
Letter to the Editor

TO THE EDITOR: I read with interest the recent Clinicopathological Conference presenting a patient with constrictive pericarditis (1), and I applaud the reinstatement of this important feature in *The Journal*. However, the discussion contained several inaccuracies pertaining to obstructive sleep apnea syndrome that I would like to correct.

The authors state that “difficulty in sleeping” suggests obstructive sleep apnea syndrome (OSAS), implying that an insomnia complaint should raise suspicion of this disorder. Although it is true that individual obstructive respiratory events typically conclude with electroencephalographic evidence of arousal, these awakenings are usually not remembered by the patient due to their brevity. Obstructive sleep apnea is diagnosed in only a small percentage of patients with an insomnia complaint, averaging only 4.7% in one recent 5-center cooperative study (2). Rather, an insomnia complaint is much more commonly the result of psychiatric illness or of psychophysiological origin (so-called “learned” insomnia).

The authors also imply that shortness of breath is a known consequence of obstructive sleep apnea. Transient dyspnea may sometimes occur in association with nighttime awakenings in the minority of patients who experience sleep-maintenance insomnia from obstructive respiratory events. However, dyspnea at other times is not usually attributable to OSAS, but is more likely a consequence of the obesity that inflicts many of these patients. Obesity is a well-known cause of exertional dyspnea largely because of the increased ventilatory demands imposed by the higher levels of oxygen consumption (VO_2) that occur for any given level of work load in the overweight individual (3). The authors also list abnormal ventilation/perfusion lung scanning as one of the findings in obstructive sleep apnea, but again this finding can be attributed to obesity (due to impaired ventilation of dependent lung units) (4) and is not reported in obstructive sleep apnea alone.

The authors go on to imply that daytime hypercapnia is a frequent complication of obstructive sleep apnea. However, most patients with obstructive sleep apnea syndrome are eucapnic when awake. Estimates of the prevalence of diurnal hypoventilation in obstructive sleep apnea syndrome vary, with published values ranging from 14% to 37% in patients with generally severe disease unselected for degree of obesity (5–7). Patients with OSAS and diurnal hypercapnia tend to be more obese (5–7) and to have coexisting airways disease (5). Daytime eucapnia is achieved in only a subset of patients with OSAS and obesity-hypoventilation syndrome receiving effective treatment for OSAS, indicating that obesity alone is primarily responsible for hypoventilation in some individuals (8). I can find no published report specifically addressing the prevalence of awake hypoventilation in nonobese patients with sleep apnea, but the foregoing considerations suggest that only a small minority will be so affected. Finally, the authors suggest that secondary erythrocytosis is a common finding in obstructive sleep apnea. Polycythemia actually occurs in only a small percent of patients with obstructive

sleep apnea (9); the transient episodes of nocturnal hypoxemia that accompany OSAS are usually insufficient to raise erythropoietin levels to a significant extent (10).

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References

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(The above letter was sent to Dr. Michael Kim, who had discussed the differential diagnosis in the original article. A response has not been received.)