Pulmonary Complications of Burns:

The Major Threat to the Burn Patient

BRUCE M. ACHAUER, M.D., PATRICIA A. ALLYN, R.N.,
DAVID W. FURNAS, M.D., ROBERT H. BARTLETT, M.D.

From the Department of Surgery, University of California, Irvine, College of Medicine and the Burn Unit of the Orange County Medical Center, Orange, California

THE ASSOCIATION between thermal burns and respiratory insufficiency has long been realized. Long[14] described hepatization and engorgement of the lung with pleural effusion in a number of fatal burn injuries in 1840. In more recent times various authors have described the increasing danger of pulmonary complications in the mortality statistics of burn patients. Phillips[23] stated that since 1939, pulmonary complications accounted for 42% of fatalities. Sochor[30] reported 41 autopsy cases in which 28 patients died from pulmonary lesions. Harrison[12] reported that bronchopneumonia was the leading cause of death in their unit. Silverstein[28] reported that between 1956 and 1964, six patients died of pulmonary complications and 12 from burn wound sepsis at Boston City Hospital, while from 1965 to 1968 there were 20 deaths from pulmonary complications and only four deaths from burn wound sepsis. Foley[10] reported that the major cause of death at Brooke Army Hospital from 1960 to 1963 was burn wound sepsis. After the use of Mafenide Acetate in 1964, the major fatal complications shifted to the respiratory system. In later articles from the same institution however, Pruitt[24] states that no actual increase in pulmonary complications in the entire population at risk has occurred.

There are several different mechanisms involved in the problem of pulmonary burn complications. These can best be considered in regard to time of onset of pulmonary findings from the time of burn injury. The acute respiratory distress seen within minutes to hours from the time of thermal injury is due to smoke inhalation, carbon monoxide poisoning, or problems secondary to airway obstruction. Following the Cocoanut Grove night club fire in Boston in 1942, there were 114 casualties taken to Massachusetts General Hospital and 75 were either dead on arrival or died within minutes.[9] This group of patients with acute onset of symptoms represents an important percentage of people who died after thermal injury but a small number treated in burn centers. Airway obstruction from oral or facial edema, or bronchospasm and alveolar damage from inhalation of toxic vapors or particles comprise most of the symptoms found in the patients in this group.

A second group of patients are asymptomatic for 24 to 48 hours, but develop tachypnea, cyanosis, and hypoxemia 1 to 5 days after being burned. Hypocarbia and respiratory alkalosis with decreased compliance and increased airway resistance follow. Patchy bilateral fluffy infiltrates are seen on X-ray. The course is usually progressive and may require mechanical ventilation and supplemental oxygen. Eventually, respiratory acidosis, high pulmonary vascular resistance, myocardial depression, and bronchopneumonia usually prove fatal.

A third group of patients are those developing late complications such as pneumonia and pulmonary embolus.

The purpose of this study is to review the incidence and course of pulmonary complications in our burn unit with the hope of identifying areas in which treatment and prevention may be improved.

Submitted for publication May 17, 1972.

Reprint requests to: Robert H. Bartlett, M.D., University of California, Irvine, Irvine, California 92664.

Materials and Methods

From August 1970 to July 1971, 100 patients were admitted to the Orange County Medical Center Burn Unit. During this period burn patients were managed similarly with respect to pulmonary problems, fluid replacement (essentially Brooke formula), and surface management (Mafenide Acetate with and without porcine xenografts).[11] In the emergency room, carboxyhemoglobin and arterial blood gas determinations were done routinely and a central venous catheter placed for fluid monitoring. After initial debridement, and while Mafenide Acetate and xenograft dressings were being applied, patients with major burns and those considered likely to develop pulmonary problems had a radial artery catheter placed for multiple arterial blood sampling and continuous blood pressure monitoring.[5] Escharotomies in circumferential chest burns were performed at this time. Nurses trained in respiratory management monitored tidal volume and minute volume, did endotracheal suction with sterile technic as necessary, provided humidification, and encouraged deep breathing and coughing. Daily chest X-rays, sputum culture and frequent physical examination for upper airway edema and bronchospasm were done routinely. In some cases a Swan-Ganz[32] catheter was employed to monitor pulmonary artery pressure. Aminophylline, (500 mg. intravenously), isoproterenol, (1:200, 2cc, via nebulizer), and dexamethasone (.3 mg./kg./6 hours) were given if bronchospasm occurred. Endotracheal intubation was performed for upper airway obstruction, or for use of mechanical ventilation. Mechanical ventilation controlled by volume was used whenever indicated (outlined later).

The 100 patients were reviewed for location of burn and physical findings (nasal or facial hair burned, soot in mouth or sputum, rales, wheezes) radiographic findings and laboratory studies including carboxyhemoglobin levels and blood gases. Patients exhibiting any objective evidence of pulmonary pathology were then reviewed in detail.

TABLE 1. *Age Census—Extent of Burns in 22 Patients.*

	Per Cent Burn				
	0–19	20–39	40–59	60–79	80+
0–2					B
2–14			B	A	
15–29		Ⓐ	B	C	ABBBB
30–49	Ⓐ	B		B	A-B
50–59		Ⓑ	B		
60–69					
70+	A-B		B		A

A = Early onset group
B = Delayed onset group
C = Late onset group
Survivors are circled

Results

Of the 100 patients 22 had clinically significant pulmonary complications (Table 1). Of these 22 patients, 19 died (86%). These were the only deaths in the 100 patients. On admission, 47 patients had facial burns and 37 had singed nasal vibrissae. Thus, only half of the patients sustaining facial or nasal hair burns developed pulmonary problems. More important than facial burns in predicting pulmonary problems was the extent of the burn and the history. The best correlation was with survival probability.[15] Those patients with very bad prognoses based on age and per cent of burn almost always developed pulmonary problems. Of 22 patients with pulmonary complications, 21 were in an enclosed space at the time of injury. Eighteen were in fires involving clothing or gasoline or both. There were two instances of scalding in a 9-month-old baby (95%) and a 95-year-old patient (10%). Two patients were involved in an industrial explosion involving "black powder." One survivor and three who did not survive were involved in the same airplane crash and all had prolonged smoke inhalation and severe respiratory problems.

