

TECHNICAL NOTE

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Scanning Electron Microscopic Ultrastructural Alterations of the Pulmonary Alveolus in Experimental Drowning

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ABSTRACT: Scanning electron microscopic structural and ultrastructural alterations of alveolar wall in experimental drowning are described. The results obtained confirm the literature's data. Peculiar microlesions affecting Type I alveolar cells, the basement membrane, and sometimes the capillary endothelium could also be observed. Finally, Type II alveolar cells show alterations which could be related with a possible direct action of the drowning liquid.

KEYWORDS: pathology and biology, cardiovascular system, drowning

Acute pulmonary emphysema is generally known as the most typical anatomic-pathological aspect of drowning. Hyperextension and sometimes laceration of the alveolar septa is found in man, and frequently in experimental animals. Such alveolar emphysema is often combined with interstitial emphysema and edema; atelectatic areas are common too.

Literature concerning the lung's morphology in drowning using the scanning electron microscope (SEM) [1,2] mainly deals with the alveolus' general architecture and the drowning liquid's effect on the red blood cells.

We have not heard of studies being made with the SEM aimed at the alveolar epithelium's modification caused by this type of asphyxia.

Materials and Methods

Young albino rats were used as experimental animals. They were completely immersed in plain water until death. Immediately after death the lungs were removed and cut into sections about 1 mm thick. The sections were repeatedly washed in 0.1M phosphate buffer pH 7.4, then fixed in glutaraldehyde 2.5% for 4 h; after another wash with phosphate buffer, they were dehydrated by passage through a series of grades of alcohol and then placed in amyl acetate. Finally, the specimens were dried at critical point and gold coated.

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The same procedure was carried out with lung sections obtained from control beheaded rats.

Observation was performed with a Cambridge 250 Stereoscan.

Results

Drowned lung looks homogeneous in all rats. Low magnification reveals atelectatic areas together with wider areas where the alveoli look dilated, with thinned and sometimes discontinuous walls. At higher resolution an abnormal stretching of the alveolar septa can be observed: they are at times so extended as to look laid down on the next alveoli's bottom.

