PRO/CON DEBATE

Routine Polysomnography is Indicated in Congestive Heart Failure

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Congestive heart failure (CHF) is a common disorder, often quite difficult to diagnose, and usually treated intensively with expensive drugs, costly gadgets and dedicated saintly staff until the patient's untimely and premature death at ~ 5 years. The main symptoms of such patients with CHF are dyspnea and cough (usually at night and paroxysmal in nature). If one looks at the sleep patterns in detail, many (~60%) patients with CHF have significant sleep disordered breathing (SDB), probably the cause of their symptoms. Moreover, these changes in breathing pattern during sleep can resolve with standard medical treatment directed at the failing heart. "Alternative" treatments (such as positive airway pressure and oxygen therapy) to standard medical therapy can be instituted (and accurately titrated) with polysomnography. Thus, I would argue that diagnostic polysomnography should be a standard investigation for all patients with CHF.

Heart failure is at epidemic proportions. It is estimated about 5 million Americans have diagnosed CHF and many more remain undiagnosed.[1] The prevalence is estimated to have doubled in past 10 years.[2] The largest and most detailed assessment of the prevalence of CHF in a community dwelling population indicates that about a quarter of the adult and elderly population have some degree of cardiac failure.[3] Based on this study, in which detailed echocardiographic studies of 2043 community dwelling people aged >45 years were analysed, 21% had mild diastolic dysfunction, 7% moderate to severe diastolic dysfunction, 5% mild systolic dysfunction and 1% had severe left ventricular systolic dysfunction.

Heart failure is not only a very costly disease, but is also currently out of control. CHF is the primary discharge diagnosis for acute hospital admissions in at least 20% of all admissions for people over 65 years, a rate that has increased by 159% in 10 years.[1] In 1997 in the USA, it was estimated that ~$5,500 was spent per admission with heart failure and an additional $1,700 per month following discharge,[2] which equates to about $40 billion per annum. In Australia, it is estimated that 12% of the healthcare budget is spent on cardiovascular disease (Health Insurance Commission, personal communication).

Heart failure is a high mortality condition. Despite a multitude of treatment options, heart failure carries a mortality equivalent to several solid organ malignancies. The 5 year mortality is estimated to be ~50% with left ventricular (LV) systolic[4] and ~25% with LV diastolic dysfunction.[3] Despite these alarming statistics, the improvement in survival over the past 40 years has been very small.[4] A non-uniform night time distribution of cardiac events[5] and of death due to heart failure[6] have been observed, suggesting that events during sleep might impact upon awake cardiac performance.

Although the main symptom of CHF is dyspnea, initially on exercise and during sleep, it is not specific to CHF. Furthermore ancilliary and standard clinical signs of CHF unfortunately are unreliable. In a study[7] of 50 patients with a wide variety of severity of documented CHF (left ventricular ejection fraction [LVEF] <45% and mean group LVEF 18%), undergoing right heart catheterisation, the classical clinical signs of CHF correlated poorly with an objective marker of CHF (i.e. pulmonary capillary wedge pressure). An elevated jugular venous pressure, peripheral oedema or the presence of pulmonary crepitations was observed in only 50%, 20% and 16% of patients respectively. The best predictors of CHF were found to be the presence of a third heart sound (96%) and the presence of orthopnea (78%). Polysomnogaphy is the best test to investigate symptoms of orthopnea and paroxysmal nocturnal dyspnea, particularly when there is doubt as to the degree of cardiac failure.

Mechanistically, CHF can be divided into LV diastolic failure, in which there is failure of relaxation and filling, and LV systolic failure in which there is a failure of contractile properties. The importance of LV diastolic dysfunction is highlighted by the observation that 50% of all patients with acute cardiogenic pulmonary oedema have normal LV systolic, but significantly impaired LV diastolic function.[8]

Sleep disordered breathing occurs in ~60% of patients with CHF. In patients selected on the basis of detailed echocardiographic evidence of LV diastolic dysfunction,[9] 55% of the group had SDB. In patients with LV systolic dysfunction, the prevalence of SDB has ranged between 51 to 70%.[10-12] Although the studies can be criticised for being either small or selective, one study was unselected for presence of SDB and the prevalence was 59%.[11] Thus a strong message is coming through that SDB is common in patients with CHF.

Sleep disordered breathing is likely to be a cause of LV systolic and diastolic dysfunction. Nocturnal hypoxia and tachycardia are

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commonly associated with impaired contractility and relaxation.[13,14] Large negative intrathoracic pressures fluctuations from SDB tend to distort and dilate the left ventricle.[15,16] Swings in sympathetic activity result in elevations in peripheral vascular resistance and endothelial damage. These effects occur at a time when cardiac function is also at its lowest ebb;[17] LVEF drops from wakefulness to sleep from 55 to 51%.

