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Upper Airway Compromise After Inhalation Injury

Complex Strictures of the Larynx and Trachea and Their Management

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Objective
Strictures of the upper airway caused by burns have features distinct from other benign stenoses. The authors reviewed their experience with burn-related stenoses to define the principles of treatment.

Summary Background Data
The combined effects of inhaled irritant gases and heat in burn victims produce an intense, often transmural, inflammation of the airway, further complicated by intubation. The incidence of laryngotracheal strictures in survivors of inhalation injury is high, but the reported experience with their treatment is limited and often unduly separated into injuries of larynx and trachea.

Methods
Presentation, treatment, and long-term follow-up are reviewed in 9 women and 9 men age 9 to 63 years, who were evaluated over a 22-year period for chronic airway compromise after inhalation injury. There were 18 tracheal stenoses, 14 subglottic strictures, and 2 main bronchial stenoses. Laryngotracheal strictures developed in three patients without history of intubation. Six underwent resection of subglottic stenosis. T-tubes were placed in 15 patients, in low subglottic or tracheal stenosis below the vocal cords, in high subglottic stenosis through the vocal cords, and as a stent after resection of subglottic stenosis.

Results
There were two deaths during follow-up, one from respiratory failure and one from an unrelated cause. Two patients underwent evaluation only. Early in this series, one tracheal and one laryngotracheal resection resulted in prompt restenosis. Of the remaining 14 patients, 9 are without airway support from 2 to 20 years later. Four have permanent tracheal tubes. One patient required tracheostomy 8 years after successful subglottic reconstruction.

Conclusions
Strictures of the upper airway related to inhalation injury are associated with prolonged inflammation and involve larynx and trachea in a majority of patients. These complex injuries respond to prolonged tracheal stenting (mean, 28 months) and resection or stenting of subglottic stenoses with recovery of a functional airway and voice in most patients. Early tracheal resection should be avoided

Despite advances in our understanding of the etiology and prevention of inhalation-induced injury to the upper airway, management of resultant strictures of larynx and trachea remains ill-defined. The initial insult is believed to be caused by irritant gases, such as aldehydes, ammonia, and hydrochloric acid, and by local effects of heat.[1] Both components are thought to produce severe tracheobronchitis with sloughing of the mucosa. In the presence of an intact basal cell layer, early repair is accomplished rapidly in clinical and experimental observation.[2,3] If the basal membrane is destroyed in the original event or due to additional trauma, delayed repair may result in granulations, cicatrization, and stenosis.[4] Effects of inhalation and intubation are often impossible to separate since intubation is performed early in the presence of respiratory symptoms to prevent acute airway obstruction.

Although injuries in the larynx and the trachea of the same etiology have similar features, collective discussions of their management are rare. One reason may be the relatively uncommon occurrence of burn-related stenoses, so that their treatment is often presented as case reports.[5,6] The reported incidence varies and is almost exclusively restricted to patients who underwent intubation. In 84 patients treated with intubation, 2 subglottic stenoses were found among 38 survivors.[7] In 99 patients with inhalation injury and tracheostomy, major airway complications ensued in 28 patients and 6 of 25 survivors had tracheal stenosis.[8] A considerable number of laryngeal and tracheal stenoses that are not severely symptomatic may not come to medical attention unless specifically sought. Lund et al. screened 17 survivors of inhalation burns for late airway sequelae and discovered 4 tracheal stenoses and 5 patients with significant tracheal granuloma formation.[9]

Management of laryngotracheal disease due to combined trauma from inhalation and intubation remains demanding and at times challenging. Acute treatment seeks to preserve reliable access to the airway. Later concerns focus on restoration of an adequate laryngotracheal lumen and vocal rehabilitation.

