

Update on mechanisms and management of atrial fibrillation

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Atrial fibrillation (AF) is a major global health concern impacting millions and causing symptoms like palpitations, dyspnea, fatigue, dizziness, and chest discomfort. Furthermore, these symptoms reduce

patients' quality of life and lead to increased mortality and morbidity.

The [medical community](#) agrees that epicardial adipose tissue (EAT), [chronic inflammation](#), imbalances in the autonomic nervous system (ANS), stretch-induced fibrosis, and genetic alterations are the main factors that influence AF pathogenesis. Despite extensive research efforts focused on uncovering the underlying mechanisms of AF, there remains an incomplete understanding. Consequently, the absence of treatments tailored to address the root cause of AF persists.

Recently, Zhicheng Hu, Ligang Ding, and Yan Yao from Fuwai Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, conducted an in-depth survey of the latest literature on AF, resulting in a comprehensive summary of their insights.

[Their findings were published in the *Chinese Medical Journal*.](#)

"This review synthesizes existing knowledge and ongoing research on AF pathophysiology and clinical management, with a specific focus on [diagnostic devices](#), next-generation anticoagulants, medications targeting underlying mechanisms, and interventional therapies. It provides a comprehensive perspective on AF, shedding light on its intricate nature," explains Dr. Hu, the article's first author.

First, the authors delve into AF's pathogenic mechanisms, highlighting the impact of stretch-induced fibrosis triggered by pressure or volume overload, leading to profound remodeling of atrial tissue. This remodeling leads to lower conductivity, calcium overload, fibrosis, fibroblast proliferation, and modified collagen degradation. The fibrosis disrupts electrical conduction in atrial tissue and creates an environment conducive to the onset of AF.

EAT aggregating around atrial muscle promotes AF. The EAT induces

fat infiltration and fibrosis that cause dysfunction in atrial muscle. The damage caused by sustained inflammation mainly results from reactive oxygen species (ROS). These ROS impair the energetic and electrophysiologic properties of myocardial tissue.

The sympathetic and parasympathetic components of the ANS can also exacerbate the onset of and sustain AF. Genetic AF traits include common and rare variants spanning more than 160 genes and underscores the complexity of these genetic alterations.

Next, the study focuses on [clinical management](#) trends for AF. While electrocardiography has traditionally traced AF's irregular rhythms for diagnosis, recent advancements in wearables present an enticing avenue for continuous, real-time monitoring outside clinical settings, eliminating the necessity for clinic visits. These devices are designed to be user-friendly and seamlessly share data with health care providers.

While hurdles to the practicality of their widespread use and accuracy of their measurements remain, the pace of development of these platforms also remains rapid.

The 4S-AF scheme (Stroke risk, Symptom severity, Severity of AF burden, and Substrate severity) is the most comprehensive approach to assessing AF. A patient's risk of stroke is determined by the CHA2DS2-VASc score. This index provides a score based on points for congestive heart failure, hypertension, age over 74 years, diabetes mellitus, stroke history, vascular disease, age between 65-74 years, and female sex.

Patients are then categorized into low, moderate, and high-risk stroke groups. Symptom severity is determined from validated questionnaires like the European Heart Rhythm Association score and the AF-Specific Health-related Quality of Life questionnaire.

Professionals gauge AF burden by tracking episode duration, frequency, and patterns. Diagnostic tools, including echocardiography, electrophysiological studies, MRI, and biomarkers, assess atrial structure and function, aiding in evaluating substrate severity.

The integrated management of AF involves treatment to improve clinical symptoms and reduce the risk of stroke and associated risk factors. Stroke prevention necessitates balancing the risk of bleeding with the risk of stroke and accordingly determining an appropriate oral anticoagulant therapy.

The HAS-BLED—the combined presentation of hypertension, abnormal renal/liver function, stroke history, bleeding predisposition/history, labile international normalized ratio, elderly, and drugs/alcohol use—is used to predict patients' bleeding risk.

It is advised that anticoagulants are administered following consideration of the CHA₂DS₂-VASc score. Once a decision to administer anticoagulants is reached, physicians can choose between warfarin and non-vitamin K antagonist oral anticoagulants.

Further, AF's main symptom, heart palpitations, can be managed by rate or rhythm control interventions. Anti-arrhythmic drugs and catheter ablation are the main routes to rhythm management. Pulmonary vein isolation is the preferred [catheter ablation](#) technique, but pulse field ablation is emerging as an ablation source with a minimal impact on non-target tissues. Patients with chronic AF usually receive medication regimens to achieve ventricular rate control over the long term.

The main risk factors and comorbidities that need to be managed to counter AF progression are cardiovascular risk factors—alcohol consumption, obesity, hypertension, diabetes, low exercise—and unhealthy lifestyles.

While AF poses considerable health care challenges due to its complex nature, ongoing research will help further understand AF and improve patient outcomes.

"The integration of wearable devices, next-generation anticoagulants, drugs targeting underlying mechanisms, and innovative interventional therapies holds the potential to transform AF management," concludes Dr. Yao.

More information: Zhicheng Hu et al, Atrial fibrillation: mechanism and clinical management, *Chinese Medical Journal* (2023). [DOI: 10.1097/CM9.0000000000002906](https://doi.org/10.1097/CM9.0000000000002906)

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