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In-hospital Testing for Sleep Disordered Breathing in Hospitalized Patients with Decompensated Heart Failure-Report of Prevalence and Patient Characteristics

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Abstract

Background—Sleep Disordered Breathing (SDB) is present in over 50% of ambulatory patients with chronic heart failure. The prevalence and type of SDB in hospitalized patients with acutely decompensated heart failure (ADHF) are not known.

Methods—In-hospital sleep studies were performed on consecutive patients with ADHF who were not previously tested for SDB.

Results—395 consecutive patients with ADHF underwent successful sleep study recording during hospitalization. 298 patients (75%, 95% CI (71, 80%)) had SDB; of these, 226 (57%, 95% CI (52, 62%)) had predominantly obstructive SDB and 72 (18%, 95% CI (14,22%)) had predominantly central SDB. Only 25% (95% CI (20%, 29%)) of patients were free of SDB. Validation polysomnography between 6 and 8 weeks after discharge on a sub-group of unselected patients with obstructive SDB revealed a 100 % positive predictive value (95% CI. 94% to 100%) for Obstructive Sleep Apnea (OSA).

Conclusion—Similar to stable chronic heart failure, ADHF is associated with a high prevalence of SDB. The prevalence of predominantly obstructive SDB exceeded that of predominantly central SDB in ADHF patients. The presence of obstructive SDB during hospitalization predicted a diagnosis of OSA on polysomnography.

INTRODUCTION

Heart failure is the only cardiovascular disease without recent improvement in incidence or related mortality (1). Approximately 5 million Americans have heart failure, with an annual incidence of 10 per 1000 in individuals over 65 years, an already rising demographic segment. In the US, heart failure is the most frequent Medicare diagnosis, and most of the cost associated with it is related to hospitalizations with acutely decompensated heart failure (ADHF)(1–3).

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Patients with ADHF have 5–20% mortality (4,5) and significant re-admission rate(6). Treatment options for ADHF remain limited and largely unchanged in the past two decades (7,8). Identification and treatment of highly prevalent co-morbidities with known detrimental effects carries a high potential for positive impact(9).

Sleep Disordered Breathing (SDB), broadly categorized into central and obstructive sleep apnea, has far higher prevalence in patients with stable heart failure than in the general middle aged population (10–12). Recent prevalence studies suggest that the occurrence of central sleep apnea (CSA) may be declining due to current changes in the management of heart failure (13–15). Other studies have consistently demonstrated a very high prevalence of obstructive sleep apnea (OSA) in 38–53% of patients with stable heart failure (16–19). Treatment of (OSA) with Continuous Positive Airway Pressure (CPAP) in ambulatory patients with systolic heart failure improves cardiac function(20,21). More recently, promising treatment options for CSA have also emerged(22). Therefore, the presence of a highly treatable and very prevalent disorder such as SDB in patients with heart failure may warrant a process for surveillance (23). A systematic approach to the diagnosis and treatment of SDB, particularly OSA, may be an important contribution to the management of heart failure. However, such an approach is not part of the current standard of practice (24,25)

OSA worsens the control of hypertension, coronary artery disease, and atrial fibrillation, all are associated with ADHF (4,26). It is likely, therefore, that OSA would be highly prevalent in patients presenting with ADHF. CSA, on the other hand, is more severe in heart failure patients with increased preload as is the case during decompensation of heart failure(27).

Therefore, we rationalized that the association between OSA and causes of ADHF (4,26), along with the relationship between CSA and worsening cardiac filling pressures, will produce a high prevalence of SDB in patients with ADHF. The fact that SDB case-finding is not the standard of practice in ADHF patients, might be explained by a conception that either the prevalence is low, the screening is not feasible, or the condition is not treatable (23). To date, no study has reported on the feasibility of inpatient testing or evaluated the prevalence and type of SDB in patients with ADHF. We attempted to evaluate an inpatient approach to the identification of SDB, and to quantify and characterize the type of SDB in patients with AHDF. We also sought to evaluate any correlation between SDB in the inpatient setting and known SDB syndromes in the stable outpatient setting.

