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memorandum

SPI

TO: The PVC Health Committee	REFERENCE
FROM: J. R. Lawrence, Technical Director	
DATE: February 10, 1981	

Attached is the long awaited report by Dr. W. A. Crosbie on "Respiratory Complications of Burns."

I have only had a brief opportunity to scan this report. It appears to contain some very interesting findings. We should plan to discuss this at a meeting of the Committee which we hope to arrange for sometime during the month of March.

R&S 115783

JRL/dg

cc: F. W. Lichtenberg
J. P. Carroll

Report by:

W A CROSBIE

Subject –

RESPIRATORY COMPLICATIONS OF BURNS

Contents –

The accummulation of knowledge which has led to the theories of how the lung is injured by fire has evolved in a sequential manner over th last century. Hence I will describe in a chronological order important papers which had an effect on the current thoughts at the time, with an interpretation of how each added to the state of previous knowledge. It will become apparent how advances in other branches of medicine such as the treatment of shock, infection and the development of mechanical ventilation of the lungs all influenced the concept f what constituted fire damage to the lung. It will also become apparent how some important observations were ignored or did not receive the attention they deserved until other evidence was found at a later date to support the primary discovery.

The first scientific report on fire victims I found was related to the Vienna Ringtheatre Fire of 1881.[1] In this report it is recorded that high levels of carboxyhaemoglobin (COHb) were present in the blood of some of th victims. Hence the concept of breathing toxic gases as a cause of death in people trapped by fire was recognised. There followed a lapse of interest until the event of the Cleveland Hospital Clinic Fire of 1929.[2] In this conflagration several victims were found to have died in the fire without sustaining any body burns. The fire had destroyed a large number of X-ray films and it was reasoned that poisonous fumes, most likely nitrous oxides, had b en produced from this source which in turn had been breathed by the victims causing their death. Once again interest lapsed until a November night in 1942 when a disastrous fire occurred in a crowded nightclub in Boston called the Coconut Grove. In this holocaust 491 people lost their lives, 114 casualties were taken to the Massachusetts General Hospital and of these 75 were either dead on arrival of died within minutes, only 39 survived long enough to be treated. A special ward was rapidly adapted for the management of the victims and the resources of a major teaching hospital organised to cope with the emergency.[3][4]

A remarkable and invaluable series of papers were subsequently produced from this hospital describing the initial findings, clinical, radiological and

biochemical abnormalities which ocurred in the subjects. For the first time th seriousness of damage to the respiratory tract of burned victims was fully recognised. All of 40 years later there has not been a comparable in depth description of the effects of fire on the lungs. I will use these reports to build up a picture of how the lungs of fire victims may be damaged. This will provide an excellent reference source for the comparison of later data from fire victims. Detailed autopsy reports were also produced hence the findings of today's fire victims can be closely compared with those of 1942. The fire started in the Bamboo Lounge which was in the basement of a single story building. It spread at an incredible rate being described as "flashing across the ceiling up the stairway to the entry hall above". Unfortunately the main exit was through a revolving door with no escape doors alongside. Survivors tell of the dramatic almost explosive onset of the fire, flames and thick smoke rapidly developed. Panic developed in the crowd and although rescue was quickly on the scene and the fire extinguished a dreadful toll was found within a few minutes. The first casualties reached the hospital within 15 minutes of the alarm being raised; of the first 39 patients seen only 3 were entirely free of respiratory symptoms. It was also quickly apparent that there was no correlation between the degree of surface burn and the extent of pulmonary damage. There appeared to be two groups of patients, the first were cyanotic, comatosed or r stless on arrival and died soon after presentation. Carbon monoxide was present in the blood of many of these victims. The second group showed a different pattern, they were not very cyanosed but some were cherry pink in colour, some were restless or excited but soon became quiet following medication and removal to the ward, most showed burns round the mouth. Several were delirious but improved when given oxygen and morphine but one rapidly deteriorated and died. Others began to deteriorate about 3 hours after admission with restlessness, cyanosis and râles in th lungs but oxygen appears to have r lieved their symptoms. About 24 hours after admission dyspnoea, cyanosis and râles returned and this was attributed to fluid in the airways. Laryngeal examination

showed damage extending beyond the vocal cords. Tracheotomies were performed on 5 patients, but only two survived. Most importantly it was noted that several of the patients in this group had been exposed to fumes and heat only and had not come in contact with flame at all, while one patient with severe face and nasal burns developed only slight lung complications.

A third phase of lung complications developed later and was thought to be due to a diffuse bronchiolitis. This was characterised by localised areas of lung collapse, hyperinflation of the chest and fine crackling râles. Several other important clinical findings are described.

The sputum produced by the victims is described as being characteristic, consisting first of heavy tenacious mucus which later became lighter and frothy. It was heavily stained with black particles resembling soot. The breath sounds were often described as being diminished in certain areas and bronchial breathing was absent.

Measurements of the vital capacity showed a marked reduction in many of the victims and the statement is made that this test represents the best quantitative index of the severity of lung burns. It also stated that diminished levels only slowly returned to normal over several weeks. Details of the pathology obtain d in 6 victims of the fire who reached hospital are reported by Drs Mallory and Brinkley (1943)[5]. 3 of the subjects were dead on arrival while the other 3 died 40 - 62 hours after admission.

One of the early statements is very revealing - "Only a small proportion of the victims had suffered extensive enough surface burns to account for death". Carbon monoxide levels of 42 and 50% were found in two of those subjects. This level would be insufficient to be considered lethal but did confirm that CO had been present in the gases breathed by the victims. The CO levels in the other subjects were not quoted. The possibility that the oxides of nitrogen were also present was considered but only slight traces of these gases were present in a sample of gas aspirated from the respiratory tract.

No methaemoglobin r porphyrin was present in the blood of one patient tested. The findings on the 3 subjects who were dead on arrival were very similar too, only one was described in detail. Second or third degree burns were present on the front and back in the areas exposed by the conventional evening gown. The muscles and internal organs were a brilliant cherry pink colour. Partially digested food was present in the trachea and larger bronchi. The lungs were bright pink heavy and voluminous, they weighed 1230 gm and on slight pressure blood stained fluid oozed from the freshly cut surfaces. Microscopically the mucosa of the trachea was markedly oedematous, the epithelial cells had desquamated, no ciliated or goblet cells persisted. The great majority f the alveoli were filled with precipitated eosinophilic material and red blood cells were present in some. The capillaries of the alveolar walls were congested, the interlobular septa widened and oedematous and the lymphatics dilated. The small bronchi showed desquamation of epithelium but no necrosis or inflammatory reaction. The picture was one of a diffuse pulmonary oedema but note "a high protein content of the oedema fluid was evidenced by the homogeneous colloid-like precipitation in the alveolar lumina and the presence of red blood cells confirmed the existence of significant capillary damage".

