International Journal of Biology and Life Sciences
ISSN: 2957-9511 | Vol. 10, No. 1, 2025

Interaction between Sleep Rhythm and Arrhythmia

-- Analysis Based on EEG Characteristics of Sleep in Atrial Fibrillation Patients

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Abstract: A regular heart rate is a critical indicator of human health. However, when the heart's rhythm or rate deviates from the norm due to various factors, an arrhythmia occurs. Atrial fibrillation (AF) is the most prevalent form of arrhythmia, with its incidence tripling over the past 50 years. Studies have shown a strong correlation between sleep disorders and the onset of atrial fibrillation. Most current studies on the sleep quality of atrial fibrillation patients rely on subjective sleep questionnaires, with a notable lack of objective sleep assessments. This study utilizes data from the public database "The Stanford Technology Analytics and Genomics in Sleep (STAGES)" to identify atrial fibrillation patients, matching them with a healthy control group to analyze sleep characteristics and influencing factors. The study found that, compared to the healthy control group, atrial fibrillation patients exhibited significantly lower total sleep time and deep sleep duration, alongside increased wakefulness after sleep onset. Correlation analysis indicated an inverse relationship between body mass index (BMI) and total sleep time in patients with atrial fibrillation. To further validate these findings, a decision tree model was employed to elucidate the relationships between various sleep indicators and the incidence of atrial fibrillation, along with the relative weights of different risk factors. The model results suggest that the most significant predictor of atrial fibrillation occurrence is reduced stage 1 sleep, followed by increased wakefulness during sleep and elevated body mass index. These findings indicate that atrial fibrillation patients generally have poor sleep quality, and elevated body mass index further exacerbates sleep disturbances, thereby increasing disease risk. The above conclusions suggest that healthcare providers and family members should monitor the sleep patterns and body mass index of atrial fibrillation patients, encouraging a balanced diet and regular exercise.

Keywords: Atrial Fibrillation; Sleep; Polysomnography System; Body Mass Index.

1. Research Background

1.1. The Rhythmic Enigma of Atrial Fibrillation

In *Heart: A History*, American cardiologist Sandeep Jauhar states that the heart exists to beat, with cardiac cells connecting through gap junctions to synchronize their rhythm, thus driving the body's metabolic cycle. From birth to death, the human heart beats approximately 3 billion times[1]. A healthy heart typically beats at a rhythm of 60 to 100 beats per minute, known as the "heart rate." A normal heart rate is essential for stable blood circulation and serves as a key indicator of health.

When the heart fails to maintain a regular rhythm, arrhythmia occurs. Specifically, atrial fibrillation (AF), also known as A-Fib, is a type of rapid and irregular atrial rhythm. Clinically, patients often experience palpitations, shortness of breath, chest tightness, and difficulty breathing. AF can lead to serious complications, including the risk of atrial thrombi dislodging and causing cerebral or peripheral arterial embolism[2]. Statistics show that 24.8% of AF patients experience stroke, leading to sudden death or long-term disability.

Globally, AF is an increasingly severe health concern. Over 33 million people worldwide are affected by AF, with this number continuing to rise[4]. A cohort study based on the Framingham Heart Study indicates that AF incidence has increased approximately threefold over the past 50 years[5], with a marked increase in prevalence as age advances. In China, the overall prevalence of AF among those aged 18 and older is 1.6%, rising to 5.9% in individuals over 80[6]. A large European prospective cohort study of individuals aged 55 and older found an AF prevalence rate of approximately 5.5%, which increased to 17.8% in those over 85[7]. Therefore, the risk of AF increases progressively throughout the human lifespan, posing a significant public health concern.

The etiology of atrial fibrillation (AF) is complex, with patients often exhibiting multiple comorbidities or risk factors, such as sleep disorders, obesity, hypertension, and diabetes. These factors complicate the course of AF, making treatment more challenging and increasing the risk of recurrence[2]. According to Interpretation *of the 2020 ESC/EACTS guidelines for the diagnosis and management of atrial fibrillation*, a comprehensive approach to AF management involves not only treatment methods such as anticoagulation, stroke prevention, heart rate control, and rhythm control, but also the evaluation of quality of life and risk factors. Lifestyle interventions are recommended to actively prevent the onset of AF[8]. Therefore, investigating and clarifying the risk factors associated with AF is of paramount importance.