Using McCoy, Micks and Lynch's[12] data on probability of survival based on age and per cent of burn only, 11 had predicted survival rates of less than 10%, six were between 10 and 40 per cent and four had a greater than 40% survival probability. Of these four, three survived and one died. Seven patients had positive blood cultures. One, a *Pseudomonas aeruginosa* septicemia, was considered to be the direct cause of death. The remaining blood cultures were *Candida albicans*, (five patients, all died), and *Escherichia coli* (one patient who survived). Cultures of tracheostomy sites and endotracheal tubes yielded *Pseudomonas aeruginosa* in three patients, *Staphylococcus aureus* in two, and yeast, *E. coli* and *Serratia marcescens* in one each. Cultures of burn wounds yielded *Staphylococcus aureus* in nine patients, *Pseudomonas aeruginosa* in eight patients, *Klebsiella-enterobacter* in seven, yeast in three and *E. coli* in one. Two of the patients were considered to have significant burn wound sepsis, one of whom died from septicemia.

Carboxyhemoglobin determinations were not particularly useful in predicting pulmonary problems. They were done as a routine on all patients in this series and all but two were below 15% carboxyhemoglobin. The patient with the highest carboxyhemoglobin (27%) was involved in an automobile accident and later developed pulmonary problems. These problems cleared, however, and the patient later developed sepsis and died (Case 4).

All but two of the fatalities and two of three survivors eventually had tracheostomies. Seven of these 19 patients (37%) had endotracheal tubes placed first. All endotracheal tubes were placed within 48 hours of admission

although some tracheostomies were performed as late as 5 days postburn.

Ten patients had arterial Po_2 values below 70 mm. Hg and many of these were on supplemental oxygen.

There was no correlation between the number of blood transfusions and severity of pulmonary problems. Ten of the 22 patients received transfusions, three of whom received more than 5000 cc. during hospitalization.

The development of radiologic abnormalities usually lagged behind clinical and laboratory evidence of pulmonary problems. Ten patients developed "positive" chest roentgenograms during the first 48 hours and another seven from 2 to 10 days.

Autopsies were performed on 16 of the 19 patients who died. Average lung weight was greater than 1,000 Gm. in six patients, 400 to 1,000 in five patients and less than 400 Gm. in three patients. In all of the autopsies the lungs showed the most abnormality of any visceral organ. Either pulmonary edema, often pink and frothy, or bronchopneumonia, usually purulent and confluent, or both, were found. One of the victims of the airplane crash had no mucosa remaining on the trachea or bronchi, only large amounts of necrotic debris. While in the burn unit, black bronchial casts were obtained during endotracheal suctioning.

There were seven patients (32%) who could be categorized as having early pulmonary complications. The two who survived had upper airway burns with edema and immediate distress. Endotracheal intubation or tracheostomy was done immediately on six of the seven patients (the seventh died 3 hours after admission and had greater than 95% burns, many had 3rd and 4th degree burns).

Case Reports

Case 1: A. C. (OCMC 140082) A 27-year-old man was found unconscious in his home in a smoke-filled room by the fire department. He had sustained 20% second and third degree burns involving the extremities, abdomen and face. The nares were burned. The tongue was swollen and coated with soot. Rhonchi were noted in both bases.

The admitting chest X-ray revealed some evidence of pulmonary edema compatable with smoke inhalation (Fig. 1A). An endotracheal tube was inserted in the emergency room and a tracheostomy performed in the burn unit. Blood gases were monitored via a radial artery catheter. Carboxyhemoglobin several hours after admission was 1%. The patient was given dexamethasone, aminophylline, penicillin, and kanamycin.

After the tracheostomy was performed copious amounts of soot, epithelial cell casts and pink tinged fluid were removed by suction and expectorated. A pneumothorax occurred 18 hours after admission and a tube thoracostomy was performed, with prompt expansion of the lung. The patient was ventilated with a mechanical ventilator for 8 days. He was confused and uncooperative for several days despite adequate oxygenation. Burn wound, sputum, and blood cultures grew *E. coli* and the patient was started on cephalothin and gentamicin 5 days after the burn.

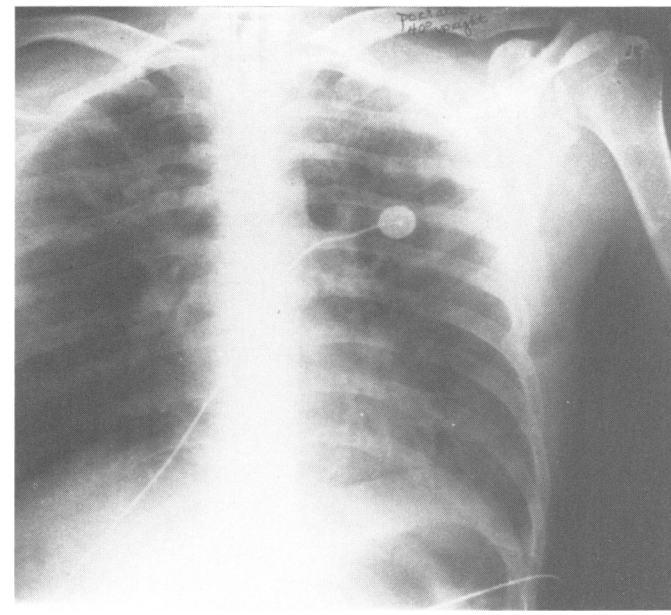

FIG. 1A. Case 1: (A.C.) on admission.