It was remarked that inhalation of flames into the respiratory passage was unlikely because of the absence of any eschar formation and the basement membrane remained intact. The aspiration of vomit also was not responsible since similar changes were found in other cases in which no evidence of such aspiration was present.

The three cases who died after arriving at the hospital were described in great detail. One patient died 40 hours after admission. At autopsy 2nd and 3rd degree burns were present in the head and neck regions. There was evidence of flame damage to the inner nose, the larynx was almost completely occluded by oedema and a fibrin purulent exudate was present. Below the larynx an extensive greenish black membrane containing charred material lined the trachea for a distance of 2.5 cm, the remainder of the tracheal mucosa showing ulceration and exudation.

The lungs were heavy and all bronchi down to 3 mm in diameter showed intense congestion ov rlaid by a greenish fibrinous membrane. Occlusive bronchial casts were present in some of the lumens. The lung parenchyma contained moderate amounts of pinkish fluid, vascular engorgement with polymorphonuclear infiltration was invariable. Colonies of micrococci were present in the cytoplasm of some of the polymorphonuclear cells. The picture was essentially of a necrotising membranous inflammatory process with pulmonary oedema in the lung tissue.

The second case died 52 hours after admission. Again there was evidence of serious burns in the head region. The larynx was congested and swollen and a fibrinous exudate containing black charred particles was present in the trachea. The lungs were heavy, moderate amounts of fluid could be expressed with pressur . Microscopically the epithelial layer of the bronchial tree was necrotic but th basement membrane appeared intact. The alveolar walls were intact but capillaries congested and their lumen was filled with colloid-like oedema. The picture was overall very similar to the first case.

The third victim died 62 hours after admission. More extensive body burns of head and trunk were present but the larynx and trachea showed only intense congestion without visible necrosis. The lungs were heavy but areas of atelectasis were present. Microscopically a pseudodiphtheritic membrane formation was present in the small bronchial radicles, a superficial necrotizing process was present on the surface of the mucosa but did not extend below the basement membrane. The major difference from the previous two cases was the presence of a brilliant acidophilic fibrinoid membrane in the respiratory bronchioles and rarely in the alveoli. The areas of infarction were lobular in size and some alveolar walls showed signs of early necrosis. Evidence of pneumonio foci were seen with organisms in pairs and short chains but always intracellular. This subject showed a membranous bronchitis extending far into the p riphery with areas of atelectasis and leukocytic infiltration in congested lungs.

In summary several important findings were noted:-

All six victims had significant burns of the head and hands. The cases dead on arrival showed significantly raised carboxyhaemoglobin levels indicating that they had breathed toxic gases. This group had a non-necrotizing tracheitis and bronchitis with acute pulmonary oedema. It is suggested that these findings indicated that they had inhaled an irritating agent either physical or chemical. The second group who survived for a time after reaching hospital showed necrotizing laryngitis with formation of a pseudodiphtheritic membrane and diffuse membranous bronchitis which extended to the smallest bronchioles. The pulmonary lesions varied but evidence of atelectases, early bronchopneumonia, multiple small emboli, lung haemorrhage and pulmonary oedema was recorded. No mention was made of any carboxyhaemoglobin levels but again it was suggested that there was evidence of the changes produced by inhaling toxic substances. The changes in the second group could be due to the increased time of survival of that group. Fortunately the evidence of photographs and thorough descriptions is available for us to use nearly 40 years later.

The events of this catastrophe so well recorded stand as a landmark on the subject of victims of fire. The effects on the lungs are clearly described but it has taken a long time for the message to be universally recognised. Several decades have passed before the hazards of being trapped by fire and dying from lung damage rather than direct effects of body burns has become accepted as a real danger.

It is an irony that the next important advance in this field came about because of war.

The war in the Pacific required the recapture of heavily fortified islands in which the defenders were entrenched in concrete bunkers. Neutralizing these bunkers by the use of high explosives proved difficult. The use of flame throwers was found to be the answer. It was then reported that when a bunker

burns. Was this due to heat or toxic gases - or both ? Two teams of investigators b gan to tackle this problem.

One team led by Dr Alan R Moritz of Harvard University set up experiments to measure the temperature and gas concentration in model bunkers which could be either well ventilated or poorly ventilated.

Another team of pathologists from the Canadian Mobile Chemical Warfare Laboratories set up a similar series of experiments in Alberta. A conflict developed between the two groups. Dr Moritz found that using a gasoline fire inside a poorly ventilated bunker that the temperature rose quickly, the oxygen concentration dropped to 14%, and the fire went out. The CO concentration rarely exceeded 1% and the CO_2 4%. In a well ventilated bunker no evidence of oxygen depletion CO or CO_2 build up occurred. They deduced that heat was probably the most important factor causing death.

The Canadians produced different findings. They used goats as the unfortunate subjects and flame throwers to model the attack. They found lethal levels of carbon monoxide in the blood of the animals after the fire assault. The controversy was resolved by further experiments when it was found that statically ignited fuel in a poorly ventilated space is self-extinguishing but the heat generated may be sufficient to kill.

When an intensely burning flame is injected into a poorly ventilated space the oxygen concentration can be forced to very low levels and CO_2 concentrations rise rapidly to lethal levels. It was concluded that in poorly ventilated spaces high carbon monoxide levels with low oxygen concentrations acting together were the most important lethal factors with heat adding to the effect. In well ventilated compartments heat was thought to be the decisive casuality producing factor.

In 1945 Dr Moritz[6] published his experimental findings when he set up experiments to look at the effects of inhaled heat on the lungs. Using anaesthetise dogs with a canmula directing hot gases into the trachea, he showed that air

at 500°C nly produced damage on the area of the tracheal wall where it impinged and th temperature of the air dropp d to 50°C at the bifurcation of the trachea. Hot gases only contain a small amount of heat. He then used st am as the medium for carrying heat and when this was blown onto the larynx local oedema was quickly produced. When steam was blown into the lungs damage to the small bronchi was produced and pulmonary oedema developed. He also showed that changes of bronchopneumonia developed within 8 hours of the steam injury.

