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EXPERIMENTAL POST-TRAUMATIC LUNG INSUFFICIENCY IN DOGS

GROSS AND LIGHT MICROSCOPIC LUNG LESIONS*

By

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LIUM, B., O. D. SAUGSTAD, A. O. AASEN, I. GULDVOG, K. NORDSTOGA and E. AMUNDSEN: *Experimental post-traumatic lung insufficiency in dogs. Gross and light microscopic lung lesions.* Acta vet. scand. 1982, *23*, 118—127. — Post-traumatic lung insufficiency in dogs was induced by a combination of haemorrhagic hypotension and liver trauma. In one experimental group, lung biopsies were sampled every 4 h. This paper describes the development of gross and light microscopic lung changes in these dogs, and the lung lesions 12 h after initiation of the trauma in another experimental group. Incipient changes were recognized as early as 4 h after initiation of the trauma. Perivascular and intraseptal oedema, scattered microthrombi, and trapping of leucocytes in lung microvessels were the most conspicuous morphological findings. These changes grew gradually more pronounced towards collapse, when also areas with interstitial haemorrhages, intra-alveolar protein-rich oedema fluid with strands of a fibrinoid material, and an increased number of partly degenerated leucocytes were found in addition to atelectasis.

The morphological lung lesions in this experimental model are similar to post-traumatic shock lungs in human patients.

p o s t - t r a u m a t i c s h o c k ; l u n g s ; p a t h o m o r p h o l o g y ; l i g h t m i c r o s c o p y ; d o g s.

Acute respiratory insufficiency has, for many years, been a feared complication in patients apparently successfully resuscitated from various forms of non-thoracic trauma (*Simeone* 1968). Approximately one-third of the patients who die after extrathoracic injury do so as a result of progressive respiratory insufficiency (*Moore et al.* 1969). Diseases of different nature, such

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as septicaemia, fractures and burns have been suggested to initiate this syndrome (*Hopewell & Murray* 1976, *Blaisdell & Lewis* 1977). Despite extensive clinical and experimental investigations during the last 20 years, the pathogenetic mechanisms leading to pulmonary damage in post-traumatic patients are still a matter of dispute and poorly understood (*Blaisdell & Lewis, Amundsen* 1980). By means of the experimental model used in our investigation we were able to study the initial pathophysiology and pathomorphology following a standardized combination of hypotension and liver damage in anesthetized dogs (*Aasen et al.* 1980, *Guldvog et al.* 1980, *Saugstad et al.* 1980), The present paper describes in detail the gradual development of gross and light microscopic lung lesions in this model.

MATERIAL AND METHODS

Sixteen labrador retriever dogs of either sex (18—22 kg) were studied. Anaesthesia was induced by intravenous administration of sodium pentobarbital (Mebumal® 25 mg/kg body weight), and maintained by supplement with small doses during the experimental period. Lung insufficiency in Group I ($n = 4$) was induced according to *Porcelli et al.* (1974). The procedure included haemorrhagic hypotension of 50 mm Hg for 2 h, combined with occlusion of the hepatic artery, the portal vein, and the common bile duct for the first 20 min of the hypotensive period. In Group II experiments ($n = 7$) the procedure also included a left-side thoracotomy. These animals were intubated endotracheally and ventilated artificially with a positive end-expiratory pressure of 5 cm H_2O. Controls ($n = 5$) also anaesthetized with sodium pentobarbital, and the femoral artery and vein were cannulated. No further surgical procedure was performed. All dogs were placed on their right side. Animals in Group I and control dogs were studied for 12 h. Animals in Group II survived for 3—14 h, and they were all studied till circulatory collapse, i.e. blood pressure less than 30 mm Hg and pulse rate less than 70 beats/min. Haemodynamic measurements, blood gases and biochemical and haematological parameters were determined as reported elsewhere (*Aasen et al.* 1980, *Guldvog et al.* 1980, *Saugstad et al.* 1980).

When the thoracotomy was completed in Group II, the left lung was inspected at hourly intervals. From these dogs 2 lung biopsies were taken from the left diaphragmatic lobe immediately

before bleeding and clamping of the portal triad, and thereafter every 4 h, and at circulatory collapse. At the end of the experiments, the lungs from all dogs were removed. Two tissue samples for morphological examinations were immediately taken from standardized localizations in all lung lobes, and from additional localizations in cases of macroscopic lesions. From each sampling, one tissue sample was immersed into 10 % Carson's modified phosphate-buffered formalin, and the other into 33 % glutaraldehyde in 3 % macrodex and Millonigs phosphate buffer for at least 24 h. All the fixatives were cold (4°C). The lungs were then instantly fixed by infusion of glutaraldehyde into the bronchial system at a pressure of 15—20 cm H_2O and immersed into a container with the same fixative.

