MEDS Chinese Medicine (2025)
Clausius Scientific Press, Canada

DOI: 10.23977/medcm.2025.070220
ISSN 2616-1753 Vol. 7 Num. 2

Current Status and Research Progress of Integrated Traditional Chinese and Western Medicine in the Diagnosis and Treatment of Atrial Fibrillation

Na Li[1], Jinli Liu[2,*], Mei Yang[2]

[1]Chengdu University of Traditional Chinese Medicine, Chengdu, Sichuan, 610032, China
[2]Neijiang Hospital of Traditional Chinese Medicine Affiliated to Chengdu University of Traditional Chinese Medicine, Neijiang, Sichuan, 641099, China
**Corresponding author*

Keywords: Atrial Fibrillation, epidemiology, pathogenesis, Western medicine treatment, Traditional Chinese medicine treatment

Abstract: Atrial Fibrillation (AF) is a common arrhythmia in clinical practice, which seriously affects the quality of life of patients and significantly increases the risks of death, stroke, heart failure and dementia. Over the past two decades, a series of breakthroughs have been made in areas such as risk prediction and screening diagnosis of AF, stroke prevention, rhythm control, catheter ablation and comprehensive management. To further promote the standardized management of AF and promptly and fully apply new technologies and concepts to clinical practice, Chinese Society of Cardiology (CSC) and Chinese Heart Rhythm Society (CHRS) organized experts to jointly formulate the "Chinese Guidelines for the Diagnosis and Treatment of Atrial Fibrillation", which elaborated on all aspects of AF management and provided a reference for standardized clinical diagnosis and treatment. However, the author believes that its treatment is confronted with problems such as side effects of Western medicine, high recurrence rate of surgery, and complex anticoagulation management, while traditional Chinese medicine has shown unique advantages through holistic dialectics and multi-target regulation. Integrated treatment of traditional Chinese and Western medicine can achieve synergistic effects, but currently there is a lack of systematic review and integration mechanism research and clinical evidence. This paper focuses on investigating the theoretical basis, mechanisms of action, and clinical applications of integrated Chinese-Western medicine in AF management, aiming to establish a foundation for therapeutic optimization.

1. Introduction

Atrial Fibrillation (abbreviated as AF) is the most common arrhythmia in clinical practice, with a high disability rate and high mortality rate. The irregular fibrillation of the atria causes the heart to lose its effective contraction and relaxation, and the heart's pumping function deteriorates or even disappears accordingly. Ischemic stroke is one of the most dangerous complications in AF, with the risk of thromboembolism increasing fivefold compared to non-atrial fibrillation patients [1]. The population in our country is gradually aging, and the number of patients with senile atrial fibrillation

is also increasing, resulting in a corresponding rise in the prevalence of ischemic stroke[2]. Therefore, it is very necessary to fully understand the pathogenesis of AF. In recent years, the understanding of the pathogenesis of AF has been continuously improved, promoting the development of the combined treatment of traditional Chinese medicine and Western medicine for AF. The understanding of the pathogenesis and management of AF in both traditional Chinese medicine and Western medicine is summarized as follows.

2. The definition and epidemiology of AF

2.1. Definition

Atrial Fibrillation, abbreviated as AF, refers to the loss of regular and orderly electrical activity in the atrium, replaced by rapid and disordered fibrillation waves. It is one of the most common arrhythmias. AF can be diagnosed when a single-lead electrocardiogram(ECG) (≥ 30 s) or a 12-lead ECG (≥ 10 s) shows the disappearance of P waves, replaced by irregular fibrillation waves (f waves) in size, shape and duration, and an absolutely irregular ventricular rate [3].

Classification: "Chinese Guidelines for the Diagnosis and Treatment of Atrial Fibrillation (2023)" classifies AF into paroxysmal atrial fibrillation, persistent atrial fibrillation, Long-standing persistent atrial fibrillation, and permanent atrial fibrillation based on the duration of atrial fibrillation attacks, the difficulty of converting and maintaining sinus rhythm for a long time, and the choice of treatment strategies. (Table 1)

Table 1: Classification of AF (Reference: 2023 Chinese Guidelines for the Diagnosis and Treatment of Atrial Fibrillation)

Clinical classification	Definition
paroxysmal atrial fibrillation	AF lasting less than 7 days (including spontaneous termination of atrial fibrillation or termination of intervention)
persistent atrial fibrillation	AF lasts 7 days or more
Long-standing persistent atrial fibrillation	AF lasts more than 1 year
permanent atrial fibrillation	It is unlikely that sinus rhythm will be restored and maintained, AF will persist for more than 10~20 years, and the ECG will show very small F waves that are nearly straight; or cardiac magnetic resonance imaging showing that the left atrial fibrosis area accounts for more than 30% of the left atrial area.

