 Effects of Continuous Positive Airway Pressure on Sleep Apnea and Ventricular Irritability in Patients With Heart Failure

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**Background**—Patients with heart failure and systolic dysfunction may develop disordered breathing during sleep. Repeated episodes of apnea and hypopnea may result in desaturation and arousals, which could adversely affect left ventricular function. The purpose of this study was to determine the short-term effects of continuous positive airway pressure (CPAP) on sleep-disordered breathing and its consequences in heart failure patients.

**Methods and Results**—The author prospectively studied 29 male patients whose initial polysomnograms showed 15 or more episodes of apnea and hypopnea per hour (apnea-hypopnea index, AHI). Twenty-one patients had predominately central and 8 patients obstructive sleep apnea. All were treated with CPAP during the subsequent night. In 16 patients, CPAP resulted in virtual elimination of disordered breathing. In these patients, the mean AHI (36 ± 12 [SD] versus 4 ± 3 per hour, \( P=0.0001 \)), arousal index due to disordered breathing (16 ± 9 versus 2 ± 2 per hour, \( P=0.0001 \)), and percent of total sleep time below saturation of 90% (20 ± 23% to 0.3 ± 0.7%, \( P=0.0001 \)) decreased, and lowest saturation (76 ± 8% versus 90 ± 3%, \( P=0.0001 \)) increased with CPAP. In 13 patients who did not respond to CPAP, these values did not change significantly. In patients whose sleep apnea responded to CPAP, the number of hourly episodes of nocturnal premature ventricular contractions (66 ± 117 versus 18 ± 20, \( P=0.055 \)) and couplets (3.2 ± 6 versus 0.2 ± 0.21, \( P=0.031 \)) decreased. In contrast, in patients whose sleep apnea did not respond to CPAP, ventricular arrhythmias did not change significantly.

**Conclusions**—In 55% of patients with heart failure and sleep apnea, first-night nasal CPAP eliminates disordered breathing and reduces ventricular irritability. (Circulation. 2000;101:392-397.)

**Key Words:** nervous system, sympathetic heart failure respiration hypoxia

Heart failure is a highly prevalent disorder with considerable morbidity and mortality despite advancements in its therapy. A recent systematic study in patients with treated, stable heart failure with left ventricular systolic dysfunction reported that ≈50% had periodic breathing during sleep.1 Periodic breathing (defined as a pattern of breathing consisting of periodically recurring cycles of apnea or hypopnea followed by hyperpnea) may result in arterial oxyhemoglobin desaturation, excessive arousals, and changes in intrathoracic pressure.1–3 If left untreated, the pathophysiological consequences of periodic breathing could affect cardiac function adversely, contributing to an increase in morbidity and mortality of patients with heart failure.4 In a study reported from this laboratory,1 we found that heart failure patients with sleep apnea had a lower left ventricular ejection fraction and a higher prevalence of atrioventricular arrhythmias than patients without periodic breathing; the cause and effect, however, could not be determined.

Nasal continuous positive airway pressure (CPAP) devices are well known to prevent upper airway occlusion, which occurs in obstructive sleep apnea (OSA).5–7 These devices have been successfully used to treat OSA in patients without heart failure. More recently, CPAP devices have been used for treatment of sleep apnea in patients with heart failure and systolic dysfunction.8–10 With regard to treatment of central sleep apnea (CSA), the most common form of disordered breathing in heart failure, although the results from one center have been promising,8 2 other centers9,10 reported no significant beneficial effects of CPAP on CSA.

During the past few years, we have been involved in studies of periodic breathing in a relatively large number of patients with heart failure resulting from systolic dysfunction.1,11 We have studied patients who were ambulatory and stable on standard therapy for heart failure, with no other major disorders (eg, chronic obstructive lung disease). Patients who were found to have an apnea-hypopnea index (AHI) of ≥15 per hour were subsequently treated with CPAP and are the subject of this report.

**Methods**

The patients in the current study belonged to the pool of 81 patients with stable heart failure and left ventricular systolic dysfunction (left
ventricular ejection fraction <45%) who have been described in detail previously. Twenty-nine of these patients whose initial polysomnograms showed an AHI of ≥15 per hour were subsequently treated with CPAP.

