Prospective Evaluation of Nocturnal Oximetry for Detection of Sleep-Related Breathing Disturbances in Patients With Chronic Heart Failure*

Frédéric Sériès, MD; R. John Kimoff, MD; Debra Morrison, MD; Marie Helene Leblanc, MD; Mark Smilovitch, MD; Jonathan Houlett, MD; Alexander G. Logan, MD; John S. Floras, MD, Dphil; and T. Douglas Bradley, MD

Background: Because patients with chronic heart failure (CHF) can benefit from specific treatment for coexisting obstructive and central sleep apnea (CSA), there is a need to develop accurate screening tools to identify or exclude these sleep-related breathing disturbances (SRBDs) in patients with CHF.

Objectives: To evaluate, prospectively, the diagnostic value of nocturnal home oximetry in identifying SRBD in CHF patients and in distinguishing central events from obstructive events.

Design: Blinded comparison of hospital and home oximetry, and polysomnographic nocturnal recordings

Setting: Cardiac heart failure and sleep clinics in three tertiary referral centers.

Patients: Fifty consecutive patients who were investigated for participation in the Canadian Continuous Positive Airway Pressure Trial for Congestive Heart Failure with Central Sleep Apnea and were recruited from three different centers.

Measurements and results: Patients underwent two oximetry recordings, one at home and one during a polysomnographic study. The criterion for an SRBD was the presence of > 15 apneas and hypopneas per hour of sleep during polysomnography or an oxygen desaturation index of > 10 events per hour during oximetry. The pattern of desaturation/resaturation during oximetry was also examined to distinguish obstructive events from central events. Using a 2% fall in pulse oximetric saturation as the criterion for oxygen desaturation, home oximetry had a 85% sensitivity and a 93% specificity (p < 0.001) for detecting an SRBD. However, the desaturation/resaturation pattern did not accurately distinguish between obstructive events and central events (eg, 100% sensitivity, 17% specificity for identifying CSA). The interpretation of the oximetry recording was highly consistent between scorers (p < 0.001).

Conclusions: Overnight home oximetry is a sensitive and specific tool for identifying SRBDs in patients with CHF, but not for distinguishing between obstructive and central events in such patients.

(CHEST 2005; 127:1507–1514)

Key words: diagnosis; oxygen; sleep

Abbreviations: AHI = apnea-hypopnea index; CANPAP = Canadian Continuous Positive Airway Pressure Trial for Congestive Heart Failure with Central Sleep Apnea; CHF = congestive heart failure; CSA = central sleep apnea; ODI = oxygen desaturation index; OSA = obstructive sleep apnea; \( \text{SpO}_2 \) = pulse oximetric saturation; SRBD = sleep-related breathing disorder

The prevalence of congestive heart failure (CHF) in the American population is 1.5 to 2% and increasing.1 Health care agencies are faced with increasing CHF-related costs2 due to the aging of the population and reduced cardiovascular mortality, but continued high health care resource utilization by CHF patients. Sleep-related breathing disorders (SRBDs) are involved in the pathophysiology and progression of CHF3 through a variety of mechanisms, including increased sympathetic activity, decreased arterial oxygen saturation, loss of the sleep-related decrease of systemic arterial pressure, and increased left ventricular wall stress.4,5 SRBDs consist of repetitive episodes of the complete or partial cessation of airflow during sleep (apnea and hypopnea, respectively). Obstructive sleep apnea (OSA) and central sleep apnea (CSA) are two major forms of SRBD. In OSA, reduced airflow is due to sleep-induced pharyngeal closure that is associated with increasing breathing efforts. In contrast, reduced airflow in CSA is due to a primary reduction in central respiratory drive. In the setting of CHF, recurrent episodes of central apnea or hypopnea alternate with hyperpnea in a crescendo/decrescendo pattern of tidal volume5 that characterize Cheyne-Stokes breathing.
SRBDs are much more common in patients with CHF than in the general population. The two largest studies completed in CHF patients have reported prevalences of 62% of 450 subjects,6 and 51% of 81 subjects,7 whereas in the general population the prevalence of SRBDs stands at 9% in women and 24% in men.8 In patients with CHF, CSA is associated with poor prognosis independent of other risk factors.9 More importantly in CHF patients, the treatment of both OSA and CSA with nasal continuous positive airway pressure improves left ventricular function,10 reduces sympathetic nervous system activity,11 and may reduce the combined mortality and heart transplantation rate.12 For these reasons, the identification of SRBDs in CHF patients is of considerable importance. The diagnosis of SRBD requires recordings of respiratory variables during sleep. Such recordings are ideally performed in a sleep laboratory to obtain supervised continuous recordings of physiologic variables. However, in-laboratory sleep studies are costly and labor-intensive, are of limited availability, and often require long waiting times to be completed. Given the high prevalence of SRBDs in the CHF population, and the relative inaccessibility of sleep laboratories for the timely performance of sleep studies, there is an urgent need to identify simple screening devices that would allow the recognition of nocturnal breathing disturbances outside sleep laboratories in these patients.

