Pathology of the Lung in Fatally Burned Patients

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While direct thermal damage to the lower respiratory tract has been demonstrated to occur only with the inhalation of steam,[7] respiratory complications are a common fatal factor in patients sustaining thermal injury.[2, 3, 5, 11, 14, 15] Some clinical studies implicate inhalation of irritating products of combustion as the primary cause of respiratory complications and recent reports of a high rate of pulmonary lesions associated with facial burns have been interpreted as reinforcing this premise.[10] Our experience, however, indicates that tracheobronchitis and pneumonia often are not directly related to facial burns or inhalation injury and are frequently a complication of tracheostomy, a procedure common in the immediate postburn period. The difficulty in separating respiratory complications due to inhalation injury from those due to tracheostomy is further compounded by the problem of clinically differentiating respiratory distress due to intrinsic pulmonary complications, from that due to alterations in cardiovascular hemodynamics following extensive burns, or as a compensatory response to acid-base imbalance. Evaluation is especially difficult when the study group also contains elderly patients who may have unrecognized as well as overt pre-existent cardiorespiratory problems.

The study reported herein attempts to clarify some of these difficulties by defining pathologic aspects of pulmonary complications that occur following cutaneous burns. The study group is unique in that it consists largely of active duty military personnel and their dependents, who are virtually free of significant pre-existent respiratory disease. The necropsies represent the largest group so studied and were performed by pathologists who autopsied burned patients almost exclusively and therefore were aware of the peculiarities of such autopsies. The pathologists at this installation customarily make daily rounds with the surgeons and the opportunity of following the clinical course of each patient renders the interpretation of the autopsy findings singularly appropriate.

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Material

There were 243 deaths from burns and 233 autopsies performed at the US Army Surgical Research Unit from 1960 through 1965 (autopsy rate, 96%). During the same period, a total of 817 patients were hospitalized for treatment of burns and Table 1 lists the types of burns encountered in both groups. Ninety per cent of patients at autopsy had sustained flash or flame injuries. Patients who sustained electrical injury without flame burns were excluded from the study group.

Distribution of autopsied patients by age and extent of thermal injury demonstrates the relative youth and magnitude of burns in patients received at this unit (Tables 2,

TABLE 1. *Type of Burns Encountered in Patients Treated and in Autopsy Population*

Year	No. Patients* Hospitalized	No. Patients* Autopsied	Flash and/or Flame	Electrical	Steam	Hot Liquid	Chemical	Contact
1960	96	30	78 (23)	2 (2)		11 (5)	1	4
1961	88	30	76 (29)			8 (1)		4
1962	142	51	124 (46)	2		13 (5)	3	
1963	148	56	136 (54)	1	1 (1)	9 (1)		1
1964	184	34	147 (31)	7 (1)	2 (1)	24 (1)	3	1
1965	159	32	125 (28)	6 (1)	1	17 (2)	4 (1)	6
TOTAL	817	233	686 (211)	18 (4)	4 (2)	82 (15)	11 (1)	16 (0)

* No. in ( ) represents autopsies.

3). Over 90 per cent of burned patients treated during this period were under 50 years of age and this is accurately reflected in the autopsy population since over 90 per cent of patients dying with burns were below 50 years of age. Also, 50 per cent of patients treated had sustained over 30 per cent total body area burns and patients with burns of this magnitude accounted for 95 per cent of deaths and autopsies.

All autopsy protocols, clinical summaries and tissue sections from the respiratory system were reviewed. A total of 1,821 histological sections of the respiratory tract, an average of 7.8 per autopsy, were available to study. Paraffin blocks were recut and restained and additional special stains were done when necessary. The pathologic diagnoses in the respiratory system were listed after review of the clinical and gross autopsy data and personal review of all sections. In addition, the causes of death were reviewed and determined from the clinical and pathologic material.

Results

Cause of Death. The major cause of death during the period 1960–3 was burn wound sepsis which, as previously defined, is due to massive bacterial invasion of the burn wound.[6, 12] In 1964–5, topical chemotherapy of the burn wound with Sulfamylon was instituted which significantly reduced mortality from burn wound sepsis and the major fatal complication shifted to the respiratory system (Fig. 1). The decline in burn wound sepsis as a cause of death in reference to both admissions and total deaths during 1964–5, compared to 1960–3, is highly significant ($p < 0.001$). The successful control of bacterial invasion of the burn wound with Sulfamylon has been reported.[4]

The major cause of death revealed at autopsy in all 233 burned patients is given in Table 4. Death was attributed to septicemia when one or more antemortem blood cultures were positive for bacteria in association with clinical features of sepsis and a source of infection that was determined at autopsy. In the absence of a source of infection elsewhere, septicemia was considered to be of pulmonary origin when extensive pneumonia was present clinically and confirmed at autopsy. Deaths with septicemia associated with pneumonia showed a proportionate increase in 1964–5 compared with 1960–3 due to decline in sepsis originating from invasive infection of the burn wound. A third and increasingly frequent cause for septicemia in the burned patient is suppurative phlebitis that involves cannulized veins (Table 4). Deaths attributed to sepsis also occurred in one pa-

tient with suppurative cholecystitis and in one pateint with a mandibular abscess, and this accounts for the two patients listed in Table 4 with miscellaneous sources of infection. Patients who had positive blood cultures and were considered to have septicemia clinically, and in whom necropsies did not disclose an origin of infection, were categorized as having septicemia of uncertain origin.