All patients were clinically stable (no change in symptoms of heart failure or their medications within the previous 4 weeks) and on standard therapy, including an angiotensin-converting enzyme inhibitor (23 patients), hydralazine (4 patients), digoxin (23 patients), isosorbide dinitrate (12 patients), and diuretics (26 patients). Exclusion criteria have been detailed previously and included major comorbid disorders (such as chronic obstructive pulmonary disease and known intrinsic liver and renal disorders) and use of morphine derivatives, benzodiazepines, theophylline, and acetazolamide. For uniformity, only male patients were studied: female patients are seldom referred to this center.

**Polysomnography**

On the first night, the patients slept in the sleep laboratory after electrodes had been placed, with the intention of familiarizing them with the environment of the sleep laboratory. On the second night, polysomnography was performed using standard techniques as detailed previously. For staging sleep, we recorded electroencephalogram (2 channels), chin electromyogram (1 channel), and electro-oculogram (2 channels). Thoracoabdominal excursions and naso-oral airflow (measured by a thermocouple) were measured qualitatively. Arterial blood oxyhemoglobin saturation was recorded using an oximeter. The output of pulse oximeter at 0 V was considered 0% saturation and at 1 V, 100% saturation. The voltage calibration was performed several times throughout each study. The variables were recorded on a multichannel polygraph (Model 78D; Grass Instrument Company).

An apnea was defined as cessation of inspiratory airflow for ≥10 seconds. All such events were counted irrespective of the degree of desaturation or presence of an arousal. An obstructive apnea was defined as the absence of airflow in the presence of rib cage and/or abdominal excursions. A central apnea was defined as the absence of rib cage and abdominal excursions with absence of airflow. It is noted, however, that in the absence of measurements of esophageal pressure, it is sometimes difficult to truly distinguish a central apnea from an obstructive apnea. Hypopnea was defined as a discernible reduction in airflow or thoracoabdominal excursions lasting ≥10 seconds and resulting in an arousal or at least a 4% drop in arterial oxyhemoglobin saturation. The author classified hypopnea as obstructive if paradoxical thoracoabdominal excursions occurred or if the airflow decreased out of proportion to the reduction in the thoracoabdominal excursions. The number of episodes of apnea and hypopnea per hour of sleep is referred to as the AHI. Similarly, the number of episodes of arousal per hour of sleep is referred to as the arousal index. The mean CPAP was calculated to have been delivered to patients with OSA- or CSA-hypopnea as defined earlier. In patients with predominate CSA, the obstructive AHI had to be <10 per hour; at least 50% of the disordered breathing events were classified as central. In these patients, the obstructive AHI varied from 0.0 to 9.1 per hour. In patients with predominately OSA, obstructive AHI had to be ≥10 per hour, and it represented at least 50% of the disordered breathing events. In these patients the obstructive AHI varied from 19.9 to 35.3 per hour. All polysomnograms were scored blindly.

**CPAP Titration**

Patients who had an AHI ≥15 were studied during the third night in the sleep laboratory with CPAP. Titration began with 5 cm H2 O, and the pressure was progressively and gradually increased by 1 cm H2 O every 15 minutes, to eliminate episodes of apnea, hypopnea, desaturation, and arousal. Pressure was decreased, at the patient’s request if awakening occurred, and titration was resumed as the patients fell asleep. We had the intention to discontinue CPAP if patients complained of dizziness or chest discomfort.

For the purpose of this study, we defined individuals whose AHI on CPAP decreased to <15 per hour, “CPAP-responsive.” This was the threshold criteria used to define presence of clinically significant sleep apnea. In all these patients, the AHI decreased by >50%.

**Other Studies**

To determine the prevalence of arrhythmia and its response to CPAP, Holter monitoring was performed during polysomnography, as detailed previously. In this study, 16 patients had paired Holter recordings. Ventricular tachycardia was defined as the presence of 3 ventricular premature beats in a row. Right and left ventricular ejection fractions were calculated from gated first pass and multi-gated radionuclide ventriculograms, respectively, using standard techniques. Pulmonary function tests and arterial blood samples were obtained as detailed previously. These studies were approved by the Institutional Review Board at the University of Cincinnati, and written informed consent of each patient was obtained.

