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EFFECT OF WATER EXPOSED TO A MAGNETIC FIELD ON STRUCTURAL CHANGES IN THE PULMONARY ALVEOLI OF ADULT RATS INDUCED BY HYPOXIA

Thesis for the degree of Doctor of Medical Sciences

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1. Introduction

Effect of a magnetic field on the body

Modern humans are increasingly exposed to magnetic fields generated by various devices of modern technology. Researchers at many prominent centers and foundations, such as the Biomagnetic Research Foundation at Evanston University (USA), the Agency Hydrographic Center in Washington, and the American Food and Drug Administration (USA), focus their attention on assessing the mechanisms and degree to which magnetic fields affect the homeostasis of living organisms. However, systematizing research findings encounters numerous difficulties because successive experiments sometimes use criteria that are not comparable (2, 6, 29, 30, 45, 73, 74).

The results of numerous theoretical, empirical, and clinical studies demonstrate that the cell membrane, as well as the entire system of intracellular cytomembranes, is the principal site through which an electromagnetic field acts on a living organism. The effects of this action are associated with intracellular Ca2+ ion flow. Exposure of cells to a magnetic field changes the structure of the cell membrane and reduces electrical potentials, which is associated with changes in its conductivity and permeability (4, 6, 11, 20, 28, 31, 32, 54).

The use of AFM (Atomic Force Microscopy) enables three-dimensional visualization of changes occurring in cell structure. Following an initial regression of microvilli, some cells show smoothing and flattening of the microvilli, followed by the formation of invaginations, and the cells gradually lose their spherical shape. Analysis of the distribution of actin filaments within the membranous cytoskeletal framework demonstrated marked nonhomogeneity within the cytoplasm and the formation of clusters immediately beneath the cell membrane. Depolymerization of actin fibers was also observed,

which is probably responsible for the reduction in cell height and the disappearance of microvilli (49). During exposure, the degree of cell-membrane hydration and the phosphorylation of specific intracellular filament proteins also changed (68).

A number of studies have addressed the effect of magnetic fields on cell proliferation. Among other findings, reduced incorporation of H3-thymidine into lymphocytes and a lower proliferation index were observed when lymphocytes were exposed to the field, together with repeated cycles of inhibition and acceleration of cell proliferation. A probable explanation for this phenomenon appears to be the influence of the magnetic field on the intrinsic biological rhythms of cells. An external stimulus in the form of energy may shift the phase of the cellular rhythm and consequently disrupt the process of proliferation (40, 43, 55, 69).

The effects of both alternating and static magnetic fields on cell adhesion have also been documented. Cells in culture that would normally adhere to the substrate remained freely suspended in the solution after the experiment, whereas cells that had been allowed to adhere before exposure to the magnetic field detached from the substrate under the influence of the field. In addition to disturbances of cell adhesion, extensive structural changes were observed, similar to those caused by ionizing radiation and chemotherapeutic agents. These changes consisted of shrinkage and granulation of the cytoplasm and, above all, involved the cell nucleus, including DNA granulation with migration toward the peripheral parts of the nuclei, which became fragmented and consequently led to cell death. These changes could indicate induction of apoptosis (12, 24, 26, 40, 48, 60).

On the other hand, when a static magnetic field was applied to cell lines in which cell death is associated with intracellular calcium-ion concentration, survival and replication were observed in cells damaged by external factors that induce apoptosis. At the same time, this result was not caused by delaying apoptosis or by changing the nature of cell death. The above effect was associated with an increase in the flow of Ca2+ ions from the extracellular environment into the cells (25, 44).

Attempts to explain the observed effects of magnetic-field exposure encounter many difficulties, mainly related to experimental methodology and the lack of highly sensitive measuring devices. Most probably, the observed result reflects the combined activation of many mechanisms at the molecular level (42, 64).

Body fluids such as blood, lymph, and cerebrospinal fluid are electrolyte solutions. An alternating magnetic field generates an alternating voltage (dependent on the area exposed to the field and on its frequency) and induces an electric field, which excites or accelerates ion movement. The flow of ions increases intermolecular friction, which in turn acts against the force of the field. Ions moving in solution are also acted upon by the Lorentz force, which causes ions of opposite charges to flow in opposite directions. In the presence of an alternating magnetic field, this leads to ion oscillation and vibration of semipermeable membranes, thereby facilitating and intensifying diffusion. The induction and summation of micropotentials lead to activation of neuronal pacemakers (10, 16, 17, 72, 73, 84, 88).

Many structural substances of living organisms have diamagnetic or paramagnetic properties; therefore, the action of a magnetic field modifies the quality and quantity of intermolecular bonds and also

affects the processes involved in forming apoenzyme-coenzyme-substrate complexes. The electromagnetic field probably also has the ability to generate piezoelectric currents in the body (57, 87, 88).

Water is essential for the proper course of most reactions occurring in living organisms, serving not only as the reaction medium but also as a substrate. It has been demonstrated that water changes its physical properties under the influence of a magnetic field (27, 88).

Spectrographic analysis of water exposed to an electromagnetic field showed that, depending on the type of field applied, absorption occurred in the 200-400 nm range of the UV spectrum. Water not exposed to the field shows no absorption properties. The electromagnetic field probably affects water dipoles, modifying the formation of bonds between electrons of individual molecules. In a sense, the structure of water in the liquid phase becomes more ordered. This effect causes the modified structure to persist for many hours after exposure (5, 66).

Water from a normal water-supply system exposed to an external magnetic field shows accelerated crystallization and an increased concentration of dissolved gases (mainly O2). Sedimentation and coagulation processes are accelerated; pH also changes, water viscosity decreases, and electrical conductivity is altered. The use of water prepared in this way, for example for irrigation, results in a 20-40% increase in plant growth and also accelerates the proliferation of microorganisms in culture (37, 82, 88).

Hypoxia and pulmonary microcirculation

The interalveolar septa separate the air spaces of the pulmonary alveoli from one another. Gas exchange between air and blood takes place at this level. The cells lining the pulmonary alveoli are closely apposed and rest on a basement membrane. Capillaries run within the septa and are lined by endothelium with its own basement membrane. In some areas, the adjacent basement membranes of the respiratory epithelium and capillary endothelium form a continuous layer. The septa also contain fibroblasts, leukocytes, macrophages, mast cells, individual contractile cells containing actin and myosin, as well as nerve, elastic, and collagen fibers. The alveolar epithelium consists of type I pneumocytes, covering 95% of the alveolar surface, and type II pneumocytes located between them.

The pulmonary microcirculation comprises arterioles (the smallest arteries), metarterioles, capillaries, and venules (postcapillary veins). Capillaries constitute the part of the pulmonary vascular system responsible for gas exchange. At branch points from the precapillaries there are clusters of myocytes forming precapillary sphincters, which regulate blood flow through specific areas of the capillary bed. The capillary endothelium forms part of the blood-air barrier. Endothelial cells rest on the basal lamina, which encloses a small number of pericytes (23).

Hypoxia contributes to increased activity of Hypoxia-Inducible Factor (HIF-1), which activates a number of genes leading to the expression of protein substances such as erythropoietin, Vascular Endothelial Growth Factor (VEGF), heme oxygenase type 1,

and inducible nitric oxide synthase. All of these subsequently participate in physiological and pathophysiological processes occurring in hypoxic lungs (58, 63, 75, 77, 81, 90).

VEGF activates endothelial cells by increasing P-selectin expression and facilitating adhesion of polymorphonuclear granulocytes (89). It also promotes the formation of fenestrae and the development of a new cellular organelle, the vesiculo-vacuolar organelle (34, 52). In addition to increasing the permeability of endothelial-cell membranes, these processes contribute to desquamation of the bronchial epithelium, increased migration of neutrophils into lung tissue, and enhanced degranulation of mast cells. The latter, clustered around venules and nerve endings, secrete prostaglandin E2, serotonin, and histamine, regulate vascular smooth-muscle tone, and modulate neural transmission (58). Chronic exposure to hypoxia increases the amount of laminin and fibronectin in the endothelial basal lamina and in the media of large and small pulmonary arteries (8, 23).

Studies of Peruvian Indians living at an altitude of approximately 4,330 m above sea level showed that their pulmonary arterioles have a thick media composed of two elastic laminae, between which lies a layer of circumferentially arranged smooth-muscle cells. Further studies of residents of La Paz documented the presence of muscle cells also in the intima of small arteries and arterioles of the alveolar septa. Similar changes can also be found in patients who do not live at high altitude but who developed hypoxemia as a result of chronic obstructive pulmonary disease (COPD). Thus, a common feature of all these cases is a well-developed smooth-muscle layer in the arterioles of the alveolar septa (39, 46, 56, 77).

The extent of vascular changes and the symptoms of primary pulmonary hypertension are determined to a large degree, in addition to the duration and intensity of the stimulus, by the age of the individual exposed to hypoxia. If exposure occurs during the perinatal or neonatal period, the response to the stimulus is much greater, despite its lower intensity, than in individuals exposed to hypoxia in adulthood. These changes do not regress completely even after physiological conditions are restored. An almost continuous layer of atrophic myocytes and the internal elastic lamina remain. These permanent alterations are of considerable importance in the development of a strong vasoconstrictor response of the pulmonary vessels in adult individuals re-exposed to hypoxia (1, 7, 9, 13).

Growth and proliferation of pulmonary vascular myocytes are induced and controlled by a system of mediators and receptors located on the surface and within smooth-muscle cells. Growth factors originating from endothelial cells and also causing vasoconstriction probably play the greatest role. There is therefore a correlation between muscle-cell proliferation and vascular contraction. The best characterized factors are endothelin-1 (ET-1) and its isoforms (ET-2 and ET-3), and platelet-derived growth factor (PDGF). In addition to hypoxia, stretching of the vessel may act as the primary stimulus for their action. Their probable antagonists are natural vasodilators: nitric oxide (NO) and atrial natriuretic factor (ANF) (3, 70, 71, 79, 80).

The mechanism by which hypoxia leads to proliferative processes in the lungs remains unknown. In addition to forces stretching the lung tissue and dilating the vascular bed, reduced concentrations of tissue growth inhibitors and increased concentrations of autocrine, paracrine, and endocrine growth factors are probably also important (22, 35, 36, 70, 71).

ET-1 and ET-3 are highly expressed in lung tissue, indicating their importance in the regulation of pulmonary vascular tone (21, 53, 76, 78). Exposure of adult rats to hypoxia increases mRNA expression for preproendothelin. This increase, however, is independent of whether the animals had already been exposed to hypoxia during the perinatal period (41). It appears likely that ET secreted by endothelial cells acts not only in a paracrine manner on myocytes but also in an autocrine manner on endothelial cells. The mechanism of endothelin-mediated vasodilation would therefore be associated with the release of nitric oxide and prostaglandins from endothelial cells (1, 41). The observed arterial vasoconstriction is associated with the release of endogenous endothelins acting through the endothelin receptor (ETB). Increased nitric oxide (NO) production under hypoxic conditions is also induced through the ETB receptor (90).

Nitric oxide (NO) derived from endothelial cells is released both into the vascular lumen, where it inactivates platelets, and outside the lumen, where it causes relaxation of vascular smooth muscle. It also affects the expression of genes responsible for the synthesis of growth factors such as VEGF and PDGF, endothelial adhesion molecules, and factors responsible for vasoconstriction, such as endothelin. Continuous inhalation of NO by rats under conditions of chronic hypoxia inhibits the development of pulmonary hypertension and partially prevents remodeling of pulmonary vessels (1, 38).

In rats that develop pulmonary hypertension as a result of chronic hypoxia, increased expression of eNOS (endothelial NO synthase) and consequently increased NO production are observed (47).

