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Original Article

Arterial oxygen saturation during Cheyne-Stokes respiration in heart failure patients: does measurement site matter?

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ABSTRACT

Objective: Despite the fact that the ear is the site to monitor arterial oxygen saturation by pulse oximetry (SpO2) closest to carotid chemoreceptors, sleep studies almost invariably use finger probes. This study aimed to assess the timing and morphological differences between SpO2 signals at the ear and finger during Cheyne–Stokes respiration (CSR) in heart failure (HF) patients.

Methods: We studied 21 HF patients with CSR during a 40-min in-laboratory resting recording. SpO2 was recorded by: (1) two identical bedside pulse-oximeters with an ear (SpO2_Ear) and a finger probe (SpO2_Finger), and (2) a standard polysomnograph with a finger probe (SpO2_PSG). We estimated the delays between signals and, for each signal, computed the mean and minimum SpO2, the magnitude of cyclic desaturations and the overall time spent with SpO2<90% (T90%).

Results: The SpO2_Finger signal was significantly delayed [bias: 12.7 s (95% limits of agreement: 2.2, 23.0 s)] and slightly but not significantly downward-shifted with respect to SpO2_Ear. SpO2_PSG was almost synchronous with SpO2_Finger; however, a further significant downward shift was observed relative to the latter. In particular, minimum SpO2_PSG was significantly lower [−2.1% (− 4.8, 0.6%)], and desaturations and T90% were significantly higher than SpO2_Finger [1.2% (−1.3, 3.7%), and 13.9% (−12.3, 40.0%), respectively].

Conclusion: During CSR in HF patients, the marked delay between SpO2_Ear and SpO2_Finger makes the interpretation of the timing relationship between peripheral chemoreceptor stimulation and ventilatory events rather difficult. The observed discrepancies between SpO2_PSG and SpO2_Finger, which may be due to differences in the processing of raw SpO2 signals, call for a standardization of processing algorithms.

1. Introduction

Moderate-to-severe sleep-related breathing disorders affect a large portion (about 50%) of chronic heart failure (HF) patients receiving therapy according to current guidelines [1,2]. These disorders most often manifest in the form of Cheyne-Stokes respiration (CSR) [1], a type of periodic breathing in which apneas and hypopneas, caused by cyclic cessations/reductions of central respiratory drive, alternate with ventilatory periods having a waxing and waning pattern of tidal volume [3]. It is widely accepted that CSR results from instability in the chemical feedback system controlling ventilation [4–6]. The most important factors determining this instability are a prolonged circulation time, causing delays in transmitting changes in arterial blood gas tensions from the lungs to the chemoreceptors, and an increased ventilatory response to chemoreceptor stimulation [4,7].

Pulse oximetry is the standard technique for noninvasive monitoring of arterial oxygen saturation (SpO2) in the assessment of sleep-related breathing disorders by overnight polysomnography [8]. Indeed, several common indices of severity, namely the oxygen desaturations index and the mean and minimum value of oxygen saturation, are derived from the SpO2 signal. Moreover, pulse oximetry values constitute a key parameter

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in scoring respiratory events because the identification of hypopneic events, which is essential for the computation of the apnea–hypopnea index (AHI), relies on the observation of corroborative reductions ≥3% in SpO2 following a reduction in breathing amplitude [8,9]. Besides its relevance in diagnosis, pulse oximetry has recently been proved to play an important role in the prognosis of patients with stable HF, because nocturnal hypoxemia, as assessed by the time spent with SpO2 < 90% (finger probe), has been shown to be a powerful and independent predictor of mortality [10].

Pulse oximetry is also frequently used in the investigation of pathophysiological mechanisms and of cardiovascular and autonomic effects of CSR [11,12], owing to the fact that this is the simplest technique for continuous monitoring of the cyclic activation and deactivation of carotid chemoreceptors associated with the ventilatory oscillation. This allows one to know the temporal relationship between chemoreceptor stimulation and the different phases of each CSR cycle, namely apnea/hypopnea and hyperpnea, which is crucial to properly interpreting observed phenomena. To reach this goal, the sensor for the recording of the SpO2 signal is placed at the ear, close to the carotid chemoreceptors. The relevance of this site of monitoring stems from the fact that, although both carotid and medullary chemoreceptors are involved in the chemical control of breathing, the former are thought to exert a dominant role in the development of periodic breathing [4,13].

Even though the ear appears to be the most appropriate site to monitor SpO2, during sleep studies oxygen saturation is almost invariably monitored using a sensor placed at the finger, as this site provides more stable readings, is comfortable for the patient, and allows easy application. The pattern of the SpO2 signal measured at the finger in healthy subjects is a delayed and distorted version of the signal measured at the ear [14,15], the delay and the distortion being essentially due to the longer path that the oxygenated blood has to travel to reach the finger, to phase shifts associated with mixing effects in the arterial vasculature, and to a greater peripheral vasomotion which might interfere with the measurement [16,17]. In heart failure patients, the delay is expected to be increased owing to reduced cardiac output; moreover, the recurrent hypoxia- and hypercapnia-induced changes in sympathetic outflow to the skeletal muscles during CSR are expected to affect peripheral vasomotion, potentially exacerbating the differences between the two signals.

