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An Experimental Study on the Pulmonary Effects of Smoke Inhalation

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=Abstract=To elucidate the patterns of acute death, the differences of toxicity of materials combusted, and histopathologic changes in lung of smoke inhalation injury, a hundred and ninty-six mice were exposed to the smokes of lauan, plywood, curtain and carpet. The rates of acute death varied with materials combusted; the higher mortality rates were obtained during the combustion of synthetic fabrics as carpet and curtain than in pyrolysis of lauan and plywood. The acute increase of carbon monoxide during incomplete combustion and the injury by the noxious gases are known for the main casuses of acute death in fire accident. The pathologic findings were not specific for the materials, but the edema of pulmonary interstitium was outstanding, accompanied with alveolar edema, localized atelectasis, congestion and the hyaline membranes. The water content of lung could be measured quantitatively by freeze-drying, which fairly correlated with the degree of the interstitial edema. ($R=0.835$)($P<0.001$)

Key words: ***Smoke inhalation, water content of lung, combustion, pulmonary edema***

INTRODUCTION

Modern fire accidents have made heavy casualties for their sizes. According to the reports from the National Safety Council, United States, the fire fatality ranks the fourth of accidental deaths and is increasing (Sopher 1983). The deaths in fire accidents were mainly caused by smoke inhalation, rather than by the direct flames (Pruitt *et al.* 1970). Trunkey(1978) observed that more than 60% of fire fatalities were secondary to inhalation injury. Among the inhalation injuries, asphyxia is the major cause of death, which results from the absolute decrease of oxygen due to its consumption during combustion, the relative lack of oxygen by mass-replacement of gases formed during the pyrolysis, and the presence of gases with higher affinity to hemoglobin than oxygen, as carbon monoxide.

Occasionally, those who had been rescued from fire accident and breathed well, developed sudden respiratory distress later, with or without thermal burn (Peters 1981). They suffered from injury and irritation of smokes on respiratory systems. Such smokes contained irritant gases (Terril *et al.* 1978), as well as particulate materials (Lee *et al.* 1976). With the development of newer synthetic chemicals, there is an increasing chance of smoke inhalation injury in fire accident.

Though many experiments with various sytems were conducted to study the occurrence and the nature of inhalation injury, some with closed exposure systems produced low oxygen level in the air (Birky *et al.* 1980), and the others were with overlapping thermal effects (Potkin *et al.* 1980; Walker *et al.* 1981).

Therefore, an exposure system excluding the low oxygen level and the thermal effect in smoke was needed, and with this, the experiment was performed to elucidate the major causes of death in fire accidents, the sequential histopathologic changes, and the objective quantitation of the water contents of lungs exposed to the smokes of different materials as lauan, plywood, and synthetic fabrics as curtain and carpet.

MATERIALS AND METHODS

1. Exposure system (Fig. 1)

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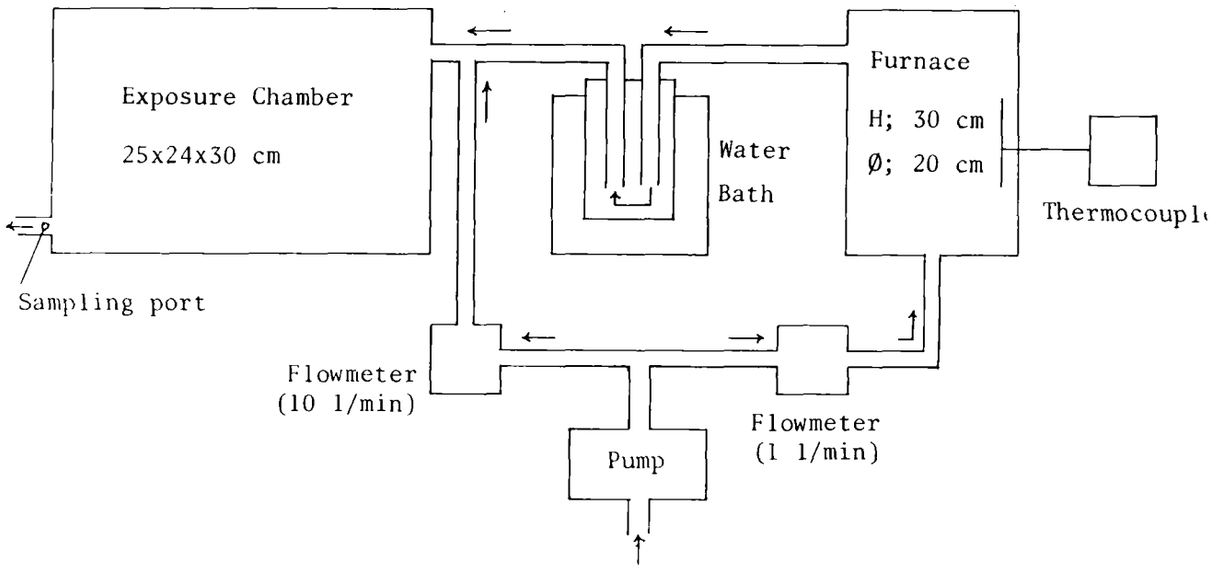

Fig. 1. Diagram of combustion-exposure system

To exculde the relative lack of oxygen and the thermal effect in respirated air, the exposure system was designed. A vacuum-compression pump (1/4 h.p.) aspirated and circulated the room air in the system, one to the furnace and the other to the exposure chamber with the flow-rate of 1 l/min and 10 l/min, respectively. The flow-rates maintained with two flowmeters. Furnace was made of 5 mm thick steel tube (20 cm in diameter, 30 cm high). A coil of nichrome (220 V, 2.5 kW) heated the furnace starting from room temperature, at the rate of 15°C/min, controlled by the thermocouple via the sensor of the wall. For the insulation, the wall of furnace is coated with 5 cm thick asbestos fibers. The inlet of room air and the oulet of smoke were at lower and higher protion of furnace, respectively. The furnace effluent was cooled in the water jacket and introduced to the exposure chamber. The cooled smoke (1 l/min) was mixed with room air (10 l/min) before the entry to maintain at least 18% of oxygen content. The volume of the exposure chamber was 30 liters. A sampling port for the detection of carbon monoxide was at the exit of fully exposed smoke.

2. Materials for combustion

Lauan and plywood, for natural matter, and curtain and carpet, for synthetic fabrics, were chosen for combustion, because they are most frequently used for the interior of the rooms. The curtain was made of 100% modacrylic polyester, the carpet was of BCF nylon and both were treated with fire retardant.