MATERIALS AND METHODS

Between 1969 and 1991, 18 patients (9 males and 9 females), whose ages ranged from 9 to 63 years (mean, 34.4 years), who had airway compromise after burn injury, were seen at the General Thoracic Surgical Unit at Massachusetts General Hospital (MGH). Most patients were seen in consultation with a laryngologist at the Massachusetts Eye and Ear Infirmary. Hospital and office charts were reviewed. Follow-up was obtained either by contacting patients by telephone or with questionnaire by mail, or through information provided by families and physicians.

Table 1. CAUSE OF BURN INJURY

Cause	No.
House fire	8
Motor vehicle accident	4
High voltage exposure	1
Gas explosion	1
Plane crash	1
Television set explosion	1
Ammonia inhalation	1
Hydrochloric acid inhalation	1
Total	18

Table 1 lists the principal causes of burn. Sixteen patients were involved in flame or combustion burns and 2 were injured by chemical agents only. Fifteen were intubated as part of their early management; an airway was established initially by tracheostomy in one of them and by translaryngeal intubation in the others. Details on exact indications for the original intubation are not available. Three patients had no history of intubation until they noted symptoms of airway obstruction 3 weeks to 5 months later. Transtracheal or translaryngeal intubation lasted between 4 days and 3 months to the point of extubation or diagnosis in 10 patients, for whom accurate information is available. The onset of symptoms in intubated patients is listed in Table 2.

The operative management of this group of patients before their presentation to MGH is detailed in Table 3. Tracheostomy for late obstruction of the airway was performed in five of the ten patients. Reconstructive tracheal procedures were attempted in three patients. One

Table 2. DIAGNOSIS OR ONSET OF SYMPTOMS IN 15 INTUBATED PATIENTS

Onset	No.
During intubation	5
After extubation	
<1 mo	4
1-3 mo	4
4 mo	2

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Table 3. OPERATIVE PROCEDURES IN 18 PATIENTS BEFORE PRESENTATION

Procedure	No.
Tracheostomy	10
Tracheal revision/repositioning	3
Attempted tracheal reconstruction	3
Tracheal dilatation	5
Laryngofissure	1
Myocutaneous flap/T-tube	1
Laser use	2

with three prior procedures underwent resection of a 4-cm segment of trachea 8 months after burn and shortly thereafter, when restenosis developed, further resection of a 2-cm segment. When recurrent obstruction ensued, the trachea below the cricoid was transected to reduce tension and a tracheal stoma was created. Another patient received costal cartilage grafts in two operative procedures 2 and 3 months after burn to relieve a long stenosis of the midtrachea, with recurrent obstruction after both procedures. The third patient acquired a high tracheal stenosis after tracheostomy and mechanical ventilation for a flail chest resulting from a car accident. Two years earlier, he had sustained a respiratory burn in a gas explosion. Two weeks after resection of a 2-cm tracheal segment, stridor developed and frequent, serial dilatation of a narrowed anastomosis became necessary.

A majority of patients (72.2%) were referred within 1 year after injury. The time period between burn and initial presentation at MGH varied between 2 and 56 months (median, 6 months). The longest interval occurred in a quadriplegic patient with permanent tracheostomy and complete subglottic obstruction, who was evaluated for recovery of voice. Two of the other three patients presenting longer than 1 year after injury had undergone prior tracheal reconstruction, and one was seen during rehabilitation subsequent to a high-voltage electrical, external burn with an extensive tracheal injury, for which a myocutaneous flap and T-tube placement had been performed.

Clinical Findings

Evaluation consisted of plain radiographs of the trachea, tomography, and fluoroscopy. Every patient underwent laryngoscopy and bronchoscopy under general anesthesia. Findings are summarized in Figure 1. Tracheal stenosis was identified in all 18 patients. A combination of subglottic and tracheal stenosis was found in 14 instances. The two lesions were contiguous in ten patients, and in four the stenoses were separated by a tracheal segment of adequate lumen. In only one of these latter patients with separated stenoses was the tracheal lesion clearly the more significant injury. In the other three cases the subglottic stenosis appeared of either equal or greater degree. In all three patients without history of intubation, an adjoining subglottic and upper tracheal stenosis was found.