2.2. Epidemiology

According to statistics, in 2023, there will be about 330 million cardiovascular diseases in China, of which about 4.87 million will be patients with AF, and the incidence rate is increasing day by day [4]. According to the Global Burden of Disease(GBD)research data [5,6], from 1990~2016, the Disability-Adjusted Life Year(DALY) of CVD increased by 33.7%, of which the increase was 51.8% for men, much higher than that for women (12.1%). The diseases with the fastest growth in disease burden were AF and atrial flutter (147.0%), Ischemic Heart Disease(IHD) (122.0%), Peripheral Arterial Disease(PAD) (108.9%), ischemic stroke (80.4%) and aortic aneurysm (49.1%). With a global prevalence of 50 million in 2020, although the prevalence of undiagnosed AF in the community is unknown, using a reverse calculation method, researchers estimated that approximately 11% (591, 000) of the 5.6 million cases of AF in the United States were undiagnosed in 2015 [7]. AF

is associated with higher health care utilization and costs, and using US data from Optum (the U.S. database of administrative claims for patients with commercial insurance [United Healthcare]), patients with episodic AF have an increased risk of hospitalization and an increased risk of cardiovascular-related emergency department visits compared to patients without AF (relative risk [RR], 2.41 [95% CI, 2.35-2.47]) AF is expensive, check the data for Optum, The annual medical cost for patients with AF is $63, 031, which is $27, 896 more than people without AF. Investigators reviewed data from public and private health insurers and estimated that AF accounted for $28.4 billion (95%CI, $24.6 billion to $33.8 billion) in healthcare spending in 2016 [8].

3. Etiology and pathogenesis of AF

3.1. Etiology

AF often occurs in patients with organic heart disease, and is more common in hypertensive heart disease, coronary atherosclerotic heart disease(CAD), rheumatoid heart disease mitral stenosis, cardiomyopathy and hyperthyroidism. Secondly, constrictive pericarditis, chronic pulmonary heart disease, pre-stimulation syndrome and aging can also cause AF. Some cases of AF have unknown causes, it can be observed in normal individuals and may occur during emotional excitement, surgical procedures, physical exercise, or excessive alcohol consumption; AF occurring in young and middle-aged individuals without structural heart disease is referred to as lone AF or idiopathic AF.

3.1.1. Heart disease

Coronary heart disease: Coronary atherosclerosis leads to myocardial ischemia and hypoxia, with surviving cardiomyocytes in the peri-infarct zone exhibiting unstable electrophysiological states that predispose them to form ectopic pacemakers, thereby disrupting the normal cardiac electrical activity rhythm.

Cardiomyopathy: The heart enlarges in patients with dilated cardiomyopathy, hypertrophy, degeneration and fibrosis of cardiac cells, which changes the electrical conduction path in the atria, slows down and is uneven. Hypertrophic cardiomyopathy Myocardial hypertrophy causes disordered atrial myocytes to arrange, reduce atrial compliance, and prone to increased pressure in the atrial and abnormal electrical activity.

Heart valve disease: such as mitral valve stenosis, mitral valve regurgitation, aortic valve regurgitation, etc., which lead to changes in atrial pressure and volume load, enlargement of the atrial muscle fibers, stretching of the atrial muscle cells, shortening of the refractory period, increasing excitability, and increasing the chance of atrial fibrillation.

3.1.2. Non-cardiac disease

Thyroid disease: excessive secretion of thyroid hormone during hyperthyroidism has a direct toxic effect on the heart, increases the excitability and automaticity of myocardial cells, shortens the action potential duration of atrial myocytes, increases the metabolic rate of the heart, increases the oxygen consumption of the myocardium, and the heart is in a state of high dynamic circulation.

Lung disease: Chronic obstructive pulmonary disease, cor pulmonale, and other conditions can lead to pulmonary hypertension, increased right heart pressure load, impaired right atrial function, and altered electrophysiological properties of atrial myocardial cells.

3.1.3. Other factors

Age: As age increases, organs such as the heart and blood vessels gradually age, cardiac function

declines, and the incidence of AF rises, with a higher prevalence among individuals over 80 years old.

Unhealthy lifestyle habits: Long-term excessive alcohol consumption, smoking, excessive caffeine intake, and high mental stress can increase the risk of AF.

3.2. Pathogenesis

AF is a significant disease that endangers the health of middle-aged and elderly populations in China and is associated with risks of stroke and mortality. Currently, the occurrence and progression of AF can be explained by pathophysiological mechanisms such as atrial structural remodeling, electrical remodeling, inflammation, autonomic nervous system dysfunction, and non-coding RNAs [9].

3.2.1. Atrial remodeling

Atrial structural remodeling and electrical remodeling serve as crucial pathophysiological foundations for the occurrence of AF.

Structural remodeling: Atrial fibrosis is a key feature, with abnormal proliferation and transformation of fibroblasts into myofibroblasts, irregular precipitation of the extracellular matrix, which disrupts local electrical activity and promotes reentrant circuits and regional conduction disturbances.

