The Evolution of Tracheal Injury Due to Ventilatory Assistance Through Cuffed Tubes: A Pathologic Study

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SEVERE clinical symptoms arising from damage to the trachea in patients who have been treated for respiratory failure by mechanical ventilatory assistance through cuffed tracheostomy tubes have been increasingly recognized.[1, 8, 10, 13] The symptoms are those of airway obstruction, massive hemorrhage or aspiration pneumonia. Symptoms arise from lesions which result from a relative balance of ulcerative and reparative processes; the lesions include tracheal stenosis, tracheal malacia and localized erosion. Similar complications have long been recognized to arise from the tracheal stoma itself. The lesions which characteristically result from assistance through cuffed tubes, however, are usually found a few centimeters below the stoma.[10, 13]

This report describes the pathology of the characteristic injuries which occurred in 30 patients who had received respiratory assistance through cuffed tubes but who died from causes other than upper airway damage. Lesions found at the site of the tracheostomy are not specifically discussed, since these occur independently of respirator assistance. The varying duration of respiratory assistance prior to death provides information on the evolution of the lesions. A small number of injuries related to ventilatory assistance through endotracheal tubes is also presented for comparison.

Surgically resected specimens were also studied from an additional group of patients who had developed tracheal stenosis in the segment located from one to 3.5 cm. below the stoma. We have previously described the management of such benign tracheal stenosis and have also briefly noted the pathology of these fully developed stenotic lesions.[10, 11]

Specimens

Tracheas were removed at autopsy from patients who had been receiving assisted respiration through a cuffed tracheostomy tube or an endotracheal tube at the time of death. When possible, specimens were removed with the tubes in place in order to correlate the site of damage to the trachea with the location of the tube and its cuff. The trachea was removed intact, including the larynx and carina. The duration of intubation and assisted ventilation was noted from the hospital records. In the 30 patients who received respiratory assistance through a tracheostomy, the duration of intubation ranged from 1 day to 8 weeks (Table 1). In some cases, an endotracheal tube had been in place for several days prior to the insertion of a tracheostomy tube. Four specimens were studied from patients who had received respiratory as-

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TABLE 1. *Pathologic Changes in Tracheas of 30 Patients Who Received Respiratory Assistance Through Cuffed Tracheostomy Tubes for Varying Periods of Time.* Autopsy Specimens*

Case No.	M.G.H. Pathology No.	Duration of Tracheostomy Tube (days)	Gross Pathologic Findings
1	31623	1	Superficial changes—tracheitis
2	31655	1	Circumferential ring of hemorrhagic change
3	31616	2	Small anterior tracheal ulceration at cuff site
4	31628	2	Early anterior ulcerations
5	31654	2	Circumferential band of fibrin at cuff site
6	31862	3	Three cartilaginous rings exposed anteriorly at the cuff site
7	31629	3	Circumferential discoloration and one exposed cartilaginous ring at cuff site
8	31780	3	Small area of exposed cartilage anteriorly at the cuff site
9	31823	3	Cartilaginous rings exposed anteriorly at the cuff site
10	31651	4	Discoloration of the anterior tracheal wall with superficial erosions over the cartilaginous rings
11	31810	4	Fibrin deposition and superficial ulcerations anteriorly
12	31828	4	Small anterior ulcerations
13	31946	5	Exposed cartilaginous rings at the cuff site and a small anterior erosion at site of tracheostomy tube tip
14	32022	6	Scattered erosions in the cuff area
15	31967	7	Extensive baring of cartilage and small perforation through the posterior tracheal wall at the cuff site
16	31818	7	One cartilaginous ring widely exposed with several erosions above and below this level. Tracheitis from the cuff site down
17	31779	8	Four cartilaginous rings widely exposed anteriorly at the cuff site
18	31825	9	Ballooning out of the trachea at the cuff site with severe exposure of cartilage. Perforation of the membranous portion of the trachea with local reaction in the esophageal wall
19	31725	9	Ulcerations in the tracheal wall in the region of the cuff
20	32004	9	Several rings exposed at various points in the region of the cuff
21	32038	10	Severe exposure of numerous cartilaginous rings in the region of the cuff with extensive fragmentation of cartilage. Large perforation through posterior wall of trachea with local reaction in adjacent esophagus
22	31646	12	Baring and softening of cartilage in the region of cuff
23	31806	16	Severely eroded and fragmented cartilage at the cuff site. Numerous pieces of cartilage missing. Severe loss of tracheal wall support
24	31851	18	Several cartilaginous rings widely exposed and thinned in the region of the cuff
25	31890	19	Two exposed, thinned and fragmented cartilaginous rings at cuff site. Small anterior ulceration at the site of the tip of the tube
26	32028	19	Extensive baring, fragmentation and exposure of cartilaginous rings at the stomal site as well as in the region of the cuff below the stoma
27	31944	21	Extensive damage to cartilaginous rings with ulceration, baring of cartilage and fragmentation of cartilage at the cuff site. Small shallow ulcer located at the tip of the tracheostomy tube
28	31821	21	Almost no visible damage except for slight discoloration
29	31694	42	Extensive fragmentation of cartilage, many areas devoid of cartilage, marked loss of support in tracheal wall at the cuff site
30	31594	60	One centimeter segment of trachea totally devoid of cartilage and totally flexible

* None of these patients had symptoms of upper airway obstruction at the time of death.

TABLE 2. *Pathologic Changes in Tracheas of 4 Patients Who Received Respiratory Assistance Through Cuffed Endotracheal Tubes only for Short Periods.* Autopsy Specimens.*

Case No.	M.G.H. Pathology No.	Duration of Endotracheal Tube (days)	Gross Pathological Findings
31	31643	4	Fibrin deposition and superficial ulcerations
32	31798	4	Fibrin deposition at cuff site with superficial shaggy ulcerations
33	31625	5	Circumferential band of discoloration and fibrin deposition with ulcerations exposing several cartilaginous rings
34	31750	7	Minor erosions at cuff site. (The cuff of this Portex tube was pre-stretched)

* None of these patients had symptoms of upper airway obstruction at the time of death.

sistance for 4 to 7 days through an endotracheal tube only (Table 2).

