

# Sleep-Disordered Breathing Occurs Frequently in Stable Outpatients With Congestive Heart Failure\*

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**Background:** Sleep-disordered breathing (SDB) has a potential role in the pathogenesis of congestive heart failure (CHF). High rates of central sleep apnea (CSA) are found in patients with severe CHF, and equal proportions of obstructive sleep apnea (OSA) and CSA are found in CHF patients referred to sleep clinics. The prevalence, type, and severity of SDB in unselected stable outpatients with CHF are unknown.

**Study objectives:** To determine the frequency and type of SDB in stable CHF outpatients and to examine the relationship between indexes of SDB and impaired cardiac function.

**Participants:** Fifty-three of 87 eligible outpatients (left ventricular ejection fraction [LVEF] < 45%) were predominantly male (77%), with an average age of 60.1 ± 9.8 years, mean body mass index of 27.9 ± 5.3 kg/m<sup>2</sup>, and mean LVEF of 34.0 ± 8.5% (± SD).

**Measurements:** Polysomnography, clinical questionnaire, echocardiography, urinary catecholamines, and amino-terminal fragment of pro-brain natriuretic peptide (NT-BNP).

**Results:** SDB (apnea-hypopnea index >10 events/h) was demonstrated in 36 patients (68%) including two subgroups: OSA (n = 28, 53%) and CSA (n = 8, 15%). SDB was associated with atrial fibrillation (0% vs 28%, p = 0.02), more severe oxyhemoglobin desaturation (percentage of time with oxygen saturation < 90%: 0.4% vs 7.9%, p = 0.003), sleep disruption (p = 0.003), and higher urinary noradrenaline levels (p = 0.013) in OSA patients and CSA patients, respectively. Subjective sleepiness (Epworth sleepiness scale, 7.5 vs 8.5; p = 0.11), indexes of impaired cardiac function including Minnesota Living With Heart Failure Questionnaire scores, shuttle walk distance, and NT-BNP levels were not related to the presence of SDB (p > 0.05). CSA patients had lower LVEF (p = 0.0013).

**Conclusions:** SDB is very common in stable outpatients with CHF, and in our sample OSA predominates. Atrial fibrillation and severe left ventricular impairment increased the likelihood of SDB (particularly CSA), whereas symptom severity, subjective daytime sleepiness, exercise capacity, and NT-BNP levels did not. If specific therapy for SDB such as continuous positive airway pressure can be shown to improve major cardiovascular end points, these results support screening of clinically stable CHF patients. (CHEST 2005; 128:2116–2122)

**Key words:** brain natriuretic peptide; congestive heart failure; sleep apnea

**Abbreviations:** AHI = apnea-hypopnea index; BMI = body mass index; BNP = brain natriuretic peptide; CHF = congestive heart failure; CI = confidence interval; CSA = central sleep apnea; CSR = Cheyne-Stokes respiration; ESS = Epworth sleepiness scale; LVEF = left ventricular ejection fraction; MLHF-Q = Minnesota Living With Heart Failure Questionnaire; nCPAP = nasal continuous positive airway pressure; NT-BNP = amino-terminal fragment of pro-brain natriuretic peptide; NYHA = New York Heart Association; OSA = obstructive sleep apnea; SDB = sleep-disordered breathing

Congestive heart failure (CHF) is a common and serious problem in Western societies,<sup>1–3</sup> and despite advances in pharmacotherapy CHF contin-

ues to cause a significant burden of morbidity and mortality.<sup>2,4,5</sup> Sleep-disordered breathing (SDB), with recurrent episodes of apnea (cessation of

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breathing) and hypopnea (decreased breathing), occurs in the general population<sup>6,7</sup> and with increased frequency in subgroups of patients with heart failure.<sup>8–10</sup> It has been argued that SDB may be a consequence of CHF, with evidence that indexes of central sleep apnea (CSA) and possibly obstructive sleep apnea (OSA) improve with measures to improve cardiac function.<sup>11,12</sup> Conversely, there is evidence that SDB may contribute to the progression of CHF by a number of mechanisms, including recurrent myocardial hypoxemia, increased oxygen demand, and sympathetic activation, with a higher morbidity in those with SDB than those without.<sup>13,14</sup> Preliminary studies<sup>15–19</sup> of small numbers of patients showed treatment of SDB with nasal continuous positive airway pressure (nCPAP) is associated with improved echocardiographic measures of cardiovascular function and reduced sympathetic activation.

