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Heat Injuries to the Respiratory System

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Summary. A steam-tube of the main boiler exploded on a ship lying in the harbour of Hamburg. The steam temperature was 283° C. Cutaneous and severe inhalational scalding occured in the 27 fatalities, the men dying after different intervals.

This paper deals with the pathological findings in the respiratory passages and the lung, describing the topographical extent of direct thermal injury and the temporal course of tissue reactions.

In cases of instantaneous death coagulation necrosis of the tracheal and bronchial wall was found to extend to alveolar ducts in central parts of the lung. The lung parenchyma showed marked congestion, alveolar edema and desquamation of alveolar epithelial cells. Death occured due to acute pulmonary dysfunction and shock.

Lethal complications following the period of primary shock consisted of fulminant confluent bronchopneumonia, the hyaline membrane syndrome or the onset of desquamative interstitial pneumonia. These changes rendered it difficult to evaluate the effects of the heavy cutaneous scalding on the pathological course of inhalational injuries in those surviving for longer periods.

Key words: Explosion – Burns – Pathohistology – Air passages – Lung.

Experimental heat injuries to the respiratory system have been reported, using different modes of heat application and measuring functional disturbances together with temperature gradients in the upper respiratory pathways (Moritz et al., 1945; Aviado and Schmidt, 1952). Pathological studies were usually performed after short periods of survival. Human pathological investigations are usually limited to casual fatalities or are derived from autopsy statistics, the latter being heterogenous with regard to the type of heat exposure (Mallory and

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Brickley, 1943; Di Vincenti et al., 1971). This report deals with the pathological findings in 27 fatalities that occured after different survival periods following the explosion of a ship's boiler.

Materials and Methods

Technical Conditions. A steam-tube of the main boiler exploded on January 6th, 1976, on the container vessel "Anders Maersk" lying in the dockyard. The boiler room was $28 \times 30 \times 38$ m of size. From a recording-instrument the pressure was known to have been 67 bar at the time of accident, and this pressure is equivalent to a steam temperature of 283° C. The steam was known to have flowed for a period of 5 min. Assuming spheroidal extension of steam, a temperature of about 100° C should have been reached in the sphere of the room. At the time of the disaster about 40 people were working in the boiler room but because of the high temperature rescue operations could not start until 20 min had passed.

Pathologic Procedures. 12 men were found dead, 15 others died within the following days (Table 1). All victims were autopsied as quickly as possible at the Institute for Forensic Medicine, Hamburg. Macroscopical and histological findings in the respiratory passages and the lung were studied in particular detail. Tissue was fixed in 10% buffered formaldehyde, embedded in paraffin, sectioned and stained with HE, PTAH, Mallory, Goldner, Elastica, van Gieson, Prussian blue and PAS. Fat was stained with Sudan III.

Table 1. General findings of the fatalities ($n=27$) of the scalding disaster

Case no.	Age	Cutaneous scalding	Survival period	Histological findings	Cause of death
1	25	95%	1 min	Coagulation necrosis of bronchial mucosa	Cerebral trauma
2–12	23–53 ($\bar{x}=35$)	90–95%	20 min	Hyperaemia, oedema, and haemorrhages of airway walls, alveoli, and interstitial spaces. Platelet aggregations. Desquamation of pneumocytes	Fulminating shock syndrome
13–16	28–53 ($\bar{x}=38$)	60–90%	$1^1/_4$–4 h	Early stages of inflammation. Microthrombosis. Early formation of hyaline membranes	Shock lung, respiratory insufficiency
17–23	23–48 ($\bar{x}=35$)	50–90%	13–42 h	Necrotizing inflammation of airways. Confluent bronchopneumonia	Confluencing bronchopneumonia
24	35	60%	63 h	Hyaline membranes. Bronchitis, tracheitis	Hyaline membranes
25–26	37–57 ($\bar{x}=47$)	60–90%	76–106 h	Interstitial oedema, mononuclear infiltration, early stage of desquamative alveolitis	Interstitial oedema, desquamative alveolitis
27	44	30%	27 days	Bronchopneumonia	Pneumonia

Results

Macroscopical Findings

The most important data on survival times, extent of external and inhalational scalds and causes of death are given in Table 1. In case 1 death was caused by direct injury from a burst tube. As a result the medulla oblongata was almost completely severed; this case will be designated as the control. Apart from second or third degree scalding of the body surface, similar lesions of the respiratory system were found in all autopsies. Case 27 suffered less severe cutaneous and inhalational scalding and died, after 27 days, from secondary pneumonia.

