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PERSPECTIVE ARTICLE

Prophylactic surgical left atrial appendage exclusion in young patients undergoing open-heart surgery for mitral valve repair: A brain-heart preventive perspective

Alfred Ibrahimi[1†], Romina Teliti[2†], Verona Beka[2], Kolja Sievert[3], Horst Sievert[3], and Jacob Zeitani[4*]

[1]Department of Anesthesiology and Reanimation, Mother Teresa University, Tirana, Albania

[2]Department of Cardiology, German Hospital, Tirana, Albania

[3]Cardiovascular Center Frankfurt, Frankfurt, Germany

[4]Department of Neurosciences and Rehabilitation, University of Ferrara, Ferrara, Italy

Abstract

Cardioembolic stroke remains one of the most severe manifestations of cardiovascular disease, with atrial fibrillation (AF) responsible for a large proportion of ischemic cerebrovascular events. Stroke prevention strategies are currently dominated by oral anticoagulation guided by CHA_2DS_2-VASc scores, which emphasize age and comorbidities and therefore have limited applicability in younger patients with low conventional risk profiles. Although bleeding risk is routinely assessed using tools such as HAS-BLED, lifelong anticoagulation may still confer a substantial cumulative hemorrhagic burden over decades of treatment. The left atrial appendage (LAA) represents the primary anatomical source of thromboembolism in AF, yet current guidelines largely restrict surgical or percutaneous LAA exclusion to patients with AF who have contraindications to anticoagulation, reflecting a predominantly reactive approach. This Perspective advocates reconsideration of prophylactic surgical LAA exclusion at the time of open mitral valve repair in younger patients. Although early mitral repair limits atrial remodeling, it does not abolish lifetime AF risk, and post-operative or late-onset AF remains frequent. Moreover, residual or recurrent mitral regurgitation promotes progressive atrial dilation and fibrosis, increasing long-term thromboembolic risk even in patients initially in sinus rhythm. By integrating atrial remodeling, LAA morphology and function, residual mitral pathology, and the limitations of lifelong anticoagulation, a selective preventive framework emerges. An anatomical brain-heart prevention strategy may reduce lifetime cerebrovascular risk and improve long-term outcomes. While prospective data are needed, existing evidence supports individualized discussion of prophylactic LAA exclusion during mitral valve repair in selected young patients with structural atrial disease.

Keywords: Cardioembolic stroke; Left atrial appendage exclusion; Mitral valve repair; Atrial remodeling; Stroke prevention

†These authors contributed equally to this work.

***Corresponding author:**
Jacob Zeitani
(Jacob.zeitani@unife.it)

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1. Introduction

Cardioembolic stroke represents a devastating intersection of heart and brain disease. While atrial fibrillation (AF) has long been emphasized as the dominant cardiac substrate for thromboembolic events, emerging evidence indicates that structural and functional atrial abnormalities contribute substantially to stroke risk, even in the absence of clinically documented AF.[1,2] Progressive left atrial (LA) remodeling, dilation, and dysfunction create a prothrombotic milieu, highlighting the need for preventive strategies targeting the anatomical origins of embolism. AF is common and strongly associated with stroke, conferring more than a five-fold increased risk of ischemic cerebral events and profoundly affecting long-term cognition and functional independence.[3,4] In AF, thrombi that embolize to the cerebral circulation originate overwhelmingly from the LA appendage (LAA), comprising over 90% of intracardiac thrombi in non-valvular AF populations.[5] This anatomical link supports the rationale for LAA-targeted exclusion strategies.

2. Limitations of current risk stratification and anticoagulation strategies

Current guidelines for LAA management focus predominantly on patients with documented AF and elevated stroke risk according to CHA_2DS_2-VASc scoring.[4,6-8] In line with contemporary AF guidelines, patient sex has been removed as an independent risk modifier, shifting emphasis toward age and vascular comorbidities, thereby further reducing the sensitivity of risk prediction in younger patients with structural atrial disease. These risk models heavily weight age and comorbidities and therefore perform well in older populations but systematically underestimate long-term cerebrovascular risk in younger patients with structural atrial disease. In such individuals, a low CHA_2DS_2-VA score may falsely convey reassurance, despite the presence of LA enlargement, remodeling, or valvular pathology that predisposes to future AF and thromboembolism.

