Nocturnal Oximetry and Transcutaneous Carbon Dioxide in Home-Ventilated Neuromuscular Patients

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BACKGROUND: Pulse oximetry alone has been suggested to determine which patients on home mechanical ventilation (MV) require further investigation of nocturnal gas exchange. In patients with neuromuscular diseases, alveolar hypoventilation (AH) is rarely accompanied with ventilation-perfusion ratio heterogeneity, and, therefore, oximetry may be less sensitive for detecting AH than in patients with lung disease. OBJECTIVE: To determine whether pulse oximetry (S_{pO_2}) and transcutaneous carbon dioxide (P_{tcCO_2}) during the same night were interchangeable or complementary for assessing home MV efficiency in patients with neuromuscular diseases. METHODS: Data were collected retrospectively from the charts of 58 patients with chronic neuromuscular respiratory failure receiving follow-up at a home MV unit. S_{pO_2} and P_{tcCO_2} were recorded during a 1-night hospital stay as part of standard patient care. We compared AH detection rates by P_{tcCO_2}, S_{pO_2}, and both. RESULTS: AH was detected based on P_{tcCO_2} alone in 24 (41%) patients, and based on S_{pO_2} alone with 3 different cutoffs in 3 (5%), 8 (14%), and 13 (22%) patients, respectively. Using both P_{tcCO_2} and S_{pO_2} showed AH in 25 (43%) patients. CONCLUSIONS: Pulse oximetry alone is not sufficient to exclude AH when assessing home MV efficiency in patients with neuromuscular diseases. Both P_{tcCO_2} and S_{pO_2} should be recorded overnight as the first-line investigation in this population. *Key words: blood gas monitoring; transcutaneous; hypoventilation; mechanical ventilation; neuromuscular diseases; oximetry.* [Respir Care 2012;57(9):1425–1430. © 2012 Daedalus Enterprises]

Introduction

Home mechanical ventilation (MV) is used to diminish alveolar hypoventilation (AH), and is generally applied initially at night.[1] Studies have shown that simultaneous recording of transcutaneous carbon dioxide partial pressure (P_{tcCO_2}) and pulse oximetry (S_{pO_2}) is the best method for assessing the efficiency of nocturnal home MV in improving blood gas values[1–5] and detecting prolonged AH and short periods of sleep-related breathing disorders (leaks, asynchrony, and glottis closure). However, to save time and diminish costs, screening with nocturnal pulse oximetry alone, which is easier than recording P_{tcCO_2}, especially at home, has been suggested as a means of identifying those patients who require further nocturnal investigations.[6–8] The criteria used to define AH differ according to the cause of respiratory failure and are more sensitive for restrictive than obstructive diseases.[1] In addition, restrictive defects due to neuromuscular diseases (NMDs) are usually characterized by higher baseline P_{aO_2} values than are obstructive defects.[9] Therefore, AH detection using S_{pO_2} alone may lack sensitivity in patients with NMDs.

The objective of the present study was to compare S_{pO_2} and P_{tcCO_2} values recorded during the same night in patients with NMDs, to determine whether these 2 parameters are interchangeable or complementary for assessing the efficiency of MV.

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The authors have disclosed no conflicts of interest.

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DOI: 10.4187/respcare.01658

Methods

The study protocol was approved by the French Data Protection Authority (Commission Informatique et Libertés), in accordance with French legislation.

Study Patients

Data were collected retrospectively from the charts of patients with NMDs receiving follow-up at the home MV unit of the Raymond Poincaré Teaching Hospital, Garches, France. The patients had chronic restrictive respiratory failure and underwent a routine evaluation of the invasive or noninvasive MV protocol they used at home. This evaluation was done during a 1-night hospital stay, as part of standard patient care, between June 2009 and July 2011. Both S_{pO_2} and P_{tcCO_2} were recorded overnight. We did not include patients on oxygen therapy or having daytime P_{aO_2} values $<$ 60 mm Hg.