These experiments showed that if respirable gases contained steam and this reached the parenchymal tissue of the lung a fatal pulmonary oedema could be produced. Hence heat alone can cause respiratory failure.

Thereafter a lull phase seemed to occur in the subject until 1952 when Aviado and Schmidt[7] reported their findings on the subject of respiratory burns in anaesthetised dogs in a series of most elegant experiments. This paper is of particular importance since the authors included new radioactiv tracer technique to study the mechanism of pulmonary oedema produced by steam inhalation. Fluid appeared in the trachea within half an hour of inhaling steam and this was accompanied by tachypnoea, hyperpnoea with decreased levels of carbon dioxide in the arterial blood. Most importantly, the tracheal fluid was found to have a specific gravity 80% that of plasma. The terminal phases were characterised by hypoxaemia.

They showed that pulmonary congestion of the lungs occurred with the pulmonary blood volume increasing by 60% - 80%. This was not due to increased blood flow since the cardiac output fell but the left atrial pressure did not rise after burns. They favoured a constriction of the pulmonary veins as the main cause of the accumulation of blood in the lungs.

At autopsy the lungs were heavy and contained a foamy sanguineous fluid. Histologically, pictures of perivascular cuffing of the adventitial lining of the venules is beautifully describ d and illustrat d. They concluded that the pulmonary oedema seen following the inhalation of steam was du to a combination of increased capillary permeability and capillary bed congestion s condary to pulmonary venous constriction. The final sentenc reads:

"a method for detecting early pulmonary oedema in a living animal is still wanting". That was a quarter of a century ago.

In 1954 there was a report of further experimental work on the subject by Fineberg, Miller and Allbritten.[8] Again steam was insufflated into the lungs of anaesthetised dogs. This resulted in a drop in the arterial oxygen tension over a period of 3 - 4 hours accompanied by the development of râles in the lungs. Pulmonary oedema then occurred shortly afterwards. At autopsy a gelatinous fluid was found to be present in the lungs which were heavy with oedema. It was also shown that giving a plasma substitute intravenously following the respiratory trauma produced an even more severe pulmonary oedema.

In 1955 the tragic Dellwood Fire[9] occurred in the United Kingdom. A fire occurred in a nursery containing 15 young babies. The main feature was considerable dense smoke - only one child sustained significant body burns. 11 of the survivors developed a progressive respiratory distress condition within 24 hours and this became severe by 48 hours. Two improved by the third day but the other 9 deteriorated and died. Postmortem examination showed membranous casts in the larger airways with bronchiolitis in a few individuals. Congestion and oedema was present in the lung tissue of all the fatalities. In one, lipoid material, thought to have come from linoleum which was in the nursery was identified in the alveolar tissue.

This disaster awakened the authorities to the danger of smoke inhalation when fires occur in confined spaces. The problems of management of such victims also received attention, the major methods being antibiotics, steroids, supplementary oxygen but assisted ventilation was not yet a common practice. It also showed that volatile chemicals could be inhaled with the smoke and pass deep into the lungs.

By 1959 another important paper was produced by Dr Aviado.[10] Again using the dog inhaling steam mod 1 he showed that the pulmonary oedematous fluid had a high protein content (85% that of blood).

He also showed that survival of the burned animals could be improved with anti-histamines, intermittent positive pressure ventilation and ethyl alcohol vapour added to the inspired gases. Not only was the mechanism of production of pulmonary oedema being investigated but the methods of treating the condition were being evaluated. The information seems not to have been heeded for quite some time.

Then in the period 1960 - 1962 an interesting contrast in the reports of two groups of workers were published.

The group at the Massachusetts General Hospital under Phillips and Cope[11] reviewed the data on burn fatalities admitted to that hospital. They showed that there was no change in the mortality rate from burns in the period 1943-47 compared with 1948-54. They did say a decrease had occurred in the 1930's which was thought to be due to the improvement in the treatment of shock. In the United Kingdom, Bull and Fisher[12] also compared the death rate in a large group of burned patients studied in the period 1949-62 and compared them with a group in 1942-47.
Theoretically there should have been a change since penicillin and other antibiotics were available to treat burned patients in the second group. Bull and Fisher made the important observation that there was no significant change in the mortality rates of the two groups, but the patients survived for a longer time before dying of sepsis. Most importantly they only allocated one sentence in their report to the effects of burns on the lungs. On the other hand Phillips and Cope[13] stressed the important part that the development of respiratory infection played on mortality rate. They also drew attention to respiratory tract damage occurring without evidence of respiratory infection and this was quoted as high as 42% in their series. They also described vividly the clinical picture of the victims of respiratory burns and the pulmonary pathological changes in commendable detail.

The pathological changes were very similar to those described in the Cocomut Grove Fire namely, laryngeal oedema, denuding of the respiratory mucosa, pulmonary congestion and oedema, plugging of the smaller bronchi and atelectases of areas of lung.

They also drew attention to the deceptive interval of easier breathing which may occur during the first 24 hours with the subsequent full blown respiratory distress occurring later. Somehow the Bull and Fisher paper fails to record any such details.

In 1963 Sochor and Mallory[14] published their report on the lung lesions found in patients dying from burns. They stated that alveolar haemorrhage was more common in the victims who had head burns compared to those without head burns: and this occurred most frequently in the first 2 days after injury. They also stated that tracheobronchitis was most frequent in the first week with bronchopneumonia occurring most frequently in the 2nd week. The pathological descriptions of the lung lesions are very similar to those reported by earlier workers. At least by now it was apparent that lung damage was a major cause of death in fire victims.

Later that year Jackson and Lee[15] analysed the progress of 629 burn victims in Memphis admitted to hospital in the period 1955–60. They reported that 17 deaths occurred in the first 48 hours and 14 of these had extensive body burns with considerable lung damage; while 92% of deaths associated with 20% – 40% body burn area was due to bronchopneumonia. They attributed only 2 deaths out of the total group as being caused by respiratory burns but nowhere is smoke toxicity mentioned in the paper. This work is really confirmatory on the reports of Phillips and Cope.