The tracheas were opened along the membranous portion with the tracheostomy tube in place, when possible. The location of the tracheostomy was noted and used as a reference point from which to measure the location of the distal damage to the

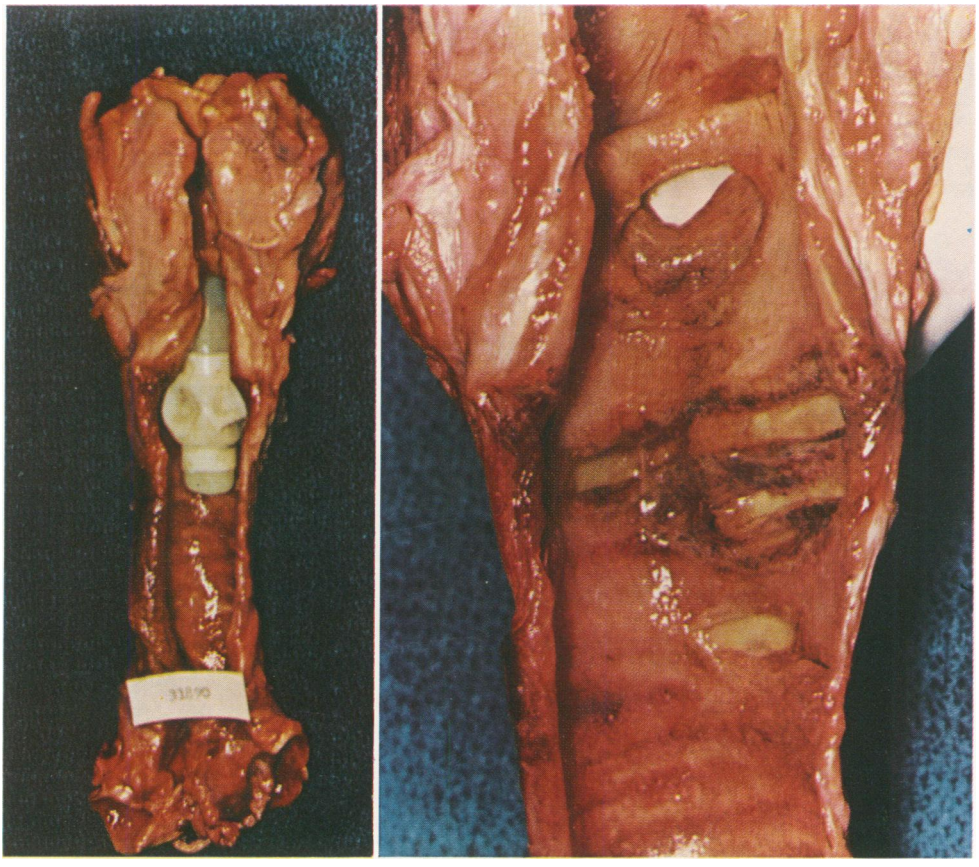

FIG. 1. (Case 25) Trachea shows moderate damage to cartilages at balloon site. Tube was in place for 19 days. a. (left) The portex tracheostomy tube is in place, deflated. The lateral tracheal walls are thinned and expanded at the balloon site. b. (right) Several rings of cartilage have been exposed and fragmented at the cuff site. There is, also, a small ulcer down to the cartilage located at the site of the tip of the tracheostomy tube.

TABLE 3. *Clinical Characteristics and Findings in 11 Patients Treated Surgically for Upper Airway Obstruction Following Respiratory Assistance Through Cuffed Tracheostomy Tubes.**

Case No.	M.G.H. No.	Disease	Age	Sex	Days of Assisted Ventilation	Symptom Free Interval (days)	Gross Pathologic Findings: Location in Relation to Stomal Site	Length of Lesion (cm.)	Circumferential Fibrous Diaphragm	Other
35	915321	pneumonia, empyema, bronchopleural fistula, lobectomy	40	M	4	10	3 cm. below	1.5	yes	
36	1439977	aneurysm of axillary artery, mediastinal sepsis	66	F	9	10	just below	3.0	yes	
37	1418409	multiple injuries, head injury	18	M	10	10	2.5 cm. below	3.5	yes	granulations at stoma
38	1424589	obesity, emphysema, myocardial infarct	62	F	30	2	2 cm. below (and at stoma)	4.0	yes	
39	1303057	toxic ingestion, bilateral pneumonia	31	F	35	64	3 cm. below	2.0	yes	
40	1458650	polyneuropathy	39	F	38	24	2 cm. below	1.5	yes	
41	1465648	crushed chest, multiple injuries, emphysema and bronchitis	61	M	38	24	3 cm. below	3.0	yes	malacia above stenosis
42	928062	rheumatic heart disease, ventricular fibrillation, multiple cardiac arrests and chest wall instability	67	F	57	2	2 cm. below	1.0	yes	malacia above stenosis
43	1507996	polyneuropathy	48	F	50	a few days	3.5 cm. below	2.0	yes	
44	1274113	aortic valve replacement, mediastinal sepsis, renal failure	36	F	61	0	3.0 cm. below	3.0	yes	diffuse destruction
45	1425995	abdominal trauma, aspiration pneumonia, emphysema	25	M	112	11	1.5 cm. below	1.0	yes	

*The surgical aspects of cases in this table except Case 43 have been previously reported[10]. During the period of this study four additional patients were treated who had undergone initial resection of stenotic lesions at other institutions, one whose stenosis occurred so late that it could not be precisely separated from late stomal cicatrization and one whose stenosis was post-traumatic.

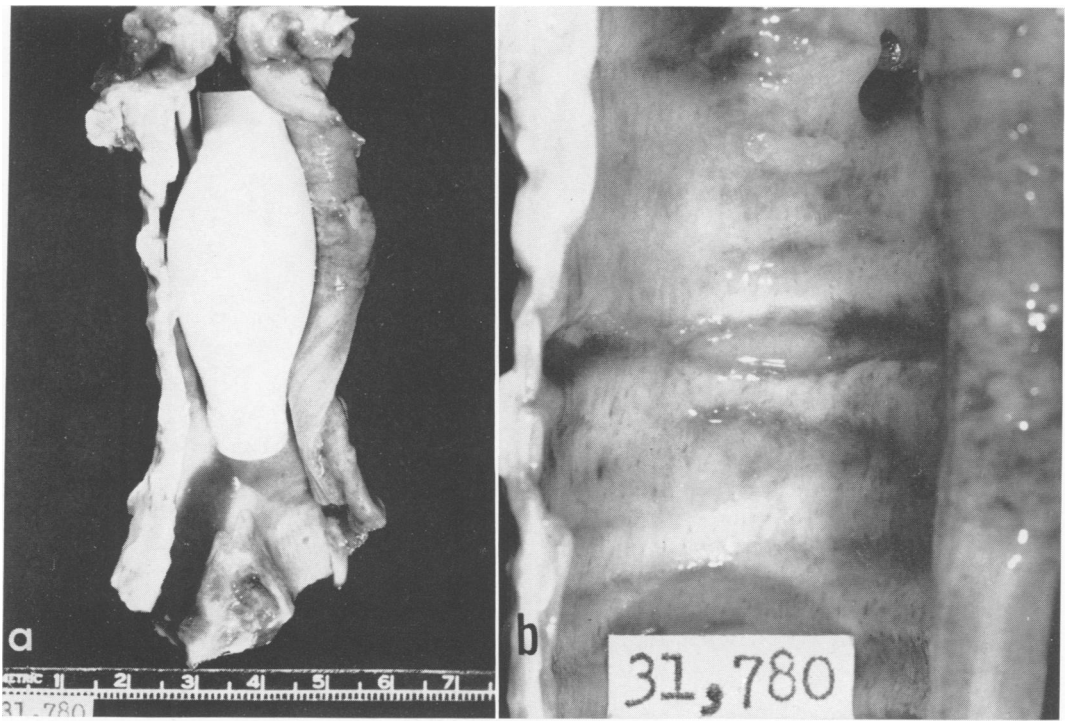

FIG. 2. (Case 8) Trachea shows slight damage from balloon cuff. Tube was in place for 3 days. Specimen opened along posterior wall. a) The tracheostomy tube in place with the balloon inflated. b) A small anterior ulceration down to cartilage is located at the site of the midpoint of the balloon cuff.

trachea. All specimens were photographed and diagrams were made of the location and nature of damage. The entire specimen was then fixed in 10% formalin and microscopic sections were taken from involved and uninvolved areas.

