Burn Therapy:

IV. Respiratory Tract Damage (An Account of the Clinical, X-ray and Postmortem Findings) and the Meaning of Restlessness

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Introduction

"Anoxia not only stops the machine, but also wrecks the machinery"—Haldane

REVIEW of the patients with burns treated at the Massachusetts General Hospital over the past 20 years has revealed that damage to the respiratory tract is today a principal cause of death.[18] In civilian practice such damage is most commonly seen following flame burns sustained in confined spaces, and second or third degree flame burns around the nose and mouth warn of its presence.[19] The present paper is concerned with the nature of the damage, the signs and symptoms it produces, the x-ray findings, the pathologic changes found at autopsy and the results of a few laboratory measurements of vital capacity, oxygen content, oxygen capacity and carbon dioxide tension. Emphasis will be laid on the importance of restlessness as a sign of inadequate oxygenation, on the ominous significance of both stupor and chest rales when the burned patient is admitted, and on the deceptive interval prior to pulmonary death when rales diminish and breathing seems easier.

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Blow torch and bunker experiments have established that dry air has such a low heat capacity that it is rapidly cooled and does not burn the lower respiratory tree.[14] It is the products of incomplete combustion, or, in some instances, steam, which appear to wreak the havoc in bronchi, bronchioles and alveoli.

Material and Plan of Study

The patients included in this investigation are described in the second paper of this series.* [18] One hundred and eighty-one of the 932 burned victims studied in detail encountered respiratory difficulties. Those with purely terminal difficulties or with common colds are not included in this number. Of the 181, 31 were ill prior to injury or developed illnesses unrelated to their burns which may have caused or contributed to their respiratory difficulties (Table 1). The balance are considered to have suffered varying degrees of respiratory tract damage due to the inhalation of noxious materials.[1] Respiratory tract damage is thought to have been the primary cause of death in 20. In 20 more, death is attributed to respiratory tract damage

* The findings in some of the cases included in this series have previously been described in the account of the management of the victims of the Cocoanut Grove Fire treated at the Massachusetts General Hospital.[1, 12, 21]

TABLE 1. *Pre-existing or Intercurrent Illnesses which May Have Caused or Contributed to the Patient's Respiratory Difficulties*

Illness	No. Cases
Cardiac disease	11
Pneumonia (pre-existing)	4
Tuberculosis	2
Chronic bronchitis	4
Chronic cough of unknown etiology (all smokers)	5
Croup	1
Asthma	2
Diabetic acidosis	1
Measles	1
	31

complicated by superimposed sepsis. The responsibility in ten other cases is divided equally between respiratory tract damage (with or without superimposed sepsis) and some other cause or causes (Table 2).

In all of the patients with respiratory difficulties, the signs and symptoms referable to the respiratory tract have been tabulated, as have the x-ray and autopsy findings, whenever available. Chest x-rays were taken of 79 of the survivors and 47 of the fatally injured. In 22 cases the chest was re-examined once and in 51, three or more films were made. Only the x-ray findings of the first week after injury are presented here. Twenty-one patients dying of respiratory tract damage (with or without superimposed sepsis) came to necropsy.

In a few cases determinations were also made of vital capacity, oxygen content, oxygen capacity and carbon dioxide tension. Although the number is small, the data is presented as contributing some information as to the burned patient's capacity for aeration and oxygenation.

Observations

I. Signs and Symptoms

Comparison of the signs and symptoms of respiratory tract damage in the fatally injured with those in the survivors reveals a startling paucity of data concerning the symptoms of the former group. Only two patients in the fatal group complained of sore throats, for example, although reddened pharynges were common. Dyspnea was hardly mentioned although labored respirations were almost universally experienced (49 of 50 cases). The explanation lies partly in the difficulty of talking: 22 of the fatal cases had tracheostomies; three had intratracheal tubes; one did not speak English and two were small children. Cerebral anoxia, however, with attendant stupor or confusion, was the major bar to communication.

A. Restlessness, Irrational Behavior and Stupor Warn of Respiratory Damage. Twenty-one of the patients dying of respiratory tract damage were *comatose* or *semi-comatose* on admission. Four were *uncooperative* and *irrational.* Eight were *confused* or *disoriented* and could not give a coherent history. Restlessness was noted in a total of 16 patients on admission, 12 of whom died within 48 hours. Two of these appeared in *panic* and *maniacal.* Of the remaining four, three, including one who was *hysterical* on the day of admission, died on the third day. The last patient survived a week.