a) Respiratory Passages

Direct thermal injuries were striking in cases of instantaneous death: The epithelium and the adjacent luminal mucosa of the upper airways exhibited a greyish colour. The epithelial layer was partly detached as strips, filling the lumen of smaller bronchi. Macroscopically the airway damage extended to medium sized bronchi in the acute cases. In those surviving longer heat damage, as estimated from the extent of the desquamation, reached the segmental bronchi in intervals up to 36 h and the lobar bronchi in longer lasting survivors. Inflammatory changes in the bronchial tree, including the trachea, were observed after as little as $1^1/_2$ h of survival time.

b) Lung Parenchyma

About 50% of all cases showed spotted subpleural haemorrhages. Focal haemorrhages were also observed in the parenchyma. Apart from case 1 congestion, oedema, and compensatory emphysema were common findings. Macroscopically early bronchopneumonia occured after 4 h survival time, becoming confluent within one to two days.

Histological Findings

a) Respiratory Passages

1. Direct Thermal Injuries were evident in different structures. The coagulated epithelial cells show vacuolar swelling of the cytoplasm giving the appearance of goblet cells (Fig. 1). The cells thus show elongation, as do the nuclei, the swollen cells sometimes narrowing the lumen of the bronchioles. Epithelial changes are usually accompanied by heat damage to the adjacent mucosal layers, consisting of swelling and roughening of the basement membrane and homogenization of the submembranous layer. There is heat fragmentation of red cells