Although most patients had been intubated for some time after their burn, the degree of tracheal damage was far more extensive than that usually seen in a group of patients with postintubation stenosis. Twelve stenoses were greater than 3 cm in length. The large proportion of high tracheal stenoses that originated in the subglottic space is also unusual for intubation-related injury. In those patients evaluated early after injury, the most pronounced damage was seen just below the vocal cords, while the pharynx and supraglottic larynx showed only superficial scarring. One patient had a short malacic ring above the stoma, a characteristic, limited postintubation tracheal injury. It was, however, accompanied by mucosal edema. There were two bronchial stenoses, one occurring in the left mainstem bronchus of a 21-year-old woman who sustained a 50% surface burn and inhalation injury with subglottic and long distal tracheal stenosis. One additional patient had a right mainstem bronchus narrowed by granulations.

A notable finding in these patients was the extent of inflammation that involved multiple levels of the larynx and trachea (Table 4). Edema and a granulating, friable, and easily bleeding tracheal surface were often seen and,

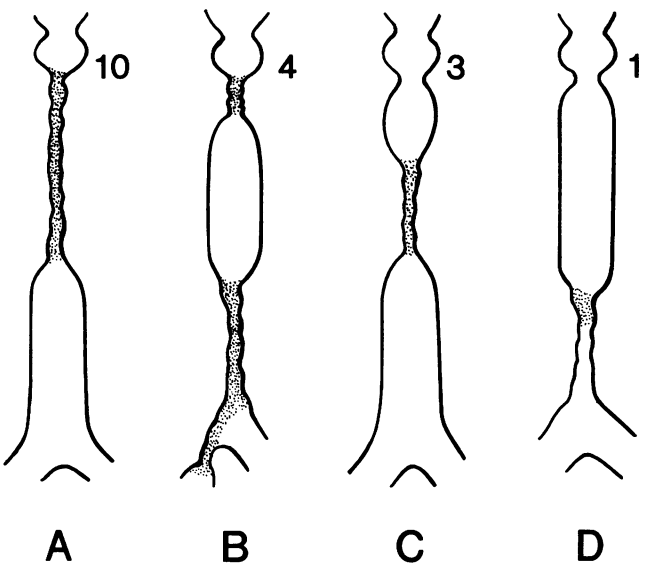

Figure 1. Distribution of airway injury in 18 patients. A: Subglottic and upper tracheal stenosis. B: Subglottic stenosis and lower tracheal stenosis with or without extension into main bronchi. C: Upper tracheal stenosis. D: Lower tracheal stenosis. The number of patients in each category is noted.

Table 4. INJURY PATTERN IN 18 PATIENTS WITH AIRWAY STENOSIS AFTER INHALATION BURN*

Patient No.	Age (yr)	Sex	Cause	Time After Burn (mo)	Glottis	Subglottic Space	Trachea	Main Bronchus
1	63	F	House fire	2	E	E	Ss, E	
2	37	F	House fire	12	E	So, E	Sl, G	
3	38	F	Television explosion	7		S	Ss, E	
4	19	F	House fire	3	E	S	Ss	
5	19	F	House fire	4	E	S	Ss, G, E	
6	53	M	Gas explosion	36	E	S	Sl, G, E	C, G
7	47	F	MVA	4		S	Ss, G	G (right)
8	28	F	Plane crash	56		So	So	
9	22	M	MVA	4		S	Sl, G	
10	11	M	House fire	25		S	Sl	
11	42	M	Ammonia inhalation	2			Sl	
12	21	F	House fire	6	S	S	Sl	Ss (left)
13	50	M	HCl inhalation	4		E	Sl, C, G	G
14	44	M	House fire	16	P (right)	S	Sl	
15	33	M	MVA	8	P (left)		Sl	
16	24	M	MVA	6	E	S	Sl	
17	62	F	House fire	8	S, E	S, E	Sl	
18	48	M	High voltage contact	36		S	Sl	

