Atrial Fibrillation and Dementia

Atrial fibrillation (AF) is the most common cardiac arrhythmia. As the global population ages, the prevalence of both AF and dementia is expected to rise substantially. Beyond stroke, growing evidence suggests that AF contributes to cognitive decline through multiple mechanisms, including different cerebral lesions, hypoperfusion, inflammation, hypercoagulable state, and brain atrophy. This review summarizes current evidence on AF-related cognitive impairment, explores underlying pathomechanisms, and identifies potential treatment strategies to prevent or delay dementia in patients with AF.

Sujeena Balasundaram MD[1,2], Viviane Herren MD[1,2], Philipp Krisai MD[1,2], Christine S. Zuern MD[1,2], Stefanie Aeschbacher PhD[1,2], Michael Coslovsky PhD[1,3], Leo H. Bonati MD[4], David Conen MD MPH[5], Felix Mahfoud MD[1,2], Christian Sticherling MD[1,2], Stefan Osswald MD[1,2], Michael Kühne MD[1,2]

ABSTRACT

Atrial fibrillation (AF) and dementia are increasingly prevalent diseases in the aging population worldwide. An association between AF, cognitive impairment, and dementia, independent of clinical stroke, has been reported across multiple studies. The underlying mechanisms behind this link are proposed to be multifactorial, including silent cerebral infarcts, cerebral microbleeds, cerebral hypoperfusion, inflammation, and brain atrophy. The impact of oral anticoagulation, rhythm and rate control, and anti-inflammatory strategies targeting these mechanisms on cognitive decline remains unclear. The aim of this narrative review is to summarize the current evidence on AF-related cognitive impairment, explore the proposed pathophysiological mechanisms, and identify potential treatment strategies to prevent or delay dementia in AF patients. Large-scale randomized controlled trials with cognitive outcomes as primary endpoints are needed to determine whether early intervention after AF diagnosis can mitigate the risk of cognitive decline. Further studies on biomarker-based risk stratification, integration of imaging, and artificial intelligence may help identify AF patients at higher risk of cognitive decline and guide future preventive strategies.

Keywords: Atrial fibrillation, dementia, cognitive impairment, cognitive decline

Introduction

Atrial fibrillation (AF) is the most common cardiac arrhythmia. Growing evidence suggests an association between AF and dementia, independent of clinical stroke (1,2). However, whether this relationship is causal or confounded by shared risk factors of AF and dementia remains uncertain. As the global health burden of both conditions rises, understanding the link between AF and dementia has important clinical implications. In Europe, AF prevalence among adults aged >55 years is expected to increase from approximately 9 million in 2010 to 14 million by 2060 (3,4). Similarly, global dementia prevalence is estimated to increase from 57 million in 2019 to 153 million by 2050 (5).

Establishing a causal link of between AF and dementia is challenging, due to the shared risk factors, including advanced age, hypertension, hyperlipidemia, diabetes, chronic kidney disease, coronary artery disease, heart failure, sleep apnea, physical inactivity, and excessive alcohol intake (6,7). Many studies are further limited by small sample sizes, short follow-up, and cross-sectional designs. The wide variety of cognitive assessment methods and terms used across studies to define cognitive dysfunction complicates interpretation. However, several mechanisms have been proposed linking AF to cognitive decline, such as cerebral hypoperfusion, cerebral microbleeds, inflammation, hypercoagulable state, and brain atrophy, some of which may represent potential treatment targets. This narrative review is based on a PubMed literature search (last updated April 2026) using keywords including atrial fibrillation combined with cognitive impairment, cognitive decline, dementia, and pathomechanisms. The aim is to provide an overview of the current understanding of the relationship between AF and dementia, explore potential underlying mechanisms, and identify knowledge gaps.

Potential mechanisms underlying the association between atrial fibrillation and dementia

The association between AF and cognitive dysfunction is complex and likely driven by multiple mechanisms *(Figure 2)*. Although cerebral infarction is considered the main contributor, additional mechanisms have been suggested, including cerebral hypoperfusion, microbleeds, inflammation, hypercoagulable state, and brain atrophy (8).

