

**Spontaneous Late Closure of Patent Foramen Ovale**

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## CORRESPONDENCE

### Letters to the Editor

#### Spontaneous Late Closure of Patent Foramen Ovale

The study by Meissner et al. (1) and the accompanying editorial by Meier (2) provide contrasting views concerning the pathologic significance of patent foramen ovale (PFO). A large autopsy study from the Mayo Clinic (3) showed a decreasing incidence of PFO with age that the investigators attributed to late spontaneous closure, although they did not suggest the mechanism whereby this might occur. Meier (2) proposes an alternative explanation, namely that the presence of a PFO is associated with a reduced life expectancy. Meissner et al. (1) and Siostrzonek et al. (4) note that a PFO is less frequently detected in patients with elevated left atrial pressure, but they imply that this is the result of a lower sensitivity of the detection technique rather than a lower incidence of PFO.

We believe it unlikely that a PFO confers a significant mortality disadvantage. If this were the case then one would expect a lower proportion of larger PFOs with age as large PFOs are considered to confer a higher risk of adverse events (2). Hagen et al. (3) found the reverse. We believe a more plausible explanation is that a chronically elevated left atrial pressure as occurs with advancing age and loss of left ventricular compliance (5) can result in late closure of a PFO, particularly if small. Indirect evidence for this assumption comes from an observation that in a personal series (R.W.H.) of 260 consecutive patients with mitral stenosis undergoing percutaneous trans-septal valvuloplasty, a PFO was crossed only twice. In this procedure the foramen ovale is always probed with a catheter stiffened with the trans-septal needle. Accordingly, if a PFO had been present in these patients with the same incidence as in the general population (25%), one would have expected a much higher crossing rate than was observed (<1%). In an otherwise normal heart it is generally very easy to cross a PFO with a catheter directed at the limbus of the fossa ovalis.

We believe that the likely explanation for the low crossing rate in mitral stenosis is that the chronically elevated left atrial pressure associated with this condition results in closure of a PFO. We also believe that the lower detection rate of PFO in conditions with elevated left atrial pressure may be the result of late closure of the PFO rather than an inability to detect the condition.

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#### REFERENCES

1. Meissner I, Khandheria BK, Heit JA, et al. Patent foramen ovale: innocent or guilty? Evidence from a prospective population-based study. *J Am Coll Cardiol* 2006;47:440–5.

2. Meier B. Patent foramen ovale, guilty but only as a gang member and for a lesser crime. *J Am Coll Cardiol* 2006;47:446–8.
3. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. *Mayo Clin Proc* 1984;59:17–20.
4. Siostrzonek P, Lang W, Zangeneh M, et al. Significance of left-sided heart disease for the detection of patent foramen ovale by transesophageal contrast echocardiography. *J Am Coll Cardiol* 1992;19:1192–6.
5. Arbab-Zadeh A, Dijk E, Prasad A, et al. Effect of aging and physical activity on left ventricular compliance. *Circulation* 2004;110:1799–805.

#### Patent Foramen Ovale and the Risk of Cryptogenic Stroke

We would like to congratulate Meissner et al. on their impressive cohort study of patent foramen ovale (PFO) and stroke. However, we hold significant reservations on the stated conclusion that PFO is not an independent risk factor for future cerebrovascular events in the general population (1).

The population studied by Meissner et al. (1) was predominately elderly, with a mean age of nearly 70 years ( $\pm$  13 years). The association of PFO and stroke is much less conclusive in older populations, with conflicting studies (2,3). Increasing age is associated with increasing incidence of traditional risk factors for atheroembolic stroke—clearly demonstrated in this study where over 50% of subjects were hypertensive and over 50% had visible aortic plaque (1). It is likely that mechanisms other than paradoxical embolism predominate in these older age groups. The plausibility of a congenital defect suddenly causing a cerebrovascular ischemic event is intuitively much less.

Additionally, it appears no attempt was made to identify those ischemic strokes that were considered “cryptogenic.” These represent the minority of the total strokes (4), but they have been most often associated with PFO (5).

The management of PFO and cryptogenic stroke is an evolving area (6). There are currently at least 3 recruiting trials of PFO closure versus medical management. Of note, all specify an upper age limit for enrollment of 60 years (7–9). The results of these trials are eagerly awaited.

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#### REFERENCES

1. Meissner I, Khandheria BK, Heit JA, et al. Patent foramen ovale: innocent or guilty? Evidence from a prospective population-based study. *J Am Coll Cardiol* 2006;47:440–5.

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