Even though the impact of probe site on SpO2 measurements has been previously investigated to some extent, we are not aware of published studies carried out in heart failure patients. Furthermore, most studies have been conducted in the experimental condition of acute hypoxemia induced by hypoxic breathing challenge [18] or breath hold [15], and the time delay between SpO2 signals at the ear and finger was evaluated either by measuring the distance between the time to detect hypoxemia (defined as SpO2 < 90%) or by measuring the distance between their respective nadirs.

Accordingly, the first aim of this study was to assess, using appropriate signal processing techniques, the timing and morphological differences between SpO2 signals measured at the ear and at the finger during the clinically important condition of CSR.

Besides physiological effects, a further potential source of timing and morphological differences is constituted by the proprietary signal processing algorithms used by manufacturers of polysomnographic recorders to increase the immunity of the SpO2 signal to movement artifacts. Hence, the second aim of our study was to compare the signal obtained using the commercial polysomnograph Embla Titanium with the signal from a bedside pulse oximeter with a finger probe.

2. Methods

2.1. Study population

Subjects for the study were recruited among the patients with chronic heart failure admitted to our Heart Failure Unit for assessment and treatment and who were part of an ongoing prospective observational study on the prevalence and clinical correlates of sleep apnea in heart failure. Patients aged >18 years, in stable clinical condition, in the absence of clinical signs of central or peripheral congestion and receiving optimal medical treatment, with left ventricular ejection fraction (LVEF) < 45% and New York Heart Association (NYHA) class II–III, were considered eligible for the study. Exclusion criteria were chronic obstructive pulmonary disease and peripheral or central nervous system disorders.

Screening was carried out performing a 10-min supine respiratory recording in a quiet, dimmed-light laboratory at a comfortable temperature during morning hours, using inductance plethysmography (Respitrace Plus; Non-Invasive Monitoring Systems, Miami Beach, FL, USA).

Patients who developed CSR were invited to participate in the study and were asked to come back to the same laboratory within two days to undergo a 40-min cardio-respiratory recording between 14:00 and 15:00.

The experimental setup comprised recording of ECG, thoraco-abdominal movements (inductance plethismography), nasal flow (nasal pressure transducer), oronasal flow (thermistor) and three SpO2 signals: one by a fast-response (processing delay: 1.5 s) pulse oximeter (Biox 3740, Ohmeda, Louisville, CO, USA) with an ear probe (SpO2_Ear), another by an identical pulse oximeter equipped with a finger probe (SpO2_Finger) and a third signal by the portable polysomnograph Embla Titanium (Embla Systems, Thornton, CO, USA) equipped with a Nonin Model 7000A finger probe (SpO2_PSG). The ear probe was placed on the ear lobe region of the right ear, and the two finger probes were placed in random order to the index and middle finger of the right hand. Patients were invited to relax and, possibly, to fall asleep. The plethysmographic reading from each unit was used to ensure that all SpO2 signals were stable and of good quality before starting the recording session. Only patients who developed stable CSR (at least 4 cycles) during the recording were considered for subsequent analysis.

The study design and protocol were approved by the local institutional review board and by the Istituti Clinici Scientifici Maugeri Ethics Committee (approval date 16/05/2017, protocol number 2115 CE). All patients signed an informed written consent form prior to participation to the study and provided written consent for the scientific treatment of their data in an anonymous form.

2.2. Data analysis

All signals (sampling frequency: 256 Hz) were resampled at 4 Hz. The O_2 saturation signal provided by the Embla-Titanium device, having a staircase waveform with 1% amplitude resolution, was smoothed using a cubic smoothing spline. The sum of thorax and abdominal signals was computed to obtain an uncalibrated lung volume signal. A continuous tidal volume signal was derived from the lung volume signal by a cubic spline interpolation of the end-expiratory and end-inspiratory points and then computing the difference [19]. The signals were plotted together, and the longest good-quality portion with stable CSR was interactively selected. During CSR, each hyperpneic phase determines, after the time needed by oxygenated blood to reach the site of measurement, a peak in the SpO2 signal, and each hypopneic/apneic phase determines a trough. Hence, the tidal volume signal was used as a common reference for SpO2 delay computations.

Because of the nonlinear relationship between tidal volume and SpO2 signals during apneas, the delay between them was estimated using the nonlinear correlation coefficient h^2 [20,21]. The latter was automatically computed at different time shifts (time resolution = 0.25 s), and the shift for which the maximum was reached was used as an estimate of the delay between the two signals [21].

This crucial part of the processing was carried out using a custom procedure specifically developed for this purpose. Briefly, the portion of tidal volume and of the SpO2 signal under consideration were plotted superimposed. Then, the analyst started to shift the SpO2 signal to the left (ie, anticipate) in 0.25-s steps. Each time, the nonlinear correlation coefficient h^2 was computed and displayed in blue if increasing and in red if decreasing. Hence, the analyst had visual feedback of when the maximum value of the correlation coefficient was reached. An example of this procedure is given in Fig. 1. In the top panel of the figure, the tidal volume and the SpO2 signal at the ear are plotted as they are recorded (tidal volume synchronous with SpO2_Ear). Left shifting the SpO2_Ear signal, the maximum value for h^2 (0.86) was reached anticipating SpO2_Ear by 30.5 s (Fig. 1, middle panel), which gives the delay between the two signals.

