Diagnosis of Patent Foramen Ovale

We read with great interest the improved echocardiographic diagnostic protocol for patent foramen ovale (PFO) by Rana et al. (1). We wish to highlight some relevant issues.

Demonstrating the presence of PFO does not establish its etiopathogenic role in systemic embolization. A small-sized PFO may be associated with higher "functional potential" and play a more important etiological role compared with its morphology. Transesophageal echocardiography (TEE) is considered the gold standard for diagnosing PFO, because it provides anatomical details and can occasionally visualize the travel of a thrombus in real time. However, TEE requires an expert echocardiographer, is poorly tolerated by patients, and the Valsalva maneuver (VM) is often impaired by sedation and the endoscope in the throat. Although these limitations do not interfere with diagnostic capability, they may affect the functional grading of PFO. Some of these limitations may be overcome with transcranial Doppler (TCD). TCD is a reliable technique with accuracy parameters comparable with those of TEE (2). Because TCD does not require sedation, an effective VM can be performed by most subjects.

Considerable variations exist in the timing of the VM during the diagnostic testing for PFO. Although some studies performed the VM simultaneously with the contrast injection (3), others initiated it after 3 to 5 s (2). Substantial hemodynamic changes occur during the "strain phase" of the VM (amplifying the interatrial left-to-right pressure gradient and counteracting right-to-left shunting) as well as the "release phase" (reversing the pressure gradient with a sudden increase in venous return and right atrial pressure facilitating right-to-left shunt). Importantly, considerable reduction and even complete stoppage of flow occurs in proximal veins during the VM. Thus, injecting contrast during the VM into a proximal vein seems counterproductive, because the high venous pressure might destroy some microbubbles. Furthermore, microbubbles created by vigorous shaking of the contrast mixture have short life and may not reach the heart in sufficient numbers, diminishing the functional grading of PFO.

Body position during the diagnosis of PFO needs special consideration. Air bubbles, being lighter, tend to "move up" because of buoyancy. Thus, patients’ left lateral position during echocardiography is not conducive for the travel of microbubbles (from the higher right atrium into the lower left atrium). We observed that the “functional grade” of PFO varies with body position, and a larger number of microbubbles are detected in the sitting position (4). Additionally, the sitting position may promote opening of the shunt flap during the VM, helping more microbubbles cross the shunt. Changing body positioning from supine to sitting upright substantially increased the microbubble count (from a median of 20 in the supine position to 72 in the sitting position) in 42% of our study population (4). Establishing the functional grade of a PFO is important because it provides useful information about the likelihood of prevalent ischemic stroke as well as the risk for recurrent events (5).

We reiterate that the simple detection of a PFO does not delineate its true etiopathogenic role. Establishing the “functional status” of a PFO is essential for planning definitive treatment. We propose that TCD, performed in various body positions, should be used for screening and establishing the functional grade of a right-to-left shunt, and echocardiography may be performed in selected patients to evaluate the morphological characteristics of PFO.

Vijay K. Sharma, MD, RVT, *Hock L. Teoh, MD, Bernard P. L. Chan, MD

*Division of Neurology, Department of Medicine, National University Hospital, 5 Lower Kent Ridge Road, Singapore 119074.

E-mail: drvijay@singnet.com.sg
doi:10.1016/j.jcmg.2010.08.004

REFERENCES