Development of Tracheal Surgery: A Historical Review. Part 2: Treatment of Tracheal Diseases

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This article is the second of a two-part historical review about the development of tracheal surgery. Part 1, "Techniques of Tracheal Surgery," appeared in the preceding issue of *The Annals*. Part 2 is a review of treatments of specific diseases of the trachea. There is, of course, considerable overlap. References for both parts are numbered sequentially. See "Selected References" at the end of this article for further explanation.

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 additional techniques to specific diseases of the airways will now be considered. The challenge of treating the rare tracheal tumors that 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 [65] collected 433 reported cases of primary tracheal tumors, beginning with Lieutaud's discovery of fibroma at autopsy in 1767. 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 one carcinoma in 9,000 autopsies at McGill University and one in 12,700 autopsies at Montreal General Hospital. Ellman and Whittaker [66] raised the total to 507 in 1947. "Cylindroma" was often classified as adenocarcinoma, and tracheopathia osteoplastica was included as a tumor. Houston and colleagues [194] collected 53 primary cancers of trachea in more than 30 years at the 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. Reporting a 30 years' experience in 1969, only two squamous cancers had been removed, one by lateral excision; six adenoid cystic cancer were also removed, none by circumferential resection and anastomosis and one mucoepidermoid by end-to-end repair. The next year, Hajdu and associates [195] described 41 patients with primary tracheal carcinoma who were treated over a span of more than 33 years—30 squamous and seven adenoid cystic carcinomas. 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 [73] resected a cervical tracheal epithelioma in 1957 with end-to-end suture. Forster and Holderbach [196] in 1960 published a voluminous report on pathology and clinical presentation of tracheal tumors, and of experimental and a few clinical trials at that early date. Nonneoplastic lesions were also included. Grillo [85] recounted treatment of three primary tumors by circumferential resection in 1965, using cross-table ventilation through the open trachea. Mathey and associates [41] reported resecting five primary tracheal neoplasms in 1966, with one early and one late postoperative death. Perelman and Korolyova [89] successfully treated 5 patients with primary tracheal intrathoracic cancer by circular resection and anastomosis in 1968. They introduced an anesthesia tube into the left main bronchus through an incision in the membranous wall of the right main bronchus. Dor and associates [93] in 1971 resected tracheal tumors in 6 patients, with three postoperative deaths. By 1973, Grillo [197] had excised 11 primary tumors and five secondary tumors in a series of 100 tracheal resections with reconstruction. Nine of the 11 patients were alive without disease; 1 patient died after the operation.

Experience with surgical management began to grow. In 1974, Eschapasse [128] reported on 152 patients with primary tracheal tumors treated by 12 French and two Russian groups. Among the treatments were 32 circumferential resections and 18 carinal reconstructions. The highest postoperative mortality and the poorest long-term results were associated with squamous carcinoma. Adenoid cystic carcinoma showed prolonged survival, but late recurrence. Also in 1974, Pearson and associates [198] accomplished five resections of adenoid cystic carcinoma with primary anastomosis, without postoperative death. In 6 other patients, a prosthetic replacement was constructed with Marlex (Chevron Phillips Chemical Company LP, Houston, TX). Grillo [87] by 1978 reported seeing 63 patients with primary tumors. Nineteen patients with primary tumors (and 5 more with secondary tumors) underwent cylindrical resection and anastomosis; 10 patients underwent carinal resection and reconstruction; 10 underwent staged reconstruction; 10 underwent laryngotracheal resection or were treated by other means. Two patients died after cylindrical resection and 3 after carinal resection and reconstruction.

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Subsequent major series began to define the long-term oncologic expectation. Grillo and Mathisen [6] in 1990 reported the largest single institutional series of 198 primary tumors treated at Massachusetts General Hospital over more than 26 years. Resection rates were 63% for squamous and 75% for adenoid cystic carcinoma. Pearson and colleagues [138] reported their experience with 44 tracheal tumors in 1984 and a subsequent report did the same for 38 adenoid cystic carcinomas [199] in 1996. That same year, Regnard and colleagues [200] provided an account of 208 patients in a multicenter series in France, and Perelman and colleagues [201] summarized their experience with 144 primary tumors.