Seven other patients, although not apparently restless on admission became *agitated, difficult* or *restless* on the day of death or from one to five days before death, as respiratory difficulties developed. An eighth managed to survive two weeks after becoming restless.

B. Chest Rales on Admission Are Ominous. Although many patients admitted with rales in their chests survived, nevertheless it proved a poor prognostic sign. Among those dying of respiratory tract damage the average survival time of 24 patients having chest rales or rhonchi on admission was 2.3 days; 14, whose chests were clear on admission, survived an average of 6.7 days. The difference is statistically significant. Auscultation of the chest of the remainder of the patients dying of

respiratory tract damage was either inconclusive because of the presence of dressings or was omitted because of the patient's critical condition.

C. Diminution in Chest Sounds Should Not Occasion Premature Optimism. In eight patients dying of respiratory tract damage a deceptive diminution in chest rales and rhonchi was noted after the first day or two, and respirations seemed less labored. X-ray and postmortem examinations indicate that atelectasis rather than improvement in pulmonary aeration, was responsible for the decreased chest sounds. The x-ray and pathologic changes occurring following respiratory tract insults are described to a greater extent below.

D. Labored Respirations Are the Most Common Finding. The respirations of 49 of the 50 fatally-injured patients were labored.* Expiration was generally more difficult than inspiration, the expiratory phase being prolonged. Obstructive breathing, characterized by stridor or crowing sounds, was observed in at least six cases and wheezing was apparent in five.

Among the survivors also, respirations were often labored, no other sign being as frequent. Forty-eight of 100 survivors breathed with difficulty. Expiratory wheezing was common and in three cases there was frank stridor.

E. Many Are Cyanotic. Cyanosis on admission often carried a grave prognosis. It was observed in 25 of the fatal cases. All but eight of them were dead within 48 hours. Among these eight an interval without cyanosis (or with diminished cyanosis) was common before its recurrence as a terminal event. Eight other patients showed no cyanosis on admission, but developed it from a few hours to five days before death. It is possible that some of them were anoxic from the start, but until shock had been corrected cyanosis was not apparent clinically. The misleading effect of carbon monoxide on the evaluation of cyanosis is dealt with below. Cyanosis was also common among survivors. Twenty-two of them were cyanotic at some time during their hospital stay, usually on admission.

TABLE 2. *Deaths from Burns Due Equally to Respiratory Tract Damage and Other Causes*

Associated Cause or Causes	No. Cases
Shock	3
Wound sepsis	2
Wound sepsis and uremia	1
Overhydration	1
Myocardial failure	1
Hepatitis and cystitis	1
Intracranial injury	1
	10

F. The Cherry Red Color of Carbon Monoxyhemoglobin May Mask Cyanosis. The extent to which anoxemic anoxia played a role in the fatal outcome of the patients of this series is not known. Carbon monoxide levels were not ascertained. In at least three cases, however, the color of the patients was reported as *normal* shortly before death. Only after death was the apparently healthy color identified as the cherry red hue of carbon monoxyhemoglobin.

Fifteen survivors were cherry red in color on admission and in one of them oxygen transport was reduced to the point of irreversible brain damage. Although only 6 per cent of her body had been burned, the damage to her central nervous system was so severe that she became permanently childish and slow mentally.

G. Hoarseness and Sore Throat Suggest Respiratory Irritation. In addition to those who had frank respiratory stridor, at least six of the fatally injured were hoarse. This difficulty also afflicted 22 of the sur-

* The fiftieth patient, a boy of 16, died of massive pulmonary collapse while under anesthesia for skin grafting seven days after injury. He had had no premonitory signs or symptoms other than a few squeaks heard in his chest on the day of admission.