Apparent second degree burn on the legs became full thickness. Because of persistent ileus, progressive sepsis, and weakness to the point of inadequate spontaneous ventilation, intravenous hyperalimentation was begun. After 3 days weight loss and nitrogen balance were reversed, the ventilator was discontinued and the granulation bed could be grafted.

The initial X-ray abnormalities resolved to a pulmonary infiltrate in the right lung on the 14th post-burn day (Fig. 1B). This increased over the next several days and fluffy infiltrates involving both lung fields, greater on the right than on the left, were noted. The infiltrates slowly resolved over the next 4 weeks (Fig. 1C). The burns on the legs required split thickness skin grafts (35 days post-burn) and the patient was discharged 12 days after grafting.

Case 2: K.D. (OCMC 264860) A 31-year-old man was involved in a crash of a cargo aircraft. He sustained second degree face and neck burns totaling 10% of his body surface area

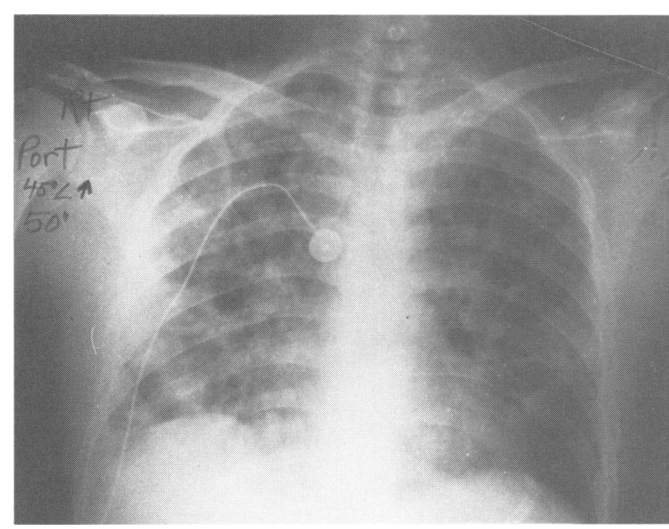

FIG. 1B. Case 1: 14 days post burn.

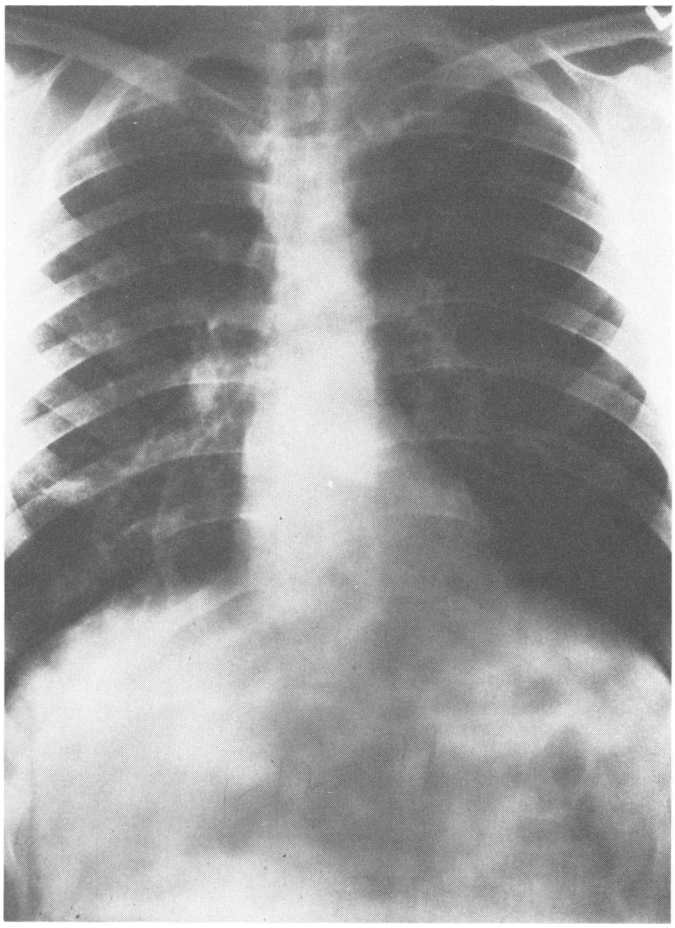

FIG. 1C. Case 1: Six weeks post burn.

and severe smoke inhalation. His entire face, ears, buccal mucosa, tongue and posterior pharynx were burned (Fig. 2). Laryngeal stridor was noted on admission and direct laryngoscopy in the emergency room revealed edema of the cords. Tracheostomy was performed over the endotracheal tube which had been inserted in the emergency room and the patient was then placed on a mechanical ventilator. A radial artery catheter was inserted, and dexamethasone (20 mg. IV then 4 mg. q 4h s 3d) was given.

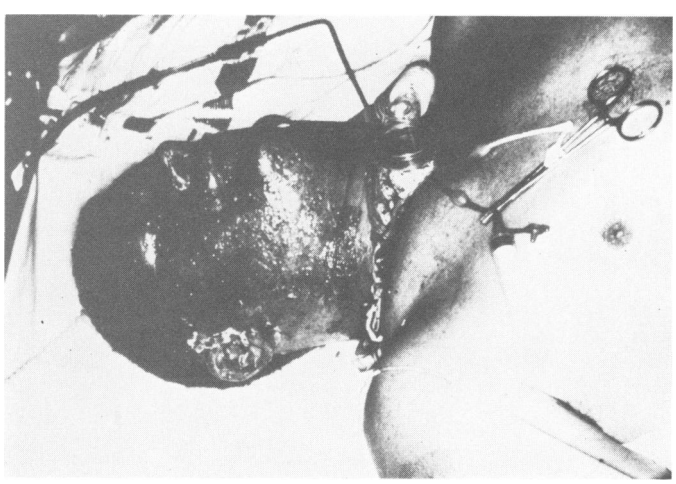

FIG. 2. Case 2: (K.D.) Appearance at admission, patient has mostly second degree burn.

