# THE PULMONARY COMPLICATIONS: A CLINICAL DESCRIPTION

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MANY BURNED patients have pulmonary lesions due to thermal or chemical burns of the lung, and an important part of burn management consists in the recognition and proper treatment of these pulmonary burns. This was particularly well emphasized in the Cocoanut Grove cases treated at this hospital, since all seven of the deaths were due to pulmonary complications. In fact, only three of the 39 patients were wholly free from respiratory symptoms, and they had covered their mouths with wet cloths or some article of clothing. A wet handkerchief appeared to have afforded adequate protection in one individual. There was little correlation between the severity of surface burns and the extent of pulmonary damage, and it was, therefore, necessary to watch for pulmonary signs even in those who were only slightly burned.

The first clue to the high incidence of pulmonary burns was afforded by the number who died within the first few minutes after reaching the hospital. They were very cyanotic, comatose or restless, and had severe upper respiratory damage. The surviving patients on arrival showed varying degrees of restlessness or excitement but soon became quiet following medication and removal to the ward. None was very cyanotic at this time; some were cherry-red in color, suggesting carbon monoxide inhalation. Most of them were burned about the mouth and nose, with singed nasal hair and reddening of the nasal mucous membranes. In general, the patients during the first three hours were breathing quietly and superficially, and coughing weakly. Chest examination at first showed distant breath sounds and this was associated with scattered basal râles in many cases. Several of the more severely burned victims were delirious, and two of these were quieted promptly by oxygen inhalation, suggesting that the delirium was due to anoxia aggravated by carbon monoxide poisoning and slowing of the respirations from morphine. Several patients, notably Case 25, soon became dyspneic, and progressed to a critical condition within a few hours.

About three hours after the fire, dyspnea suddenly appeared in others associated with cyanosis, restlessness, and increased râles. Since there was a rapid accumulation of edema in the external burns at this time, it is probable that burned pulmonary areas were undergoing similar changes. Thus, Case 25, who had shown early dyspnea but had remained quiet during the first three hours, became so short of breath that he insisted on getting on his hands and knees to facilitate breathing. An oxygen tent promptly

relieved cyanosis but had no effect on the dyspnea. Complications in this stage were (1) acute dilatation of the stomach in two of the most severely dyspneic patients; (2) a wildly delirious state, apparently due to anoxia; (3) auricular fibrillation in Case 2, eventually relieved by oxygen administration. This period of dyspnea subsided within a few hours.

A more critical period occurred about 24 hours after the fire and continued for the next 36. Dyspnea and cyanosis became much aggravated in certain patients, and râles again spread. This fulminating state was obviously due to edema from burns of the upper air passages, trachea and bronchial tree. Laryngeal examination had demonstrated reddening, edema, and burned areas extending beyond the vocal cords. Because of the critical nature of symptoms at this time, radical therapy appeared indicated and intubations and tracheotomies were done in several instances. Only two of the five treated by tracheotomy survived. In all, seven patients died. Necropsies on three indicated that the lesions had been too widespread to be relieved by these procedures even though additional oxygen was fed by catheter through the tracheal cannula. It is clear that an accurate estimate of the extent of a pulmonary burn cannot be made soon after occurrence, but it would appear that tracheotomy or tracheal intubation is indicated since it affords a chance of reducing the labor of respiration in a weakened patient by facilitating the passage of oxygen to the alveoli.

Whether phosgene or nitrous fumes were present in the smoke, as might be suspected from the delayed edema, apparently must remain a matter for speculation. Several of the patients exhibiting symptoms (Cases 5, 6, and 19) were exposed only to fumes and heat, and those for but a short period, and did not come in contact with flame at all. Case 13, on the other hand, had severe face and nasal burns, with denudation of her lower turbinate bones, but developed only slight lung complications. Professor Alan Moritz found carbon monoxide in a high proportion of those dead on arrival at the hospital, but no methemoglobin or porphyrin in the blood of the one patient tested. He is of the opinion that oxides of nitrogen were in the smoke, and it is noteworthy that the pattern of pulmonary reaction in the Cocoanut Grove survivors was not unlike that of the Cleveland Clinic fire.