The eleven surgical specimens were all removed to relieve clinical airway obstruction due to stenosis occurring below the tracheostomy site (Table 3). These patients had received ventilatory support for periods ranging from 4 to 112 days and had been asymptomatic from 2 to 64 days following removal of the tracheostomy tubes. Surgical management of most of these patients has been previously reported.[10]

Observations

Autopsy Specimens

A consistent pattern of tracheal damage was observed. The location of major damage correlated with the site of the balloon cuff. In general, the amount of damage increased as the period of mechanical ventilation was prolonged (Table 1). Early changes were superficial tracheitis with fibrin deposits. Tracheitis was maximal in the area of the cuff but sometimes extended to the carina. Small shallow ulcerations overlying the anterior portion of the cartilaginous rings at the level of the cuff were seen early (Fig. 2a, b). The size and extent of the ulcers increased with time, leading to exposure of the cartilaginous rings. Softening, splitting and fragmentation of cartilage followed at a later stage. In most cases the tracheal wall bulged where the balloon had been located, indicating that the cuff had exerted significant pressure on the trachea. In general, this area of damage began one to 1.5 cm. below the inferior

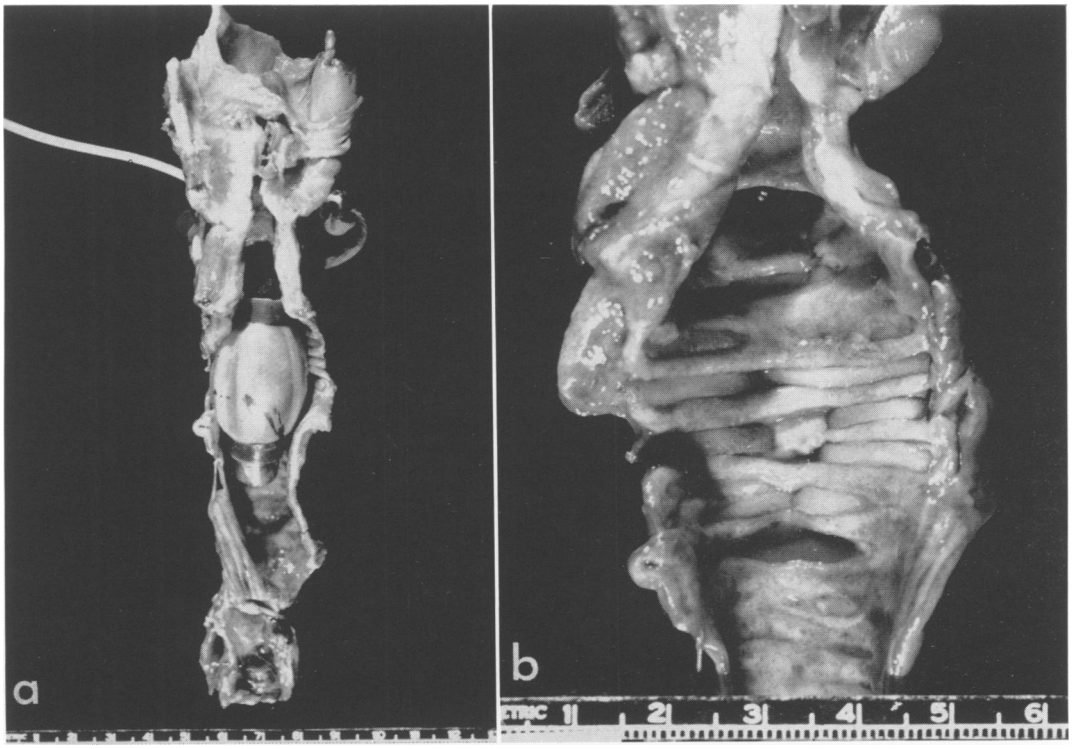

FIG. 3. (Case 23) Trachea shows severe damage at balloon site. Tube was in place for 16 days. a) The silver tracheostomy tube is in place with rubber cuff expanded. b) Several rings located at the balloon site have been extensively bared, softened and fragmented. There is considerable loss of support at this location.

margin of the tracheostomy stoma and extended downward for a length of 1.5 to 2.5 cm. With time two to four cartilaginous rings became completely bared, softened and fragmented so that segments of cartilage were totally missing (Fig. 1a, b). In the final stage, the segment of trachea at the balloon site was completely devoid of cartilage, had lost any firm substance and presented as a completely flexible and collapsible tracheal segment (Figs. 3a, b and 4a). Wherever a cuff had been in place for 2 days or more the balloon site demonstrated definite changes. Within 10 days to 2 weeks, several rings of cartilage were usually widely exposed, softened and fragmented. At a level slightly below that of the cuff damage, usually anteriorly, additional injury was sometimes identified at the site of the tip of the tracheostomy tube. Damage at the level of the tip of the tracheostomy tube was never circumferential and was usually of considerably lesser extent than that seen at the balloon site. Frequently, lateral instability was present at the stoma, as well, whenever the tracheostomy was of large size. In the four patients who had received assistance through endotracheal tubes only, the changes were comparable with those seen in tracheostomy patients (Table 2).

