REVIEW ARTICLE

CONTENTS

# Atrial fibrillation as a driver of cognitive decline

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## ABSTRACT

Traditionally, Atrial Fibrillation (AF) has been managed to reduce the risk of stroke, but there is an increasing amount of evidence to suggest that atrial fibrillation is also related to a decline in cognitive function (dementia) and not necessarily related to blood clots or interruptions to blood flow. The aim of this review is to evaluate different mechanisms contributing to the relationship between atrial fibrillation and dementia through hemodynamic and inflammatory pathways. In order to achieve this, a systematic review of longitudinal studies, randomized trials from the past five years, and neuroimaging studies (2013–2026) was conducted to examine the neurocognitive variables relative to atrial fibrillation through the PubMed and Cochrane database. The results of this study suggest that patients with atrial fibrillation are at increased risk of developing dementia independent of thromboembolic events (1.4–2.2 times). Primary risk factors affecting this relationship include silent cerebral infarcts (40% of patients will have these) and chronic cerebral hypoperfusion associated with beat-to-beat variability. Of particular note, patients with atrial fibrillation who achieve early rhythm control using catheter ablation demonstrate a 27%–30% lower risk of cognitive dysfunction as compared to those with rate control. There are numerous and complex physiological changes that can occur from atrial fibrillation that can lead to cognitive decline.

**KEY WORDS:** cognitive dysfunction, dementia, neuroprotection, catheter ablation

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## INTRODUCTION

According to the World Health Organization, it is estimated that the global prevalence of atrial fibrillation (AFib) will reach 12 million in the United States by 2030 [1]. Previously, cognitive dysfunction related to AFib was thought to be exclusively related to thromboembolic strokes, but recent longitudinal studies have uncovered a cognitive gap, referring to AFib patients with no history of stroke and an increased probability of experiencing dementia compared with those in sinus rhythm [2]. This indicates that AFib is likely a chronic and silent source of erosion of the brain's cognitive reserve; therefore, there needs to be a shift from macro-vascular disease as the primary pathological process affecting patients with AFib to micro-vascular integrity.

## AIM

The main aim of this literature review is to classify the various non-embolic modes of contribution that atrial fibrillation has to neurodegenerative disease. Additionally, this study will investigate the measurable neuroprotective effect of the modern treatment modalities utilized on elderly patients, namely catheter ablation and direct oral anticoagulants (DOACs).

## MATERIALS AND METHODS

A systematic literature search of PubMed, Google Scholar and the Cochrane Library was performed between 2013 and 2026. The data extraction for the review was based on randomised controlled trials (RCTs), systematic reviews and neuroimaging studies using 3T/7T MRI as preferred data sources. The inclusion criteria for the studies were limited to a minimum sample size of n = 1,000, or longitudinal follow-up (> 5 years) of sufficiently high-fidelity. The evidence was synthesised according to the pathophysiological drivers, and the relative effectiveness of rhythm-control compared to rate-control strategies of cognitive preservation scores were compared.