METHODS

Participants

Consecutive patients admitted to the heart failure services at the Ohio State University (OSU) Ross Heart Hospital with ADHF between December 30, 2006 and January 31, 2008 were eligible. The definition of ADHF was as follows: A primary admission diagnosis of congestive heart failure and elevated left ventricular pressure as indicated by at least one sign and one symptom of volume overload (pedal edema, crackles, consistent chest X-ray, increased left ventricular end-diastolic dimension (LVEDD), or elevated B-type natriuretic peptide (BNP) level (7). This definition of ADHF included new onset acute heart failure or an exacerbation of already recognized heart failure. In particular, there was no cut-off criterion for left ventricular ejection fraction (LVEF). The study protocol was approved by the Ohio State University Institutional Review Board (#2007H0055).

A clinical process was established to perform in-hospital sleep studies on all newly admitted patients with ADHF. While the clinical process is still ongoing, this report includes patients who were hospitalized between December 30, 2006 and January 31, 2008.

Study Protocol

An order for the sleep study was incorporated in the computerized order entry set (28) for all patients hospitalized with ADHF at the Ohio State University Ross Heart Hospital. Patients were subsequently excluded if they had a preexistent diagnosis of SDB. Training of nursing and support staff on administering the study was concluded prior to implementation of the computerized order entry process. As such, the study was ordered by default and subsequently performed on the second night of admission on all newly admitted patients with ADHF. No previous knowledge of the patient's history or weight was available or included in the ordering or interpreting processes. While no screening for SDB occurred, a clinical study coordinator ensured every day that the orders generated by the system included patients with ADHF who met the inclusion criteria and did not have an existing diagnosis of SDB.

The administering nurses had the discretion to withhold the study if a patient was hemodynamically unstable or developed clinical deterioration during the study requiring administration of supplemental oxygen. Patients with hemodynamic instability, defined as mean arterial pressure (MAP) less than 55 mmHg off vasopressors or being on vasopressor treatment, and patients requiring mechanical ventilatory support were included after stabilization and on the night prior to discharge.

In the first 6 months of the study (12/2006-6-2007), patients who had abnormal studies in and resided in the hospital's draw area were asked to return to the OSU Sleep laboratory. The remainder of patients was referred to either the OSU Sleep Laboratory or a local AASM accredited sleep laboratories. After 06/01/2007 we delegated the follow up arrangement to the case managers and regional sleep centers. Figure 1 describes the disposition of patients in the study.

Procedures

1- In-hospital sleep study—This was an attended cardiorespiratory study that measures nasal pressure, respiratory effort, oxygen saturation, heart rate, and body position (Stardust II, Respirationics, Inc Murrysville, PA). The sleep study is classified as a type III sleep study according to the American Academy of Sleep Medicine (AASM) (29). The study was attended by previously trained night shift nurses who explained the purpose of the study to the patient and noted light out and light on times, along with any interruption to sleep. On the following morning, the study records were downloaded and transmitted via the hospital network for interpretation by a Sleep specialist who was blinded to patients' diagnosis or risk factors for SDB.

2- Polysomnography—This was a standard clinical sleep study performed in the Ohio State University Sleep Laboratory within 8 weeks of discharge from the hospital. The polysomnography montage (MedCare Diagnostics, Buffalo, NY) was a standard overnight clinical montage with flow measured by nasal pressure and oro-nasal thermistor, thoraco-abdominal wall movement measured by inductance plethysmography; cardiac frequency by ECG; Arterial oxygen saturation measured by pulse oximetry. A bilateral electro-oculogram, four channels of electroencephalogram, chin and anterior tibial electromyograms, and body position sensor were all available.

Interpretation of Sleep Studies

For the in-hospital sleep study, SDB was defined as an Apnea Hypopnea Index (AHI) ≥ 15 events/hour. Apnea was scored when complete or near complete cessation of flow occurred. Hypopnea was scored when a 50% reduction in the flow signal occurred in association with 3% desaturation. A duration of 10 seconds was required for all events, and a minimum 3% desaturation was required for scoring hypopneas (29). An AHI cutoff of 15 events/hour was

selected for the in-hospital study to mitigate against expected increase in SDB events during severe heart failure. Mostly apneas were used to classify SDB as central or obstructive. If more than 50% of the apneas were central the SDB was considered as Central SDB (CSDB). If more than 50% of the apneas were obstructive the disorder was considered as Obstructive (OSDB). If hypopneas occurred in the setting of periodic breathing (crescendo-decrescendo breathing with a cycle length >50 seconds), they were classified as central. The scoring criteria for the polysomnography were according to event definition by the AASM(30). The polysomnography was scored by technicians blinded to the findings of the in-hospital studies. The sleep physicians interpreting the polysomnography were aware that the patient underwent an in-hospital sleep studies, but were not aware of the results.