1.2. The Circadian Rhythm of Cardiac Rhythm

Human organs are regulated by circadian rhythm genes, maintaining periodic fluctuations approximately every 24 hours. The cardiac rhythm also follows a distinct circadian pattern.

Under normal physiological conditions, the average heart rate drops by about 10%-20% during sleep compared to daytime. Upon waking, the heart rate quickly accelerates, peaking within 3 to 4 hours, and then gradually decreases,

reaching its lowest point between 3:00 and 5:00 a.m., establishing a day-night cycle [9]. This rhythmic cycle is primarily regulated by the autonomic nervous system and hormonal secretion [10]. Maintaining a normal circadian rhythm is essential for cardiac health.

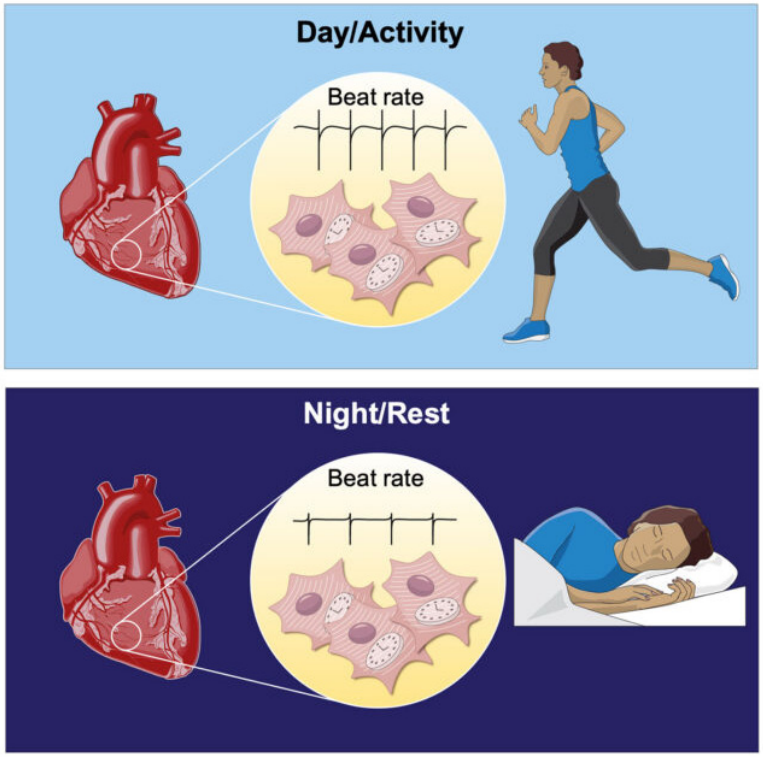

Figure 1. Circadian Rhythm of Cardiac Rhythm
Image Source: The article *How Cellular Clocks within Heart Cells Coordinate Daily Cardiac Rhythms* on the website of the MRC Laboratory of Molecular Biology

In fact, the brain uses external cues such as daylight to adjust or reset the body's biological clock[11]. As shown in Figure 1, during nighttime sleep, the heart is affected by both sympathetic and vagal nerve activities, resulting in reduced myocardial contraction and a slower heart rate[12]. However, in modern society, common issues like late-night habits, artificial lighting, jet lag, and shift work disrupt normal sleep schedules, desynchronizing the body's internal clock from external environmental changes[13]. This disruption in the circadian rhythm of cardiac rhythm is a significant driver of arrhythmias, including atrial fibrillation[14].

Scholars have conducted studies using subjective and objective measures to investigate the relationship between sleep and the onset of atrial fibrillation. Results indicate that sleep issues such as insomnia, frequent awakenings, excessively long (≥8 hours) or short (≤6 hours) sleep duration, and low sleep efficiency can all contribute to the incidence of AF[15-18]. A study based on statistical data regarding AF patients' subjective sleep quality, sleep onset time, sleep duration, sleep disturbances, sleep efficiency, and daytime dysfunction found that over half of AF patients report poor sleep quality[16]. In a pioneering longitudinal study, Genuardi et al. objectively measured sleep duration and found that each 1-hour reduction in sleep time increases the risk of AF by approximately 9%[17]. Additionally, a meta-analysis conducted by researchers indicated that sleep durations of 8 hours or more can increase the risk of AF by 18%[18]. Beyond sleep duration, there is a strong relationship between AF and obstructive sleep apnea syndrome (OSAS). In a study on nocturnal arrhythmias and sleep-disordered breathing, a comparison between 228 individuals with sleep-disordered breathing and 338 without showed that the incidence of nocturnal AF was approximately five times higher in those with sleep-disordered breathing[19].

