Effects of Nasal Continuous Positive Airway Pressure on Oxygen Body Stores in Patients With Cheyne-Stokes Respiration and Congestive Heart Failure*

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Study objectives: The mechanism(s) by which nasal continuous positive airway pressure (CPAP) is effective in the treatment of Cheyne-Stokes respiration (CSR) in patients with congestive heart failure (CHF) remains uncertain, and may involve an increase in total oxygen body stores (dampening), changes in central and peripheral controller gain, and/or improvement in cardiac function. The purpose of this study was to evaluate the effects of nasal CPAP on total oxygen stores, as measured by the rate of fall of arterial oxyhemoglobin saturation (dSaO₂/dt), to determine if dampening may play a role in the attenuation of CSR in patients with CHF.

Design: Prospective controlled trial.

Setting: University hospital.

Patients: Nine male patients (mean ± SD age, 59 ± 8 years) with CHF and a mean left ventricular ejection fraction (LVEF) of 16 ± 4%.

Interventions and measurements: All patients had known CSR, as identified on a baseline polysomnographic study. Patients then underwent repeat polysomnography while receiving nasal CPAP (9 ± 0.3 cm H₂O). The polysomnography consisted of recording of breathing pattern, pulse oximetry, and EEG. dSaO₂/dt was measured as the slope of a line drawn adjacent to the falling linear portion of the arterial oxygen saturation (SaO₂) curve associated with a central apnea. All patients underwent echocardiography and right-heart catheterization within 1 month of the study to measure LVEF and cardiac hemodynamics, respectively.

Results: There was a significant decrease in the apnea-hypopnea index (AHI) with nasal CPAP, from 44 ± 27 events per hour at baseline to 15 ± 24 events per hour with nasal CPAP (p = 0.004). When compared to baseline, dSaO₂/dt significantly decreased with nasal CPAP from 0.42 ± 0.15%/s to 0.20 ± 0.07%/s (p < 0.001). The postapneic SaO₂, when compared to baseline, significantly increased with nasal CPAP, from 87 ± 5% to 91 ± 4% (p < 0.05). The preapneic SaO₂ did not significantly change, from a baseline of 96 ± 2% to 96 ± 3% with nasal CPAP (p = 0.8). When compared to baseline, the apnea duration and heart rate did not change with nasal CPAP. While there was a significant correlation noted between baseline postapneic SaO₂ and dSaO₂/dt (r = 0.8, p = 0.02), no correlation was seen between baseline preapneic SaO₂ and dSaO₂/dt (r = 0.1, p = 0.7). A significant correlation was noted between baseline dSaO₂/dt and the AHI (r = 0.7, p = 0.02). With CPAP, there was a significant correlation noted between dSaO₂/dt and the AHI (R = 0.7, p = 0.04), but no correlation was noted between dSaO₂/dt and postapneic SaO₂ (R = 0.1, p = 0.8).

Conclusion: Nasal CPAP significantly decreases dSaO₂/dt and thus increases total body oxygen stores in patients with CSR and CHF. By increasing oxygen body stores, dampening may be one of the mechanisms responsible for the attenuation of CSR seen with nasal CPAP.

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Key words: Cheyne-Stokes respiration; congestive heart failure; dampening; nasal continuous positive airway pressure

Abbreviations: AHI = apnea-hypopnea index; BMI = body mass index; CHF = congestive heart failure; CPAP = continuous positive airway pressure; CSR = Cheyne-Stokes respiration; dSaO₂/dt = rate of fall of arterial oxyhemoglobin saturation; FRC = functional residual volume; LVEF = left ventricular ejection fraction; SaO₂ = arterial oxygen saturation; Svo₂ = mixed venous oxygen saturation; TST = total sleep time; Vo₂ = oxygen consumption

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Approximately 45 to 56% of patients with congestive heart failure (CHF) and a left ventricular ejection fraction (LVEF) of < 45% have Cheyne-Stokes respiration (CSR) during sleep. Characterized by a crescento-decrescendo alteration in tidal volume separated by periods of apnea or hypopnea, CSR has been associated with an increased mortality in patients with CHF. We have demonstrated that nasal continuous positive airway pressure (CPAP) and oxygen therapy were equally effective at decreasing the apnea-hypopnea index (AHI) in patients with CSR and CHF. Yet, the mechanism(s) by which these treatment modalities improve CSR is uncertain and may involve increasing total body oxygen stores (dampening), changes in central and peripheral controller gain, and/or improvement in cardiac function. In particular, it has been suggested that nasal CPAP is effective by decreasing interstitial edema and pulmonary vagal afferent stimulation, resulting in a decrease in central and peripheral controller gain. But an increased controller gain does not appear to be the sole mechanism responsible, as not all patients with CSR and CHF demonstrate an increased sensitivity to CO₂.