By 1967 attention was turning to the mechanism of damage to the lungs caused by fire and smoke. Stone, Rhame, Corbitt, Given and Martin[16] reviewed the sequence of events in 27 patients admitted with this condition to their hospital. They reported that if there were physical signs in the chest on admission this was an ominous sign. They also stated that clinical evidence of lung damage always preceded radiographic changes. Ten subjects developed pulmonary oedema, six of whom died. At autopsy the lungs were oedematous, haemorrhagic, showed damage to the alveolar capillary membrane and the fluid in the airways had a high protein c ntent. They felt that the most likely

cause of damage to th lungs was that due to inhaled products. They then reported the results of their animal experiments using rats. By subjecting alive rats t breathing increasingly hot air with increasing humidity, they showed that a direct relationship existed between the temperature and humidity and the mortality rate. They then added smoke from smouldering cotton and this showed an even higher lethal rate. Hence temperature, humidity and smoke adversely affect the change of survival.

That same year (1967) Webster, McCabe and Karp[17] described the clinical picture of 3 patients rescued from a fire who required hospital treatment. They showed -

(1) a deceptively mild initial period in which the patients may be asymptomatic then follows;

(2) an acute drop in the forced vital capacity;

(3) 6-48 hours later rapid onset of bronchiolitis and pulmonary oedema;

(4) acute right heart failure may also develop at this time;

(5) eventually some victims return to normal lung function.

An Editorial article in the British Medical Journal in 1967[18] somehow was not in tune with the current thoughts of the time. It stated that most cases of respiratory burns in the United Kingdom occur in children whose clothes have caught fire and they then run about breathing normal air. Only about 5% of patients admitted to hospital have received the burn in a conflagration and the pulmonary lesion tends to be missed! It reported that pulmonary insufficiency due to narrowing of the smaller bronchi may occur in the 24-36 hour period after admission but pulmonary oedema was uncommon within the first 8 hours and pulmonary infection occurred in the later stages of the management of burns. While technically correct this editorial failed to direct attention to the need for better diagnosis of fire damage to the lungs, improved awareness of the condition in anybody who has b en involved in a fir and the increasing possibility of breathing toxic gases and fumes in these circumstances. Unfortunately the subsequent dearth of inter st in this condition in the United Kingdom is reflected in th paucity of clinical and exp rim ntal reports

emanating from this country.
The f ll wing year (1968) another report on the pathological changes found in the lungs of burned patients was published in the United States by Foley, Moncrief and Mason.[19] They reviewed the findings of 233 autopsies in patients with cutaneous burns who had been gathered over a 6 year period. They also showed that the major cause of death had changed from wound infection to pneumonia. This was thought to be due to better control of wound infection but tracheotomy and mechanical breathing support may have played a part. They also showed that the presenoe of facial burns was associated with a higher incidence of respiratory complications but this may only reflect that facial burns were found in the most severely burned patients.

In 1969 Cornish and Abar[20] described the results which they obtained when they tested the effects of the pyrolysis products of vinyl plastics on rats. They stated that the major factor was the production of carbon monoxide which lead to high levels of COHb in the blood. They also observed that when oxygen was added to the inspired air of the exposed animals pulmonary oedema and haemorrhage was seen in the lungs at autopsy. Hence the effects of breathing toxio products from burning plastics produces a complex picture from the interplay of direct and indirect damage to the lungs.

In 1969 Stone and Martin[21] reviewed the progress of 1306 burned patients admitted to their care over the period 1961-68. They confirmed that pulmonary clinical signs were often absent for the first 36 hours and radiographic changes until the 2nd - 3rd day. But they then say that pulmonary oedema occurred in 42 of their subjects somewhere between 5 hours and the 7th day. They attributed this condition to too much intravenous fluid. Hence they moved away from toxic damage to the alveolar capillary membrane as the cause of the pulmonary oedema. But by 1970 suspicion was arising that inhalation of toxic fumes or gases could be an important factor in th lung complications of burned patients.
But new publications were soon t appear which brought fresh evidence of this possibility. Later that year a report of lung diseas caused by the inhalation of nitrous fumes was published in France (Larcaw, Calamai, Lambert & Memtre[22])

They described a patient who developed br athlessness, ral s and lung infiltrates foll wing the inhalation of the fumes from burning dolls which w re made from nitro-cellulose. Fortunately he recovered fully within 10 days.

In the same year Tse and Bockman[23] described the olinical events in 4 firemen who had been exposed to nitrous oxide fumes in a fire. There appeared to be two distinot phases in this illness. The first phase characterised by acut pulmonary oedema developed 24-48 hours after exposure but they all recovered. Thre of the group developed an illness characterised by breathlessness and wheezing 4 - 6 weeks after the primary exposure. Biopsy at this time is reported as showing the changes of abronchiolitis in an organising stage but diffuse infiltration of the alveolar tissue with monomuclear cells was also seen. A further biopsy 6 weeks after the fire showed a healed bronchiolitis with only scattered lymphocytic infiltration and collagen thickening of the alveolar walls. This paper does describe a speoific example of damage due to inhalation of a known toxic chemical with the production of lung damage. The time scale of the two phases is interesting and supports the possibility that two pathological proc sses occur in the lungs with a different rate of development of signs and symptoms.

Another report on the pulmonary complications of burn patients by Pruitt, Flemma Divicenti and Foley[24] appeared that year with findings similar to the previously reported series.

Then in 1971 Lloyd and MacRae[25] described the clinical course and management of tw patients admitted under their care with respiratory tract damage following rescue from burning buildings. They reviewed the entity of fire damage to the lungs. They also comment on the comparative lateness of the United Kingdom to really become involved in this field. This article reviewed the state of knowledge on the condition at that time and described the management of such cases in considerable d tail. They made a plea for the use of the phrase "respiratory tract damage in burns"
rather than pulmonary burn or smoke toxicity. This ofcourse implies that the two conditions can be readily separated on clinical grounds.

That same year Woolley[26] described the decompensation products produced by the pyrolysis of PVC. He stated that mainly non-toxic aromatic and aliphatic hydrocarbons were produced. He did not detect phosgene but carbon monoxide and hydrogen chloride were released and would form the main risk in a fire.

In another review Haynes[27] again confirmed the previous reports that direct injury to the upper respiratory tract can occur when individuals are exposed to and breath hot gases in a closed area and that pneumonia secondary to burn injury was a major cause of death. He felt that airborne infection of the lungs had now become commoner and that haematogenous pneumonitis less common due to better treatment of systemic burns.