Statistical Analysis

Descriptive statistics, such as frequencies and percentages and their confidence interval or standard error, were used to characterize the subjects and to report the prevalence and type of SDB in the study population. Comparisons on a profile of patient characteristics were used to understand the differences between normal obstructive SDB, and central SDB groups. Pearson's correlations were used first in all patients then in the SDB group to evaluate potential relationships between the severity of SDB according to the AHI and selected clinical variables: Body mass index (BMI), LVEF, LVEDD, BNP, and age. The BMI and age were selected because they correlate with OSA in the general population(31). LVEF, LVEDD, and BNP were chosen as markers of heart failure disease severity. For correlations, 95% confidence intervals were provided. Polychotomous logistic regression (SAS version 9.1, SAS Institute Inc., Cary, NC) was used to model the relation between the three groups as referents (obstructive SDB, central SDB, and negatives) and the selected variables.

RESULTS

Patient Characteristics

During the study period (December 30, 2006 – January 31, 2008) we identified 559 patients who met the described criteria of ADHF. Of these, 93 patients have had a test for SDB and were excluded. The remaining 466 patients received orders for the in-hospital sleep study. Successful recording on the second day of hospitalization was obtained in 395 patients. Based on the 395 consecutive ADHF patients, Table 1 provides a profile of baseline characteristics. For example the mean LVEF was 33 (95% CI (32,35)). The 71 patients who did not have successful recording had similar characteristics as the 395 patients with successful recording. In particular there was no difference in age, BMI, type of cardiomyopathy, LVEFF, or LVEDD. The most common reason for recording failure was device malfunction, inadequate number of recorders on a particular night, or multiple channel failure on the recorded study.

Prevalence and Type of Sleep Disordered Breathing

Using a cutoff AHI of ≥ 15 events/hour, 298 patients (75%, 95% CI (71, 80%)) had SDB; 226 (57%, 95% CI (52, 62)) had predominantly obstructive SDB (OSDB) and 72 (18%, 95% CI (14,22)) had predominantly central SDB (CSDB). Only 97 patients (25% (95% CI 20%, 29%)) were free of SDB.

Characteristics and predictors of the type of SDB

The characteristics of patients in the three groups (negative, obstructive, and central) are compared in Table 2. Note that the significance values were not corrected for multiplicity here, because this analysis is considered exploratory. The same applies to the confidence intervals. Compared to patients without SDB, patients with OSDB were more likely to be older males, have higher body mass index (BMI), and have ischemic cardiomyopathy.

When compared to negatives, patients with CSDB were also more likely to be males. Additionally, central SDB patients had lower LVEF, and higher LVEDD, and BNP than both negatives and patients with OSDB. There was no difference in BMI between the negatives and central SDB patients.

Correlation between SDB and selected predictors

In order to further evaluate the relationships described above and to confirm potential predictors of SDB, we calculated Pearson correlation coefficients between AHI and five variables (LVEF, LVEDD, BMI, Age, BNP). We also evaluated the association with atrial fibrillation using logistic regression. For the overall group of ADHF patients, there was a correlation between AHI and BMI (r^2 0.17, (CI: 0.07, 0.26) and AHI and LVEDD (r^2 0.19, (CI: 0.07, 0.30). There was no significant association between the four other variables (LVEF, age, BNP, and atrial fibrillation) and AHI. For the SDB group (n=298), similar correlations between LVEDD and BMI with AHI were found. The correlation between BMI and AHI was r^2 0.18 (CI 0.07, 0.29); and between LVEDD and AHI was r^2 0.15 (CI: 0.01, 0.28). This further analysis identified only elevated BMI and LVEDD as predictors of having SDB in patients with ADHF. For the group of patients with OSDB the correlation between BMI and AHI was largest (Pearson r^2 = 0.3, (CI: 0.17, 0.41)). For the group with CSDB (n=72), no significant correlations were found between any of six clinical variables and severity of SDB (AHI). Multi-regression modeling did not change these relationships.