E: edema; S: stenosis; s: short (<3 cm); l: long (>3 cm); o: occluding; G: granulations; C: scarring; P: vocal cord paralysis.
* Age and findings at the initial bronchoscopic examination are recorded.

when present, covered a sizable portion of the remaining unstenosed airway. Granulations of the tracheal wall unrelated to the location of the tracheostomy were seen on bronchoscopy in six patients and extended into either mainstem bronchus in three of them. Irregularities of the tracheal wall and scarring not only at the stenosis, but also for some distance above and below, suggest that consequent to thermal and chemical factors structural integrity was altered, though not lost beyond the obvious stenosis. Tracheal cartilage appears to remain intact in these segments. On initial evaluation, edema and granulation in the larynx or trachea were observed in all patients seen 12 months or less after burn and were also seen 2 years after respiratory burn in another. In most patients, the inflammatory component tended to subside over a period of 3 to 5 years, and granulation tissue on the tracheal wall became firm and less friable.

Treatment

Evaluation

Two patients were evaluated without further therapy. An obliterating subglottic and upper tracheal stenosis in a patient without phonation and speech articulation was considered unreconstructible. A patient with subglottic stenosis, treated previously with dilatation and referred for consultation, underwent further dilatations by his physician.

Tracheal Reconstruction

Two patients underwent resection and reconstruction of their stenosis using a standard technique.[10] Tracheal resection of a 2.5-cm segment was performed in one patient. Investigation for recurrent symptoms shortly thereafter demonstrated a previously unsuspected subglottic stenosis above the anastomosis over the anterior cricoid arch. A laryngofissure and prolonged T-tube placement was required. In the second patient, who had a subglottic stenosis and a distal trachea of satisfactory caliber, laryngotracheal resection of a 3.5-cm segment was performed. Early edema developed at the anastomosis, which did not respond to dilatation. Airway obstruction required tracheostomy. Evaluation elsewhere 9 years later showed a residual anterior tracheal stenosis, which responded to laser treatment. The patient was subsequently lost to follow-up. In a patient with three previous procedures, cervicotracheal reconstruction using laryngeal release was performed to reanastomose larynx and trachea over a T-tube. This patient regained her voice and is alive 20 years later with a T-tube.

Resection of Subglottic Stenosis

Chronic subglottic stenoses were resected by consulting laryngologists according to the technique of Mont-

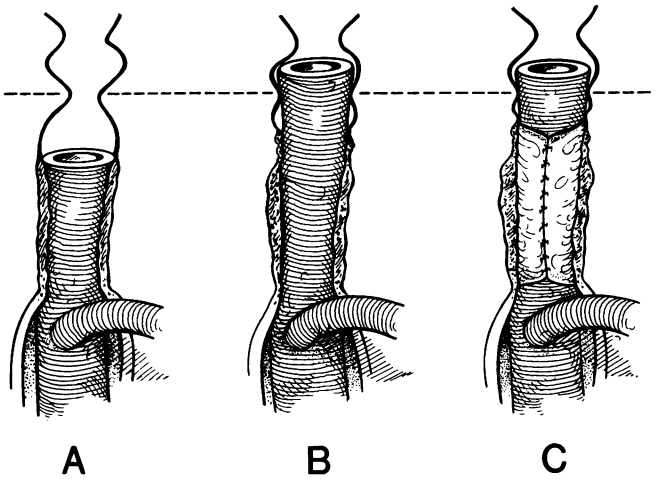

Figure 2. Use of T-tube in subglottic stenosis. The dotted line indicates level of true vocal cords. A: The tip of T-tube lies in the subglottic space below the vocal cords, it does not touch the conus elasticus. B: The T-tube traverses the vocal cords to rest in the laryngeal ventricle just beneath the false cords. C: After laryngofissure and excision of scar, the T-tube is placed in the subglottic space stenting a buccal mucosal graft.

gomery.[11] After removal of scar, the laryngeal defect was usually covered with a buccal mucosal flap or a split-thickness skin graft over a stent, either a T-tube or a conforming laryngeal stent. If the degree of stenosis was severe with structural narrowing of the cricoid arch, the arch and posterior cricoid lamina were widened with grafts from the thyroid lamina.