We have previously demonstrated that home oximetry is a sensitive tool for identifying OSA.13 However, these data were obtained in subjects without CHF who were mostly found to have OSA. Since OSA and CSA occur in approximately equal proportions in CHF patients, and considering the less pronounced falls in pulse oximetric saturation (SpO2) that result from CSA compared to OSA,6,14 the sensitivity of nocturnal oximetry for identifying SRBDs may be different in CHF patients. In addition, the characteristics of ventilatory resumption and the length of the apnea-hypopnea/hyperventilation cycle differ between OSA and CSA, which may lead to differences in the desaturation/resaturation pattern.15

The primary aims of the present study were to evaluate the diagnostic value of nocturnal oximetry in the identification of SRBDs in CHF patients, and to compare its reliability during home and laboratory recordings. As a secondary end point, we also evaluated whether CSA and OSA can be reliably distinguished based on the evaluation of the SpO2 desaturation-resaturation pattern.

**Materials and Methods**

**Study Population**

Fifty consecutive patients with CHF who were undergoing investigation for eligibility for the Canadian Continuous Positive Airway Pressure Trial for Congestive Heart Failure with Central Sleep Apnea (CANPAP)16 participated in this study. All patients were accepted to participate. The eligibility criteria are detailed in Table 1. These were identical to those of the CANPAP trial16 except that polysomnographic results were excluded from inclusion/exclusion criteria. Patients were recruited from three centers participating in the CANPAP trial (Montreal, Halifax, and Quebec City). The protocol had been approved by the ethical review board of these institutions. Written informed consent was obtained from each subject prior to their participation in the protocol.

**Oximetry Recordings**

Continuous nocturnal SpO2 monitoring was obtained with a recording system (Stardust; Respironics; Murrysville, PA) using

**Table 1—Eligibility Criteria**

<table>
<thead>
<tr>
<th>Eligibility Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inclusion</strong></td>
</tr>
<tr>
<td>Men and women between 18 and 79 years of age with a history of at least one clinical episode of congestive heart failure due to ischemic, hypertensive, or idiopathic dilated cardiomyopathy</td>
</tr>
<tr>
<td>Left ventricular systolic dysfunction as shown by a left ventricular ejection fraction at rest determined by equilibrium radionuclide angiography of &lt; 40% while receiving optimal drug therapy at the time of recruitment</td>
</tr>
<tr>
<td>New York Heart Association functional class 2 to 4</td>
</tr>
<tr>
<td>Stable condition and stable optimal cardiac medications for at least 4 weeks before entry</td>
</tr>
<tr>
<td>Written informed consent</td>
</tr>
<tr>
<td>Exclusion</td>
</tr>
<tr>
<td>History of unstable angina, cardiac surgery, and/or documented myocardial infarction &lt; 3 mo before study entry</td>
</tr>
</tbody>
</table>
only the pulse oximeter with a finger probe and the body position sensor. \(\text{SpO}_2\) was recorded from a finger probe using at 1-Hz sampling frequency and an 8-s averaging time, allowing for adequate resolution and stability of the signal. For home recordings, the subjects came to the sleep laboratory for an explanation of the installation procedures. Patients were instructed to initiate the recording by pushing the key button of the device when turning lights off and to stop the recording when they awoke in the morning. Patients then returned to the sleep laboratory the next day where their home monitoring data were downloaded to a computer with dedicated software for data interpretation.

