Sleep-disordered breathing in congestive heart failure: an opportunity missed?

The review by Vazir and colleagues in this issue of the journal (see pages 219–23) comes as a timely and practical update on the implications, diagnosis and treatment of sleep-disordered breathing (SDB) in congestive heart failure (CHF).

Despite advances in the treatment of CHF, it remains a leading cause of morbidity and mortality. An understanding of the factors responsible for the inexorable progression of heart failure is essential to facilitate the development of new therapies. The identification of specific subgroups of CHF patients such as those with intra-ventricular conduction delay[1] allows the tailoring of therapy for patients with proven benefits.

Undiagnosed SDB is a common problem affecting 24% and 9% of men and women while obstructive sleep apnoea hypopnoea syndrome (OSAHS) affects 2% and 4%, respectively.[2] The prevalence of sleep-disordered breathing is however much higher in the CHF population, affecting 51 to 68% of patients.[3,4] Within these cohorts the contribution of OSAHS to SDB was significantly higher than in a non-CHF population, affecting 12 to 32% of patients, the remainder being due to Cheyne-Stokes respiration-central sleep apnoea syndrome (CSR-CSA).

Cardio-respiratory effects

Sleep-disordered breathing has multiple adverse cardio-respiratory effects even in the absence of CHF.

Repeated obstructive events are associated with oscillations of negative intrathoracic pressure, with consequent increased venous return and increased cardiac transmural systolic pressures, ultimately increasing afterload and compromised cardiac output. Increased sympathetic activity occurs as a consequence of apnoea (which causes loss of the pulmonary stretch-receptor mediated reflex, normally responsible for suppressing central sympathetic discharge) and of the effects of hypoxia and hypercapnia on central and peripheral chemoreceptors.[6,7] The results are increases in heart rate, vasoconstriction and raised peripheral vascular resistance.

Obstructive sleep apnoea hypopnoea syndrome results in increased left ventricular wall thickening in normotensive subjects,[8] is an independent risk factor for hypertension[9] and furthermore is associated with depressed left ventricular function in subjects without CHF.[10] Additional negative cardiovascular effects of OSAHS are insulin resistance[11] and endothelial dysfunction, as evidenced by elevated levels of plasma C-reactive protein (CRP) in comparison to controls.[12]

Pathogenesis

Though the pathogenesis of CSR-CSA in CHF is now well understood, that of OSAHS in CHF is less clear. The studies citing a high prevalence of OSAHS in CHF contain subjects who are classified only as overweight to mildly obese.[3,4] Increased fluid shift at night during supine positioning with central pooling involving the upper airway tissues could affect oropharyngeal diameter and be of significance.[13]

Treatment

There is now clear evidence, on the basis of randomised controlled trials, for the beneficial effect of treatment of CSR-CSA in CHF with continuous positive airway pressure (CPAP).[14,15] Though these studies contain small numbers of patients and follow-up is considerably shorter than the randomised controlled trials of beta blockade[16] and ACE inhibition[17] in CHF, they demonstrate comparable significant improvements in indices of cardiac function, symptomatology and markers of adrenergic activity for this specific subgroup of CHF patients.

Studies of the treatment of OSAHS have demonstrated increments in ejection fraction in the absence of CHF or other cardiac diseases.[10,18] These findings have been confirmed and extended by the first landmark randomised controlled trials of CPAP in OSAHS associated with CHF.[19,20] Again, even though the numbers are small (55 and 24), treatment for one and three months resulted in an absolute mean increment in left ventricular ejection fraction of 5%[19] and 8.8%,[20] respectively. Treatment with CPAP was associated with a reduction in mean heart rate, mean systolic daytime blood pressure, left ventricular end systolic dimensions[20] and overnight norepinephrine excretion.[19] Further larger scale trials of longer duration are required (such as the ongoing and eagerly awaited CANPAP trial[21]) to clarify the role of CPAP therapy for this subgroup of patients precisely.

It is important that CHF patients are questioned about the symptoms of sleep-disordered breathing and investigated if necessary. There is now good evidence that this subgroup of CHF patients may benefit significantly from appropriate therapy. In time, larger and longer-term randomised controlled trials will clarify the mortality benefits of this emerging therapy.

Conflict of interest

None declared.

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