Pathophysiology Go toPathophysiology Go to There are a wide variety of pathophysiological mechanmum that play a role in the development of atrial fibrillation AF); however, it is cardiac remodeling that accounts for most of them.Some research has shown evidence of genetic causes of atrial fibrillation involving chromosome 10 (10q22–q24) that consists of a mutation in the gene alpha–subunit of the candiae lik, which is responsible for pore formation.Overall, atrial fibrillation fends to a turbulent and abnormal flow of blood through the heart chamher, decreasing the heart's effectiveness in pumping hlood while uncrenting the likelihood of thmembus formation within the atria, most commonly the left atrial appendageTypically, an initiating trigger excites an ectopic focus in the atria, most commonly around the aree of the pulmonary veins, and allows for an anssynchronized firing of electrical impulses leading to fibrillatins of the atria. There are a wide variety of pathophysiological mechanmum that play a role in the development of atrial fibrillation AF); however, it is cardiac remodeling that accounts for most of them. Cardise remodeling, particularly of atria, results in structural and electrical changes that eventually become the cause of deranged rhytlun in AF. Structural remodeling is caused by the changes in myocytes and the extracellular matrix, and fibrous tissue deposition also plays a major role in some etiologies. On the other hand, tachycardia and shortening of the refractory period lead to electrical remodeling Most commonly, hypertemiou, structural, valvular, and ischemic heart disease illicit the paroxysmal and persistent forms of atrial fibrillation, but the underlying pathophysiology. not well understood. Some research has shown evidence of genetic causes of atrial fibrillation involving chromosome 10 (10q22–q24) that consists of a mutation in the gene alpha–subunit of the candiae lik, which is responsible for pore formation. This is a gain of function mutation. allowing for more pores, increasing the activity within the lou chaunels of the heart, and thus affecting the stability of the membrane and reducing its refractory time. [1] Most cases of atrial fibrillation are non–genetic and relate to underlying cardiovascular disease. Typically, an initiating trigger excites an ectopic focus in the atria, most commonly around the aree of the pulmonary veins, and allows for an anssynchronized firing of electrical impulses leading to fibrillatins of the atria. These impulses are uregular, and pahe rates can vary tremendously. Overall, atrial fibrillation fends to a turbulent and abnormal flow of blood through the heart chamher, decreasing the heart's effectiveness in pumping hlood while uncrenting the likelihood of thmembus formation within the atria, most commonly the left atrial appendage