Disturbances of NO synthesis are therefore both a cause and a consequence of vascular changes in the lungs. However, the progression of pulmonary hypertension appears to depend on the NO concentration (65).

In the walls of small pulmonary arteries in rats with hypoxia-induced pulmonary hypertension, expression of angiotensin-converting enzyme (ACE) increases. Studies assessing the degree of H3-thymidine incorporation into isolated smooth-muscle cells of peripheral pulmonary arteries in rats demonstrated an increase under the influence of angiotensin II (ANG II). On this basis, it may be suspected that local release of ANG II in pulmonary vessels affects vascular remodeling (33, 59, 61).

Various stimuli leading to increased pressure in the pulmonary circulation cause remodeling of the pulmonary vessels. Studies evaluating the degree of regression of these changes after restoration of physiological conditions demonstrated mechanisms such as regression of pulmonary vascular-cell hyperplasia, apoptosis, reduced production, or even degradation of extracellular matrix substances (19, 50, 85, 86).

In rats exposed to chronic hypoxia, after the exposure was discontinued the content of fibronectin and laminin in the walls of pulmonary vessels decreased. Changes in the arrangement of elastic-membrane fibers were also observed.

These observations demonstrate the reversibility of vascular changes that developed under the influence of hypoxia-induced pulmonary hypertension.

2. Rationale and aim of the study

The present study is intended to provide an ultrastructural assessment of the morphological elements comprising the microcirculation of lung tissue, with particular emphasis on the vascular endothelium and the epithelium lining the pulmonary alveoli in animals given water exposed to an external magnetic field.

On the basis of earlier studies (37) using appropriately prepared water, a beneficial effect on metabolic processes occurring in the liver of experimental animals was found. This was also reflected in the assessment of hepatocyte ultrastructure. Normal compartmentalization of the cytoplasm, model organization of cellular organelles, and the absence of any signs of destructive effects indicate an unequivocally beneficial influence on hepatocyte structure.

In recent years, the development of methods using magnetic fields in clinical studies has repeatedly supplemented or replaced conventional pharmacological therapy.

Not all mechanisms of magnetic-field action that produce beneficial effects in the treatment of certain disorders have been fully elucidated. Nevertheless, there is extensive literature concerning the beneficial effects of magnetic fields. Among numerous reports, particular emphasis should be placed on results obtained in the treatment of wound healing, burns, leg ulcers, trophic skin lesions, and in osteosynthesis in cases of delayed bone union or pseudoarthrosis. The positive effect of magnetic fields on the circulatory system through the observed vasodilatory effect should also be taken into account (15).

It cannot be ruled out that, at the molecular level, these effects may result from interaction of the field with diamagnetic molecules and paramagnetic atoms of water exposed to a magnetic field,

which, as the most common solvent and an ideal semiconductor, is known to acquire exceptional properties of proton and electron recombination under the influence of a magnetic field.

Basic research analyzing the effects of so-called magnetized water is important, among other reasons, because magnetizers intended to improve the quality of drinking water are becoming increasingly widespread.

However, the question may always arise whether consumption of water prepared in this way entails any adverse effects.

I selected hypoxic hypoxia, which in its course and consequences may lead to effects dangerous to health and life as a result of changes developing in lung tissue, as an experimental animal model in order to obtain precise answers to the following questions:

1) What effects does chronic experimental hypoxic hypoxia produce in the cells comprising the pulmonary microcirculation of experimental animals?

2) What effect does water exposed to a magnetic field have on changes induced by hypoxic hypoxia in experimental animals?

3. Material and methods

a) material

Animal experiments were conducted after approval was obtained from the Local Ethics Committee for Animal Experiments in Katowice at the Silesian Medical Academy in Katowice (opinion no. 35/00).

Female Sprague-Dawley rats came from the Central Animal Facility of the Silesian Medical Academy in Katowice-Ligota, managed by Prof. Artur Stojko.

The study used 20 sexually mature animals weighing 250 g (+/-20), maintained under standard conditions: room temperature 20-22 C; lighting was changed on a 12-hour cycle, with a light phase from 7:00 to 19:00 and a dark phase from 20:00 to 7:00.

The animals were fed GLM mixed feed (GAN-RAT) and had unrestricted access to drinking water (both ordinary and magnetized water).

Hypoxic hypoxia was induced in a low-pressure chamber with a volume of 3 m3; the experimental parameters were set to the following values:

• pressure: 380 mm Hg
• oxygen content: 10%
• temperature: 22 C
• humidity: 65.7%

(corresponding to conditions at an altitude of 5,000 m above sea level).
The animals were divided into the following groups:

Control group Ia: 5 rats given water from the normal water-supply system were kept outside the low-pressure chamber.

Control group Ib:	5 rats given water previously exposed to a magnetic field were also kept outside the low-pressure chamber.
Experimental group IIa:	5 rats kept in the low-pressure chamber where hypoxic conditions were induced were given water from the normal water-supply system.
Experimental group IIb:	5 rats kept in the low-pressure chamber were given water previously exposed to a magnetic field.

Experimental-group IIa and IIb rats remained in the hypoxic chamber for 30 days. They were removed every day for 1 hour to replace the bedding and replenish food and water. Carbon dioxide was eliminated using chemical absorbers.

b) apparatus

Running water from the normal water-supply system, at a temperature below 30 C and a flow rate of at least 0.5 m/s, was magnetized in a specially designed device called RAM (tubular magnetic apparatus), owned by Feniks. The device is protected by patent no. 155856 (Fig. 1).

The above device for magnetizing liquids contains a magnetic stack within a tubular element, consisting of permanent magnets made of sintered anisotropic barium or strontium ferrite with an energy density of not less than 26 kJ/m3 and a magnetic-field strength of not less than 150 kA/m, as well as non-magnetized barium or strontium ferrite discs with remanent magnetization equal to one-half of saturation magnetization, amounting respectively to not less than 420 mT and 210 mT.

The apparatus used in the study contains a magnetic stack that produces dispersion of the magnetic flux, as a result of which the magnetic relaxation time (the time required for a water molecule to rotate around its own axis) is prolonged in water flowing through the RAM, consequently changing the viscosity, surface tension, and electrical conductivity parameters of the outflowing water.

The magnetic relaxation time for ordinary water, water after passage through the RAM, and demineralized water is 1100 microseconds, 1300 microseconds, and 1500 microseconds, respectively (results obtained at the Institute of Nuclear Physics in Krakow). The viscosity of distilled water and of water after passage through the RAM is 262.69 s and 243.84 s, respectively (measurements performed at the Rybnik Power Plant Laboratory using a Tomson TV-2000 AKV capillary viscometer).

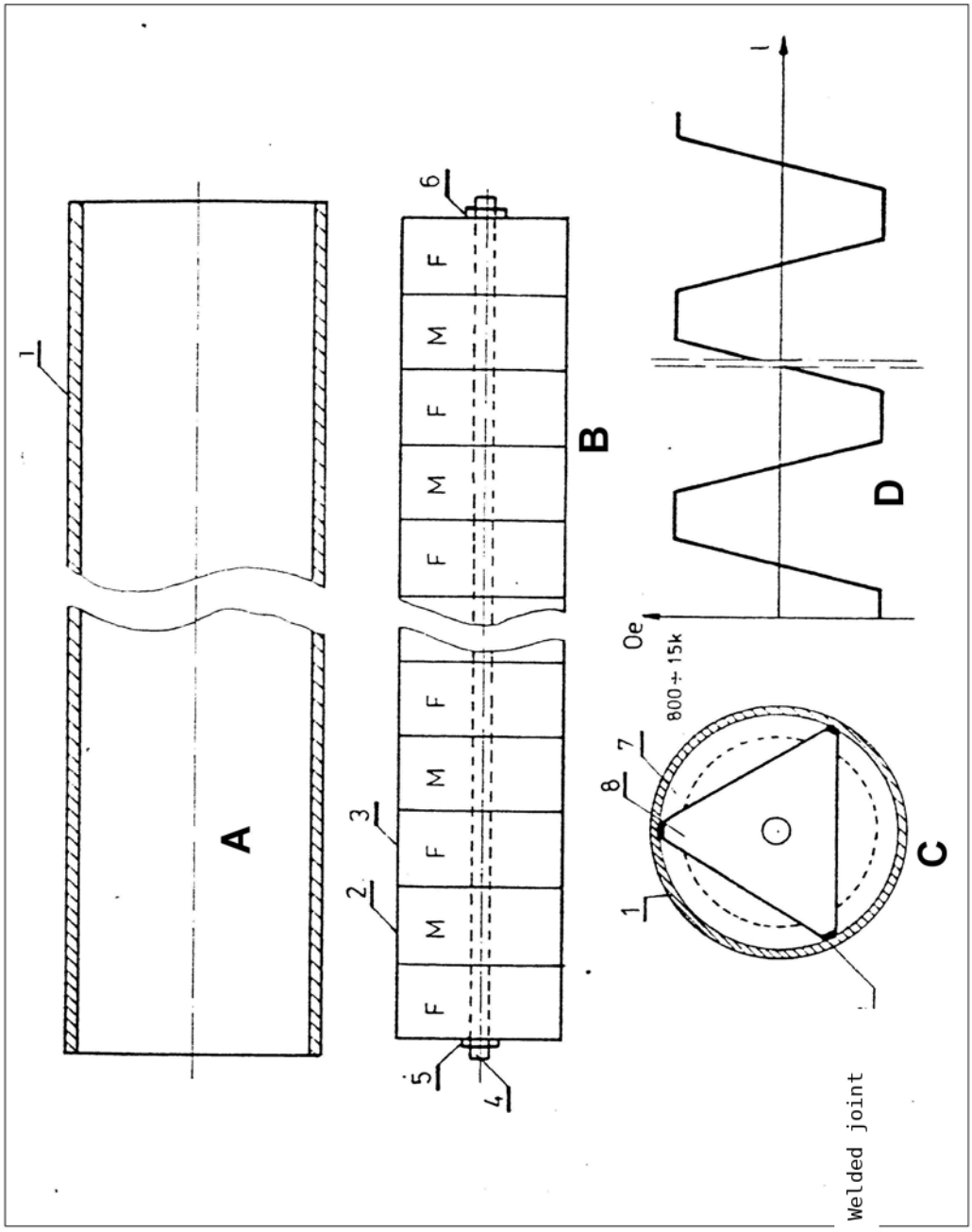
Diagram of the RAM device

A - section of steel pipe

B - magnetic stack consisting of ferrite discs and permanent magnets

C - steel fitting centering the magnetic stack and housing

D - magnetic-field characteristics



c) morphological examinations

In both control and experimental rats, tissue samples measuring 1 mm on each side were collected from the left upper lung lobe under general anesthesia (2.5% thiopental solution administered intraperitoneally at a dose of 40 mg/kg body weight). The tissue samples were immediately placed in cold fixative (2.5% glutaraldehyde buffered with cacodylate buffer at pH 7.2), where they remained for 2 hours at 4 C. They were then rinsed twice (12 hours each time) in cacodylate buffer. The samples were post-fixed for two hours at 4 C in a 1% solution of osmium tetroxide (OsO4) buffered with cacodylate buffer at pH 7.2.

Routine dehydration of the tissues through an ascending alcohol series was completed with propylene oxide, after which the tissue samples were embedded in a mixture of epoxy resins (Epon 812) and polymerized in gelatin capsules in an incubator for 36 hours (12 hours each at 36, 42, and 60 C).