Admitting carboxyhemoglobin level was 8.9%. Chest X-ray revealed bilateral diffuse densities which increased markedly during the next 3 days. The arterial catheter was removed on the third post-burn day and the patient began on daily tubbing and debridement. The tracheostomy was removed on the 7th post-burn day. The patient responded well, did not require skin grafting and was discharged 3 weeks after admission.

Prompt recognition of this first group and aggressive therapy seem to be the key to management. Blood gases should be monitored closely during the first 24 hours and intubation performed if any respiratory embarassment occurs. Although the use of steroids in burn patients is controversial[6,31] their effectiveness in epiglottitis and croup prompts us to use a short course of high dose steroid to minimize upper airway edema. Bronchodilators are often helpful when bronchospasm (wheezing) is prominent.

The second or delayed complications group of patients was the largest group with 14 patients. This group contained those with very massive burns. The day of onset of respiratory symptoms was inversely related to the per cent burn. Typically, a patient with massive burns will exhibit no respiratory problems on admission, have a negative chest X-ray and normal blood gases. However from 24 hours to 5 days after admission the patient will become dyspneic, develop pulmonary infiltrates and hypoxia and require mechanical ventilation. Two patients were not intubated in this group, one patient with a minor burn was noted to have atelectasis that responded to inflation and tracheal cleaning, and a 9-month-old baby with 95% scald burns.

Case 3: R. L. (OCMC 292461) A 42-year-old man was cleaning his garage with gasoline when it exploded, engulfing him in flames. He ran from the garage and rolled in the grass finally succeeding in extinguishing the fire by jumping into a swimming pool. He was admitted to the Orange County Medical Center Burn Unit with 90% second and third degree burns including singed eyelashes and nasal vibrissae and soot in his oropharynx. A central venous catheter, arterial line via a radial artery cutdown, naso-gastric tube and bladder catheter were placed. His chest X-ray was clear on admission. He was started on Mafenide Acetate and Neosporin surface treatment. Admission blood gases were Po_2 82, Pco_2 34 and pH 7.43. During the first 24 hours, he was given large amounts of fluid and had wide fluctuations in central venous pressure. Two days after admission he became very hoarse with labored respiration. A naso-tracheal tube was placed. X-ray on this day demonstrated pulmonary edema. After the naso-tracheal tube has been in place 48 hours a tracheostomy was performed. Blood pressure and urinary output became difficult to maintain and he died 6 days after admission. At autopsy pulmonary edema was noted and the lungs weighed 2,050 and 1,610 Gm.

Case 4: G. R. (OCMC 026219) An 11-year-old boy was trapped in a burning automobile following an accident. He sustained a burn of 55% of his body surface involving the upper one half of his body (mostly fourth degree) (Figure 3). On admission to the Orange County Medical Center Burn Unit a central

venous pressure line, arterial catheter, nasogastric tube and bladder catheter were placed. Surface therapy using Mafenide Acetate and xenografts was instituted. He was given several transfusions in addition to Brooke formula to maintain his hemoglobin and blood pressure. On the fifth post-burn day a tracheostomy was done after the patient developed crowing respirations and he was placed on a volume regulated ventilator. Bilateral pulmonary infiltrates were noted six days after admission. *Staphylococcus aureus* was cultured from his sputum and he was started on methicillin. Later the sputum grew *Pseudomonas aeruginosa* and gentamycin was added. Enzyme treatment (Travase®) resulted in debridement of eschar with exposure of subcutaneous fat and no granulation. On the 19th day he was taken to the operating room in which sharp debridement and split thickness skin grafting was done. The patient died 7 days after operation. At this time *Pseudomonas aeruginosa* was cultured from the blood. X-rays were negative (Fig. 4A) until the fifth post-burn day (Fig. 4B) when they demonstrated bilateral pulmonary infiltrates which cleared by the 12th day except for a residual right upper lobe infiltrate (Fig. 4C). Confluent bronchopneumonia was found at autopsy. This patient died from *Pseudomonas aeruginosa*, septicemia and was the only such patient in this series of 100.

The late group of pulmonary complications was represented by only one patient in this series. This was a 20-year-old woman who sustained burns (mostly second degree) over 70% of her body surface in an automobile accident. She had facial edema and a tracheostomy was performed at an outlying hospital. However, she had no pulmonary problems initially with normal blood gases and chest X-rays. She responded well for 3 weeks, was eating well, afebrile and apparently a survivor when she suddenly died without antecedent or terminal symptoms. The only finding at autopsy was an area of bronchopneumonia. It was assumed that the patient died of arrhythmia without any clinical signs of pulmonary insufficiency, but the autopsy findings required that she be included here. Although this group was not well represented in this series it is a common one in other reports[7] and usually included bacterial pneumonia or pulmonary embolism.

Discussion

Incidence

Physiologic fluid replacement and topical antibiotics have reduced fatality from shock and sepsis, unmasking the high incidence and mortality of pulmonary complications. The 22% incidence of pulmonary complications agrees well with other series. Stone[13] reported 15.1%, Shook[14] 23% and Pruitt[7] 24%. The mortality rates reported in this series also agree with other reported series. Stone has reported up to an 89% mortality rate, Shook reported that 84% of the deaths in his series exhibited pulmonary findings as a major cause of death. Walder[15] from Brooke Army Hospital reported that bronchopneumonia carried an 86% mortality rate and Pruitt[7] also from Brooke reported that pulmonary edema carried a 71% mortality rate.