**Polysomnographical Study**

Polysomnography consisted of in-laboratory continuous acquisitions of the EEG, the electrooculogram, the submental and tibial electromyogram, the arterial oxyhemoglobin saturation from transcutaneous sensing (ie, \(\text{SpO}_2\)) with a pulse oximeter and finger probes, the nasooral airflow with thermistors and nasal prongs connected to a pressure transducer, the chest and abdominal movements by inductive plethysmography (Respirtrac; Ambulatory Monitoring Inc; Ardsley, NY), and the ECG. All variables were digitally recorded (Sandman Elite system; Tyco; Ottawa, ON, Canada). Sleep-wake state and arousals were scored according to standardized criteria. Breathing abnormalities were scored as apneas (ie, the absence of flow for at least 10 s) or hypopneas (ie, a decrease of \(\geq 50\%\) in the sum of plethysmography or nasal pressure tracings baseline for at least 10 s) without any requirement for subsequent arousal or a minimal \(\text{SpO}_2\) fall. Events were identified as central in the absence of movements (apnea) or as a proportional reduction with in-phase (hypopnea) movements of the rib cage and abdomen. Obstructive events were characterized by out-of-phase thoracoabdominal movements and/or increasing respiratory efforts. The apnea-hypopnea index (AHI) was defined as the number of apneas plus hypopneas per hour of sleep. According to CANPAP eligibility criteria, the diagnosis of sleep apnea was defined by an AHI of > 15 events/h, and its classification as either OSA or CSA was determined by the predominant (ie, > 50%) type of breathing abnormality.

**Study Design**

The following two oximetry recordings were performed using the same pulse oximeter: one at home and the other in the sleep laboratory during the polysomnography recording in addition to above-detailed recorded variables. The two recordings were carried out within 2 weeks of each other in random order. Home oximetry tracings were considered to be adequate if the subjects reported that they had slept > 4 h and that their sleep was not disturbed or was minimally disturbed by the recording procedure. If these criteria were not met, a second home recording was performed.

**Data Analysis**

**Interpretation of the Oximetry Recordings**: For each subject, the home and in-laboratory oximetry tracings were interpreted blindly with respect to each other and to the polysomnography results. An event-by-event interpretation was performed manually by one investigator at each center (D.M., J.K., and F.S.) from a review of the continuous real \(\text{SpO}_2\) signal using a 5-min window display resolution. Desaturation events were defined as a transient (ie, \(\leq 1\) min) fall in \(\text{SpO}_2\) of \(\geq 2\%\) followed by a rise in \(\text{SpO}_2\). There was no absolute \(\text{SpO}_2\) threshold to be reached for a desaturation event to be scored in either analysis. The oxygen desaturation index (ODI) was calculated as the number of desaturations per hour of recording.

In view of the inherent problems involved in comparing AHI values from polysomnography (ie, the No. of events per hour of sleep) with ODI from oximetry recordings (ie, the No. of events per hour of recording), we considered a priori that the cutoff value for the ODI should take into account differences in sleep time and recording duration. Given that the number of sleep hours is equivalent to the recording time multiplied by the sleep efficiency, and that the mean sleep efficiency was approximately 70% (see below), the ODI threshold was set at 10 events/h of recording (AHI cutoff on polysomnography = 15 events per hour of sleep = 15 events/[recording hour × sleep efficiency] = 15 events/[recording hour × 0.7] = 10 events/recording hour).

The interpreter also had to identify the obstructive or central nature of the SRBD-related desaturations according to the desaturation/resaturation pattern. The following two different types of desaturation could be scored: (1) progressive desaturation followed by a progressive rise in \(\text{SpO}_2\) (rounded, smooth wave pattern) compatible with CSA (Fig 1, top, A); or (2) progressive desaturation followed by a rapid increase in \(\text{SpO}_2\) (sawtooth waveform pattern), suggestive of OSA (Fig 1, bottom, B). Each abnormal oximetry recording was classified as obstructive or central in nature according to the types of the most frequent desaturations. The interscorer variability for the analysis of the oximetry tracings was assessed by conducting an indepen-

---

**Figure 1.** Typical examples of oximetry recordings demonstrating regular \(\text{SpO}_2\) falls that are suggestive of SRBDs. **Top, A:** the symmetry of the desaturation/resaturation pattern suggests CSA. **Bottom, B:** a slow desaturation pattern is followed by a rapid increase in \(\text{SpO}_2\) characteristic of OSA.
dent blinded analysis by all three scorers of the oximetry tracings from 14 subjects who were randomly selected from the three centers.