Follow-up Testing and Validation of inpatient testing

Patients who underwent an abnormal in-hospital study in the first 6 months of the study period (January – June 2007) were invited back to undergo a standard outpatient in-lab polysomnography in our sleep laboratory (Figure 1). Sixty two patients out of the 111 validation cohort of OSDB patients returned for their validation polysomnography. The most common reason for not returning for the validation polysomnography was living away from the hospital's draw area. Twelve patients (out of 26 invited) with CSDB underwent their validation polysomnography. Table 3 presents the comparison in sleep and cardiac characteristics between the group of OSDB patients with validated studies and those without a validation study. Overall, while there was a slight difference in BMI, no other differences existed in cardiac parameters, symptoms, or AHI on the in-hospital study between the two groups. Additionally, the severity of SDB (average AHI) remained unchanged on the validation Polysomnography (mean $\pm$ standard error 37.4 ± 2.5 for the in-hospital study vs. 41.7 ± 3.9 on the outpatient polysomnography) (Table 3.3). All patients with OSDB were diagnosed with OSA on the validation polysomnography in the OSDB group with a validated study. This gives the inpatient test 100% positive predictive value (95% CI 94,100%) for a diagnosis of OSA on a subsequent outpatient polysomnography. In the validated group of patients with CSDB the average AHI did not change on the validation polysomnography (table 3.3). Among the 12 patients with CSDB who returned for validation polysomnography, 4 patients were diagnosed as having OSA, and 8 were diagnosed as CSA.

DISCUSSION

In this study, we describe a systematic surveillance program for SDB in patients hospitalized with ADHF. This is the first study to evaluate the prevalence of SDB in this population and demonstrate feasibility and reliability of inpatient testing. Using an attended inpatient sleep study, with an AHI cut-off of 15 events/hour, we studied 395 consecutive unscreened patients hospitalized with ADHF. We found that 75% of these ADHF patients demonstrated SDB. Of these, the majority (57%) had a predominantly obstructive type and a minority (18%) demonstrated central type of SDB. Although only 56% of invited patients returned for the validation polysomnography, we found that those who returned were quite similar to those who

did not. The validity of inpatient diagnosis of central SDB for an outpatient diagnosis of CSA was not tested in this study. However, the presence of obstructive SDB during the hospitalization was 100% predictive of having OSA on follow up polysomnography after resolution of the decompensation. Increased LVEDD and BMI were the only risk factors for having SDB. Patients with OSDB tended to be older males with higher BMI. Patients with CSDB in this series of ADHF patients had worse cardiac function parameters (lower LVEF, higher LVEDD, and higher BNP).

Prior studies of SDB prevalence in heart failure mainly included stable outpatients with chronic systolic heart failure. In one landmark study, Javaheri et al (11) evaluated ambulatory elderly males with systolic heart failure and found that 51% of these patients had SDB. Forty percent of all patients with heart failure had CSA and 11% had OSA. Sin et al (12) also studied predominantly male patients with systolic heart failure who were previously referred to the sleep laboratory, and reported similar prevalence of SDB with a higher occurrence of OSA. Patients with ADHF are a distinct population from ambulatory patients with stable systolic heart failure. And the available data on prevalence and distribution of SDB in stable patients with systolic dysfunction may have limited application to hospitalized patients with ADHF. Our study enrolled consecutive hospitalized patients with decompensated heart failure regardless of sex, type of cardiac dysfunction, or the duration of prior cardiac dysfunction. As a result, 38% of our cohort was women (compared to 15% in Sin et al, and 0% in Javaheri et al) (12). In addition, our sample did not exclude patients with predominantly diastolic etiology of their decompensated heart failure. These patients represent a large portion of heart failure patients (32) (3). In more recent studies of ambulatory heart failure patients, the prevalence of SDB was still very high and similar to that reported by Javaheri and Sin. However, the distribution of central and obstructive sleep apnea appeared to change; with higher prevalence of OSA was in these ambulatory patients with systolic heart failure (16–19). This was particularly the case in one study that included hospitalized as well as ambulatory patients with heart failure (19). There is evidence that recent changes in the treatment of chronic heart failure and the use of β -blockers may affect a decline in the occurrence of CSA (14). These changes in the management of chronic heart failure, along with the increasing obesity in the general adult population (33,34) may explain the increasing prevalence of OSA in these studies and in ours.