Oral anticoagulants (OACs), when prescribed according to these scores, effectively reduce ischemic stroke risk but simultaneously introduce a clinically meaningful risk of major bleeding.[9,10] Assessment of bleeding risk is therefore integral to balanced clinical decision-making regarding long-term anticoagulation. The HAS-BLED score is a validated clinical tool developed from the Euro Heart Survey cohort to estimate the 1-year risk of major bleeding in patients with AF. It incorporates common clinical parameters—such as hypertension, abnormal renal and/or liver function, prior stroke, bleeding history or predisposition, labile international normalized ratio, elderly age (≥65 years), and concomitant drugs or alcohol use, each contributing one point to the total score.[4,7] A HAS-BLED score ≥3 identifies patients at increased bleeding risk; however, this classification does not represent a contraindication to anticoagulation. Rather, it highlights the need for closer surveillance and correction of modifiable risk factors, such as uncontrolled blood pressure, interacting medications, suboptimal international normalized ratio control, and excessive alcohol intake.

While the HAS-BLED score was designed to estimate short-term bleeding risk, its implications are particularly relevant in younger patients who may otherwise face decades of continuous anticoagulation. Even with contemporary direct OACs, which offer improved safety compared with Vitamin K antagonists, major bleeding, especially gastrointestinal and intracranial, remains a significant limitation to lifelong therapy.[11,12] Over time, cumulative bleeding events, treatment interruptions, medication non-adherence, and patient intolerance may compromise effective stroke prevention. Thus, in younger individuals with low CHA_2DS_2-VASc scores but underlying atrial remodeling or valvular disease, the apparent balance between thromboembolic and bleeding risk may shift unfavorably when considered over a lifetime horizon rather than a single-year estimate.

When used alongside stroke-risk assessment tools, the HAS-BLED score supports an individualized, longitudinal approach to cerebrovascular prevention. In this context, strategies that mechanically address the primary anatomical source of thromboembolism, such as surgical LAA exclusion performed during otherwise indicated open-heart procedures, gain conceptual relevance. By potentially reducing or eliminating future dependence on systemic anticoagulation, prophylactic LAA exclusion may mitigate cumulative bleeding risk while preserving long-term protection against cardioembolic stroke, particularly in carefully selected young patients undergoing mitral valve repair.

3. Surgical and percutaneous LA appendage exclusion: Techniques and preventive potential

Percutaneous LAA occlusion (LAAO) devices, such as Watchman and Amulet, offer alternative strategies for patients with AF who cannot tolerate OAC. Randomized clinical trials demonstrate non-inferiority to warfarin in stroke prevention; however, these interventions are inherently reactive and typically deployed after AF onset or once formal risk indication is established.[13] Procedural complications, device-related thrombus, and residual peri-

device leaks further limit their applicability as preventive strategies.[13,14] While transcatheter LAAO represents an important therapeutic option in selected high-risk or anticoagulation-intolerant patients, it does not address the concept of early, upstream prevention during already indicated open-heart surgery. In contrast, surgical LAA exclusion performed concomitantly with other cardiac procedures represents a unique preventive opportunity.

Several surgical techniques for LAAO are available during open-heart surgery, each with distinct advantages and limitations that may influence long-term efficacy. Traditional suture ligation, a simple and rapid procedure, is widely used but has been associated with variable rates of incomplete closure and residual LAA stumps, which may remain thrombogenic. Recently, Yuan *et al.*[15] evaluated prophylactic LAA closure in patients undergoing valvular surgery with CHA_2DS_2-VASc scores ≥2 but without preoperative AF and reported no significant reduction in a composite endpoint of ischemic stroke, transient ischemic attack, and cardiovascular mortality at 1-year follow-up. Importantly, the surgical strategy predominantly relied on LAA ligation followed by appendage amputation and suturing of the residual stump over the ligation site. While effective in many cases, ligation-based techniques are inherently associated with a risk of incomplete exclusion and residual LAA stump formation, which may preserve thrombogenic potential if not fully eliminated. In addition, the relatively short follow-up duration may be insufficient to capture the cumulative cerebrovascular benefit of anatomical exclusion, particularly in younger patients, for whom stroke risk accumulates over decades rather than within the 1st post-operative year.

Surgical resection of the appendage with subsequent atrial wall closure offers direct removal of the embolic source and may provide more definitive exclusion, although it requires meticulous technique to avoid bleeding and preserve atrial geometry (Figure 1). Stapler-based devices have emerged as efficient alternatives, allowing rapid exclusion or resection with reproducible results; however, concerns persist regarding residual stumps, tissue necrosis, and long-term durability depending on appendage anatomy. Contemporary epicardial clip devices, when applied under direct visualization, have demonstrated high rates of complete closure with minimal residual flow, offering a standardized and reproducible approach in experienced hands.