Microscopic examination mirrored the progressive changes found grossly (Figs. 5a–c and 6a, b). Acute inflammation, hemorrhage and fibrin appeared early. Microscopic ulceration next appeared overlying the cartilaginous rings. The ulcers deepened progressively and widened to expose totally the surface of the rings. The surface of the cartilage was bared and inflammatory changes could be seen superior and inferior to the cartilage. Later, acute in-

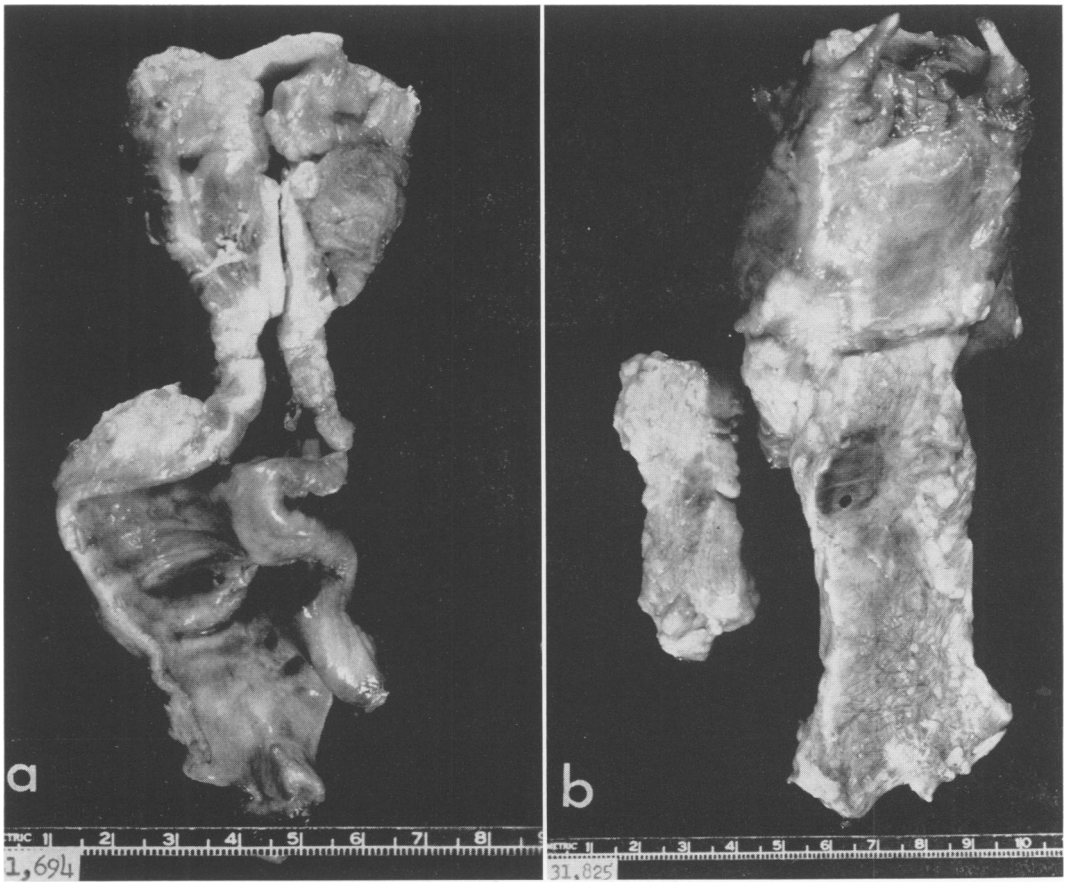

FIG. 4a. (Case 29) Trachea shows severe loss of supporting structures. The tube was in place for 42 days. There is extensive loss of cartilaginous rings. Specimen has been folded to illustrate the extent of loss of rigidity. b) (Case 18) Unopened trachea shows perforation of the posterior tracheal wall located at the site of balloon cuff. The tube was in place for 10 days. A 2 by 1.5 cm. area of the posterior wall is paper thin and perforation has occurred in the lower portion of this area. The adjacent portion of the esophagus is shown to the left of the tracheal specimen. The wall of the esophagus adjacent to the perforation shows inflammatory changes. Cases 15 and 21 showed large perforations through the posterior wall of the trachea at the site of the cuff.

flammatory changes extended completely around the exposed rings. The cartilage fragmented and broke up leaving an ulcer crater where the cartilage had been. Between ulcerations resulting from destruction of adjacent rings, a septum of tissue remained, initially containing residual glands which showed squamous metaplasia. At a later stage the base of the ulcerations began to re-epithelize but showed squamous metaplasia. Prior to epithelization, granulation tissue covered the ulcer base.

Severe damage to the compliant membranous portion of the trachea was seen less often than severe damage to the more rigid, non-yielding cartilaginous portion. Nonetheless, three instances of perforation of the membranous wall occurred (Fig. 4b). These perforations were all located at the balloon site. In all there were some inflammatory changes in the adjacent esophagus —but no perforation of the esophagus itself. It was obvious that in a short time a tracheo-esophageal fistula would have developed, as has been encountered clinically in such situations.[4, 8]