Increased cardiac filling pressure in patients with severe systolic heart failure correlates with respiratory control instability and subsequently CSA (27,35). Therefore one might expect higher prevalence of CSA than OSA in patients with worse cardiac function. However, the role of increased cardiac filling pressure in the pathogenesis of SDB in heart failure patients remains incompletely understood. Recently it was demonstrated that increased venous return worsens upper airway collapsibility and gives rise to obstructive events (36,37). One can speculate that this effect may be more pronounced in patients with ADHF who have more cervical venous congestion than patients with stable heart failure. A complimentary explanation may be the strong relation between respiratory control instability itself and upper airway collapsibility. It is known that upper airway obstruction may follow central apnea (38) and that central sleep apnea may result from upper airway instability (39). Therefore, it is conceivable that patients who are predisposed to increased upper airway resistance due to obesity or cervical venous congestion may manifest predominantly obstructive, but also some central events during states of further increased filling pressure. The high prevalence of OSA, a disorder of obesity and aging, may be simply a corollary of the rising weight of the middle-aged population, and the older age of heart failure patients (1). Elevated BMI correlated with having OSDB in our patients with ADHF, and OSDB patients tended to be older males, all of which are the same risk factors for OSA in the general population (40) and in patients with stable heart failure (12). This supports that OSDB is independent from the heart failure and its decompensation. The persistence of SDB on the validation portion of our study after resolution of the

decompensation further supports that SDB in these patients, while possibly worsened by the decompensation, has an independent etiology from the underlying decompensation.

We used a cardiorespiratory sleep recording device to evaluate for SDB. Similar devices have been validated in the unattended setting (41–43), and their sensitivity is likely enhanced with use in the in-hospital attended setting. Additionally, an identical device was shown to have excellent ability in the unattended setting to discriminate between OSA and CSA in patients with heart failure compared to polysomnography (44). These devices are now approved by the American Academy of Sleep Medicine (AASM) for the diagnosis of OSA (45) in populations with high pretest probability; albeit, not yet in the heart failure population. If the in-hospital technique had a high rate of false positives due to a possible role for ADHF in worsening SDB (27,36,46), the polysomnography after stabilization of the decompensation would have produced at least some false positives. The confidence interval on the positive predictive value was quite narrow. The persistence of OSDB into the outpatient setting is well supported by these findings. Due to the limited number of patients, the persistence of CSDB is less clear. The reason for the persistence of OSDB is not evaluated in this study. Increased cervical venous congestion during decompensation may have contributed to the higher than expected prevalence of OSDB in the inpatient setting. Increased cardiac filling pressure is not expected to be a significant problem 8 weeks after discharge. However, a recent study suggests that AHI in patients with OSA is related to fluid shift in the supine position (47). Such fluid shift may be even more significant in patients with heart failure and subsequently may account for the persistence of OSDB into the outpatient setting. Improved sleep efficiency after discharge and the ability to measure sleep on the polysomnography may also result in higher AHI since sleep time is the denominator for AHI in polysomnographic studies.

According to WHO criteria for case-finding, highly prevalent conditions with known negative impact, for which treatment is available and diagnosis is feasible, warrant screening (23). This study demonstrated the feasibility of systematic inpatient testing for SDB and the high prevalence of this independent comorbidity. The benefit of such practice will require further studies to evaluate the effect of inpatient treatment of OSA on ADHF outcomes. Once these studies are concluded and the benefit of inpatient treatment of OSA is demonstrated, such systematic approaches could be considered in patients with ADHF.

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Figure 1.

Disposition of the 395 consecutive patients with acutely decompensated heart failure (ADHF) who underwent successful recording during hospitalization between 12/30/2007 and 1/31/2008. SDB: Sleep Disordered Breathing; OSDB: Obstructive Sleep Disordered Breathing; CSDB: Central Sleep Disordered Breathing; OSA: Obstructive Sleep Apnea; CSA: Central Sleep Apnea; AHI Apnea hypopnea index. The figure includes all patients with ADHF who were screened during the study period (559). Note that there was no difference in any of the characteristics listed in table 1 between the 71 patients with failed sleep studies and the 395 with successful sleep studies.

Table 1

Characteristics of all 395 patients with ADHF

Patient characteristic (number of Patients with data)	Mean or % (SE)	95% Confidence Interval
Age (395)	59 (0.7)	(57, 60)
Sex (male) (395)	62 % (2%)	(58 %, 67 %)
Ischemic cardiomyopathy (395)	57 (0.8)	(56, 59)
Left ventricular ejection fraction (370)	33 (0.9)	(32, 35)
BMI kg/cm ² (393)	32 (0.4)	(31, 33)
Admission BNP pg/mL (294)	888 (59)	(773, 1003)
Atrial fibrillation (394)	35 % (2%)	(30 %, 39 %)

BNP: B-type natriuretic peptide on admission; BMI: body mass index; SE: Standard error