Data Collection

The following were collected: anthropometric parameters; type of NMD; symptoms of sleep-disordered breathing or nocturnal hypoventilation (numerous awakenings, nocturnal agitation, diurnal asthenia, sleepiness); lung function test results (vital capacity, maximal inspiratory and expiratory static mouth pressures); time since MV initiation; time on MV per 24-hour cycle; ventilatory parameters; and interface.

P_{tcCO_2} and S_{pO_2} Measurements

Overnight continuous noninvasive P_{tcCO_2} and S_{pO_2} monitoring was achieved using a monitor (Digital Monitoring System, SenTec, Therwil, Switzerland) equipped with a noninvasive combined P_{tcCO_2} (Severinghaus-type electrode), S_{pO_2}, and heart rate sensor (V-Sign, SenTec, Therwil, Switzerland). Recording was started at the patient's usual bedtime and lasted 8 hours, which was the maximal possible recording duration with this device.

As recommended by the manufacturer, the electrode was calibrated in a docking station before each measurement, using a service gas (mixture of 8% CO_2, 12% O_2, and 80% N_2) (SenTec, Therwil, Switzerland), which took approximately 4–11 min. The sensor membrane was changed every 14 days. The skin was cleansed thoroughly with isopropanol 70%, then dried. A small drop of sensor gel (SenTec, Therwil, Switzerland) was applied to the center of the sensor membrane surface, which was then secured to the ear lobe with a low-pressure ear clip and a tape in front of the ear. The electrode temperature was set at 42°C to increase blood flow, thereby improving skin permeability to gases and arterializing the capillaries in order to record P_{tcCO_2} values and subsequently to estimate P_{aCO_2}. P_{tcCO_2} monitoring began after 5–8 min (required for warming of the measurement site, complete local arterialization, and equilibration of CO_2 concentrations between the skin and sensor) and lasted 8 hours. In the event of P_{tcCO_2} signal interruption due to patient movements or passive body mobilization, the electrode was recalibrated and the sensor secured again to the earlobe. According to the manufacturer, measurement resolution was typically 0.8 mm Hg, with an in vitro drift estimated at $<$ 1%/hour and a response time shorter than 80 s.

Overnight S_{pO_2} was recorded in the first patients using both pulse oximetry with a finger sensor (BlueNight, Sleep-Innov Technology, Moirans, France) and the pulse oximetry sensor incorporated in the combined P_{tcCO_2}, S_{pO_2}, and heart rate sensor described above. No significant differences were found between the values supplied by the 2 S_{pO_2} sensors, and, consequently, only the combined sensor was used in the remaining patients.

QUICK LOOK

Current knowledge

Nocturnal ventilation in patients with neuromuscular disease is often indicated for reversal of alveolar hypoventilation. These patients are often monitored with oximetry for detection of gas exchange abnormalities.

What this paper contributes to our knowledge

Pulse oximetry alone cannot exclude alveolar hypoventilation when assessing home mechanical ventilation efficiency in patients with neuromuscular diseases. Both transcutaneously measured partial pressure of carbon dioxide (P_{tcCO_2}) and S_{pO_2} should be recorded overnight as the first-line investigation.

Daytime Blood Gas Measurement

According to routine clinical practice in the unit, daytime blood gas values were obtained in all patients receiving invasive MV, on the morning after the overnight recording. The blood sample was drawn at rest, in the sitting position. In addition, in patients on noninvasive MV, daytime blood gas values were measured in the afternoon on room air.