Electrical remodeling: Alterations in ion channels lead to a shortened refractory period, while rapid atrial pacing increases Ca^{2+} influx, thereby inducing calcium overload and promoting inactivation of L-type calcium channels as well as downregulation of calcium currents. Concurrently, calcium-dependent K^+ channels and Cl^- channels facilitate the efflux of K^+ and Cl^-, which results in a reduction of the atrial effective refractory period and action potential duration, ultimately generating reentrant waves [10]. The renin-angiotensin-aldosterone system (RAAS) is involved in atrial structural and electrical remodeling. Angiotensin II (Ang II) is a known profibrotic factor, and its profibrotic process is mediated through the TGF-β1/Smads signaling pathway [11]. Moreover, Ang II can increase intracellular Ca^{2+} concentration by regulating L-type Ca^{2+} channels, leading to Ca^{2+} overload, thereby shortening the plateau phase and inducing rapid atrial excitation, which results in atrial electrical remodeling [12]. This may explain why sacubitril-valsartan can effectively reverse atrial remodeling in AF and be used to delay its progression and prevent recurrence.

3.2.2. Inflammatory response

Inflammation and atrial fibrillation are mutually causative. Inflammatory responses can lead to degeneration and fibrosis of atrial myocytes, slow atrial conduction velocity, and participate in atrial structural and electrical remodeling. Multiple inflammatory factors are significantly elevated in the serum of AF patients. For instance, TNF-αcan promote collagen synthesis via the TGF-β/Smad pathway, leading to atrial fibrosis. It can also alter the Ca^{2+} handling capacity of cardiomyocytes and downregulate Cx40, thereby contributing to atrial electrical remodeling [13]. Existing research has demonstrated that certain anti-inflammatory medications, including nonsteroidal anti-inflammatory drugs (NSAIDs), glucocorticoids, colchicine, and statins, can reduce the recurrence rate of atrial fibrillation [14]. By further exploring the role of inflammatory factors in the pathogenesis of atrial fibrillation, more options may become available for the pharmacological management of patients with this condition in the future.

3.2.3. Imbalance of the autonomic nervous system(ANS)

On one hand, after sympathetic nerve activation, the postganglionic nerve terminals release norepinephrine, which acts on adrenergic receptors to increase myocardial cell automaticity and contractility, accelerate conduction, and trigger abnormally rapid discharges from AF foci. On the other hand, excessive sympathetic excitation leads to increased intracellular Ca^{2+} influx, resulting in calcium overload within cells, shortening the atrial effective refractory period, and inducing AF. The vagus nerve releases acetylcholine that binds to cholinergic receptors, activating IKach and increasing outward K^+ current, thereby shortening the action potential duration, slowing intra-atrial conduction velocity, increasing dispersion of atrial effective refractory periods, and facilitating reentry to trigger AF [15,16].

3.2.4. Impact of non-coding RNAs

Long non-coding RNAs (lncRNAs) and microRNAs (miRNAs) have garnered particular attention. lncRNAs can play a role in cardiac electrical and structural remodeling by regulating key signaling pathways, thereby participating in the occurrence and progression of atrial fibrillation. miRNAs are involved in the post-transcriptional regulation of gene expression and are associated with the development of various cardiovascular diseases. Current research indicates that multiple miRNAs such as miR-146b-5p [17] and miR-29b [18] participate in atrial fibrosis, while miR-145 [19] and miR-21 [20] alter atrial ion channels, contributing to cardiac electrical remodeling. Non-coding RNAs have emerged as molecular biomarkers for the clinical diagnosis of AF.

3.2.5. Other factors

Obesity has become an independent risk factor for AF. A meta-analysis showed that for every 5-unit increase in BMI, the risk of AF increases by 10%–29% [21]. For overweight and obese AF patients, relevant guidelines recommend weight loss to reduce the frequency and duration of AF episodes [22]. Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a common chronic sleep disorder in the population, which may promote the development of AF through various potential mechanisms, including intermittent hypoxia, recurrent arousal, and increased negative intrathoracic pressure. In addition to pharmacological treatment, continuous positive airway pressure (CPAP), uvulopalatopharyngoplasty, and hypoglossal nerve stimulation may become new therapeutic strategies for OSAHS complicated with AF [23]. Furthermore, alcohol consumption, hypertension, and diabetes are all risk factors for AF [24]. Therefore, lifestyle and dietary modifications, including weight loss, alcohol abstinence, and blood glucose and blood pressure control, will be the cornerstone of AF prevention.

4. Diagnosis of AF

According to current medical practice, the diagnosis of AF should primarily refer to the "2023 ACC/AHA/ACCP/HRS Guideline for the Diagnosis and Management of Atrial Fibrillation, " which was jointly issued by the American College of Cardiology (ACC), the American Heart Association (AHA), the American College of Chest Physicians (ACCP), and the Heart Rhythm Society (HRS). This guideline is internationally recognized as the authoritative standard.