Formalin-fixed tissues were embedded in paraffin, sectioned at about 5 μm, and stained with haematoxylin and eosin (HE) and elastin van Gieson (e.v.G). Selected sections were also stained with phosphotungstic acid haematoxylin (PTAH), periodic acid-Schiff (PAS), and the Martius scarlet blue (MSB) methods. Glutaraldehyde-fixed samples were rinsed in Millonigs phosphate buffer overnight, post fixed for 1—2 h in 2 % phosphate buffered OsO_4, dehydrated and embedded in Araldite. Semithin sections, 1 μm, cut with glass knives on a Reichert OmU_2 ultramicrotome were stained by 0.5 % Toluidine blue for light microscopic examinations. From each biopsy semithin sections from at least 4 blocks were examined, and at the end of the experiments at least 10 blocks from each animal. From all dogs in Group I and 4 dogs in Group II at least 5 blocks from each lung lobe were studied. The major part of our light microscopic studies were performed on semithin sections.

RESULTS

Macroscopic examination

Controls. Lungs from control dogs were well inflated, and had a homogeneous, orangy-pink colour when examined at 12 h. In all lungs, however, a few subpleural bluish-red discoloured areas with a maximum extension of 0.5—1 cm were seen. There were no visible fluid increases in the airways.

Group I. The macroscopic lung changes in this group were moderate and consisted of a few scattered atelectatic areas dorsally, near the lung hilus. A faint, diffuse bluish-red discolouration and incipient consolidation were observed in 2 of the dogs.

