

Heart Failure, Obesity, and Sleep Apnea

To the Editor:

The article written by Zamora et al¹ provided an excellent description of the phenomenon known as the obesity paradox in heart failure patients, in this case in a population of heart failure outpatients who had received adequate concomitant treatment for heart failure.

Obstructive sleep apnea–hypopnea syndrome (OSAHS) is highly prevalent among the general population and is related to the development of various cardiovascular complications that determine patient prognosis, among them, ventricular dysfunction, systemic and pulmonary hypertension, cerebrovascular disease, and cardiac rhythm disorders.²⁻⁵ In addition, obesity is the main risk factor of OSAHS, with a direct relation observed between the degree of obesity and the severity

of the sleep disorder. It has been estimated that as many as 40% to 90% of obese patients present OSAHS.⁶ Furthermore, OSAHS is very common in patients with heart failure, both those with left ventricular systolic dysfunction and those with normal systolic function.⁷ There is also evidence that OSAHS in subjects with heart failure is an additional independent predictor of mortality in this population.⁸

Zamora et al¹ did not analyze the possibility of OSAHS in the patients of their series, something also not undertaken in various previous studies that have uncovered the phenomenon of the obesity paradox in heart failure.⁹ In view of the data discussed, it appears plausible to assume that OSAHS diagnosis and treatment in obese subjects with heart failure could widen the differences in mortality compared to nonobese subjects even further, something that would enhance the magnitude of the obesity paradox in heart failure. Evidently, future studies in this field will provide an answer to this hypothesis.

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Response

To the Editor:

We would like to express our appreciation for the comments of Arias et al. As mentioned, our study did not analyze the presence of obstructive sleep apnea-hypopnea syndrome (OSAHS) and its possible relationship with patient mortality. It is true that a potential OSAHS was not actively investigated in a generalized manner; however, the history of this syndrome was compiled from the patients' demographic data despite the fact that such details were not listed in the article. In fact, only 18 patients had been diagnosed with OSAHS (3.6%). An analysis of these patients' data did not show significant differences in terms of 2-year mortality, despite the low number of patients with the syndrome (mortality of OSAHS patients, 16.6%; mortality of OSAHS-free patients, 22.3%; $P=.57$). It is also true that the prevalence of OSAHS was higher among the obese ($BMI \geq 30$): 8.3% versus 1.4% ($P < .001$). A comparison of the mortality rates for obese patients with OSAHS (15%) and obese patients without OSAHS showed similar results (16%) ($P=.94$). Moreover, most of the patients diagnosed with OSAHS were receiving adequate therapy in the pneumology department, although exact figures have not been recorded. The assumption proposed by Arias et al with regard to the differences in mortality is interesting. As far as we are aware, however, it has not yet been shown that continuous positive airway pressure therapy improves the survival of patients with heart failure and OSAHS, even though ventricular function can be improved in some patients.¹⁻³ Therefore, cannot be affirmed that the difference in mortality in favour of obese persons, would have been even higher if all obese individuals with OSAHS had been diagnosed and actively treated. As Arias et al mentioned, the results of future studies are needed to answer this hypothesis.

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