4.1. Clinical presentation

4.1.1. Symptom

AF is the most clinically concerned reentrant arrhythmia, which is characterized by irregular contraction of atrial myocytes, and usually abnormal rapid contraction, which can lead to various clinical symptoms such as palpitations, dizziness, chest tightness, fatigue, and amaurosis fugax, or no clinical symptoms. Serious complications caused by AF, such as stroke and heart failure, seriously affect the quality of life of patients. The most common symptoms of AF are palpitations, decreased activity tolerance, and chest discomfort, and some patients may also have symptoms such as dizziness, anxiety, and increased urine output (due to increased atrial natriuretic peptide secretion). The severity of AF symptoms varies widely between individuals, with some patients gradually tolerating symptoms due to nonspecific or mild symptoms, with about one-quarter of patients reporting no symptoms. Thromboembolism or heart failure and other complications may also be the initial manifestations of AF.

4.1.2. Signs

The main signs of AF include an absolutely irregular heart rhythm, unequal first heart sound, and a short pulse.

4.2. Laboratory examination

For initial diagnosis of AF patients, routine blood tests, electrolyte levels, liver and kidney function tests, coagulation function tests, thyroid function tests, B-type natriuretic peptide (BNP) or N-terminal pro-brain natriuretic peptide (NT-proBNP) tests, as well as relevant laboratory examinations for comorbid conditions should be performed.

Surface ECG: The typical ECG manifestations of AF include: (1) Absence of P waves, replaced by irregular fibrillatory waves (f waves) with a frequency of 350–600 beats/min; (2) Absolutely irregular R-R intervals. When interpreting an ECG of a patient with AF, attention should also be paid to signs of myocardial ischemia, myocardial hypertrophy, pre-excitation syndrome, electrolyte imbalances, pulmonary embolism, etc. Additionally, indicators such as heart rate, QRS duration, and QT interval should be assessed.

Holter monitoring and other long-term ECG monitoring methods are helpful in diagnosing asymptomatic AF, assessing AF burden, and evaluating ventricular rate during AF episodes.

5. Treatment of AF

5.1. Western medicine treatment

The management of AF primarily includes anticoagulation, rhythm control, and rate control. Rhythm control and rate control remain the cornerstone of AF treatment, with the restoration and maintenance of sinus rhythm being a key long-term goal for improving patient survival [9]. Additionally, emerging therapies such as anti-inflammatory approaches, autonomic nervous system modulation, and genomic interventions may become novel treatment strategies for AF in the future.

5.1.1. Anticoagulation

Although AF is a disorder in the electrical activity of the heart, it can cause abnormalities in the mechanical activity of the atria. This impaired mechanical activity causes blood stasis within the atrial

chambers. The left atrial appendage (LAA), due to its unique anatomical structure, becomes a particularly vulnerable site for thrombus formation. Once formed, LAA thrombi carry a persistent risk of embolization. Detached thrombi travel through the circulatory pathway: left atrium → left ventricle → aorta → systemic arterial circulation. The cerebral arteries are the most frequent destination for these emboli. When a cerebral artery becomes occluded by such an embolus, it results in cerebral infarction - clinically manifested as stroke ("brain attack"). These pathophysiological mechanisms constitute the rationale for anticoagulant therapy in AF patients. The purpose of anticoagulation is to prevent intracardiac thrombus formation, with the ultimate goal of avoiding stroke occurrence. According to epidemiological data cited in the latest clinical guidelines (ESC, AHA/ACC), compared to the non-AF population, AF patients not receiving anticoagulation therapy face a 3-5 times higher risk of: stroke, transient ischemic attack (TIA), systemic embolism (thromboembolism affecting other organs). Furthermore, AF-related strokes typically demonstrate more severe clinical manifestations than non-AF strokes, characterized by: higher disability rates, greater mortality, increased recurrence frequency.

Therefore, for patients with AF, anticoagulation therapy to prevent stroke is a critical component of treatment. Clinically, anticoagulant drugs primarily include: Warfarin (a vitamin K antagonist), Direct oral anticoagulants (DOACs) such as rivaroxaban, dabigatran, apixaban, and edoxaban.

The decision to anticoagulate does not primarily depend on the type of AF (paroxysmal vs. persistent) but rather on the patient's stroke risk. The latest clinical guidelines endorse a simplified scoring system tailored for Asian populations: the CHA_2DS_2-VASc-60 score. It is a novel risk stratification tool introduced in the 2023 Chinese Guidelines for the Diagnosis and Treatment of Atrial Fibrillation. This adaptation was developed in response to epidemiological evidence demonstrating that Asian AF patients face elevated stroke risks at a younger age threshold compared to Western populations. The detailed scoring criteria are as follows, as shown in Table 2.