3. Animal exposure to smoke

One hundred and ninty-six male mice of closed colony of ICR strain were used, weighing 32.3 ± 4.04 gm in average. Among them, 10 were for the control, 84 for lauan combustion, each 34 for the plywood, the curtain and the carpet. Those were exposed to various amounts of smoke of materials. For the observation of histopathologic changes, 60 mice were exposed to the smoke of 10 gm of lauan and sacrificed at 0, 6, 12, 24, 48 and 72 hours after exposure. And the other groups of 10 gm held 10 mice which were sacrificed at 24 hours after exposure (Table 1).

With animals in exposure chamber, the pump supplied the room air to the furnace and to the exposure chamber. Thereafter, the materials to be combusted were introduced to the furnace and heated.

Concentrations of carbon monoxide were detected semiquantitatively by the gas detectors (Gastec™) at every 5 minutes, while 15 gm of each

Table 1. Numbers of animal in experimental groups

Materials	Amount of combustion (gm)						Total
	5	10#	15	17.5	20	25	
Control							10
Wood		60*	6	6	6	6	84
Plywood		10	6	6	6	6	34
Curtain	6	10	6	6	6		34
Carpet	6	10	6	6	6		34
Total	12	90	24	24	24	12	196

#; Group chosen to study for different effects of materials combusted.

*; Animals sacrificed sequentially for the changes of lungs.

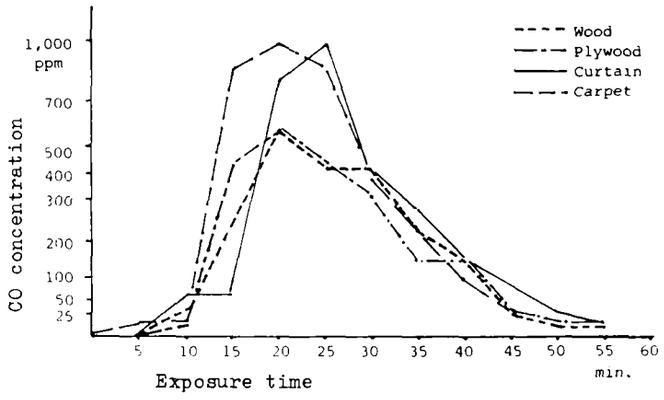

Fig. 2. Concentration during combustion of 15 gm of materials

materials combusted.

4. Water content of lung

Animals sacrificed at predetermined period were dissected and the right lungs were removed. The wet lung weighed immediately with electrical balance (A). Then evaporated for 72 hours in freeze-dryer (LABSCO®) and weighed (B). The difference of wet and dry lung weight (A−B) represented the original volume of water of lung. Water contents were expressed as (A−B)/A.

5. Microscopic observation

After fixation with 10% buffered formalin, the longitudinal sections of left lungs were stained with hematoxylin-eosin. The presence of intra-alveolar and interstitial edema, hemorrhage, localized atelectasis, neutrophilic infiltration, congestion of small vessels and epitheilal changes of bronchioles were evaluated. Small pieces of subapical protion of left lung fixed with 2.5% glutaraldehyde solution and examined with transmission electron microscope (Hitachi® , H300).

RESULTS

1. Concentration of carbon monoxide

The increasing patterns and the times for peak concentration of carbon monoxide liberated during the combustion were different in each materials, though they produced carbon monoxide abruptly. The concentration elevated more steeply in the combustion of curtain and carpet, and peaked at 20 minutes in lauan, plywood and carpet, while for curtain, at 25 minutes (Fig. 2).

2. Mortality rate

With the small increment of material, there was a sharp increase of mortality when the weight of material was critical. For example, during the combusion of 15 gm of lauan, no animal died, while two and five of six animals died at 17.5 gm and 20 gm of lauan (Table 2). The death or incapacitation of animal during the exposure was generally concurrent to the peak time of carbon monoxide, and the temperature of furnace was near to autoignition temperature of each material. The postmortem examinations showed the peculiar cherry red livor of tissues and organs.

Table 2. Moratlity by the materials combusted

Materials	Amount of combustion (gm)					
	5	10	15	17.5	20	25
Wood		0/10	0/6	2/6	5/6	6/6
Plywood		0/10	1/6	2/6	5/6	6/6
Curtain	0/6	3/10	5/6	6/6	6/6	
Carpet	0/6	0/10	1/6	5/6	6/6	

The LC_{50} of materials in the condition according to Litchfield & Wilcoxon (1949) demonstrated that the smaller amount of carpet or curtain than of lauan or plywood could cause the deaths of animals (Table 3).

Table 3. LC_{50} by the each material

Materials	LC_{50} (mg/l)
Wood (lauan)	574.1
Plywood	589.9
Curtain (polyester)	307.6
Carpet (nylon)	469.6

3. Water content of lung

A total of 50 mice, exposed to the smoke of 10 gm of each materials and control group, were sacrificed at 24 hours after exposure. The control lungs revealed 78.66 ± 0.53% of water contents (Table 4), which increased in experimental groups of lauan ($p<0.005$), and curtain and carpet ($p<0.001$)(Table 5). Meanwhile, the sequential

Table 4. Water contents of lung at 24 hrs after smoke inhalation (combustion of 10 gms of each materials)

Materials	No. of animal	Water contents(%)
Control	10	78.66±0.53
Wood	10	79.88±1.71
Plywood	10	79.95±2.01
Curtain	10	81.97±2.63
Carpet	10	80.92±1.44

Table 5. T-values of water contents of lung at 24 hrs (combustion of 10 gm each materiala)

Material	Control	Wood	plywood	Curtain
Wood	2.156*			
Plywood	1.914	0.083		
Curtain	5.622#	2.110*	1.913	
Carpet	3.907#	1.599	1.300	1.159

*; $P<0.005$, #; $P<0.001$, and others; $P>0.1$

Table 6. Sequential change of water contents of lungs by smoke inhalation (combustion of 10 gm of lauan)

Time after exposure (hrs)	Water content (%)
0	78.71±0.05
6	78.65±0.59
12	78.14±0.69
24	79.88±1.71
48	79.13±1.08
72	78.06±1.62

Table 7. Histopathologic changes at 24 hrs after smoke inhalation(%)#(combustion of 10 gms of each materiala)

Pathology	Wood	Plywood	Curtain	Carpet	Mean
Alveolar edema	18.2	12.4	14.6	17.2	15.6
Hemorrhage	—	—	*	—	—
Atelectasis	38.6	42.0	52.3	42.7	43.9
Pneumonia	—	—	—	—	—
Interstitial edema	84.2	85.5	89.4	90.2	87.3
Congestion	27.2	28.3	28.4	31.2	28.8
Bronchiolar mucosal lesion	—	—	—	—	—

*; one case demonstrates multifocal hemorrhages.
#; Frequencies of lesions per 100 or 20 high power fields. Findings are not specific for any material combusted.

changes of lung water content were not significant statistically in the group of 10 gm of lauan (Table 6).