For the sake of comparison, we also computed the square of conventional Pearson's correlation coefficient (r^2) and the corresponding estimate of the delays. Using the estimated delays, the three SpO2 signals were aligned and visually checked.

For each cycle of CSR and for all the three aligned signals, the minimum and maximum of SpO2 were computed, as well as the corresponding desaturations (difference between the maximum and minimum value of SpO2). These values were then averaged across all CSR cycles. The mean value of SpO2 across all cycles was also computed. Finally, the proportion of time spent with SpO2 < 90% (T90%), was calculated for the three SpO2 signals.

2.3. Statistical methods

The central tendency and the dispersion of considered variables are reported as mean ± standard deviation. Repeated-measures analysis of variance (ANOVA) was used to compare the measurements from the three SpO2 signals. A significant result from ANOVA was followed up by post hoc analysis (Tukey–Kramer) to compare pairs of signals: SpO2_Finger vs SpO2_Ear and SpO2_PSG vs SpO2_Finger. We also computed the bias and the 95% limits of agreement (LoA), which give an estimate of the interval within which 95% of the differences are expected to lie. To investigate the association between computed delays and demographic, anthropometrical, and clinical variables (age, height, weight, left ventricular ejection fraction, heart rate, and systolic arterial pressure), multiple regression analysis was carried out (backward variables selection at the 0.15 significance level).

3. Results

After screening, 46 eligible patients agreed to participate in the study, but only 21 of them developed stable CSR during the 40-min recording and could therefore be included in the analysis. The number of CSR cycles analyzed was 7.5 ± 2.4 (range: 4–12) and the CSR cycle length was 56.3 ± 13.9 s (range: 34.5–100.0 s).

Demographic, anthropometric, and clinical characteristics of the studied patients are reported in Table 1. An example of the portion

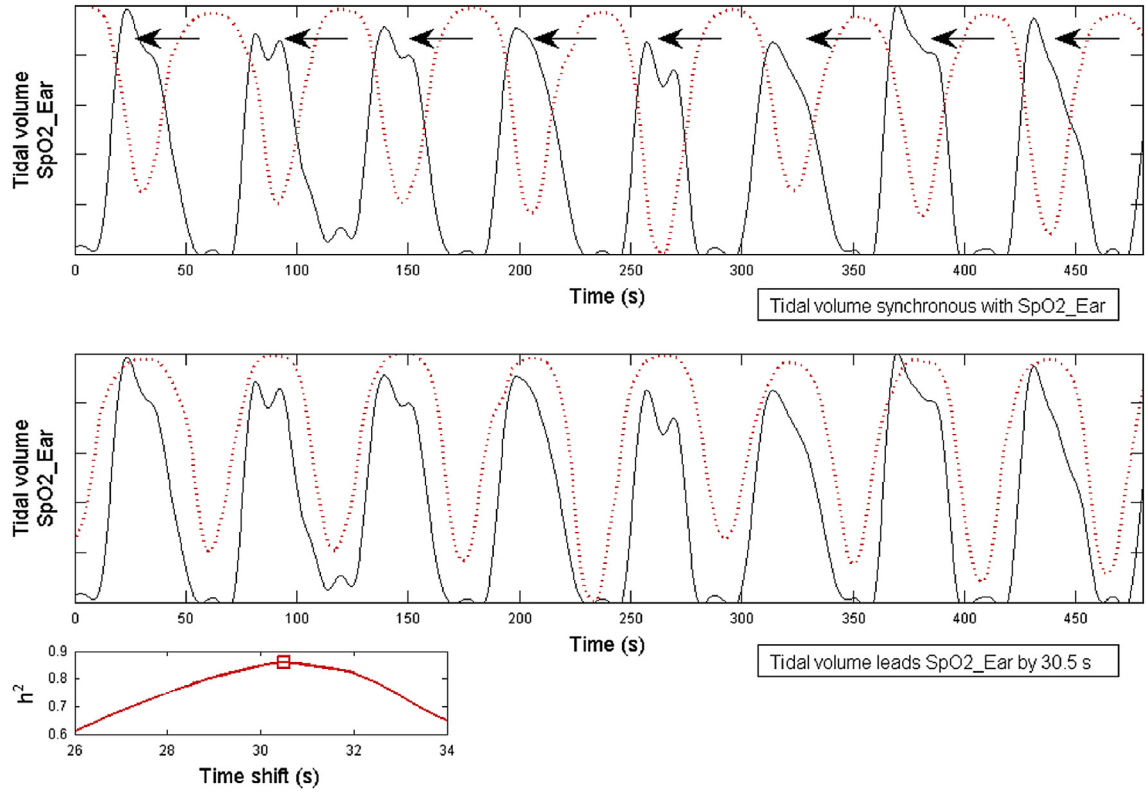

Fig. 1. Example of the procedure used to compute the delay between tidal volume and SpO2 signals (SpO2_Ear in this example). From top to bottom: the tidal volume is plotted superimposed to SpO2_Ear with no time shift applied. The nonlinear correlation coefficient h^2 is computed and displayed. The SpO2_Ear signal is then left shifted (time resolution: 0.25 s) until the maximum of h^2 is reached (second panel from the top). In this example, the maximum value for h^2 was 0.86, at 30.5 s. The bottom small panel shows the values assumed by h^2 as the time shift is changed around the point of maximum.

