# **April 27 Highlights and Commentary**

## Patent foramen ovale closure and migraine

Post et al. studied 76 patients with a patent foramen ovale (PFO) and found that the prevalence of migraine with aura decreased from 18.2% before to 5.3% after percutaneous PFO closure.

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Schwerzmann et al. observed that percutaneous PFO closure in 215 patients after presumed paradoxical embolism unexpectedly reduced the frequency of migraine attacks by >50%, but had no effect on other headaches.

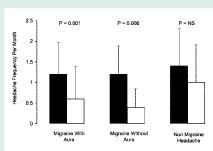
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## Is patent foramen ovale closure a treatment for migraine?

Commentary by Deborah Friedman, MD

The foramen ovale, a remnant of the fetal circulation, allows oxygenated placental blood entering the right atrium to flow to the arterial system during gestation. In 20 to 25% of people, the foramen fails to close completely within the first 2 years of life, producing a right-to-left shunt. 1,2 PFO is implicated in the pathogenesis of stroke, migraine headache, and decompression sickness. The inter-relationship between PFO, stroke, and migraine has received recent attention as an area of clinical investigation. A reduction in migraine frequency was previously reported in patients with PFO undergoing either surgical closure or anticoagulation following a stroke.3 However, considerable uncertainty exists regarding the management of PFO and stroke and no randomized trials comparing surgical and medical therapy have been performed.<sup>2</sup>

The above two retrospective studies examined whether PFO closure changed migraine frequency. PFO closure was performed after patients experienced a paradoxical or embolic event or



Frequency of headache attacks per month before PFO closure and  $\square$  after PFO closure.

systemic desaturation. There was both a high prevalence of migraine in patients with PFO preoperatively, and a significant decrease in migraine frequency and impact after percutaneous PFO closure.

Proposed mechanisms of PFO contributing to stroke include paradoxical embolus, associated atrial arrhythmias, concomitant hypercoagulable conditions, and thrombosis within the canal of the PFO. The role of PFO in the pathogenesis of migraine is uncertain but there are two suggested mechanisms: paradoxical embolism or possibly a "trigger substance" from the venous circulation that is transported to the arterial system via the right-to-left shunt. At this time, there is not enough evidence to suggest that treatment of otherwise asymptomatic interatrial septal defects is indicated in migraineurs. However, prospective studies assessing the impact of treating symptomatic PFO on migraine are warranted. Moreover, these two articles and others that have linked PFO with migraine are providing a clue as to a cause or trigger of migraine. 1,3

#### References

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