Data Analysis

Normal blood gas values were defined based on the normal values for our laboratory: pH 7.38–7.42, P_{aO_2} 90–100 mm Hg, bicarbonate 24–28 mmol/L, and base excess

0 ± 2 mmol/L. Daytime hypercapnia was defined as $P_{aCO_2} > 45$ mm Hg.[1]

Specifically designed software (V-Stats 3.00, SenTec, Therwil, Switzerland) was used to transfer and analyze stored nocturnal S_{pO_2} and P_{tcCO_2} data. Graphs and tables were established, and times spent above or below predefined P_{tcCO_2} or S_{pO_2} cutoffs were determined. We visually examined the graphs for each entire recording night to identify artifact-free periods. We then determined the artifact-free recording times; the maximal, minimal, and mean nocturnal P_{tcCO_2} and S_{pO_2} values; and the times spent with $P_{tcCO_2} \geq 49$ mm Hg and with $S_{pO_2} < 90\%$.

Nocturnal AH was defined as abnormal P_{tcCO_2} or S_{pO_2} values. P_{tcCO_2} was considered abnormal when the maximal value was ≥ 49 mm Hg.[10] Three different criteria were used to define abnormal S_{pO_2}, in separate analyses: a value $\leq 88\%$ for at least 5 consecutive minutes (criterion 1),[1] mean nocturnal $S_{pO_2} < 90\%$ or $S_{pO_2} < 90\%$ during $\geq 10\%$ of the total recording time (criterion 2),[6,11] and mean nocturnal $S_{pO_2} < 92\%$ or $S_{pO_2} < 92\%$ during $\geq 10\%$ of the total recording time (criterion 3).[5]

Statistics

The data are described as median and IQR. We used the chi-square test to compare AH detection by S_{pO_2} alone and P_{tcCO_2} alone, to compare clinical characteritics between patients with and without nocturnal elevated P_{tcCO_2} values and to correlate presence of symptoms with nocturnal gas exchange abnormalities. The Mann-Whitney test was used to compare continuous variables between patients with and without nocturnal hypercapnia.

P values $< .05$ were considered significant. All analyses were performed using statistics software (Prism 5, GraphPad Software, San Diego, California).

Results

Population Characteristics

We identified 169 patients with nocturnal P_{tcCO_2} recordings performed at our home MV unit during the study period. Among them, 58 (34%) were home MV users. None of these 58 patients was on oxygen therapy. Table 1 shows the main characteristics of the study population.

Of the 58 study patients, 33 (57%) had severe respiratory muscle disease, with vital capacity values $< 20\%$ of predicted, and maximal inspiratory and expiratory static mouth pressure values $< 30\%$ of predicted in 41 (71%) patients and 43 (74%) patients, respectively. Forty-one (71%) patients used volume controlled ventilators. Body mass index was ≥ 30 kg/m^2 in 3 patients (one each with kyphoscoliosis, Steinert myotony, and facioscapulohumeral muscular dystrophy). No patient had obstructive lung disease. The symptoms questionnaire was retrieved in 29 medical records among the 58 patients included: 18 patients showed symptoms of sleep-disordered breathing or nocturnal hypoventilation, and 11 patients were completely asymptomatic.

Table 1. Characteristics of the Study Population ($n = 58$)

	Median (IQR)	Range	No.
Age, y	28.5 (25.0–37.0)	19–71	
Male/female			49/10
Body weight, kg	45.5 (35.5–62.5)	22–101	
Body mass index, kg/m^2	17.1 (14.6–24.8)	7.9–34.2	
Neuromuscular Disease			
Duchenne muscular dystrophy			41
Steinert myotony			5
Spinal cord injury			2
Other dystrophies			11
Sitting vital capacity, % of predicted	10.5 (6.8–23.8)	0–86	
Supine vital capacity, % of predicted	10.5 (5.0–21.5)	0–84	
P_{Imax}, cm H_2O	12.0 (4.5–27.0)	0–77	
P_{Emax}, cm H_2O	10.0 (5.0–24.0)	1–101	
MV duration, mo	96.0 (47.0–143.5)	14–276	
Time since tracheotomy, mo	96.0 (51.0–183.0)	1–276	
MV use, h/d	24.0 (9.0–24.0)	4–24	
NIV			29
Nasal mask			20
Oronasal mask			6
Nasal mask and mouthpiece			3
Tracheostomy			29
Time of MV use			
Night only			23
Night and day			35
Ventilatory Mode			
Volume controlled			41
Pressure controlled			17
Blood Gas Under MV			
pH	7.42 (7.39–7.46)	7.47–7.57	
P_{O_2}, mm Hg	84 (70–111)	60–149	
P_{CO_2}, mm Hg	36 (31–42)	20–57	
Bicarbonate, mmol/L	24.8 (21.6–27.2)	16.9–33.5	
Base excess, mmol/L	0.8 (-1.9–2.5)	−5.3 to 19.3	
Blood Gas While Breathing Room Air			
pH	7.41 (7.39–7.43)	7.26–7.47	
P_{O_2}, mm Hg	74 (68–86)	60–119	
P_{CO_2}, mm Hg	45 (41–49)	31–70	
Bicarbonate, mmol/L	27.2 (25.4–28.4)	21.4–38.8	
Base excess, mmol/L	2.3 (0.4–3.7)	−3.7 to 7.8	

