sity was not as great as was described previously. However, the fact that there was a change in the vascular network in an investigation conducted by the method of examination of preparations which we adopted, confirms the previous results. Our data indicate some specific structural changes in the vessels in hypertension. The structural changes observed can evidently be explained by two processes: a decrease in caliber of the vessels and complete closing of some arterioles and capillaries. The change in the vascular network is an important fact which must evidently affect the pathogenesis of hypertension.

LITERATURE CITED

CHANGES IN PULMONARY MICROVESSELS OF RATS WITH BRONCHIOLO-ALVEOLAR FIBROSIS INDUCED BY INTRABRONCHIAL INJECTION OF TRYPSIN

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The interest of research workers has recently been drawn to interstitial diseases of the lungs (IDL), which are characterized by diffuse septo-alveolar fibrosis and alveolitis [6, 10]. All IDL are characterized not only by immunopathological processes of various kinds [6, 9], but also by systemic lesions of the microcirculation and the development of an alveolar-capillary block, which is the cause of the patients' death [8, 10]. Meanwhile the histogenesis of these lesions and, in particular, relations between specific (immunopathological) and non-specific (regenerative) changes in the microvessels in IDL has not been adequately studied.

It was accordingly decided to study changes in the microvessels of the lungs in experimental bronchiolo-alveolar fibrosis induced by intrabronchial injection of trypsin.

EXPERIMENTAL METHOD

Experiments were carried out on 35 noninbred albino rats of both sexes weighing 260-320 g, into which 0.5 ml of a solution of trypsin (from Spofa, Czechoslovakia) in a concentration of 50 mg in 1 ml of isotonic sodium chloride solution was injected intrabronchially under ether anesthesia by tracheotomy. The animals were killed 1, 3, 5, 8, 10, and 15 days after the injection (five to seven rats at each time). Pieces of the lungs were fixed for light and electron microscopy by the usual methods [5, 7]. Serial frozen sections 5-7 μm thick for immunomorphological investigations were stained by the direct Coons' method with luminescent serum against rat globules, and also by the method of Goldwasser and Shepard to reveal complement [2]. The vascular system of the lungs was injected with a mixture of ink and gelatin in two or three animals at each time post mortem [5].
Fig. 1. Morphology of rat lung tissue on first days after intrabronchial injection of trypsin. a) First day of experiment: alveoli slightly collapsed, round cells (alveolar macrophages and single polymorphs) in their lumen and what are evidently trypsin crystals also visible (arrow). Impregnation with silver by Gomori's method. 250x; b) 3rd day of experiment: an eosinophil (E), a leukocyte (L), a degenerating alveolocyte (Al), and finely granular material (albuminous fluid) visible in alveolar lumen. Transmission electron microscopy (TEM). 3500 x; c) 5th day of experiment: lung tissue moderately collapsed, marked edema present around artery at level of intralobular bronchus. Silver impregnation by Gomori's method. 100 x; d) alveolar septum (S) swollen and thickened due to edemas of interstitial substance, basement membrane (BM) of alveolocytes swollen, two leukocytes (L) visible in lumen of one capillary (CL), Al) Alveolocyte. TEM. 3500 x.

Sections for light microscopy were stained with hematoxylin and eosin, with fuchsin by Weigert's method and counterstaining by Van Gieson's method, impregnated by Gomori's method, and treated by the PAS reaction with amylase control. Ultrathin sections were studied on the Tesla BS-500 electron microscope.

EXPERIMENTAL RESULTS

Traces of yellowish fluid were found in the pleural cavities of the rats 24 h after injection of trypsin. The lungs were enlarged, doughy in consistency, with multiple dark red areas up to 1-2 μm in diameter. Examination with the light microscope revealed focal atelectasis, and the lumen of the alveoli contained erythrocytes, pinkish fluid, desquamated epithelial cells and macrophages, and trypsin crystals (Fig. 1a). Eosinophilic masses resembling hyaline membranes were seen on the alveolar walls. Marked interstitial edema was present with a few polymorphs in the edematous connective tissue (especially peribronchial and perivenous). On electron microscopy the alveolar lumen was seen to contain granular masses, eosinophils, polymorphs, macrophages, and desquamated alveolocytes (Fig. 1b). The type I alveolocytes were swollen, with marked edema and focal destruction of their cytoplasm, as well as vesicular dilatation of the tubules of the endoplasmic reticulum.

On the 3rd day the lungs remained variegated: small lobular and acinar bluish-red areas alternated with red and pale pink areas. Microscopically, concentrations of large round cells of alveolar macrophage type and