Clinically Overt and Silent Cerebral Infarcts

AF is associated with an increased risk of ischemic stroke, with approximately one-third of such events attributed to

[1]Cardiovascular Research Institute Basel, University Hospital Basel, University of Basel, Basel, Switzerland
[2]Department of Cardiology, University Hospital Basel, University of Basel, Basel, Switzerland
[3]Department of Clinical Research, University of Basel, University Hospital Basel, Basel, Switzerland
[4]Rheinfelden Rehabilitation Clinic, Rheinfelden, Switzerland
[5]Population Health Research Institute, McMaster University, Hamilton, Canada

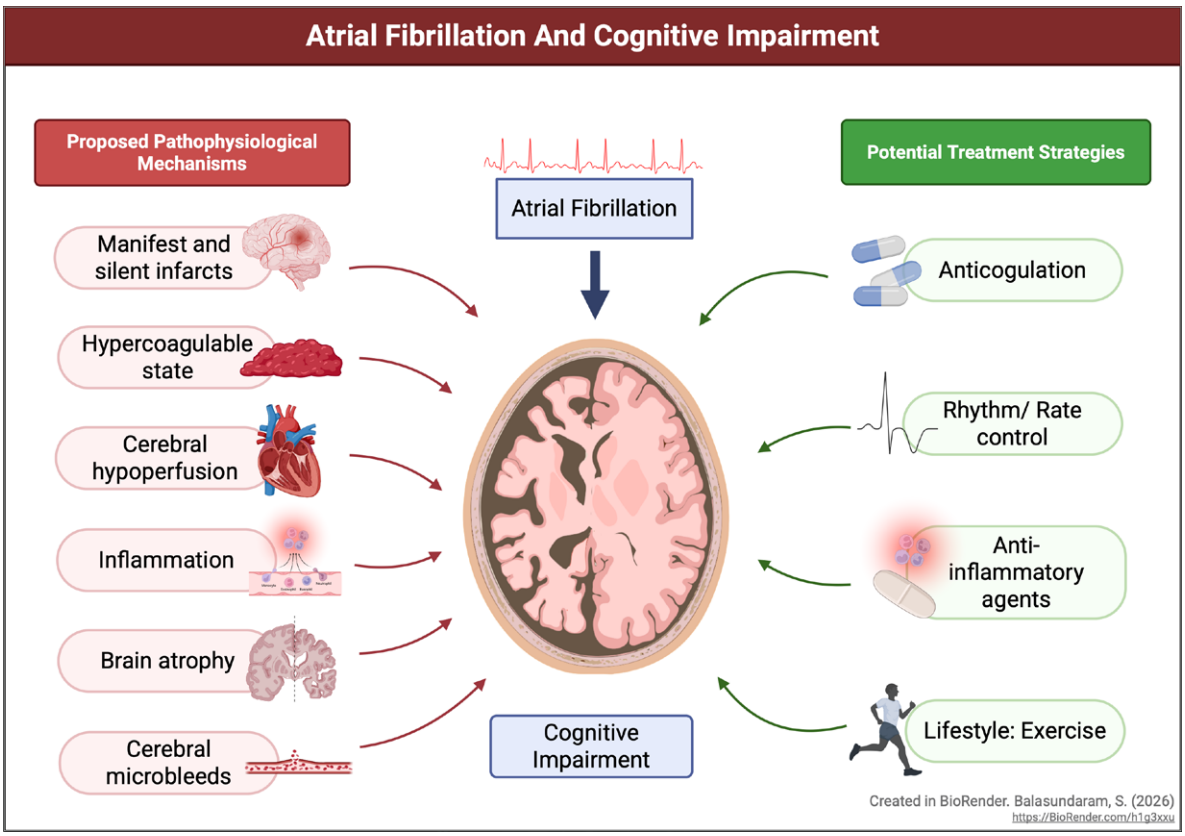

Figure 1. Graphical Abstract

AF (9). Stroke, in turn, increases the risk of dementia, with incidence rates approaching 24% within 3 years after stroke (1,10). This risk is further amplified in patients with AF, which has been associated with a more than two-fold increased likelihood of post-stroke dementia (10). However, the association between AF and cognitive decline is not limited to overt stroke. Silent cerebral infarcts, asymptomatic and detected incidentally on imaging, have been associated with increased risk of dementia and accelerated cognitive decline (11,12). Patients with AF have a high burden of both overt and clinically silent cerebral infarcts. The prospective Swiss Atrial Fibrillation study (Swiss-AF), using systematic brain magnetic resonance imaging (MRI), reported a 22% cerebral infarct rate in a cross-sectional analysis and found that 5.5% of AF patients developed new ischemic infarcts over two years, of which 85% were clinically silent, and occurred predominantly in anticoagulated patients (12,13). Large cortical and noncortical infarcts were associated with poorer cognitive function, with the impact of silent infarcts on cognition comparable to that of overt infarcts (12). These findings suggest that oral anticoagulation alone may not be sufficient to prevent cerebral injury in patients with AF.