1.3. The Interaction Between the Heart and Brain During Sleep

Neuroscientist Matthew Walker once referenced a wise adage from British writer Charlotte Brontë: "A restless heart makes for poor sleep[11]." This evocative literary expression likely has scientific merit and suggests a link between sleep and cardiac arrhythmias.

In reality, the communication between the heart and brain is continuous and mutually influential[20]. When the brain experiences anxiety, it sends signals to internal organs, causing the heart to beat faster. In turn, an increased heart rate stimulates the brain's occipital cortex, triggering anxiety-like responses[21]. The brain constantly monitors signals from internal organs to maintain body movement and balance, processing sensations like fullness or palpitations to help sustain bodily homeostasis. This process is known as interoception[20]. Those with dysregulated interoception may experience organ dysfunction. For instance, individuals with certain mental disorders, such as phobias, display arrhythmic symptoms more frequently than healthy individuals[20]. This interplay persists even during sleep. Throughout sleep, the parasympathetic nervous system activates, slowing the heart rate to induce calm and relaxation. This decelerated rhythm is also observable in EEG patterns during sleep.

Current research on atrial fibrillation and sleep quality predominantly uses the Pittsburgh Sleep Quality Index (PSQI). This index relies on a subjective questionnaire to assess sleep quality metrics in AF patients. Consequently, these studies depend on patients' personal perceptions and

memory recall, which may compromise the accuracy and reliability of the findings. An accurate and objective evaluation of sleep quality is essential for analyzing the relationship between AF and sleep architecture. The rhythmic features of the human body during sleep are primarily reflected in electrophysiological metrics; thus, monitoring EEG data in AF patients allows for an objective assessment of sleep characteristics and analysis of the correlation between sleep rhythms and arrhythmias [22]. Polysomnography (PSG) collects comprehensive electrophysiological metrics from subjects during sleep, including EEG, chin and leg EMG, nasal and oral respiration, chest and leg movement, body position, and ECG. This method provides an objective assessment of sleep quality.

2. Research Objectives

The prevalence of atrial fibrillation (AF) remains high, yet awareness among family and friends is limited, and preventive measures are poorly understood. Last year, my grandmother was diagnosed with AF. My initial research into this common yet unfamiliar disease led me to a deeper interest in exploring its risk factors.

Several articles on AF reference an influential scientific statement by the American Heart Association titled *Lifestyle and Risk Factor Modification for Reduction of Atrial Fibrillation*, published in *Circulation*, which I found particularly enlightening. Many factors contribute to AF, some of which may be mitigated through lifestyle changes, especially sleep disorders, obesity, and sedentary habits—common issues in modern society. However, our understanding and research on these aspects remain insufficient[23].

I have long been interested in sleep science. Through reviewing relevant literature, I learned that the United States hosts several public databases on sleep medicine, collecting data across age groups on patients evaluated for sleep disorders. These databases include polysomnographic data, demographic details, psychological assessment scales, and lifestyle information for some AF patients. The public can access and download shared datasets via the National Sleep Research Resource (NSRR) portal[24]. My goal is to analyze these data to objectively and quantitatively examine the interactions between AF and sleep rhythms and to evaluate the weight of various risk factors contributing to AF. This research aims to enhance public awareness of AF risk factors, promote healthier lifestyles, and reduce AF incidence.

3. Theoretical Foundation of the Study

3.1. Principles of EEG Data Recording in Sleep

Electroencephalogram (EEG) recordings during sleep are considered the gold standard for assessing sleep quality. In laboratory settings, polysomnography (PSG) is typically used to collect various physiological data from subjects during sleep, including EEG signals, eye movements, chin electromyography (EMG), electrocardiogram (ECG) signals, respiratory patterns, and body position.

To collect data, electrodes are strategically placed on the subject's scalp in accordance with the American Academy of Sleep Medicine's international 10-20 electrode placement system[25], as illustrated in Figure 2. The chin EMG setup includes a reference electrode and two active electrodes. The reference electrode is positioned 1 cm below the chin's midline, while the two active electrodes are placed 2 cm below the chin's midline and horizontally 2 cm to the left and right. For ECG signal collection, a reference electrode is placed below the right clavicle, and an active electrode approximately 2.5 cm below the left clavicle. Prior to data recording, the electrode impedance for ECG and chin EMG signals must not exceed 20 kΩ, while EEG electrode impedance should be kept below 10 kΩ. Laboratory staff must maintain a sampling rate of 500 Hz and carefully control the humidity, temperature, and sound insulation in the sleep environment.

