



# Quantitative Anatomical Findings for Lung Parenchyma

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## Introduction

The ventilatory disturbances produced by centri-lobular emphysema have been shown by the use of a model to be almost exclusively caused by an increased diffusion time of gas molecules through distended centriacinar or centri-lobular spaces before they reach the respiratory exchange area of the lung [1]. However, such a model hardly accounts for the severity of most of the changes observed in cases of centri-lobular emphysema. Usually only the upper zones of the lung and less than 40% of the parenchymal volume show centriacinar spaces, and it is uncertain whether, in addition to the centriacinar spaces, some other obstacle to alveolar ventilation may be present throughout the lung. Such an obstacle might be caused by bronchiolar narrowings which were described by McLean [2]. Previously we have shown, using a quantitative method, that widespread bronchiolar stenoses were present in chronic obstructive broncho-pulmonary disease with or without emphysema. Recently it has been emphasized that in chronic obstructive pulmonary disease the obstruction to airflow is situated in small bronchi of less than 2 mm [3]. diameter. The present study, employing morphometric methods, was designed to investigate the quantitative relationship between the alveolar and bronchiolar damage in centri-lobular emphysema, and the severity of the resulting chronic pulmonary hypertension and right ventricular hypertrophy. The importance of permanent structural changes in the pulmonary arteries as a cause of the pulmonary hypertension was also investigated. Eight cases of pure centri-lobular emphysema were studied. Post-mortem examinations were carried out according to a method described previously. The lungs were fixed by endo-bronchial formalin infusion and expanded by a partial vacuum of -30 cm. H20 for 72 hours [4]. The lung volume was measured after fixation by weighing the volume of water it displaced. The lungs were then sliced into five or six sagittal macro-sections with a thickness of 1 cm, and the appearances of CLE were similar to those described by Leopold and Gough [5]. The proportion of the total lung parenchyma made up by the centri-lobular spaces was evaluated by submerging the slices of lung in water and using a stereomicroscope combined with the point-counting method [6]. The proportion of the lung tissue volume due to CLE was calculated for the upper half, the lower half, and the whole lung separately. Twenty standardized blocks of lung tissue, and taken from each lung, were sampled by a stratified randomized technique. These blocks, measuring about 20 x 30 mm, were then processed in the usual way and embedded in paraffin under vacuum. Sections, 5, thick, were stained with haematoxylin and eosin and by Weigert/van Gieson for elastic fibres [7]. The mean linear shrinkage for processing was determined in each case by measuring the length and breadth of fixed blocks and processed slides on the 20 randomized samples calculated from the formula [8]. Elastic and muscular pulmonary arteries as well as pulmonary arterioles were studied qualitatively both in microscopical sections and by post-mortem arteriography. Arteriograms were carried out by a method described previously by Schlesinger [9]. The left and right ventricles were carefully dissected free from fat and weighed separately according to the method described by Fulton, Hutchinson, and Jones. A right ventricular weight less than 65 g. and a Fulton's ratio higher than 2-2 were regarded as normal [10].

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None

## Conflict of Interest

None

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