A limited number of studies have examined the prevalence of SDB in heart failure populations. Early studies<sup>18,20–22</sup> of small numbers of patients with moderate-to-severe disease or decompensated CHF showed high rates of SDB, predominantly CSA-Cheyne Stokes respiration (CSR). In the largest series,<sup>10</sup> 450 patients were recruited from a population who had been referred to a sleep unit for investigation of suspected sleep breathing problems and therefore did not include those without symptoms suggestive of SDB. Javaheri et al<sup>23</sup> studied an unselected CHF population but recruited male patients only; although the patients were ambulatory, the average left ventricular ejection fraction (LVEF) was low in both those with and without SDB ( $22 \pm 8\%$  and  $27 \pm 9\%$ , respectively).

Cardiac peptides have received attention as markers of cardiovascular disease and have been shown to be independent markers of both cardiac status and prognosis in patients with CHF.<sup>24,25</sup> Plasma atrial natriuretic peptide and brain natriuretic peptide (BNP) levels are mildly elevated in OSA patients during sleep and drop with continuous positive airway pressure therapy.<sup>26</sup> In normal subjects, the plasma concentrations of BNP and the amino-terminal fragment of pro-BNP (NT-BNP) are similar; however, in patients with left ventricular dysfunction, NT-BNP rises more than BNP, suggesting that NT-BNP may be a more sensitive marker of cardiac failure.<sup>27</sup> NT-BNP has prognostic significance in patients with ischemic cardiomyopathy and following myocardial infarction.<sup>28,29</sup> To our knowledge, no previous studies have compared cardiac hormone levels in patients with both CHF and SDB.

Research<sup>30</sup> suggests that the New Zealand adult population has a similar burden of SDB when compared to subjects in overseas studies, but that the indigenous Maori population has a higher prevalence

of OSA. The prevalence of SDB in the New Zealand heart failure population is unknown.

The aims of the study were to determine the frequency of SDB, both OSA and CSA, in a population of stable CHF outpatients (both male and female), and secondly to examine the relationship between SDB and indexes of cardiac function (echocardiography, symptoms, exercise tolerance, sympathetic activation, and plasma NT-BNP). We hypothesized that in a group of heart failure patients, the presence and magnitude of SDB would be related to commonly used markers of impaired cardiac function.

## MATERIALS AND METHODS

### Patients

Fifty-three stable outpatients (41 men and 12 women) with heart failure due to systolic dysfunction were studied. Patients were recruited on the basis of a diagnosis of heart failure and an initial echocardiogram showing LVEF < 45% from cardiology outpatient clinics, either from a designated CHF clinic database ( $n = 26$ , referred by the patient's family practitioner) or from patients attending for echocardiography with CHF during the recruitment period ( $n = 27$ ). Diagnosis and clinical stability on optimal therapy, with no change in medication in the 4 weeks prior to study, were confirmed by a cardiologist.

Exclusion criteria included unstable angina, primary valvular and congenital heart disease, primary pulmonary hypertension, and intrinsic pulmonary disease, including interstitial lung disease or obstructive lung disease (FEV<sub>1</sub>/FVC ratio < 65%), significant liver or renal disease, and daily use of theophylline, benzodiazepines, or morphine derivatives. At the time of recruitment, no information was sought regarding symptoms or risk factors for sleep apnea. The study was approved by the Wellington Ethics Committee.

### Study Protocol

After giving written informed consent, patients were admitted to WellSleep, Sleep Investigation Centre, Wellington School of Medicine for 24 h (10 AM to 10 AM). Medical history, physical examination, determination of New York Heart Association (NYHA) class, along with an in-house sleep questionnaire including Epworth sleepiness scale (ESS) score<sup>31</sup> were completed by a medical practitioner (B.Y./K.F.). Quality of life was assessed using the Minnesota Living With Heart Failure Questionnaire (MLHF-Q).<sup>32,33</sup> Spirometry (Microloop 3535; MicroMedical Ltd; Rochester, Kent, UK) to determine FEV<sub>1</sub>, FVC, and FEV<sub>1</sub>/FVC ratio, and arterial blood gas sampling (i-STAT Portable Clinical Analyzer; i-STAT Corporation; East Windsor, NJ) while breathing room air were performed. An incremental shuttle walking test was used to assess functional capacity.<sup>34–36</sup>