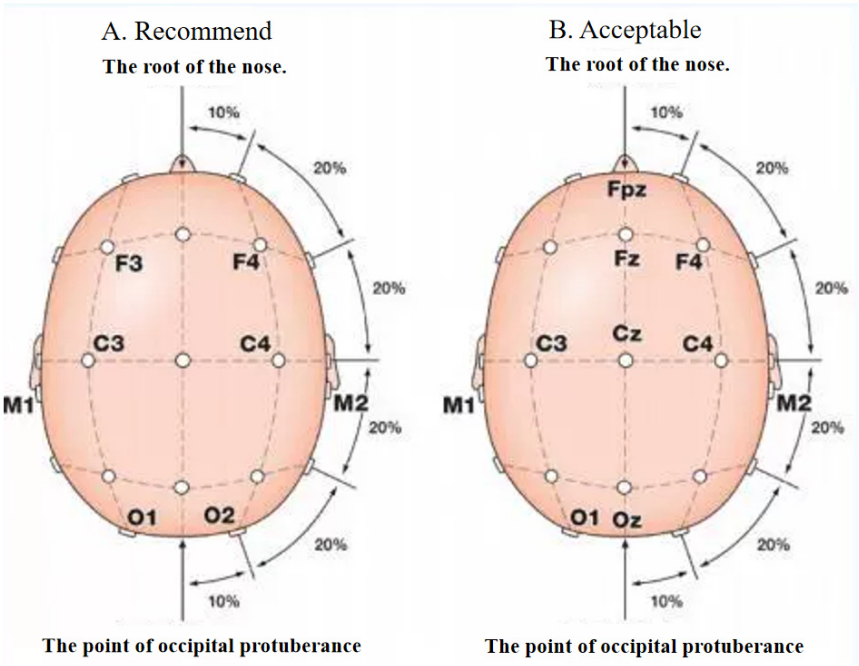

Figure 2. International 10-20 Electrode Placement System

Note: According to the International 10-20 Electrode Placement System, EEG electrodes are positioned over the frontal, parietal, and occipital lobes of the brain, with reference electrodes typically placed behind the mastoid areas of both ears

Image source: *The American Academy of Sleep Medicine and Sleep Related Event Interpretation Handbook (Rules, Terms, and Technical Specification) [25]*

3.2. Sleep Architecture Staging

For the sleep data in this database, staging is conducted using 30-second epochs based on *The American Academy of Sleep Medicine and Sleep Related Event Interpretation Handbook (Rules, Terms, and Technical Specificat*ion) [25].

Sleep architecture is categorized into rapid eye movement (REM), non-REM (NREM), and wakefulness stages. NREM sleep comprises stages NREM1, NREM2, and NREM3, representing progressively deeper sleep stages. By examining various electrophysiological changes during sleep, several parameters are derived, including: (1) Total Sleep Time (TST), the sum of time spent in each sleep stage; (2) Sleep Onset Latency (SOL), the time taken to fall asleep after lying down; (3) Wake After Sleep Onset (WASO), the cumulative duration of wakefulness after initially falling asleep.

4. Data and Methods

4.1. Data Mining in the U.S. Sleep Medicine Public Database

The National Sleep Research Resource (NSRR) is a comprehensive system that organizes and stores extensive sleep data from multiple sources, including polysomnography, questionnaire-based data, and activity monitor counts. Researchers can utilize these public datasets for secondary data analysis in studies on sleep and circadian rhythms[24].

In this study, we followed NSRR website guidelines to use the NSRR gem browser to download and access data from the Stanford Technology, Analytics, and Genomics in Sleep (STAGES) database[24]. STAGES is a prospective, cross-sectional, multi-site study that collects data from 20 sites associated with six institutions, including Stanford University and the Mayo Clinic. The dataset includes sleep disorder assessments for over 1,800 patients aged 13 to 84. The data primarily consists of polysomnographic records (including EEG, chin and leg EMG, ECG, respiratory, and body position records), demographic and key health data gathered through the online Alliance Sleep Questionnaire (ASQ), and multi-week activity monitor counts. With NSRR authorization, individuals can access the entire dataset and samples to conduct analytical research on existing data.