The mortality table (Table 4) underestimates the true incidence of suppurative phlebitis as well as Curling's ulcers in burned patients since these complications are successfully treated in most instances. Seven of 8 patients whose cause of death was gastroduodenal ulceration died from repeated episodes of hemorrhage; one died from peritonitis following perforation of a duodenal ulcer.

Those deaths attributed to renal failure have been due to inadequate resuscitation in the early postburn period and no deaths were placed in this category when renal insufficiency was but a terminal feature of the major cause of death, for example, burn wound sepsis. There were eight deaths attributed to renal failure on the basis of oliguria with a rapidly rising serum potassium. In addition, there were five deaths attributed to severe hypotension in the early postburn period that are listed in the miscellaneous category and these were also thought to have resulted from inadequate restoration of fluid and colloid. There were, therefore, 13 deaths in these two groups that might be considered as deaths due to inadequate resuscitation prior to evacuation to this unit.

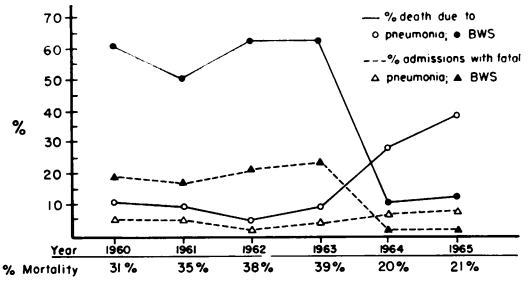

FIG. 1. Incidence of fatal pneumonia and burn wound sepsis in 817 burned patients hospitalized and 233 autopsies.

Other causes of death in the miscellaneous group include: coronary heart disease (4), acidosis (3), adrenal hemorrhage (2), pancreatitis (2), pulmonary emboli (2), peritonitis (2), cerebral vascular thrombosis (1), hepatic cirrhosis (1), subacute bacterial endocarditis (1), aortic-esophageal

TABLE 2. *Distribution of Patients by Age and Per Cent Mortality*

Age	0–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80	80–90	Total
No. Burned	228	101	232	138	62	29	17	7	3	817
Deaths	79	27	46	42	29	11	3	4	2	243
% Mortality	35	27	20	30	47	38	18	57	67	29.7
Autopsies	78	27	44	38	28	10	3	4	1	233

TABLE 3. *Distribution of Patients by Extent of Total Body Burn and Per Cent Mortality*

% Body Burn	0–10	10–20	20–30	30–40	40–50	50–60	60–70	70–80	80–90+	Total
No. burned	150	146	126	103	80	71	46	49	46	817
Deaths	1	5	7	26	40	39	37	43	45	243
% Mortality	0.7	3	6	25	50	55	80	88	98	29.7
Autopsies	1	5	7	23	40	39	35	39	44	233

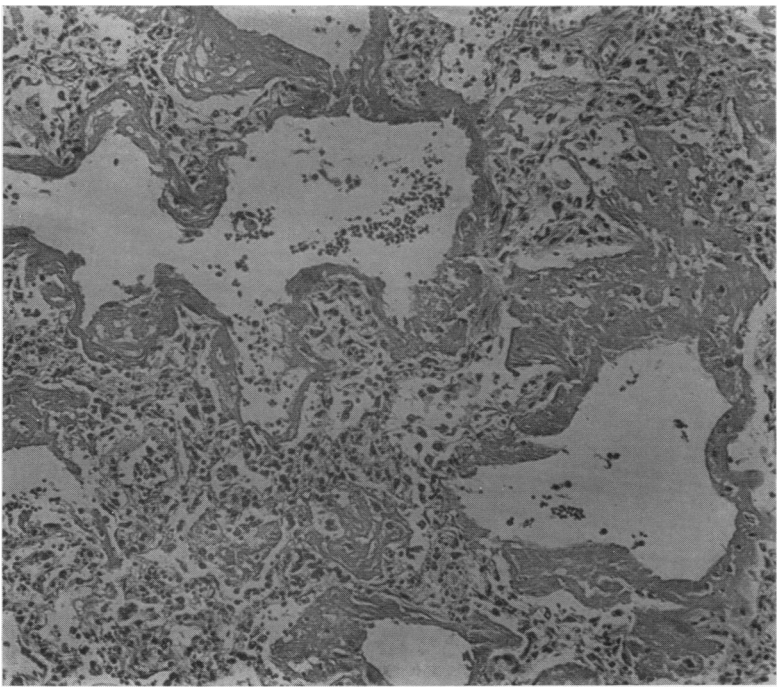

FIG. 2. Photomicrograph demonstrating hyaline membrane lining alveolar ducts and alveoli with interstitial edema and inflammation (H&E, ×100).

fistula (1), and retroperitoneal hemorrhage (1).

In the category, "Cause of Death Uncertain" are many patients who died, often within the first week postburn, with pulmonary edema and congestion as the sole finding at autopsy, in addition to the burn. There are also a few patients in this group whose death was a puzzle to the clinician and pathologist although there may have been no lack of autopsy findings.