Twenty milliliters of venous blood was sampled after 1 h in a semirecumbent position for cardiac natriuretic peptides at 4 PM, centrifuged at 5,000 revolutions per minute for 10 min, plasma divided, and stored at  $-40^{\circ}\text{C}$  before being analyzed at the Cardioendocrine Unit at Christchurch School of Medicine. Extracted NT-BNP was assayed by radioimmunoassay with a mean detection limit of 1.7 pmol/L after extraction.<sup>27</sup>

Echocardiography was performed by a single operator blinded

to the results of other investigations (Vivid 3; GE Medical Systems; Chalfont St. Giles, UK; using 2.1 E software). Echocardiography measured parameters of systolic left ventricular function. Biplane methods of disk left ventricular volume measurements were used to calculate ejection fraction.

Urine was collected over 24 h in containers acidified with 20 mL of 6 mol/L hydrogen chloride and stored at 4°C prior to analysis. Urinary catecholamines were measured by high-performance liquid chromatography with electrochemical detection. Urine samples were first prepared using a column cation-exchange procedure followed by alumina adsorption. High-performance liquid chromatography was accomplished with ion pair, reverse-phase chromatography and electrochemical detection (ESA Coulochem 5100A; ESA Biosciences; Chelmsford, MA). Results are expressed with reference to creatinine excretion.

Comprehensive polysomnography over a single night (S series Sleep System; Compumedics P/L; Melbourne, Australia) was performed, measuring arterial oxygen saturation (SatLite Trans; Datex-Engstrom; Helsinki, Finland); heart rate; oronasal airflow (thermistor); nasal pressure; thoracoabdominal movement (piezo-electrical bands); body position (mercury switch transducer); sleep (EEG, electromyography, electro-oculography [Grass gold cup electrodes; Grass-Telefactor; West Warwick, RI]); sound (Rion integrating sound meter; Rion; Tokyo, Japan); leg movements (piezo-electrical sensors); and transcutaneous CO<sub>2</sub> (TCM3; Radiometer; Copenhagen, Denmark). The presence or absence of sleep apnea was determined from polysomnographic analysis (Replay v2.0; Compumedics P/L) with manual scoring of sleep staging and respiratory events by an experienced sleep technologist (A.C.).

An apnea was defined as a clear decrease (> 50%) from baseline in the amplitude of a valid measure of breathing during sleep (either nasal pressure or two of airflow, thoracic movement, and abdominal movement), lasting ≥ 10 s. A hypopnea was defined as a clear amplitude reduction of a validated measure of breathing during sleep lasting ≥ 10 s that does not reach the above criterion but is associated with either an oxygen desaturation of > 3% or an arousal.<sup>37</sup>

CSA was distinguished from OSA by a cessation of both airflow and respiratory effort. An arousal was defined as the appearance of α waves on the EEG for at least 3 s in duration.<sup>38</sup> The number of episodes of apnea plus hypopnoea per hour of sleep is referred to as the apnea-hypopnea index (AHI). Diagnosis was confirmed by an experienced sleep physician (A.N.).

#### Classification of Patients

As with previous studies,<sup>21,39,40</sup> an AHI threshold of 10 events/h was used to define the presence of SDB: group 1 (AHI < 10 events/h) and group 2 (AHI > 10 events/h). Group 2 patients were further classified into those with OSA (group 2 [obstructive] > 50% events obstructive) or CSA (group 2 [central] > 50% events central) by an experienced sleep physician.

#### Statistical Analysis

Appropriate parametric and nonparametric tests were used to assess significant differences of cardiac and sleep variables between groups 1 and 2. We have included these data for interest to the reader but acknowledge that no allowance has been made for the multiple statistical tests performed. A value of  $p < 0.05$  was considered significant. Values are reported as mean ± SD. The relationship between LVEF and category of sleep apnea type was analyzed by analysis of variance. The relationship between LVEF and AHI was analyzed by simple regression. The relation-

ship between MLHF-Q and shuttle test, and LVEF and SDB was analyzed by multivariate analysis of covariance using MLHF-Q and shuttle test as response variables and LVEF and SDB as predictor variables. Analysis of covariance was used to examine the relationship between NT-BNP and LVEF and the presence of SDB. The logarithm of NT-BNP was used in the analysis to meet normality assumptions.