Cerebral Microbleeds

Cerebral microbleeds (CMBs), particularly located in lobar regions, have been associated with poorer cognitive performance in patients with AF (14). This relationship persists after adjustment for vascular risk factors and imaging markers of cerebral small vessel disease (CSVD) (15). Patients with AF have a higher prevalence of CMBs, estimated at approximately 28%, compared to individuals without AF (16). Findings from the Swiss-AF cohort suggested that newly occurring CMBs may not directly lead to cognitive decline, as no association between new CMBs and cognitive dysfunction was observed (13). Anticoagulation treatment may further modulate CMB burden. A prospective MRI study reported that the development of new microbleeds at one year in patients with AF was associated with warfarin therapy, whereas no such association was observed with direct oral anticoagulants (DOACs) or antiplatelet therapy (15).

In summary, CMBs appear to represent underlying vascular brain damage rather than a primary driver of cognitive impairment, with ischemic infarcts considered stronger mediators of cognitive decline (12). However, CMBs remain clinically relevant given their association with increased risk of intracerebral hemorrhage (17).

Cerebral Hypoperfusion

AF has been linked to transient or chronic cerebral hypoperfusion, which may contribute to cognitive impairment (18). Beat-to-beat variations and atrioventricular asynchrony eliminate the atrial contribution to left ventricular filling during AF, leading to reduced cardiac output. Although the brain has intrinsic mechanisms to preserve adequate cerebral blood flow across varying blood pressures, several studies have reported decreased cerebral perfusion in patients with

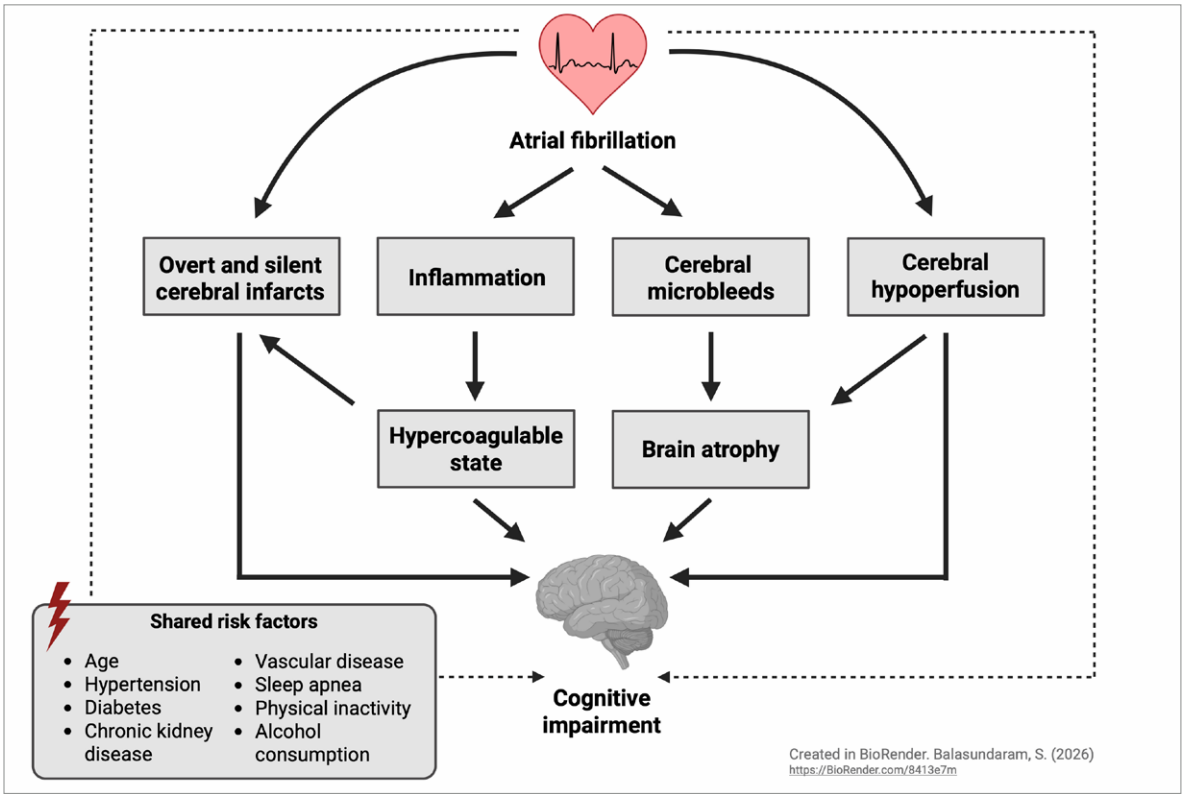

Figure 2. Proposed mechanisms underlying the association between AF and cognitive impairment.