Table 1
Demographic, anthropometric, and clinical characteristics of studied patients.

Age (yr)	62.9 ± 10.1
Gender (% male)	100
LVEF (%)	29.4 ± 6.6
NYHA class II–III (%)	100
HR (bpm)	62.7 ± 6.3
SPB (mm Hg)	107 ± 15
DPB (mm Hg)	70 ± 7
Weight (kg)	85 ± 11
Height (cm)	173 ± 6
Creatinine (mg/dL)	1.33 ± 0.24
Bilirubin (mg/dL)	1.14 ± 048
Sodium (mEq/L)	140.1 ± 5.1
Mitral regurgitation 2–3 (%)	38
Treatment (%):	
ACE-I/ARBs	100
β-Blockers	100
Diuretics	100
MR antagonists	65
Digitalis glycosides	15
Nitrates	35
Ivabradine	20

ACE, angiotensin-converting enzyme inhibitor; ARBs, angiotensin receptor blockers; DBP, diastolic arterial blood pressure; HR, heart rate; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; SBP, systolic arterial blood pressure.

of recorded signals analyzed in a representative patient is shown in Fig. 2. The results of the procedure to align the signals are displayed in Fig. 3. From the lung volume (Fig. 3, top panel), the continuous tidal volume signal is computed (Fig. 3, middle panel), and the delay computed for each SpO2 signal is used to align these signals with tidal volume (Fig. 3, bottom panel).

Bland–Altman plots of the difference between couples of measurements against their average are reported in Fig. 4.

3.1. Measurements of delay

The delay between continuous tidal volume and SpO2_Ear, SpO2_Finger, and SpO2_PSG and their respective relevant differences are reported in Table 2, top line. As expected, the SpO2 at the finger was significantly delayed with respect to SpO2 measured at the ear, with an average extra delay of 12.7 s. Reflecting a large patient-by-patient variability, the range of this extra delay was quite wide (5.8–25.7 s), with the 95%LoA extending from 2.2 to 23.0 s. The SpO2_PSG signal was almost synchronous with the SpO2_Finger signal.

3.2. Measurements of SpO2

Compared to SpO2_Ear, a slight decrease in mean, maximum, and minimum SpO2 was observed in SpO2_Finger (Table 2), with statistical significance reached only for the minimum. Similarly, the desaturations tended to be slightly deeper at the finger than at the

