Editorial

Patent Foramen Ovale Is Indicted, but the Case Hasn’t Gone to Trial

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In humans, birth itself is primarily a cardiorespiratory event. The fetus has outgrown its inefficient circulatory dependence on the mother and needs an effective oxygen-delivery system: the umbilical cord clamps off, the lungs aerate, the ductus arteriosus involutes, and the foramen ovale seals shut. A patent foramen ovale (PFO) is, by far, the most commonly persistent abnormality of fetal origin, occurring in 10% to 15% of the normal adult population. Because the thin, left-sided septum primum is pushed against the thicker septum secundum by higher left atrial pressures, a PFO only permits intracardiac shunting during those transient periods (eg, sudden changes in intrathoracic pressures or right heart compliance) when right atrial pressure exceeds left atrial pressure.

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Although common, a PFO is invariably, or almost invariably, benign. The key word here is almost. The first real suspicion of pathological potential came from case reports. One such report included a picture of the heart from a patient who died of a stroke; incredibly, a large thrombus straddled the patient’s persistently patent foramen ovale. A formal indictment of the PFO as a source of embolic stroke was lodged by Lechat et al in 1988, when they reported that patients with an embolic stroke of unknown cause had a much higher incidence of PFOs than control subjects. Although this finding was challenged by some studies that used differing diagnostic techniques, the large majority of studies now support the notion that the presence of a PFO is strongly associated with the occurrence of an embolic stroke of unknown cause. Bridges et al strengthened the prosecution’s case by demonstrating that closure of the PFO reduced the rate of recurrent strokes, albeit in an unusual patient population that was both young and plagued by very high recurrence rates.

The defense has countered by observing, with considerable justification, that another culprit must be at large. More than 30 million Americans have PFOs, but only 1 in 1000 will have an embolic stroke of “unknown” origin. If the PFO were the sole perpetrator, the recurrence rate would be tiny, yet recurrence rates have been estimated to be 3.4% to 11% per patient-year. The defense contends that these patients have a primary clotting diathesis, and lifelong warfarin therapy has emerged as the treatment of choice.

In this month’s issue of Circulation, Windecker et al respond with the largest series to date of patients with PFO and strokes who have undergone transcatheter closure to prevent recurrent strokes. The annual recurrence rate after PFO closure in 80 patients was reasonably low at 2.5% for transient ischemic attack and 3.4% for all embolic events. Most importantly, using multiple different devices with differing closure rates, they found that the presence of a residual defect predicted recurrent events.

The PFO stands accused. The evidence is strong and getting stronger. Acquittal or conviction will only occur after a randomized trial in patients who have a PFO and a first event, comparing closure (either by catheter or surgery) with anticoagulation. Given the accumulating data, one hopes that agencies interested in adult stroke prevention will organize such a trial. Until such a trial is completed, neurologists and cardiologists have real patients with real strokes to manage. A review of the available data, including those presented by Windecker et al, would seem to support the following recommendation: those embolic stroke patients who are younger, who have large PFOs and no other stroke source, and who fail anticoagulant therapy or should not take anticoagulants may be considered candidates for anatomic closure of their PFO.

References

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Dr Lock is a co-inventor of the CardioSEAL device, and his institution receives royalties on the commercial sales of the device.

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