Pneumonia. Table 5 lists the pathologic findings in the lungs of the 233 patients at autopsy and indicates those considered a cause of death. We have used the term "airborne pneumonia" in preference to bronchopneumonia or lobar pneumonia so as not to confuse this complication with the pneumonic lesions that we believe to be hematogenous in origin. This distinction is important because no deaths were attributed to hematogenous pneumonia, but rather to the original infective complication that resulted in dissemination to the lung, e.g., burn wound sepsis or suppurative phlebitis. In contrast, airborne pneumonia, which was found in 69 burned patients at autopsy, was considered a major cause of death in 37 patients. This type of penumonia was listed as a cause of death, in addition to the burn, when progressive lower respiratory infection was dominant in the clinical course and extensive inflammatory consolidation of pulmonary parenchyma was found at autopsy. Although noted annually as the leading cause of respiratory deaths, airborne pneumonia has increased in frequency and has replaced burn wound sepsis as the major cause of death in 1964–5 (Fig. 1). The increased incidence of pneumonia is of more significance in reference to deaths ($p < 0.01$) than total admissions ($0.05 < p < 0.1$) when both periods are compared. Pneumonia has recently been more of a problem in our adult population and this is substantiated

TABLE 4. *Cause of Death in 233 Fatally Burned Patients*

	1960	1961	1962	1963	1964	1965
Septicemia, origin, burn wound infection	18	15	31	34	3	4
Septicemia, origin, respiratory infection	(3)	(5)	(3)*	(1)*	(2)	(6)
Septicemia, origin, phlebitis		1		2	1	2
Septicemia, origin, miscellaneous		1	1			
Septicemia, origin, uncertain	2	3	4	2		1
Respiratory disease	5	5	3	6	12	13
Gastrointestinal ulceration			3	2	2	1
Renal failure			2	2	2	2
Miscellaneous	1	1	2	8	9	4
Cause of death uncertain	4	4	9	7	7	11
Deaths	30	31	54	57	37	34
Autopsies	30	30	51	56	34	32
Patients treated	96	88	142	148	184	159
% Mortality	31.3	35.2	38.0	38.5	20.2	21.4

( ) Subtotal, already listed under Respiratory Disease.
* Also had other possible causes for sepsis.
Note: There are more causes of death listed than patients who were autopsied since two causes of death were occasionally considered of equal importance.

if one compares the incidence of fatal pneumonia in hospitalized patients over 15 years of age during each period. Twelve of 318 adult patients died of pneumonia during 1960–3 vs. 19 of 237 patients during 1964–5 ($p < 0.05$).

The pathologic aspects of airborne lower respiratory infection were demonstrated by the usual suppurative response in a bronchopneumonic, lobular or lobar distribution with varying degrees of organization, in a few cases, that were consistent with the duration of this complication. Table 6 lists all the organisms cultured from sputum or tracheal secretions and postmortem lung cultures in the 37 patients dying of pneumonia. Mixed gram-negative infections were commonly encountered throughout the entire period. Both antemortem and postmortem cultures frequently yielded coliform organisms particularly during 1964–5. It was not uncommon however to recover coagulase-positive *Staphylococci* from initial sputum cultures which subsequently yielded gram-negative organisms following potent antistaphylococcal chemotherapy.

Certain unique pathologic features became prominent in the lungs of patients dying from airborne pneumonia in 1964–5. Pulmonary hyaline membranes have previously been noted in fatally burned patients.[13] Hyaline membranes were found in 4 of 16 patients who died from pneumonia in 1960–3. In the 1964–5 group of 21 patients whose cause of death was pneumonia, 14 had hyaline membrane formation ($p < 0.05$). There were additional histologic features in the areas of pneumonia and hyaline membrane formation that were not thought to represent the customary pulmonary response to bacterial infection alone. These included hypervascularity and thickening of the alveolocapillary membrane due to interstitial edema and inflammation (Fig. 2). Also, there were numerous alveolar macrophages and septal epithelial cells lining the alveoli.

The pulmonary lesion secondary to hematogenous dissemination was common during 1960–3 when burn wound sepsis was prevalent, but was not considered an intrinsic respiratory complication. This lesion is believed to evolve from dissemination

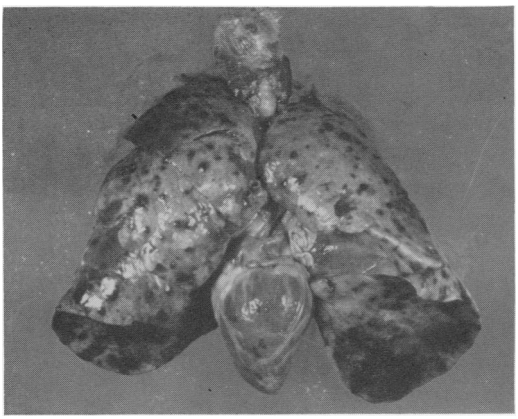

FIG. 3. The hemorrhagic areas are infected pneumonic lesions of hematogenous origin.
(Reprinted from J. Trauma., 4:233–245, 1964.)

of bacteria from the infected burn wound to the lung. The pathologic features of hematogenous pneumonia are described in the discussion. This and other embolic visceral lesions originating from infection of the burn wound have been noted previously.[12] Postmortem bacteriological cultures of lungs with hematogenous pneumonia yielded *Pseudomonas sp.* and *Staphylococcus aureus* as the most frequent pathogens, but other gram-negative organisms were frequently present.

