Use of Pulse Oximetry to Recognize Severity of Airflow Obstruction in Obstructive Airway Disease*

Correlation With Pulsus Paradoxus

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Study objectives: The purpose of this cross-sectional study was to confirm the observation that pulse oximetry tracing correlates with pulsus paradoxus, and is therefore a measure of the severity of air trapping in obstructive airway disease.

Design: Cross-sectional survey.

Setting: The ICU in a tertiary care academic hospital.

Patients: Twenty-six patients consecutively admitted to the ICU with obstructive airway disease, either asthma or COPD.

Measurements and results: Forty-six percent of the study patients required mechanical ventilation, and 69% had an elevated pulsus paradoxus. We defined the altered pulse oximetry baseline tracing as the respiratory waveform variation (RWV). The RWV was measured in numerical form as the change in millimeters from the baseline. Pulsus paradoxus was significantly correlated with the RWV of the pulse oximetry tracing ($p < 0.0001$). An analysis of the respiratory variations in the pulse oximetry waveforms in obstructive lung disease patients reflects the presence and degree of auto-positive end-expiratory pressure (auto-PEEP; $p < 0.0001$).

Conclusions: We describe the characteristic alterations in the pulse oximetry tracings that occur in the presence of pulsus paradoxus and auto-PEEP. Since pulse oximetry is available universally in ICUs and emergency departments, it may be a useful noninvasive means of continually assessing pulsus paradoxus and air trapping severity in obstructive airway disease patients.

Key words: asthma; critical care; lung disease, obstructive; oximetry; positive-pressure respiration, intrinsic; pulsus paradoxus

Abbreviations: AC = alternating current; auto-PEEP = auto-positive end-expiratory pressure; DC = direct current; RWV = respiratory waveform variation; VMICU = Vanderbilt Medical Intensive Care Unit; WOB = work of breathing

There are few objective measures available in the ICU for determining the severity of air trapping. Peak expiratory flows and forced expiratory volumes often cannot be utilized if the patient is sedated, unable to cooperate, mechanically ventilated, or experiencing increased work of breathing (WOB). The only clinical signs that have ever been shown to correlate well with severe airways obstruction are pulsus paradoxus and sternocleidomastoid muscle retraction.1,2 Pulsus paradoxus has been shown to correlate with obstructive airway disease severity as measured by the FEV$_1$ in critically ill patients.1 Abnormal pulsus paradoxus has been described in 80% of the cases when the FEV$_1$ is reduced to approximately 0.5 to 0.7 L.1,3 The purpose of this cross-sectional study was to confirm the observation that pulse oximetry tracing waveform variations correlate with pulsus paradoxus and increased WOB, and are therefore a measure of air trapping severity in obstructive airway disease. Conacher and McMahon4 describe a similar phenomenon in a case report of three patients, each patient with a different reason for air trapping. Perel et al5 and Pizov et al6 report arterial waveform variations related to dynamic hyperinflation that are similar to the pulse oximetry waveform variations we investigate in this study.

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The utilization of pulse oximetry in ICUs and operating rooms has become a standard means of obtaining valuable blood oxygenation data in a non-invasive and continuous manner. Oximeters use two wavelengths of light to solve for hemoglobin saturation. The waveforms are created by the absorption produced by pulsatile arterial blood volume, which represents the alternating current (AC) signal. The absorption produced by nonpulsatile arterial blood, venous and capillary blood, and tissue absorption is depicted by the direct current (DC) signal (Fig 1). Most pulse oximeters, including the one that was used in our study, are engineered with a fixed filter to diminish the respiratory variation in the waveform to the degree that it is found in normal subjects. Although the amplitude of the pulsatile arterial waveform can be altered and dampened, this does not affect the baseline of the pulse oximeter trace.

We hypothesize that pulse oximetry baseline tracings vary as pleural pressures change, affecting stroke volume. High negative intrapleural pressure during inspiration decreases the stroke volume. This is reflected, we hypothesize, in the altered baseline tracing. To investigate the relationship between airflow obstruction severity, as measured by pulsus paradoxus, and the variations seen in the pulse oximeter waveforms, we studied 26 patients admitted to the Vanderbilt Medical Intensive Care Unit (VMICU) with asthma or COPD. Within the first 24 h following admission, and at follow-up within the next 24 h, we measured each patient’s pulsus paradoxus, their auto-positive end-expiratory pressure (auto-PEEP) if they were mechanically ventilated, and their pulse oximetry, respiratory, and ECG tracings. We noted that the pulse oximeter tracing baseline, which is generally linear with positive deflections with each peripherally detected heart beat, varies with respiration. Once pulsus paradoxus and/or auto-PEEP has normalized, the linear baseline returns, without variation with respiration (Fig 2).

**Materials and Methods**

**Study Patients/Asthma and COPD**

In a cross-sectional survey, our study population consisted of 26 patients with obstructive airway disease, either asthma or COPD, who were consecutively admitted to the VMICU over two 2-month periods, August to September 1996, and December to February 1997. The patients were breathing spontaneously or were on some form of positive pressure ventilation. The admitting diagnosis of asthma or COPD was used to identify this group. The study was approved by the Vanderbilt University School of Medicine Institutional Review Board.