**Statistical Analysis**

We used the Wilcoxon rank sum test for paired and Mann-Whitney U statistic for unpaired data, because our data were not normally distributed; χ2 was used for comparison of proportions. A 2-tail P value ≤0.05 was considered significant. Values are reported as mean±SD. Calculations were done using GraphPad Instat V2.03.

**Results**

There were 29 heart failure patients, 8 with OSA and 21 with CSA. Fifty-five percent (16 of 29) patients responded to CPAP, 7 with OSA and 9 with CSA.

Patients with OSA were significantly heavier and had a higher prevalence of habitual snoring than patients with CSA (Table 1). In patients with OSA, application of nasal CPAP resulted in significant improvement in disordered breathing and arterial oxyhemoglobin desaturation, with a significant drop in arousals due to periodic breathing (Figure 1). These arousals decreased from 22±10 to 3±3 per hour and accounted for most of the decrease in overall arousal index from 42±31 to 14±9 per hour (P=0.008). Sleep stages did not change significantly. CPAP values varied from 5 to 18 cm H2 O, with a mean of 8±5 cm H2 O.

In one patient with predominate OSA (AHI 54 per hour, obstructive AHI 35 per hour, and central apnea index 14 per hour), CPAP resulted in elimination of all obstructive episodes (index=0.0 per hour), but episodes of central apnea increased (central apnea index increased to 21 per hour with CPAP). This patient was considered nonresponsive to CPAP.

There were 21 patients with CSA, 9 of whom responded to CPAP (43% of patients with CSA). CPAP significantly improved disordered breathing and arterial oxyhemoglobin desaturation with a significant decrease in the number of arousals due to periodic breathing (Figure 2). These arousals decreased from 12±8 to 2±2 per hour (Figure 2) and accounted for the decrease in the overall arousal index from 21±16 to 11±9 per hour (P=0.098). There were no significant changes in sleep stages, however. The CPAP values varied from 5 to 12 cm H2 O and the mean was 7±2 cm H2 O.

In the 12 heart failure patients with CSA who did not respond to CPAP, the mean values for AHI (62±22 versus 62±29 per hour), baseline versus CPAP, respectively), central apnea index (43±21 versus 49±21 per hour), total dark time (397±21 versus 383±33 minutes), total sleep time (255±51 versus 212±83 minutes), most sleep stages, and arousal index (40±27 versus 40±31 per hour) did not change significantly. However, CPAP modestly improved arterial oxyhemoglobin desaturation with percent total sleep time below 90% decreasing from 30±17% to 9±15%, P=0.02. Values for CPAP varied from 5 to 18 cm H2 O, and the mean was 13±4 cm H2 O.
Although there were significant differences in demographics of patients with OSA versus CSA (Table 1), demographics of heart failure patients with CSA who did not respond to CPAP did not differ from those who did (Table 1). However, the AHI was significantly higher in nonresponsive than responsive patients (Table 1).

In patients whose sleep apnea responded to CPAP, the number of premature ventricular contractions, couplets, and ventricular tachycardias decreased with CPAP (Table 2). In contrast, CPAP had no significant effect on ventricular irritability in patients whose disordered breathing did not improve (Table 2).

**TABLE 1. Demographics, Arterial Blood Gas Values, Left Ventricular Ejection Fraction, and Disordered Breathing Events in Heart Failure Patients With OSA and CSA**

