pressure ventilation is possible and protection from aspiration of material into the lower airway is provided. Patients are considered candidates for a cuffed fenestrated tracheostomy tube when swallowing function is adequate. Airway protection can be evaluated with a modified barium swallow or a bedside test using blue-stained ice chips or water. Prior to the use of these devices, patients must 1) be able to protect their lower airway, 2) be able to clear secretions with minimal assistance, 3) have only a modest amount of secretions, and 4) be capable of spontaneous ventilation for at least a 2-hour period.

Upper airway patency should be evaluated prior to the placement of a fenestrated tube. A simple bedside method is to remove the tracheostomy tube, occlude the stoma with gauze, and evaluate the patient's breathing pattern using the patient's natural airway. If a patient has difficulty breathing, as evidenced by inspiratory stridor or retractions, then the fenestrated tube should not be placed.

To insure proper alignment of the fenestration within the tracheal lumen, either a lateral neck x-ray or bedside measurements should be obtained (Figure 38-3). At the bedside, the distance from the skin to the anterior and posterior tracheal walls can be measured using a simple technique. A sterile pipe cleaner, as depicted, is useful for this approach. These maneuvers help to avoid occlusion of the fenestration with tracheal tissue, by insuring proper position within the tracheal lumen. This procedure should be performed for cuffless fenestration as well.

Manufactured prefenestrated tubes may be adequate for only a small percentage of patients, due to anatomical variation and pathology. Serious upper airway obstruction has been reported in the presence of improperly positioned fenestrations.[17] Therefore, custom fenestrated tubes can be ordered or, in some cases, crafted at the bedside. The size of the fenestration generally should not be larger than the lumen of the tracheostomy tube; large fenestrations allow more contact with the tracheal wall, often stimulate granulation tissue growth, and weaken the structure of the tube. It is suggested that this type of tube be examined by direct vision (flashlight or flexible bronchoscope) on a routine basis.[18]

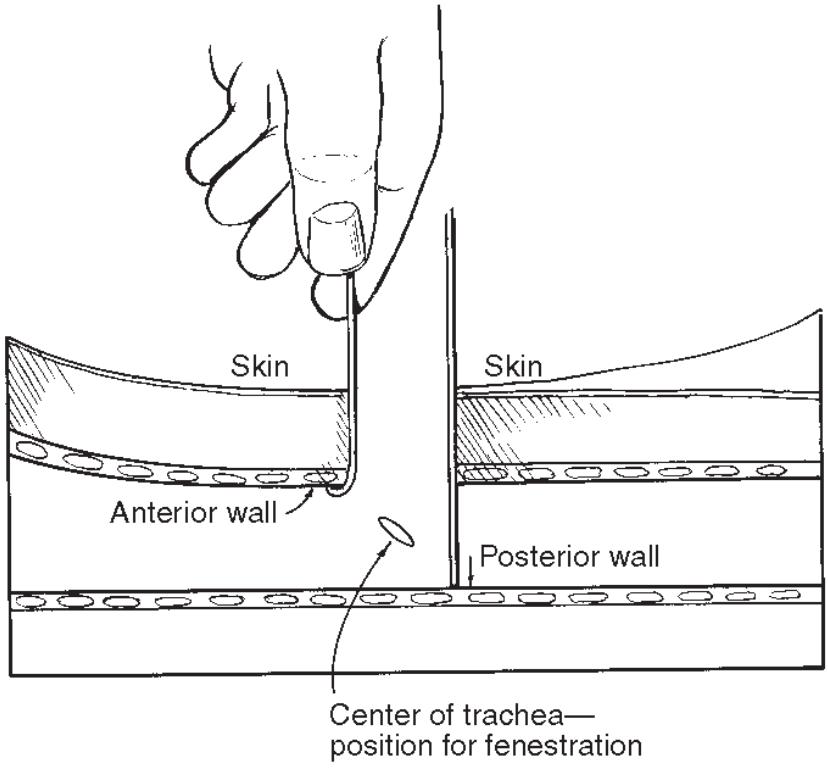


FIGURE **38-3** *Bedside technique of measuring for fenestration.*

An alternative approach is to use a nonfenestrated tracheostomy tube with the cuff deflated and the proximal opening occluded or "capped" to provide the ability to talk as air flows around the tube and through the cords. This method can be dangerous due to resistance and the substantial effort required to move air up and around the nonfenestrated tube through the upper airway.[19] If this method is used, monitor the patient's pattern and rate of respiration for increased effort to breathe, and listen to airflow characteristics over the lateral neck with a stethoscope. Patients may have difficulty clearing secretions using a nonfenestrated capped tracheostomy tube. Pulse oximetry is recommended throughout the entire weaning procedure.

Stents

Tracheal stoma stents or tracheostomy buttons are used to maintain stomal patency. This provides access for suctioning or replacement of a tracheostomy tube for emergency ventilation (Figure 38-4). The closure plug on the button allows for normal respiration and phonation. Several designs of stomal stent are available (Figure 38-5), including the Montgomery tracheal cannula system, made of silicone (Boston Medical Products, Inc.), the Olympic tracheostomy button made of Teflon (Olympic Medical, Seattle, WA),[20] and the straight and curved stents made of silicone (Hood Laboratories, Pembroke, MA) (see Figure 38-4). Each manufacturer provides a wide range of sizes and lengths, and thus, these devices can be adapted to fit most pathological situations in any patient. Before a stomal stent is placed, the patient's swallowing function must be evaluated. In addition, a chest x-ray should be obtained to confirm the absence of progressive parenchymal disease. The inspired oxygen requirement should be no greater than 40%.

Prior to placement of a stomal stent, the tracheostomy tube is removed. The stomal depth is measured by placing a sterile small hooked pipe cleaner, or a device called a stoma gauge (Hood Laboratories),

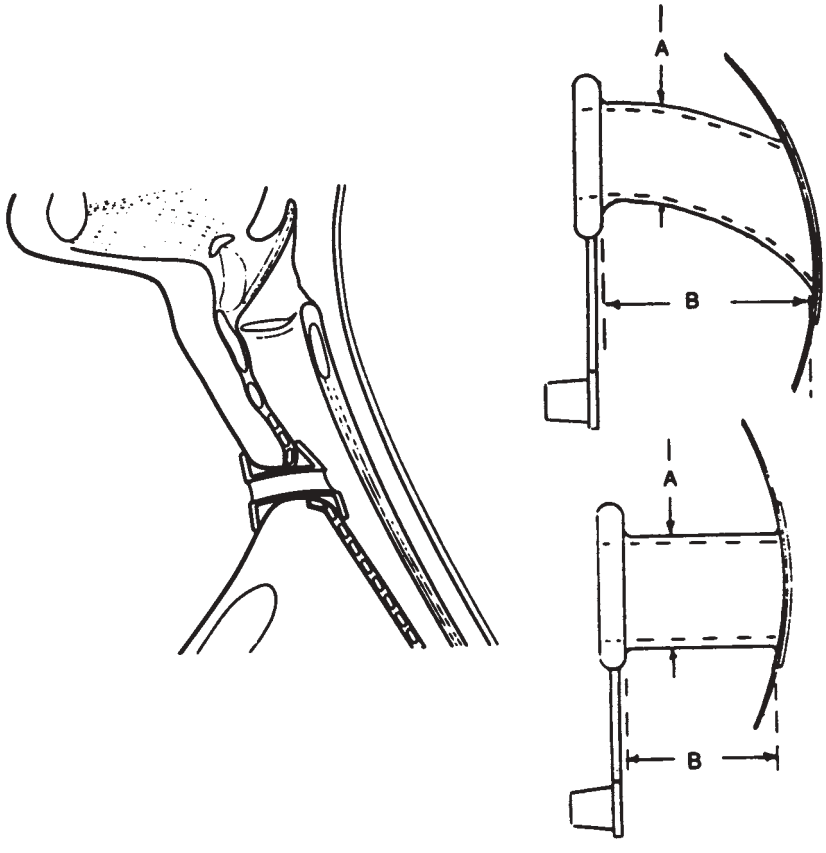


FIGURE **38-4** *Stomal stent is available in both curved and straight designs (Hood Stoma Stent, Hood Laboratories, Pembroke, MA). Both designs are available in several diameters* (A) *and lengths* (B).

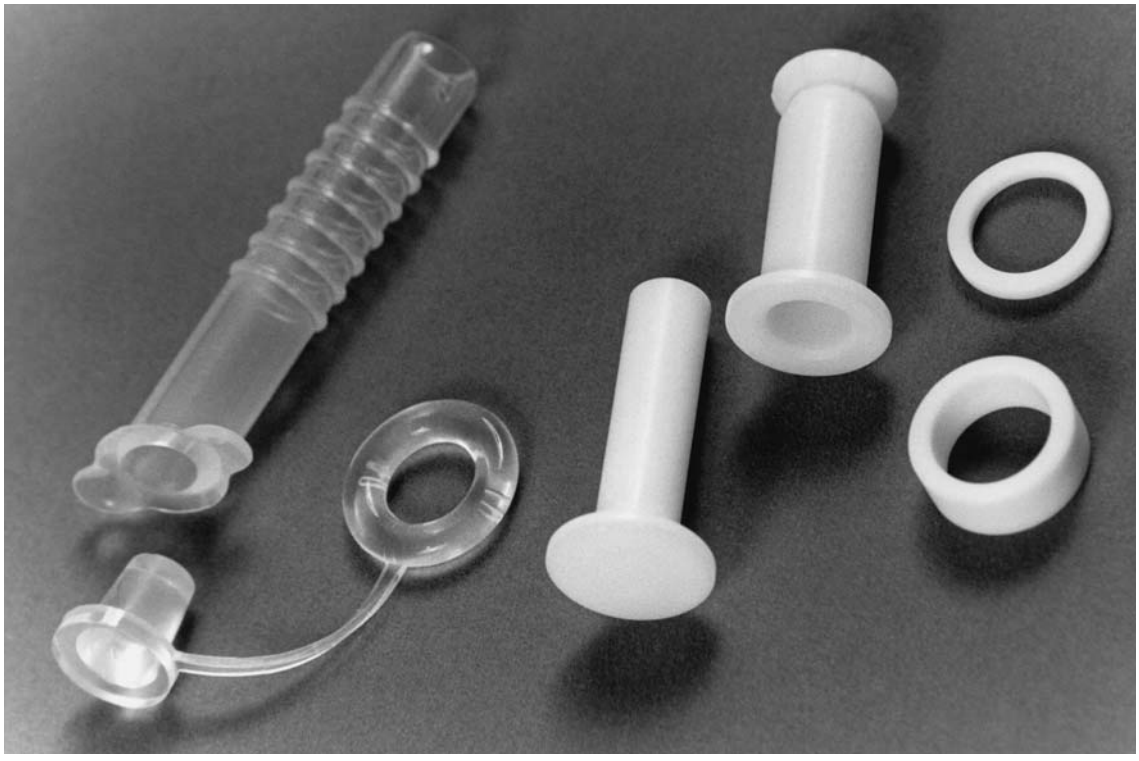
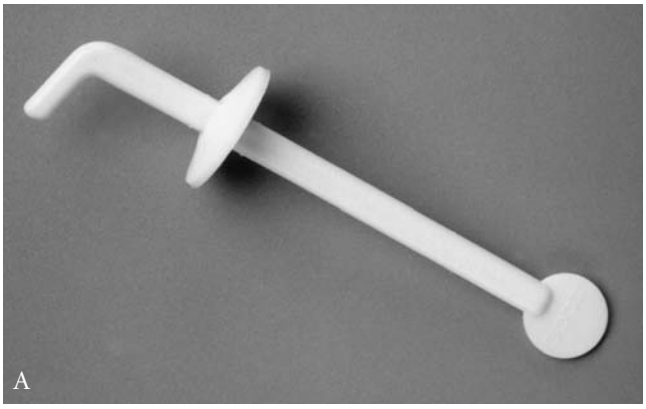


FIGURE **38-5** *Montgomery stomal stent on the left (Boston Medical Products Inc.). The oblique end-plate resides in the trachea. The plug fits the opposite end. The Olympic button lies on the right.*

into the stoma and hooking the anterior tracheal wall (Figure 38-6). The length is marked at the skin surface. The length of the stent is determined using this measurement. The length of stomal stent must not exceed that of the stomal track, otherwise the stent can slip backward into the trachea, causing obstruction of the airway. The stent is lubricated and placed into the stoma, and then the closure plug is inserted. The patient should be encouraged to cough, breathe deeply, and talk using normal glottic function. The closure plug should be removed, and a light source used to ensure that no obstruction exists around the pedicles of the stent against the anterior tracheal wall. If the stomal stent protrudes, then the closure plug is removed and gentle pressure is applied to reposition the stent flush with the neck. Improper positioning may limit function and increase the risk that the stent is only partially in the stoma and that the anterior tracheal wall will close. Stomal stents may be left in place for several days or weeks. Complications are primarily related to malpositioning. Many patients go home with an appliance in place.

Speaking Valves

Placement of a cuffed tracheostomy tube generally causes dysphagia, aphonia, and reduces the ability of the patient to taste and smell. Patients who are stable on mechanical ventilation, or those capable of maintaining spontaneous ventilation for a few hours, are candidates for a one-way speaking valve to restore voice communication. There are suitable valves for the ventilator-dependent and nonventilated patient.[21–23] The one-way valve is placed on the tip of the tracheostomy tube with the cuff deflated; on inspiration, air enters the trachea through the valve, and on expiration, the valve closes and air exits through the upper airway, allowing normal glottic function. Speaking valves assist in the reestablishment of laryngeal reflex activity,



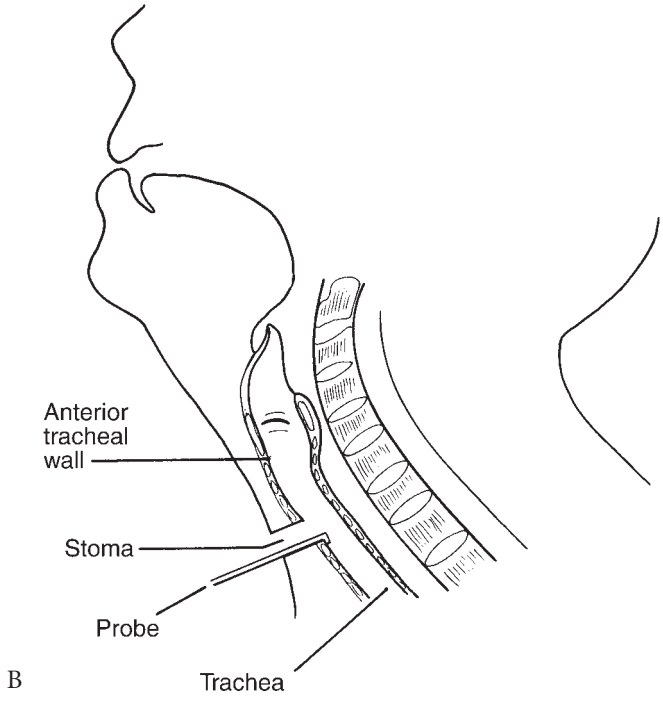


FIGURE **38-6** A, *Device to measure stomal depth.* B, *Technique of measurement. Place the sterile pipe cleaner or device through the stoma and hook it onto the anterior tracheal wall; then mark at the skin to determine the depth of the stoma track.*

phonation, and coughing without the need for finger occlusion. The inspiratory airway pressure that the patient requires to open the valve is relatively low (airflow resistance 2.6 cm H_2O or less in all of the valves).[24] The low resistance to open the valve should not add to increased effort to breathe, but it may do so, especially in patients with respiratory muscle weakness. The patient's breathing pattern and oxygen saturation should be monitored initially with the use of a speaking valve. Commonly, patients can clear secretions more easily, their sense of smell is stimulated, and their swallowing function improves. The valves are durable and easy to clean.

There are several one-way speaking valves designed to fit on standard tracheostomy tubes and stomal stents equipped with a 15 mm hub adapter. The most common one is the Passy-Muir tracheostomy speaking valve (Passy-Muir Inc., Irvine, CA) (Figure 38-7).[22,24] The Passy-Muir (PMV) 2000 series is a low profile, lower resistance tracheostomy and ventilator speaking valve. The PMV 2000 frame is clear, designed to be less visible, whereas the PMV 2001 is bright purple to facilitate staff awareness. The PMV 2000 series valves are lightweight, smaller, open with less resistance, and can be used on or off the mechanical ventilator. These valves have the "Secure-It." This is an attachment that connects the PMV to the tracheostomy tie to prevent valve loss. The PMV 2000 series valves are closed position "no leak" valves offering benefits including improved swallowing, reduced aspiration, improved secretion management, reduced weaning, and decannulation time.[25]

The Shiley phonate speaking valve is available with or without an oxygen supplement port and cap (Mallinckrodt–Shiley). This valve is lightweight and the diaphragm has a hinged cap for easy access for cleaning. The Montgomery tracheostomy and cannula speaking valve is the only valve with a pressure release feature. This release feature reduces the risk of the valve popping off the 15 mm adapter of the tracheostomy tube during a forceful cough (Boston Medical Products, Inc.). The Olympic "Trach-Talk" (Olympic Medical) is a spring-loaded valve that clicks with opening and closing of the valve. The spring assists in reopening the valve on expiration. This speaking valve is T-shaped so it can be used for oxygen delivery and

FIGURE 38-7 *Passy-Muir speaking valve (Passy-Muir Inc., Irvine, CA).*

has a cap that is easily removed to permit suctioning without removing the valve from the tracheostomy tube. There are two Hood speaking valves (Hood Laboratories). One of these is designed to fit stoma stents of sizes 11.0 and 13.0 mm OD. The stomal stent plug can be removed from the attachment and the speaking valve attached to the holder to prevent loss of the valve. The other valve is adapted to fit all 15 mm connectors for tracheostomy tubes.

Heat and Moisture Exchanger

Any tracheostomy device that bypasses the upper airway interferes with the normal heat and moisture exchange that is usually provided to inspired air. Heat and moisture exchanger (HME) devices provide some of this function. In spontaneously breathing patients, in whom the tracheostomy tube cannot be capped or a speaking valve used, an HME can minimize the loss of moisture and protect the lower airway. The heat and moisture exchanger, Thermovent T (Portex, Inc.), has an oxygen adapter that clips onto the exchanger to provide an oxygen flow to the patient. The HME is easy to use, has low resistance to flow, and filters foreign particles from entering the lower airway.

Summary

In summary, there are a few but important aspects of tracheostomy tube management. First, it is essential to recognize the size-to-length relationship—the smaller the inner diameter of a tube, the shorter the shaft length of the tube. It is important to remember that the length of a tube for a given size varies among different manufacturers.

Second, evaluation of tube position using the chest x-ray and an estimate of diameter cuff to tracheal diameter ratio is important. Airway injury may increase if the range is greater than 1.5:1.0. The tracheostomy tube may have to be changed to a different size or cuff design. Third, speciality tubes are important to provide vocalization and comfort for the patient. It is essential that the staff caring for these patients be knowledgeable about the design and function of these devices. Listed in the appendix are manufacturers and their Web sites. Most importantly, all have excellent educational handbooks and guides to care of the tracheostomy patient.

Appendix

Manufacturer Information

Bivona Medical Technologies
5700 West 23rd Ave.
Gary, IN 46406
1-800-348-6064
1-219-989-9150
Fax: 1-219-989-7435
<http://www.portexusa.com>

Boston Medical Products, Inc.
117 Flanders Road
Westborough, MA 01581
1-800-433-2674
1-508-898-9300
Fax: 1-508-898-2373
<http://www.bosmed.com

Hood Laboratories
575 Washington Street
Pembroke, MA 02359
1-800-942-5227
1-781-826-7573
Fax: 1-781-826-3899
<http://www.hoodlabs.com>

Mallinckrodt Inc.
PO Box 5840
St. Louis, MO 63134
1-888-744-1414
<http://www.mallinckrodt.com>

Olympic Medical
5900 First Avenue South
Seattle, WA 98108
1-800-426-0353
1-206-767-3500
Fax: 1-206-762-4200
<http://www.olymed.com>

Passy-Muir Inc.
4521 Campus Drive, Suite 273
Irvine, CA 92612
1-800-634-5397
1-948-833-8255
Fax: 1-949-833-8299
<http://www.passy-muir.com>

Portex, Inc.
10 Bowman Drive
Keene, NH 03431-0724
1-800-258-5361
Fax: 1-603-352-3703
<http://www.portexusa.com/>

References

1. Ayshford CA, Walsh RM, Proops DW. Corrosion of a silver Negus tracheostomy tube. J Laryngol Otol 1999;113:68–9.
2. Dixon L, Wasson D. Comparing use and cost effectiveness of tracheostomy tube securing devices. MEDSURG Nursing 1998;7:270–4.
3. Grillo HC, Cooper JD, Geffin B, Pontoppidan H. A low-pressure cuff for tracheostomy tube to minimize tracheal injury: a comparative clinical trial. J Thorac Cardiovasc Surg 1971;62:898–907.
4. Plunkett PF, Astravas-Perrault S. The effect of tracheal shape on the pressure dynamics of seven commercially available endotracheal tube cuffs: an in vitro study. Respir Care 1981;26:45–52.
5. Crimlisk JT, Horn MH, Wilson DJ, Marino B. Artificial airways: a survey of cuff management practices. Heart Lung 1996;25:225–35.
6. Off D, Braun SR, Tompkins B. Efficacy of the minimal leak technique of cuff inflation in maintaining proper intracuff pressures for patients with cuffed artificial airways. Respir Care 1983;28:1115–20
7. Demers RR, Saklad M. Intratracheal inflatable cuffs: a review. Respir Care 1977;22:29–36.
8. Lee T, Hsu D. Ischemic damage to the trachea: lateral wall pressure versus intracuff pressure [letter]. Crit Care Med 1991;19:1328.
9. Goodnough SK. Reducing tracheal injury and aspiration. Dimens Crit Care Nurs 1988;7:324–32.
10. Tyler DO, Clark AP, Ogburn-Russell L. Developing a standard for endotracheal tube cuff care. Dimens Crit Care Nurs 1991;10:54–61
11. Power KJ. Foam cuffed tracheal tubes: clinical and laboratory assessment. Br J Anaesth 1990;65:433–7.
12. Burns SM, Spilman M, Wilmoth D, et al. Are frequent inner cannula changes necessary? A pilot study. Heart Lung 1998;27:58–62
13. Diehl JL, El Atrous S, Touchard D, et al. Changes in the work of breathing induced by tracheostomy in ventilator-dependent patients. Am J Respir Crit Care Med 1999;159:383–8.
14. Safer P, Grenvik A. Speaking cuffed tracheostomy tube. Crit Care Med 1975;3:23–6.
15. Trout C. Artificial airways: tubes and trachs. Respir Care 1976;21:513–20.
16. Wilson DJ. Airway appliances and management. In: Kacmarek RM, Stoller J, editors. Current respiratory care. Philadelphia: BC Decker; 1988. p. 80–9.
17. Beard B, Monaco FJ. Tracheostomy discontinuation: impact of the tube selection on resistance during tube occlusion. Respir Care 1993;38:267–70.
18. Snyder GM. Individualized placement of tracheostomy tube fenestration and in-situ examination with the fiberoptic laryngoscope. Respir Care 1983;28:1294–8.
19. Hussey JD, Bishop MJ. Pressure required to move gas through the native airway in the presence of a fenestrated vs a nonfenestrated tracheostomy tube. Chest 1996;110:494–7.
20. Long J, West G. Evaluation of the Olympic trach button as a precursor to tracheostomy tube removal. Respir Care 1980;25:1242–3.
21. Manzano JL, Lubillo S, Henriquez D, et al. Verbal communication of ventilator-dependent patients. Crit Care Med 1993;21:512–7.
22. Passy V. Passy-Muir tracheostomy speaking valve. Otolaryngology 1986;95:247–8.
23. Kaut K, Turcott JC, Lavery M. Passy-Muir speaking valve. Dimens Crit Care Nurs 1996;15:298–306.
24. Zajac DJ, Fornataro-Clerici L, Roop TA. Aerodynamic characteristics of tracheostomy speaking valves: an updated report. J Speech Lang Hear Res 1999; 42:92–100.
25. Dettlebach T, Gross R, Mahkmann J, Eibling D. Effect of the Passy-Muir valve on aspiration in patients with tracheostomy. Head Neck 1995;17:297–300.

Tracheal T Tubes

Hermes C. Grillo, MD

Critical stenosis of the upper airway not amenable to surgical resection often requires a tracheostomy, either as a temporary measure or for long-term relief. Transient airway obstruction after operative reconstruction may also require temporary intubation. Although tracheostomy tubes have the virtue of simplicity in insertion, management, and change, they divert the airflow from the nose, mouth, and larynx. Speech is then possible only when air passes around the tube or through a fenestration in the tube, and then only with a flutter valve or an occluding finger. Because of these disadvantages and the potential of additional injury to the trachea from quite rigid tubes, an alternative solution to maintenance of a patent airway was desirable. The tracheal T tube lies halfway between a tracheostomy tube and an internal airway stent. It has advantages and disadvantages relative to both and so merits its own chapter.

In 1891, Bond devised a tracheal T tube made of two rigid parts, a proximal half and a distal half, divided horizontally along the line of the sidearm which emerged from the tracheal stoma.[1] The two were held together with a collar (Figure 39-1). The T tube in its present incarnation was developed by William Montgomery, at the Massachusetts Eye and Ear Infirmary, and introduced in 1965.[2,3] Although an initial model consisted of a two-part rigid tube, the present design, which is now widely used, is made of flexible medical silicone rubber. It was originally hoped that the tube would provide definitive treatment for tracheal stenosis. Because of the innate cicatricial tendency of scar tissue, this has rarely been successful, but the Silastic T tube tracheal stent has found extensive use. If the sidearm of the T tube remains occluded, except during treatment, then humidity is maintained in the tube by the normal upper airway protective mechanisms. The airway remains open and the patient breathes normally through the mouth and nose. When the tube spans an obstructing tracheal lesion, normal glottic speech is maintained. Excellent long-term acceptance with absence of injury to the tracheal wall makes this a preferable way to maintain airway patency. The T tube has been used for long-term management of many problems.[4–6] Modifications have been made to deal with special problems at the laryngeal and carinal ends of the airway.

The T tube has been used at Massachusetts General Hospital since 1968 for three general *indications*: 1) for temporary stenting of the airway, 2) as a definitive procedure for palliation of airway obstruction, and

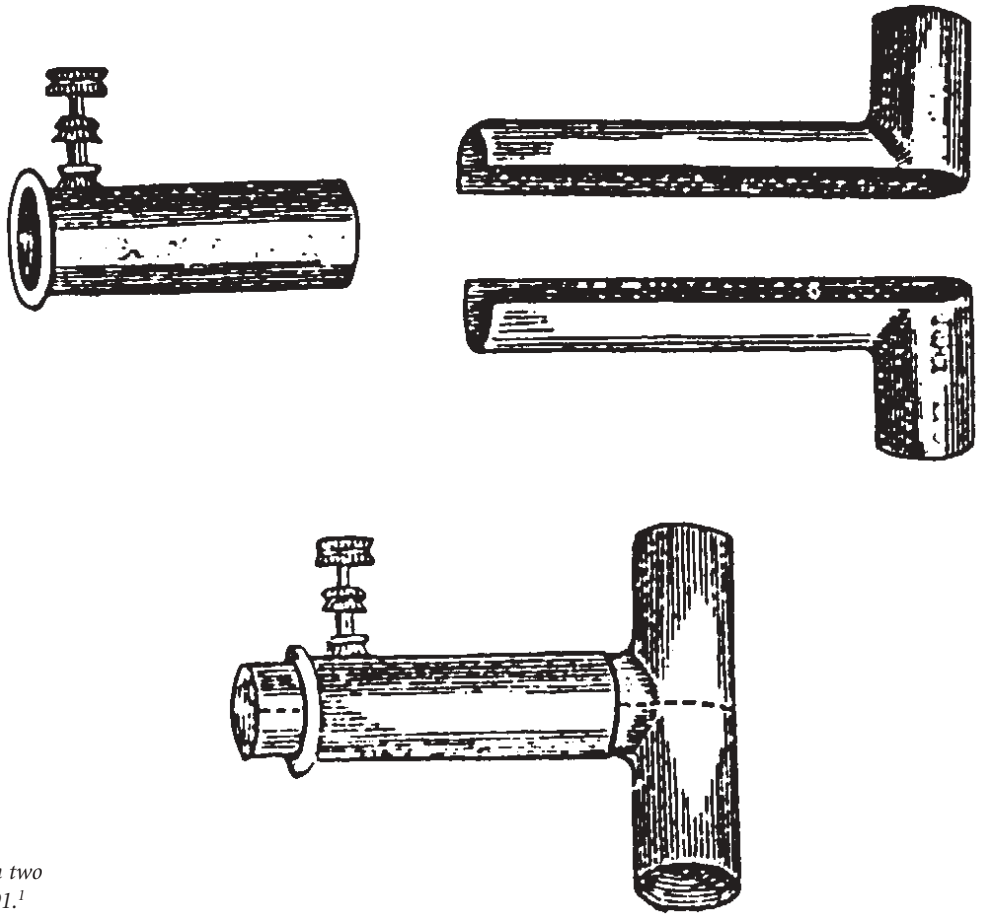


FIGURE **39-1** *Rigid T tube in two parts, devised by Bond in 1891.*[1]

3) for complications of airway reconstruction. Gaissert and colleagues described the use of the T tube or its modifications in 140 patients from the ages of 7 months to 95 years, between 1968 and 1991.[7] *Temporary airway support* was provided in 31 patients, either prior to tracheal resection or as the only procedure. In 49 patients, the T tube was the *definitive procedure for airway palliation*. *Obstruction following operative reconstruction* led to placement in 32 patients.

The T tube can provide *temporary airway support* while determination is made as to whether all or part of a complex injury will stabilize, where, for example, cartilages are partly intact. Time is also gained for severe inflammation to subside prior to resection at a safe stage, for a patient's medical status to improve, as in Guillain-Barré syndrome, for reduction in corticosteroid medication, or for completion of other procedures (such as orthopedic repairs) prior to tracheal reconstruction. If the stenosis is severe—and indeed in most cases, resolution after T tube splinting is illusory and the stricture will reassert itself—then either resection or permanent splinting is required.

The T tube may be the *definitive airway* where insufficient trachea remains for adequate reconstruction or where irresectable tumor obstructs after other modalities have been used, especially extrinsic tumor, or where other severe disease precludes tracheal repair. Some malacic or inflammatory processes, such as relapsing polychondritis, may also be so palliated. Severe inhalation burns may be palliated by prolonged intubation but may eventually resolve sufficiently to allow extubation.

A T tube can provide a satisfactory *airway following failure of tracheal anastomosis*, until such time as reoperation is appropriate, or permanently if reconstruction can not be repeated.

The T Tube and Its Placement

The tube consists of a vertical column of flexible medical silicone, which resides in the airway and is intended to span the entire length of a stenosis or other obstructive pathology. A sidearm of the same material emerges from the tube at a right angle and passes to the surface. A plug or stopper of silicone is provided to occlude the sidearm. Tubes are made in a variety of outer diameters ranging from 6 to 16 mm. The size numbers of tubes correspond to their outer diameter. Tubes are provided in pediatric, standard, long, and extra-long lengths (up to 15 cm) (Figure 39-2). The tubes may be trimmed to precisely the size required for the individual patient but care must be taken to smooth the cut edges. Densely radiopaque tubes are available.

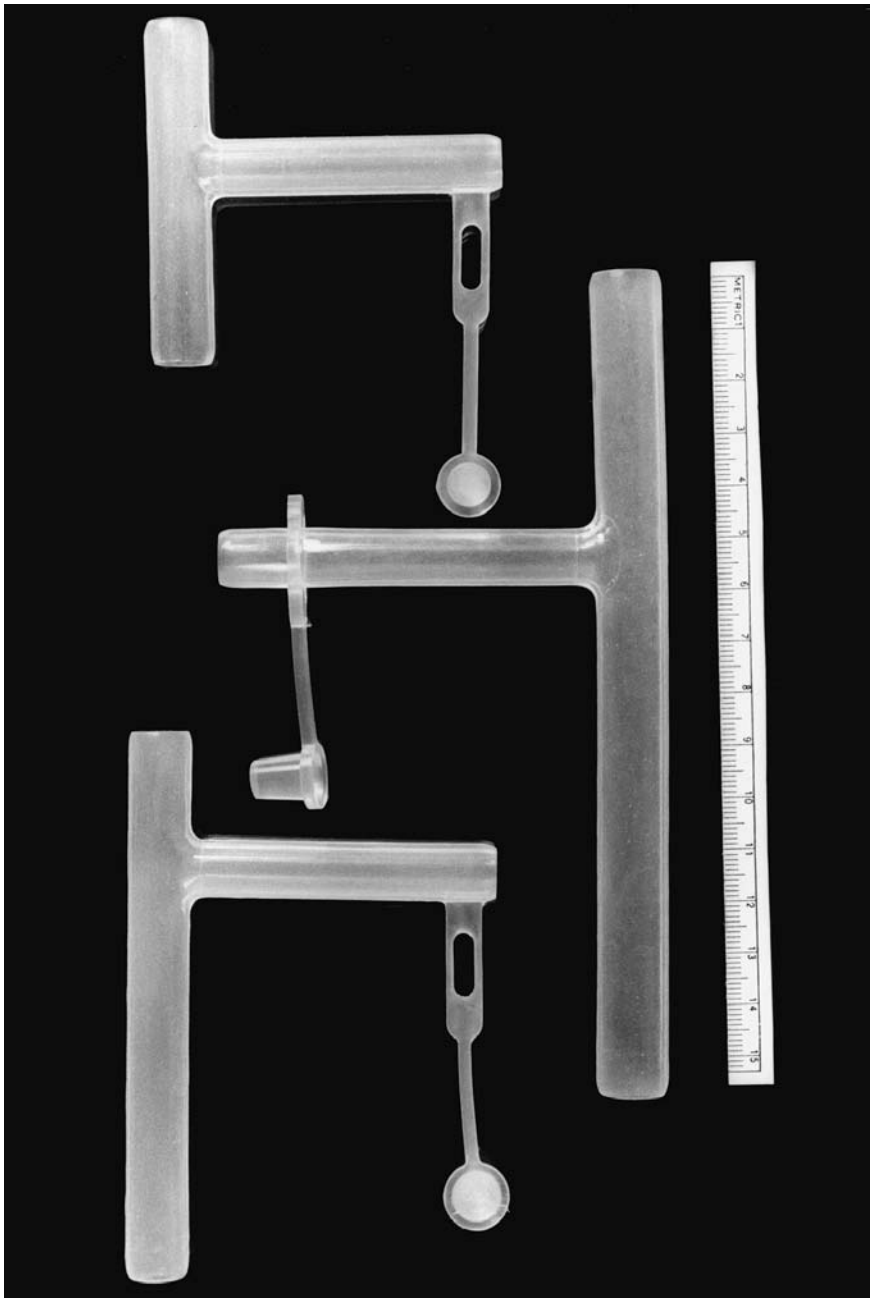


FIGURE **39-2** *Silicone tracheal T tubes; standard, long, and extra-long tubes. Two tubes are 12 mm outer diameter and the extra-long tube is 14 mm (Hood Laboratories, Pembroke, MA).*

Following an instance of airway obstruction, when an entire tube including the sidearm was inexplicably aspirated into the distal trachea, Montgomery produced a model of a tube characterized by a ridged sidearm over which a circular ring of silicone is passed to prevent such aspiration. I have never observed this problem using the modified T tube.

The technique of placement is conceptually simple, but in individual cases with very varied pathology, fitting a T tube may be difficult and time-consuming and demands close cooperation between the anesthetist and surgeon. Preliminary standard tracheal x-rays provide maximum information about the lesion, including precise and relative dimensions of the diseased airway, distances between the glottis and superior margin of the lesion, and relationship of the lesion to the carina and to an existing stoma. T tube placement is best done under general anesthesia with spontaneous respiration. Rigid bronchoscopy is performed initially, a stenosis is dilated, and tracheal measurements are made as described in Chapter 5, "Diagnostic Endoscopy." The diameter of the tube is selected on the basis of radiologic findings and observations. I prefer to select a tube of larger size to provide the patient with the best possible airway palliation, but which will not fit too tightly against the mucosa proximally or distally. A tight fit can produce inflammation of normal trachea above and below the lesion. In many patients, a standard or long T tube will suffice to span the lesion without creating additional problems such as laryngeal irritation. If a tracheostomy is already present, it is usually used for the sidearm of the T tube. In some patients, because of the obliquity of the tracheostomy tube tract, a T tube sidearm will not rest appropriately, and it is necessary to modify the stomal tract and the opening into the trachea so that the sidearm will emerge without kinking (Figure 39-3). If the stoma is insufficiently large, it may be dilated with Hegar uterine dilators. During these manipulations, the ventilating bronchoscope is withdrawn to a point above the stoma, but it is not removed from the airway. The rigid bronchoscope provides a safe airway for these maneuvers. A flexible endotracheal tube should also be available to be slipped into the stoma for direct distal ventilation, intermittently if necessary. Liberal suctioning keeps the airway free of blood. Profuse granulations at the tracheal stoma and within the stomal tract and trachea are removed. In some patients, opportunity is taken at this point to transfer a tracheostomy stoma that may have been placed below a cervical stenotic lesion to a location in the stenotic lesion. This will permit the stoma that was within normal trachea to heal and so recapture this portion of trachea for later reconstruction (see Chapter 24, "Tracheal Reconstruction: Anterior Approach and Extended Resection"). The sidearm of the T tube should never rest against the brachiocephalic artery any more than a tracheostomy tube should.

In most patients, the T tube must be modified at one or both ends to obtain a precise fit. The proximal end of the T tube should not abut the conus elasticus of the subglottic larynx, to avoid inciting a ring of potentially obstructive granulations. It is acceptable to seat the proximal end of the tube within the subglottic larynx, but below the curve of the conus. Distally, the tube should extend below the lesion, but not so far that evacuation of secretions is made more difficult, or that the lower tip of the tube abuts and irritates the carina, giving the potential for forming granulations.

Final tube lengths are determined in the following manner. After initial approximation of the length from bronchoscopy, the tube is trimmed conservatively, inserted into the trachea, and position observed carefully. The proximal end is examined with the 0° Hopkins telescope. If a short (25 cm) ventilating bronchoscope is used at this point, then the telescope can be passed through the longest of T tubes and the location of the distal end of the T tube observed. Alternatively, a flexible bronchoscope is passed through a sealing gasket via the rigid bronchoscope to check the position of the distal end of the T tube relative to the lesion and carina. The lower end of the tube must lie lower than the obstructing lesion, or granulations will form distal to the tube.

Trimming the tips of the tube may be done in a number of ways. The tube is cut sharply with a scalpel, resting the tube on a firm surface while this is being done. A sterile paper millimeter ruler included in the kit permits precise measurement of lengths removed and lengths remaining. The cut end of the

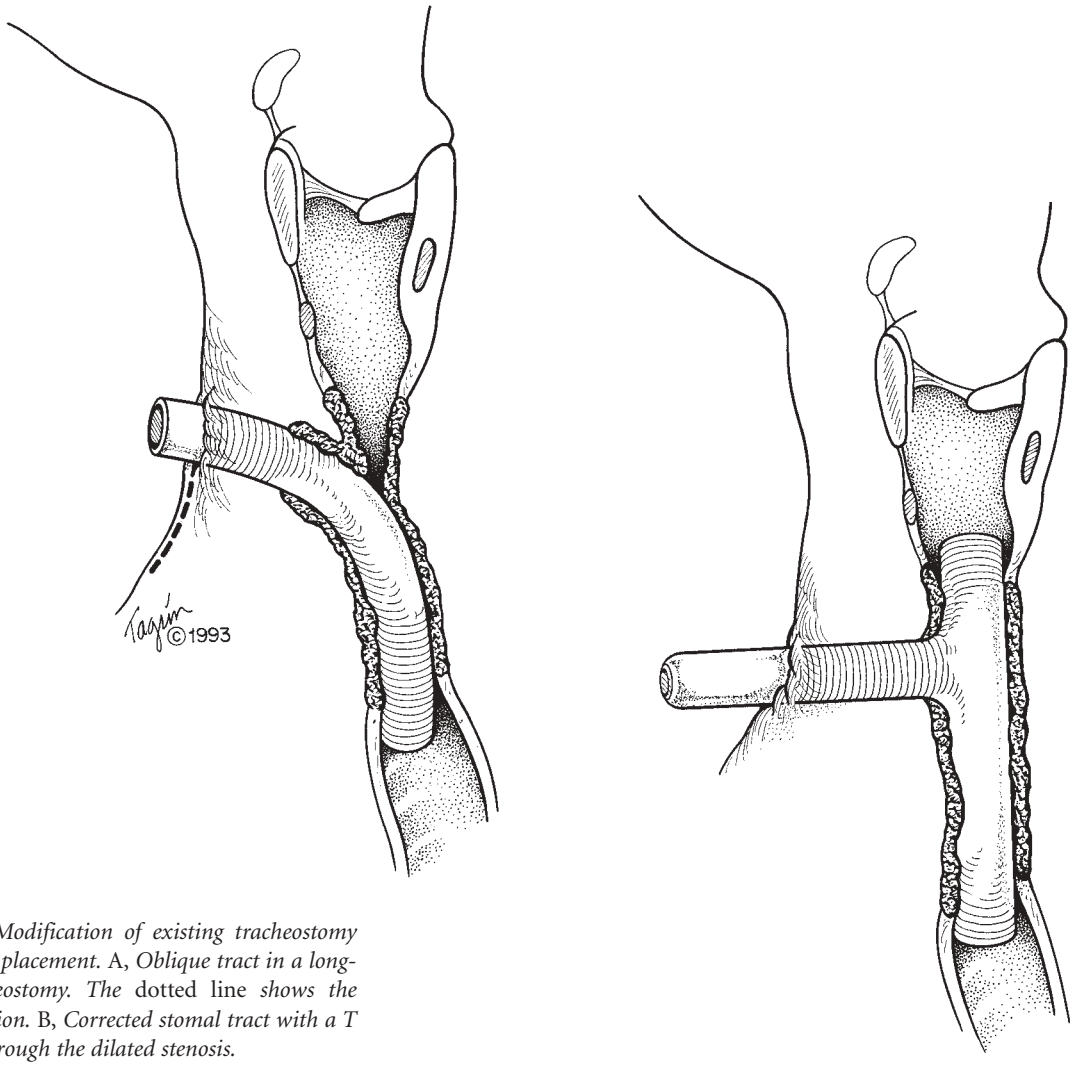


FIGURE **39-3** *Modification of existing tracheostomy tract for T tube placement.* A, *Oblique tract in a long-standing tracheostomy. The* dotted line *shows the modifying incision.* B, *Corrected stomal tract with a T tube in place through the dilated stenosis.*

tube is sanded smooth with sterile emery paper to produce a rounded end similar to that of the original manufacture. Excess bits of silicone produced by the grinding process are, of course, washed off with saline. This process can be speeded a bit by trimming the sharp outer circumference of the freshly cut edge with scissors on a 45º obliquity and then sanding the resulting rough cut. The precise lengths of proximal and distal vertical limbs of the final tube and its total length are measured and recorded. This will facilitate later changes and revisions. When a tube that has been trimmed in this fashion is changed, it provides a template for a replacement tube. During final bronchoscopic examination of the tube's position, an adaptor from an endotracheal tube may be slipped into the sidearm of the T tube and adequate ventilation carried out with high flow directly through the tube, despite proximal loss of gases.

The technique of insertion is done according to Montgomery's description (Figure 39-4). The distal end of the tube is grasped with Kelly forceps. I prefer to invert the distal portion of the tube into a U-shape and grasp it from either side, thereby compressing the tube into a relatively small leading diameter (see Figure 39-4*A*). With liberal use of a water-soluble surgical lubricant, the tube is passed through the existing stoma into the distal trachea. A second Kelly clamp grasps the tube higher up, close to the sidearm, thrusting the tube distally as the first Kelly clamp is released and withdrawn carefully (see Figure 39-4*B*). The tube is now pushed

distally so that the upper limb is inserted into the distal trachea first and then permitted to snap up into the upper trachea above the stoma (see Figure 39-4*C*). Passage of a suction catheter distally, to clear out secretions and blood, is facilitated by angulating the sidearm upward towards the chin with a finger.

If great difficulty is encountered in seating the upper end of the tube, then it may be overcome by a technique described by Cooper and colleagues.[6] A long tracheostomy tape is passed through the sidearm of the tube, out through the proximal vertical limb, and thence into the stoma, where it is grasped by forceps through the rigid bronchoscope and pulled out through the bronchoscope proximally (Figure 39-5*A*). The distal tube is pushed downward into the distal trachea and a clamp is placed across the sidearm, fixing the tape firmly in the T tube (Figure 39-5*B*). The bronchoscopist next pulls firmly and tightly on the portion of the tape that passes through the bronchoscope, which serves to snap the proximal limb into the tracheal lumen (Figure 39-5*C*). The clamp on the sidearm is removed and the tape withdrawn. Cooper and colleagues described passing a T tube over a bronchoscope perorally, using an occlusive balloon on the bronchoscope.[6] We have not found this technique necessary. Once the T tube is seated, ventilation continues through the sidearm. If strenuous manipulation is necessary, then Decadron is frequently given intraoperatively and for a short period postoperatively. Racemic epinephrine can also be considered.

The final position of the tube is always checked bronchoscopically. A further check on its effectiveness is whether ventilation through the sidearm is satisfactory. Where pathology permits, and where the patient can tolerate it and breathes well, I prefer to cap the sidearm of the tube immediately. The patient leaves the operating room breathing spontaneously through a normal route across the pharynx. It is almost impossible to ventilate satisfactorily through a T tube even if a Fogarty or Pruitt catheter is placed in the proximal vertical limb

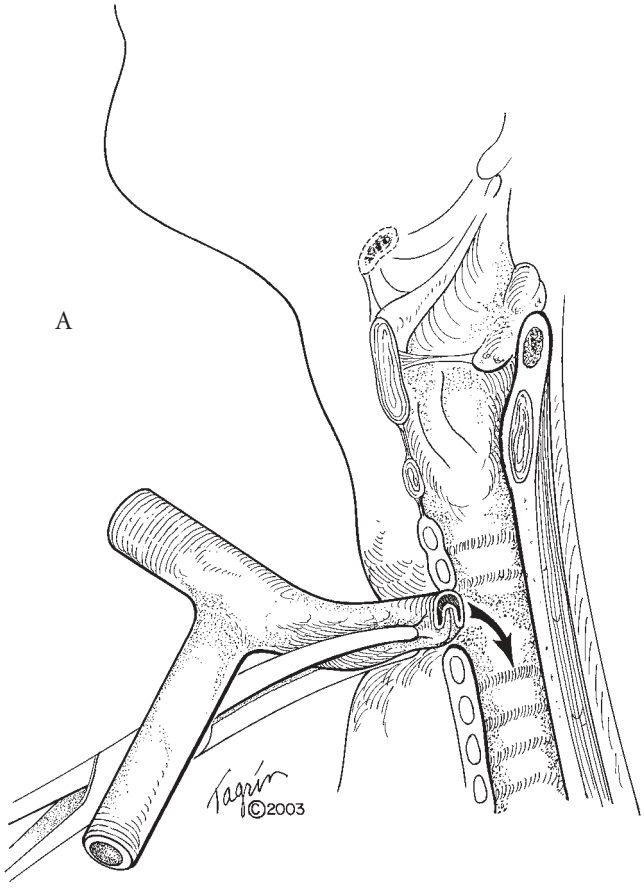


FIGURE **39-4** *Technique of insertion of T tube.* A, *Folding the distal end of the T tube on itself and grasping it with a curved clamp close to its tip produces a "probe," which is easily directed into the stoma and distal trachea.*

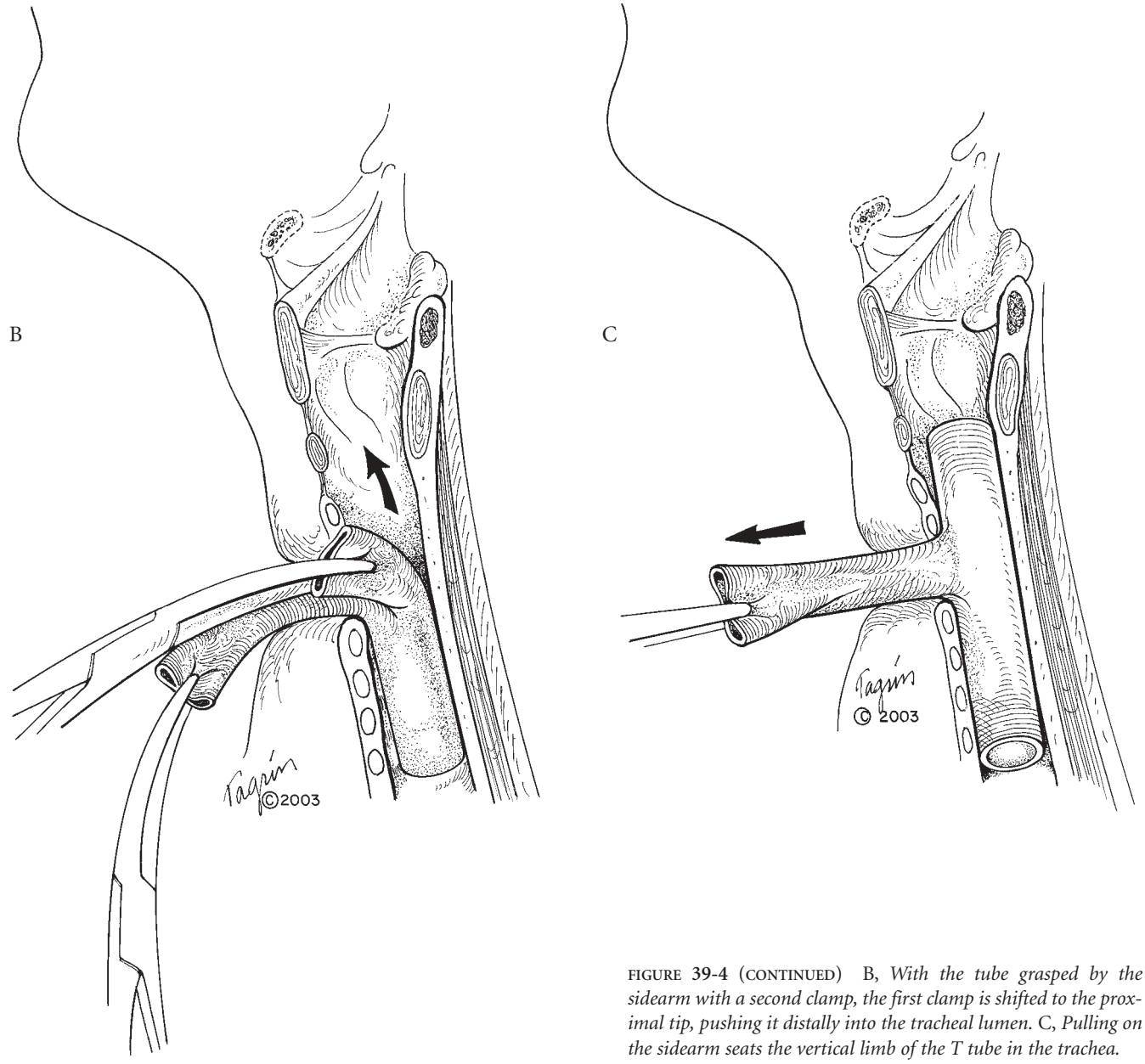


FIGURE **39-4** (CONTINUED) B, *With the tube grasped by the sidearm with a second clamp, the first clamp is shifted to the proximal tip, pushing it distally into the tracheal lumen.* C, *Pulling on the sidearm seats the vertical limb of the T tube in the trachea.*

and an adaptor is placed in the sidearm of the T tube. The T tube is too flexible to be a safe conduit for ventilation except briefly in the operating room, even though the lesion itself provides a seal around the distal tube.

In patients who have undergone multiple prior tracheal procedures (failed reconstruction, several tracheostomies at different levels, lasering, etc) with destruction of long lengths of trachea, and in whom proximal and distal tracheal remnants are wholly separated by scar, cervical exploration may be necessary in order to insert a tracheal T tube. In others, an extent of injury precluding safe end-to-end reconstruction only becomes evident after exploration. In several patients with tracheoesophageal fistula in addition to extended tracheal stenosis, the fistula was reparable but a T tube was used to restore a functional airway. Pedicled strap muscles are used to cover the bridging T tubes in these patients. The results can be very satisfactory.

