Atrial fibrillation (AF) is the most common arrhythmia in the clinical practice. It can occur at any age; however, it becomes extremely common in the elderly, with a prevalence approaching more than 20% in patients older than 85-years. AF is associated with a wide range of cardiac and extra-cardiac complications and thereby significantly contributes to morbidity and mortality. Present therapeutic approaches to AF have major limitations, which have inspired substantial efforts to improve our understanding of the mechanisms underlying AF, with the premise that improved knowledge will lead to innovative and improved therapeutic approaches.

Our understanding of AF pathophysiology has advanced significantly over the past 10 to 15 years through an increased awareness of the role of “atrial remodeling”. Any persistent change in atrial structure or function constitutes atrial remodeling. Both rapid ectopic firing and reentry can maintain AF. Atrial remodeling has the potential to increase the likelihood of ectopic or reentrant activity through a multitude of potential mechanisms.

The present lecture reviews the main novel results on atrial tachycardia-induced electrical, structural and contractile remodeling focusing on the underlying pathophysiological and molecular basis of their occurrence. Special attention is paid to novel strategies and targets with therapeutic significance for atrial fibrillation.

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