The Epon blocks were cut with a Reichert ultramicrotome (Oum-3) into semithin sections 0.5-1 micrometers thick, which were mounted on microscope slides and stained with an aqueous toluidine-blue solution. These preparations were used for light-microscopic observation and for obtaining histomorphological documentation. They were also used to localize and determine the site for ultrathin sectioning.

Ultrathin sections 0.05 micrometers thick were placed on copper grids (300 mesh) and contrasted with uranyl acetate and lead citrate solutions (67). Ultrastructural observations of the tissue material and photographic documentation were performed using a JEM 100CX electron microscope manufactured by JEOL.

The examinations were carried out in the Electron Microscopy Laboratory of the Department of Histology and Embryology in Zabrze, Silesian Medical Academy in Katowice.

4. Results

Photographic documentation of morphological and ultrastructural examinations

Photographs of semithin sections of rat lung tissue stained with toluidine blue (Figs. 1-8).

Fig. 1. Section of lung tissue from a rat in group Ia.

Alveolar ducts and pulmonary alveoli are visible. Magnification 250x.

Fig. 2. Section of lung tissue from a rat in group Ia.

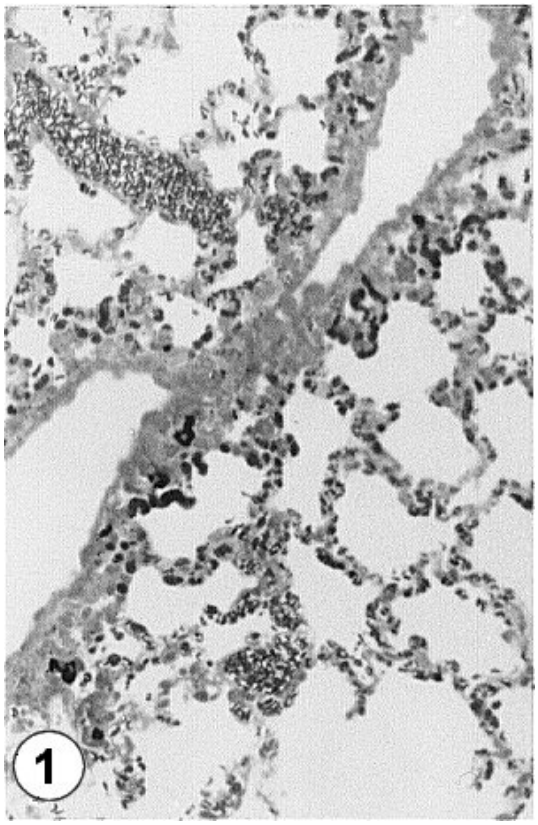
At higher magnification, adjacent pulmonary alveoli are visible. Magnification 1000x.

Fig. 3. Section of lung tissue from a rat in group Ib.

Alveolar sacs and pulmonary alveoli with slightly widened lumina are visible. Magnification 250x.

Fig. 4. Section of lung tissue from a rat in group Ib.

At higher magnification, pulmonary alveoli separated from one another by delicate interalveolar septa are visible. Magnification 1000x.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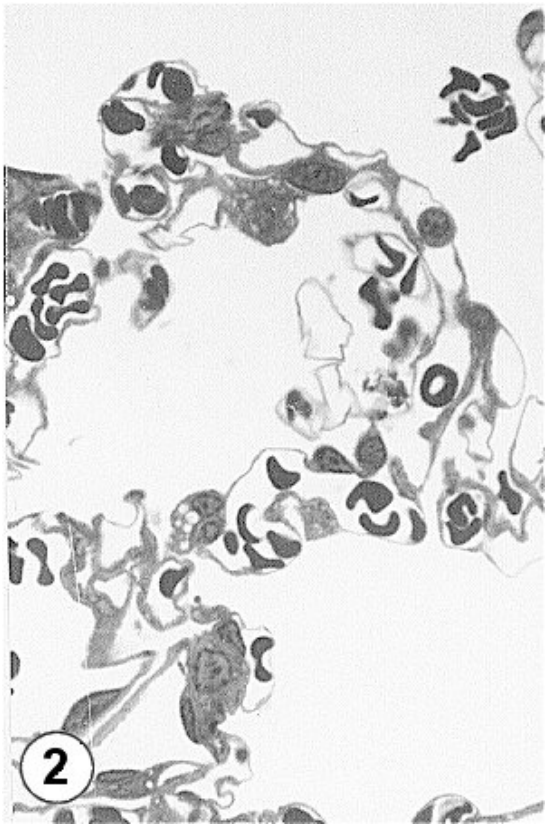
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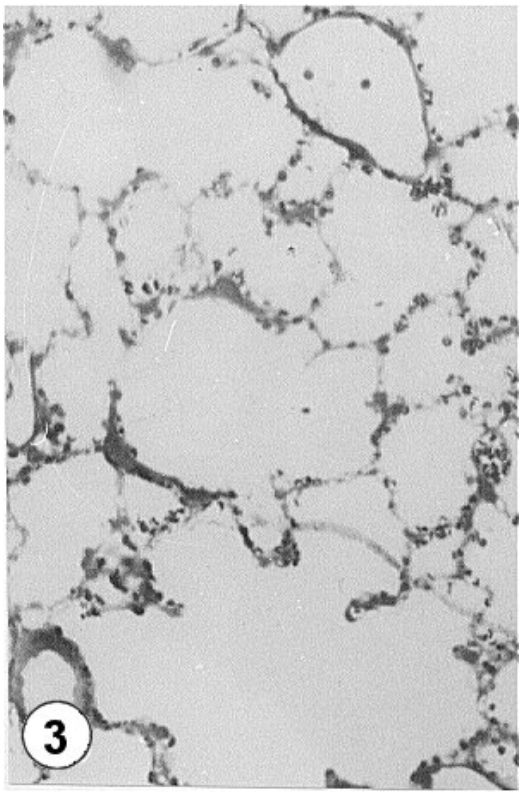
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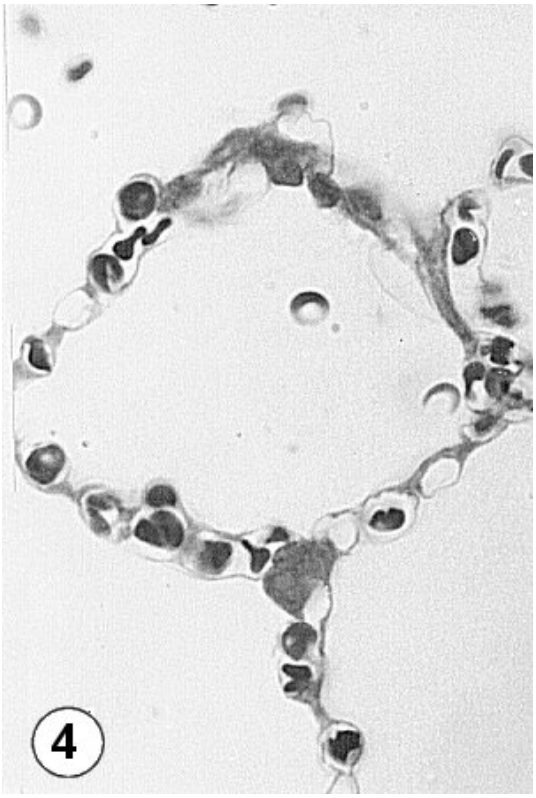
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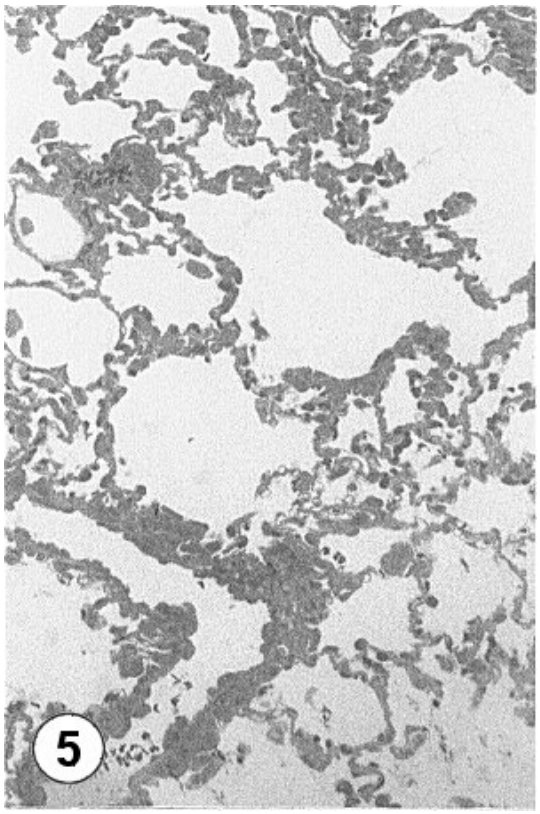
Fig. 5. Section of lung tissue from a rat in group IIa.

Alveolar ducts and pulmonary alveoli of various sizes are visible. Magnification 250x.

Fig. 6. Section of lung tissue from a rat in group IIa. Thickened basement membranes of the interalveolar septa are visible. Magnification 1000x.

Fig. 7. Section of lung tissue from a rat in group IIb. A respiratory bronchiole, alveolar sacs, and pulmonary alveoli are visible. Magnification 450x.

