Abnormal Breathing During Sleep and Increased Central Chemoreflex Sensitivity in Congestive Heart Failure

To the Editor:

The data of Narkiewicz et al1 expand our understanding of the importance of the imbalance between enhanced central versus peripheral chemoreflex sensitivity in the pathophysiology of congestive heart failure. The authors comment on the links between central cardiovascular and respiratory control, but despite this, patients in this study were not assessed for sleep apnea, which may have confounded their study conclusions.

It is known that ≈50% of patients with stable congestive heart failure have associated sleep-disordered breathing, predominantly central sleep apnea (CSA), whereas a minority have obstructive sleep apnea (OSA). We have previously shown that hypercapnic ventilatory responses are increased in patients with CSA and left ventricular dysfunction,2,3 but hypoxic responses are normal. In contrast, patients with OSA have relatively normal central and peripheral chemoreflex responses whether they have congestive heart failure or not.3,4

Increased central chemosensitivity is associated with hypocapnia during the awake state and unstable breathing during sleep. Although the etiology of this enhanced central chemosensitivity is not known, hyperventilation occurs as a consequence of this and is accompanied by increased sympathetic activity. Naughton et al5 reported that treatment of central sleep apnea in congestive cardiac failure with nocturnal continuous positive airway pressure reduced awake sympathetic nerve activity.

Thus, it does appear likely that the increased sympathetic efferent activity that characterizes left ventricular systolic dysfunction and congestive heart failure may be due in part to the interaction between abnormal breathing during sleep and increased central chemoreflex sensitivity.

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Response

We appreciate the interest of Drs Wilcox and McNamara in our work. We do not think that the enhanced central chemoreflex sensitivity in our patients was secondary to occult central or obstructive apnea for the following reasons. First, 4 of our heart failure patients underwent sleep studies, which ruled out sleep-disordered breathing. Ventilatory and autonomic responses to hypercapnia were markedly increased in these subjects. Second, in all subjects, we evaluated respiratory patterns over 2 hours of supine rest. None of the patients had evidence of apneas or hypopneas. Third, there was no difference in baseline CO2 between heart failure patients and controls in our study, whereas Wilcox et al4 demonstrated that patients with central sleep apnea had low CO2 during wakefulness. Fourth, in contrast to findings in patients with heart failure, patients with obstructive sleep apnoea who are free of any other diseases have enhanced peripheral but not central chemoreflex sensitivity compared with closely matched controls.2

Wilcox and McNamara make important points regarding the complex interaction between heart failure, chemoreflex function, and sleep-disordered breathing. Chemoreflex function in heart failure may be influenced by several factors, including species,3,4 the origin of the heart failure,3,4 and the presence of coexisting disease states. Furthermore, it has also been proposed that enhanced sensitivity to CO2 may contribute to the development of central sleep apnoea in patients with heart failure.5

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