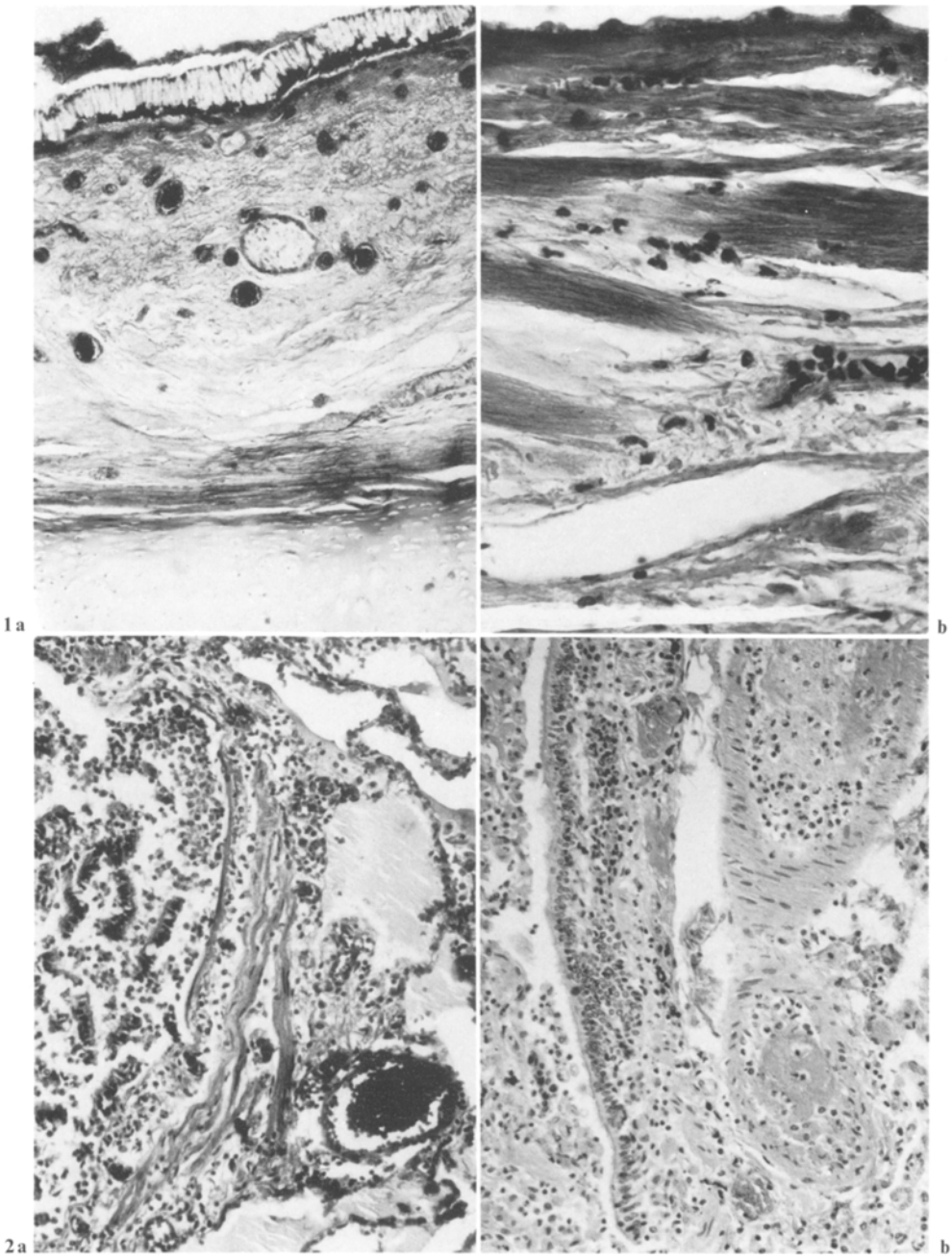


Fig. 1. a Acute death. Heat-induced vacuolar swelling of tracheal epithelium, the swollen cells being longitudinally extended. Structural density of subepithelial mucosa together with fading of the blood vessels due to heat coagulation. Deeper mucosa showing hyperaemia, oedema and dilatation of lymphatic spaces. Mallory, ×480. **b** 2 h survival. Tracheal epithelium has been desquamated (top). Apparently enhanced density of superficial mucosa due to heat coagulation. Early polymorph infiltrates and leucocytosis together with oedema in the deeper bronchial mucosa. HE, ×480

Fig. 2. a 4 h survival. Purulent bronchiolitis and early stage of bronchopneumonia. The epithelium has been lifted off the basement membrane and is lying in the bronchiolar lumen. HE, ×180, **b** 13 h survival. Purulent and focally necrotizing bronchiolitis together with bronchopneumonia. Thrombosis and leucocytosis of accompanying blood vessels. HE, ×180

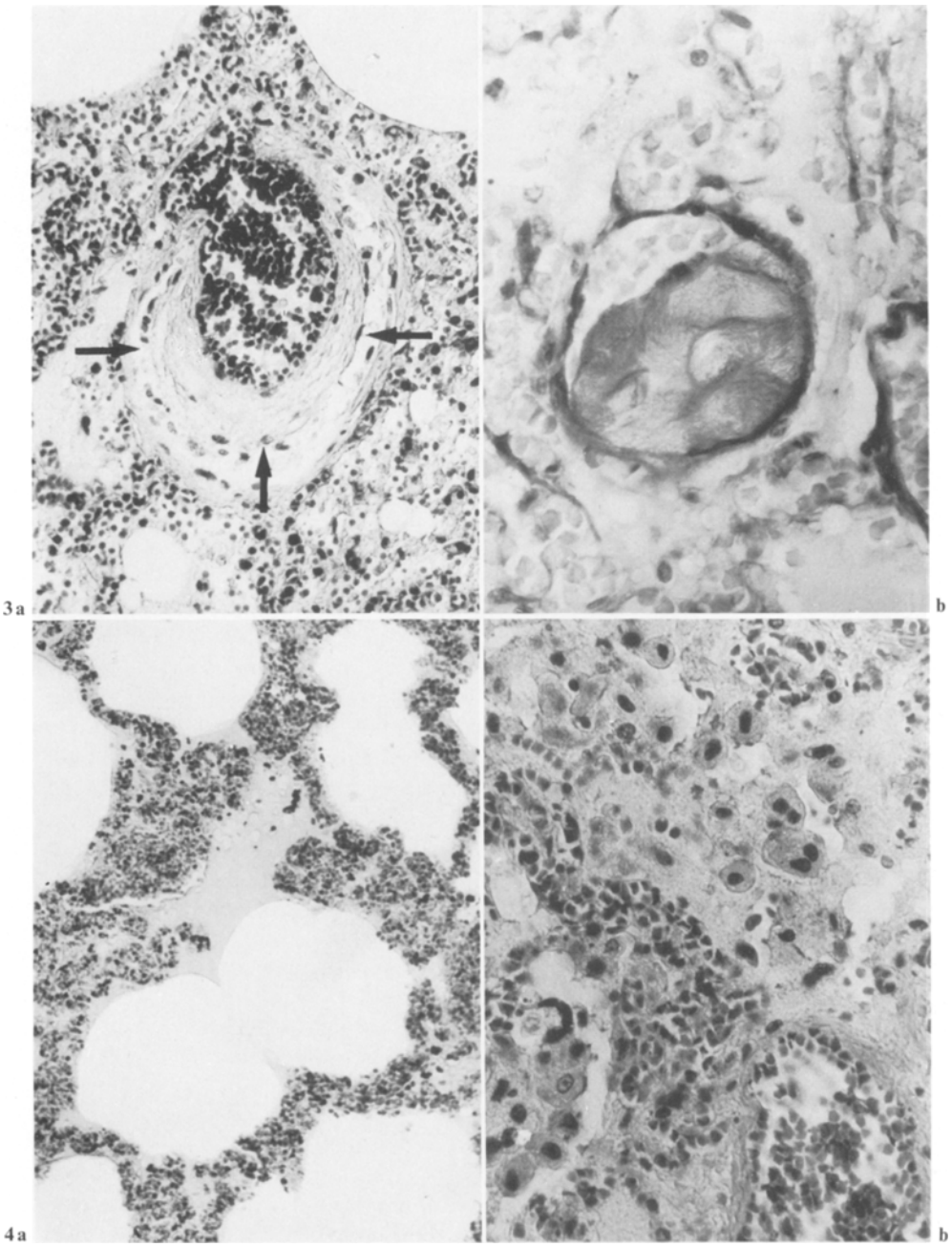
and broadening of collagen fibres. The peripheral extension of the heat damage is roughly the same as estimated macroscopically. The transitional zone to the undamaged mucosa in peripheral bronchioles shows multiple forms of incomplete and discontinuous epithelial damages. In survivors up to 2 h the heat coagulation injuries extend to the alveolar ducts in the central zone and to the smallest bronchi and terminal bronchioles in the cortex. It is estimated, in those surviving two to four days, that epithelial damage extends to the lobar bronchi only.