4. Histopathologic examination

The microscopic findings were not specific for the materials, but showed the difference in frequencies (Table 7). The interstitial edema of lung was most prominent (Fig. 3), the degree of which fairly correlated with the water contents of lung ($R=0.835$)($p<0.01$). Others were exudative fluids in alveolar spaces (Fig. 4), localized atelectasis (Fig. 5), congestion of small vessels (Fig. 6), and focal hemorrhage (Fig. 7). Also noted was the presence of multifocal hyaline membrane in alveolar space associated with neutrophilic infiltration (Fig. 8).

Table 8. Sequential histopathologic changes of the lung after smoke inhalation(%)#(combustion of wood, 10 gm)

Pathology	Time after exposure (hr)					
	0	6	12	24	48	72
Alveolar edema	8.8	16.2	15.3	18.2	19.0	22.4
Atelectasis	36.6	40.7	35.4	38.6	42.6	39.2
Pneumonia	—	—	—	—	—	*
Interstitial edema	68.3	70.4	76.4	84.2	82.6	87.5
Congestion	52.3	55.8	46.2	27.2	30.9	31.2

*; two cases develop bronchopneumonia.
#; frequencies of lesions per 100 or 20 high power fields

The frequencies of histologic findings showed the slight difference according to the time of sacrifice, which showed that the congestion of small vessels at 6 hours was twice frequent as at 24 hours, and the interstitial edema was prominent at 72 hours (Tabe 8).

The electron microscopic examination demostrated the carbon particles in alveolar macrophage and at the surface of pneumocytes, and the swelling and degeneration of mitochondrias in pneumocytes (Fig. 9). In bronchiolar epithelia, there were loss of cilia and the marked increase of secretory graules particularly adjacent to the surface (Fig. 10). The interstitial edema was identified as collection of amorphous fluid in connective tissue.

DISCUSSION

Smoke inhalation can be defined as the inhalation of toxic or noxious gases and particles which can produce such symptoms as hypoxemia, as well as injuries on respiratory tract (Achauer *et al.* 1973). It is partly because of urbanization and crowdedness in a place that there are heavy casualties for the sizes of fire accidents. Furthermore, as the newer chemical synthetics have developed, various kinds of noxious or toxic gases are liberated when they are burned (Crapo 1981).

Terril *et al.* (1978) reported that the major causes of death on fire are 1) direct thermal burn,

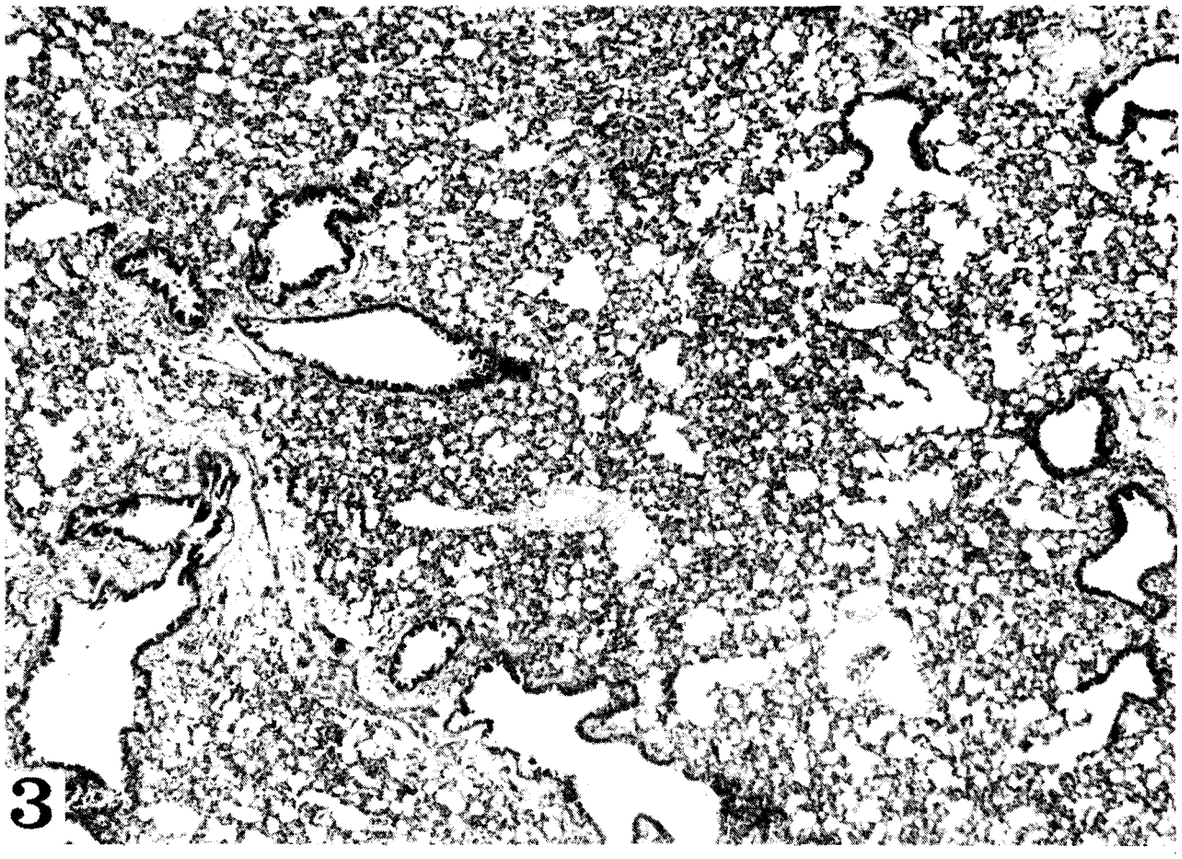

Fig. 3. Alveolar spaces obliterated by diffuse thickening of interstitium and interalveolar septa. (H&E, X40)

