COMPARATIVE ANALYSIS OF MORPHOLOGICAL CHANGES IN LUNG TISSUE CAUSED BY HIGH-ENERGY YAG LASER RADIATION AND HIGH-FREQUENCY ELECTRIC CURRENT

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In recent years high-energy lasers and high-frequency electric currents have been widely used in clinical practice for resection of the lung and for endoscopic operations [1, 2, 4]. Meanwhile morphological changes in lung tissue under the influence of a neodymium YAG laser have not yet been adequately studied. There have been reports that the radiation of a YAG laser can be used by a contact method [3]. The aim of this investigation was to determine the character of morphological changes in lung tissue under the influence of radiation of an Nd:YAG laser (by contact and no-contact methods) and of the high-frequency electric current traditionally used in lung surgery.

EXPERIMENTAL METHOD

Radiation from an Nd:YAG laser with wavelength of $1.06~\mu$ and power of 40 and 60 W was used. The laser radiation was applied through a quartz light guide $400~\mu$ in diameter. The source of the high-frequency electric current was a type S-500 electrosurgical apparatus. The electric current was applied through electric probes. Albino rats weighing 150-200 g, male and female, were used in acute and chronic experiments. In three series of experiments, the animals were exposed to laser radiation with a power of 40 and 60 W and to a high-frequency electric current. Laser radiation was used by contact and no-contact methods. A lateral thoracotomy was performed on the animals under inhalation anesthesia, the lung was brought out into the wound, the laser radiation and electric current were applied and the lung tissue perforated to a thickness of 1 cm. Columns of lung tissue with regions of exposure to laser radiation and electric current were excised for microscopic study and fixed in 10% formalin solution. Histological sections were stained with hematoxylin-eosin, alcian blue, and by Weigertls and McManus' methods. The depth of thermal injury to the lung tissue was measured by means of a type MOB-1-15 screw-operated ocular micrometer (objective 10), and the results subjected to statistical analysis. The animals were taken from the experiment on the 3rd and 7th days.

EXPERIMENTAL RESULTS

After irradiation of the unchanged lung parenchyma by YAG laser with a power of 40 W, the formation of a coagulation film, grayish black in color, was observed microscopically on the surface of the visceral pleura. This film appeared locally only in the zone which was irradiated. The visceral pleura surrounding the zone of irradiation did not change in its appearance. Leakage of air from the lung at the site of the coagulation scab was not observed. Perforation of the segmental vessels by laser radiation led to profuse bleeding, and in such cases the hemostatic effect was inadequate. When laser radiation with a power of 60 W was used, a canal 2 mm in diameter was formed quickly in the lung tissue. The time required to form a canal 10 mm deep in the lung tissue depended on the power of laser radiation and the mode of its

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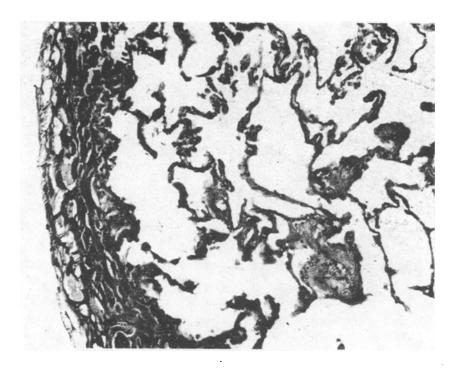


Fig. 1. Zone of necrosis with uniform basophilic masses and separate fragments of elastic fibers. Exposure to radiation of YAG laser with a power of 40 W. Staining with hematoxylin and eosin. $180\times$.

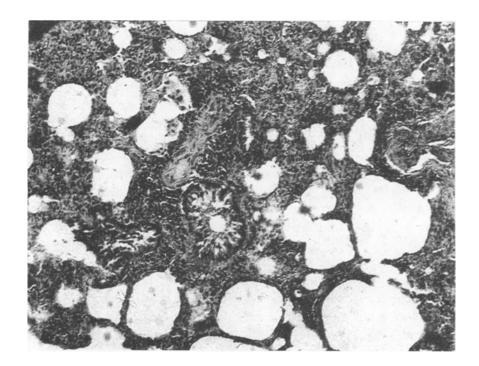


Fig. 2. Lumen of alveoli reduced in size, some vessels destroyed, some thrombosed; bronchi in spasm. Exposure to radiation of YAG-laser with a power of 40 W. Stained with hematoxylin and eosin. $180\times$.

application. If the no-contact method and a power of 40 W were used this time was 15 ± 0.4 sec. Contact application of laser radiation reduced this time to 7.0 ± 0.6 sec. An increase in the power of laser radiation to 60 W shortened this time to 10.0 ± 0.1 sec, and with the no-contact method, to 4.0 ± 0.2 sec. Perforation of the wall of a bronchus by irradiation

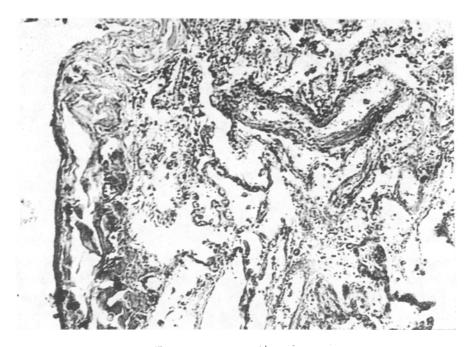


Fig. 3. Lung tissue 3 days after laser coagulation. Serous exudate present in alveolar lumen. Alveolar septa in a state of plasmarrhagia. Stained with hematoxylin and eosin. $180\times$.

