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Atrial Fibrillation and Psychosocial Stress: Epidemiology, Risk, Mechanisms and Future Directions

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Abstract

Atrial fibrillation (AF), the most common cardiac arrhythmia links to significant morbidity, mortality, and healthcare costs through typical risks like ageing, hypertension, and structural heart disease. Recent studies show chronic psychosocial stress, negative emotions, and mental health factors in AF susceptibility. This review integrates AF epidemiology, focusing on mental stress and adverse emotional states interacts with atrial electrophysiology. Key mechanisms include sympathetic upregulation, parasympathetic attenuation, Hypothalamic-Pituitary-Adrenal (HPA) axis overdrive, inflammation, and myocardial remodeling. Observational data associate persistent stress with higher AF incidence yet causation and stress reduction effectiveness remain uncertain. Targeting modifiable psychosocial factors that contribute to improve risk stratification management and quality of life.

Keywords: Atrial fibrillation, Mental health, Sympathetic upregulation, Parasympathetic attenuation, Epidemiology, Psychosocial factors, Hypothalamic-Pituitary-Adrenal axis, Myocardial remodeling.

1. Introduction

Atrial fibrillation is characterised by irregular and often rapid atrial activity that reduces cardiac efficiency. Its global occurrence continues to increase due to population ageing and improved detection methods [1]. AF is associated with severe adverse outcomes including stroke, heart failure, and increased mortality, making it a major public health concern. While traditional risk factors such as hypertension, diabetes, obesity, and structural cardiac disease are widely recognised, growing evidence suggests that psychosocial stress and mental health conditions including anxiety and depression may also influence the initiation and progression of AF. These factors affect cardiac rhythm regulation through

alterations in autonomic balance, neuroendocrine function, and inflammatory pathways [2,3].

Historically, AF has been addressed through a biomedical framework. However, recent developments highlight the importance of the "neurocardiac axis," recognising the mutual relationship between psychological states and cardiac function. Stress and emotional distress may not only worsen arrhythmic risk but also affect treatment adherence and quality of life among individuals with AF.

This short review analyses current evidence on the relationship between psychosocial stress and atrial, fibrillation, outlining potential biological mechanisms that connect emotional and cardiac health. It emphasises the need to incorporate psychosocial factors into AF prevention and management to achieve more holistic and effective care.

1.1. Epidemiology of AF

Atrial fibrillation affects an estimated 33 million of people worldwide and forms a major public health challenge due to its tendency to cause significant complications such as stroke and heart failure [1]. Particularly in older adults, the rate and occurrence of AF rise sharply, reflecting not just the ageing of populations but also improved detection through ambulatory monitoring devices and heightened clinical awareness. Over the past several decades, efforts to assess AF's burden have resulted in reliable data suggesting a strong association between chronic AF and greater risk of fatal and non-fatal outcomes. Large population cohorts such as Framingham report that AF confers approximately a five-fold increase in stroke risk and a near two-fold increase in all-cause mortality after adjustment for conventional cardiovascular risk factors. Incidence increases with age, rising from less than 1% in adult adults under 60 to over 8-10% in people aged 80 and above [4].

Beyond these well-recognized biomedical contributors, epidemiological investigations increasingly point toward psychosocial elements that may contribute to the risk of AF development. Specifically accumulating data suggests that situational, occupational, and express exposure can act as triggers or worsening factors of cardio metabolic dysfunction affecting atrial rhythm stability [5]. Within the older adult group who inherently face greater vulnerability to both cardiovascular and mental health conditions, new research highlights that extended stress states could significantly increase the risk of AF [6].

1.2 Impact

The consequences of AF extend far beyond its cardiovascular burden. Stroke and heart failure remain the most feared complications, but the broader impact also includes reduced quality of life, cognitive decline and increased healthcare utilisation. Cross sectional studies consistently report that depression, anxiety, and other mood disturbances are more prevalent among individuals with AF compared to those without the condition. While the direction of causality remains complex, evidence shows that psychological distress can aggravate symptoms, reduce treatment adherence and increase hospitalisation rates.

Collectively these findings underscore that the AF epidemic must be understood within both biomedical and psychosocial framework. Effective management therefore requires

integrated strategies that not only target traditional cardiovascular risk factors but also identify and mitigate mental health contributors influencing arrhythmic outcomes.

1.3 Psychosocial Stress and AF Risk

Emotional states have been linked to cardiac electrophysiological changes for decades. Stress and negative emotions including anger, anxiety and depression have been considered arrhythmogenic for certain ventricular rhythm disorders [7,8]. In the realm of supra-ventricular arrhythmias particularly AF, multiple observational studies have shown associations between psychological distress and the beginning of AF episodes. One of the most cited human population analyses stems from sub-studies within the Framingham Offspring population which highlight that chronic anger, hostility, and distinct anxiety traits can predict a higher occurrence of AF [9,10].