Regardless of technique, complete and durable exclusion is paramount, as incomplete LAA closure may negate protective effects and perpetuate thromboembolic risk. Accordingly, the selection of the surgical strategy should be individualized, accounting for LAA morphology,

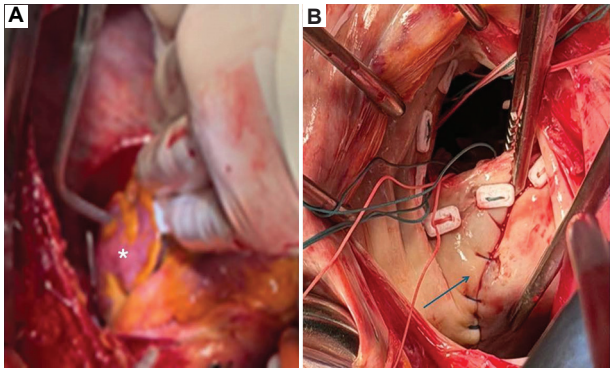


Figure 1. Intraoperative view of the left atrial appendage (LAA). (A) External surgical view of the LAA before resection. (B) Left atrial view after complete LAA exclusion, demonstrating closure of the LAA orifice; the suture line is indicated by the arrow. Image is original from authors.

surgical exposure, concomitant procedures, and operator experience, with the overarching goal of achieving reliable, lifelong cerebrovascular protection. The preventive value of mechanical LAA exclusion is further supported by contemporary evidence. Baudo *et al.*[16] demonstrated that LAA exclusion provides sustained protection against ischemic stroke, reinforcing the central role of the appendage as a therapeutic target beyond pharmacological anticoagulation alone. Similarly, the LAAO study III study demonstrated that concomitant LAA exclusion in patients with AF reduces ischemic stroke without increasing perioperative morbidity.[17] These findings underscore the additive preventive value of mechanical exclusion in surgical populations.

Despite these advantages, anatomical variability of the appendage represents an important limitation of surgical LAA exclusion. Complex LAA morphologies, particularly multilobed, cauliflower-type, or deeply trabeculated appendages, may pose technical challenges to complete exclusion, increasing the risk of residual stumps or incomplete closure even with surgical techniques. In such cases, appendage exposure, orientation, and depth may limit the feasibility of stapling or resection without compromising atrial geometry or hemostasis. These considerations underscore the importance of preoperative imaging, intraoperative assessment, and individualized selection of the exclusion technique, particularly when a prophylactic strategy is contemplated.

Accordingly, surgical and percutaneous LAA exclusion should be viewed as complementary rather than competing strategies, each addressing distinct stages along the atrial disease continuum. Percutaneous LAAO has been shown to reduce major bleeding events compared with long-term oral anticoagulation in patients with AF, particularly with respect to hemorrhagic stroke and late

bleeding complications.[8] Randomized clinical trials and extended follow-up studies demonstrate that, after the initial procedural period, LAAO permits discontinuation of systemic anticoagulation in most patients, thereby mitigating cumulative bleeding risk. This advantage is particularly relevant in elderly or high-bleeding-risk populations, for whom prolonged anticoagulant therapy poses substantial hazards.[10-12] However, percutaneous approaches are typically implemented after the onset of AF and therefore do not address early structural atrial disease or the lifetime cerebrovascular risk of younger patients undergoing cardiac surgery.

4. The LA appendage: Physiology, morphology, and thromboembolic risk

The LAA has physiological roles in sinus rhythm, contributing to LA compliance, reservoir function, and atrial natriuretic peptide secretion.[18,19] In the context of LA enlargement, high-pressure load, or volume overload, as seen in mitral regurgitation (MR) and other valvular lesions, the functional contribution of the LAA diminishes while its thrombogenic potential rises.[20,21] Accordingly, the appendage shifts from a functional reservoir to a thrombus-prone site, suggesting a window for prophylactic intervention. The risk of thrombus formation and subsequent embolization is further modulated by the morphological variability of the LAA. Differences in appendage shape influence blood flow velocity, regions of stasis, and shear stress, thereby affecting thrombogenic potential. Di Biase *et al.*[22] described four principal LAA morphologies with distinct associations with thromboembolic risk. The chicken wing morphology is associated with the lowest embolic risk, whereas increasingly complex architectures, windsock, cactus, and particularly cauliflower morphology, are associated with progressive blood stasis, spontaneous echo contrast, and higher thromboembolic risk. These morphological considerations reinforce the concept that structural features of the LAA, independent of rhythm status, meaningfully contribute to embolic risk.