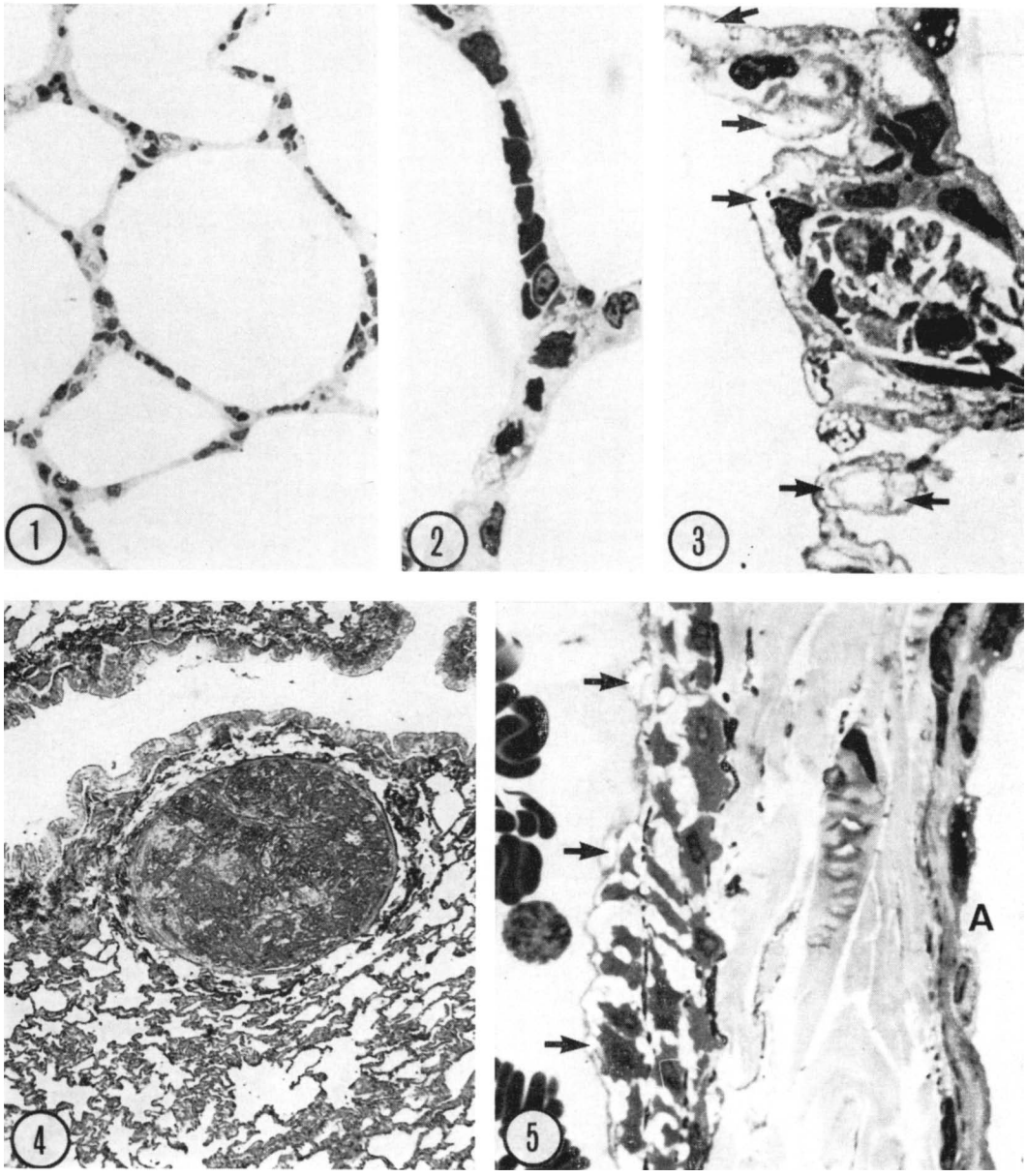


Figure 1. Normal alveolar septa and well inflated alveoli. Control dog. Semithin section, Toluidine blue, × 520.

Figure 2. Normal alveolar septa. Group II experiments, 0 h biopsy. Semithin section, Toluidine blue, × 1300.

Figure 3. Intravascular aggregate of platelets and leucocytes. Perivascular and intraseptal interstitial oedema (arrows). Group I experiments. Semithin section, Toluidine blue, ×1440.

Figure 4. A pulmonary vessel occluded by a thrombus stained as fibrin. Group I experiments, MSB, × 70.

Figure 5. A pulmonary vessel with vacuolated endothelial cells (arrows) and intramural oedema. Alveolar lumen (A). Group I experiments. Semithin section, Toluidine blue, × 1300