Six patients underwent either laryngofissure or laryngocricotracheal fissure, four of them soon after referral, one at 4 years after tracheostomy, and one after failed tracheal resection. Two patients had two procedures and one patient three procedures. Stents (two patients) and T-tubes (four patients) were usually removed after 1 to 3 months. One T-tube remained for 3 years. Vocal cord lateralization was performed at the time of laryngofissure in one patient for vocal cord paralysis.

T-tube

A tracheal T-tube was placed in 15 patients. Indications were combined subglottic and tracheal stenosis in 13 patients and distal tracheal stenosis in 2 patients. Successful use of the T-tube to bridge the subglottic space occurred in three situations: (1) in the presence of a low subglottic stenosis with sufficient space to permit the tip of the tube to rest above the stenosis, but not in contact with the undersurface of the cords; (2) in high subglottic stenosis by passing the T-tube through the vocal cords; and (3) after resection of the stenosis, when the T-tube is placed as a stent (Fig. 2). If a tracheostomy is constructed at the time of T-tube placement, the stoma is placed through the area of maximal injury to preserve tracheal length. Persistence on the part of surgeon and patient was occasionally necessary, when several attempts were required to achieve good seating of the tube and a satisfactory airway.

Two patients failed repeated placement due to obstructive symptoms. T-tubes remained in the airway for a mean duration of 36 months (range, 2 months to 20 years). In six patients, the upper end of the T-tube was placed above the true cords at the level of the laryngeal ventricle for a period of between 4 and 21 months. A hoarse voice ensues in all patients due to phonation with the false cords, but improves usually with enhanced pharyngeal speech articulation. Aspiration is prevented by closure of the false cords and deflection of the epiglottis. Generally patients can swallow without signs of aspiration after 24 hours, with the exception of elderly patients who often fail to swallow properly.

Dilatation

Dilatation was often used as a temporary measure to restore airway caliber to allow diagnostic bronchoscopy or T-tube placement. In one patient with extensive stenosis of the larynx, trachea, and left mainstem bronchus, repeated dilatations over the course of 2 months resulted in prolonged patency of the bronchus, while the upper airway was managed with laryngofissure and T-tube. We generally employ esophageal Jackson dilators and pediatric Jackson bronchoscopes for this purpose. If a tracheal stoma is present, Hegar dilators are also used successfully.

RESULTS

One patient was lost to follow-up and one with unreconstructible injury was not observed after evaluation. There were two deaths in the interval, one of unrelated disease 20 years after injury and 18 years after removal of the T-tube following a failed tracheal resection. The other patient, who did not tolerate a T-tube and left the hospital with a tracheostomy tube, died of airway obstruction 4 months after presentation. She had a very distal tracheal stenosis close to the carina and scarring of the right mainstem bronchus with granulations.

The remaining 14 patients were observed from 33 months to 20 years (mean, 10.8 years) after injury. Four patients have permanent tracheal tubes, of whom two have T-tubes and two have tracheal cannulas. One additional patient, deemed irreparable and palliated with a T-tube, underwent skin flap reconstruction of the trachea elsewhere with unsatisfactory result.

Nine patients are without airway support. Five were treated with T-tube only, four with laryngofissure and

subsequent T-tube. Decannulation was achieved at 4 to 61 months (mean, 27.9 months) after injury. The remaining patient underwent several tracheal dilatations for a relative distal tracheal stenosis by his physician resulting in a stable airway. One of the patients treated with laryngofissure and T-tube, who has a very extensive stenosis with left main bronchial component, currently undergoes bronchial dilatation approximately every 8 months. Of six patients with open repair of their subglottic stenosis, four had good results without further operative treatment. One patient, referred to us with total obliteration of the subglottic space, failed to improve and required a tracheal cannula. One patient remained without airway support for 8 years, until he had recurrent symptoms and an unreconstructible recurrence, necessitating tracheostomy.

