the alveoli, their perforation and atrophy; it is more marked at the apex, and along the margins of the lung. If it follows a violent and persistent cough, mechanical obstruction appears to be the method of production. If, as sometimes occurs, it comes on insidiously without cough, the changes are due to some constitutional dyscrasia. Senile changes affect the lung tissue, as well as other parts of the body. In lobar emphysema, fatty degeneration of the walls surrounding the air sacs, has been affirmed; fibrosis of lung tissue from prolonged bronchial congestion, is another explanation. From the fact that there is an hereditary tendency to the transmission of emphysema, as in gout, forces the opinion that there is an underlying blood dyscrasia in most of these cases not explainable by mechanical obstacle.

The general nature of the affection is shown by its insidious progress, its hereditary character, implication of both lungs in their entire extent, and the effects of remedial measures, which are likewise found useful in degenerative changes of other organs.

Two theories of the formation of Emphysema are:—1. That forced inspiration dilates and breaks through the air cells; 2. That forced expiration is the efficient factor. The first view was based upon the supposition that the air cells contained retained air—which forced inspiration compressed—a mucous plug obstructing its outflow. The expiratory effort being the stronger would force air past any obstruction which allowed air to pass on inspiration, and the shape of the bronchial tube would permit an obstructing plug to move more easily in the expiratory direction than the inspiratory. The result would be atelectasis, not emphysema. Traube's experiments support this explanation. The expiratory theory of emphysema is further borne out by the fact that in forced expiration after cough the supra-clavicular spaces become bulged out and hyper-resonant; the margins of the base and the anterior borders of the lungs being outside of the direct axis of pressure, and containing less air than other portions of the lungs are apt to become emphysematous. In forced expiration the diaphragm and thoracic parietes contract with force to press the air toward the apices and anterior borders—the cartilaginous portions of the ribs yield to the pressure, allowing expansion—as in a case of absence of the sternum, on forced expiration, the anterior borders of the lungs become prominent through the opening present. Thus it is seen that the causes, pathology, and mechanism of lobular and lobar emphysema differ much. A prominent symptom of emphysema is a constant sense of oppression, or smothering, which is most painful to witness, and may be aggravated by bronchial, gastric, or emotional causes. Such patients are usually more tranquil, and suffer less in summer. Such patients cannot sleep, and are troubled with real asthmatic attacks, with cyanosis, lowering of temperature, icy extremities. Expectoration abundant. After intense and painful cough during the day, the expectoration is small in quantity, though the cough be severe. The sputa may be tinged with blood, owing to rupture of some of the altered capillary vessels. Pain is sometimes present at the epigastric region, and may be increased by pressure. The right side of the heart may be enlarged and the liver congested. It is only in very advanced cases that we find evidences of valvular incompetency of the heart.

The physical signs are numerous. The shape of the chest, if the affection be bilateral, is larger; the ribs more prominent, the intercostal muscles sunken, the movements peculiar—moving up and down instead of expanding—the back is curved antero-posteriorly, the shoulders carried forward, the abdomen prominent, and its movements exaggerated. The inspirations are short and quick; expirations, long-drawn and jerky.