## RESULTS

Of the 87 subjects identified by initial recruitment, 53 agreed to participate: 41 men and 12 women. The main reason for refusing to participate was an unwillingness to spend 24 h in the hospital. Nonparticipants were not different from participants in terms of age, gender, and LVEF.

The demographic characteristics, baseline symptoms, and measures of cardiac function for the 53 study patients are shown in Table 1. Patients were predominantly male (77%) with an average age of  $60.1 \pm 9.8$  years, overweight (body mass index [BMI]  $27.9 \pm 5.3$  kg/m<sup>2</sup>), and had mild-to-moderate heart failure with an average LVEF of  $34.0 \pm 8.5\%$  (range, 8 to 45%); 75% of patients were NYHA class 1 or 2. Excessive daytime sleepiness (ESS > 8) was reported by 47% (ESS range, 0 to 18 of 24 total), and 55% snored. The study population included 28% Maori or Pacific Islanders (compared with Wellington population census, 12.9%).

Polysomnography demonstrated SDB in 36 patients (68%; group 2). This included subgroups with predominantly OSA ( $n = 28$ , 53%) and CSA ( $n = 8$ , 15%). Seventeen subjects (32%) did not have SDB (group 1). Those with SDB were heavier, had larger neck sizes ( $p < 0.05$ ), and the expected differences in polysomnographic indexes of SDB (Table 2).

**Table 1—Characteristics of Heart Failure Patients ( $n = 53$ )\***

Variables	Data
Age, yr	$60.1 \pm 9.8$ (35–79)
Male gender, %	77
Ethnicity (Maori or Pacific Islander), %	28
BMI, kg/m <sup>2</sup>	$27.9 \pm 5.3$ (19.8–42.8)
Neck circumference, cm	$41.0 \pm 4.1$ (33–52)
Snoring (often or always), %	55
ESS score (total of 24)	$8.2 \pm 4.4$ (0–18)
NYHA class 1 or 2, %	75
MLHF-Q (total of 105)	$40.8 \pm 24.6$ (0–105)
Shuttle test, m	$442.5 \pm 237.5$ (20–1,020)
FEV <sub>1</sub> , %	$84.1 \pm 16.5$
FVC, %	$82.3 \pm 13.3$
FEV <sub>1</sub> /FVC ratio	$82.3 \pm 8.8$ (65–115)
LVEF, %	$34.0 \pm 8.5$ (8–45)
NT-BNP, pmol/L	$120.3 \pm 151.9$ (5.7–1,020)

\*Data are presented as mean ± SD or mean ± SD (range) unless otherwise indicated.

**Table 2—Sleep and Demographic Characteristics in Patients Without SDB (Group 1) or With SDB (Group 2)\***

Variables	Group 1 (n = 17)	Group 2 (n = 36)
Male gender	64.7	83.8
Ethnicity (Maori or Pacific Islander)	29	25
Weight, kg	78.1 ± 14.4	88.7 ± 19.2†
BMI, kg/m <sup>2</sup>	26.9 ± 3.9	28.4 ± 5.9
Neck circumference, cm	39.4 ± 3.7	41.7 ± 4.1†
Diagnosis of hypertension, %	58.8	50
ESS	7.5 ± 3.6	8.5 ± 4.7
PO <sub>2</sub> awake, mm Hg	83.2 ± 11.1	76.0 ± 12.1†
PCO <sub>2</sub> awake, mm Hg	40.0 ± 3.3	40.0 ± 7.3
HCO <sub>3</sub> awake, mmol/L	25.6 ± 2.6	25.6 ± 2.4
Sleep efficiency, %	67.9 ± 14.3	67.5 ± 12.7
Rapid eye movement sleep, min	57.9 ± 27.4	56.4 ± 28.4
Non-rapid eye movement sleep, min	244.6 ± 63.9	252.4 ± 70.3
Arousals, events/h	21.0 ± 11.0	33.0 ± 15.9†
AHI, events/h	4.4 ± 3.0	31.3 ± 18.8†
Central apnea, events/h	0.3 ± 0.4	7.9 ± 16.1†
Obstructive apnea, events/h	4.0 ± 2.9	22.4 ± 16.0†
Oxyhemoglobin desaturation (< 90% of total sleep time), %	0.4 ± 0.6	7.9 ± 14.2†

\*Data are presented as mean ± SD or %.

