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DETECTION WITHOUT DIRECTION: THE CLINICAL SIGNIFICANCE OF SUBCLINICAL ATRIAL FIBRILLATION IN STROKE PREVENTION

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ABSTRACT

The increasing use of long-term cardiac monitoring in patients with cryptogenic stroke has led to a marked rise in the detection of arrhythmias, particularly subclinical atrial fibrillation (AF). While this improved detection represents a clear step forward, it also introduces new clinical uncertainty.

AF has traditionally been viewed as a direct cause of thromboembolic stroke. However, emerging evidence suggests that this relationship is more complex, especially in the context of short, device-detected episodes. Major trials, including ASSERT, CRYSTAL-AF, LOOP, ARTESiA, and NOAH-AFNET 6, consistently show that although subclinical AF is associated with increased stroke risk, its detection alone does not reliably translate into improved outcomes when used as a trigger for anticoagulation under current guideline frameworks.

This uncertainty is further reinforced by the lack of clear temporal association between AF episodes and stroke, as well as the absence of validated burden thresholds defining clinically meaningful arrhythmia.

Concepts such as atrial cardiopathy suggest that AF may act more as a marker of an underlying prothrombotic substrate than as a direct causal mechanism. This perspective shifts the focus away from AF as a binary finding toward a model in which stroke risk emerges from the interaction between arrhythmic burden, clinical risk profile, and structural atrial disease.

Taken together, these findings highlight the limitations of current AF-centered approaches and support the need for a more integrated and clinically nuanced strategy for risk stratification and treatment decisions.

KEYWORDS

Atrial Fibrillation, Subclinical AF, Cryptogenic Stroke, AF Burden, Implantable Loop Recorder, Atrial Cardiopathy

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1. Introduction

The diagnosis of stroke causes is a complex issue. This is especially important because of the major long-term impact of stroke and its complications that we were not able to prevent. Approximately 80% of all strokes are ischemic, caused by blood clots. Within ischemic strokes, up to 40% are classified as cryptogenic. Of these, about a half are categorized as Embolic Stroke of Undetermined Source (ESUS), which accounts for roughly 10-18% of all ischemic strokes [1]. The risk of stroke recurrence is approximately 30% and is classically described as 5-10 times higher than in the general population. The risk is the highest in the first year after the initial event, reaching approximately 10-15% [2]. While primary prevention strategies reduce population-level stroke incidence, their impact at the individual patient is often limited. The limitation is caused by the captious and multifactorial nature of cerebrovascular disease. In contrast, secondary prevention targets a population with much higher baseline risk, that makes it the most effective strategy for reducing recurrent events. Additionally, improved understanding of underlying mechanisms and progress in development of new methods enable new ways of detection with wider detection windows, extending to continuous monitoring with greater sensitivity than current standard methods.

The increase in the use of implantable loop recorders (ILRs), with enhanced AF detection, provides new opportunities, and better diagnostic sensitivity. However, this increased diagnostic sensitivity introduces a critical clinical dilemma: whether the detection of atrial fibrillation, particularly in its subclinical form, should directly translate into therapeutic intervention.

The aim of this paper is to evaluate if the detection of device detected, subclinical atrial fibrillation should be interpreted in a binary manner as a direct trigger for consideration of anticoagulation, or if its clinical relevance depends on a broader assessment of arrhythmic burden, patient risk profile, and atrial substrate.

2. Pathophysiological Background

2.1 Pathophysiological background of AF-related thromboembolism

Atrial fibrillation (AF) is the most common arrhythmia. AF, as a risk factor for thromboembolism, is one of the most common causes of cardioembolic events. Cardioembolic causes account for 20-25% of all ischemic strokes [19].

Mechanisms connected to AF-related strokes are commonly described using Virchow's triad, although they are not fully understood yet [3].