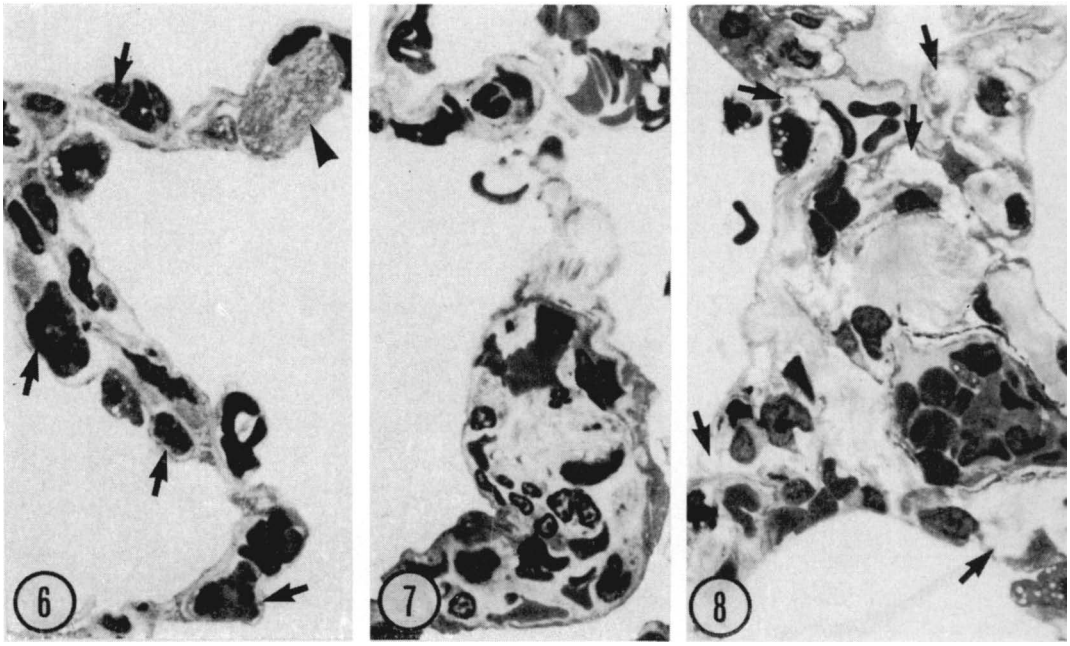
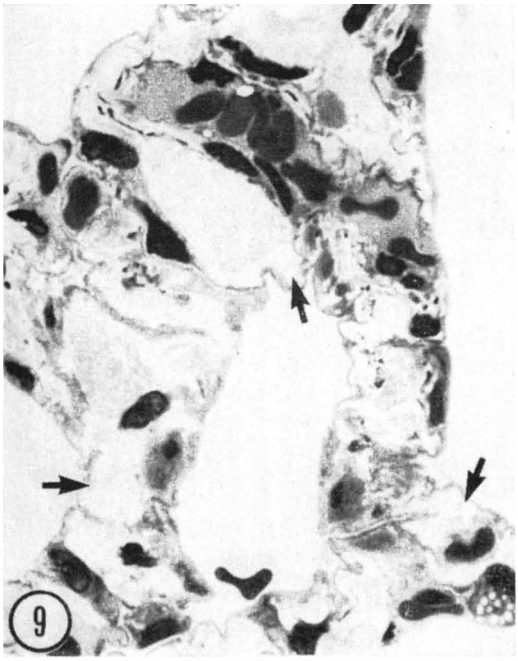
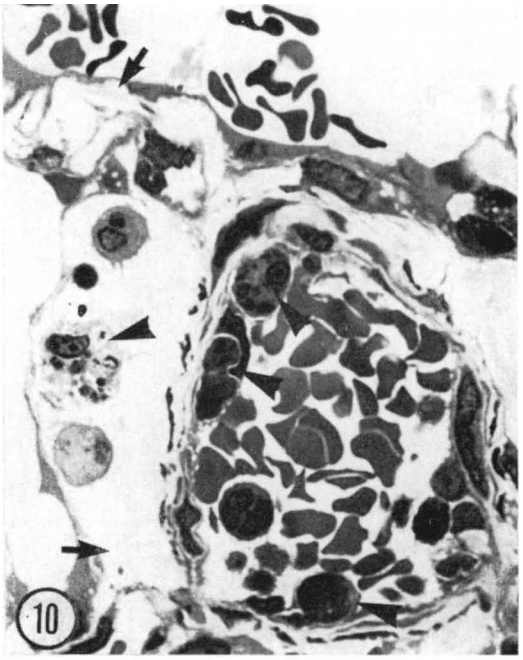


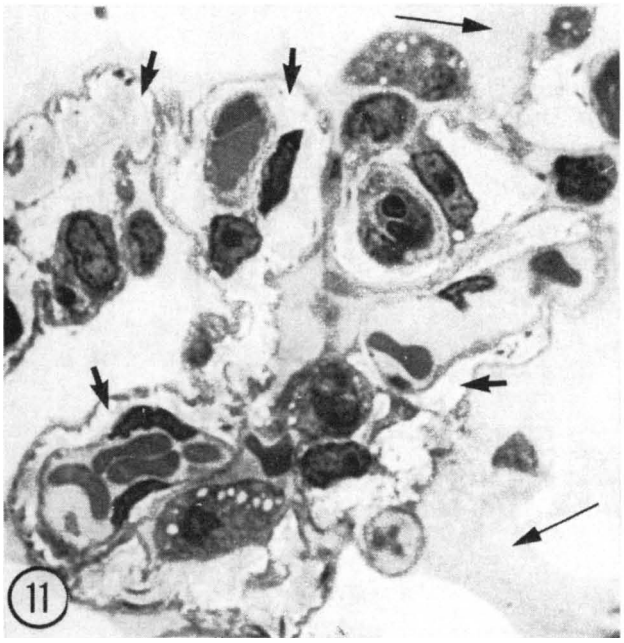
Figure 6. Alveolar septa with increased number of polymorphonuclear leucocytes (arrows) and a hyaline body (arrow head) in a respiratory capillary. Group I experiments. Semithin sections, Toluidine blue, × 1400.

Figure 7. A markedly dilated respiratory capillary occluded by aggregated leucocytes and a fibrillar material. Group II experiments, 4 h biopsy. Semithin section, Toluidine blue, × 1300.

Figure 8. A pulmonary arteriole partly occluded by aggregated leucocytes. Incipient intraseptal oedema is indicated by arrows. Group II experiments, 4 h biopsy. Semithin section, Toluidine blue, × 1000.







