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EFFECT OF LOW-FREQUENCY ULTRASOUND ON THE PLEURA AND ADJACENT LUNG TISSUE

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KEY WORDS: pleura, low-frequency ultrasound, morphology, exposure.

The use of low-frequency ultrasound (US) in clinical practice has expanded widely in the last decade. To treat open suppurative lesions US is used with an energy that has the property of actively moving fluid and creating cavitation, and so on [3, 6]. The bactericidal action of low-frequency US has been confirmed by clinical and experimental research [5, 7]. Under the influence of ultrasound waves, intracellular metabolism is accelerated and energy processes stimulated [3, 7]. Low-frequency US is used to treat biological tissues, during pleurectomy, and also to prevent suppurative complications and for the treatment of empyema [1, 4, 9].

For the reasons given above there is good reason to use the energy of low-frequency US for the treatment of chronic dieases of the pleura. However, the possibility that ultrasonic radiation may have a harmful action on the serous and mucous membranes must be taken into account. It has been shown that if low-frequency US is passed through solutions in the abdominal cavity of rabbits, foci of necrosis and hemorrhage are found in the zone of exposure, especially if the wave guide is in contact with the abdominal wall [8]. Low-frequency US also acts on the bronchopulmonary system when applied by the endobronchial route [2]. It has been shown experimentally that a single or multiple exposure to ultrasound for 30 sec causes virtually no pathological changes in the bronchopulmonary tissue, but lengthening the exposure is accompanied by destruction and desquamation of the bronchial epithelium, by necrosis of the cells of the mucous membrane, and by the development of an inflammatory reaction in the zone of exposure.

There is no information in the literature on the effect of low-frequency US on tissues or on its use in closed cavities such as the pleural cavity.

The aim of this investigation was to study morphological changes in the pleura and adjacent lung parenchyma and also to determine optical therapeutic schedules for experimental exposure to low-frequency US.

EXPERIMENTAL METHOD

Experiments were carried out on 58 male rabbits aged 4-5 months and weighing from 2.5 to 3 kg. The animals were divided into two groups: group 1) control, consisting of eight intact animals; group 2) 50 animals, in which the harmful action of US on the pleura and lung tissue was investigated. The control and experimental animals were kept under identical conditions.

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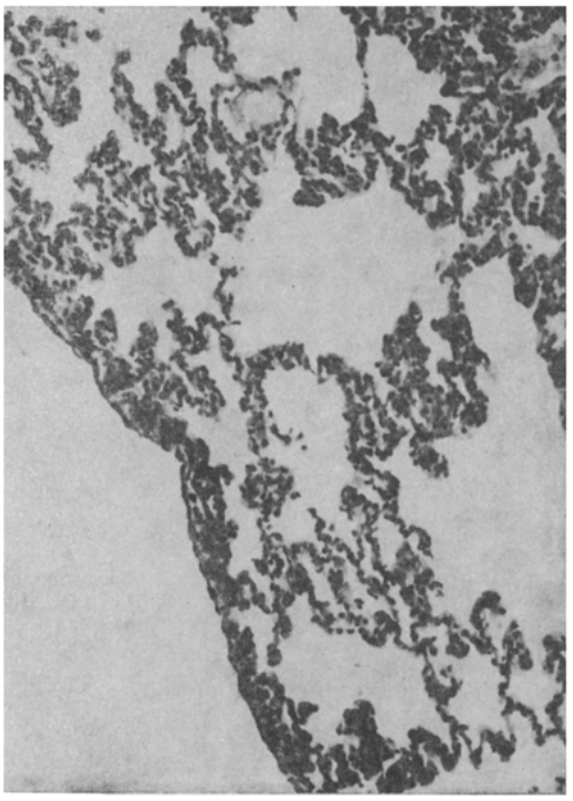

Fig. 1

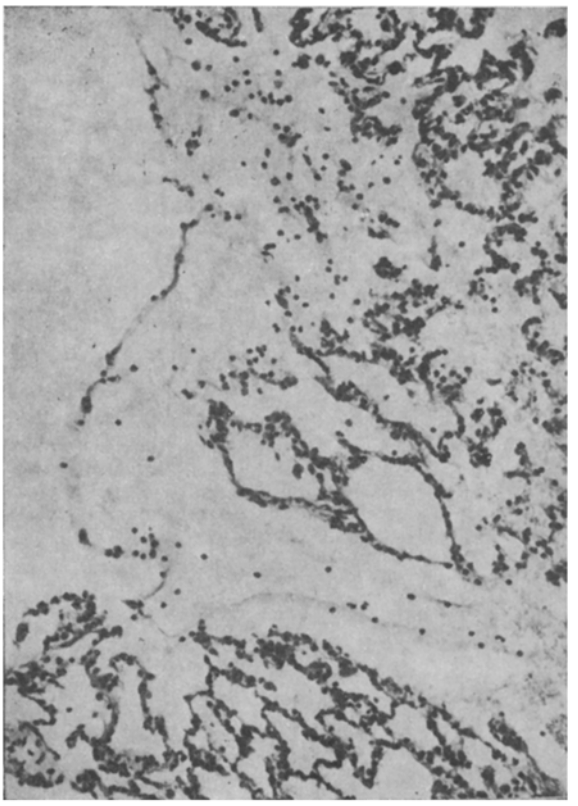

Fig. 2

Fig. 1. Normal structure of pleura and adjacent lung tissue. Control group. Hematoxylin-eosin.