AF (19,20). Computational modelling has shown that higher ventricular rates during AF progressively increase cerebral hypoperfusion in the distal cerebral circulation (21). Over time, hypoperfusion associated with AF may contribute to CSVD, including white matter hyperintensities, although a direct causal relationship remains to be established (22,23).

Inflammation and Hypercoagulability

Both AF and dementia have been linked to a proinflammatory state (24). Inflammation is thought to play an important role in the initiation and maintenance of AF, with markers such as C-reactive protein (CRP) and interleukin-6 (IL-6) elevated during AF compared to sinus rhythm (25,26). The hypercoagulable state promoted by endothelial dysfunction and inflammation increases the risk of thrombus formation. Furthermore, AF may compromise blood-brain barrier integrity, allowing increased passage of circulating inflammatory mediators into the cerebrospinal fluid and potentially leading to deposition of β-amyloid peptide, the primary pathological hallmark of Alzheimer's disease (AD) (25,26). In the Swiss-AF study, inflammation biomarkers, including growth differentiation factor-15, were associated with cognitive decline in AF patients despite high anticoagulation rates, suggesting inflammation as an important non-ischemic mechanism of cognitive decline (27).

Brain Atrophy

The association between AF and reduced brain volume remains inconsistent across studies. Hypoperfusion and microbleeds have been proposed as contributing mechanisms to brain atrophy (1,28). A cross-sectional study of AF patients without a history of stroke found that hippocampal volume was reduced, while total brain volume did not differ between AF patients and controls (29). Hippocampal damage is also observed in AD. AF patients showed poorer memory, learning, attention, and executive function. In contrast, a larger cross-sectional analysis of 4251 participants without dementia found that AF was associated with smaller brain volume, specifically reduced grey and white matter volume, with a stronger association observed in persistent compared with paroxysmal AF (30). The potential of sinus rhythm maintenance to mitigate AF-related brain atrophy remains to be established in future prospective studies.

Cognitive Assessment

Screening for mild cognitive impairment in patients with AF is recommended when concerns arise from patients or family members regarding memory and daily activities. The most commonly used brief screening tools are the Montreal Cognitive Assessment (MoCA) and the Mini-Mental State Examination (MMSE), with additional tools, such as the Trail Making Test, assessing specific domains, including processing speed and mental flexibility (31). While these tools do not confirm a diagnosis of dementia, they help clinicians to identify patients who may need referral to a specialist. In case of abnormal screening, reversible causes of cognitive impairment, such as medications, depression, and sleep apnea should be excluded.

The MoCA assesses multiple cognitive domains *(Figure 3)* (32). The MMSE has lower sensitivity for mild cognitive impairment but higher specificity compared to MoCA (32). In a cross-sectional study, AF patients have been associated with poorer performance in all cognitive domains compared to patients without AF (33). In general, it should be noted that the variety of cognitive screening tools used in AF studies makes interpretation of findings difficult.

Impact of Treatment Strategies

Role of Oral Anticoagulation

Several studies suggested that oral anticoagulation (OAC) may reduce the risk of cognitive decline and dementia in AF patients *(Table 1)* (34–36). Whether DOACs provide superior cognitive protection compared with vitamin K antagonists (VKAs) remains unclear. Evidence from a population-based cohort demonstrated that OAC use was associated with a significantly lower risk of dementia compared with non-OAC use (hazard ratio [HR] 0.59; 95% CI 0.44-0.80; $p < 0.001$) (34). DOAC use was associated with lower dementia incidence compared with both non-OAC use and warfarin use, whereas no significant difference was observed between warfarin and non-OAC use (34). However, a Danish study found no clinically meaningful difference in dementia incidence between patients using DOACs and those taking warfarin (37).

Several knowledge gaps remain, including whether some type of anticoagulation should be prescribed in AF patients based on new risk stratification tools to protect cognitive function, and how to balance potential benefits against bleeding risk. Despite OAC therapy, silent cerebral infarcts continue to occur in AF patients, suggesting that anticoagulation alone may not fully prevent subclinical brain injury (12,13). Overall, current evidence supports a potential protective effect of OAC against cognitive decline.