5. MR, atrial remodeling, and lifetime cerebrovascular risk

MR, particularly degenerative MR (DMR), provides a paradigm for early atrial remodeling. Chronic volume overload leads to progressive LA dilation, increased wall stress, and fibrosis, predisposing to electrical and mechanical dysfunction.[23] Evidence from the MR International Database registry highlights the prognostic impact of AF in DMR. Among 2,425 patients (mean age 67 ± 13 years), AF—present in roughly one-third—was independently associated with long-term mortality, even after mitral valve repair (10-year post-surgical survival: sinus rhythm 82%, paroxysmal AF 70%, persistent AF 57%; $p<0.0001$).[24] In degenerative pathology, lesion complexity, such as anterior leaflet prolapse, influences the risk of residual or recurrent regurgitation and long-term outcomes, as residual MR may contribute to progressive LA enlargement over time.[25] Surgical planning must integrate atrial and ventricular dimensions, remodeling, lifestyle, projected thromboembolic risk (CHA_2DS_2-VASc), and lesion-specific repair risk to guide the potential addition of prophylactic LAAO, optimizing functional and prognostic results.

Under the current paradigm, prophylactic surgical LAA exclusion is rarely considered in younger patients without AF and low traditional, age-weighted scores.[4] These scores systematically underestimate lifetime cerebrovascular risk in young populations. Stroke is a cumulative process, and silent infarctions contribute to cognitive decline and irreversible brain injury.[26] Integrating LA size, remodeling indices, and structural atrial pathology into risk assessment enables a more mechanistic, lifetime-oriented approach.[18]

6. Early mitral valve repair and the persistent risk of AF

Critics of prophylactic LAA exclusion cite physiological LAA roles and potential post-operative arrhythmias.[26,27] However, when performed by experienced surgeons during indicated cardiac procedures, incremental risk is minimal and does not substantially affect outcomes.[16] Compared with the currently available percutaneous devices, surgical exclusion offers direct visualization, reliable closure, and minimal residual leaks, maximizing preventive potential.[13,14,16] Contemporary management of DMR increasingly favors early surgical repair once repair is feasible, even before symptom onset.[28] Early intervention limits atrial fibrosis and prevents extreme LA dilation, reducing, but not eliminating, the lifetime risk of AF. Despite this proactive strategy, post-operative and late-onset AF remain frequent, and a subset of patients progresses to persistent AF, sustaining long-term thromboembolic risk even after technically successful repair. These observations strengthen the rationale for selective prophylactic LAA exclusion during mitral valve repair, particularly in younger patients.[27]

7. Conclusion

Expanding surgical LAA exclusion indications to younger patients undergoing mitral valve repair represents a logical evolution of preventive strategy. Integrating anatomical, functional, and valvular factors, including LA size, residual MR risk, and atrial remodeling, enables

nuanced, individualized decision-making that extends beyond traditional age-weighted stroke-risk scores. In parallel, the incorporation of bleeding-risk assessment tools, such as the HAS-BLED score, highlights the cumulative limitations of lifelong oral anticoagulation, particularly in younger patients who may otherwise face decades of therapy. Although an elevated bleeding risk does not contraindicate anticoagulation, it underscores the importance of strategies that minimize long-term exposure while preserving effective stroke prevention. In contrast with transcatheter approaches reserved for later-stage disease, prophylactic surgical LAA exclusion during mitral valve repair offers an upstream, anatomy-driven brain–heart prevention strategy. This integrated approach aligns with broader preventive paradigms in cardiovascular and neurological care, emphasizing lifetime cerebrovascular protection rather than short-term event reduction alone. Future progress will require prospective registries, interdisciplinary collaboration, and inclusion of long-term neurocognitive outcomes alongside stroke incidence. Until definitive trials are available, thoughtful, individualized discussion regarding prophylactic surgical LAA exclusion at the time of open-heart mitral valve repair is warranted. By addressing the anatomical source of embolism early, this brain–heart prevention perspective promotes durable protection against thromboembolic events and integrated stewardship of cardiac and neurological health.

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Conflict of interest

The authors declare that they have no competing interests.

Author contributions

Conceptualization: Jacob Zeitani, Horst Sievert, Kolja Sievert
Visualization: Verona Beka
Writing – original draft: Alfred Ibrahimi, Romina Teliti
Writing – review & editing: Kolja Sievert, Jacob Zeitani, Verona Beka

Ethics approval and consent to participate

Not applicable.

Consent for publication

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