Attention was now being directed to machanisms of damage to the lungs associated with burns. The term "smoke poisoning" was now being used to describe victims of fires who had little or no body burns but developed respiratory symptoms or who had succumbed at the scene of the fire. In 1971 Zikria, Ferrer and Floch[28] produced their paper describing some experimental work with anaesthetized dogs made to breath two types of smoke. The first smoke was produced by burning kerosene while the second from burning wood. They showed a significant difference in the mean post mortem lung weights due to an increased water and sodium content when the effects of kerosene smoke were compared to the effects of wood smoke. The latter was associated with a heavier lung which had a higher water and sodium content. Wood smoke was associated with pulmonary oedema in all the exposed animals. Two dogs were independently exposed to wood soot insufflated down a bronchoscope but no significant pathological changes were seen in the lungs at autopsy. Detailed chemical analysis of the two types of smoke showed that wood smoke had a higher content of carbon monoxide and total aldehydes. This was particularly marked for the acrolein content, a chemical known to be particularly irritating to the upper respiratory tract and likely to cause pulmonary oedema at concentrations as low as 10 ppm.

This work was important in identifying factors which helped explain pathological findings in fire victims. Smoke poisoning could be seen to be an entity by itself.

In 1972 Zikria, Weston, Chodoff and Ferrer[29] produced another important paper on the subject of smoke and carbon monoxide poisoning in fire victims. They analysed the autopsy reports on 257 such victims which occurred in New York City in the period 1966 - 67. They decided to divide the group into two categories according to whether they survived longer than 12 hours post burn period or not. They reported that respiratory tract involvement was found in 70% of the deaths which occurred in less than 12 hours after the fire compared with only 46% of those surviving longer than 12 hours. In addition smoke poisoning and asphyxia was present in 54% and respiratory tract pathology in only 6% of those dying within 12 hours, while the respectiv figures were 6% and 39% for those dying after 12 hours.
Looking at the pathological changes present in the lungs, they showed that pneumonia and pneumonitis occurred more often in those surviving longer than 12 hours while pulmonary congestion and oedema was associated with those surviving less than 12 hours. Analysis of the levels of carboxyhaemoglobin in the victims was not so helpful since in many cases the value was not available especially in those surviving longer than 12 hours. They state that of the 124 victims attributed to smoke poisoning or asphyxia 79% had carboxyhaemoglobin levels above 10%. They also made the observation that raised levels of carboxyhaemoglobin were present almost equally in the presence or absence of body burns. Using the Brooke model to predict the probability of survival according to the surface area of the burn they showed that many victims who should have survived died because of respiratory involvement. They concluded that almost a third of all fire fatalities were due to "smoke poisoning".

In 1972 two papers relating to the clinical aspects of fire victims were published: The first by Oldenburger, Maurer, Beltaos and Magnim[30] described the case of a 24 year old man whose job exposed him to the inhalation of burning fats. He had the signs and symptoms of an interstitial lung disease and this was confirmed on biopsy where interstitial granulomas, foamy histiocyt s and giant cells containing fat stainable globules were found.

The point was mad that when animal fats were inhaled the fat is hydrolysed liberating fatty acids which cause local inflammation. This paper showed that fat may be inhaled into the gas exchange tissue of the lung producing a serious pathological change.

The second paper by Lauda, Avery and Sackner[31] described the clinical and physiological findings of 6 subjects who were trapped in a smoke filled elevator in a burning building. They found elevated carboxyhaemoglobin levels and evidence of bronchospasm in all 6 victims. They all survived but 2 patients developed an acute myocardial infarction during their time in hospital. Arterial blood gas measurements on admission showed that hypoxaemia and metabolic acidosis were present in the group.

In 1973 an important paper on the possible mechanisms of pathological changes in the lungs found in association with burns was produced by Rapaport, Nemirovsky, Badoslav and Ball.[32] Using Wister rats as their model they produced a standard full thickness skin burn on anaesthetized animals. Groups of burned and normal control rats then received intravenous suspension of colloidal carbon at different intervals after the burn. They showed that a precipitous clearance of carbon from the blood occurred within 1 - 4 hours after the injury and this was due to a massive embolization of carbonfloccules into the pulmonary tree. This in turn caused a profound hypoxaemia with anoxic death of many of the rats. Most importantly they also showed that anti-coagulant doses of heparin prevented this effect. Hence anyone sustaining a significant burn could develop a similar sequence of events with hypoxaemia developing there being no exposure to inhalation of toxic substances.

The lungs can be seriously damaged from either the air or the blood aspects of the alveolar capillary membrane. Looking at reported cases those with known smoke toxicity who also have body burns have a higher mortality than either alone.

In 1973 German, Alkyn and Bartlett[33] r ported their studies on 10 patients in the 2 - 5 days after being burned. They showed how the mean pulmonary artery pressure was reduced during the first few hours after th burn but rose to normal

levels with good r suscitation. Th y warn that raised levels of pulmonary artery diastolic pressure c rrelate well with left atrial pressures measured at the same time and indicate the state of fluid overload. They also make the statement that some patients with smoke inhalation damage develop pulmonary oedema when the pulmonary artery diastolic pressure is within the normal range. This evidence is in favour of smoke damaging the lung capillaries and making them vulnerable to increased fluid loads.

In the same year Wanner and Cutchavaree[34] described the fibre-optic bronchoscopy findings in a group of 45 patients seen in the emergency room soon after rescue from a fire. Pharyngitis and laryngeal oedema were seen in the group 2 of whom developed acute respiratory distress requiring a period of intermittent positive pressure ventilation. They recommended early fibre-optic brochoscopy as a means of assessing the degree of upper airway damage in fire victims.

In 1974 Faling, Medici and Chodosh[35] made a study of the cell population of the sputum of 29 patients with smoke inhalation damage. They reported a high correlation between their indices of bronchial mucosal damage and the ventilatory measurements of lung dysfunction. The highest correlation was with the level of COHb in the blood. Hence cellular damage in the airways follows closely the other measurements of lung damage.

Carter, Bafus, Warrington and Harris[36] in 1974 described the effects of exposing male rats to the thermal degradation products of 4 fluoropolymers. They showed how death occurred within 48 hours due to pulmonary oedema and haemorrhage in some of the animals and this was greatest in the case of P.T.F.E when it was heated to 800°C.

Several reports appeared in 1975 of the respiratory complications of burns. McArdle and Finlay[37] described the typical clinical, radiological and arterial blood gas changes in two patients who had sustained upper b dy burns. In one of these they showed how the use of p ak end expired pressure improved the ventilation of the subject.