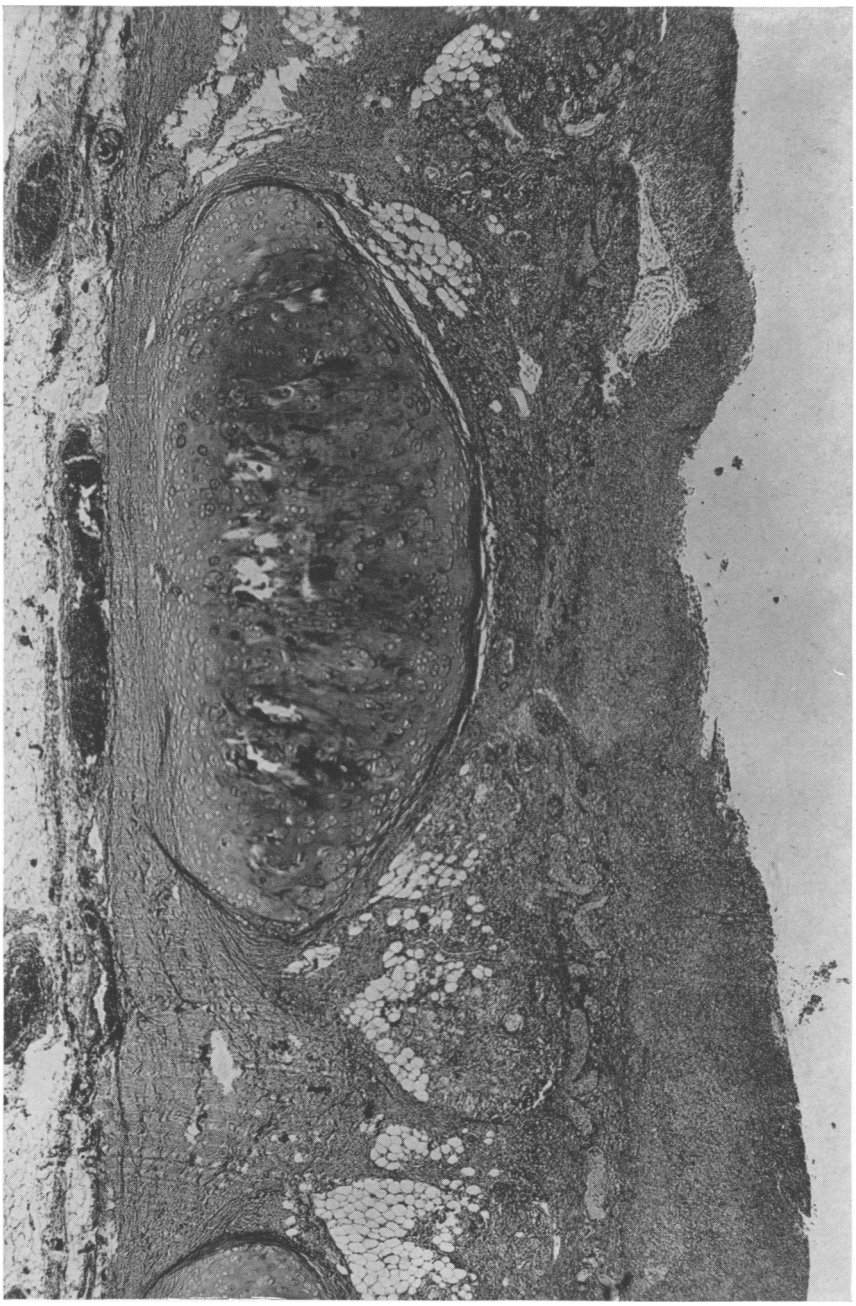

FIG. 5a. Microscopic evolution of destruction of cartilages. Slight damage to tracheal mucosa is shown. Fibrin deposition is present on the surface and an extensive acute inflammatory change with many polymorphonuclear leukocytes. Tube was in place for 2 days (Case 5). H&E, ×27.

Surgically Resected Specimens

In those patients in whom healing had occurred and who had developed acute respiratory obstruction following extubation, characteristic cicatricial stenosis was found (Table 3). A firm circumferential web of dense fibrous tissue was found located between 1.0 to 3.5 cm. *below* the tracheostomy site with a central airway measuring from 2 to 5 mm. (Fig. 7a, b). From outside the trachea it was usually possible to identify an hourglass-shaped constriction with loss of the usual cartilaginous structure in the center of the con-

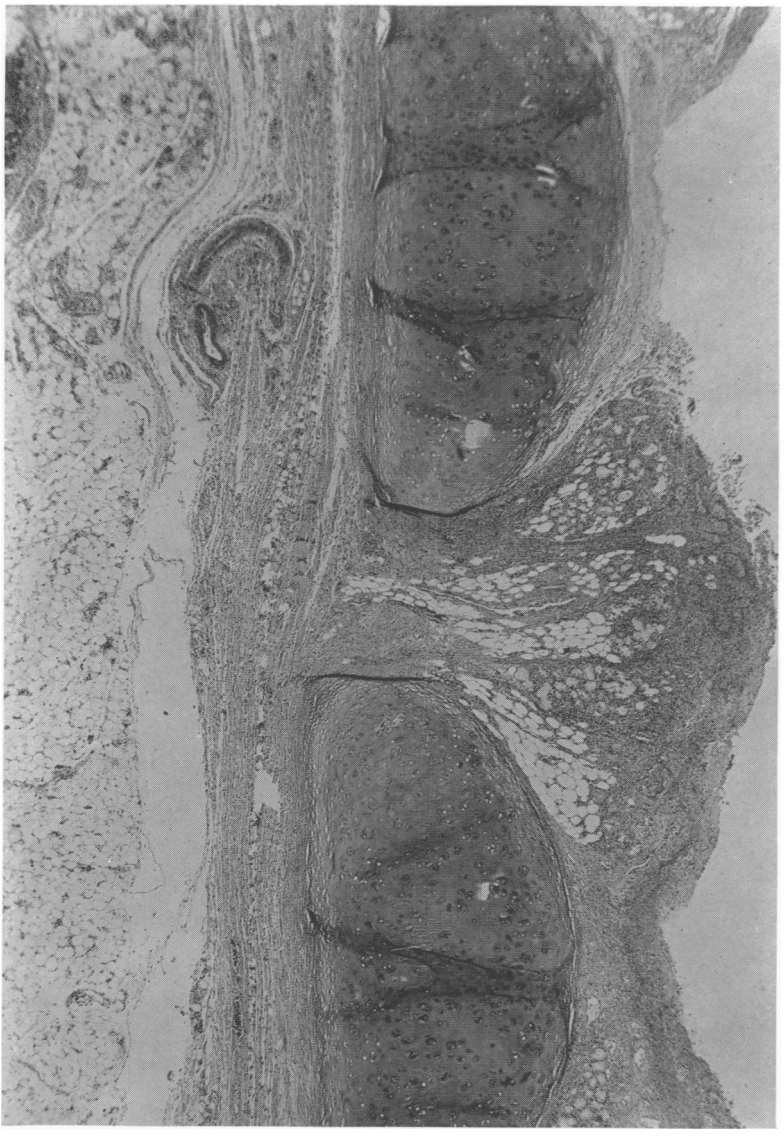

FIG. 5b. Ulcerations are present over the prominent points of the cartilaginous rings located at the site of the balloon cuff. Initial ulceration developed over the central portions of the cartilaginous rings. One ring shown here has been almost totally exposed. Tube was in place 3 days (Case 7). H&E, ×24.

striction and lesser degrees of damage for one centimeter proximal and distal to the mid-point of the narrowing. Within, there was further airway narrowing by the dense web of scar tissue. The superior and inferior surfaces of this cicatricial tissue were usually unepithelized, although in some of the prolonged cases metaplastic epithelium crept close to the remaining lumen. In many cases there was some degree of damage between the tracheostomy site and the level of this cicatricial web. This consisted of residual chronic tracheitis, some fibrosis or loss of cartilaginous substance and, in extreme cases, a collapsible segment due to tracheomalacia. The trachea distal to the stenosis was usually relatively normal grossly.

Microscopically, the fully developed lesion consisted of dense scar tissue with only some attempts at squamous epithelization laterally. The residue of cartilages was sometimes present within the wall of the trachea adjacent to the stenosis. Variable