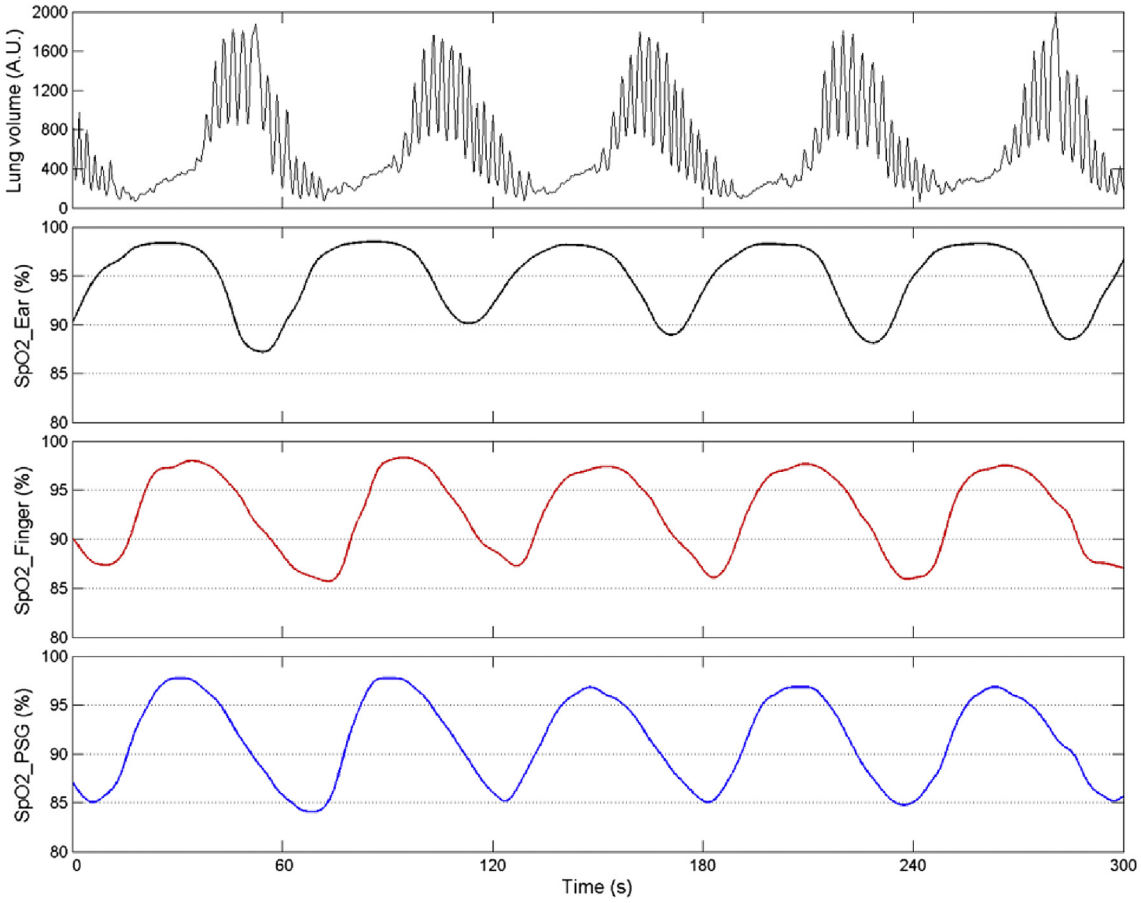

Fig. 2. Example of a 5-min portion of the signals recorded during Cheyne–Stokes respiration in a representative patient. From top to bottom: the uncalibrated lung volume signal obtained summing thorax and abdomen signals, the SpO2 signal measured by a bedside pulse oximeter equipped with an ear and a finger probe (SpO2_Ear and SpO2_Finger, respectively), and the SpO2 signal recorded by the polysomnography device (Embla Titanium) with a finger probe (SpO2_PSG).

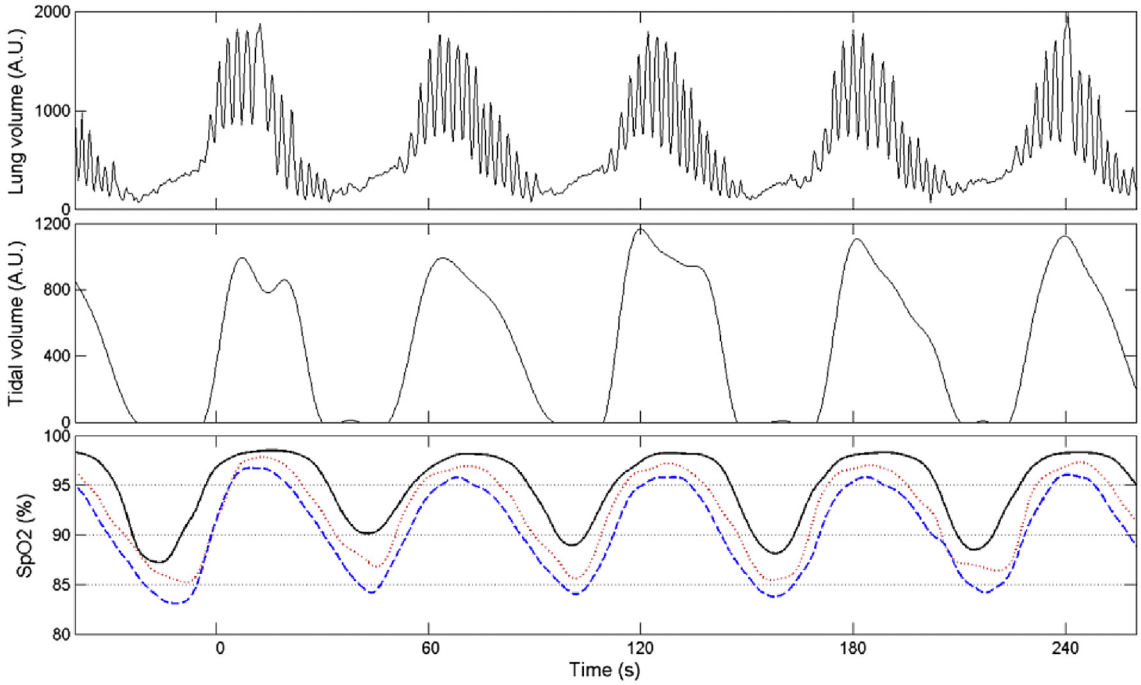

Fig. 3. Example of the results of the procedure used to compute the delays and to align the SpO2 signals. From the lung volume signal (top panel), a continuous tidal volume signal was derived (middle panel) and used as a common reference to align the three SpO2 signals. In the bottom panel, the SpO2 signals after alignment are shown (solid line, black: SpO2 by the bedside device with ear probe, dotted line, red: SpO2 by the bedside device with finger probe, dashed line, blue: SpO2 by the polysomnography device with finger probe). (For interpretation of the references to color/colour in this figure legend, the reader is referred to the Web version of this article.)

ear (0.9% increase in SpO2_Finger vs SpO2_Ear). Although the differences in each measurement were small on average, they were large in specific patients. The large patient-by-patient variability is shown by the wide 95%LoA, ranging from −3.9% to 2.2% for the mean, from −2.9% to 2.1% for the maximum, from −4.9% to 2.5% for the minimum, and from −2.4% to 4.1% for desaturations. SpO2_PSG measurements were significantly lower than those obtained by SpO2_Finger. Moreover, desaturations were significantly deeper (1.2% increase, $p < 0.01$). An example of a patient showing marked differences among the three SpO2 signals is given in Fig. 5.

The percentage of time spent with SpO2 < 90% was almost double in SpO2_Finger than in SpO2_Ear, passing from 5.3% to 11.0%, but this difference did not reach statistical significance. Of note, a further increase of 13.9% in T90% was observed in the SpO2_PSG measurement with respect to SpO2_Finger (24.9% vs 11.0%, $p < 0.001$).

3.3. Nonlinear vs linear estimates of delays

The difference between nonlinear and linear estimates of the delays was negligible for all three SpO2 signals considered (0.4 ± 0.5, 0.1 ± 0.4, and 0.0 ± 0.5 s for SpO2_Ear, SpO2_Finger, and SpO2_PSG, respectively), indicating that a satisfactory estimate of the delays between SpO2 and tidal volume signals can be obtained using the simple product moment correlation coefficient.