Mellins and Park[38] reviewed the literature and made the statement that if chemical or thermal injury to the lung is going to develop in fire victims, it will do so within the first 24 hours. They felt that respiratory disease occurring several days after the burn injury was invariably related to appreciable surface burns. They also warn against too vigorous use of colloid fluids in the resuscitation phase and also excessively high concentrations of inspired oxygen.

Esrig, Stephenson and Fulton[39] described their experimental findings using anaesthetised dogs breathing wood smoke spontaneously for 6 minutes. They make the statement that the initial pulmonary changes of smoke inhalation are due to both bronchial and vascular spasm with resultant changes in ventilation/perfusion balance of the lungs producing hypoxaemia. They also instilled pseudomonas aerogenosa bacteria by way of an intracheal injection into the smoke being inhaled by the animals. They showed that the combination of insults was more lethal compared to smoke alone.

Stephenson, Esrig, Polk and Fulton[40] in another paper described the detailed changes which occurred in the cardio-pulmonary system of anaesthetized dogs made to breath wood smoke spontaneously. They followed the changes which occurred as the animals came close to asphyxia when breathing the smoke for a 4 - 6 minute period. An 8 minute period of smoke inhalation was found to be uniformly fatal. When the lungs of the animals were examined at autopsy 4 hours after the toxic insult there was a significant increase in lung water. In the survivors a progressive increase in the alveolar - arterial oxygen tension difference occurred accompanied by a decrease in compliance. Cardiac output decreased for the first 24 hours and both the systemic and pulmonary vascular resistances were substantially elevated for prolonged periods. The pulmonary wedge pressure did not change significantly during this time. Administration of crystalloids produced no change in the cardiac output or in the vascular resistances They then state that the pulmonary injury from smoke inhalation can b divided into four phases -

Stage (1) - while in the smoke chamber the arterial oxygen tension falls and the carbon dioxide tension rises producing an acidosis. They noted that the animals breathed the toxic smoke easily without developing glottic closure once the animal was unconscious. This observation suggests that in man near unconsciousness must occur before significant smoke inhalation occurs.

Stage (2) is the immediate period up to 30 minutes and is characterised by a severely depressed cardiac output and increased vascular resistanc , the mechanism of which is unknown.

Stage (3) develops as blood flow increases through the lungs but damage to the alveoli now becomes apparent. Interstitial oedema is seen and alveolar closure occurs.

Stage (4) occurs between 24 - 72 hours and is thought to be related to the onset of pulmonary infection with lung oedema now being a common finding. Death was usually due to respiratory failure.

This excellent paper describes the sequence of pulmonary changes which occur in smoke damaged lungs with considerable clarity. The mechanisms underlying the changes are more obscure and serve as leads for those studying this condition. Levels of COHb would have been most useful.

In the same year another important study was published by Zawacki, Jung, Joyce and Rimion.[41] They used male mice which were exposed to smoke produced from burning crude cotton in a specially constructed chamber where the O_2, CO_2 and CO content could simulate that found in domestic fires. They also eliminated fluid resuscitation and pulmonary infection as variables as far as possible in their study. In this way they hoped to show a dose-response effect from the exposure of the animals. They did show that the inhalation injury was a simple function of the smoke and burn dosage and that the COHb level measured soon after the injury was a good indicator of th absorbed dose of smoke. As well as subjecting the mice to smoke they also inflicted a skin burn of known degree by using a hot brass block applied to the skin. They also reported the results of their experiments where fluid r suscitation and inhalation of pseudomonas aeroginosa

were included in the experiment. They state that plumonary oedema occurred in association with too little rather than too much fluid therapy and fatal bacterial pneumonia was difficult to produce with inhalation injury alone but common when associated with a surface burn. There is much in this paper which is relevant to the clinical picture of fire victims. The use of the COHb as an indicator of the possible degree of lung inhalation or smoke toxicity damage is now accepted and the need for a tissue burn as well as smoke damage as a necessary combination before fatal bacterial pneumonia occurs is also recognised. The findings on too little fluid resuscitation being more important than too much fluid is more difficult to substantiate.

That same year a review paper by Barlett, Nicole, Tavis, Allyn and Furnas[42] where 740 burned patients admitted between 1972 – 75 to their hospital showed that 36 required mechanical ventilation and 24 of these died. They say that smoke inhalation alone does not cause serious respiratory failure but the combination with body burns does.

Further reports appeared in 1976. Stewart, Stewart, Stamm and Seelen[43] reported their findings where they measured the COHb of fire fighters using a portable apparatus to measure CO in the expired breath at the scene of a fire. They showed that COHb of the men measured after three training sessions was significantly raised in both the smokers and non-smokers the highest level being 13%. Hence firemen do develop raised COHb levels when exposed to the smoke of fires.
On the practical side of observing the effects of being exposed to the smoke and fumes from burning buildings, Dyer and Esch[44] reported on the clinical findings in a group of firemen who had been exposed to toxic fumes in an office fire. Many of the fire fighters developed acute respiratory symptoms soon after returning to their quarters and one died suddenly the next day. At autopsy he had evidence of a chemical pneumonitis. This paper then described the evidence for incriminating the release of HCL from burning PVC as the major cause of the fatality. They then reviewed the animal experimental work on the toxic products released from the pyrolysis of PVC, Kishitani,[45] In these experiments it was shown that the combination of exposure to PVC fumes with the release of HCL and CO from th

burning of c llul se had a reciprocal potentiating adverse eff ct. They also show d how chlorine, phosgene and HCL were all released from heated PVC. It seemed that HCL could be adsorbed onto soot particles which could b inhaled into the most distal parts of the lungs and hence damage the gas exchange tissue.

They also commented on the work of D P Dressler[46] who showed that CO_2 may rise dramatically in an enclosed space when plastics and modern fibres burn in such a space. The possible cardio toxic effects of HCL were also described from the mice experiments of Kishitani. While this paper performs the excellent function of making all those involved in fighting fires and those who also look after the victims become aware of the potential hazards, there is a lack of scientific data necessary to establish a correlation between circumstances and effect in the subjects described.

In 1976 a paper relating to the clinical assessment of inhalation injury caused by fire was presented by Petroff, Hander, Clayton and Pruitt.[47] They used the Xenon 133 scintophographic lung soan technique and the analysis of the maximum expiratory flow volume curve to try to detect and measure the degree of damage in patients who had been burned. They concluded that both techniques are useful in evaluating smoke inhalation damage and for following the progress of the patients. They felt they could separate out those patients who had inhalation injury from those who did not. In the group with abnormal 133Xe scans 87% had sustained their injury in a closed space, 53% had carbonaceous sputum and the mortality rate was twice those with normal scans. The major problem with these techniques are that some patients may have abnormalities due to pre-existing lung disease and not due to the exposure to the harmful products of the fire.

