Fluid shift in obstructive sleep apnea: Does it really have no role in the pathogenesis?

To the Editor:

The study by Jafari and Mohsenin published in a recent issue of CHEST (October 2011) tested the effect of nighttime rostral fluid shift on the development of obstructive sleep apnea (OSA) and its severity. The strengths of their study were stringent methodology and statistical processing of the received data. In contrast to some prior works, they did not find any effect of fluid shift on sleep disordered breathing severity. The studied population was likely free of clinically significant excessive fluid, evidenced by the absence of limb edema. Only three individuals received a diagnosis of heart failure (HF), which was mild and likely clinically compensated. However, most patients with OSA were obese and had thicker necks, which is known to be an OSA risk factor. The authors concluded that fluid shift unlikely has any role in the pathogenesis of OSA.

Some issues are noteworthy. First, it is well known that OSA is a risk factor for arterial hypertension (AH), kidney disease, and HF. That, in turn, may result in fluid retention. Second, several studies have shown a detrimental effect of fluid shift on OSA severity in comorbid conditions like HF. However, most patients with OSA were obese and had thicker necks, which is known to be an OSA risk factor. The authors concluded that fluid shift unlikely has any role in the pathogenesis of OSA.

It is likely that in the early course of OSA rostral shift may not play a major role, but with the time needed for OSA complications to develop (eg, AH and HF), it may further worsen apnea-hypopnea index and, in turn, complicate the course of comorbid disorders, making a vicious cycle. Therefore, it seems that rostral fluid shift likely plays a role in the pathogenesis of OSA and its severity.

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Response

To the Editor:

We appreciate the interest of Dr Mirrakhimov in our recent study in CHEST investigating the role of rostral fluid shift in the pathogenesis of obstructive sleep apnea (OSA).1 We enrolled 135 patients who had been referred for sleep-disordered breathing. However, we only included those who had a rostral fluid shift with increased neck circumference in the study population. The OSA group consisted of typical patients with OSA with a mean age of 52.5 years, marked obesity with a BMI of 36.2 kg/m², and wide ranging OSA severity. Hypertension and heart failure were present in 47% and 9%, respectively, of the patients with OSA. Despite significant fluid shift into the neck in both patients with OSA and control subjects without OSA, there was no increase in the apnea-hypopnea index or the breathing disturbance index (which, in addition to apnea and hypopnea, includes respiratory effort-associated arousals). We did not specifically examine the influence of rostral fluid shift in subjects with a gross edematous state. As we mentioned in our article,1 we cannot exclude some contribution of fluid shift in a worsening of OSA in this population. But this is far from suggesting rostral fluid shift as a significant mechanism in the pathogenesis of OSA in the majority of these patients.

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