With widespread clinical use of blood gas monitoring, volume-controlled ventilators and nursing and inhalation therapy personnel who are highly skilled in caring for patients with pulmonary problems, a more scientific approach can be elicited for better understanding and management of these patients.

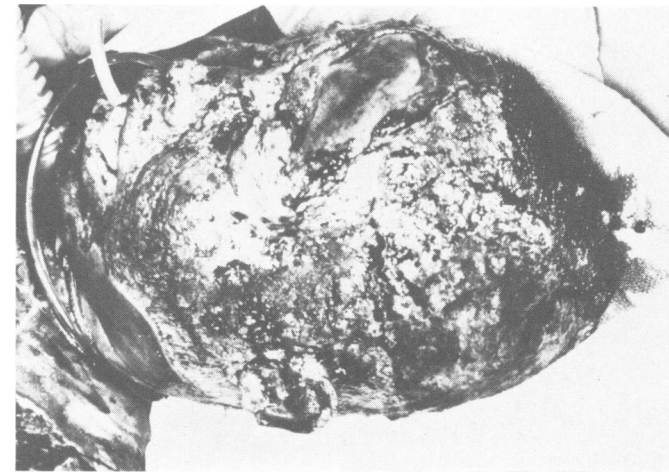

FIG. 3. Case 4: (G.R.) Appearance 6 days after admission. All third degree burn plus area of denuded calvarium.

Pathogenesis

Direct Injury

Many etiologic factors are interrelated as shown in Figure 5. Direct heat injury to the lung is not a major contributing factor. Moritz[21] demonstrated that a direct burn of the respiratory tract below the larynx is extremely difficult to produce because of the very effective cooling of air by the upper airway. A blast of hot air causes reflex closure of the vocal cords reducing even further the chance of direct heat injury to the lower respiratory tract. Live steam with a heat-carrying capacity 4,000 times that of hot air can produce direct heat injury of

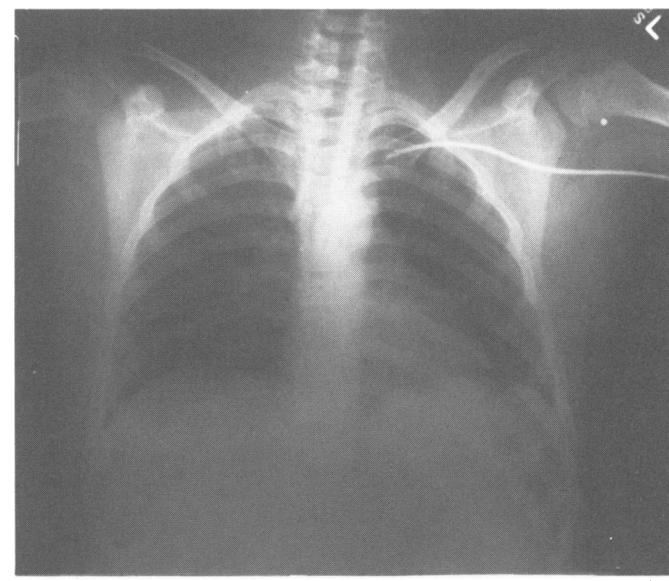

FIG. 4A. Case 4: (G.R.) at admission.

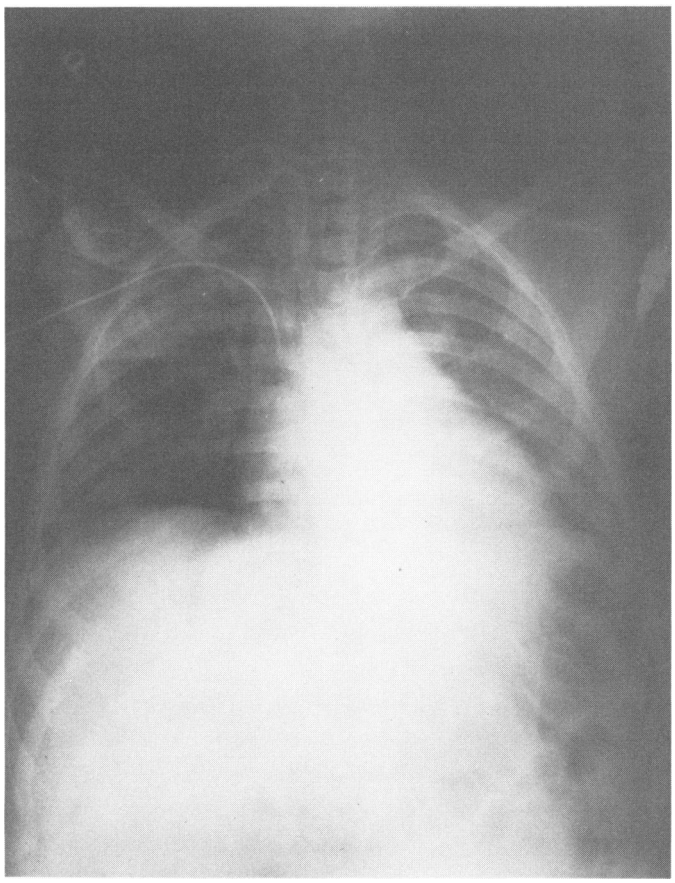

FIG. 4B. Case 4: Five days post burn.

the lower respiratory tract; however, this is unusual and did not occur in this series. Only one patient of 697 burn cases at Brooke Hospital demonstrated evidence of direct heat necrosis, and this extended only a few centimeters below the glottis.[24] This does not indicate that the lungs do not sustain direct injury. The products of combustion of various common substances are very toxic to airways