Following subsidence of the epidemic-like attack of pulmonary edema, the final, subacute stage of pulmonary manifestations set in, of which the pathologic basis was diffuse bronchiolitis. This resulted in (1) obstruction of the air passages, particularly in the bases, sufficient to produce localized lobular collapse; and (2) trapping of air at the apices with acute emphysema. Both gave characteristic physical signs; the percussion note was highly resonant over emphysematous areas, and normal or slightly dull over the small areas of collapse, while the most striking finding was the surprisingly diminished breath sounds over the entire chest, most evident in the lower portions. Bronchial breathing was not heard except in Case 6, and at one time in Case 27. Râles, remarkably few in number were fine and crackling in the first few days and later coarse in character.

The absence of bronchial breathing suggested that the bronchi were plugged with secretion which prevented bronchial breath sounds from being transmitted to the periphery. This interpretation was borne out in many of the patients when the usual physical signs of pulmonary collapse disappeared after the coughing up of mucus and when similar signs appeared elsewhere. Thus, Case 20 had typical signs of collapse of the left lower lobe on the second day which disappeared on the next day after she coughed up a plug of pinkish-black material the shape of a medium-sized bronchus. On the fourth day, massive collapse of the other side was found to be present. The next day both lungs had cleared considerably, followed by appearance in the sputum of numerous brown plugs of mucus. A similar sequence occurred in Case 27 (who died on the third day). Postmorten examination confirmed these observations. The sputum raised by all who had lung involvement was of the same character, consisting at first of a heavy, tenacious mucus, later of a lighter, more frothy material. All of it was heavily stained with black particles resembling soot.

The areas of acute emphysema showed the same migratory tendency as those of atelectasis and lobular collapse. Here, too, auscultation showed that the breath sounds were almost absent and râles were rare but the emphysematous areas, which predominated in the upper lobes, were distinguished by their extreme resonance to percussion. Emphysema was also seen roentgenologically and at necropsy. The areas of collapse and of emphysema appeared to have a common origin in obstruction of the bronchioles—complete obstruction causing collapse, and partial obstruction producing emphysema.

Although breath sounds could barely be heard, the patients appeared to be breathing easily and with normal depth. As one might expect in this type of lesion the vital capacity of the lungs of most of the survivors (previously healthy adults) was markedly reduced, frequently to levels of only 800 to 1300 cc. The vital capacities in 19 patients averaged 73 per cent of the theoretical normal on the seventh day after the fire, with extremes of 25 and 120 per cent. Those which were diminished returned to normal only slowly. This diminished vital capacity is probably not unlike that found in cardiac decompensation. The lack of lung elasticity due to burns and edema probably accounted for it as much as the lung collapse and emphysematous areas. It doubtless was a factor in the anoxia experienced by these patients. The vital capacity test represents our best quantitative index of the severity of lung burns, even though it obviously cannot be used in the first few days after an accident.

Three patients, not previously afflicted with the disease, developed typical asthma. This was clearly present on the second day and persisted for over a week; then it disappeared, for the remainder of the hospital stay. They were relieved by steam inhalations and by adrenalin or by aminophylline several times, though the response was not invariable. This type of reaction can be described best by the reports of Cases 5 and 19. Other

patients with less obvious symptoms, but with asthma-like breathing, also obtained relief from the use of aminophylline. The response to these drugs indicated that the asthma was due in part to a muscular constriction of the bronchi, which could be relaxed, for it appears quite unlikely that the drugs would materially reduce the mucosal edema of the bronchioles. It is very interesting that intense asthmatic breathing could be precipitated by the bronchiolar lesions due to inhalation burns.

