When the lungs are exposed to harmful substances like tobacco an inflammatory response occurs. The lungs experience a chronic inflammatory response if the exposure is repeated which results in lung parenchymal damage (emphysematous changes) and fibrosis that traps air and gradually reduces airflow. The airways lung parenchyma and pulmonary vasculature are the primary sites of these inflammatory alterations with tissue damage and fibrosis which typically worsen with increased exposures. Smokers who develop COPD have been shown to exhibit particular inflammatory patterns marked by an increase in CD8+ and Tcl lymphocytes. Together with neutrophils and macrophages these cells release enzymes and inflammatory mediators that interact with lung parenchyma pulmonary vasculature and airway structural cells. Second in patients with COPD there is an imbalance between ant proteases that prevent connective tissue breakage and proteases that degrade connective tissue components. Patients with emphysema frequently exhibit protease–mediated destruction of elastin a significant component of connective tissue in the lung parenchyma. Reduced FEV1 is caused by peripheral airway constriction and inflammation. Airflow restriction is also a result of parenchymal destruction brought on by emphysema. The degree of inflammation fibrosis and luminal exudates in small airways is directly correlated with FEV1 and the FEV1/FVC ratio. Hyperinflation is caused by this peripheral airway obstruction which gradually traps air during expiration. Increased dyspnea and limited exercise capacity result from hyperinflations reduction of inspiratory capacity especially during exercise. Pulmonary hypertension results from hypoxic vasoconstriction of the small pulmonary arteries which also causes intimal hyperplasia and smooth muscle hypertrophy. Right ventricular hypertrophy and ultimately .right–side heart failure are caused by progressive pulmonary hypertension

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