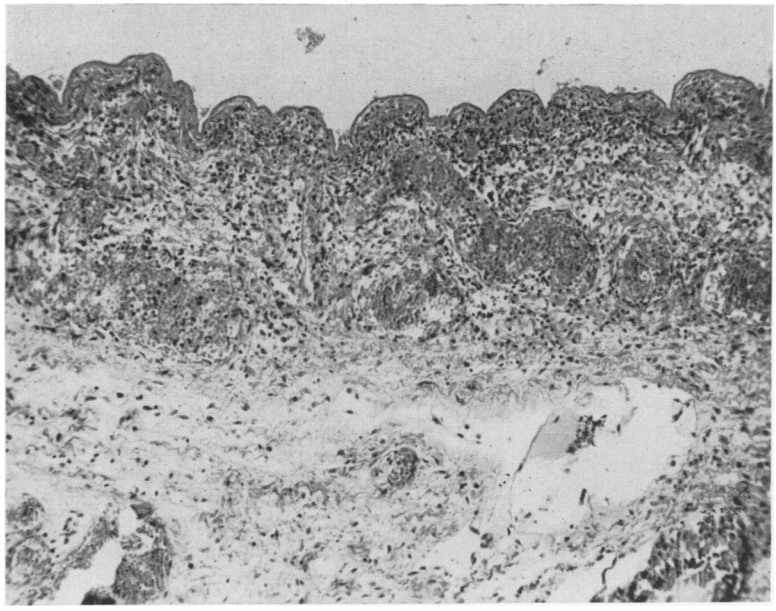

FIG. 1. Complete loss of the normal respiratory epithelium is seen in this section. Only the basement membrane is retained. Capillary dilatation, congestion and edema are marked. The inflammatory infiltrate consists of both polymorphonuclear and lymphocytic cells.

vivors, two of them losing their voices altogether. Thirty-three survivors also suffered from soreness in their throats. Reddening of the pharynx was common.

Among the fatally injured laryngoscopic examination generally revealed edema and reddening of the hypopharynx, larynx, and epiglottis, with patchy debris adherent in some cases. In two cases the tongue was swollen as well. In several patients because of edema the larynx could not be visualized at all.

H. Coughing Is the Respiratory Victim's Most Irritating Friend. Next to labored respirations, coughing was the most frequent sign of respiration tract injury in both the survivors and the fatal cases. The presence of blood-tinged material in the sputum during the early days after injury was noted in a number of the fatal cases, but was rare among the survivors, occurring only in the most critically ill. Carbon particles were coughed up, or found in the airway on tracheostomy, in both groups, although more commonly in the serious cases. Similarly in both groups bronchial casts were either coughed up or withdrawn by suction through the tracheostomy tube. These casts were noted as early as the first day after injury. In some instances the patient's color and condition improved dramatically once the cast was dislodged from the respiratory tree.

As a rule, purulent material did not appear in the sputum until after the third or fourth day. As will be seen below, bacterial invasion began much earlier.

II. The Pathologic Changes

The outstanding autopsy findings in the patients with respiratory tract damage are denuding of the respiratory mucosa, pulmonary congestion, edema of the tracheobronchial tree, plugging of the smaller bronchi with epithelial debris and edema fluid and atelectasis distal to the plugs. In the patients surviving 48 hours or more, superimposed bacterial infection was generally evident as well.

A. The Airway Is Obstructed, Most Commonly in the Smaller Bronchi. Congestion and edema of the respiratory tree was found in every case in which the airway was examined (18 cases). Almost total

occlusion of the larynx was noted in two cases, but in the majority obstruction occurred only in the smaller bronchi. The narrowed lumina of the secondary and tertiary divisions were often partially or completely plugged with epithelial debris, fibrin, edema fluid, carbon particles, white cells and mucus. In seven cases (including one who died 4 weeks after injury) large bronchial casts were present. In two *all* of the smaller bronchi examined were found to be obstructed.

B. The Tracheobronchial Mucosa Is Denuded. In 16 of the 21 cases the trachea was examined microscopically; all showed intense inflammation. The same was true of the bronchi in all 21 cases. In three, patchy necrosis and ulceration was observed, while in the remainder the epithelium was completely destroyed (Fig. 1).* Complete membranous replacement of the denuded epithelium had occurred in four cases. It is interesting to note that had microscopic examination not been made, this denuding of the respiratory epithelium would have been missed in three cases, for in each the report of the gross examination stated that the tracheal and bronchial mucosa was intact, while microscopic examination revealed complete epithelial destruction.

C. Atelectasis, Trapped Air and Emphysema Are the Progeny of Obstruction. Scattered patchy atelectasis was observed at necropsy in 13 cases. Complete collapse of one or more lobes had occurred in six. The posterior portions of the lower lobes or the entire lower lobes were most frequently involved. In four cases trapped air was seen distal to bronchial plugs. In the nonatelectatic areas compensatory emphysema was often present.**

D. The Alveoli Themselves May Be Plugged. Plugging of the alveoli was seen in 12 cases, including three in whose necropsy protocols atelectasis was not mentioned. In two patients *almost all the alveoli* were filled and in one, half of the lung was involved. In the remainder the extent was somewhat less widespread, involving many alveoli in scattered areas or in a single lobe. For the most part, edema fluid, cellular debris and occasional bits of fibrin filled the alveoli of those who succumbed early, although hemorrhage into the alveoli also occurred. Those who lived a week generally had alveoli filled with polymorphonuclear leucocytes, serous exudate and debris, the walls of the alveoli themselves appearing partially destroyed. In only one of the cases examined were the alveolar walls intact.