Loss of synchronized atrial contraction leads to blood stasis, particularly in the left atrial appendage, and it is the primary site of thrombus formation. In addition, abnormalities of the vessel and atrial wall (e.g., left atrial remodeling), as well as changes in blood constituents, contribute to a hypercoagulable state [3,19]. This pathophysiological complexity is to some extent reflected in current treatment guidelines, which consider the presence of AF as a gateway to risk stratification for treatment decisions [13].

Parts of this complex process can be visualized in many ways, such as transesophageal echocardiography (TEE) for thrombus detection in the left atrial appendage or the spontaneous echo contrast indicating blood stasis. Cardiac magnetic resonance imaging can help with identifying atrial fibrosis and/or heart muscle remodeling. Both of those examples are well documented risk factors of stroke [4].

2.2 Stroke phenotype in radiological tools

The typical stroke phenotype associated with AF-related stroke is an embolic stroke with characteristic clinical and radiological features. The infarctions are usually large and cortical. Typically involving major cerebral artery territories or multiple vascular territories. Due to that, those strokes are usually characterized by greater clinical severity [4,5]. Those characteristics show a clear contrast with lacunar strokes, which are typically on much smaller territories and also deeper into the brain structure. Caused by small vessel pathology and much less associated with cardioembolic processes. Recognition of these patterns may guide clinical attention towards the right epidemiology and point into the need for greater cardiac monitoring or administering of anticoagulation/ therapy [4,5].

2.3 Transition to clinical uncertainty

While the classical understanding of AF-related ischemic stroke is well established, it is derived from a clinical data limited by previous diagnostic capabilities and related to patients with sustained and clinically "loud" arrythmia. However, modern studies such as ASSERT suggest the previously underdiagnosed subclinical arrhythmias such as AF are also associated with an increased risk of stroke. Even though the temporal consistency between AF and thromboembolic event seems to be lacking [6].

These observations seem to indicate that a stroke risk derived from subclinical AF may be related to the quantitative expression of AF burden rather than its clinical presence. The consensus on a set threshold that would help distinguish between relevant and clearly incidental arrhythmia occurence is not there yet [6,8].

That raises an important question: at what point does the AF act as a cause for a thromboembolic event an justifies the stratifying use of CHA_2DS_2-VA scale for administering of therapy/ anticoagulation, and when does it serve as a marker prompting the use of observation and less intense therapy methods. Current findings suggest a need for deeper investigation of the temporal relationship between AF and ischemic stroke and its relation to radiological findings [6,8].

Consequently, the detection of AF, especially the subclinical one, cannot be interpreted in isolation, but must be considered within a broader clinical context that includes arrhythmic burden, imaging findings, and overall thromboembolic risk [7,8].

3. Implantable Loop Recorders - Diagnostic Revolution

Implantable loop recorders (ILRs) allow for continuous long-term cardiac rhythm monitoring for up to 2-3 years, increasing the detection of atrial fibrillation (AF) compared with the current gold standard, 24-72-hour ECG Holter monitoring [7].

In patients with cryptogenic stroke, AF detection using 24-72 hour Holter monitoring remains low at approximately 1-3%, while extended external monitoring over ~30 days increases detection to around 5-16%. In contrast, data from the CRYSTAL-AF trial demonstrate significantly higher detection rates with ILRs, reaching 8.9% at 6 months, 12.4% at 12 months, and approximately 30% at 3 years, showing that detection depends largely on monitoring duration [7].

However, this increased sensitivity also creates important challenges. It creates the question about the definition of clinically meaningful AF, as traditional thresholds (≥30 seconds) are applied to increasingly shorter and often asymptomatic episodes detected by devices [7,14]. Supporting the validity of such detection, findings from ASSERT trial indicate that atrial high-rate episodes identified by implanted devices correlate well with electrocardiographically confirmed AF, suggesting that single-lead device recordings reliably reflect true arrhythmic events, albeit with limitations depending on device programming [6].

Therefore, while ILRs have effectively solved the problem of arrhythmia detection, they have simultaneously exposed a more complex issue - how to interpret and act upon findings while clinical relevance remains uncertain. In addition to AF, ILRs frequently detect other rhythm disturbances, including pauses and bradyarrhythmias, the significance of which in relation to stroke inducement is not well defined, further complicating clinical decision-making [14].