†p < 0.05

**Table 3—Cardiac Variables, and Urinary Catecholamine and NT-BNP Levels in Patients Without SDB (Group 1) or With SDB (Group 2)\***

Variables	Group 1 (n = 17)	Group 2 (n = 36)
Idiopathic etiology	59	53
LVEF, %	35.5 ± 8.0	33.4 ± 9.3
Atrial diameter, cm	4.36 ± 0.60	4.86 ± 0.68†
Atrial fibrillation, %	0	28†
Angiotensin-converting enzyme inhibitor	100	94
β-Blocker	29	33
Spiroonolactone	24	36
NYHA class III or IV	35	28
MLHF-Q score (total of 105)	38.4 ± 23.9	42.0 ± 25.2
Shuttle test, m	455.9 ± 258.9	436.3 ± 230.4
NT-BNP, pmol/L	90.9 ± 92.7	134.2 ± 172.4
Urinary epinephrine, nmol/nmol creatinine	2.1 ± 1.0 (n = 15)	1.0 ± 1.9
Urinary norepinephrine, nmol/nmol creatinine	19.7 ± 6.0 (n = 15)	29.1 ± 11.7†
Urinary dopamine, nmol/nmol creatinine	113 ± 31 (n = 15)	110.1 ± 41.2

\*Data are presented as % or mean ± SD.

†p < 0.05, group 1 vs group 2.

Witnessed apnea or often/always snored was not associated with the presence of SDB (witnessed apnea, 23% [no SDB] vs 42% [SDB];  $p = 0.20$ ; and often/always snore, 47% [no SDB] vs 58% [SDB];  $p = 0.40$ ).

Cardiac variables were compared between those with and without SDB (Table 3). No difference in etiology and severity of heart failure (as measured by LVEF and NYHA class) was found in the two groups. The prevalence of atrial fibrillation and urinary norepinephrine levels was significantly greater in those with SDB. Plasma NT-BNP concentrations were similar in both groups ( $p = 0.75$ ).

By multivariate analysis, there was no relationship between the MLHF-Q score, shuttle walk and LVEF ( $p = 0.16$ ), presence of sleep apnea ( $p = 0.85$ ), or the interaction between LVEF and presence of SDB ( $p = 0.91$ ). There was the expected relationship between LVEF and NT-BNP ( $p < 0.0001$ ,  $\log[\text{NT-BNP}]$ ) used to meet normality assumptions ( $R^2 = 0.33$ ; slope, 0.93; 95% confidence interval [CI], 0.91 to 0.96). There was no difference in the relationship between LVEF and NT-BNP according to whether the subject had SDB or not ( $p = 0.89$ ).

The AHI was weakly associated with LVEF. The slope parameter was  $-0.55$  (95% CI, 1.168 to 0.07;  $p = 0.08$ ). As LVEF increased, AHI decreased.

Group 2 subjects (those with SDB) were further subclassified into those with OSA ( $n = 28$ , 53% of all

subjects) and CSA ( $n = 8$ , 15% of all subjects) as defined previously. Subjects classified as group 2 obstructive had a mean of 92.5% respiratory events considered obstructive (range, 64.5 to 100%). Subjects classified as group 2 central had a mean of 82% central events (range, 56.3 to 100%). The mean LVEF in patients with CSA was lower than those with OSA and those without SDB (Fig 1;  $p = 0.0013$ ; 95% CIs [OSA-CSA], 5.6 to 18.3% and [no SDB-CSA] 4.7 to 18.3%). Urinary noradrenaline was increased in the CSA group ( $31.9 \pm 10.5$  nmol/nmol creatinine) when compared to those without SDB ( $19.7 \pm 6.0$  nmol/nmol creatinine,  $p = 0.013$ ).

## DISCUSSION

In the first (to our knowledge) systematic study of unselected stable heart failure outpatients, we found that OSA (53%) and CSA (15%) were remarkably common. The presence of SDB was associated with anthropomorphic features of OSA, atrial fibrillation, more severe oxyhemoglobin desaturation, sleep disruption, and higher urinary norepinephrine levels, but not increased daytime sleepiness. Indexes of impaired cardiac function including LVEF, MLHF-Q, shuttle walk distance, and NT-BNP were not related to the presence of SDB. Patients with CSA had more severe left ventricular dysfunction and increased norepinephrine levels than those with heart failure alone.