Fig. 2. Edema and separation of fibers of pleura. More marked edema in "delta" region. Hematoxylin-eosin.

Low-frequency ultrasound waves were generated at a frequency of 26.5 kHz, with an amplitude of displacement of the cylindrical instrument (wave guide) of 40 μ, and a diameter of 4 mm. After premedication (atropine 0.1-0.2%, trimeperidine 1-0.5%), under intravenous anesthesia with 2% thiopental sodium solution (25 mg/kg), together with local anesthesia (0.25% procaine solution — 20-30 ml), thoracotomy was performed in the fourth intercostal space. After the chest had been opened the pleural cavity was filled with physiological saline. A zone of the lung with an area of 1 cm^2 in the upper lobe, with a distance of 5-10 mm from the end of the wave guide, was irradiated. The endothoracic exposure was carried out only once.

There were five series of experiments with different durations of exposure to low-frequency US: I) 5 sec/cm^2, II) 10 sec/cm^2, III) 20 sec/cm^2, IV) 30 sec/cm^2, and V) exposure for 5 sec by the contact method; 10 animals were used in each series. Participation of the rabbits in the experiments was terminated by injection of air into the marginal vein of the ear. Zones of the lung and visceral pleura, irradiated with US, were studied histologically. Paraffin sections were stained with hematoxylin and picrofuchsin by Van Gieson's method.

EXPERIMENTAL RESULTS

The visceral pleura in the control, as usual, consisted of several layers (Fig. 1). The covering cells, with a round or oval nucleus, lay on a limiting membrane. The connective-tissue structures consisted of loosely arranged collagen fibers with reserve folds. The superficial and deep elastic network formed slit-like spaces. Blood vessels and lymphatics were located in the deep collagen layer. Only small vascular plexuses appeared on the surface — so-called "glomeruli" and "arcs." Larger lymphatic vessels were present in the deep layers of the pleura, where they joined lymphatic vessels embedded in the interlobular, intersegmental, and interlobar layers of the lungs. Their junctions are called "deltas." This arrangement of the vessels enables the pleura to participate actively in transsudation processes and in the resorption of fluid from the pleural cavity.