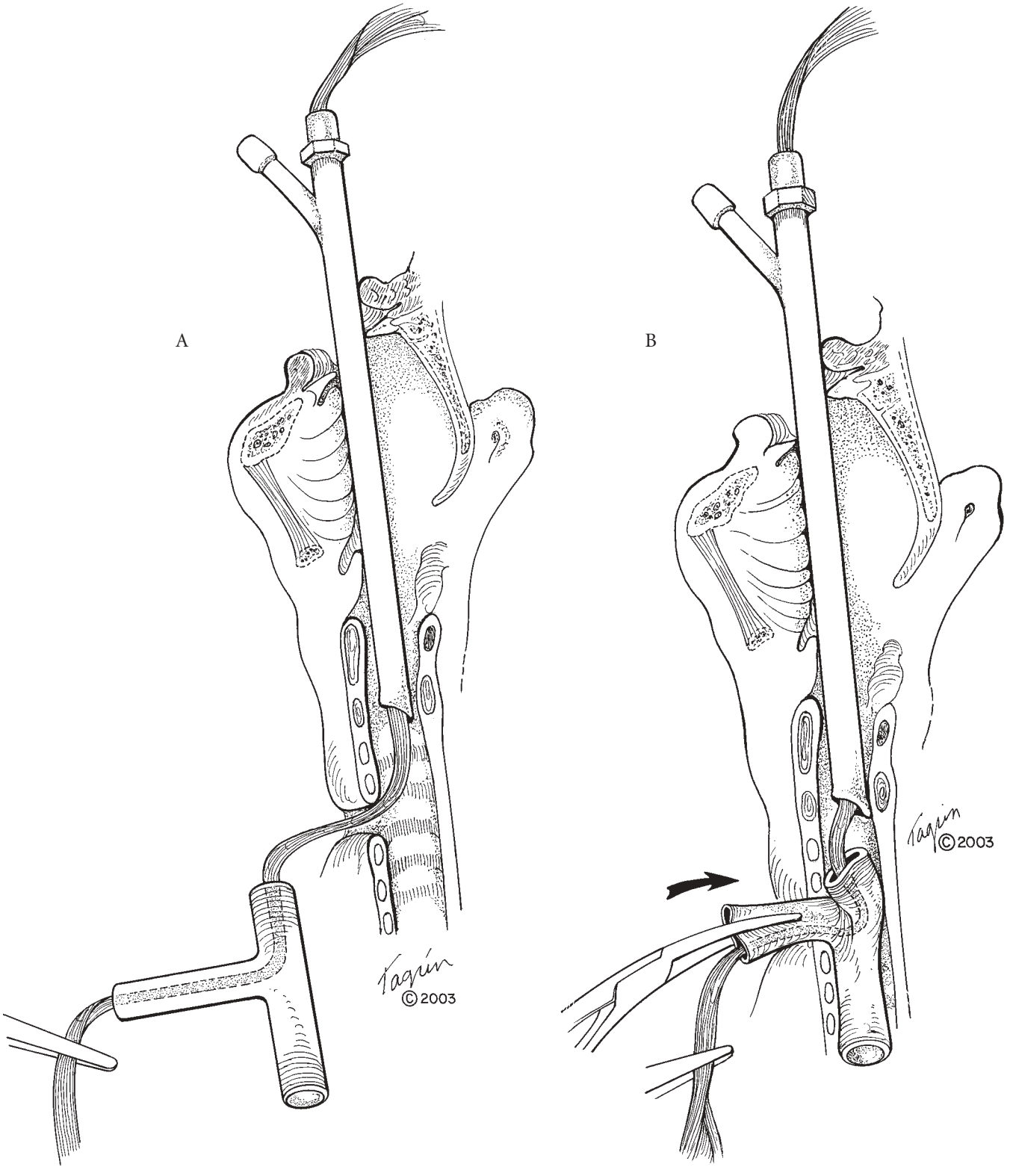


FIGURE **39-5** *Insertion technique if placement is unusually difficult.* A, *Umbilical tape passed through the T tube sidearm and the proximal vertical arm is retrieved from the tracheal stoma via the rigid bronchoscope and pulled through the bronchoscope.* B, *The T tube insertion is commenced in the usual way, and then a clamp placed across the sidearm of the tube to fix the tape firmly.*

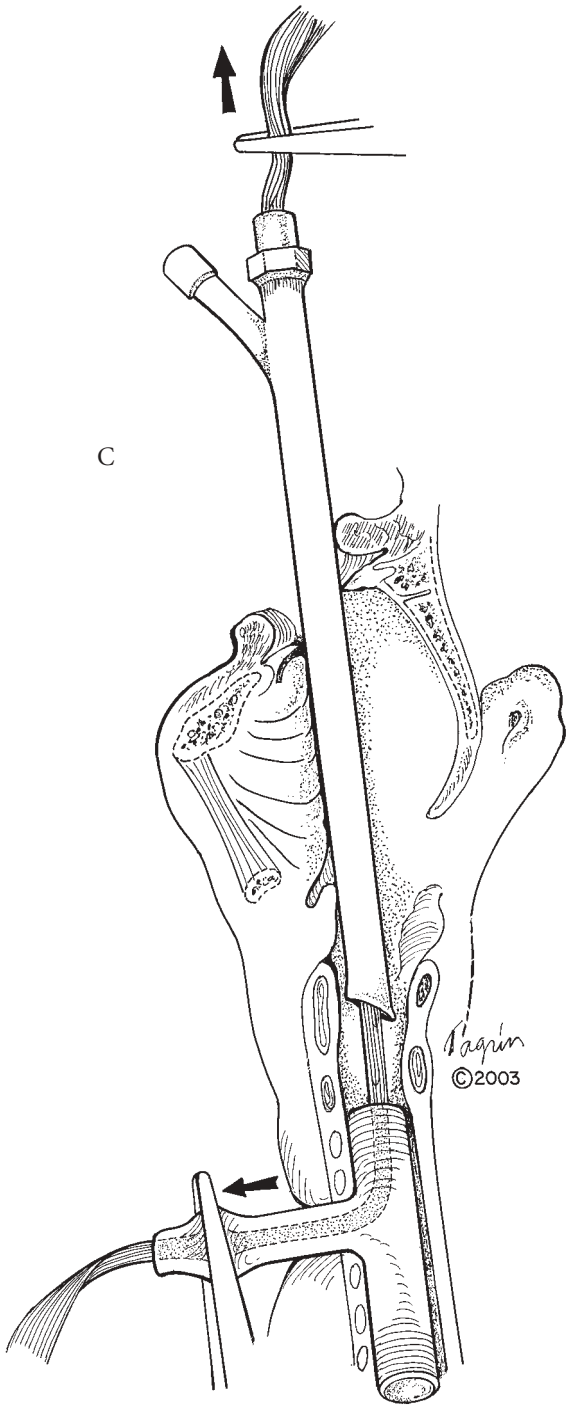


FIGURE **39-5** (CONTINUED) C, *Firm traction is made on the tape through the bronchoscope to draw the proximal vertical arm into the trachea. The tape is then withdrawn.*

Immediate Care

If there is any immediate difficulty in ventilation due to glottic edema from the manipulations, pharyngeal secretions, or soft tissue obstruction in the not fully awake patient, then 100% humidity is provided through a tracheostomy mask placed over the sidearm of the tube. This is necessary to prevent inspissation of secretions in the tube. If the patient must remain initially without a cap on the tube, it is important that the nursing staff be aware of the critical need for continuous humidification. A T tube is not a tracheostomy

tube. On principle, once placed, it should never be left uncapped except when treatment is being given or in an initial period of airway adjustment and patient awakening.

In most patients, the long sidearm will protrude significantly from the skin of the neck. Once edema of the incision has regressed, the tube may indeed be trimmed to within a couple of centimeters of the skin surface.

In some T tubes, the cap is attached to the sidearm to prevent its loss. The connecting piece is designed so that it may be cut off and a slot in the strap slipped over the sidearm if the sidearm must be shortened. If the stopper is free, we prefer to place a loop of suture through the cap and attach this to the patient's neck with a tracheostomy tape passed loosely through the loop.

The irritation of the procedure and the presence of a foreign body, however inert, may initially stimulate considerable secretions. A brief course of antibiotics appropriately directed to organisms present may favorably affect this. The tube is ordinarily not fixed to the skin unless the patient is uncontrolled. In a rare patient of particularly heavy build, or in whom the trachea at the level of the stoma is far from the surface of the skin, it has been necessary to cement an extension to the sidearm with silicone adhesive, for easy suctioning. The best way to do this is with a segment cut from another tube of the same size as the sidearm, held with a short collar cut from the next larger size of silicone tubing.

Modifications of Tubes and Placement

Modifications of the silicone tracheal T tube are useful for special circumstances. Westaby and colleagues fashioned a T-Y tube for lesions involving the carina and main bronchi (Figure 39-6).[8] The T-Y tube may be inserted over two ureteral catheters, which pass through the tube and its right and left bronchial limbs into the tracheal stoma, and by bronchoscopic placement, into right and left main bronchi. A simpler alternative is to squeeze the right and left limbs together, with a bronchoscopic foreign body forceps placed through the T-Y tube, and insert the tube directly into the stoma. The proximal end of the tube is snapped upward into the proximal trachea. The placement is confirmed by a flexible bronchoscopy.

Because the angles of the bronchi are not the same in all patients, it is necessary in some cases to design a tube with special dimensions and angles. The length of the tube and the point at which the external sidearm emerges may also need to be specified. In general, the T-Y tube is designed so that the sidearm enters at an angle which is obtuse distally. An advantage of the T-Y tube over a Y stent is that the carinal Y will not be displaced upward because of the fixation by the sidearm. Furthermore, access for suctioning is always available and the tube can be extracted with ease emergently and a tracheostomy tube replaced. In special cases, we have removed one of the sidearms of the T-Y tube, converting it to a T-L tube. If necessary, a bronchial limb may extend into the bronchus intermedius, and a further side hole is cut out for the right upper lobe bronchial orifice.

With patients who have had a right pneumonectomy, but have airway obstruction of the carina or left main bronchus, a very long tube has been fashioned, using a segment of a Hood Montgomery silicone salivary tube (Figure 39-7). The next larger size of the standard T tube is cut to produce a short collar attached to a sidearm. A side opening is made at the appropriate level of the salivary tube segment and the collar with the sidearm is slipped over the tube and cemented in place. For long-term management, special tubes may be designed and ordered individually. Bronchial stents may be preferred for some patients (see Chapter 40, "Tracheal and Bronchial Stenting").

In the special case of a subglottic stenosis that extends close to the vocal cords, the T tube must pass through the vocal cords to be effective. If placed directly beneath the cords, irritation may produce granulation tissue or edematous obstruction. Subglottic and upper tracheal inhalation burns are similarly managed.[9] A tube placed in this fashion should end between the false and true cords and not impinge on the epiglottis. Because of the small size of the glottic aperture, Robert H. Lofgren has designed a T tube with a proximal upward tapering vertical limb that traverses the glottis (see Chapter 35, "Laryngologic Problems Related To Tracheal Surgery"). A tube through the glottis interferes with the voice, leaving the patient with

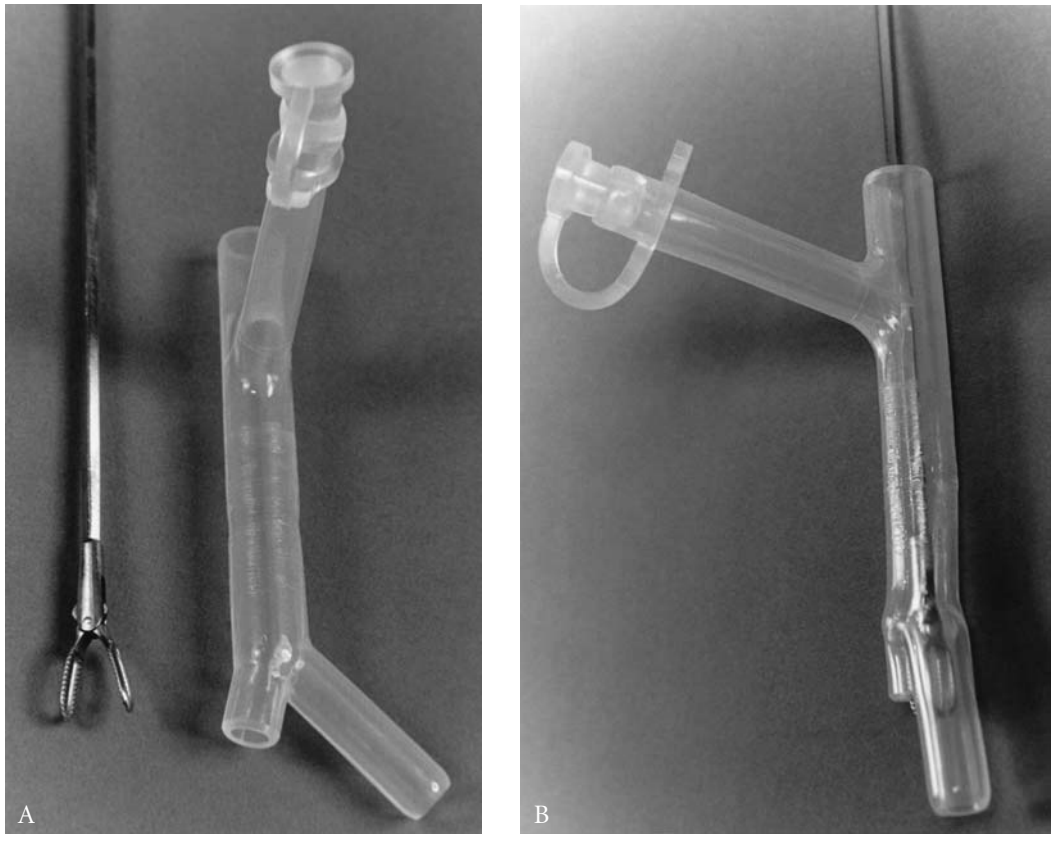


FIGURE **39-6** *Silicone T-Y tracheal tube for carinal placement.* A, *The tube with a stout bronchoscopic foreign body forceps beside it.* B, *The bronchial limbs of the Y tube are squeezed together by the forceps to allow insertion via the stoma. Water-soluble lubricant eases the passage.*

a hoarse or a whispered voice, but one that is intelligible. Usually, the patient is able to swallow without aspiration after a short period of time. A speech pathologist can help to train the patient to avoid aspiration. Generally, the epiglottis and false cords protect sufficiently against aspiration.

In order to meet patients' objections to the protrusion of the sidearm of a T tube through a cervical stoma, Keszler removed most of the sidearm in some patients and placed the capped end subcutaneously, depending on the stump to hold the tube in place.[10] I have not used this technique, which today becomes another form of inlying stent that is complicated in placement.

Tube Management

As noted, the tube should be kept capped at all times except as described. A few cc's of saline are instilled once or twice a day.[11] The patient usually clears secretions with a cough. Suctioning should be available, especially initially, even at home, using a portable machine. The patient and family are taught to instill saline and also to pass the suction catheter distally by angling the protruding tube upward or proximally, if necessary, by angling it downward. Proximal suctioning, which will tickle the undersurface of the vocal cords, is likely to produce cough and sometimes gagging. If the catheter is passed too far distally, it produces cough from carinal reflex. Saline instillation may be used more frequently, if necessary.

Acetylcysteine instillation for care of a T tube is not routinely necessary. In some patients, acetylcysteine appears to produce more irritation and cough. The stoma itself is cared for in the usual manner.

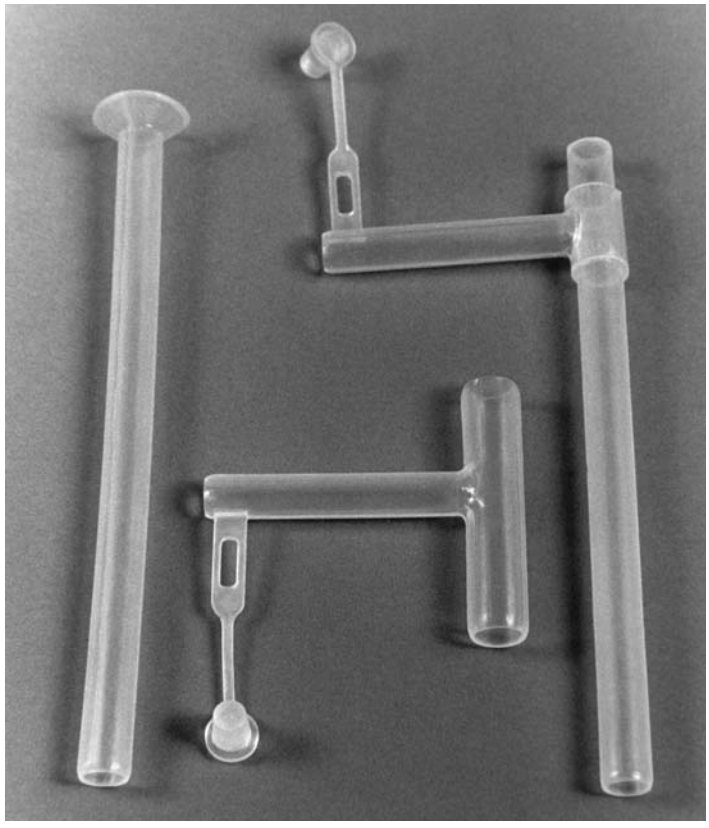


FIGURE 39-7 *Fabrication of a very long T tube in the operating room. The tube is fashioned from a Montgomery silicone "salivary tube," shown at the left, by removing the funnel and cutting it to the desired length. The cut edge is sanded smooth. A side opening is cut out at the level desired for the sidearm. The sidearm is constructed from a standard T tube of next larger size (at the center of the illustration), by cutting off the proximal and distal vertical limbs to produce a short collar. The collar is then cemented into place, as seen on the right.*

Swabs with hydrogen peroxide or saline are used to clean the peritubal area daily, or more frequently, as needed. No dressing is required. Granulation tissue around the stoma is treated by removal and cautery with silver nitrate sticks.

Patients have kept T tubes in place for years without difficulty. In general, however, we recommend that the tube be changed about once a year. Silicone rubber ages, and eventually, the sidearm may crack or even separate from the vertical tube. Inspissated material inevitably sludges inside the tube after long periods of time. The rate of formation of such sludge, even with the best of care, will vary from patient to patient. Patients should also be instructed that if they experience shortness of breath on exercise or hear wheezing, then their tube patency should be examined promptly by a physician. This is easily done with a flexible bronchoscope through the sidearm.

Experience with T Tubes

In general, tracheal reconstruction after resection should stand on its own merits and not require splinting. This is also true for laryngotracheal reconstruction. In only two unusual patients were T tubes used after resection and reconstruction. In one, there was concern about a segment of uncertain stability *above* the anastomosis.

The majority of 140 patients in whom the T tube was used suffered from postintubation lesions, burns, or malignant tracheal tumor (Table 39-1).[7] The indications for *temporary use of a T tube* were stated above. In 14 patients, it preceded reconstruction, and in 16 patients, it was the sole treatment for lesions that had resolved with splinting from 3 months to 9 years—most often in 4 to 6 months.

The tube was used *definitively as palliation* in 49 patients.[7] Six had an unresectable tumor; 4 with squamous cell carcinoma survived 1 and 17 months, whereas 2 with adenoid cystic carcinoma survived

10 months and 2 years, respectively. The remaining 43 patients had a benign tracheal stenosis that was unresectable, either because of limited residual length of normal trachea which was suitable for reconstruction or, less often, because of contraindications such as advanced cardiopulmonary disease. Two of these patients had a left main bronchial stenosis after prior right pneumonectomy, 27 then living had had T tubes in place between 5 months and 16 years (mean 55 months), and 15 who died between 1 month and 6 years had the tubes in place for a mean of 21 months. A patient died of tracheal hemorrhage, but the causes of death in the others were not related to the T tube.

Thirty-two patients used the T tube *postoperatively after tracheal resection*. Sixteen were referred from other institutions and 16 had reconstruction at the Massachusetts General Hospital. If reconstruction fails, I prefer to wait 4 to 6 months before attempting a second reconstruction. If mechanical reconstruction is delayed, a T tube is the preferred method of maintaining the patient while the postoperative inflammation resolves. In 13 of the 32 patients, the T tube was placed in the immediate postoperative period, and in the remaining 19, after delay of 1 month to 14 years following reconstruction. Five of these patients achieved decannulation without other procedures, whereas in another 5 patients the T tube permitted successful re-resection later, but 11 patients continued to have tracheal tubes—8 T tubes and 2 T-Y tubes and 1 tracheostomy tube (due to subglottic stenosis).

In 28 of our patients (20%), the T tube did not create a reliable airway and had to be removed within 2 months of placement. This was due primarily to airway obstruction (19), but also in a few to aspiration (3), obstruction and aspiration (2), patient noncompliance (2), secretions (1), and tracheobronchitis (1). Most commonly, the obstruction was proximal to the subglottic area. In 2 patients, the problem proved to be that of sleep apnea, not permitting a closed T tube nocturnally. In these patients, the T tube served to demonstrate that a tracheal reconstruction should not be done, and a tracheostomy tube was reinserted.

It should be noted that success was less satisfactory in children than in adults. The T tube failed in 5 of 10 patients under 10 years of age, reflecting the small dimensions of the pediatric airway and particularly the subglottic space.

Table 39-1 Diagnosis in 140 Patients in Whom T Tubes Were Used

Diagnosis	*Number of Patients*
Postintubation stenosis	86
Burn	13
Malignant airway tumor	12
Radiation stenosis	4
Relapsing polychondritis	4
Tracheomalacia	4
Vascular malformation	3
Sarcoidosis	2
Trauma	2
Necrotizing tracheitis	2
Mucopolysaccharidosis	2
Postpneumonectomy syndrome	2
Tracheobronchomegaly (Mounier-Kuhn)	1
Tuberculosis	1
Idiopathic stenosis	1
Tracheopathia osteoplastica	1

Reproduced with permission from Gaissert HA et al.[7]

There were two episodes of airway obstruction with a T tube in place. Both patients died in outside hospitals and the role of the T tube was not entirely clear. It is not known whether the tube was removed when the asphyxiation occurred. The episode of hemorrhage in a 12 year old, presumably from the innominate artery, after placement of a T tube, has been noted.

The tracheal T tube can be handled successfully by many patients in the long term. The longest period noted was 20 years. Once the tube is tolerated for 2 months, late problems appear unlikely. In 49 patients, intubation was continued for 1 year, and in 12 patients for over 5 years. Review of 33 patients, who lived with a T tube chronically, showed mild or absent respiratory symptoms in most of them. Exercise tolerance was normal in 7, mild dyspnea on severe exertion was present in 16, and 10 patients had marked dyspnea on exertion, plus wheezing in another 10. Most of this latter group had a pulmonary parenchymal disease or reduced functional reserve for varying reasons.

T tubes are also used as the basis for reconstruction in a unique situation: correction of obstructing tracheopathia osteoplastica (see Chapter 32, "Surgery for Tracheomalacia, Tracheopathia Osteoplastica, Tracheal Compression, and Staged Reconstruction of the Trachea").

Conclusions

A silicone T tube developed by Montgomery is an important adjunct in surgery of the airways.[2,3] In a patient with a functional larynx, it offers the closest approximation to a normal airway with good humidification of the lower airways, preservation of voice, and social acceptability. Daily care is easy and not rigidly demanding. I generally prefer the immediate access provided by these tubes over fixed or inlying stents that are inaccessible from the outside. A T tube does not preclude later surgical treatment. In 10 of 12 patients with subglottic stenosis, a tube placed through the vocal cords was tolerated quite well, as pointed out by Cooper and colleagues.[4] At present, for the patient with a benign but unreconstructible lesion of the trachea, the T tube appears to be an excellent method of providing a stable and dependable airway.

Inlying silicone stents of the Dumon type, with projecting knobs to hold the stent's position, tend to migrate, especially when used in the upper trachea. Expandable metallic stents, coated or not, may incite serious, sometimes irreversible, obstructing granulations, limiting their advisability for treatment of benign lesions (see Chapter 40, "Tracheal and Bronchial Stenting").[12] The silicone T tube obviates all these problems but presents the disadvantage of an opening to the surface of the neck.

References

1. Bond CJ. Treatment of tracheal stenosis by a new T-shaped tracheostomy tube. Lancet 1891;1:539.
2. Montgomery WW. The surgical management of supraglottic and subglottic stenosis. Ann Otol Rhinol 1968; 77:534–46.
3. Montgomery WW. Silicone tracheal T-tube. Ann Otol Rhinol Laryngol 1974;83:71–5.
4. Cooper JD, Todd TRJ, Ilves R, Pearson FG. Use of the silicone tracheal T-tube for the management of complex tracheal injuries. J Thorac Cardiovasc Surg 1981;82:559–68.
5. Landa L. The tracheal T-tube in tracheal surgery. In: Grillo HC, Eschapasse H, editors. International trends in general thoracic surgery. Vol 2. Philadelphia: WB Saunders; 1987. p. 124–32.
6. Cooper JD, Pearson FG, Patterson GA, et al. Use of silicone stents in the management of airway problems. Ann Thorac Surg 1984;47:371–8.
7. Gaissert HA, Grillo HC, Mathisen DJ, Wain JC. Temporary and permanent restoration of airway continuity with the tracheal T-tube. J Thorac Cardiovasc Surg 1994;107:600–6.
8. Westaby S, Jackson JW, Pearson FG. A bifurcated silicone rubber stent for relief of tracheobronchial obstruction. J Thorac Cardiovasc Surg 1982;83:414–7.
9. Gaissert HA, Lofgren RH, Grillo HC. Upper airway compromise after inhalation injury: complex strictures of larynx and trachea and their management. Ann Surg 1993;218:672–8.
10. Keszler P. The tracheal T-tube for indwelling intubations as an alternative management method. In: Grillo HC, Eschapasse H, editors. International trends in general thoracic surgery. Vol. 2. Philadelphia: WB Saunders; 1987. p. 133–7.
11. Dempsey R. Nursing manual and home care instructions: tracheal and thoracic T-tubes. Pembroke (MA): Hood Laboratories; 1992.
12. Gaissert HA, Grillo HC, Wright CD, et al. Lengthening and complication of benign tracheobronchial strictures by self-expanding metal stents. J Thorac Cardiovasc Surg 2003. [In press]

Tracheal and Bronchial Stenting

Douglas E. Wood, MD

Stenosis or obstruction of the central airways produces symptoms of dyspnea, stridor, and obstructive pneumonia. Depending on the location and extent of the obstruction, patients may suffer from impending suffocation, and a slight exacerbation of the narrowing by edema, secretions, or blood may be immediately life threatening. In most cases, resection and surgical reconstruction provide the best opportunity for definitive management. However, bronchoscopic management is the first step in providing a diagnosis, stabilizing the obstructed airway, and evaluating resectability. Many patients may be found to be unresectable, and it is in these patients that endoscopic techniques, including airway stenting, can provide a minimally invasive and effective palliation of their airway obstruction. Although the long-term outlook in these cases is often dismal, the temporary or permanent relief of airway obstruction provides significant palliation, with marked improvement in quality of life and potential prolongation of life.

A variety of endoscopic techniques have been developed for the palliation of central airway obstruction. Endobronchial débridement (core-out), laser resection, endobronchial brachytherapy, and photodynamic therapy have all been used successfully for malignant airway stenosis. Similarly, dilation and laser resection have been used for the management of benign tracheobronchial stenosis. Although advocates for each of these techniques have reported success with individual modalities, most would agree that a flexible application of these techniques, even combined in the same patient, provide the best chance for a successful outcome.[1]

In both benign and malignant diseases, tracheobronchial stents have been used to palliate the effects of large airway obstruction caused by extrinsic compression, intraluminal disease, or loss of cartilaginous support. As experience has been gained with specially designed stents for the biliary tree, esophagus, urinary tract, and blood vessels, there has likewise been a proliferation of devices for use in airway stenting (Table 40-1). Advances in airway prosthetics have produced a variety of silicone stents, expandable metal stents, and pneumatic dilators, enabling the correction of increasingly complex anatomic problems.

Although airway resection and reconstruction is the preferred therapy for both benign and malignant lesions, a variety of factors, including a long stenosis, failed previous repair, metastatic or unresectable malignancy, or patient refusal, may dictate nonsurgical management.[2,3] Tracheobronchial stents also pro-

Table 40-1 Characteristics of Major Airway Stents

Stent Type	*Manufacturer*	*Construction*	*Delivery*	*FDA Approved ?*	*Sizes (mm)*	*Costs (approx US$)*
Hood	Hood Corporation	Molded silicon rubber	Tube stent	Yes	6 × 13 – 18 × 70 plus Y stents	$100–$350
Dumon	Bryan Corporation	Molded silicon rubber	Tube stent	Yes	9 × 20 – 16 × 60 plus Y stents	$350
Dynamic	Rüsch Incorporated	Silicone with anterolateral steel struts	Y stent	Yes	Y stents only 3 sizes: 13, 15, 17 mm tracheal diameter	$1,600
Wallstent	Boston Scientific	Woven cobalt / chrome alloy monofilaments coated with silicone	Self-expandable constrained within delivery catheter	Yes	8 × 20 – 24 × 60	$1,495
Ultraflex	Boston Scientific	Single-strand woven nitinol, with and without silicone coating	Self-expandable constrained within delivery catheter	Yes	8 × 20 – 20 × 80	$1,625
Strecker	Boston Scientific	Single-strand tantalum mesh	Balloon expanded deployment	Yes	8 × 20 – 11 × 40	
Palmaz	Johnson and Johnson	Expandable slotted stainless steel tube	Balloon expanded deployment	Yes	8 × 10 – 12 × 40	1,000–$2,000
Wallgraft	Boston Scientific	Woven cobalt / chrome alloy monofilaments with polyethylene covering	Self-expandable constrained within delivery catheter	Yes	6 × 20 – 12 × 70	$1,950–$2,250
Polyflex	Rüsch Incorporate	Polyester mesh coated with silicone	Self-expandable constrained within delivery catheter	Yes	6 × 20 – 22 × 80	$1,300
Gianturco	William Cook Europe	Cylindrical zigzag stainless steel monofilament	Self-expandable constrained within delivery catheter	Yes	6 × 25 – 35 × 50	$150–$450

FDA = US Food and Drug Administration.

vide an alternative to open surgical procedures that may be preferred by some patients or physicians. In other cases, endoscopic management may act as temporizing palliation while stabilizing or evaluating a patient for subsequent resection.

Benign and malignant airway obstruction is uncommon, resulting in limited clinical experience for most thoracic surgeons and pulmonary physicians, even at tertiary medical centers. Experience with interventional bronchoscopy, including stent placement, is an important part of any thoracic surgical program that performs airway resections or lung transplantation, providing for management of postoperative anastomotic complications. Similarly, the surgeon or pulmonologist considering stenting for airway palliation should be experienced with both the indications and techniques of airway resection, or be a part of a closely linked multidisciplinary team that allows a balanced consideration of airway resection versus therapeutic bronchoscopy. Because of the relative rarity of these lesions, it may be preferable to manage patients with central airway obstruction in a specialized tertiary center that has the experience and multidisciplinary team to offer consideration and application of the full spectrum of techniques of complex airway management.

Historical Perspective

Charles Stent was a British dentist who practiced in the late nineteenth century and developed dental impression material that was used as a template to support healing skin grafts. His pioneering work resulted in the term "stent" being used to describe such artificial structural methods for preserving the viability and function of tissue. Today, the term is used most commonly for methods of maintaining patency of tubular structures, including the tracheobronchial tree.[4] The concept of the airway stent dates back to the late 1800s, with publications by Trendelenburg in 1872 and Bond in 1891.[5,6] The first modern treatment of a major airway obstruction by placement of an endotracheal stent was reported by Harkins in 1952, describing the use of metal tubes for a benign tracheal stenosis resulting from trauma.[7] Successful stenting of the airway by a silicone prosthesis was first described in 1965 by Montgomery, who used a silicone T tube for stenting of the trachea.[8] Some of the earliest attempts at airway stenting consisted of surgical placement of silicone tubes in the trachea.[9,10] The best known of these surgical prostheses was the Neville stent, which was a silicone stent with Dacron extremities sutured into place during thoracotomy.[11]

The current generation of bronchoscopically placed endoluminal stents was introduced by Duvall and Bauer, who modified the Montgomery T tube design so that it could be inserted by bronchoscopy.[12] Other authors modified the Montgomery T tube, lengthening the distal limb and adding a bifurcation to provide support for the mainstem bronchi.[13] Ultimately, the external side limb of the T tube was removed and the subsequent cylinder stent inserted into the airway during laryngoscopy, rigid bronchoscopy, or over bronchial bougies.[14] In 1989, Cooper and colleagues reported on a modified Silastic stent, used in 11 cases for malignant tracheobronchial obstruction.[15]

Dumon published his preliminary results of a newly designed stent, used in 66 patients in the late 1980s.[16] Dumon had experimented with modified Montgomery T tubes throughout the 1980s and subsequently patented what is the most widely used tube stent today, the Dumon stent (Bryan Corporation, Woburn, MA). This stent is made of molded silicone with external studs at regular intervals to prevent dislodgment. A Y-modification for stenting of the carina and main bronchi has also been introduced. Silicone stents by other manufacturers are also available, which may have proximal and distal flanges for stabilization or have external studs like the Dumon stent (Hood Laboratories, Pembroke, MA). One of the most recent modifications has been designed by Freitag (Dynamic stent, Rüsch, AG, Kernan, Germany) and is a silicone Y stent with the anterolateral walls reinforced with metal hoops. The nonreinforced silicone posterior wall of the stent is collapsible, mimicking the dynamics of the membranous trachea during inspiration and expiration, with the theoretical benefits of facilitating expectoration and avoiding secretion accumulation.[17,18]

Although the silicone stents have been proven to be effective for the treatment of tracheobronchial stenosis from both benign and malignant etiologies, they have several disadvantages that limit their usefulness as an airway prosthesis. One of the shortcomings is wall thickness, which offers a low internal to external diameter ratio, resulting in a relatively smaller usable lumen. These stents also interfere with mucociliary function and may become obstructed by inspissated secretions. Silicone stents may also have problems with dislodgment and potential obstruction of lobar or segmental orifices. Furthermore, in general, placement of silicone stents requires general anesthesia and rigid bronchoscopy and may be technically challenging for endoscopists who do not perform rigid bronchoscopy routinely. Because of these limitations, a number of investigators have developed other types of stents, most notably the metal expandable stents.

Several self-expanding wire stents originally designed for biliary or vascular strictures have been used for malignant as well as benign tracheobronchial stenosis. Besides the initial report by Harkins of a metal alloy stent, others suggested the use of metallic endoprostheses for surgical reconstruction of tracheal and bronchial stenoses.[19,20] Although these stents were not expandable, an inner skeleton comprised of a wire mesh adequately supported the airway. This initial experience, combined with the successful use of metal stents in the vascular and biliary trees, led to the development of first-generation expandable metal stents. Cesare Gianturco developed the Gianturco stent in the animal laboratory at the MD Anderson Cancer Center in the 1980s. The Gianturco stent (Cook, Inc., Bloomington, IN) is a continuous zigzag loop of stainless steel wire that is compressed into a cylinder, allowing delivery into a vascular or airway stenosis. Wallace and colleagues reported their experience with the Gianturco stent in the tracheobronchial tree in dogs in 1986, and soon thereafter, the Gianturco stent was approved for use in humans.[21]

The Palmaz stent (Johnson and Johnson Interventional Systems, Warren, NJ) was first used in 1988 for emergency treatment of tracheal stenosis in a child.[22] The Palmaz stents are examples of fixed diameter balloon expandable stents. These stents do not exhibit any intrinsic radial force and are positioned and seated by balloon expansion. This stainless steel mesh stent has been predominantly used for pediatric patients because of the availability of small diameter sizes. However, because of the inflexibility and complications from both the Gianturco and Palmaz stents, these are now uncommonly used in adult clinical practice in the United States.

The second generation of metal stents was led by the Wallstent, initially marketed by Schneider, Inc., which has subsequently become a subsidiary of Boston Scientific Corp. (Natick, MA). Wallstent is a self-expandable intravascular stent, is delivered in a constrained form and, once released, expands to a preset diameter. The tubular mesh structure of the Wallstent makes it more able to conform to the airway contours than first-generation expandable stents, and its ease of delivery has led to widespread use for tracheobronchial stenoses.

Another second-generation stent, the Ultraflex stent, is also a self-expanding prosthesis woven from a single strand of nitinol. This nickel-titanium alloy stent exhibits properties of "shaped memory," meaning that at low temperatures, the alloy deforms plastically (martensitic state), whereas at higher temperatures, it regains its original shape (austenitic state).[22] The Ultraflex stent (Boston Scientific Corp., Natick, MA) has only recently been released for use in the United States.

Initially, there was enthusiasm for the porous structure of expandable wire mesh stents, theoretically allowing neoepithelialization, resumption of mucociliary clearance, and avoidance of lobar or segmental obstruction if placed across airway orifices. However, complications of granulations and tumor ingrowth through the stent interstices have resulted in further refinement of expandable stents by merging the strengths of silicone and metal stent technologies. The Gianturco, Wallstent, and Ultraflex stents are now all available in covered versions, in which the mesh structure is coated with a silicone or polyurethane coating. Obviously, this prevents the potential benefit of neoepithelialization, but helps alleviate the known complications of ingrowth of tissue through the stent.

Although refinements have continued to be made over the last two decades, the existing stent designs are still flawed. The ideal stent should possess the following characteristics: 1) ease of insertion; 2) ability to adjust or remove; 3) ability to reestablish and maintain airway patency; 4) stability to prevent migration; 5) be firm enough to resist compressive forces yet compliant enough to prevent airway erosion; 6) abiltiy to conform to airway contours; 7) biocompatibility with low incidence of infection or granulation tissue; and 8) normal mobilization of secretions and minimal interference with mucociliary clearance. Although both silicone and metal stents possess some of these characteristics, each also lacks other significant characteristics, and the ideal stent is yet to be created.

Types of Stents

Silicone Stents

The most commonly used silicone stents are the Dumon and Hood stents (Figure 40-1*A*). These are both manufactured from molded silicone. Both of these stents come in a variety of diameters (6 to 18 mm) and lengths (20 to 80 mm). Both also have regularly placed external studs to engage the airway wall and prevent migration. The Hood stents also come in a version that has proximal and distal flanges in the smaller bronchial sizes to prevent proximal or distal migration in an area of stricture. Both the Hood and Dumon stents come in a bifurcated version for stenting of the carina, distal trachea, and proximal mainstem bronchi, simultaneously.

The Orlowski stent is created from polyvinyl chloride with internal metal armor.[23] In order to prevent migration, these stents are long, with the intent of being placed across the carina into one of the main bronchi with a bronchial side port to allow contralateral lung ventilation. This feature complicates stent placement and mucociliary clearance.

The Dynamic stent (Rüsch AG, Kernan, Germany) is Y-shaped, with a long tracheal and left main bronchial limb and a short right main bronchial limb (Figure 40-1*B*). This stent has an anterolateral silicone wall reinforced with a metallic hoop, and a nonreinforced posterior wall to mimic the dynamics of membranous wall of the trachea.

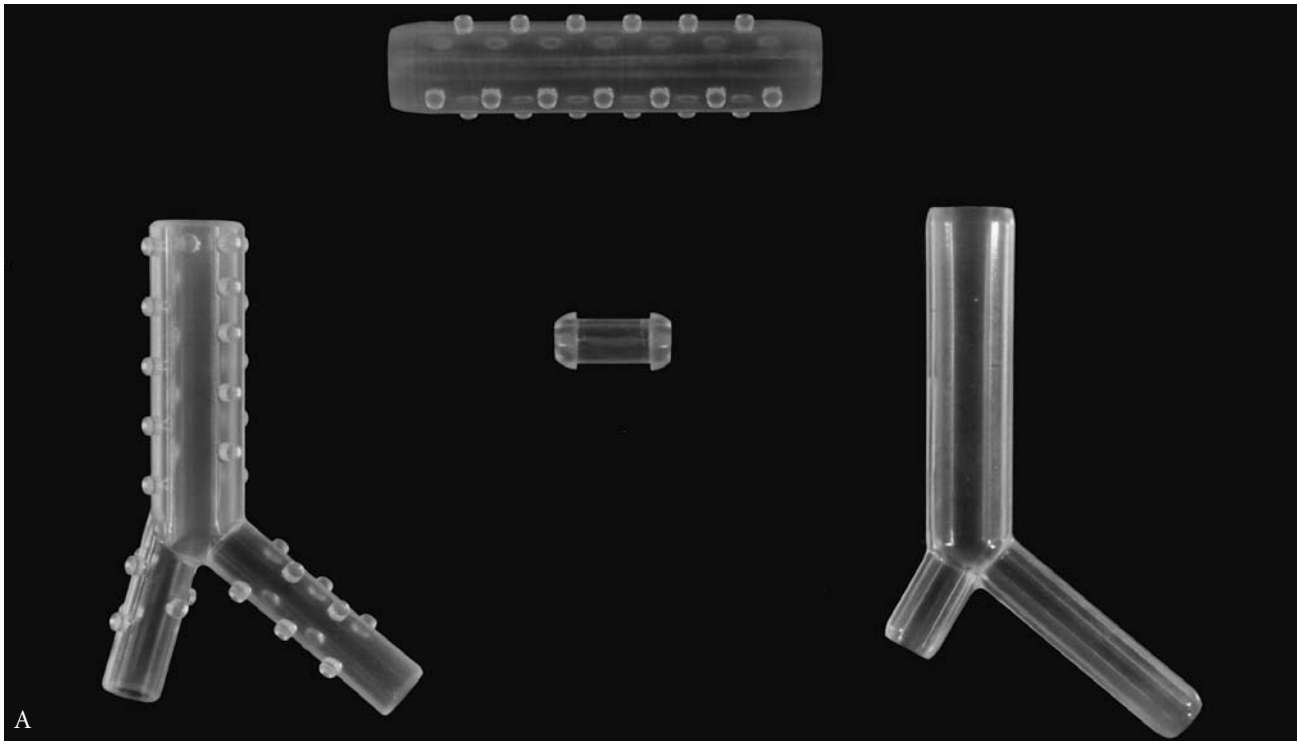
A newer design of silicone stents is the Nova stent (Novadis, St. Victoret, France), which consists of a thin sheet of silicone rolled like cigarette paper to form a tube. Small metal bands are embedded within the silicone that allow it to progressively reexpand to its preformed diameter after insertion.[24]

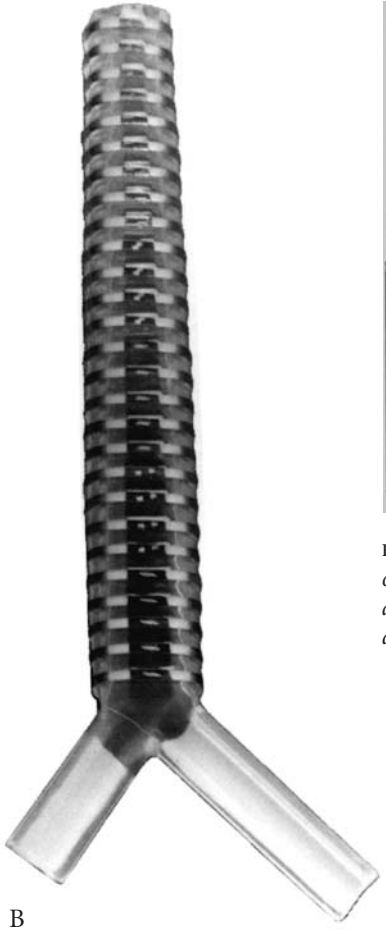
Metal Stents

Gianturco stents are constructed from metal stainless steel monofilament wires, fashioned in a zigzag pattern with 5 to 10 bands.[4] The lengths of the stents are 2.5 cm for tracheal stents and 2 cm for bronchial stents. These stents can be delivered through a 12F introducer sheath. These stents are also available as tandem stents with double the length of the single stents. Because of problems with migration, small hooks are placed along the proximal and distal ends to allow for anchoring into the airway wall.

The Wallstent airway prostheses are made from woven stainless steel monofilament wires, fashioned to form a cylindrical mesh. These stents are longitudinally stretched to compress into an introducer sheath and, as such, shorten upon the self-expansion of deployment (Figure 40-1*C*). Because of the larger contact area than the Gianturco stent, Wallstents do not require hooks to prevent migration. The Wallstent has excellent flexibility and conformance to the airway anatomy. Problems with ingrowth of tumor or granulations through stent interstices have been largely alleviated by coating the Wallstent with a thin layer of silicone rubber (Permalume). There is still 5 mm of bare metal stent at each end to help stabilize the stent and prevent migration, but these areas may still result in tissue ingrowth and secondary stent obstruction.

The Ultraflex stent is made from a single strand of nickel-titanium alloy (nitinol) woven into a cylindical mesh. The increased heat of the body produces self-expansion of the thermal-triggered shape





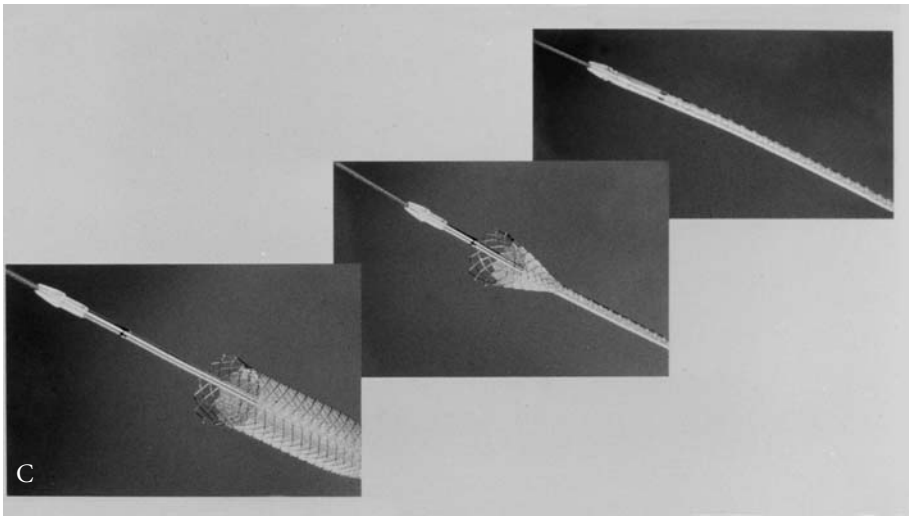


FIGURE **40-1** *Photographs of airway stents.* A, *Hood tracheal, bronchial, and Y stents with flanges or studs for airway seating.* B, *Dynamic stent with bifurcated mainstem limbs and reinforced anterolateral walls.* C, *Wallstent with Permalume coating within the delivery sheath, partially deployed, and fully deployed over the delivery sheath.*

memory property of the stent, once released from its delivery catheter. Both covered and uncovered versions are available, and because of the single-strand nature of the construction, endoscopic retrieval is more feasible than with a Wallstent or Gianturco stent.

The Palmaz stent is a fixed diameter stent made of stainless steel, with staggered rows of rectangular slots around the entire circumference. These stents require balloon expansion to deploy them in the airway and expand the stent to its functional diameter. Once expanded, the Palmaz stent does not exert a continual expanding pressure on the airway. There is no covered version of the Palmaz stent, and after tissue ingrowth occurs, removal is very difficult.

The Strecker stent consists of a single tantalum filament interwoven into a cylindrical wire mesh. These stents are very flexible and do not shorten longitudinally when dilated, making placement easier, particularly in short segment stenoses. Like the Palmaz stent, Strecker stents require balloon expansion during deployment to produce the desired stent diameter and they do not produce the intrinsic radial force of self-expandable stents.

Indications

The large variety of potential indications for endobronchial stent placement are listed in Table 40-2. The majority of patients requiring tracheobronchial stents have malignant disease, most commonly bronchogenic carcinoma. A patient with an unresectable lung cancer and endobronchial tumor producing central airway obstruction may receive rapid and satisfying palliation by laser or core-out of the endobronchial disease. However, this requires patent distal airways and is not feasible when a tumor also produces obstruction at the

Table 40-2 Etiology of Obstructive Airway Lesions

Malignant	*Benign*
Primary airway tumor	Postintubation
Squamous cell	Cuff stenosis
Adenoid cystic	Stomal stenosis
Carcinoid	Idiopathic
Mucoepidermoid	Anastomotic
Miscellaneous	Lung transplantation
Lung cancer	Sleeve resection/bronchoplasty
Endobronchial tumor	Inflammatory
Extrinsic compression	Tuberculosis
Thyroid cancer	Histoplasmosis
Head and neck cancer	Wegener's granulomatosis
Esophageal cancer	Bacterial or fungal tracheitis
Airway obstruction	Trauma
Tracheoesophageal fistula	Tracheobronchial malacia
Metastases	Vascular compression
Renal cell carcinoma	Postpneumonectomy syndrome
Breast cancer	Aortic aneurysm
Colon cancer	Pulmonary artery dilatation
	Miscellaneous
	Tracheopathia osteoplastica
	Tracheobronchomegaly
	Relapsing polychondritis
	Compression by esophageal stent

lobar and/or segmental levels. Proximal endobronchial tumor without distal lobar obstruction is the situation most amenable to airway palliation (Table 40-3). In these cases, airway stenting may prolong the period of palliation achieved by mechanical or laser reestablishment of the airway (Figure 40-2, [Color Plate 10]). In this setting, stenting may be reserved to palliate rapidly recurrent areas of an endobronchial tumor.

Patients with locally advanced lung cancer may also exhibit significant extrinsic compression of the airway, with or without concomitant endoluminal tumor. These patients may require urgent stenting to stabilize the airway while the primary tumor and bulky mediastinal lymph nodes are treated with systemic therapy or radiation. However, many times, these patients have already undergone definitive treatment and have recurrent or persistent airway compromise with impending suffocation. In these patients, airway stenting is the only treatment for the extrinsic airway compression, and can produce a gratifying improvement in quality of life and avoidance of death from airway obstruction (Figure 40-3 [Color Plate 10]).

Primary airway tumors such as squamous cell carcinoma, adenoid cystic carcinoma, and carcinoid tumors are most commonly treated with definitive surgical resection unless the length or extent of tumor involvement precludes operability. Radiation, with or without chemotherapy, is the usual second-line therapy for patients who are not candidates for surgical resection. However, patients with endobronchial obstruction are candidates for stent placement, both for stabilization of the airway during treatment, or for persistent airway stenosis after definitive therapy.

Adjacent primary tumors may produce airway obstruction by direct airway invasion or extrinsic compression (Figures 40-4*A*, 40-5, [Figures 40-4*B* and 40-5*C* through *E*, see Color Plates 10, 11]). Esophageal cancer, head and neck malignancies, and thyroid cancer may all result in tracheal obstruction that can be palliated by stent placement. Esophageal cancer may also result in a malignant tracheoesophageal fistula. These patients commonly have a short life expectancy, but the contamination of the airway by salivary and gastric contents may be minimized by a covered esophageal stent or covered tracheal stent, or both. Expandable endoesophageal stents have produced a marked improvement in the palliation of patients with malignant esophageal obstruction. However, when these stents are placed in the upper esophagus, they may result in a secondary extrinsic compression of the trachea or mainstem bronchi. If the esophageal stent cannot be removed, then the airway can be palliated with a second stent in the airway to maintain airway patency.

Several primary tumors are known to metastasize occasionally to the airway or to the paratracheal or subcarinal lymph nodes with secondary airway involvement (Figures 40-6 [Color Plate 11], 40-7) The most common of these is renal cell carcinoma, followed by breast cancer and colon cancer metastases. If these lesions are isolated to the central airways, with patent lobar and segmental orifices, then these patients are also candidates for laser and/or endobronchial core-out as previously described for central lung cancer. In these cases, extrinsic compression or rapid recurrence of endobronchial tumor is a strong indication for endobronchial stenting.

The most common etiology of benign tracheal stenosis is a postintubation injury resulting in either a cuff stenosis or a stomal stenosis. The vast majority of these patients are best treated by a tracheal sleeve resection as a definitive therapy with reliable results.[3] Even patients with a high surgical risk because of medical comorbidities are usually best treated by definitive surgical correction rather than by bronchoscopic palliation or stenting, for several reasons. First of all, few benign stenoses are permanently corrected by dilatation, laser resection, or stenting. Second, tracheal resection and reconstruction is usually only a

Table 40-3 Anatomic Criteria for Airway Stenting

Distal to cricoid
Proximal to lobar orifice
Patent lobar/segmental orifices

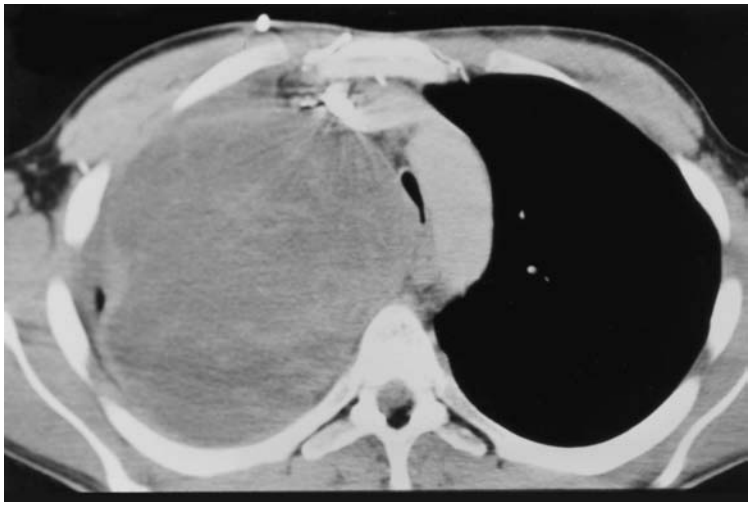


FIGURE **40-4** A, *Tracheal compression from recurrent metastatic sarcoma producing marked tracheal compression and critical airway stenosis.*

neck operation, with a relatively trivial physiologic insult that is well tolerated by most patients. Third, patients with significant co-morbidities may not tolerate the often-repeated interventions that are necessary with palliative techniques, including stenting. However, some patients may refuse surgery and others may have a very long segment stenosis that is not amenable for surgical reconstruction. Other patients may benefit from temporary endoluminal stenting while maximizing conditions for a planned surgical correction. Rarely, patients with an early postintubation stenosis may remodel the airway over an indwelling tracheal stent that can ultimately be removed with a narrowed but adequate and stable airway.

Patients with traumatic airway disruption are not candidates for early stent placement and should have their tracheobronchial laceration corrected surgically. Likewise, patients with a benign stricture from trauma are best corrected with resection and primary reconstruction, as for patients with postintubation stenosis. However, these patients may also be candidates for stenting if the stricture is uncorrectable or if a stent is being used as temporary palliation in preparation for surgery.