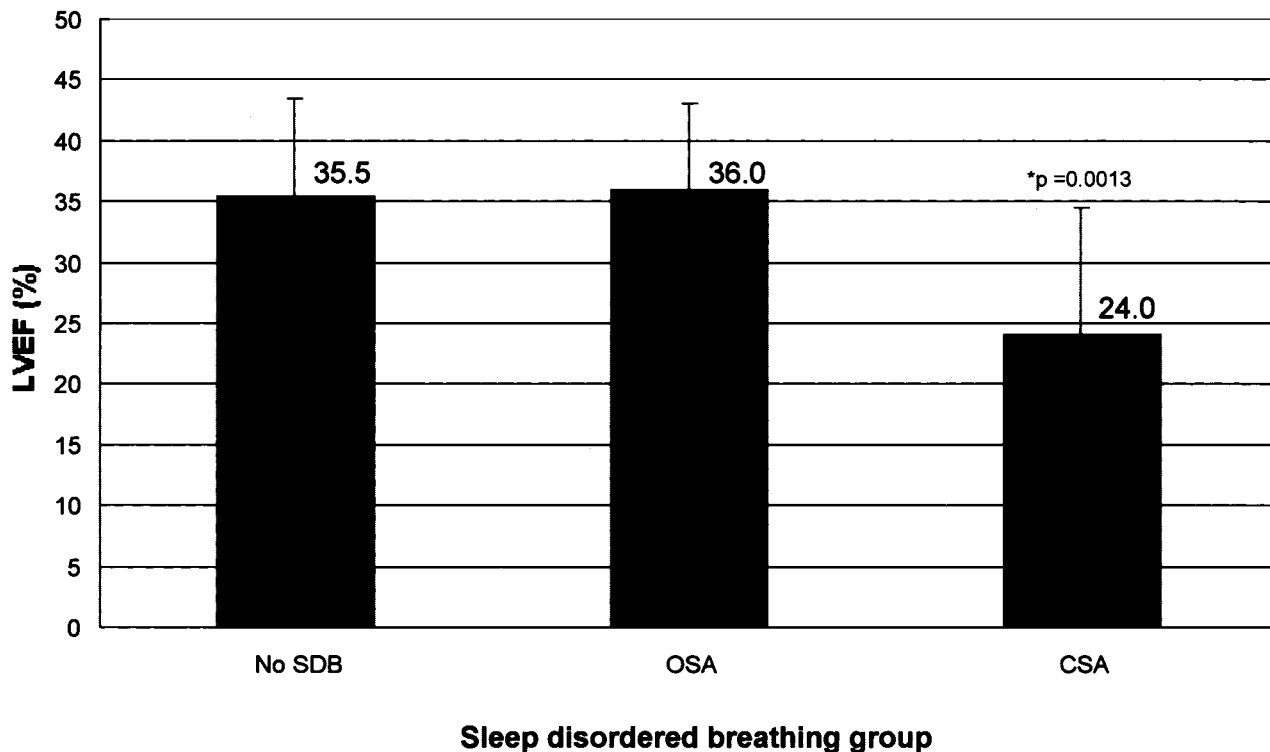


FIGURE 1. LVEF (mean  $\pm$  SD).

The frequency of SDB is similar to the largest study of patients with heart failure referred to a sleep clinic,<sup>10</sup> but the predominance of OSA over CSA contrasts with studies<sup>10,21,23</sup> in heart failure populations. It has been shown that CSA is correlated to severity of heart failure,<sup>41</sup> and therefore the relatively low proportion of CSA may be a reflection of the milder heart failure in our study patients (average LVEF, 34%) compared with those of other studies (mean LVEF, 27.3%,<sup>10</sup> LVEF < 25%<sup>21</sup>). Criteria for determining CSA vs OSA were the same as those used by Sin et al<sup>10</sup> (> 50% events), but the studies have used different monitoring techniques to measure airflow. The American Academy of Sleep Medicine guidelines recommend the use of nasal pressure over thermistor for determining airflow. It is possible that we have seen a high prevalence of SDB due to this technique, but we would not expect the difference in technology to alter the OSA-vs-CSA prevalence.