3.4. Clinical correlates

Multiple regression analysis revealed that, among all considered explanatory variables, only left ventricular ejection fraction was independently associated with the delay of SpO2_Ear (inverse association, $p = 0.009$). The difference in delay between SpO2_Finger and SpO2_Ear, was significantly associated with age ($p = 0.019$) and systolic arterial blood pressure ($p = 0.018$).

4. Discussion

This is the first study to investigate the impact of probe site on SpO2 measurements in patients with heart failure during the clinically relevant condition of Cheyne–Stokes respiration. We have provided comprehensive results for the comparison between measurement of SpO2 obtained at the ear and at the finger and between measurements obtained at the finger by a commercial polysomnography device and by a bedside pulse oximeter.

The main findings were as follows: (1) a large delay between the SpO2 signal at the finger and that at the ear was observed in most patients; (2) on average, the SpO2_Finger signal was slightly downward shifted and the measurements were only slightly lower than those at the ear, but interindividual variability was high; and (3) the SpO2 signal from the polysomnographic recorder was further downward shifted, with an increase in the depth of desaturations and a large increase in the measurement of the percentage of time spent with SpO2 < 90%, and with a high patient-by-patient variability.

4.1. Delay

The lung-to-ear delay and the lung-to-finger delay (ie, the delay between tidal volume and SpO2_Ear and SpO2_Finger, respectively) were measured using a nonlinear time domain technique that provides measurements that include both the pure transport delay and the time lag due to distortion effects. We found an average lung-to-ear delay equal to 32.7 s, similar to the 30.9 s previously obtained using spectral methods in heart failure patients during nonapneic Cheyne–Stokes [22]. Our measurement of the lung-to-ear delay corresponds to 1.16 times half the observed Cheyne–Stokes cycle length, in excellent agreement with what is expected according to the hypothesis of instability of the chemoreflex loop [22]. We also found that the lung-to-ear delay was