E. Pulmonary Edema May Not Parallel Cutaneous Edema. Pulmonary edema was an invariable companion of respiratory tract damage in all of the cases autopsied. Congestion of the pulmonary blood vessels was often intense, with extravasation or hemorrhage from the dilated lumina. In eleven patients the edema was massive. The course of the development of edema and its duration are not clear. Death with pulmonary edema occurred in at least one patient of the series each day from the day

* In two cases the loss of epithelium was not interpreted as an antemortem phenomenon. In one it was described as an "artifact due to stripping" and in the other as evidence of "postmortem disorganization."

** The possibility that inhalation of 100 per cent oxygen might be an important factor in the development of atelectasis in the burned patients has been investigated. Because many patients in this series received oxygen in varying concentrations there is not sufficient evidence for statistical analysis, as yet, but on the basis of the data available it does not appear that the absence of nitrogen as an alveolar splint was the predominant factor in the atelectasis seen. Atelectasis occurred prior to oxygen administration in many instances and did not appear to develop more frequently in patients treated with intratracheal oxygen in high concentrations than in those kept in tents containing greater amounts of nitrogen.

TABLE 3. *Times of Death from Massive Pulmonary Edema*

Days
0.7
1.7
2.3
3.4
3.8
5.6
6.0
6.7
7.0
7.4
8.5

of admission (Day 0) through the eighth day with the exception of the fourth (Table 3). Two patients died on each of Days three, six, and seven. It has not been possible to correlate the course of this edema with that of the cutaneous burns, with the depth of the pulmonary injury, or with the fluid therapy.

F. The Damaged Airway Is Wide Open to Bacterial Invasion. Invasion of the damaged respiratory tract tissues probably begins immediately after injury. As early as 17 hours after injury, cocci were found in the alveoli of one patient. In a second, at a day and half after the burn, both pneumococci and streptococci were present in the exudate in one lobule. Frank bronchopneumonia had developed to the point of abscess formation in others as early as the third day, but the majority of the patients with bronchopneumonia did not come to necropsy until five days after injury. Since only five of the 22 patients who were tracheostomized survived beyond the first 48 hours and only two of them were autopsied, it is not possible to determine whether or not tracheostomy alters the hazard of bronchopneumonia.

III. The Roentgenographic Findings

Chest x-rays revealed abnormalities in 89 patients, 55 survivors and 34 of the fatally injured. In nine cases the abnormalities are known to have existed prior to the burn injury and in five the development of pulmonary edema is thought to have been on a cardiac rather than a pulmonary basis. Seventy-five patients remain whose abnormal chest findings are presumed due to the inhalation of noxious materials.

A. A Single Negative X-ray Does Not Rule Out Respiratory Tract Damage. The majority of the patients with positive chest x-ray examinations showed abnormalities at the time of their first examination, but in ten patients, whose x-rays shortly after admission were negative, a second chest examination done within 72 hours revealed clearcut changes. Scattered areas of atelectasis had developed in the lungs of nine, and trapped air could be recognized in three.

A single chest film was also taken and interpreted as negative in each of three fatal cases. Permission for autopsy was obtained from the medical examiner in two. Both showed extensive ulceration of the tracheobronchial mucosa, dilated, partially filled alveoli and intense pulmonary congestion. Flecks of black material were found in the debris filling their bronchi.

B. Chest X-ray Examinations Should Be Repeated in Patients with Second or Third Degree Flame Burns of the Face Sustained Indoors. In the third paper of this series it is shown that 88 per cent of patients with second or third degree flame burns of the respiratory area of the face, sustained in enclosed spaces, develop respiratory difficulties.[19] The usefulness of facial burns as an omen of possible respiratory damage is also borne out by the x-ray findings. All ten of the patients whose initial x-rays were negative, but who showed pathologic changes on re-examination during the next 72 hours, had just such burns of the face. In only one patient so burned did the x-ray findings remain negative repeatedly. During the fire he had breathed through his hands and avoided getting more than a few whiffs of smoke. He had a slight inspiratory wheeze on the day of

admission, but was symptom-free from the respiratory standpoint thereafter.

