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An Invitation for Rethinking About Echocardiographic Abnormalities in Patients With Sleep Apnea Syndrome

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Dear Editor,

We read with great interest the article entitled "Echocardiographic abnormalities in patients with sleep apnea syndrome", published online in Archives of Cardiovascular Imaging on January 12th, 2014 (1). The authors sought to determine the cardiovascular disorder at an early stage in patients with sleep apnea syndrome (OSA) with different echocardiographic parameters. They found both relative left ventricular systolic and diastolic dysfunction and dysfunction at some echocardiographic parameters of the right ventricle.

We have some comments about this study.

Firstly, there is a major concern about the methodology of the study. The study group consists of male subjects whose disease duration is not known. To our knowledge, the disease is frequently seen in woman patients too (2). Furthermore, the disease duration is not known for the subjects. Thus, comparison of the results is not appropriate: for example, suppose that there is a patient with mild OSA of 10 years' duration and there is also another patient with severe OSA of 3-4 years' duration. The authors have mentioned this point in the limitations section, but it should be written in the patients and methods section.

Secondly, the fact that the study is not the first study in the literature on the subject was not mentioned in the references. Aslan et al. published their work in Cardiology Journal in 2013 (3). They studied 80 patients with the OSA syndrome while excluding those with hypertension, diabetes, or any known cardiac disease, which makes their study more reliable. They divided the patients into two groups of normal/mild OSA group and moderate/severe OSA group. Their study, however, was not as comprehensive as the original study. Tissue Doppler echocardiography showed that early diastolic myocardial velocity was lower in the second group (21 ± 5.6/s in group 1 vs. 18.3 ± 5.3 cm/s in group 2; P = 0.01). They concluded that left ventricular (LV) diastolic dysfunction, hypertrophy, and left atrial dilatation occurred in the OSA patients even before the development of hypertension and other cardiovascular disease. In the original study, strain imaging was used and changes at the basal septum strain rate (P =0.005) were associated with OSA severity. We should note that strain imaging is a new and more reliable method in terms of diastolic function.

Thirdly, Cicek et al. prospectively studied 64 patients in terms of echocardiographic and aortic parameters (4). Their study was not as comprehensive as the present one with respect to echocardiographic examinations. They found that the LV systolic function did not differ between groups, which was a different result from the present study. They also concluded that the LV diastolic function and aortic elastic parameters deteriorate with the severity of OSA. The different result in terms of the LV systolic function might be due to the small number of subjects in both studies.

In conclusion, we thank our colleagues for their effort to encourage us to think about this disease. We believe that the methodology of the study is inappropriate for drawing a conclusion in terms of the LV and right ventricular function and future research is needed to make a strong conclusion. We may obtain better information on the cardiovascular function in OSA patients with another study. Indeed, future research should recruit female subjects and subjects with known disease duration and exclude individuals with hypertension, diabetes, and other cardiovascular diseases.

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