Squamous and adenoid cystic carcinoma together compose about three-quarters of all primary tracheal tumors, in comparable numbers. Squamous cancer of the trachea has behaved in etiology, curability, and associated aerodigestive carcinoma much like squamous lung cancer. Surgery for adenoid cystic carcinoma, combined with radiotherapy, improved 5-year survival rates, but was associated with a continued decrease in survival at 10 years and thereafter, because of local recurrence and the appearance of metastases. The idiosyncratic and prolonged course of the rare adenoid cystic carcinoma clearly requires prolonged observation for complete clinical definition. A wide variety of tumors of other histology, most often benign or of low-grade malignancy, compose the remaining one-quarter of cases of primary tracheal tumor [6]. Operative mortality in all patients ranged from 5.3% to 10.5% in various series. Mortality and morbidity decreased with surgical experience [185], but remained highest for carinal reconstruction.

Secondary Tracheal Tumors

Resection of the carina for bronchogenic carcinoma was discussed last month in Part 1 of this review (under "Carinal Pneumonectomy"). The trachea and lower larynx are susceptible to invasion by thyroid cancer because of the proximity of the gland [202]. Localized tracheal invasion by thyroid neoplasms has been resected episodically as tracheal surgery evolved. Rob and Bateman [31] 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. Tantalum gauze-fascia lata reconstruction was carried out, leaving a strip of posterior mucosa. After a checkered course, the patient survived. Conley [69] did a staged repair with tantalum mesh and fascia plus skin flaps after resection of anterior tracheal wall invaded by "adenocarcinoma of the thyroid." Lazo [203] in 1957 resected the anterior wall of cervical trachea to treat thyroid cancer, and implanted a prosthesis for speech. Grillo [85] in 1965 resected a six-ring segment of trachea, including a portion of cricoid invaded by papillary carcinoma, which paralyzed the left cord and which obstructed the tracheal lumen. Tracheal reconstruction was staged with a cutaneous tube supported by inlying polypropylene rings. The result was satisfactory. In 1966, Mathey and colleagues [41] 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.

An aggressive approach was accepted early in Japan, but only slowly in the west. Ishihara and colleagues [204] in 1978 reported operative results for 11 patients, 8 of whom had recurrent papillary adenocarcinoma after prior surgery. Sleeve resections were done with resection of anterior cricoid in 3 patients. Two died from the operation and 3 developed laryngeal stenosis; 5 were long-term survivors. This same group reported on 60 patients by 1991 [205]. In 1985, Tsumori and colleagues [206] reported 18 resections with anastomosis. In 1986, Fujimoto and colleagues [207] performed sleeve resection in 6 patients and window resection in 3. A survey of tracheobronchial surgery in Japan by Maeda and colleagues [208] in 1989 revealed 151 cases of tracheoplasty for thyroid cancer compared with 147 tracheobronchial tumors over a period of 30 years.

In the west, Grillo [87] in 1978 reported on 3 patients who underwent resection for thyroid carcinoma and recommended that this treatment be applied more widely. In 1986, Grillo and Zannini [209] described outcome of 16 patients who underwent resection and reconstruction, and Grillo and colleagues [210] reported 27 by 1992. The rationale for resection and reconstruction is adherence to the oncologic principle of thyroid surgery, that local disease be removed totally. The surgery is not high risk or radical in competent hands. Given the proclivity of papillary tumors to become more aggressive in time, plus 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 [211].

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 [212] in 1963 in 11 patients with 5 long-term survivors and 5 alive without disease from 4 to 16 years. In 1958, Frazell and Foote [213] noted 3 of 4 patients who had laryngeal and tracheal resection for thyroid cancer lived 4.5 to 5 years. Grillo and colleagues [210] in 1986 reported radical extirpation of tumor in 7 patients by cervicomediastinal exenteration including esophagectomy. Palliation is a principal goal of these procedures.

Postintubation Lesions

The poliomyelitis epidemics of the mid-20th 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 that was attributable to ventilatory apparatus–endotracheal and tracheostomy tubes and the cuffs necessary to seal the trachea [86, 92, 188, 214]. Principal among these lesions were circumferential stenosis, which appeared at the level of the sealing cuff, and 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 fistulas occurred principally between the trachea and the esophagus at cuff level, usually with accompanying circumferential tracheal damage. Tracheal innominate artery fistulas were rare but disastrous when they occurred. These fistulas proved to be of two types, one in which a tracheostomy tube rested immediately on the innominate artery near the stoma, and another, in which the cuff or, even less often, the tube tip eroded through the trachea anteriorly into the innominate artery.