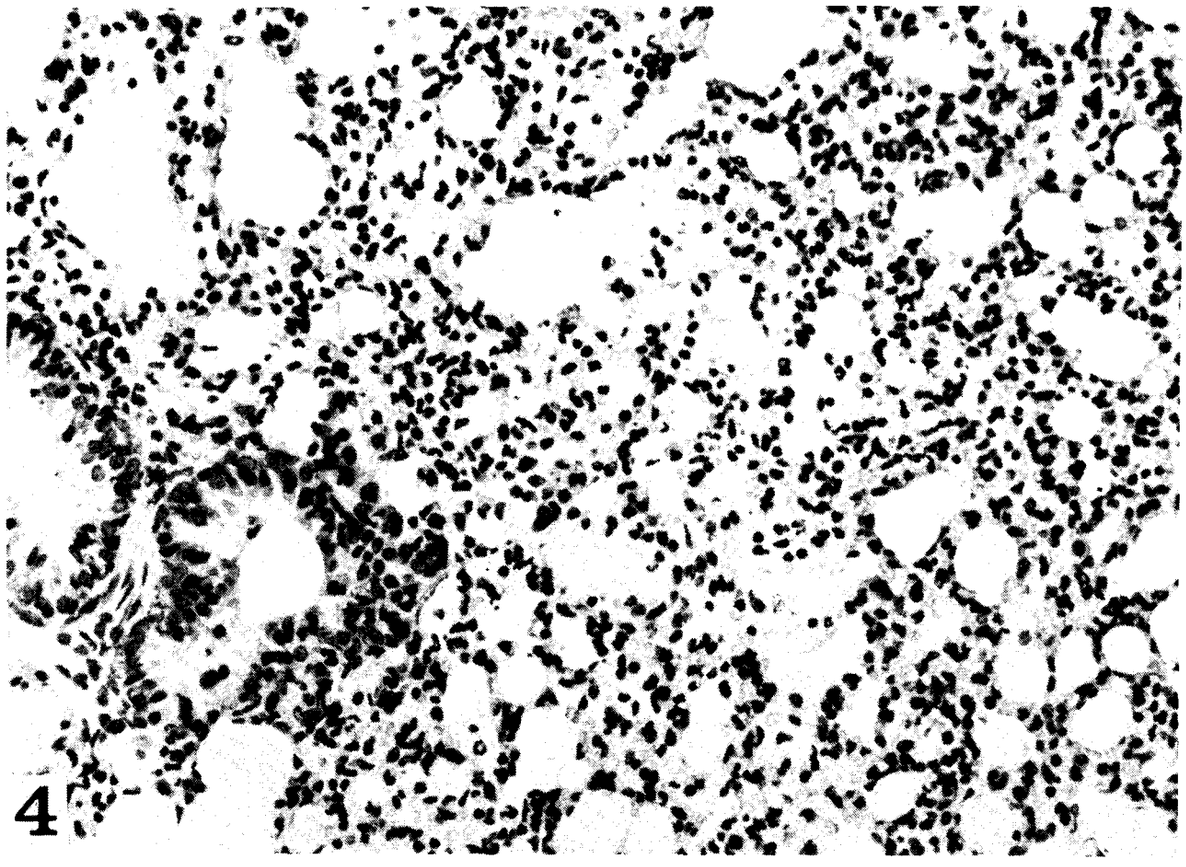

Fig. 4. Respiratory units filled with homogeneously eosinophilic fluid. (H&E, X200)

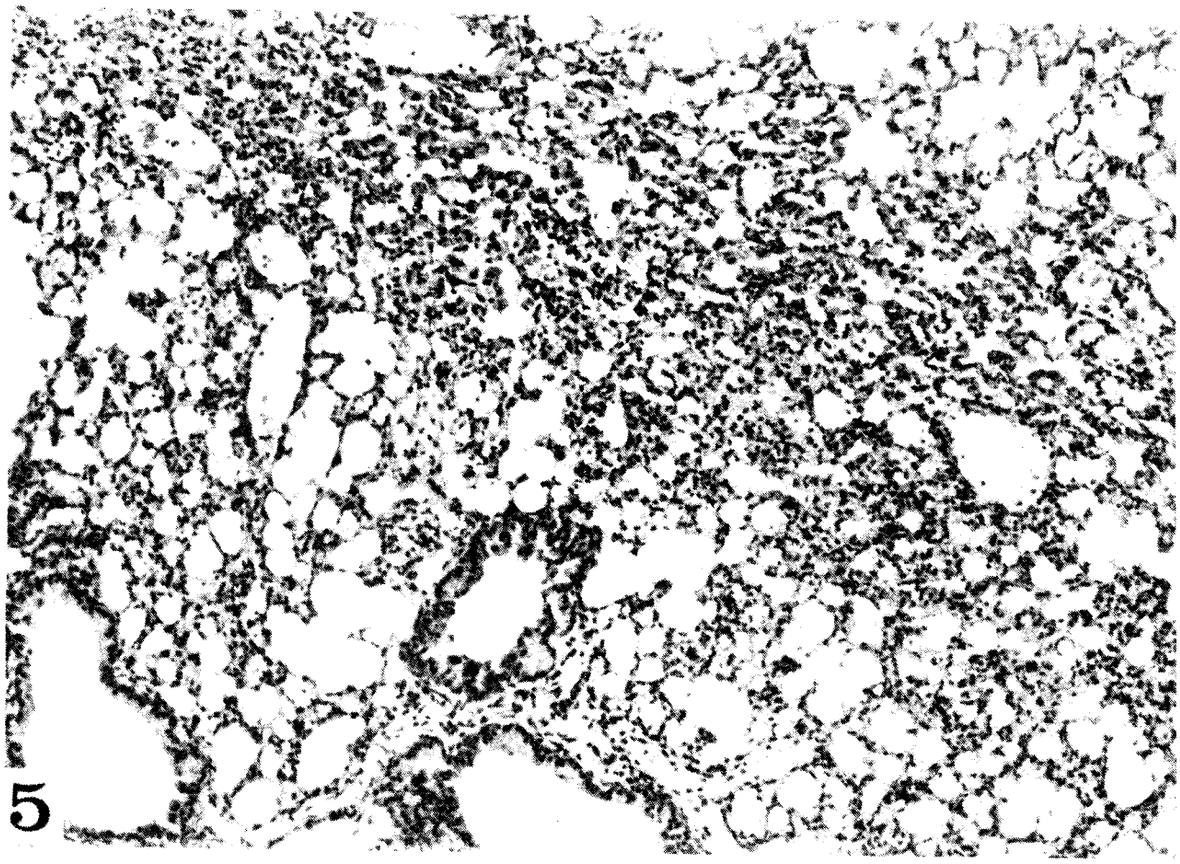

Fig. 5. Slit-like spaces representing localized atelectasis. (H&E, X100)