2. Inflammatory Reactions. The earliest reaction occurs in the mucosa beneath the coagulated layer and consists of hyperaemia with some capillary haemorrhage and marked interstitial oedema with dilatation of the lymphatic vessels (Fig. 1). These changes are also seen in the acute cases and they are absent or minimal in the control. In those surviving longer than 20 h larger cuff-like haemorrhages occur in the deeper mucosal layers and even in the peribronchial and peritracheal tissues. Intravascular and extravascular deposits of fibrillar or amorphous fibrin are not found before 4 h of survival. In the 20 h to 36 h survivors many of the mucosal vessels are occluded by fibrin-rich thrombi (Fig. 2b). The necrotic mucosal surface is then covered by a fibrinous membrane sometimes incorporating the fragmented basement membrane.

Signs of cellular inflammation are observed at first in the $1^1/_4$ h case. These consist of peribronchiolar intravascular leucocytosis, margination, and sometimes diapedesis. The 2 h survivor presents some polymorph leucocyte infiltration in the oedematous bronchial and tracheal mucosa (Fig. 1) and dense inflammatory infiltration of the bronchiolar wall. Necrotizing inflammation of terminal bronchioles is observed in those surviving longer than 13 h (Fig. 2) and is accompanied by bronchopneumonia. In early cases (up to 22 h of survival) the central lung regions are selectively affected by bronchopneumonia. In longer survivors the distribution of the bronchopneumonic foci is disseminated, and bronchopneumonia becomes confluent. It is then accompanied by extensive microthrombosis and focal necrosis. The bronchial and tracheal mucosa show dense cellular infiltration in the 13 h case; this change is followed by focal ulceration and fibrinous exudation or subtotal mucosal necrosis in the 24 h and 36 h cases (Fig. 6).

b) Lung Parenchyma

1. Acute Changes. The most striking feature in those surviving up to 20 min consists of extensive congestion of the alveolar capillaries. The alveoli are filled with oedema fluid containing red cells, fibrin, and masses of desquamated alveolar epithelial cells, the latter sometimes even filling the lumen of bronchioles and small bronchi (Fig. 4b). Accompanying these initial changes are varying degrees of oedema of the interlobular septa, dilatation of lymphatic vessels, and multiple forms of microthrombosis together with changes in the vascular walls (Fig. 3). Microthrombi usually consist of platelet aggregations often combined with leucocytes or with fragments of red cells and some fibrin in the small branches of



3a b

4a b

Fig. 3a and b. Acute death. **a** Oedema of an arteriolar wall together with microdissection (arrowed). Some red cells lying in the fissure. PTAH, ×240. **b** Fibrinous insudation of the intima of a small vessel leading to pad-like narrowing of the lumen. Van Gieson, ×480

Fig. 4a and b. Acute death. **a** Extensive congestion of alveolar capillaries together with microhaemorrhages and alveolar oedema. PTAH, ×730. **b** Multiple desquamated alveolar cells lying in the lumen of a terminal bronchiole. Note signs of phagocytosis and double nucleated cells. PTAH, ×360