The lung complications encountered may be classified into four degrees of severity:

Grade 1: (9 patients.) This group showed minimal abnormal physical signs manifested by râles. There was no significant diminution of vital capacity, where it was estimated.

Grade 2: (8 patients.) The second group showed râles and emphysema; there was marked diminution in breath sounds together with roentgenologic evidence of trapping of air. There was slight diminution of vital capacity in all. (This grade is illustrated by Case 36.)

Grade 3: (7 patients.) The third degree of severity added persistent atelectasis, attributed to edema sufficiently marked to obstruct the passage of air into certain areas of the lungs during either phase of respiration. Vital capacities were reduced in varying degrees between the limits of 25 and 83 per cent of the theoretic normal. Cases 5, 6 and 19, who showed special features of interest, are described as examples.

Grade 4: (12 patients.) This group included the patients with the most severe degree of injury. Seven died (Case 27 is discussed). Five survived (Case 20 is described).

## CASE REPORTS

**Case 36.**—This case illustrates Group 2 of lung complications—râles and emphysema. The patient retained consciousness throughout. On admission, he was cyanotic and there were burns of the mouth and nose. For 48 hours he was dyspneic. There were markedly diminished breath sounds over the right lower lobe and râles at both lung bases. He could phonate only in whispers. The roentgenogram showed a small area of atelectasis on the fourth day and later also evidence of some trapped air. After 12 days, his chest was clear except for occasional râles, but he was still raising a half ounce of sputum. His vital capacity, which was 58 per cent of normal six days after the fire, had risen to 79 per cent three days later. On discharge, after seven weeks in the hospital, there was no evidence by physical examination or roentgenogram of residual damage to the lungs.

**Case 5.**—This case illustrates Group 3 of the degrees of lung complications—râles, emphysema, and persistent atelectasis plus asthma. This man, whose mother has asthma, had never had any previous manifestation of allergy beyond slight exertional dyspnea with chest colds; he had a heavy cold at the time of the fire.

He was admitted unconscious and cyanotic, with burns of the lips, mouth, nares, tongue, and trachea. There were râles in both chests. Twenty-seven hours after admission, his breathing became much more difficult, and was relieved following intravenous aminophylline. This was interpreted as bronchial spasm superimposed upon bronchial edema. The asthmatic breathing persisted for four days, and râles, with suppression of breath sounds, for eight days in all. Roentgenograms showed air trapping and atelectasis. His vital capacity rose from 54 per cent of normal on the sixth day to 69 per cent three days later.

This patient was seen one month after the fire, at which time there was complete freedom from symptoms together with a normal roentgenogram.

**Case 6.**—A female, age 16, well except for previous sinusitis. In the fire she lost consciousness, and awakened in the hospital, nauseated and vomiting. On entrance, no râles were heard in the lungs, though the nares, mouth, and pharynx showed evidence of the effect of the heat. She was not otherwise much burned, but was in mild shock, the systolic pressure falling from 120 to 80 mm. Hg., which was improved by a plasma transfusion. Within three hours, she developed hoarseness, with moist râles throughout both chests, signs of consolidation in left lower lobe, and showed marked cyanosis and an elevation of respirations to 40 per minute. She was much improved in color and breathing by oxygen, by Boothby mask, after aminophylline had failed to relieve her. However, she became more restless, raised tenacious mucus, and 12 hours after the fire she was put in an oxygen tent because of her poor responsiveness, cyanosis, and increasing respiratory difficulty. She had to breathe oxygen-rich mixtures nearly continuously until the evening of the fifth day.

Prior to the third day, physical signs indicated transient blocking of the large bronchi, but thereafter there remained a constant block of the lower portion of the left lower lobe where constant dullness to percussion and bronchial breath sounds were heard (unique in this series). The rest of the chest examination showed constant and marked diminution of breath sounds with variable and migrant areas of râles, dullness, and hyperresonance though collapse of the right lower and emphysema of right upper lobe were usually found. She could exert no force on expiration or cough and could not dislodge the mucus plugs in her bronchi.

