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PATHOPHYSIOLOGICAL ASPECTS OF EMPHYSEMA

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The primary cause of emphysema is cigarette smoking, which is responsible for up to 90% of all cases. Cigarette smoke contains many harmful chemicals, including tar, carbon monoxide, and nicotine, which can damage the alveoli and trigger an inflammatory response. Other environmental factors, such as exposure to air pollution, can also contribute to the development of emphysema.

Genetic factors also play a role in the development of emphysema. In particular, a deficiency of alpha-1 antitrypsin (AAT), a protein that helps to protect the lungs from damage, can increase the risk of developing the disease. Individuals with AAT deficiency are more susceptible to the damaging effects of cigarette smoke and other environmental toxins.

The pathogenesis of emphysema involves a complex interplay of inflammatory cells, enzymes, and cytokines. Inflammatory cells, such as neutrophils and macrophages, are activated by cigarette smoke and other environmental toxins, leading to the release of enzymes that can break down the walls of the alveoli. This breakdown of the alveoli results in a loss of surface area for gas exchange, which in turn leads to shortness of breath and other symptoms of emphysema.

In addition to the destruction of the alveoli, emphysema can also lead to the development of pulmonary hypertension, a condition in which the blood vessels in the lungs become narrow and stiff. This can further exacerbate the symptoms of emphysema and lead to additional complications.

In conclusion, emphysema is a chronic and progressive respiratory disease with a complex of etiological and pathogenetic factors. While cigarette smoking is the primary cause of the disease, genetic factors also play a role in its development. Understanding the mechanisms underlying the development of emphysema is essential for the development of effective treatments and prevention strategies.