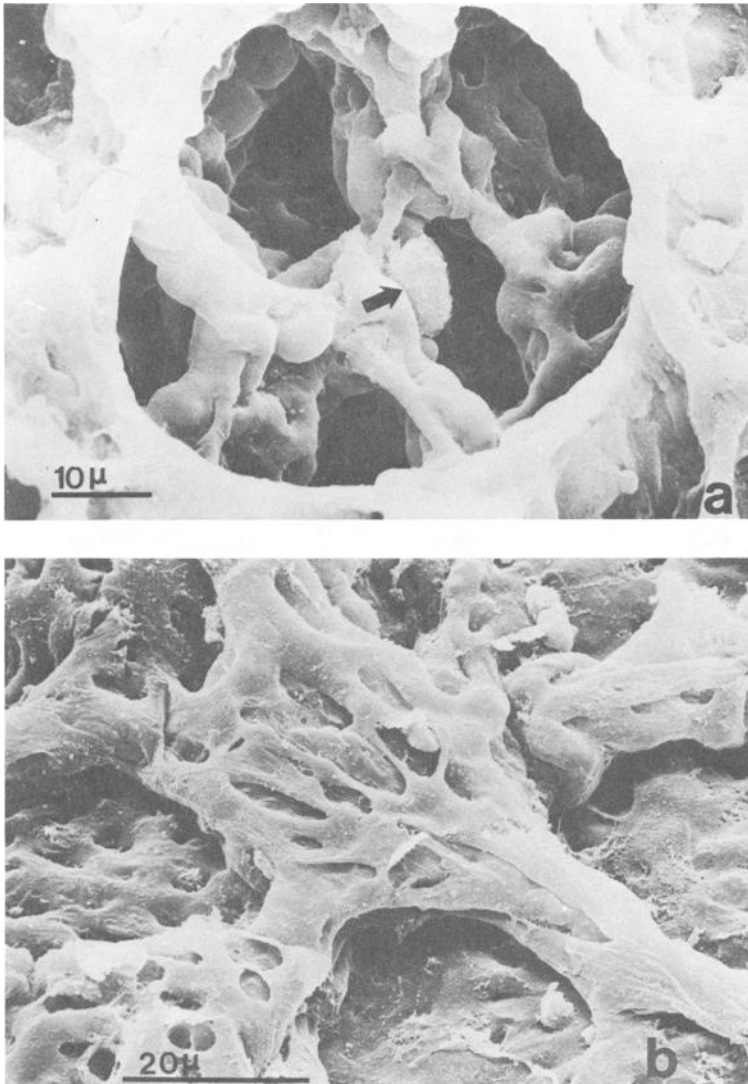


FIG. 1—(a) Normal aspect of control rat's lung: alveola with interalveolar septa. The arrow indicates an alveolar macrophage. (b) Aspect of drowned rat's lung: all parts of alveolar walls look stretched and the capillaries are thinned.

Capillaries in such septa are so thinned that they cannot possibly contain red blood cells (Fig. 1). Areas of emphysema also show numerous lacerations of Type I alveolar cells, causing exposure of the basement membrane and of the underlying capillary net (Fig. 2a). A clear disjunction, creating an empty space between epithelium and capillaries, is often evident. Some fields show wall discontinuity involving the capillary endothelium with exposure of entire red blood cells (Fig. 2b). Type II alveolar cells appear at times "sunk" in their usual sites, or projecting into the alveolar lumen. Surface morphological characters seem con-

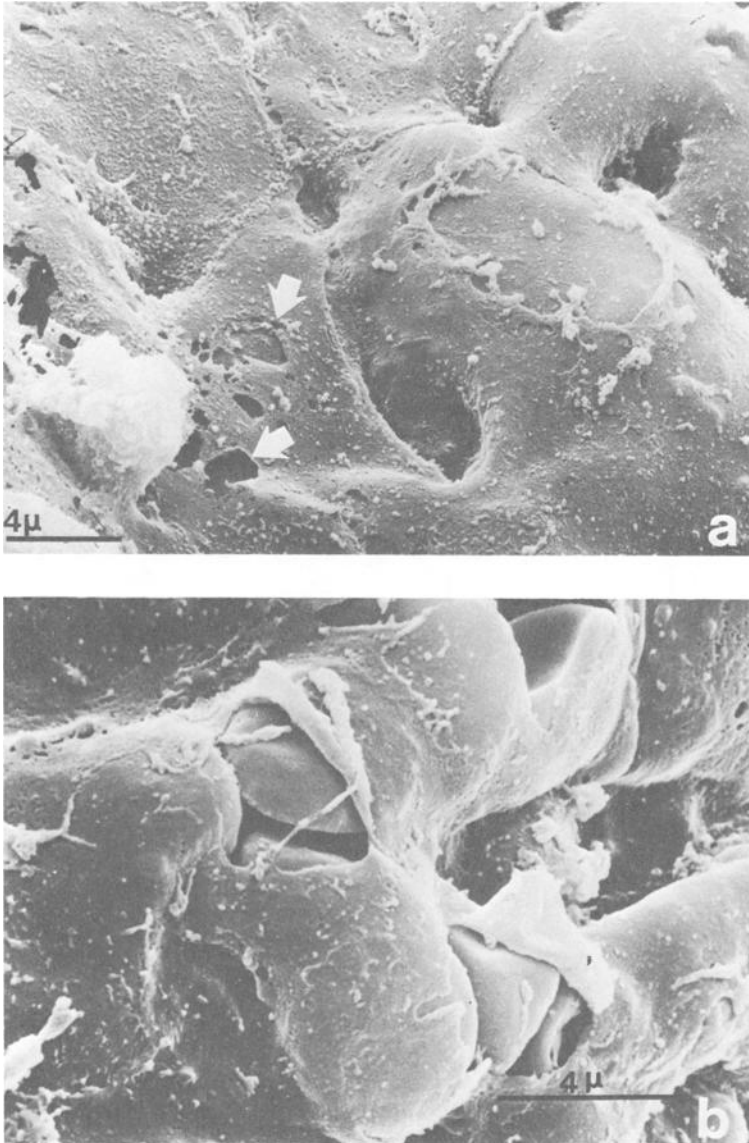


FIG. 2—Two aspects of the alveolar wall in drowned rat's lung. Type I pneumocytes appear lacerated (a) with disjunction (arrow) from the basement membrane. (b) Lacerations involving the capillary wall with exposure of red blood cells.

stantly affected, showing disorganization, shortening, and reduction of the number of microvilli (Fig. 3).

