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# Comments on Nonsustained Atrial Fibrillation and Stroke Risk: Methodological and Interpretive Considerations

LETTER TO THE EDITOR

To the Editor,

We read with interest the study by Yurtseven et al,[1] which investigates the association between nonsustained atrial fibrillation (NS-AF) episodes lasting less than 30 seconds and ischemic stroke risk.[1] While the study addresses a clinically relevant question, we identified several issues that warrant discussion, including inconsistencies within the manuscript, discrepancies with existing literature, and methodological limitations.

The study reports a significant association between NS-AF episodes (<30 seconds) and stroke risk (OR = 3.930, 95% CI: 1.235-12.510, $P = .021$), with a CHA2DS2-VA score ≥2 showing high sensitivity (85.7%) for predicting stroke. However, a discrepancy in the reported sample size raises concerns. The methods section states that 133 patients with NS-AF and 113 controls were included (totaling 246), but the results section references 163 NS-AF patients (Table 1). This inconsistency questions the accuracy of patient inclusion and statistical analyses. Could the authors clarify the correct sample size and its impact on the study's findings?

The conclusion that NS-AF episodes <30 seconds independently increase stroke risk contrasts with studies suggesting that brief AF episodes carry minimal risk unless prolonged. The ASSERT trial found that subclinical AF episodes >24 hours were strongly associated with stroke, while shorter episodes (<6 minutes) showed weaker links.[2] Similarly, the RATE Registry reported no significant stroke risk for AF episodes of 10-20 seconds.[3] Although cited, these studies are not reconciled with the current findings. How do the authors explain this divergence, particularly given the limitations of 24-hour Holter monitoring in capturing AF burden?

Methodologically, the retrospective design and reliance on 24-hour Holter monitoring may underestimate AF burden, as longer monitoring (e.g., implantable devices) is more sensitive. The authors acknowledge this but do not discuss its impact. Additionally, excluding patients with paroxysmal or persistent AF during follow-up relied on hospital records and phone interviews, which may miss subclinical AF, potentially confounding results. Propensity score matching failed to eliminate age differences ($P = .045$ post-matching), a critical confounder given age's association with stroke risk.[4] Why did age remain unmatched, and how does this affect the reported odds ratio for NS-AF? Furthermore, the definition of NS-AF as "more than 3 consecutive irregular atrial contractions without visible P waves" lacks specificity, risking inclusion of non-specific atrial ectopy.[5] How was this definition validated and were inter-observer agreement metrics assessed?

We pose the following questions: (1) Can the authors resolve the sample size discrepancy (133 vs. 163)? (2) What mechanistic insights explain the elevated stroke risk in this cohort compared to conflicting literature? (3) Why was 24-hour Holter monitoring chosen over longer-term monitoring, and how was subclinical AF accounted for? (4) Why was CHA2DS2-VA used instead of CHA2DS2-VASc, and how does this affect generalizability?



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While the study highlights a potential stroke risk in brief NS-AF episodes, these issues limit the robustness of the findings. We encourage the authors to address these concerns and suggest larger prospective studies with extended monitoring to guide anticoagulation strategies.

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