POSTINTUBATION STRICTURES. In the 1960s, numerous articles, often single case reports, appeared in Europe and North America describing surgical resection of postintubation strictures. Included among these were: Forster [73] in 1957, Flavell [77] in 1959, Witz [215] in 1960, Binet and Aboulker [75] in 1961, Van Wien [216] in 1961, Mathey [41] in 1966, Byrn [217] and Fraser [218] in 1967, Jewsbury [219], Dor [220], Dolton [221], Schaudig [222], Lindholm [223], and Naef [90] in 1969, along with their colleagues. Series of cases also were reported: Deverall [88] detailed 6 patients in 1967, Pearson [214] 15 patients in 1968, Grillo [86] 14 patients and Couraud [91] 9 patients in 1969, Dor [93] 9 patients, Levasseur [94] 10 patients, and Harley [95] 11 patients in 1971. These last authors, especially Pearson, Grillo, and Harley, having 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 [224]. Deverall [88], Pearson [214], Grillo [86], and Couraud [91] and their colleagues stressed the importance of allowing florid inflammation to subside before surgical correction. 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 [225], and, more lately with much more disastrous results, the attempted use of stents [193] to treat postintubation stenosis.

Postintubation lesions became, and remain, the most common indication for tracheal resection and reconstruction. Generally good results have been obtained in major cumulative series of patients with iatrogenic tracheal and subglottic laryngotracheal stenosis: Bisson and colleagues [226] achieved 87.5% "cure" in 200 patients in 1992, Couraud and colleagues [227] achieved 96% success in 217 patients in 1994, and Grillo and colleagues [100] achieved 94% success in 503 patients in 1995.

SUBGLOTTIC LARYNGEAL STENOSIS. Correction of postintubation stenosis involving the subglottic larynx remains more difficult than lesions confined to 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 associates [169] applied this approach in infants and children, also with encouraging success.

REOPERATIVE TRACHEAL RESECTION. Reoperative tracheal resection and reconstruction for unsuccessful repair of postintubation stenosis proved to be surprisingly manageable. Donahue and colleagues [228] in 1997 tallied 92% good or satisfactory results in 75 patients who had failed prior surgical repairs, 59 of whom were referred.

TRACHEOESOPHAGEAL FISTULA. Tracheoesophageal fistula due to erosion by tracheal cuffs and often of "giant" size were noted early, in 1966 by Le Brigand and Roy [229] and several other French surgeons during the same period, Flege [230] in 1967, and Hedden and colleagues [231] in 1969. Scattered attempts of repair by sometimes multistaged techniques, including Braithwaite's [232] successful use of a cutaneous flap to seal the tracheal side of a large fistula in 1961, did not often meet with success [229, 233]. Grillo and associates [234] in 1976 described a definitive one-stage technique for esophageal closure, tracheal resection (in which a circumferential cuff lesion was present), and strap muscle interposition, with good results in 7 patients.

Postintubation injury, however infrequent, has become the most frequent cause of acquired tracheoesophageal fistula. It is now effectively managed by the type of procedure noted, and has been further described [235–237]. When the fistula is small and the tracheal lesion not circumferential, tracheal closure is performed. The techniques developed have been applied effectively to closure of tracheoesophageal fistulas from a variety of causes, including trauma and inflammation.

TRACHEAL INNOMINATE ARTERY FISTULA. Tracheal innominate artery fistula, described in the mid- to late-1960s by several groups [238–242], as a consequence of tracheostomy and ventilation was effectively approached surgically by Grillo [243] in 1976, Cooper [244] in 1977, and Couraud [245] in 1984 and their associates. 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 [246] reviewed the topic extensively in 1976, including delineation of types of erosion, emergency management, safety and desirability of arterial resection, and success rates.

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

Posttracheostomy stenosis had been pointed out as early as 1886, when Colles [247] found four strictures in 57 patients treated for diphtheria. 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 [188] identified inflammation as a major factor in the spectrum of posttracheostomy stenosis. Based on 12 autopsy studies of patients who were ventilated through a tracheostomy for differing time periods, Bignon and Chrétien [248] 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. 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. Severity of lesions did not correlate with the length of ventilation.

