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Echocardiography in Sleep Apnea Patients: A Long Way to Go

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Dear Editor,

Sleep apnea undoubtedly stands among the most influential diseases in cardiovascular medicine. Albeit simply characterized by frequent pauses in breathing or instances of shallow or infrequent breathing during sleep, it usually goes overlooked since the patient is rarely aware of having difficulty breathing, until the sequelae put a big burden on the cardiovascular system.

In their article, Moshkani et al. (1) have taken an in-depth look into echocardiographic findings regarding sleep apnea patients. Fifty-five sleep apneic patients diagnosed by standard polysomnographic study were examined and analyzed in three groups of mild, moderate, and severe sleep apnea, based on the apnea-hypopnea index value (5-15, 15-30, and > 30, respectively). Though exposed to significant confounders and not revealing any information about the clinical findings, the authors have shown very similar echo indices on the left and right ventricles: left ventricular ejection fraction (LVEF); right and left Tei index; pulmonary artery pressure (PAP); and strain and strain rate in a row of right and left ventricular segments.

The authors have also been extremely comprehensive in their discussion, yet there seems to be a great deal missing in looking at the echocardiography of sleep apnea patients. There is a significant body of literature supporting the association between sleep apnea and LV diastolic dysfunction. Sleep apnea literally comes with a state of sympathetic overactivity, with a resultant rise in systemic blood pressure. Obesity, a common comorbidity seen in obstructive sleep apnea, adds to the risk of hypertension and diastolic dysfunction. Many investigators have proved the presence of significant diastolic dysfunction, which is responsive to the treatment of sleep apnea, pointing to the benefits of therapy in terms of cardiovascular morbidity and mortality (2-4).

The impact of sleep apnea on pulmonary circulation is certainly a turning point in the field of pulmonary hypertension. Investigators are nowadays making their ways toward better explanation of pulmonary hypertension in sleep apnea patients. The interesting point is that many sleep apnea patients are found to have significant pulmonary hypertension, yet they are classified as having a mild disease regarding their apnea-hypopnea index, posing the question of coincidence. More important than the true relation between these entities is the dramatic subjective and objective improvement of pulmonary hypertension after implementing the treatment of sleep apnea (5, 6).

Moshkani and colleagues have shown significant differences in strain and strain rate of the basal septal segment between different degrees of sleep apnea. Despite the fact that this can be considered a marker of abnormal septal motion with degrees of right ventricular dysfunction, it certainly needs to be analyzed in the context of other right ventricular contractility indices as well as clinical findings of these patients. Lack of the integration of clinical findings in their study has resulted in significant deviation from what the parameters are truly showing. It is worthy of note that the small sample size of the study compounds this problem. Changes in basal septal segment strain and strain rate after standard sleep apnea treatment could have helped to answer these ambiguities, which unfortunately have not been investigated. Further echocardiographic investigations need to focus on what parameters should be more representative of the impact of sleep apnea on the cardiovascular system, how they should be interpreted in terms of further prognosis, and what constitutes the best therapeutic strategies for these patients.

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