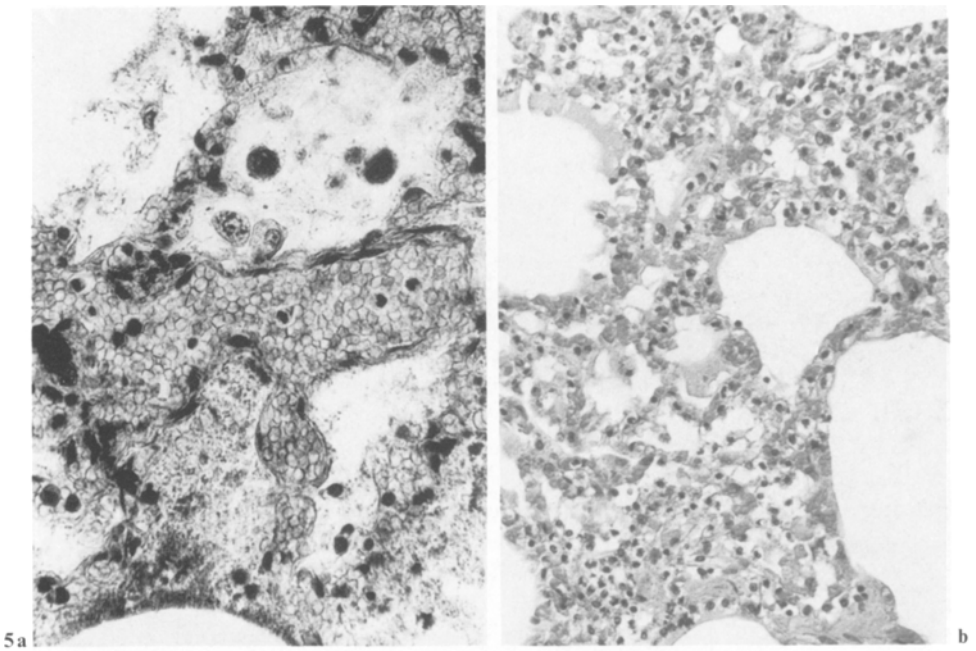
the pulmonary arteries. The walls of the arterioles show oedematous thickening and focal microdissection. Furthermore there is concentric and excentric intimal oedema sometimes with fibrinous insudation. The capillary endothelial cells show swollen appearance. All acute changes are more marked in the hilar and medullary zones than in the cortex. They are either absent or slight in the control.

2. Fibrin Formation. In longer survival periods increasing degrees of fibrin formation are observed. The intraalveolar oedema gradually diminishes and becomes mixed diffusely with fibrin forming hyaline membranes (Fig. 7) or with clumps and networks of fibrin. Fibrin phagocytosis of the desquamated alveolar cells is seen (Fig. 5). These changes are associated with extensive intravascular fibrin formation with small globules and clumps with subsequent capillary occlusion. In the 2 and 4 h survivors there are larger thrombi usually occluding the lumen of arterioles and unfrequently of smaller arteries. The areas of pneumonia are separated by multiple tyre-shaped intracapillary fibrin thrombi associated with alveolar wall necrosis (Fig. 6). The intravascular coagulation syndrome begins during the first hour of survival and is marked in 2 and 3 days survivors.

3. Interstitial Changes. From survival periods of about 4 h alveolar interstitial oedema develops progressively; in the early stages this gives the interstitial spaces a watery appearance but these areas stain more intensely during the next few days. The ground substance then stains strongly with PAS, focally with alcian blue, collagen fibres show a swollen appearance and disintegration (Fig. 8). Reticulin fibres also undergo fragmentation and disintegration. In the 4 day survivors these changes have caused enormous thickening of the alveolar septa which show mild mononuclear infiltration and reduced capillary blood content, this latter change being due in part to thickening of vascular walls and narrowing of the lumen of smaller arterial branches.

4. Inflammatory Changes. Early polymorph leucocytosis of the alveolar capillaries is found in the $1^1/_4$ h survivor (Fig. 5b). It is accompanied by slight intraalveolar leucocytic infiltration without topographic relation to the injured bronchi, the whole picture resembling the early spreading stage of pneumococcal pneumonia. Subsequent stages show a gradual increase in the pulmonary inflammation up to 4 h. Later on this is overlapped by a rapidly spreading bronchopneumonia, ending fatally within the first two days. Another type of inflammation is associated with extensive interstitial oedema and gradually increases in the 2 to 4 days survivors. Some alveoli become lined by swollen pneumocytes. Alveolar lumina are filled with masses or swollen granular alveolar cells or macrophages staining strongly positive with PAS and negative with lipid or iron dyes. Bi-nuclear cells are frequently observed and some multinucleated forms are present, however, only few of these cells are to be found in the interstitial spaces (Fig. 8). This type of inflammation strongly resembles the initial stages of desquamative interstitial pneumonia (DIP).