Role of Rhythm and Rate Control

Growing evidence suggests that AF itself impairs cerebral perfusion independently of embolic events. A large MRI-based observational study found that persistent AF was associated with reduced total cerebral blood flow and brain perfusion compared with paroxysmal AF and sinus rhythm (38). A prospective study showed that successful electrical cardioversion significantly improved cerebral perfusion, while perfusion remained unchanged in those who failed to convert to sinus rhythm (39). Regarding rhythm control, earlier data from the AFFIRM trial found no cognitive benefit of pharmacological rhythm control over rate control, though follow-up was limited to 3.5 years (40). Notably, AFFIRM compared rhythm versus rate control strategies rather than sinus rhythm versus AF, and not all patients in the rhythm control arm were in sinus rhythm. The recently published EAST-AFNET 4 (Early Treatment of Atrial Fibrillation for Stroke Prevention Trial) found that early rhythm control within 12 months of AF diagnosis was associated with a 21% lower risk of adverse cardiovascular outcomes compared with usual care (41). However, cognitive function assessed by the MoCA did not differ significantly between groups after a limited follow-up duration of 2 years. Supporting the cognitive benefit of rhythm control, a recent systematic review and meta-analysis, including 193,830 patients with AF, demonstrated that rhythm control was associated with a 26% lower risk of dementia compared with rate control (42). While the effect of rate control on cognitive outcomes remains unknown, a 10-year cohort study showed that both low (< 50 bpm) and high (> 90 bpm) ventricular rates had a 7-fold increased risk of dementia in the elderly with cognitive impairment, suggesting that adequate ventricular rate control may be relevant for cognitive outcomes (43). In summary, whether rhythm and rate control treatment may reduce dementia risk warrants further investigation.

Role of Catheter Ablation

Compared to antiarrhythmic drug therapy, catheter ablation is more effective in reducing AF burden and maintaining sinus rhythm (44). The effect of ablation on dementia risk is debated, with earlier studies reporting post-ablation cognitive decline and acute brain lesions (45–47). A recent large nationwide Korean cohort, with a median follow-up of 4 years, found that ablation was associated with a lower dementia risk, even after adjusting for clinical confounders (48). This protective effect was evident only in patients with successful ablation, suggesting that sustained sinus rhythm restoration underlies the neuroprotective benefit (48). The Swiss-AF study found no consistent association between pulmonary vein isolation (PVI) and neurocognitive function at 1-year follow-up (49), but larger studies with long-term follow-up are needed.

Role of Anti-Inflammatory Agents

Given the relevance of inflammation in AF-related cognitive impairment, anti-inflammatory strategies represent a potential therapeutic target. A large-scale Taiwanese cohort study of patients with AF found that statin treatment was associated with a lower risk of non-vascular dementia (HR 0.83; 95% CI 0.80-0.86; $p < 0.001$) compared with non-users, with stronger protective effects observed among patients receiving more potent statins and longer treatment duration (50). In a randomized placebo-controlled trial, intensive lipid-lowering therapy with atorvastatin 40 mg and ezetimibe 10 mg was found to reduce cognitive decline in older patients with AF (51). These findings require further validation in randomized trials before statins can be recommended for improved cognitive outcomes in patients with AF. Colchicine and canakinumab have shown anti-inflammatory effects in AF, though their impact on cognitive decline remains to be investigated (52,53).

Role of Lifestyle: Physical Activity and Coffee Consumption

Physical inactivity has been identified as a modifiable risk factor for dementia in the older population (54). The Swiss-