4. Evidence from Clinical Trials

The contemporary evidence base surrounding subclinical atrial fibrillation (AF) is currently characterized by a gap between detection, association, and therapeutic benefit. While early studies established the clinical relevance of AF detection, more recent trials have increasingly challenged its clinical utility as a treatment trigger [8,13].

The CRYSTAL-AF trial demonstrated a clear superiority of implantable loop recorders in detecting AF. The detection rates approaching 30% at 3 years are confirming that subclinical arrhythmia is fairly common in patients with cryptogenic stroke [7]. However, the study did not address whether this increased detection translates into improved outcomes, it being a limitation frequently overlooked in clinical interpretation.

Building on this, the ASSERT trial established an association between device-detected AF and stroke risk [6]. Notably, the lack of consistent temporal proximity between AF episodes and thromboembolic events raised concerns regarding causality and suggesting that AF may function more as a marker of underlying atrial pathology than a direct trigger.

These concerns were further reinforced by the LOOP study. It demonstrated that increased detection and subsequent clinical management did not result in a significant reduction in stroke incidence [8]. This represents an important turning point, directly challenging the assumption that identification of AF alone is sufficient to improve outcomes.

Finally, randomized evidence from ARTESiA and NOAH-AFNET 6 addressed the therapeutic implications of subclinical AF. While ARTESiA demonstrated a reduction in thromboembolic events at the cost of increased bleeding, NOAH-AFNET 6 was terminated early due to the lack of net clinical benefit and safety concerns. Both of those trials show the absence of a clearly defined threshold at which anticoagulation provides an unequivocal benefit [9,10].

These findings point to a certain paradox: we have achieved high sensitivity in detecting AF, established its statistical association with stroke, but failed to demonstrate consistent clinical benefit from treatment based solely on its presence. This disconnect suggests that current paradigms overestimate the causal role of subclinical AF and underscores the need for a more nuanced approach to risk stratification.

5. Subclinical AF: Burden, Duration, and Risk

The relationship between subclinical atrial fibrillation (AF) and thromboembolic risk is not defined only by its presence. It's defined by its quantitative characteristics, including duration, frequency, and overall burden. While earlier frameworks treated AF as a binary entity, emerging evidence suggests a continuum in which increasing arrhythmic burden correlates with higher stroke risk. But in a way that is non-linear and still not fully understood [6,14].

Data from device-based studies, including ASSERT, indicate that episodes exceeding approximately 5.5 hours are associated with increased stroke risk, when shorter episodes, often defined using the ≥30 second threshold, show inconsistent or weak associations [6]. This highlights a key limitation: thresholds developed for conventional ECG diagnosis are now applied to continuous monitoring systems, where it becomes unclear how clinically relevant the finding is [14].

The absence of a clearly defined threshold that would justify therapeutic intervention further complicates interpretation. Even though longer and more frequently appearing episodes are associated with higher risk, current evidence does not establish one cut-off separating clinically meaningful from incidental arrhythmia [6,8,14].

Additional challenge lies in the temporal relationship between AF and stroke. Studies show thromboembolic events frequently occurring without preceding AF episodes [6,8]. This weakens the direct causal model and supports the view that AF may act more as a marker of an underlying prothrombotic substrate than a trigger [4].

What's important, the increasing ability to detect AF seems to have outpaced our ability to interpret its clinical significance. Lowered detection thresholds improve sensitivity but reduce specificity and that leads to identification of episodes whose relevance remains unclear [14]. The conclusion is that AF presence is not equivalent to AF relevance, and its detection, particularly in low burden forms, should be interpreted within a broader context including the clinical risk profile and structural atrial disease [4,13].