Table 2: CHA2DS2-VASc-60 score (Reference: 2023 Chinese Guidelines for the Diagnosis and Treatment of Atrial Fibrillation)

	Risk factors	Clinical Specification	Points
C	Congestive heart failure	Including HFrEF, HFmrEF, HFpEF, and left ventricular systolic dysfunction(LVSD) (LVEF <40%)	1
H	Hypertension	≥140/90 mmHg or treated	1
A2	Age ≥65 years	Asian AF patients aged ≥65 years	2
D	Diabetes mellitus	Including type 1 and type 2 diabetes, the longer the disease duration, the higher the risk of stroke.	1
S2	Stroke	Prior stroke(Including ischemic and hemorrhagic stroke), systemic embolism, or TIA	2
V	Vascular disease	CAD, Prior myocardial infarction, (Peripheral Artery Disease)PAD, or aortic plaque (imaging-confirmed)	1
A	Age 60~64 years	Asian AF patients aged 60-64 years	1
Sc	Gender (female)	Modifiable factors for stroke risk, but not independent risk factors	1

As a general rule, if a male AF patient scores ≥2 points using the aforementioned assessment tool, anticoagulant therapy should be initiated. Similarly, if a female patient scores ≥3 points, anticoagulation treatment is also required. The CHA_2DS_2-VASc-60 scoring system reveals that certain seemingly healthy individuals actually face elevated stroke risks. For example: All patients aged ≥65 years: Males automatically score ≥2 points (meeting the threshold for anticoagulation). Females score ≥3 points (similarly requiring anticoagulant therapy). Thus, even with paroxysmal AF, most patients over 65 will likely need anticoagulants. Additionally, Patients aged 60–

64years witheither hypertension or diabetes: Males score ≥2 points, females ≥3 points, Anticoagulation is generally recommended for this group as well. AF prevalence increases with age, making stroke prevention through anticoagulation a critical treatment component, in conclusion，Even infrequent AF episodes warrant anticoagulants if the CHA_2DS_2-VASc-60 score indicates high stroke risk.

Bleeding risk assessment is equally important as anticoagulant therapy. Guidelines recommend that bleeding risk should be regularly assessed both at the initiation of anticoagulant therapy and during its course, to allow for timely detection and intervention of modifiable bleeding risk factors. It is suggested to use the HAS-BLED scoring system to evaluate bleeding risk, where a score ≥3 indicates high bleeding risk. However, a high bleeding risk score should not be considered as a contraindication for the use of OAC.

5.1.2. Rhythm control

Contemporary strategies for rhythm control in AF predominantly encompass antiarrhythmic drugs（AADS）and catheter ablation procedures. Early rhythm control in AF can effectively halt disease progression, reduce adverse events, and significantly improve patients' quality of life. The Early Treatment of AF for Stroke Prevention Trial (EAST-AFNET 4), a multicenter randomized open-label study, demonstrated that early rhythm control significantly reduced the risk of primary composite endpoints (stroke, cardiovascular death) compared to usual care. Earlier studies relied heavily on AADs such as amiodarone and sotalol (accounting for >⅔ of AAD use), which were associated with potential mortality risks and adverse effects, possibly offsetting the benefits of sinus rhythm maintenance. However, in the EAST-AFNET 4 trial [25], more advanced rhythm control methods were employed, with more rational use of AADs. The primary AADs used were flecainide, amiodarone, and dronedarone. Dronedarone, a novel class III antiarrhythmic drug with multi-channel blocking effects, shares a similar molecular structure with amiodarone while retaining its efficacy but demonstrating improved safety [26]. Additionally, approximately 20% of patients underwent catheter ablation as a rhythm control strategy. Safe and effective rhythm control remains the ideal treatment approach for atrial fibrillation. Growing evidence supports the active adoption of early rhythm control strategies for patients with newly diagnosed AF or AF complicated by heart failure. Current guidelines recommend early rhythm control for patients diagnosed within one year who have cardiovascular risk factors (including asymptomatic, persistent, or heart failure cases) to improve prognosis. AADs and catheter ablation are the primary methods for rhythm control. Substantial evidence has accumulated supporting catheter ablation for rhythm control. Compared with AADs, catheter ablation significantly reduces AF recurrence risk and decreases cardiovascular hospitalizations. As first-line therapy for paroxysmal AF, catheter ablation demonstrates clear superiority over AADs in reducing symptomatic AF recurrence and improving quality of life. However, reliable evidence regarding whether catheter ablation can improve long-term prognosis in AF patients remains lacking [27].

5.1.3. Rate control

The ventricular rate control strategies for AF include: Strict rate control (resting heart rate ≤80 bpm, heart rate <110 bpm during moderate exercise Lenient rate control(resting heart rate <110 bpm). For initial management, the target ventricular rate can be set at resting heart rate <110 bpm (Class IIa, Level B evidence). If symptoms persist, stricter rate control should be considered. AF complicated with heart failure represents a common clinical challenge. International guidelines show inconsistency in recommending optimal heart rate targets for these patients, with symptom relief being the primary treatment goal. Common medications for long-term rate control include: β-blockers,

Non-dihydropyridine calcium channel blockers (ND-CCBs), Digoxin and Partial AADs [28].