Fig. 8. Section of lung tissue from a rat in group IIb. Cross-sections through pulmonary alveoli together with interalveolar septa are visible; thickening of the septa is less pronounced. Magnification 1000x.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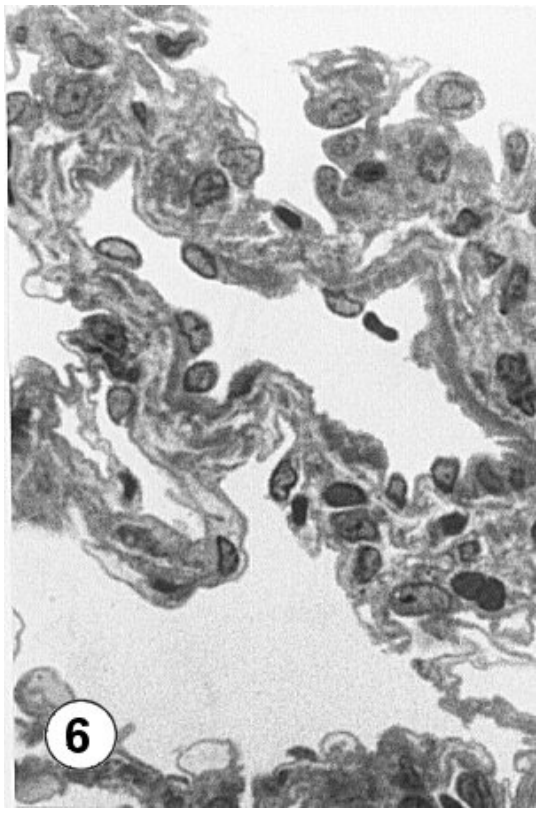
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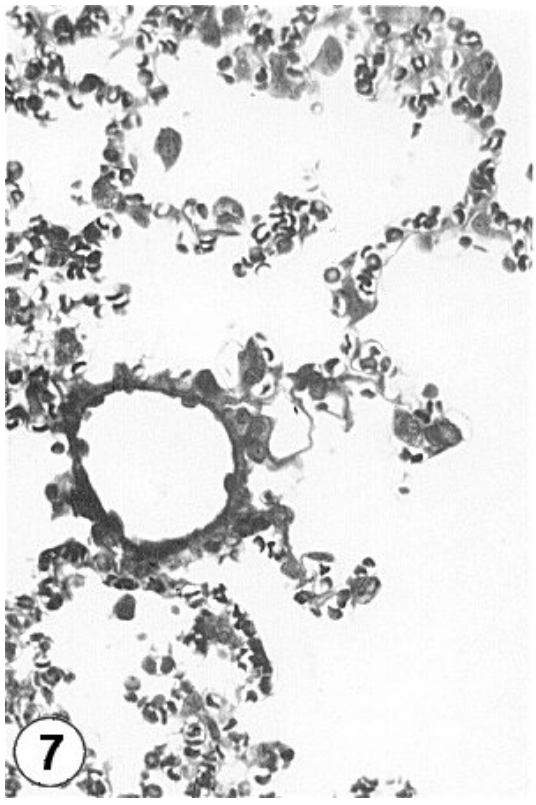
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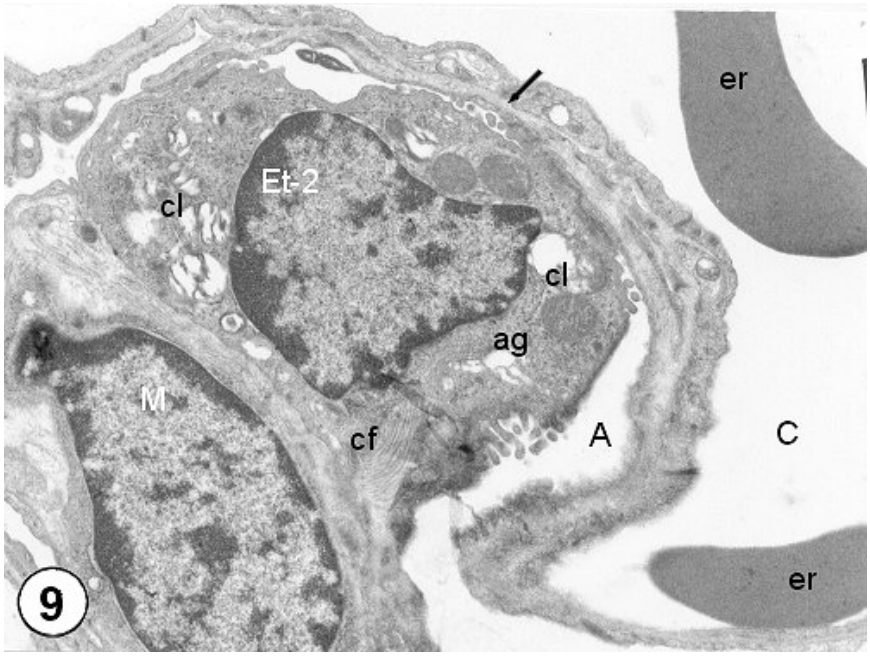
Electron micrographs of lung-tissue preparations from control group Ia animals given ordinary water (Figs. 9-12).

Fig. 9. Fragment of lung tissue with a type II pneumocyte (Et-2) in close proximity to a monocyte (M). On the right, erythrocytes (er) are visible in the lumen of a capillary (C). The type II pneumocyte is characterized by the presence of numerous lamellar bodies (cl). Microvilli are visible in the apical part of the cell facing the alveolar lumen (A). A well-developed Golgi apparatus (ag) is observed in the cytoplasm. The blood-air barrier (arrow) shows a regular arrangement of vascular endothelial cells, fused basal laminae, and the cytoplasmic plate of type I epithelial cells. Clusters of connective tissue (cf) are visible in the stroma of the interalveolar septum. Magnification 5000x.

Fig. 10. Fragment of a pulmonary alveolus. On the left side of the electron micrograph there is a portion of a type II pneumocyte (Et-2) with numerous lamellar bodies (cl). In the stroma of the alveolar septum, on the right, a fragment of a monocyte nucleus (M) and a cluster of connective-tissue fibers (wk) are visible. The arrow indicates the endothelium lining a capillary and resting on the basement membrane. Magnification 19000x.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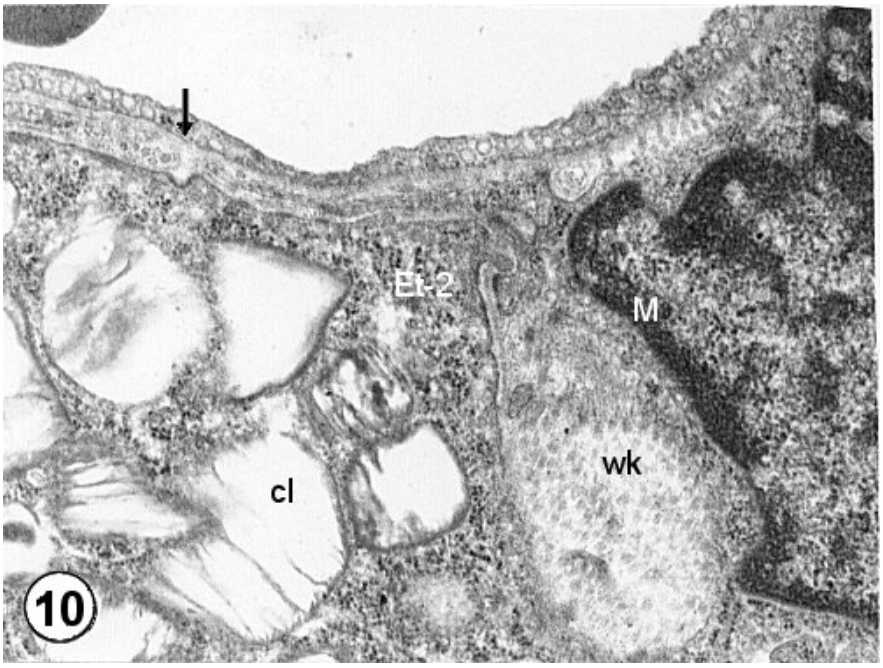
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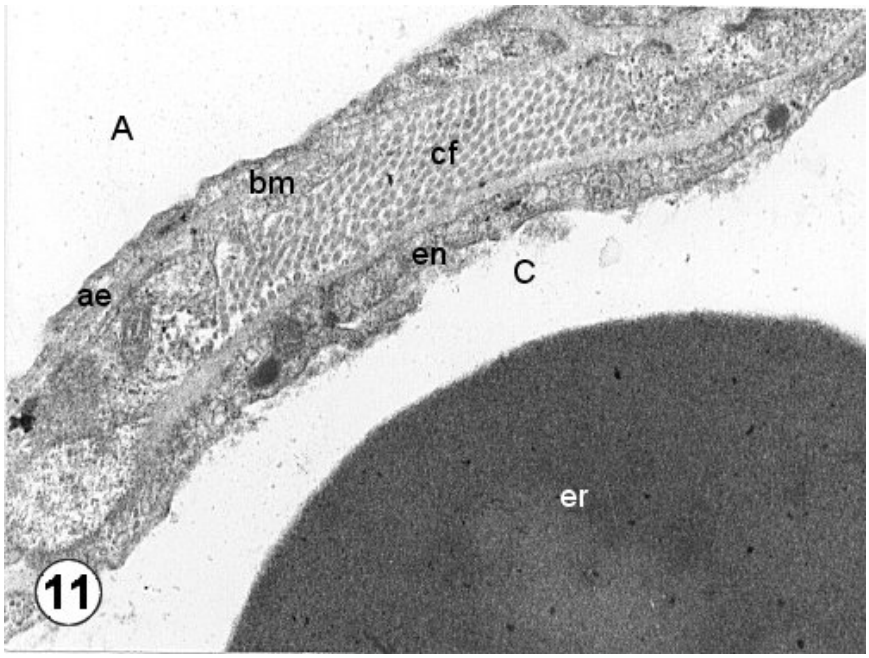
Fig. 11. Fragment of a flattened interalveolar septum; within its stroma cross-sections of connective-tissue fibers (cf) are visible. Endothelial cells (en) of the capillary (C) rest on the basement membrane. Pinocytotic vesicles are present in the cytoplasm of these cells. A fragment of an erythrocyte (er) is visible in the vascular lumen. The pulmonary alveolar lumen (A) is lined by a flat cytoplasmic plate (ae) of a type I pneumocyte covering a thin basement membrane. Magnification 16500x.

Fig. 12. Fragment of an endothelial cell with a cell nucleus (Nu), with chromatin arranged peripherally. On the left side of the image, the capillary lumen is lined by a narrow band of cytoplasm (en). In the right corner of the electron micrograph, the alveolar lumen is lined by a narrow cytoplasmic band (ae) resting on the basement membrane. Connective-tissue fibers (cf) are visible in the widened portion. Magnification 19000x.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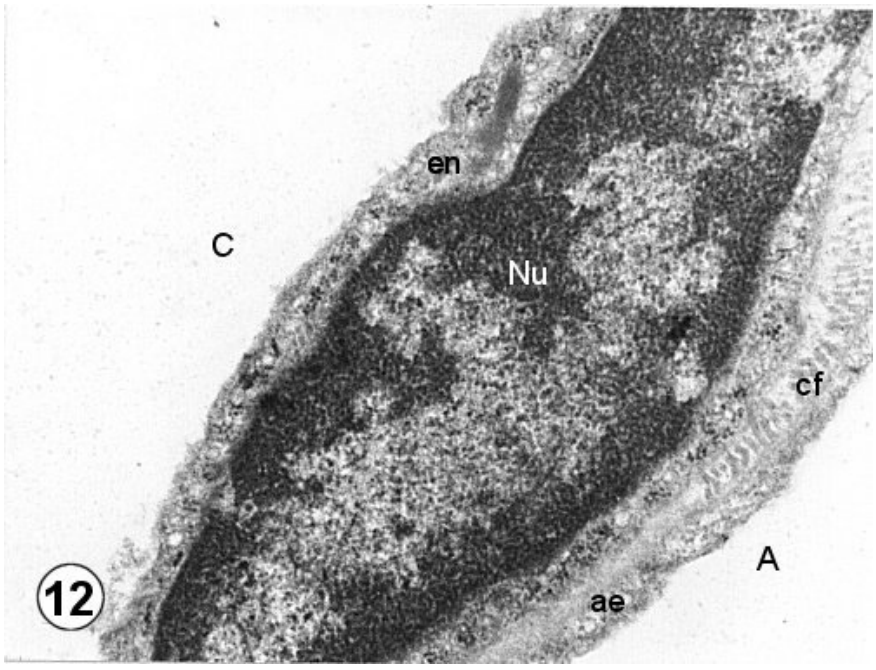
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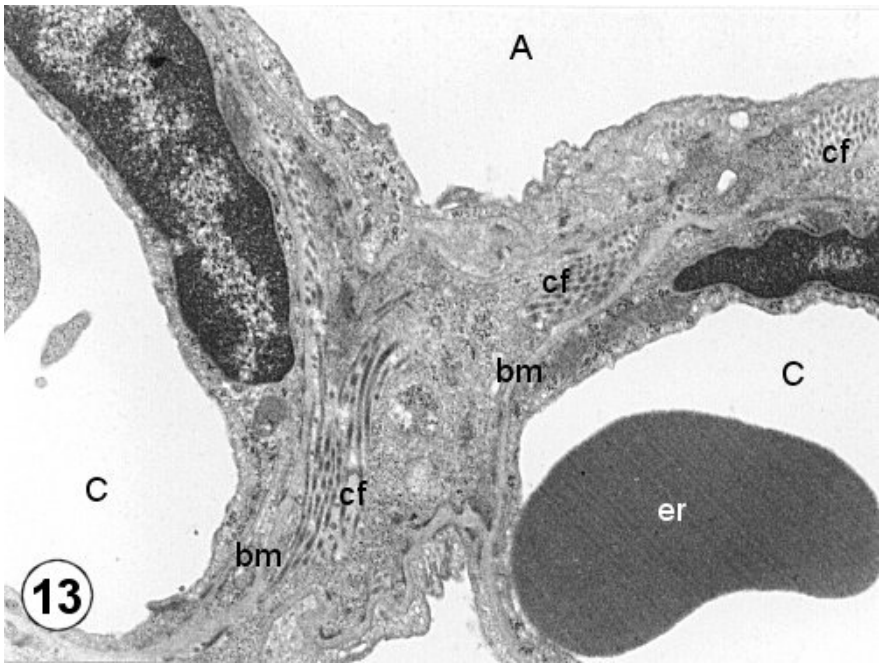
Electron micrographs of lung-tissue preparations from control group Ib animals given magnetized water (Figs. 13-16).

