Discussion Remarks: Cellular Contraction in Pulmonary Hypertensive Disease

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The problem of pulmonary hypertensive disease (PHD) is not only that of a reduction of the vascular lumen, but also that of an active mechanism of cellular contraction, in which cells of the arterial wall are involved. These cells are not only smooth muscle cells, but also non-muscular cells.

In PHD, the wall of the muscular pulmonary artery shows a thickening of the media and the intima. The latter contains cells which have features of smooth muscle cells. This same appearance is also seen in a cell found in the sub-intimal layer of a fibrotic intimal lesion. This cell is a myo-intimal cell which has features of a smooth muscle cell (myo-fibril) and of a fibroblast (rough endoplasmic reticulum).

Cells containing microfilaments are also seen in endothelial cells of arteries of PHD, which show an increase of myofibrils when compared with human control cases. Similar filaments of actin have been identified in alveolar interstitial cells of human lungs by Kapanci et al. [1974]. These cells show bundles of myofibrils attached on an elastic fibre and on the alveolar capillary basal membrane. They contain also actin put in evidence by fluorescent anti-actin containing serum. Strips of human pulmonary parenchyma submitted to repeated hypoxia in a saline bath show measurable contraction. All these features suggest that alveolar interstitial cells are contractile and may reduce the pulmonary flow.

References

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