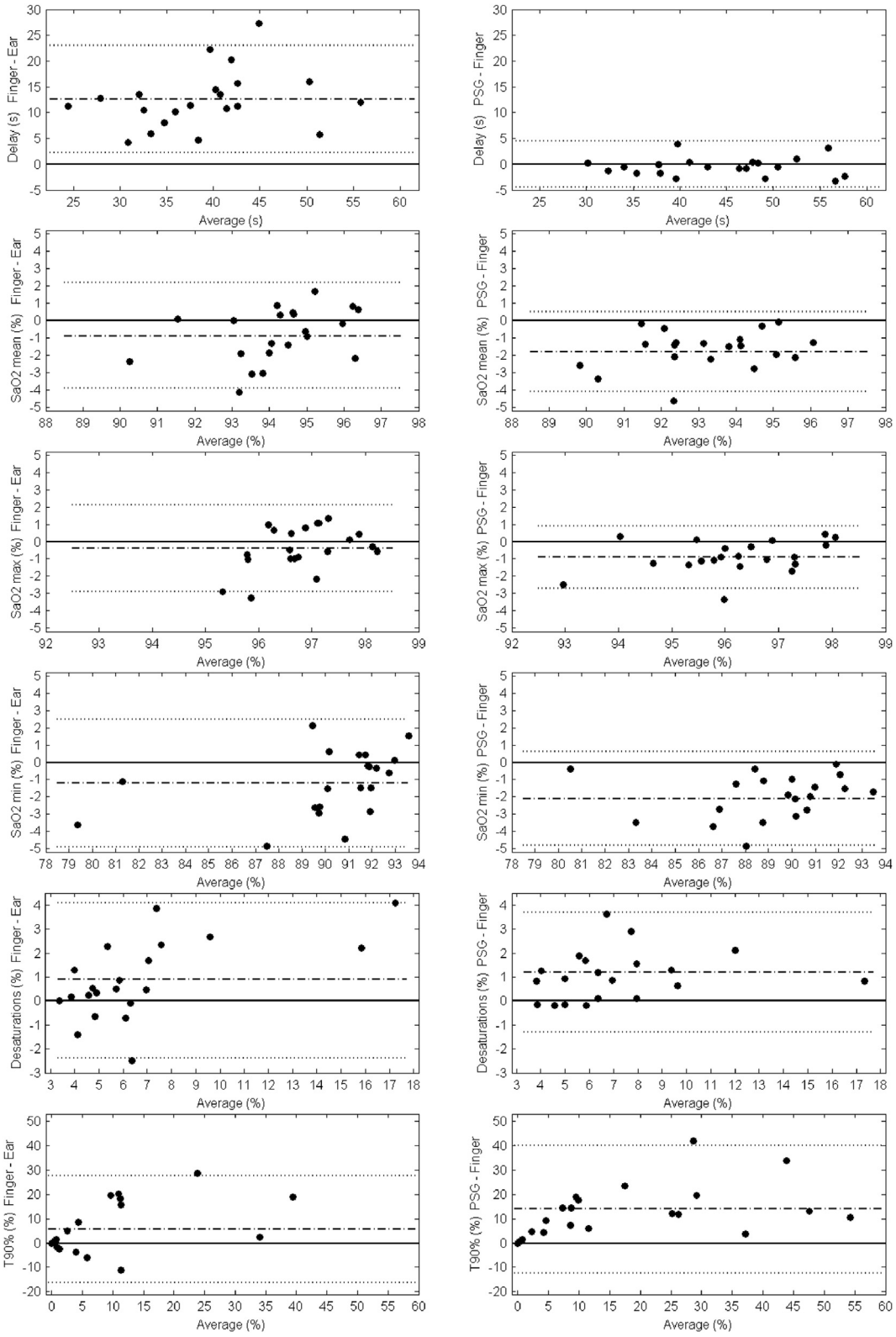

Fig. 4. Bland–Altman plots of the difference between couples of measurements against their average. Left panels show the difference between finger and ear measurements, right panels show the difference between PSG and finger measurements. From top to bottom, differences in measurements of delay, SaO2 mean, SaO2 max, SaO2 min, desaturations, and T90% are shown.

Table 2
Measurements of delay (with respect to tidal volume), mean value, average value of minima, maximma and desaturations and percentage of time spent with SpO2 < 90% for SpO2 measurements at the ear by Biox 3740 pulse oximeter (SpO2_Ear), SpO2 measurements at the finger by Biox 3740 pulse oximeter (SpO2_Finger), and at the finger by the Embla titanium polysomnograph (SpO2_PSG).

Variable	SpO2 Ear	SpO2 Finger	SpO2 PSG	Bias (95%LoA) Finger-Ear	Bias (95%LoA) PSG-Finger	p[a]
Delay (s)	32.7 ± 7.5	45.4 ± 8.6	45.3 ± 9.5	12.7 (2.2,23.0)[c]	−0.1 (−4.4, 4.5)	<0.0001
SaO2 mean (%)	94.7 ± 1.4	93.8 ± 1.9	92.0 ± 2.4	−0.9 (−3.9, 2.2)	−1.8 (−4.1, 0.5)[c]	<0.0001
SaO2 max (%)	97.0 ± 0.8	96.6 ± 1.2	95.7 ± 1.5	−0.4 (−2.9, 2.1)	−0.9 (−2.7, 0.9)[c]	0.0008
SaO2 min (%)	90.7 ± 3.3	89.5 ± 4.0	87.4 ± 4.5	−1.2 (−4.9,2.5)[b]	−2.1 (−4.8, 0.6)[c]	<0.0001
Desat (%)	6.3 ± 3.2	7.2 ± 4.1	8.4 ± 4.8	0.9 (−2.4, 4.1)	1.2 (−1.3, 3.7)[c]	<0.0001
T90% (%)	5.3 ± 9.6	11.0 ± 15.0	24.9 ± 20.6	5.7 (−16.2,27.7)	13.9 (−12.3,40.0)[c]	<0.0001

[a] p Value from repeated-measures analysis of variance.
[b] $p < 0.05$.
[c] $p < 0.001$ for post hoc comparison with SpO2_Ear (Bias Finger-Ear) or with SpO2_Finger (Bias PSG-Finger).

inversely associated with left ventricular ejection fraction. This association, which confirms the results of earlier studies in heart failure patients during central sleep apnea [23], is explained by the fact that impaired hemodynamics determines longer circulatory times.

The lung-to-finger delay was on average 12.7 s longer than the lung-to-ear delay.

We found that the difference between lung-to-finger and lung-to-ear delays was not associated with left ventricular ejection fraction but only with age and systolic pressure, which are major determinants of arterial stiffness [24].

The average delay between the SpO2 signal measured by the Embla-Titanium device and the signal measured at the finger by the laboratory pulse oximeter was close to 0 s. While the averaging time of the Biox 3740 device was known (3 s), no such information is provided for the Embla Titanium device. Given the observed synchronicity, we argue that a fast processing time, similar to the bedside device, was used in the polysomnographic system by the manufacturer. Indeed, the use of fast processing time in devices for polysomnographic studies has been advocated to provide accurate and timely scoring of desaturations [25].

The differences in the delays computed using the nonlinear and the linear methods were negligible, indicating that a satisfactory estimate of the time relationship between SpO2 and tidal volume can be obtained also using the Pearson's correlation coefficient.

4.2. SpO2 measurements

The measurements of SpO2 at the finger by the bedside device tended to be slightly lower than those at the ear, the only difference reaching statistical significance being the minimum value. Desaturations and T90% were increased on average by about 1% and 6%, respectively, not statistically significant. However, the differences in some patients were quite large, indicating that, when considering individual data, the site of measurement matters. Since the fingers have a high degree of vasoconstrictor activity whereas the vessels supplying the ear do not vasoconstrict as much [17], one possible factor contributing to the observed interpatient variability might be a different magnitude of hypoxia- and hypercapnia-induced changes in sympathetic outflow to skeletal muscles.