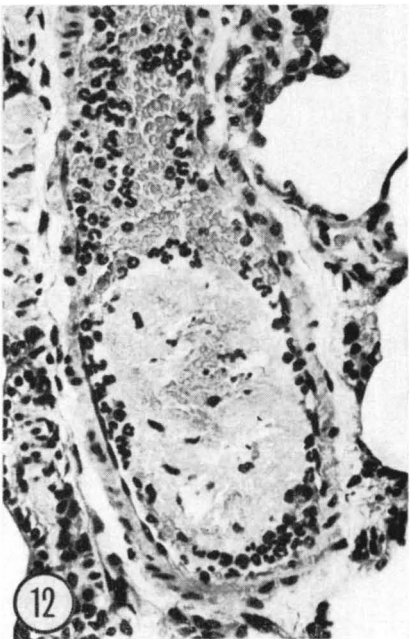
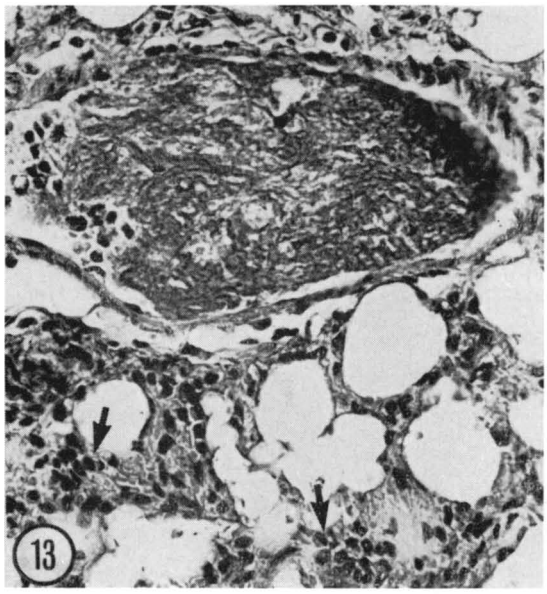


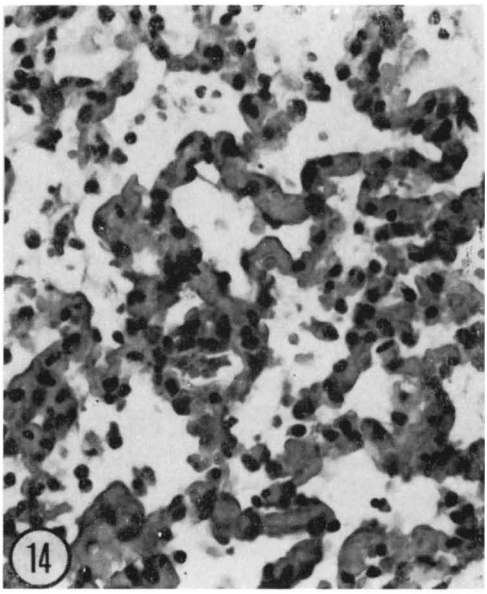
Figure 9. Thickened alveolar septa with evident interstitial and perivascular oedema indicated by arrows. Group II experiments, 4 h biopsy. Semithin section, Toluidine blue, × 1300.

Figure 10. A pulmonary arteriole with margination and extravasation of leucocytes (arrow heads). Perivascular and interstitial oedema indicated by arrows. Group II experiments, collapse 12 h. Semithin section, Toluidine blue, × 1300.

Figure 11. Thickened alveolar septa with marked interstitial oedema (thick arrows), and incipient alveolar oedema (thin arrows). Group II experiments, collapse 12 h. Semithin section, Toluidine blue, × 1700.

Figure 12. A pulmonary arteriole partly occluded by a hyaline to granular material and aggregates of red and white blood cells. Group II experiments, collapse 12 h, HE, × 325.