Dampening refers to the ability of the body to stabilize Pao₂ and Paco₂ during changes in ventilation. In patients with CHF, interstitial edema causes a decrease in functional residual capacity (FRC), leading to a decrease in total body oxygen and CO₂ stores. As a result, the respiratory system becomes unstable (underdamped), with exaggerated changes in Pao₂ and Paco₂ during transient changes in ventilation. CSR can develop as a result of these changes in blood gas tensions.

The rate of fall of arterial oxyhemoglobin saturation (dSaO₂/dt) has been shown to correlate with total body oxygen stores and is dependent on thoracic volume, resting total body oxygen consumption (V̇O₂), preapneic arterial oxygen saturation (SaO₂), and mixed venous oxygen saturation (SvO₂). We prospectively studied a group of patients with severe CHF (LVEF < 40%) and CSR to evaluate the effects of nasal CPAP on total body oxygen stores, as measured by dSaO₂/dt, to determine if dampening may play a role in the attenuation of CSR; and to determine which factors correlate with dSaO₂/dt in patients with CSR and CHF.

**Materials and Methods**

**Patient Selection**

Nine male patients with severe CHF (New York Heart Association class IV) were studied in a nonblinded manner. Patients were all in medically stable condition for at least 1 month prior to the start of the study, with no change in their treatment regimens during the study period. The patients were all known to have CSR, as identified during a baseline polysomnographic study as part of a previous study. Patients were excluded from the study for the following: (1) if there was an episode of acute pulmonary edema within 1 month prior to the study, (2) if there was a history of a cerebral vascular accident, or (3) if patients refused to complete all the required polysomnographic studies. Our institutional review board approved the protocol, and informed consent was obtained from all patients prior to the study.

Patients were admitted to a special inpatient heart failure unit where they were evaluated and listed for heart transplantation, as previously described. The patients’ treatment regimens were optimized, and all patients were ambulatory and participating in physical conditioning classes at the time of the study.

**Cardiac Hemodynamics and Echocardiography**

All patients underwent right-heart catheterization within 1 month of the study. Parameters that were obtained included measurement of right atrial pressure, pulmonary artery pressure, and pulmonary capillary wedge pressure. In addition, cardiac output was measured as the mean of three recordings using thermodilution technique. An echocardiogram was also obtained on each patient to determine LVEF.

**Therapeutic Interventions**

All patients were acclimated to nasal CPAP (REmsstar; Respironics; Murrysville, PA) during the daytime prior to their study night, as previously described. In brief, patients received nasal CPAP bid for 1- to 2-h periods while awake at an initial pressure of 5 cm H₂O. During a 5- to 7-day period, nasal CPAP was increased 2 cm H₂O/ld toward a goal of 10 to 12 cm H₂O, as tolerated. Nasal CPAP was administered during the study night at the maximal determined pressure (9 ± 0.3 cm H₂O).

**Sleep Studies**

Polysomnography consisted of recordings of abdominal and rib cage motion (Resp-EZ; EPM Systems; Midlothian, CA), finger pulse oximetry (model N-100; Nellcor Puritan Bennett; Pleasanton, CA), oral and nasal thermistors, ECG, electro-oculogram, digastic electromyogram, and EEG. The pulse oximeter used a signaling average for 3 to 15 s. Total sleep time (TST), and sleep efficiency (defined as TST divided by time in bed) were also determined. Arousals were defined as the appearance of α activity for 3 to 15 s. Central apneas were defined by the lack of airflow for > 10 s, associated with the absence of rib cage and abdominal movement.