These findings support a psychosomatic interaction, where negative affective patterns promote maladaptive psychological responses. Notably, other prospective data sets describe how occupational strain can lead to enhanced cardiovascular sympathetic responses overtime, reliably linking overcommitment, low reward or high job demands to an increased risk of developing AF [11].

Recent studies propose that short-term mental stress incidences can trigger acute physiological changes leading to premature atrial contractions which are considered precursors of paroxysmal AF. These findings are supported by clinical investigations noting that changes from acute stress states such as emotional disturbance or anxiety related events can trigger incident or recurrent AF [12].

Moreover, chronic exposure to high stress levels can induce systemic inflammation and endothelial dysfunction both implicated in remodeled myocardial substrates conductive to AF [2]. An important aspect within psychosocial research on AF risk concerns the quantification of stress. Stress is multifactorial, spanning acute, situational episodes, 2 persistent, pervasive life stressors (for example, caregiving, stress, financial stress, or social isolation).

While more studies do not adopt a uniform measure of stress, overall evidence suggests that both acute and long-term stress exposures may collectively increase AF vulnerability highlighting complex interaction between emotional well-being, autonomic regulation, and atrial electrophysiology.

1.4 Psychological tests and study designs

Across the literature, psychosocial stress and emotional states are mainly measured using validated self-report questionnaires. Prospective AF studies, commonly assess anger, hostility and anxiety traits and depressive symptoms using tools such as Beck Depression Inventory, while studies on work related stress use job-strain models and effort-reward imbalance measures to describe occupational stress.

Most evidence linking psychosocial stress to incident or recurrent AF drives from observational cohorts, case control designs and case crossover analysis that relate questionnaire defined stress or mood to subsequent AF episodes. **Table 1** summarises representative studies with their design, sample size, exposure definition and main findings.

Author (year)	Design and population	n (participants)	Exposure/ psychological test	AF outcome	Main result
[4]	Framingham heart study	4618 adults (mean age 69) free of AF at baseline	Baseline cardiovascular risk factors; no formal stress scale	Incident AF	AF associated with markedly Higher/and all causes mortality compared with people without AF
[1]	Global burden of disease systematic analysis	184 countries, pooled population-based data	Not applicable	Prevalence and incidence of AF	Around 33 million people worldwide living with AF with rising global prevalence and incidence
[9]	Prospective cohort, Framingham offspring	3682 participants, mean age 48.5; 52% women	Standardised trait anger, hostility, and anxiety scales	10-year incidence of AF, CHD, mortality	Higher trait anger, hostility, predicted increased incidence of AF over follow up in the Framingham offspring cohort
[10]	Prospective cohort, Framingham offspring	3682 participants, mean age 48.5 ± 10.1 y; 52% women	Self-reported tension and anxiety scales	10-year incidence of AF, CHD and mortality	Higher tension, anxiety scores in men predicted greater long-term risk of AF and cardiovascular events
[11]	Prospective occupational cohort, Swedish workers	13200 employees without prior AF	Job demand-control questionnaire defining high strain	Incident AF identified from national registers	High Job strain modestly increased the risk of incident AF compared with low jobs strain
[13]	Prospective cohort, Women's Health Study	30000-34000 middle-aged women free of CVD and AF	Global physiological wellbeing/ optimism scale	Incident AF during long term follow-up	Higher psychological wellbeing/ optimism was associated with a lower risk of developing AF
[12]	Prospective cohort with nested analyses	Several hundred patients with stable coronary disease	Mental stress-induced ischemia, depressive and PTSD symptom scales	Incident and recurrent AF during follow-up	Mental stress-induced ischemia and higher depressive, PTSD symptoms were linked with more frequent AF episodes,
[2]	Narrative/syste matic review of AF and psychological factors	Not applicable	Summarises, use of depression, anxiety and perceived stress scales in AF cohorts	AF incidence	Depression, anxiety are common in AF and associated with worse symptoms, more hospitalisation and possibly higher AF recurrence
[14]	Nonrandomised interventional study, AF patients	Small AF cohort	Relaxation/ yogabased behavioural programme	AF symptoms and arrhythmia burden	A stress reduction intervention was associated with fewer AF symptoms and better quality of life

Table 1: *Summary of key epidemiological and prospective cohort studies evaluating the association between psychological stress, emotional factors, and the incidence, prevalence, and outcomes of atrial fibrillation.*