Looking at measurements by the Embla Titanium device, it can be appreciated that their average difference with the finger bedside

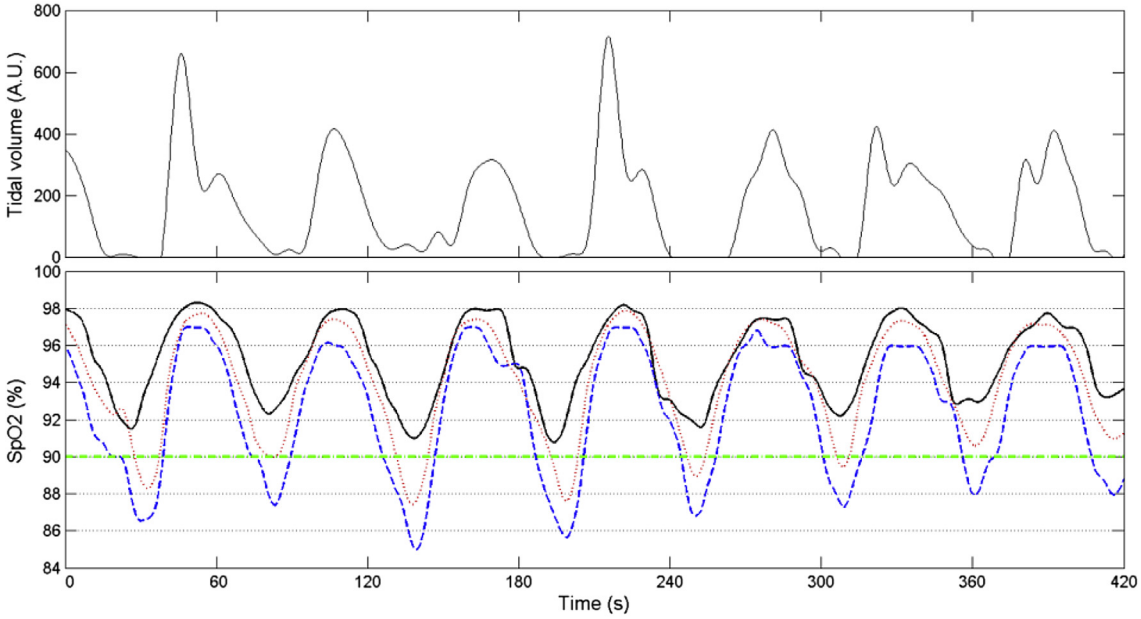

Fig. 5. Example of a portion of recording from a patient showing marked differences among the three SpO2 signals. The tidal volume signal is shown in the top panel, and the three SpO2 signals after alignment are shown in the bottom panel, together with a line marking the 90% value. Passing from SpO2 by the bedside device with ear probe (solid line, black) to SpO2 by the bedside device with finger probe (dotted line, red) and to SpO2 by the polysomnography device with finger probe (dashed line, blue), a progressive decrease in mean SpO2, in the value of maxima and minima and an increase in the depth of desaturations, can be appreciated. Focusing on the portion of the three signals crossing the 90% line (ie, on the percentage of time spent with SpO2 < 90%), very large differences can be seen, with corresponding T90% values passing from 0% (bedside device, ear) to 11.4% (bedside device, finger) and to 30.9% (polysomnography device, finger). (For interpretation of the references to color/colour in this figure legend, the reader is referred to the Web version of this article.)

pulse oximeter measurements was always statistically significant and nearly twice as large as the difference between the latter and ear measurements, with narrower 95% LoA (ie, intersubject variability). With respect to ear measurements, this negative bias led to a remarkable 3.3% underestimation of the minimum values of SpO2, to an amplitude of the desaturations 2.1% greater, and to an impressive increase close to 20% of the T90%. On the whole, the T90% measured by the Embla Titanium device was more than double that measured at the finger by the bedside pulse oximeter and more than four times that measured at the ear. Because these differences might depend on technical characteristics and device-specific signal processing algorithms (calibration curves, artifact elimination, averaging time, etc), we cannot exclude that different polysomnography devices might provide different results with discrepancies of potentially significant clinical impact. Indeed, a recent study comparing the oxygen desaturation index obtained by two different sleep software systems found clinically significant differences, which were attributed to differences in the signal processing [26]. Thus, it would be advisable for technical specifications and recording parameter to be disclosed by device manufacturers, and for standardization of relevant signal processing algorithms to be recommended by polysomnography guidelines.

5. Conclusion

Our results indicate that in heart failure patients with CSR, changes in oxygen saturation at the finger are markedly delayed with respect to those sensed by carotid bodies chemoreceptors. Thus, in the specific context of research studies investigating the pathophysiological mechanisms of sleep disordered breathing in HF patients, pulse oximetry should be recorded at the ear, close to carotid bodies, to obtain information that is close in time to the cyclic activation and deactivation of carotid chemoreceptors associated with the ventilatory oscillation. In the general clinical setting, both ear and finger probes provide valid measurements with different data distributions: one should be aware of the site of measurement to allow for appropriate interpretation of reported data, taking into account the existence of an extra delay when the SpO2 signal is measured at the finger.

Although on average the SpO2_Finger signal was only slightly downward shifted with respect to SpO2_Ear, the limits of agreement for the differences in relevant oxygen saturation measurements were wide. and remarkable differences were observed in some patients.

The SpO2 signal from the commercial polysomnographic recorder Embla-Titanium was, furthermore, significantly downward shifted, with an increase of the cyclic oxygen saturation reductions brought about by apneas/hypopneas and with a marked increase in the measurement of the percentage of time spent with SpO2 < 90%. These discrepancies, which may, at least in part, be due to differences in the processing of raw SpO2 signals, call for a standardization of processing algorithms.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2018.10.043.

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