Table 1. Trials with Cognitive Outcomes as Endpoints

Trial and Research Question	Methods	Cognitive Endpoint	Status and Results
Oral anticoagulation			
BRAIN-AF *Does rivaroxaban reduce the risk of stroke/TIA and cognitive decline in AF patients at low risk? (58)*	Inclusion criteria: NVAF, low risk of stroke, age ≥ 30 to ≤ 62 years RCT, double-blind, n = 1235 Groups: rivaroxaban 15 mg vs. standard of care (placebo or ASA 100 mg) Mean follow-up: 3.7 years	Primary: composite of stroke/TIA and cognitive decline (MoCA decreased ≥ 2 points from baseline)	Stopped early for futility: no benefit observed for rivaroxaban compared to placebo in reducing stroke, TIA or cognitive decline
CAF *Does dabigatran reduce cognitive decline and dementia compared to warfarin? (59)*	Inclusion criteria: NVAF, CHA_2DS_2VASc score ≥2, age >65 years RCT, open-label, n = 101 Groups: dabigatran and warfarin Follow-up: 24 months	Primary: Incident dementia (formal diagnosis of dementia by a neurologist) or moderate cognitive decline at 24 months (ADAS, DAD)	Completed: similar risks of cognitive decline and dementia between dabigatran and warfarin, suggesting either strategy is acceptable to mitigate these risks
NOAH *Is edoxaban superior to current therapy in preventing cardiovascular death, stroke or SE in patients with AHRE? (60)*	Inclusion criteria: AHRE (≥ 6 minutes), CHA_2DS_2VASc score ≥ 2, age ≥ 65 years RCT, double-blind, n = 2536 Groups: edoxaban or placebo/ASA Median follow-up: 21 months	Primary: composite of cardiovascular death, stroke, or SE Secondary: change in cognitive function (MoCA)	Stopped early for safety and informal futility assessment: edoxaban did not significantly reduce the risk for primary endpoint but increased the risk of death or major bleeding
OCEAN *Should OAC be continued after successful AF ablation to prevent stroke? (61)*	Inclusion criteria: absence of AF recurrence ≥ 12 months after AF ablation procedure, CHA_2DS_2VASc score ≥ 1 RCT, open-label, n = 1284 Groups: rivaroxaban or aspirin Follow-up: ≥ 3 years	Primary: stroke, SE, or new covert embolic stroke (associated with increased risk for cognitive decline)	Completed: rivaroxaban did not significantly reduce the risk for primary endpoint compared to aspirin, higher rates of minor bleeding were observed with rivaroxaban
DaRe2THINK *Does DOAC therapy reduce cognitive decline in younger AF patients at low or intermediate risk for stroke? (62)*	Inclusion criteria: low or intermediate risk for stroke, age ≥55 years to ≤ 73 years RCT, open-label, n = 3000 Groups: DOAC vs. usual care Follow-up: 5 years, longer-term outcomes (cognitive function) reassessed at 10 years	Primary: time to first event of cardiovascular mortality, ischemic cerebrovascular events, all thromboembolic events Secondary: change in cognitive function (fluid intelligence test, trail making test, symbol digit substitution test)	Ongoing: expected completion 2031 (NCT04700826)
Swiss-AF *What is the relationship between AF, structural alterations on brain MRI and cognitive decline? (63)*	Inclusion criteria: AF patients ≥ 65 years Prospective national multi-center cohort (14 centres), n = 2415 Yearly follow-up visits; brain MRI at baseline, FU2, and FU7	Primary: stroke, SE, death, bleeding, hospitalization for heart failure, myocardial infarction, and any unplanned hospitalization Cognition: cognitive decline (MoCA, TMT SFT, DSST) and structural brain damage (MRI)	Ongoing, sub-study results available: 5.5% had a new brain infarct on MRI after 2 years, silent brain infarcts occurred in AF patients despite high anticoagulation rates, overt and silent brain infarcts had a similar impact on cognitive decline (13)
Rhythm control			
EAST-AFNET 4 *Does early rhythm control reduce AF-related complications? (41)*	Inclusion criteria: AF diagnosed ≤ 12 months, age ≥ 75 years RCT, open-label, n = 2789 Groups: early rhythm control vs. usual care Follow-up: 5 years	Primary: A composite of cardiovascular death, stroke, and hospitalization due to worsening of heart failure or due to acute coronary syndrome. Secondary: change in cognitive function (MoCA) at 2 years	Completed: early rhythm control was associated with a lower risk of adverse cardiovascular outcomes compared to usual care, no significant difference in MoCA scores between groups

Table 1. Trials with Cognitive Outcomes as Endpoints

AFCOG *Do acute AF episodes cause measurable changes in cognitive function?*	Inclusion criteria: AF patients in active episode of AF undergoing rhythm conversion Prospective cohort, n = 600	Primary: change in cognitive function during AF episodes (CANTAB)	Ongoing: expected completion 2027 (NCT04033510)
AF ablation			
FALCON *Does catheter ablation of persistent AF improve cognitive function in patients with mild cognitive impairment?*	Inclusion criteria: persistent AF, MoCA 18–25 points (MCI), age 60–80 years RCT, single-blind, n = 120 Groups: AF ablation + antiarrhythmic drugs vs. antiarrhythmic drugs alone Follow-up: 6–12 months	Primary: change in cognitive function (MoCA) at 6 months	Ongoing: expected completion 2027 (NCT05790707)
EASThigh-AFNET 11 *Does early AF ablation reduce cardiovascular events in patients with high comorbidity burden?*	Inclusion criteria: CHA_2DS_2VASc score ≥ 4, age ≥ 18 years RCT, single-blind, n 2312 Groups: early atrial ablation vs. usual care Follow-up: mean 4 years	Primary: composite of cardiovascular death, stroke, or hospitalization for worsening of heart failure Secondary: change in cognitive function (MoCA)	Ongoing: expected completion 2030 (NCT06324188)