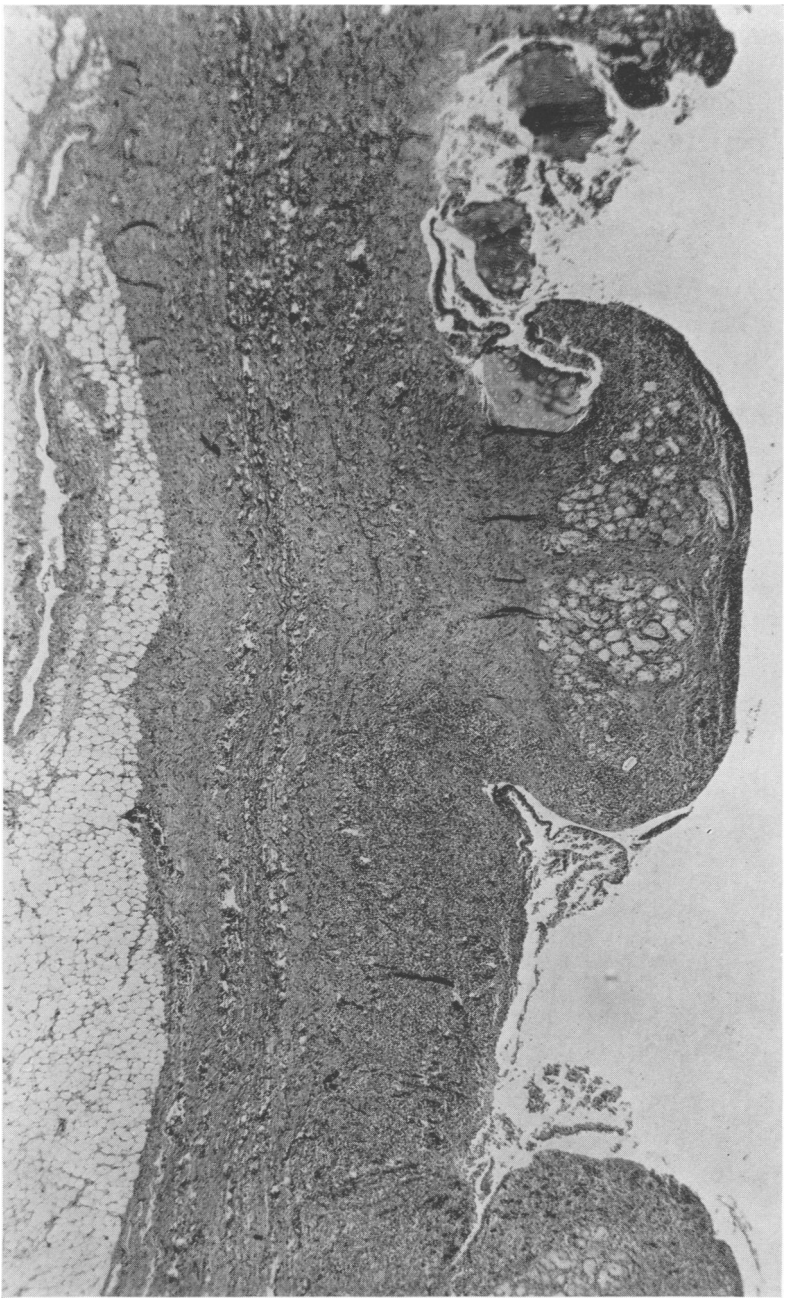

FIG. 5c. Specimen shows severe damage to cartilaginous rings and tracheal wall. In one ulcer crater only a few fragments of cartilage remain at the site of the ring. In the other ulcer crater the cartilaginous ring has been totally sloughed. The full thickness of the tracheal wall shows extensive inflammatory changes (Case 29). H&E, ×24.

degrees of fibrotic scarring were present in these mature lesions at the site of maximal damage in the tracheal wall. In a number of patients, when the lesion was relatively lengthy and quite acute, there was total replacement of the tracheal wall by granulation tissue and scar tissue in varying stages of maturity. No tracheal structure was identifiable at all in such cases (Fig. 8a, b).

Discussion

These pathologic studies show that some damage occurs consistently in essentially

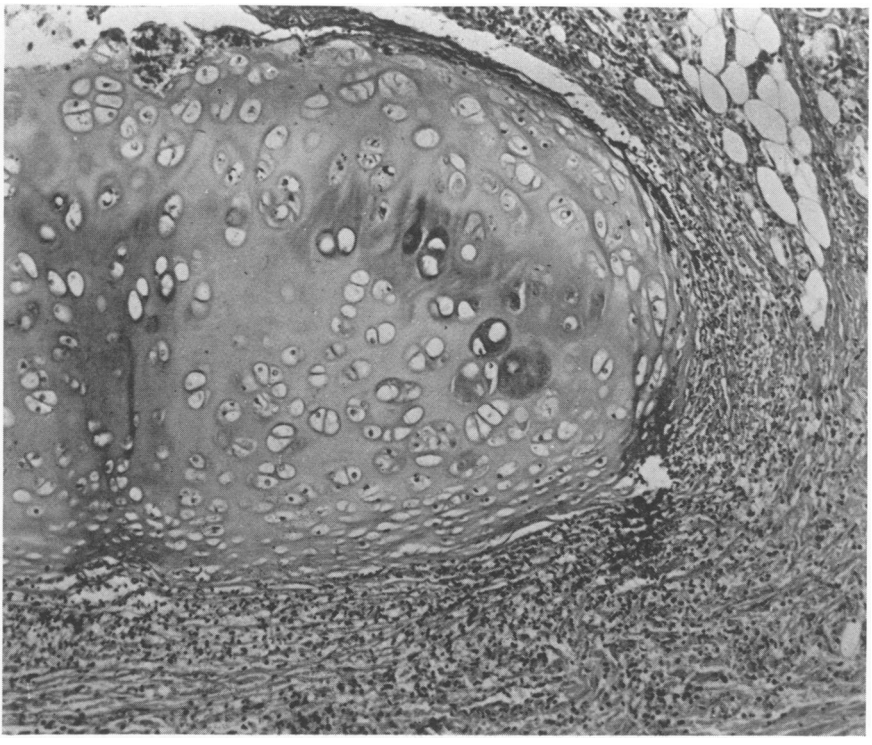

FIG. 6a. High power details of early destruction of cartilage. The changes of acute inflammation are seen along the side and lower surface of the cartilaginous ring. This occurred after the superficial surface had been totally exposed by ulceration from the balloon cuff. Tube was in place for 12 days (Case 22). H&E, ×75.

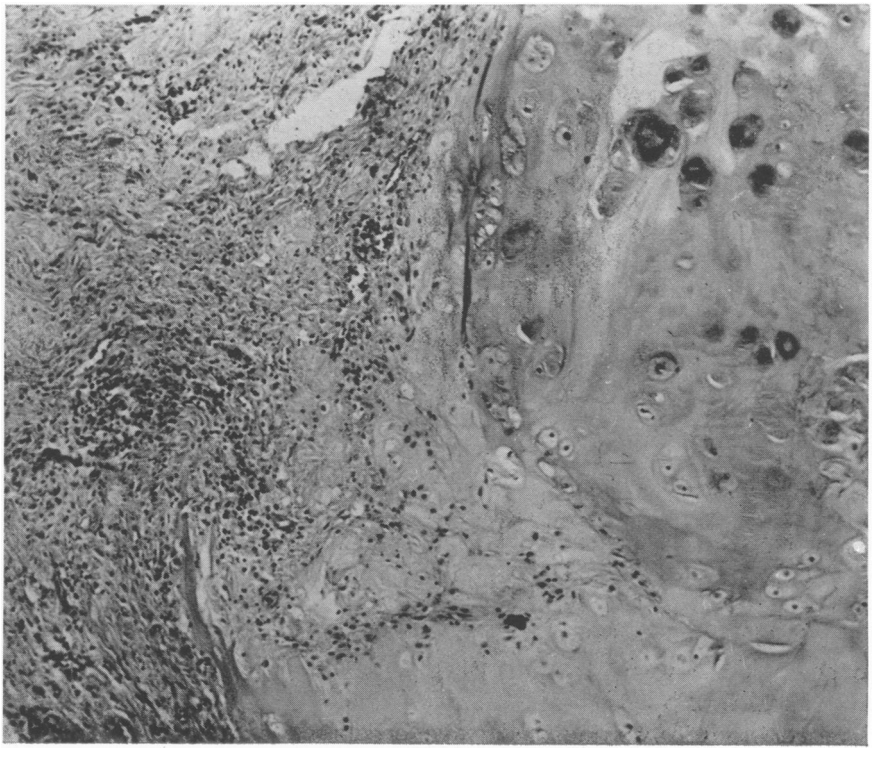

FIG. 6b. Cartilaginous ring shows ingrowth of inflammatory cells. The surface of the cartilage was totally exposed and the ring was surrounded by acute inflammatory cells. (Case 30). H&E, ×100.