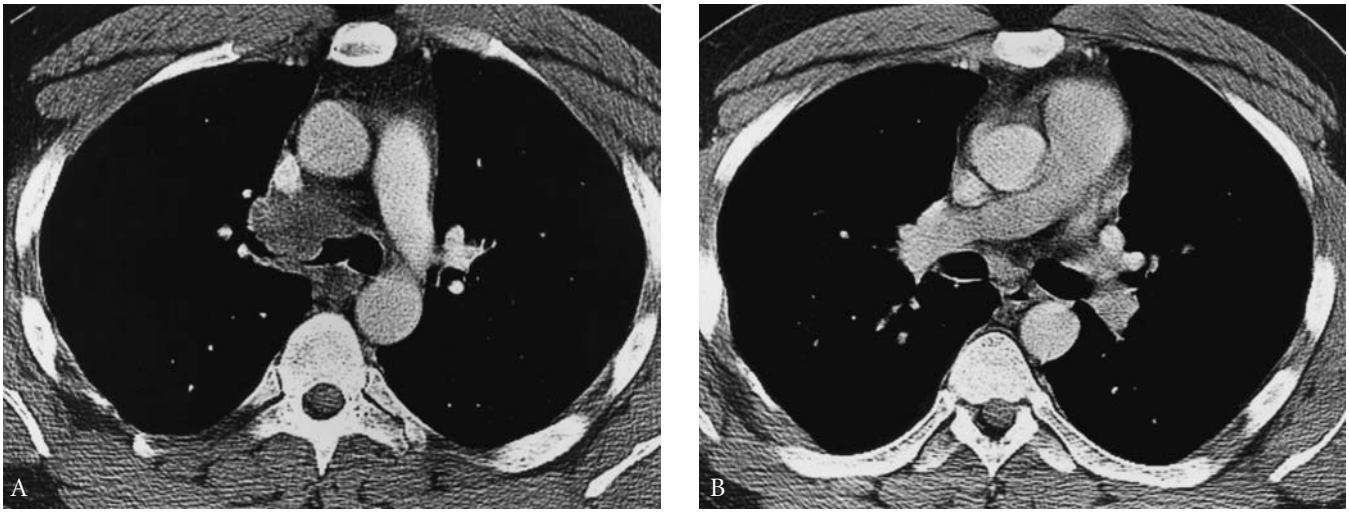


FIGURE **40-5** A, *Computed tomography (CT) scan showing a right mainstem bronchial tumor and compression from a metastatic sarcoma.* B, *CT scan showing a clear bronchus intermedius, confirming a patent distal airway favoring successful stenting.*

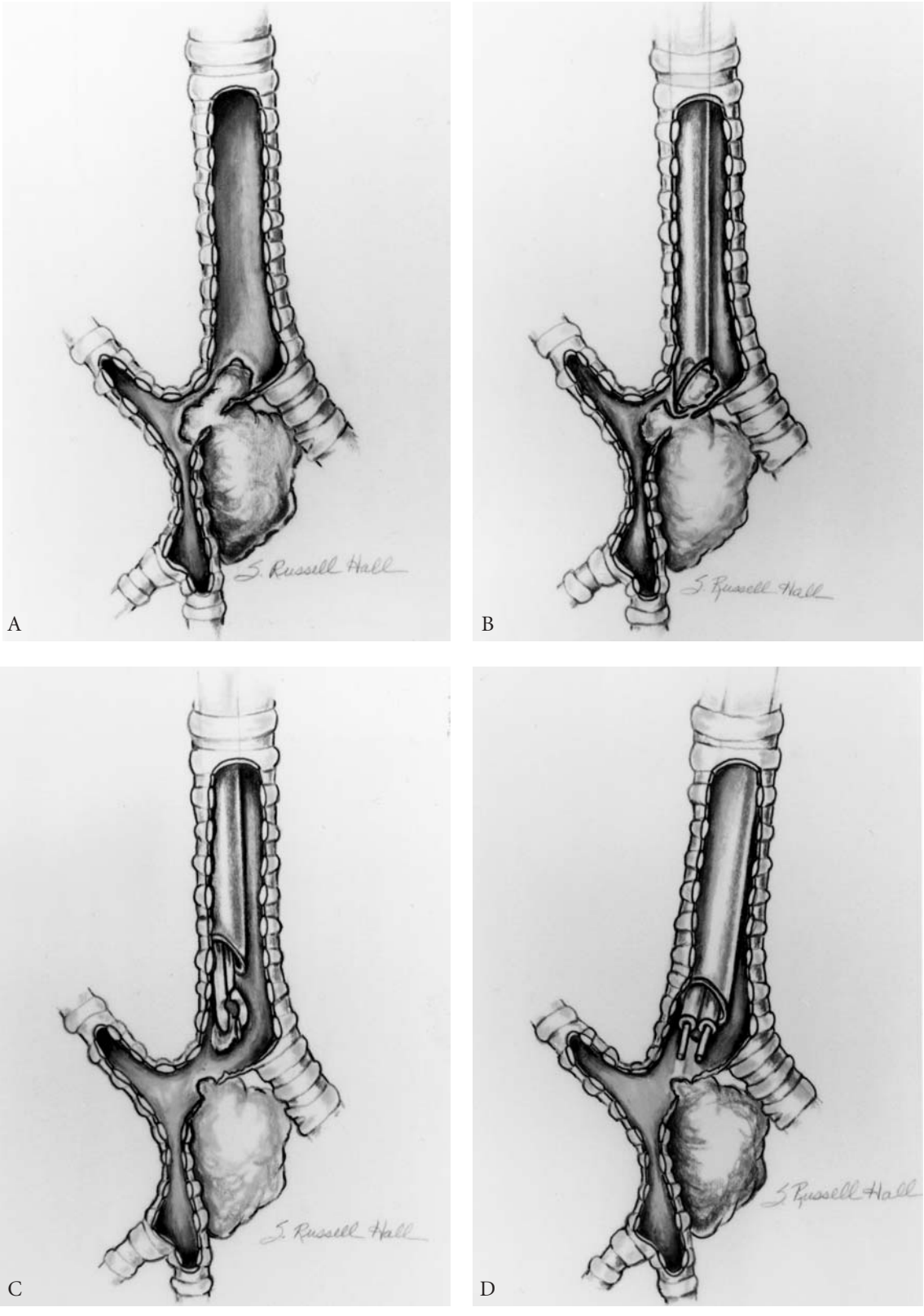
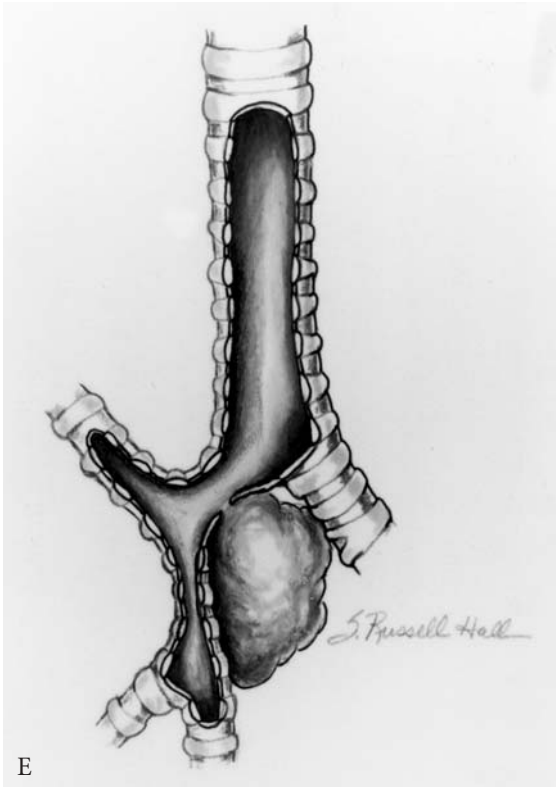
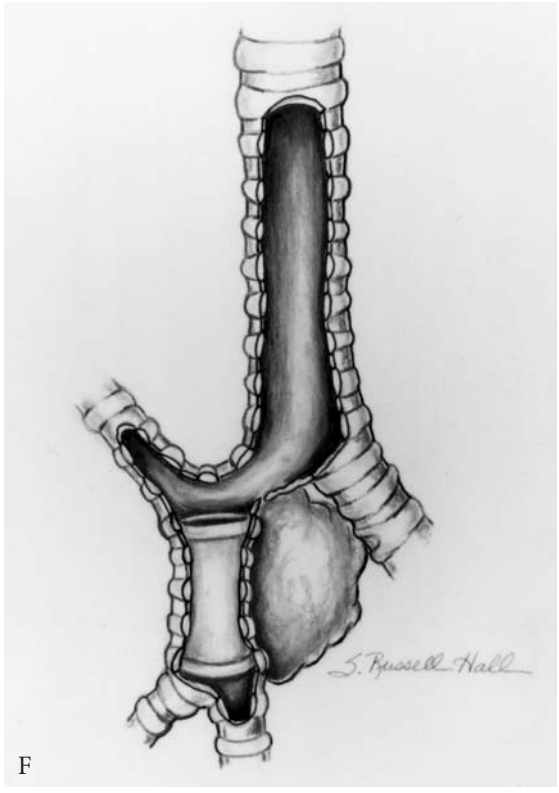


FIGURE **40-7** A, *Metastatic renal cell carcinoma to subcarinal lymph nodes with both endoluminal and extrinsic obstruction of the right lung.* B, *Mechanical core-out of tumor with the tip of the rigid bronchoscope.* C, *Further débridement of the endoluminal tumor with biopsy forceps.* D, *Hemostasis and vaporization of the tumor bed with an Nd:YAG laser.*



E



F

G

FIGURE **40-7** (CONTINUED) E, *Residual extrinsic compression of the bronchus intermedius after resection of the endoluminal tumor.* F, *Silicone stent with flanges seated in the bronchus intermedius.* G, *Separate silicone stent with external studs seated in the mainstem bronchus. Note the medial intussusception and lateral separation to allow ventilation of the right upper lobe. This can be augmented by cutting the distal portion of the right mainstem stent at an angle to prevent obstruction of the right upper lobe orifice.*

Idiopathic tracheal stenosis most commonly involves the very proximal trachea, often extending into the subglottic larynx. These patients may be palliated with periodic dilatation or definitively corrected with tracheal resection or reconstruction. Because of the anatomic location of these stenoses, idiopathic tracheal stenosis is generally not a good indication for airway stenting. This is because of the significant difficulty in seating a stent proximal to the cricoid into the subglottic larynx, as well as a significant risk of producing subglottic granulations that may actually extend and further complicate the original stenosis. If these patients do require airway stenting, then this is probably best accomplished using external stabilization with a tracheal T tube, as discussed in Chapter 39, "Tracheal T Tubes."

Anastomotic stenosis after lung transplantation, sleeve resections, and bronchoplastic procedures now provide a common indication for endobronchial stenting. Anastomotic complications occur in 4 to 15% of lung transplant anastomoses.[25] Anastomotic stenoses occur due to technical complications, dehiscence with subsequent granulation and cicatricial scarring, and ischemia. Ischemia can also occur distal to the anastomosis and can cause ischemic stricture of the bronchus intermedius after right lung transplantation. Other patients may develop secondary bronchomalacia due to ischemia, airway distortion, or steroids. Most of these patients are not candidates for direct surgical reconstruction or anastomotic revision and so provide a good indication for endobronchial stenting.

Anastomotic stricture after tracheal resection or bronchial sleeve resection is uncommon, occurring in approximately 5% of primary airway resections.[3,26] Many of these patients may be candidates for reoperative surgical resection and reconstruction, but most of these patients benefit from a period of airway stenting to allow maturing of the fibrous scar to facilitate reoperative surgery.[27] Many of these patients may no longer be surgical candidates for a variety of reasons, and provide further indications for prolonged endobronchial stenting.

Inflammatory or infectious conditions producing benign airway stenosis are infrequently appropriate for definitive surgical correction. The exceptions are the occasional patient with a short segment residual tracheal or bronchial stenosis after tuberculosis or a fungal or bacterial tracheitis, or from Wegener's granulomatosis in remission. The majority of these lesions, however, are best treated by dilation and endobronchial stenting, due to the extent of disease and the natural history of the underlying etiology. This is also true of a variety of miscellaneous causes of airway obstruction such as a relapsing polychondritis and tracheobronchomegaly.

Vascular compression of an adjacent airway may occur in the setting of post-pneumonectomy syndrome, aortic aneurysm, or marked pulmonary artery dilatation. In each of these cases, every attempt should be made to correct the underlying problem rather than using an airway stent simply to palliate the secondary consequences of an airway compression. Many would consider vascular compression of the airway to be a relative contraindication to stenting due to the concern of stent erosion and a fatal bronchovascular fistula.

Finally, tracheobronchial malacia may result in functional airway obstruction in the absence of a fixed stenosis. Patients may have true loss of cartilaginous support or a functional malacia, with anterior–posterior collapse in patients with severe chronic obstructive pulmonary disease. This provides another indication for stenting, which may result in excellent palliation, although these patients have a difficult problem with stent seating due to the lack of a malignant or benign stricture.

In general, patients are candidates for endobronchial stenting when they have malignant or benign obstruction that is not amenable to definitive surgical correction (Table 40-4). Temporary stenting may also be useful to stabilize the airway during initiation or preparation for definitive therapy. It is important that the ability to place an airway stent does *not* become its primary indication. A large number of patients are best treated by definitive surgery, and many other patients benefit from other endobronchial therapeutics that provide reliable results without the disadvantages of an indwelling prosthesis.[1]

Table 40-4 Criteria for Airway Stenting

Surgically Correctable Lesions	*Surgically Uncorrectable Lesions*
Patient refuses surgery	Extrinsic compression
Short life-expectancy	Recurrent stricture (after dilation)
Planned delay of surgical intervention	Recurrent endoluminal tumor
High surgical risk	Stabilization during chemoradiation
Trial of airway remodelling	Malacia

Clinical Presentation

Besides the symptoms of their primary disease, patients with significant large airway obstruction are likely to present with dyspnea, wheezing or stridor, cough, and hemoptysis. Patients may also have postobstructive pneumonia or recurrent infections. Not infrequently, stridor may be misinterpreted as bronchospastic disease and patients may be treated with increasing doses of bronchodilators and steroids before the diagnosis of central airway stenosis or tumor is considered. Patients with critical but compensated airway stenosis may present in extremis from a relatively minor exacerbation of their stenosis, due to infection, secretions, or mucosal edema. Patients with known malignancies may attribute their symptoms to progression of their tumor. However, increasing dyspnea or stridor may actually be caused by endobronchial tumor or extrinsic airway compression, and should be considered when symptoms dictate.

A physical examination often reveals upper airway stridor or decreased breath sounds over the lung fields. Coarse rhonchi may be present due to difficulty in clearance of secretions. Absence of breath sounds and consolidation may be present in areas of more distal obstruction with pneumonia or mucous impaction.

Chest radiographs are useful if they show a tracheobronchial mass or distortion of the tracheobronchial air column. They may also show pneumonia, consolidation, or provide evidence of a lung or other primary malignancy. A high kilovoltage anterior–posterior chest x-ray and linear tomograms often provide the best definition of the longitudinal extent of airway involvement. However, in most facilities, a neck and chest computed tomography (CT) scan is more readily available. With today's generation of CT scanners, a fine-cut CT through the airway, with or without three dimensional (3-D) reconstruction or coronal or sagittal views, provides excellent detail of airway anatomy. For benign lesions, this helps predict the location and extent of stenosis prior to endoscopy. For malignant lesions, this helps to identify the 3-D extent of tumor for planning the potential therapeutic options and anticipating their potential hazards and chances for success.

Pulmonary function testing and flow volume loops may occasionally be helpful, but the sensitivity is low in the absence of significant tracheal obstruction.[24] Pulmonary function tests provide little help in assessing symptomatic airway obstruction as long as the diagnosis has been considered. The primary benefit may be in providing an objective measure of improved airflow after tracheal bronchial stenting. Independent of pulmonary function values or flow volume loops, the patient with symptoms of upper airway obstruction should undergo bronchoscopic evaluation.

Bronchoscopic Evaluation

Bronchoscopy is essential in the evaluation of the patient with central airway obstruction who may be a candidate for airway stenting. Bronchoscopy is also useful for delivery and adjustment of an endoluminal stent and is preferred for refining the accuracy of stent delivery by a majority of pulmonary physicians and thoracic surgeons. Although stents may also be placed under fluoroscopic guidance without bronchoscopy, initial evaluation of the airway by bronchoscopy is critical to assess the nature of the obstructing lesion and the appropriateness for airway stenting. Bronchoscopy should be performed on any patient with symptoms

of central airway obstruction combined with a history of prolonged mechanical ventilation or tracheostomy. Other patients with a history or findings suggestive of a potential tracheobronchial stenosis should also prompt bronchoscopic inspection, since imaging studies are not yet sensitive or specific enough to replace direct visualization of the airways.

Bronchoscopy performs five critical functions in the evaluation and treatment of patients with symptomatic central airway obstruction: 1) definition of the existence and pathology of the airway abnormality; 2) temporary stabilization of the critically narrowed airway; 3) definition of the extent, severity, and complexity of the stenosis; 4) assessment of the treatment modalities that may be successful given the pathology and anatomy; and 5) direct therapeutic bronchoscopic intervention for temporary or long-term airway palliation.

When the pathology of a tracheobronchial stricture is unknown, biopsies should be obtained to establish a diagnosis. Establishment of a stable airway and adequate access for secure ventilation is the top priority and should take place simultaneously with the initial bronchoscopic assessment. A careful map of the airway anatomy should be created that defines the extent of the lesion and its relation to the normal airway anatomy; that is, directly measuring the distance of the stenosis from the vocal cords, cricoid cartilage, carina, and if necessary, the length of the mainstem bronchi and bronchus intermedius. The anatomy should be clearly documented along with the associated findings of granulation tissue, mucosal inflammation, or loss of cartilaginous support. This assessment should document the length and severity of stenosis as well as the degree of stenosis due to endoluminal disease versus fibrotic scar or extrinsic compression. The initial bronchoscopy should also evaluate the remainder of the airway for signs of postobstructive inspissated secretions, inflammation, or infection.

When the initial bronchoscopic assessment has defined the pathology and anatomy, it is easier to choose the optimal therapeutic approach. Patients with central T3-4 N0-1 nonsmall cell lung cancer are usually best treated by an extended surgical resection with a sleeve lobectomy or sleeve pneumonectomy, as discussed earlier in this text. Patients with benign strictures of the trachea or patients with tracheal tumors that involve less than half the length of the trachea may usually be definitively treated with primary tracheal resection and reconstruction. Tumor or granulations can be debulked by mechanical core-out with the tip of the bronchoscope or biopsy forceps. In some cases, this may be augmented by laser vaporization. Benign or malignant strictures can be dilated with esophageal bougies, with serially sized rigid bronchoscopes, or with hydrostatic balloon dilatation. Patients with significant extrinsic compression or malacia, however, do not have other directly applicable strategies other than airway stenting. Stenting also provides an adjunct to débridement of an intraluminal lesion if the initial therapy fails or has the appearance of likely early failure (see Figure 40-7). Similarly, stenting provides a useful adjunct to the dilation of a benign stricture to maintain patency, with less need for repeated dilations.

The endoscopic techniques for airway palliation are not mutually exclusive. Each of these modalities, including definitive surgical correction, should be available and considered by the physician evaluating a patient with symptomatic central airway obstruction. The treatment can then be tailored to fit the pathology and anatomy, with treatments combined to achieve the optimal patient outcome.[1] An algorithm for the application of therapeutic bronchoscopy to central airway obstruction, including stenting, is provided in Figure 40-8.

Many authors have come to favor either flexible bronchoscopy or rigid bronchoscopy for their therapeutic interventions. A number of pulmonary physicians and thoracic surgeons alike have abandoned rigid bronchoscopy in favor of the relative ease of flexible bronchoscopy. However, in patients with central airway obstruction and complex airway anatomy, rigid bronchoscopy provides a spectrum of airway interventions that are not easily duplicated by flexible bronchoscopy. Although rigid bronchoscopy has the disadvantage of requiring a general anesthetic, it has the significant advantage of providing ventilation concurrent with air-

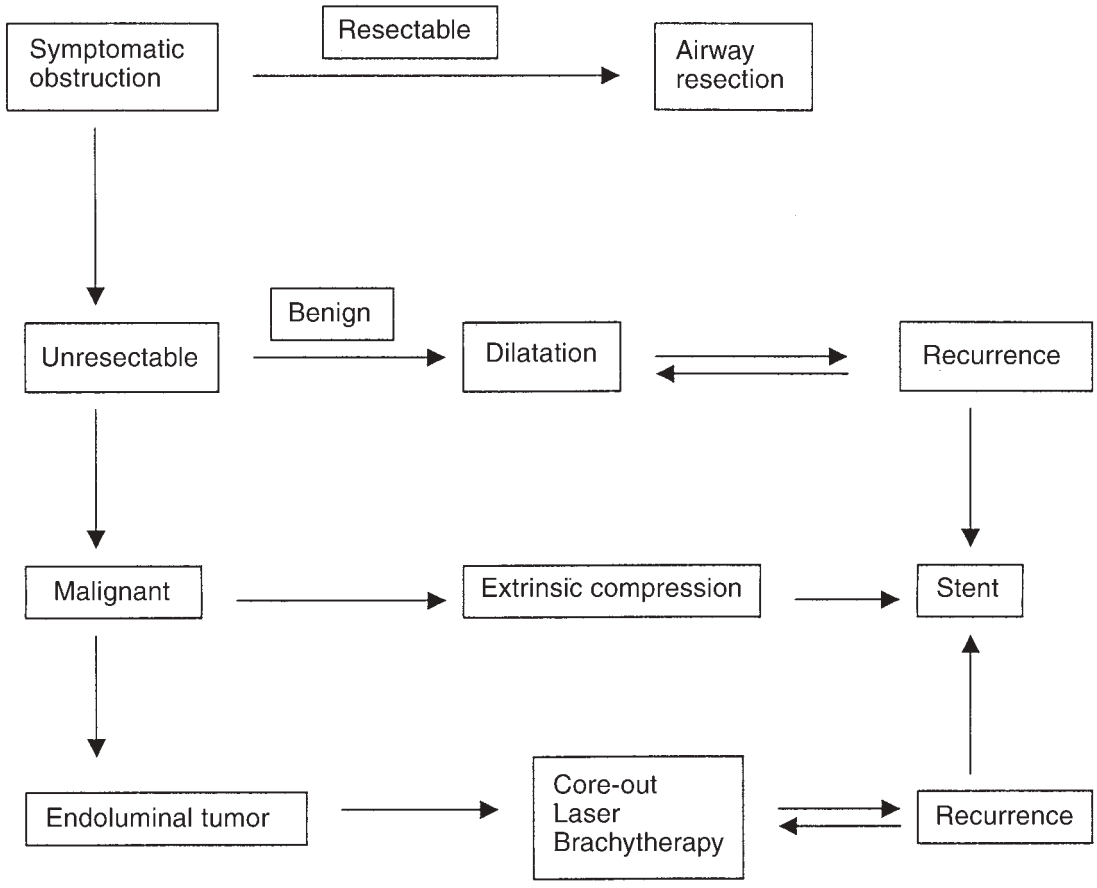


FIGURE **40-8** *Algorithm for management of central airway obstruction*

way assessment, and allowing the endoscopist to directly secure airway control distal to a critical stenosis. Rigid bronchoscopy has the advantage of larger instrumentation to facilitate mechanical débridement of endoluminal tissue and aspiration of secretions and blood.

The availability of rigid bronchoscopy is especially important for airway stenting since rigid bronchoscopy allows delivery of silicone or expandable stents. This allows the choice of the best stent for the patient and anatomy rather than a stent chosen because of the inability to deliver a nonexpandable stent. Furthermore, it is much easier to manipulate, adjust, and even remove stents by rigid bronchoscopy. Although expandable stents are typically considered permanent, most of these can be removed, albeit with significant difficulty, by rigid bronchoscopy, even after the development of tissue ingrowth. Flexible bronchoscopy provides little ability to adjust expandable stents once they have become firmly seated within the airway.

Flexible bronchoscopy has the advantage of allowing excellent airway assessment in an endoscopy suite without general anesthesia. Flexible bronchoscopy also allows an easier assessment of the lobar and segmental orifices than rigid bronchoscopy does. The increasingly sophisticated instrumentation available for the flexible bronchoscope allows dilation of strictures, retrieval of foreign bodies, and manipulation of airway prostheses. However, this is a far more limited repertoire than that available to the endoscopist skilled in rigid bronchoscopy. Flexible bronchoscopy has the added disadvantage of exacerbating the obstruction by the diameter of the bronchoscope, although this may be minimized by use of pediatric bronchoscopes. This may not allow distal airway evaluation in critical tracheal stenosis until after dilatation.

The complete interventional bronchoscopist should be skilled and adept in both rigid and flexible bronchoscopies. The bronchoscopist should have a broad spectrum of instrumentation and a team that is familiar and comfortable with both approaches to the airway. Although the skills and procedures for therapeutic bronchoscopy are not particularly difficult, application of these skills is challenging in the patient with a severely compromised airway, even for physicians with significant experience in airway therapeutics. Because of this, maintenance of skill in rigid bronchoscopy is critical so that this expertise can be readily available when a patient presents with a complicated and threatened airway.

Choice of Stent

Neither silicone nor the available metal stents conform to all the ideal characteristics for an endobronchial stent. Although each has its advocates, both of these general types of stents have advantages and disadvantages that should be considered when choosing the best stent for the individual patient (Table 40-5). There is no one stent that ideally fits all circumstances, and the full variety of silicone and expandable stents should be considered to maximize positive outcomes. Each category of stent now has significant clinical experience on which to base expectations of outcomes.

The primary advantage of the silicone stent is that it can be repositioned as many times as needed and removed easily. The molded silicone rubber has very little tissue reactivity, with minimal granulations. Because it is a solid stent, there is no tumor ingrowth or granulations, although each may occur at the ends of the stent. Silicone stents are inexpensive and can be easily modified by cutting a portion of the stent to allow customization to the applicable airway anatomy. Finally, silicone stents have a defined diameter that prevents uncontrolled expansion.

There are three major criticisms of silicone stents that prevent their universal application. The most common criticism, and the least important, is the need for rigid bronchoscopy and general anesthesia with more difficult initial delivery than the expandable stents. This consideration alone should not prevent their use in the appropriate clinical setting. A legitimate criticism of silicone stents is the reduced inner diameter due to the thicker wall necessary to provide adequate rigidity. This, combined with a loss of mucociliary clearance, may lead to inspissated secretions within the stent, interfering with its function. Another criticism of silicone stents is their potential for dislodgment or distortion. Because of the rigid nature of these stents, if they are inappropriately sized or not well seated within an area of stenosis, they may migrate, producing the complication of distal or lobar segmental obstruction or proximal airway obstruction.

Expandable metal stents overcome some of the disadvantages of the silicone stents. Perhaps the most profound, but least important, characteristic is the ease of delivery of expandable stents, using flexible bronchoscopy with fluoroscopy under topical anesthesia. Once seated, these stents are extremely stable with

Table 40-5 Relative Advantages and Disadvantages of Silicone versus Expandable Metal Stents

Silicone		*Metal*	
Advantages	*Disadvantages*	*Advantages*	*Disadvantages*
Adjustable	Rigid bronchoscopy	Flexible bronchoscopy	Permanent
Removable	Difficult placement	Easy delivery	Difficult adjustment
No ingrowth	Dislodgement	Stable placement	Needs fluoroscopy
Unreactive	Decreased inner diameter	Conformation	Granulations
Controlled expansion	Distortion	Epithelialization	Tumor ingrowth
		Ventilation through interstices	Erosion
Inexpensive			Expensive

minimal complications of stent migration. The second-generation stents (Wallstent and Ultraflex stent) also conform well to distortions or curves within the airway anatomy. Uncovered stents have the theoretical benefit of neoepithelialization with incorporation of the stent into the airway wall and resumption of mucociliary clearance. These stents may also allow ventilation through the interstices in cases where a lobar or segmental orifice is partially covered. Obviously, these characteristics of neoepithelialization and ventilation through the stent are lost in expandable stents that are coated with silicone rubber or polyurethane.

Unfortunately, the disadvantages of the expandable stents are significant. The most serious disadvantage is that these stents are permanent and, once seated, are nearly impossible to reposition or remove. This may be especially troubling when tumor ingrowth or granulation tissue produces recurrent obstruction inside the stent, requiring repeated débridement or a repeat stenting within the preexisting stent (Figure 40-9 [Color Plate 11]). Both the silicone-coated Wallstent and Ultraflex stent cause much less difficulty with tumor and granulation ingrowth. These stents are also easier to remove if necessary, due to their lack of incorporation within the airway wall. However, both of these prostheses still employ bare metal portions at each end of the stent to allow stable seating. These areas still limit the ability to remove the stent and can be complicated by growth of tissue through the interstices.

The significant radial force exerted by expandable stents creates the potential for erosion into surrounding structures with possible life-threatening bronchovascular fistula. Series reporting the use of the Gianturco stent have shown a significant incidence of strut fracture with occasional fatal vascular erosion.[28] Finally, expandable stents are markedly more expensive than silicone stents, but this device expense may be offset by the costs of the operating room and anesthesia for delivery of silicone stents.

For patients with advanced malignancy and airway obstruction, both silicone stents and covered expandable stents may offer reliable palliation. In this setting, the optimum choice of stent may be determined by the anatomy of the lesion and the experience and preference of the pulmonary physician or thoracic surgeon. It is in patients with benign disease where more debate exists. Some have pointed to the similar rate of complications reported for both silicone stents and expandable stents as a rationale to support the use of expandable stents in benign disease.[28] However, others feel that the severity and irreversibility of the complications from expandable stents are a strong relative contraindication to their use in benign disease.[1,29,30] The permanence of the expandable stents, the inability to reposition them, the possibility of extension of airway injury by stent-related granulations, and concern about potential erosion of a long-standing stent, all discourage consideration of these stents for benign disease. However, there are some settings where the anatomy or position of the lesion may make adequate stenting with silicone stents impossible, and expandable stents may provide the only remedy. Patients with malacia may require an expandable stent due to the difficulty in seating a silastic stent in the absence of a fixed stenosis. However, in this setting, other techniques for stent fixation, such as placement of a carinal Y stent or a tracheal T tube, may still provide a better alternative than an expandable stent in these benign conditions.

Stent Placement

Anesthesia

Patients undergoing flexible bronchoscopy can routinely be managed with topical anesthesia, with or without intravenous sedation. However, even when planning flexible bronchoscopy, careful consideration should be made regarding the degree of airway compromise, and the potential benefit of the equipment and anesthesia back-up available in the operating room. Extreme caution should be exercised in initiating endoscopy with minimal support in the endoscopy unit, when one is evaluating a patient with potentially critical central airway obstruction. In this setting, flexible bronchoscopy with the immediate availability of general anesthesia and rigid bronchoscopy provides the safest options for establishment of airway control.

Patients undergoing rigid bronchoscopy require a general anesthetic. Careful preoperative planning and intraoperative communication between the surgeon and anesthesiologist is essential for safe management of the airway. The endoscopist should be present at the bedside for the entire induction sequence, and prepared for emergency airway control with a rigid bronchoscope. The safest anesthetic induction maintains spontaneous ventilation using a combination of inhaled or short-acting intravenous agents until a secure airway can be established.[31] Once the obstruction has been assessed and airway control achieved with a ventilating rigid bronchoscope, then muscle paralysis can be added if necessary.

The vast majority of patients can be managed with routine ventilation through the ventilating side limb of a rigid bronchoscope. Occasional catheter jet ventilation can also be used but this generally adds unnecessary complexity without added benefit over standard ventilation techniques. Jet ventilation distal to a fixed obstruction is relatively contraindicated since it may lead to distal barotrauma. In order to facilitate adequate ventilation in some cases, the endoscopist may need to negotiate the stenosis and direct the ventilating bronchoscope into a mainstem bronchus. This is facilitated by the immediate availability of a smaller rigid bronchoscope that can traverse the stenosis. With a closed telescope-guided system, it is possible to maintain ventilation even during delivery and positioning of the endoluminal stent. Temporary cessation of ventilation, however, may be useful to minimize aerosolization of blood or secretions that may obscure the endoscopic field. An experienced and dedicated team of nurses and anesthesiologists familiar with the problems and equipment used for endoscopic airway management greatly facilitates these procedures, particularly during the critical moments of airway control or stent deployment.

Bronchoscopic Equipment and Preparation for Stenting

Although flexible bronchoscopic assessment can be performed even with small pediatric bronchoscopes, therapeutic intervention usually requires an adult bronchoscope with a large working channel (2.8 to 3.2 mm). This is necessary to allow adequate suction and delivery of therapeutic instrumentation such as laser catheters, balloon dilators, and some of the smaller stent delivery devices. For expandable stents to be placed under fluoroscopic guidance, the initial steps of bronchoscopy serve to define the proximal and distal extent of the stenosis and these can be marked with external radiopaque markers. Laser resection of endoluminal tumor or balloon dilation of a stricture prepares the airway for maximal stent deployment.

Rigid bronchoscopy serves the dual function of preparation of the airway for stent delivery as well as direct stent deployment. Endobronchial tumor can be cored out with the tip of the rigid bronchoscope and extrinsic compression or stenosis can be dilated with either rigid bronchoscopes or esophageal bougies.[1] For airway dilation, Jackson bronchoscopes (Pilling-Weck, Research Triangle, NC) have the advantage of blunt and rounded tips, making it easier to dilate the airway with less risk of perforation or mucosal laceration. These bronchoscopes come in a variety of sizes (3.5, 4, 5, 6, 7, 8, and 9 mm) and serial dilatation can establish an adequate airway lumen while maintaining ventilation through the bronchoscope. If the lesion is too narrow to accept the 3.5 mm Jackson bronchoscope, then esophageal bougies ("Jackson" flexible, Karl Storz Endoscopy America, Culver City, CA) can be used to enlarge the airway enough to allow bronchoscopic dilatation. Hydrostatic balloon dilatation can also be performed through the rigid bronchoscope to produce an even larger diameter airway before stent deployment.

The final stage of bronchoscopic preparation is the assessment of the style and size of stent(s) to be deployed. At the University of Washington, we prefer silicone stents for the majority of both benign and malignant lesions, due to the ease of repositioning and removal, and the low rate of serious (permanent) complications. The length is determined by direct measurement. The diameter is estimated using the diameter of the rigid bronchoscope as a benchmark. The potential need for multiple stents and the degree of external compression is also assessed. For patients in whom satisfactory stent seating cannot be obtained by silicone stents, or in whom marked external compression produces distortion of a silicone stent, an

expandable metal stent may provide a better result. The Hood Corporation (Pembroke, MA) has recently produced silicone stents with greater or lesser rigidity to improve the results when these stents are positioned in an area of firm external compression.

A variety of techniques have been described for the deployment of stents through the rigid bronchoscope. Expandable stents have different delivery systems consisting of constrainment within a sheath (Wallstent, Ultraflex stent) or expansion over a balloon (Palmaz stent, Strecker stent). The same technique of fluoroscopic guidance with external skin markers is performed as during flexible bronchoscopy. It is also possible to simultaneously view the deployment through the magnified view of the bronchoscopic telescope to improve the accuracy of stent delivery. One can then revise the position of the proximal and distal ends of the stent endoscopically, and the Wallstent or Ultraflex stent can be more completely expanded and seated by dilatation with an appropriately sized intraluminal angioplasty balloon.

Silicone stents are more difficult to deploy and four strategies have been described for the accurate placement of these stents by rigid bronchoscopy. One technique places the stent on the outside of an appropriately sized rigid bronchoscope with an endotracheal tube inserted as a sheath over the proximal portion of the bronchoscope. The patient is then intubated with the rigid bronchoscope and the overlying stent and "pusher tube." This "pusher tube" or sheath prevents the stent from riding up on the bronchoscope as it is inserted. After the tip of the bronchoscope is placed through the stenosis, the bronchoscope is gradually rotated and withdrawn, but with the external sheath held in place, thus pushing the stent off the tip of the bronchoscope at the desired location. Grasping forceps can then be used to modify and adjust the final stent position.[15] The problems associated with this technique are due to the bulk of the bronchoscope–stent–pusher tube apparatus, both in passing through the vocal cords and in passing through the area of stenosis.

The second technique, popularized by Dumon, uses a specialized stent delivery system, the Dumon-Harrell Universal bronchoscope (EFER, La Ciotat, France, and Bryan Corporation, Woburn, MA).[16] This system features changeable tubes of various sizes that can be used to calibrate and dilate the stenosis. Since these tubes telescope over each other, they can also be used as introducers for prostheses of all sizes. Once the stenosis has been dilated and a stent chosen, the stent is collapsed into the distal end of the delivery tube, which is passed through the bronchoscope to the site of stenosis. A plunger system is then used for pushing the stent out of the introducer. Again, the fine-tuning of stent positioning is performed with grasping forceps under direct visualization.

The third technique delivers the endoluminal stent through the lumen of the rigid bronchoscope without any specialized equipment.[1,32] Rigid bronchoscopy is performed using a Storz bronchoscope ("Shapshay" laser bronchotracheoscope, Karl Storz Endoscopy America, Inc., Culver City, CA). The Storz bronchoscope has no internal light carrier and a smooth internal lumen, allowing placement of 14 mm or smaller silicone stents through the lumen of the bronchoscope. This is facilitated by lubricating the stent with a silicone lubricant. The stent can then be pushed through the rigid bronchoscope with a standard grasping forceps, and directly positioned. This has the benefit of requiring fewer manipulations, with less airway trauma and without the need for specialized equipment.

The fourth technique involves direct laryngoscopy to place the stent through the vocal cords into the proximal airway using grasping forceps. Rigid bronchoscopy is then performed with the bronchoscope, and graspers used to position the stent into the appropriate position. Although this can be performed with any size of stent, this technique is most useful for stents larger than 14 mm in diameter or carinal Y stents that will not fit through the Storz bronchoscope in the technique described above. This technique has the advantage of allowing larger stents to be placed through the vocal cords with minimal trauma. Since the stents are flexible, they are easily inserted through the glottis into the proximal airway, with rigid bronchoscopy then guiding the appropriate placement.

Carinal Y stents produce a unique set of problems in stent delivery and seating. Cooper and colleagues have placed these bifurcation stents over a rigid bronchoscope placed through the left mainstem limb, with a Fogarty catheter placed through the right mainstem limb and into the right main bronchus to guide proper placement.[15] Acuff and colleagues described placing carinal stents through direct laryngoscopy as described above, and have attempted to place the stent all the way to the carina blindly with a release of the grasping forceps at the carina, allowing delivery into the respective mainstem bronchi.[33] A modification of the technique described by Acuff does not require accurate delivery during the initial placement. Rigid bronchoscopy through the tracheal limb of the Y stent can straighten and seat the mainstem bronchi into their appropriate locations. Sometimes, it is easiest to invert the shorter right mainstem limb "inside out" up into the tracheal limb. When the left mainstem limb is seated at the carina, the right mainstem limb can then be pushed out into the right mainstem using the grasping forceps or the tip of the telescope.

Careful endoscopic evaluation of the stent position is important prior to completion of the procedure. Care should be taken to prevent obstruction of adjacent orifices. Placement of a cylinder stent in the distal trachea can potentially result in partial obstruction of a mainstem bronchus. Likewise, a mainstem bronchial stent extending above the carina may produce partial obstruction to the contralateral mainstem bronchus. A stent in the bronchus intermedius should avoid coverage of the middle lobe orifice or superior segment orifice distally, or the right upper lobe orifice proximally. The most difficult site for stenting is the right mainstem bronchus because of its short length. Here, a silicone stent can be modified by cutting it at an angle with a shorter lateral length than medial length. This then facilitates seating while maintaining patency to the right upper lobe orifice. This also requires attention to proper stent rotation at the time of positioning.

After expandable stent deployment, minor adjustments of the proximal or distal end can usually be made with grasping forceps through either the flexible or rigid bronchoscope. Inexact deployment with partial obstruction of a lobar or segmental orifice, or incomplete coverage of the tumor or stenosis, should not be accepted. In these cases, the stent should be repositioned endoscopically, or removed and a repeat placement attempted. This high degree of stent accuracy is necessary in order to achieve optimal palliation and minimal complications.

In some cases, it may be necessary to place multiple stents to achieve optimal palliation. Bilateral mainstem bronchial obstruction can be managed with a solitary carinal stent or bilateral bronchial stents if the stenosis does not extend into the distal trachea (see Figures 40-2, 40-3 [Color Plate 10]). Sometimes, subcarinal extrinsic compression or endobronchial tumor produces simultaneous obstruction of the right mainstem bronchus and bronchus intermedius but a patent right upper lobe orifice. Some have described placement of an expandable stent over the upper lobe orifice, allowing ventilation through the interstices of the stent.[28] However, two separate stents can be placed, even with solid silicone stents, to provide good airway palliation, as well as maintain unobstructed ventilation of the right upper lobe (see Figure 40-7). If the stent for the right mainstem bronchus is modified by cutting the distal end at an angle as described above, then the medial edge of the bronchus intermedius stent and right mainstem stent may intussuscept while providing a completely unobstructed orifice laterally into the right upper lobe.

The final endoscopic assessment should provide an estimate of the adequacy of airway palliation, the stability of the stent, and anticipate potential complications. Careful documentation of these factors facilitates the evaluation of outcomes and prepares the patient and endoscopist for the expectation of potential complications. The expandable stent should be firmly seated in nearly the full circumference of the airway wall in order to maximize the diameter and minimize complications of migration, granulations, and secretion retention. Silicone stents should be firmly seated in order to avoid dislodgment. However, a stent that is too big for the stenosis may buckle and result in partial airway obstruction by the stent itself. Granula-

tion tissue can develop at the proximal or distal end of stents, particularly in the bare metal portion of the coated expandable stents. This should be considered in the final stent position, if possible keeping these areas away from lobar and segmental orifices where granulation tissue may lead to recurrent obstruction.

Management after Stent Placement

Many authors recommend routine saline nebulization treatments starting immediately after stent placement in an effort to avoid any encrustations within the stent.[17,34,35] Other authors have recommended routine nebulized Mucomyst twice a day for the same reasons.[15] Some endoscopists routinely employ prophylactic antibiotic prophylaxis, and some employ inhaled or systemic steroids.[36,37]

There are no studies that have elucidated any post-stent medical interventions for minimizing stent complications. Stent obstruction with encrusted secretions is a potential complication, particularly in silicone stents. At the University of Washington, we have not routinely recommended any of these post-stent measures since most patients will have an uncomplicated course without the added inconvenience of a home nebulizer or Mucomyst treatments. It is advisable to have the patient stay well hydrated, in an effort to minimize the thickness of the secretions. Postobstructive infection should be treated with appropriately tailored antibiotics, with no need for routine antibiotic therapy due to the stent placement itself. Mucomyst and nebulizer treatments can be reserved for those patients who develop stent obstruction from inspissated secretions. There is no known use of post-stent steroids except for the potential treatment of glottic edema secondary to trauma during stent placement.

A variety of authors have recommended routine follow-up bronchoscopy in order to evaluate stent patency.[15,37] However, clinical symptoms provide the primary indication for follow-up bronchoscopy. Obviously, urgent bronchoscopy is indicated in the setting of acute respiratory decompensation, but it is also indicated if there is an unexplained worsening in the patient's respiratory status, or even an unanticipated lack of significant symptomatic improvement. These points are true at any time after stent placement, ranging from immediate post-placement in the post-anesthesia care unit, or years after stent placement after a prolonged period of palliation and stability. The purpose of endoscopy is to assure correct positioning and patency of the stent, evaluate possible extension of disease that may be amenable to further treatment or stent revision, or débride obstructing tumor or granulations within or at the ends of the stent. In the absence of stent abnormalities, other etiologies can be pursued for the progression of the patient's respiratory complaints, but stent malposition or obstruction should be definitively excluded by endoscopy in any patient with an indwelling stent and new respiratory complaints.

Stents that have become obstructed with inspissated secretions can usually be adequately cleaned in vivo with either the rigid or flexible bronchoscope. This may require physically débriding the hardened secretions and irrigating the thickened secretions. With silicone stents, it is also easy to remove these temporarily, cleaning them in a basin of hydrogen peroxide and rinsing with saline and simply reinserting the same stent. This usually takes a few minutes and is very easily accomplished once the stent position has been established by the original placement.

Granulations or progressive tumor may infiltrate through the interstices of a metal expandable stent or at the bare metal ends of a coated expandable stent. Both tumor and granulation tissue can be débrided mechanically and this can be easily augmented by a Nd:YAG laser. In some cases, this may be managed by placement of an additional stent within the original stent.[38]

Granulations obstructing the end of a silicone stent are uncommon and can also be débrided mechanically and with a laser. However, with silicone stents, the most likely etiology of proximal or distal granulations is uneven seating within the airway, with one lip of the stent excessively embedded within the bronchial mucosa. Therefore, a stent revision with a slightly different position, diameter, or length of Silastic stent may prevent recurrence of this problem.

Occasional surveillance bronchoscopy may be useful. In cases where stent seating is less secure, a follow-up endoscopy can confirm appropriate positioning or provide the opportunity for early stent revision. In some cases, the degree of airway inflammation, endobronchial tumor, or obstructive pneumonia may make it difficult to assess the distal airways. Here, follow-up bronchoscopy after airway palliation can provide a better definition of the distal anatomy. Finally, routine follow-up bronchoscopy may be indicated after radiation therapy or systemic treatment where there is an intrathoracic malignancy producing airway obstruction. Post-treatment bronchoscopy may reveal adequate tumor response to allow stent removal. We favor prompt removal of any stent that is no longer needed, to avoid airway inflammation, potential complications, and the need for long-term stent maintenance.

Results and Complications

As with many other procedures, good results are largely dependent on proper patient selection. Overall, satisfactory or excellent results can be achieved in 90 to 96% of patients undergoing therapeutic bronchoscopy including stenting.[1,35] All results usually depend more on the site and distal extent of tumor infiltration rather than the histology, or the style of stent chosen. More distal lesions that extend into lobar and/or segmental orifices are much less likely to achieve successful palliation than focal tracheal, mainstem bronchial, or bronchus intermedius obstruction. Although short-term outcomes after airway stenting are mostly dependent upon patient selection and proper preparation of the airway, long-term outcomes are more dependent upon the underlying pathology and the type of stent employed.

Dumon and Hood Stents

The major disadvantages of silicone stents include stent migration and obstruction of the stent with inspissated secretions. Although granuloma formation is more common after metal stent placement, it can also occur after placement of silicone stents. Diaz-Jimenez and colleagues reported 11% stent migration and 10% granuloma formation in their series of 125 silicone stent placements.[39] In the University of Washington's experience of 181 stent placements, there was a 5% rate of stent migration and a 4% rate of stent obstruction with inspissated secretions or granuloma. Cavaliere and colleagues analyzed 393 silicone stent placements in 306 patients with malignant airway obstruction and reported a migration rate of 5% and granuloma formation rate of 1%.[35] Dumon and colleagues reported the largest experience with silicone stents, collected from several European centers.[40] In this series, a total of 1,574 stents were placed in 1,058 patients, two-thirds for malignant airway obstruction. Overall, stent migration occurred in 9.5%, granulation formation in 8%, and stent obstruction by secretions in 4%.

The University of Washington experience confirms the palliative nature of silicone stent placement, with 39% of patients requiring repeat procedures for stent revision or clearance of obstructing secretions. However, because of the lack of incorporation of silicone stents into the airway wall, these complications are fairly minor and easily managed by endoscopy and stent revision or stent débridement. Overall palliation of central airway obstruction has been accomplished in 318 of 325 patients (97.8%) at the University of Washington, with no operative or procedure-related mortality, and no known subsequent mortality related to a stent complication. Similar results were achieved in the series by Cavaliere and colleagues, with 93% of patients achieving good or excellent palliation.[35] Other groups have reported 85 to 95% successful airway palliation, but these results are often poorly tabulated with different criteria for determining outcomes, making direct comparison between stents and studies difficult.[34,37] Overall, it appears that in properly selected patients, good results can be achieved in over 90% of patients receiving silicone stents, with 10 to 40% of patients requiring further endoscopies or stent revision. However, these complications are relatively minor, with only 1 apparent stent-related death reported in the literature.[37]

Gianturco Stent

The Gianturco stent has historically been one of the most commonly used stents, primarily because it is one of the earliest metal stents developed and used in the airway. Carrasco and colleagues described their experience with 36 patients receiving 64 Gianturco stents, predominantly for malignant disease.[41] In this series, 78% of patients had improvement in their symptoms. Other authors have reported similar rates of airway palliation in several small series.[36,42,43] Complications have frequently been reported after a Gianturco stent placement. Nashef and colleagues reported severe granuloma formation in patients receiving Gianturco stents for fibro-inflammatory stenosis.[43] Fracture of the stent has been reported in several series.[28,41,44] Metal stent fracture is a serious complication after Gianturco placement because of the potential for airway perforation. This has the possibility of being a fatal complication because of the overlying great vessels and the potential of tracheo-bronchial–vascular fistula.[45,46] In the series by Rousseau and colleagues and Nashef and colleagues, stent disruption or migration occurred in 22 to 32% of patients, with a 5% stent-related mortality.[36,43]

Because of the significant complications reported with this first-generation expandable metal stent, Gianturco stents are no longer routinely recommended for either benign or malignant tracheo-bronchial obstructions.

Palmaz Stent

The balloon-expanded Palmaz stainless steel stent exhibits plastic rather than elastic behavior, preventing further expansion after stent deformation. Palmaz stents have been predominantly used in the pediatric patients because of the presence of small sized stents. Successful airway palliation has been achieved in 85 to 95% of patients with both benign and malignant diseases.[47,48] However, obstructing granulations have been reported in 5 of 8 patients receiving Palmaz stents for tracheo- or bronchomalacia.[49] This same series noted obstructive granulations in 38% of patients and stent migration in 13% of patients overall. In the same series, 1 of 16 patients (6%) died after attempted stent removal. Other series have likewise noted a high percentage of obstructive granulations and stent migration after placement of Palmaz stents.[28] Given these results, the Palmaz stent is not routinely used in the adult population. However, the Palmaz stent continues to be used in the pediatric population because of the small size availability.

Strecker Stent

Similar to the Palmaz stent, the Strecker stent has plastic rather than elastic properties and is deployed over an expandable balloon. The advantage of the Strecker stent is that it does not shorten longitudinally during deployment, providing for ease and accuracy of placement. Hautmann and colleagues reported on the placement of 27 Strecker stents for tracheal or bronchial stenosis in 2000.[50] Eighty percent of patients had an immediate clinical improvement in respiratory symptoms, which was confirmed by improvement in objective lung function. In the overall series, which included patients with nitinol Accuflex stents, 11% had early difficulty with stent positioning, requiring removal, revision, or replacement. Late stent migration occurred in 12% of patients, but over half of these cases were due to tumor regression or were clinically insignificant. The majority of stents in this series were placed for advanced malignancy and so a majority of the patients died from their underlying disease. However, 3 of 65 patients (5%) died shortly after stent placement due to stent complications. Like the Palmaz stents, Strecker stents have had problems with obstructive granulations after placement.[4] Overall, the results and indications are similar for both the Palmaz and Strecker stents, neither of which have a large experience for the management of tracheobronchial obstruction. The Strecker stent has less of a problem of deformability than the Palmaz stent and may be useful for accurate stenting of short segment stenoses since it does not foreshorten on deployment. However, the absence of a covering over the metal stent makes both the Palmaz and Strecker stents poorly suited for malignant lesions since they do not offer protection against tumor ingrowth.

Wallstent

The Wallstent has been the most frequently used expandable stent in the United States for tracheobronchial obstruction. These stents are popular because of their ease of delivery under fluoroscopy. They are self-expanding, with a moderate radial force and good flexibility. Rousseau and colleagues reported their experience with 39 Wallstents in the airway in 1993.[36] In this series, 89% of patients had an improvement in their respiratory status immediately after stent placement that was maintained for a mean follow-up of 10 months (range 3 to 27 months). Complications occurred in 6 of 39 patients (15%) with Wallstents, which all consisted of tumor or granulation ingrowth leading to stent obstruction. These were managed by repeat stenting or balloon dilation within the stent. Dasgupta and colleagues reported on the placement of 51 Wallstents in 37 patients, with over half due to malignant obstruction, 30% due to tracheobronchial malacia, and 15% due to benign tracheal stenosis.[38] In these patients, symptomatic improvement was achieved in 97% and bronchoscopic evidence of airway patency was seen in all patients. Technical problems related to inappropriate length of the stent occurred in 5 of 37 patients (14%). On long-term follow-up, granuloma formation occurred in 16% of patients, and in 1 patient (3%), stent removal was mandated because of staphylococcal bronchitis.

Tsang and Goldstraw reported their results using the Wallstent in 12 patients with airway obstruction in 1992.[51] All of the patients with benign strictures had subjective and functional improvement after placement of the stent, with no evidence of restenosis within the stented segment in 6 of 7 patients. Symptoms were relieved in 4 of 5 patients with malignant strictures, but 3 of these patients (60%) required revision or had recurrent obstruction by tumor ingrowth through the stent. In another report, Tsang and colleagues recommended against using a metal stent for inflammatory strictures because of the propensity for granulation ingrowth through the interstices of the stent.[52] In this setting, the authors recommended using a sequential stenting approach, using a silicone stent during the first stage to allow healing of the inflamed area, followed by replacement with an expandable stent after a mature fibrous scar had been achieved.

Wallstents were used for the treatment of bronchial stenosis following lung transplantation.[53] Although good initial results were accomplished in all cases, granulation tissue again complicated stent management in 37% of patients.

Similar to other metallic stents, the main disadvantage of the Wallstent has been poor stent–tissue interaction, with significant local reaction and granuloma formation. Because of this, the Wallstent has recently become available with a coating of silicone rubber to prevent granulation and tumor ingrowth through the stent interstices (Wallstent with Permalume coating, Boston Scientific, Natick, MA). The American version of this stent has bare metal ends to improve anchorage of the stent, but these ends may still allow for tumor or granulation ingrowth in the uncovered areas. For most indications, the covered stent is preferred, but the stent with covering loses some of the desirable characteristics of an expandable bare metal stent; that is, reepithelialization and return of mucociliary clearance. Few data exist currently regarding the results or complications of the coated Wallstents.

Ultraflex Stent

Few studies have been published regarding the use of the Ultraflex stent. In a series placing uncovered Ultraflex stents in 6 patients with benign disease, all patients were reported to have good results with no granulation tissue at short-term follow-up.[54] Jantz and Silvestri have inserted 49 Ultraflex stents into 34 patients with both benign and malignant tracheobronchial obstructions.[28] At a mean follow-up of nearly 20 months, none of the 16 patients with benign tracheobronchial disease had suffered complications of granuloma formation or secretion retention. Stent removal was possible in 1 patient who had recurrent obstruction secondary to tracheobronchial malacia with recurrent dynamic airway collapse despite Ultraflex stenting.

The only published comparison between the Wallstent and Ultraflex stent has been in the palliative treatment of esophageal obstruction. In these patients, the Wallstent was associated with a higher procedure-related mortality and early complication rate. However, the stent dysfunction and reintervention rate was higher for patients receiving the Ultraflex stent.[55] In this series, the Wallstent was found to have a greater dynamic expansive force and the Ultraflex stent was more flexible and easier to reposition or remove after deployment. Because of its single woven wire construction, the Ultraflex stent, particularly its covered version, is significantly easier to remove than the Wallstent. Although this provides a potential advantage to use of the Ultraflex stent, there are no direct comparisons between these stents in the airway. These covered second-generation stents are now the stents of choice for expandable metal stents in the airway, pending development of a better stent.

Dynamic Stent

The major experience with the Dynamic stent has been reported by Freitag and colleagues in 1997.[18] These authors have treated 135 patients with a bifurcated Dynamic stent, 94 with malignant and 41 with benign diseases. Stent placement is potentially more complicated with a bifurcated stent, but it was achieved in all patients without major complications. The authors did not report a quantitative result, but stated that the stent provided immediate relief of dyspnea in most cases. One patient died during stent placement, but this was most likely due to the patient's underlying disease. Five of the 135 patients (4%) died of hemoptysis. Three of them had terminal lung cancer, but 2 suffered from tracheal compression by the aorta and it is not clear whether stent erosion may have resulted in an aortotracheal fistula. Three patients developed a unilateral recurrent nerve palsy and 4 patients suffered stent migration after tumor regression from radiation or systemic therapy. Five patients (4%) had marked formation of granulation tissue at the stent edges, requiring endoscopic resection. The theoretical advantage of the Dynamic stent is facilitated mucus clearance due to the flexible posterior wall. Problems with secretion retention were minimal in the Freitag series, although some patients required endoscopic suctioning early after stent placement.