P_{Imax} = maximal inspiratory static mouth pressure
P_{Emax} = maximal expiratory static mouth pressure
MV = mechanical ventilation
NIV = noninvasive ventilation

Table 2. Cross-Tabulation of P_{tcCO_2} and S_{pO_2} Monitoring ($n = 58$)

	Normal P_{tcCO_2}, no.*	Abnormal P_{tcCO_2}, no.*	Total, no.
S_{pO_2} criterion 1			
Normal	33	22	55
Abnormal	1	2	3
Total	34	24	58
S_{pO_2} criterion 2			
Normal	33	17	50
Abnormal	1	7	8
Total	34	24	58
S_{pO_2} criterion 3			
Normal	30	15	45
Abnormal	4	9	13
Total	34	24	58

* Normal and abnormal definitions in text.
P_{tcCO_2} = transcutaneously measured partial pressure of CO_2

Table 3. Combination of S_{pO_2} and P_{tcCO_2} Results

	Diagnosis of Alveolar Hypoventilation, no. (%)
S_{pO_2} criterion 1	3 (5.2)
S_{pO_2} criterion 2	8 (13.8)
S_{pO_2} criterion 3	13 (22.4)
P_{tcCO_2}	24 (41.4)
P_{tcCO_2} or S_{pO_2} criterion 2	25 (43.1)
P_{tcCO_2} or S_{pO_2} criterion 3	28 (48.3)

P_{tcCO_2} = transcutaneously measured partial pressure of CO_2

Nocturnal S_{pO_2} and P_{tcCO_2} Monitoring

Abnormal peak P_{tcCO_2} values indicated AH in 24 (41%) of the 58 patients, and abnormal S_{pO_2} values in 3 (5%), 8 (14%), and 13 (22%) patients, with criteria 1, 2, and 3, respectively. The chi-square test comparing P_{tcCO_2} and S_{pO_2} showed highly significant differences using both S_{pO_2} criteria ($P < .001$ for criteria 1 and 2, and $P = .03$ for criterion 3).

Table 2 shows the results of combining P_{tcCO_2} and S_{pO_2} values for diagnosing AH. Among the 34 patients with normal P_{tcCO_2} values, 33 (97%, 57% of the overall study population) had normal S_{pO_2} values and one (3%, 2% of the overall study population) had abnormal S_{pO_2} values, according to criteria 1 or 2; this latter number increased to 4 (12%, 7% of the overall study population) patients with normal P_{tcCO_2} and abnormal S_{pO_2} considering criterion 3. Among the 24 patients with abnormal P_{tcCO_2} values, only 2 (8%) had abnormal S_{pO_2} values according to criterion 1, 7 (29%) according to criterion 2, and 15 (38%) according to criterion 3. Thus, the prevalence of high P_{tcCO_2} values among patients without hypoxemia was 40% (22/55) when criterion 1 was used, 34% (17/50) when criterion 2 was used, and 33% (15/45) when criterion 3 was used.