Tracheobronchitis. The most frequent intrinsic pulmonary complication following thermal injury is tracheobronchitis, which was manifest at autopsy in 109 patients—almost half of all autopsies. During the 6-year period, erosive tracheobronchitis due to abutment of the tracheostomy tube on the anterior tracheal wall has been a direct cause of death in 6 patients (Table 5). In two, the erosion was deeply invaded with bacteria, with peritracheitis and positive antemortem blood cultures, in the absence of infection elsewhere in the lung or burn wound. Another patient developed mediastinitis via bacterial invasion of the eroded trachea. Diffuse invasive bacterial tracheobronchitis occurred in one patient and was thought to have originated in the tracheal ulcer. Two patients died with hemorrhage from the anterior tracheal erosion. The lesions encountered in the tracheobronchial tree and their relation to tracheostomy and suspected inhalational injury are presented in the discussion.

Tracheostomy may have been indirectly responsible for other deaths although not listed as such in Table 5, *e.g.*, patients who die of pneumonia subsequent to temporary obstruction of a main stem bronchus by the tracheostomy tube early in the course of treatment. Also, in patients who develop pneumothorax as a tracheostomy complication, death may be attributed to the pneumothorax, as in one of our patients, or to the subsequent development of pneumonia. In addition, bacterial infection of tracheal erosions may represent a nidus for airborne dissemination of lower respiratory infection. In any case, the hazards of tracheostomy in the burned patient are underestimated by the mortality table.

Laryngitis. Laryngeal ulcerations were found in 27 autopsies. This is an underestimate of incidence since the larynx was not described in adequate detail in some autopsies. Of the 27 examples that were available for study, 21 involved the vocal or ventricular folds. The remainder of the erosive lesions occurred just above or below the folds or over the corniculate process or epiglottis. Bacterial infection of these lesions was demonstrated in 20 of the 27 autopsies.

Thromboemboli. Thrombi, without bacteria, were found in the lungs of 59 burned patients at autopsy and were assumed to be embolic in nature. These were usually of microscopic proportions, involved pulmonary arterioles and small musculo-elastic pulmonary arteries 100–200 micra in diameter and were few in number in relation to the total cross-sectional area of the pulmonary vasculature. Pulmonary infarcts

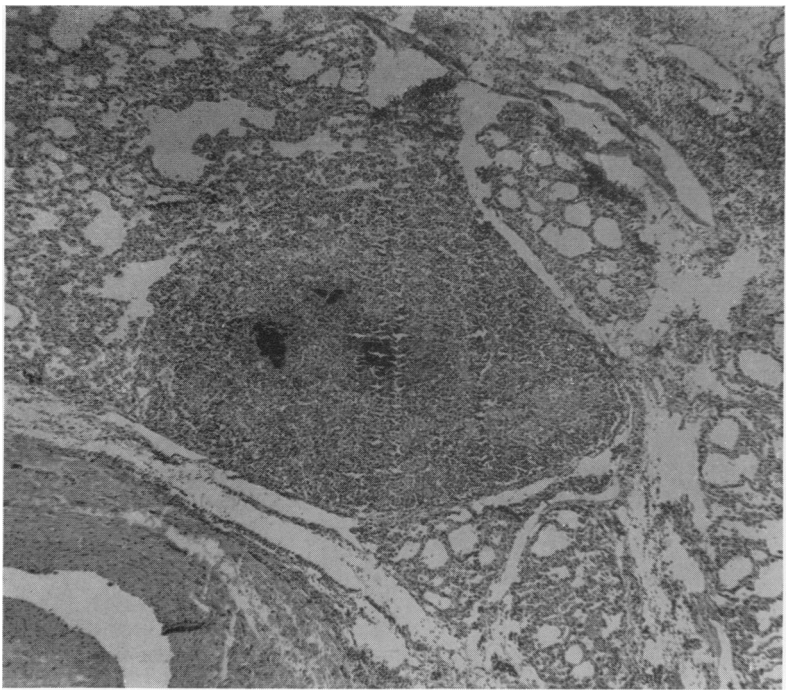

FIG. 4. Photomicrograph demonstrating hematogenous (Stapyhlococcal) pneumonia. The central dark spots are masses of bacteria (H&E, ×37).

were occasionally encountered, as indicated in Table 5, and these were associated with thromboemboli that were observed grossly. In 1960, a 7-year-old boy died following hemoptysis of an estimated 950 ml., eight days after sustaining 35 per cent total body burns. A large right lower lobe pulmonary infarct and pulmonary thromboemboli, both recent and organizing, were found at autopsy. In 1964, a 26-year-old man died 14 days after sustaining 65 per cent total body burns. Pulmonary infarcts were found at autopsy with recent thromboemboli in each main pulmonary artery. These two fatal complications were not classified in the mortality table as deaths due to respiratory disease.