Shiraishi and colleagues have reported using the Dynamic stent for 6 patients with inoperable esophageal cancer and tracheal invasion or advanced pulmonary or mediastinal malignancy.[56] Two of these patients with esophageal carcinoma presented with a tracheo- or bronchoesophageal fistula. The stent was effective in minimizing secretions from the tracheoesophageal fistula as well as maintaining a sufficient airway in all cases.

Overall, initial results with the Dynamic stent show promise for management of distal tracheal, carinal, or mainstem bronchial obstruction. No direct comparisons have been made between the Dynamic stent and the bifurcated versions of the Hood or Dumon stents, which are also used for obstructing pathology around the carina. However, these stents do not appear to be appropriate for routine use if a simpler cylinder stent in the trachea or bronchi can accomplish a complete palliation of the area of obstruction.

The University of Washington Experience

From May 1992 to February 1999, 165 patients underwent 325 endoscopic procedures for central airway obstruction. Fifty-three percent of the patients were male and ages ranged from 13 to 82 years (mean of 54 years). Fifty-six percent of patients had malignant lesions, where 64% of these were due to local invasion of nearby malignancies, 23% from metastatic tumors, and 12% from primary tracheobronchial histology. All patients were symptomatic and 56% had severe respiratory distress. Another 21% presented with stridor or wheezing and 16% with pneumonia. Ten of 165 patients (6%) presented with intubation due to respiratory failure. Fifty-one percent of these procedures were classified as urgent or emergent because of the severity of presenting symptoms. The degree of obstruction was estimated by initial bronchoscopy as > 50% of the airway in 78% of patients, > 75% obstruction in 62% of patients, and > 90% obstruction in 46% of patients.

These 165 patients underwent a total of 466 discrete interventions. These included dilation (22%), endobronchial core-out (20%), laser (12%), and brachytherapy (9%). Of all procedures performed, 110 were primary stent placement (22%) and another 71 were stent revision. Forty-five percent of patients underwent stenting for a benign disease that was not amenable to definitive surgical correction, and 55% of stent procedures were for malignancy.

Patients underwent an average of 2 procedures per patient, ranging from 1 to 22 therapeutic interventions per patient. Thirty-nine percent of patients had 2 or more procedures, confirming the palliative nature of endoscopic therapeutics and the need for ongoing interventions to obtain or maintain airway patency. Sixty-nine patients underwent 110 stent procedures. Eighty-five percent of stents placed were silicone stents (Hood Corporation) and 15% were expandable metal stents (Wallstent with or without Permalume coating, Boston Scientific). The majority of patients (87%) had only 1 stent placed, whereas 2 or 3 stents were necessary to achieve airway patency in 11% and 2% of patients, respectively. Sixty-five of 110 stents (59%) were placed in a bronchial position, and 21% in the trachea. Carinal Y stents were placed in 11%, and T or T-Y stents in 9% of patients. Twenty-five percent of patients had surgical customization of the silicone stent to maximize stent seating or aeration of lobar orifices.

Overall, successful airway palliation was achieved in 158 of 165 patients (96%). There was no operative or procedure-related mortality, and the hospital and 30-day mortality was 1.2%. None of the early deaths were related to stent complications and were a result of the progression of the patient's underlying disease. Complications occurred in 19 of 165 patients (11%). Stent-related complications were migration, occurring in 5% of stents, with stent occlusion occurring in 3% of stents placed.

Conclusion

A wide variety of airway pathology may result in central airway obstruction. For patients with both benign and malignant diseases, definitive surgical correction by tracheobronchial resection and reconstruction is preferred. However, a number of patients will have unresectable airway obstruction due to the extent of the obstruction, the natural history of the underlying disease, or medical or surgical contraindications. These patients can be palliated by a number of endoscopic strategies, employing endoscopic dilation, core-out of endobronchial tumor, laser resection, endobronchial brachytherapy, or photodynamic therapy. Airway stenting with silicone or expandable metal stents provides reliable and durable palliation in 80 to 95% of properly selected patients. The major advantages of silicone stents are the ease of customization, repositioning, and removal, with the major drawbacks being stent migration or stent obstruction. Expandable metal stents have the advantage of ease of insertion, conformation to the airway, a low inner diameter to outer diameter ratio, and stent stability. However, this is offset by the development of tumor ingrowth or granulations at the ends or through the interstices of these stents, and difficult or impossible repositioning or removal once the stent has been completely seated within the airway.

Management of the patient with central airway obstruction requires a thorough knowledge and consideration of both the surgical and endoscopic management options. This will usually require a multidisciplinary approach, with experienced thoracic surgical consultation to evaluate the potential for definitive surgical correction. The interventional bronchoscopist must then fully consider the spectrum of endoscopic therapeutics. Most patients will benefit from combining strategies in a flexible algorithm directed at optimizing patient outcomes. The benefits and risks of airway stenting must be considered in comparison to the other options for airway palliation. In refractory strictures, rapidly recurrent tumor, or extrinsic compression, endobronchial stenting will likely be necessary to achieve durable palliation of airway obstruction. The short- and long-term implications of airway stenting, including the complications of silicone versus expandable metal stents, should be considered thoroughly, with decisions regarding individual patients being based upon their anatomy and expected natural history.

References

1. Stephens KE Jr, Wood DE. Bronchoscopic management of central airway obstruction. J Thorac Cardiovasc Surg 2000;119:289–96.
2. Wood DE. Tracheal tumors. In: Wood DE, Thomas CR, editors. Mediastinal tumors. Berlin: Springer-Verlag; 1995. p. 87–93.
3. Grillo HC, Donahue DM, Mathisen DJ, et al. Postintubation tracheal stenosis: treatment and results. J Thorac Cardiovasc Surg 1995;109:486–93.
4. Nesbitt JC, Carrasco H. Expandable stents. Chest Surg Clin North Am 1996;6:305–28.
5. Trendelenburg F. Beitrage zu den operationen an den luftwegen. Langenbecks Arch Chir 1872;13:335.
6. Bond CJ. Treatment of tracheal stenosis by a new T-shaped tracheotomy tube. Lancet 1891;1:539.
7. Harkins WB. An endotracheal metallic prosthesis in the treatment of stenosis of the trachea. Ann Otol Rhinol Laryngol 1952;61:932–5.
8. Montgomery WW. T-tube tracheal stent. Arch Otolaryngol 1965;82:320–1.
9. Graziano J, Spinazolla A, Neville WE. Prosthetic replacement of the tracheal carina. Ann Thorac Surg 1967;4:1–11.
10. Rainer WG, Newby JP, Kelble DL. Long term results of tracheal support surgery for emphysema. Dis Chest 1968;53:765–72.
11. Neville WE, Hamouda F, Andersen J, Dwan FM. Replacement of the intrathoracic trachea and both stem bronchi with a molded silastic prosthesis. J Thorac Cardiovasc Surg 1972;63:569–76.
12. Duvall AJ, Bauer W. An endoscopically introducible T-tube for tracheal stenosis. Laryngoscope 1977;87:2031–7.
13. Westaby S, Jackson JW, Pearson FG. A bifurcated silicone rubber stent for relief of tracheobronchial obstruction. J Thorac Cardiovasc Surg 1982;83:414–7.
14. Amemiya R, Matsushima Y, Kunii T, et al. Palliative tracheal tube stent without tracheotomy in tracheal stenosis [letter]. J Thorac Cardiovasc Surg 1985;90:631–4.
15. Cooper JD, Pearson FG, Patterson GA, et al. Use of silicone stents in the management of airway problems. Ann Thorac Surg 1989;47:371–8.
16. Dumon JF. A dedicated tracheobronchial stent. Chest 1990;97:328–32.
17. Freitag L, Eicker R, Linz B, Greschuchna D. Theoretical and experimental basis for the development of a dynamic airway stent. Eur Respir J 1994;7:2038–45.
18. Freitag L, Tekolf E, Stamatis G, Greschuchna D. Clinical evaluation of a new bifurcated dynamic airway stent: a 5-year experience with 135 patients. Thorac Cardiovasc Surg 1997;45:6–12.
19. Belsey R. Resection and reconstruction of the intrathoracic trachea. Br J Surg 1951;38:200–5.
20. Bucher RM, Burnett WE, Rosemond GP. Experimental reconstruction of tracheal and bronchial defects with stainless steel wire mesh. J Thorac Surg 1951;21:572–83.
21. Wallace MJ, Charnsangavej C, Ogawa K, et al. Tracheobronchial tree: expandable metallic stents used in experimental and clinical applications. Radiology 1986;158:309–12.
22. Vinnograd I, Klin B, Brosh T, et al. A new intratracheal stent made from nitinol, an alloy with "shape memory effect." J Thorac Cardiovasc Surg 1994;107:1255–66.
23. Orlowski TM. Palliative intubation of the tracheobronchial tree. J Thorac Cardiovasc Surg 1987;94:343–8.
24. Colt HG, Dumon J-F. Airway stents present and future. Clin Chest Med 1995;16:465–78.
25. Wood DE, Vallières E, Karmy-Jones R. Current status of airway management in lung transplant patients. Curr Opin Organ Transp 1999;4:264–8.
26. Wood DE, Vallières E. Tracheobronchial resection and reconstruction. Arch Surg 1997;132:850–7.
27. Donahue DM, Grillo HC, Wain JC, et al. Reoperative tracheal resection and reconstruction for unsuccessful repair of postintubation stenosis. J Thorac Cardiovasc Surg 1997;114:934–8.
28. Jantz MA, Silvestri GA. Silicone stents versus metal stents for management of benign tracheobronchial disease: pro: metal stents. J Bronchol 2000;7:177–83.
29. Rodriguez AN, Diaz-Jimenez JP, Edell ES. Silicone stents versus metal stents for management of benign tracheobronchial disease: con: metal stents. J Bronchol 2000;7:184–7.
30. Kim H. Stenting therapy for stenosing airway disease. Respirology 1998;3:221–8.
31. Wilson RS. Anesthetic management for tracheal reconstruction. International trends in general thoracic surgery. Vol 2. Philadelphia: WB Saunders; 1987. p. 3–12.
32. Noppen M, Dhondt E, Meysman M, et al. A simplified insertion technique for tracheobronchial silicone stents. Chest 1994;106:520–3.
33. Acuff TE, Mack MJ, Ryan WH. Simplified placement of a silicone tracheal Y stent. Ann Thorac Surg 1994; 57:496–7.
34. Sonett JR, Keenan RJ, Ferson PF, et al. Endobronchial management of benign, malignant, and lung transplant airway stenosis. Ann Thorac Surg 1995;59:1417–22.
35. Cavaliere S, Venuta F, Foccoli P, et al. Endoscopic treatment of malignant airway obstruction in 2,008 patients. Chest 1996;110:1536–42.
36. Rousseau H, Dahan M, Lauque D, et al. Self-expandable prostheses in the tracheobronchial tree. Radiology 1993;188:199–203.
37. Martinez-Ballarin JI, Diaz-Jimenez JP, Castro MJ, Moya JA. Silicone stents in the management of benign tracheobronchial stenoses: tolerance and early results in 63 patients. Chest 1996;109:626–9.
38. Dasgupta A, Dolmatch BL, Abi-Saleh WJ, et al. Self-expandable metallic airway stent insertion employing flexible bronchoscopy: preliminary results. Chest 1998;114:106–9.
39. Diaz-Jimenez JP, Farrero Munoz E, Martinez Ballarin JI, et al. Silicone stents in the management of obstructive tracheobronchial lesions: a two year experience. J Bronchol 1994;1:15–8.
40. Dumon JF, Cavaliere S, Diaz-Jimenex JP, et al. Seven-year experience with the Dumon prosthesis. J Bronchol 1996;3:6–10.
41. Carrasco CH, Nesbitt JC, Charnsangavej C, et al. Management of tracheal and bronchial stenosis with the Gianturco stent. Ann Thorac Surg 1994;58:1012–7.
42. Sawada S, Tanigawa N, Kobayashi M, et al. Malignant tracheobronchial obstructive lesions: treatment with Gianturco expandable metallic stents. J Radiol 1993;188:205–8.
43. Nashef SA, Dromer C, Velly FJ, et al. Expanding wire stents in benign tracheobronchial disease: indications and complications. Ann Thorac Surg 1992;54:937–40.
44. Hramiec JE, Haasler GB. Tracheal wire stent complications in malacia: implications of position and design. Ann Thorac Surg 1997;63:209–12.
45. Alfaro J, Varela G, De-Miguel E, Martin de Nicolas JL. Successful management of a tracheo-innominate artery fistula following placement of a wire self-expandable tracheal Gianturco stent. Eur J Cardiothorac Surg 1993;7:615–6.
46. Maynar M, Lopez L, Gorriz E. Massive brachiocephalic artery bleeding due to a Gianturco tracheal stent. J Vasc Intervent Radiol 1993;4:289–91.

47. Slonim SM, Razavi M, Kee S, et al. Transbronchial Palmaz stent placement for tracheo-bronchial stenosis. J Vasc Intervent Radiol 1998;9:153–60.
48. Beer M, Wittenberg G, Sandstede J, et al. Treatment of inoperable tracheobronchial obstructive lesions with the Palmaz stent. Cardiovasc Intervent Radiol 1999; 22:109–13.
49. Filler RM, Forte V, Chait P. Tracheobronchial stenting for the treatment of airway obstruction. J Pediatr Surg 1998;33:304–11.
50. Hautmann H, Bauer M, Pfiefer KJ, Huber RM. Flexible bronchoscopy: a safe method for metal stent implantation in bronchial disease. Ann Thorac Surg 2000;69:398–401.
51. Tsang V, Goldstraw P. Self-expanding metal stent for tracheobronchial strictures. Eur J Cardiothorac Surg 1992;6:555–60.
52. Tsang V, Williams AM, Goldstraw P. Sequential silastic and expandable metal stenting for tracheobronchial strictures. Ann Thorac Surg 1992;53:856–60.
53. Carre P, Rousseau H, Lombart L, et al. Balloon dilatation and self-expanding metal Wallstent insertion for management of bronchostenosis following lung transplantation: The Toulouse Lung Transplantation Group. Chest 1994;105:343–8.
54. Ducuc Y, Khalafi RS. Use of endoscopically placed expandable nitinol tracheal stents in the treatment of tracheal stenosis. Laryngoscope 1999; 109:1130–3.
55. Schmassmann A, Meyenberger C, Knuchel J, et al. Self-expanding metal stents in malignant esophageal obstruction: a comparison between two stent types. Am J Gastroenterol 1997;92:400–6.
56. Shiraishi T, Kawahara K, Shirakusa T, et al. Stenting for airway obstruction in the carinal region. Ann Thorac Surg 1998;66:1925–9.

Editor's Note

Dr. Wood firmly advises consideration of all therapeutic options for each patient with airway obstruction, rather than hustling to stent placement, especially for benign disease. The lengthening of tracheobronchial strictures and the production of additional complications by use of expandable stents, which we have seen recently, underlines this wisdom. Gaissert and colleagues report 13 patients who failed to obtain palliation with such stents (Wallstent, Microvasive Ultraflex) and suffered stent-related extension of strictures and granulations in the previously normal airway.[1] With coated stents, rings of stenotic tissue formed at either end.

Special problems arise in the removal of obstructing expandable wire stents. Sometimes, the stent may be plucked out wire-by-wire bronchoscopically. Extended transsternal linear tracheostomy, cricoid to carina, is required to remove some uncoated stents. That portion of the stent incorporated into the membranous wall is best left in place, to avoid a tracheoesophageal fistula. A permanent long T tube is then necessary. If a coated stent has produced obstruction at either end due to wires protruding beyond the coating, and if the lumen of the stent is sufficiently wide, then a longer T tube may be inserted through the stent. Rarely is the original lesion, which might have been easily resected originally, any longer resectable. Examples of these injuries are to be seen in the Tracheobronchial Endoscopic Atlas (Figures 29 and 30 [Color Plate 15]).

In an earlier editorial, I deplored the use of expandable stents for benign disease, where simple surgical excision is likely to produce complete cure in most patients.[2] Silicone stents such as the Dumon stent also produce additional injury, but these are often reversible after extraction of the stent; not so with expandable wire stents.

The problem, which is a growing one, represents uncritical application of new gadgets and techniques without full understanding of the pathology treated or the range of therapeutic options available. Many of these unfortunate patients are first seen by stent placers, who unwittingly produce major and uncorrectable complications.

Hermes C. Grillo

References

1. Gaissert HA, Grillo HC, Wright CD, et al. Lengthening and complication of benign tracheobronchial strictures by self-expanding metal stents. J Thorac Cardiovasc Surg 2003. [In press]
2. Grillo HC. Stents and sense [editorial]. Ann Thorac Surg 2000;70:1142.

Radiation Therapy in the Management of Tracheal Cancer

Noah C. Choi, MD

Tracheal carcinoma is rare and represents 0.2% of respiratory tract malignancies.[1–3] Etiologic factors for squamous cell cancer include tobacco and environmental pollutants, which are also the main causes for malignancies of the larynx, bronchus, and lung. Histologic types of tracheal malignancies include squamous cell carcinoma, adenoid cystic carcinoma, and other less common types of tumors.[4]

Squamous cell carcinoma accounts for 50% of all primary tracheal malignancies. It occurs predominantly in male smokers between the ages of 45 and 80 years, more often in the distal one-third of the trachea, and presents as an exophytic and usually ulcerated mass obstructing the lumen. Many develop another primary carcinoma in the larynx or lung. Squamous cell carcinoma of the trachea has an aggressive behavior characterized by early invasion into the adjacent mediastinal structures and metastasis to mediastinal and cervical lymph nodes. Metastasis to lung, liver, and bone is a presentation of late stage disease. Tracheomediastinal and tracheo-esophageal fistulae are late complications of advanced stage tracheal carcinoma.

Adenoid cystic carcinoma comprises approximately 30% of all primary tracheal malignancies. The characteristics of this tumor include a growth rate slower than squamous cell carcinoma, a predilection for the upper one-third of the trachea, and extensive endophytic infiltration beyond the exophytic portion of the tumor.[4] This endophytic infiltration may extend as much as 2.0 cm proximal or distal to the boundary of the gross tumor. Perineural infiltration beyond the gross tumor in vertical directions is another characteristic of adenoid cystic carcinoma. Metastasis to the regional lymph nodes is found in about 10% of cases at the time of surgery.

Staging Work-Up

Assessment of the tumor extent and the physiologic condition of a patient by means of performance status, and cardiac and pulmonary functional reserve, should provide a comprehensive basis for selecting proper treatment and estimating results.[5–9] The extent of the primary tumor (T factor) is evaluated by chest radiographs, computed tomography (CT) scan of the chest, and bronchoscopy. A locally advanced squamous

cell carcinoma of the trachea, which was judged unresectable, is shown in an axial view of a CT scan of the chest (Figure 41-1). A CT scan of the chest and upper abdomen provides critical information for tumor staging that is otherwise unavailable or less than satisfactory from conventional radiographs. The size and shape of the primary lesion and its relationship to the surrounding normal structures are very well defined by this method. A coronal and sagittal reconstruction of the CT scan provides the superior and inferior extent of the tumor—critical information for treatment planning. The mediastinum can be satisfactorily evaluated for direct tumor invasion and metastases. Barium swallow is important in obtaining additonal information in patients who are suspected of having a possible tracheoesophageal fistula. It is also important to perform esophagoscopy at the time of bronchoscopy, when tumor invasion through the membranous wall of the trachea into the esophageal wall is suspected.

Indications for Radiation Therapy

Surgery remains the treatment of choice for resectable tracheal carcinomas.[1–3,7–9] The following sections present situations for radiation therapy use.

Postoperative Radiation Therapy in Resected Tracheal Cancer

The fate of patients with either incomplete resection, metastatic disease in the regional lymph nodes, or both is poor. It has also been well demonstrated that radiation therapy is capable of sterilizing residual cancer in either the tumor bed region, the regional lymph nodes, or in both, when an adequate radiation dose is administered in thoracic and other malignant tumors.[10] In squamous cell carcinoma of the lung, postoperative radiotherapy using a moderate dose of radiation (45 to 54 Gy in 25 to 30 fractions over a period of 5 to 6 weeks) showed a significant improvement in locoregional tumor control.[11] If it is assumed that the

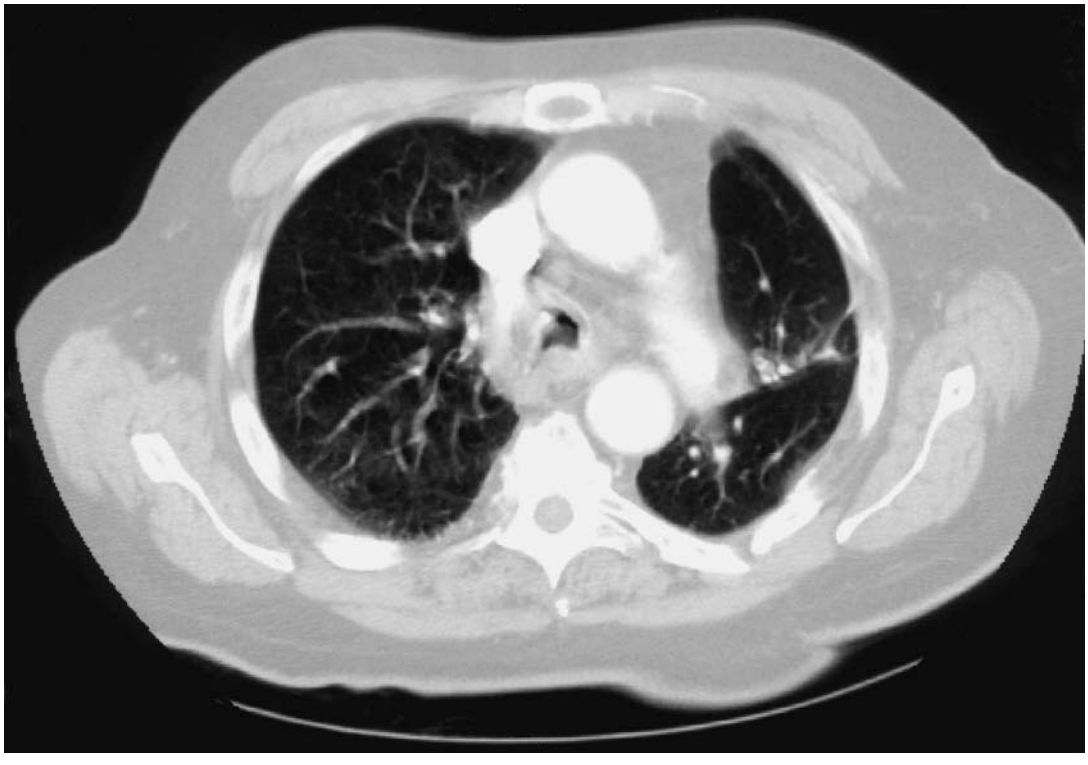


FIGURE **41-1** *An axial view of the thorax with a squamous cell carcinoma involving the right lateral and posterior wall of the trachea.*

radiation response of primary squamous cell carcinoma of the trachea and carina are similar to that of bronchogenic carcinoma, then postoperative radiotherapy would be a valuable adjuvant treatment for patients with either incomplete resection, metastatic disease in the regional lymph nodes, or both.

Even though there are not enough reports on the role of postoperative radiotherapy in adenoid cystic carcinoma of the trachea and carina,[12,13] an analogy, which can be drawn from the clinical experience in adenoid cystic carcinoma of the minor salivary glands in the head and neck region, may be a useful guide. Douglas and colleagues reported results of neutron radiotherapy for patients with gross residual adenoid cystic carcinoma of minor salivary glands after attempted surgical resection.[14] The median dose was 19.2 neutron Gy in 1.2 neutron Gy doses, given 4 times a week. The median duration of follow-up was 32 months. Sites of the primary disease and the number of patients treated per disease site were as follows: paranasal sinus, 31; oral cavity, 20; oropharynx, 12; nasopharynx, 11; trachea, 6; and other sites in the head and neck, 4. The 5-year actuarial locoregional tumor control rate for all patients treated with curative intent ($n = 72$) was 47%. The 5-year actuarial overall survival and cause-specific survival were 59% and 64%, respectively. Patients with adenoid cystic carcinoma of the oral cavity and oropharynx were able to receive the intended dose of neutron radiation (19.2 neutron Gy), whereas those with a primary lesion in the nasopharynx and paranasal sinus with skull base involvement were treated with a lower dose of neutron radiation (12 neutron Gy) because of the risk of normal tissue injury at the skull base and brain stem. Five-year locoregional tumor control was 59% for patients treated with a high neutron dose for the primary oral cavity and oropharyngeal lesions, whereas it was 18% for those treated with a low neutron dose for the nasopharynx and paranasal sinus lesions involving the skull base and brain stem.

Both photons and neutrons have been used for postoperative radiotherapy for residual tracheal adenoid cystic carcinoma. Douglas and colleagues treated 5 patients with residual tracheal adenoid cystic carcinoma postoperatively with neutron radiation.[14] Five-year locoregional control, overall survival, and cause-specific survival were 40%, 67%, and 67% respectively. Ogino and colleagues treated 7 patients with adenoid cystic carcinoma of the trachea for incomplete resection margins with photon radiation.[12] Local tumor control was obtained in 3 of 4 patients when the radiation dose administered was 60 Gy or higher, and in 1 of 3 patients when the administered radiation dose was less than 60 Gy.

Therefore, it seems likely that a radiation dose of 60 Gy in 2 Gy dose fractions, 5 fractions per week, over a period of 6 weeks of photon radiation, or a biologically equivalent dose of neutron radiation, is necessary to convert a surgical resection from incomplete to complete, by sterilizing residual microscopic carcinoma in the tumor bed and regional lymph nodes in both squamous cell carcinoma and adenoid cystic carcinoma. Such postoperative radiotherapy is likely to result in an improvement in survival. For gross residual carcinoma, the radiation dose needs to be increased to 68 to 70 Gy in 2 Gy dose fractions, 5 fractions per week, over a period of 6.8 to 7 weeks.

High-Dose Radiation Therapy for Unresectable and Medically Inoperable Tracheal Cancers

The relationship between radiation dose and tumor control has been described in nonsmall cell carcinoma of the bronchus.[15,16] Limited data from studies of tracheal tumors also suggest that a radiation dose higher than 60 Gy/30 fractions/6 weeks is necessary to achieve local tumor control in squamous cell carcinoma of the trachea.[3,17–25] If we assume that squamous cell carcinoma of the trachea and carina does respond to radiation in a similar fashion to primary squamous cell carcinoma of the bronchus, then high dose radiotherapy is warranted for unresectable and medically inoperable squamous cell carcinoma of the trachea and carina. Although it is based on limited data, it is likely that a radiation dose higher than 60 Gy/30 fractions/6 weeks, and preferably in the order of 70 Gy/35 fractions/7 weeks, is necessary in order to provide local tumor control in the majority of patients with unresectable squamous cell carcinoma.

For adenoid cystic carcinoma of the trachea, the data on the radiation dose–tumor control relationship are very sparse.[12,13,18–20,26] However, the limited data suggest that a radiation dose higher than 60 Gy/30 fractions/6 weeks, and preferably near 70 Gy/35 fractions/7 weeks, is necessary to achieve local tumor control. Adenoid cystic carcinoma of the trachea has a long natural history, and the risk period for local tumor recurrence after radiation therapy is much longer than that of squamous cell carcinoma.

In a retrospective study, Huber and colleagues compared neutrons (median dose of 16 neutron Gy), photons (median dose of 64 Gy), or mixed beam (8 neutron Gy and 32 photon Gy) for local tumor control and survival in 75 patients with inoperable, recurrent, or incompletely resected adenoid cystic carcinoma of the head and neck region.[27] The median follow-up was 51 months and surviving patients had a minimum follow-up of 3 years. The actuarial 5-year local control was 75% for neutron, 32% for photon, and 32% for mixed beam. Multivariate analysis showed that postoperative radiotherapy and small tumor size were associated with better local control.

The dose-response data from clinical studies on adenoid cystic carcinoma of the trachea in the head and neck regions support high-dose radiotherapy for primary adenoid cystic carcinoma of the trachea.

Palliation of Distressing Symptoms

Relief of distressing symptoms is another important goal in caring for patients with advanced stage of tracheal carcinoma. Hemoptysis, which is alarming to patients and their families, is relieved in over 80% of patients receiving a moderate dose (40 to 45 Gy) of irradiation.[28,29] Dyspnea associated with obstruction of the trachea, carina, or mainstem bronchi is also relieved in over 80% of patients with 45 to 50 Gy of radiation. Cough associated with carcinoma in either the trachea or carina is more likely to be relieved than cough associated with a peripheral tumor of the lung. Local pain associated with direct spread of the tumor to the surrounding normal structures such as the mediastinum, vertebral bodies, brachial plexus, and chest wall, has also been well relieved with radiation.

Radiotherapeutic Factors and Techniques

The importance of radiotherapeutic factors and techniques cannot be overemphasized, inasmuch as the outcome of the treatment depends heavily on the success or failure of sterilization of the tumor in the target volume and the relative frequency of serious complications associated with the techniques used.[30] The optimum time for the initiation of postoperative radiation is approximately 6 weeks after the tracheal resection and reconstruction. It is important that the site of tracheal or tracheobronchial reconstruction is adequately healed and that it can withstand a moderate to high dose of radiation.

Target Volume

A comprehensive review of the preoperative CT scan of the chest is essential in evaluating the extent of gross tumor and tumor bed region relative to the surrounding normal structures and the regional nodal status. The treatment volume consists of three components.[30,31] The gross target volume (GTV) includes residual tumor at the resection margins and involved regional lymph nodes. The clinical target volume (CTV), which is added to the GTV to cover potential microscopic disease in adjacent tissue and organs, includes a radial margin of 2.0 cm and a margin of 4.0 cm in the cranial and caudal directions. Adenoid cystic carcinoma is a good example of spread along the perineural lymphatics of the tracheal wall beyond the boundary of the gross tumor. In order to take into account organ motion and daily variation in patient set-up, an additional 1.0 cm margin is added to the CTV as a planning target volume (PTV). Therefore, the combined CTV and PTV, beyond the GTV, include a radial margin of 3.0 cm and a margin of 5 cm in the cranial and caudal directions.

If regional lymph nodes are involved, then one sentinel nodal station beyond the involved lymph nodes is covered as the CTVn. The margins given for the CTV need to be reduced after 50 Gy. Therefore,

the combined CTV and PTV after 50 Gy includes a radial margin of 2.0 cm and 3.0 cm for the cranial and caudal margins.

Optimal Dose and Fractionation Schedule

The radiation dose schedule for postoperative therapy varies with the degree of residual tumor burden. For patients with clear but close margins with clearance of < 0.2 cm, the optimal radiation dose schedule for prevention of locoregional recurrence is in the order of 50.4 to 54 Gy, administered with daily fractions of 1.80 Gy, 5 days a week over a period of 5.6 to 6 weeks. Assuming that the radiation response of tracheal squamous cell and adenoid cystic carcinoma is similar to that of bronchogenic carcinoma, data from previous studies showed that this level of radiation dose schedule is near the optimum in forestalling a relapse of the tumor in the tumor bed region or regional lymphatic areas.[15] For residual microscopic tumor at the resection margins or regional lymph nodes, a total radiation dose of 60 Gy administered in 30 fractions over a period of 6 weeks is recommended. For gross residual tumor, a total radiation dose of 66 to 70 Gy in 33 to 35 fractions over a period of 6.6 to 7 weeks is required for a high rate of locoregional tumor control.[3,21]

For unresectable and medically inoperable tracheal carcinomas, the radiation dose schedule should be high enough for the sterilization of gross tumor. However, such high-dose radiation therapy should be guided by the tolerance of the trachea and esophagus. A combination of external radiation and endotracheal brachytherapy may meet the need for such high-dose radiotherapy. I have used a total dose of 76 Gy which is administered in two sessions: the initial 68 Gy in 34 fractions over a period of 6.8 weeks with three-dimensional (3-D) conformal radiotherapy, and the subseqent boost dose of 8 Gy at 0.5 cm from the source to the gross tumor by means of endotracheal brachytherapy. The dose limiting organs in high-dose radiotherapy are the esophagus, the membranous wall of trachea, and the spinal cord. Thus, the potential risk for inducing a tracheoesophageal fistula, when endotracheal brachytherapy is combined with external radiotherapy, needs to be addressed and discussed with the patient. To avoid a direct contact between the brachytherapy source wire and the tracheal wall, particularly the membranous wall, it is desirable to use a special device with which the radiation source wire is kept centered within the lumen of the trachea.[32]

Conventional Two-dimensional Radiation Therapy

The arrangement of radiation portals is dependent on the planned total dose, the energy levels of radiation, and the shape of the target volume. Because of the scatter irradiation of low energy beams and the large penumbra of a ^{60}Co unit, high energy beams (10 to 25 MV photon) are preferred for curative radiation therapy, with an aimed total dose of 66 to 68 Gy.

An arrangement of two parallel opposed portals (POP) applied anteroposteriorly (AP) and posteroanteriorly (PA) to the chest is simple and accurate, with the least risk of a geographical miss. The maximum dose that can be administered with this technique is, however, in the range of 44 Gy because of the tolerance limit of the spinal cord. When a posterior spinal cord block was used to keep the spinal cord dose within the tolerance limit, while a total dose of 60 Gy was administered at the midplane, an increase in local tumor recurrence was noted because of a zone of a low tumor dose at the posterior mediastinum along the vertebral column in lung cancer. An outright three-field arrangement of AP, right posterior oblique (RPO), and left posterior oblique (LPO) has also been used for a small target volume located centrally. Drawbacks to this technique are a relatively higher radiation dose (40 to 50% of total dose) to the pulmonary tissue in the path of the oblique beams, and the difficulty of dealing with a large target volume because of poor pulmonary tolerance. The rotational technique alone is undesirable because of the potential risk of increased pulmonary complications.

A treatment technique that I have used is a combination of AP–PA–POP with a subsequent three-field of AP–RPO–LPO, and right and left lateral opposed boost portals. We have administered a total dose of 60 to 68 Gy to the target volume, whereas normal vital structures are kept below the threshold dose for serious

complications; that is, ≤ 44 Gy to the spinal cord and ≤ 12 to 14 Gy to the pulmonary tissue in the path of the oblique beams. For gross tumor involving the trachea and/or carina, a total dose of 66 to 68 Gy is administered by a combination of AP–PA–POP for the initial 36 Gy and AP–RPO–LPO for an additional dose of 30 to 32 Gy, using daily fractions of 2.0 Gy, 5 days a week.[33] An alternative approach to the combination of AP–PA–POP (36 Gy) and AP–RPO–LPO (30 to 32 Gy) is to add right lateral (RL) and left lateral (LL) opposed portals for the last 8 to 10 Gy. By doing so, the radiation dose to the pulmonary tissue in the path of RPO and LPO portals is significantly reduced. The same technique is also used for patients with involved regional lymph nodes.

Three-dimensional Conformal Radiation Therapy

With 3-D CT scan simulation of the chest for tracheal cancer, the radiation treatment plan is significantly improved over that of a two-dimensional treatment plan.[30] A 3-D radiation plan provides accurate body contours at different levels of the target volume, improved definition of tumor size and location relative to other normal vital structures, and a precise measurement of the thickness of the pulmonary tissue in the path of the radiation beams for which a correction for the increased transmission in pulmonary tissue is made. With the beam's eye-view of the portals and dose–volume histogram (DVH), one can achieve the optimum treatment plan for individual patients. The maximum differential between the radiation dose of GTV and that of the adjacent normal vital organs can be achieved. DVH in 3-D conformal treatment plan is an essential tool to determine the optimum treatment plan. The optimized treatment plan is studied to achieve the desired balance between normal tissue complication probability (NTCP) and tumor control probability (TCP). By using a DVH, one can adjust the relative risk of pneumonitis by modifying the arrangement of radiation portals. It may require 5 to 7 portals to achieve the optimum treatment plan. Axial and sagittal views of a composite isodose plan, and DVHs for GTV, spinal cord, lung, and esophagus, from a 3-D radiation plan for a patient with medically inoperable squamous cell carcinoma of the trachea, are shown in Figures 41-2 through 41-4. This patient was treated with a total dose of 75.4 Gy by a combination of 68.4 Gy in 38 fractions over a period of 7.6 weeks of external radiation, and a boost dose of 7 Gy at 0.5 cm from the radiation source by endotracheal brachytherapy using ^{192}Ir sources 2 weeks after the completion of the external radiation therapy. The endotracheal catheters are shown in Figure 41-5.

Concurrent Chemotherapy

Assuming that the biological characteristics of squamous cell carcinoma of the trachea and carina and their response to radiation and chemotherapy are very similar to those of primary carcinoma of the bronchus and lung, it is recommended that radiation therapy be combined with chemotherapy for patients with unresectable or gross residual squamous cell carcinoma.

Dillman and colleagues compared radiation therapy alone (60 Gy/30 fractions/6 weeks) with sequential chemoradiotherapy, in which two cycles of induction chemotherapy (cisplatin 100 mg/m^2 IV on days 1 and 29, and vinblastine 5 mg/m^2 IV on days 1, 8, 15, 22, and 29) were administered first, and radiation therapy (60 Gy/30 fractions/6 weeks) was administered subsequently starting on day 50.[34] The 5-year survival rates were 7% with radiation therapy alone as compared to 16% with chemoradiotherapy. Arriagada and colleagues also compared radiation therapy alone with chemoradiotherapy, in which 3-monthly cycles of VCPC (vindesine 1.5 mg/m^2 on days 1 and 2; cyclophosphamide 200 mg/m^2 on days 2 to 4; cisplatin 100 mg/m^2 on day 2; lomustine 50 mg/m^2 on day 2 and 25 mg/m^2 on day 3) were administered before and after radiation therapy (65 Gy/26 fractions/6.5 weeks) for patients with stage IIIA and IIIB nonsmall cell lung carcinoma.[35] The eligibility criteria were less restrictive in this study than those of Dillman and colleagues.[34] The locoregional failure that was assessed by bronchoscopy and biopsy at 3 months after the completion of the therapies was found to be in the order of 80% for both chemoradiotherapy and radiation therapy alone groups. However,

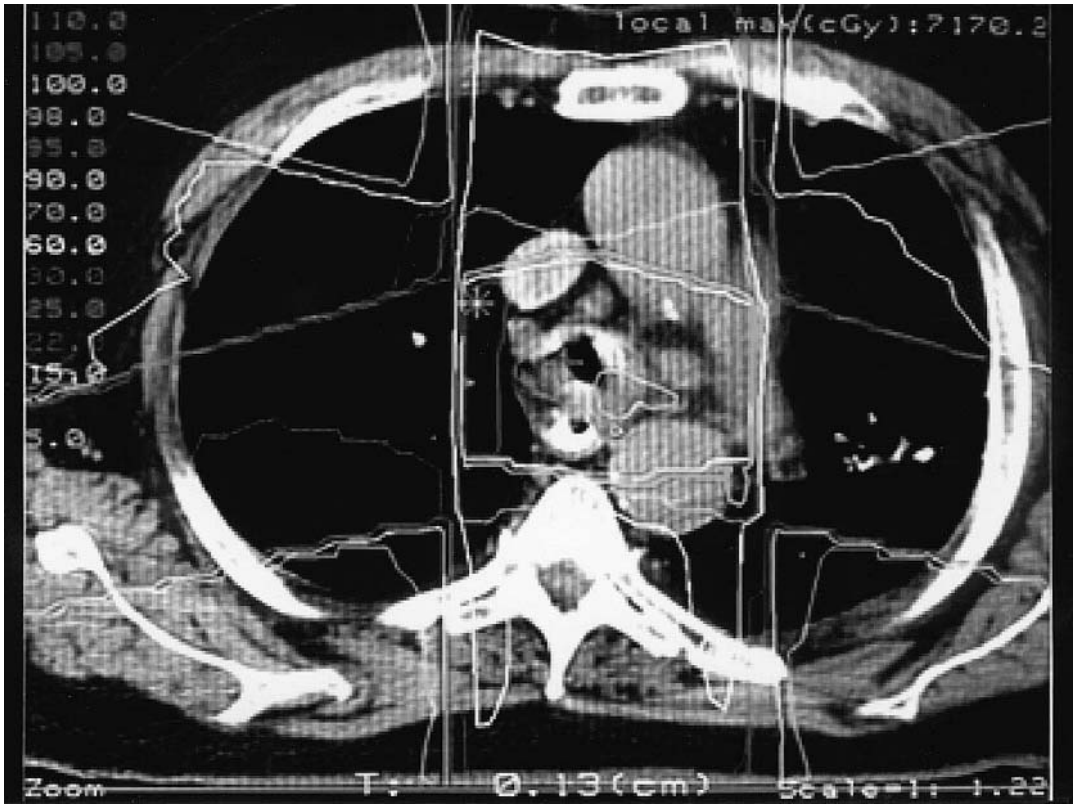


FIGURE **41-2** *An axial view of the thorax with a composite isodose plan of three-dimensional conformal radiotherapy using 6 portals (AP–PA, RAO–LPO, LAO–RPO). The planning target volume (PTV) is covered with 95% of the isodose line. AP = anteroposteriorly; LAO = left anterior oblique; LPO = left posterior ablique; PA = posteroanteriorly; RAO = right anterior oblique; RPO = right posterior oblique.*

the distant failure rate at 5 years was reduced from 70% with radiation therapy alone to 49% by addition of chemotherapy. Thus, a combination of radiotherapy and chemotherapy seems most appropriate for unresectable squamous cell carcinoma of the trachea and/or carina. Chemotherapy for adenoid cystic carcinoma has not been studied extensively, and its role in curative therapy remains to be determined.

Results

Postoperative Radiation Therapy

For patients with resectable tumor, the cure rate by surgery alone is in the range of 40 to 50%, and the majority of failures are due to presumably a combination of locoregional and distant metastases. Because of the rareness of these tumors, data on the pattern of failure after surgery are very sparse.

The ultimate goal of postoperative radiation therapy is to improve the probability of cure for patients with a high risk for local and regional failure. However, postoperative radiation therapy is a local and regional treatment, and its benefit may not result in a gain in survival should there be preexisting distant metastases. Patients with a positive margin for residual cancer have a high risk for local recurrence and subsequent failure.

When we compared the outcome of therapy in patients who received postoperative radiotherapy for either positive resection margins for residual cancer or close margins of 0.2 cm or less with those who had clear margins and did not receive postoperative radiation therapy, the median survival time was 136 months

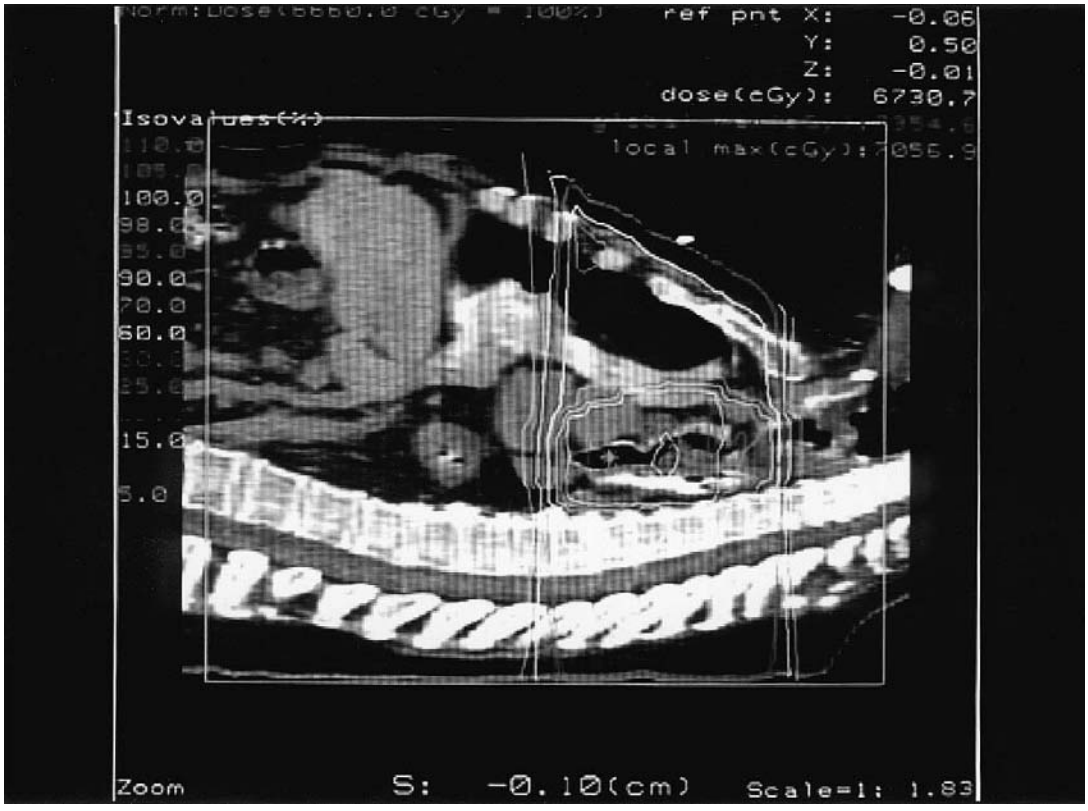


FIGURE **41-3** *A sagittal view of the thorax with a composite isodose plan of three-dimensional conformal radiotherapy. The planning target volume (PTV) is covered with 95% of the isodose line.*

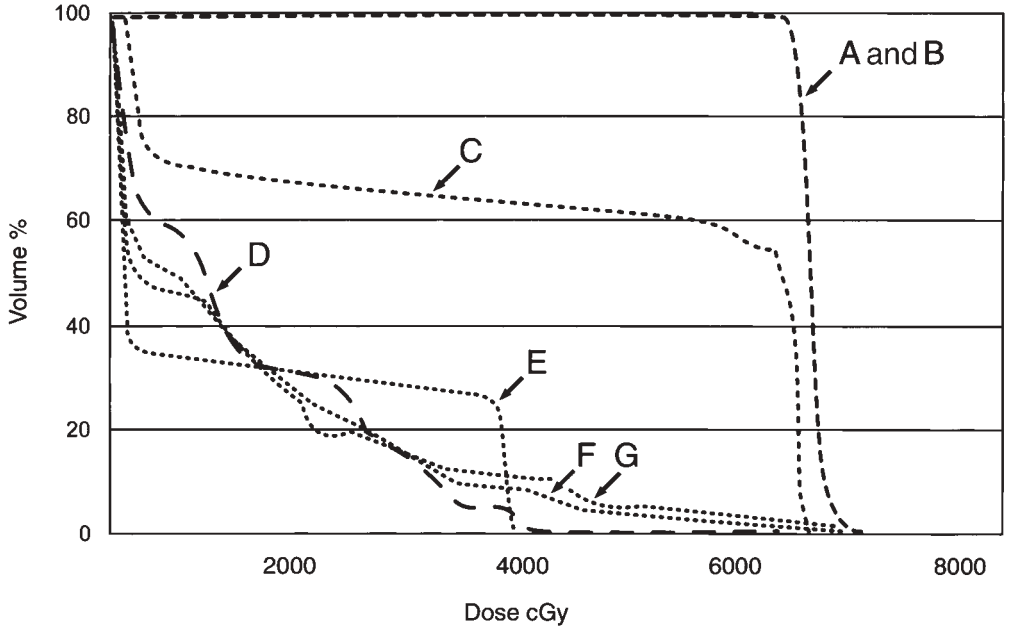


FIGURE **41-4** *Dose–volume histogram (DVH) showing the coverage of gross target volume (GTV) and planning target volume (PTV)* (A and B) *with the prescribed radiation dose of 68.4 Gy. The DVH for normal organs is shown:* C, *esophagus;* D, *left lung; E, spinal cord; F, both right and left lungs; G, right lung.*

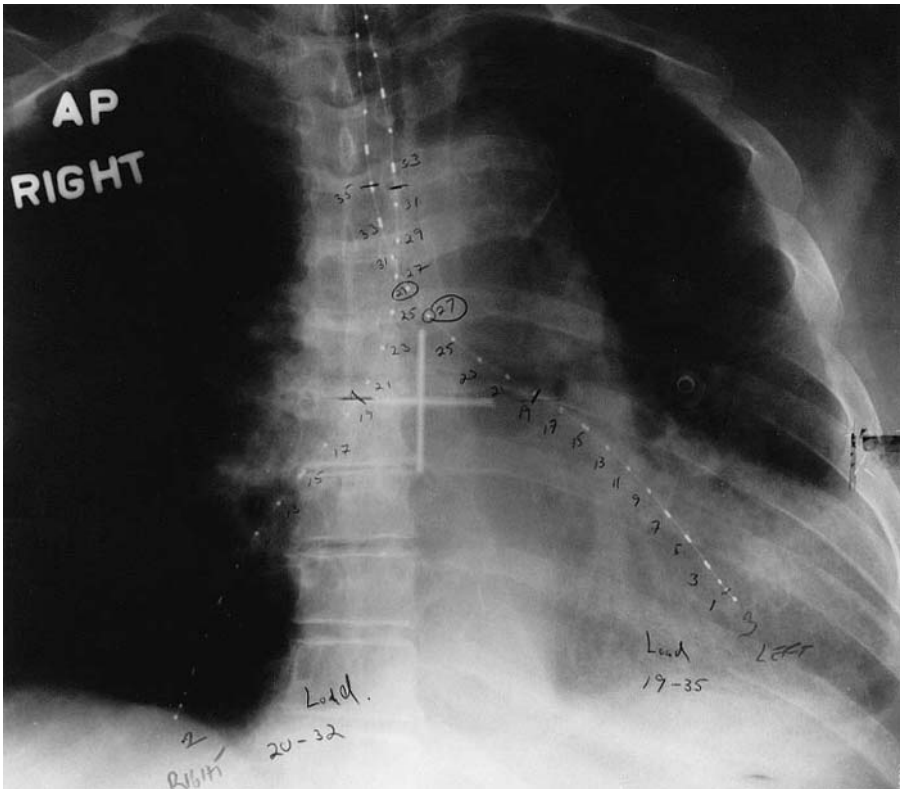


FIGURE 41-5 *An anteroposterior view of a chest after placement of two endotracheal catheters for endotracheal brachytherapy. The patient received 7.0 Gy at 0.5 cm from the source (^{192}Ir) as a boost dose of radiation after a total dose of 68.4 Gy in 38 fractions over a period of 7.6 weeks by three-dimensional conformal radiation therapy.*

for patients treated with surgery and postoperative radiation therapy ($n = 52$) as compared to 127 months for patients treated with surgery alone ($n = 31$).[36] There were no significant differences in 5- and 10-year survival rates.

These data suggest that postoperative radiotherapy is beneficial in improving survival in patients with either resection margins positive for residual cancer or close margins.

High-Dose Radiotherapy for Unresectable or Medically Inoperable Tracheal Cancers

The primary goal of high-dose radiation therapy is cure for patients with unresectable tracheal carcinoma. Radiation therapy is, however, a local and regional treatment that cannot alter the course of the disease if there is preexisting distant metastasis. Although high-dose radiation therapy is employed to obtain long-term control of unresectable tracheal cancer, associated distressing symptoms (ie, dyspnea secondary to airway obstruction, cough, and hemoptysis) are also relieved.

The prognosis of patients with unresectable tracheal cancer is poor. For squamous cell cancer, median survival time is of the order of 6 to 10 months after radiation therapy. The natural course of adenoid cystic carcinoma is much longer than that of squamous cell carcinoma. Thus, the time to recurrence after high dose of radiation therapy in adenoid cystic carcinoma can be as long as 6 to 8 years. Manninen and colleagues reported results of primary radiation therapy in 44 patients with squamous cell carcinoma.[20] The median survival time was 8 months from the time of diagnosis. Complete response to radiation therapy was a favorable prognostic sign. For those who achieved complete response, the survival rates at 1, 2, and 5 years were 45%, 18%, and 9%, respectively. Mornex and colleagues reported results of radiation therapy in 84 patients with primary tracheal carcinoma, which included squamous cell carcinoma ($n = 70$), undifferentiated carcinoma ($n = 9$), adenocarcinoma ($n = 3$), and small cell carcinoma ($n = 2$).[3] The survival rates at 1, 2, and 5 years were

46%, 21%, and 8%, respectively. Prognostic factors included tumor size (< 3 cm versus > 3 cm), performance status, and radiation dose. Five-year survival rates were 12% versus 5% with radiation dose > 56 Gy versus < 56 Gy, respectively. Local tumor control was also found to be radiation dose dependent. It was achieved in 69% versus 34% with radiation dose > 56 Gy versus < 56 Gy, respectively.

Reports on long-term follow-up after high-dose radiation therapy for patients with unresectable adenoid cystic carcinoma are sparse and incomplete.[12,13,18–20,26] Most reports include a relatively small number of patients and it is difficult to draw a meaningful conclusion. Given the long natural history of this tumor, it is important to aim for control of the local regional disease with a high dose of radiation therapy. This approach offers an opportunity for local tumor control and possible cure, even though the risk for distant failure from microscopic seeding is high.

New Trends in Prospect

New approaches are being sought to achieve a better control of the local and regional tumors than what the current radiation dose schedule has been able to attain.

Altered Fractionation Schedules

Accelerated or hyperfractionated radiation therapy may exploit the radiobiologic advantages of both a reduced fraction size for late-reacting tissues (lung, spinal cord, connective tissue) and a shortened overall treatment time against rapidly proliferating tumors such as squamous cell carcinoma of the trachea.[37,38] Repair of sublethal radiation damage in aerobic mammalian cells is essentially complete within 2 to 4 hours.[39,40] When a rapidly proliferating tumor cell population such as squamous cell carcinoma of the trachea is growing in normal tissue whose cells are nonproliferative or slowly proliferating, an advantage accrues to the tumor cells if the treatment interval is greater than 4 hours. In such a situation, greater radiotherapeutic efficacy is expected with the use of two or three treatment sessions per day with an essentially normal dose per fraction, total dose levels, and intervals of at least 5 hours between fractions.

In a clinical study, we compared a qd schedule with bid and tid schedules in the same patients with multiple sites of metastatic carcinoma to evaluate the response of normal human skin to accelerated fractionation schedules using 200 kV deep x-rays.[41] The intertreatment interval was kept at 4 hours. This study showed that the bid or tid dose schedules required an 8 to 10% decrease in the total dose of the qd schedule to yield the same level of acute skin response. In Burkitt's lymphoma, complete response was obtained in 74% (25 of 34) of patients with a hyperfractionation schedule (120 cGy tid) as compared with 11% (1 of 9) by a conventional dose schedule (220 cGy qd).[42]

In recent studies, Saunders and colleagues tested a concept of accelerated high-dose fractionated radiation administered within a short period of time (12 days) before accelerated tumor proliferation begins in standard qd radiation therapy.[43,44] Their pilot study, with a continuous, hyperfractionated, accelerated radiotherapy (CHART) regimen (54 Gy/36 fractions/12 days by 1.5 Gy/fraction, 3 treatments/day, 7 days/week), showed an encouraging result when it was compared with historical control at the same institution. The dose limiting toxicity of this CHART regimen was acute esophagitis. This led to a phase III trial in which the same CHART regimen, as stated above, was compared with a standard qd radiation schedule (60 Gy/30 fractions/42 days) in patients with unresectable nonsmall cell carcinoma of the lung (stages I to III). The 2-year survival rates were 29% by CHART as compared with 20% by standard qd radiation ($p = .006$).