Statistical Analysis

Results of continuous measures were expressed using the mean ± SD. One-way analysis of variance was used to compare parameters among the three groups of patients (OSA, CSA, and normal). AHI and sleep efficiency (percent) were log-transformed and square-root-arc sinus-transformed, respectively, to stabilize the variances. Statistical analyses for these parameters were performed using transformed values. The SpO₂ percentage < 90% was analyzed with the rank transformation on data and using the one-way analysis of variance. A randomized block design was used to compare the results of the oximetry and polysomnography recordings. The normality assumption was verified with the Shapiro-Wilk test, and the Brown and Forsythe variation of the Levene test statistic was used to verify the homogeneity of the variances. The diagnosis of SRBD as well as its classification as mainly obstructive or central in nature were analyzed with the Fisher exact test. Bland-Altman plots were used to assess the agreement between home and in-laboratory ODIs. Intraclass correlation was used to calculate reliabilities among raters.¹⁹ The results were considered to be significant at p values of ≤ 0.05. The data were analyzed using a statistical software package program (SAS, version 8.2; SAS Institute; Cary, NC).

Results

The characteristics of the subjects are reported in Tables 1, 2. Three oximetry recordings were not completed during polysomnography (two recording failures and one omission), and three other recordings were not completed at home (one recording failure, one omission, and one refused by the patient). Two tests had to be repeated at home because of insufficient sleep recording. There was no difference in the baseline SpO₂ values between the polysomnography and the in-laboratory and/or home oximetry recordings (Table 3). In all cases, the ODI was significantly lower than the AHI (Table 3). The percentage of time spent at < 90% SpO₂ was significantly less during the home oximetry recording than during the laboratory recording. As represented in Figure 2, there was a good agreement between the sleep laboratory and home oximetry recordings. There was no systematic discrepancy between the values of the ODI obtained inside vs outside the sleep laboratory (mean difference in ODI, −5.2 events/h) [Fig 2]. No difference was found between the percentage of time spent in the supine position and the nonsupine position among the different recordings.

Based on polysomnography results, the presence of SRBDs was identified in 36 of 50 subjects (72%), with 12 of 36 subjects (33%) having OSA and 24 of 36 subjects (66%) having CSA. The home ODI was abnormal in 28 of 33 subjects, with SRBD false-positive results found in 1 of 14 subjects (Table 4). This corresponded to an 85% sensitivity and a 93% specificity. Abnormal oximetry findings were significantly more common in subjects with SRBDs (χ² test, 23.6; p < 0.0001). Six patients (12%) had atrial fibrillation, and all patients had abnormal AHIs on polysomnography. The oximetry tracing concluded that SRBDs were present in five of six patients, corresponding to an 83% sensitivity. The likelihood ratio for SRBDs on polysomnography was 11.8. Therefore, in our study population, given that 72% of our patients had sleep apnea, the likelihood of SRBDs increased to 97% with a positive home oximetry recording and decreased to 5.8% with a negative home oximetry recording. Separating the results obtained in different body positions did not help to refine the diagnosis value of oximetry recordings.

### Table 2—Anthropomorphic, Left Ventricular Ejection Fraction, and Polysomnographic Characteristics of Participating Patients