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Central hypopneas were defined by a 50% decrease in airflow for > 10 s, associated with a decrease in rib cage and abdominal excursion and a lack of abdominal-rib cage paradox. The central AHI was expressed as the number of apneas and hypopneas per hour of sleep. CSR was determined to be present when the central AHI was ≥ 10 events per hour,7 with events associated with a crescendo-decrescendo alteration in breathing pattern characteristic of CSR. dSaO₂/dt was measured as the slope of a line drawn adjacent to the falling linear portion of the SaO₂ curve associated with a central apnea (Fig 1).17,18 The SaO₂ prior to the linear fall in SaO₂ is referred to as the preapneic SaO₂ (Fig 1), and the postapneic SaO₂ refers to the SaO₂ at the flattened portion of the curve, following the linear descent (Fig 1). For each study, the dSaO₂/dt, preapneic SaO₂, and postapneic SaO₂ were determined by calculating the mean of 10 consecutive measurements obtained during an episode of CSR. The 10 consecutive apneas were chosen during the second sustained episode of CSR, during the first half of the night, while the patient was in stable stage 2 sleep. There were no awakenings or movement times during the sequence of CSR that was utilized for the measurements.

Protocol

All patients underwent baseline polysomnography that identified the presence of CSR. Repeat polysomnography while administering nasal CPAP at the maximized tolerated pressure was then performed 12 ± 6 days after the baseline study. All patients were in stable condition with no change in their medical regimens between the two studies.

Statistical Analysis

Data are represented as mean ± SD. Paired t tests were used to compare baseline variables to those obtained with nasal CPAP.

The relationship between dSaO₂/dt and the preapneic SaO₂, postapneic SaO₂, and AHI were assessed by Pearson correlation coefficients.

RESULTS

Patient Characteristics

Nine male patients (mean age, 59 ± 8 years; body mass index [BMI], 28 ± 6) were studied (Table 1). The mean LVEF was 16 ± 4% on a maximized treatment regimen, which included a continuous infusion of an inotropic agent. Baseline cardiac hemodynamics demonstrated a cardiac index of 2.5 ± 0.6 L/min/m², mean pulmonary artery pressure of 31 ± 11 mm Hg, pulmonary capillary wedge pressure of 20 ± 8 mm Hg, and a heart rate of 78 ± 21 beats/min. The baseline AHI was 44 ± 27 events per hour, with a TST of 324 ± 59 min, sleep efficiency of 82 ± 8%, and an arousal index of 14 ± 13 arousals per hour (Table 1).

Effects of Nasal CPAP

Nasal CPAP resulted in a significant decrease in the AHI, from 44 ± 27 events per hour at baseline, to 15 ± 24 events per hour with nasal CPAP (p = 0.004). The apnea index significantly decreased with nasal CPAP, from 34 ± 31 to 14 ± 32 events per hour per hour with nasal CPAP.
Table 1—Patient Characteristics (n = 9)*

<table>
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<tr>
<th>Characteristics</th>
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<tbody>
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<td>Age, yr</td>
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<tr>
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<tr>
<td>BMI</td>
<td>28 ± 6</td>
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<td>LVEF, %</td>
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<tr>
<td>Cardiac index, L/min/m²</td>
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<tr>
<td>PCWP, mm Hg</td>
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<tr>
<td>Heart rate, beats/min</td>
<td>78 ± 21</td>
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<tr>
<td>AHI, events/h</td>
<td>44 ± 27</td>
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<tr>
<td>TST, min</td>
<td>324 ± 59</td>
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<tr>
<td>Arousal index, arousals/h</td>
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<td>Sleep efficiency, %</td>
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<td>11 ± 5</td>
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<td>Stage 2</td>
<td>67 ± 13</td>
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<td>Stage 3/4</td>
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<tr>
<td>REM</td>
<td>17 ± 12</td>
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<td>Dobutamine (n = 6)</td>
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<tr>
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*Data are presented as mean ± SD or No. PCWP = pulmonary capillary wedge pressure; REM = rapid eye movement; NYHA = New York Heart Association; ACE = angiotensin-converting enzyme.

An increase in total body oxygen stores, resulting in a dampening effect on the respiratory control system, is thought to be responsible for the effectiveness of oxygen in the treatment of CSR in patients with CHF.23 The effects of nasal CPAP on total oxygen body stores, and thus dampening, in these patients has not previously been investigated. There are three major findings in this study: (1) nasal CPAP significantly reduced dSao₂/dt in patients with

Figure 2. Effects of nasal CPAP on dSao₂/dt. Compared with baseline measurements, nasal CPAP resulted in a significant decrease in dSao₂/dt (*p < 0.001).