Squamous cell carcinoma of the trachea has rapidly proliferating characteristics for which an accelerated fractionation regimen using a bid schedule, or a hybrid schedule of a qd mixed with a bid schedule, seems more logical than a conventional qd schedule. On the contrary, adenoid cystic carcinoma is a slowly growing tumor and a conventional qd schedule seems more appropriate than accelerated fractionation schedules.

Innovations in Improving Therapeutic Ratio

Three-Dimensional Conformal Radiation Therapy. Three-dimensional conformal radiation therapy is a new treatment method in radiation therapy. This 3-D treatment plan provides a DVH of the target tissue and normal organs in the thorax. With this new technology, it is feasible to optimize the radiation dose schedule and total dose to individual patients.[30,31] This new technology also offers an opportunity to study the maximum tolerated dose of radiation for the given size and length of tracheal carcinoma. With 3-D radiation therapy, one should attempt to administer a radiation dose of 70 Gy in 35 fractions over a period of 7 weeks to the tracheal tumor in the majority of patients with unresectable disease. It is anticipated that results of high-dose radiation therapy using a 3-D conformal radiation plan will become available over the next 5 to 10 years.

Endotracheal Brachytherapy. Endotracheal brachytherapy is a reasonable approach for tracheal carcinoma. However, this treatment may not be able to administer an adequate dose of radiation to the extramural tumor without causing an unacceptably high dose to the tracheal mucosa and cartilages. It is not feasible to administer a uniform dose of radiation throughout the large target volume. Therefore, it has been used as supplemental therapy for an additional dose of 8 to 15 Gy in 1 to 2 sessions, after 60 to 68 Gy of external beam radiation. Studies using the combined therapy of external beam radiation and endotracheal brachytherapy showed an improvement in local tumor control.[32,45] Further studies are necessary to determine the maximum and optimum dose of supplemental radiation by endotracheal brachytherapy after external radiation therapy. In such studies, an applicator that is capable of positioning the catheter to the center of the tracheobronchial lumen is desirable.[32]

Summary and Conclusion

Tracheal carcinoma is rare and the therapy outcome depends on the tumor extent and histological type. Survival rates beyond 5 years are 50 to 60% after resection for operable disease, and 5 to 20% after radiation therapy for unresectable or medically inoperable tumors. Adenoid cystic carcinoma has a much longer natural history than squamous cell carcinoma.

Surgery remains the choice of treatment for tracheal carcinoma. Postoperative radiation therapy should be considered for patients with either resection margins positive for residual carcinoma or inadequate margins of resection of 2 mm or less.

With the advent of 3-D conformal radiation therapy, it is feasible to quantitate the radiation dose administered to the tumor as well as the normal tissue or organs in the thorax by means of a DVH. Future studies should use 3-D conformal radiation therapy to determine the maximum tolerated dose of radiation as well as for the determination of the optimum dose for individual patients with unresectable or inoperable tracheal carcinomas.

Reference

1. Grillo HC. Tracheal tumors: diagnosis and management. In: Choi NC, Grillo HC, editors. Thoracic oncology. New York: Raven Press; 1983. p. 271–8.
2. Grillo HC, Mathisen DJ, Wain JC. Management of tumors of the trachea. Oncology 1992;6:61–7.
3. Mornex F, Coquard R, Danhier S, et al. Role of radiation therapy in the treatment of primary tracheal carcinoma. Int J Radiat Oncol Biol Phys 1998;41:299–305.
4. Mark EJ. Pathology of thoracic neoplasms. In: Choi NC, Grillo HC, editors. Thoracic oncology. New York: Raven Press; 1983. p. 257–69.
5. Karnofsky DA, Golbsy RB, Pool JL. Preliminary studies on the natural history of lung cancer. Radiology 1957;69:477–87.
6. Stanley KE. Prognostic factors for survival in patients with inoperable lung cancer. J Natl Cancer Inst 1980;65:25–32.
7. Pearson FG, Todd TR, Cooper JD. Experience with primary neoplasms of the trachea and carina. J Thorac Cardiovasc Surg 1984;88:511–8.
8. Grillo HC, Mathisen DJ. Primary tracheal tumors: treatment and results. Ann Thorac Surg 1990;49:69–77.
9. Regnard JF, Fourquier P, Levasseur P. Results and prognostic factors in resections of primary tracheal tumors: a

multicenter retrospective study. The French Society of Cardiovascular Surgery. J Thorac Cardiovasc Surg 1996;111:808–13.
10. Choi NC, Grillo HC, Gardiello M, et al. Basis for new strategies in postoperative radiotherapy of bronchogenic carcinoma. Int J Radiat Oncol Biol Phys 1980;6:31–5.
11. Lung Cancer Study Group. Effects of postoperative mediastinal radiation on completely resected stage II and stage III epidermoid cancer of the lung. N Engl J Med 1986;315:1377–81.
12. Ogino T, Ono R, Shimizu W, Ikeda H. Adenoidcystic carcinoma of the tracheobronchial system: the role of postoperative radiotherapy. Radiat Med 1995;13:27–9.
13. Azar T, Abdul-Karim FW, Tucker HM. Adenoidcystic carcinoma of the trachea. Laryngoscope 1998;108: 1297–300.
14. Douglas JG, Laramore GE, Austin-Seymour M, et al. Treatment of locally advanced adenoidcystic carcinoma of the head and neck with neutron radiotherapy. Int J Radiat Oncol Biol Phys 2000;46:551–7.
15. Choi NC, Doucette JA. Improved survival of patients with unresectable non-small cell bronchogenic carcinoma by an innovated high-dose en-bloc radiotherapeutic approach. Cancer 1981;48:101–9.
16. Perez CA, Stanley K, Grundy G, et al. Impact of irradiation technique and tumor extent in tumor control and survival of patients with unresectable non-oat cell carcinoma of the lung. Cancer 1982;50:1091–9.
17. Green N, Kulber H, Landman M, Pierce M. The experience with definitive irradiation of clinically limited squamous cell cancer of the trachea. Int J Radiat Oncol Biol Phys 1985;11:1401–5.
18. Cheung, AY. Radiotherapy for primary carcinoma of the trachea. Radiother Oncol 1989;14:279–85.
19. Fields JN, Rigaud G, Emami BN. Primary tumors of the trachea. Results of radiation therapy. Cancer 1989;63: 2429–33.
20. Manninen MP, Pukander JS, Flander MK, et al. Treatment of primary tracheal carcinoma in Finland in 1967–1985. Acta Oncol 1993;32:277–82.
21. Chow DC, Komaki R, Libshitz HI, et al. Treatment of primary neoplasms of the trachea. The role of radiation therapy. Cancer 1993;71:2946–52.
22. Schraube P, Latz D, Wannenmacher M. Treatment of primary squamous cell carcinoma of the trachea: the role of radiation therapy. Radiother Oncol 1994;33:254–8.
23. Jeremic B, Shibamoto Y, Acimovic L, Milisavljevic S. Radiotherapy for primary squamous cell carcinoma of the trachea. Radiother Oncol 1996;41:135–8.
24. Makarewicz R, Mross M. Radiation therapy alone in the treatment of tumours of the trachea. Lung Cancer 1998;20:169–74.
25. Chao MW, Smith JG, Laidlaw C, et al. Results of treating primary tumors of the trachea with radiotherapy. Int J Radiat Oncol Biol Phys 1998;41:779–85.
26. Muller A, Stockamp B, Schnabel T. Successful primary radiation therapy of adenoid cystic carcinoma of the lung. Oncology 2000;58:15–7.
27. Huber PE, Debus J, Latz D, et al. Radiotherapy for advanced adenoid cystic carcinoma: neutron, photons or mixed beam? Radiother Oncol 2001;59:161–7.
28. Line D, Deeley TJ. Palliative therapy. In: Deeley TJ, editor. Modern radiotherapy—carcinoma of the bronchus. New York (NY): Appleton-CenturyCrofts; 1971. p. 298–306.
29. Schulz MD. Palliation by radiotherapy in bronchogenic carcinoma. JAMA 1966;196:850.
30. Choi NC. Radiation therapy for carcinoma of the lung. In: Wang CC, editor. Clinical radiation oncology: indications, techniques, results. New York (NY): Wiley-Liss, John Wiley & Sons; 2000. p. 295–333.
31. International Commission on Radiation Units. Prescribing, recording and reporting photon beam therapy. Bethesda (MD): International Commission on Radiation Units and Measurements; 1993. Report No. 50.
32. Fritz P, Schraube P, Becker HD, et al. A new applicator, positionable to the center of tracheobronchial lumen for HDR-Ir-192-afterloading of tracheobronchial tumours. Int J Radiat Oncol Biol Phys 1991;20:1061–6.
33. Choi NC. Curative radiation therapy for unresectable non-small-cell carcinoma of the lung: indications, techniques, results. In: Choi NC, Grillo HC, editors. Thoracic oncology. New York: Raven Press; 1983. p. 163–200.
34. Dillman RO, Seagren SL, Propert KJ, et al. A randomized trial of induction chemotherapy plus high-dose radiation versus radiation alone in stage III non small cell lung cancer. N Engl J Med 1990;323:940–5.
35. Arriagada R, Le Chevalier T, Quoix E, et al. for the GETCB, the FNCLCC and the CEBI trialists. ASTRO plenary: effect of chemotherapy on locally advanced non-small cell lung carcinoma: a randomized study of 353 patients. Int J Radiat Oncol Biol Phys 1991;20:1183–90.
36. McGrath JJ, Choi NC, Mathisen DJ, et al. Role of radiation therapy in the management of tracheal tumors [abstract]. Int J Radiat Oncol Biol Phys 2001;51:344
37. Withers HR. Biologic basis for altered fractionation schemes. Cancer 1985;55:2086–95.
38. Thames HD, Peters LJ, Withers HR, et al. Accelerated fractionation vs. hyperfractionation: rationales for several treatments per day. Int J Radiat Oncol Biol Phys 1983;9:127–38.
39. Elkind MM, Sutton H. Radiation response of mammalian cells grown in culture. 1. Repair of x-ray damage in surviving Chinese hamster cells. Radiat Res 1960;13:556–93.
40. Suit HD, Urano M. Repair of sublethal radiation injury in hypoxic cells of a C3H mouse mammary carcinoma. Radiat Res 1969;37:423–34.
41. Choi CH, Suit HD. Evaluation of rapid radiation treatment schedules utilizing two treatment sessions per day. Radiology 1975;116:703–7.
42. Noron T, Onyango I. Radiotherapy in Burkett's lymphoma: conventional vs. superfractionated regime—early results. Int J Radiat Oncol Biol Phys 1977;2:399–406.
43. Saunders MI, Dische S, Barrett A, et al. on behalf of the CHART Steering Committee. Randomised multicentre trials of CHART vs conventional radiotherapy in head and neck and non-small-cell lung cancer: an interim report. Br J Cancer 1996;73:1455–62.
44. Saunders MI, Dische S, Barrett A, et al. on behalf of the CHART Steering Committee. Continuous, hyperfractionated, accelerated radiotherapy (CHART) versus conventional radiotherapy in non-small-cell lung cancer: mature data from the randomised multicenter trial. Radiother Oncol 1999;52:137–48.
45. Harms W, Latz D, Becker H, et al. HDR-brachytherapy boost for residual tumour after external beam radiotherapy in patients with tracheal malignancies. Radiother Oncol 1999;52:251–5.

The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation

Hermes C. Grillo, MD

Anatomy and Mobilization of the Omentum

The omental flap has many uses in airway surgery. An omental flap based on a right gastroepiploic vascular pedicle is sufficiently long to reach any part of the airway. The omentum seems to possess unique abilities to enhance neovascularity, provide fibroplasia, and reestablish lymphatic drainage. Its bulk serves to fill space. It functions in the face of infection and helps to clear it. These functions have long been recognized.[1,2] In 1988, Mathisen and colleagues described the uses of the omentum in the management of complicated cardiothoracic problems.[3] This chapter deals only with its application to airway problems.

The greater omentum is supplied by branches from the right and left gastroepiploic arteries, which form variable arcades (Figure 42-1). Commonly, three major vessels branch more distally in the omentum to anastomose in inconstant vascular arcades.[4] The right gastroepiploic artery is almost always of good caliber and with good flow, except if previously divided. The left gastroepiploic artery is smaller and less constant, especially in its anastomosis with the right gastroepiploic artery. The right artery extends well to the left of the midpoint of the greater curvature of the stomach. The pulse in the right gastroepiploic artery should always be palpated. Although 5 to 13 arteries to the omentum originate from the right gastroepiploic artery, only 1 usually continues from the left.[2] It may or may not form an anastomotic arcade distally with other omental arteries. From the gastroepiploic arcade, multiple small branches flow to the greater curvature of the stomach. The short gastric arteries supply the greater curvature of the stomach above the left gastroepiploic artery. The veins parallel the arteries and empty into the portal system, most often via the superior mesenteric vein.

The omentum varies in length from 14 to 36 cm and in width from 23 to 46 cm.[5] In a few patients, the omentum may be turned upward into the chest through an appropriate entry point without any mobilization. This is in effect an omentopexy. In most cases, it is necessary to detach the omentum from the transverse colon and also laterally from attachments to other abdominal structures. If the lesser omental sac is partially or completely obliterated, separation from the transverse mesocolon must be done with

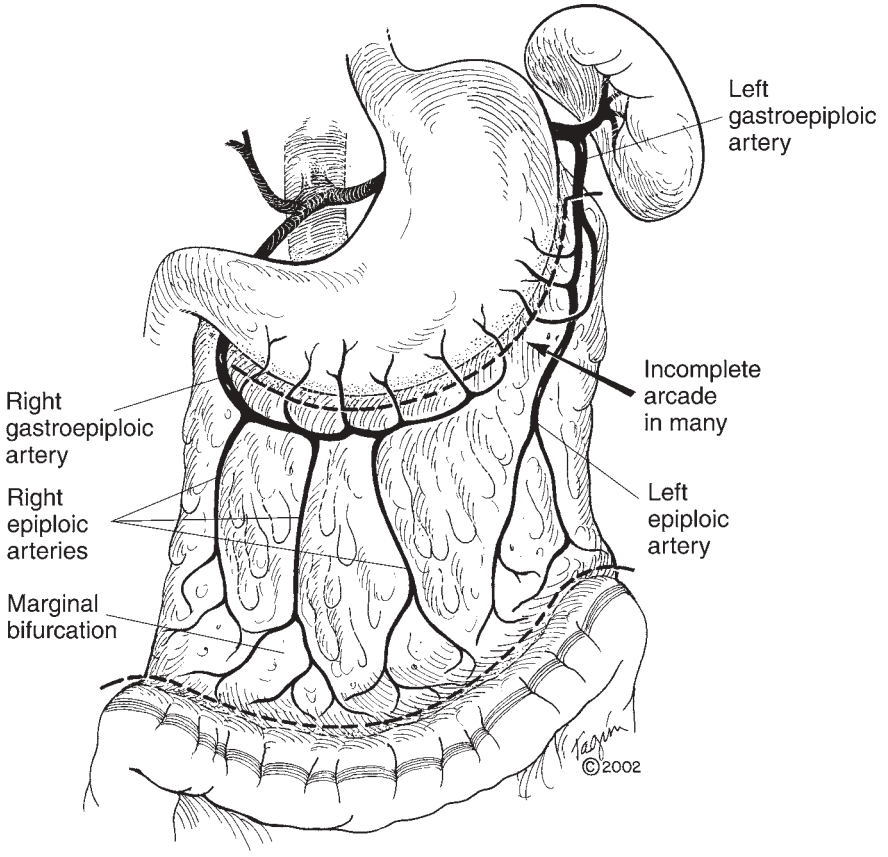


FIGURE **42-1** *Distribution of omental arteries and mobilization of the omentum. The gastroepiploic arcade is incomplete in about one-third of patients. The left gastroepiploic artery is inconstant in size and pattern. Distal epiploic arterial arcades are also extremely variable and may be incomplete. The* superior dashed line *indicates the line for omental mobilization with preservation of right gastroepiploic arterial supply. The* lower dashed line *represents the plane of separation of the omentum from the transverse colon. In many patients, this alone provides sufficient mobility for omental transfer.*

care to avoid injury to middle colic vessels. Detachment of the omentum from the transverse colon will provide sufficient length to reach nipple level in 75% of patients. The precise plane of dissection between the omentum and epiploic appendices of the colon is often unclear. The epiploic appendices should be left attached to the colon to minimize bleeding. The omental pedicle thus established often reaches the desired point in the thorax.

For further mobilization, I prefer to divide the left gastroepiploic artery and detach the gastroepiploic arch from the greater curvature of the stomach, carefully protecting the origin of the right gastroepiploic artery. The numerous short arteries to the stomach are individually ligated and divided one by one. With careful dissection, injury to the gastroepiploic arcade or to the stomach wall will not occur. When thus pedicled, the omentum will reach the base of the neck in 88% of patients. We have found that this degree of mobilization meets almost all the needs of the thoracic surgeon (Figure 42-2). A flap based on the left gastroepiploic artery is less dependable and provides little, if any, advantage. It is possible to obtain further length by dividing the arcade at one or more points to produce a longer pedicle.[4,5] In such a case, atraumatic vascular clamps are placed at the points of the anticipated division of vascular arcades, and the distal omentum is examined for viability after about 10 minutes. Such division permits the omentum to reach as high as the skull, but there is an attendant risk of partial loss of blood supply.

Omental mobilization is most often done through an upper midline abdominal incision from the tip of the xiphoid to the umbilicus. The timing of omental mobilization and the route of transposition to the chest vary with the intended use. In treating the airway, there is no indication to transfer the omentum sub-

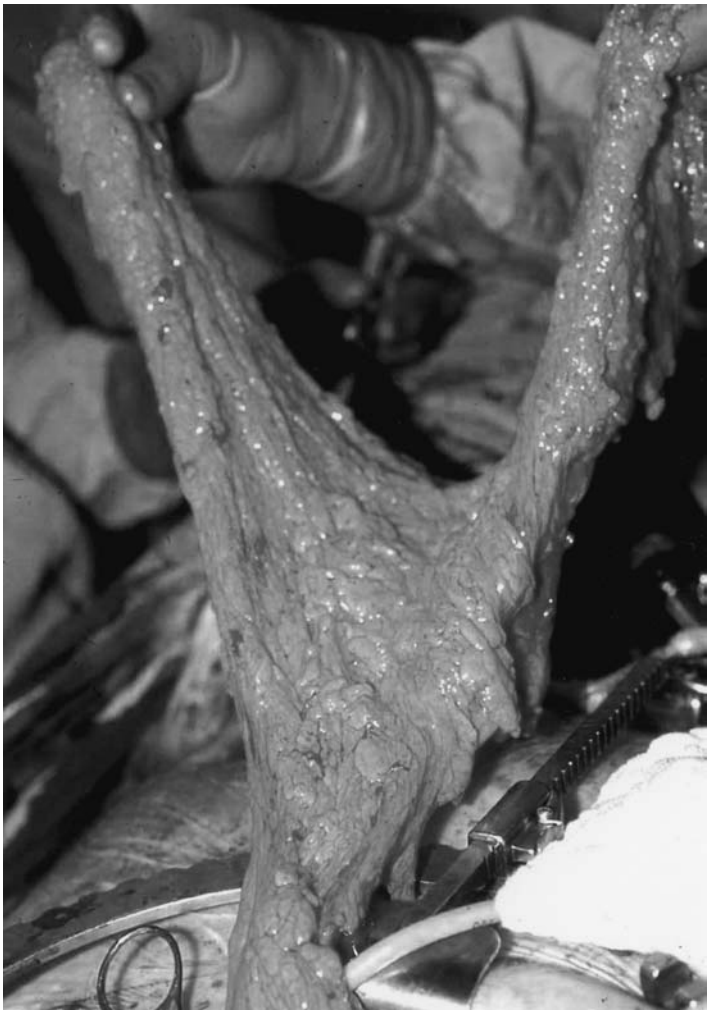


FIGURE **42-2** *Omentum mobilized from the colon and pedicled on the right gastroepiploic artery. The extent of mobility is apparent.*

cutaneously. The routes for transposition of the omentum into the chest are most commonly a substernal tunnel immediately behind the retrosternal fascia or through incisions made through the diaphragm on either the right or left side anteriorly just beyond the costal attachments (Figure 42-3). Lateral incisions must be adequate to permit the omentum to pass through without compression of blood supply, and at the same time, snug enough to prevent herniation of intestine. A few sutures between the diaphragm and the omentum protect against herniation.

If a decision is made in advance to use the omentum in a patient who will be operated upon by thoracotomy, I prefer first to mobilize the omentum through a midline abdominal incision, with the patient supine, and place the omentum in a substernal tunnel or pocket that has been created. The abdominal incision is closed and the patient positioned for thoracotomy. Mobilization should therefore be generous, most often using a right gastroepiploic artery pedicle. At thoracotomy, the mediastinal pleura is opened, and the omentum is drawn into the hemithorax when needed. With cervicomediastinal approach to the trachea, a change of position is of course not required. If it is not clear whether the airway lesion is resectable prior to exploration by thoracotomy, the patient is placed in a modified thoracoabdominal position and the abdominal incision is deferred. In this case, transfer of the omentum to the chest may sometimes be more easily done through a small anterior diaphragmatic incision.

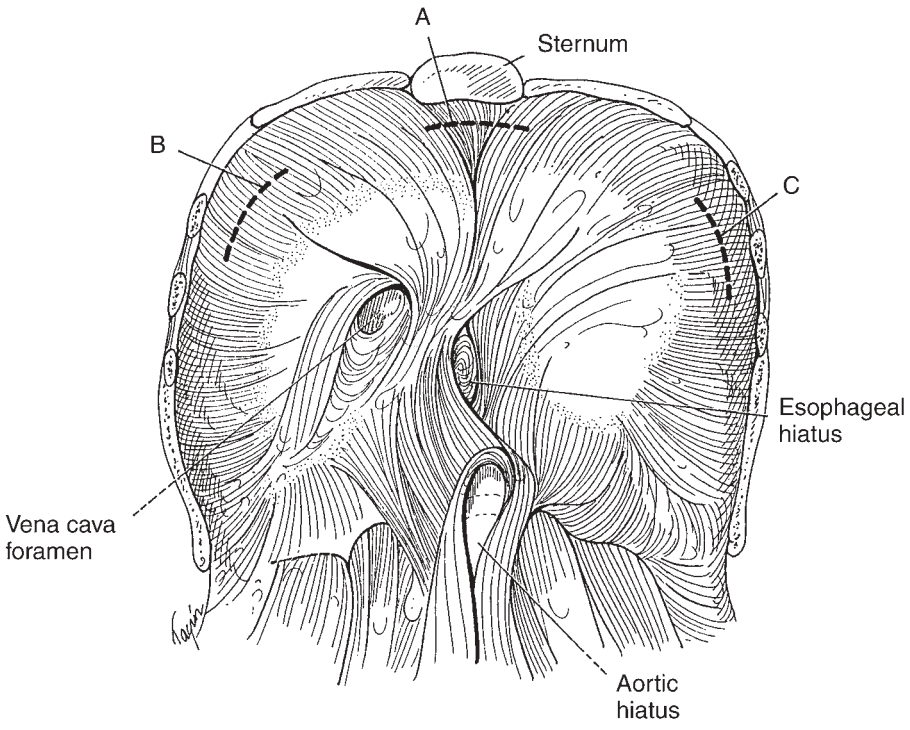


FIGURE **42-3** *Diaphragmatic apertures for transposition of omentum.* A *indicates the area of opening for substernal transfer.* B *and* C *indicate the regions of incisions for anterolateral passage.*

Airway Problems for Which the Omentum is Used

The omentum is useful to *prevent* airway problems in 1) cervicomediastinal exenteration with mediastinal tracheostomy, 2) tracheobronchial reconstruction following prior high-dosage irradiation to the airways, 3) high-risk bronchial closure, and 4) lung transplantation. It is also used for *treatment* of established airway problems including 1) bronchopleural fistula and 2) tracheal dehiscence. Since most of these problems are discussed elsewhere in this book, they will be noted only briefly here.

Cervicomediastinal Exenteration

When mediastinal tracheostomy is established close to the carina, even a partial separation of the trachea from the skin can lead to mediastinal sepsis and then to fatal hemorrhage from the brachiocephalic artery or aortic arch (see Chapter 34, "Cervicomediastinal Exenteration and Mediastinal Tracheostomy"). Prior irradiation increases the danger. Elective division of the brachiocephalic artery plus use of the omentum to surround the trachea beneath the cutaneous anastomosis and cover the large vessels prevents these disasters. A tongue of omentum is also placed over the pharyngoesophageal closure or pharyngoenteric anastomosis. The omentum is advanced directly from the upper abdominal incision through a substernal tunnel.[6]

Of 8 patients who underwent radical operations of this type with omental support, 4 had received a prior high-dose irradiation, 1 was irradiated postoperatively, and 2 suffered problems related to ^{131}I treatment. Colon bypasses were used for reconstruction in 5 patients, the stomach in 1, and esophagoplasty in another. One patient did not require esophageal resection. None of the patients developed either esophageal fistula or vascular erosion. One patient experienced a complete separation of the trachea from the skin and another experienced a partial separation. Healthy granulations arose from the underlying omentum, and the vessels remained protected while healing proceeded satisfactorily.

Prevention of Bronchopleural Fistula

Patients at increased risk for bronchopleural fistula, particularly after a right pneumonectomy, include 1) those with prior mediastinal irradiation, 2) those who require a radical mediastinal lymph node dissection, which can devascularize the exposed right main bronchial stump, 3) those with intrathoracic infections such as aspergillosis, or 4) those requiring a pneumonectomy due to pulmonary destruction from tuberculosis. The bronchial stump should be specially reinforced after meticulous suture closure. A variety of tissues are satisfactory. These include a bulky and well-vascularized pedicled pericardial fat pad, a pedicled intercostal muscle flap, or pedicled omentum. All must be fixed with multiple sutures to provide a second layer closure and not simply "tacked" into place. Chest wall muscular pedicles are preferred by some surgeons. The omentum has been elected 1) where a bulkier flap was felt to be useful, 2) where prior chest wall surgery, such as a preceding thoracoplasty, militated against the use of intercostal muscle, or 3) where the chest wall had been irradiated. Results have been excellent.[7]

It is not the purpose of this chapter to discuss detailed management of the underlying problems in a pneumonectomy; however, if the pleural space is heavily contaminated during or preceding a pneumonectomy, it will remain so even after surgical débridement of infected tissue, both parenchymal and parietal. In these cases, two chest catheters have been left in place; a small one superiorly and a larger one posteriorly and inferiorly. After 48 hours to permit sealing of the tissue planes of the applied flap, the patient is started on a 1-week program of antibiotic irrigation of the cavity, prior to removal of chest tubes. This might be termed a "preemptive" Clagett procedure.

Lung Transplantation

The problem of bronchial blood supply in lung transplantation is discussed in Chapter 44, "Airway Management in Lung Transplantation." In an effort to minimize anastomotic devascularization problems and their sequelae, which were seen so often after lung transplantation, Cooper advocated wrapping the anastomosis with omentum.[8] Experiments demonstrated the survival of a completely detached segment of main bronchus wrapped with omentum.[9] A network of fine vessels originating from the omentum appeared in 4 days. As long as healing of anastomoses following lung transplantation continues to present problems, the omental adjunct will likely remain useful, at least in certain instances.[7]

Completely free transplantation of the trachea and even of the larynx, surrounded by an omental wrap, has been proposed experimentally by a number of investigators. It has not been generally successful (see Chapter 45, "Tracheal Replacement").

Closure of a Bronchopleural Fistula

Effective treatment of a chronic bronchopleural fistula after pneumonectomy, or after lobectomy with empyema and destroyed residual lung, requires three steps. First, the empyema must be widely and completely drained. Second, the fistula must be permanently closed. Third, the residual pleural space must be cleared of infection and closed, completely filled, or obliterated. Techniques for closure of a bronchopleural fistula are many and are variably applicable to individual cases. This chapter seeks only to review the use of the omentum for this problem. Detailed consideration of bronchopleural fistula is given in Chapter 43, "Postpneumonectomy Bronchopleural Fistula."

Virkkula and Ecrola described the use of the omentum for closure of fistulae.[10] We have used the omentum to close fistulae in patients in whom the original disease was lung neoplasm—a number with prior irradiation, chronic infection, and trauma.[11] Some had undergone a lobectomy with subsequent destruction of the remaining lung. Several patients were previously treated unsuccessfully with chest wall muscle flaps. It is our conclusion that the omentum is more dependable than myoplastic flaps in difficult situations such as these.[7]

Adequate dependent drainage of the chronic empyema is first assured. At the time of definitive closure, the omentum is fully mobilized through an upper abdominal incision and placed in a retrosternal "pocket." The abdomen is closed and chest exposure is obtained. The bronchial stump is dissected, any excess is resected, and the stump is then closed with 4-0 Vicryl sutures.

The mediastinal pleura is opened and the omentum extracted. The massively thickened pleura may make location of the "pocket" difficult. The omentum is sutured over the closed stump in at least two layers, folding it on itself. In patients in whom the fistula was completely flush with the carina, the omentum was initially sutured to the margins of the opening, in the manner of closure of a large perforation due to duodenal ulcer. Postoperative bronchoscopy demonstrated the omentum to fill the fistula. Carinal resection seems an unwise alternative in this densely scarred, contaminated, and often irradiated field.

Most commonly, the hemithorax was allowed to remain widely drained for a later Clagett procedure when healing was firm and the cavity was clean. In some cases, a small residual pleural space was filled with the bulky omentum or with the adjunct of a pedicled chest wall muscle flap, permitting closure with a draining chest tube.

A patient with an unsuturable fistula flush with the trachea, which is plugged with an omentum, may leak briefly several days after operation, but spontaneous closure should follow as granulation tissue proliferates. One failure of omental reclosure after multiple prior attempts at closure occurred, but residual carcinoma was discovered in the stump.

Tracheal Dehiscence

Resuture may not be possible in the very rare occurrence of frank separation early after a tracheal anastomosis. Management may be attempted by a T tube or an endotracheal tube (if the patient requires ventilation) that spans the area of separation. If the innominate artery or aortic arch is adjacent, it seems judicious to wrap the dehisced airway and its splinting tube with the omentum, interposing it between airway and vessels.

Fortunately, the problem very rarely occurs and experience is therefore minimal. A patient who had massive irradiation for squamous cell carcinoma of the upper trachea prior to transfer to this hospital was found at resection to have an abscess adjacent to the necrosing tumor. The anastomosis following resection initially appeared satisfactory. Subsequent localized necrosis and leakage in the lateral wall of the trachea at the anastomosis was managed by débridement, omental wrapping, and a splinting T tube. The airway remained functional for 6 months until death, which occurred from multiple pulmonary metastases from a highly aggressive tumor.

Reconstruction after Irradiation

Airway injury from irradiation is encountered in two forms. The first is stenosis, and the second is loss of healing capacity following reconstruction.

Postirradiation Stenosis

Tracheal stenosis subsequent to irradiation is seen only occasionally. Our experience is therefore related anecdotally. Following external irradiation of the larynx and upper trachea for thyroid cancer in a young child, the larynx failed to develop to proper size and, years later, subglottic and upper tracheal stenosis became evident. In a small number of adults, high-dose external irradiation for thyroid and laryngeal carcinomas resulted, decades later, in severe subglottic and upper tracheal stenoses. In 3 additional patients with mediastinal Hodgkin's disease, severe stenosis of the midtrachea appeared a decade or more after apparently successful management, primarily by irradiation in doses in the range of 4,500 cGy. None of these patients had recurrence of neoplasm. In 1 patient who was explored, massive fibrosis was found, encasing the area of tracheal stenosis, presumably residual scar from the destroyed lymphoma.

Another patient suffered destruction and stenosis of the larynx, the trachea, and the upper esophagus following treatment of a stubborn Graves' disease by administration of an excessive dose of radioactive iodine (RAI) (Figure 42-4). Inappropriate use of brachytherapy in the left main bronchus to deliver irradiation to the carinal area, after right pneumonectomy for adenoid cystic carcinoma, produced an obliterative stenosis of the bronchus, as might be expected.

These patients have, in general, been managed as conservatively as possible. Two adults with upper laryngotracheal stenoses were handled by dilation at intervals. Fortunately, they did not require further intervention. There appeared to be no surgical route to correct the lesions because of their disposition and location,

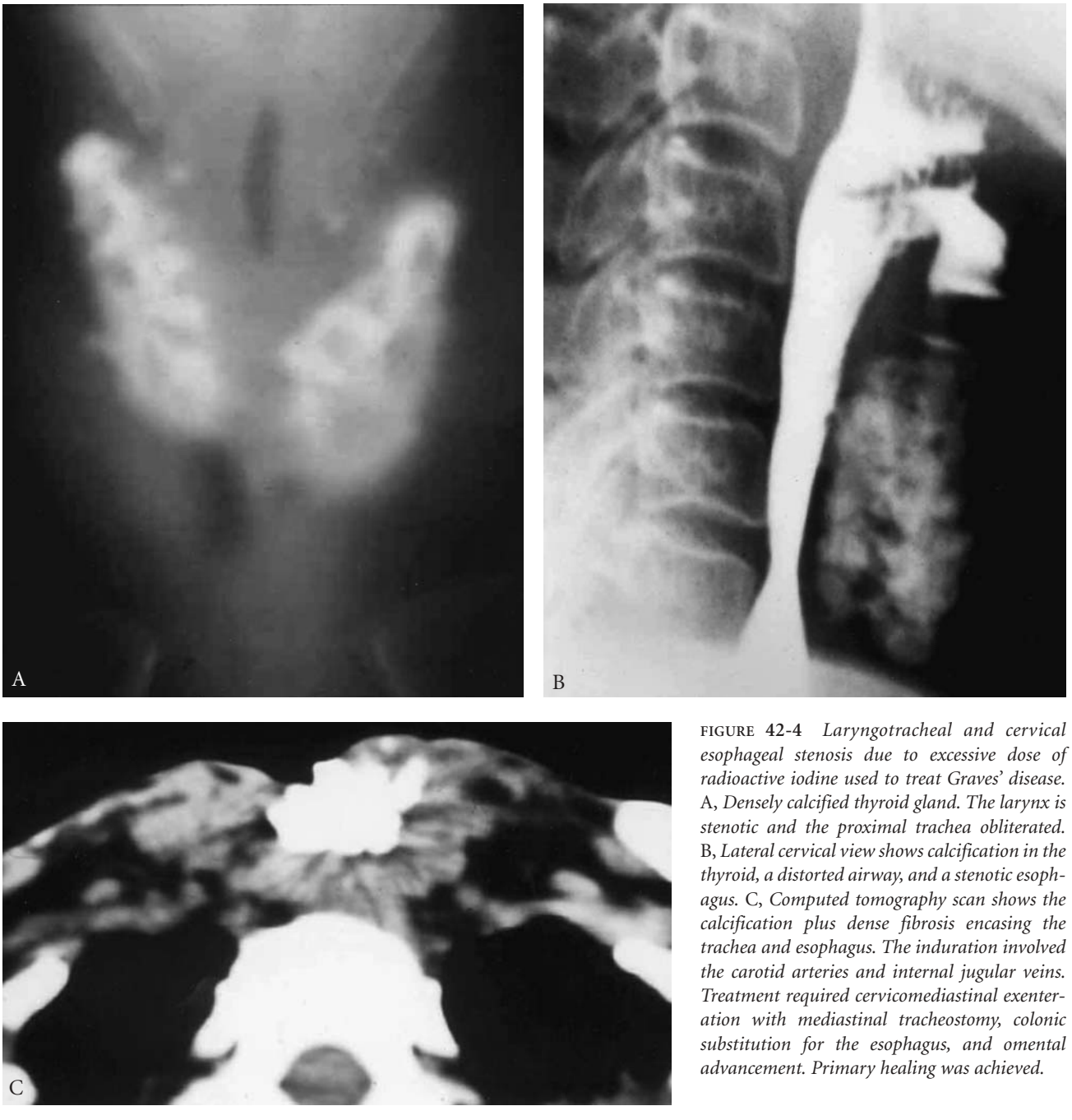


FIGURE **42-4** *Laryngotracheal and cervical esophageal stenosis due to excessive dose of radioactive iodine used to treat Graves' disease.* A, *Densely calcified thyroid gland. The larynx is stenotic and the proximal trachea obliterated.* B, *Lateral cervical view shows calcification in the thyroid, a distorted airway, and a stenotic esophagus.* C, *Computed tomography scan shows the calcification plus dense fibrosis encasing the trachea and esophagus. The induration involved the carotid arteries and internal jugular veins. Treatment required cervicomediastinal exenteration with mediastinal tracheostomy, colonic substitution for the esophagus, and omental advancement. Primary healing was achieved.*

even if healing could have been obtained. A tracheal "button" distal to the stenosis was advised in the patient who underwent irradiation in childhood, with subsequent severe laryngeal and upper tracheal stenosis.

The patient with destruction of laryngotracheoesophageal structures by RAI had a large inflammatory mass, rigid cervical fibrosis, and multiple fistulae. She suffered from bleeding from a tracheostomy. Cervicomediastinal exenteration of the entire process, with establishment of mediastinal tracheostomy protected with the omentum, and restitution of swallowing with a colon bypass, produced a satisfactory result. The left main bronchial stenosis due to brachytherapy proved undilatable and irresectable.

Our first patient with a midtracheal stenosis and severe surrounding fibrosis due to prior treatment of Hodgkin's disease was managed by excision and end-to-end anastomosis with omental wrapping. Failure of healing led to separation, and death occurred from hemorrhage from the brachiocephalic artery. Subsequent patients were managed by dilation and placement of a T tube, with satisfactory results.

Postirradiation Inhibition of Healing after Reconstruction

Damage to the healing potential of tissues as a result of significant doses of irradiation has long been recognized. Factors that impair wound repair are increased dose of irradiation and a prolonged interval between irradiation and a surgical procedure. Traditionally, the surgeon's anxiety increases when the irradiation dose exceeds 4,500 cGy and when irradiation has been given longer than a year prior to operation. Whereas excisional surgery, such as lymph node dissection or lobectomy, may be accomplished with only moderate increase in complications of wound healing, anastomoses of the airway or intestine are likely to dehisce, with disastrous consequences. The ability of irradiated tissues to respond to injury with normal capillary budding and fibroblastic proliferation is minimized or lost. Because of hyalinizing changes in small vessels, necrosis may occur once the integrity of the organ is disturbed by surgery (Figure 42-5).

Failure of healing following anastomosis of the irradiated intestine has long been noted. A similar wariness of prior irradiation exists in resectional surgery of the lung, although the concern is not uniformly accepted. Experimental studies in dogs by Tsubota and colleagues demonstrated detrimental effects of irradiation on tracheal anastomoses.[12] When more than 3,500 cGy of irradiation was delivered over a 3-week period prior to resection and reconstruction, the incidence of subsequent tracheal stenosis increased proportionally with increasing dose of irradiation. This was associated microscopically with more edema, inflammation, and fibrosis. Grillo and Potsaid, and Ross, demonstrated inhibition by irradiation of capillary proliferation and of local appearance of a fibroblast population in experimental wounds.[13,14] These are key components in the reparative response that normally follows injury and which finally serves to unite the anastomosis. The late histological effects following irradiation include hyalinization of arterioles, which then lose the ability to respond with a reparative response after injury.

Clinical Experience

In 1967, I saw a 54-year-old patient who 6 years earlier had undergone a thyroid lobectomy and radical neck dissection for differentiated carcinoma of the thyroid, and who subsequently received 4,800 cGy of radiotherapy because of invasion of the tracheal wall by tumor. She presented with severe airway obstruction due to recurrent cancer. The invaded tracheal segment was resected and an end-to-end anastomosis was performed. The trachea failed to heal, turned grayish-green, and necrosed, and the patient ultimately died from brachiocephalic artery hemorrhage. It appeared judicious to withhold tracheal reconstructive surgery in patients who had had a significant dose of irradiation given remotely.

This rule was later broken for an 8-year-old boy who had undergone very high-dose radiotherapy (3,728 and 5,040 cGy), 93 and 67 months earlier, for rhabdomyosarcoma in the neck. A short segment of the cervical trachea obstructed by a recurrent nodule of tumor was resected, and the anastomosis was buttressed with a substernally pedicled omental flap. Healing was satisfactory, except for a minor air leak at the fifth post-

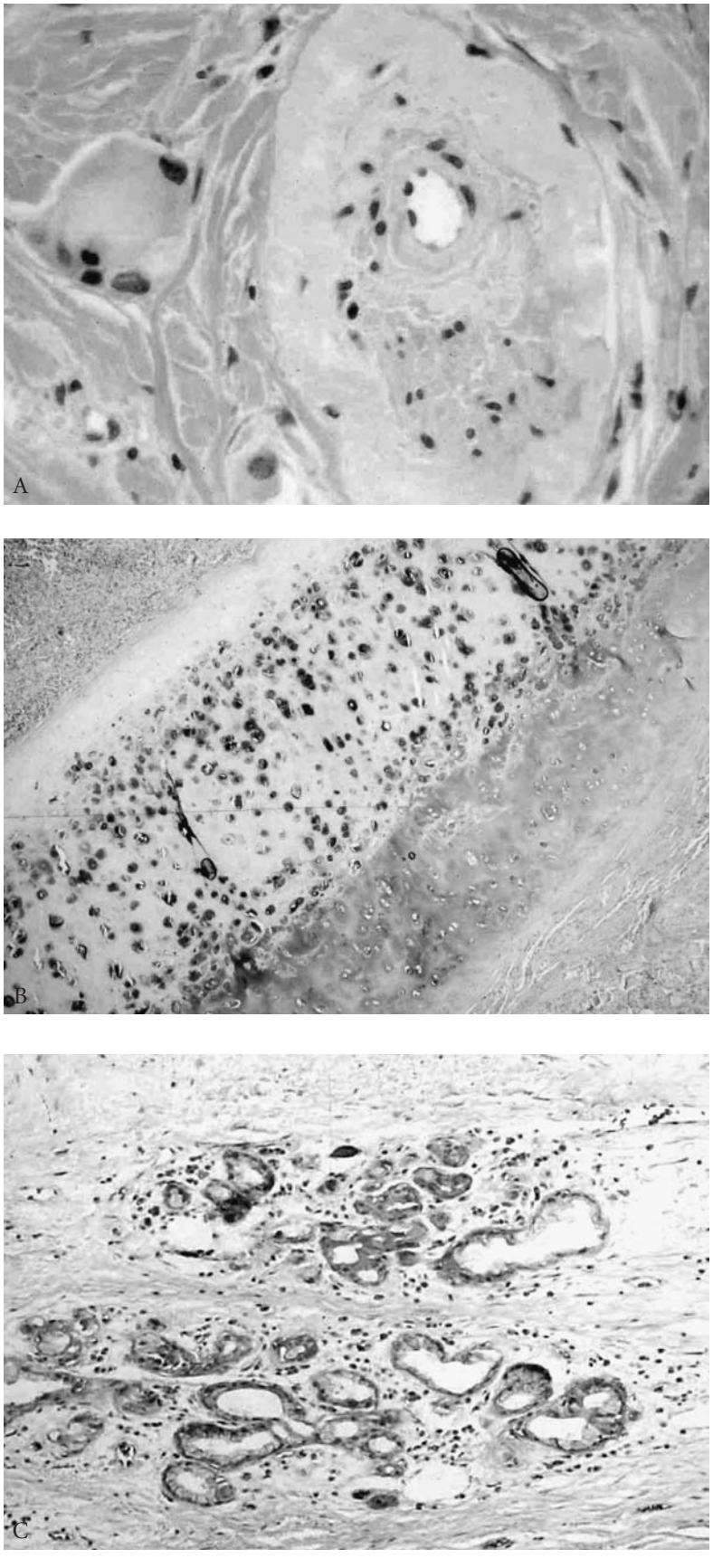


FIGURE **42-5** *Histological damage to the trachea due to irradition. A 76-year-old female underwent subtotal thyroidectomy for invasive papillary carcinoma of the thyroid and subsequently received irradiation to a total dose of 6,080 cGy to the tumor bed. Four years later, she manifested laryngotracheal invasion, with hemoptysis. Laryngotracheal resection was performed with substernal omental advancement. A temporary tracheostomy was necessary. The anastomosis healed slowly, but well.* A, *Photomicrograph of an irradiated small artery, showing characteristic hyalinization of the wall, intimal hyperplasia, and small lumen.* B, *Degeneration of cartilage. The hyaline cartilage at the left shows typical grouped chondrocytes. The fibrocartilage at the right is also abnormal.* C, *Atrophic tracheal glands.*

operative day. This healed with a tiny granuloma at the suture line, which was managed bronchoscopically.

Between 1979 and 1992, 22 consecutive patients underwent major airway resection and reconstruction following significant irradiation.[15] Excluded were patients who underwent resectional surgery only (ie, pulmonary resection or exenteration) without anastomoses, since these are lesser albeit still major problems. The average dose of irradiation received by these patients was 4,979 ± 1,112 cGy (range 3,150 to 7,768 cGy) in between 20 to 44 fractions, with an average fractional dose of 150 to 200 cGy. The time interval between irradiation therapy and surgery was 42.6 ± 105 months (range 1 to 488 months).

Patients fell into four categories: 1) symptomatic postirradiation tracheal stenosis, 2) recurrent tracheal tumor after irradiation either primary or adjunctive, 3) recurrent extratracheal tumor invading the trachea, and 4) preoperative radiotherapy. Six patients had postirradiation tracheal stenosis, causing dyspnea at rest. Three had been irradiated for positive margins after resection of adenoid cystic carcinoma of the trachea, 1 had received mediastinal irradiation for stage IA Hodgkin's lymphoma 13 years before, and a fifth developed stenosis following radiation for cervical tuberculosis as a child 40 years earlier. A sixth patient had been irradiated for recurrent thyroid cancer. Ten patients had recurrent primary tracheal tumors after prior radiation therapy. In 5 patients, the therapy had failed as primary treatment. Two had received radiotherapy for recurrence after resections for primary squamous cell carcinoma. Others included squamous cancer in an area of radiation for a primary lingual cancer, and another had been radiated following two prior tracheal resections for squamous cell carcinoma and now had yet another. There were 3 patients who had had a previously resected extratracheal tumor, with recurrence that now invaded the trachea. Three additional patients had preoperative irradiation and chemotherapy shortly before tracheal resection as part of a protocol extended to stage IIIB lesions.

Six patients underwent resection for benign stricture, 4 of these resulting from prior tracheal resections, whereas the other 2 had received radiation many years before but had not undergone surgery. Ten of the patients had primary tracheal cancer, 5 of who were referred because of failure of irradiation and laser therapy. The diagnoses of the patients who had extratracheal tumors invading the airway were rhabdomyosarcoma, laryngeal squamous cell carcinoma, and carcinoma of the lung.

It was planned to buttress the anastomosis with a vascularized and unirradiated tissue flap if the patient had received more than 4,500 cGy of irradiation or if irradiation therapy had been completed more than 12 months prior to surgery. This was intended to provide a healthy source of regenerating tissue for healing of the anastomosis and also to provide a sealing layer and tissue interposition between the suture line and major blood vessels. In 19 of the patients, the anastomoses were wrapped with a vascularized tissue flap. In 15 patients, the airway was buttressed with a pedicled omental flap, our first choice for this type of support. In 2 patients, in whom the omentum was unavailable due to a prior gastrectomy, a pericardial fat pad flap was accepted in 1 and a pedicled intercostal muscle flap in the second whose pericardial fat pad was insubstantial. In 3 additional cases, the sternohyoid muscle was interposed between the anastomosis and the brachiocephalic artery in 1 patient, the sternohyoid muscle was placed between the tracheal anastomosis and the esophagus in the second patient, and no flap was used in the third patient who had received only 4,200 cGy of irradtiation 12 months prior to operation. Hilar releases and suprahyoid laryngeal releases were used freely as indicated (32%). Fifteen of the 22 patients had undergone prior surgery in the area of resection. Tracheal resection and reconstruction was accomplished in 20 patients and mainstem sleeve resections in 2. The cervical trachea was resected in 7 cases, the midtrachea in 8, and the carina or distal trachea in 5.

One patient died following development of respiratory distress syndrome after a right carinal pneumonectomy. The patient with late stenosis due to Hodgkin's lymphoma died after his anastomosis had separated and innominate artery hemorrhage ensued. Although no active lymphoma was detected, the trachea was encased in massive scar, presumed to have resulted from regression of his original pathology. The qual-

ity of tracheal wall used for the anastomosis was unsatisfactory. This dehiscence occurred despite an omental wrap. Two subsequent patients with the same pathology were treated preferentially with T tubes.

One of the surviving patients, in whom a paratracheal abscess was encountered at resection of a necrotic tumor, suffered a dehiscence of the anastomosis, which was treated with placement of a T tube and an omental wrap. Two other patients developed wound infection, with granulations at the anastomotic site occurring in 1 of them. One patient, who had stenosis induced by primary irradiation, suffered a recurrent stenosis that required continued dilations. Seventeen of the 19 survivors had excellent results with no evidence of exertional dyspnea. The other 2 patients had dyspnea with moderate exercise.

Two subsequent patients, who underwent upper tracheal resection for squamous tracheal carcinoma after full-dose irradiation years previously for carcinoma of the base of the tongue and of a vocal cord, and who were buttressed with omentum, developed late (about 2 weeks after resection) limited necrosis of a portion of the anastomosis. Both were managed with T tubes. One of them healed slowly, with full epithelization. However, a partial stenosis occurred at the anastomosis and this was treated with an expandable uncoated stent, apparently successfully. The other patient is under treatment and may evolve similarly.

Shrager and colleagues recently described 14 patients who underwent prophylactic omental wrapping of high-risk tracheobronchial anastomoses.[7] These included 4 carinal pneumonectomies and 10 tracheal reconstructions, a number of which had received prior irradiation. Primary healing was attained in 12 patients. One, previously noted here, had been remotely irradiated for Hodgkin's lymphoma. Omentum was also used in 20 lung transplantation patients with 19 successes, in 7 cervicomediastinal exenterations with success, and in 6 pneumonectomy patients thought to present special risk (irradiation in 4, immunosuppression in 3, and infection in 3). Four of the pneumonectomy patients healed without complication. The overall success in these patients was 89%. Thirteen of 15 patients with complex postpneumonectomy bronchopleural fistulae were successfully closed with omental transposition and reclosure of a bronchial stump.

With careful surgical technique and avoidance of excessive tension on tracheal and bronchial anastomoses and of interruption in blood supply, separation or stenoses are relatively uncommon. Massive necrosis, such as that described in our earliest patient, has not subsequently been seen. With the known effects of irradiation on tissue healing, it becomes important to approach the patients in whom this factor is present with great caution. The limited experience described indicates that airway reconstruction can be performed fairly safely in irradiated patients, despite the hindrance to healing that radiation causes. The use of vascularized tissue flaps and preferably the omentum to enhance blood supply and fibroplasia is always advised. In this group of patients, there is unquestionably a greater likelihood of anastomotic problems, including localized necrosis and subsequent evolution to stenosis or malacia. Ultimately, the final resort in such patients may be to use a permanent T tube or other type of stent.

Reference

1. Goldsmith HS. The omentum: research and clinical applications. New York: Springer-Verlag; 1990.
2. Liebermann-Meffert D, White H. The greater omentum. New York: Springer-Verlag; 1983.
3. Mathisen DJ, Grillo HC, Vlahakes GJ, Daggett WM. The omentum in the management of complicated cardiothoracic problems. J Thorac Cardiovasc Surg 1988; 95:677–84.
4. Alday ES, Goldsmith HS. Surgical technique for omental lengthening based on arterial anatomy. Surg Gynecol Obstet 1972;135:103–7.
5. Das SK. The size of the human omentum and methods of lengthening it for transplantation. Br J Plastic Surg 1976;29:170–4.
6. Grillo HC, Mathisen DJ. Cervical exenteration. Ann Thorac Surg 1990;49:401–9.
7. Shrager JB, Wain JC, Wright CD, et al. Omentum is highly effective in the management of complex cardiothoracic surgical problems. J Thorac Cardiovasc Surg 2003;125:526–32.
8. Cooper JD. Lung transplantation. Ann Thorac Surg 1989;47:28–44.
9. Morgan E, Lima O, Goldberg M, et al. Successful revascularization of totally ischemic bronchial autografts with omental pedicle flaps in dogs. J Thorac Cardiovasc Surg 1982;84:204–10.
10. Virkkula L, Ecrola S. Use of omental pedicle for treatment of bronchial fistula after lower lobectomy. Scand J Thorac Cardiovasc Surg 1975;9:287–90.
11. Puskas JD, Mathisen DJ, Grillo HC, et al. Treatment strategies for bronchopleural fistula. J Thorac Cardiovasc Surg 1995;109:989–96.

12. Tsubota N, Simpson WJ, Van Nostrand AWP, Pearson FG. The effect of preoperative irradiation on primary tracheal anastomosis. Ann Thorac Surg 1975;20:152–60.
13. Grillo HC, Potsaid MS. Studies in wound healing. IV. Retardation of contraction by local x-irradiation and observations relating to the origin of fibroblasts in repair. Ann Surg 1961;154:741–50.
14. Ross R. The fibroblast and wound repair. Biol Rev 1968; 43:51–96.
15. Muehrcke DD, Grillo HC, Mathisen DJ. Reconstructive airway operation after irradiation. Ann Thorac Surg 1995;59:14–8.

Postpneumonectomy Bronchopleural Fistula

Cameron D. Wright, MD

Bronchopleural fistula following pneumonectomy remains a dreaded complication, despite advances in thoracic surgical care. Postpneumonectomy bronchopleural fistula is often associated with an empyema, although some early fistulae have a sterile hemithorax. Most bronchopleural fistulae are a result of a faulty mechanical closure of the main bronchial stump. An empyema may cause a fistula by necessitatising through the previously closed bronchus. Late bronchopleural fistulae are usually the result of an empyema acquired either by pleural soilage or hematogenous spread. Treatment of a bronchopleural fistula usually entails treatment of both the bronchopleural fistula and empyema. Despite appropriate treatment, mortality remains high in patients with postpneumonectomy bronchopleural fistula.