Pneumothorax. Thirteen patients had pneumothorax sometime during their course. However this is another complication that is underestimated in a study limited to fatally burned patients. The pathogenesis of pneumothorax in burned patients almost invariably evolves from tracheostomy complications rather than from trauma to the chest at the time of thermal injury. The single patient who died *directly* after pneumothorax was a 30-month-old infant who sustained 80 per cent total body burns 2 days previously. Tracheostomy had been placed between the 7th and 8th tracheal rings prior to evacuation to this unit and he subsequently developed mediastinal emphysema and bilateral pneumothorax.

Chronic Pulmonary Disease. Pre-existent pulmonary disease has been a significant factor in mortality in only two patients over the 6-year period. One was a 61-year-old man who had previously undergone pneumonectomy for bronchogenic carcinoma and at autopsy his opposite lung was emphysematous with acute tracheobronchitis. The second was a 75-year-old man who had acute bronchopneumonia superimposed on severe chronic bronchitis and emphysema.

Miscellaneous. Pulmonary edema and congestion is the most frequent autopsy finding in the lungs of burned patients, but

TABLE 5. *All Fatal and Non-Fatal Pulmonary Complications in 233 Burn Autopsies*

	1960		1961		1962		1963		1964		1965	
	Total	COD*	Total	COD	Total	COD	Total	COD	Total	COD	Total	COD
Pneumonia, airborne	7	4	8	4	11	2	13	6	15	9	15	12
with hyaline membranes		(1)		(1)		(0)		(2)		(7)		(7)
Pneumonia, hematogenous	5		11		25		11		1		1	
Tracheobronchitis, erosive	17	1	20	1	18	1	24		13	2	17	1
Laryngeal ulceration	8		4		5		7		1		2	
Pulmonary thromboemboli	13		9		8		16		5	1	8	
with infarction	(6)	1	(2)		(2)		(1)		(3)		(1)	
Pneumothorax tracheostomy	2		3		2		2		2	1	2	
complication	(1)		(3)		(2)		(1)		(2)		(1)	
Preexistent pulmonary disease	2		3				2		6	1	3	1
Miscellaneous:												
Pulmonary edema and congestion	15		20		32		42		26		22	
Atelectasis					5		1		2			
Pulmonary megakaryocytes	3		9		11		13		2		11	

* COD—Cause of Death ( )—Subtotal

is considered an unrevealing pathologic diagnosis. Although it is frequently the only finding in patients with extensive burns who die in the early postburn period, no effort was made to correlate this finding with examples of overhydration, renal failure, cardiac arrest with and without arrhythmias, or other clinical events that might result in pulmonary edema and congestion since retrospective clinical material is incomplete and arbitrary in this regard, and also because pulmonary edema and congestion may develop rapidly as an agonal phenomenon. Atelectasis is another autopsy diagnosis of uncertain significance, but in no instance was it thought to be an important or contributory cause of death.

Pulmonary megakaryocytes were frequently found in increased numbers in burned patients' lungs. This probably reflects a general posttraumatic response in which megakaryocytic production or mobilization from bone marrow is increased and entrapment in the pulmonary capillaries occurs.

Other features could not be evaluated or were thought not to be of any unique interest, e.g., pulmonary septal macrophages are prominent in most lungs of burned patients, however, this response is probably too nonspecific to warrant meaningful interpretation. The frequency of pulmonary fat emboli could not be properly evaluated since the tissue available was paraffin-embedded and therefore had been processed through fat solvents. Insignificant foci of bronchiolitis, pleuritis overlying areas of pneumonia and small inactive pulmonary granulomas were considered irrelevant. Pleural effusion and small foci of intra-alveolar hemorrhage were invariably associated with pulmonary edema and congestion and were not individually analyzed.

Discussion

There is no readily apparent reason for the recent increase in deaths due to pneu-

monia in our adult population. Although the incidence of burn wound sepsis declined in the period 1964–1965, the increase in pneumonia as a cause of death does not appear simply for lack of a better cause since the clinical and pathologic interpretation of death *from* pneumonia was consistent during both periods. Also many patients died *with* small foci of pneumonia that were considered insignificant in 1964–5 as they were in 1960–3. The average extent of total body burn has also remained fairly constant throughout the entire 6-year period. Furthermore, fatally burned patients are not surviving longer in the absence of burn wound sepsis to subsequently develop pneumonia since the distribution of patients as to the postburn day on which death occurred reveals no difference between patients who died of burn wound sepsis and those who died of pneumonia, during either period. Although tracheobronchitis in our patients has been associated with tracheostomy, as discussed below, the increase in fatal pneumonia is not similarly related. Tracheostomy was performed in 30 per cent of 474 burned patients treated from 1960–3 and in 21 per cent of 343 patients treated in 1964–5. The number of tracheostomies performed on fatally burned patients during the above periods was 56 per cent (96 of 172) and 61 per cent (43 of 71), respectively. Also, there has been no increase in inhalational injury, either suspected clinically or documented at autopsy, that might account for the rise in deaths due to pneumonia.