The chest findings in four other cases, all of whom experienced moderate respiratory difficulty, remained negative.* Two of these had no facial burns. One patient had only first degree burns of the respiratory area and stated that he had crawled from the burning building to avoid the smoke. The fourth, although severely burned about the nose and mouth, was burned out-of-doors.

C. Patchy Pulmonary Densities Are Seen in Patients with Bronchial or Alveolar Plugging and Pulmonary Edema. During the first four days after injury 20 previously healthy patients showed increased densities scattered through their lungs which might be called hazy, mottled, flecky or patchy. Clinically all 20 displayed signs and symptoms of respiratory tract damage. Eight of them survived and 12 died. Seven came to necropsy. Exudate and debris filled the bronchi or alveoli of all seven, and the lungs of each were edematous. Five of the seven showed focal atelectasis as well.

D. Atelectasis Is Not Uncommon in Flame Burn Cases; Its Presence Is Suggested by Linear Densities, Vascular Crowding, Elevation of the Diaphragm or Displacement of a Hilus. In this series chest x-rays taken during the first week after injury demonstrated atelectatic changes in 25 patients, 17 of them survivors and eight fatally injured. One of the fatal cases, a 62-year-old man with a brain hemangioma and bronchopneumonia, had been burned by a hot water bottle, but all the others were burned by flames.

The x-ray findings are such as might be expected when air is absorbed from the alveoli distal to a bronchial plug and the affected area of the lung collapses. The collapsed area itself, containing less air than the surrounding tissue, appears as an area of increased density. The adjacent blood vessels are displaced toward the collapsed tissue so that the vascular markings appear to have pulled inward or, in roentgenologic parlance, *crowded* together. If atelectasis is sufficiently extensive, pressure in adjacent areas similarly displaces a hilus or the diaphragm toward the collapsed portion of the lung.

Massive collapse, involving one or more lobes of the lung was seen on x-ray examination in four of the fatal cases in this series and in four survivors. An entire lung collapsed in one of those who died.

E. Atelectasis May Shift from Lobe to Lobe or Vanish with Effective Coughing. In at least seven cases serial x-rays showed atelectasis first in one portion of the lungs and then in another. In two additional patients atelectasis decreased markedly and then recurred. One of the former group showed dramatic clinical and roentgenologic improvement when she coughed up a plug of pinkish-black material, which appeared to be a bronchial cast.

F. Bronchial Plugs May Also Trap Air in the Lungs. During the first few days after injury trapped air was observed on x-ray in the lungs of 13 survivors and three fatal cases. It was not seen roentgenologically on the day of injury, but was found at autopsy as early as the following day in another patient who was not x-rayed.

G. Diminution of the Oxygenating Capacity of a Part of the Lungs Is Associated with Compensatory Emphysema in Other Portions. Emphysema was seen on x-rays taken within the first three days after injury in 16 cases and was present in seven who were x-rayed on their first day in the hospital. In two emphysema may have antedated the burns, but in the remainder it is thought to have been com-

* It will be noted that although this report includes the Cocoanut Grove cases treated at this hospital a smaller number of repeatedly negative x-rays are reported than were mentioned in the Symposium.[21] It is believed that minor positive findings were present in some of the cases reported as negative at that time.

TABLE 4. *Vital Capacity Changes in a Patient with Respiratory Tract Damage**

Day after Injury	Vital Capacity (average of 3 determinations) %
11	31
14	68
19	80
21	90
22	98
111	98

* X-rays of the chest revealed partial collapse of the left lower lobe on the day of admission followed by extensive atelectasis in the right lower lobe the following day. These changes had regressed markedly by 11th day. By the 18th day atelectasis on the right was minimal and by the 21st the right side of the chest was normal. Some persisting atelectasis was visible in the left lower lobe on the 21st day but had cleared by the 111th day after injury.

pensatory. The patients' ages ranged from 16 to 58 years, the average being 33 years.

H. Gastric Dilatation Should Not Be Overlooked. Gastric distention was noted on x-rays taken shortly after admission in seven of the fatally injured patients and in two survivors all of whom had clinical evidence of respiratory tract damage. In three cases nasal oxygen administration may have played a part in the distention of the stomach, but in the remainder this possible causative factor was absent. All nine of these patients had burns of the mouth. The records of these patients do not show whether or not emptying the distended stomach by aspiration improved the patient's ability to breathe by permitting further descent of the diaphragm, but it is not unreasonable to suppose it did.