**DISCUSSION**

An increase in total body oxygen stores, resulting in a dampening effect on the respiratory control system, is thought to be responsible for the effectiveness of oxygen in the treatment of CSR in patients with CHF.23 The effects of nasal CPAP on total oxygen body stores, and thus dampening, in these patients has not previously been investigated. There are three major findings in this study: (1) nasal CPAP significantly reduced dSao₂/dt in patients with per hour (p = 0.006), with no significant change in the hypopnea index, from 11 ± 21 to 1 ± 1 events per hour (p = 0.2). When compared to baseline, nasal CPAP significantly decreased the dSao₂/dt, from 0.42 ± 0.15% to 0.20 ± 0.07%/s (p < 0.001; Fig 2). The postapneic Sao₂, when compared to baseline, significantly increased with nasal CPAP from 87 ± 5% to 91 ± 4% (p < 0.05; Fig 3). There was no significant change in the preapneic Sao₂, from a baseline of 96 ± 2% to 96 ± 3% with nasal CPAP (p = 0.8; Fig 4). When compared to baseline, there was no significant change in apnea duration with nasal CPAP, from 21 ± 3 to 21 ± 6 s (p = 0.9). Heart rate, when compared to baseline, did not significantly change with nasal CPAP, from 78 ± 21 to 73 ± 16 beats/min, respectively (p = 0.3).

At baseline, there was a significant correlation between postapneic Sao₂ and dSao₂/dt (r = −0.8, p = 0.02; Fig 5, top, A). There was no correlation noted between baseline preapneic Sao₂ and dSao₂/dt (r = −0.1, p = 0.7; Fig 5, bottom, B). Additionally, a significant correlation was noted between baseline dSao₂/dt and the AHI (r = 0.7,

Figure 3. Effects of nasal CPAP on postapneic Sao₂. When compared to baseline measurements, nasal CPAP significantly increased the postapneic Sao₂ (*p < 0.05). See Figure 1 legend for expansion of abbreviation.
CSR and CHF; (2) in patients with CHF and CSR, the baseline postapneic but not the preapneic \( \text{Sa}_2 \) correlated with \( \text{dSa}_2/\text{dt} \); and (3) there was a significant correlation between \( \text{dSa}_2/\text{dt} \) and the AHI.

The endogenous stores of oxygen of the body are rather limited (approximately 2 L), as compared to that of \( \text{CO}_2 \) (120 L), due to differences in tissue-binding affinities. \(^{24,25}\) Approximately 25% of the total oxygen stores of the body are contained in the lung and 15% in the tissues, with the majority (60%) contained in the blood due to the binding affinity for hemoglobin. \(^{25}\) During breath-holding, as well as with central and obstructive apneas, \( \text{dSa}_2/\text{dt} \), as a reflection of total oxygen body stores, has been shown to correlate with a number of factors, including thoracic volume, \( \dot{\text{V}}_\text{O}_2 \), preapneic \( \text{Sa}_2 \), and \( \text{Sv}_2 \). \(^{13-19}\)

The volume of oxygen stored in the lungs is equal to lung volume times the alveolar fractional concentration of oxygen. \(^{24}\) Findley et al. \(^{13}\) examined the impact of lung volume on the fall in \( \text{Sa}_2 \) during voluntary breath-holding in seven normal males. Initial lung volume was found to be the most important determinant for both the fall in \( \text{Sa}_2 \) as well as the minimal \( \text{Sa}_2 \). The most severe decreases in \( \text{Sa}_2 \) were noted at lower initial lung volumes (<2 to 3 L), more specifically at lung volumes below closing capacity where dependent airways are more susceptible to closure. Similarly, Hurewitz and Sampson \(^{14}\) demonstrated that lung volume (measured at FRC) was an important determinant for the rate of fall in alveolar oxygen tension during breath-holding. The decrease in alveolar oxygen tension was found to be responsible for the associated decrease in \( \text{Sa}_2 \).