<table>
<thead>
<tr>
<th></th>
<th>OSA (n=8)</th>
<th>CSA (n=21)</th>
<th>P</th>
<th>CSA-Nonresponsive (n=12)</th>
<th>CSA-Responsive (n=9)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>61±10</td>
<td>66±9</td>
<td>0.2</td>
<td>65±10</td>
<td>68±8</td>
<td>1.0</td>
</tr>
<tr>
<td>Height, cm</td>
<td>175±5</td>
<td>174±8</td>
<td>0.7</td>
<td>173±8</td>
<td>174±8</td>
<td>0.8</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>111±29</td>
<td>85±19</td>
<td>0.02</td>
<td>82±18</td>
<td>90±21</td>
<td>0.4</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>36±8</td>
<td>28±5</td>
<td>0.02</td>
<td>27±5</td>
<td>29±5</td>
<td>0.3</td>
</tr>
<tr>
<td>Habitual snoring, %</td>
<td>88</td>
<td>50</td>
<td>0.07</td>
<td>50</td>
<td>50</td>
<td>1.0</td>
</tr>
<tr>
<td>P&lt;sub&gt;O&lt;/sub&gt;, mm Hg</td>
<td>77±11</td>
<td>84±9</td>
<td>0.1</td>
<td>84±9</td>
<td>84±10</td>
<td>0.9</td>
</tr>
<tr>
<td>P&lt;sub&gt;CO&lt;/sub&gt;, mm Hg</td>
<td>41±6</td>
<td>37±4</td>
<td>0.048</td>
<td>35±4</td>
<td>38±4</td>
<td>0.2</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>26±7</td>
<td>22±9</td>
<td>0.1</td>
<td>20±6</td>
<td>25±11</td>
<td>0.3</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>132±20</td>
<td>122±19</td>
<td>0.3</td>
<td>120±16</td>
<td>127±22</td>
<td>0.6</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>79±12</td>
<td>72±11</td>
<td>0.1</td>
<td>72±12</td>
<td>73±9</td>
<td>0.4</td>
</tr>
<tr>
<td>AHI, n/hr</td>
<td>37±9</td>
<td>51±23</td>
<td>0.09</td>
<td>62±22</td>
<td>36±15</td>
<td>0.01</td>
</tr>
<tr>
<td>OAI, n/hr</td>
<td>8±10</td>
<td>1±2</td>
<td>0.01</td>
<td>0.4±1.2</td>
<td>1.7±2.9</td>
<td>0.03</td>
</tr>
<tr>
<td>CAI, n/hr</td>
<td>28±6</td>
<td>1±3</td>
<td>0.0001</td>
<td>1.0±2.6</td>
<td>2.1±3.1</td>
<td>0.05</td>
</tr>
</tbody>
</table>
| SBP indicates systolic blood pressure; DBP, diastolic blood pressure; LVEF, left ventricular ejection fraction; OAI, obstructive apnea-hypopnea index; and CAI, central apnea index. Values are mean±SD.

**Figure 1.** Polysomnographic findings in 8 heart failure patients with OSA with and without continuous positive airway pressure CPAP. OAHI indicates obstructive apnea-hypopnea index; SaO₂, arterial oxyhemoglobin saturation; TDT, total dark time; TST, total sleep time; WASO, wakefulness after sleep onset; SE, sleep efficiency; DBArI, arousal index due to disordered breathing.

**Discussion**

This is the largest study of the effects of CPAP on sleep-disordered breathing in patients with stable, treated heart failure and systolic dysfunction. There were 29 heart failure patients with sleep apnea, 55% of whom (16 of 29 patients) responded significantly to CPAP. In these patients, AHI, arousal index, and arterial oxyhemoglobin saturation improved. Ventricular arrhythmias also improved in patients whose disordered breathing responded to CPAP; there was no significant change in nonresponders.
Effects of CPAP in Heart Failure Patients
With OSA
In 8 patients with OSA, application of nasal CPAP (8±5 cm H₂O) resulted in a significant reduction in AHI from 37 to 5 per hour (Figure 1). As a result, arousal index due to sleep apnea and hypopnea decreased significantly from 22 to 3 per hour, and time below saturation of 90% decreased from 31% of the total sleep time to 1% (Figure 1).

Continuous positive airway pressure is known to maintain upper airway patency by increasing transmural pressure of upper airways.5–7 The relief of upper airway occlusion in heart failure patients with OSA should be similar to OSA patients without heart failure. However, therapeutic implications of improvement in obstructive apnea-hypopnea in the setting of heart failure with established left ventricular systolic dysfunction and/or coronary artery disease could be different from those in OSA without heart failure. These may include several factors,3,4 such as improved myocardial oxygen delivery, decreased sympathetic activity, left ventricular transmural pressure, and afterload.

Effects of CPAP on Heart Failure Patients
With CSA
In 43% (9 of 21) of the patients with CSA, application of CPAP (7±2 cm H₂O) resulted in virtual normalization of disordered breathing (Figure 2). In these patients, the average AHI decreased from 36 to 4 per hour and central apnea index decreased from 23 to 2 per hour. As a result, arterial oxyhemoglobin desaturation was virtually eliminated and arousal index decreased considerably from 21 to 11 per hour. The latter was due to reduction in disordered breathing–related arousal index, which decreased from 12 to 2 per hour (P=0.008).