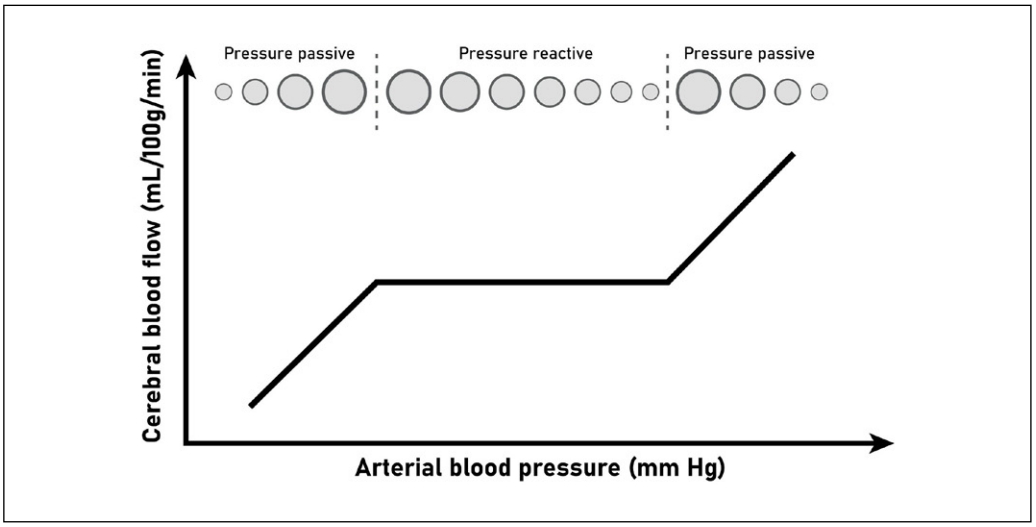


**Fig. 1.** Two critical physiological „barriers" and „flows" that connect heart health to brain function [4]

## REVIEW AND DISCUSSION

### HEMODYNAMIC DISTURBANCE AND INSUFFICIENT BLOOD PERFUSION

The altered variance within the R-R interval of atrial fibrillation is responsible for beat-to-beat variation in stroke volume. Compared with normal sinus rhythm, which has stable pulsatile flow through the arteries into the brain, the chaotic nature of hemodynamics caused by atrial fibrillation overwhelms the ability of the brain to autoregulate blood flow [3]. The rapid ventricular responses associated with atrial fibrillation shorten the duration of diastole, resulting in a significant reduction in stroke volume. Figure 1 represents the two critical physiological "barriers" and "flows" that connect heart health to brain function [4].

When the brain experiences this type of long-term instability (cerebral hypoperfusion), it receives 10-20% less adequately profused (oxygenated) blood than it needs to adequately perform its functions [5]. The current study indicates that chronic cerebral hypoperfusion is especially deleterious to watershed areas of the brain (the areas, throughout the brain, where blood supply comes from at least two different arteries), the white matter of the brain, and the hippocampus, which all have particularly high susceptibilities to oxygen debt [6].

### SILENT CEREBRAL INFARCTS (SCIS) AND MICROBLEEDS

High-resolution MRI studies involving the Swiss-AF cohort have demonstrated that silent brain lesions are found in as many as 40% of patients with atrial fibrillation, or AF [7]. Silent cerebral infarcts (SCIs) are small clusters of tissue death (infarction) that occur in the brain in an asymptomatic fashion and are essentially a cumulative process that decreases the structural integrity of the brain [8]. While anticoagulant therapy may prevent strokes, the results of this study suggest that they do not completely eliminate the risk of suffering microembolic showers—microscopic clots that do not cause any motor deficits, but negatively impact cognitive processing speed [9].

### NEUROINFLAMMATION AND DYSFUNCTION OF THE BLOOD-BRAIN BARRIER (BBB)

AFib is considered a pro-inflammatory state with high levels of C-reactive protein and interleukin-6 present in patients with this condition [10]. This systemic inflammatory process begins to disrupt the tight junctions of the BBB, allowing both neurotoxins and peripheral immune cells to gain access to the brain parenchyma .

It has been suggested through developing theoretical models in 2026 that the loss of the rhythmic pulsating motion of the heart has disrupted the function of the glymphatic system (the mechanism that removes waste from the brain). The rhythmic pumping action created by the arteries allows for the efficient clearance of amyloid-beta/Tau proteins, two of the biological characteristics of Alzheimer's Disease [11, 12].

### NEUROPROTECTION AND THERAPEUTIC INTERVENTIONS

The current debate in the field revolves around the question of whether a restoration of normal cardiac rhythm via catheter ablation can be considered to slow down the rate of dementia progression. In light of data obtained through early rhythm control trials

(EAST-AFNET 4) suggesting a 27-30% decrease in the likelihood of developing dementia following successful intervention [13]. Additionally, when compared to Warfarin, DOACs appear to provide greater neuroprotective properties based upon their ability to reduce the incidence of subclinical microbleeds [14, 15]. However, they are less effective than ablation at rectifying issues related to mechanical hypoperfusion resulting from irregular heart rhythms due to hypoperfusion [16-18].

## CONCLUSIONS

Atrial Fibrillation is an independent and powerful contributor to cognitive erosion. Primary treatment goals should expand beyond preventing stroke to encompass total brain preservation. It is suggested by the present study to achieve this through early and aggressive rhythm control, consistent cognitive assessments via accepted instruments such as the MoCA, and an overall holistic approach to cardiovascular wellness.