6. Clinical Implications and Decision-Making

Having previous chapters in mind, the topic of Subclinical AF raises a key question: when should anticoagulation be initiated? Current guidelines, including those of the European Society of Cardiology, recommend stroke prevention strategies based on clinical risk stratification scores such as CHA_2DS_2-VA. There, AF serves as an entry point into a risk-based model rather than a standalone therapeutic trigger [13]. In practice however, the detection of AF (especially having in mind device detected subclinical AF) often ends up acting as an indication for treatment. Especially once a certain, although ill-defined burden threshold is perceived.

Recent changes in guideline based risk assessment, including the transition from CHA_2DS_2-VASc scale to CHA_2DS_2-VA one, simplify decision-making by removing less predictive modifiers [13]. While surely methodologically justified, this simplification may reduce the inclusion of broader clinical contest and in borderline cases increasing the relative clinical weight assigned to the presence of AF itself. In the context of highly sensitive monitoring technologies producing subclinical AF readings, this shift risks reinforcing an implicit binary logic: AF detected → risk stratify → anticoagulate.

Current guidelines also emphasize that thromboembolic risk in AF is largely independent of its clinical pattern, with comparable risk observed across paroxysmal, persistent, and permanent forms [11,12]. However, this principle is derived from populations with clinically overt AF and should not be directly extrapolated to subclinical, device detected arrhythmias. Device detected AF episodes are frequently brief, asymptomatic, and identified only through prolonged monitoring. Emerging evidence suggests that their clinical relevance is more closely related to quantitative burden than solely their presence. Applying a model developed for sustained clinically “loud” arrhythmia to low-burden subclinical episodes introduces a mismatch between diagnosis and treatment [6].

This mismatch becomes most apparent in real-world clinical scenarios. For example, a patient with high clinical risk but only seconds long, device detected AF presents a fundamentally different problem than one with sustained, clinically evident arrhythmia. In addition, a younger patient with longer but less frequent episodes may not get the same benefit from anticoagulation treatment despite a seemingly higher arrhythmic burden. In both cases, decision-making occurs in a clear absence of validated thresholds, forcing clinicians to navigate on a knives edge between over-treatment and under-protection [14].

The resulting tension reflects an unresolved risk-benefit trade-off. Anticoagulation reduces thromboembolic risk. But it does so at the cost of increased bleeding. This balance becomes even more uncertain as AF burden decreases. Evidence from recent randomized trials suggests that in subclinical AF, the net clinical benefit of anticoagulation is not uniform, even further undermining a current, simple treatment paradigm [9,10].

Taken everything into account, these observations show clear limitations of a model in which AF detection alone drives management decisions. The increasing sensitivity of ILR devices has outpaced the specificity of therapeutic guidelines. Therefore, creating a landscape in which the presence of AF is often overinterpreted in relation to its true clinical significance. In practice, this likely means a shift toward a more integrated approach that considers AF burden, overall risk profile, and underlying atrial substrate. Instead of relying on detection as a sufficient trigger for intervention [8,13].

7. Beyond AF - The Atrial Cardiopathy Model

The limitations of an AF causality of stroke model become clear when considering that thromboembolic risk is not fully explained by arrhythmia alone. The concept of atrial cardiopathy proposes that structural, functional, and biochemical abnormalities of the atrium may as a conglomerate promote thromboembolism. Even in the absence of clinically overt atrial fibrillation [4].

In this context, AF is reinterpreted not as a causal trigger, but more as a marker of an underlying diseased atrium (characterized by fibrosis, dilation, and impaired contractility) [4]. This perspective is supported by imaging-based studies such as DECAAF II, which demonstrate a relationship between atrial fibrosis and adverse cardiovascular outcomes [18], as well as by biomarker-based approaches linking elevated natriuretic peptides and electrocardiographic abnormalities to increased stroke risk [4].

Having that in mind, attempts to translate this model into clinical practice have yielded mixed results. The ARCADIA trial, which evaluated anticoagulation in patients with atrial cardiopathy without documented AF, failed to demonstrate a significant benefit over antiplatelet therapy [15]. Quite similarly, trials in ESUS, such as NAVIGATE ESUS and RE-SPECT ESUS, did not show superiority of anticoagulation [16,17], further suggesting that not all embolic events are driven by AF or respond to its treatment paradigm.

