Risk of a residual shunt following patent foramen ovale closure: A myth?

Bryan Kluck

Interventional Cardiology-Emeritus, Lehigh Valley Hospital, Allentown, PA, USA

https://doi.org/10.17925/HI.2026.20.1.6

This letter challenges the prevailing assumption that residual shunt following patent foramen ovale (PFO) closure is a primary driver of recurrent stroke risk. While residual shunt has been associated with higher rates of neurologic events in observational studies, the author argues that this relationship is not supported by randomized data and may be confounded by diagnostic ambiguity, particularly the inclusion of transient ischemic attack alongside stroke outcomes. The complex pathophysiology of PFO-related stroke, requiring thrombus formation, transient right-to-left shunting, and specific anatomic factors, is often substantially mitigated by the closure procedure itself. Emerging evidence suggests that device-related factors, including incomplete endothelialization and altered atrial hemodynamics, may independently contribute to thromboembolic risk after closure, irrespective of residual shunting. The letter proposes that residual shunt may be an inadequate surrogate endpoint and that clinical focus should instead remain on true stroke recurrence. It further cautions against routine re-intervention to eliminate residual shunt, advocating instead for consideration of prolonged antithrombotic therapy and alternative closure strategies, such as suture-based approaches, which may avoid device-related complications. Overall, this perspective calls for a reassessment of current paradigms in post-PFO closure management and outcome evaluation.

Keywords

Antithrombotic therapy, atrial hemodynamics, cardiac shunt, cryptogenic stroke, device closure, endothelialization, heart septal aneurysm, atrial, patent foramen ovale (PFO) closure, stroke recurrence, suture-mediated closure

Disclosures: Bryan Kluck has no financial or non-financial relationships or activities to declare in relation to this article.

Acknowledgements: No artificial intelligence (AI) or large language model (LLM) tools were used in the development of this article.

Review process: Double-blind peer review.

Compliance with ethics: This article is an opinion piece and does not report on new clinical data or any studies with human or animal subjects performed by any of the authors.

Data availability: Data sharing is not applicable to this article as no datasets were generated or analyzed during the writing of this article.

Authorship: The named author meets the criteria of the International Committee of Medical Journal Editors for authorship for this manuscript, takes responsibility for the integrity of the work as a whole and has given final approval for the version to be published.

Access: This article is freely accessible at touchCARDIO.com. © Touch Medical Media 2026.

Received: 18 March 2026

Accepted: 21 April 2026

Published online: 2 June 2026

Citation: *Heart International.* 2026;20(1):Online ahead of journal publication

Corresponding author: Bryan Kluck, Cardiovascular Experts of PA, 3040 Market St, Camp Hill, PA 17011, USA. E: cathsalot@gmail.com

Support: No funding was received in the preparation of this article.

After decades of debate, patent foramen ovale (PFO) closure is now a well-established strategy to reduce recurrent stroke in patients aged 18–60 years who have had a PFO-associated stroke.[1] Patients who underwent PFO closure continue to have a higher stroke risk than the general population. A recent study demonstrated a 2.1% higher stroke rate in the patients who underwent PFO closure compared with age-matched controls at 4 years.[2] The reasons for this elevated post-closure risk are multifactorial. These factors would include incomplete re-endothelialization of the device placed, altered left atrial hemodynamics, residual shunt and possibly that the PFO was not causative of the stroke at all.

Kim et al. investigated patients who underwent device PFO closure by computed tomography (CT), looking at re-endothelialization.[3] Incomplete re-endothelialization was present for far longer than previously suspected. Larger devices, males and older patients were more prone to incomplete re-endothelialization.

Gaspardone et al. compared atrial function post-closure with devices and with suture-based closure.[4] This demonstrated that only device closure altered atrial hemodynamics.

There are no randomized studies that show elevated stroke rate with residual shunt. The literature that does exist citing elevated neurologic event rates following closure lumps true stroke and transient ischemic attack (TIA).[5] TIA is less of a hard endpoint compared with stroke. TIA symptoms are often subtle and, in the post-stroke patient (a significant majority of closure patients), easily confused with residual stroke symptoms. Differentiating these is purely clinical, as TIA has no permanent imaging signal. Thus, the only legitimate comparator would be stroke alone. Gaspardone et al. followed over 700 patients with suture-mediated closure for over 4 years.[6] There were no recurrent neurologic events. Beyond that, a sizeable fraction of patients with residual shunt chose not to have further PFO closure of any sort. In this cohort, there were no recurrent strokes.

Thus, a reasoned and less invasive approach should be taken in the post-closure patient as a residual shunt may, in fact, not be the cause of recurrent ischemic events. With the passage of time, residual shunts reduce and often close, irrespective of the device or procedure selected.[7] Prolonged antithrombotic therapy may well be a better approach and one that should be clinically evaluated. Beyond that, the placement of additional devices or plugs to seal a residual shunt would certainly seem to unfavorably affect both re-endothelialization and atrial hemodynamics.

As the contemplated role of PFO closure broadens, the development of lower profile devices, a suture-mediated closure strategy and the role of ongoing medical therapy all need consideration. ❑

1. Saver JL, Carroll JD, Thaler DE, et al. Long-term outcomes of patent foramen ovale closure or medical therapy after stroke. *N Engl J Med*. 2017;377:1022–32. DOI: 10.1056/NEJMoa1610057.
2. Bonnesen K, Korsholm K, Andersen A, et al. Risk of ischemic stroke after patent foramen ovale closure. *J Am Coll Cardiol*. 2024;84:1424–33. DOI: 10.1016/j.jacc.2024.07.015.
3. Kim AY, Woo W, Lim BJ, et al. Assessment of device neoendothelialization with cardiac computed tomography angiography after transcatheter closure of atrial septal defect. *Circ Cardiovasc Imaging*. 2022;15:e014138. DOI: 10.1161/CIRCIMAGING.122.014138.
4. Tarsia C, Gaspardone C, Sgueglia GA, et al. Atrial function analysis after percutaneous umbrella device and suture-mediated patent fossa ovalis closure. *Minerva Cardiol Angiol*. 2023;71:83–90. DOI: 10.23736/S2724-5683.21.05710-0.
5. Deng W, Yin S, McMullin D, et al. Residual shunt after patent foramen ovale closure and long-term stroke recurrence: A prospective cohort study. *Ann Intern Med*. 2020;172:717–25. DOI: 10.7326/M19-3583.
6. Gaspardone A, Sgueglia GA, De Santis A, et al. Suture-mediated patent fossa ovalis closure: Late clinical outcomes and predictors of technical success. *JACC Adv*. 2025;4(8):101991. DOI: 10.1016/j.jacadv.2025.101991.
7. Witte LS, El Bouziani A, Beijk MAM, et al. Persistence of residual shunt at 6 and 12 months after transoesophageal echocardiography-guided percutaneous closure of a patent foramen ovale for cryptogenic stroke. *Heart*. 2024;110:1172–9. DOI: 10.1136/heartjnl-2024-323905.