5.2. Traditional Chinese Medicine (TCM) treatment

Ancient medical texts did not document "AF" as a distinct condition. Modern practitioners of TCM generally classify AF within the categories of "palpitations", "fright palpitations", and "severe palpitations". The Chinese Association of Traditional Chinese Medicine's Diagnostics Committee has formally designated AF as "heart-throbbing palpitations" in TCM terminology. The earliest documentation of palpitations appears in "Huangdi Neijing", which provides descriptions of its clinical manifestations and pulse characteristics. The Treatise on Cold Damage Disorders: On Pulse Patterns and Treatment of Contraindicated Purgation records: "When moving qi manifests in the right [abdomen], purgation is forbidden. If purgation is erroneously applied, it will deplete internal body fluids, leading to dry throat and nostrils, dizziness, and palpitations"The Treatise on Cold Damage Disorders: On Pulse Patterns and Treatment of Taiyang Diseases states: "For patients experiencing bodily heaviness and palpitations, diaphoresis [induced sweating] is contraindicated. Resolution should occur through spontaneous perspiration. This is because a faint pulse at the chi position indicates internal deficiency; recovery requires internal reinforcement to restore fluid harmony. "In the General Treatise on the Causes and Symptoms of Diseases, Chao Yuanfang classified palpitations into two primary categories: deficiency and excess, proposing that palpitations result from "excess pathogenic factors overwhelming deficient healthy qi". Zhu Danxi explicitly identified "fright palpitations" as stemming from blood deficiency, stating: "The heart is nourished by blood. When heart-blood is deficient, the spirit loses its anchorage". The "Standards of Diagnosis and Treatment" identifies two primary causes of palpitations: Deficiency and Fluid Retention. Dual deficiency of qi and blood creates emptiness below the heart, allowing internal fire to stir, manifesting as palpitations. Water-dampness stagnating near the heart disrupts stability, triggering palpitations. Wang Qingren in Corrections of Medical Errors Proposed the groundbreaking "blood stasis-induced palpitations" theory. Zhang Xichun's Etiological Theory of Palpitations in "Records of Medicine with Chinese-Western Reference": he proposed that fright palpitations primarily arise from constitutional deficiency and phlegm-fluid retention [29].

Contemporary TCM Scholars' Development in AF Treatment Building upon the clinical wisdom of ancient masters in treating "palpitations", modern TCM practitioners have gradually developed distinctive academic approaches. Through ongoing research on: Classical formulas, Historical prescriptions, Empirical recipes and by integrating their own clinical experience, they have formulated personalized prescriptions that demonstrate significant efficacy in managing atrial fibrillation. Ye Haitao et al. conducted a therapeutic study where Zhigancao Decoction was administered alongside metoprolol to patients with persistent AF. The results demonstrated that: The combination therapy showed significant improvements in both clinical symptoms and laboratory parameters compared to metoprolol alone [30]. A prospective clinical trial conducted by Ji Meng et al. involving 40 patients with paroxysmal non-valvular AF demonstrated that: The modified Chaihu Plus Longgu Muli Decoction showed significant clinical efficacy in: Symptom relief, decreased frequency and duration of AF episodes, enhanced echocardiographic parameters [31]. Recent large-scale clinical studies have confirmed that Shensong Yangxin Capsule demonstrates good efficacy either alone or in combination with AADS for various types of AF and post-AF ablation. It also shows significant effectiveness in treating AF complicated with other diseases. Shensong Yangxin Capsule can improve cardiomyocyte metabolism to reduce apoptosis, regulate ion channels, ameliorate electrical remodeling, enhance sinoatrial node function, decrease myocardial fibrosis to inhibit cardiac remodeling, and modulate neural function to improve heart rate variability, demonstrating multi-component, multi-target, and multi-pathway effects [32]. A study by Li Min et al., which

included 24 randomized controlled trials involving 2, 246 AF patients, demonstrated that Wenxin Granule alone or in combination with Western medications shows good therapeutic efficacy in AF treatment [33]. Modern pharmacological and clinical studies demonstrate that various Chinese herbs and compound formulas exhibit anticoagulant and antithrombotic effects: active components in Danshen (Salvia miltiorrhiza) can increase warfarin plasma concentration, thereby enhancing anticoagulant efficacy when used in combination with warfarin [34].

6. Conclusion

AF is the most common clinical arrhythmia, which can cause symptoms such as palpitations and fatigue, though some patients may remain asymptomatic. The pathogenesis ofAF is complex and diverse. Western medicine primarily employs anticoagulation, ventricular rate control, cardioversion, ablation, and left atrial appendage closure for treatment, but these approaches often come with significant adverse drug reactions. In contrast, TCM adopts a holistic approach with syndrome differentiation and treatment. Based on the patient's complex clinical condition, TCM tailors classical prescriptions through individualized modifications, creating personalized treatment plans with fewer adverse reactions. Non-pharmacological TCM therapies are also well-accepted by patients, demonstrating minimal side effects, convenient application, and notable efficacy. This approach provides comprehensive safety assurance, effectively alleviates symptoms during AF episodes, reduces attack frequency, shortens episode duration, and leads to favorable patient outcomes, making it worthy of clinical promotion.