This study included 74 subjects from the STAGES database, comprising 38 atrial fibrillation patients (average age 53 years) and 36 healthy controls (average age 53 years), with a gender distribution of 36 males and 38 females. Detailed case information is accessible via https://sleepdata.org/ datasets/ stages/ variables? folder=Demographics.

4.2. Statistical Analysis

Data analysis was performed using SPSS version 27. Independent sample t-tests ($P < 0.05$) were used to compare demographic data, lifestyle habits, and sleep architecture between the two groups. Pearson's correlation test ($P < 0.05$) was employed to assess the correlation between demographic data and sleep structure. A decision tree model was applied to fit the existing data, resulting in a basic predictive model for AF occurrence.

5. Research Results

This study included data from 100 atrial fibrillation (AF) patients and healthy controls from the STAGES database. After evaluating psychological assessment scales and sleep architecture, 26 cases were excluded, resulting in a final sample of 74 participants (36 males and 38 females). Among them, there were 38 AF patients (average age 53) and 36 healthy controls (average age 53). All participants underwent laboratory-based polysomnography (PSG).

5.1. General Demographic Analysis

As shown in Table 1, the demographic scores for both groups fell within the normal range. However, AF patients showed higher total scores on the depression scale compared to the healthy controls, indicating mild depressive symptoms in the AF group.

Table 1. Demographic and Psychological Assessment

Characteristic	Control	Atrial fibrillation	*p*
	(n=36)	(n=38)	
Gender(M/F)	13/23	23/15	0.60
Age	55.11±1.74	52.94±1.57	0.36
BMI	32.61±1.47	31.53±0.97	0.54
Days Per Week at Work	4.84±0.21	4.83±0.21	0.98
FSS Total Score	29.84±2.09	34.71±3.01	0.19
GAD Total Score	3.50±0.73	4.89±0.80	0.20
PHQ Total Score	3.89±0.57	7.71±0.97	0.00
Family History of Insomnia	36	35	0.93

Note: BMI, Body Mass Index; FSS, Fatigue Severity Scale; GAD, Generalized Anxiety Disorder Scale; PHQ, Patient Health Questionnaire

5.2. Lifestyle and Chronic Disease History

As shown in Table 2, The scores for lifestyle habits and chronic disease history in both groups were within the normal range, indicating that both groups maintained typical daily dietary habits and showed no signs of hypertension or neurological conditions.

Table 2. Lifestyle and Behavioral Health Aspects

Characteristic	Control (n=38)	Atrial fibrillation (n=36)	*p*
Caffeine Total servings	2.23±0.28	3.13±0.59	0.17
Age Stopped Smoking Cigarettes	36.50±3.71	36.10±3.13	0.94
Hypertension	0	0	0.34
Neurological problems	0	0	0.22
Nap duration hours	0.81±0.21	1.00±0.24	0.56
Easily awakened after nap	2.86±0.25	2.69±0.33	0.68
Exercise Number of times	0.50±0.08	0.47±0.08	0.81
Alcohol Number of times	17	13	0.27

5.3. Sleep Architecture Analysis

As shown in Figure 3, further, the study evaluated differences in EEG sleep architecture between the two groups throughout the night. Independent sample t-tests were conducted to compare sleep architecture, revealing that, compared to the healthy controls, AF patients had significantly lower total sleep time and deep sleep duration, as well as a noticeably higher wake after sleep onset (WASO).