5. Patterns of Morphologic Alterations. Fatalities that are thought to be due to shock lung show all the typical changes described above in 1. and 2. and



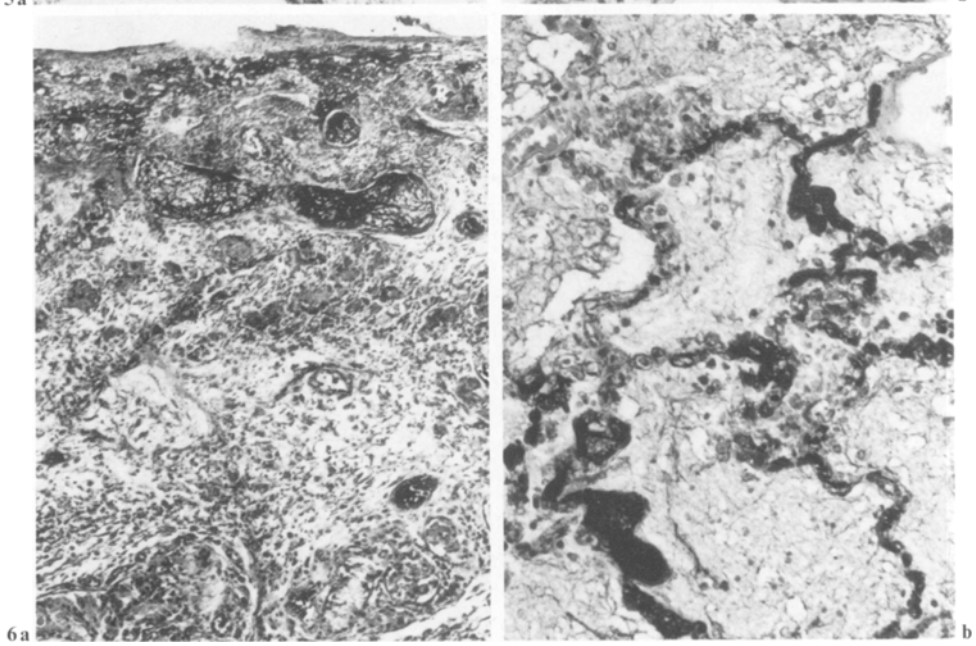


Fig. 5a and b. $1^1/_4$ h survival. **a** Hyperaemia of blood vessels together with extravascular fibrin precipitation forming intraalveolar granular and fibrillar deposits. Note fibrin phagocytosis of alveolar cells. PTAH, ×360. **b** Diffuse leucocytosis of alveolar capillaries and venules. Focally some intraalveolar exudates containing leucocytes (top-right-hand corner). HE, ×180.

Fig. 6a and b. 34 h survival. **a** Ischemic and/or thermal necrosis of tracheal mucosa. Swollen basement membrane at the top. Intravascular fibrin networks. Disappearance or fading of cellular structures. PTAH, ×180. **b** Multiple tyre-shaped intracapillary fibrin thrombi together with intraalveolar fibrin networks. Obvious necrobiosis of the alveolar walls. Section from non-infiltrated part of a lobe showing confluencing pneumonia. PTAH, ×180