Fig. 13. Fragment of an interalveolar septum at the junction of two capillaries (C), with endothelial-cell nuclei. Numerous bundles of fibrous structures (cf) are present in the septal stroma. The basement membranes (bm) located between the endothelial cells and the epithelium lining the pulmonary alveolus (A) have a regular course. er - erythrocyte. Magnification 6600x.

Fig. 14. Large fragment of a type II pneumocyte with numerous lamellar bodies (cl) filled with lamellar structures. Mitochondria (m) show normal morphology. The ground cytoplasm contains both rough endoplasmic-reticulum tubules and free ribosomes. Numerous fibrous structures (cf) are present in the septal stroma. A fragment of a monocyte (M) is visible in the upper-right corner of the image. Magnification 6600x.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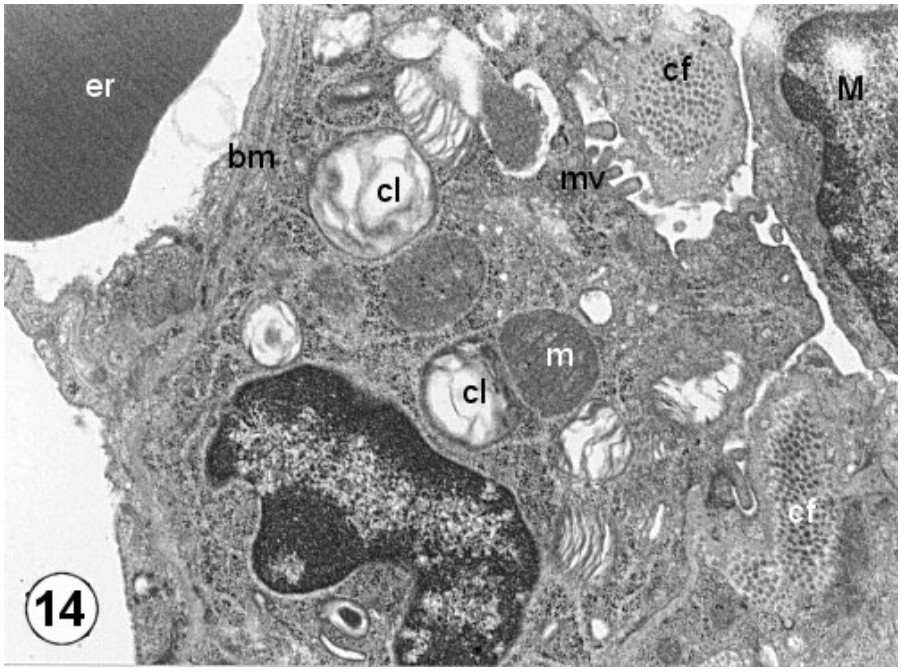
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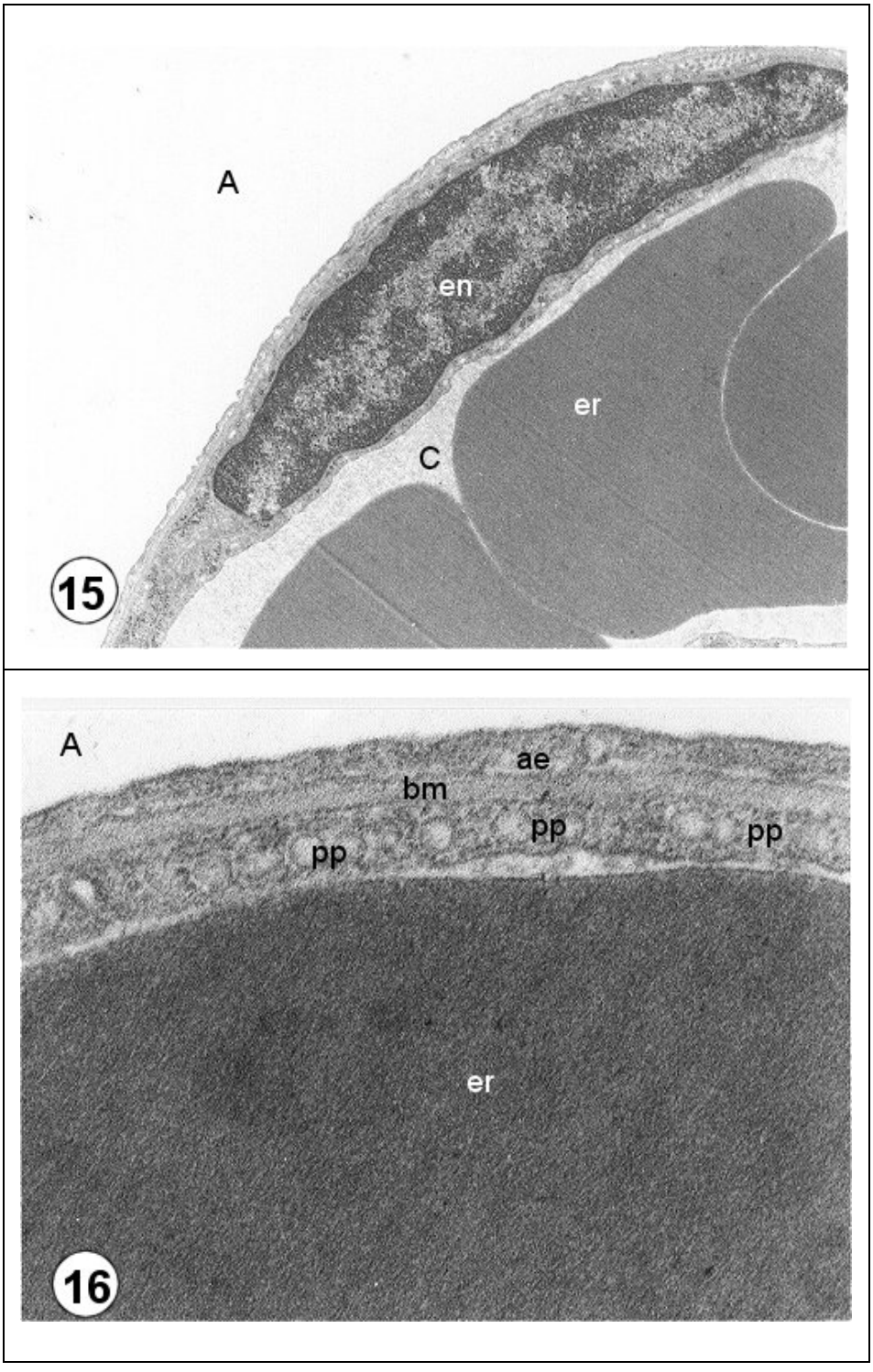
M
bm
cl
mv
m
cl
cf
14

Fig. 15. Fragment of a capillary (C) with a flattened nucleus of an endothelial cell (en). On the side facing the vascular lumen (C), the cell has a narrow band of cytoplasm. On the side facing the pulmonary alveolar lumen (A), there is an equally delicate cytoplasmic lining of a type I pneumocyte. Magnification 6600x.

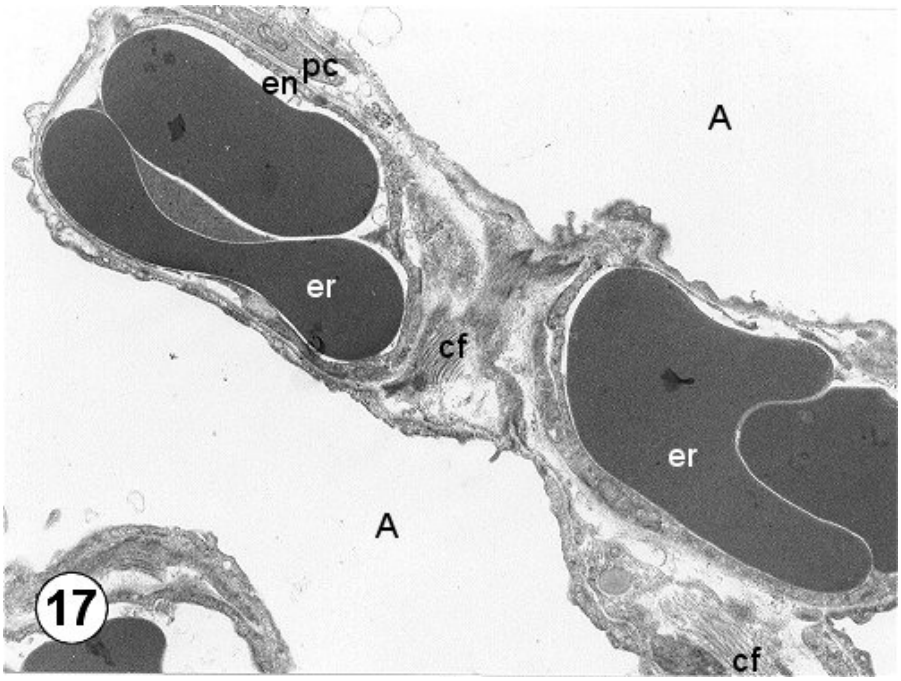
Fig. 16. Fragment of the blood-air barrier at high magnification. The cytoplasmic plate of the capillary endothelium contains numerous pinocytotic vesicles (pp). An erythrocyte (er) adjoins the capillary lumen. On the side facing the pulmonary alveolar lumen (A), a cytoplasmic plate of a type II pneumocyte (ae) is visible, adjacent to the common basement membrane (bm). Magnification 33000x.


A
en
er
C
15
A
ae
bm
pp
pp
pp
er
16

Electron micrographs of lung-tissue preparations from experimental group IIa animals exposed to hypoxia and given ordinary water (Figs. 17-22).

Fig. 17. Cross-section through an interalveolar septum with two capillaries filled with erythrocytes (er). Between the endothelia (en) and alveolar cells, a pericyte adjoining the capillary (pc) is visible. Fibrous structures (cf) are present in the stroma of the interalveolar septum. A - lumen of pulmonary alveoli. Magnification 5000x.