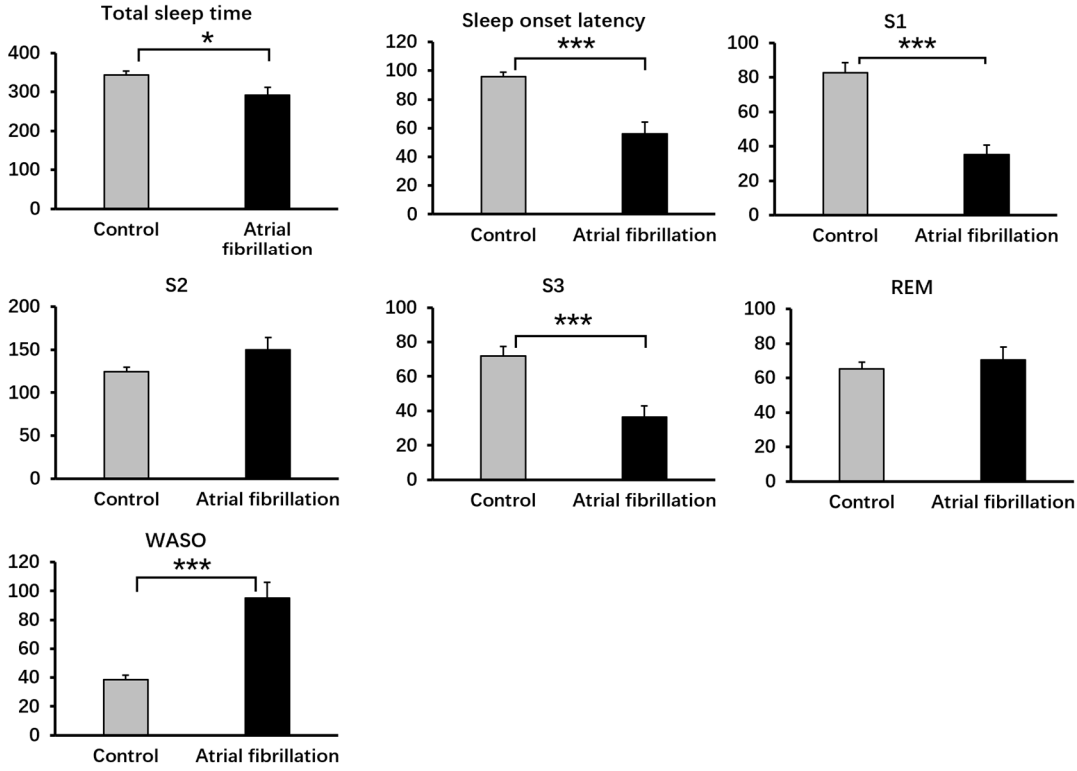

Figure 3. Analysis of EEG Sleep Architecture Differences Between AF Patients and Healthy Controls

Note: S1, Stage 1 Sleep; S2, Stage 2 Sleep; S3, Stage 3 Sleep; REM, Rapid Eye Movement Sleep; WASO, Wake After Sleep Onset.

5.4. Relationship Between Lifestyle Habits and Sleep Architecture in AF Patients

This study also analyzed the relationship between lifestyle habits and sleep architecture in AF patients. Using body mass index (BMI) as the independent variable and EEG sleep architecture as the dependent variable, Pearson's correlation analysis revealed a significant negative correlation between BMI and total sleep time in AF patients ($P < 0.05$), as shown in Figure 4.

These findings indicate that the higher the BMI in AF patients, the lower their total sleep time. This suggests that AF patients generally experience poorer sleep quality, with elevated BMI further aggravating sleep disturbances.

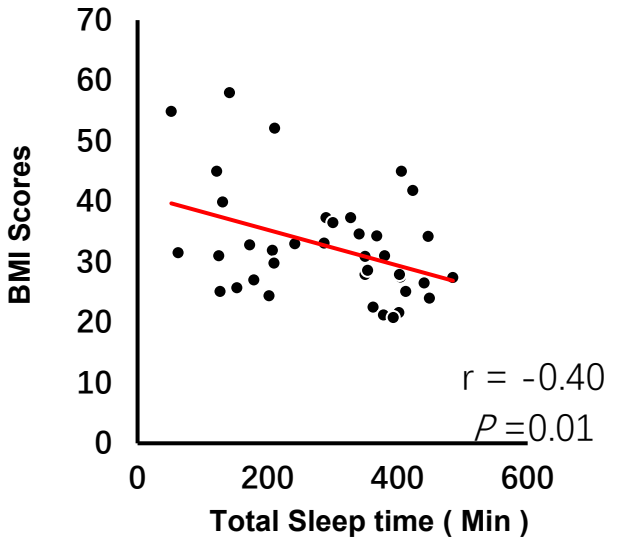

Figure 4. Relationship Between BMI and Sleep Architecture in AF Patients

Note: The horizontal axis represents Total Sleep Time, and the vertical axis represents Body Mass Index (BMI)

5.5. Comparative Analysis of Risk Factor Weights in AF Patients

To further validate these data, a decision tree model was applied to assign weights to various risk factors. The model used sleep architecture indices, demographic data, psychological assessment scores, and lifestyle habits as independent variables, with the occurrence of AF as the dependent variable.