1.5 Biological mechanisms linking stress to AF

A defining feature of chronic or acute psychological stress is a shift in the neuroendocrine and autonomic systems. Stress typically activates the HPA axis, leading to increased cortisol and other glucocorticoid levels as well as the sympathetic nervous system, which is characterised by elevated catecholamine secretion [15]. This interaction is sometimes referred to as the neurocardiac axis, framing the concept that the brain and heart interact in complex ways under conditions of emotional disturbance [16]. Heightened sympathetic activity can promote arrhymogenic substrates in the atria. Mechanistically, elevated catecholamines (eg. Norepinephrine, epinephrine) can shorten the atrial refractory period, increase the likelihood of ectopic beats and augment electrical heterogeneity across atrial tissue. When combined with reduced parasympathetic activity, as often seen in prolonged stress or anxiety, the typical autonomic balance that maintains cardiac rate and rhythm can be disrupted leading to arrhythmic events [2,17].

Simultaneously, chronic stress can stimulate systemic inflammation marked by elevated levels of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumour necrosis factor alpha (TNF-α) and C reactive protein (CRP). These inflammatory markers in turn may speed up pathological remodelling of the atrial substrate, including fibrosis and conduction abnormalities that can prolong AF [2,5]. Moreover, oxidative stress, which is often heightened in conditions of chronic mental distress can intensify these changes, further stabilising AF once triggered. These neurohormonal and inflammatory processes present an opportunity for in-depth analysis. Ongoing research aims to differentiate between short-term stress bursts (for example, acute grief response) and long-term stress burdens (such as severe workplace strain) in terms of their specific mechanistic impacts for arrhythmia, it is initiation and continuation. Additionally, factors like epigenetic, which may underlie differential responses to psychosocial stress are just beginning to be examined in the context of AF pathophysiology.

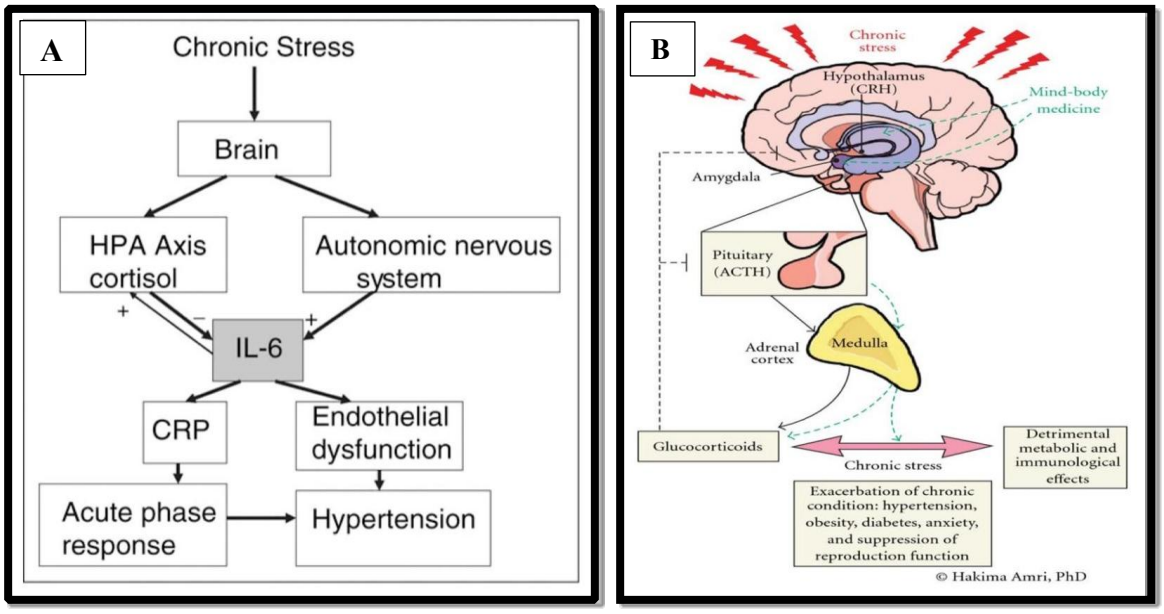

Figure 1: [A] *Mechanism showing link in chronic stress to cardiovascular dysfunction through activation of the HPA axis, autonomic nervous system, and inflammatory mediator IL-6, which leads to endothelial dysfunction, hypertension, and acute phase inflammatory responses. [18].* ***[B]*** *Chronic stress pathway involving the HPA axis and its metabolic and immunological effects [19].*

1.6 Protective role of positive emotions

While the adverse effects of negative mood and high stress on cardiovascular health have been documented, there is a parallel body of evidence suggesting that psychological well-being and positive emotional states may act as protective factors against AF. A study by Dr. Whang found that individuals reporting higher levels of global psychological health experienced a reduced occurrence of AF suggesting that optimism, life satisfaction, and other positive affective states could balance some biological pathways implicated in AF initiation [13].

