Left Ventricular Dysfunction and Sleep Apnea Syndrome

Cause or Consequence?

To the Editor:

In an article published recently in CHEST (October 2002), Dr. Laaban and colleagues conclude that obstructive sleep apnea syndrome (OSAS) may be a direct cause of daytime left ventricular systolic dysfunction (LVSD) that can reverse following nocturnal apneas by nasal continuous positive airway pressure (CPAP). We would like to take this opportunity to make some suggestions.

First, this finding can not definitely rule out the hypothesis that LVSD and/or congestive heart failure may be the cause of OSAS and not the opposite. Indeed, a high prevalence and persistence of sleep apnea is observed in patients with chronic left ventricular failure. Rather, as CPAP improves cardiac output in patients with LVSD, the useful effect of nasal CPAP observed in the study by Yan et al. could be a direct effect of positive pressure ventilation on heart. Thus, we are not sure about the validity of these results and we may expect that after a time of optimal medical treatment (angiotensin-converting enzyme inhibitors [23% patients treated in the study], diuretics, . . .) the episodes of OSAS may be treated.

Second, the authors have stated that associated cardiac disease had been excluded in the present study. We may expect that this statement may be erroneous, as to our knowledge the onset of an idiopathic dilated cardiomyopathy in patients with LVSD could not be excluded. Finally, we believe that more research is required to better understand the pathophysiologic association between OSAS and LVSD and to define the potential role of CPAP in the treatment of chronic heart failure.

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REFERENCES


To the Editor:

We thank Dr. Bendjelid for his interest in our study. He suggested that obstructive sleep apnea syndrome (OSAS) was not the cause but the consequence of left ventricular (LV) systolic dysfunction and that LV systolic dysfunction could be due to an idiopathic dilated cardiomyopathy. I agree that a high prevalence of OSAS has been reported in patients with chronic LV failure.

However, in our study we observed a normalization of LV systolic function following treatment of OSAS with nasal continuous positive airway pressure (CPAP). There was no confounding factor during the follow-up period. We did not start any medical treatment of LV failure in any of the study patients. Twenty-three percent of the patients with LV systolic dysfunction had been receiving angiotensin-converting enzyme inhibitors for several years as antihypertensive medication. Antihypertensive medications were not modified during the follow-up period. No weight loss that could have improved LV function was observed during the follow-up period.

Dr. Bendjelid stated that the improvement of LV function could be a direct effect of nasal CPAP on the heart, independently of reversal of nocturnal apneas. I agree that nasal CPAP could increase cardiac output in patients with LV failure; however, to my knowledge, a normalization of LV function had not been demonstrated in clinically stable patients with LV systolic dysfunction, and without OSAS, following nasal CPAP.

Therefore, the results of our study strongly suggest that OSAS may be a direct cause of LV systolic dysfunction that can resolve...