THERE IS GROWING EVIDENCE SUGGESTING THAT OBSTRUCTIVE SLEEP APNEA (OSA) PROMOTES VASCULAR ENDOTHELIAL DYSFUNCTION AND atherosclerosis.¹ OSA stimulates several intermediate pathways that are harmful to the cardiovascular system and includes sympathetic activation, increased production of reactive oxygen species, inflammatory mediators and lipid metabolism. The primary mechanisms by which OSA triggers cardiovascular disease include intermittent hypoxia, generation of negative intrathoracic pressure during occluded breaths and arousals from sleep.² All these disturbances are entangled, and it is difficult to isolate the relative importance of each factor. In this context, we read with much interest the recent work by Lee et al. that provided evidence that snoring should be also regarded as a primary mechanism for the genesis of carotid atherosclerosis.³ The rationale is that the vibration during snoring is transmitted through the surrounding tissues to the carotid artery wall triggering an inflammatory cascade leading to early changes of atherosclerosis. This concept may to some extent parallel the vascular lesions observed in subjects submitted to prolonged exposure to hand-transmitted vibration.⁴ Lee et al. studied 110 participants, with 107 community volunteers and 3 patients undergoing diagnostic polysomnography for suspected OSA.³ The authors found that heavy snoring was significantly associated with carotid atherosclerosis but not with femoral atherosclerosis even after adjustments for potential confounding factors. Although there are important strengths in the Lee study, including the objective measurement of snoring during polysomnography and the evaluation of vascular parameters in the femoral bed (therefore a distant site from the upper airway), we were particularly concerned with the overinterpretation of the data as suggested by the title: “snoring causes carotid atherosclerosis.” First, the study is a cross-sectional design, which makes it impossible to establish a cause-effect relationship between heavy snoring and atherosclerosis. Although there is biological plausibility and a dose-response relationship, it will be necessary to demonstrate that the elimination of snoring reverses carotid atherosclerosis. Second, this study did not involve patients with primary snoring. Instead, there were several patients with OSA (particularly in patients with heavy snoring), and the relative importance of sleep parameters such as apnea-hypopnea index and other hypoxic parameters are not clear in the multivariate logistic regression (Table 2). Third, as acknowledged by the authors, the sample was neither representative of the general population nor of an OSA population, and therefore the results should be interpreted with caution. Finally, the relative impact of snoring on cardiovascular outcomes remains to be established. For instance, in the Spanish cohort study, Marin et al. found no increase risk of fatal and non-fatal cardiovascular events in the subset of 377 patients with primary snoring.⁵

In conclusion, the current relationship between heavy snoring and carotid atherosclerosis is an exciting new area of research. However, the conclusion so far is that there is an independent association between snoring and carotid atherosclerosis. Future treatment studies focusing on patients with primary snoring and the impact of snoring treatment on atherosclerosis of the carotid arteries should be performed in order to clarify this relationship.

DISCLOSURE STATEMENT

The authors have indicated no financial conflicts of interest.

REFERENCES