As reported in Table 3, using both P_{tcCO_2} and S_{pO_2} (criterion 2) showed AH in 25 (43%) patients. The 2 tests combined increased the AH diagnosis rate by 29.3%, compared with S_{pO_2} alone (criterion 2), and by 1.7% compared with P_{tcCO_2} alone.

No correlation was found between symptoms of sleep-disordered breathing or nocturnal hypoventilation (18/29) and abnormalities of P_{tcCO_2} or S_{pO_2} with criteria 1, 2, or 3 ($P = .81$, $P = .43$, $P = .36$, and $P = .14$, respectively).

The 5 myotonic dystrophy patients were all ventilated using noninvasive MV. Four of them presented elevated nocturnal P_{tcCO_2}, but none of them presented oxygen desaturation according to criterion 1, whereas 3 of them presented oxygen desaturation according to criterion 2. The myotonic dystrophy patient without abnormal P_{tcCO_2} presented oxygen desaturation according to criterion 3 ($S_{pO_2} < 92\%$).

Nocturnal P_{tcCO_2} Versus Morning Blood Gas Values

Among the 24 patients with nocturnal hypercapnia, 8 (33%) had high P_{aCO_2} values the next morning during objective wakefulness, whereas the remaining 16 patients (67%) had normal P_{aCO_2} values ($P < .001$). Moreover, patients with nocturnal hypercapnia had significantly higher values for the morning bicarbonate concentration (27.2 mmol/L [24.9–28.5 mmol/L] vs 22.4 mmol/L [20.1–25.4 mmol/L], $P < .001$) and base excess (1.8 mmol/L [0.6–3.6 mmol/L] vs −1.6 mmol/L [-3–1.4 mmol/L], $P = .001$), compared to patients with normal nocturnal P_{tcCO_2} values. We did not find any other significant difference between the 2 groups of patients with and without nocturnal elevated P_{tcCO_2} values, respectively, when comparing anthropometric parameters, respiratory functional test values, and MV characteristics.

Discussion

In our study of patients with NMDs, nocturnal pulse oximetry used to assess the efficiency of MV was not sufficient to exclude AH. About one third of the patients had AH manifesting as high P_{tcCO_2} values without substantial oxygen desaturation according to the usual S_{pO_2} criteria.[1,6] On the other hand, oxygen desaturation without nocturnal hypercapnia was rare.

The usual criteria used to determine whether home MV is needed[1] were challenged recently by a study in which nocturnal hypercapnia predicted diurnal hypercapnia in patients with NMDs, and correcting nocturnal hypercapnia

by MV prevented acute hypercapnia crises.[10] The cutoff used to define hypercapnia in this study was 49 mm Hg.[10] We also used this cutoff in our study, as no consensus exists about the best nocturnal P_{CO_2} criterion for assessing MV efficiency. Like Ward et al,[10] we did not use a minimal duration of $P_{tcCO_2} \geq 49$ mm Hg. In fact, 2 patients had a hypercapnia duration of < 5 min. Transferring these 2 patients in the non-hypercapnic group would not have changed the statistics significance and therefore the interpretation of the study. The cutoff used to define daytime hypercapnia is 45 mm Hg,[1] which takes into account the normal P_{CO_2} increase during sleep in adults.[12]

For oxygen saturation we used the recommended cutoff for determining when MV is indicated,[1] considering that S_{pO_2} should not be $\leq 88\%$ for ≥ 5 consecutive minutes when MV is efficient (S_{pO_2} criterion 1). According to a study of MV in patients with kyphoscoliosis, a reasonable goal is a mean nocturnal $S_{pO_2} \geq 90\%$, with $< 10\%$ of the total recording time at $S_{pO_2} < 90\%$ after the correction of leaks.[6,11] We used this possibly more sensitive cutoff for a separate analysis in our study (S_{pO_2} criterion 2). Finally, we also studied a third criterion for S_{pO_2} analysis (mean nocturnal $S_{pO_2} \geq 92\%$, with $< 10\%$ of the total recording time at $S_{pO_2} < 92\%$),[5] considering that the value of 90% may be too severe. With this latter criterion we increased detection of S_{pO_2} abnormalities, including patients with normal P_{tcCO_2} (see Table 2), suggesting another mechanism than AH for explaining oxygen desaturation.