Yanagisawa and Kirchner [249] and Atkins [250] in 1964 described severe damage to the trachea and stenosis from use of cuffed tracheostomy tubes. In 1965, after careful autopsy studies of tracheostomized and ventilated subjects, Florange and associates [251] reconstructed the evolution of tracheal necrosis from mucosal inflammation to erosion of mucosa, loss of cartilage, and localized mediastinitis. They concluded that this damage could result in stenosis. Stiles [240] in 1965 described severe changes at stomal, cuff, and tube tip levels in 23 patients in 37 consecutive tracheostomies, all of whom died after ventilation. He was inclined to relate the damage to the materials from which the tubes were manufactured. Gibson [252] in 1967 concluded that the "main factors" in producing stenosis were cuff trauma plus infection at the stoma. Most tracheas of patients who died while being ventilated through a tracheostomy showed necrosis. Murphy and colleagues [253] in 1966 could produce stenosis in dogs with cuff tracheostomy only when infection was also present. In 1968, Foley and colleagues [242] described the tracheal changes due to abutment of tubes and cuffs in patients with fatal burns.

Grillo [86] in 1969 showed similar changes as a result of ventilation. Cooper and Grillo [254] presented a detailed pathologic study of autopsy specimens from patients dying on respirators. A spectrum of changes was described similar to that noted by Florange and associates [251]. Lesions appeared within 48 hours and progressed from tracheitis to ulceration of mucosa, to fragmentation of cartilage, to replacement of tracheal wall with scar tissue. The location and nature of the lesions also correlated with surgically removed stenotic lesions. Lindholm [223] in 1969 presented a detailed study of lesions that developed in the larynx and also in the trachea from ventilation. The severity of histologic changes was vastly greater than those described after tracheostomy alone [255]. Andrews and Pearson [256] in 1971 prospectively examined the trachea of 103 patients receiving ventilator support. Twelve stomal and six cuff stenoses developed. Bronchoscopic examination was of little value in predicting which patients would develop stomal stenosis, but circumferential mucosal ulceration at the cuff level dependably predicted cuff stenosis. Additional statistically significant factors observed in this study were large tracheostomy tubes and administration of high-dose steroids. The same erosive processes were observed to cause tracheoesophageal fistulas and tracheoinnominate artery fistulas.

PREVENTION. Prevention of postintubation injury quickly became a priority once the origin of these lesions was evident. Adriani and Phillips [257] in 1957 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) to develop ventilatory pressures of 10 to 20 mm Hg. Cooper and Grillo [258] later pointed out that excessive pressures were necessary to seal the irregularly shaped trachea by distending the relatively rigid small volume cuffs then in use. Knowlson and Bassett [259] also noted that small increments over minimal occlusive volume necessary for sealing conventional cuffs at 20 cm H_2O caused rapid rise in pressure exerted on tracheal mucosa. Grimm and Knight [260] in 1943 had proposed that the ideal cuff "should have sufficient volume when inflated without stretching to fill the diameter of the trachea." Lomholt [261] in 1967 offered a cuff of thin and elastic Teflon (DuPont, Parkersburg, WV) lying in folds so that intracuff pressure would be identical with the pressure on the mucosa. Carroll and colleagues [262] in 1969 recommended a cuff with large residual volume, a large sealing area, and a centered tube, leading to only small increases in tracheal wall pressure with overinflation.

Cooper and Grillo [258] in 1969 reproduced severe stenosing cuff lesions in dogs that were entirely parallel with lesions seen in humans. 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. Because this experiment proved conclusively that tracheal lesions were due to cuff pressure, a large-volume compliant cuff was designed for clinical use by Grillo and coworkers [263]. 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 evaluated and compared. Any degree of injury severe enough to evolve into stenosis was produced by

the standard (high-pressure) cuff. Average intracuff pressure in the new cuff was 33 mm Hg compared with 270 mm Hg in the standard. In extensive clinical use, no tracheal lesions resulted from use of this large-volume compliant cuff.

For economic reasons, manufacturers later abandoned latex in favor of plastic cuffs, which lack extensibility. Thus, when slightly overinflated, the present-day large-volume cuffs produce steep rises in intracuff pressure [259] with the potential for tracheal injury severe enough to result in stenosis. Careful attention to cuff inflation and pressures, however, has helped to avoid any incidents of cuff stenosis since 1970 at Massachusetts General Hospital. 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 large-volume cuffs properly used.

After adopting the lightweight, swivel trachea connectors used at Massachusetts General Hospital, Andrews and Pearson [256] observed a decline in the incidence of stomal stenosis from 17.5% to 6.9%. Since then, the addition of suspension of the connecting tubing to avoid leverage of the tube against the tracheal stoma has essentially eliminated stomal stenosis at Massachusetts General Hospital.