## REFERENCES


1. Koh YH, Lew LZW, Franke KB, et al. Predictive role of atrial fibrillation in cognitive decline: a systematic review and meta-analysis of 2.8 million individuals. EP Europace. 2022 Jan 21;24(8):1229–39. doi: 10.1093/europace/euac003. DOI
2. Zhai Y, Hu F, Yuan L, et al. Atrial fibrillation increases the risk of all-cause dementia, Alzheimer's disease, and vascular dementia: A cohort study of 373, 415 participants in the UK Biobank. J Affect Disord. 2024;351:323-330. doi:10.1016/j.jad.2024.01.224. DOI
3. Kühne M, Krisai P, Coslovsky M, et al. Silent brain infarcts impact on cognitive function in atrial fibrillation. Eur Heart J. 2022;43(22):2127-2135. doi:10.1093/eurheartj/ehac020. DOI
4. Dimitrios Varrias, Tinatin Saralidze, Borkowski P, Sumant Pargaonkar, Spanos M, Bazoukis G, et al. Atrial Fibrillation and Dementia: Pathophysiological Mechanisms and Clinical Implications. Biomolecules. 2024 Apr 8;14(4):455–5. doi: 10.3390/biom14040455 DOI
5. Guo J, Zhao X. Atrial fibrillation and risk of cognitive impairment and dementia: Relationships, potential mechanisms, and current therapies. Brain & Heart. 2024 Dec 3;0(0):4702. doi:10.36922/bh.4702. DOI
6. Bretzman JP, Tseng AS, Graff-Radford J, et al. Silent cerebral infarcts in patients with atrial fibrillation: Clinical implications of an imaging-adjusted CHA2DS2-VAScscore. Cardiol J. 2022;29(5):766-772. doi:10.5603/CJ.a2022.0055. DOI
7. Krisai P, Aeschbacher S, Coslovsky M, et al. Ischemic brain infarcts, white matter hyperintensities, and cognitive impairment are increased in patients with Atrial Fibrillation. Commun Med (Lond). 2026;6(1):120. doi: 10.1038/s43856-026-01389-w DOI
8. Hennings E, Bhend K, Paladini RE, et al. Brain lesions and cognitive decline in patients with atrial fibrillation. EP Europace. 2023;25(Suppl.1):euad122.050. doi: 10.1093/europace/euad122.050. DOI
9. Carbone G, Ercolano E, Bencivenga L, et al. Atrial fibrillation and dementia: focus on shared pathophysiological mechanisms and therapeutic implications. J Am Med Dir Assoc. 2024;25(3):465-469. doi:10.1016/j.jamda.2024.01.010. DOI
10. Krisai P, Eberl M, Coslovsky M, et al. Biomarker and cognitive decline in atrial fibrillation: a prospective cohort study. Sci Rep. 2025;15(1):12921. doi:10.1038/s41598-025-89800-9. DOI
11. Huang LJ, Chen JS, Song YZ, Chang P. Atrial fibrillation and cognitive impairment: mechanisms, influencing factors, and prospects. Front Cardiovasc Medic. 2025 Feb 6;12. doi:10.3389/fcvm.2025.1527802. DOI
12. He X, Xiao H, Guo H, et al. Atrial fibrillation-related ischemic stroke and cognitive impairment: Research progress on the characteristics and pathogenesis. Brain Res Bull. 2025;227:111392. doi:10.1016/j.brainresbull.2025.111392. DOI
13. Petersen M, Chevalier C, Naegele FL, et al. Mapping the interplay of atrial fibrillation, brain structure, and cognitive dysfunction. Alzheimers Dement. 2024 Jun 5;20(7):4512–26. doi:10.1002/alz.13870. DOI
14. Agarwal A, Mostafa MA, Ahmad MI, Soliman EZ. Exploring the Link between Anticoagulation, Cognitive Impairment and Dementia in Atrial Fibrillation: A Systematic Review. J Clin Med. 2024;13(8):2418. doi: 10.3390/jcm13082418. DOI
15. Fong, K, Chan, Y, Wang, Y. et al. Dementia Risk of Direct Oral Anticoagulants Versus Warfarin for Atrial Fibrillation: Systematic Review and Meta-Analysis. JACC: Asia. 2023 Oct, 3 (5) 776–786. doi:10.1016/j.jacasi.2023.07.012. DOI
16. Sagris D, Ntaios G, Buckley BJR, et al. Direct oral anticoagulants are associated with lower risk of dementia in patients with atrial fibrillation. Eur J Intern Med. 2024 Mar;121:114–20. doi:10.1016/j.ejim.2023.10.033. DOI
17. Lin KJ, Singer DE, Bykov K, et al. Comparative Effectiveness and Safety of Oral Anticoagulants by Dementia Status in Older Patients With Atrial Fibrillation. JAMA Netw Open. 2023;6(3):e234086. doi: 10.1001/jamanetworkopen.2023.40866. DOI
18. Li GY, Chen YY, Lin YJ, et al. Ablation of atrial fibrillation and dementia risk reduction during long-term follow-up: a nationwide population-based study. Europace. 2023;25(5):euad109. doi: 10.1093/europace/euad109. DOI


## CONFLICT OF INTEREST

The Authors declare no conflict of interest

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