Hyaline membrane formation and hyperplasia of alveolar lining cells with congestion and interstitial edema of the alveolocapillary membrane represents a constellation of unique histologic features that we have seen previously in the lungs of burned and nonburned patients who received prolonged positive pressure ventilatory assistance with oxygen. Since these pathologic

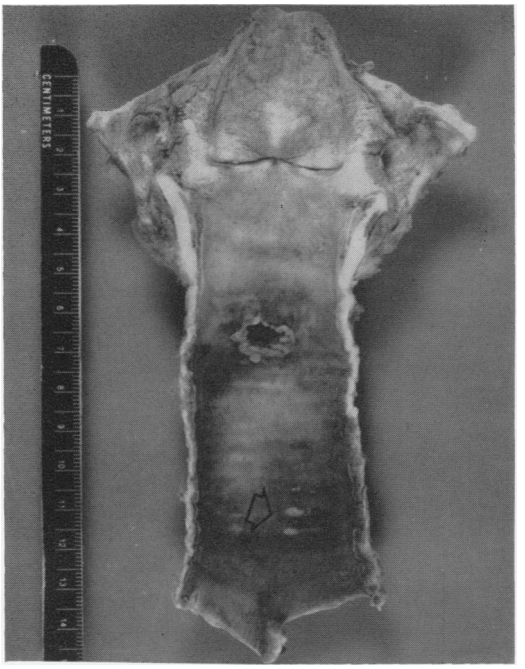

FIG. 5. Note the erosion below the level of the tracheostomy (arrow) and relatively normal larynx. A skip area below the tracheostomy stoma is characteristic.

findings were significantly more frequent in lungs of patients dying from pneumonia during 1964–5 (Table 5) and were concurrent with an overall increase in deaths due to pneumonia during this period as compared with the 1960–3 period, we examined the utilization of ventilatory assistance and oxygen in patients who died from pneumonia both with and without these unique pathologic features. Thirty-four records of 37 patients dying from pneumonia were available to study. Eleven of 17 patients with, and 10 of 17 patients without hyaline membrane formation and the associated histologic alterations described above, received positive pressure ventilatory assistance with an oxygen source. Hyaline membranes have also been seen in association with oxygen therapy, however, all patients dying from pneumonia in our group, both with and without hyaline membranes, re-

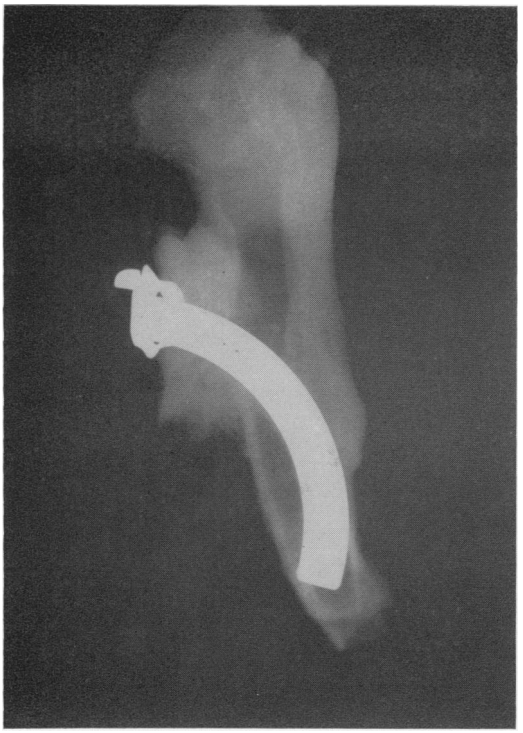

FIG. 6. Postmortem roentgenogram demonstrating the mechanism of trauma from abutment of the tracheostomy tube against the anterior tracheal wall.

ceived oxygen at some time in their terminal illnesses. There was no difference therefore in the use of ventilatory assistance or oxygen *per se,* in patients with or without hyaline membrane formation. These histologic findings have been attributed by others to oxygen and positive pressure ventilatory assistance, particularly when this type of inhalational therapy is given for prolonged periods, viz., over 10 days.[1, 8, 9] We were unable to analyze the association of hyaline membrane formation with duration of ventilatory assistance and level of oxygen tensions achieved due to limitations in the retrospective study. Increased incidence of hyaline membrane formation and the associated histologic changes, during 1964–5, may be the result of earlier and more prolonged use of oxygen and positive pressure ventilatory assistance in burned patients who display respiratory symptoms. There is no question that this histologic response has not only become more frequent, but has increased in extent in the lungs of patients dying from pneumonia in the last two years of the study. Furthermore, this unique histologic response also occurred in areas of gross pulmonary consolidation that were free of suppuration and bacteria and therefore presumably free of bacterial infection. The increased extent of pulmonary consolidation and consequently the increased frequency of deaths attributed to pneumonia during 1964–5, therefore, may be partly a result of respirator or oxygen toxicity.