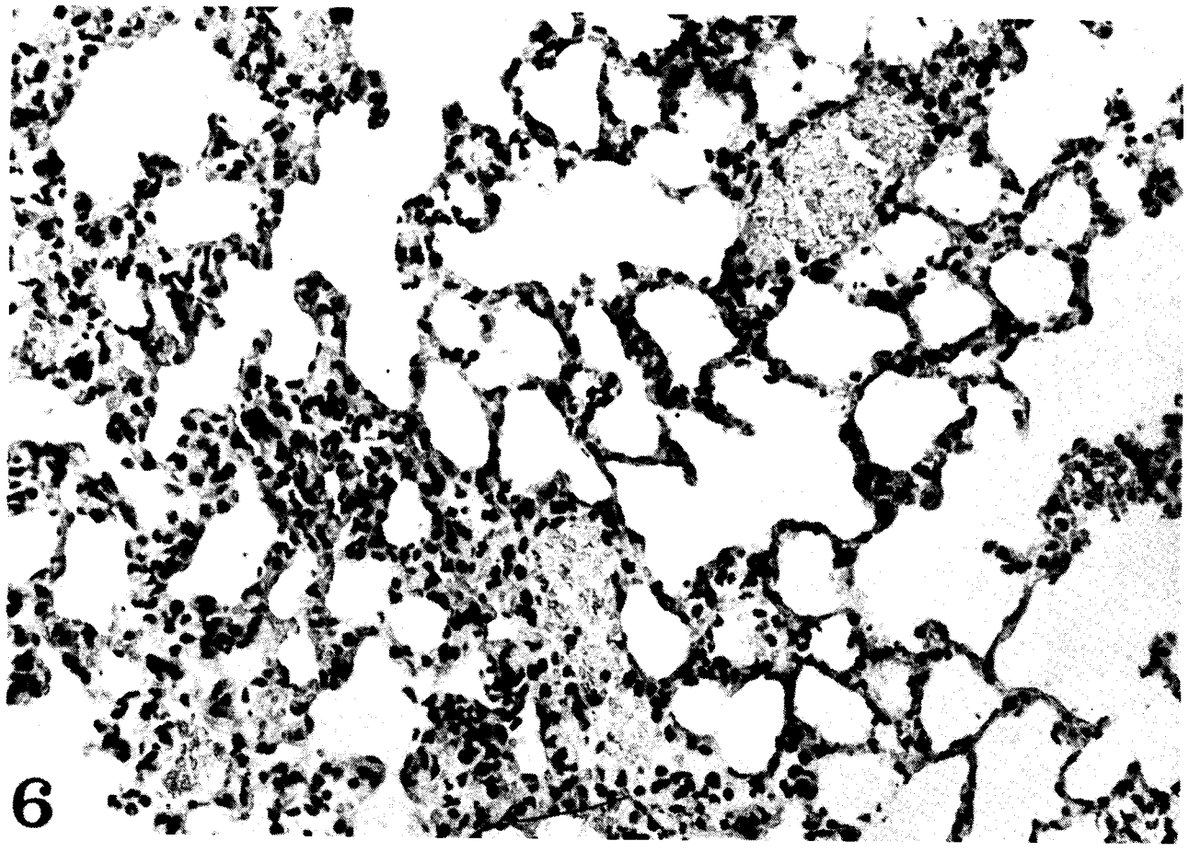

Fig. 6. Dilated arterioles and venules, as well as alveloar capillaries, with packed RBC's. (H&E, X200)