Abbreviations: ADAS, Alzheimer's Disease Assessment Scale; AF, atrial fibrillation; AFCOG, Acute Cognitive Changes During Atrial Fibrillation Episodes; AHRE, atrial high rate episodes; ASA, acetylsalicylic acid; BRAIN-AF, Blinded Randomized Trial of Anticoagulation to Prevent Ischemic Stroke and Neurocognitive Impairment in Atrial Fibrillation; CANTAB, Cambridge Neuropsychological Test Automated Battery; CAF, Cognitive Decline and Dementia in Patients With Non-Valvular Atrial Fibrillation; CHA2DS2VASc score, congestive heart failure (1 point), hypertension (1 point), age ≥75 (2 points), diabetes (1 point), previous stroke or transient ischemic attack (2 points), vascular disease (1 point), age 65 to 74 (1 point), and sex (female; 1 point); DAD, Disability Assessment for Dementia; DOAC, direct oral anticoagulant; DSST, digit symbol substitution test; EAST-AFNET 4, Early Treatment of Atrial Fibrillation for Stroke Prevention Trial; EASThigh-AFNET 11, Early Atrial Fibrillation Ablation for Stroke Prevention in Patients With High Comorbidity Burden; FALCON, Effect of Ablation of Persistent Atrial Fibrillation on Cognitive Function in Individuals with Mild Cognitive Impairment; MCI, mild cognitive impairment; MRI, magnetic resonance imaging; MoCA, Montreal Cognitive Assessment; NOAH, Non-Vitamin K Antagonist Oral Anticoagulants in Patients With Atrial High Rate Episodes; NVAF, nonvalvular atrial fibrillation; OAC, oral anticoagulation; OCEAN, Optimal Anticoagulation for Higher Risk Patients Post-Catheter Ablation for Atrial Fibrillation Trial; RCT, randomized controlled trial; SE, systemic embolism; SFT, semantic fluency test; TIA, transient ischemic attack; TMT, trail making test.

AF cohort study investigated the association between physical activity and brain health in 1,490 patients with AF (55). Regular exercise (at least once weekly) was associated with a lower prevalence of ischemic infarcts and moderate to severe white matter hyperintensities, as well as higher global cognitive performance. Furthermore, increasing weekly physical activity was associated with higher brain volume. While prospective studies are needed to establish causality, these findings suggest that patients with AF should be advised to remain physically active. Beyond physical activity, higher coffee consumption has been associated with better cognitive performance and reduced inflammatory markers, including IL-6 and hs-CRP (56). Contrary to the conventional wisdom that caffeinated coffee is proarrhythmic, the randomized DECAF trial (Does Eliminating Coffee Avoid Fibrillation?) found that coffee consumption following successful cardioversion was associated with a lower risk of AF recurrence compared with coffee abstinence (57). Whether the effects of coffee consumption translate into long-term cognitive benefits warrants further investigation.

Future Investigations

Several observational studies have found an association between AF and cognitive impairment. Given that AF and dementia share several risk factors, a complete understanding of the relationship still requires further investigation in long-term and larger prospective cohorts to determine whether the link is causal. The proposed underlying mechanisms, including cerebral hypoperfusion, inflammation, prothrombotic state, and microbleeds, are potential targets for delaying or preventing cognitive impairment in patients with AF. The impact of anti-inflammatory agents, rhythm, and rate control on dementia onset and progression in AF patients should be examined in randomized controlled trials. BRAIN-AF, the largest randomized trial of OAC for cognitive decline prevention, was recently stopped early due to futility, with rivaroxaban showing no benefit over placebo in reducing cognitive decline, stroke, or transient ischemic attack (TIA) in younger AF patients at low stroke risk (58). These findings suggest that non-ischemic mechanisms are involved in this age group and that additional treatment targets warrant investigation (58).

Beyond anticoagulation, the optimal blood pressure target for cognitive protection in AF remains uncertain. A cohort study of midlife AF patients reported a U-shaped association, with both low and elevated blood pressure levels associated with increased dementia risk (64). However, the Swiss-AF cohort study found no consistent association between blood pressure and cognitive decline over 6 years of follow-up (65).