Currently, the following issues exist in research on AF:

(1) The standardization process of TCM syndrome differentiation for AF lags severely behind. Currently, the TCM community has not established a unified and standardized classification system for AF syndromes. Significant differences exist in the theories and clinical experiences applied by different practitioners during syndrome differentiation, resulting in diversified and fragmented syndrome classifications. This situation substantially hinders the consistency and comparability of clinical research outcomes in TCM. (2) In terms of clinical research methodology, there is a severe lack of high-quality randomized controlled trials for TCM interventions targeting AF, making it difficult to fully validate and gain recognition from modern evidence-based medical systems regarding the efficacy and safety of TCM in treating AF. (3) Currently, there exists a gap in integrated Chinese-Western medicine guidelines for AF. The lack of authoritative and systematic diagnostic and treatment guidelines for integrated therapy leaves clinicians without clear references when combining Chinese and Western medical approaches, making it difficult to achieve organic integration and synergistic effects between the two systems. Moreover, significant individual differences among AF patients in terms of constitution, underlying diseases, and disease progression pose considerable challenges for precise personalized treatment. Determining the optimal balance between TCM syndrome differentiation and Western disease diagnosis, and developing the most suitable integrated treatment plan based on each patient's specific condition, has become a critical issue requiring urgent resolution.

References

[1] Huang Congxin, Zhang Shu, Huang Dejia, et al. Atrial Fibrillation: Current Understanding and Recommendations for Treatment-2018 [J]. Chinese Journal of Cardiac Pacing and Electrophysiology, 2018, 32 (04): 315-368.

[2] Jiang Junrong, Xue Yumei. Anticoagulation in atrial fibrillation[J]. Chinese Journal of Practical Internal Medicine, 2023, 43(2): 106-109.

[3] Wang Yanpeng, Li Jingbo. Interpretation of the "Chinese Guidelines for the Diagnosis and Treatment of Atrial Fibrillation[J]. "CARDIO-CEREBROVASCULAR DISEASE PREVENTION AND TREATMENT, 2024, 24(11): 5-9.

[4] National Center for Cardiovascular Diseases, The Writing Committee of the Report on Cardiovascular Health and