with a power of 40 W took place after 13.0 ± 0.8 sec, and with a current of 60 W, after 3.0 ± 0.8 sec. With the contact method of application the time required to form a canal was sharply reduced, to 0.1 sec. During electrocoagulation of the lung tissue, a coagulation scab, indistinguishable in density from that produced by the laser, was formed and was visible macroscopically on the surface of the visceral pleura, but it was three times larger than the area of contact of the electrode. During application of the electric current, coagulation and drying of the lung tissue took place with the formation of a carbon deposit at the end of the electrode, as a result of which coagulation ceased completely. For this reason perforation of lung tissue with a thickness of 10 mm cannot take place without periodic interruption of exposure to the electric current.

Histological study of the lung parenchyma exposed to laser radiation with a power of 40 W revealed three distinct zones of injury. The first zone, measuring $25.3 \pm 2.8 \,\mu$, consisted of a zone of uniform basophilic masses, with a parallel arrangement of the separate fragments of elastic fibers on the surface of the scab (Fig. 1). The second zone was one of damage to the alveolar septa: lesions measuring $44.0 \pm 5.5 \,\mu$ were characterized by basophilia, oxyphilia, picrinophilia, and a decrease in size of the alveolar lumen; coagulation thrombi, mainly eosinophilic, and also concentrations of erythrocytes were found in the vessels. The blood vessel walls were destroyed and their remains oxyphilic, with signs of segmental picrinophilia; the small bronchi in this zone were completely destroyed (Fig. 2). The third zone of laser damage was characterized by vascular disorders in the form of spasm of the vessels. The bronchi were also in a state of spasm. The thickness of this zone was $4950.5 \pm 112.3 \,\mu$. On the boundary with unchanged lung tissue there was a narrow strip of hyperemia with foci of dystalectasis and acute emphysema.

With an increase in the power of laser radiation to 60 W, the same changes as with a power of 40 W were observed, but the second zone of destructive changes was enlarged to $125.6 \pm 6.2 \,\mu$. The character of the lung damage was not found to depend on the method of laser irradiation.

After 3 days the laser scab showed no tendency to enlarge $(22.3 \pm 5.5 \,\mu)$. The inflammatory manifestations were increased in severity. A serous exudate containing a few erythrocytes, desquamated epithelial cells, single lymphocytes and polymorphonuclear leukocytes, together with a large number of siderophages were identified in the lumen of the alveoli. In some alveolar septa signs of plasmarrhagia were present, together with perivascular infiltration with lymphocytes and polymorphs. Some vessels were empty, and in others coagulation thrombi were found (Fig. 3). Widening of the zone of vascular disorders could be seen. Inflammatory manifestations 7 days after laser irradiation were replaced by signs of organization in the zone of necrosis, in the form of invasion by young granulation tissue. However, no evidence of normal organization could be found at these times.

TABLE 1. Morphometric Characteristics of Zone of Lung Damage Following Exposure to Nd:YAG Laser and High-Frequency Electric Current ($M \pm m$)

Procedure to which exposed	Dimensions, µ			
	first zone	second zone	third zone	all zones of injury
High- frequency electric current YAG laser	253±18,0		2000,4±145,2	2160±153,2
40 W	25.3 + 2.8	44.0 + 5.5	4950.4+112.3	5085.5 + 180.3

 $26,2\pm4,2$ $125,6\pm16,2$ $4825,8\pm142,0$ $4976,7\pm162,8$

Histological changes in the lung tissue under the influence of a high-frequency electric current were similar to those observed under the influence of the YAG laser. However, with the electric current, a considerable increase in size of the zone of necrosis was observed (253.4 \pm 18.0 μ). Only the collagen and elastic carcase of the alveolar septa, with no cells, could be seen in this zone. The muscular coat of the blood vessels was completely destroyed and homogeneous in structure. The bronchi also were totally destroyed. The first zone of destructive lesions bordered on a zone 2000.4 \pm 145.2 μ wide, which it was impossible to divide into subzones, and which consisted of a carcass of basophilic structures from the outlines of the destroyed alveoli with blood vessels filled with erythrocytes and erythrocytic debris; in some vessels coagulation thrombi could be found. The carcass was immured in the erythrocytic debris. Vascular disorders, characterized by marked congestion, by recent hemorrhages into the alveoli, and by edema of the lung tissue were more marked than after laser irradiation. On the boundary with the unchanged lung tissue there were areas of microtelectasis. The morphometric characteristics of the zones of injury to the lung after exposure to the electric current and laser irradiation are given in Table 1.

The investigation thus showed that radiation from a neodymium YAG laser is characterized by high penetrating power, especially with a dose of radiation of 60 W and the contact method of application. Laser radiation with a power of 40 W can be used to coagulate the lung. The dimensions of the area of laser necrosis are 10 times smaller than those of electronecrosis. The perifocal reaction of the lung tissue to laser irradiation is characterized by marked vascular and bronchial spasm, a favorable factor when laser radiation is used in clinical practice. The morphological changes in the lung were found not to be dependent on the mode of application of laser radiation.

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