Incidence

The incidence of postpneumonectomy bronchopleural fistula varies in large series, ranging from 0 to 12% (Table 43-1).[1–9] Many factors probably contribute to this variation, such as the number of patients with infectious lung disease, induction treatment, and attention to detail in the perioperative period, but the major factor is likely to be the technical details in closing the main bronchus stump. In general, there appears to be a lower risk of postpneumonectomy bronchopleural fistula with sutured closure as opposed to stapled closure (see Table 43-1). An incidence of greater than 5% should give cause for concern to a surgeon or thoracic surgery unit, and prompt examination of the closure methods used.

Risk Factors

Many putative risk factors for postpneumonectomy bronchopleural fistula exist and seem to be evident (Table 43-2). Right pneumonectomy is thought to be a risk factor since the right bronchus projects uncovered into the hemithorax after pneumonectomy, whereas the left bronchus retreats deeper into the mediastinum under the aortic arch. Radiation given long before surgery seems to be more of a risk factor than radiation given immediately preoperatively. Radiation doses exceeding 50 Gy are associated with higher

Table 43-1 Bronchopleural Fistula after Pneumonectomy

Author	*Year*	*Number of Pneumonectomies*	*Incidence BPF %*	*Closure*	*Risk Factors*	*Mortality %*
Jack[1]	1965	450	0	Sutured	NA	NA
Sarsam and Moussali[2]	1989	332	0	Sutured	NA	NA
Vester et al[3]	1991	489	4.1	Stapled	NS	NS
Asamura et al[4]	1992	464	4.5	Sutured	Cancer, RT, DM	71
Al-Kattan et al[5]	1994	530	1.3	Sutured	Experience	29
Wright et al[6]	1996	256	3.1	Sutured	Postop ventilation, R side	25
Hollaus et al[7]	1997	797	12	Stapled	R side, Male	67
Hubaut et al[8]	1999	209	2.4	Sutured	Neoadjuvant therapy	40
de Perrot et al[9]	1999	67	9	Stapled	Postop ventilation, Cancer	43

BPF = bronchopleural fistula; DM = diabetes mellitus; NA = not applicable; NS = not stated; R = right; RT = radiation therapy.

rates of bronchopleural fistula in induction programs.[10] Many reported studies lack sufficient numbers to make meaningful statistical statements regarding risk factors. Reported risk factors by multivariate or univariate analyses include right pneumonectomy,[4,6,7] postoperative ventilation,[6,9] pleuropulmonary infection,[6] cancer in the stump,[4,9] radiation therapy,[4,8] diabetes mellitus,[4] experience of the surgeon,[5] and induction chemoradiotherapy.[8]

Prevention

The optimal approach to postpneumonectomy bronchopleural fistula is prevention. Patients with infections that might result in empyema should be optimally treated medically prior to pneumonectomy, including use of the appropriate antimicrobial therapy. Resection for tuberculosis requires prolonged preoperative antituberculosis therapy (generally at least 3 months) unless done for emergency indications. Excessive bronchial devascularization should be avoided by ligation of bronchial arteries close to the bronchial transection point rather than more proximal. Excessive use of cautery (as opposed to fine suture ligation) to control large bronchial arteries is to be avoided in order to minimize ischemic and necrotic tissue around the bronchus. Inexperienced surgeons often make the stump too long, creating a reservoir which can harbor infected secretions. Transection of the bronchus should be done by sharp division to minimize tissue trauma (avoiding electrocautery). Clamping the proximal bronchus risks injury to the microcirculation of the stump and seems wise to avoid. The mucosal point to be closed should be visually inspected to make sure it is free of cancer (by bronchoscopy if a stapler is used) and frozen section analysis should be performed if there is any suspicion of residual disease. The particular method of bronchial closure is very individual and controversy remains as to what method is best. No randomized trial data exist to allow statistically valid comparisons. Review of reported retrospective series (see Table 43-1) suggests that hand suturing leads to somewhat lower rates of bronchopleural fistula than stapled closure. In fact, the only two large series that report *no* bronchopleural fistulae are from an unusual tension-reducing hand-sewn method reported by Gordon Jack.[1,2] Jack's

Table 43-2 Possible Risk Factors for Postpneumonectomy Bronchopleural Fistula

Pleural Factors	Postoperative Factors
Empyema	Mechanical ventilation
Lung abscess	
Bronchiectasis	Adjuvant Factors
Tuberculosis	Radiation therapy
Fungal infection	Chemotherapy
	Bronchial artery embolization
Technical Factors	
Right pneumonectomy	Systemic Factors
Experience of surgeon	Diabetes mellitus
Bronchial devascularization	Chronic steroid therapy
Mediastinal lymph node dissection	Immunosuppression
Residual disease at bronchial mucosa	Malnutrition
Excessive stump length	Advanced age
Bronchial closure under tension	

method is shown in Figure 43-1 and is most applicable when a cancer encroaches close to the carina, making a stapled closure impossible and an ordinary sutured closure problematic. This technique aims to reduce tension on the suture line and thus reduce the mechanical stress that promotes failure of bronchial closure. Another way to reduce tension on the suture line is to cut the bronchus so that a flap of the membranous wall is left longer to rotate up to the divided cartilage (Figure 43-2). This technique was originally popularized by Lyman Brewer and colleagues[11] and is used in our unit. Closure of the evenly divided bronchus with the typical rigid cartilage ring invariably leads to tension and is probably one of the causes for the slightly higher rate of bronchopleural fistula with the stapler. Good results have been reported with permanent monofilament (Prolene) and braided absorbable (Vicryl) sutures. Nonabsorbable braided sutures (silk and polyester) are to be avoided as granulomas have been reported. Tying the sutures should be done very carefully as the membranous wall is easily torn (especially in women and patients on steroids), leading to very troublesome stump leaks. If a stapler is used (not my preference), it must be used just as carefully as if a hand-sutured closure is done. When closing a main bronchial stump, a 4.8 mm staple height should be used to avoid excessive crushing and resultant ischemia of the bronchial stump. One of the disadvantages of using a stapler is that not all stumps can be closed with a stapler, leading the surgeon to close a difficult stump without much experience or confidence in a sutured closure.

Vascularized tissue coverage of the closed main bronchial stump is probably an important component to reduce the incidence of bronchopleural fistulae, especially if risk factors (radiation, right pneumonectomy) exist. In 1942, Rienhoff and colleagues reported the first detailed study of bronchial healing after pneumonectomy (both experimentally in dogs and clinically in humans) and concluded that the bronchus healed by scar formation at the divided end of the bronchus.[12] In 82% of the experimental animals, the primary closure with sutures failed, and healing occurred by secondary intention aided by local mediastinal tissue at the cut end of the bronchus. This fact led Rienhoff to the concept of buttressing the closure (which he viewed as temporary only) with local tissue coverage of the stump, both experimentally and in humans. Smith and colleagues reported an experimental study of stapled main bronchial closures and reported primary healing of only 50% of all stapled bronchi.[13] Hence, whether the surgeon sutures or staples the bronchus, a fair proportion of bronchi will heal by secondary intention with the aide of local mediastinal tissues. Since there is little disadvantage to placing a flap over the bronchus, it seems to be a

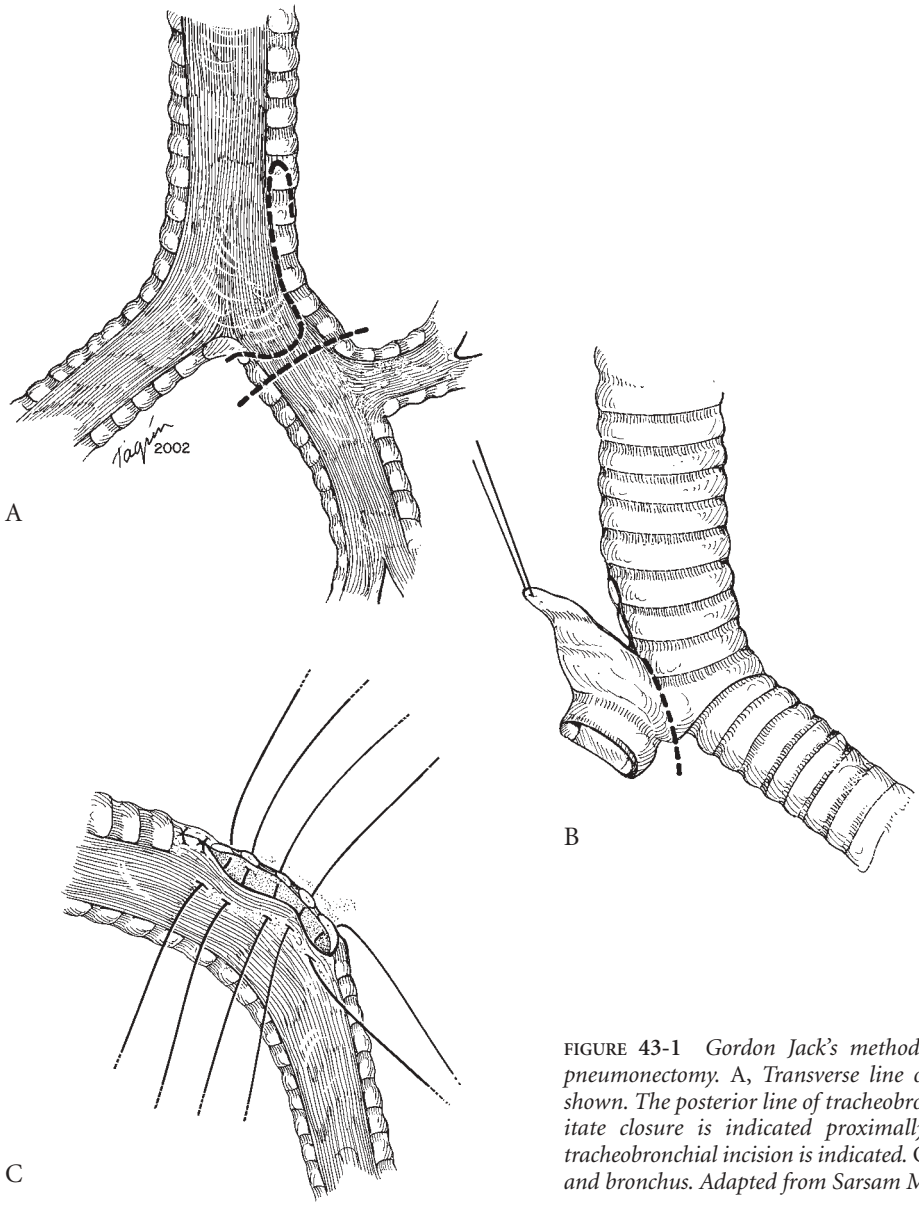


FIGURE **43-1** *Gordon Jack's method of closure after right pneumonectomy.* A, *Transverse line of bronchial division is shown. The posterior line of tracheobronchial division to facilitate closure is indicated proximally.* B, *Anterior line of tracheobronchial incision is indicated.* C, *Closure of the trachea and bronchus. Adapted from Sarsam MA and Moussali H.*[2]

prudent precaution. In 1953, Brewer and colleagues reported favorable results with the pericardial fat pad, both experimentally and clinically.[11] The incidence of bronchopleural fistula in humans was reduced from 8% to zero in a consecutive case series with routine use of this flap. Current options include pleura, local mediastinal tissue, pericardium, pericardial fat pad, intercostal muscle, pedicled diaphragm, chest wall muscles (serratus or latissimus), and the omentum.[14–17] Our preference is intercostal muscle or the pericardial fat pad for low-risk closures, and extrathoracic muscles or the omentum for high-risk closures. Pleura is usually too thin and flimsy to provide adequate coverage. The tissue flap should be carefully sutured around the circumference of the main bronchus to ensure adequate coverage.

Prolonged drainage of a sterile hemithorax after pneumonectomy is to be avoided since the chest tube can act as a site of ingress by skin bacteria, turning the site into a perfect culture medium of pleural fluid. When pleural contamination has occurred intraoperatively (such as a ruptured lung abscess), additional measures should be employed beyond culturing the pus, copious irrigation of the cavity, and pro-

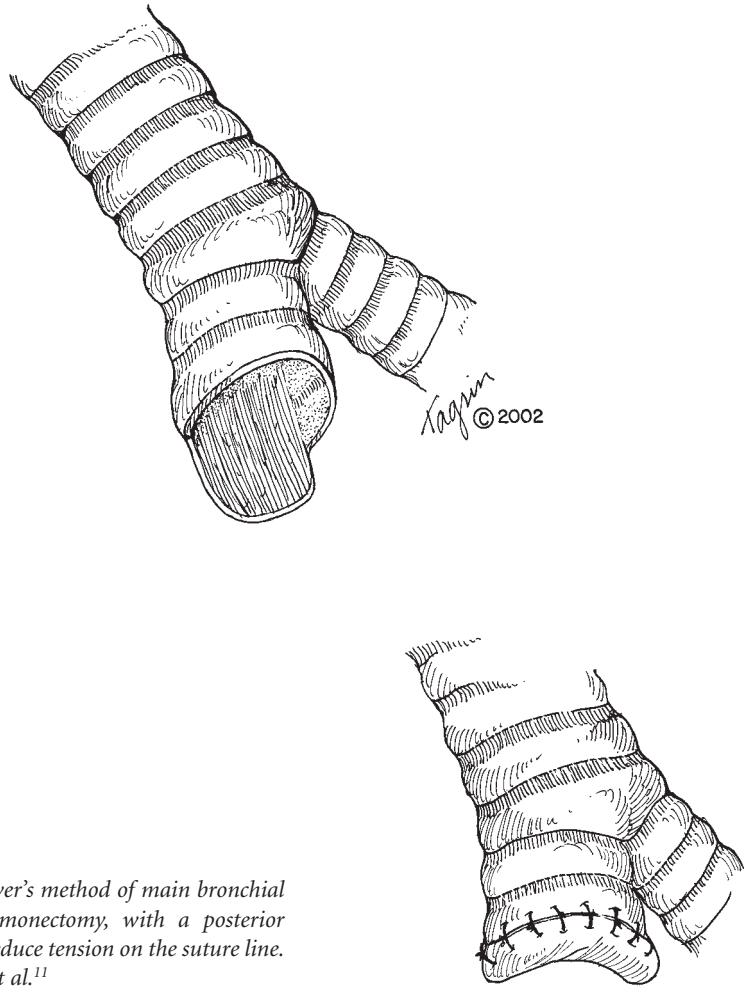


FIGURE **43-2** *Lyman Brewer's method of main bronchial stump closure after pneumonectomy, with a posterior membranous wall flap to reduce tension on the suture line. Adapted from Brewer LA et al.*[11]

longed postoperative antibiotics. We have found it useful to irrigate the hemithorax postoperatively through a high anteriorly-placed intercostal catheter, with drainage through a basilar intercostal catheter connected to an underwater seal drainage. Irrigation for several days with warm saline containing dilute antibiotic selected by intraoperative cultures has met with uniform success in preventing postoperative empyemas. We considered this to be a "preemptive" Clagett procedure.

Diagnosis

The diagnosis of postpneumonectomy bronchopleural fistula can be very difficult if only a pinhole fistula exists, or it can be very easy if a large bronchopleural fistula occurs with expectoration of copious amounts of serosanguineous fluid. Bronchopleural fistulae that occur early postoperatively do not usually present with infection but rather dyspnea and cough. Aspiration of even small amounts of pleural fluid into the remaining lung can cause an almost continuous irritative cough and can lead to severe respiratory distress and adult respiratory distress syndrome (ARDS). Expectoration of large amounts of brown fluid after a pneumonectomy is rare, but is pathognomonic of a bronchopleural fistula. With an early bronchopleural fistula, patients often have low-grade fever, elevated heart rate and respiratory rate, and leukocytosis. Later presentations of postpneumonectomy empyema are often those of indolent

infection. Fatigue, weight loss, dull deep chest discomfort, poor appetite, cough, low-grade fever, and night sweats are seen. A high index of suspicion is necessary to make the diagnosis, especially in the very late postoperative period.

The classic radiographic finding is lowering of the fluid level in the hemithorax (Figures 43-3*A*,*B*). Caution must be used in interpreting early postoperative films that are done portably, and therefore, the degree of uprightness and location of fluid level may vary. Ventilation scan may be useful in diagnosing occult bronchopleural fistulae by observing xenon gas in the operated hemithorax.[18] Computed tomography (CT) scans of the chest can be very helpful, especially in patients presenting late (Figure 43-4). Despite an opacified hemithorax by plain radiography, a CT scan can demonstrate air around the stump, which suggests bronchopleural fistula. The amount of residual fluid can be ascertained, the length and position of the bronchus can be delineated, the status of the remaining lung can be examined, and the possibility of locoregional recurrence of neoplasm can be documented.

Thoracentesis of the pneumonectomy space can be readily performed if an occult empyema is suspected. Careful sterile aspiration is necessary in order not to infect the space if it is indeed sterile. Patients diagnosed late have thickened pleura and markedly elevated hemidiaphragms, making thoracentesis more difficult. Ultrasound guidance and long needles are often helpful. Bronchoscopy is always an important step in thorough evaluation of a patient for possible bronchopleural fistula. Bronchoscopy can be performed in the early postoperative patient under local anesthesia with proper sedation and monitoring. Valuable information can be gained, such as the state of the bronchial closure, and presence of space fluid and infected secretions in the remaining lung. Watching a patient cough under local anesthesia while exam-

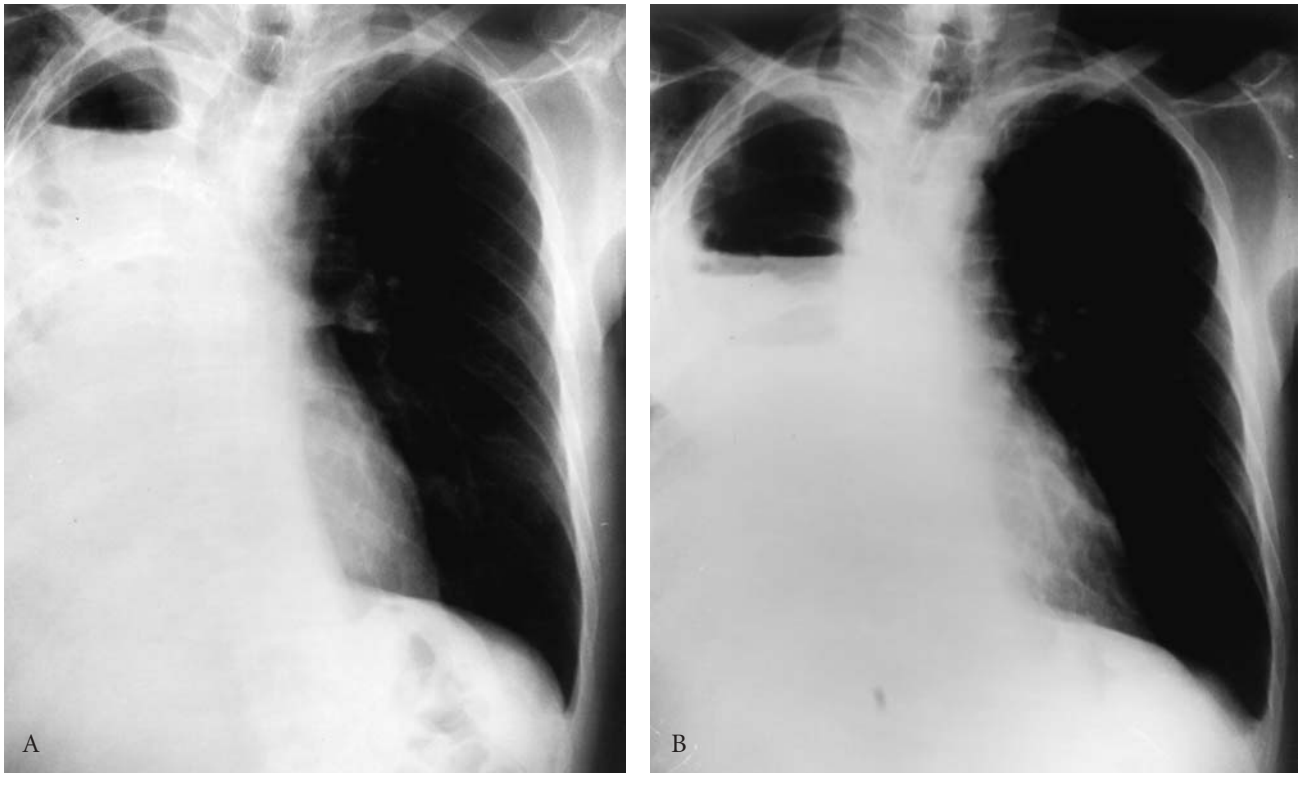


FIGURE **43-3** A, *Chest radiograph, 5 days after right pneumonectomy, with typical high air–fluid level.* B, *Chest radiograph of the same patient, 1 day later after cough and dyspnea were reported by the patient. Notice the drop in the air–fluid level. Subsequent investigation demonstrated a very small bronchopleural fistula.*

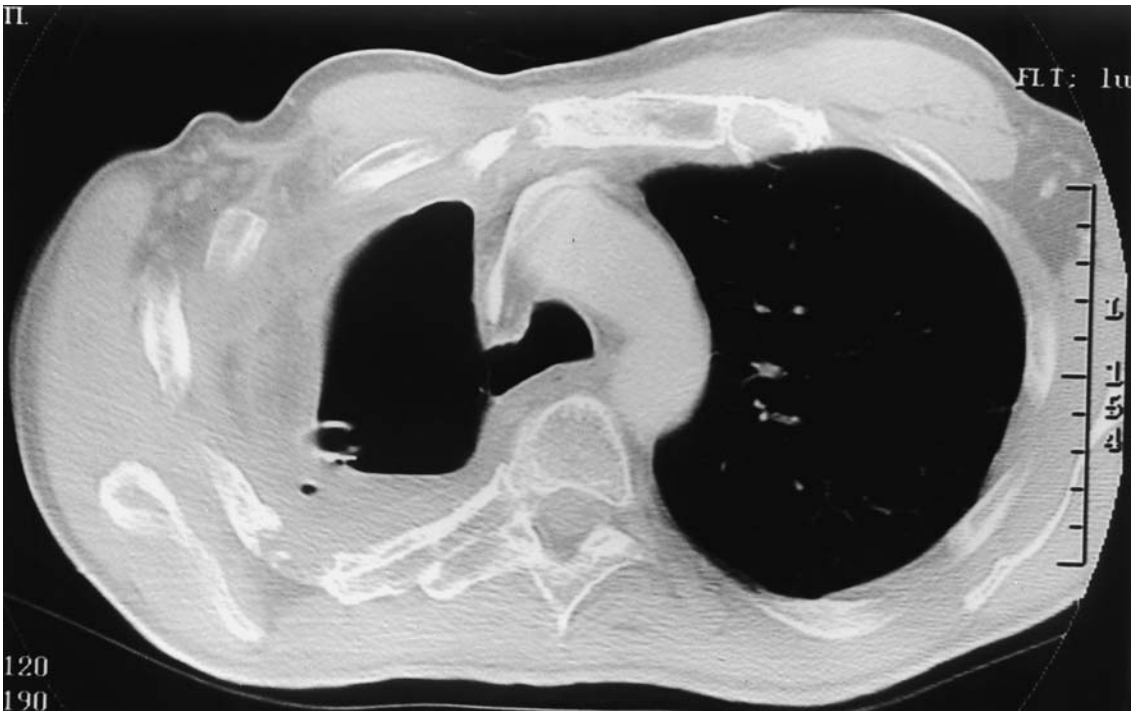


FIGURE **43-4** *Computed tomography (CT) scan of the chest of a patient, 6 months after right pneumonectomy complicated by a bronchopleural fistula. The empyema space is drained by a large bore catheter. Notice the markedly thickened pleura. The bronchial stump is widely dehisced.*

ining the stump may help to diagnose occult fistulae. The absence of a fistula at bronchoscopy does not eliminate the possibility of a pinhole fistula.

The differential diagnosis of an early postoperative postpneumonectomy bronchopleural fistula is fairly broad and includes pneumonia, ARDS, fluid overload, pulmonary embolism, myocardial infarction, and postpneumonectomy pulmonary edema.

Management

Immediate Treatment

Upon strong suspicion or diagnosis of an early postpneumonectomy bronchopleural fistula, the patient should be positioned in the lateral decubitus position with the operated side down and the head of the bed elevated to prevent further aspiration of noxious and potentially infected space fluid into the remaining lung. Intercostal catheter drainage of the space should then be performed to eliminate the risk of drowning the remaining lung with pleural fluid. Usually, the diaphragm is elevated almost to the level of the typical posterolateral thoracotomy incision, so chest tube insertion should be higher than usual. Culture of the pleural fluid and sputum should be performed and appropriate antibiotics administered. Patients who sustain an injury to the remaining lung and require mechanical ventilation present a major challenge in management. Selective ventilation of the remaining lung is usually required, either by selective intubation of the normal bronchus by an uncut standard endotracheal tube (confirmed by bronchoscopy) or by a double-lumen endotracheal tube. Early bronchoscopy is necessary to confirm the fistula and assess the degree of disruption of the closure. Patients with pinhole fistulae may close with drainage alone or with endoscopic glue techniques. Most fistulae involve separation of a significant amount of the bronchial closure and require urgent reclosure.

Historically, treatment of an early postpneumonectomy bronchopleural fistula was usually by closed drainage (until the mediastinum became stable), followed by prolonged open drainage and then reclosure of the fistula. This approach is no longer appropriate for the majority of patients diagnosed early, who can be treated by urgent reoperation and reclosure of the operated stump. Since the majority of these patients do not have truly infected spaces, there is a good chance for success with immediate reclosure. Patients without grossly purulent fluid and who have reasonable function in the remaining lung are candidates for urgent reoperation. In our own unit, we have successfully closed early postpneumonectomy bronchopleural fistulae as late as 37 days postoperatively (range 1 to 37 days, mean 12 days).[6] The important point is not the exact postoperative day but the underlying condition of the patient. Bronchoscopy to assess the fistula and to perform tracheobronchial toilet of the remaining lung is important. The intact bronchus is selectively intubated with a long endotracheal tube, preferably with a flexible distal segment. The tube is positioned bronchoscopically. The thoracotomy wound should be reopened and all fluid and fibrinous debris removed. Necrotic or obviously infected mediastinal tissue should be débrided. The stump must be débrided back to healthy tissue and reclosed with fine interrupted sutures (absorbable monofilament or multifilament). Extra attention needs to be paid to minimizing tension on the closure by resecting more of the anterior cartilaginous wall than the posterior membranous wall to allow the posterior wall to serve as a flap. This closure should be considered a temporary one, and it should be buttressed ideally with either intercostal muscle, extrathoracic muscle, or the omentum. In this high-risk situation, the vascularized tissue buttress should be sutured to the bronchus very carefully as if it were an anastomosis. The chest cavity should be extensively irrigated after the flap is placed and irrigation and drainage catheters placed as previously described for a "preemptive" Clagett procedure. Irrigation with warm saline (with dilute antibiotics) is continued for several days until the chest tube effluent is confirmed to be sterile by culture and Gram stain. Immediate reclosure with vascularized tissue coverage and postoperative irrigation was successful in all 5 patients from our unit treated early for postpneumonectomy bronchopleural fistula.[6] de Perrot and colleagues reported success in 3 of 4 patients treated in a similar fashion.[9] Gharagozlov and colleagues reported uniform successes in all 22 of their patients treated similarly.[19]

Patients with severe lung injury, who require ventilation and who are critically ill, and those presenting late with an established empyema, all require open drainage. Simple chest tube drainage alone is inadequate since a dependent pool of pus remains in the chest. This can be readily visualized by looking through the fistula or the chest tube tract with a flexible bronchoscope. One option is a rib resection and placement of a large empyema tube (our unit uses soft silicone Montgomery salivary bypass tubes made by Hood) in a dependent portion of the chest for drainage and irrigation. My preference for drainage is an open window thoracostomy (Eloesser window) with daily dressing changes. This is usually done by reopening the anterior aspect of the thoracotomy incision and resecting two or three ribs for 5 or 10 cm and then suturing the skin to the pleura to create a permanent stoma. The advantages of this approach include being able to inspect the entire cavity daily, elimination of retained infected material in the chest cavity, the débriding action of dressings, and the absence of a tube resting against the ribs which sometimes causes significant discomfort. Daily dressing changes can subsequently be carried out by a visiting nurse or the patient's family. Closure of the fistula is described below.

Endobronchial Treatment

A minority of patients with a very small postpneumonectomy bronchopleural fistula have been successfully treated by bronchoscopic delivery of a glue or sclerosing agent (Table 43-3).[20–23] Most authors report success only in small fistulae (usually less than 5 mm), and no success has been reported in patients with a totally open bronchial stump. Silver nitrate was originally used to stimulate granulation tissue but with relatively poor results. Acrylic tissue glues have been used to occlude fistulae but they are difficult to use

Table 43-3 Endobronchial Closure of Bronchopleural Fistulae

Author	*Year*	*Number of Patients*	*Substance Used*	*Success (%)*
Scappaticci et al[20]	1994	12	Cyanoacrylate	83
Torre et al[21]	1994	16	Fibrin glue	50
Hollaus et al[22]	1998	36	Fibrin glue	31
Varoli et al[23]	1998	35	Polidocanol	66

due to quick setting of the glue once the two components of the glue are mixed (and may glue the biopsy port of the bronchoscope closed!). Fibrin glue is increasingly reported as being useful in the management of small fistulae, perhaps since it is now commercially available. Polidocanol, an agent used for endoscopic sclerosis of esophageal varices, has been reported to be useful to stimulate granulation tissue which may occlude a fistula. The underlying empyema, if present, must of course still be treated.

Minimally Invasive Treatment

Video-assisted thoracoscopic closure of a postpneumonectomy bronchopleural fistula has been reported, using a stapler to reclose the bronchus.[24] This approach requires that a long stump be present, and closure must be done early enough to allow ready separation of the bronchus from the adjacent mediastinal tissue. Satisfactory harvesting and anchoring of a tissue flap is very difficult with this approach, thus limiting its application. An interesting transmediastinal approach via a cervical mediastinoscopy incision has been reported, using a videomediastinoscope.[25] This approach also requires the presence of a long bronchus and subsequent stapled closure by an endoscopic linear stapler introduced along the pretracheal plane. This approach does not allow coverage of the stapled bronchus and does not allow removal of the distal blind bronchial segment. Infection in the chest cavity must be treated if present.

Treatment of Chronic Postpneumonectomy Bronchopleural Fistula

Anterior Transsternal Approach. An anterior approach to closure of a postpneumonectomy bronchopleural fistula was reported by Padhi and Lynn in 1960, using an anterior thoracotomy, and was modified by Abruzzini in 1961, using a median sternotomy.[26,27] This approach was originally advocated for patients with long bronchial stumps and was thought to be an advantage since the operation was largely carried out through an undisturbed field. After a median sternotomy is performed, the anterior trachea and carina is exposed by retracting the superior vena cava laterally (see Figure 23-7 in Chapter 23, "Surgical Approaches"). Entry into the pericardium is usually necessary. Reamputation of the main pulmonary artery stump is sometimes necessary. Closure of the exposed main bronchus can then be performed either by sutures or staples. Dissection of the distal (fistula end) end of the bronchus can be exceedingly difficult due to the narrow confines of the operative field. Division of the stapled bronchus is critical to allow the bronchus to heal, since simply stapling the bronchus shut will invariably lead to recanalization because apposed mucosal surfaces do not heal together. If the divided but unresected distal bronchus is left in situ, then a mucocele or infection may develop or a repeat fistulization may occur. These undesirable consequences may be reduced by cauterization and destruction of the bronchial mucosa or resection of the remaining stump. If this approach is chosen, then strong consideration should be given to transposing the omentum (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation") at the same time to cover the reclosed stump. A sternohyoid muscle may be dissected through a cervical collar incision and pedicled to cover the bronchial closure. Again, the empyema cavity must be dealt with by a separate approach. The advantage of this approach includes operating through a relatively

uninvolved field by a muscle-sparing incision. Disadvantages involve contaminating the mediastinum, pericardium, and sternum, having major vascular structures surround and limit access to the bronchus, the problem of the divided (but unresected) bronchial stump, reduced (or nonexistent) efficacy in treating patients with short stumps, and the need to still deal with the empyema cavity by a separate approach. Patients with deep barrel chests and those with marked ipsilateral mediastinal shift are especially difficult to manage by this approach. If the mediastinum has previously been irradiated, then the procedure can become very difficult and dangerous. Several series are reported in Table 43-4 with success rates of 50 to 70% and mortality rates of 0 to 24%.[28–31] Perelman[28] and Ginsberg[29] and their colleagues have reported giving up on this approach and only use it if the lateral approach is no longer feasible.

Lateral Approach (Ipsilateral Hemithorax). Reopening the original posterolateral thoracotomy incision is almost always the approach of choice for large, chronic bronchopleural fistulae after pneumonectomy. Usually, rib resection is advantageous in reopening the thoracotomy, due to contraction and calcification of the hemithorax. The pleura is usually very thickened (often more that 1 cm) and must be fully opened to expose the usually small hemithorax. Most fistulae are immediately evident or can be made so by positive pressure ventilation. If doubt exists as to the exact location of the bronchus, intraoperative bronchoscopy can delineate the bronchus with the aid of transillumination. The thickened pleura, which can be calcified if very late, must be carefully dissected off the end of the bronchus to fully expose it. By staying directly on the wall of the bronchus, inadvertent injury of the contiguous pulmonary artery, superior vena cava, and superior pulmonary vein can usually be avoided. Once the dense scar overlying the mediastinum has been opened, the deeper tissues are not infrequently less scarred and easier to dissect. If the patient has received irradiation, then dissection may be exceedingly difficult. In order to avoid injury to major vessels, it is sometimes advisable to open the pericardium and re-divide the stump of pulmonary artery and superior vein more centrally. Irradiation may also obliterate the pericardial space, making dissection beneath the pericardium difficult. Another helpful maneuver on the right is to discover and follow the esophageal border to the plane posterior to the bronchus.

If the bronchus is long, it can be débrided back to healthy tissue, similar to that described in the acute situation. Reclosure with fine interrupted absorbable sutures can then be performed. This closure is again done with the expectation that a fair percentage will fail, mandating that a second layer of pedicled vascularized tissue be carefully sewn around the closure. Our preference is for the pedicled gastrocolic omentum based on the right gastroepiploic artery and transposed retrosternally (see Chapter 42, "The Omentum in Airway Surgery and Tracheal Reconstruction after Irradiation"). Other groups prefer extrathoracic muscle, usually the serratus anterior. The advantages of the omentum include its angiogenic abilities to facilitate neovascularization, its ability to fight infection, and its ability to conform to the underlying nooks and crannies of the empyema cavity. It has been employed successfully where prior muscle flaps had failed. If the bronchus is quite short and can not be closed, it is sealed instead by carefully suturing the muscle or omen-

Table 43-4 Transsternal Closure of Postpneumonectomy Bronchopleural Fistula

Author	*Year*	*Number of Patients*	*Successful Closure (%)*	*Mortality (%)*
Perelman et al.[28]	1987	39	74	23
Ginsberg et al.[29]	1989	13	77	0
Stamatis et al.[30]	1996	19	74	11
Brutel de la Riviere et al.[31]	1997	55	49	24

tal flap circumferentially around the edges of the open bronchus. The bronchus will subsequently reepithelialize as it heals. Obviously, planning and conducting the operation so that the patient can be extubated and breathe spontaneously at the conclusion is very important to minimize the chances of a repeat fistula. In rare circumstances, a right-sided approach may be used for a left-sided fistula with a very short bronchial stump. Advantages of the lateral approach include the opportunity to deal with the empyema cavity through the same incision, ability to resect the entire redundant bronchial stump (if present), and wide access to the bronchus. The results of our own unit, reported by Puskas and colleagues in 1995,[32] compare very favorably with the anterior transsternal approach (Table 43-5).[33–35] We found the omentum to be more effective (92%) than muscle (64%) for maintaining secure closure of chronic bronchopleural fistulae.[32] It has also been successful in closure of chronic fistulas in patients who had received high-dose mediastinal irradiation.[32]

Space Management. Following closure of a postpneumonectomy bronchopleural fistula, the space may be managed by continued drainage, the Clagett procedure, tissue transposition to fill the space, thoracoplasty, or a combination of these procedures. Continued wide drainage is appropriate for spaces that are still heavily contaminated. Closure by another method can be accomplished at a later date or deferred, especially if, for oncologic reasons, a poor outcome is expected. In 1963, Clagett and Geraci reported a technique to successfully close an infected pneumonectomy space that was relatively easy to accomplish and that involved little morbidity.[36] Clagett's technique involved creation of a large open window thoracostomy to facilitate debridement and cleansing of the empyema space and return of healthy granulation tissue covering the walls of the cavity. Once the patient is in good clinical shape with a visually clean space, cultures are taken to determine the types of bacteria present and their antibiotic sensitivities. The patient is hospitalized, administered appropriate intravenous antibiotics, and the space is irrigated repetitively over several days with the same antibiotics. Finally, the thoracostomy stoma is closed after filling the cavity with antibiotic solution. Despite this apparent violation of surgical principles (ie, closure of a contaminated space), this procedure usually works. In our experience, the procedure was successful on the first attempt in 60% of the patients and was ultimately successful in 80% (after two to three attempts).

Transposition of vascularized tissue into the empyema cavity both obliterates the cavity and provides the assistance of healthy tissue in managing the contaminated but clean cavity.[34,37] The omentum and serratus anterior, latissimus dorsi, pectoralis major, and rectus abdominis muscles have all been used to fill the cavity. The size of a postpneumonectomy cavity is quite variable; small cavities are obviously ideal for closure by this technique. Thoracoplasty, once used as first-line treatment of postpneumonectomy bronchopleural fistulae, is now relegated to the treatment of last resort due to its functional and disfiguring limitations.[33,38] It is most commonly used in conjunction with muscle transposition, when the amount of tissue available is insufficient to fill the space. A tailoring thoracoplasty is done to obliterate a cavity where necessary. Kergin's modification of the Schede thoracoplasty, by leaving the intercostal muscles attached at both ends, is preferred.[39]

Table 43-5 Closure of Postpneumonectomy Bronchopleural Fistula by Lateral Thoracotomy and Tissue Transfer

Author	*Year*	*Number of Patients*	*Successful Closure (%)*	*Mortality (%)*
Perelman et al.[28]	1987	37	95	3
Puskas et al.[32]	1995	31	86	10
Jadczuk[33]	1998	8	100	0
Duan et al.[35]	1999	32	94	0

References

1. Jack GD. Bronchial closure. Thorax 1965;20:8–12.
2. Sarsam MA, Moussali H. Technique of bronchial closure after pneumonectomy. J Thorac Cardiovasc Surg 1989;98:220–3.
3. Vester SR, Faber P, Kittle CF, et al. Bronchopleural fistula after stapled closure of bronchus. Ann Thorac Surg 1991;52:1253–8.
4. Asamura H, Naruke T, Tsuchiya R, et al. Bronchopleural fistulas associated with lung cancer operations: univariate and multivariate analysis of risk factors, management, and outcome. J Thorac Cardiovasc Surg 1992;104:1456–64.
5. Al-Kattan K, Cattalani L, Goldstraw P. Bronchopleural fistula after pneumonectomy with a hand suture technique. Ann Thorac Surg 1994;58:1433–6.
6. Wright CD, Wain JC, Mathisen DJ, et al. Postpneumonectomy bronchopleural fistula after sutured bronchial closure: incidence, risk factors, and management. J Thorac Cardiovasc Surg 1996;112:1367–71.
7. Hollaus PH, Lax F, El-Nashef BB, et al. Natural history of bronchopleural fistula after pneumonectomy: a review of 96 cases. Ann Thorac Surg 1997;3:1391–7.
8. Hubaut JJ, Baron O, Al Habash O, et al. Closure of bronchial stump by manual suture and incidence of bronchopleural fistula in a series of 209 pneumonectomies for lung cancer. Eur J Cardiothorac Surg 1999;16:418–23.
9. de Perrot M, Licker M, Robert J, et al. Incidence, risk factors and management of bronchopleural fistulae after pneumonectomy. Scand Cardiovasc J 1999;33:171–4.
10. Fowler WC, Langer CJ, Curran WJ, et al. Postoperative complications after combined neoadjuvant treatment of lung cancer. Ann Thorac Surg 1993;55:986–9.
11. Brewer LA, King EL, Lilly LJ, et al. Bronchial closure in pulmonary resection: a clinical and experimental study using a pericardial fat graft reinforcement. J Thorac Cardiovasc Surg 1953;26:507–32.
12. Rienhoff WF, Gannon J, Sherman I. Closure of the bronchus following total pneumonectomy. Ann Surg 1942;116:481–531.
13. Smith DE, Karish AF, Chapman JP, et al. Healing of the bronchial stump after pulmonary resection. J Thorac Cardiovasc Surg 1963;46:548–55.
14. Anderson TM, Miller JI. Use of pleura, azygos vein, pericardium, and muscle flaps in tracheobronchial surgery. Ann Thorac Surg 1995;60:729–33.
15. Rendina EA, Venuta F, Ricci P, et al. Protection and revascularization of bronchial anastomoses by the intercostal pedicle flap. J Thorac Cardiovasc Surg 1994;107:1251–4.
16. Smolle-Jüettner FM, Pinter H, Pongratz G, et al. Closure of bronchial defects using a pedicled pericardial flap. Ann Thorac Surg 1993;56:923–6.
17. Mathisen D, Grillo H, Vlahakes G, et al. The omentum in the management of complicated cardiothoracic problems. J Thorac Cardiovasc Surg 1988;95:677–84.
18. Pigula FA, Keenan RJ, Naunheim KS, et al. Diagnosis of postpneumonectomy bronchopleural fistula using ventilation scintography. Ann Thorac Surg 1995; 60:1812–4.
19. Gharagozlov F, Trachioltis G, Wolfe A, et al. Pleural space irrigation and modified Clagett procedure for the treatment of early postpneumonectomy empyema. J Thorac Cardiovasc Surg 1998;116:943–8.
20. Scappaticci E, Ardissone F, Ruffini E, et al. Postoperative bronchopleural fistulas: endoscopic closure in 12 patients. Ann Thorac Surg 1994;57:119–22.
21. Torre M, Chiesa G, Ravini M, et al. Endoscopic gluing of bronchopleural fistula. Ann Thorac Surg 1994; 58:901–2.
22. Hollaus PH, Lax F, Janakiev D, et al. Endoscopic treatment of postoperative bronchopleural fistula: experience with 45 cases. Ann Thorac Surg 1998;66:923–7.
23. Varoli F, Roviaro G, Grignani F, et al. Endoscopic treatment of bronchopleural fistulas. Ann Thorac Surg 1998;65:807–9.
24. Saw E, Ramachandra S, Franco M. Video-assisted thoracoscopic closure of postpneumonectomy bronchopleural fistulas. Surg Laparosc Endosc 1997;7:73–6.
25. Azorin JF, Francisci MP, Tremblay B, et al. Closure of postpneumonectomy main bronchus fistula using video-assisted mediastinal surgery. Chest 1996; 109:1097–8.
26. Padhi RK, Lynn RB. The management of bronchopleural fistulas. J Thorac Cardiovasc Surg 1960;39:385–93.
27. Abruzzini P. Trattamento chirurgico delle fistole del bronco principale consecutive a pneumonectomia per tubercolosi. Chirur Torac 1961;14:165–71.
28. Perelman MI, Rymko LP, Ambatiello GP. Bronchopleural fistula: surgery after pneumonectomy. In: Grillo H, Eschapasse H, editors. International trends in thoracic surgery. Vol 2. Philadelphia: WB Saunders; 1987. p. 407–12.
29. Ginsberg RJ, Pearson FG, Cooper JD, et al. Closure of chronic postpneumonectomy bronchopleural fistula using the transsternal transpericardial approach. Ann Thorac Surg 1989;47:231–5.
30. Stamatis G, Matini G, Freitage L, et al. Transsternal transpericardial operations in the treatment of bronchopleural fistulas after pneumonectomy. Eur J Cardiothorac Surg 1996;10:83–6.
31. Brutel de la Riviere A, Defauw JJ, Knaepen PJ. Transsternal closure of bronchopleural fistula after pneumonectomy. Ann Thorac Surg 1997;64:954–9.
32. Puskas JD, Mathisen DJ, Grillo HC, et al. Treatment strategies for bronchopleural fistula. J Thorac Cardiovasc Surg 1995;109:989–96.
33. Jadczuk E. Postpneumonectomy empyema. Eur J Cardiothorac Surg 1998;14;123–6.
34. Garcia-Yuste M, Ramos G, Duque JL, et al. Open-window thoracostomy and thoracomyoplasty to manage chronic pleural empyema. Ann Thorac Surg 1998;65:818–22.
35. Duan M, Chen G, Wang T, et al. One-stage pedicled omentum majus transplantation into thoracic cavity for treatment of chronic persistent empyema with or without bronchopleural fistula. Eur J Cardiothorac Surg 1999;16;636–8.
36. Clagett OT, Geraci JE. A procedure for the management of postpneumonectomy empyema. J Thorac Cardiovasc Surg 1963;45:141–5.
37. Michaels BM, Orgill DP, Decamp MM, et al. Flap closure of postpneumonectomy empyema. Plast Reconstr Surg 1997;99:437–42.
38. Harrigan TP, Snow JH. Thoracoplasty: current application to the infected pleural space. Ann Thorac Surg 1990;50:695–9.
39. Kergin FG. An operation for chronic pleural empyema. J Thorac Surg 1953;26:430–4.

Airway Management in Lung Transplantation

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Achieving a successful anastomosis of the airway has been an elusive goal in the development of lung transplantation. The airway anastomosis has been properly termed the "Achilles heel" of lung transplantation. Impaired healing at this site was a consistent impediment to successful human lung transplantation in the past, accounting for the majority of deaths in patients surviving more than 10 days following the transplant procedure. However, systematic investigations into the processes involved in the healing of airway anastomoses have resulted in the evolution of techniques that now allow for routinely successful results in most transplant patients.[1,2]

Three important factors for effective airway anastomosis have been identified in lung transplant patients. The first and most important issue relates to preservation of the *blood supply* of the airway. The distal trachea and bronchi are supplied primarily from the bronchial circulation.[3] The anatomy of this circulation and the maneuvers required to preserve airway perfusion by it are the most critical issues to be addressed in regards to the transplant airway anastomosis. A second determinant of a successful airway anastomosis is the *immunosuppressive regimen* used in lung transplant patients. Rejection in the transplanted lung may impair airway healing by compromising perfusion at the anastomotic site[4] and by the indirect effects of mechanical ventilation and other therapies instituted for the management of concomitant respiratory failure. The use of immunosuppressive regimens based on cyclosporine or FK-506 (tacrolimus) has markedly diminished the incidence of rejection-related postoperative graft failure. This improved immunosuppression may therefore augment anastomotic healing by diminishing the adverse effects of rejection on airway.

A third factor impacting on the transplant airway anastomosis is the use of high dose *steroids*. The potential adverse effects of steroids on the healing of airway anastomoses are numerous. The anti-inflammatory and lympholytic effects of steroids impair the process of wound healing in general. The use of steroids has been associated with anastomotic complications in other types of airway surgery.[5] In lung transplants, steroids have been shown to explicitly impair the healing of bronchial anastomoses and are associated with an increased incidence of bacterial sepsis.[6] Despite these findings, the routine use of steroids following lung transplantation

has *not* been associated with impaired anastomotic healing in clinical circumstances when airway blood supply is meticulously preserved and cyclosporine-based immunosuppressive regimens are used.[7] The pertinent conclusion from this finding is that, of the factors related to the outcome of a transplant airway anastomosis, preservation of blood supply and a cyclosporine-based immunosuppressive regimen are more significant determinants of successful anastomotic healing than the avoidance of steroid use per se.

Anatomy of the Bronchial Circulation

Preservation of the blood supply of the donor and recipient airway is the most critical factor in obtaining a successful airway anastomosis in transplant patients. The primary blood supply to the common sites of anastomosis, the distal trachea and main bronchi, is derived from the bronchial circulation. A thorough understanding of the anatomy and physiology of this circulation is important in the management of the airway in lung transplantation. The bronchial circulation arises embryologically from the primitive pulmonary plexus, formed from the ventral branches of the dorsal aorta. The sixth branchial arch forms the pulmonary arteries, which invade the lung buds and fuse with the separately formed pulmonary microvasculature. The pulmonary and bronchial arteries therefore share a related embryologic derivation, which endures in the adult as a network of extensive anastomotic communications between the two systems, although the sites of origin of the respective circulations are by then separated into systemic and pulmonic circuits.

The bronchial arterial circulation may be divided into *anterior* branches, arising from the subclavian, internal mammary, or coronary arteries, and *posterior* branches, arising from the thoracic aorta or intercostal arteries. Anterior branches are uncommon and assume clinical significance only in instances in which the posterior branches are interrupted, such as following en bloc heart-lung transplantation. In these instances, anterior branches, particularly from the coronary circulation, provide significant systemic collateral blood flow to the airway for anastomotic healing and maintenance of structural integrity of the tracheobronchial tree.[8]

The branches of the posterior circulation are divided into left- and right-sided arteries. A left bronchial artery arises directly from the descending thoracic aorta in over 90% of cases. A right bronchial artery arises in relationship to a right intercostal artery as a right intercostobronchial artery (RICBA) in 95% of cases. Additional right- and left-sided bronchial arteries may arise directly from the aorta or from ipsilateral intercostal arteries. The most common patterns of posterior bronchial circulation consist of one right and two left bronchial arteries (in approximately 25% of patients) or two right and two left bronchial arteries (in approximately 20% of patients). The RICBA arises from the descending thoracic aorta, crossing posterior to the esophagus and then passing between the azygous vein and the esophagus to the right main bronchus (Figure 44-1). It serves as a common trunk for the first or second right intercostal artery and the right bronchial artery. The RICBA supplies the right main bronchus and carina, and may supply the left bronchial tree as well, through subcarinal collaterals (Figure 44-2). This branch is useful for direct bronchial revascularization following en bloc double lung transplantation.[9,10]

The bronchial arteries enter the lungs through the hila and divide within the peribronchial connective tissue sheath surrounding the main bronchi. Typically, two or three branches of the bronchial artery wind around the wall of the bronchus, supplying the bronchial tree to the level of the terminal bronchioles. The diameter of the vessels decreases in size from 1.5 mm at the hilum to a 0.5 mm diameter at a bronchopulmonary segment. The arteries anastomose freely, forming a peribronchial plexus, and also bear branches that penetrate the wall to the bronchial mucosa, forming a submucosal plexus (Figure 44-3). Additional branches are given off to the visceral pleura, the lymph nodes, and the vasa vasorum of the pulmonary arteries and veins.

Along the airways and beyond the terminal bronchioles, bronchopulmonary anastomoses are found. Two types of large anastomoses have been identified: short, narrow vessels (1 mm × 100 microns) and longer, wider coiled vessels (20 mm × 400 microns).[11] The exact function of these vessels is not known,

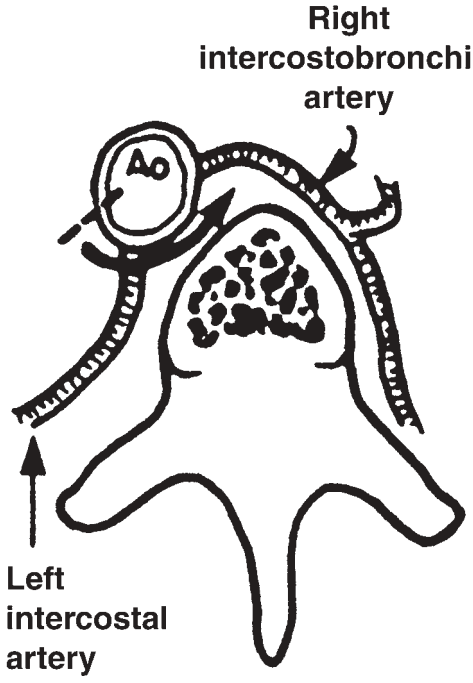

FIGURE **44-1** *Anatomic location of the right intercostobronchial artery (RICBA). The RICBA is found in 95% of patients, originating from the right side of the descending thoracic aorta. It travels extrapleurally, posterior to the esophagus, and then passes between the esophagus and the azygous vein to the right main bronchus. The RICBA commonly bifurcates into a right bronchial artery and an intercostal artery to the first or second intercostal space. Reprinted with permission from Schreinemakers HHJ et al.*[9]

although a role in pressure-dependent regulation of bronchopulmonary anastomotic flow via a Starling resistor mechanism has been postulated.[12] These large anastomoses may be found within the pleura, along the bronchi, and between bronchial arteries and the pulmonary veins. In addition, at the level of the pulmonary lobules, the bronchial microvasculature merges with the pulmonary capillary network. These anastomoses become more numerous at the periphery of the lung.