During 1964–5, due to control of septicemia originating from infection of the burn wound, hematogenous pneumonia virtually disappeared from autopsy subjects, except for isolated examples, some of which originated from an infected cannulized vein. The pathologic features of this type of pneumonia are distinct and readily recognized. The lesions are commonly small, subpleural, hemorrhagic and discrete (Fig. 3), or may be confluent. The larger single lesions characteristically resemble pulmonary infarcts. Focal necrosis of alveoli is presently histologically and in hematogenous *Pseudomonas* lesions there are myriads of gram-negative bacteria proliferating in the alveolocapillary membrane and particularly in the perivascular and peribronchial spaces. Although the peribronchiolar sheath may be filled with gram-negative bacteria, the early lesion is relatively free of exudate within the bronchiolar lumen, thus distinguishing it from bronchopneumonia. Staphylococcal emboli, however, did not show this perivascular distribution even though nests of cocci were found within vessels (Fig. 4). The hematogenous derivation of this type of pneumonia is also suggested by the fre-

TABLE 6. *Bacteria Cultured From Sputum, Tracheal Secretions and Postmortem Lung Cultures in 37 Burned Patients Dying from Bronchopneumonia*

	1960–1963		1964–1965	
	Ante-mortem	Post-mortem	Ante-mortem	Post-mortem
Aerobacter—E. coli	5	6	10	17
Pseudomonas sp.	6	3	6	12
Staphylococcus aureus	4	6	2	5
Proteus sp.	1	3	3	9
Miscellaneous	5	6	3	11
TOTAL	21	24	24	54

quent presence of fibrin and neutrophil thrombi containing bacteria, in the small pulmonary arteries and arterioles within the lesions.

Although tracheobronchitis is the most frequent finding in the lungs of fatally burned patients, the prominent lesions encountered in the present study were clearly related to tracheostomy in most instances, rather than to inhalation injury. Ninety-nine of the 109 patients with tracheobronchitis had tracheostomies performed. The lesions that are convincingly tracheostomy complications include erosions or ulcers due to abutment of various parts of the tracheostomy tube or its inflatable cuff on the tracheal mucosa (Figs. 5, 6). Erosions in the tracheobronchial mucosa beyond the range of the tracheostomy tube occurred less frequently, but were also focal and thought to be traumatic due to frequent suctioning. These erosions may become infected, as noted previously, with the development of bacterial tracheobronchitis.

Although similar lesions were occasionally found at the margin of the vocal folds (Fig. 7), laryngeal and tracheal mucosa above the tracheostomy stoma was grossly normal in the majority of patients at autopsy who had tracheitis. The normality of the laryngeal mucosa, when focal erosions were present below the tracheostomy stoma, is regarded as further evidence that tracheobronchitis is not due solely to inhalation injury, the effect of which might be expected to be diffuse or at least manifest proximal as well as distal to the tracheostomy stoma. Nevertheless, clinical evidence for thermal or chemical laryngotracheitis due to inhalation injury exists. We frequently see severely burned patients with marked dyspnea and bronchospasm postinjury and with carbonaceous sputum, whose laryngeal and tracheal mucosa is inflamed and edematous at bronchoscopy, prior to the performance of a tracheostomy. A tracheostomy is invariably performed, however, and if the injury is fatal, the lesions commonly seen at autopsy are those related to the tracheostomy.

The interpretation of the discrepancy between clinical evidence for inhalational injury and the lack of autopsy documentation, is twofold. Carbon deposition and thermal necrosis of laryngeal and upper tracheal epithelium is frequent in patients who die in fires and this evidence of inhalational injury is commonly recorded by forensic pathologists. The experience of pathologists who autopsy patients who survive cutaneous thermal injury beyond the burn day, however, is in contrast to pathologists who examine those who died in fires. We have demonstrated soot deposit and thermal necrosis of laryngotracheal mucosa in a few burned patients who expired before

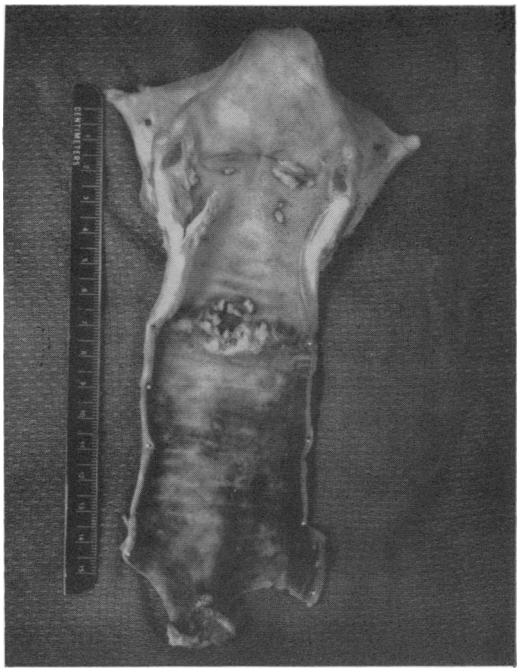

FIG. 7. Laryngeal erosions are present on the vocal and ventricular folds. Tracheitis is prominent below the level of the tracheostomy.

the first or second postburn day (Fig. 8), but these features are usually absent by the time postmortem examinations are performed. Clinically, we also noted diminishing expectoration of soot beyond the second postburn day and therefore the lack of pathological documentation of inhalational injury is not unexpected since 94 per cent of the autopsies were performed beyond the second postburn day. Secondly, the residual of inhalation injury that is encountered above the tracheostomy site has been minimal in comparison with the extensive, ulcerative tracheitis due to the tracheostomy tube in the lower trachea. Since the carbon deposit and necrotic respiratory epithelium has been expectorated, the residual inflammatory infiltrate in the lamina propria of the respiratory mucosa, with regeneration of respiratory epithelium, above the level of the tracheostomy, is overshadowed by the impressive gross and histologic alterations inferior to the tracheostomy.