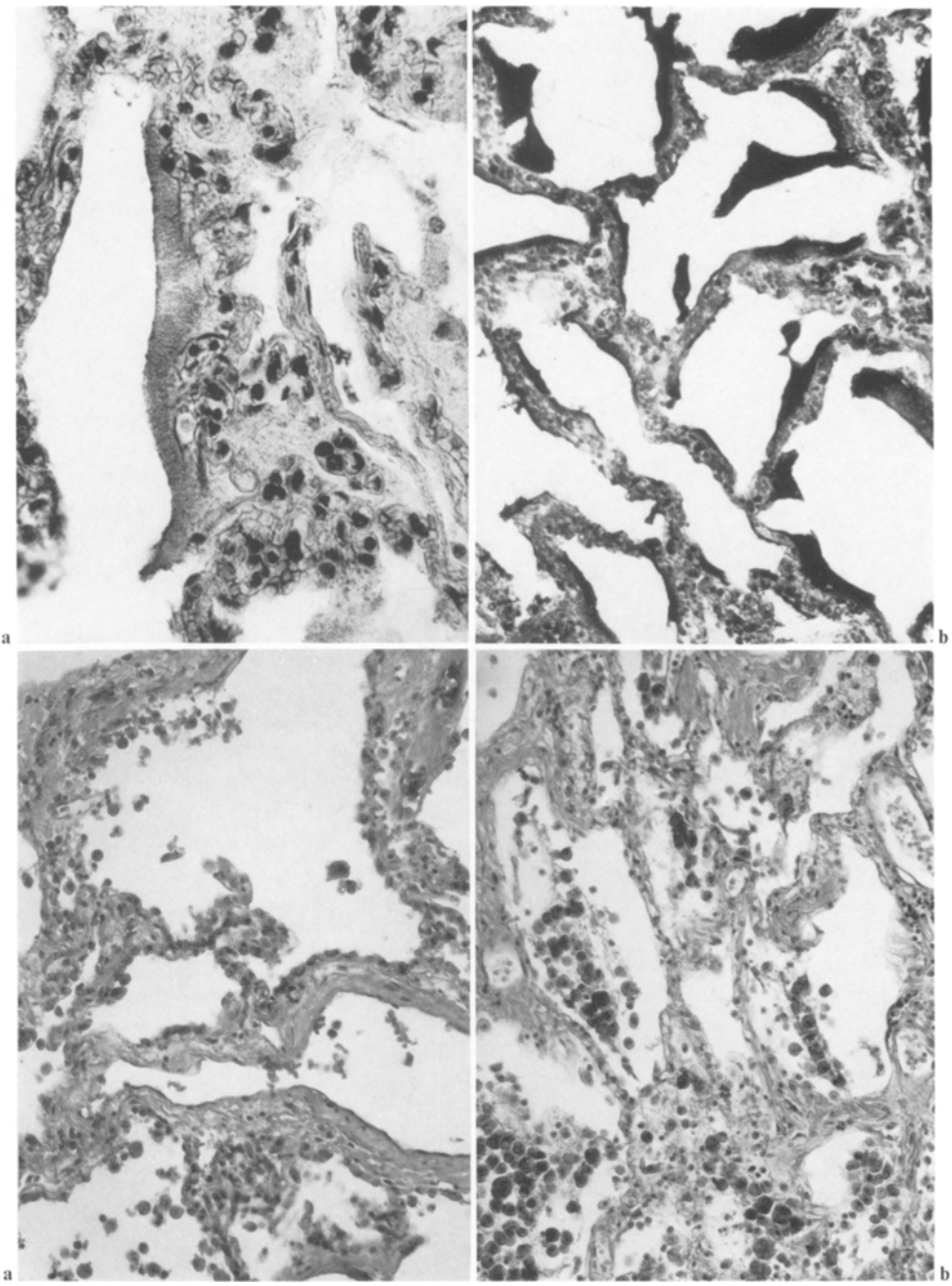


Fig. 7. a $1^1/_2$ h survival. Early formation of fibrin-rich hyaline membrane, other alveolar spaces showing oedema. PTAH. ×480. **b** 63 h survival. Extensive hyaline membranes. PTAH, ×180

Fig. 8 a and b. 4 days survival. **a** Swollen appearance of alveolar septa. Focally interstitial cell infiltrates. Lining of the alveolar lumen with swollen pneumocytes. Desquamated alveolar cells. HE, ×180. **b** Desquamative alveolitis. HE, ×180

the early stages of pulmonary and bronchopneumonary inflammation. Fatalities due to pneumonia are combined with intra- and extravascular fibrin formation and some hyaline membranes. The hyaline membrane syndrome and desquamative alveolitis with interstitial oedema are combined with some polymorph infiltrates. Purulent tracheitis and bronchitis are also frequently observed.

Discussion

It is necessary to clarify how far the striking lesions in the respiratory system represent direct thermal injuries. The effects of burn shock following the excessive cutaneous scaldings also complicate the pathological findings.

Shock-Lung. The histological aspects of shock lungs have been extensively described by Mittermayer et al. (1970) and Remmele and Goebel (1973): general congestion and oedema, microthrombosis, endothelial lesions, hyaline membranes, and dystelectasis. In the development of microthrombosis a phase of cell aggregation is followed by the formation of fibrinoid-rich thrombi (Hardaway, 1966; Lasch et al., 1966; Sandritter and Lasch, 1967). Chronologically microthrombosis and interstitial oedema precede the manifestation of intraalveolar oedema.

Respiratory Alterations After Cutaneous Burns. Specific reactions in the lungs following cutaneous scalds are due to aggregation of thermolabile gammaglobuline (Wolf and Neuhof, 1975). According to Hagedorn et al. (1975) the microcirculatory changes of burn shock in rabbits are as follows: sludging, diapedesis of leucocytes after 40 min, leucocyte sticking, aggregation of thrombocytes after 65 min, haemolysis. Pulmonary changes consisted of endothelial swelling and microthrombosis accompanied by interstitial and perivascular oedema; these changes were observed after 2–3 h. Acute intraalveolar oedema was never found in burned humans by Allgöwer and Siegrist (1957). Pulmonary oedema, congestion, bronchiolotis, bronchitis and tracheitis were considered to be secondary complications of severe cutaneous burns despite the presence of facial burns (Foley et al., 1968; Achauer et al., 1973). This interpretation is supported by experiments with rats: within 48 h after cutaneous burns of the back the animals developed degenerative lesions of the tracheal epithelium (Femenic, 1969).

