

Study of Lung Function in Cor Pulmonale

The anoxemia and hypercapnia of cor pulmonale have long been thought to originate in difficulty in the exchange of gases between the lungs and the blood. An alternative explanation is that the cor pulmonale subject reacts abnormally to respiratory stimuli, so that the anoxia and hypercapnia may reflect alterations in the lung gas pressures rather than difficulty in gas exchange.

Our findings suggest that afferent reflexes arising from the lungs are of great importance in determining the response of the respiratory centers to anoxia and hypercapnia in normal men. This has not previously been recognized. Whether the reflexes arising from the abnormal lungs in cor pulmonale can also account for the low oxygen pressure and high carbon dioxide pressure in the lungs and blood in such subjects should be determined by experimental investigation.

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Our preliminary investigations demonstrate the importance of these active reflexes. We have studied a single subject with cor pulmonale secondary to deformity of the chest, and found a relatively low proportion of oxygen and a relatively high proportion of carbon dioxide in the alveolar air. This subject tolerates far higher carbon dioxide pressures and lower oxygen pressures at a given chest volume than a normal subject who has been extensively studied. These observations agree with the theory that the anoxia and hypercapnia characteristic of cor pulmonale reflect a depressed sensitivity of the respiratory centers, rather than difficulty in the exchange of gases between the lungs and blood.

We have searched for a possible explanation of respiratory depression independent of the extent of anoxia and hypercapnia. ^{We have found that} A restricted chest volume is equally important as anoxia, and far more important than hypercapnia in stimulating breathing. All these variables act independently and throughout their range, as quantitative measurements show.

To demonstrate the importance of these afferent reflexes, - normal men can rebreathe pure nitrogen with the chest volume unrestricted, for a much longer time than they can rebreathe pure oxygen if the chest volume is restricted. The oxygen pressure in the lungs falls to far lower levels when nitrogen is rebreathed than when oxygen is rebreathed. It was quite impossible for a normal subject to rebreathe a gas mixture with a high oxygen pressure and a normal carbon dioxide pressure for more than a short period of time if the chest volume is restricted, but if the volume of rebreathed air is not limited, the only normal subject tested tolerated the gradual accumulation of sufficient carbon dioxide to pass beyond the point where carbon dioxide stimulated breathing, and into the first stage of anaesthesia.

In order to explore the importance of the above effects in subjects with cor pulmonale, a series of studies of a group of such subjects need to be made. First, the tolerance limits to chemical and mechanical stimulation of breathing may be measured by the techniques noted above, in order to determine their relative importance in the particular subject, and in subjects with cor pulmonale as compared to normal subjects. Next, the alveolar and mixed lung gas pressures of oxygen and carbon dioxide, and the volume exchange of oxygen and carbon dioxide between the lungs and blood may be measured, to determine if the gas exchange at determined pressures is altered. Third, the gas content and pressures of the mixed venous and arterial bloods may be measured, in order to determine their correspondence with the lung gas pressures and volume exchange. Last, the mechanical function of the lungs may be measured, by recording the breathing pattern of the subject at rest, the tidal air, the reserve air, the inspiratory and expiratory vital capacity, the residual air, and the respiratory dead space.

The study in its entirety will determine whether the anoxemia and hypercapnia characteristic of subjects with cor pulmonale reflects some difficulty in the exchange of gases between the lungs and the blood, or an altered sensitivity of the centers controlling respiration as a result of afferent reflexes from the lungs. If, as our observations suggest, the latter explanation is the true one, a fundamental reorientation of the accepted theory of the pathogenesis of the disability in cor pulmonale will be necessary.

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