One plausible explanation is that the stable positive effect helps maintain balanced autonomic tone, thereby reducing frequent swings toward sympathetic overdrive or vagal withdrawal. Moreover, healthier stress management often correlates with better lifestyle habits, as individuals who maintain positive outlooks more inclined toward regular exercise, improved sleep patterns and nutritious diets, factors also recognised to reduce the risk of cardiovascular disease. Indeed, existing data points to stressreduction interventions such as mindfulness, training, yoga, or cognitive behaviour therapy as offering at least minor benefits for individuals with a range of cardiovascular or arrhythmia related conditions [2,20].

2. Critical discussion

Current evidence from community, occupational and clinical cohorts indicates that psychosocial factors show consistent but modest associations with atrial fibrillation. Higher levels of anger, hostility, tension, anxiety and job strain are repeatedly linked with greater AF incidence, whereas higher psychological well-being and optimism tends to relate to a lower risk of developing AF and patients with AF who report significant depressive or anxiety symptoms frequently experience more severe symptoms and increased healthcare use. Altogether, these findings support a contributory role of psychosocial stress and emotional states in AF vulnerability and clinical burden, most likely through interaction with established cardiovascular risk factors and shared neuroendocrine and inflammatory mechanisms. Despite accumulating evidence that links psychosocial stress and adverse emotions to AF, significant limitations percents the ability to reach definitive conclusions.

One recurring challenge is that much of the data arise from observational or cross-sectional designs, limiting conclusion about causation. Typically, these studies can identify strong associations but cannot conclusively demonstrate that the presence of psychological stress leads to AF, as opposed to individuals developing stress in reaction to symptomatic or anxiety provoking arrhythmic episodes [20].

3. Limitations

Additionally, definitions of stress differ across research protocols. Some researchers focus on acute stress triggers such as highly emotional personal events or major life changes. Others measure chronic stress through validated instruments, but these instruments vary between trials. Further uncertainty arises from subjective reporting as stress perception can differ significantly among individuals. Although device-based metrics (like heart rate variability under mental stress tasks) offer objective measures, they are not commonly used in large epidemiological studies limiting the ability attempts to generalise findings [21].

It is also notable that confounding factors often cluster among populations exposed

to higher levels of psychosocial stress. Behavioural risk factors such as tobacco use, excessive alcohol intake or unhealthy dietary patterns, may directly increase AF risk and correlate with stress exposure. Socioeconomic disadvantages such as lower educational level or job insecurity can be both a source of stress and a cause of limited access to preventive healthcare further increasing AF risk. Hence separating the specific impact of stress from other linked factors remains a complex task. Moreover, mental stress is static, it may vary over a person ‘s life. AF too, often changes between paroxysmal and persistent forms or progresses from an initial diagnosis to a chronic state. Understanding whether psychological interventions affect these trajectories will require carefully designed, prospective randomise trials with long-term follow-up periods. Despite these challenges, clarifying these pathways is essential for advancing AF prevention and for developing better clinical management strategies that target both the emotional and physiological drivers of the arrhythmia.

4. Future directions

Future research should focus on using standardised tools to measure both acute and chronic psychosocial stress, including validated, questionnaires, biological markers, such as cortisol and inflammatory cytokines and physiological measures like heart rate, variability. Randomised controlled trials of stress-reduction interventions, including cognitive behavioural therapy, structured exercise programmes, or yoga are needed to determine causality and clinical benefit in atrial fibrillation (AF). Mechanistic studies using longitudinal biomarker assessment may help explain individual differences in stress responses and AF progression. Incorporating psychosocial assessment into routine AF management, particularly for vulnerable groups, such as older adults and socio-economically disadvantaged populations, may improve risk stratification and patient outcomes [22].

5. Conclusion

Atrial fibrillation is a complex condition with significant implications for patient morbidity and healthcare burden. While traditional risk factors such as ageing, hypertension and structural heart disease remain central to AF pathogenesis, growing evidence suggests that psychosocial stress and negative emotional states may also influence AF onset and progression [22]. Proposed mechanisms include autonomic imbalance, heightened sympathetic activity, reduced parasympathetic tone, systemic inflammation, and oxidative stress, all of which may impair atrial electrical stability. However, existing studies are limited by heterogeneous stress assessments and a scarcity of randomized controlled trials, restricting causal inference. Future research should address these gaps and evaluate whether targeted stress management strategies can improve AF outcomes. Integrating psychosocial assessment into clinical practice may enable a more holistic person centred approach to AF prevention and management.

Conflict of Interest Statement

Disha Jain declares no conflicts of interest. The work was conducted independently, ensuring objectivity and scientific integrity.

Data Availability Statement

All data discussed in this review are available from the cited sources. No new data were generated for this review.

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