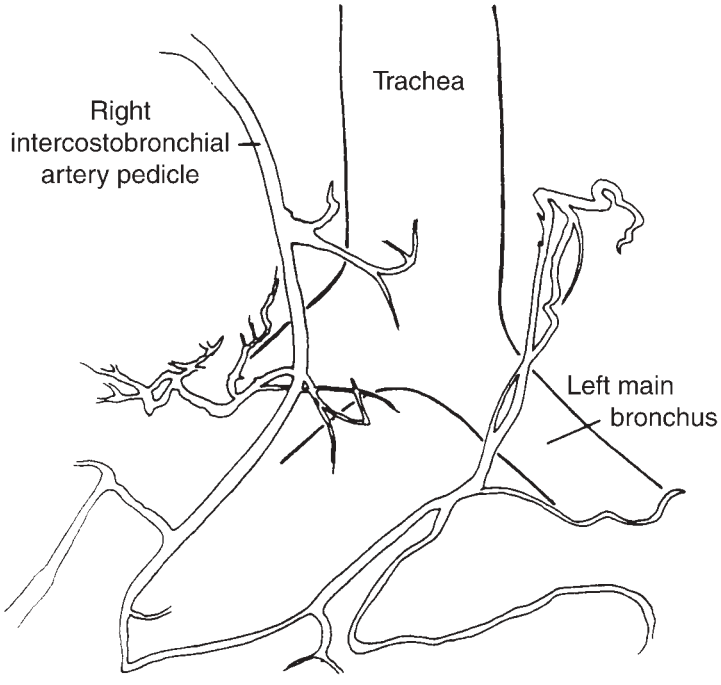

FIGURE **44-2** *A line drawing from a selective arteriogram indicating the distribution of the right intercostobronchial artery. There is extensive arborization to the distal trachea, proximal right main bronchus, and carina. Significant subcarinal collaterals to the left main bronchus are also demonstrated. Reprinted with permission from Schreinemakers HHJ et al.*[9]

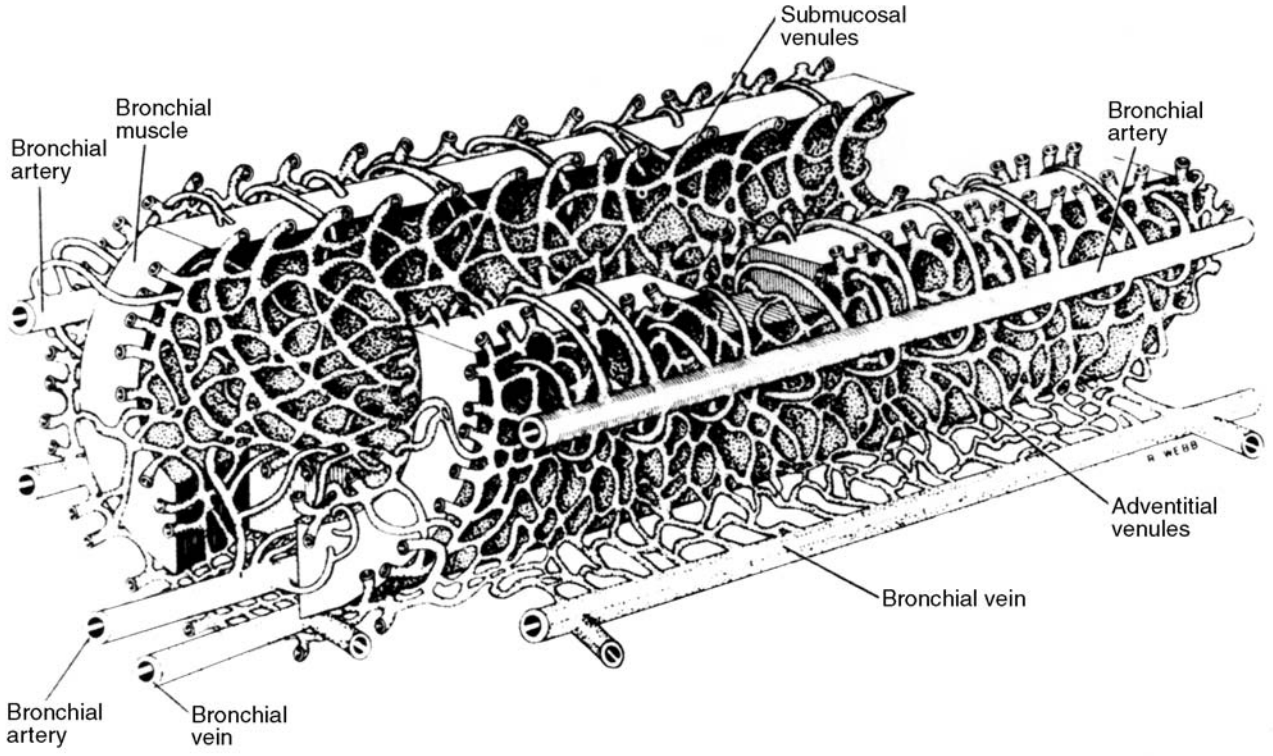

FIGURE **44-3** *Schematic representation of the bronchial circulation in a bronchiole. The bronchial arterial circulation forms extensive peribronchial and submucosal plexi, which anastomose to bronchial venous plexi in these locations. Reprinted with permission from Deffenbach ME, Charan NB, Lakshminarayans S, Butler J. The bronchial circulation. Am Rev Respir Dis 1987;135:463–81.*

The venous blood from the proximal tracheobronchial tree to the level of the lobar bronchi drains in an extrapulmonary route via bronchial veins to the azygous and hemiazygous veins. In addition, one or more deep bronchial veins are found in each lung. These deep veins drain intraparenchymal bronchial blood into the left atrium or inferior pulmonary vein near its left atrial confluence. In normal circumstances, however, the majority of intraparenchymal bronchial venous flow passes into the pulmonary circulation at the precapillary level via bronchial veins or the previously mentioned anastomotic pathways (Figure 44-4).

Functionally, flow in the bronchial circulation in the normal state is unidirectional, from bronchial arteries to both the bronchial veins and the pulmonary circulation via direct anastomoses. The bronchial veins also empty into the pulmonary circulation. Total bronchial blood flow and anastomotic flow in this state is directly proportional to the gradient between systemic arterial pressures and pulmonary arterial pressures.

The most common technique used for single or double lung transplantation consists of an isolated lung transplant without bronchial revascularization. In this instance, arterial pressure in the graft bronchial arteries remains close to zero after implantation of the graft, as the systemic connection to the bronchial arteries is not restored. Reversal of flow from the pulmonary circulation into the bronchial circulation via the bronchial venous network occurs, resulting in reperfusion of the peribronchial and submucosal plexi of the airway.[13] The relative contributions of the pulmonary arterial and pulmonary venous anastomoses to this flow via the bronchial veins is not known, but is likely to be pressure dependent. In addition, the contribution of the two types of direct bronchopulmonary arterial anastomoses to the reversed bronchial blood flow is not known. Clinical experience suggests, however, that the amount of perfusion achieved by this

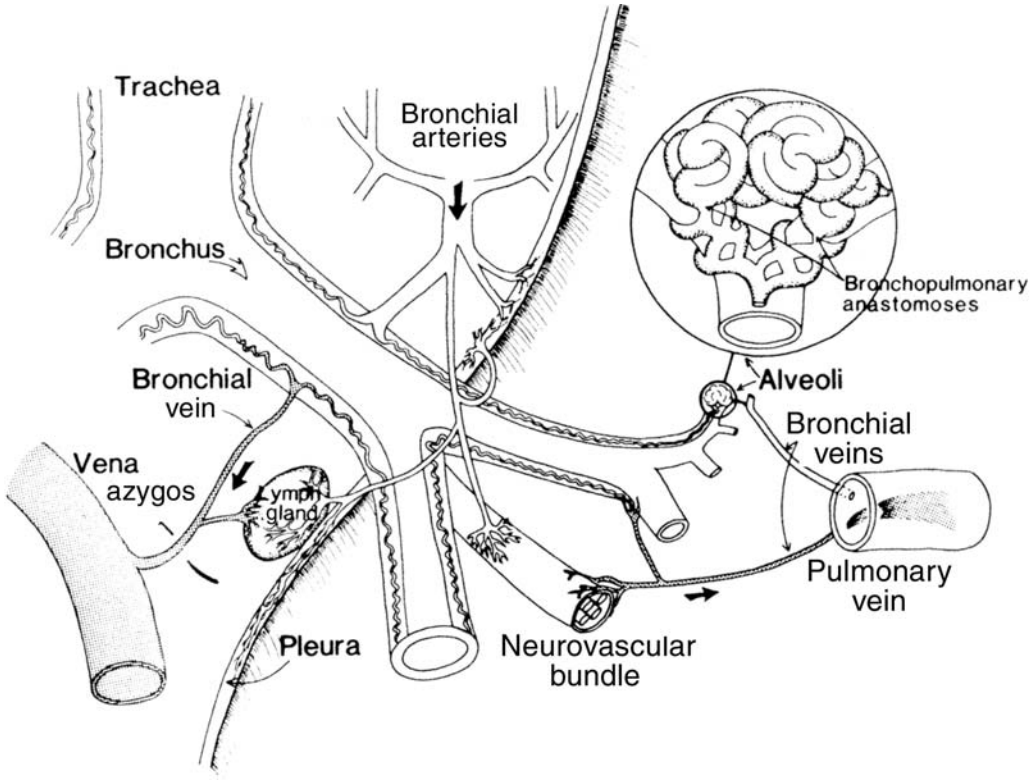

FIGURE **44-4** *Bronchial arterial and venous circulations. The bronchial arterial circulation to the extraparenchymal airways drains to systemic venous (azygous) vessels. The bronchial arterial circulation to the intraparenchymal airways may drain to deep veins, which anastomose to the left atrium or pulmonary veins or into the pulmonary circulation. The majority of flow is via the latter pathway. In addition, direct bronchial artery–pulmonary artery anastomotic vessels may be found. Reprinted with permission from Deffenbach ME, Charan NB, Lakshminarayans S, Butler J. The bronchial circulation. Am Rev Respir Dis 1987;135:463–81.*

reversal of flow from the pulmonary to the bronchial circulation is sufficient to maintain airway viability and allow for anastomotic healing.

Techniques of Airway Anastomosis

A crucial tenet of the airway anastomotic technique is precision in both the preparation and execution of the surgical procedure. The importance of a precise technique is especially underscored in lung transplant patients in whom the combination of ischemia, histologic incompatibility, and requisite pharmacologic therapy may combine to create an environment particularly averse to anastomotic healing. Despite differences in the details of the techniques among various centers, the principle of optimal preservation of airway blood supply remains paramount.

Maintenance of airway vascularity begins at the time of procurement of the donor lung tissue. Improvements in techniques of pulmonary preservation have led to significant improvements in healing of airway anastomoses.[14] At the present time, optimal lung preservation is accomplished by the administration of a pulmonary vasodilator (eg, prostaglandin E_1, prostacyclin) into the central circulation, followed by perfusion of the pulmonary circuit using either modified Euro-Collin's or University of Wisconsin solution at 10°C. Perfusion is carried out during mechanical ventilation of the lungs at a tidal volume of 10 to 15 cc/kg and a positive end-expiratory pressure of 5 cm of water. The active ventilation of the lungs allows for optimal distribution of the perfusate and cooling of the parenchyma by this solution. The lungs are then

extracted using a technique that preserves the nodal and pericardial tissues adjacent to the carina and main bronchi, which contain significant bronchopulmonary collateral vessels. The RICBA and a portion of the descending thoracic aorta may also be included in the donor bloc when bronchial revascularization using this vessel is contemplated. The lungs are transported in a partially inflated state (approximately two-thirds of the total lung capacity), immersed in cold preservation solution at 10°C.

In spite of these preservation efforts, a postoperative reimplantation response, identified as perihilar and peribronchial edema, is uniformly seen in patients after lung transplantation. Although this response does not usually interfere with gas exchange, in up to 15% of cases, graft dysfunction may occur which requires augmented respiratory support. An intriguing hypothesis is that this reimplantation response is primarily related to the effects of ischemia or reperfusion in the bronchial circulation.[15] Since the bronchial arterial circulation is *not* flushed with preservative solutions using current techniques, modification of these methods to correct this deficiency may further improve lung preservation. An optimally preserved lung provides the best substrate for a successful airway anastomosis.

Maintenance of donor airway blood supply relates primarily to the preservation of all available bronchopulmonary collateral circulation and maximizing reversed flow through this circuit. Keeping "excess" peribronchial tissue intact, including attached nodal and pericardial tissues, is valuable in this respect. The donor airway is trimmed to within 5 to 10 mm of its first bifurcation to minimize the length of airway that must be perfused at the periphery of the collateral circulation. Reflection of the peribronchial tissues away from the airway prior to its final transection preserves additional collateral blood flow within the peribronchial anastomotic plexus. Division of the left atrium to include the identifiable openings of the deep bronchial vein(s) within the cuff of atrial tissue will assure patency of this collateral channel. Finally, accurate orientation of pulmonary arterial and venous anastomosis, and intraoperative assessment of anastomotic pressure gradients, will assure the optimal perfusion is present in the pulmonary circuit to provide reverse flow into the bronchial circulation (Table 44-1).

Preservation of recipient airway blood supply is achieved by preserving the local systemic arterial inflow and collateral circulation. The bronchial artery is ligated distally on the native airway, at approximately the first lobar origin. The artery and surrounding peribronchial tissue containing the anastomotic plexus is then reflected proximally, away from the wall of the airway. The recipient airway is transected within the confines of the mediastinum and surrounding peribronchial tissues. Dissection of the proximal portion of the recipient airway within the mediastinum is confined to the anterior aspect of the cartilaginous wall, to avoid disruption of the segmental vessels located at a midlateral position which may augment the blood supply of the airway (Table 44-2).

Techniques for approximation of the donor and recipient airway vary among transplant centers. Basic principles of minimizing mucosal trauma and preventing strangulation of the mural tissues during suture tying are always followed. Both monofilament nonabsorbable and multifilament absorbable sutures have been used. Our own technique consists of the use of interrupted 4-0 Vicryl sutures, placed sequentially in a fashion to allow the sutures to be tied on the extramural aspect of the airway. Stay sutures of

Table 44-1 Optimizing Donor Airway Blood Supply

Optimal preservation of donor lung
Preservation of pericardium, peribronchial tissue
Inclusion of bronchial vein orifice in left atrium
Short donor airway segment
Unobstructed pulmonary arterial and venous anastomoses

Table 44-2 Optimizing Recipient Airway Blood Supply

Distal ligation of bronchial arterial supply
Preservation of peribronchial tissues
Mobilization within the mediastinum
Short recipient airway segment

2-0 Vicryl, placed in a lateral position on both the donor and recipient airway, are used and help to preserve orientation during suture placement and minimize tension during tying of the 4-0 sutures.

Some authors have emphasized the need for telescoping of the anastomosis, typically with the donor airway being positioned within the recipient airway.[7,16] In general, a size discrepancy between the two airways is always found and some degree of anastomotic intussusception is commonly seen, as is noted with routine sleeve-type bronchial resections. The specific use of telescoping the airway in lung transplant patients, in combination with other maneuvers to optimally preserve airway vascularity and promote anastomotic healing, can not be conclusively demonstrated. In our experience, we have not attempted to achieve a deliberately telescoped anastomosis and have not identified any anastomotic complication related to this approach.

The role of a vascularized wrap of the airway anastomosis in lung transplant patients has been widely debated. The use of a pedicle of vascularized tissue to wrap the anastomosis has been shown to restore systemic flow to the bronchial arterial circulation[17,18] and to diminish the incidence of mediastinitis and bronchovascular fistula.[19] The omentum has been most commonly employed as the anastomotic wrap, although intercostal muscle, pericardial fat, pleura, and thymus have been used. However, proponents of a deliberately telescoped airway anastomosis have reported excellent results without the use of extrabronchial tissues to wrap the anastomosis. A recent study showed no difference in airway anastomotic healing in a comparison of omentum and telescoped anastomoses.[20] However, the deliberate telescoping of the donor into the recipient airway effectively creates a wrap of vascularized tissue, in this case, the recipient airway with its intact systemic arterial circulation, about the site of anastomosis. The use of some method of anastomotic wrap, therefore, is an integral part of the anastomotic technique in lung transplantation. Our own practice is to use the omentum when it is available, given its superb vascularity and angiogenic capabilities. We have used the thymus and pericardial fat pad in instances when the omentum was unavailable. In all cases, no complications related to the use of these extrabronchial tissues were noted and no instances of anastomotic ischemia, dehiscence, or fistulization were identified. Our current incidence of anastomotic complication using this technique is 3%.

A technique for successful reimplantation of the bronchial vessels has been demonstrated for patients undergoing en bloc double lung transplantation.[10] The method employs the RICBA, which is procured with the donor bloc to its point of origin from the descending aorta. The segment of donor aorta containing the origin of the RICBA is either anastomosed directly or via a saphenous vein graft to the recipient aorta. En bloc double lung transplantation without bronchial revascularization is associated with a significant incidence of airway anastomotic ischemia, which is frequently fatal.[19] With bronchial revascularization in the above manner, no instances of airway ischemia or impaired healing have been noted in an early experience.[10] The technique of direct bronchial revascularization is also applicable to isolated single lung transplantation (SLTx) and to en bloc heart-lung transplantation. However, the necessity for this maneuver with these procedures, which currently have excellent results in achieving airway healing, remains to be determined. Although it has been hypothesized that small airway ischemia may lead to scarring and obliterative bronchiolitis (OB), which is currently considered a manifestation of chronic rejection, there is no evidence to support the routine use of bronchial revascularization as a method to diminish the incidence of OB in lung transplant recipients.

Results

The use of an anastomotic technique based on the principle of preserving airway vascularity has an excellent chance of providing successful airway healing in a lung transplant patient receiving cyclosporine-based immunosuppression. The precise likelihood of success, however, is related to the specific type of lung transplant procedure performed as well as to the indication for transplantation.

Isolated single lung transplantation is performed most commonly for nonseptic restrictive or obstructive lung disease or for progressive pulmonary hypertension, either primary or secondary. Anastomoses of the main bronchus, pulmonary artery, and left atrium are performed. The majority of cases are performed without bronchial revascularization. Cardiopulmonary bypass is required for 5 to 15% of the parenchymal disease recipients and all of the pulmonary hypertensive patients. Airway anastomotic complications are seen in 5 to 10% of patients. Most commonly, an inflammatory stenosis secondary to limited donor airway ischemia or necrotizing infection is identified. The majority of these complications are not fatal and may be managed successfully with preservation of graft function. Anastomotic complications occur more frequently in single lung transplants performed for restrictive lung disease and in left-sided implantations. The use of cardiopulmonary bypass may also increase the incidence of airway anastomotic complications in these patients, although heparinization or the use of antithrombotic agents per se may actually improve healing of the airway in the early postoperative period.[21]

Double lung transplantation is performed for bilateral septic lung disease, severe pulmonary hypertension or, on occasion, for obstructive pulmonary disease in a young recipient. The techniques employed for double lung transplants include sequential single transplants and en bloc transplants, with or without bronchial revascularization. Bilateral sequential single lung transplantation consists of sequential implantation of a pulmonary graft in each hemithorax, with vascular and bronchial anastomoses identical to isolated SLTx. Cardiopulmonary bypass is not usually required for this procedure. The incidence of anastomotic complications for this procedure is slightly greater than for SLTx, being approximately 15%, perhaps due to sepsis from the recipient airway in the anastomotic field. The majority of these complications, however, are nonfatal occurrences.[14,22] En bloc double lung transplantation consists of an anastomosis of the main pulmonary artery, the left atrium, and the airway. The airway anastomosis may either be performed as a single anastomosis at the level of the supracarinal trachea or as individual anastomoses to the main bronchi. This procedure requires cardiopulmonary bypass. The incidence of airway complication is highest for en bloc transplantation without bronchial revascularization. With a supracarinal anastomosis, over 40% of patients will develop airway ischemia and necrosis. In half of these patients, the airway complication will be a fatal event. A similar incidence of airway complication is noted for en bloc transplants with bilateral bronchial anastomoses, although the ischemic injury may be less severe than that seen with the tracheal anastomosis.[19] The experience with bronchial revascularization in en bloc double lung transplantation using a tracheal anastomosis has been most encouraging. In an initial report, the incidence of airway complications was 0%, and the only manifestation of ischemia was an insignificant delay in mucosal healing in 50% of patients. The incidence of airway complications in en bloc heart-lung transplantation is less than 10%, despite the routine use of a supracarinal anastomosis without bronchial revascularization. The preservation of large amounts of tissue about the airway containing peribronchial collaterals, as well as the potential for recruitment of collateral flow from the anterior branches of the bronchial circulation, particularly from the coronary circulation, are thought to account for successful anastomotic healing. However, isolated instances of diffuse airway necrosis have been reported. These events typically have a fatal outcome. The use of the omentum to wrap the tracheal anastomosis has been employed as a preemptive measure to lessen the likelihood of this event.

Anastomotic Complications and Their Management

Complications involving the airway anastomosis in lung transplant patients occur infrequently when appropriate surgical techniques are employed. The majority of pulmonary transplant recipients can be expected to have acceptable healing of the airway anastomosis. However, a consistent subpopulation of patients does develop airway complications in spite of meticulous surgical methods. These complications usually require some form of operative intervention for their resolution.

The etiology of lung transplant airway complications in the majority of cases is related to airway *ischemia*, almost invariably involving the donor airway. The vascular insult may be manifested acutely, within the first week following transplantation, or in a more subacute fashion in the third or fourth post-transplant week. In many cases, a technical error can be identified.

The most common technical error is using a relatively long segment of donor airway devoid of peribronchial tissues. The resultant ischemia of the proximal donor airway may lead to mucosal ulceration, and subsequent granulation tissue formation, or to transmural necrosis. This type of complication usually manifests within the first week following transplantation. Necrosis of the donor airway typically results in anastomotic dehiscence and perianastomotic sepsis. A wrap of vascularized tissue around the anastomosis can localize the septic focus and prevent mediastinitis. If the tissue used for the wrap has sufficient vascularity and if the ischemic region is localized to the proximal portion of the donor airway, then granulation tissue will replace the necrotic region. The final presentation of postischemic granulation tissue is a focal stenosis of the airway at the anastomotic site. In the event of free perforation or fistulization at the site of the anastomosis, reoperation and repair of the site with interposition of a vascularized tissue flap may allow restoration of airway continuity and preservation of graft function.

En bloc double lung transplantation using a supracarinal tracheal anastomosis without bronchial revascularization presents a special circumstance of airway ischemia. The donor airway is essentially an arch comprised of the right main bronchus, the carina, and the left main bronchus. Perfusion to this entire arch is by bronchopulmonary collaterals in the peribronchial tissue. Ischemia is most likely to occur in the middle of this span, corresponding anatomically to the proximal left main bronchus. The resultant necrosis and granulation tissue formation will lead to an anastomotic stenosis at this site, with varying degrees of involvement of the right main bronchus and carina.[19]

Airway ischemia may also be related to diminished pulmonary flow into the bronchial circulation. Angulation or constriction of the pulmonary arterial or venous anastomoses should be identified at the time of implantation. Compromise of bronchopulmonary collateral flow by this mechanism leads to ischemic sequelae at the anastomosis, resembling those seen with excess donor airway. Surgical revision of a suboptimal vascular anastomosis, as soon as it is identified, will not only prevent airway ischemia but will assure optimal respiratory function of the lung graft.

On rare occasions, diffuse necrosis of the donor airway to the level of the segmental bronchi may occur in the acute postoperative period. The etiology of this event is unknown, although impaired bronchopulmonary collateral flow due to an unidentified anatomic anomaly is a likely cause, based on the distribution of the ischemic necrosis. The only therapy for this otherwise fatal complication is emergent retransplantation, which is also associated with a high morbidity and mortality.[14]

Anastomotic airway complications may present in a delayed fashion, after apparently adequate healing initially. In such cases, a localized necrosis of the donor airway leading to anastomotic stenosis is identified more than 3 weeks following transplantation. Late anastomotic stenoses are less common and usually less severe than airway complications occurring in the early postoperative period. The etiology in these instances may be related to recurrent airway ischemia exacerbated by episodes of graft *rejection* (resulting in a decrease in bronchopulmonary collateral flow) or to a necrotizing *infection* of a poorly perfused donor airway.

Routine bronchoscopic surveillance should be continued for 6 weeks following transplantation. Such studies allow for inspection of the anastomotic site, for assessment of rejection by transbronchial biopsy, and for monitoring of microbiologic flora in the graft airway. Appropriate preemptive interventions may lessen the incidence of late airway complications from these etiologies.

Anastomotic stenosis is the most common airway complication following lung transplantation.[19,23] Most cases are related to donor airway ischemia, and the severity of the stenosis is directly related to the amount of donor airway that was ischemic. Patients are typically symptomatic with progressive exertional dyspnea, stridor, and diminished expired lung volumes. Diagnosis is made by bronchoscopy. Tracheal tomograms or virtual bronchoscopy employing image data acquired from helical chest computed tomography scanners have also been used as diagnostic modalities, but accurate evaluation and therapy will always require primary bronchoscopic evaluation. Management consists of rigid and flexible bronchoscopy with endobronchial dilatation using either graduated esophageal or balloon dilators. In many cases, two or three serial dilatations over the course of several months will suffice to manage the stenosis during maturation of the anastomotic site and stabilization of the stenotic segment.

In other cases, the initial severity of the stenosis or its rapid recurrence following dilatation will require endoscopic stent placement. Careful measurements of the length and caliber of the stenotic region following dilatation are made. An appropriate stent is selected and is positioned through the stenosis under bronchoscopic visualization. A self-anchoring Silastic stent is preferred, although permanent metal stents have been used. The Silastic stents have the advantage of ease of exchange and/or modification. Insertion of the Silastic stent involves its placement over a rigid bronchoscope to which an endoscopic fixation

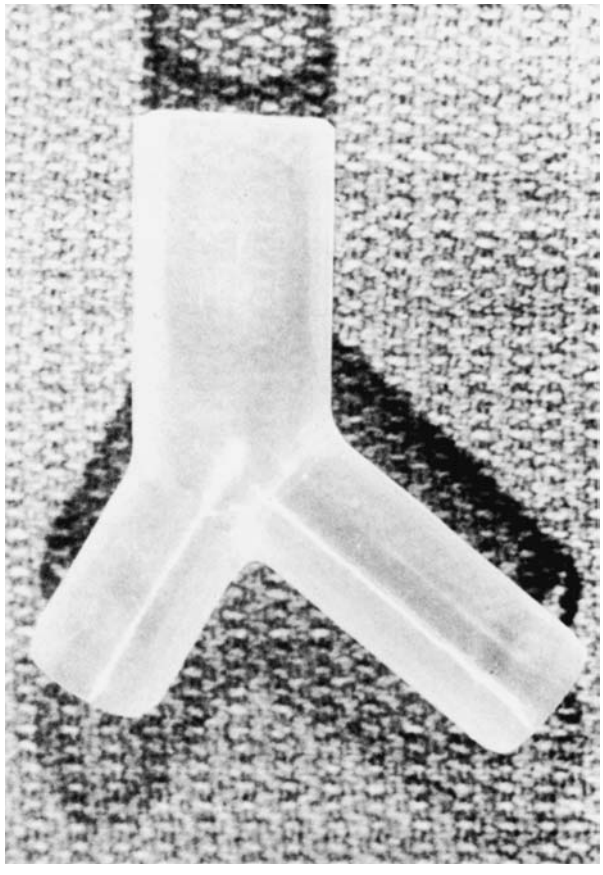

FIGURE **44-5** *Endotracheal Y stent. Silastic stents are useful for the management of anastomotic stenoses. A Y stent is commonly required for en bloc double lung transplant patients.*

device, such as a 40 F catheter placed over the bronchoscope proximal to the stent, is attached. The bronchoscope is then withdrawn as the stent is held in position by an endoscopic fixation device. The fixation device is then withdrawn and the position of the stent is confirmed by bronchoscopy.

Endobronchial stents are most commonly used for isolated SLTx or for bilateral sequential SLTx. Endotracheal Y stents are useful for stenoses occurring with en bloc double lung transplantation without bronchial revascularization (Figure 44-5). A modified tracheal T tube may be used for stenosis of tracheal anastomoses or for complex carinal stenoses. Unlike other inflammatory airway stenoses, anastomotic strictures in transplant patients may be very malleable. Endobronchial stents may be removed in more than one half of cases within 1 year. Sufficient remodelling of the anastomotic scar will have occurred at this interval to prevent the recurrence of an anastomotic stricture (Figure 44-6). The development of a fibrous stenosis, however, augurs poorly for the possibility of eventual elimination of the stent.[24,25] The likelihood of anastomotic stabilization and permanent stent removal is also less for carinal or tracheal stenoses. Although the presence of an indwelling stent does increase the incidence of tracheobronchial infection in transplant patients, the risk of reoperation is sufficiently great to preclude this as an alternative for the management of most such patients.

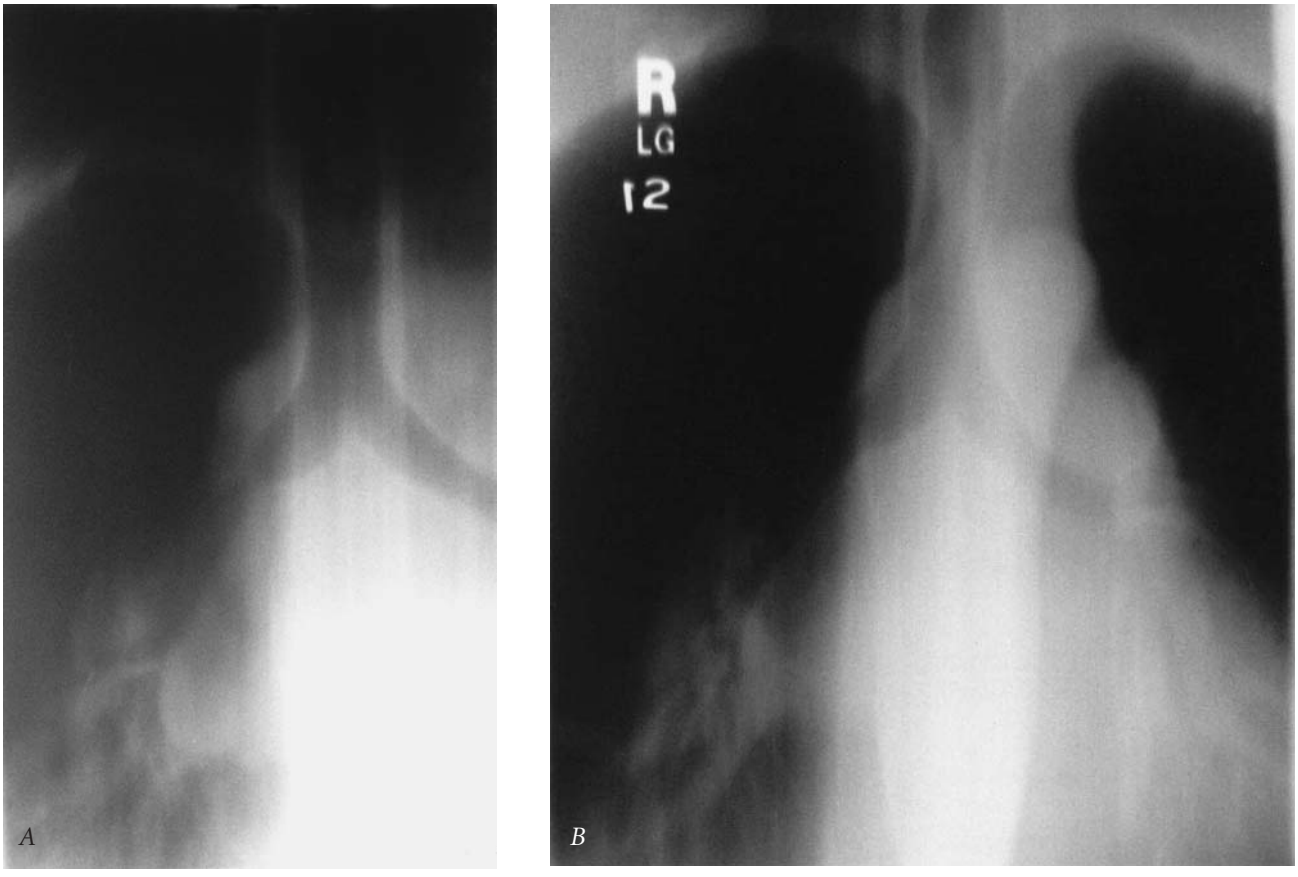

FIGURE **44-6** A, *Tracheal tomogram demonstrating an anastomotic stenosis. The airway stenosis can be seen at the level of the anastomosis in this patient's post-right single lung transplantation for pulmonary fibrosis with secondary pulmonary hypertension. The stenosis was treated by dilatation and stent placement.* B, *Tracheal tomogram following stent removal. The patient demonstrated in* A *had an indwelling Silastic stent placed after dilatation of the anastomotic stenosis. The stent was removed after 14 months and subsequently did not recur. A normal anastomotic caliber can be appreciated on this radiograph.*

Summary

Success in the management of the airway anastomosis has inaugurated the clinical application of lung transplantation for end-stage pulmonary disease. The critical issue for healing of the transplanted airway is maintenance of the blood supply of both the donor airway (primarily from bronchopulmonary collateral flow) and the recipient airway (primarily from systemic arterial flow). The exact role of cyclosporine-based immunosuppression and other factors in augmenting healing of the airway anastomosis in transplant patients is unknown, but they are thought to be important elements.

Precise surgical technique, optimal lung preservation, conservation of peribronchial collateral vessels, and wrapping of the anastomosis with vascularized tissue are the significant points of the anastomotic method in lung transplantation.[26] Bronchial artery revascularization is possible, but is useful primarily for en bloc double lung transplantation. Anastomotic stenosis, the most common airway complication following lung transplantation, is usually due to donor airway ischemia and is optimally managed by bronchoscopic dilatation and stent placement.

References

1. Veith FJ. Lung transplantation. Transplant Proc 1977; 9:203–8.
2. Cooper JD. Evolution of techniques and indications for lung transplantation. Ann Surg 1990;212:249–58.
3. von Sommering ST, Reisseissen FD. Ueber den bau der lungen. Berlin: Voss; 1808.
4. Inui K, Wada H, Yokomise H, et al. Evaluation of a bronchial anastomosis by laser Doppler velocimetry. J Thorac Cardiovasc Surg 1990;996:14–9.
5. Grillo HC, Zannini P, Michelassi F. Complications of tracheal reconstruction: incidence, treatment and prevention. J Thorac Cardiovasc Surg 1986;91:322–38.
6. Lima O, Cooper JD, Peters WJ. Effects of methylprednisolone and azathioprine on bronchial healing following lung autotransplantation. J Thorac Cardiovasc Surg 1981;82:211–5.
7. Calhoon JH, Grover FL, Gibbons WJ, et al. Single lung transplantation: alternative indications and technique. J Thorac Cardiovasc Surg 1991;101:816–25.
8. Reitz BA, Wallwork JL, Hunt SA, et al. Heart-lung transplantation. Successful therapy for patients with pulmonary vascular disease. N Engl J Med 1982;306: 557–64.
9. Schreinemakers HHJ, Weder W, Miyoshi S, et al. Direct revascularization of bronchial arteries for lung transplantation: an anatomical study. Ann Thorac Surg 1990;49:44–54.
10. Couraud L, Baudet E, Martigne C, et al. Bronchial revascularization in double-lung transplantation: a series of 8 patients. Ann Thorac Surg 1992;53:88–94.
11. Tobin C. The bronchial arteries and their connections with other vessels in the human lung. Surg Gynecol Obstet 1952;95:741–50.
12. Baile EM, Albert RK, Kirk W, et al. Positive end-expiratory pressure decreases bronchial blood flow in the dog. J Appl Physiol 1984;56:1289–93.
13. Ladowski JS, Hardesty RL, Griffith BP. The pulmonary artery blood supply to the supracarinal trachea. J Heart Transplant 1984;4:40–2.
14. de Hoyos AL, Patterson GA, Maurer JR, et al. Pulmonary transplantation: early and late results. J Thorac Cardiovasc Surg 1992;103:295–306.
15. Traber DL, Lentz CW, Traber LD, Herndon DN. Lymph and blood flow responses in central airways. Am Rev Respir Dis 1992;146:515–8.
16. Veith FJ, Richards K. Improved technique for canine lung transplantation. Ann Surg 1970;171:553–8.
17. Dubois P, Choiniere L, Cooper JD. Bronchial omentopexy in canine lung allotransplantation. Ann Thorac Surg 1984;38:211–4.
18. Fell SC, Mollenkopf FP, Montefusco CM, et al. Revascularization of ischemic bronchial anastomoses by an intercostal pedicle flap. J Thorac Cardiovasc Surg 1985;90:172–8.
19. Patterson GA, Todd TR, Cooper JD, et al. Airway complications after double lung transplantation. J Thorac Cardiovasc Surg 1990;99:14–21.
20. Miller JD, DeHoyos A. An evaluation of the role of omentopexy and of early perioperative corticosteroid administration in clinical lung transplantation. J Thorac Cardiovasc Surg 1993;105:247–52.
21. Borst HG, de Hoyos AL, Patterson GA, et al. Pulmonary transplantation: early and late results. J Thorac Cardiovasc Surg 1992;103:306.
22. Pasque MK, Cooper JD, Kaiser LR. Improved technique for bilateral lung transplantation: rationale and initial clinical experience. Ann Thorac Surg 1990;49:785–91.
23. Patterson GA, Cooper JD. Status of lung transplantation. Surg Clin North Am 1988;68:545–58.
24. Kshettry VR, Kroshus TJ, Hertz MI, et al. Early and late airway complications after lung transplantation: incidence and management. Ann Thorac Surg 1997;63:1676–83.
25. Orons PD, Amesur NB, Dauber JH, et al. Balloon dilation and endobronchial stent placement for bronchial strictures after lung transplantation. J Vasc Intervent Radiol 100;11:89–99.
26. Wain JC, Wright CD, Donahue DM, et al. Lung transplantation and the bronchial anastomosis: an alternative approach. [In preparation]

Tracheal Replacement

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When I commenced work on the trachea in 1961, a large body of literature on experimental tracheal replacement and transplantation already existed. There had also been a few clinical trials, fewer successes, and these were qualified. Biological principles seemed to prevent consistent success of prosthetic implants in the trachea. This directed me to reassess the extent of resection that was possible by employing anatomic maneuvers directed to primary anastomotic reconstruction.[1] Forty years later, the experimental literature on tracheal replacement has increased greatly, many ingenious and even promising ideas have been developed, an equal number have receded, and there is still not a dependable or predictable tracheal substitute.

This chapter is a necessarily brief commentary on these voluminous efforts to solve what at first glance seems to be such a simple problem—development of an acceptable conduit to replace the native trachea. Related literature published in English is extensively reviewed here. A complete review is nearly impossible, since so attracted have surgeons been by the illusory simplicity of the problem. I hope that a fairly complete recitation of past efforts may forestall the tedious repetition of similar experiments using techniques that have long been shown to fail. My intent is not to discourage thoughtful exploration of this biosurgical challenge but to provide a helpful background for possible development of new concepts. At very least, it may save the lives of countless dogs, sheep, pigs, rats, and other experimented-on creatures from the altars of dubious science. I hope to be excused for slighting copious additional literature, especially that in German-, French-, and Japanese-language journals.

Need for Tracheal Replacement

Most tracheal lesions can now be resected and primary reconstruction safely effected, as detailed in this book. The general limits of safe resection are about one-half of the tracheal length in adults and probably one-third in small children. Limits vary widely depending upon age, body build, local anatomy, pathology, and prior treatment. More lengthy, non-neoplastic lesions which cannot be safely removed and reconstruction done primarily, can usually be managed in the long term with T tubes or stents (see Chapter 39, "Tracheal T Tubes"

and Chapter 40, "Tracheal and Bronchial Stenting"). The silicone airway so provided is at least as satisfactory as any prosthetic device yet fashioned for surgical tracheal replacement. Any tracheal replacement technique offered as an alternative must therefore be wholly dependable and provoke minimal complications or risk of fatality.[2] The special case and previously challenging problem of long segment congenital tracheal stenosis is now safely and effectively managed by slide tracheoplasty (see Chapter 6, "Congenital and Acquired Tracheal Lesions in Children" and Chapter 33, "Repair of Congenital Tracheal Lesions").

The remaining need for tracheal substitution is to permit extirpation of those few tumors of extended length, chiefly adenoid cystic carcinoma, where invasion of the larynx or mediastinum does not prohibit complete resection with salvage of a functional larynx. Few such patients are seen annually, even in a clinic such as ours where airway surgery is frequently performed. Currently, these patients are managed palliatively with irradiation, stents, and T tubes (see Chapter 7, "Primary Tracheal Neoplasms," Chapter 39, "Tracheal T Tubes," Chapter 40, "Tracheal and Bronchial Stenting," Chapter 41, "Radiation Therapy in the Management of Tracheal Cancer"). A safe and dependable method of tracheal replacement would indeed be useful and welcome for these few patients.

Requirements for Replacement

Belsey iterated the requirements for tracheal replacement to be 1) a laterally rigid but longitudinally flexible tube, and 2) a surface of ciliated respiratory epithelium.[3] The second criterion has proved to be desirable, but not essential.[4,5] Patients can clear secretions by cough despite conduits lined with squamous metaplastic epithelium, skin, or foreign materials (silicone tubes, coated stents, metallic and other solid prostheses). The conduit must further be initially airtight and become integrated into adjacent tissues, so that chronic inflammation, granulation tissue, infection, and erosion do not occur. A requirement for immunosuppression is undesirable for many reasons, especially since the principal need for tracheal replacement is extensive carcinoma (adenoid cystic and squamous cell). Finally, for a method to be practically considered, the technique of construction or insertion of the conduit must be surgically straightforward and the results must be predictably successful.

Other authors have further added that materials for tracheal replacement must be biocompatible, nontoxic, nonimmunogenic, noncarcinogenic, must not dislocate or erode over time, should ideally provide or facilitate epithelial resurfacing, should avoid stenosis or late buckling, resist bacterial colonization, avoid accumulation of secretions, and must be permanent constructions.[6,7]

Approaches to Replacement

The belief which persisted for many years, that only a few centimeters of trachea could be resected *circumferentially* and the trachea safely anastomosed, stimulated earlier search for a tracheal prosthesis.[3,8] Today, the need for replacement is recognized to be much more limited. The quest continues because of the more closely defined needs which have been presented. In general, lines of research have included the following:

1) Trials of multiple *foreign materials* with many technical modifications to avoid complications of implantation;
2) Implantation of *nonviable tissues*, including fixed trachea;
3) Adaptation and transfer of *autogenous tissues*, with or without scaffolding of foreign materials as patches or tubes;
4) *Tissue engineering* of needed components such as cartilage; and
5) *Transplantation* of allografts with and without immunosuppression, preservation, and vascularization procedures.

Success has episodically been announced over the decades in each of these categories, but thus far, has not held for the long term in any safe and practicable manner.

The technique of *lateral tracheal excision*, principally for tumors, was practiced for many years, because of fear of creating tracheal discontinuity which, it was believed, could not be reapproximated. The defect in the trachea was patched with a variety of foreign material and tissues.[9] The technique has been largely abandoned because of the frequency of recurrence of tumor due to inadequate margins resulting from the compulsion to save sufficient tracheal wall for structural reason. Furthermore, necrosis and leakage of devascularized patches were often fatal, if in the mediastinum. However, combinations of vascularized tissue flaps, such as pericardium, and supporting foreign materials (such as Marlex) did succeed in healing in some cases, even if rarely successful oncologically.

Foreign Materials

The apparent simplicity of tracheal replacement encouraged trials of tubular conduits, initially of solid material. Since these could not become incorporated by local tissues, problems of migration, dislodgement, erosion, infection, and obstruction usually arose. Epithelization, of course, was impossible. Furthermore, solid tubes can never be removed, since the connective tissue tract formed around them proceeds to obstruct with new connective tissue formation and by contraction in the absence of a stent.[10] Attention then turned to porous materials, often meshes of various substances, which might encourage tissue ingrowth and possibly even epithelization in time.

Solid Prostheses

Foreign materials were earlier used to form tubular replacements for resected trachea. The trachea was replaced *experimentally*, most often in dogs, with the following materials: tubes of stainless steel[11,12]; tight coils of steel wire[13]; Vitallium[11,14]; glass[11]; polyethylene[10,15–19]; Lucite[13,14]; silicone[20–23]; Teflon[12,24,25]; Ivalon[14,26]; polyvinyl chloride[27]; polyethylene, polyurethane, Silastic, and Teflon combinations[28]; rigid and flexible Silastic with Dacron cover[29]; and tubes of Teflon, Ivalon, and Silastic.[30]

Solid prostheses were also tried *clinically* in occasional cases, using the following materials: stainless steel[31,32]; steel coil[13]; silicone[33,34]; polythene (polyethylene)[35,36]; Teflon[37]; polyethylene and Tantalum[28]; and Lucite.[15] Anastomoses after short resections were also done over splinting polythene stents.[38]

Borrie, Redshaw and colleagues added suturable subterminal fabric cuffs to a silicone prosthesis, which was intussuscepted into the tracheal ends (experimentally in sheep), to prevent granulation tissue from obstructing the lumen and to encourage fixation of the prosthesis.[21,22]

Neville and colleagues employed a similarly cuffed silicone tube clinically, whereas Toomes and colleagues experienced obstructive granulation tissue, migration, and vascular erosion with the same prosthesis.[33,34] I saw patients in consultation, who were suffering severe, recurrent, and insoluble granulation tissue obstruction at either end of a Neville prosthesis. Long segments of normal trachea had been excised to remove shorter stenotic lesions in order to accommodate the nonadjustable prostheses. In a number of patients, the original lesion could have been easily managed with a short resection and end-to-end anastomosis. In later cases, the prosthesis had been placed inside an unresected stenotic trachea, where a silicone stent or T tube might have served better.

Solid prostheses, despite some successes for varied lengths of time, eventually tended to migrate, become dislodged, obstruct with granulation tissue at either end (with subsequent stenosis), and encourage infection at the interface between foreign material, tracheal epithelium, and the granulation tissue bed. Erosion of the brachiocephalic artery, not infrequently, followed with fatal result. Complete epithelization rarely occurred beneath the prosthesis. Despite all of this, a rigid prosthesis can maintain an open airway for some time, even though healing has not occurred in any complete sense. Such a result, however, is episodic and unpredictable. Daniel mistakenly believed that tracheal rings regenerated structurally over a solid tube and Longmire appeared to accept this unlikely event.[11,15]

To date, nearly all (if not all) surgical prostheses that have been successful (ie, vascular conduits, heart valves, orthopedic devices) have been cited in potentially sterile mesenchymal tissues. No example of comparable success has been cited in the respiratory, gastrointestinal, or genitourinary tracts. Here, an interface inevitably persists among foreign material, chronically repairing connective tissue, and epithelium, which is a source of bacterial contamination. The trial of one material after another cannot be expected to solve this basic biologic incompatibility.

Porous Prostheses

In order to counter the usual failure of impervious solid prostheses, meshes made from a wide selection of materials were used *experimentally*. The porous structure was calculated to permit ingrowth of host connective tissue, incorporating the prosthesis into the tracheal site. A minimal porosity of 40 to 60 μm is necessary for capillary ingrowth.[7] It was hoped that this bed of autogenous new connective tissue would serve as a base for migration of adjacent tracheal epithelium. Regeneration of the epithelium was described, with evolution from squamous to cuboidal to pseudostratified to ciliated cells.[39,40] Meshes were sometimes supported with wire, plastic rings or coils, and sealed with tissues (such as omentum, fascia, pericardium) or biopolymers (such as fibrin sponge or reconstituted collagen) to prevent early air leakage. Among the meshes tried experimentally were the following: steel wire, variously wrapped with tissue for air-tightness[10,13,19,25,41–43]; stainless steel wire lined with dermis or various synthetic materials[12]; Tantalum with or without pleura or fascia[8,13,19,25,44–47]; coated titanium fiber metal[48]; Marlex,[19,42,49,50] Marlex with collagen,[51] or with collagen reinforced by a polypropylene spiral[52,53]; polytetrafluoroethylene (PTFE)[25,54–57]; polyurethane[58]; Ivalon and wire[14,59,60]; Dacron and polyurethane[61]; and Teflon.[25,36]

The list of combinations is almost endless. Dacron was employed in patches of low and high porosity, with sloughing of the low-porosity material, and eventual incorporation and epithelization of small patches of high-porosity Dacron over a prolonged period of time.[61,62] Experimental Z-plasty allowed tracheal approximation by relaxation, and the sites of Z-plasty were patched with Dacron.[63] McCaughan noted that Silastic-reinforced Dacron produced granulations and stenosis.[64] Surprisingly, ready connective tissue incorporation and epithelization (in 5 to 7 weeks) of a 5 cm Goretex tube with reinforcing rings was reported, but details of epithelization and long-term results were lacking.[65] Ingrowth of connective tissue in Dacron prostheses led to narrowing and obstruction.[66] Wide mesh Teflon with polypropylene rings, with an inner liner of solid polypropylene tube, was also attempted.[36]

Meshes were also employed *clinically* as patches or circumferentially.[67] These were made of the following materials: steel wire[13,41]; Tantalum covered with fascia lata,[68] or skin and Tantalum gauze[69]; heavy Marlex,[50,70–73] sometimes covered with pedicled pericardium, Ivalon, and wire.[58] Pagliero and Shepherd managed late postoperative dehiscence of the trachea by operative insertion of a stainless steel wire coil to splint open a connecting conduit of partly epithelized scar tissue.[74]

Connective tissue ingrowth served to fix and incorporate some porous prostheses as noted. However, the continued proliferation of scar tissue most often led to obstruction and stenosis. Frequently, large sections of mesh, especially in longer segment replacements, remained uncovered by connective tissue, and bacterial colonization followed. Epithelium generally failed to migrate sufficiently to cover the entire neotracheal surface, allowing continued cicatrix formation, usually in the center of the replacement. Although very short interpositions might achieve full epithelization, which seemed to prevent further cicatrization, the rate and completeness of epithelial migration was usually insufficient to prevent central granulations, cicatrization, and stenosis. Obviously, very short tracheal replacements offered no solutions to the problem addressed. Pearson and colleagues experimentally endeavored to provide a source of epithelization by advancing partially-detached terminal rings of cartilage, with the mucous membrane intact, obliquely over Marlex replacements.[50] Stenotic obstruction and brachiocephalic artery erosion attended some Marlex

tracheal replacements in humans as well as in experimental animals.[50,73] Death occurred similarly with steel mesh prosthesis.[13] A Marlex mesh that fails to become fully covered harbors bacteria (notably *Pseudomonas aeruginosa*), which produce purulent sputum and foul odor. One patient, who over many years had lost his wife, his job, and his friends as a result of intolerable halitosis from a Marlex reconstruction of cervical trachea, was finally relieved by its excision and placement of a silicone T tube. Thus, the dual problem of infection or of granulation tissue and scar formation, sometimes in irregular patterns, tends to prevent even incorporation of porous prostheses.[36] An impervious inner liner prevents the overgrowth of obstructing granulation tissue, but full epithelization was not seen and such an internal liner can never be removed.[36]

Implantation of Nonviable Tissues

Cadaver trachea and other tissues, fixed chemically, frozen, or lyophilized, have been used both experimentally and clinically as tracheal replacements. Such implantation has mistakenly been called "transplantation" or described as a "tracheal allograft," despite its nonviable and denatured status. "Bioprosthesis" may be a better term, since a fixed tissue is more akin to tanned leather than it is to transplanted tissue in the usual sense. Preserved or lyophilized tissues are usually replaced in time by host scar tissue, the rate possibly dependent upon the locus of implantation.[75] Dead tissue cannot be expected to function as a template for regeneration of the complex structure of the trachea, in accord with any basic biologic processes yet identified.

Experimentally, Scherer and colleagues processed rat, guinea pig, and pig tracheae chemically, including tanning with glutaraldehyde, and variously implanted tracheal segments and window patches as autografts, allografts, and xenografts.[7] Rejection seemed to be avoided. Variable long-term morphologic survival of cartilage and epithelization occurred in rats, but not in pigs. Pressman and Simon used sterilized and lyophilized aortic homografts over polyethylene tubes in dogs to exclude granulation tissue.[18] Stenosis was prevented by the stent, but linear contraction stretched and deformed the proximal and distal ends of the trachea. The damaged rings calcified and tracheal epithelium surfaced the aortic graft. Marrangoni in 1951 and Greenberg and Williams in 1960 observed that lyophilized canine tracheal allografts lost their cartilage and were replaced by scar tissue.[76,77] Björk and Rodriguez implanted freeze-dried, formalin-fixed, and 95% alcohol treated allografts, with fatal stenosis or necrosis in all.[26] Related laboratory experiences are described below (see Preserved Allografts). These are included in the section on tracheal transplantation, for completeness, since many investigators analyzed both fresh and preserved grafts in their experiments.

More recently, this avenue has been reexplored. *Clinically*, a cadaver tracheal graft, fixed in formalin and stored in Merthiolate, and then implanted and observed over 13 months, showed histologic disintegration and reduced allogenicity.[78] Jacobs and colleagues further described "tracheal allograft reconstruction" in adults and children, using cadaver trachea treated with 4% formalin, followed by thimerosal and stored in acetone.[79] The cartilaginous portion of long stenoses of various etiologies (or the anterolateral portion in the case of congenital "O" rings) were excised and replaced with the fixed tissue, supported by Dumon or Hood silicone intraluminal stents. Frequent bronchoscopy was necessary to remove exuberant granulation after stent removal, but complete epithelization was described after unspecified intervals, based on bronchoscopic visualization and histologic samples. Of 6 patients, 1 died of innominate artery fistula, 2 had tracheomalacia requiring stents, 1 with a stent replacement that was subsequently "recently" removed needed frequent bronchoscopy for granulations, 1 was stented with a tracheostomy tube, and finally, 1 who was operated upon 6 months previously, and who needed a tracheostomy tube, was decannulated for an unspecified time. In summary, 1 patient in 6 was free of stents with an adequate airway over the short term. Somewhat better results were cited for 18 of 31 children (58%) so treated in Europe, but detailed follow-up data were not given to permit full appreciation of their final status.[80]

If full epithelization does occur by migration of the tracheal epithelium from the intact posterior wall strip, it must be over host granulation tissue that has replaced the cadaveric graft. This would explain the

exuberant granulations described and also the development of serious malacia, as the dead cartilage is resorbed and replaced by cicatrix. It is biologically inconceivable that wholly dead cartilage is reconstituted as living cartilage. In effect, we appear to be dealing with an oversized patch tracheoplasty using fixed tissue rather than fresh autologous costal cartilage or pericardium. Indeed, in an earlier commentary, one of the authors stated that the graft "becomes very fibrotic according to the samples of the few patients...who have died.... There is no live cartilage in the donor trachea."[81]

Autogenous Tissues

Free Grafts With and Without Foreign Material Support

The use of autogenous tissue, such as the omentum, to seal mesh prostheses was noted earlier. Foreign materials have, on the other hand, been used to support free grafts of autogenous tissues, either as patches or in tubular form. *Experimental* patches or tubular constructions have used the following materials: fascia[11,46,82,83]; tracheal wall[84]; diced cartilage against wire mesh and glass cylinders[13]; dermal grafts laced with wire[14]; fascia with Tantalum wire support[8]; pericardium[85]; pericardium with Marlex, free periosteum with omental wrap[86,87]; bone strips and fibrocollagen[25]; cartilage and perichondrial strips over silicone stents[88]; costal cartilage, periosteum, and rib over a polyethylene stent[89]; periosteal patch applied to staggered intercartilaginous relaxing incisions, which was also applied clinically[90]; composite patches of buccal mucosa and auricular cartilage[91]; dura mater with wire[92]; bladder mucosa with Silastic or polyurethane stents,[93,94] with the epithelial face of the mucosa turned away from the lumen in order to form bone[69]; and jejunal patch with microvasclar reconstruction.[95] Tantalum-supported skin grafts, perichondrium from ear and rib, the latter on fascial flaps which formed cartilage, became epithelized, but stenosed.[77,96] Nasal cartilage with attached mucosa served as a patch in dogs.[97] The patch healed, but the cartilage was almost completely resorbed. The same occurred with tracheal wall.[84] Marshak and colleagues later interposed cylinders of bladder mucosa surfaced over Silastic tubes in dogs and achieved stable tubes lined with epithelium.[98] However, edema followed removal of the stent and caused death by obstruction. A combination of scar tissue formation, contraction, and epithelization has been described in the healing of pericardial patches, sometimes misinterpreted as regeneration of cartilage from scar tissue.[11,23,85,99]

Clinically, in 1943 and 1944, Belsey performed two radical tracheal resections for "cylindroma," leaving a narrow strip of tracheal wall intact and reconstructing the defect with fascia lata supported by a coil of steel wire.[3] Clagett and colleagues also used this technique.[35] A free bronchial patch and free fascia lata were also used in patients.[100,101] The use of wire-supported dermal grafts to widen bronchi and tracheae was extensively reported by Gebauer and others.[102,103] Larger dermal grafts for tracheal repair were frequently unsuccessful.[104,105] Dermal grafts wrapped around Tantalum tubes failed when used intrathoracically.[4] Caputo and Consiglio used a patch of auricular cartilage to widen the lower trachea.[106] Free fascial grafts in small tracheal windows healed and epithelized.[107] Crafoord and Lindgren used skin and cartilage to repair cervical tracheae in 1945.[108]

Costal cartilage and, later, pericardium were successfully employed as patch grafts for treatment of long congenital stenosis, without specific revascularization.[109,110] Only occasionally did necrosis occur.[111] Reepithelization was noted to occur.[99] Pericardium was replaced with mature scar tissue.[99] Tracheal growth was also reduced.[112] Even with addition of an omental pedicled flap, a cartilage patch is in time resorbed.[113]

Vascularized Autogenous Tissue Flaps

Another route of reconstruction, either as a flap for tracheal repair after lateral or "window" resection, or as a tube after circumferential resection, was to use the patient's own tissues, preserving or reanastomosing blood supply. Since relative rigidity is necessary, free grafts of cartilage, plastic rings, or meshes were added

for support. Foreign material or cartilage autografts were implanted in mesenchymal tissue. In general, lateral resection of tumor is not favored because of the likelihood of inadequate resection, as well as problems in healing of free tracheal patches, especially when intrathoracic. A vascularized patch is less likely to necrose than a free patch. On rare occasions, this technique is still used when a long segment of lateral tracheal wall must be removed to effect complete resection of an invasive secondary neoplasm. Where circumferential resection is not possible because of the length of neoplastic involvement, lateral excision of the wall and reconstruction with a pedicled pericardial flap, supported by materials such as Marlex, may be effective.

