Prevalence of Patent Foramen Ovale and Its Contribution to Hypoxemia in Patients With Obstructive Sleep Apnea

To the Editor:

Shanoudy and colleagues1 presented a study that addressed an interesting and important question concerning the incidence of patent foramen ovale (PFO) in patients with obstructive sleep apnea (OSA). Although their methods were detailed, several important points were omitted. How were OSA patients selected? Did they include all patients with OSA that did not meet exclusionary criteria and consented, or were potential biases introduced? Patients with “obstructive or restrictive lung disease as documented by pulmonary function tests” were excluded. How many patients were excluded? Did all patients undergo pulmonary function testing? What criteria were used for the diagnosis of obstructive and restrictive lung disease? The data for FEV1/FVC ratio were presented, but the FEV1 and FVC are at least as important. How were control subjects selected, and did they all have pulmonary function tests? All control subjects had “normal cardiac dimensions and functions with no valvular pathologic findings by [transesophageal echocardiography] TEE.” It seems unusual that 24 patients undergoing diagnostic TEE would have totally negative TEE unless stipulated by the selection criteria. How exactly was the ΔSaO2 assessed? It was noted that SaO2 recordings were printed serially, but it was not stated whether the recordings were continuous. This is an important point since failure to use continuous values could result in subtle bias through unintentional closer attention to the nadir SaO2 in patients with OSA. These are basic methodologic points that are important to the interpretation of any study and hopefully were picked up by the reviewers of this article.

There appears to be a discrepancy in the reporting of pulmonary artery pressure (PAP). Table 2 notes that patients with OSA and PFO had a PAP of 34 mm Hg, while those without PFO had a PAP of 22 mm Hg. Using these numbers, the group of all patients with OSA should have a PAP of 30.3 mm Hg, rather than 32 mm Hg as reported in Table 1. Furthermore, the difference in PAP between the two OSA groups was reported as not significant, while a lesser difference between OSA patients and control subjects was significant. Should the PAP in OSA patients without PFO be 27.6 mm Hg instead? Finally, how significant were the PFO as detected by TEE? Did the majority of patients with PFO show a small number of microcavitations or did they show significant opacification of the left atrium? Although the ΔSaO2 was significantly different in OSA patients with and without PFO, the absolute difference was small.

James R. Gossage, MD, FCCP
Section of Pulmonary Diseases
Medical College of Georgia
Augusta, Georgia

Correspondence to: James R. Gossage, MD, Medical College of Georgia, Section of Pulmonary Diseases, BBR-5513, Augusta, GA 30912-3135

REFERENCES


A Critical Commentary and Treatise on Myocardial Repolarization

To the Editor:

The article “T-Wave Inversion in Pulmonary Embolism” by Dr. Nikolic1 prompted the study of Ferrari and colleagues,2 merits additional clinical commentary.

It is not my intent to discuss or debate the comments brought forth by Dr. Nikolic regarding his interpretation of that study and its content regarding the enigmatic right precordial T-wave pattern as may be seen in pulmonary embolism. Suffice it to say that the possibility of the pathophysiologic electrical events discussed by the authors as well as those entertained by Dr. Nikolic have a certain degree of merit. I am unable, however, to accept Dr. Nikolic’s statement that, the ‘anterior subepicardial ischemic aspect’ refers to a pattern that is neither subepicardial nor ischemic.” Likewise, his statement, “for most cardiologists, T-wave inversion connotes, if anything, subendocardial rather than subepicardial ischemia, even through subendocardial infarction is no longer a tenable diagnostic entity,” leaves much to be desired.

Unless the basic electrophysiology of myocardial repolarization3 has changed drastically without my realization, there is still a distinction between subepicardial and subendocardial events, even though there are exceptions to the rule. From the outset, I would agree that the term ischemia, as ill-defined, abused, and misleading as it may be, is often used to imply “ischemic-type” T-wave changes, and does not necessarily imply a deficiency in myocardial vascular supply. I would further disagree that “ischemia is an uncommon cause of isolated precordial T-wave inversion.” It may not be the commonest expression of anteroseptal ischemia, but is, nevertheless, a presence to be reckoned

Communications to the Editor