Biomarker-based prediction of cognitive decline in patients with AF is a growing area of research interest. The development of cognitive risk prediction scores, potentially

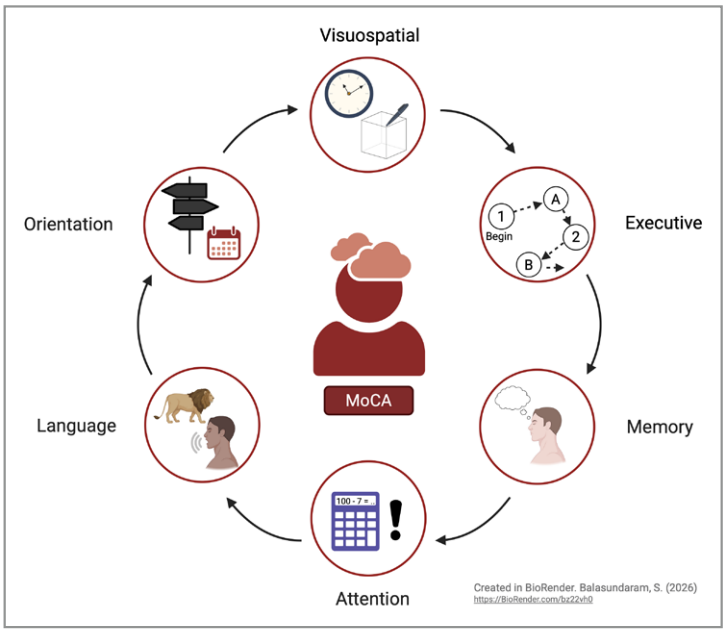

Figure 3. Cognitive domains assessed by the Montreal Cognitive Assessment (MoCA)

integrating clinical variables with biomarkers, imaging, and artificial intelligence, may help identify AF patients at risk of cognitive decline.

Conclusion

AF and dementia are prevalent and expected to further increase, in part due to population aging. A consistent association between AF and dementia has been reported, although a causal relationship remains to be established. The underlying mechanisms behind this link are multifactorial. Incorporating cognitive decline as an endpoint in future AF studies is crucial to identify effective preventive treatment strategies. Randomized trials are needed to determine whether early intervention after AF diagnosis can reduce the risk of cognitive decline and dementia. □

Corresponding author:
Michael Kühne, MD
University Hospital Basel
Petersgraben 4
4031 Basel
Switzerland
E-Mail: Michael.Kuehne@usb.ch

Conflicts of interest:
M.K. reports grants from the Swiss National Science Foundation (Grant numbers 33CS30_148474, 33CS30_177520, 32473B_176178, 32003B_197524, and 33IC30_229733), the Swiss Heart Foundation, the Foundation for Cardiovascular Research Basel and the University of Basel, grants from Abbott, Bayer, BMS, Boston Scientific, Daiichi Sankyo, Medtronic, Pfizer, Roche. Royalties from Springer (ECG book). P.K. reports speaker fees from BMS/Pfizer. Grants from the Swiss National Science Foundation, Swiss Heart Foundation, Foundation for Cardiovascular Research Basel, Machaon Foundation. C.S.Z. reports grants from Freiwillige Akademische Gesellschaft Basel and speaker honoraria/consulting fees from AstraZeneca, Boehringer Ingelheim Medtronic, Pfizer, and Vifor Pharma. L.H.B. received grants from the Swiss National Science Foundation (PBBSB-116873,33CM30-124119, 32003B-156658,32003B-197524; Berne, Switzerland), the Swiss Heart Foundation (Berne,Switzerland) and the University of Basel (Basel, Switzerland). He received an unrestricted research grant from AstraZeneca, and consultancy or advisory board fees or speaker's honoraria from Amgen, Bayer, Bristol-Myers Squibb and Claret Medical, and travelgrants from AstraZeneca and Bayer. D.C. received advisory board fees from Abbott. F.M. has been supported by Deutsche Forschungsgemeinschaft (SFB TRR219, Project-ID 322900939), and Deutsche Herzstiftung. FM's previous institution, Saarland University, received scientific support from Ablative Solutions, Medtronic, and ReCor Medical. Until May, 2024, FM has received speaker honoraria or consulting fees from Ablative Solutions, AstraZeneca, Inari, Medtronic, Merck, Novartis, Philips, and ReCor Medical. C.S. Member of Medtronic Advisory Board Europe and Boston Scientific Advisory Board Europe, received educational grants from Biosense Webster and Biotronik and a research grant from the European Union's FP7 program and Biosense Webster, and lecture and consulting fees from Abbott, Medtronic, Biosense Webster, Boston Scientific, Microport, and Biotronik, all outside the submitted work. S.O. received research grants from the Swiss Heart Foundation, the Foundation for Cardiovascular Research Basel, the Swiss National Science Foundation and from Roche. He has also received educational and speaker office grants from Roche, Bayer, Novartis, Sanofi, AstraZeneca, Daiichi-Sankyo and Pfizer. The remaining authors have no disclosures.

References in the online version of the article at www.ch-cardiology.ch