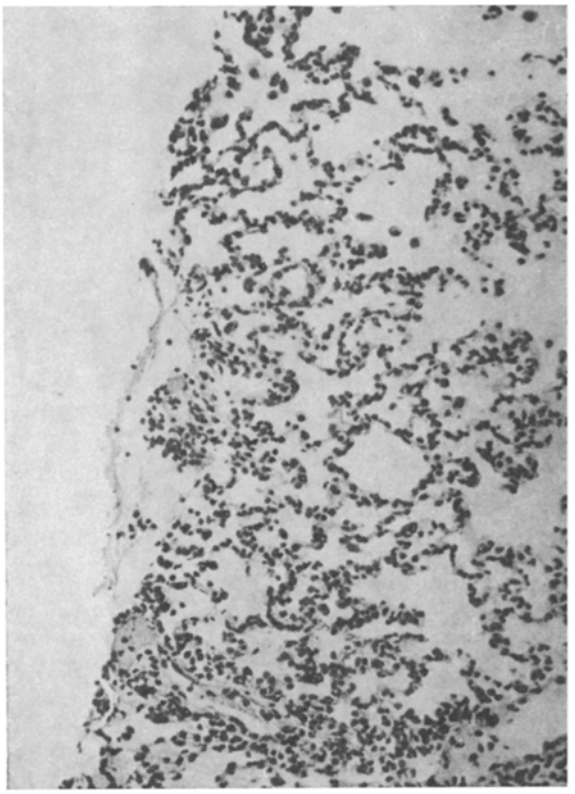

Fig. 3

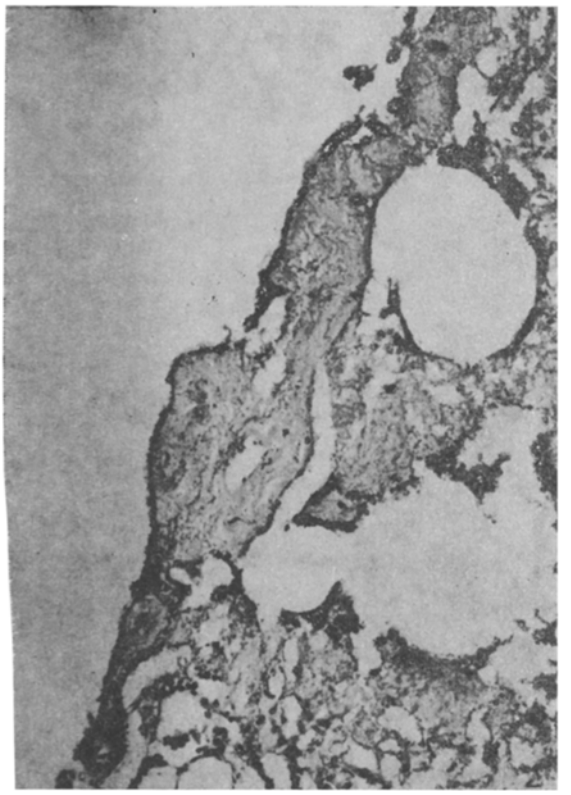

Fig. 4

Fig. 3. Hemorrhage in "delta" region, focal infiltration with neutrophils. Hematoxylin–eosin.

Fig. 4. Coagulation necrosis of pleura and adjacent lung tissue, involving alveoli and bronchioles. Hematoxylin–eosin.

Histological study of the visceral pleura in the animals of series I revealed edema and separation of the fibers. Edema was most marked in the region of the "delta," where dilated lymphatics could be seen (Fig. 2). In one case edema in the "delta" region was combined with hemorrhage. Capillaries in the adjacent alveoli were dilated and congested. In some alveoli hemorrhages were present, and in some places the hemorrhages were acinar in character.

In the animals of series II the visceral pleura, on microscopic examination, just as in the rabbits of series I was edematous and showed loosening of its fibers. In some places the edema was more severe. In the boundary zones moderate leukocytic infiltration of all layers of the pleura was observed. In the adjacent alveoli congestion of the capillaries and small hemorrhages still remained.

On histological examination of the visceral pleura of the rabbits in series III it was observed to be twice the normal thickness due to more severe edema, separation of the fibers, and leukocytic infiltration. The edema was uneven in character and increased in severity toward the "delta." Marked congestion of the capillaries and arterioles was observed in the adjacent alveoli. Areas of leukostasis and single diapedetic hemorrhages were observed.

In the animals of series IV examination of histological sections revealed a serous effusion on the pleura in addition to marked edema of the pleura and focal infiltration with neutrophils, and small hemorrhages (Fig. 3). In the adjacent alveoli congestion of the capillaries and arterioles and solitary diapedetic hemorrhages were still present.

Direct contact of the wave guide with the visceral pleura (series V) was accompanied by coagulation necrosis of the pleura and of the adjacent lung tissue, extending to the alveoli and bronchioles (Fig. 4).

It must be emphasized that in all series of experiments described above the histological changes were focal and mild in character. Comparison of the changes over a period of time shows that the first response was lymphostasis and edema of the pleura. Next followed small

diapedetic hemorrhages in the adjacent alveolar parenchyma, and only after exposure to US for 30 sec/cm^2 did a serous effusion on the pleura develop.

Endothoracic application of low-frequency ultrasound for therapeutic purposes must therefore have limited exposure: not more than 20 sec/cm^2. Contact of the wave guide with the tissue of the lung and pleura is unacceptable.

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SCLEROTIC CHANGES IN THE LIVER IN EXPERIMENTAL CHOLESTASIS AND THEIR REVERSIBILITY AFTER RE-ESTABLISHMENT OF BILIARY DRAINAGE

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KEY WORDS: liver, cholestasis, cirrhosis, reversibility.

The problem of reversibility of pathological changes in organs, and in particular, of sclerotic changes in the liver is of considerable urgency.

In the case of complete occlusion of the common bile duct in rats cirrhosis of the liver develops after 20 days, and progressive cirrhosis with ascites after 25-28 days [6]. This model of cirrhosis of the liver in rats can be obtained within a much shorter time than by the use of hepatotropic agents. Investigation of the reversibility of these cirrhotic changes in the liver after restoration of bile drainage into the intestine is of great theoretical and practical interest, but there is as yet no adequate experimental model with which to study this problem.

To investigate the morphogenesis of the sclerotic changes in the liver and their reversibility, the writers have created an experimental model of long-term cholestasis in rats followed by restoration of the outflow of bile into the intestine.

EXPERIMENTAL METHOD

An experimental model of mechanical jaundice was produced in 54 rats weighing 140-160 g by ligation of the common bile duct and its division between two ligatures. In order to observe the time course of the morphological changes, some rats (n = 24) with a ligated bile duct were killed after 5, 10, 20, and 30 days of cholestasis. The drainage of bile was restored in the remaining rats (n = 30) after cholestasis for 10 and 20 days. At these times the stumps of the duct were considerably dilated and contained transparent bile. Choledochoduodenostomy was performed by gluing the dilated duct to the duodenum. To restore the drainage of bile into the intestine, the bile duct was punctured through the duodenal wall, and the

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