Importantly, implantable loop recorders capture a full spectrum of arrhythmias beyond atrial fibrillation, including bradyarrhythmias and pauses, whose independent contribution to stroke risk remains uncertain [14]. In featured in this chapter Cardiopathy Model, those arrhythmias instead of acting as an embolic substrate and causal mechanisms, may reflect a broader atrial and conduction system pathology, reinforcing the concept that thromboembolic risk is not exclusively linked to AF itself [4,14].

These observations position AF within a wider pathophysiological context, and no more as a treatment target. While the atrial cardiopathy model provides a compelling explanation for the inconsistencies observed in AF-related stroke research, its current inability to guide effective therapeutic decisions limits its incorporation into clinical guidelines [13]. As such, it cannot serve as replacement for existing frameworks but more as a lens through which current guidelines limitations can be understood.

8. Synthesis and Future Directions

8.1 Reframing the problem

Over the past decades, advances in continuous cardiac monitoring have significantly improved the detection of subclinical atrial fibrillation (AF). However, as demonstrated in this review, the ability to detect AF has outpaced our understanding of its clinical significance. AF has traditionally been regarded as a direct cause of thromboembolic stroke but emerging evidence suggests that this relationship is not universally causal.

Findings from studies such as ASSERT and LOOP highlight a fundamental inconsistency: increased detection of AF does not consistently translate into expected reductions in stroke risk [6,8]. This makes the simple binary model harder to justify.

8.2 A multidimensional model of stroke risk

Overall, the evidence presented in this Thesis suggests a shift from a binary to a multidimensional model of thromboembolic risk in AF. In this framework, stroke risk is not determined solely by the presence of arrhythmia, but rather by the interaction of three key domains:

- AF burden (duration and frequency of episodes).
- Clinical risk profile (e.g., CHA_2DS_2-VA score) [13].
- Atrial substrate (structural and functional abnormalities) [4].

This approach shows where currently used models fall short, where highly sensitive detection methods identify increasingly shorter and clinically ambiguous episodes of AF without clear guidance on their significance.

The concept of atrial cardiopathy provides a unifying explanation for these observations, suggesting that AF may act more of as a marker of a broader prothrombotic state rather than its sole driver [4].

8.3 Limitations of current clinical practice

Current guideline based management, largely derived from populations with clinically overt AF, does not account for the complexity of subclinical AF [13]. Several limitations emerge:

- Lack of validated AF burden thresholds defining clinically meaningful risk [6,14]
- Insufficient integration of temporal relationships between AF episodes and stroke events [6].
- Equating device-detected AF with clinical AF, despite differing pathophysiological implications [14].
- Uncertain net benefit of anticoagulation in low-burden or asymptomatic cases [9,10].

As a result, clinical practice fixed in current guidelines may lead to both overdiagnosis and overtreatment, particularly in patients with minimal AF burden but elevated clinical risk scores.

8.4 Toward integrated risk stratification and future perspective

Future strategies should move beyond AF detection alone and toward multimodal risk assessment models. Potential directions include:

- incorporation of AF burden metrics into clinical decision-making [6,14].
- use of advanced imaging to assess atrial structure and fibrosis [18].
- development of biomarker-based approaches reflecting atrial dysfunction [4].
- refinement of temporal risk models linking arrhythmia episodes with thromboembolic events [6].

Such approaches may allow for more precise identification of patients who really benefit from anticoagulation, while avoiding hemorrhagic complications and unnecessary treatment in low-risk individuals.

The evidence suggests that subclinical AF should not be interpreted as a uniform entity, but rather as a spectrum of findings with variable clinical relevance. The presence of AF is not equivalent to its clinical significance. Recognizing this distinction is essential for the evolution of both research and clear pathways through clinical practice and supports a shift toward interpreting AF as part of a broader pathophysiological process rather than a standalone therapeutic trigger [4,13].

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