A successful outcome of treatment does not result in a normal airway. The quality of voice is often diminished and some degree of hoarseness is present. Mild chronic wheezing and frequent recurrent episodes of upper respiratory tract infections are commonly reported; no patient has had acute airway obstruction.

DISCUSSION

The distribution of laryngotracheal damage deserves comment. The observation of an exaggerated necrotizing process of the airway immediately below the vocal cords was first reported by pathologists examining victims of the Cocoanut Grove Fire and attributed to eddies in the subglottic airflow with increased exposure to toxins.[2] The respiratory epithelium below the true vocal cords may also be more susceptible to injury than squamous epithelium above. Confluent destruction extending from the glottic level to the cervical trachea with extensive mucosal injury is typical, but not pathognomonic for burn injury. Stenoses are also observed after prolonged endotracheal intubation, in one study in 4% of patients intubated between 6 to 10 days and 6% of patients intubated between 11 to 24 days.[12] However, the degree of damage after inhalation injury, the presence of mucosal signs of inflammation at a distance from the tracheal tube, and the hypertrophic cicatricial response, if resection is undertaken, create a different disease pattern. Since most patients undergo intubation as part of their early management, clear distinction between burn-related and postintubation injury is difficult, if not altogether impossible. The fact that three of our patients experienced airway stenosis without prior intubation suggests that the initial injury has in itself the potential for stricture formation and that the presence of a tube is not required. Since inhalation and intubation produce upper airway obstruction independent of each other in similar location, they presumably potentiate their individual destructive effects.

Some of our patients sustained laryngotracheal stenosis without, or with negligible contribution of, heat. Two patients with chemically induced inhalation injury from agents known to be toxic to the airway mucosa, who also had early intubation, sustained a combined subglottic and cervical tracheal stenosis in one case and a long distal tracheal stenosis in the other. In the three patients without intubation before onset of symptoms, thermal injury to the face was minimal and a grafting procedure, of the nose only, was required in one patient. These patients represent the best available evidence that upper respiratory burn injury is caused in some, if not most, patients by irritant gases, even in the presence of fire. At the time of observation, injury to the pharynx and supraglottic larynx was negligible or absent in all patients.

The purpose of treatment is to restore airway patency, to preserve a satisfactory quality of voice, and to rid the patient eventually, if possible, of tracheal tubes. To achieve these goals, patient and physician must be prepared to accept an extended period of treatment. The location of the stenosis, the degree of inflammation, and the amount of uninjured larynx and trachea are noted on initial evaluation. If the subglottic space is well preserved, symptomatic tracheal stenosis can be treated initially with dilatation or direct insertion of a T-tube. If the infraglottic vestibulum is stenosed to the level of the vocal cords, a T-tube is placed with its tip resting above the true vocal cords.[13] The subglottic stenosis may be resected with grafting and stenting of the subglottic space in selected patients at an appropriate time. Stenoses involving the carina and the mainstem bronchi are most difficult to treat. Acutely, the area may require repeated and frequent dilatation. We have not used a T-Y-tube in this situation for fear of inciting more granulations, but it is an option.

The optimal time for resection of a subglottic stenosis remains undetermined, but delayed repair is thought to lead to fewer recurrences.[14] Three stenoses recurred after laryngofissure performed between 10 months and 3 years after burn. Stents were removed after 1 and 2 months in two patients, and a successful outcome was accomplished with longer stenting in both. In the remaining patient, recurrent narrowing above the laryngeal stent was treated with placement of a T-tube through the vocal cords with good result. Prolonged intubation of the subglottic space with a T-tube may therefore suppress or remodel scar formation, particularly in the case of recurrent stenosis.