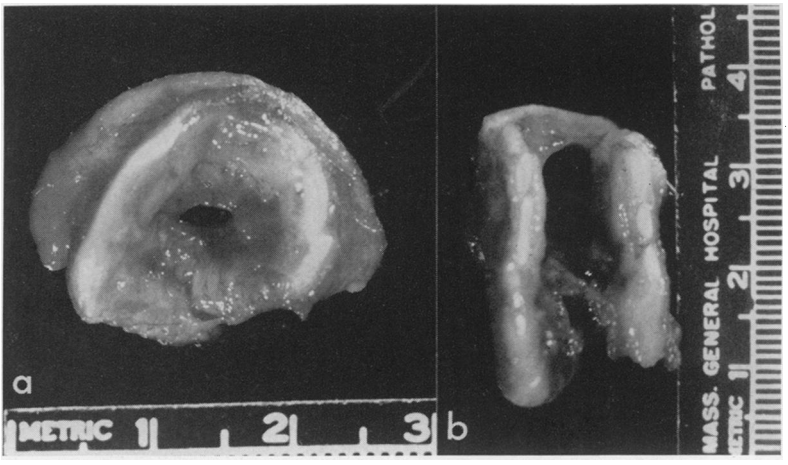

FIG. 7. Surgically resected stenotic segments of trachea. a) (Case 45) Severe stenosis at balloon site. The effective airway measures 2 × 3 mm. b) (Case 37) Specimen opened longitudinally along posterior wall. The tracheostomy may be seen above, with a fibrous tissue diaphragm below. There is destruction and fibrosis in the wall of the entire specimen. Thinned cartilages are visible in the upper portion of the tracheal wall. Contiguity of the severe changes required inclusion of the stoma in the resected specimen.

every human trachea through which respiratory assistance has been given using a standard cuffed tracheostomy tube for a period of 48 hours or more, although in none of the autopsy subjects had there been any evidence of upper airway obstruction at the time of death. Most of these patients were cared for in a respiratory unit where meticulous attention was given to periodic deflation of the cuff and where the amount of air used to inflate the cuff was just sufficient to provide a seal. Plastic tubes with plastic balloons and silver tubes with rubber cuffed balloons were used primarily; injury occurred with both types of tube. A spectrum of lesions occurs which commences with superficial tracheitis, progresses to necrosis and loss of cartilage and, in an occasional case, to posterior necrosis with tracheo-esophageal fistula. Maximal damage was always seen at the site of the cuff, although non-circumferential erosions were occasionally also evident in relation to the tip of the tube (Fig. 9). Such uniform damage has been described in a prospective study by Pearson, Goldberg and da Silva.[13] They observed bronchoscopically through the stoma that every patient who had been on respiratory assistance with a cuffed tracheostomy tube had developed significant de-

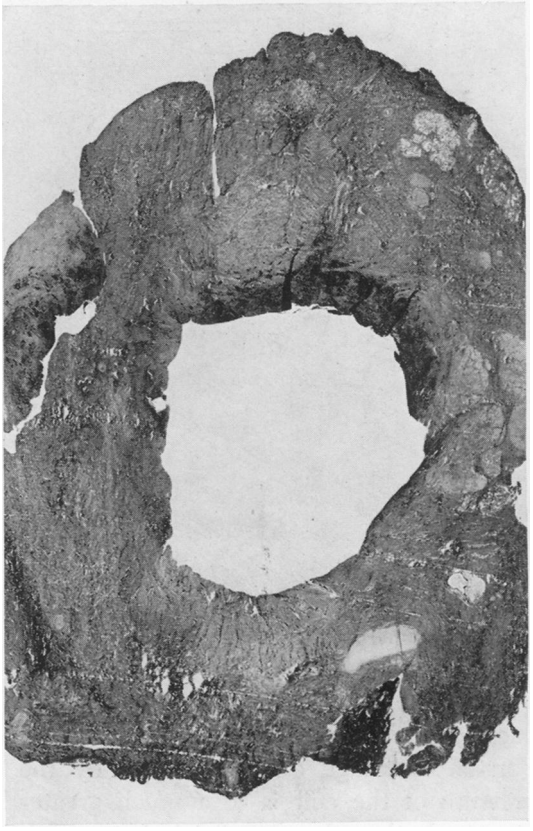

FIG. 8a. Microscopic appearance of surgically removed, fully developed, circumferential stenoses at balloon cuff site. Cross section of stenotic diaphragm shows thickening of the tracheal wall, loss of all cartilages and normal structure and severe degree of fibrous stenosis (Case 35). H&E, ×11.