The high prevalence of OSA might be explained by traditional risk factors, including age, male gender, increased neck circumference,<sup>42</sup> BMI, and ethnicity (Maori and Pacific Island).<sup>30</sup> On average, our patients were overweight with a BMI of 28 kg/m<sup>2</sup>, but this is very similar to other studies.<sup>10,23</sup> One fourth of the sample were of Maori or Pacific Island ethnicity, which is more than twice the population

ethnic prevalence; and as Maori (and probably Pacific Island) ethnicity is associated with a threefold increase in the prevalence of SDB, this may have contributed to the high proportion of OSA in this group.<sup>30</sup> There were, however, a similar proportion of Maori and Pacific Islanders in group 1 (29%) and group 2 (25%). Maori and Pacific Island New Zealanders suffer disproportionately higher morbidity and mortality,<sup>43,44</sup> but the prevalence of CHF in these populations is not known.

The second finding of this study is that, although SDB is common in this population, its presence did not correlate with other prognostic markers of heart failure, nor did it impact on the patient's physical performance or their perception of disease impact on their life. The significantly higher presence of atrial fibrillation in those with SDB has previously been reported.<sup>10,23</sup> The size of this study limits our ability to determine whether the presence of atrial fibrillation may have independently affected the presence of SDB. Sin et al<sup>10</sup> determined that atrial fibrillation was a risk factor for CSA rather than OSA, and although the CSA group in the current study had the highest proportion of atrial fibrillation, this did not reach statistical significance. This study also supports previous findings<sup>41</sup> that CSA is correlated to severity of left ventricular dysfunction, measured by LVEF.

We found increased urinary norepinephrine levels among our CHF/SDB patients compared to those with CHF alone, as had been reported in previous studies.<sup>39,45</sup> Previous literature<sup>46</sup> indicates that the relative contribution of CHF to sympathetic activation is significantly larger than the effect of SDB, but those with CHF and CSA-CSR have increased urinary norepinephrine levels than those with CHF and OSA. However, in the current sample, urinary norepinephrine levels in those with CHF/OSA and CHF/CSA-CSR were not different and may reflect the milder CHF status of our patients.

As expected, the presence of sleep apnea was associated with sleep disruption and more prolonged periods of oxyhemoglobin desaturation, although despite these findings, daytime sleepiness (ESS) was not predictive of SDB. Therefore, we would suggest that subjective daytime somnolence (as measured by ESS) is not useful in determining which heart failure patients may have SDB. There are other reasons why patients with CHF may have daytime sleepiness, including nocturnal symptoms of CHF, nocturia, and medication effects. Previous research<sup>47,48</sup> looking at predictive variables for sleep apnea have shown that the relationship between subjective sleepiness and AHI is weak. Larger epidemiologic studies<sup>49</sup> show a significant relationship, but again this relationship is weak. The Sleep Heart Health Study<sup>49</sup> reports a mean ESS of 7.2 for subjects with an AHI of < 5 events/h and a mean ESS of 9.3 for subjects with an AHI > 30 events/h.

Currently hospital/sleep laboratory polysomnography is the "gold standard" for diagnosis and can readily distinguish between OSA and CSA-CSR, but it is expensive and not always readily available. Portable devices are being increasingly used to detect OSA; however, few can clearly distinguish between CSA and OSA. This is of direct practical relevance, as current treatment pathways are different. Unattended home polysomnography is a potentially cost-effective method that meets the technical diagnostic requirements, but it has not been adequately evaluated in this patient population.

nCPAP is an established treatment of symptomatic moderate-to-severe OSA in patients without CHF. There is evidence from two randomized, controlled trials<sup>50,51</sup> of small numbers of CHF patients that OSA can be successfully treated with nCPAP, with an improvement in short-term cardiovascular outcomes (LVEF, BP, sympathetic activation). However, these studies are of selected, highly motivated patients, are not placebo controlled, and lack hard cardiac end points including exercise capacity, hospital admission rates, and mortality.<sup>52</sup>

We conclude that most stable heart failure patients have either OSA or CSA. There was no clear

relationship between markers of impaired cardiac function and the presence of SDB apart from the finding that all patients with atrial fibrillation had SDB and that low LVEF was associated with CSA. Given the high prevalence of OSA and its potential adverse cardiovascular consequences, if treatment such as continuous positive airway pressure can be shown to be effective in improving major cardiovascular end points, these results would support the adoption of screening of stable CHF patients (LVEF < 45%).

We would encourage physicians to be alert to the possibility of SDB in their patients with stable CHF. However, further research into whom to target and what the long-term benefits of treatment are in this population is still required.

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