The aim of this investigation was to study the ultrastructure of the alveoli in experimental induced pneumonia in pigs inoculated with Haemophilus pleuropneumoniae serotype 1 as a complement of a previous work with light microscopy in order to disclose the pathogenesis of the lesions observed.

Materials and Methods: Two pigs of conventional cross breed, 32 days old were used. They were free of Bordetella bronchiseptica and Haemophilus spp. by nasal sampling and the lungs appeared normal at roentgenographic examination. The pigs were inoculated intranasally with 2 ml of a suspension of Haemophilus pleuropneumoniae serotype 1 containing 1.5 x 10^9 C.F.U. Both animals were observed several times daily and carefully examined once each day when their rectal temperature was recorded and roentgenological studies were performed. The pigs were killed by electrocution and bleded, one at 24 hours and the other at 48 hours post inoculation (P.I.). Samples were taken for bacteriology and histology, for electron microscopic examination, sections of lung tissue were cut into pieces of approximately 1 mm^3, fixed in 10% buffered formalin, post fixed in 25% OsO_4 and embedded in Araldite. Ultrathin sections were stained with uranyl acetate and lead citrate and examined in a Philips electron microscope, E.L. 201.

Results: Pathological changes found in the ultrastructural investigation comprised all components of the alveolus. At 24 hours P.I. both types of epithelial cells showed degenerative changes characterized by different degrees of intracellular edema, cells formation and detachment from the basement membrane. At 48 hours P.I. almost all type II pneumocytes were lacking dense lamellar bodies, and type I pneumocytes were filling up the alveolar space. At 24 hours P.I. all capillaries were dilated and congested, some of them in different stages of disintegration. Blood monocytes and occasional neutrophils were also observed. A few platelets were seen associated with disintegrated erythrocytes, fibrin was not observed. At 24 hours P.I. the alveolar septa were distended by edema, fibrin and mononuclear cells. At 24 hours P.I. the alveolar spaces were distended with fluid, cellular debris, monocytes and few neutrophilic leukocytes. At 48 hours P.I. the lumen of most alveoli was collapsed. The content appeared the same as at 24 hours P.I. Few bacteria were observed free in the lumen or inside macrophages.

Discussion and Conclusions: The degenerative changes observed in the epithelial cells were similar to those reported in pneumonia produced by Salmonella cholerae suis and also to those seen in connection with histoplasmosis, radiation injuries and haemorrhagic shock indicating a non specific cellular response. The packed erythrocytes within the alveolar capillaries appeared to be in different stages of disintegration. This material, identified as fibrin with light microscopy, was found consisting in erythrocytes debris with electron microscopy. These findings are in many respects rather similar to the histology of fibrinoid thrombi found in the glomerular capillaries in association with Haemophilus infections and in vivo salmonellaosis, an experimental model of generalized sharkan response. The marked degenerative changes seen in the endothelial cells at 48 hours P.I. could be due to hypoxemia caused by the thrombosis formation or by a combination of toxic, endotoxins and hypoxic effects and explain the coagulative and infiltrative changes found in the alveolar septa and the interstitial connective tissue. In this study most of the inflammatory cells were identified as macrophages, some with phagocytosed eosinophilic material or bacteria. In addition some neutrophilic leukocytes were observed.