She gradually improved until the eleventh day, when it appeared safe to tip head and chest to drain the lung. This had a remarkably good and rapid effect, and though she raised no sputum the lung collapse in both lower lobes disappeared; aeration thereafter was much improved and the diaphragms descended well for the first time. Her vital capacity rose immediately from 0.8 liters (25 per cent theoretic normal) to 1.2 liters (37 per cent of normal), and by the end of the third week this had returned to 3.0 liters, or 90 per cent of normal. Her convalescence was uneventful.

**Case 19.**—A male, age 42, with no previous history of asthma or chronic cough, was largely a problem of inhalation burns, with minor burns particularly evident in the nose and mouth. On entrance, he had râles in both lung bases followed, six hours after the fire, by signs of blocking of his right lower lobe. Six hours later he had asthmatic breathing and moist râles and expiratory stridor, mostly in right upper lobe. After 29 hours, he became very ill, with intense dyspnea, evidence of obstructed bronchus of right lower lobe, and universal râles; and later, signs of emphysema in right upper lobe. Tracheotomy was considered but not done. Asthmatic breathing from the beginning was relieved by aminophylline. On the third day, he could not phonate audibly and local examination disclosed diffuse swelling and redness throughout the nasopharynx, with chest signs of diffuse emphysema sufficient to depress the diaphragm and obscure the heart. The breathing was typically asthmatic and the clinical picture consistent with asthma of long standing. The patient was very ill, required repeated stays in an oxygen tent, and was frequently relieved by aminophylline and codeine as well as by adrenalin injections. This continued with gradual improvement, with increasing amounts of mucoid sputum (containing 1.8 Gm.% protein) and lessening of asthmatic breathing and physical chest signs. The vital capacity was greatly reduced by the expiratory push-test (see Schatzki) and after ten days was 1.4 liters, only 34 per cent of the theoretic normal and 74 per cent of normal by the nineteenth day. At the end of a month the lungs still sounded far from normal. There was no dullness to percussion, but breath sounds were very distant and faint over the whole back, most marked down the right lower lobe. Many coarse bronchial râles were heard over the left lower lobe, particularly after coughing. Vital capacity was 2.6 liters, or 65 per cent of

theoretic normal. The patient felt weak but well, complained of no asthma or shortness of breath, and only of the persistent cough and sputum.

**Case 27.**—A female, age 18, was very seriously burned. On admission, her color was slightly cherry-red, and she was wildly delirious. After an hour she developed respiratory failure; she was given artificial respiration followed by oxygen and carbon dioxide; an airway was inserted and she was then put into an oxygen tent. One hour later her lungs were wet, her systolic blood pressure varied from zero to 120; she was unresponsive, and six hours later she was still unresponsive. Her lungs were full of râles. Given aminophylline, she awakened and coughed, and repeated tracheal aspiration produced a 7 cm. bronchial cast streaked with black pigment. Roentgenograms 11 hours after admission, showed partial collapse of the right upper lobe and probably of the middle lobe. There was also acute dilatation of the stomach and esophagus. The next roentgenogram, after a four-hour interval, during which a Levine tube had been used, showed that the dilatation of the stomach had decreased and the esophagus was no longer dilated.

The next morning her breathing was labored, almost Cheyne-Stokes in character, and a patch of bronchial breathing was heard at the base of the right lung. An electrocardiogram showed sinus tachycardia, with a rate of 150; the tracing was otherwise within normal limits.

She continued to have a good airway for a day, then developed gurgling noises in her trachea. Suction with an intratracheal tube evacuated a very thick mucus, but it could not all be removed. The patient became cyanotic and died 62 hours after admission. An autopsy was performed.