Resection for the treatment of postintubation tracheal injury may often be accomplished soon after the patient is weaned from the ventilator and has recovered suffi-

ciently from his or her original illness, in the absence of florid inflammation.[10] The trachea in victims of inhalation burns, in contrast, retains a capacity for hypertrophic scar proliferation during extended periods of time, prohibiting early reconstruction. In the acute stage, the tracheal wall is still involved in an inflammatory process extending beyond the anatomically defined stenosis. Even where a generous airway diameter is preserved, the trachea is abnormal. Under these circumstances, reconstruction is far more likely to fail and the anastomosis all too often becomes the site of a new stenosis in a shortened trachea. The best effort at resection should therefore be reserved until inflammation has disappeared, conditions are optimal, and no immediate reintubation is expected. Further, as the inflammation gradually subsides, the patient may prove not to require resection. Similarities to the hypertrophic response of cutaneous burn scars and its treatment with compression garment are obvious.[5] We do not suggest avoidance of resection at all cost and without regard to the location of the injury. Subglottic stenoses, for example, may respond well to earlier operative repair[15] and, when resection is combined with T-tube placement, allow concurrent internal splinting of a tracheal stenosis.

Our experience with this selected group of patients with inhalation injury conveys several important messages. First, chronic upper airway compromise resulted from a combination of subglottic laryngeal and extensive tracheal stenosis in a majority of patients. Second, most of these tracheal stenoses responded to prolonged intraluminal stenting with a T-tube (mean, 28 months). Third, tracheal resection, performed early after injury, was followed by recurrent stenosis. And finally, even in these challenging circumstances, most patients will eventually regain a satisfactory airway and voice without a need for tracheal tubes.

References

1. Crapo RO. Causes of respiratory injury. *In* Haponik EF, Munster AM, eds. Respiratory Injury: Smoke Inhalation and Burns. New York: McGraw-Hill, 1990; pp 51–56.
2. Mallory TB, Brickley WJ. Pathology: with special reference to the pulmonary lesions. Ann Surg 1943; 117:865–884.
3. Shimazu T, Yukioka T, Hubbard GB, et al. A dose-responsive model of smoke inhalation injury. Ann Surg 1987; 206:89–97.
4. Colice GL. Long-term respiratory complications of inhalation injury. *In* Haponik EF, Munster AM, eds. Respiratory Injury: Smoke Inhalation and Burns. New York: McGraw-Hill, 1990; pp 336–337.
5. Eliachar I, Moscona R, Joachims HZ, et al. The management of laryngotracheal stenosis in burn patients. Plast Reconstr Surg 1981; 68:11–16.
6. Adams C, Moisan T, Chandrasekhar AJ, Warpeha R. Endobronchial polyposis secondary to thermal inhalation injury. Chest 1979; 75:643–645.
7. Venus B, Matsuda T, Copiozo JB, Mathru M. Prophylactic intubation and continuous positive airway pressure in the management of inhalation injury in burn victims. Crit Care Med 1981; 9:519–523.
8. Jones WG, Madden M, Finkelstein J, et al. Tracheostomies in burn patients. Ann Surg 1988; 209:471–474.
9. Lund T, Goodwin CW, McManus WF. Upper airway sequelae in burn patients requiring endotracheal intubation or tracheostomy. Ann Surg 1985; 201:374–382.
10. Grillo HC, Mathisen DJ. Surgical management of tracheal strictures. Surg Clin North Am 1988; 68:511–524.
11. Montgomery WW. Subglottic stenosis. Int Surg 1982; 67:199–207.
12. Whited RE. A prospective study of laryngotracheal sequelae in long-term intubation. Laryngoscope 1984; 94:367–377.
13. 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; 82:559–568.
14. Miller RP, Gray SD, Cotton RT, Myer CM. Airway reconstruction following laryngotracheal thermal trauma. Laryngoscope 1988; 98:826–829.
15. Flexon PB, Cheney ML, Montgomery WW, Turner PA. Management of patients with glottic and subglottic stenosis resulting from thermal burns. Ann Otol Rhinol Laryngol 1989; 98:27–30.