Fig. 18. Fragment of a type II pneumocyte with a cell nucleus (N) and a nucleolus located within the karyoplasm. The nuclear outlines are irregular. A well-developed Golgi apparatus (aG), a lamellar body (cl), and a mitochondrion (m) are visible in the ground cytoplasm. On the left side of the electron micrograph there is a fragment of the cytoplasmic wall of a capillary endothelial cell (C). Magnification 13000x.


pc
en
A
er
cf
er
A
17
cf

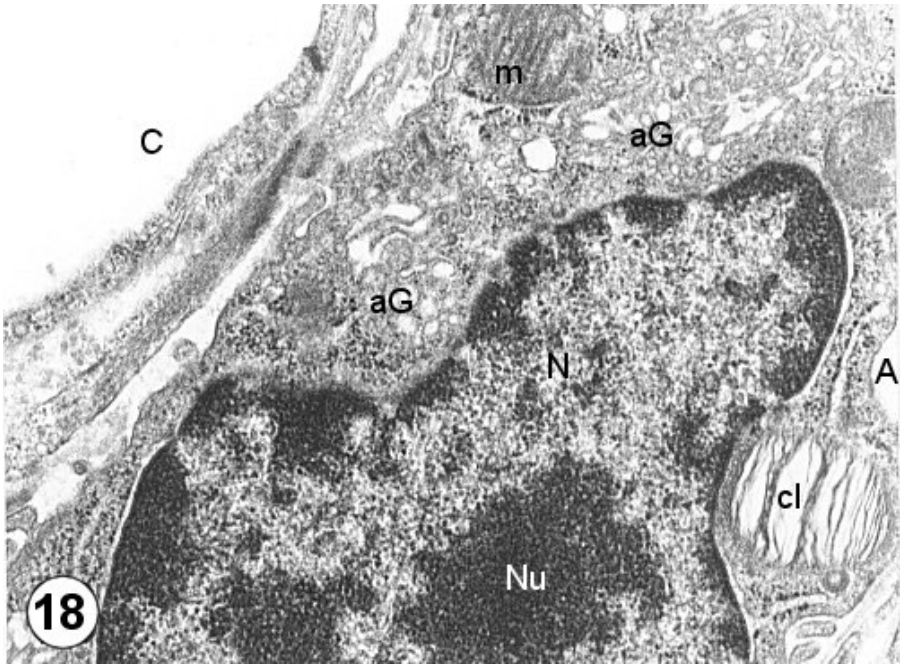
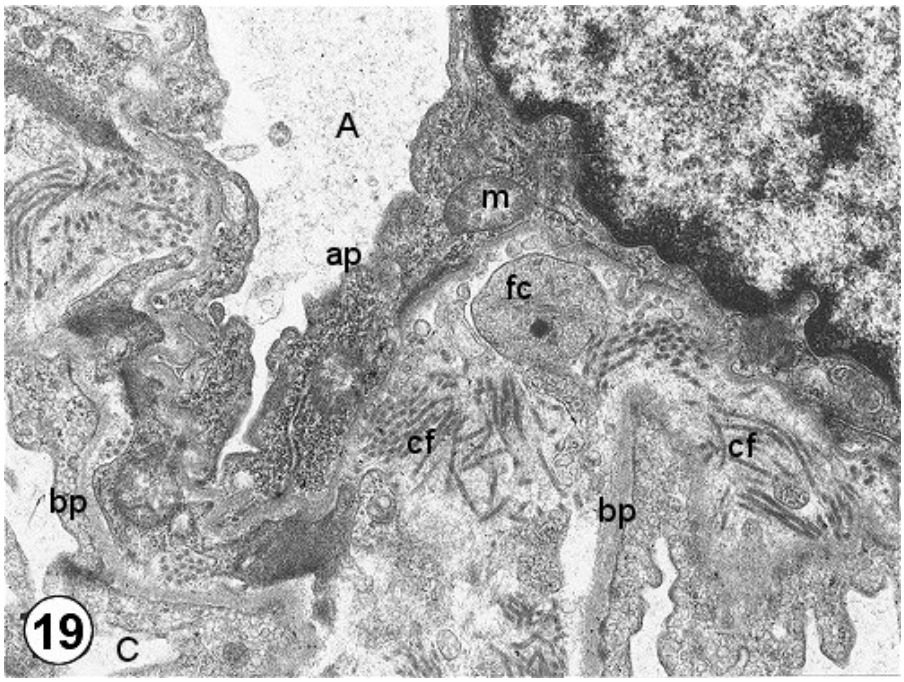

m
C
aG
aG
N
A
cl
Nu
18

Fig. 19. Fragment of an interalveolar septum. The connective-tissue stroma contains numerous irregularly arranged connective-tissue fibers (cf). The pulmonary alveolar lumen (A) is lined in places by a thickened cytoplasmic plate (ap), in which mitochondria (m) are visible. The basement membrane (bp) follows an irregular course and is thickened in places. In the upper-right corner, a fragment of the nucleus of an epithelial cell lining the pulmonary alveoli and a fibrocyte process (fc) are visible. Magnification 13000x.

Fig. 20. Capillary (C) with markedly widened cytoplasm of an endothelial cell (en). Surfactant granules (sb) are visible in the pulmonary alveolar lumen (A). The basement membrane (bp), with an irregular course, is covered by epithelium bulging into the alveolar lumen. tc - thrombocyte. Magnification 6600x.


A
m
ap
fc
cf
cf
bp
bp
19
C

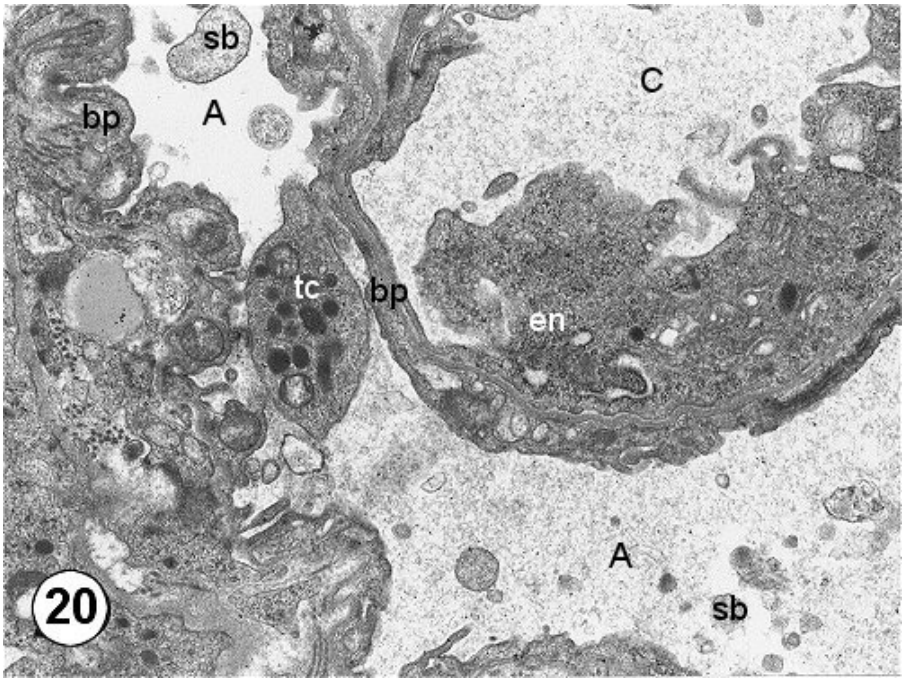
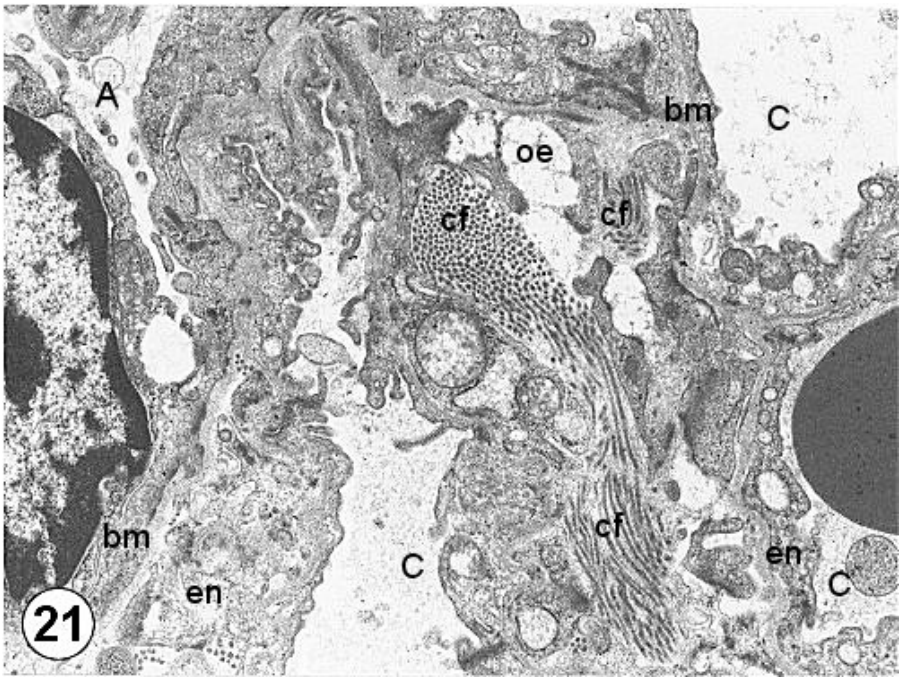
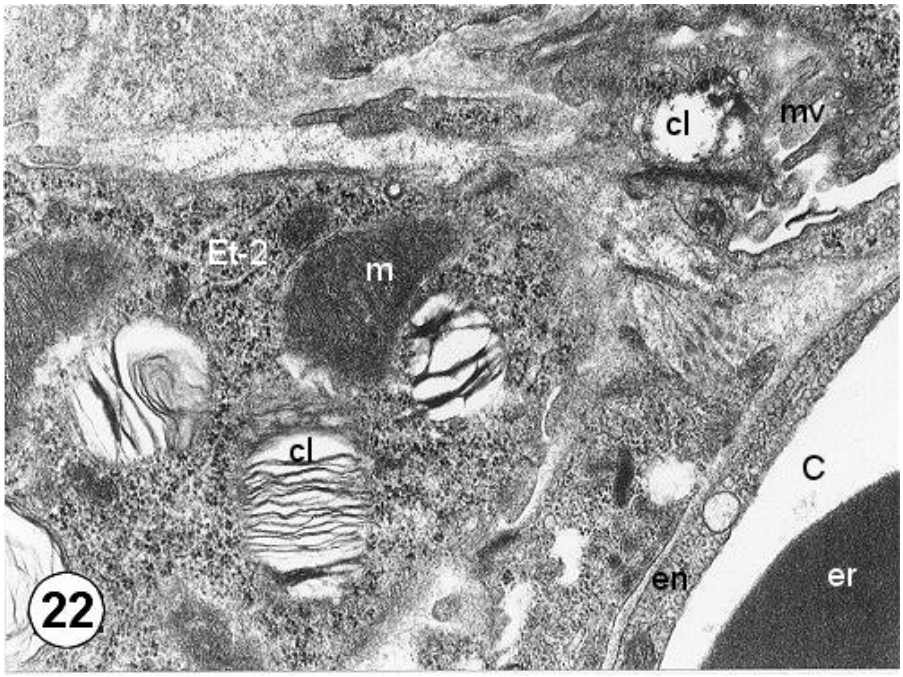

sb
C
bp
A
tc
bp
en
A
sb
20

Fig. 21. Stroma of an interalveolar septum with numerous connective-tissue fibers (cf). Spaces indicating interstitial edema (oe) are visible. The alveolar lumina (A) are narrowed. Both the capillary endothelium (en) and the epithelium lining the pulmonary alveoli are markedly thickened. C - capillary lumen. Magnification 6000x.

Fig. 22. Fragment of a type II alveolar cell (Et-2) with lamellar bodies (cl) filled with numerous lamellar structures. Mitochondria (m) with an electron-dense matrix are visible in the ground cytoplasm. In the upper-right part of the electron micrograph, evacuation of a lamellar body toward the microprojections (mv) is observed. C - capillary lumen, er - erythrocyte, en - endothelial cytoplasm. Magnification 13000x.

