Patent Foramen Ovale and Stroke

To the Editor:

I wish to congratulate the authors of the current “Controversies in Stroke” on their critical discussion of secondary prevention in patients with patent foramen ovale (PFO) and cryptogenic stroke.1–3 In the absence of definitive data in this scenario, they focus a large part of their discussion on the possible treatment options quoting the 2 most cited recently published prospective multicenter studies that evaluated the stroke recurrence risk in patients with PFO.4,5 I fully agree with the authors that the best medical therapy has not yet been determined; furthermore, there is no doubt that another trial randomizing patients <55 years of age either to device closure or medical therapy is necessary. More or less all authors accept that there is an association between the presence of PFO and stroke. Unfortunately, despite the controversy surrounding PFO identification/quantification, I am missing a critical discussion on the limitations and flaws of contrast echo technique which is currently used. In my opinion, there is an urgent need for standardization of PFO identification and quantification, which at present does not exist. In the French PFO-ASA study, a PFO was defined if at least 3 contrast-bubbles appeared in the left atrium. The degree of shunting was defined to be small if 3 to 9 contrast-bubbles appeared, it was moderate if 10 to 30 contrast-bubbles appeared, and large if more than 30 contrast-bubbles appeared in the left atrium.4 The authors of this study did not conceal that sonographers disagreed on the presence of patent foramen ovale in 13.9% of patients and the degree of shunting in 26.6%.6 In the Patent Foramen Ovale in Cryptogenic Stroke Study (PICCSS) a PFO was considered to be present if >1 contrast-bubble appeared in the left atrium, and the authors used a cut-off point for a large shunt if more than 10 bubbles could be demonstrated in the left atrium.5 Very recently it was nicely shown that for a given PFO, the amount of right-to-left contrast shunting is a matter of expiratory pressure during the Valsava maneuver.7 Previously, we and others have shown that in any PFO right-to-left shunting varies considerably and that the magnitude of contrast shunting does not necessarily correlate with the true anatomical size of the PFO.8–10 Due to the orientation of the inferior vena cava blood (which potentially contains an embolus arising from pelvic or deep vein thrombi) to the fossa ovalis, even a large PFO may be missed if contrast agent is administered through a cubital vein, as these bubbles may be redirected from the fossa ovalis by this blood flow.5,11 These flow patterns are aggravated by an Eustachian valve (VE) which directs the blood from the inferior vena cava preferentially to the area of the fossa ovalis can be studied by contrast administration in to foot vein.9,11,13 By the way, this valve is frequently seen in patients with patent foramen ovale.12,13 Moreover, there are reports showing that transthoracic contrast echocardiography with harmonic imaging mode may be too sensitive at the expense of a decreased specificity for PFO detection.14 Furthermore, the time appearance of contrast-bubbles in the left atrium which is used as one of the distinguishing features between intracardiac and (physiologi- cal) intrapulmonary shunts has shown to be unreliable.15–18

Tong and Backer2 state that the relevance of PFO may be analogous to the presumed association between mitral valve prolaps and stroke reported over the last decade. Contrary to this assumption, I believe that by neglecting the underlying pathophysiology of right atrial flow pattern and the method- ological limitation of the commonly used contrast echo technique, there is a higher chance of underestimating the genuine risk of a large PFO. Consensus in the identification/ quantification of this valve like structure is needed to avoid confusion and to make future interventional studies easier to interpret. I agree with Tong and Becker that studies for PFO closure may be premature in this patients with cryptogenic stroke, not due to doubts on causality but rather due to the fact that we should first acknowledge and close the “hole” of our ignorance on morphological/functional PFO characteristics. Unless we are not able to optimize and standardize our available methods for PFO identification/quantification, we will not be able to randomize comparable patients and the issue of PFO and stroke will not be conclusively resolved.

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