Hampton[48] also described the clinical findings which he found in 29 seamen who had been exposed to a fire in ship where they were trapped in a smoke filled compartment. 8 - 24 hours after rescue most developed dyspnoea cough, exp ctoration and wheezing while 2 progressed to pulmonary oedema. All subsequently recovered without apparent perman nt damage.

In 1976 there was published the proceedings of an International Symposium on the T xicology and Physiol gy of Combustion Products held by the University of Utah in Salt Lake City.[49] This symposium brought together many of the leading experts on the effects of the products of fires on the lungs. Zikria et al[49] reviewed the findings in 57 patients who had been admitted to the Columbian-Presbyterian Medical Centre between 1960 - 72 with a diagnosis of smoke inhalation, CO poisoning or respiratory burns. He states that where a clinical diagnosis of smoke poisoning is made more than 80% will have COHb above 15%. All the patients with the diagnosis of respiratory burns had some visible evidence of respiratory tract injury on laryngoscopy or bronchoscopy. They stress the important points that the level of the COHb is a good indicator of the degree of pulmonary injury by other injurous gases, and in many smoke poisoned patients, pulmonary injury may not become clinically evident for 12 - 48 hours. He states that smoke poisoning is a chemical respiratory injury with concomitant CO poisoning.

Smith, Crane, Saunders, Abbot and Endercott[49] reviewed the toxicological findings in the victims of two commercial aircraft accidents. They come out strongly in favour of CO poisoning as being a major cause of death in these victims and feel that the evidence for other toxic gases being involved is very sparse. Blood cyanide levels were measured but a poor correlation was attained with the COHb level.

Austin[49] also reviewed the evidence on the medical aspects of toxicity resulting from fire exposure. He confirmed the findings reported previously, no new evidence being produced.

Radford, Pitt, Halpin, Caplan, Fisher and Schmeda[49] reported their findings on a study of 107 fatal fire victims in Maryland over the period 1971 - 74. All th victims had died at the scene of the fire, had an autopsy carried out by the Medical Examiner's office and had COHb measured in th School of Hygiene Laboratory. They drew attention to the large number of these fatalities (34%) who had coronary vascular diseas . A COHb of over 65% was found in 44% of the group. In 18 individuals the degree of skin burn was considered to have been

th caus of death. In 26% f the total a bl d alcohol level of greater than th considered int xicating level (0.15 gm/100 ml) was pr sent. Most of the cases had inhaled smoke as shown by soot particles in airways. They make the statement that some of the cases showed significant effects of smoke products on the respiratory tree and pulmonary oedema was present in a few cases.

In 1976 there was a detailed study of an 18 year old burn victim whose clinical course is described in the Western Journal of Medicine.[50] Those in charge of his management advocate early fibre-optic bronchoscopy to assess the upper airway damage but also state that systemic steroid therapy may increase the chance of lung infection.

Later that year, Axford, McKerrow, Jones and Le Quesme[51] described the respiratory history of 35 firemen who had been exposed to spilled toluen diosocyanate while fighting a fire. Most of the men developed respiratory symptoms while fighting the fire or within 3 weeks thereafter. Serial ventilatory measurements showed a marked decline in the first 6 months but thereafter improved. 20 of the men still had persistent symptoms 4 years after the incident.

In 1977, Levine and Radford[52] reported on the outcome of all victims of fires identified by the Baltimore Fire Department over a period of 14 months. In that particular group, 16% out of the total investigated were fatal. The majority of deaths were due to pulmonary injury or CO poisoning, the latter being the major cause of deaths at the scene of the fire or in the first 24 hours.

On the practical experimental aspect Ballantyne and Clifford[53] examined the respiratory response of a group of volunteers who were exposed for 10 minutes to annamic acid smoke. They were unable to show any short term effects on the breathing characteristics or total airway resistance of the group.

An interesting report on the pathophysiology of acute smoke inhalation in anaesthetised dogs was published by Clark, Webb, Wars and Mieman.[54] They exposed the animals to sawdust and kerosene smoke using a respirator t administer

12 breaths. Using cinephot microscopy they state that after two breaths atelectases was observed and photographed grossly and microscopically. The arterial PO_2 fell, the pH declined, the PCO_2 rose and the COHb increased dramatically. After 5 minutes of smoke exposure the airway pressure had increased 3 fold, the work of breathing similarly and the dynamic compliance was less than half the control value. They also measured the surfactant in a tissue sample of the lung and showed that the surfactant activity was severely compromised. The normal hysteresis of the surface tension curve was abolished after smoke exposure. Even more remarkably they report that the incorporation of 0.5 μ pore sized filter protected the surfactant system, prevented atelectases and significantly blunted the changes seen in lung mechanics afte. smoke exposure.

This is a remarkable example of practical experimentation to help answer the problem of smoke toxicity. If these findings can be transcribed to the human situation the speed of onset of the pathological changes quite overwhelm the generally accepted concepts. As stated only acute surfactant inactivation could explain the time course of these events. A filter seems to trap what is causing this dramatic change.

A paper by Trunkey[55] described the current thinking on inhalation injury associated with burns. He divided the condition into two distinct categories of carbon monoxide poisoning and smoke toxicity. The latter he further subdivided into direct injury and smoke poisoning. Each category is described very much according to previous workers but he does make the point that some plastics release benzene on pyrolysis and this acts as an anaesthetic hence allowing more toxic but normally irritant chemicals to reach the lung alveolar tissue. The description of the pathophysiological changes are again those described in previous papers. Excellent practical advice on the management of th se victims is given and a plea is made for smoke detectors to be installed in buildings and hom s.

Then in 1978 Schwerd and Shultz[56] made the interesting observation that no COHb was present in the blood of some victims who were burnt in a fire. They reason that acute cessation of breathing can occur when a person suddenly inhales very hot gases. High levels of COHb were found in victims who survived for several minutes. In addition soot particles were found in the small bronchi. High levels of MetHb were also found in three victims who survived for more than 5 minutes after the conflagration.