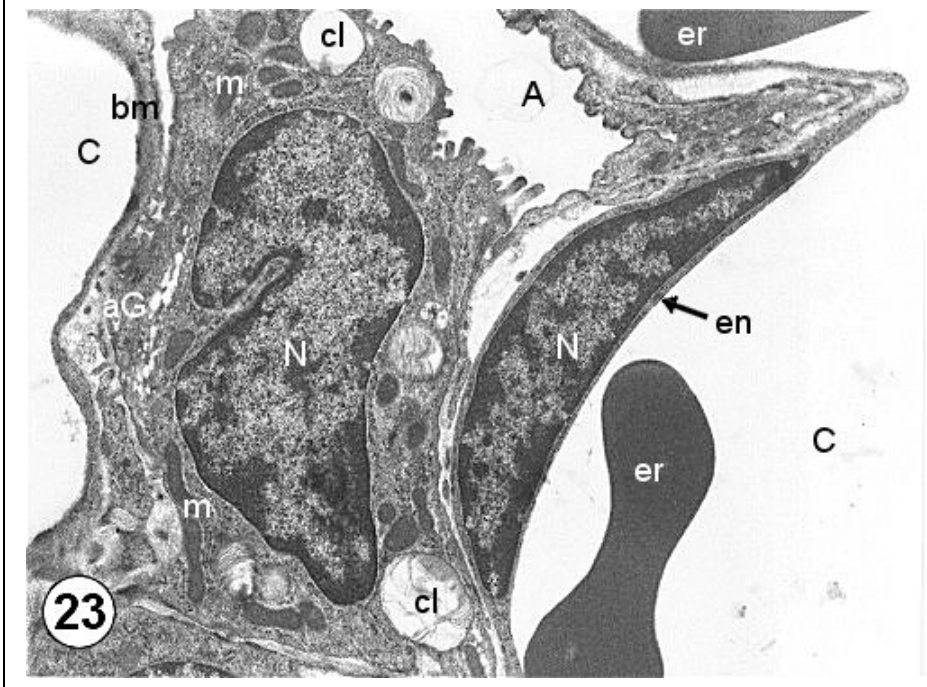

A
bm
C
oe
cf
cf
bm
C
cf
en
C
en
21


cl
mv
Et-2
m
cl
C
en
er
22

Electron micrographs of lung-tissue preparations from experimental group IIb animals exposed to hypoxia and given magnetized water (Figs. 23-28).

Fig. 23. Type II pneumocyte in a niche between two capillaries (C). On the right, an arrow indicates the nucleus (N) of a cell lining the capillary, whose lumen contains erythrocytes (er). The apical surface of the pneumocyte (Et-2) is covered with microprojections. The ground cytoplasm contains a small number of lamellar bodies (cl), a well-developed Golgi apparatus (aG), and numerous mitochondria (m) of various shapes. The vascular endothelial lining is flattened and regular in course. Magnification 5000x.

Fig. 24. A fragment of a type II pneumocyte similar to that in the previous image, with a large number of lamellar bodies. In the lower-right part of the image there is a section through the nucleus (N) of a capillary endothelial cell (en). er - erythrocyte, C - capillary lumen, A - pulmonary alveolar lumen. Magnification 5000x.


cl
er
m
A
bm
C
aG
N
N
en
C
er
m
cl
23

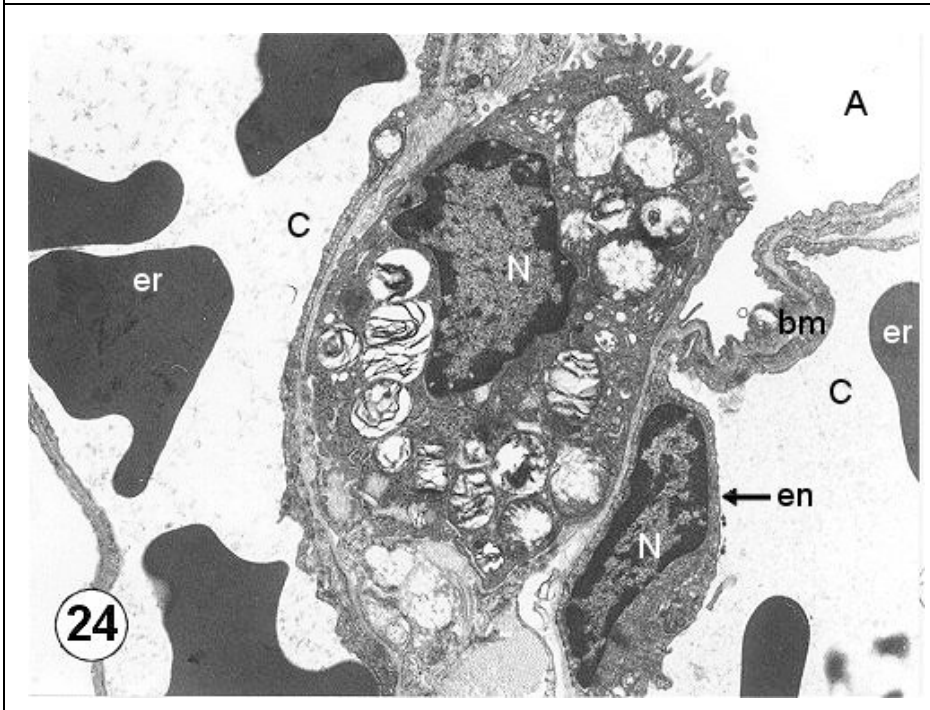
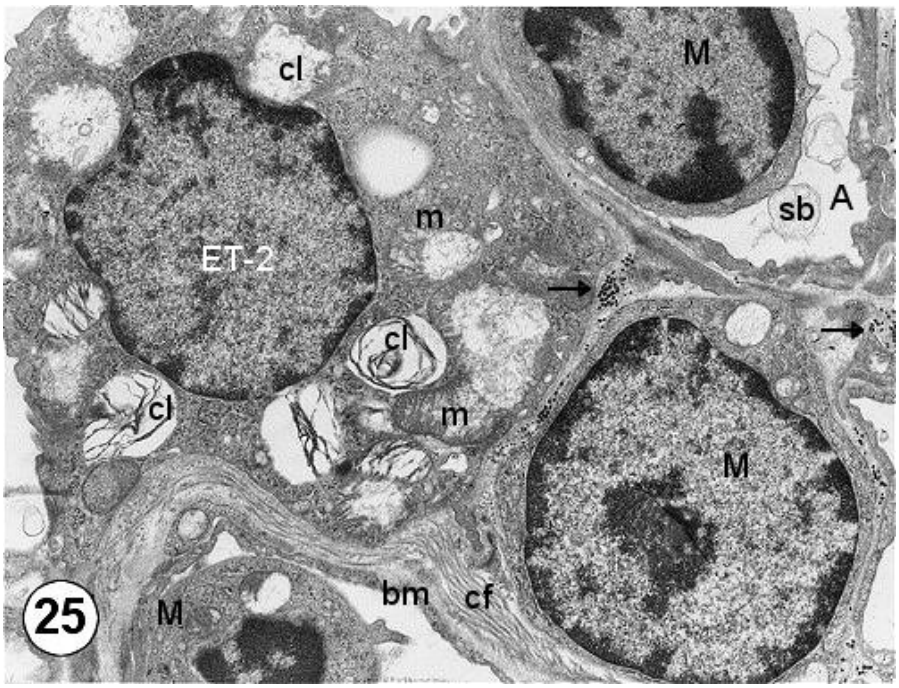

A
C
er
N
bm
er
C
en
N
24

Fig. 25. Stroma of an interalveolar septum with a large fragment of a type II pneumocyte. Lamellar bodies (cl) are visible in the ground cytoplasm; some of them lack lamellar structures. Monocytes (M) are visible nearby. Electron-dense granules (arrows), which may represent granules from degranulated mast cells, are visible in the intercellular space. Surfactant granules (sb) are visible in the pulmonary alveolar lumen (A) near the monocyte. Magnification 5000x.

Fig. 26. Cross-section through the wall of a capillary. In the center, the nucleus of an endothelial cell (en) adjacent to the basement membrane (bm) is visible. On the side facing the pulmonary alveolar lumen (A), a thicker cytoplasmic plate of a type I pneumocyte lining the alveolus can be seen. On the right side of the electron micrograph, the apical surface of a type II pneumocyte with microvilli (mv) is visible. tj - intercellular junction. Magnification 13000x.


cl
M
sb
A
m
ET-2
cl
cl
m
M
25
M
bm
cf

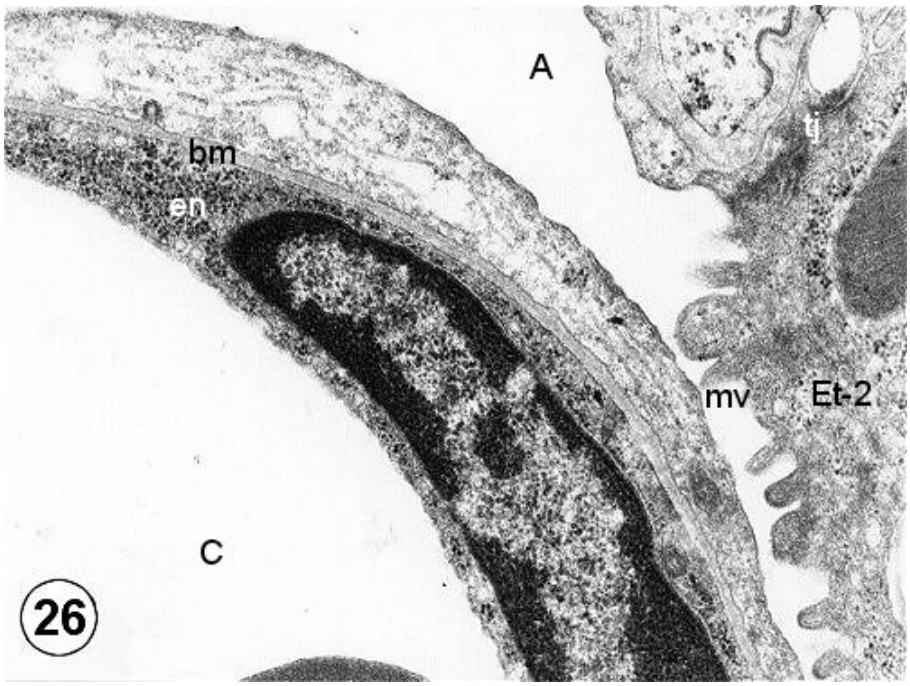
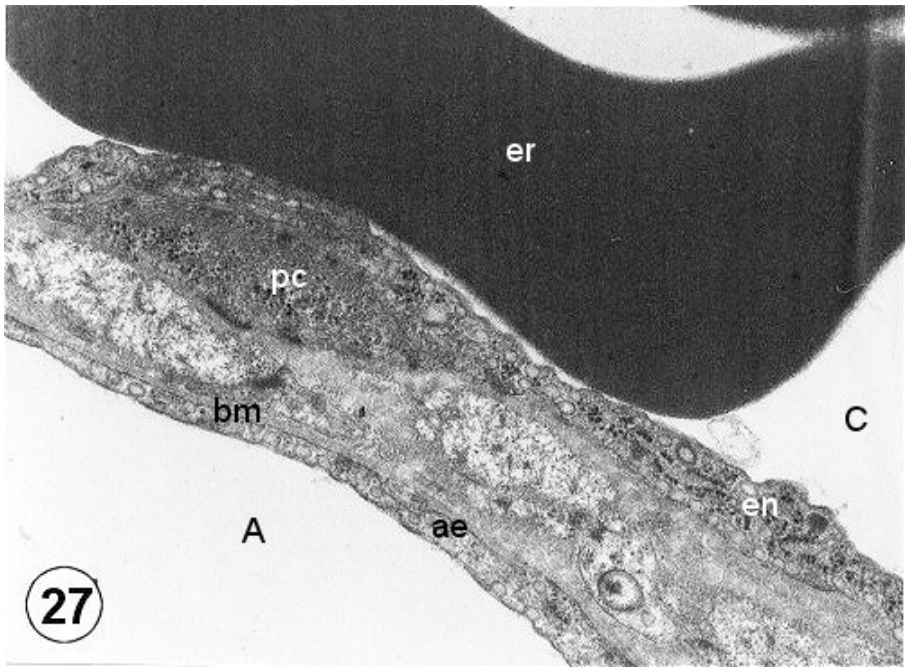
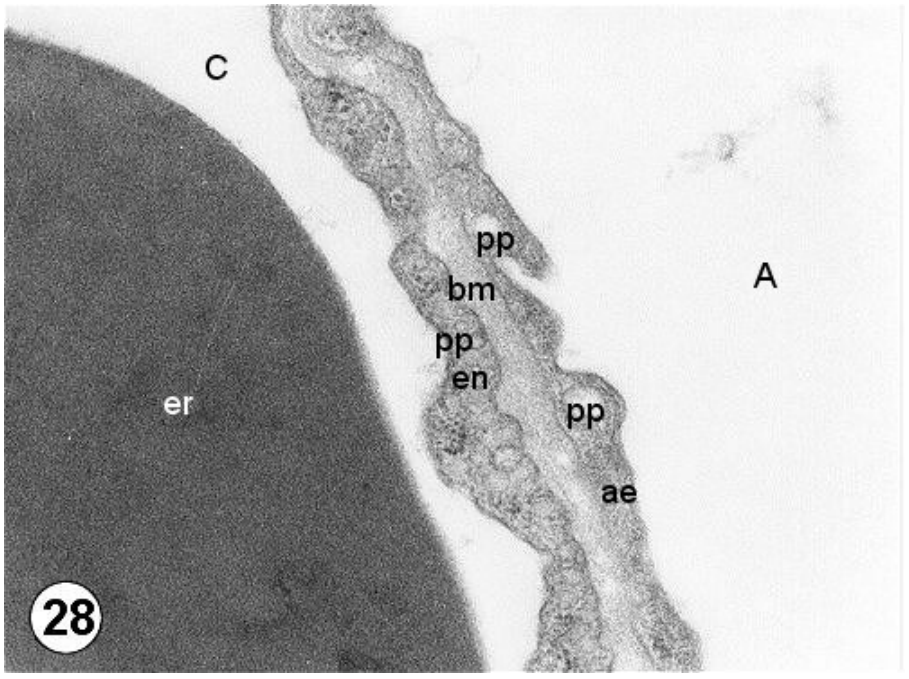

