COMMENTARY

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A Nuisance or Nemesis: The Adverse Effects of Snoring

Commentary on Jin-Gun Cho et al. Tissue vibration induces carotid artery endothelial dysfunction: a mechanism linking snoring and carotid atherosclerosis? SLEEP 2011;34(6):751-757.

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In the report by Cho and colleagues[1] in this issue of *SLEEP*, the authors provide provocative data that simple snoring is no longer a benign complaint or something to be ignored in the absence of obstructive sleep apnea (OSA). The authors have created a novel model of snoring in the rabbit that produces the effect of a "physiologic dose" of snoring vibration. In a single 6-hour exposure to their model of snoring, Cho et al. show impressive reductions in endothelial function, an intermediate in the development of cardiovascular disease (CVD).

It has been 25 years since the original epidemiologic studies first demonstrated the association between self-reported snoring and CVD.[2,3] However, self-reported snoring has been relegated to surrogate status for OSA. The association of snoring with CVD has been attributed to the consequences of OSA and linked to intermittent hypoxemia, sleep fragmentation, and negative intrathoracic pressure swings.[4]

There is no current polysomnography (PSG) standard to objectively measure snoring or its resultant vibration. When snoring has been measured, it is usually done in the prediction of OSA, but not quantified in the way apneas and hypopneas are counted, or in the manner sleep disordered breathing events are quantified. Recent human data have suggested that while snoring has been ignored in the current age of PSG, it may represent a distinct pathophysiologic perturbation of disordered breathing during sleep, with its own cardiovascular implications.[5] Snoring leads to vibration of the upper airway that is transmitted to the carotid artery, possibly resulting in direct vibratory injury to the artery.[6] This insult may be distinct from the pathophysiologic changes described above that are traditionally associated with OSA.[4]

A number of important details of the model of snoring by Cho et al. warrant closer evaluation. Importantly, this model excludes the perturbations of OSA to which the cardiovascular complications of OSA are usually attributed. In prior work, the authors have determined the "dose" of energy associated with snoring in the rabbit.[5] Using a speaker overlying the right neck and the rabbit's carotid artery, they have delivered vibrations at 60 Hz for 6 hours, to replicate a single night of snoring.[1] During the snoring exposure, oxygenation was stable in the tracheotomized rabbits that were anesthetized and mechanically ventilated. Furthermore, the negative intrapleural pressure swings that accompany apneas and hypopneas were eliminated. Lastly, unlike other models of vibratory injury, this more ecologically valid model resulted in reduced endothelial dysfunction in the absence of histologic evidence that endothelial architecture was disrupted.

In the carefully controlled experiments by Cho et al. reported in this issue of *SLEEP*, the external vibration from the speaker that was delivered to the right carotid artery (vRCA) at a level commensurate with snoring was different from the much lower level of vibration measured in the left carotid artery (vLCA).[1] These vibrations resulted in differential effects on the vRCA vs. the vLCA in both biochemical outcomes and functional measures of endothelial function. They showed that acetylcholine (ACH) stimulated cyclic guanosine monophosphate (cGMP) was reduced in the vRCA. This effect was eliminated with the addition of sodium nitroprusside, a nitric oxide (NO) donor, establishing reduced NO bioavailability as a putative mechanism. Then, they demonstrated reduced *ex vivo* ACH stimulated vasorelaxation of the vRCA in comparison to the vLCA and the unvibrated control animals.

In total, these findings suggest that a single 6-hour period of vibration led to impaired endothelial function, in the absence of the usual perturbations of OSA. Consequently, it is possible that snoring represents its own vascular risk in the non-apneic, as well as an additional vascular insult in those with OSA and snoring.

So how do we reconcile the findings of Cho et al.[1] that are at odds with the existing published longitudinal data on snoring and OSA? OSA has been associated with incident stroke only in those with moderate and severe OSA as measured by the apnea-hypopnea index (AHI).[7,8] In a large epidemiologic study from Europe, simple snorers appeared to have no excess cardiovascular risk after 10 years.[9] The fact that snoring has never been objectively measured in the large epidemiologic studies may account for this difference. The reliance on self-reported snoring is inaccurate, particularly in the absence of an attentive bed partner.[10] Objective measures of snoring quantity and characteristics are not part of the major OSA studies in existence. Therefore snoring and its associated vibration could represent an unmeasured covariate (related to the magnitude and duration of the vibratory insult), not equally distributed across the current categories of OSA used in studies. As a result, current epidemiologic studies are unable to answer the question of whether snoring predicts vascular risk. It is quite possible that differential degrees of snoring exist across the AHI distribution, confounding the association between OSA and CVD outcomes.

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The novel data of Cho et al.[1] therefore, have implications for public health and future sleep research. If snoring, independent of OSA, confers excess vascular risk, the public health ramifications are potentially significant. Recent estimates of self-reported snoring in over 68,000 US adults place its prevalence at 48% (47.2–48.8%).[11] This includes nearly 40% of women surveyed. Thus, even a small effect of snoring alone on vascular risk could have large population effects.

In the future, it will be important to clarify the cardiovascular consequences of snoring, independent of OSA severity. Given that a small physiologic vibration, analogous to snoring, in a single 6-hour period, is able to result in endothelial dysfunction, the data of Cho et al. suggest that future studies of OSA should include an objective snoring assessment. The challenge at hand is that we currently do not know the best method for measuring snoring (e.g., microphones, piezoelectric vibration sensors, or airflow oscillations in the nasal pressure signal), or what characteristics of snoring are most relevant to vascular injury (amplitude, frequency, intensity, or duration of exposure). Studies that link quantitatively measured snoring with vascular risk should help to determine the nature of these relationships. We congratulate the authors for advancing the field with their pioneering studies of snoring and vascular risk.

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