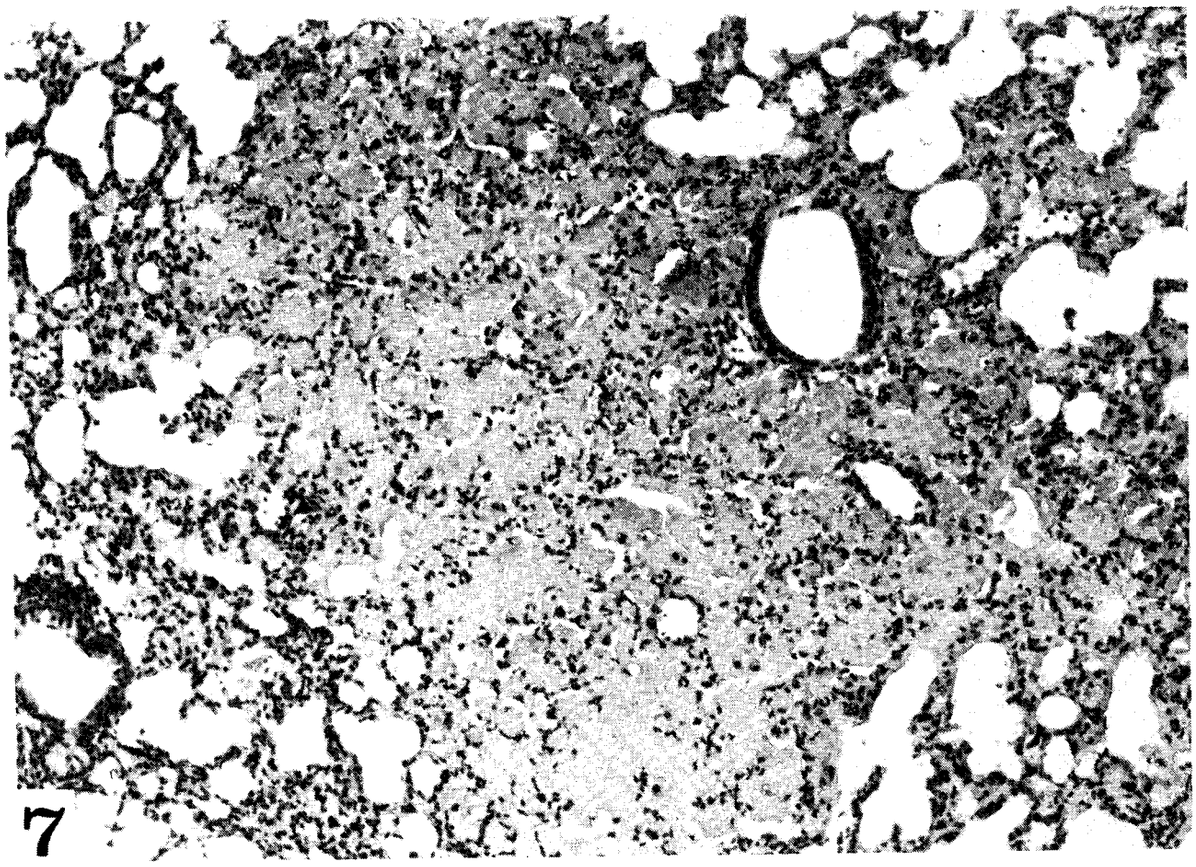

Fig. 7. Localized hemorrhage with clotted blood in alveolar spaces. (H&E, X100)

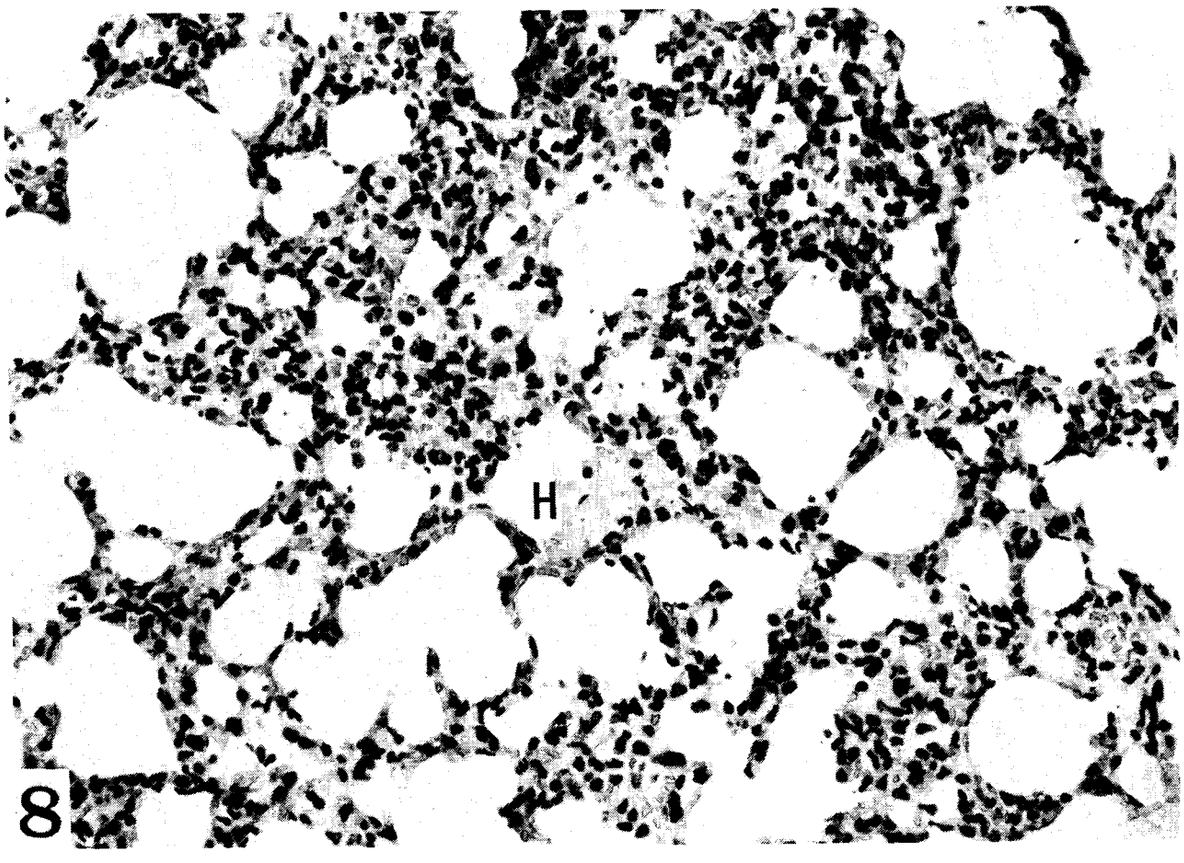

Fig. 8. An area showing dense hyaline membrane (H) in an alveolar sac. (H&E, X200)