Discussion

This preliminary study shows that SEM observation confirms literature data concerning the aspect of the lung in drowning asphyxia. The technique employed also allows a much

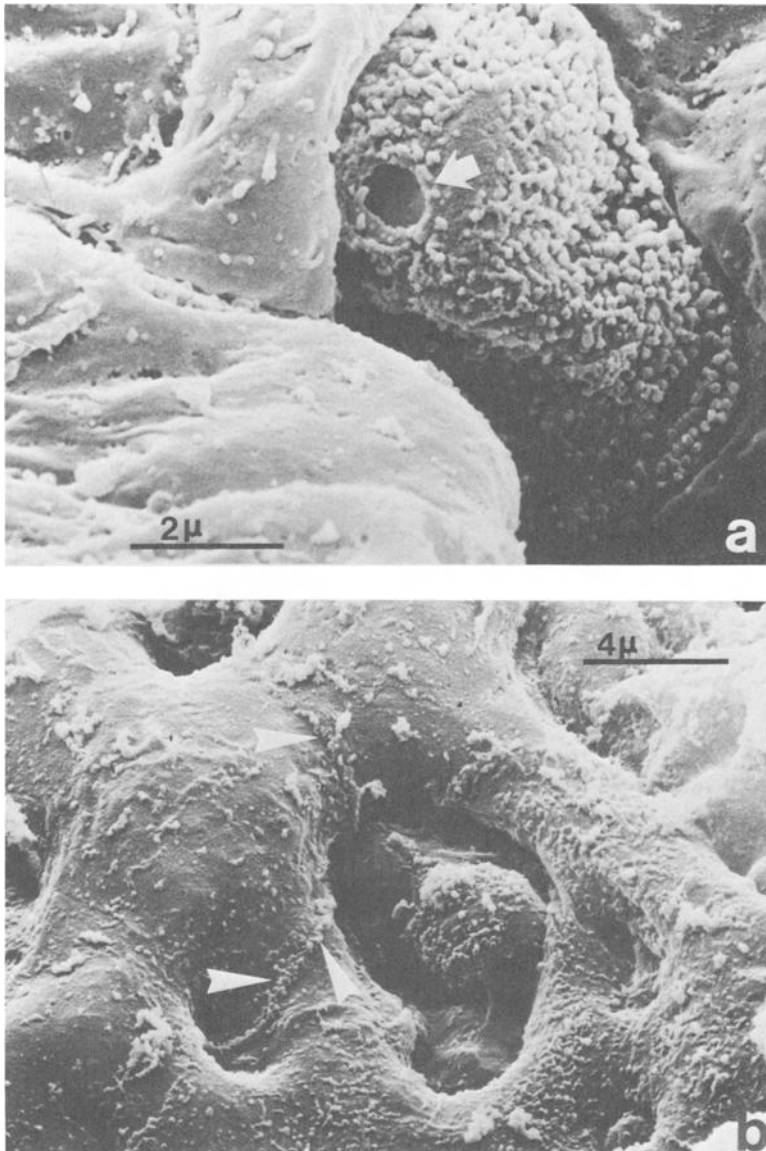


FIG. 3—Type II alveolar cell with numerous microvilli and evident (arrow) sign of secretory activity. (b) Type II pneumocyte in drowned rat's lung. The cell looks shrunken, and the microvilli disarranged. Arrows indicate the cell limit between two Type I pneumocytes.

finer structural and ultrastructural research on the alveolar modifications caused by such an asphyxia.

In fact we could observe along with coarse alterations of the alveolar septa, specific microlesions affecting the epithelium, the basement membrane, and the capillary walls.

It must be mentioned that no judgement can be made concerning the pulmonary edema, because the technique requires the removal of all intra-alveolar liquid before the fixation.

As to the ultrastructural anatomical damage of the alveolar wall, we think that it can be a real entry for red cells into the alveola, which is also frequently observed in light microscopy.

Up to now observations on modifications of Type II alveolar cells seemed insufficient to express hypotheses. Nevertheless we believe that the described surface disorganization can give an answer to how the cell membrane makes contact with an extraneous liquid, hypotonic in this case.

The obtained results are, however, sufficient to induce further studies on possible different behavior of these cells when exposed to drowning liquids of various natures.

References

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