Experiments in *vascularized flap repair of window defects* have included use of intercostal muscle pedicled patch graft, pedicled periosteum, pedicled bronchus, and rib and pleural transfer with microvascular anastomosis for long anterior tracheal defects in dogs.[86,114–116] Half of these latter animals survived. Lofgren and colleagues performed, in three stages, a pedicled composite graft of buccal mucosa, Proplast (PTFE and pyrolithic graphite), with a connective tissue "external" layer, for repair of lateral tracheal defects in beagles.[117]

Clinically, Nowakowski in 1909 employed local skin flaps to close cervical tracheal defects.[118] Other flaps used included pedicled intercostal muscle and pleura, without the wire supports for flaps which had been previously used in dogs, periosteum on a muscular pedicle for lateral repair, rotated bronchus, pedicled diaphragm to posterior tracheal wall, and pedicled patch of pericardium supported by Marlex for repair of a long lateral defect (J. C. Wain, unpublished, c. 1998).[119–122] Pedicled intercostal periosteal flap was used to repair tracheoesophageal fistulas.[123]

The use of cartilage grafts and hyoid bone transfers, to enlarge the stenotic *larynx* either anteriorly or posteriorly, is a very large subject by itself and will not be discussed here.[124]

Autogenous Tube Construction

The cervical trachea has been reconstructed *experimentally* by formation of a cutaneous trough, variously supported by cartilage or plastic rings, with staged closure of the trough.[4,125,126] Edgerton and Zovickian reviewed early attempts at creation of skin flaps, including tubed pedicles, variously supported with rib or costal cartilage and sometimes lined with split grafts.[4] In 1964, Grillo and colleagues proposed a staged repair of the cervical trachea to replace a cervical tracheal segment devolved with its blood supply into the mediastinum for primary intrathoracic anastomosis.[127] A cutaneous tube was formed in stages, supported by polypropylene rings inserted between the dermis and the attached platysma.[126] Serrano and colleagues inserted bilaterally a series of hemi-rings of cartilage carved from the costal arch to provide support when a skin trough was finally closed anteriorly.[128] Drs. Joo Hyun Kim and H. C. Grillo (unpublished) constructed staged tracheal replacements in dogs by allowing the perichondrium of two costal cartilages to form a cartilaginous tube around a silicone mold in situ, later lining this tube with buccal mucosa and, finally, transferring it to a thoracic site with blood supply from the still-attached but mobilized intercostal muscle pedicles. As might be expected in such a complex repair, only a percentage of preparations fully succeeded. The work was, therefore, not published.

Papp and colleagues wrapped intercostal muscle, stiffened with cartilage and lined with skin around a stent, in dogs.[129] The complexity of the procedure led to high mortality. Krespi and colleagues wrapped a pleural periosteal flap around a Silastic stent and, after 6 weeks in a subcutaneous location, moved the tube to an orthotopic location, but with uncertain results.[130] Botta and Meyer, working on rats, prepared in multiple stages a connective tissue tube with allografts of tracheal cartilage, previously prepared to reduce antigenicity, around a Silastic tube, later lined with buccal mucosa, with regional supply from implantation, which would eventually be anastomosed by microsurgical technique to cervical vessels.[131] The complexity appears clinically daunting. Kon and Van den Hooff also produced cylinders of cartilage by wrapping rib perichondrium around a silicone rod implanted in tissue.[132]

Another line of experimental reconstruction with host tissue has been the use of adjacent esophagus to replace a long segment of trachea. Mural splinting is required to maintain patency, although in some experiments this, amazingly enough, seems to have been unneeded. In my laboratory, placement of a series of discontinuous polypropylene rings failed when the adjacent supported segments obstructed by valving across one another. A suggestion that an esophageal segment be substituted for trachea just to provide a channel for a T tube seems to overlook the magnitude of surgery that is then needed for esophageal replacement.[133] In an effort to avoid the requirement of major surgery for later esophageal replacement, revascularized segments of small intestine have also been employed experimentally.[134] Observation that a segment of small intestine remained rigid enough for normal respiration is difficult to understand if the interposition is of any length at all.

Clinically, multistaged cutaneous tubes have been widely employed for construction of cervical trachea. Multiple trough techniques have been devised and used.[4,135–144] Edgerton and Zovickian attained success in multistaged flap reconstruction of cervical trachea clinically.[4] Grillo described clinical application of his two-staged technique.[5] Although successful in a few patients, the technique is not recommended because of an overall low success rate. It should be noted that staged repairs of the type listed have no application intrathoracically, where an airtight airway must be attained immediately. Failure of healing, likely to occur in some cases after complex repairs, could cause fatal mediastinitis in the chest.

Esophagus in situ has also been used to patch the membranous tracheal wall and to serve as a long linear patch for congenital stenosis.[145,146] Fonkalsrud and colleagues unsuccessfully tried to replace the trachea for agenesis and for stricture with a segment of esophagus.[147,148]

Tissue Engineering

As described in the previous section, inducing formation of a cartilaginous tube by wrapping perichondrium around an inert cylinder (providing a lining with buccal mucosa or skin graft) is, in a sense, tissue engineering. However, current use of the term implies formation of tissue by cultivated cells introduced into a framework of biodegradable synthetic polymer, which guides the shape and size of a macrostructure, such as a tube.[149] Thus, chondrocytes, placed on a template of polyglycolic acid fibers after incubation and implantation, produced cartilage of selected shape and size over 4 weeks.[150] Vascularized connective tissue surrounded the implants and lined the inner surface. The structure resisted collapse and was used to replace segments of trachea in rats. In further experiments, effort was made to seed the inner surface with tracheal epithelial cells.[151] Work in this area continues. A combination of these two technical concepts, that is, the "growth" of a segment for tracheal replacement and its implantation, where the vascularized tube may later be swung into place, may in time become a useful technique. In benign disease, a T tube could provide an interim airway.

Tracheal Transplantation

Nonrevascularized Grafts

Autografts: Fresh and Devascularized. In 1918, Burket reported successful fresh autografts of three- to nine-ring segments in 4 of 8 dogs.[152] The other animals, plus two allografts, died with tracheal strictures. The latter died in 7 and 8 days in contrast with longer survival for autografts (except for one early "breakdown"). The author did not comment on this difference between autografts and allografts. Experiments have since shown that immediate orthotopic reimplantation of an animal's own trachea after excision, a *fresh autograft*, was most often unsuccessful, since regeneration of local blood supply is too slow to nourish the complex tissues of the trachea.[21,26,45,84,153-155] However, variable results have been reported experimentally with devascularized *autografts* of different lengths, with survival of the graft likely with short segments, or with bronchial patches placed in the trachea.[20,44,154,156] With very short segments replanted, animals survived, but

the cartilage was often resorbed and the segment converted to fibrous tissue.[30] With longer segments, dissolution, stenosis, and obstruction followed due to loss of blood supply.[154]

Mayer and colleagues found that continuous local infusion of basic fibroblast growth factor appeared to enhance early revascularization and epithelial regeneration in tracheal isograft segments in rats when implanted in the omentum.[157] Olech and colleagues, in contrast, detected no beneficial effect of growth factor, but basic fibroblast growth factor was administered as a single local application in a carrier.[158]

Allografts: Fresh and Devascularized. Fresh tracheal allografts without the aid of immunosuppression will initiate rejection.[156,159–161] Uniform failure of fresh allografts of any length occurs, even with immune suppression (Imuran, Decadron) in the absence of revascularization.[4,20,30,84,154] Neville and colleagues identified the same processes as seen with autografts, with survival only in very short grafts where the resulting fibrous tissue was too limited to obstruct the lumen.[154] With longer allografts, all their dogs died. Allografts of partial tracheal wall removal prevented obstruction, but grafts were replaced by flaccid scar.[162]

Fresh tracheal *allografts* were earlier demonstrated to necrose and liquefy, or if short, to result in fibrous stenosis.[6,44,45,156,163,164] Beigel and colleagues found from experimentation in inbred rats that tracheal transplants carry antigens and have an immunogenic action from the donor, and that mucosa is rejected and replaced by the recipient's cells, hence concluding that tracheal transplants follow the same rules as other tissues, in opposition to earlier belief that the trachea had weak antigenicity.[159,165] Bujia and colleagues also agreed that mucosa from the human trachea was likely the major antigenic structure responsible for the immunogenic action of tracheal allografts.[160]

Allografts: Preserved and Devascularized. In 1950, Jackson and colleagues found that partly deepithelized, Merthiolate-treated, and cold-preserved canine allografts failed in their repair of extensive defects.[6] Cartilage resorption and scar replacement produced collapse and obstruction. In 1952, Davies and colleagues found that fresh canine allografts, preserved in Tyrode's solution or in 4% formaldehyde, narrowed to complete obstruction in 1 to 3 weeks.[166] Epithelium and cartilage disappeared and fibrosis occurred. Pacheco and colleagues described the destruction and fibrosis of fresh allografts (short enough so that autografts in concurrent experiments survived), and the stenosis of allografts that were preserved in alcohol or saline or lyophilized.[156] Björk and Rodriguez confirmed this in grafts preserved in Tyrode's solution at –4°C, in lyophilized grafts, and in grafts formalinized or preserved in alcohol.[26] Keshishian and colleagues observed liquefaction and necrosis of canine allografts preserved in Tyrode's solution at –4°C.[13] Cryopreserved cartilaginous allografts, placed on tracheal window defects in piglets, appeared to survive with some resorption and to become epithelized.[167]

Keshishian and colleagues transplanted six-ring allografts in dogs after about 2 weeks of preservation in Tyrode's solution.[13] All necrosed. Diced autogenous costal cartilage molded between glass and stainless steel mesh survived in rectus muscle, but not as a tracheal replacement. When half the cartilage was allograft, the implant failed in muscle, too. Lenot and colleagues demonstrated in pigs the necrosis of devascularized tracheal *allografts* pretreated with glutaraldehyde, glycerol, lyophilization, or cryopreservation.[168] Immunosuppressive agents and steroids were not given. All died from resulting airway obstruction. Although there was a variation of response in some accordance with the methods of preservation, the results were uniformly disastrous/all leading to ischemic necrosis of the epithelium, submucosa, and cartilage.

This slow accumulation of evidence in both autografts and allografts treated in a variety of ways made it clear that blood supply was critical if successful tracheal transplantation was ever to be possible. Macchiarini summarized this as follows: "(D)evascularized tracheal allografts necrose whatever the conservation procedure. Only a living substitute, therefore vascularized, can pretend to fulfill the anatomic mechanical and anti-infectious functions of the trachea."[169]

Vascularized Grafts

Revascularization of an orthotopic tracheal graft can be accomplished *indirectly* with pedicled omentum, intercostal muscle, musculofascial flap, or other pedicles, or *directly* by vascular anastomoses.

Autografts: Fresh and Indirectly Vascularized. Hirata, Messineo, and Borro and their colleagues used omentopexy to revascularize free tracheal grafts in rats and in dogs, with considerable success.[170–172] Nakanishi and colleagues demonstrated that omental wrapping allowed fresh tracheal autografts to recover from the early ischemic changes as new vessels connected to the graft.[155] Morgan and colleagues had earlier demonstrated the neovascularizing potential of the omentum for bronchi.[173] Fell and colleagues found an intercostal pedicle flap to function similarly in bronchial autografts.[174] Li and colleagues noted better vascularization and survival of autografts, initially reimplanted in the omentum abdominally and then transferred 2 weeks later to a tracheal site on an omental pedicle.[175] However, even with omental revascularization, canine autografts longer than 4 cm (8 to 10 rings) frequently showed stenosis or dissolution in their central portions due to ischemia.[176] Introducing the omentum into an autograft split at the midpoint did, however, provide sufficient vascularity to prevent central graft necrosis.[177]

On the negative side, Balderman and Weinblatt did not find that omental wrapping sustained chondrocyte viability in eight-ring tracheal autografts in dogs.[178] Li and colleagues improved epithelial and chondrocyte survival by preliminary implantation of autografts into the omentum, about 2 weeks prior to transfer to an orthotopic location. Tojo and Mukaida and their colleagues showed survival of omental-wrapped autografts with a normal histologic structure.[179,180] Murai and colleagues removed cartilage rings from long autografts to improve the omental contact and revascularization of the epithelium.[181]

Allografts: Fresh or Preserved and Indirectly Vascularized. *Fresh allografts* with omental revascularization failed, most probably due to rejection.[178–180,182–184] Recovery of the epithelium from initial damage prior to establishment of vascular supply from the omentum, which is seen in autografts, failed to occur in allografts.[185] *Cryopreserved allografts* with omental flap revascularization survived without immunosuppression.[179,180,186] Cryopreservation seems to inhibit allogenicity while structural integrity appears to be maintained. Mukaida and colleagues observed a gradual replacement of graft epithelium by recipient epithelial cells after 50 to 60 days.[187] Deschamps and colleagues noted some deterioration of cartilage in allografts implanted in abdominal muscle for vascularization, despite cryopreservation.[188] Other tracheal elements seem to be unchanged. Moriyama and colleagues found that cryopreservation of allografts reduced acute rejection and permitted early revascularization, but that chronic rejection then led to vascular occlusion and atrophy.[189] Working with orthotopic cryopreserved tracheal allografts in rats without immune suppression, Tojo and colleagues also identified the late (2 months) graft epithelium to be of recipient origin, whereas the chondrocytes were of donor origin.[190] They concluded that the recipient origin of the epithelium over the donor trachea accounted for the reduced antigenicity of the cryotransplanted trachea. Inutsuka and colleagues noted the feasibility of cryopreservation of tracheocarinal allografts in dogs (for 3 weeks at –80°C) and subsequent viability in 75% without immunosuppression.[191] The effectiveness of cryopreservation is unexplained.

Based on the likelihood that the mucosa and not the cartilage is the major antigenic structure of the trachea, Liu and colleagues removed the epithelium with a detergent.[184] Omental revascularization was provided and the cartilage remained viable. Incorporation of allografts (without immunosuppression) without stenosis was observed, in contrast to granulation tissue formation and stenosis in untreated controls. Yokomise and colleagues reported long-term (greater than 1 year) survival of heavily irradiated five-ring allografts (100,000 cGy) with an omental pedicle for vascularization.[182] No immunosuppression was

given. This is likely the result of ablation of the tracheal epithelium by irradiation, since the epithelium seems to have strong antigens, whereas cartilage does not.[159,192]

Major histocompatibility complex antigens (MHC II, MHC I) are expressed in the epithelium and mixed glands of the trachea and not in cartilage.[160] Moriyama and colleagues found that *immunosuppression* was essential for tracheal graft survival along with omentopexy in dogs.[183] Davreux showed that tracheal allograft viability in rats, wrapped in omentum and heterotopically implanted, was improved with cyclosporin A.[193] Central portions of longer grafts were less well vascularized. Late endothelial repopulation by host cells was cited. The trachea survived where control skin grafts did not. Ueda and Shirakusa experienced failure in 4 of 6 carinal allografts in dogs with omental wrapping and immunosuppression.[194] Failure was attributed to failure of blood supply, even in short-segment grafts. Delaere and colleagues concluded that initial heterotopic tracheal transplantation (in rabbits) using a fascial vascular carrier flap, with a 2-week delay to orthotopic placement with immunosuppression, improved success in allotransplantation.[195,196]

Clinical tracheal allotransplantation has been tried in humans, with success reported in 1 patient after 9 weeks.[165] The donor trachea was first implanted heterotopically in the sternocleidomastoid muscle, and pedicled orthotopically in 3 weeks. No immunosuppression was employed. In another human long-segment allograft with omental revascularization and immunosuppression, necrosis and stenosis occurred, requiring a stent that could not be removed.[197] Its "success" must be questioned.

Direct Revascularization

Experimental. The arterial and venous supply of the trachea (see Chapter 1, "Anatomy of the Trachea") does not lend itself easily to direct revascularization. The vessels are of tiny diameter and segmental in distribution. Khalil-Marzouk faced this problem by preparing a composite thyrotracheal graft, anastomosing the thyroid artery to the common carotid artery.[198] Venous anastomosis was *not* done. In the absence of immunosuppression, the cartilage was preserved but the tracheal soft tissues necrosed. With cyclosporin and hydrocortisone, all tracheal tissues survived.

Macchiarini and colleagues, in a heterotopic pig model using cyclosporin, observed the preservation of tracheal grafts, including the epithelium, but only where both arterial and venous anastomoses were accomplished.[199] Venous infarction occurred in the absence of venous anastomosis. Khalil-Marzouk's observations are difficult to reconcile with this.[198] Grafts were 9 to 11 cm long. Isolated areas of submucosal necrosis, especially in the membranous wall, repaired within a week.[200] These investigators also explored experimental thyrotracheoesophageal transplantation, adding perfusion of the arterial system of the graft with Euro-Collin's solution.[201]

Clinical. In double-lung transplantation, direct bronchial artery revascularization has been done to prevent the still major threat of ischemia at the tracheal anastomosis. Couraud and colleagues harvested an aortic patch at the origin of the right intercostobrachial artery, connecting it to the recipient aorta with a saphenous vein graft.[202] Daly and colleagues anastomosed the largest bronchial artery in a patch of donor descending aorta to the left internal thoracic artery.[203]

Strome and colleagues described a fresh laryngeal transplantation with complete human leukocyte antigen matching, which included a five-ring segment of trachea, thyroid, and parathyroids, and a portion of attached pharyngeal wall, plus both superior laryngeal nerves and right recurrent nerve, to replace a totally scarred larynx due to trauma years before.[204] Arterial, venous, and neural anastomoses were performed, and perfusion was established early in the procedure. Over time, the patient regained vocal cord function and normal deglutition. Despite one episode of rejection, health and function were good at 40 months, with continued immunosuppression. This success, if generally repeatable, would seem to encour-

age a similar trial for the trachea. However, the quality of life issues involved in providing a functional larynx and voice are not comparable with providing a conduit for the trachea. Since tracheal transplantation would presently be applicable only to benign disease, the efficiency and safety of a T tube, currently in use, must be compared with the hazard of surgery and the chronic disease state of immunosuppression, now necessary for any tracheal transplantation.

Conclusions

The flood of experimental tracheal transplantation experiments in recent years, briefly noted above, has clarified some issues. A free autograft—an experimental procedure by definition—can sometimes succeed if it is short and if it is at least vascularized by omentum. Fresh allografts require immunosuppression to counteract rejection and will only succeed (and then somewhat unpredictably) with omental revascularization, if very short in length. Cryopreserved allografts may survive under the same conditions without immunosuppression, but for unknown duration. Optimal regimens for immunosuppression and preservation have not been established. Other techniques for reducing antigenicity have been tried in the laboratory. Long grafts show necrosis or proliferation of granulation tissue, progressing to stenosis in their center. Epithelization fails to proceed centrally. Arterial revascularization, because of the multiplicity and small size of blood vessels supplying the trachea, thus far demands transplantation of adjacent organs which share blood supply with the trachea; that is, the thyroid gland and at least part of the esophagus. With these present limitations, the prospects for a justifiable tracheal transplantation as a solution for extended tracheal resection, which is most often needed for malignant disease, remain remote.

References

1. Grillo HC. Notes on the windpipe. Ann Thorac Surg 1989;47:9–26.
2. Grillo HC. Tracheal replacement [editorial]. Ann Thorac Surg 1990;49:864–5.
3. Belsey R. Resection and reconstruction of the intrathoracic trachea. Br J Surg 1950;38:200–5.
4. Edgerton MT, Zovickian A. Reconstruction of the trachea and infraglottic larynx. Plast Reconst Surg 1954; 13:167–92.
5. Grillo HC. Circumferential resection and reconstruction of the mediastinal and cervical trachea. Ann Surg 1965;162:374–88.
6. Jackson TL, O'Brien EJ, Tuttle W, Meyer J. The experimental use of homogenous tracheal transplants in the restoration of continuity of the tracheobronchial tree. J Thorac Surg 1950;20:598–612.
7. Scherer MA, Ascherl R, Geissdorfer K, et al. Experimental biosynthetic reconstruction of the trachea. Arch Otorhinolarynol 1986;243:215–23.
8. Rob CG, Bateman GH. Reconstruction of the trachea and cervical oesophagus. Br J Surg 1949;37:202–5.
9. Grillo HC. Surgery of the trachea. Current problems in surgery. Chicago: Year Book Med; July 1970.
10. Morfit HM, Neerken AJ, Prevedel A, et al. Sleeve resection of the trachea. Experimental studies on regenerative capacity and principles of reconstruction and repair. Arch Surg 1955;70:654–61.
11. Daniel RA Jr. The regeneration of defects of the trachea and bronchi. An experimental study. J Thorac Surg 1948;17:335–49.
12. Wykoff TW. A preliminary report on segmental tracheal prosthetic replacement in dogs. Laryngoscope 1973;83:1072–77.
13. Keshishian JM, Blades B, Beattie EJ. Tracheal reconstruction. J Thorac Surg 1956;32:707–27.
14. Michelson E, Solomon R, Maun L, Ramirez J. Experiments in tracheal reconstruction. J Thorac Cardiovasc Surg 1961;41:748–59.
15. Longmire WP Jr. The repair of large defects of the trachea. Ann Otol Rhinol Laryngol 1948; 57:875–83.
16. Grindlay JH, Clagett OT, Moersch HJ. Resection and anastomosis of the trachea: an experimental study. Ann Otol Rhinol Laryngol 1949;58:1225–9.
17. Craig RL, Holmes GW, Shabart EJ. Tracheal resection and replacement with a prosthesis. J Thorac Surg 1953; 25:384–96.
18. Pressman JJ, Simon MB. Tracheal stretching and metaplasia of the tracheal rings from cartilage to bone following the use of aortic homografts. Am Surg 1959;25:850–6.
19. Shaw RR, Aslami A, Webb WR. Circumferential replacement of the trachea in experimental animals. Ann Thorac Surg 1968;5:30–5.
20. Spinazzola AJ, Graziano JL, Neville WE. Experimental reconstruction of the tracheal carina. J Thorac Cardiovasc Surg 1969;58:1–13.
21. Borrie J, Redshaw NR. Prosthetic tracheal replacement. J Thorac Cardiovasc Surg 1970;60:829–35.
22. Borrie J, Redshaw NR, Dobbinson TL. Silastic tracheal bifurcation prosthesis with subterminal Dacron suture cuffs. J Thorac Cardiovasc Surg 1973;65:956–62.
23. Demos NJ, Mitnick H, McCally D, et al. Tracheal regeneration in long-term survivors with silicone prosthesis. Ann Thorac Surg 1973;16:293–300.
24. Kramish D, Morfit HM. The use of a Teflon prosthesis to bridge complete sleeve defects in the human trachea. Am J Surg 1963;106:704–8.
25. Bailey BJ, Kosoy J. Observations in the development of tracheal prostheses and tracheal transplantation. Laryngoscope 1970;80:1553–65.
26. Björk VO, Rodriguez LE. Reconstruction of the trachea and its bifurcation. An experimental study. J Thorac Surg 1958;35:596–603.

27. Moncrief WH Jr, Salvatore JE. An improved tracheal prosthesis. Surg Forum 1958;9:350–2.
28. McCall JW, Whitaker CW. The use of prostheses in the larynx and trachea. Ann Otol Rhinol Laryngol 1962;71:397–403.
29. Cheng WF, Takagi H, Akutsu T. Prosthetic reconstruction of the trachea. Surgery 1969;65:462–9.
30. Aronstam EV, Nims RM, Winn DF Jr. Studies in segmental replacement of the thoracic trachea. J Surg Res 1961;1:108–10.
31. Jarvis FJ. Discussion. J Thorac Surg 1950;20:649.
32. Cotton BH, Penido JRF. Resection of the trachea for carcinoma. J Thorac Surg 1952;24:231–45.
33. Neville WE, Bolanowski PJP, Kotia GG. Clinical experience with the silicone tracheal prosthesis. J Thorac Cardiovasc Surg 1990;99:604–13.
34. Toomes H, Mickisch G, Vogt-Moykopf I. Experiences with prosthetic reconstruction of the trachea and bifurcation. Thorax 1985;40:32–7.
35. Clagett OT, Moersch HJ, Grindlay JH. Intrathoracic tracheal tumors: development of surgical techniques for their removal. Ann Surg 1952;136:520–30.
36. Atamanyuk MY, Melrose DG. The treatment of circumferential defects of the trachea. Br J Surg 1965;52:59–65.
37. Ekeström S, Carlens E. Teflon prosthesis in tracheal defects in man. Acta Chir Scand 1959;245(Suppl):71–5.
38. Clagett OT, Grindlay JH, Moersch HJ. Resection of the trachea: an experimental study and a report of a case. Arch Surg 1948;57:253–66.
39. Condon WB. Regeneration of tracheal and bronchial epithelium. J Thorac Surg 1941;11:333–46.
40. Wilhelm DL. Regeneration of tracheal epithelium. J Pathol Bacteriol 1953;65:543–50.
41. Bucher RM, Burnett E, Rosemond GP. Experimental reconstruction of tracheal and bronchial defects with stainless steel wire mesh. J Thorac Surg 1951; 21:572–83.
42. Greenberg SD, Williams RK. Tracheal prostheses. An experimental study in dogs. Arch Otolaryngol 1962; 75:335–41.
43. Poticha SM, Lewis FJ. Experimental replacement of the trachea. J Thorac Cardiovasc Surg 1966;52:61–7.
44. Carter MG, Strieder JW. Resection of the trachea and bronchi. An experimental study. J Thorac Surg 1950;20:613–27.
45. Ferguson DJ, Wild JJ, Wangensteen OH. Experimental resection of the trachea. Surgery 1950;28:597–619.
46. Swift EA, Grindlay JH, Clagett OT. The repair of tracheal defects with fascia and tantalum mesh. J Thorac Surg 1952;24:482–92.
47. Kiriluk LB, Merendino KA. An experimental evaluation in the dog of bronchial transplantation, bronchial, tracheal and tracheobronchial resection with reconstruction. Ann Surg 1953;137:490–503.
48. Mendak SH Jr, Jensik RJ, Haklin MF, Roseman DL. The evaluation of various bio-absorbable materials on the titanium fiber metal tracheal prosthesis. Ann Thorac Surg 1984;38:488–93.
49. Beall AC Jr, Harrington OB, Greenberg SD, Morris GC Jr. Tracheal replacement with heavy Marlex mesh. Arch Surg 1962;84:390–6.
50. Pearson FG, Henderson RO, Gross HE, et al. The reconstruction of circumferential tracheal defects with a porous prosthesis. An experimental and clinical study using heavy Marlex mesh. J Thorac Cardiovasc Surg 1968;55:605–16.
51. Sekine T, Nakamura T, Matsumoto K, et al. Carinal reconstruction with a Y-shaped collagen-conjugated prosthesis. J Thorac Cardiovasc Surg 2000;119:1162–8.
52. Okumura N, Nakamura T, Natsume T, et al. Experimental study on a new tracheal prosthesis made from collagen-conjugated mesh. J Thorac Cardiovasc Surg 1994;108:337–45.
53. Teramachi M, Okumura N, Nakamura T, et al. Intrathoracic tracheal reconstruction with a collagen-conjugated prosthesis: evaluation of the efficacy of omental wrapping. J Thorac Cardiovasc Surg 1997;113:701–11.
54. Bottema JR, Wildevuur CRH. Incorporation of microporous Teflon tracheal prostheses in rabbits: evaluation of surgical aspects. J Surg Res 1986;41:16–23.
55. Shaha A, MiMaio T, Morrey S, et al. Prosthetic reconstruction of the trachea. Am J Surg 1988;156:306–9.
56. Cull DL, Lally KP, Mair EA, et al. Tracheal reconstruction with polytetrafluoroethylene graft in dogs. Ann Thorac Surg 1990;50:899–901.
57. Guijarro Jorge R, Sanchez-Palencia Ramos A, Cueto Ladrón de Guevara A, et al. Experimental study of a new porous prosthesis. Ann Thorac Surg 1990;50:281–7.
58. Nelson RJ, Goldberg L, White RA, et al. Neovascularity of a tracheal prosthesis/tissue complex. J Thorac Cardiovasc Surg 1983;86:800–8.
59. Taber RE, Tomatis L. Experimental and clinical utilization of a prosthesis for replacement of the trachea. Arch Surg 1958;77:576–83.
60. Yasargil EE, Hess R. Beitrag zur plastichen. Überbrückung der thoracalen trachealdefekte. Thoraxchirurg 1958;5:470–84.
61. Jacobs J. Investigations into tracheal prosthetic reconstruction. Laryngoscope 1988;98:1239–45.
62. Kaiser D. Alloplastic replacement of canine trachea with Dacron. Thorac Cardiovasc Surg 1985;33:239–43.
63. Worman LW, Starr C, Narodick BG. Tracheoplasty techniques for reconstruction after extensive resection. Am J Surg 1966;111:819.
64. McCaughan JS Jr. Tracheal reconstruction with Silastic reinforced Dacron. Arch Otolaryngol 1968;87:150–1.
65. Trojján I, Kecskés L, Vécsei B, et al. Tracheal substitution in dogs with reinforced Gore-tex prosthesis. Thorac Cardiovasc Surg 1985;33:337–40.
66. Leake D, Habal M, Pizzoferrato A, Vespucci A. Prosthetic replacement of large defects of the cervical trachea in dogs. Biomaterials 1985;6:17–22.
67. Podoshin L, Fradis M. Reconstruction of the anterior wall of the cervical trachea using knitted Dacron. Ear Nose Throat J 1976;55:294–6.
68. Cahan WG. Carcinoma of intrathorcic trachea: excision and repair by tantalum gauze-fascia lata graft. J Thorac Surg 1952;23:513–27.
69. Edgerton M. One stage reconstruction of the cervical esophagus or trachea. Surgery 1952;31:239–50.
70. Ellis PR Jr, Harrington OB, Beall AC Jr, DeBakey ME. The use of heavy Marlex mesh for tracheal reconstruction following resection for malignancy. J Thorac Cardiovasc Surg 1962;44:520–7.
71. Beall AC Jr, Harrington OB, Greenberg SD. Tracheal reconstruction with heavy Marlex mesh. Arch Surg 1963;86:970–8.
72. Yeh TJ, Batayias G, Peters H, Ellison RG. Metastatic carcinoma to the trachea: report of a case of palliation by resection and Marlex graft. J Thorac Cardiovasc Surg 1965;49:886–92.
73. Moghissi K. Tracheal reconstruction with prosthesis of Marlex mesh and pericardium. J Thorac Cardiovasc Surg 1973;69:499–506.
74. Pagliero KM, Shepherd MP. Use of stainless steel wire coil prosthesis in treatment of anastomotic dehiscence after cervical tracheal resection. J Thorac Cardiovasc Surg 1974;67:932–5.
75. Grillo HC, McKhann CF. The acceptance and evolution of dermal homografts freed of viable cells. Transplantation 1964;2:48–59.
76. Marrangoni AG. Homotransplantation of tracheal seg-

ments preserved by lyophilization; an experimental study. J Thorac Surg 1951;21:398–401.
77. Greenberg SD, Williams RK. Tracheal reconstruction: an experimental study. Arch Otolaryngol 1960;72:565–74.
78. Bujia J, Pitzke P, Krombach F, et al. Immunological behavior of preserved human tracheal allografts: immunological monitoring of a human tracheal recipient. Clin Transplant 1991;5:376–80.
79. Jacobs JP, Elliott MJ, Haw MP, et al. Pediatric tracheal homograft reconstruction: a novel approach to complex tracheal stenosis in children. J Thorac Cardiovasc Surg 1996;112:1549–60.
80. Jacobs JP, Quintessenza JA, Andrews T, et al. Tracheal allograft reconstruction: the total North American and worldwide pediatric experiences. Ann Thorac Surg 1999;68:1043–52.
81. Elliott MJ, Haw MP, Jacobs JP, et al. Tracheal reconstruction in children using cadaveric homograft trachea. Eur J Cardio-Thorac Surg 1996;10:707–12.
82. Davis JS. The transplantation of free flaps of fascia. An experimental study. Ann Surg 1911;54:734–48.
83. Taffel M. The repair of tracheal and bronchial defects with free fascia grafts. Surgery 1940;8:56–71.
84. Daniel RA Jr, Taliaferro RM, Schaffarzick WR. Experimental studies on the repair of wounds and defects of the trachea and bronchi. Dis Chest 1950;17:426–41.
85. Bryant LR. Replacement of tracheobronchial defects with autogenous pericardium. J Thorac Cardiovasc Surg 1964;48:733–40.
86. Fonkalsrud EW, Plested WG, Mulder DG. Tracheobronchial reconstruction with autologous periosteum. J Thorac Cardiovasc Surg 1966;52:666–74.
87. Cohen RC, Filler RM, Konuma K, et al. A new model of tracheal stenosis and its repair with free periosteal grafts. J Thorac Cardiovasc Surg 1986;92:296–304.
88. Eckersberger F, Moritz E, Wolner E. Circumferential tracheal replacement with costal cartilage. J Thorac Cardiovasc Surg 1987;94:175–80.
89. Sato R, Hasegawa I, Nakagawa J. Experimental study of tracheal reconstruction. J Thorac Surg 1957;34:526–38.
90. Narodick BG, Worman LW, Pemberton AH. Relaxation technique for tracheal reconstruction. Ann Thorac Surg 1965;1:190–6.
91. Farkas LG, Farmer AW, McCain WG, Wilson WD. Replacement of a tracheal defect in the dog by a preformed composite graft. Plast Reconstr Surg 1972;50:238–41.
92. Sabás AA, Uez JB, Rojas O, et al. Replacement of the trachea with dura mater. Experimental work. J Thorac Cardiovasc Surg 1977;74:761–5.
93. Rush BF, Clifton EE. Experimental reconstruction of the trachea with bladder mucosa. Surgery 1956;40: 1105–10.
94. Barker W, Litton W. Bladder osteogenesis aids tracheal reconstruction. Arch Otolaryngol 1973;98:422–5.
95. Jones RE, Morgan RF, Marcella KL, et al. Tracheal reconstruction with autogenous jejunal microsurgical transfer. Ann Thorac Surg 1986;41:636–8.
96. Ohlsen L, Nordin U. Tracheal reconstruction with perichondrial grafts. An experimental study. Scand J Plast Reconstr Surg 1976;10:135–45.
97. Drettner B, Lindholm CE. Experimental tracheal reconstruction with composite graft from nasal septum. Acta Otolaryngol Scand 1970;70:401–7.
98. Marshak G, Porter JH, McAdams AJ. Reconstruction of the canine trachea with urinary bladder wall. Laryngoscope 1973;83:1090–5.
99. Cheng ATL, Backer CL, Holinger LD, et al. Histopathologic changes after pericardial patch tracheoplasty. Arch Otolaryngol Head Neck Surg 1997;123:1069–72.
100. MacHale SJ. A new technique for repairing the major air passages. J Thorac Cardiovasc Surg 1972;64:6–10.
101. Levit H. Deckung von Trachealdefecten durch line freie Plastik aus der Fascia lata femoris. Arch Klin Chir 1912;97:686–99.
102. Gebauer PW. Further experiences with dermal grafts for healed tuberculous stenosis of the bronchi and trachea. J Thorac Surg 1950;20:628–51.
103. Evans BH. Myxochondroma of the trachea. A case report. J Thorac Surg 1951;22:585–91.
104. Paulson DL. Plastic reconstruction of the trachea and bronchi. Am Rev Tubercul 1951;64:477–88.
105. Abbott OA, Van Fleit WE, Roberto AE. Experiences with extending the indications for the use of tracheal and bronchial grafts. J Thorac Surg 1955;29:217–37.
106. Caputo V, Consiglio V. The use of patient's own auricular cartilage to repair deficiency of the tracheal wall. J Thorac Cardiovasc Surg 1961;41:594–6.
107. Neuhof H. Fascia transplantation into visceral defects. An experimental and clinical study. Surg Gynecol Obstet 1917;24:383–427.
108. Crafoord C, Lindgren AGH. Mucous and salivary gland tumours in the bronchi and trachea. Acta Chir Scand 1945;92:481–506.
109. Kimura K, Mukohara N, Tsugawa C, et al. Tracheoplasty for congenital stenosis of the entire trachea. J Pediatr Surg 1982;17:869–71.
110. Idriss FS, DeLeon SY, Ilbawi MN, et al. Tracheoplasty with pericardial patch for extensive tracheal stenosis in infants and children. J Thorac Cardiovasc Surg 1984;88:527–36.
111. Backer CL, Mavroudis C, Dunham CE, et al. Re-operation after pericardial patch tracheoplasty. J Pediatr Surg 1997;32:1108–12.
112. Dykes EH, Bahoric A, Smith C, et al. Reduced tracheal growth after reconstruction with pericardium. J Pediatr Surg 1990;25:25–9.
113. Isugawa C, Nishijima E, Muraji T, et al. The use of omental pedicle flap for tracheobronchial reconstruction in infants and children. J Pediatr Surg 1991;26:762–5.
114. Blair E. Study of the viable intercostal pedicle graft in tracheobronchial surgery. J Thorac Surg 1958;36: 869–78.
115. Akl BF, Mittelman J, Smith D, Butler C. A new method of tracheal reconstruction. Ann Thorac Surg 1983; 36:265–9.
116. Watson J, Donald PJ, Gourley IM, et al. Composite vascularized free-rib and pleural transfer for laryngotracheal reconstruction. Otolaryngol Head Neck Surg 1983;91:384–95.
117. Lofgren LA, Lindholm CE, Jansson B. Reconstruction of the airway with a composite alloplastic and autogenous graft. Acta Otolaryngol 1985;100:140–50.
118. Nowakowski K. Beitrag zur tracheoplastik. Arch Klin Chir 1909;90:847–61.
119. Penton RS, Brantigan OC. The use of a viable pedicle graft for repairing an extensive tracheobronchial defect. Ann Surg 1952;135:709–14.
120. Narodick BG, Pemberton AH, Worman LW. Tracheoplasty by means of a periosteal pedicle graft. J Thorac Cardiovasc Surg 1964;47:572–6.
121. Kergin FG. Carcinoma of the trachea. J Thorac Surg 1952;23:164–8.
122. Westaby S, Shepherd MP, Nohl-Oser HC. The use of diaphragmatic pedicle grafts for reconstructive procedures in the esophagus and tracheobronchial tree. Ann Thorac Surg 1982;33:486–90.
123. Ross RR. Repair of tracheal and esophageal defect by use of a pedicle graft. Surgery 1956;39:654–62.
124. Cotton RT. The problem of pediatric laryngotracheal stenosis: a clinical and experimental study on the efficacy of autogenous cartilaginous grafts placed between the vertically divided halves of the posterior

lamina of the cricoid cartilage. Laryngoscope 1991;101(Suppl):1–34.
125. Fairchild FR. Reconstruction of the trachea. Surg Gynecol Obstet 1927;44:119–22.
126. Grillo HC, Dignan EF, Miura T. Experimental reconstruction of cervical trachea after circumferential resection. Surg Gynecol Obstet 1966;122:733–8.
127. Grillo HC, Dignan EF, Miura T. Extensive resection and reconstruction of mediastinal trachea without prosthesis or graft: an anatomical study in man. J Thorac Cardiovasc Surg 1964;48:741–9.
128. Serrano A, Ortiz-Monasterio F, Andrade-Pradillo J. Reconstruction of the cervical trachea: a technique to obtain a permanently patent airway. Plast Reconst Surg 1959;24:333–40.
129. Papp C, McCraw JB, Arnold PG. Experimental reconstruction of the trachea with autogenous materials. J Thorac Cardiovasc Surg 1985;90:13–20.
130. Krespi YP, Siller HF, Baek SM. Tracheal reconstruction with a pleuroperiosteal flap. Otolaryngol Head Neck Surg 1983;91:610–4.
131. Botta Y, Meyer R. Reconstruction expérimentale de la trachée par microchirurgie. Praxis 1978;67:1588–92.
132. Kon M, Van den Hooff A. Cartilage tube formation by perichondrium: a new concept for tracheal reconstruction. Plast Reconstr Surg 1983;72:791–7.
133. Kato R, Onuki AS, Watanabe M, et al. Tracheal reconstruction by esophageal interposition: an experimental study. Ann Thorac Surg 1990;49:951–4.
134. Letang E, Sánchez-Lloret J, Gimferrer JM, et al. Experimental reconstruction of the canine trachea with a free revascularized small bowel graft. Ann Thorac Surg 1990;49:955–8.
135. LeJeune F, Owens N. Chronic laryngeal stenosis. Ann Otol Rhinol Laryngol 1935;44:354–63.
136. Conley JJ. Rehabilitation of the airway system by neck flaps. Ann Otol Rhinol Laryngol 1962;71:404–10.
137. Bryce D, Lawson V. The "trough" method of laryngotracheal reconstruction. Ann Otol Rhinol Laryngol 1967;76:793–803.
138. Kapur TR. Tracheal injury and reconstruction. Br J Surg 1967;54:689–94.
139. Keim WF. Total reconstruction of the cervical trachea. Arch Otolaryngol 1970;91:501–4.
140. Williams GT. New material for a prosthesis used in tracheal reconstruction. J Laryngol Otol 1974;88:1175–84.
141. Friedman WA, Biller HF, Som ML. Repair of extended laryngotracheal stenosis. Arch Otolaryngol 1975; 101:152–6.
142. Meyer R, editor. Reconstructive surgery of the trachea. New York: Thieme; 1982.
143. Biller HF, Lawson W, Weisberg V. Staged repair of extensive tracheal and laryngotracheal stenosis. Ann Otol Rhinol Laryngol 1986;95:586–9.
144. Eliachar I, Goldsher M, Moscona AR, Hurwitz DJ. Reconstruction of the larynx and cervical trachea with the pectoralis major myocutaneous island flap. Head Neck Surg 1984;6:880–3.
145. Ein SH, Friedberg J, Williams WG, et al. Tracheoplasty—a new operation for complete congenital tracheal stenosis. J Pediatr Surg 1982;17:872–8.
146. Sasaki S, Hara F, Oowa T, et al. Esophageal tracheoplasty for congenital tracheal stenosis. J Pediatr Surg 1992;27:645–9.
147. Fonkalsrud EW, Martelle RR, Maloney JV Jr. Surgical treatment of tracheal agenesis. J Thorac Cardiovasc Surg 1963;45:520–5.
148. Fonkalsrud EW, Suinida S. Tracheal replacement with autologous esophagus for tracheal stricture. Arch Surg 1971;102:139–42.
149. Mooney DJ, Vacanti JP. Tissue engineering using cells and synthetic polymers. Transpl Rev 1993;7:153–62.
150. Vacanti CA, Paige KT, Kim WS, et al. Experimental tracheal replacement using tissue engineered cartilage. J Pediatr Surg 1994;29:201–5.
151. Sakata J, Vacanti CA, Schloo GC, et al. Tracheal composite tissue engineered from chondrocytes, tracheal epithelial cells and synthetic biodegradable scaffolding. Transplant Proc 1994;26:3309–10.
152. Burket WC. Transplantation of the trachea. Bull Johns Hopkins Hosp 1918;29:35–40.
153. Strandness DE Jr, Gustafson IJ, Payne JT. Surgical resection of the thoracic trachea: an experimental study in dogs. J Thorac Surg 1957;34:269–77.
154. Neville WE, Bolanowski PJP, Soltanzadeh H. Homograft replacement of the trachea using immunosuppression. J Thorac Cardiovasc Surg 1976;72:596–601.
155. Nakanishi R, Shirakusa T, Takachi T. Omentopexy for tracheal autografts. Ann Thorac Surg 1994;57:841–5.
156. Pacheco CR, Rivero O, Porter JK. Experimental reconstructive surgery of the trachea. J Thorac Surg 1954;27:554–64.
157. Mayer E, Cardoso PFG, Puskas JD, et al. The effect of basic fibroblast growth factor and omentopexy on revascularization and epithelial regeneration of heterotopic rat tracheal isografts. J Thorac Cardiovasc Surg 1992;104:180–8.
158. Olech VM, Keshavjee SH, Chamberlain DW, et al. Role of basic fibroblast growth factors in revascularization of rabbit tracheal autografts. Ann Thorac Surg 1991;52:258–64.
159. Beigel A, Steffins-Knutzen R, Muller B, et al. Tracheal transplantation. III. Demonstration of transplantation antigens on the tracheal mucosa of inbred rat strains. Arch Otorhinolaryngol 1984;241:1–8.
160. Bujia J, Wilmas E, Hammer C, Kastenbauer E. Tracheal transplantation: demonstration of HLA class II subregion gene products on human trachea. Acta Otolaryngol (Stockh) 1990;110:149–54.
161. Kalb TH, Chuang MT, Marom Z, Mayer L. Evidence for accessory cell function by class II MHC antigen-expressing airway epithelial cells. Am J Respir Cell Mol Biol 1991;4:320–9.
162. Alonso WA, Bridger GP, Bordley JE. Tracheal transplantation in dogs. Laryngoscope 1972;82:204–9.
163. Greenberg SD. Tracheal homografts in dogs. Am Med Assoc Arch Otolaryngol 1958;67:577–86.
164. Harrington WT, Hung WC, Binns PM. Experimental tracheal homografting. J Laryngol Otol 1977;91:101–10.
165. Rose KG, Sesterhenn K, Wustrow F. Tracheal allotransplantation in man. Lancet 1979;1:433.
166. Davies OG, Edmiston JM, McCorkle HJ. The repair of experimental tracheal defects with fresh and preserved homologous tracheal grafts. J Thorac Surg 1952;23:367–76.
167. Messineo A, Filler RM, Bahoric A, Smith CR. Repair of long tracheal defects with cryopreserved cartilaginous allografts. J Pediatr Surg 1992;27:1131–5.
168. Lenot B, Macchiarini P, Dulmet E, et al. Tracheal allograft replacement. Eur J Cardio-Thorac Surg 1993;7:648–52.
169. Macchiarini P. La transplantation de trachée et trachéo-oesophagienne [thesis]. Berançon: Université de Franche-Comte; 1997.
170. Hirata T, Yamazaki F, Fukuse T, et al. Omentopexy for revascularization of free tracheal grafts in rats. Thorac Cardiovasc Surg 1992;40:178–81.
171. Messineo A, Filler RM, Bahoric B, et al. Successful tracheal autotransplantation with a vascularized omental flap. J Pediatr Surg 1991;26:1296–300.
172. Borro JM. Successful revascularization of large isolated tracheal segments. Eur J Cardio-Thorac Surg 1992; 6:621–3.
173. Morgan E, Lima O, Goldberg M, et al. Successful revascu-

larization of totally ischemic bronchial autografts with omental pedicle flaps in dogs. J Thorac Cardiovasc Surg 1982;84:204–10.
174. Fell SC, Mollenkopf FP, Montefusco CM, et al. Revascularization of ischemic bronchial anastomosis by an intercostal pedicle flap. J Thorac Cardiovasc Surg 1985;90:172.
175. Li J, Xu H, Chen H, et al. Improvement of tracheal autograft survival with transplantation into the greater omentum. Ann Thorac Surg 1995;60:1592–6.
176. Nakanishi R, Shirakusa T, Mitsudomi T. Maximum length of tracheal autografts in dogs. J Thorac Cardiovasc Surg 1993;106:1081–7.
177. Yokomise H, Inui K, Wada H, et al. Split transplantation of the trachea: a new operative procedure for extended tracheal resection. J Thorac Cardiovasc Surg 1996;112:314–8.
178. Balderman SC, Weinblatt G. Tracheal autograft revascularization. J Thorac Cardiovasc Surg 1987;94:434–41.
179. Tojo T, Niwaya K, Sawabata N, et al. Tracheal replacement with cryopreserved tracheal allograft: experiments in dogs. Ann Thorac Surg 1998;66:209–13.
180. Mukaida T, Shimizu N, Aoe M, et al. Experimental study of tracheal allotransplantation with cryopreserved grafts. J Thorac Cardiovasc Surg 1998;116:262–6.
181. Murai K, Oizumi H, Masaoka T, et al. Removal of cartilage rings of the graft and omentopexy for extended tracheal autotransplantation. Ann Thorac Surg 1999;67:776–80.
182. Yokomise H, Inui K, Wada H, et al. High dose irradiation prevents rejection of canine tracheal allografts. J Thorac Cardiovasc Surg 1994;107:1391–7.
183. Moriyama S, Shimizu N, Teramoto S. Experimental tracheal allotransplantation using omentopexy. Transplant Proc 1989;21:2596–600.
184. Liu Y, Nakamura T, Yamamoto Y, et al. Immunosuppressant-free allotransplantation of the trachea: the antigenicity of tracheal grafts can be reduced by removing the epithelium and mixed glands from the graft by detergent treatment. J Thorac Cardiovasc Surg 2000; 120:108–14.
185. Nakanishi R, Shirakusa T, Hanagiri T. Early histopathologic features of tracheal allotransplant rejection. A study in nonimmunosuppressed dogs. Transplant Proc 1994;26:3715–8.
186. Yokomise H, Inui K, Wada H, et al. Long-term cryopreservation can prevent rejection of canine tracheal allografts with preservation of graft viability. J Thorac Cardiovasc Surg 1996;111;930–4.
187. Mukaida T, Shimizu N, Aoe M, et al. Origin of regenerated epithelium in cryopreserved tracheal allotransplantation. Ann Thorac Surg 1998;66:205–8.
188. Deschamps C, Trastek VE, Ferguson JL, et al. Cryopreservation of canine trachea: functional and histological changes. Ann Thorac Surg 1989;47:208–12.
189. Moriyama H, Sasajima T, Hirata S, et al. Revascularization of canine cryopreserved tracheal allografts. Ann Thorac Surg 2000;69:1701–6.
190. Tojo T, Kitamura S, Gojo S, et al. Epithelial regeneration and preservation of tracheal cartilage after tracheal replacement with cryopreserved allograft in the rat. J Thorac Cardiovasc Surg 1998;116:624–7.
191. Inutsuka K, Kawahara K, Takachi T, et al. Reconstruction of trachea and carina with immediate or cryopreserved allografts in dogs. Ann Thorac Surg 1996; 62:1480–4.
192. Gibson T, Davis WB, Curran RC. The long-term survival of cartilage homografts in man. Br J Plast Surg 1959; 131:177–87.
193. Davreux CJ. Improved tracheal allograft viability in immunosuppressed rats. Ann Thorac Surg 1993; 55:131–4.
194. Ueda H, Shirakusa T. Carinal transplantation. Thorax 1992;47:968–70.
195. Delaere PR, Liu ZY, Hermans R, et al. Experimental tracheal allograft revascularization and transplantation. J Thorac Cardiovasc Surg 1995;110:728–37.
196. Delaere PR, Liu Z, Sciot R, Welvaart W. The role of immunosuppression in the long term survival of tracheal allografts. Arch Otolaryngol Head Neck Surg 1996;122:1201–8.
197. Levashov YN, Yablonsky PK, Cherny SM, et al. One-staged allotransplantation of thoracic segment of the trachea in a patient with idiopathic fibrosing mediastinitis and marked tracheal stenosis. Eur J Cardio-Thorac Surg 1993;7:383–6.
198. Khalil-Marzouk JF. Allograft replacement of the trachea. Experimental synchronous revascularization of composite thyrotracheal transplant. J Thorac Cardiovasc Surg 1993;105:242–6.
199. Macchiarini P, Lenot B, de Montpréville V, et al. Heterotopic pig model for direct revasularization and venous drainage of tracheal allografts. J Thorac Cardiovasc Surg 1994; 108:1066–75.
200. Macchiarini P, Mazmanian G-M, de Montpréville VT, et al. Maximal preservation time of tracheal allografts. Ann Thorac Surg 1995;60:1597–604.
201. Macchiarini P, Mazmanian G-M, de Montpréville VT, et al. Experimental tracheal and tracheoesophageal allotransplantation. J Thorac Cardiovasc Surg 1995;110: 1037–46.
202. Couraud L, Baudet E, Martigne C, et al. Bronchial revascularization in double-lung transplantation: a series of eight patients. Ann Thorac Surg 1992;53:88–94.
203. Daly RC, Tadjikarimi S, Khagani A, et al. Successful double-lung transplantation with direct bronchial artery revascularization. Ann Thorac Surg 1993;56:885–92.
204. Strome M, Stein J, Esclamado R, et al. Laryngeal transplantation and 40 month follow-up. N Engl J Med 2001;344:1676–9.

Index