We think that a combination of factors is responsible for the focal ulcerative laryngeal and tracheal lesions found in fatally burned patients. The lesions mechanically related to trauma from the tracheostomy tube seem to occur more readily following suspected inhalation injury. Similar erosions limited to the vocal folds with a normal surrounding laryngeal mucosa may also represent the combined effects of laryngitis due to inhalation injury in combination with the trauma of endotracheal intubation, e.g., during anesthesia, or simply from voice trauma. Our findings are consistent with Moritz's experimental studies on the occurrence and location of inhalational thermal injury [7] and also suggest that inhalational laryngeal or tracheal injury undergoes rapid repair in the absence of superimposed trauma. The focal ulcerative lesions that occur so frequently in the tracheobronchial tree emphasize the hazards of tracheostomy in the burned patient.

The association of facial burns with respiratory complications implies that inhalation injury may occur at the time of burning and result in tracheobronchitis and pneumonia. For purposes of discussion patients with facial burns include those with over 1 per cent of total body surface, partial or full thickness, burn that involves the anterior face between the eyes and chin and between each malar eminence, i.e., around the nose or mouth. Thirty-eight of 498 patients with facial burns and 5 of 319 patients without facial burns died from tracheobronchitis or pneumonia ($p < 0.01$). Although these data support the premise that fatal respiratory complications occur more frequently with facial burns, we think it is equally important that the majority of fatally burned patients with facial burns died from nonrespiratory complications over the 6-year period. Of 187 patients, at autopsy, who had facial burns, 149 died of complications other than tracheobronchitis or pneumonia (80 per cent).

If thermal injury around the nose or mouth implies that inhalation injury is responsible for subsequent respiratory complications, then one might expect to encounter respiratory complications in patients who suffer facial burns without extensive thermal injury elsewhere. There were 59 patients treated over this 6-year period who sustained facial burns, as defined above, in association with less than 15 per cent total body surface thermal injury. Not one suffered either a fatal or nonfatal respiratory complication. This supports the premise that facial burns alone do not constitute a hazard to the burned patient. Also, there was no difference in mortality between the 498 patients with, and 319 patients without facial burns, using probit analysis ($LD_{50} \cong 50$ per cent total body surface burn).

All deaths attributed to tracheobronchitis or pneumonia, in association with facial burns, occurred in patients with over 39 per cent total body burns. There were 299 patients with 39 per cent, or more, total body surface burns and 241 had facial burns (81 per cent). The increased frequency of respiratory complications in patients with facial burns, therefore, may indicate that severely burned patients are more prone to develop tracheobronchitis and pneumonia for reasons other than inhalation injury since extensive thermal injury occurs more often with, than without, facial involvement.

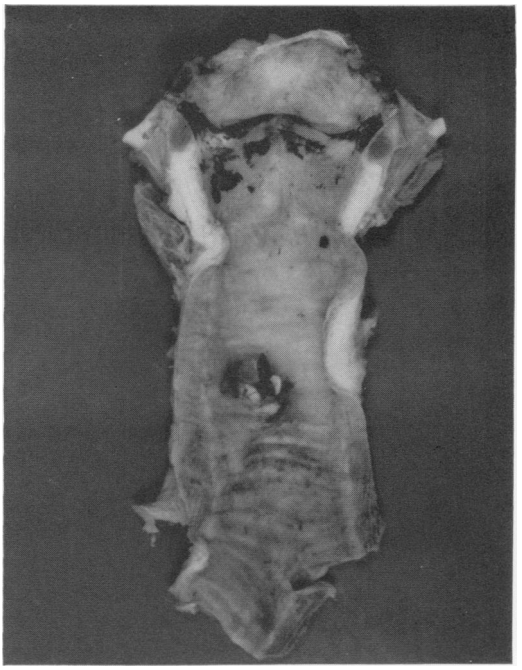

FIG. 8. Inhalational injury with deposition of soot in the larynx.

Summary and Conclusions

A clinicopathological examination of 233 autopsies was performed to analyze the frequency of fatal complications following cutaneous thermal injury with particular reference to pulmonary complications.

Over a 6-year period, the major fatal complication in burned patients treated at the Surgical Research Unit has shifted from invasive burn wound infection to pneumonia. The decline in burn wound infection has been due to effective topical chemotherapy. The recent increase in deaths attributed to pneumonia is considered real in patients over 15 years of age and results from the greater frequency of infective lower respiratory disease found clinically. A part of the increase in deaths attributed to pneumonia may be due to effects of earlier and more prolonged ventilatory assistance and oxygen therapy.

Although inhalational injury occurs, our experience indicates that the majority of laryngeal and tracheal lesions found at autopsy are due to repeated trauma from tracheostomies or endotracheal tubes.

Respiratory complications occur more frequently in patients with facial burns, but the presence of facial burns alone does not constitute a particular hazard to the burned patient. The increased incidence of respiratory complications in patients with

facial burns, in our material, may be due to the increased occurrence of facial burns in severely burned patients who are more susceptible to infective lower respiratory disease. Respiratory complications in less severely burned patients with facial burns have not occurred.

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