Can SDB in CHF be identified based upon history? There is no evidence to show this is the case. Indeed, with the re-analysis of our data[11] in which 48 patients referred by cardiologists with severe CHF for transplant assessment who all had detailed cardiac and polysomnographic assessment plus the Epworth Sleepiness Score (ESS),[18] we were unable to show any significant correlation between ESS and pulmonary capillary wedge pressure (PCWP) (R^2=0.06), apnea-hypopnea index (AHI) (R^2=0.00) or LVEF (R^2=0.04).

Does the identification of SDB confer an effect upon prognosis? Based upon limited data, the answer is likely to be yes. At least 6 studies[19-24] have now assessed mortality in 318 patients over a mean follow-up period of 41 months. The most recent data[24] from our laboratory (78 patients followed over 51 months) found a step wise reduction in survival at 500 days in the no apnea, obstructive and central apnea groups. However at the termination of this uncontrolled and unblinded study, survival curves drifted together. One interpretation was that the identification and treatment of SDB brought the survival curves together.

Does the identification of SDB alter management? There are now several medium term trials that support the use of positive airway pressure in patients with SDB. In the case of patients with predominantly OSA, the studies by Kaneko[25] and Mansfield[26] highlight the improvements in objective and subjective markers of CHF. Cumulative results revealed highly significant improvements in various aspects of quality of life (general and disease specific) in addition to objective markers such as LVEF, systemic blood pressure, reductions in awake resting end-diastolic and end-systolic LV chamber size, reductions in urinary noradrenaline excretion.

Central sleep apnea is a condition characterised by waxing and waning hyperventilation during the lighter levels of sleep associated with worse cardiac systolic function, higher PCWP,[11] greater LV chamber size,[27] greater cardiac and total body norepinephrine activity[28] and greater ventilatory responses to chemical stimuli.[29] The response to positive airway pressure has been shown to improve cardiac function (symptoms and objective markers),[30] via mechanisms of reduced left ventricular transmural pressure,[31] size,[32] mitral regurgitation[33] and cardiac work,[34] reduced respiratory work,[35] increased lung volume, stabilisation of upper airway, assistance of inspiratory respiratory pump muscles, and reduced systemic vascular resistance and blood pressure.[36] An attenuation of hyperactive chemosensors due to a reduction in sympathetic activity and a reduction in arousal frequency and stabilization in sleep stage[37] may explain the rise in PaCO2 and associated stabilization of ventilation.

In contrast to patients with OSA in whom the AHI and snoring on polysomnography is used as a guide to continuous positive airway pressure (CPAP) titration, patients with CSA have CPAP titration monitored against comfort, orthopnoea, cardiac function. In time, the frequency of central events falls as cardiac function improves.[11, 31]

More recently (and possibly following the cardiac pacemaker trend), positive airway pressure devices have incorporated servo controlled mechanisms that allow minute ventilation to be tracked and maintained at ~85% during the central apnea[38] with associated improvements in sleep quality, reductions in arousals, and elevations in CO2 levels during sleep (presumably due to greater slow wave and REM sleep and less arousals).

Correction of any hypoxemia can be also titrated with supplemental oxygen during polysomnography, with caution to avoid hyperoxia. Although large studies of the benefits of supplemental oxygen are limited, some[39, 40] evidence exists of a reduced CSA AHI (presumably via reduced activity of peripheral chemosensitivity) and associated rise in prevailing PaCO2 and thus stabilization of ventilation. Avoidance of hyperoxia is likely to be equally important, as too much of a good thing, may result in impaired cardiac function as has been shown in two recent studies.[41, 42]

Finally, given that there is a correlation between CSA AHI and the PCWP, polysomnography may be the best investigation to follow-up the response of patients with CHF to treatments which include both standard (digoxin, beta blockers, diuretics, rehabilitation), to the extravagant (biventricular pacing, transplantation, left ventricular assist devices) and alternative treatments (positive airway pressure or oxygen). Recently, atrial pacing has been used for patients with CHF and sleep apnea (possibly via improved chronotropic performance) and again polysomnography should be used to assess the response.[43]

Thus it would appear cogent to determine SDB type with polysomnography. Can this be done with simple screening tools, such as oximetry or cardiorespiratory monitoring? Differentiation of obstructive from central SDB does require knowledge of relationship of several aspects of the polysomnogram including sleep stage, body position, placement of arousal in relation to ventilation, transcutaneous PCO2 levels and supervision to record height of pillows which are not always available using simpler monitoring techniques.

In summary, routine polysomnography is required in patients with CHF in order to classify presence and type of SDB, plus response to treatment.

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