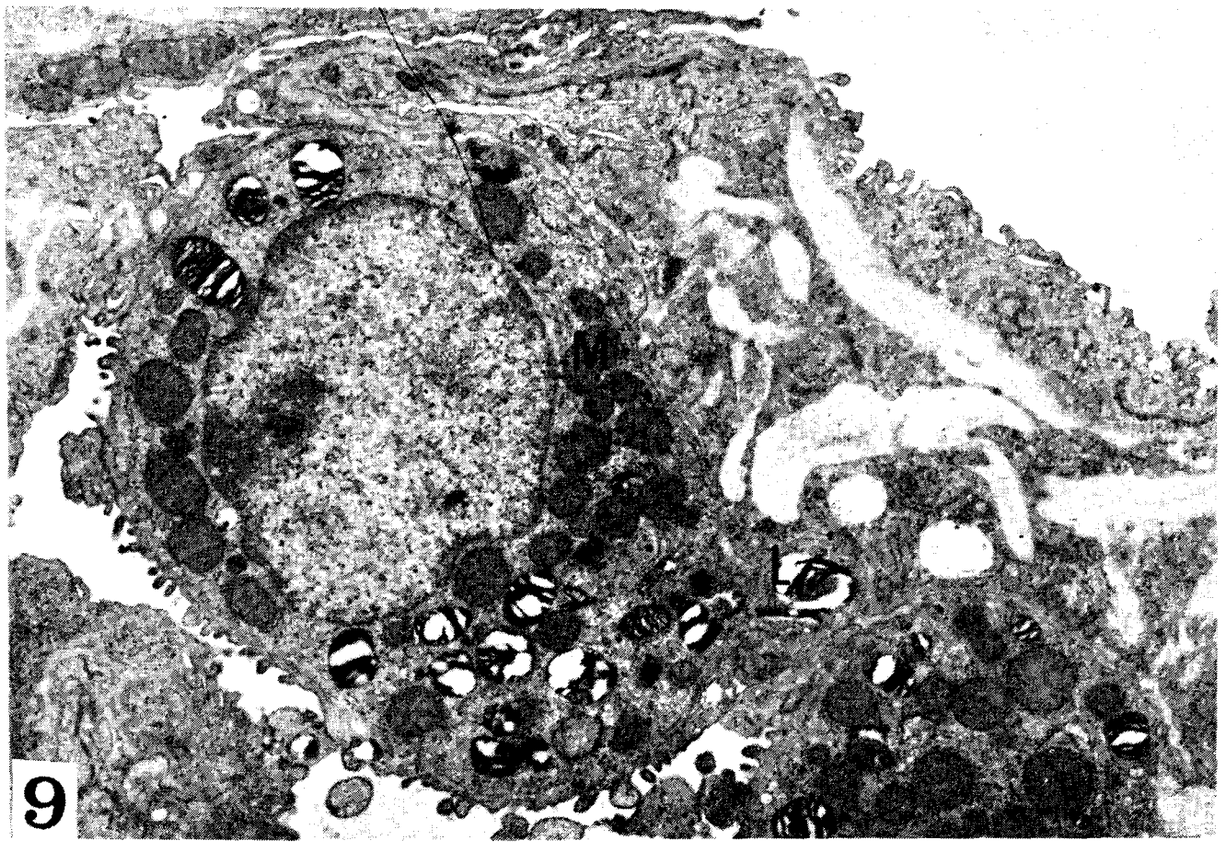

Fig. 9. Type II pneumocyte demonstrating swollen and degenerated mitochondrias (M). L; laminated electron dense materials (surfactant). (X3,000)

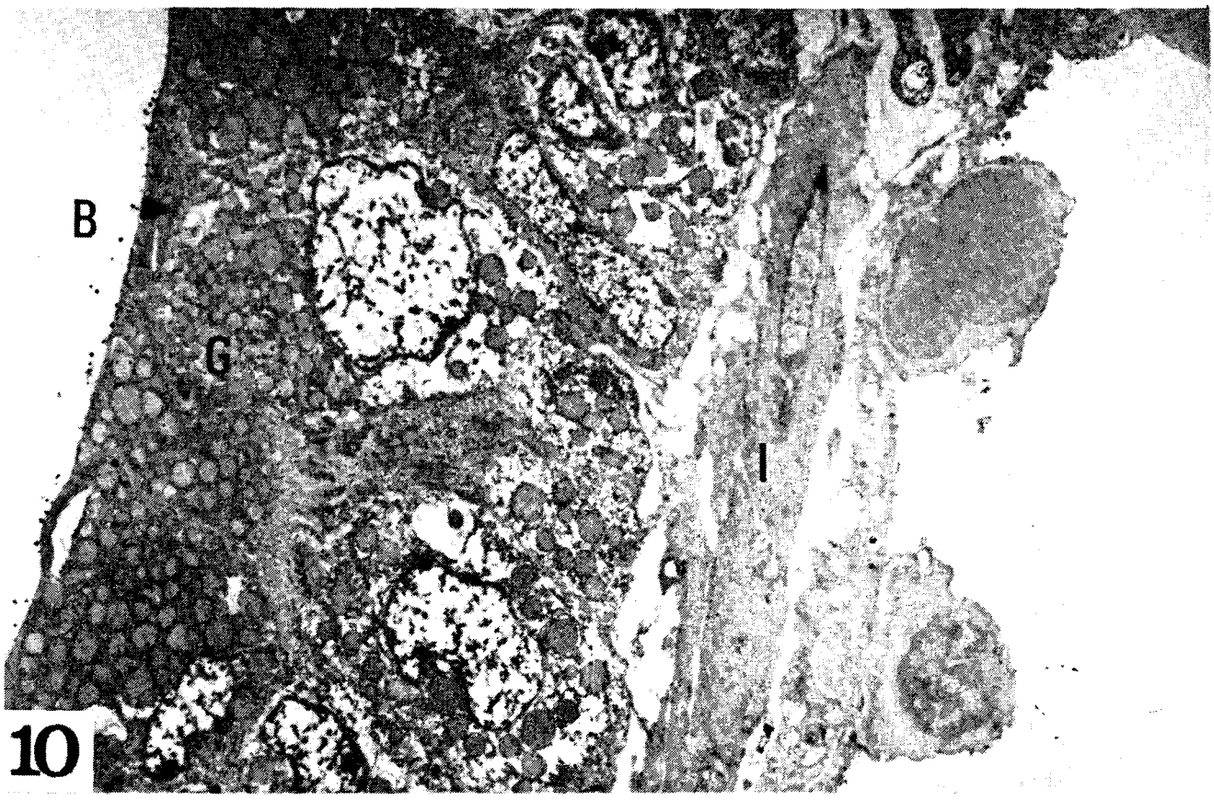

Fig. 10. Bronchiolar epithelia have lost their cilia on luminal surface(B), and show increase of secretory granules(G). Also noted are fluid collections around interstitial cell(I). (X1,500)

2) hypoxia and 3) toxicity of the inhaled smoke. The incidence of respiratory injury from flame heat is low. Moritz *et al.* (1945) proved that the temperature in dog's trachea reached 50°C maximally, which had respirated the dry air of 260°-280°C. The injury by steam heat, which carries the heat as 4,000 times as the dry air, does not occur frequently.

In fact the large portion of fire fatality are of carbon monoxide poisoning. Haplin *et al.* (1976) reported that more than half were proved to have the lethal level of carboxyhemoglobin and the half of remainders showed the poisoning of carbon monoxide and other diseases. As the affinity to hemoglobin of carbon monoxide is over 200 times greater than oxygen, the carboxyhemoglobin level reaches 50% in minutes at the concentration of 800 ppm in room air (Smith 1980). In this experiment, the concentration of carbon monoxide was at or over 1,000 ppm, when 0.5 gm of materials per unit liter of exposure chamber was burned. In general, the concentration of carbon monoxide elevated highest at 50°C below the natural flaming temperature of the materials, when the incomplete combustion took place abruptly. The high mortality obtained in carpet and curtain burning mainly depended on the pattern of carbon monoxide generation. And the LC_{50}'s of the synthetic fabrics were lower than of the lauan and plywood, with narrow safety margins which were similar with Alarie & Anderson's work(1979).