Table 2

Comparison of characteristics among the three groups of patients with ADHF

Characteristic	OSDB Mean (SE)	CSDb Mean (SE)	Negative Mean (SE)	P Value negatives vs. OSDB	P Value negative vs. CSDb	P Value CSDb vs. OSDB
Age	60 (0.9)	58 (1.8)	56 (1.6)	0.03	0.37	0.40
Male sex	69% (3%)	75% (5%)	38% (5%)	0.0001	0.0001	0.30
Cardiomyopathy						
Ischemic	62% (3%)	64% (6%)	44% (5%)	0.003	0.01	0.82
Dilated	23% (3%)	14% (4%)	35% (5%)	0.02	0.001	0.11
Others	15% (2%)	22% (5%)	21% (4%)	0.22	0.80	0.16
LVEF	34 (1.2)	27 (1.7)	38 (1.8)	0.06	0.0001	0.0008
BMI kg/cm2	33 (0.6)	29 (0.9)	31 (0.8)	0.03	0.12	0.0001
LVEDD	57 (1.1)	63 (1.6)	54 (1.2)	0.14	0.0001	0.0037
BNP pg/dl	746 (66)	1341 (161)	873 (130)	0.35	0.02	0.001
Atrial fibrillation	59% (3%)	32% (6%)	28% (5%)	0.06	0.57	0.31

SDB: Sleep Disordered Breathing; OSDB: Obstructive Sleep Disordered Breathing; CSDb: BNP: B-type natriuretic peptide on admission; SE: Standard error; LVEF: left ventricular ejection fraction; LVEDD: Left ventricular end diastolic diameter; BMI: body mass index; SE: Standard error

Table 3**Table 3-1. Comparison between OSDB patients with validation outpatient polysomnography and eligible patients who did not have validation polysomnography OSDB**

	OSDB patients with validation PSGs (62) Mean or % (SE)(N)	OSDB Non-validated patients (49) Mean or % (SE)(N)	Difference between validated OSDB patients and non-validated patients	95% CI for the Difference
Age	55 (1.8) (62)	61 (2.1) (49)	-6	-11.4, 0.4
Male	69% (6%) (62)	67% (7%) (49)	2%	-16%, 20%
BMI	35.5 (1.1) (62)	30.8 (1.3) (49)	4.7	1.4, 8.0
LVEF	35.3 (2.2) (60)	31.2 (2.8) (48)	4.1	-2.8, 11.0
LVEDD	56.4 (2.1) (51)	58.6 (2.2) (38)	-2.2	-8.2, 3.9
BNP	475 (79) (46)	1113 (180) (44)	-637	-1023, -252
Ischemic cardiomyopathy	56% (6%) (62)	69% (7%) (49)	-13%	-31%, 5%
Snoring	54% (6%) (61)	80% (6%) (40)	-26%	-45%, -7%
ESS	10.4 (0.7) (43)	9.7 (0.9) (43)	0.7	-1.5, 2.9
Atrial fibrillation	41% (6%) (61)	39% (7%) (49)	2%	-17%, 21%
Inpatient AHI	37.4 (2.5) (62)	32.5 (1.9) (49)	5.0	-1.7, 11.6

Table 3-2. Comparison of AHI between the in-hospital study and the polysomnography in the validated OSDB and CSDB patients

	PSG AHI Mean (SE) (N)	Inpatient AHI Mean (SE) (N)	Difference between PSG AHI and Stardust AHI	95% CI for the Difference
OSDB	41.7 (3.9) (62)	37.4 (2.5) (62)	4.3	-1.1, 9.6
CSDB	36.4 (7.2) (12)	49.1 (5.9) (12)	-12.7	-29.9, 4.5

OSDB: Obstructive Sleep Disordered Breathing; CSDB: BNP: B-type natriuretic peptide on admission; SE: Standard error; LVEF: left ventricular ejection fraction; LVEDD: Left ventricular end diastolic diameter; ESS: Epworth Sleepiness Scale; BMI: body mass index; SE: Standard error; N: number of patients with available data; Inpatient AHI: Apnea hypopnea index on the inpatient sleep study

OSDB: Obstructive Sleep Disordered Breathing; CSDB: Central Sleep Disordered Breathing; LVEDD: Left ventricular end diastolic diameter; LVEF: Left ventricular ejection fraction; BNP: B-type natriuretic peptide on admission; SE: Standard error; ESS: Epworth Sleepiness Scale; AHI: Apnea hypopnea index, Afib: Atrial Fibrillation, PSG: Polysomnography