Respiratory Injuries. Airway lesions caused directly by toxic combustion products and flame inhalation were described by Mallory and Brickley (1943) in six victims of the "cocoanut grove disaster". All of them suffered from disproportionately severe burns of the head. Three cases dead on arrival at the hospital presented with acute pulmonary oedema and a serohaemorrhagic exudation in the upper tracheo-bronchial tree. The cause of death was carbon monoxide poisoning. Three cases with survival periods ranging from 40 to 62 h demonstrated necrotizing and focal membranous laryngitis, located below rather than above the vocal cords, tracheitis and bronchitis, pulmonary oedema, massive haemorrhage into the alveoli, dystelectasis, bronchopneumonia. Similar changes were described by Sochor and Mallory (1963) in a series of 41 autopsies, includ-

ing a number of 28 patients with burns of the head. The frequency of each of the lung lesions varied with the length of survival. A causal relationship between bronchiolitis and the later onset of bronchopneumonia was inferred from the data.

Out of 2297 patients treated for thermal injuries, inhalational injuries occured in 2.9% (Di Vincenti et al., 1971). In these 66 patients, however, the cause of death was directly related to the respiratory tract injury in 73%.

Histological findings after experimental heat inhalation in dogs were described by Förster (1933) who found distended epithelial cells, fragmented elastic fibres and obturation of smaller bronchi by desquamated material. Similar changes were observed by Goldbach (1956) after experimental heat inhalation (600° C) in guinea pigs.

Inhalation of Steam. The sequelae of steam inhalation were tested in dogs by Moritz et al. (1945) and Aviado and Schmidt (1952). According to these authors steam has a heat carrying capacity 4000 times that of hot air. In contrast to inhalation of hot air or flames it can produce direct thermal lesions even in the lower respiratory tract. The histopathological changes range from rapidly fatal obstructive oedema of the glottis, severe thermal tracheitis and destruction of the bronchial mucosa to haemorrhagic oedema of the centrally located alveoli. Temperature peaks recorded by a deep tracheal thermocouple reached more than 90° C (Moritz et al., 1945) and blood temperature rose temporarily to 12.6° C (Aviado and Schmidt, 1952).

From our study the following points emerge:

(1) Despite individual variability in age, sex, and state of health there was remarkable homogeneity in pathological findings.

(2) No evidence of contributory causes of death (carbon monoxide poisoning, smoke inhalation) was found.

(3) The distribution of the heat induced changes could be accurately determined and by examining survivors was studied over a period of time, although in longer term survivors complicating lesions made the pathogenesis of the observed changes less certain.

(4) The thermal energy involved in this incident was high.

Summarizing our pathological findings and the data from literature of direct and secondary thermal damages to the respiratory system we conclude that:

The acute changes are due to a severe lesion of the alveolar-capillary membranes and haemodynamic dysregulation, hypoxia and shock contribute to the lethal course. The histopathological pattern differs from the findings as described following cutaneous burns alone. It is uncertain whether the severe lesion of the basement membranes, combined with alterations of endothelium and alveolar epithelium, is caused by a direct thermal insult or whether it is induced by toxigenic side effects of the inhalational scalding. The theory of direct thermal effects is supported by the fact that the acute lesions are found preferentially in the central parts of the lung. On the other hand, fibrinous insudation and swelling of the intima of small vessels may be better explained by effects of vasotropic material causing pulmonary hypertension.

The acute membrane alterations combined with the coagulation of the tracheal and bronchial mucosa permit different complications to ensue. In cases of instantaneous death or in those who survive for a few hours acute pulmonary dysfunction and shock are the cause of death. Later on three different types of lethal course may develop: A fulminant confluencing bronchopneumonia, the hyaline membrane syndrome, or interstitial oedema combined with desquamative alveolitis, which may be interpreted as onset of desquamative interstitial pneumonia.

Inflammation and necrosis of the tracheal and bronchial mucosa are correlated with the extent of the thermal injury. The topographical extent and grading of the airway lesions is apparently correlated to the survival time and individual differences in health are of minor importance. It may be concluded that the temperature distribution was inhomogenous in the engine room, and that the victims were apparently working at varying distances from the center of the explosion. Direct thermal injury of the airways and lung parenchyma constitute a great hazard to the patient, much more serious than cutaneous burns. For this reason it makes no sense to attempt to prevent thermal injury by means of thermoresistant overalls alone.

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