Th se authors suggest that a sudden arrest of the circulation could be produced by the inhalation of very hot gases. Perhaps nearly 40 years later it may be se n that the occupants of the concrete defence bunkers subjected to assault by flame died of fright.

ANALYSIS OF PREVIOUS REVIEW

Analysis of th literature review requires certain questions to b asked the main one being, -

"is there any difference in the respiratory pathology of modern fire victims ?"

On review of the evidence presented in the previous section there is no real change in the pathological features in the lungs of present day fire victims compared with 30-40 years ago. The detailed changes described in the autopsy reports of the Cocomut Grove Fire of 1942 confirm this finding. The same changes of mucosal damage lung oedema and haemorrhage are characteristics of fire fatalities today. It is apparent that the effects of inhaling the products of fire in the 1940's is tha same as in the 1970's. While we do have more physiological evidence of the progress of the burned patient today the clinical description of the sequence of events and the time scale of respiratory symptoms and signs remained the same. The initial irritative phase, breathlessness with/ without cyanosis, expectoration of mucoid sputum containing soot particles followed by a period of apparent recovery or quiescence is common place today. The difference is that the modern fire victim can be shown to have significant lung function abnormalities at this stage although the reduction in the vital capacity was noted in the early papers. The subsequent recurrence of respiratory distress and function is now a well recognised feature of patients who have been exposed to the products of fire. It has also become apparent that the prognosis of fire victims who have body burns and lung damage is significantly worse than lung damage alone. This is in keeping with the experimental work where the effects of systemic injury leads to damage to the vascular compartment of the lung from the release of vasoactive amines from the damaged tissues. Perhaps today the major change is in the awareness of the possible damage to the lungs of chemical mediators carried in the blood stream. It seems that the lungs respond to trauma in a predictable manner although the rate of onset and progression of the pathological changes may vary from patient to patient. Hence the fir victim is yet another example of the shock lung syndrome and as such any difference

in the degr e f lung injury merely reflects the time course f th response to treatment. The pattern of respiratory symptoms and signs seen in modern burned patients is very similar to the sequence described in those rescued from the Cocomut Grove Fire in 1942. The radiographic and biochemical changes also are very similar. Today we may be more aware of the usual sequence of events but the pattern remains the same. Our understanding of the patho-physiological processes taking place in the lungs is very much dependent on the investigations designed to find out the cellular and humoural processes underlying the respiratory distress syndrome. We are now aware that clumping of polymorphonuclear leucocytes occurs at an early stage of the injury in the lung capillary bed and that release of vasoactive amines by the leucocytes then occurs. This in turn damages the capillary endothelium which then allows increased quantities of protein to pass into the lung interstitium. This in turn disturbes the control of fluid balance across the capillary membrane. Although the lymphatic system of the lung can remove some of the excess proteins and fluid this defence may be overwhelmed and the excess flow through the alveolar cell layer into the alveoli themselves. This in turn seriously disturbs the gas exchange function of the lung producing hypoxaemia and cyanosis. The excess fluid in the lungs also makes the lung tissue less pliable making them very difficult to ventilate. Hence is seen the combination of loss of lung compliance with a progressive hypoxaemia, leading to respiratory failure. When in the case of the fire victim there may also be damage to mucosa of the airways which cause the lumen to become narrowed and even obstructed it soon becomes apparent how a fire victim may die because of lung failure. It is also apparent why there is a large range of types of respiratory damage in people rescued from fires. At one end of the spectrum are the patients with upper airway damage only and no body burns, while at the other end are seen the victims with severe b dy burns and no r little upper airway damage. Both will in tim show respiratory disease as the lung responds to assault from either the gaseous or blood routes. The limitations of pr sent knowledge are very much related t amount of information which is available on the cellular and humoural response to trauma. This is

a field which has attracted much attention over the past 5–10 years but most studies ar more related t sepsis in the body, multiple trauma or haemorrhage rather than inhalation of the toxic products of fire. The effects of vasoactive amines such as bradykinims, prostoglandims and histamine have all been studied in animal experiments but much remains to be explained and the interaction of these metabolites remains to be worked out. Other chemicals such as the iso-nzymes released from damaged leucocytes must now be seen as harmful to the pulmonary vasculature. The role of the iso-enzymes requires special investigation since the cascade effect of the release of one injurous product leads to the release of another and soon until the combined effect is disastrous to the function of the lung. The earlier the chain of events can be broken the less damage there will be repair. Hence a good animal model where the pulmonary insult can be controlled and the sequence of biochemical amine or enzyme release studied should provide good leads as to the best therapy for the burned patient. While this will provide useful data on the basic pathophysiological processes underlying the lung damage in fire victims much remains to be learned about the prevalence of types and outcome of the various degrees of lung injury. It appears that this topic suffers from many departments and branches of medicine being aware of the aspects of burn casualties which interests them yet lacking in knowledge about the aspects which interests others. The emergency Physician, Surgeon, Anaesthetist, Renal Physician, Plastic Surgeon, Bacteriologist, Pathologist, Forensic Expert, all are involved but rarely pool their expert knowledge on the subject. A good avenue of communication and means of gathering facts on pulmonary complications of the fire victim would be beneficial for all those involved in the management of this common condition. While progress is being made in the field of general awareness of pulmonary complications of burns the unfortunate established case still presents a difficult therapeutic problem to those responsible for their management.

Future lines of research would best be advised to focus on the mechanisms of ellular damage and release of harmful chemicals into th lungs. This sequence of events may possibly be interrupted at certain points henoe limiting or even

making it possible to reverse the damage to th lung interstitium. The animal model where wood smoke is blown into the lungs should be developed so that the products released into the blood stream can be detected and measured. Differences in the concentrations in the arterial and venous blood of these chemical mediators may then show the time scale and sequence of events. Aviado showed that histamine release was a feature of the pulmonary burn but anti-histamine therapy did not seem to be effective as a therapy. Today many more amines can be measured and their effects blocked by other drugs hence some indication of how to manage burned patients may be learned. A closer study of the surfactant metabolism in the burned patient may also be fruitful. There seems no doubt that the turnover of these chemicals is changed following xposure to the products of combustion. Whether there is decreased producti n of increased breakdown of the surfactant is not known. Again there are pharmacological methods of influencing surfactant metabolism but when and how to use these drugs is at present left to individuals managing the fire victims. The combination of good olinical and physiological investigation of fire victims with the design of animal experiments which focus on damage to lung interstitium seem to offer the greatest hope in the investigation and saving of the fire victim

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