According to their mechanism of action, the toxic gases of fire site are classified as the asphyxiant and the irritant. Carbon monoxide, carbon dioxide, and hydrogen cyanide are asphyxiant. And for the irritant, gases of nitrogen compound as nitrogen dioxide, ammonia, hydrogen chloride, sulfur dioxide, isocyanates, and aldehydes are well-known. Their sources and inhalation effects can be summarized as Table 9. However, the actual levels of such gases at the fire site, besides carbon monoxide, were not fatal or morbid. Zawacki *et al.* (1977) reported that the actual concentration of oxygen was 17.7%, carbon dioxide, 0.94% and carbon monoxide, 0.19%. The aldehyde was 0.11% by Zikria *et al.* (1972). Terill *et al.* (1978) suggested that the repsiratory injury could resulted from the synergistic effect of smokes. Chu(1981) and Teixidor *et al.* (1983) indicated that such chemical burn occurred more in the relatively closed environment, and a close relationship between carbon monoxide poisioning and chemical intoxication. Their characteristics were summarized at Table 10., according to the time of symptom onset.

Many are known for the histopathologic changes on smoke inhalation injury. Among them, necrosis and edema of trachea and main bronchi, detachment of respiratory mucosa, interstitial edema, and congestion of alveolar capillary could be seen in initial stage of injury. Hemorrhage, presence of fibrin in alveolar spaces and localized atelectasis predominated in severe cases (Mellins and Park 1975; Sochor and Mallory 1963). Particularly, the necrotizing tracheitis and bronchitis were one of the main causes of death in smoke inhalation injury, in consequence of loss of ciliary movement, susceptability to infection, and increase permeability of vessels adjacent to the tracheobronchi (Loke *et al.* 1984; Thorning *et al.* 1982). The pulmonary edema, the initial finding of parenchymal injuries, developed at 6 to 72 hours after the smoke inhalation (Stone 1979). In this experiment, the patholo-

Table 9. Source and inhalation effects of thermocombustion gases*

Gas	Source	Effects of inhalation
Carbon monoxide	Incomplete combustion of all organic matter	Oxygen deprivation
Carbon dioxide	Complete combustion of organic materials	
Hydrogen cyanide	Wool, silk, nylons, polyurethane,,	Interferes w/ oxygen utilitzation at cellular level
Nitrogen dioxide	Cellulose nitrate, fabrics,,	Pulmonary irritation
Amionia	Wool, silk, nylon, melamine,,	Irriation to eyes and nose
Hydrogen chloride	PVC, acrylics,,	Respiratory irritant
Aldehyde	Wood, cotton, paper,,	Respiratory irritant
Sulfur dioxide	Compounds containing sulfur	Respiratoyr irritant

*; modified from Surveyer JA(1980), and Terrill JB, Montgomery RR, Reinhardt CF(1978).

Table 10. Characteristics of respiratory distress syndromes in fire victims classified by time of onset*

	Acute	Delayed	Late
Days	Less than 1	1 to 5	5 to weeks
Causes	Heat, smoke	Gases, sepsis	Pneumonia, aspiration, embolism
Symptoms	Dyspnea, stupor, wheezes	Tachypnea, ARDS	Various
COHb	High	Low or absent	Various
Pathology	URT damage, bronchiolitis	Congestion, edema	Various

*; modified from Zawacki BE, Jung RC, Joyce J, Rincon E(1977).

gic changes were mild, because of the short period of exposure and small amount of burned materials. Among them interstitial edema was outstanding, which resulted from the changes of capillary permeability and was accentuated by alveolar collapse due to decrease of surface tension (Head 1980). It is reported that the interstitial edema begins at the perivascular spaces (Teixidor *et al.* 1983). The degree of interstitial edema is assessed as the toxicity of the gases of materials (Peitzman *et al.* 1981; Transbauch *et al.* 1983). With the fair correlation between the degree of interstitial edema and the water content of lungs, it can be recommended for the indirect evaluation of acute lung injury.

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=국문초록=

燃燒가스 吸入이 肺에 미치는 影響에 關한 實驗的 硏究

서울大學校 醫科大學 病理學敎室

李允聖 · 咸毅根

火災時 有毒性 燃燒가스의 吸入은 肺를 위시한 呼吸器系에 刺戟과 損傷을 일으키며 이 吸入性 損傷은 燃燒材의 種類에 따라 差異가 있다. 본 硏究에서는 우리나라 建築資材의 主要 部分을 차지하는 羅王과 合板 그리고 合成 纖維材인 커튼지와 카페트 등 서로 成分이 다른 燃燒材로, 考案된 燃燒가스 吸入裝置를 使用하여, 可能한 熱과 無酸素性 窒息의 영향을 排除하고 각 燃燒材의 고유한 燃燒가스 依한 毒性의 程度, 病理組織學的 變化의 特徵 및 死亡의 樣相을 196마리의 '마우스'에서 觀察하여 다음의 結果를 얻었다.

1. 急性 死亡은 燃燒材의 種類에 따라 差異가 있었다.
2. 化學的 合成 纖維材인 커튼지와 카페트가 自然材인 羅王에 비하여 毒性이 강했으며, 이는 급격한 不完全 燃燒에 의한 一酸化炭素의 增加와 刺戟性 가스에 의한 損傷에 기인한다고 이해되었다.
3. 病理組織學的 所見은 燃燒材의 種類와 無關한 非特異的이었으며, 가장 현저한 病變은 肺胞壁 間質의 浮腫이었고, 肺胞內 浮腫, 局所的 無氣肺, 鬱血 및 硝子樣 膜의 出現등이 觀察되었다.
4. 乾燥法에 의한 肺의 水分含量의 測定은 肺浮腫의 評價에 좋은 客觀的 지표가 될 수 있었으며, 肺胞壁 間質의 浮腫 出現頻度와 비교적 좋은 相關關係를 보였다.($R=0.835$)($p<0.001$)

이상의 結果로 보아 肺에 미치는 燃燒가스 吸入의 効果는 현저하였으며 특히 急性 死亡의 경우 無酸素性 窒息이나 熱에 의한 직접 損傷이 아니고 一酸化炭素를 비롯한 燃燒材의 고유 有毒性 가스에 의한다고 이해되며, 肺의 主要 病理組織學的 所見은 肺浮腫, 特히 肺胞壁 間質의 浮腫으로 온다는 사실을 乾燥處理로 客觀化할 수 있었다.