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Total</th>
<th>OSA</th>
<th>CSA</th>
<th>NL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects, No.</td>
<td>50</td>
<td>12</td>
<td>24</td>
<td>14</td>
</tr>
<tr>
<td>Age, yr</td>
<td>63 ± 10</td>
<td>61 ± 9</td>
<td>64 ± 9</td>
<td>61 ± 12</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>41</td>
<td>11</td>
<td>22</td>
<td>8</td>
</tr>
<tr>
<td>Female</td>
<td>9</td>
<td>1</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>29.5 ± 5.2</td>
<td>31.5 ± 5.3</td>
<td>27.7 ± 4.6</td>
<td>30.8 ± 5.6</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>29.0 ± 8.2</td>
<td>29.3 ± 9.2</td>
<td>28.5 ± 8.2</td>
<td>29.7 ± 7.9</td>
</tr>
<tr>
<td>TST, h</td>
<td>5.7 ± 1.2</td>
<td>6.2 ± 1.1</td>
<td>5.4 ± 1.0</td>
<td>5.8 ± 1.4</td>
</tr>
<tr>
<td>SE, %</td>
<td>7.1 ± 14</td>
<td>77 ± 13</td>
<td>68 ± 12</td>
<td>73 ± 18</td>
</tr>
<tr>
<td>AHF events/h</td>
<td>27.2 ± 20.2</td>
<td>31.8 ± 23.2</td>
<td>37.1 ± 14.9</td>
<td>6.3 ± 4.2†</td>
</tr>
<tr>
<td>Baseline SpO₂, %</td>
<td>96.5 ± 1.5</td>
<td>96.7 ± 1.1</td>
<td>96.0 ± 1.7</td>
<td>97.2 ± 1.1</td>
</tr>
<tr>
<td>SpO₂ &lt; 90%, % TST</td>
<td>8.4 ± 18.1</td>
<td>11.0 ± 21.0</td>
<td>11.8 ± 21.1</td>
<td>0.7 ± 1.8‡</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD, unless otherwise indicated. BMI = body mass index; LVEF = left ventricular ejection fraction; TST = total sleep time; SE = sleep efficiency; NL = normal, no SRBD.

†p < 0.05 compared to other groups.
The accuracy of the oximetry analysis in predicting the type of nocturnal breathing disorder was evaluated using in-laboratory recordings in patients with an abnormal AHI. Patients with CSA were all accurately identified by the analysis of the oximetry tracing (100% sensitivity) [Table 5]. However, the majority of desaturation events were incorrectly attributed to central events in 10 patients with predominant OSA (specificity, 17%). All patients with atrial fibrillation were found to have had Cheyne-Stokes respiration during polysomnography. This was confirmed by home oximetry in five of six subjects (83% sensitivity).

Anthropometric characteristics and polysomnographic results for the subset of patients that was used to assess the interscorer reproducibility of oximetry scoring did not differ significantly from those of the other 36 subjects. The identification of the presence or absence of breathing abnormalities was highly consistent among the three scorers (κ = 0.99; p < 0.001). A significant consistency was also found when quantifying the interscorer reliability of the analysis of the desaturation/resaturation pattern (κ = 0.50; p < 0.001).

Discussion

We demonstrated that home nocturnal oximetry accurately identifies SRBDs in patients with CHF. Apart from case reports or observational reports, the literature on the accuracy of oximetry recording to identify SRBDs in CHF patients is scanty. Staniforth et al investigated this issue by conducting home oximetry in 104 CHF patients and 21 healthy volunteers. Polysomnography was performed in only 41 patients. Based on the fact that the oximetry tracing was equivocal or abnormal in 81% of patients, and in 28% of healthy volunteers, it was concluded that overnight oximetry is a useful screening tool for CSA in patients with known CHF. Unfortunately, this conclusion was not supported by validation of the oximetry data using the “gold standard” of polysomnography for most of the subjects. Accordingly, it was not possible to determine the prevalence of SRBD, CSA, and OSA in that study. Considering the high rate of cardiac rhythm disturbances such as ventricular tachycardia in CHF patients with CSA, other home monitoring techniques, such as 24-h ambulatory ECG recording, could be potentially used to identify SRBDs in these patients. However, some results have suggested that such abnormalities are predominantly observed in patients with severe CSA, and the applicability of 24-h ambulatory ECG recording as a tool to identify the whole range of SRBDs remains to be determined.