Diseases in China. Report on Cardiovascular Health and Diseases in China 2023: an Updated Summary[J]. Chinese Circulation Journal, 2024, 39(7): 625-660.
[5] Li Yichong, Liu Shiwei, Zeng Xinying, Zhou Maigeng. Report on Burden of Cardiovascular Diseases From 1990 to 2016 in China[J]. Chinese Circulation Journal, 2019, 34(8): 729-740.
[6] Liu S, Li Y, Zeng X, et al. Burden of Cardiovascular Diseases in China, 1990-2016: Findings From the 2016 Global Burden of Disease Study[J]. JAMA Cardiol, 2019, 4(4): 342-352.
[7] Tsao C W, Aday A W, Almarzooq Z I, et al. Heart Disease and Stroke Statistics-2023 Update: A Report From the American Heart Association[J]. Circulation, 2023, 147(8): e93-e621.
[8] Joseph L. Dieleman, Jackie Cao, et al. US Health Care Spending by Payer and Health Condition, 1996-2016 [J]. JAMA, 2020, 323(9):863-884.
[9] Li Han, Cai Wei. Progress in pathogenesis and treatment of atrial fibrillation[J]. Journal of Practical Electrocardiology, 2024, 33(1): 87-92.
[10] Que Dongdong, Song Xudong. Roles of atrial remodeling in atrial fibrillation[J]. Journal of Practical Electrocardiology, 2021, 30(6): 404-407.
[11] Hu J, Zhang J, Li L, et al. PU. 1 inhibition attenuates atrial fibrosis and atrial fibrillation vulnerability induced by angiotensin-II by reducing TGF-beta1/Smads pathway activation[J]. Journal of Cellular and Molecular Medicine, 2021(8): 6746-6759.
[12] Wang Yao, Chen Guiying. Research progress of renin angiotensin system and atrial fibrillation[J]. Hebei Medical Journal, 2022, 44(2): 278-281+288.
[13] Liew R, Khairunnisa K, Gu Y, et al. Role of tumor necrosis factor-alpha in the pathogenesis of atrial fibrosis and development of an arrhythmogenic substrate[J]. Circ J, 2013, 77(5): 1171-1179.
[14] Zhou Shuye, Xie Meili, Wang Zhihao. Research Progress on the Relationship Between Inflammation and Atrial Fibrillation[J]. International Journal of Geriatrics, 2022, 43(6): 742-746.
[15] Qin M, Zeng C, Liu X. The cardiac autonomic nervous system: A target for modulation of atrial fibrillation[J]. Clin Cardiol, 2019, 42(6): 644-652.
[16] Xi Yutao, Cheng Jie. Dysfunction of the autonomic nervous system in atrial fibrillation[J]. Journal of thoracic disease, 2015, 7(2):193-198.
[17] Ye Q, Liu Q, Ma X, et al. MicroRNA-146b-5p promotes atrial fibrosis in atrial fibrillation by repressing TIMP4[J]. J Cell Mol Med, 2021, 25(22): 10543-10553.
[18] Han X, Wang S, Yong Z, et al. miR-29b ameliorates atrial fibrosis in rats with atrial fibrillation by targeting TGFbetaRIota and inhibiting the activation of Smad-2/3 pathway[J]. J Bioenerg Biomembr, 2022, 54(2): 81-91.
[19] Hu Hu, Wu Tao, et al. Effects of miR-145 on the Atrial Effective Refractory Period, Atrial Fibrillation Induction Rate and Atrial Electrical Remodeling in a Rapid Atrial Pacing Model of New Zealand Big-Eared White Rabbits[J]. Molecular Cardiology of China, 2022, 22(1): 4495-4500.
[20] Zhang Song, et al. Resveratrol reduces electrical remodeling in atrial fibrillation by downregulating microRNA-21 in neonatal rat atrial myocytes[J]. Chinese Journal of Pathophysiology, 2017, 33(8): 1353-1358.
[21] Wong C X, Sullivan T, Sun M T, et al. Obesity and the Risk of Incident, Post-Operative, and Post-Ablation Atrial Fibrillation: A Meta-Analysis of 626, 603 Individuals in 51 Studies[J]. JACC Clin Electrophysiol, 2015, 1(3): 139-152.
[22] January C T, Wann L S, Calkins H, et al. 2019 AHA/ACC/HRS Focused Update of the 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society in Collaboration With the Society of Thoracic Surgeons[J]. Circulation, 2019, 140(2): e125-e151.
[23] Dong Xue, et al. Research Progress on the Pathogenesis and Treatment of Atrial Fibrillation in Patients with Obstructive Sleep Apnea-Hypopnea Syndrome[J]. Shandong Medical Journal, 2022, 62(30): 111-114.
[24] Liu Guangzhong. Risk factors analysis and strategies of prevention and treatment of atrial fibrillation[J]. China Journal of Modern Medicine, 2022, 32(7): 1-5.
[25] Xie Yong, Wang Jingfeng. Application of Antiarrhythmic Drugs in Rhythm Control for Atrial Fibrillation[J]. Chinese Journal of Cardiac Arrhythmias, 2025, 29(02): 89-93.
[26] Li Jingdong, Xia Chaorui, Chen Yuan. Progress of electrophysiology and the clinical application of dronedarone[J]. Journal of Clinical Cardiology, 2022, 38(3): 172-176.
[27] Huang Guanming, Wang Jingjing, et al. Exploration of Pathogenesis and Treatment Strategies for Atrial Fibrillation Recurrence After Catheter Ablation Based on"Unity of Body and Spirit"[J]. Jiangsu Journal of Traditional Chinese Medicine, 2025, 57(5): 21-24.
[28] Hauk L. Newly Detected Atrial Fibrillation: AAFP Updates Guideline on Pharmacologic Management[J]. American Family Physician, 2017, 96(5): 332-333.
[29] Zhang Linbo, Rong Chao, et al. Research Progress of Atrial Fibrillation in Traditional Chinese Medicine and Western Medicine[J]. Henan Traditional Chinese Medicine, 2024, 44(5): 790-796.
[30] Ye Haitao, et al. Observation on Therapeutic Effect of Zhigancao Decoction Combined with Metoprolol on

Persistent Atrial Fibrillation[J]. World Journal of Integrated Traditional and Western Medicine2021, 16(4): 731-735.
[31] Ji Meng, et al. Clinical Observation on the Treatment of Non-Valvular Paroxysmal Atrial Fibrillation with Self-Prescribed Chaihu Jia Longgu Muli Decoction Derivative Based on the Pathogenesis Theory of"Mutual Fanning of Liver Wind and Heart Fire"[J]. Journal of Nanjing University of Traditional Chinese Medicine, 2025, 41(3): 405-410.
[32] Xiang Na, Hu Jiayun, Wu Bin, et al. Research Progress on the Treatment of Atrial Fibrillation with Shensong Yangxin Capsule[J]. Chinese Journal of Cardiac Pacing and Electrophysiology, 2024, 38(6): 391-396.
[33] Li Min, et al. The Clinical Study of Wenxin Keli in the Treatment of Atrial Fibrillation: a Systematic Review[J]. Modernization of Traditional Chinese Medicine and Materia Medica-World Science and Technology, 2018, 20(10): 1761-1771.
[34] Xie Hongjuan, Fu Haiying, Zhu Caifeng, et al. Effects of Danshen Injection(Salvia miltiorrhiza) on Pharmacodynamics and Pharamcokinetics of Warfarin in Rats[J]. Herald of Medicine, 2009, (01): 41-44.