With both S_{pO_2} cutoffs, P_{tcCO_2} was more sensitive than oximetry for detecting AH. Our criteria were similar to those used by Paiva et al, who compared S_{pO_2} and P_{tcCO_2} in a pediatric population with a variety of causes of respiratory failure.[5] This study provided the first evidence that hypercapnia was common during MV in patients without oxygen desaturation. Moreover, we did not find significant correlation between the clinical examination and nocturnal gas exchange abnormalities.

The sensitivity of S_{pO_2} for detecting AH is probably higher in patients with COPD. First, the diurnal hypercapnia cutoff used to determine when MV is required is higher in patients with COPD than in those with restrictive lung diseases.[1] Second, when COPD is so severe as to require MV, there is usually substantial heterogeneity in the ventilation-perfusion ratio.[9,13,14] Therefore, independently from AH severity, hypoxemia is common, and P_{aO_2} values may fall to levels at the steep portion of the oxyhemoglobin dissociation curve, where small P_{aO_2} decreases induced by worsening AH are associated with large drops in S_{pO_2}.[13,15–17] For instance, a P_{aCO_2} increase of 5 mm Hg diminishes P_{aO_2} by about 6 mm Hg, which is largely sufficient to markedly decrease S_{pO_2} when P_{aO_2} is initially low. Thus, in patients with COPD, pulse oximetry alone may be accurate for detecting nocturnal respiratory anomalies, including AH.[6]

An advantage of S_{pO_2} monitoring is the ability to detect brief decreases in MV efficiency, possibly explained by residual obstructive events, decreases in the central ventilatory command, or unintentional leaks.[6] Theoretically, these brief anomalies are not associated with P_{aCO_2} increases. In our population, however, nocturnal oxygen desaturation without hypercapnia was rare. Nevertheless, in practice, and given that currently available P_{tcCO_2} devices can accurately measure instantaneous S_{pO_2}, the issue is not choosing between S_{pO_2} and P_{tcCO_2}, but adding P_{tcCO_2} to S_{pO_2}.

The limitations of transcutaneous CO_2 monitors have been largely overcome in recent years. First, although electrode calibration before each recording remains necessary, membrane changes can be performed less frequently. Second, the lag time has decreased from 5 min on average[4] to approximately 2 min,[18] and the issue of sensor drift over the night has been solved by introducing a compensation that allows continuous recording for 8 hours without substantial signal drift[19] and without requiring 2 P_{aCO_2} determinations at the beginning and end of monitoring.[18] Third, several studies have shown good agreement between arterial and transcutaneous values, even during MV.[18,19] Finally, P_{tcCO_2}-S_{pO_2} measurement devices are now sufficiently easy to use that they are suitable for home monitoring.[5]

Conclusions

In conclusion, given the design and performance improvements in transcutaneous CO_2 monitors, together with their ability to simultaneously monitor S_{pO_2} recording, and our results showing that P_{tcCO_2} is considerably more sensitive than S_{pO_2} for detecting MV inefficiency in patients with NMDs, we recommend the first-line use of nocturnal P_{tcCO_2} and S_{pO_2} monitoring rather than pulse oximetry alone for evaluating MV efficiency. If CO_2 monitoring is not available, a criterion usable in clinical practice for determining oximetry abnormalities should be adjusted individually with the knowledge of diurnal P_{aO_2} in order to appreciate the facility of S_{pO_2} decreasing during hypoventilation. In addition, morning blood gas determination should be preferred to evening blood gas determination for appreciating nocturnal MV effectiveness, as morning bicarbonate and base excess levels may accurately reflect nocturnal respiratory acidosis.

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