Despite considerable improvement in periodic breathing, CPAP failed to improve sleep efficiency and wakefulness after sleep onset. This appears to be unique to patients with heart failure, both with OSA and CSA even after long-term use.8 In contrast, even short-term application of CPAP in OSA without heart failure usually improves sleep characteristics.5

The majority of patients with heart failure and CSA failed to acutely respond to CPAP. We progressively increased

TABLE 2. Effects of CPAP on Ventricular Arrhythmias, Arousals, and Oxyhemoglobin Saturation During Sleep in Heart Failure Patients With Sleep Apnea

<table>
<thead>
<tr>
<th></th>
<th>CPAP-Nonresponsive</th>
<th>CPAP-Responsive</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVCs, n/h</td>
<td>223±357</td>
<td>192±321</td>
</tr>
<tr>
<td>Couplets, n/hr</td>
<td>4.7±9</td>
<td>5±8</td>
</tr>
<tr>
<td>VT, n/hr</td>
<td>0.4±1</td>
<td>0.9±2</td>
</tr>
<tr>
<td>Baseline SaO₂</td>
<td>96±1</td>
<td>95±3</td>
</tr>
<tr>
<td>Lowest SaO₂, %</td>
<td>74±12</td>
<td>86±6</td>
</tr>
<tr>
<td>SaO₂&lt;90%, %TST</td>
<td>31±17</td>
<td>12±17</td>
</tr>
<tr>
<td>ARI, n/hr</td>
<td>31±25</td>
<td>36±31</td>
</tr>
</tbody>
</table>

PVCs indicates premature ventricular contractions; VT, ventricular tachycardias; SaO₂, saturation measured by pulse oximetry; TST, total sleep time; ARI, arousal index. Two patients in the CPAP-responsive group had OSA, and the remaining patients had CSA; n=8 in each group. Values are mean±SD.
CPAP level to eliminate central apnea. However, the number of episodes of central apnea remained relatively unchanged (see Results). Upper airway occlusion is known to occur in patients with CSA,15 and Issa and Sullivan16 showed that high level (9 to 16.5 cm H2O) CPAP was required to eliminate CSA in patients without known left ventricular systolic dysfunction. In the present study in heart failure patients with CSA, we increased the pressure hoping to prevent CSA, but high level CPAP failed to eliminate these apneas in 57% of the patients and the mean AHI and central apnea index remained unchanged. These data suggest that even if upper airway collapse occurred in CSA, elimination of occlusion by CPAP did not eliminate CSA, indicating that pathogenesis of CSA and OSA differs at least in most patients with heart failure. This conclusion is further supported by the only patient with predominant episodes of OSA, which were completely eliminated by CPAP (obstructive AHI was zero); episodes of CSA increased (see Results).

We can only speculate why in 57% of patients with heart failure, CSA does not respond to CPAP. We found no significant differences in any of the demographics between CPAP-responsive and nonresponsive (Table 1). The only significant difference, however, was that nonresponsive patients had a more severe sleep apnea than responsive patients (AHI=62±22 versus 36±15 per hour). In the face of severe CSA, during the periods of repetitive cyclical hyperventilation after episodes of central apnea, CPAP may result in arousability that promotes ventilatory instability.17 Irrespective of the mechanism, however, severe CSA could be an important predictor of long-term CPAP nonresponsiveness in heart failure patients.

Effects of CPAP on Ventricular Irritability During Sleep
Comparing baseline to CPAP, there were no significant changes in the hourly rates of various ventricular arrhythmias in CPAP nonresponders (Table 2). However, in heart failure patients whose disordered breathing improved with CPAP, ventricular arrhythmias decreased. The hourly rate (during sleep) of premature ventricular contractions decreased from 66 to 18 (P=0.055), couplets from 3.2 to 0.2 per hour (P=0.031), and ventricular tachycardias from 1.1 to 0.05 per hour. We acknowledge that the number of patients was small (n=8 in each group) and normally there is considerable intra-individual variability in ventricular arrhythmias, yet the mean values of the various ventricular arrhythmias did not change significantly in CPAP nonresponders (Table 2).