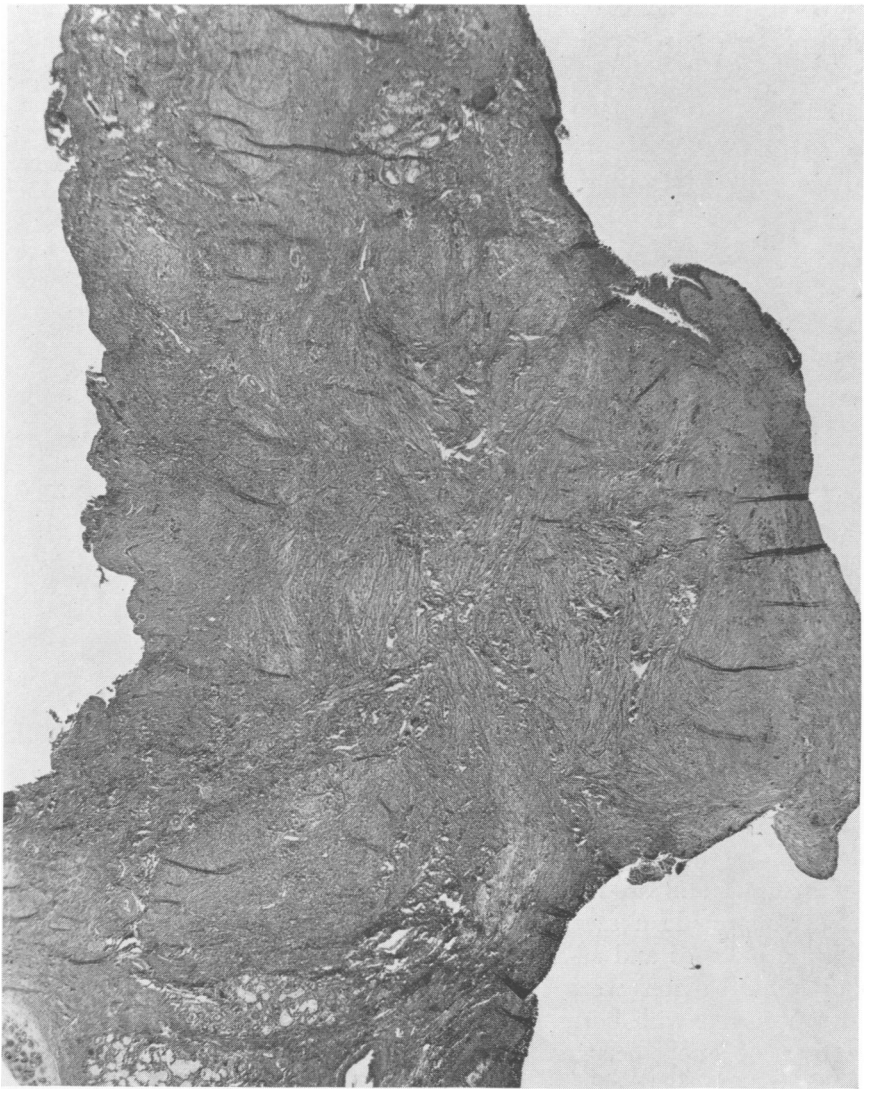

FIG. 8b. Longitudinal section shows total loss of cartilage and replacement of the tracheal wall with fibrous tissue. The prominent projection in the photomicrograph was part of the stenotic diaphragm (Case 40). H&E, ×25.

grees of damage primarily at the site of the cuff. Tracheal damage due to cuffed tubes has also been described by numerous observers [1, 6, 9, 15-17] and the pathology has been described by Florange, Muller and Forster.[5] Damage due to erosion by the pressure of the cuff is of a much greater magnitude than the superficial changes following tracheostomy alone.[2, 7, 14]

These present observations confirm that pressure necrosis is the primary cause of damage. With shorter periods of injury, recovery may occur more easily. The degrees of pressure exerted by the standard tracheostomy cuff are remarkably high. We have studied these relationships in more detail and have produced comparable lesions experimentally.[3] The results of these studies will be reported in detail elsewhere. The role of bacteria, which are always present, is not clear [10, 12-14] but may be that of an accessory factor.

Study of the surgically resected specimens demonstrates destruction of the normal structure of the tracheal wall and evolution of a stenotic ring by attempts at ci-

catricial healing (Fig. 9). Total loss of structure with replacement by granulation tissue permits no alternative to a concentric cicatrix which tends to contract with time. The process explains the general failure of dilatation as a method of treatment—unless the lesion is not circumferential. The location and nature of the fully developed stenosis correlates with the location and nature of the tracheal injury found at the balloon site in the 30 autopsy specimens. It is clear that stenosis is the end stage of healing of the erosive processes seen in the autopsy specimens.

Since the lesion occurs at the site of the cuff it is not surprising that prolonged intubation with an endotracheal tube led to the same type of erosion. The endotracheal tube itself may also cause pressure erosion at a narrow point of the airway—at cricoid level. Significant lesions may also occur at the tracheostomy sites; such lesions are not necessarily related to respiratory assistance and have been well identified in the past.

As more patients with respiratory failure survive as a result of expert care and as awareness of this clinical syndrome increases, the number of patients recognized to have serious degrees of stenotic or malacic tracheal damage has increased steadily. The incidence of recognized clinical symptoms has risen as high as 15% of patients surviving treatment for respiratory failure by these methods.[8, 13] A remarkable degree of narrowing may be accepted by the relatively sedentary patient without symptoms. Various attempts have been made to prevent these lesions, including use of alternately inflated double-cuffed tubes, intermittently inflated cuffs cycled to the respirator, serial readjustment of the level of the cuffed tube using spacers of various types and the use of large bore tubes with a respirator of high volume flow. Each of these methods presents certain difficulties and complexities. We have recently demonstrated that these lesions can be prevented experimentally by use of a new type of cuff which differs in pressure-volume characteristics from the standard cuff.[3]

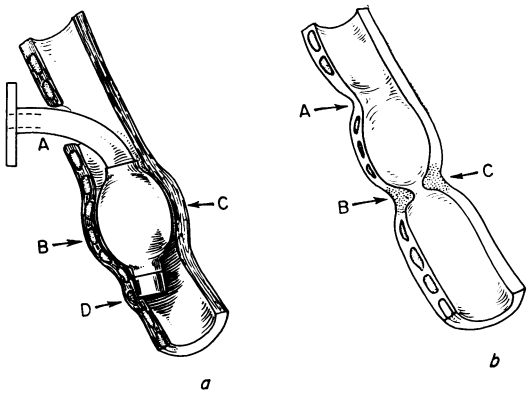

FIG. 9. Diagram summarizes (a) points of injury and (b) results of healing at these sites. At site A, the stoma, local necrosis occurs which may heal with various deformities of the anterior wall of trachea, including a shelf-like, partial obstruction. This is where pseudo-polyps of granulation tissue occur. These lesions are independent of ventilatory assistance. B and C are locations of compression by the inflated cuff. With deep erosion and destruction of cartilages, a stenotic diaphragm results during healing (b). The cartilages between A & B may be thinned and the trachea loses rigidity. Tracheo-esophageal fistula may occur at C. The tip of the cannula may erode, most often at D, anteriorly. The innominate artery crosses the trachea at about this point and may be eroded occasionally.

Conclusions

Tracheal stenosis, malacia and erosion have increasingly occurred as serious complications of ventilatory assistance through cuffed tracheostomy tubes. Pathologic study of the tracheas of 30 patients, who died from causes other than upper airway lesions while receiving ventilator assistance, demonstrated the universal presence of tracheal damage at the site of the cuff. Such lesions appeared within 48 hours and progressed from tracheitis to ulceration of mucosa to fragmentation of cartilage to replacement of the tracheal wall with scar tissue. Correlation of the surgically resected specimens from 11 patients treated for ventilatory obstruction arising from such assisted respiration, showed the evolution of the circumferential lesion at the cuff site

into a tight, infrastomal stenosis by the usual processes of wound healing. The lesions clearly result from pressure necrosis even with well-managed conventional cuffs. Preventive measures must be based upon these facts.

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