Elimination of tracheoesophageal fistula has followed proper use of large-volume cuffs for ventilation along with avoidance of inlying rigid nasogastric tubes. Tracheoesophageal fistula 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 [27] described their 18 experiences with tracheal injuries in a series of 91 tracheobronchial cases from the literature; these injuries were more commonly linear lacerations. Shaw and colleagues [28] in 1961 provided results from repair of nine cervical and four intrathoracic tracheal ruptures, recommending primary repair of acute injuries and resection of scar with accurate anastomosis for posttraumatic stenosis. Baumann [264] reviewed the limited knowledge about tracheal trauma in 1960, recommending tracheal bronchoscopy in all serious thoracic trauma. Ogura and Powers [97] approached traumatic stenosis of the subglottic larynx aggressively in 1964. Chodosh [265], Ashbaugh and Gordon [266] and others described laryngotracheal avulsion injuries.

Beall and associates [267] presented 23 tracheal injuries in 1967 and favored immediate treatment, advising airway maintenance and reanastomosis where possible. Ecker and colleagues [268] described 21 tracheal injuries in 1971, with 18 successfully treated. Bertelson and Howitz [269] reported cervical tracheal rupture and perforating wounds in 1972, recommending tracheostomy alone for small wounds. Symbas and colleagues [270] by 1976 progressed from tracheostomy alone to repairs of penetrating wounds in a series of 20 patients. Grover and colleagues [271] reported experience with a variety of tracheobronchial injuries in 1979. Angood and associates [272] added to experience in extrinsic trauma to larynx and cervical trachea in 1986. Mathisen and Grillo [273] in 1987 reported good results with immediate repair of acute tracheal injuries and also of concurrent esophageal transection in one, and good results in 16 of 17 chronic patients, 14 with vocal cord paralysis and 4 with esophageal injury. They emphasized the importance of acute airway control, assessment of glottic competence where recurrent nerves may be damaged, 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 associates [274] in 1989 addressed the especially difficult problem of traumatic disruption of the laryngotracheal junction, describing 19 patients with restoration of airway and voice in all.

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

INHALATION BURNS. Gaissert and colleagues [275] described principles of management of inhalation burns of the trachea in 1993, recommending a conservative approach and patient use of T-tubes. Any surgery required is performed late, after subsidence of cicatricial response.

IRRADIATION. The irradiated trachea heals poorly when transected and reanastomosed, particularly with rising doses of irradiation and increased intervals between radiation and surgery. Muehrcke and coworkers [276] showed that improved results may be obtained in these difficult problems by wrapping an anastomosis with pedicled omentum. Such anastomoses, however, remain at greater risk for serious complications.

Congenital and Pediatric Lesions

GROWTH. Concern whether growth would occur after resection and anastomosis of the trachea in infants and small children was allayed early by experiment, although an occasional success had also been noted clinically. Kiriluk and Merendino [46] had observed growth of main bronchi after anastomosis experimentally. Borrie [277] had found stenosis to occur after excision of more than three segments in lambs. Sorensen and associates [278] in 1971 noted somewhat limited growth in anastomotic sites in puppies after resection of zero to five rings. Maeda and Grillo [279] in 1972 noted only mild narrowing of the anastomotic site in puppies without resection, at full growth. 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 vs 1,750 g) [280]. Kotake and Grillo [281] observed in puppies that tracheal "stay sutures" reduced

anastomotic tension. Murphy and associates [282] in 1973 noted unpredictably variable growth at anastomosis in piglets after resection of only two rings. Mendez-Picon and colleagues [283] confirmed anastomotic growth in 1974 in puppies. In 1978, Burrington [284] found that cartilage grew continuously by proliferation on the convex surface without specific growth centers. Vertical incisions, hence, do not interrupt growth.

CONGENITAL TRACHEAL STENOSIS. Cantrell and Guild [285] 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. Tracheal resection and primary anastomosis in children was explored by Carcassonne and associates [286] in 1973, and others through the early 1980s [287–290]. Couraud and associates [291] demonstrated long-term growth of anastomotic scars in 1990 particularly after resection of stenosis and anastomosis. Monnier and colleagues [169] showed that single-stage laryngotracheal resection and anastomosis was also applicable in small children. This procedure appeared likely to largely replace cartilage graft procedures [292] developed earlier. However, the length of many congenital tracheal stenoses prohibited resectional treatment.