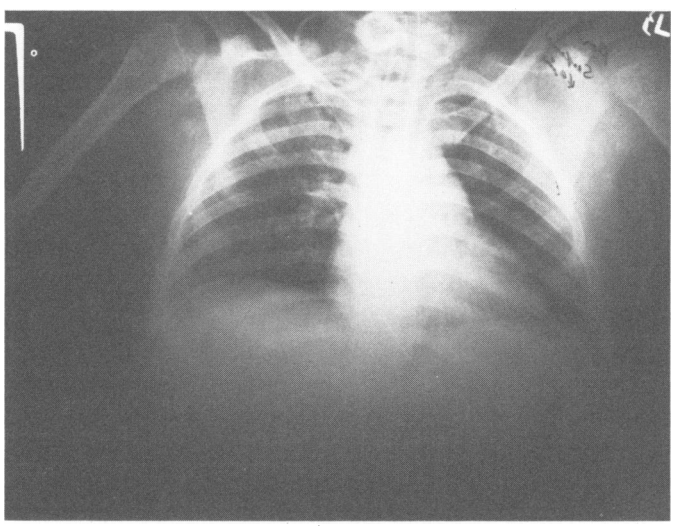

FIG. 4C. Case 4: 12th post burn day.

and alveoli. Initially, mucosal cilia are paralyzed (Beal[6]) and cease to clear the airway of debris. Upper airway obstruction secondary to inflamed mucosa or plugs of soot and mucus may occur. Bronchospasm may be triggered by irritating chemicals. A loss of surfactant has been reported as a consequence of thermal injury.[17]

Shallow Breathing

Atelectasis may occur because of decreased movement of the chest wall secondary to eschar, pain, narcotics, and altered mental status. Tidal breathing is normally punctuated by a spontaneous deep breath, a sigh or yawn every 5 to 10 minutes.[16] These deep respirations expand alveoli which are underinflated during tidal breathing. A patient with painful burn, cerebral depression or curass-like eschar loses this important variation in the pattern of breathing. Continuous tidal breathing results in gradual alveolar collapse, (decreased functional residual capacity) causing decreased compliance and shunting.[3,8,13] (When the shunt exceeds 25%, cyanosis and tachypnea are noted and the decreased lung volume is visible on X-ray as atelectasis.) Atelectasis also occurs in areas where pulmonary blood flow is increased by gravity in the patient who constantly maintains the same body posture. Capillary transudation occurs first in these areas (usually the same areas that are poorly ventilated during tidal breathing).

Increased Lung Water

Chemical pulmonary edema is one of the major problems in dealing with burn patients. There is evidence of increased pulmonary capillary permeability from direct chemical injury to pulmonary endothelium from a variety of sources. Initially irritating chemical products of combustion are the major insult.[19] Good biochemical evidence has recently been presented for the often postulated burn toxin. This peptide may prove to play an important part in lung and cardiac damage.[1,26] A third factor is the low flow state that may occur resulting in inadequate perfusion of peripheral tissue and lactic acidosis. Prolonged poor perfusion results in cell damage and clotting in capillaries. When adequate flow is reestablished these products are flushed back into the venous circulation. The lungs acts as a sieve for this material and may sustain damage from direct effects on the pulmonary capillary endothelium from activation of vasoactive kinins, or from systemic intravascular coagulation.[7] Prolonged exposure to high oxygen concentration[20] (albeit necessary) and the micro embolic effects of blood transfusion[22] may be contributing factors.

Cardiac

Compounding these factors which alter capillary permeability are the shifts in circulating blood volume and

hydrostatic pressure which lead to preferential edema in the lung. During the acute edema phase the entire extracellular space is enlarged (especially when non-colloid fluid is infused) and pulmonary transudation occurs when the left atrial pressure exceeds 25 mm. Hg. Right atrial central venous pressure does not reflect the minute-to-minute changes occurring in the left atrium. The left ventricle may become transiently overloaded and go in and out of failure for short periods, leaving extra water in the lung interstitium without causing gross changes in right atrial pressure. The efficiency of the left ventricle improves with increasing atrial filling pressure up to about 25 cm. of water, therefore, error on either side may be disastrous. Small amounts of proteinaceous fluid in alveoli will appear as hyaline membranes on histologic examination.[10]

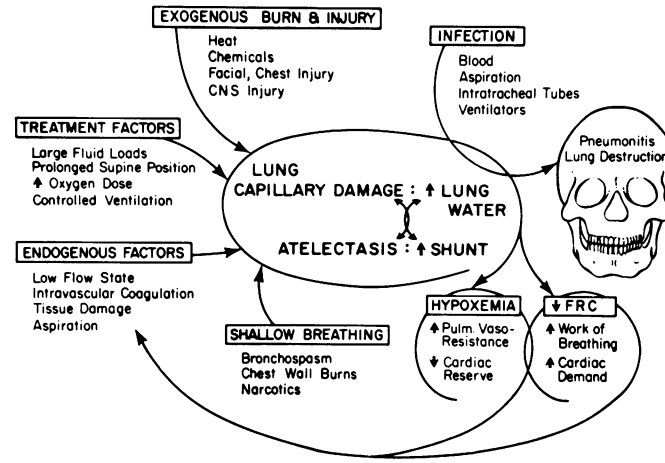

FIG. 5. Pathogenesis of pulmonary insufficiency in burn patients.

Infection

Pneumonitis originating from the burn wound or from the endotracheal tube, suction apparatus, or ventilator supervenes in areas of boggy atelectasis. The development of pneumonia is a constant threat and seems to occur after the patients have survived a series of difficult hurdles and are on the way to recovery.

Pulmonary Embolism

Sevitt[27] found only a 5.5% incidence of pulmonary embolism in 163 autopsies on burn patients as compared with 20.3% in 468 cases of trauma. No pulmonary emboli were recognized in our group of patients.

Prevention and Management

Review of the literature and this series of patients has resulted in the evolution of the following protocol for prevention and management, which has been used in our unit since July, 1971.

