Response to Letter by Lavie and Lavie

Response:

We are very grateful to Lavie and Lavie for their polite correction. As they point out, this variable (T90) should be expressed in Table 2 in “percentage of sleep time spent below 90% saturation,” not in seconds.

After revising data referring to T90 variable in both groups, we didn’t find any mistakes (ischemic stroke: median 14.6, SD 18.44, nonischemic stroke: median 16.12, SD 22.51). We think it could be possible to have some explanations for these findings. First, as you can see in Table 2, differences of AHI between 2 groups are significant ($P=0.049$) but very small (ischemic stroke: median 28, SD 17, nonischemic stroke median 20.1, SD 17), so T90 could be similar in both groups. Moreover, median AHI is moderate in severity, and for that reason it is not surprising that T90 is not very high. Second, it is important to remind that we have studied old people with many associated medical conditions not considered in exclusion criteria (as for example moderate chronic obstructive pulmonary disease or chronic bronchitis) that could have influence in respiratory function, and for that reason we think that T90 could have been determined not only by AHI.

Even though hypoxia and reoxygenation phenomena are responsible for oxidative stress and endothelial damage, as Lavie and Lavie have demonstrated, this is probably not the single mechanism involved in the increased vascular risk in obstructive sleep apnea hypopnea (OSAH) patients. For example, it is well know that repeated episodes of apnea and hypopnea predispose to stroke by generating arousals that are associated with repetitive rises in blood pressure, heart rate, and sympathetic nerve activity.1 In addition, episodes of obstructive sleep apnea determine changes in cerebral blood flow and intracranial pressure.2-4 Because multiple and diverse physiopathological pathways can lead to stroke, we should consider different mechanisms for OSAH patients. In this sense, our study suggests that, in elderly people, AHI could be a better stroke risk marker than T90.

For the second question, it is important to highlight that our study was not designed as an incidence study. A crucial difference from the study by Hollander et al5 was that, for practical reasons, we selected our cohort between noninstitutionalized living-home people, excluding people living in homes for the elderly. This could be the main reason of our lower number of incident events, as we assume that we are selecting healthier subjects. We think that inclusion of people with higher degree of disability could have implied people with higher risk to experience cerebrovascular events.

Disclosures

None.

Response to Letter by Lavie and Lavie
Roberto Munoz, Joaquín Duran-Cantolla, Eduardo Martínez-Vila, Jaime Gallego, Ramón Rubio, Felipe Aizpuru and Germán De La Torre

*Stroke*. 2007;38:250; originally published online December 21, 2006;
doi: 10.1161/01.STR.0000254543.50628.2d

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2006 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/38/2/250