There are at least 2 explanations for reduction in ventricular irritability in CPAP-responsive patients. CPAP improved arterial oxyhemoglobin desaturation and the number of arousals (Table 2), both of which are associated with sympathetic overactivity.18–23 In addition, improved saturation may have also improved myocardial oxygen delivery.

Although we did not measure sympathetic activity, this has been shown repeatedly in various studies and under a variety of circumstances. The studies of Somers and colleagues have shown increased sympathetic activity with arousals in normal men18 and men with central19 and OSA.20 Studies by Naughton and associates21 have shown increased plasma norepinephrine concentration in heart failure patients with CSA when compared with heart failure patients without CSA; and application of nasal CPAP decreased both norepinephrine plasma concentration and urinary excretion. Furthermore, it has been shown that major ventricular arrhythmias are associated with and potentially caused by cardiac sympathetic overactivity.24 Therefore, it is perhaps reasonable to speculate that the decrease in ventricular arrhythmias by CPAP was mediated by decreased sympathetic activity secondary to decrease in the number of arousals and improved saturation.

Decreased sympathetic activity by CPAP also decreases systemic vascular resistance and transmural left ventricular pressure, the latter in part due to changes in intrathoracic pressure as well.25 Thus, CPAP decreases left ventricular afterload resulting in an increase in left ventricular ejection fraction and improved pump failure.8 Because sudden death, presumably caused by ventricular arrhythmias, and pump failure are the 2 major causes of death in heart failure patients, CPAP by decreasing ventricular arrhythmias, as noted in the present study, and improving ejection fraction may improve survival in heart failure.

Decreased cardiac output and hypotension are potential complications of CPAP. We did not monitor hemodynamics during sleep; however, in our study none of the patients complained of chest discomfort or dizziness with use of CPAP. Regarding safety of CPAP in heart failure patients, 2 points are emphasized. Our patients were ambulatory and stable, and no one was in class IV of New York Heart Association classification. Most importantly, CPAP was increased gradually by increments of 1 cm H2O. In contrast, in studies which have reported decreased cardiac output, CPAP was used at predetermined high pressures, eg, 10 cm H2O. One study using the latter approach reported that heart failure patients with atrial fibrillation were particularly sensitive to CPAP.26 In our study, there were 5 patients with atrial fibrillation, and no one reported any adverse effects with gradual titration. Heart failure patients with low intravascular volume, receiving vasodilators or β-blockers may not be able to increase venous return to compensate for the sudden increases in intrathoracic pressure. Our data in 29 heart failure patients suggest that gradual titration of CPAP is a safe procedure. We avoid application of CPAP at a predetermined pressure of >5 cm H2O to patients with heart failure.

Limitations of This Study
As noted in Methods, for uniformity only male subjects were studied. We therefore acknowledge that the results of this study may not necessarily be applicable to female patients with heart failure.

As also noted in Methods, in the absence of esophageal pressure, classification of central versus obstructive disordered breathing events might have not been always accurate and some misclassification might have occurred. However, independent of the kind of sleep apnea, the results of this study show that CPAP virtually eliminated sleep-disordered breathing in 55% of the heart failure patients and improved ventricular arrhythmias. Therefore, the impact of this finding in a typical patient with heart failure and sleep apnea cannot be overemphasized.
Meanwhile, regarding classification of disordered breathing into central versus obstructive disordered breathing, the results of our studies and others suggest that misclassification of events should be limited. In all our studies, polysomnograms were scored blindly. Afterward, when the disordered breathing events were matched with demographics of patients, we have consistently observed that those patients classified as OSA are significantly heavier and have a higher prevalence of snoring than those classified as CSA (Table 1). Furthermore, with our pharmacological studies, eg, theophylline, as expected, only disordered breathing events classified central in nature decreased, with episodes of obstructive apnea remaining unchanged. Finally, in a study using esophageal balloon, it was concluded that thoracoabdominal excursions (measured qualitatively by mercury strain gauges) alone had a positive predictive value of $\approx 0.96$ for classifying central or obstructive apneas.

In summary, the results of this study show that CPAP is effective in eliminating sleep apnea in the majority of heart failure patients. As a result, arterial oxyhemoglobin saturation improves and the number of arousals and nocturnal ventricular irritability decrease. We speculate that long-term CPAP may favorably influence the natural history of heart failure and its morbidity and mortality.

Acknowledgment

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References

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