IV. Vital Capacity Measurements

Determinations of vital capacity using the McKesson-Scott Apparatus were carried out on 19 survivors. Three measurements were made on each occasion that the vital capacity was tested. Unfortunately, because of severe dyspnea and inability to cooperate, it was not possible to make this type of measurement on the patients with the most severe respiratory embarrassment. Values as low as 30 to 35 per cent of normal for the patients' heights were observed in four of the survivors. X-ray examination revealed massive collapse of one lobe of the lungs in each of the four. Two lobes had collapsed in one, and patchy atelectasis had affected two additional lobes in a second. The vital capacity changes in a typical case are shown in Table 4.

V. Chemical Determinations

A. Oxygen Saturation Does Not Tell the Whole Story—Anemia and Carbon Monoxyhemoglobin May Both Decrease Oxygen Capacity. Unless the oxygen capacity of the blood is known, oxygen saturation figures may be misleading. In the patient described above, who became hopelessly childish, it was known that her oxygen saturation was low. A value of 80 per cent was reported, the normal being 96 to 100 per cent in this hospital. However, her actual oxygen content (10 cc. of oxygen per 100 ml. of blood) * was much lower than the oxygen saturation figure suggests because her oxygen capacity was reduced from the normal range of 15 to 23 volumes per cent to 12.4 volumes per cent. She was severely anoxemic due to carbon-monoxide inhaled during the Cocoanut Grove Night Club holocaust. Similar low oxygen contents were seen in other patients. The lowest observed was 8.6 volumes per cent in a patient who died the day she was burned.

B. Carbon Dioxide Accumulation May Also Produce Toxic Effects. High carbon dioxide tensions occur in some burned patients as a result of their respiratory tract damage. The normal pCO_2 in this hospital ranges between 35 and 45 mm. Hg. Levels as high as 70 and 99 mm. Hg. were seen among the patients in this series who failed to survive.

* Or, in the terminology of the respiratory physiologist, 10 volumes per cent.

Discussion

The oxygenation problems of the burn victim are often only beginning when he is removed from the scene of the fire, where combustion may have brought about a deficiency of oxygen in the atmosphere. The following are all factors which cut down the effective oxygenation of his tissues: 1) Obstruction to oxygen inflow through the airway (debris in the airway, edema of the respiratory tract walls and possibly bronchospasm); 2) Insufficient functioning lung tissue (atelectasis distal to bronchiolar plugs and/or due to shallow breathing and insufficient mobilization); 3) Poor diffusion of oxygen across the alveolar capillaries (pulmonary edema and alveolar exudation); 4) Diminution in hemoglobin available for oxygen transport (anemia due to blood destruction or preexisting disease; carbon monoxyhemoglobinemia and met-hemoglobinemia); 5) Inadequate circulation of blood (shock); 6) Increased tissue demands (restlessness, increased metabolic rate **); 7) Interference with oxygen delivery (tissue edema); and 8) Morphine or other respiratory depressants, whether iatrogenically administered or inhaled.

Intelligent burn therapy calls for an attack on the problem all along the line. To compensate for the low oxygen intake and uptake, the oxygen supply must be high. In view of the 88 per cent incidence of respiratory complications in patients sustaining second and third degree flame burns around the nose and mouth while in enclosed spaces, we believe that such patients should be placed on adequately humidified oxygen at the time of admission. It may be a fatal error to wait for cyanosis or respiratory distress to become obvious.*** One hundred per cent oxygen, however, should probably be reserved for those in evident difficulty.†

Whenever possible, obstruction of the airway must be relieved. We are not fond of tracheostomy, because of the sepsis and increased damage which the suctioning catheter may bring. Nevertheless, we know that in those few cases where the larynx is obstructed, there is no alternative. But it is by no means a cure-all when the trouble lies, as it usually does, in the smaller bronchi. If tracheostomy and suctioning are resorted to, they must be done with all the nicety of a surgical procedure. Asepsis must be rigid. The catheter used in the nose and mouth must never be introduced into the trachea. It has to be realized that tracheal and bronchial suctioning in a burned bronchial tree is quite different from the same maneuver in a patient with an uninjured airway. Ruthless suctioning may avulse the last few remaining islands of variable bronchial epithelium or start small hemorrhages which will further obstruct the respiratory passages. To avoid these additional insults to the airway, suc-

** Following burns of 25 per cent or more of the body surface the metabolic rate is elevated, often to levels of plus 30 or even plus 60.[8] The role of the increased epinephrine released following the burn stress in the production of this elevation of the metabolic rate has not yet been fully assessed.

