

# THE HUMAN LUNG AND PLEURA IN VIBROACOUSTIC DISEASE

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**Introduction** Vibroacoustic disease (VAD) is caused by long-term exposure (years) to low frequency noise (LFN) ( $\leq 500$  Hz, including infrasound) [1]. In VAD animal models, the respiratory system was shown to be a target for this acoustic aggressor [2]. Simultaneously, the 11 respiratory tract tumors we have observed in VAD patients, were all of a single type of cellularity: squamous cell carcinomas. In this report, fragments of lung and pleura of a VAD-lung cancer patient are studied through light and electron microscopy.

**Methods** The patient is a retired 62 year old, smoker, male Caucasian. He worked as an aircraft technician for over 30 years. With the patient's informed consent, fragments of lung and pleura were removed during the surgery for the lung carcinoma. Specimens for light microscopy were formalin-fixed, paraffin-embedded, hematoxylin, eosin and fuchsin-rhesorcin stained. For transmission electron microscopy, sections of lung tissue were fixed at room temperature in an aldehyde mixture, washed in buffer, postfixed in a ferricyanide-reduced osmium solution, dehydrated through a graded ethanol series, and embedded in Epon. Samples were then sectioned in an LKB ultramicrotome (Sweden), stained with uranyl acetate and lead citrate, and viewed with a JEOL 100C (Japan) electron microscope.

**Results.** Vessel walls are thickened, the bronchioli and the rest of the lung parenchyma have numerous macrophages, some with brown pigment, and other with tar.

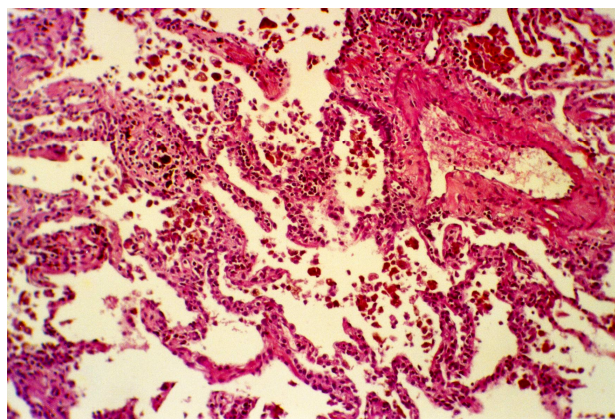


Fig. 1. Light microscopy – Lung parenchyma. A functional unit consisting of a very thickened artery, and a bronchiole filled with macrophages containing brown pigment. (x200)

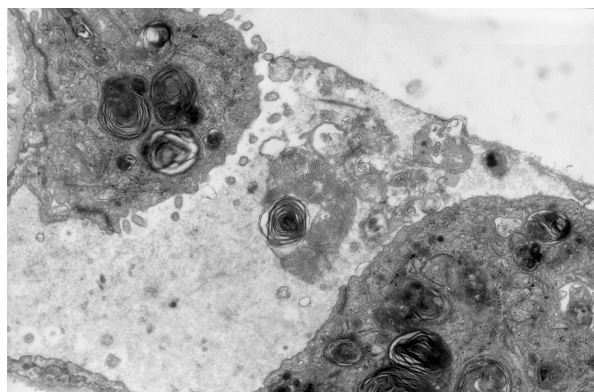


Fig. 2. TEM – Lung parenchyma. Two type II pneumocytes surrounded by hypophase where a lamellar body is releasing surfactant that covers the surface of the liquid film. (x16000)

TEM images disclosed a marked reduction of type I pneumocytes with a large increase of type II pneumocytes. Interstitial fibrosis is not uniformly distributed but in some fields, it becomes a very prominent feature.

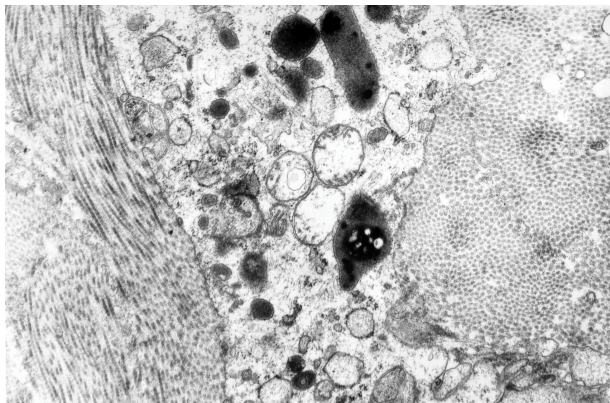


Fig. 3. TEM – Lung parenchyma. An apoptotic pneumocyte is visible, filled with apoptotic vesicles and surrounded by intense bundles of collagen (x20000)

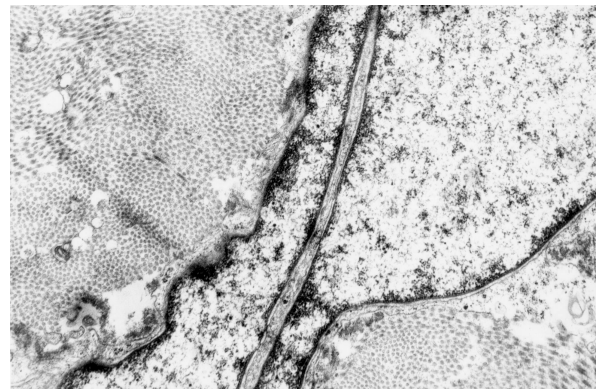


Fig. 4. TEM – Pleura. What appears to be a fibroblast, with marginal condensation surrounded by bundles of collagen. (x20000)

Cells under apoptotic processes are very common. The most remarkable feature within the pleura is the increased thickness due to the intense proliferation of collagen.

**Discussion** The increase in interstitial fibrosis and the increased thickness of the pleura and vessels is an expected situation since these same aspects have been observed in LFN-exposed rodents [2]. The reduction of the number of type I pneumocytes in contrast with the increased amount of type II pneumocytes is also seen in other pulmonary stress situations. The very frequent images of apoptotic processes were unexpected and indicate possible avenues of research in the future. All these aspects, taken together with the discrete results obtained with high-resolution CT Scan of the lungs of VAD patients [3], and with the almost normal pulmonary functional tests, strongly emphasizes the insidious and almost silent progression of VAD.

**Keywords:** low frequency noise, interstitial fibrosis, pleural fibrosis, pneumocytes, apoptosis

## References

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