Table 3—Respiratory Variables (Polysomnography vs Oximetry)*

<table>
<thead>
<tr>
<th>Variables</th>
<th>PSG (n = 50)</th>
<th>In-Laboratory Oximetry (n = 47)</th>
<th>Home Oximetry (n = 47)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI, events/h of TST</td>
<td>27.2 ± 20.2</td>
<td>23.5 ± 17.2†</td>
<td>17.1 ± 14.9†</td>
</tr>
<tr>
<td>ODI with 2% threshold, events/h of TRT</td>
<td>96.5 ± 1.5</td>
<td>96.7 ± 1.1</td>
<td>97.1 ± 1.5</td>
</tr>
<tr>
<td>Baseline Spo2, %</td>
<td>8.4 ± 18.1</td>
<td>8.3 ± 17.5</td>
<td>3.3 ± 9.6†</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD. PSG = polysomnography; TRT = total recording time. See Table 2 for abbreviation not used in the text.
†p < 0.05 compared to PSG.

Table 4—Diagnostic Value of Home Nocturnal Oximetry in Identifying SRBDs*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normal</th>
<th>SRBD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oximetry</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>13</td>
<td>5</td>
</tr>
<tr>
<td>SRBD</td>
<td>1</td>
<td>28</td>
</tr>
</tbody>
</table>

*See Table 3 for abbreviation not used in the text.
Table 5—Diagnostic Value of Oximetry Recording to Identify OSA vs CSA in Patients With SRBD*

<table>
<thead>
<tr>
<th>Variables</th>
<th>OSA</th>
<th>CSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oximetry</td>
<td></td>
<td></td>
</tr>
<tr>
<td>OSA</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>CSA</td>
<td>10</td>
<td>24</td>
</tr>
</tbody>
</table>

*See Table 3 for abbreviation not used in the text.

It is important to emphasize that our study fulfills some recommendations24 that diagnostic techniques should be evaluated in subjects who are representative of the population in which the technique is to be applied. In this regard, home oximetry was applied directly to patients in the population of interest, that is, stable but symptomatic outpatients with CHF who were being evaluated for eligibility for inclusion in the CANPAP trial,12 rather than applied to patients who had been referred to a sleep disorders clinic. Furthermore, the cutoff values of the main dependant variable (ie, ODI) were determined a priori in opposition to an explorative study in which receiver operating characteristic curves are used to determine the best cutoff diagnostic values.

The following several factors could have theoretically altered the discriminative power of oximetry recordings: the normal baseline SpO2 value; the fact that oxygen desaturation was not included in the definition of SRBD during polysomnography recordings; and the more shallow dips in SpO2 following CSA than those following OSA.25 These issues were possibly counterbalanced by the age and CHF-related decline in pulmonary function parameters, which are significant determinants of apnea-related desaturation.26,27 The absence any absolute threshold value for scoring desaturation events and the consideration of a 2% SpO2 desaturation amplitude threshold also contributed to the highly discriminative value of oximetry recordings. Under these conditions, our data indicated that home oximetry is a highly reliable screening tool for identifying SRBDs in patients with CHF.

The second aim of the study was to evaluate the accuracy of the desaturation/resaturation pattern in distinguishing OSA from CSA on the oximetry tracing. Animal studies28 and human studies25 have suggested that the pattern of oxyhemoglobin desaturation/resaturation is likely to differ between OSA and CSA as a consequence of the differences in the amplitude of SpO2 fall23 and in the pattern of the resumption of ventilation following OSA and CSA (ie, abrupt or progressive rise, respectively, to peak ventilation following apneic event).21 In subjects with normal cardiac function, ventilation and cardiac output abruptly increase following the resumption of OSA,20,30 which accounts for abrupt rises in SpO2 in the lung and its rapid transmission to the systemic circulation. In contrast, following CSA, ventilation rises gradually to a peak 10 to 20 s after the end of the apnea in CHF patients.31 As a result, SpO2 in the lung rises slowly and is transmitted slowly to the systemic circulation owing to low cardiac output. As a result, oximeters detect rapid increases in SpO2 following OSA, but detect a gradual rise in SpO2 following CSA. This justified our assumption that the desaturation/resaturation pattern is only reliable when it indicates the presence of OSA in patients with CHF. There are two potential explanations for such a finding. The first relates to factors influencing the rate of rise of SpO2 following central vs obstructive events. In patients with CHF, OSA causes more pronounced reductions in cardiac output that persist longer into the postapneic period than in subjects with normal cardiac function.32 Hence, even if there is an abrupt increase in ventilation in the postapneic period, the persistent depression of cardiac output limits the rise in SpO2 in the systemic circulation. In this instance, in CHF patients, the rate of rise in SpO2 following OSA could resemble the pattern of resaturation following CSA. This may have limited our ability to distinguish between central and obstructive events on the basis of the SpO2 desaturation/resaturation pattern.