Patients with CHF have a restrictive ventilatory pattern with a reduction in lung volumes. \(^{12}\) By recruiting alveoli and redistributing lung water, positive end-expiratory pressure may increase total oxygen body stores by increasing end-expiratory lung volume. \(^{26,27}\) CPAP has been shown to increase lung volumes, including FRC, at levels of 5 to 11 cm \( \text{H}_2\text{O} \) \(^{28-31}\) which is similar to the mean CPAP level of 9 cm \( \text{H}_2\text{O} \) used in our patients. Although lung volumes were not directly measured in our study, an increase in FRC could be responsible for the observed decrease in \( \text{dSa}_2/\text{dt} \) and thus the increase in total oxygen body stores seen with nasal CPAP.
The preapneic \( \text{Sao}_2 \) has been shown to be an important determinant for \( \text{dSaO}_2/\text{dt} \) during breath-holding as well as during central and obstructive apneas.\(^{15,17}\) Yet, other studies have not demonstrated as strong a correlation between the two variables,\(^{18}\) and a component of the relationship was attributed to a decrease in lung oxygen stores created by having the subjects breath hypoxic gas mixtures.\(^{15}\) In the present study, there was no correlation noted between baseline \( \text{dSaO}_2/\text{dt} \) and preapneic \( \text{Sao}_2 \). In addition, there was no significant change in preapneic \( \text{Sao}_2 \) with nasal CPAP. One possible explanation may involve the fact that the preapneic \( \text{Sao}_2 \) was 96% at baseline, with a further increase in \( \text{Sao}_2 \) expected to be marginal with nasal CPAP.

Another important determinant of \( \text{dSaO}_2/\text{dt} \) is the preapneic \( \text{SVO}_2.\)\(^{17,18}\) Since the majority of the oxygen stores of the body are contained in the blood, and 70% of the total blood volume is venous, it is understandable that a change in \( \text{SVO}_2 \) could effect \( \text{dSaO}_2/\text{dt} \), especially in the presence of underlying lung disease. In our study, we did not directly measure \( \text{SVO}_2 \) but we did measure postapneic \( \text{Sao}_2 \), which at baseline was found to inversely correlate with \( \text{dSaO}_2/\text{dt} \) (Fig 5, top, A). Postapneic \( \text{Sao}_2 \) may influence \( \text{SVO}_2 \), especially in patients with CHF where there is an increase in both the circulation time and oxygen extraction.\(^{7}\) The use of nasal CPAP resulted in a significant increase in postapneic \( \text{Sao}_2 \), with a possible corresponding increase in \( \text{SVO}_2 \).

Although apnea duration may affect postapneic \( \text{Sao}_2 \) values, nasal CPAP was noted to have no effect on apnea duration in our patients.

With nasal CPAP, an inverse correlation was no longer noted between \( \text{dSaO}_2/\text{dt} \) and postapneic \( \text{Sao}_2 \). Yet, it should be noted that with nasal CPAP, the postapneic \( \text{Sao}_2 \) had increased from 87 ± 5 to 91 ± 4 mm Hg. With an increase in the postapneic \( \text{Sao}_2 \) to >90%, its effect on \( \text{SVO}_2 \) may be less significant, with other factors, such as lung volume, becoming more important in determining total body oxygen stores.

A decrease in \( \text{VO}_2 \) could also account for the change in \( \text{dSaO}_2/\text{dt} \) seen with nasal CPAP, but this seems less likely. Differences in body weight are responsible for the correlation between the rate of fall of alveolar oxygen tension and \( \text{VO}_2 \) noted during breath-holding.\(^{14}\) Obese subjects have higher preapneic \( \text{V} \text{O}_2 \) measurements and thus a greater rate of fall of alveolar oxygen tensions. In the present study, our patients were not morbidly obese (BMI, 28 ± 6), and thus would not be expected to have an elevated baseline \( \text{VO}_2 \). Furthermore, the mechanism(s) by which nasal CPAP would decrease \( \text{VO}_2 \) is uncertain. Nasal CPAP may increase cardiac output in patient with CHF,\(^{32,33}\) with a corresponding increase in oxygen delivery resulting in either no change\(^{34}\) or an increase\(^{35}\) in \( \text{VO}_2 \).

Other mechanisms may be responsible for the decrease in \( \text{dSaO}_2/\text{dt} \) seen with nasal CPAP. As noted, nasal CPAP can have significant effects on cardiac function in patients with CHF.\(^{32,33}\) An increase in cardiac output may result in a significant increase in \( \text{SVO}_2 \), with a corresponding increase in total oxygen body stores. Others have suggested that a reduction in cardiac output could result in a decrease in \( \text{dSaO}_2/\text{dt} \), by its effect on the lung to chemoreceptor circulation time.\(^{20}\) Yet, it has previously been demonstrated that nasal CPAP, at levels similar to those used in the present study, have no effect on circulation time in patients with CSR and CHF.\(^{4}\)