The model results indicated that the most significant predictor of AF occurrence was a reduction in stage 1 sleep (Chi-Square=28.008, *P*-value<0.001), followed by increased wakefulness after sleep onset (Chi-Square=26.372, *P*-value < 0.001) and elevated BMI (Chi-Square=10.000, *P*-value< 0.05), as shown in Figure 5.

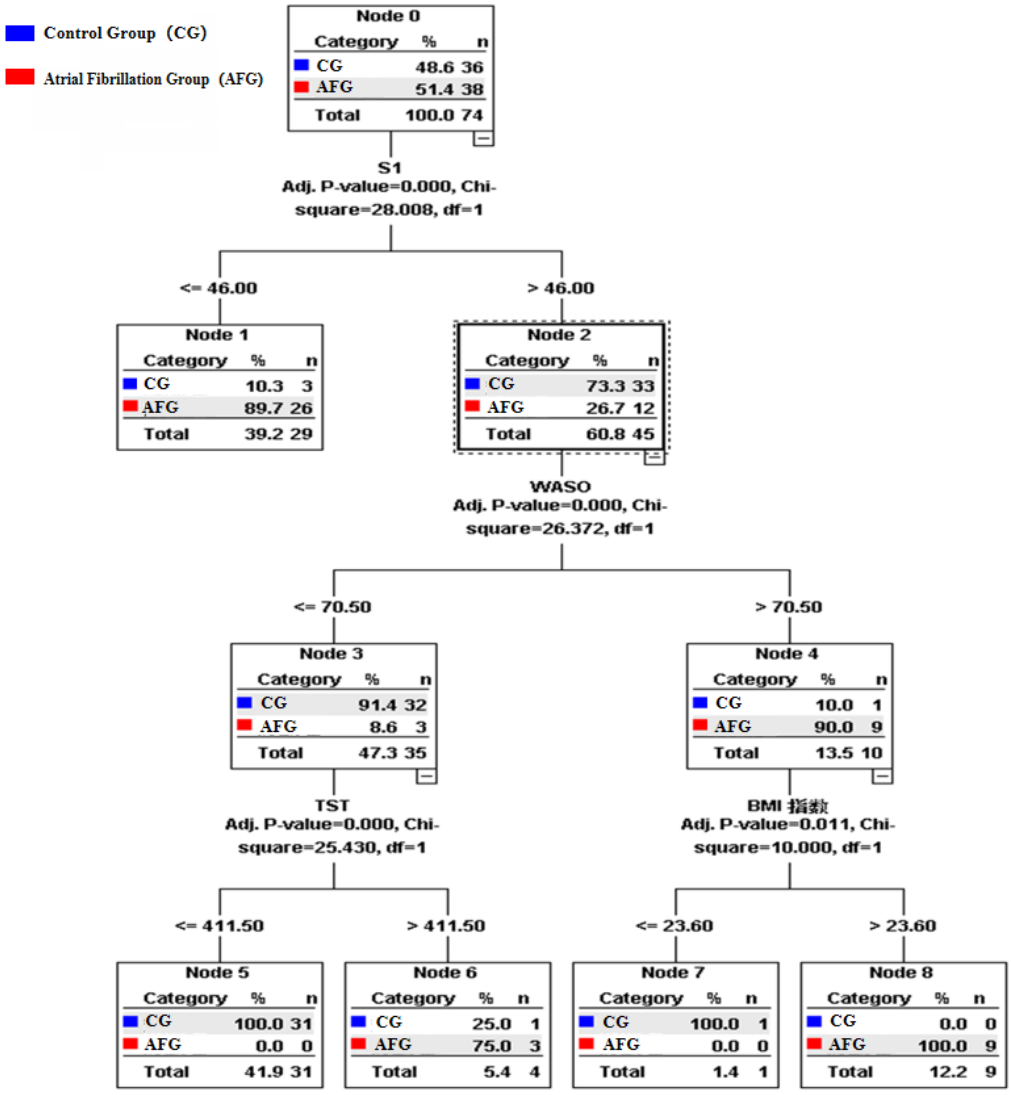

Figure 5. Analysis of Risk Factors for Atrial Fibrillation Based on a Decision Tree Classification Model
Note: S1, Stage 1 Sleep; WASO, Wake After Sleep Onset.