A
tj
bm
en
mv
Et-2
C
26

Fig. 27. Cross-section through the blood-air barrier separating the lumen of a capillary (C) from the pulmonary alveolar lumen (A). On the alveolar side, the cytoplasmic plate of a type I pneumocyte (ae), resting on the basement membrane (bm), is visible. On the vascular-lumen side, the endothelial plate with a small number of pinocytotic vesicles also rests on a basement membrane that encloses a fragment of a pericyte (pc). Fibrous structures are visible between the plates. Magnification 13000x.

Fig. 28. Cross-section showing the blood-air barrier between a capillary (C) and the pulmonary alveolar lumen (A). The components of this barrier, i.e. the alveolar epithelium (ae) and vascular endothelium (en), are separated by a common basement membrane (bm). Numerous pinocytotic vesicles (pp) are visible in the cytoplasmic plates of both cell types. Magnification 20000x.


er
pc
bm
C
en
A
ae
27


C
pp
A
bm
pp
en
er
pp
ae
28

5. Summary of morphological and ultrastructural findings

No significant structural differences are seen in semithin lung-tissue preparations from control groups Ia and Ib given ordinary and magnetized water (Figs. 1-4).

Analysis of semithin lung-tissue preparations from animals in groups IIa and IIb exposed to hypoxic conditions shows a widened stroma of the interalveolar septa and signs of cellular proliferation in the rats that were given water from the normal water-supply system (Figs. 5-8).

Comparison of electron micrographs prepared from lung-tissue specimens of animals in groups Ia and Ib reveals structural differences in the blood-air barrier. In animals given ordinary water, numerous fibrous connective-tissue structures are accumulated in the stroma of this barrier. This is particularly evident between the basement membranes of the capillary endothelium and the epithelium lining the pulmonary alveoli (Fig. 11).

In animals given magnetized water, the layering of these cellular elements is normal (Fig. 16).

The amount of connective-tissue fibers and their location in the stroma of the interalveolar septa are similar in both groups.

Electron micrographs (Figs. 17-22) showing the stroma of the interalveolar septa in the lungs of animals exposed to hypoxic conditions and given water from the normal water-supply system demonstrate "structural disorder." This is manifested by swelling of the cells lining the capillary lumina and of the cytoplasmic processes of alveolar cells - type I pneumocytes. An increase in the amount of collagen fibers is also observed, while electron-lucent intercellular spaces may indicate the presence of edema fluid (Fig. 21).

Comparative evaluation of electron micrographs showing the structure of the pulmonary microcirculation in animals exposed to hypoxia and given magnetized water (group IIb - Figs. 23-28) reveals significant differences compared with group IIa.

In different areas of lung tissue, type II pneumocytes show varying submicroscopic structure of the cellular elements analyzed. Type II cells with numerous lamellar bodies occur simultaneously with cells in which the number of lamellar bodies is minimized in favor of a well-developed Golgi apparatus, together with evacuation of the contents of lamellar bodies into the extracellular space (Figs. 23-24). Monocytes are also observed in the stroma of the interalveolar septa, both those that have penetrated into the capillary lumen and those located within the connective-tissue area of the interalveolar septa (Fig. 25). Also noteworthy is the presence in the intercellular spaces of electron-dense granules corresponding to mast-cell granules after degranulation (Fig. 25).

The essential difference within the blood-air barrier itself lies in the normal morphology of the cytoplasmic linings of both the capillary endothelium and the pulmonary alveoli, as well as in the orderly arrangement of the submicroscopic structures of the interalveolar-septal stroma in the group of animals that were given magnetized water under hypoxic conditions (Figs. 23-28).

6. Discussion of results

In recent years, many leading research centers have intensified their interest in the use of therapeutic methods based on magnetostimulation and magnetotherapy in clinical practice, where physical medicine is increasingly becoming a beneficial alternative to pharmacological therapy.

Numerous studies demonstrate beneficial effects of magnetic fields in a range of conditions, particularly because of their anti-inflammatory and analgesic properties, which are probably associated with the vasodilatory effect of these fields. Many favorable results after the use of magnetic fields have also been reported in regenerative processes and soft-tissue repair, acceleration of bone union, and intensification of tissue-respiration processes.

Publications from recent years indicate that interference by magnetic fields with enzymatic and molecular processes occurring in cells cannot be excluded, especially within the liquid-crystalline structures of cell membranes with their abundance of receptors. This also applies to the system of intracellular membranes surrounding cellular organelles and to the membranes forming the cellular cytoskeleton. The latter is responsible for compartmentalization, which substantially facilitates the proper course of biochemical reactions in defined regions of the cytoplasm.

To date, relatively few publications have addressed the possible effect of a magnetic field on tissue water, which is the principal component of the body and, by serving as a cellular filler, is involved in all biochemical processes occurring throughout the organism.

At a time when magnetizers are being used increasingly widely to treat drinking water in households as well as in industrial

flow-through systems intended to reduce scale deposition, there is a need for a thorough analysis of the effects of using these devices. A negative assessment of the effects of magnetizers would have to lead to their removal from the market, whereas a positive assessment of their effects should contribute to their promotion for both economic and health reasons.

It appears important to answer the question of how water exposed to a magnetic field affects the course of a disease process induced in experimental animals. It may be assumed that, when used for a prolonged period as the only source of fluid, it effectively replaces the body's entire water pool while simultaneously imparting its altered physicochemical properties.

In my experiment, the animals remained for 30 days in a hypoxic chamber in which conditions analogous to those prevailing at an altitude of 5,000 m above sea level were created. An equal number of animals remained under normal conditions outside the low-pressure chamber. Both control and experimental animals were given water from the normal water-supply system and the same water previously exposed to a magnetic field.

The preparations obtained from control-group animals and illustrated by electron micrographs showed no significant differences between animals given ordinary water and those given magnetized water, apart from a greater accumulation of fibrous structures in the intercellular septa of rats given ordinary water.

Electron micrographs prepared from lung-tissue specimens of animals given magnetized water indicate that water prepared in this way protects the terminal portions of the respiratory tract against hypoxic stress, and the changes

induced by hypoxia are substantially less pronounced than when ordinary water is used. It may therefore be hypothesized that water exposed to a magnetic field has a beneficial effect on the microcirculation of lung tissue. However, there remains a need to seek explanations for the mechanisms of this phenomenon. Answers should be sought in further studies expanded to include interdisciplinary methods, for example immunobiological and molecular investigations (13, 18, 33, 59, 61, 62, 83).

7. Conclusions

9. Chronic experimental hypoxic hypoxia leads in adult rats to remodeling of lung tissue as a result of damage to cells forming the interalveolar structures. This mainly involves capillary endothelial cells, the epithelium lining the pulmonary alveoli, and connective-tissue structures.

10. Giving "magnetized water" to rats subjected to experimental conditions, if it does not prevent the changes, substantially minimizes those induced by hypoxia.

11. It may be assumed that "magnetized water" protects the cellular structures of the pulmonary microcirculation against the adverse effects of molecular reactions occurring in the region of cell membranes and the cytoskeleton as effectors of magnetic-field action.

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FEBS Lett. 296; 117-122, 1992

9. Summary

The results of numerous studies in recent years indicate a beneficial effect of magnetic fields with specified parameters on the human body. However, the mechanisms of this action are still not fully understood. One possible pathway is modification of the structure of water, which constitutes not only the medium but also a substrate for most chemical reactions occurring in living organisms.

Exposure to chronic hypoxia is an important factor leading to remodeling of the pulmonary circulation and, consequently, to the development of pulmonary hypertension. The content of laminin and fibronectin increases in the endothelial basal lamina as well as in the media of the pulmonary arteries. The most important changes concern the distribution of myocytes between two elastic laminae. Myocytes are also present in the intima of small arteries and arterioles. Muscularization of small pulmonary vessels that normally lack myocytes is a persistent pathological feature of chronic hypoxia. Changes in the vessels of the pulmonary microcirculation are associated with factors such as HIF-1, VEGF, and NO.

In the present experiment, the effect of magnetized water was studied in rats exposed to hypoxia. Twenty adult Sprague-Dawley rats were divided into four groups: Ia (control group) - 5 animals kept under normoxic conditions and given unmodified water; Ib (control group) - 5 animals under normoxic conditions given magnetized water; IIa (experimental group) - 5 animals kept under hypoxic conditions (10% O2) and given unmodified water; IIb (experimental group) - 5 animals under hypoxic conditions (10% O2) given magnetized water. After 30 days, lung-tissue samples were collected for examination by electron microscopy. The results demonstrate changes in lung tissue induced by hypoxia. The endothelial cells of the vessels and the epithelium of the

pulmonary alveoli are swollen. An increase in the amount of collagen fibers is also observed. The beneficial effect of magnetized water is reflected in normalization of the structure of endothelial cells and the alveolar epithelium, a reduction in the amount of collagen fibers, and migration of monocytes into the connective-tissue regions of the interalveolar septa.

Because hypoxia plays a significant role in the development of pulmonary hypertension, the results of the above experiment may be helpful in the search for new methods of treating a number of pulmonary disorders.