**Case 20.**—This case represents the most severe grade of lung complications encountered among the survivors. This patient, a female, was one of those who were badly burned, and her dressings made examination of the chest unsatisfactory. She was cyanotic and had râles on admission, but did not begin to cough until about 24 hours later. During the next few hours she sounded like an asthmatic, until she coughed up a plug of pinkish-black material, which suggested a bronchial cast. On the third day following the fire, she developed a massive collapse of the right lower lobe. Laryngoscopy showed definite edema of the cords and a tracheotomy was performed. That evening she was unconscious and had a respiratory rate of 38 in her oxygen tent. The lung collapse diminished in the course of about 24 hours, though localized areas of atelectasis remained for 20 days. During the period of reexpansion she produced many brown mucoid plugs.

She improved gradually. The tracheotomy tube was removed after 29 days. Scattered wheezes, clearing after deep breathing, were heard for some days more. A roentgenogram, in the eighth week after the fire, showed no evidence of any abnormality.

The sequence here was partial bronchial obstruction due to a plug, associated with clinical asthma. Subsequent complete plugging produced massive collapse. The patient survived this acute phase and the bronchial edema subsided. As laryngoscopy showed only edema and no third-degree burn of the cords, it is improbable that she had a deep burn of the bronchial mucosa. Seven weeks after the fire her lungs were apparently normal, as determined both by physical and roentgenologic examination.

Therapy for severe lung complications of burns is difficult and in a sense inadequate, but it is important that it be carried out vigorously and with a clear conception of the underlying pathology. At first, the problems are essentially those of getting sufficient oxygen to the lungs, of reducing edema of the mucous membranes, and of avoiding pulmonary infection. Oxygen in high concentration is the obvious emergency therapy. Patients

receiving morphine should be watched closely. Although intubation and tracheotomy were not highly successful in our cases, we believe that they fulfill a definite function in relieving labored breathing and in facilitating the delivery of oxygen, and should be resorted to in patients with acute cyanosis and in those with severe upper respiratory lesions. Intravenous saline solution is contraindicated since it will increase edema and exudate in the bronchial tree; plasma transfusions will apparently not do this if given in moderate amounts, and obviously they are the essential therapy after burns. It should be remembered that loss of fluid from burned areas of skin and superficial edema can occur with little harm, while similar occurrences within the respiratory tract may be dangerous.

It is, of course, most important to avoid infection. None of the patients in this group developed pneumonia or pulmonary abscess, and this was in all probability due to their isolation on one floor and the precautions taken to avoid cross infections by the continuous use of masks and gowns, and by scrupulous cleanliness. The early use of sulfadiazine was probably also an important factor.

In the later subacute phase, the main problem was to lessen the viscosity of the sputum and to liberate blocked bronchi. A moist atmosphere, produced by means of steam kettles and by liberating steam from the room heating units, gave considerable comfort and improved breathing. Aminophylline appeared helpful, not only in the asthmatics but also in the other dyspneic patients. To loosen secretions, ammonium chloride appeared to be of some value. Gravity drainage during convalescence had a dramatic effect in liberating one collapsed lung (Case 6), and should be used when the patient is well enough to endure it.

## CONCLUSIONS

Of the numerous lessons to be learned from these lung burns, the following should be emphasized: (1) That covering the mouth with a wet cloth may afford complete protection against pulmonary burns; (2) that in most patients the degree of inhalation burn was by no means ascertainable directly after the fire, and the extreme edema, which occurred later could not be predicted; (3) the resuscitation of patients in acute attacks of edema was difficult and unsatisfactory, and these acute attacks must be watched for with great vigilance, even in patients with minimal surface burns.

The pulmonary complications were bizarre and characterized by extreme variability, with areas of lung collapse and emphysema, which were often quite transient and migratory. As the injury to the bronchioli healed, these signs disappeared and the lungs sounded as though no permanent damage had occurred. Roentgenologic examination confirmed this, but only time will tell whether bronchial scars will constrict and produce bronchiectasis in the future.