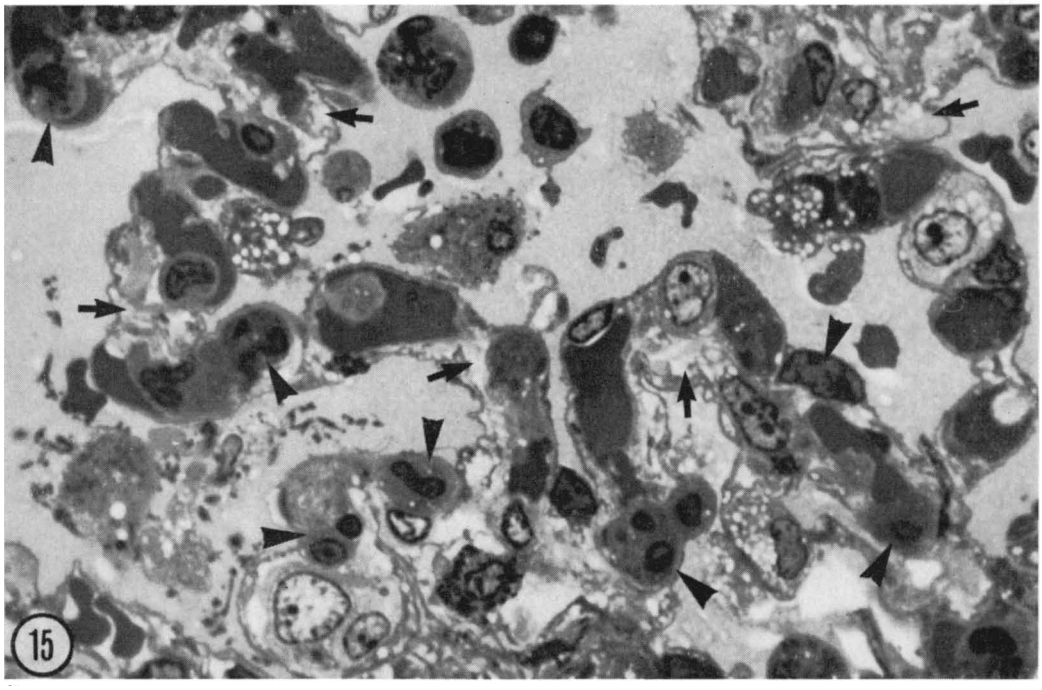


Figure 13. The pulmonary arteriole is occluded by fibrin, and the alveolar septa thickened and cellular. Microatelectasis is indicated by arrows. Group II experiments, collapse 12 h, MSB, × 325.

Figure 14. Dilated respiratory capillaries tightly packed with red blood cells. Group II experiments, collapse 12 h, HE, × 425.

Figure 15. Thickened alveolar septa with interstitial oedema (arrows) and distended respiratory capillaries occluded by leucocytes (arrow heads) and tightly packed red blood cells. A fine-granular fluid, partly degenerated leucocytes, and some few strands of a fibrinoid material (F) are seen in alveolar lumina. Group II experiments, collapse 12 h. Semithin section, Toluidine blue, × 1450.

Group II. Immediately before the hypotension and clamping procedure, the lungs completely filled the left thoracic cavity and showed a smooth and homogeneous orangy-pink surface. During the first 4 h the pink colour turned to a red discolouration, and the left lung volume was temporarily reduced. At 8 h parts of the left lung were discoloured bluish-red, and there were scattered petechial haemorrhages subpleurally. The lung surface had a glistening, oedematous appearance. The left lung volume increased again during the last 1—2 h prior to collapse.

At collapse the lungs were heavy with a glistening, smooth and bulging pleural surface, with a varying bluish-red to grayish discolouration. The changed areas showed no tendency to elastic collapse and no crepitation, and samples from these parts of the lungs sank in water. The cut surface revealed consolidated tissue with a liver-like appearance and consistency. Petechial haemorrhages were seen subpleurally and scattered throughout the parenchyma; the cut surface was glistening and oedematous. The haemorrhages and oedema fluid were particularly localized to the interstitium surrounding the bronchi and blood vessels, but no or only minor amounts of oedema fluid were found in the bronchi. Incipient signs of congestion and consolidation were found near the lung hilus and were distinctly more pronounced in dogs who lived for 8—12 h compared with those who died before 8 h. The periphery of most lung lobes frequently revealed emphysematous areas. In dogs surviving for about 12 h, haemorrhagic consolidation of entire lobes was sometimes seen. Gross pathological changes were most pronounced in the lower half of the lung.

Microscopic examination

Controls. Histological examination of lung sections from control dogs revealed normal structures (Fig. 1), except for moderately increased number of leucocytes in a few arteries and respiratory capillaries.

