



Small Pulmonary Artery Showing A Distinct Coat of Circular Muscle

Kjell E J Hakansson*

Department of Respiratory Medicine, Copenhagen University Hospital - Hvidovre, Denmark, Germany

Introduction

Ventilatory disturbances in Centri-Lobular Emphysema cannot be accounted for only by decrease of internal surface area. Indeed, in the majority of the cases studied the intact parenchyma accounted for 50 to 77% of the volume of the lungs and the internal alveolar surface area was only slightly reduced. These results, as previously stressed by Dunnill, are the reverse of the findings in severe PLE cases in which there is frequently minimal respiratory failure and right ventricular hypertrophy, although emphysema may destroy as much as 80% of the lung parenchyma, thus seriously reducing the internal ventilatory surface area [1]. Staub and Gomez emphasized that the ventilatory disturbances in Centri-Lobular Emphysema were mostly due to the enlarged centri-acinar spaces, which were situated in a strategic position between the conductive zone and the respiratory exchange area, and slowed down gas diffusion. As a result, Dunnill pointed out that the number and the distribution of these abnormal air spaces were a more reliable guide than their size and the total lung volume involved [2]. However, the predominant location of the centri-lobular foci in the upper half of the lung, demonstrated here as well as by Snider and Thurlbeck, suggests that this theory cannot account for all the facts. Indeed, more than half of the lung parenchyma and particularly the lower halves of the lungs, which are the best perfused and ventilated generally show much less centri-lobular emphysema [3]. This suggests that other mechanisms are responsible; especially increase of terminal airways resistance. Leopold and Gough noticed inflammatory narrowings in 12% of the respiratory bronchioles supplying Centri-Lobular Emphysema spaces, and narrowing of membranous bronchioles was not reported by them. The present morphometric study of the membranous bronchioles showed that bronchiolar narrowings were scattered randomly inside the lung, including zones without emphysema [4]. Moreover, the degree of these bronchiolar narrowings seemed related to the severity of chronic respiratory failure. These inflammatory stenosis situated at the end of the conductive air passages could play an important part in ventilatory disturbances, as discussed in a previous paper, and were demonstrated in recent physiological studies by Hogg. Right ventricular hypertrophy was noted in 55% of the 75 cases studied by Leopold and Gough, and the majority of authors have noticed the frequency of RVH in Centri-Lobular Emphysema even in cases with moderate emphysema and sometimes at an early stage. Nevertheless, the cause of the PAH or RVH is often uncertain or controversial [5]. Permanent structural changes in the pulmonary arterial system are a matter of debate, and even if pulmonary arterial destruction occurs, it is of minor importance as a cause of chronic pulmonale in Centri-Lobular Emphysema, because in some cases RVH develops with only moderate parenchymal destruction [6]. Compression of the pulmonary arterioles by the centri-lobular air-distended spaces, which was suggested by Dunnill as a cause of pulmonary hypertension, was not found in this study. Heath showed that the small pulmonary arterial vessels often presented characteristic histological features in emphysema when there was associated right ventricular hypertrophy [7]. These changes included development of longitudinal muscle in the intima of pulmonary arterioles, the development of medial circular muscle in the pulmonary arterioles, and a lack of hypertrophy of the medial muscle in the large muscular pulmonary arteries [8]. This triad of structural changes might

account for the increased pulmonary vascular resistances in Centri-Lobular Emphysema [9]. Such vascular changes, however, are not only characteristic of emphysema but are found in all conditions of chronic hypoxia [10].

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Conflict of Interest

None

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*Corresponding author: Kjell E J Hakansson, Department of Respiratory Medicine, Copenhagen University Hospital-Hvidovre, Denmark, Germany, E-mail: Kjell@gmail.com

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