*** Comroe *et al.* have shown that the patient's color is a poor guide for judging oxygen deficiency even in normal subjects breathing oxygen in reduced concentrations.[6, 7] In the shocked burned patient, in whose constricted peripheral capillaries very little blood is circulating, the danger of overlooking hypoxia is even greater. The cherry-red tint of carbon monoxyhemoglobin further conceals the state of oxygenation.

† There is evidence in the literature that 100 per cent oxygen breathed by normal subjects may of itself cause alveolar damage,[2, 5, 16] but such damage should not necessarily be expected in burned patients with respiratory obstruction and pulmonary edema, since in these patients neither a high alveolar O_2 content nor a high circulating oxygen content will be achieved. Experimentally Ohlsson has shown that protection from oxygen poisoning, which ordinarily occurs in rabbits exposed to 80 or 90 per cent oxygen, can be achieved by prior inhalation of diphosgene, a respiratory tract damaging agent.[15]

tion should always be discontinued whenever the catheter is moved.*

We have not measured the extent to which bronchospasm may interfere with oxygen inflow in the burned patient. Its role and the effectiveness of anti-spasmodics remains to be investigated. In some cases adrenalin and aminophylline have relieved respiratory distress but in others we have found them utterly useless.

It is possible that intermittent deep breathing and coughing may do much toward reducing atelectasis in these patients. Recumbent posture, restrictive dressings, pain, anesthetics and analgesics all tend to reduce ventilation, and it has been shown that whenever deep breaths are not a part of the ventilating pattern, atelectasis develops.[4, 10, 11, 13] ** Higher than normal air pressures are then required to re-open the collapsed air spaces.[4, 10, 11, 13] We believe that the hazard of anoxia so far outweighs any possible deleterious effects of interrupted sleep that the victims of respiratory tract injury should be turned from side to side and encouraged to take deep breaths or cough hourly, day and night.*** Controlled regular pulmonary inflation by mask up to 30 or 40 cm. of water may also do much to minimize the danger of atelectasis.† If the patient is not too dyspneic and can lean over the side of the bed, resting his elbows on a chair, postural drainage may assist in raising obstructing material and aerating the lung bases.

It is curious that very little attention has been given to the possibility that tracheobronchial suction might lead to atelectasis. Recent studies show that it does, indeed, cause air cells to collapse and that deep breaths or inflation are necessary to re-inflate them.[20] In the light of this knowledge, the encouragement of deep breathing after each suctioning of the burned patient's airway should be rigidly enforced.

Regarding the treatment of shock in the presence of respiratory tract damage, we do not know, as yet, whether plasma and electrolytes should be used in smaller amounts than in the burned patients without respiratory tract damage. This question is under study. As to the administration of blood, we feel that whole blood should be used promptly if anemia is present in order to increase oxygen transport.

When shock has been averted, overhydration should be scrupulously avoided to prevent further pulmonary edema. It is essential that intravenous fluids be curtailed as soon as diuresis is established. We suggest that in these patients the occurrence after the thirtieth hour of two successive hourly urine volumes of 40 ml. or more should be considered evidence that diuresis has begun. At that point the I.V. catheter should be either removed, or, if only one or two veins are available, heparinized and plugged.

The treatment of carbon monoxide poisoning in the burned patient needs further attention. Perhaps oxygen under positive pressure will prove helpful, the high oxygen tension aiding in the dissociation of carbon monoxide from hemoglobin. The idea of using positive pressure oxygen in treating burned cases is not new. It was tried and abandoned two decades ago, because the patients went into shock, perhaps due to interference with the thoracic pump.

* This is easily achieved by having a small vent or side tube between the catheter and the source of the suction. When suction is desired, the vent can be closed by placing a finger over the opening.

** Normal people have been shown to take a deep breath, or sigh, nine or ten times per hour. The average volume of the deep breath being a little more than three times the average tidal volume.[4]

*** Pulmonary compliance has been shown to decrease 26–40 per cent during quiet breathing in human subjects whenever deep breaths were prevented or omitted. This decrease could be reversed or prevented by intermittent inflation of the lungs.[11]

† At the time of the Nantucket catastrophe high pressures, exceeding 60 centimeters of water, were required in two victims treated at this hospital to produce normal tidal volumes.

It is possible that another trial will have the same result, but we certainly know how to treat shock far better today than we did 20 years ago.[17] If positive pressure is tried again, we suggest the patients be watched carefully for signs of shock and also for twitching of the lips or other portions of the body. In the normal person the administration of oxygen under pressures of more than one atmosphere produces undesirable central nervous system reactions.[9] If either of these signs develop it might be wise to discontinue the positive pressure.