Group I. The most constant and predominant findings in lung sections from these dogs were perivascular and intraseptal oedema together with accumulations of leucocytes and platelets in arteries and respiratory capillaries (Figs. 3 and 6). Thrombosed arteries (Fig. 4) and perivascular haemorrhages were occasionally observed. High magnification of pulmonary vessels revealed vacuolization of endothelial cells and mural oedema

(Fig. 5). Respiratory capillaries were distended in some areas and showed moderate congestion. In addition fine granular or hyaline round to oval bodies were frequently seen intracapillarily (Fig. 6). Lung sections from all animals in this group showed incipient intra-alveolar oedema and moderate atelectasis.

Group II. Biopsies obtained at 0 h revealed mainly normal structures (Fig. 2), although local congestion and capillary dilatations were observed. Leucocytes were only occasionally seen in lung vessels and respiratory capillaries.

At 4 h leucocytes were more frequently seen. Occasionally aggregates of leucocytes and platelets occluding lung microvessels were found, particularly in arterioles (Figs. 7 and 8). Intracapillary hyaline bodies, as described for Group I, were seen in 2 biopsies at this time. Incipient perivascular oedema was demonstrated in most of the dogs, and in 3 of 7 animals the alveolar septa were thickened with moderate interstitial oedema (Fig. 9). Sections from all dogs revealed pleural thickening with oedema and infiltration of leucocytes.

Biopsies performed at 8 h showed lung changes similar to those described for biopsies at 4 h. The interstitial oedema and leucocyte as well as platelet aggregations were, however, more pronounced. Vacuolated endothelial cells and periarterial haemorrhages were occasionally seen, and alveolar lumina sometimes contained moderate oedema fluid and an increased number of free mononuclear cells.

Collapse. The most characteristic light microscopic lung alterations at collapse were perivascular oedema and marked intra-arterial and intracapillary trapping and margination of polymorphonuclear leucocytes (Fig. 10). Perivascular lymphatics were dilated, and alveolar septa markedly thickened with distinct interstitial oedema (Fig. 11). Arteries and arterioles occluded by amorphous masses of a granular, eosinophilic material containing red and white blood cells were observed in one or more sections from all dogs (Fig. 12). Some of these thrombi showed a positive stain for fibrin (Fig. 13). A varying degree of congestion was seen in all lungs, and periarterial haemorrhages frequently observed in most of them. Vacuolated endothelial cells and mural oedema were occasionally seen. Intracapillary hyaline bodies, as described for Group I, also occurred at collapse. In some areas the respiratory capillaries were dilated and tightly packed with red blood cells and/or leucocytes (Figs. 14 and 15). Interstitial

and intra-alveolar haemorrhages were common in these areas, together with strands of a fibrinoid material and an oedema fluid that was densely eosinophilic, indicating high protein concentration. In addition polymorphonuclear leucocytes and macrophages with swollen and vacuolated cytoplasm were found in alveolar lumina (Fig. 15). In all these dogs the pleura of the left half was oedematous with fibrin deposits, infiltration of polymorphonuclear leucocytes, and focal haemorrhages.

DISCUSSION

This report clearly shows that a combination of hypotension and clamping of the portal triad in dogs initiates morphological lung changes similar to those described in human post-traumatic shock lungs (*Mittermayer et al.* 1977, *Missliwetz et al.* 1978, *Sandritter et al.* 1978).

The most characteristic light microscopic lung changes in both experimental groups were interstitial oedema and trapping of leucocytes in lung microvessels. In Group II experiments, the gradual development of these morphological changes were seen, starting with incipient perivascular and intraseptal oedema recognized in biopsy specimens obtained at 4 h after the initiation of the trauma. As demonstrated in this investigation, semithin sections stained with Toluidine blue were very suitable for detecting intraseptal oedema, leucocyte aggregates, and platelet thrombi. Conventional 5 μm sections stained with HE, were, however, unfitted to demonstrate early accumulation of oedema fluid within alveolar septa. Since most morphological descriptions of shock lungs are based on traditional light microscopy, we believe that this may have misled some authors into underestimating the significance of increased vascular permeability and intraseptal oedema in the initial stage of post-traumatic shock lungs. The site of fluid leakage in pulmonary oedema is disputed, and probably varies with the model used (*Staub et al.* 1967, *Ratliff et al.* 1970, *Gil* 1971, *Cunningham & Hurley* 1972). In the present experiments, oedema of the perivascular and peribronchial connective tissue seemed to occur concurrently with intraseptal oedema. Even not proven by tracer techniques, we believe that increased permeability in respiratory capillaries was one of the initial events landing to the development of shock lung in our experimental models. The occurrence of protein-rich intra-alveolar fluid and fibrin depositions, together with interstitial and

intra-alveolar haemorrhages and leucocytes infiltrations, are consistent with an increase in capillary permeability. This does not, however, preclude that increased permeability of pulmonary or bronchial arterioles and venules may occur simultaneously. The observation of vacuolated endothelial cells and intramural oedema supports this possibility. Perivascular oedema is thought to interfere with vascular resistance and the distribution of blood flow (*West et al.* 1965) and may have contributed to the increase in pulmonary arterial resistance observed in our experiments (*Saugstad et al.* 1980).