Kimura and his associates [293] provided a solution in 1982 by inserting a cartilage patch longitudinally the length of the stenosis. Idriss and associates [294] in 1984 used pericardium for the same purpose. Heimansohn [295] and Jaquiss [296] and their associates confirmed the use of pericardium and cartilage insets, respectively. Although their efforts were successful in most cases, a considerable incidence of repetitive granulations formed on the mesenchymal patch until epithelization eventually occurred [297], and in some patients necrosis of the patch required reoperation or tracheostomy [298].

Tsang and associates [299], working with Goldstraw, solved these ploblems with slide tracheoplasty, described in 1989. Grillo's [300] report in 1994 describing four successful cases so treated established the procedure. A subsequent publication [301] reporting a total of 8 successful patients, one 10 days old, confirmed that satisfactory long-term growth occurred after slide tracheoplasty. The procedure corrected long stenosis by providing a firm reconstruction with tracheal tissue, lined with ciliated epithelium and hence with little tendency to form granulomas, that did not require postoperative intubation for support and—absent left pulmonary artery sling or other cardiac anomaly—did not require cardiopulmonary bypass for surgery.

LARYNGOTRACHEOESOPHAGEAL CLEFT. Complete laryngotracheoesophageal cleft was successfully repaired in 1984 by Donahoe and Gee [302].

Infectious, Inflammatory, and Other 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 granulomatosus. These techniques are not referenced individually because no new principles were necessary for their treatment. The "new" techniques replaced the wire-supported dermal grafts pioneered by Gebauer [53] for tuberculous airway strictures.

IDIOPATHIC LARYNGOTRACHEAL STENOSIS. Idiopathic laryngotracheal stenosis had been identified in scattered case reports in the 1970s [303–305]. Grillo and associates [306] in 1993 presented a series of 49 such patients, 39 treated by one-stage tracheal or laryngotracheal resection, with 32 good or excellent results. Twenty-six had been followed from 1 to 15 years with extension of fibrosis 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 [307] of repetitive laser treatment used in 30 patients who suffered recurrent progressive stenosis, and of failure in 7 patients who did undergo open operation.

TRACHEOPATHIA OSTEOPLASTICA. Tracheopathia osteoplastica, a rare condition characterized by submucosal cartilaginous nodules with calcification, most often involving the entire trachea, but also the main bronchi, is sometimes severely obstructive. This condition was treated successfully by Grillo [308, 309] with a tracheoplasty over a T-tube that was later removed. 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. Tracheomalacia appears in many forms. Short segments related to postintubation lesions have been resected [224], longer segments have sometimes been splinted with external polypropylene rings [224] or internal stents or T-tubes. Respiratory collapse associated with chronic obstructive pulmonary disease was treated by Herzog [310, 311] and Nissen [312] in the 1950s with posterior membranous wall splinting and quilting, pulling the ends of the splayed, softened cartilaginous rings into a more normal C-shape. Thin bone slabs, fascia lata, and later Gore-Tex (W.L. Gore and Assoc, Flagstaff, AZ) were used as splint materials. Rainer and colleagues [313] in 1968 reported results with perforated plastic splints of several designs. Grillo and associates (in preparation) found posterior splinting of intrathoracic trachea and both main bronchi with strips of Marlex to be effective. Importantly, Marlex becomes permanently incorporated by scar tissue, preventing recurrence. Principal clinical benefits are 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 commonly in adults. The occurrence of airway compression remains unpredictable. The same effect was observed in patients with agenesis of the lung by Maier and Gould [314] in 1953. The phenomenon was also recognized to occur after left pneumonectomy with a right aortic arch [315, 316] and, rarely, even after left pneumonectomy with a normal aortic arch [317, 318]. Johnson and colleagues [319] in 1949 suggested filling a hemithorax with Lucite balls to prevent "overdistension" of a remaining lung. Adams and colleagues [320] used this technique in a symptomatic child. Kaunitz and Fisher [321] in 1966 proposed continued refills of air to maintain normal mediastinal position after pneumonectomy. Powell and associates [322] used a prosthesis preventively.

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

Rasch and colleagues [325] placed an expandable prosthesis in an infant therapeutically in 1990. Grillo and associates [316] produced the first report of a series of any size, 11 adult patients, in 1992. Ten underwent repositioning with implants. Good results were obtained generally, but not in 4 patients who showed severe residual tracheobronchial malacia after mediastinal repositioning. Malacia was more likely to be present in patients with a 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 used with success.

Conclusion

Tracheal surgery has largely developed and matured in the last 40 years. 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 and for 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 [79] wisely counseled that the simplest solution was likely to be the best and that sacrifice of even a half centimeter of trachea might unnecessary force a change in surgical plan.

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