There are two aspects to the problem of reducing the patient's oxygen utilization, first, that of reducing muscular activity, and second, that of reducing tissue metabolism. The patient with respiratory tract damage is caught in a vicious cycle. The more anoxic he is the more restless he becomes and the more oxygen he consumes.* Obviously, if oxygen administration relieves the burned patient's anoxia, it may also decrease his oxygen consumption by decreasing his restlessness and agitation.

Hypothermia is an obvious theoretical means of reducing tissue metabolism and may be of value in the burned patient, but thus far we have no experience regarding its possible use and dangers.

Beecher has warned of the use of morphine in the burned patient, and other severe respiratory depressants are similarly contra-indicated.[3] If sedation must be given (and often after oxygenation is improved it will be found unnecessary) it should be given intravenously. Otherwise the sedative may remain useless in the tissues during shock only to be absorbed with disastrous consequences later on when additional medication has been given. For the average restless burned adult 50 mg. of pentobarbital sodium given intravenously is a reasonable dosage.[3]

* Comroe and Dripps have pointed out that a 35 per cent decrease in oxygen dissociation in a single leg can be brought about if it is brought to rest after vigorous exercise.[7]

Summary and Conclusions

The respiratory difficulties of 181 patients culled from the records of 932 burned patients admitted to the Massachusetts General Hospital over a 20-year period have been investigated. In 150 the difficulties are attributed to damage to the respiratory tract resulting from the inhalation of noxious products of incomplete combustion.

Autopsy data in 21 cases of respiratory tract damage are presented. The outstanding autopsy findings are denuding of the respiratory mucosa, pulmonary congestion, edema of the tracheobronchial tree, plugging of the smaller bronchi with epithelial debris and edema fluid, and atelectasis distal to the plugs. Bacterial invasion of the damaged tissues probably begins at once and has been detected at postmortem examination as early as 17 hours after the injury.

Symptomatically, the most common features of the respiratory tract damage are labored respirations, cough, hoarseness, sore throat, restlessness, stupor, and confusion.

In the burned patient who has sustained second or third degree flame burns around the nose and mouth while indoors, restlessness, irrational behavior, confusion, stupor and coma should be considered due to anoxia until proved otherwise.

Chest rales on admission may herald severe respiratory difficulties. Their disappearance or diminution after the first day or two may be the result of atelectasis rather than improvement in aeration.

The victim of respiratory tract damage often has a deceptive interval of easier breathing between his initial difficulties and subsequent fullblown respiratory distress.

Cyanosis may be masked by shock or carbon monoxyhemoglobinemia. If present on admission, it often carries a grave prognosis.

The burned patient should be encouraged to cough. Displacement of plugs in

the airway may lead to marked improvement.

Deep breathing and turning from side to side should be insisted upon hourly to combat atelectasis.

Obstruction of the larynx occurs in the burned patient, but in the majority of cases of respiratory tract damage obstruction takes place only in the smaller bronchi. Bronchi and alveoli may be filled with debris and edema fluid.

In the respiratory damage victim who survives 48 hours or more, clinical evidence of infection in the airway is almost always present.

A single negative chest x-ray does not rule out respiratory tract damage. Chest x-rays should be repeated in patients with second or third degree flame burns of the respiratory area of the face sustained indoors, preferably at 48-hour intervals.

Patchy pulmonary densities seen on early chest x-ray in the burned patient suggest bronchial or alveolar plugging and pulmonary edema.

The presence of atelectasis is suggested by linear or segmental densities, crowding of the vascular markings, elevation of the diaphragm or displacement of a hilus. Serial x-rays may show shifting of the atelectasis from lobe to lobe.

Although 100 per cent oxygen inhalation may contribute to atelectasis in the burned patient by reducing the nitrogen available for alveolar splinting, it is not a major factor. Atelectasis occurs in many burned patients before any oxygen is given.

Trapping of air distal to a bronchial plug and compensatory emphysema in portions of the lungs still capable of aerating gives rise to areas of decreased density in the chest films of the victims of respiratory tract damage.

Gastric dilatation is a frequent complication in patients with respiratory tract damage. Although nasal oxygen may contribute to such dilatation, it is not the principal cause of it.

Vital capacity may be seriously reduced by respiratory tract damage.

Oxygen content measurements are more informative than oxygen saturation data, since anemia or carbon monoxide poisoning may seriously decrease oxygen capacity.

Abnormally high carbon dioxide tensions are sometimes seen in patients with respiratory tract damage also and may produce toxic effects.

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