Interstitial oedema is, by itself, not expected to contribute essentially to changes in the arterial blood oxygen tension (*Wischer et al.* 1956, *Said et al.* 1964). This is supported by our experiments. A marked decrease in oxygen tension was only seen terminally in Group II (*Saugstad et al.*) and corresponded in time to the occurrence of intra-alveolar fluid and marked terminal consolidation of the lungs. It is reasonable to assume that the intra-alveolar oedema may have been more pronounced than was suggested from our histological examination. Only fluid with a high protein content is stainable with HE, and visible in the light microscope. In addition, intratracheal fixation and routine histological procedure are known to wash out some of the intra-alveolar fluid.

Many authors suggest that microembolization is the primary pathogenetic factor in human post-traumatic shock lungs (*Cheney et al.* 1978, *Saldeen* 1979). This study showed that thromboemboli of arteries and arterioles occurred in our shock models, and they may have contributed to the increase in pulmonary arterial resistance. Such thrombosed vessels were, however, only occasionally found, and a simple mechanical action of the thromboemboli could hardly account for all the lung changes observed. The pulmonary vascular reserve should be more than enough to compensate for these obstructions (*Hyland et al.* 1963). In a recent report *Cheney et al.* (1978) concluded that anatomical arteriovenous shunting does not contribute significantly to the disturbance of gas exchange after pulmonary microembolism in dogs, and they suggested that pulmonary oedema is the major cause of hypoxia seen in such dogs. Thrombi may, however, cause local damage of the arterial walls and thereby contribute to the periarterial haemorrhages seen in our experiments. It is suggested that perivascular haemorrhages are particularly prone to occur

following lysis of thromboemboli and resumption of blood flow (*Blaisdell & Lewis* 1977).

Our study clearly shows that polymorphonuclear leucocytes are trapped in the microvessels of the lungs after hypotension and liver trauma in dogs. This might initiate other morphological changes. Degenerating leucocytes release substances that may cause endothelial and interstitial damage (*Cohn & Hirsch* 1960, *Janoff & Zweifach* 1964, *Turino et al.* 1974). Leucocyte aggregates may also contribute to anatomical shunting of blood and thereby to increased resistance in lung vessels. The role of leucocytes will be the subject of further studies in this experimental model.

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SAMMENDRAG

Eksperimentell post-traumatisk lungesvikt hos hund. Makroskopiske og lysmikroskopiske lungeforandringer.

En kombinasjon av blødningshypotensjon og leverskade ble benyttet for å framkalle post-traumatisk lungesvikt hos hund. I en forsøksgruppe ble det tatt biopsier hver 4. time. Denne artikkelen beskriver utviklingen av makroskopiske og lysmikroskopiske lungeforandringer hos disse hundene. Dessuten beskrives lungeforandringene 12 timer etter initiering av leverskaden i en annen forsøksgruppe. Morfologiske forandringer i lungene ble observert allerede etter 4 timer. De mest framtredende forandringene var ødem i alveolesepta og i bindevevet rundt kar, spredte hyaline mikrotromber og ansamlinger av polymorfkjernede leukocytter i små lungekar. Forandringene ble gradvis mer uttalte fram mot kollaps, da det i tillegg såes interstitielle blødninger, proteinrik ødemvæske med fibrinoid materiale og økt antall, delvis degenererte leukocytter i lumen av alveolene. Det var dessuten betydelig atelektase ved kollaps.

Det foreliggende arbeidet viser at en kombinasjon av hypotensjon og leverskade hos hund resulterer i lungeforandringer som i stor grad ligner post-traumatisk sjokklunge slik den beskrives hos menneske. Den patogenetiske betydning interstitielt og alveolært ødem, mikrotromber og leukocyttaggregering har for utvikling av „sjokklunge" diskuteres.

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