ELECTRON-MICROSCOPIC INVESTIGATION OF THE AIR–BLOOD BARRIER IN PNEUMOSCLEROSIS AND PULMONARY EMPHYSEMA

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Chronic inhalation of a copper aerosol leads to the development of pneumosclerosis and emphysema. Thickening of the basal layer of the air–blood barrier can be regarded as the structural basis of the disturbance of gas exchange.

Few electron-microscopic investigations of the lungs have been made in pneumosclerosis and emphysema, and they have not yet given a clear picture of the fine structural changes taking place in various components of the air–blood barrier in these pathological states [1, 8, 12-15]. Yet data of this type are vital to the further elucidation of the pathogenesis of respiratory disturbances in chronic bronchitis [7], pneumoconiosis [4, 5, 11], and disorders of the pulmonary circulation.

The object of the present investigation was to study the submicroscopic changes in the basal layer of the air–blood barrier in experimental pneumoconiosis produced by prolonged administration of a copper oxide aerosol.

EXPERIMENTAL METHOD

Electron-microscopic studies were made of the lungs of nine rats aged 270 days and weighing 310-340 g; six of these animals had inhaled a copper oxide aerosol daily for 150 days, while the other three were controls. Pieces of lung tissue were studied histologically and electron-microscopically. Ultrathin sections were examined in the UÉMV-100A electron microscope.

EXPERIMENTAL RESULTS

Electron-microscopic investigation of the lungs of the experimental animals inhaling copper oxide showed considerable changes in the structure of the air–blood barrier. These changes varied in different parts, evidently because of the uneven distribution of the inhaled dust.

The mucoprotein matrix of the basal layer under normal conditions is a layer 310-360 A in thickness, of low electron-optical density, and located between the basement membranes of the endothelial cells on one side and of the alveolar cells on the other side. These three structures together (endothelial cell, basal layer, alveolar cell) constitute the air–blood barrier through which the gas exchange takes place (Fig. 1a).

Considerable thickening of the basal layer was found in the experimental animals. In some places it was 14 times wider than the basal layer of the control animals. Its electron-optical density was also increased. Characteristically the other components of the air–blood barrier (endothelial and alveolar cells) were only very slightly changed. The increased thickness of the basal layer of the air–blood barrier was

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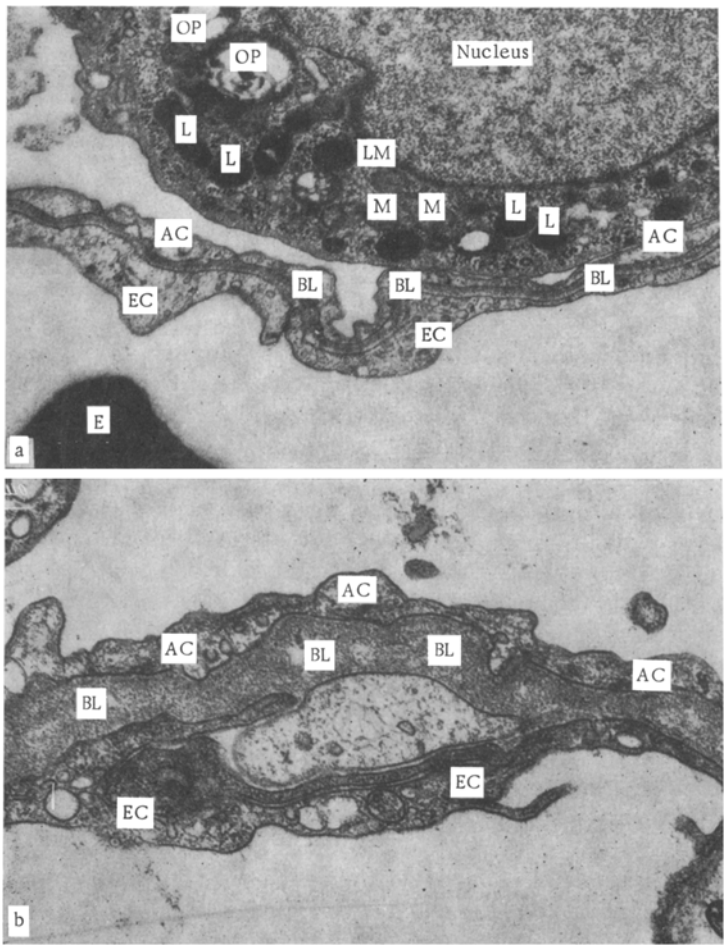

Fig. 1. Ultrastructure of air–blood barrier under normal conditions and in pneumosclerosis: a) thin basal layer of air–blood barrier of control animal (29,400×): AC) cytoplasmic process of alveolar cell; BL) basal layer 310-360 A in thickness; EC) cytoplasmic processes of endothelial cell; LM) lung macrophage; OP) osmiophilic particle; L) lyosome; M) mitochondria; b) considerable thickening of air–blood barrier of experimental animal (29,400×): BL) basal layer 2500-5000 A in thickness.

not uniform (Fig. 1b): in some places its width reached a maximum (5000 A), while in others its increase in thickness was less marked (800-2500 A).

In many areas the changes in the air–blood barrier were more pronounced still. Not only was the basal layer thickened, but large number of collagen fibers appeared in it. Connective-tissue cells were only infrequently found.

The characteristic spur projections [2, 6] of pneumosclerosis and pulmonary emphysema consist of greatly thickened and shortened parts of the air–blood barrier projecting into the lumen of the dilated alveoli. The ultrastructure of a typical spur consists of a pulmonary capillary 6-8μ in diameter, located terminally on a projection of the thickened air–blood barrier measuring from 15 to 40μ in length and 3-4μ in section. The pulmonary capillaries are often found on the lateral surface of the spur (Fig. 2) or at its base. Frequently the spur consists of ground substance of connective tissue, or reticulin and elastic fibers, and no capillary is present. The surface of this spur of the thickened part of the air–blood barrier is covered with alveolar cells. If the spur undergoes marked shortening, it forms folds and reduplications of the basal layer thickened to 1700-2500 A. Spurs were also observed in the control, but they were much less common. They are probably an age feature of lung tissue [6].

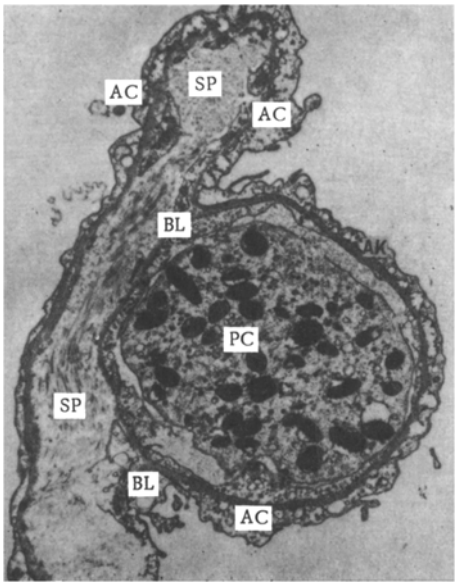

Fig. 2. Ultrastructure of spurs on air–blood barrier in pneumosclerosis and pulmonary emphysema (7500×). AC) Cytoplasmic process of alveolar cell; BL) basal layer 1700–2400 A in thickness; PC) pulmonary capillary; SP) spur.

These results also indicate that the changes taking place in lung tissue during prolonged inhalation of a copper oxide aerosol are noninflammatory in character and arise as the result of intercellular and cellular hyperplasia [9, 10], developing in the various structural components of the air–blood barrier.

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