Preventing Obstruction

Close attention to obstruction secondary to edema, sloughed mucosa and bronchospasm is essential. Frequent endotracheal suction or bronchoscopy, examination of the upper airway, and the use of bronchodilators and steroids (especially in the early phases of upper airway complications) as mentioned earlier are recommended. Endotracheal intubation is now used in preference to tracheostomy when the major problem is upper airway edema. A naso-tracheal tube is passed at the first sign of stridor and left in place (with humidification) for several days if necessary. The patient is allowed to breath spontaneously and mechanical ventilation is instituted only if needed (see below). It is our impression that bacterial contamination is less with this procedure than with primary tracheostomy.

Preventing Atelectasis

Maximal inflation hourly is emphasized. This can be done by use of deep breathing exercises, incentive spirometer,[4] or carefully administered volume controlled positive pressure breathing.[2] Escharotomies are done on patients with circumferential chest burns.

Preventing Pulmonary Edema

Because of the relative insensitivity of right atrial pressure monitoring, pulmonary artery pressure monitoring has been instituted for fluid replacement in addition to the CVP in patients with large burns or difficult respiratory and cardiac problems. A balloon-tipped catheter[32] is inserted in a peripheral vein and fed through the right atrium, and right ventricle into the pulmonary artery. Pressure is recorded on an oscilloscope. These pressure readings also serve as a navigational aid during introduction of the catheter. PA pressure is used as an upper limit to volume infusion in a fashion similar to CVP. If the mean is less than 10 cm. of water, infusions are accelerated until normal peripheral perfusion is achieved. If mean PAP reaches 20 cm. water fluids are curtailed and inotropic drugs are instituted. The plan of fluid management in the acute phase has been radically changed. There is no doubt that patients in hypovolemic shock can be resuscitated with large amounts of crystalloid solution, but the price is often pulmonary complications for the reasons mentioned above. Plasma is lost into the burn wound, and we believe that Moore[19] is right in stating that plasma must be replaced. Fluid management is based on infusion of 5% protein solution in saline to maintain normal perfusion (normal arterial pressure, no metabolic acidosis). The amount required usually comes out remarkably close to the 10% of body weight predicted by Moore,[19] and is much less than the total volume previously given to large burns using crystalloid. The only

other fluid given is "dry" estimate of daily water requirement.

Diminishing the polypeptide "burn toxin" with early eschar excision is an attractive hypothesis worthy of laboratory and clinical investigation.[1,18] Some of the effects of this or other myocardial depressant factors can be reversed with inotropic drugs. We routinely digitalize patients with burns over 40%.

Ventilator Management

The principles of ventilator management on our surgical services have been described in detail elsewhere.[2] The need for mechanical ventilation is based on clinical judgement and measurements of vital capacity, tidal volume, and arterial blood gases breathing air and 100% oxygen (Table 2). Tracheostomy is avoided in favor of an endotracheal tube to minimize the possibility of airway contamination. A volume controlled ventilator is used with large tidal volumes (10 to 15 cc./Kg.), continuous positive airway pressure, and intermittent maximal inflation to total lung capacity. Daily sputum cultures and chest X-rays are routines. Systemic antibiotics are given only for two consecutive positive sputum cultures.

Using the technics outlined, mortality figures have demonstrated improvement. Since this series was completed (July 1971–January 1972) there have been 99 admissions to the burn unit with 21 pulmonary problems (incidence of 22.6%) and six deaths (28% mortality). Although the series are not similar (Table 3) we feel that we are making progress and this program of management has a sound scientific basis.

Summary

Pulmonary complications cause or contribute to the death of most patients who die after thermal injury. Twenty-two of 100 patients in this series developed pulmonary complications with an 86% mortality. The severity of the burn and the history of being in an enclosed space are the most important predicting factors. The pulmonary complications of burns can be divided into three categories with reference to time of onset and pathogenesis: 0 to 24 hours (direct inhalation damage and airway edema), 2 to 5 days (atelectasis, pulmonary edema, pneumonitis), and after 5 days (late pneumonitis and pulmonary embolism). A program of management based on this review is presented emphasizing early intubation, mechanical ventilation for specific indications, pulmonary artery pressure monitoring, and no crystalloid fluid infusion. An improvement in mortality statistics has been noted since this program was insituted.

TABLE 2. *Indication for Mechanical Ventilation.**

	Needs Ventilator			Breathing Adequate
Inspiratory (cmH_2O) Force	5		20	80
Vital capacity / Tidal Volume	1		2	10
PaO_2 [100% O_2] (mm. Hg)	50		100	600
PaO_2 [AIR] (mm. Hg)	20	35	45	100
$PaCO_2$ (mm. Hg)	100	60	50	20
		↓	↳	Patients with normal lungs
	Patients with chronic pulmonary insufficiency			

* (Adapted from Bartlett, R. H., Post Traumatic Pulmonary Insufficiency in Surgery Annual, 1971, (with permission).

