In response to questions raised about our study, we would like to make some comments. The literature indicates that there is a bidirectional association between obstructive sleep apnea (OSA) and systemic arterial hypertension (SAH). With regard to the role of SAH and OSA as risk factors for the development of OSA and SAH, respectively, some data merit special attention: OSA as a causal risk factor for the development of SAH has been more extensively studied than has the reverse; in addition, the treatment of OSA with continuous positive airway pressure usually improves blood pressure levels in patients with hypertension. In contrast, the treatment of SAH with antihypertensives in order to improve OSA yields conflicting results; except, perhaps, for the treatment of SAH with diuretics, especially spironolactone, because it reduces parapharyngeal edema and secondary airway obstruction.

With regard to whether or not patients were being treated, we do not believe that this would have influenced the magnitude of association between SAH and OSA, because the main clinical questionnaires that use the variable SAH do not discriminate between patients who are on antihypertensive treatment and those who are not. In our study, SAH was found to be a prognostic factor in the univariate analysis at three different cut-off points—apnea-hypopnea index (AHI) ≥ 5 events/h; AHI ≥ 15 events/h; and AHI ≥ 30 events/h—however, when used in the multivariate analysis, SAH was found to be an independent prognostic factor only at the cut-off point of AHI ≥ 5 events/h, showing that, in the more severe forms of OSA, SAH acts as a confounding factor, which is why it was not used in our questionnaire.

Metabolic syndrome is defined as the presence of three or more of the following five factors: 1) waist circumference > 80 cm in women and > 94 cm in men; 2) serum triglycerides ≥ 150 mg/dL (or on drug treatment for elevated triglycerides); 3) HDL cholesterol < 40 mg/dL in men and < 50 mg/dL in women (or on drug treatment for reduced HDL cholesterol); 4) SAH (or on antihypertensive drug treatment); and 5) fasting glucose ≥ 100 mg/dL (or on drug treatment for elevated glucose). Metabolic syndrome was not used in our questionnaire because its confirmation requires serum level determinations, which would make it impossible to produce an easy-to-administer questionnaire for physicians and patients.

The aim in designing a clinical questionnaire is not to replace polysomnography (PSG). The aim of a questionnaire is to screen patients, selecting those at high risk and enabling faster and cheaper diagnosis with portable monitoring devices. Type I PSG, despite being the gold standard for the diagnosis of OSA, has its inherent costs and is associated with long waiting lists. Since the use of portable methods has been validated in the bariatric population, our questionnaire, specifically developed for this population, can indeed change practice, selecting high-risk patients for home diagnosis, limiting costs, decreasing the waiting time for surgery, and decreasing the waiting time for laboratory PSG.

REFERENCES