Mechanisms other than underdampening appear to be important in the development of CSR in CHF. An increase in central and peripheral controller gain is thought to be responsible for the hyperventilation-induced resting hypocapnia seen in these patients.\(^{7-11}\) With an arousal induced hyperpnea, \( \text{PaCO}_2 \) decreases below the sleeping apneic threshold, resulting in a central apnea. In the presence of an increased central and peripheral controller gain, the ventilatory response to the increase in \( \text{PaCO}_2 \) at the end of an apnea is exaggerated, resulting in the development of CSR. Javaheri\(^{11}\) demonstrated that the ventilatory response to \( \text{CO}_2 \) was significantly elevated in those patients with, as compared to those without CSR, despite a similar degree of heart
failure. More recently, Solin et al\textsuperscript{10} reported that both central and peripheral CO\textsubscript{2} ventilatory responses are increased in patients with CSR as compared to those without CSR, as well as normal control subjects. Yet, in both studies, there was a considerable amount of overlap in the ventilatory response to CO\textsubscript{2} noted between the groups, emphasizing the importance of other mechanisms in the development of CSR. In addition, our study demonstrated a significant correlation between the baseline dSao\textsubscript{2}/dt and the AHI (r = 0.7, p = 0.02; Fig 6), suggesting that other mechanisms, such as under-dampening, may be important in the development of CSR.

Nasal CPAP has been shown to be effective in the treatment of CSR in CHF when used over a variable amount of time.\textsuperscript{4,9,33,36} One mechanism that appears to be responsible is the ability of nasal CPAP to increase Paco\textsubscript{2}, so that it remains well above the apneic threshold. Naughton et al\textsuperscript{8} demonstrated that 1 month of nasal CPAP resulted in an increase in nocturnal transcutaneous PCo\textsubscript{2}, secondary to a decrease in tidal volume. The authors postulated that an improvement in left ventricular function with nasal CPAP reduced interstitial edema and pulmonary vagal afferent stimulation, resulting in a decrease in central controller gain. Yet, the effects of nasal CPAP on central or peripheral controller gain have not been evaluated. In addition, although there was a decrease in tidal volume, other lung volumes, including FRC, will increase with nasal CPAP, resulting in an increase in the volume of oxygen stored in the lungs. Finally, it is important to note that although both the AHI and dSao\textsubscript{2}/dt decreased with nasal CPAP in our study, the correlation between the two indexes remained significant (R = 0.7, p = 0.04). Therefore, other mechanisms, including a dampening effect, may play a role in the effectiveness of nasal CPAP on CSR in patients with CHF.

There are a number of limitations with our study that need to be discussed. First, none of the variables that determine total oxygen body stores, and thus dSao\textsubscript{2}/dt, including measurement of lung volumes, were evaluated in the present study. Thus, it is uncertain which variables were affected by nasal CPAP that led to the significant attenuation in dSao\textsubscript{2}/dt. Second, the present study evaluated the acute effects of nasal CPAP on dSao\textsubscript{2}/dt. Whether more prolonged therapy will have a similar and sustained effect has yet to be determined. Third, the studies were performed in a nonblinded manner. We believe this did not effect the measurement of dSao\textsubscript{2}/dt, as it was performed in a standardized manner in a sleeping patient. Finally, in addition to apnea duration, the repetitive nature of the apneas may effect dSao\textsubscript{2}/dt, mostly by its effects on preapnea Sao\textsubscript{2} and Svo\textsubscript{2}.\textsuperscript{17} The dSao\textsubscript{2}/dt was calculated by taking the mean of 10 consecutive apneas at baseline and while administering nasal CPAP. We found no correlation between the 10 sequential baseline central apneas and their corresponding dSao\textsubscript{2}/dt, for all nine patients individually (data not shown), as well as for the group as a whole (r = −0.2, p = 0.6). Therefore, the repetitive nature of the central apneas did not effect total oxygen body stores, and the decrease in dSao\textsubscript{2}/dt observed with nasal CPAP is independent of its effects at decreasing the number of central apneas during the night.

In conclusion, nasal CPAP is effective at decreasing dSao\textsubscript{2}/dt and thus increasing total body oxygen stores in patients with CSR and CHF. By increasing total body oxygen stores, dampening may be one of the mechanisms partially responsible for the attenuation of CSR seen with nasal CPAP. In patients with CSR and CHF, baseline AHI and postapneic but not preapneic Sao\textsubscript{2} correlate with total oxygen body stores, as reflected in the measurement of dSao\textsubscript{2}/dt. Which physiologic variables are affected by nasal CPAP and are responsible for the decrease in dSao\textsubscript{2}/dt, and whether similar findings are seen after more prolonged use, awaits further investigation.

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