TABLE 3. *Comparison of Series A and B. Series A had an 86% Mortality and Series B a 28% Mortality.*

Survival Probability	Series A 1970–71	Series B 1971–72
<10%	11	2
10–40%	6	6
>40%	4	11

References

1. Allgower, M., Burri, C., Cueni, L., Engley, F., Fleisch, H., Gruber, U. F., Harder, F. and Russel, R. G. G.: Study of Burn Toxins. Ann. N. Y. Acad. Sci., **150**:807, 1968.
2. Bartlett, R. H.: Post-Traumatic Pulmonary Insufficiency. (Ed: Cooper & Nyhus) Appleton-Century-Crofts, N. Y. Surgery Annual, 1971.
3. Bartlett, R. H., Krop, P., Hanson, E. L. and Moore, F. D.: The Physiology of Yawning and its Application to Postoperative Care. Surg. Forum, **21**:222, 1970.
4. Bartlett, R. H., Gazzaniga, A. B. and Geraghty, T.: The Yawn Maneuver: Prevention and Treatment of Postoperative Pulmonary Complications. Surg. Forum, 1971.
5. Bartlett, R. H. and Munster, A. M.: An Improved Technique for Prolonged Arterial Cannulation. N. Engl. J. Med., **279**: 92, 1968.
6. Beal, D. D., Lambeth, J. R. and Conner, G. H.: Follow-up Studies on Patients Treated with Steroids Following Pulmonary Thermal and Acrid Smoke Injury. Laryngoscope, **78**:396, 1967.
7. Blaisdell, F. W., Lim, R. C., Jr. and Stallone, R. J.: The Mechanism of Pulmonary Damage Following Traumatic Shock. Surg. Gynecol. Obstet., **130**:15, 1970.
8. Caro, C. G., Butler, J. and DuBois, A. B.: Some Effects of Restriction of Chest Cage Expansion of Pulmonary Function in Man: An Experimental Study. J. Clin Invest., **39**:573, 1960.
9. Cope, O. and Rhinelanch, F. W.: The Problem of Burn Shock Complicated by Pulmonary Damage. Ann. Surg., **117**:915, 1943.
10. Foley, D. F., Moncrief, J. A. and Mason, A. D.: Pathology of the Lung in Fatally Burned Patients. Ann. Surg., **167**: 251, 1968.
11. German, J., Achauer, B. Woolley, T., Furnas, D. and Bartlett, R.: The Use of Porcine Skin in the Care of Burns: A Clinical Reappraisal. Arch Surg., 1972.
12. Harrison, H. N.: Respiratory Tract Injury, Pathophysiology

and Response To Therapy Among Burned Patients. Ann. N. Y. Acad. Sci., **150**:627, 1968.

13. Laver, M. B., Morgan, J., Bendixen, H. H. and Radford, E.: Long Volume Compliance with Arterial Oxygen Tension During Controlled Ventilation. J. Appl. Physiol., **19**:725, 1964.
14. Long, J.: On the Post-Mortem Appearance Found After Burn. London Medical Gazzett, **25**:743, 1840.
15. McCoy, J. A., Micks, D. W. and Lynch, J. B.: Discriminant Function Probability Model for Predicting Survival on Burned Patients. J.A.M.A., **203**:644, 1968.
16. McCutcheon, F. H.: Atmospheric Respiration and Complex Cycles in Mammalian Breathing Mechanisms. J. Cell Physiol., **41**:291, 1953.
17. Matsuura, Y.: Pulmonary Compliance and Surfactant Activity in Thermal Burn. Surg. Forum, **17**:86, 1966.
18. Monafo, W. W.: Early Tangential Excision of Burn Eschar. Arch. Surg., **104**:503, 1972.
19. Moore, F. D.: The Body Weight Burn Budget Basic Fluid Therapy for the Early Burn. Surg. Clin. North Amer., **50–6**:1249, 1970.
20. Morgan, A. P.: The Pulmonary Toxicity of Oxygen. Anesthesiology, **29**:570, 1968.
21. Moritz, A. R., Henriques, F. C. Jr., McLean, R.: The Effects of Inhaled Heat on the Air Passages and Lungs: An Experimental Investigation. Am. J. Pathol., **21**:311, 1945.
22. Mosley, R., Doty, D. F.: Changes in the Filtration Characteristics of Stored Blood. Ann. Surg., **171**:329, 1970.
23. Phillips, A. W., Cope, O.: Burn Therapy: II The Revelation of Respiratory Tract Damage as A Principal Killer of the Burned Patient. Ann Surg., **155**:1, 1962.
24. Pruitt, B. A., DiVincenti, F. D., Mason, A. D., Foley, F. D. and Flemma, R. J.: The Occurrence and Significance of Pneumonia and Other Pulmonary Complications in Burned Patients: Comparison of Conventional and Topical Treatments. J. Trauma, **10**:519, 1970.
25. Pruitt, B. A., Flemma, R. J., DiVincenti, F. C. Foley, F. D. and Mason, A. D., Jr., Pulmonary Complications of Burn Patients. A Comparative Study of 697 Patients. J. Thorac. Cardiovasc. Surg., **59**:7, 1970.
26. Schoenenberger, G. A., Allgower, M.: Isolation, Activity and Physiological Characterization of a Burn Toxin from Mouse Skin. Br. J. Surg., **55**:704, 1969.
27. Sevitt, S. and Gallagher, N.: Venous Thrombosis and Pulmonary Embolism. A Clinical-Pathologic Study in Injured and Burned Patients. Br. J. Surg., **48**:475, 1961.
28. Silverstein, P. and Dressler, D:. Effect of Current Therapy on Burn Mortality. Ann. Surg., **171**:124, 1970.
29. Shook, C. D., MacMillan, B. G. and Altemeier, W. A.: Pulmonary Complications of the Burn Patient. Arch. Surg., **97**:215, 1968.
30. Sochor, F. M. and Mallory, G. K.: Lung Lesions in Patients Dying of Burns. Arch. Pathol., **75**:303, 1963.
31. Stone, H. H., Martin, J. D.: Pulmonary Injury Associated with Burns. Surg. Gynecol. Obstet., **129**:1242, 1969.
32. Swan, H. J. C., Ganz, W., Forrester, J. *et al*: Catheterization of the Heart in Man with Use of a Flow Directed Balloon-tipped Catheter. N. Engl. J. Med., **283**:447, 1970.
33. Walder, A. I., Summerlin, W. T., Mason, A. D., Foley, F. D. and Moncreif, J. A.: Respiratory Complications in the Acutely Burned Patient. A Clinical Study. Milit. Med., 1967.