

Epidemiology and Mechanism of Atrial Fibrillation

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Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia. AF is associated with increased mortality and morbidity, such as stroke and congestive heart failure. Prevalence of AF increases with aging. In the United States, it is expected that AF prevalence will be double by the year 2050. A global burden of disease 2010 study, showed the prevalence rates were 0.5962% in men and 0.3731% in women. The prevalence rates were increased moderately between 1990 and 2010 in both sexes. The low prevalence rates (2010) were estimated in the Asia-Pacific region for both men and women (0.3402% and 0.196%, respectively). The highest rates were estimated in North America (0.9257% for men and 0.5208% for women). AF incidence rates were lowest in Asia-Pacific region for both men and women (0.038% and 0.0198%, respectively). The highest rates were estimated in North America (0.2645% for men and 0.1963% for women). The age-adjusted mortality rate for AF were increased 2-fold and 1.9-fold for men and women between 1990 and 2010. The AF mortality was higher in women overall.

External stress such as hypertension, diabetes, AF itself induce a slow and steady progressive structural remodeling of the left atrium. Fibrosis is the hallmark of this process. Structural remodeling induces reentry arrhythmia. AF induced important ionic changes which are downregulation of the Ca^{2+} inward current and upregulation of inward rectifier K^+ currents, these are associated with decreased action potential duration and atrial refractory period. Acetylcholine activation potassium current ($I_{K_{ACh}}$) is constitutively active in chronic atrial fibrillation, it has major role in persistence and perpetuation of AF. Alteration of calcium handling processes due to hyperphosphorylation of Ca^{2+} handling proteins were developed during AF, these are associated with increased spontaneous Ca^{2+} release and triggered activity, and it causes ectopic beats and AF promotion. Pulmonary vein (PV) is focal source of AF, it is due to triggered activity and localized reentry within the PVs.

Hypercoagulability is another important major pathology associated with AF, it is due to hypocontractility, which may be due to reduced endothelial shear stress induced increases PAI-1 expression, and enhances inflammatory process and expression of endothelial adhesion molecules, resulting in tissue factor exposure to the blood stream, these changes are associated with increased thrombogenesis of AF.