6. Discussion

The relationship between atrial fibrillation (AF) and sleep quality represents a frontier topic at the intersection of neuroscience and cardiovascular medicine. During various stages of sleep, the brain influences circulatory activity, while cardiac function, in turn, affects sleep architecture. Patients with sleep disorders may experience autonomic dysfunction, which can lead to circulatory diseases, including AF[26]. In

the clinical treatment of cardiovascular diseases, researchers have explored methods to improve circadian rhythms, such as extending sleep duration, light therapy, and melatonin supplementation[27], aiming to prevent cardiovascular disease and improve patient outcomes by enhancing sleep quality.

This study selected 38 AF patients from 1,881 samples of PSG data in the STAGES public database. An additional 36 healthy controls were chosen based on psychological scale assessments and sleep architecture evaluations. Both groups underwent polysomnographic staging to analyze parameters such as total sleep time and REM duration. Results indicated that compared to the healthy control group, AF patients had shorter total sleep time, reduced deep sleep duration, and a relatively unstable sleep state, identifying abnormal sleep structures and related risk factors. Compared to most studies using subjective questionnaires like the PSQI, this study provides a more objective reflection of sleep architecture, revealing pathological sleep characteristics in AF patients and confirming the correlation between AF and sleep rhythms. These findings may increase awareness of AF prevention from the perspective of sleep improvement.

Additionally, this study analyzed demographic data, lifestyle habits, and chronic disease history in AF patients, correlating these with EEG data. The analysis revealed a significant correlation between BMI and total sleep time, showing that higher BMI is associated with poorer sleep quality. Thus, both sleep architecture indices and BMI may serve as risk factors for AF in these patients.

To validate these conclusions and further understand the weight of each risk factor, a decision tree model was employed to fit the data. The model identified reduced stage 1 sleep as a key risk factor closely associated with AF onset, followed by increased wakefulness during sleep and elevated BMI. This model suggests that both sleep disturbances and BMI are significant risk factors for AF, highlighting the importance of a healthy lifestyle—such as dietary regulation, physical exercise, and improved sleep quality—for AF prevention in both healthy individuals and AF patients.

This study used polysomnographic data from a public database to evaluate sleep architecture and the ASQ questionnaire to analyze demographic, psychological, and lifestyle information. This approach avoids the reliability and accuracy issues common with subjective assessments like the PSQI, allowing for objective, quantitative analysis of sleep architecture indices. Additionally, the questionnaire facilitated tracking of lifestyle habits and critical medical histories, enabling a comparative analysis of sleep characteristics and lifestyle differences between AF and control groups.

It is noteworthy that many studies on sleep issues in AF patients rely on data from a specific region or single hospital, potentially introducing bias due to local environmental or cultural conditions. In contrast, this study utilized data from the STAGES database, which includes 20 different collection sites, thereby minimizing regional influence and offering broader coverage, making the conclusions more objective.

Admittedly, the methodology in this study has limitations. First, EEG signals can be influenced by various confounding factors, such as age and genetic predispositions. Second, the sample size in this study was relatively small; smaller sample sizes generally reduce data usability and increase susceptibility to systematic errors and uncertainty. In future research, expanding the sample size by gathering data from additional public databases would improve the reliability, comprehensiveness, and generalizability of the findings.

7. Conclusion and Prospect

Overall sleep quality in AF patients is poor, and increased BMI further exacerbates sleep disturbances, thereby raising disease risk. Risk factor weighting analysis indicates that reduced stage 1 sleep and increased wakefulness are critical indicators for healthcare providers to monitor and address through interventions.

In modern society, the accelerated pace of life and the flourishing nighttime economy offer countless opportunities for late-night activity. However, the adverse effects of chronic sleep deprivation manifest first in major organs. The heart, in particular, requires regular rest to function optimally, and prolonged sleep loss can significantly disrupt cardiac rhythms. Evaluating vital signs during sleep in AF patients is essential for both prevention and treatment of the condition. Specifically, exploring the interactions between the brain and heart during sleep, and understanding how each indicator contributes to restoring physical energy, holds great significance. We hope that more research platforms will enhance data collection and sharing for AF patient sleep data, promoting greater openness and transparency in sample databases. This would enable big data research to deepen our understanding of heart and sleep patterns. Disease itself is not daunting; the key lies in knowledge and understanding. By recognizing health risks from a preventive perspective, we can foster healthier lifestyles.

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