The second explanation relates to the characteristics of the oximeter used in this study. The particular oximeter (Stardust; Respironics) was used because it has a reasonably short signal-averaging time of 8 s and provides a continuous recording of raw data to allow the manual interpretation of SpO2 recordings. Signal-averaging time is an important determinant of the desaturation/resaturation pattern (ie, the dampening and smoothing of the SpO2 signal increasing with averaging time).33 This dampening would impair the ability of the oximeter to discriminate between obstructive and central events. It would be interesting to determine whether the identification of the obstructive or central nature of an SRBD could be improved by the use of an oximeter with a shorter averaging time. However, it is our clinical experience from in-laboratory sleep recordings that a slow resaturation pattern can be observed with oximeters having signal-averaging times as low as 2 s. On the other hand, caution is required when extending the results of our study to oximeters with longer signal-averaging times (which may be set as long as 21 s in some devices) since the results of oximetry recordings are significantly influenced by the aver-
aging time. It is also conceivable that additional data characterizing the specific patterns of the \( \text{SpO}_2 \) changes have to be considered in the distinction between obstructive and nonobstructive events such as reoxygenation levels as well as the variability of the amount of desaturation across the night. From a clinical point of view, the identification of the obstructive or nonobstructive nature of SRBDs in CHF patients may have a marginal impact on the management of these patients since the proportion of each event can vary throughout the night and over time with fluctuation in left ventricular function, and the treatment of both OSA and CSA with nasal continuous positive airway pressure improves left ventricular function and may reduce the combined rate of mortality and heart transplantation.

Methodological issues can be raised concerning the criteria retained to categorize the presence of sleep apnea in our CHF patients. In the Sleep Heart Heath Study, an AH\( \text{i} \) of more than five events per hour has been found to be associated with an increased cardiovascular risk. However, in that large population-based study, breathing abnormalities had to be associated with a 3% fall in \( \text{SpO}_2 \) to be scored. Such interpretation criteria cannot be applied to the identification of CSA, which is characterized by periodic changes in ventilation independently of the resulting changes in \( \text{SpO}_2 \). Any difference in scoring rules obviously has an impact on the AH\( \text{i} \) threshold; for example, in our study 43 of 50 patients would have been classified as apneic if the Sleep Heart Heath Study criteria had been applied. In this context, the scoring rules used in the CANPAP study justify the use of a higher AH\( \text{i} \) threshold. Furthermore, such a choice is further strengthened by its ability to limit the influence of night-to-night variability of the SRBD on the sleep apnea status of our subjects.

The present results establish that overnight home oximetry is a reliable tool to screen for the presence of SRBDs in patients with CHF. Presently, owing to the limited awareness of SRBDs among physicians caring for patients with CHF, as well as limited access to, and the relatively high cost of in-laboratory polysomnography, only a minority of CHF patients benefit from the diagnosis and treatment of SRBDs. Home oximetry therefore provides a readily available, inexpensive means of detecting SRBDs in these patients. Although it does not rule out OSA in the presence of symmetrical \( \text{SpO}_2 \) fluctuations, a positive oximetry recording would assist in prioritizing patients for full polysomnography based on the severity of their SRBD. In this way, many more CHF patients may benefit from the timely diagnosis and treatment of SRBD than might otherwise be the case.

ACKNOWLEDGMENT: The authors acknowledge the research nurses and assistants (Isabelle Simard, Diane Page, Danielle Petit, Sylvie Lavallée, Charlene Barber, and Kathy Spurr) for their valuable contribution to this study through their sustained efforts to recruit patients for the CANPAP trial.

REFERENCES

36 Collop NA. Cheyne-stokes ventilation converting to obstructive sleep apnea following heart transplantation. Chest 1993; 104:1288–1289