**Study Patients/Normal Subjects**

Normal, healthy volunteer members of our laboratory group and nursing staff served as the control subjects. Pulmonary breathstacking with hyperinflation was achieved by breathing rapidly through an expiratory resistance exerciser (Threshold breathstacking with hyperinflation; Healthscan/Respironics; Cedar Grove, NJ). The measurements of BP, pulsus paradoxus, and pulse oximetry tracings were recorded.

**Data Collection**

Within 12 h of VMICU admission, and within the next 24 h at follow-up, pulse oximetry tracings in real time with respiratory and ECG tracings were recorded in each patient during quiet breathing. If patients were able to cooperate, they were asked to take several deep breaths during the follow-up visit, and these variables were again documented. The measures of BP, respiratory rate, oxygen saturation, clinical evaluation of volume status, and pulsus paradoxus were also recorded.

We used the following clinical indicators and technology. One of the investigators (TH) was present for all data collection.

**Pulsus Paradoxus**

Each patient’s pulsus paradoxus was measured using a sphygmomanometer. The average of two values was used. Before the initiation of the study, the VMICU staff was provided with written instructions on how to measure pulsus paradoxus.

**Auto-PEEP**

The auto-PEEP in patients on mechanical ventilation was measured by one of the investigators, the patient’s physician, or a respiratory therapist. The auto-PEEP was measured by the “occlusion-M” technique, or by programming on the ventilator (Model PB 7200a; Nellcor Puritan Bennett; Pleasanton, CA) by a respiratory therapist. The average of two values was used. None of the patients in this study were paralyzed, as was decided by each patient’s clinical team. All of the patients on mechanical ventilation were heavily sedated, and none had respiratory rates of > 16.

**Pulse Oximetry and Respiratory Waveform Variation**

Continuous real-time pulse oximetry (Model N-100; Nellcor Puritan Bennett) viewed on monitors (Models M1046A-66 and

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**Figure 1.** A schematic illustration of light absorption through living tissue. The AC signal is a result of the pulsatile component of the arterial blood. The DC signal represents all the nonpulsatile absorption in tissue, nonpulsatile arterial blood, and venous and capillary blood. Reprinted from the Ohmeda Pulse Oximeter Model 3700 service manual, with permission granted by Ohmeda Inc.
M1046A-54; Hewlett Packard; Andover, MA) was used to record initial and follow-up waveforms. The patients were studied within the first 12 h of admission, and again during a follow-up period over the next 24 h. A line representing the DC signal (absorption produced by nonpulsatile arterial, venous, and capillary blood) is the bottom of each pulsatile deflection (Fig 3). A line connecting these points defines the baseline (Fig 3). The change in the baseline tracing caused by the respiratory variation was defined as the respiratory waveform variation (RWV). The RWV is documented in numerical form using the paper scale, and is recorded as the number of millimeters (1 mm = 1 box) of change of the baseline between expiration and inspiration (Fig 3). The respiratory tracing is recorded in real time with the pulse oximetry tracing in order to define the phases of respiration. Each pulse oximetry tracing was evaluated for the following: (1) the oscillating change in the oximetry tracing with the respiratory cycle and, if present, the numeric change in baseline with respiratory variation between expiration and inspiration recorded in millimeters, the RWV; (2) the change in RWV over time with treatment; (3) the relationship to respiratory tracing; and (4) the relationship to ECG tracing. The average value of the RWV over four consecutive breaths was used for each patient in the analysis.

**Clinical Indicators**

Pulsus paradoxus, which has been shown to correlate with the severity of obstructive lung disease compared with the FEV1, was used as an objective measure of the clinical course and clinical improvement.1,2

**Statistical Analysis**

Unless otherwise noted, the data are reported as means (± SD) using Student's two-sided t test for Gaussian variables. Linear regression was used to examine the relationship between

![Figure 2](http://example.com/image2.png)

**Figure 2.** Pulse oximetry tracings from a 60-year-old woman with an exacerbation of chronic obstructive lung disease who was admitted to the ICU in ventilatory failure. Top, Panel A: the patient's pulse oximetry tracing at the time of admission, revealing the respiratory variability in the pulse oximeter plethysmography tracing. Her measured pulsus paradoxus at this time was 16 mm Hg. The patient was managed with noninvasive nasal ventilation. Bottom, Panel B: the patient's pulse oximetry tracing after 12 h of aggressive therapy. Her pulsus paradoxus at this time was 8 mm Hg. Note the absence of respiratory waveform variation (RWV) in the baseline of the oximeter tracing after the clinical improvement in airflow and the resolution of elevated pulsus paradoxus.

![Figure 3](http://example.com/image3.png)

**Figure 3.** The baseline represents the line that connects the lowest portion of each waveform that is due to pulsatile arterial blood, and represents the border between pulsatile arterial blood and absorption that is due to nonpulsatile blood. Upper panel: a real-time respiratory tracing is illustrated. The respiratory waveform variation (RWV) is calculated as the number of millimeters of change of the baseline from expiration to inspiration, which corresponds with each full respiratory cycle. The RWV is calculated as the average over 4 consecutive breaths.
pulsus paradoxus and RWV, pulsus paradoxus and auto-PEEP, and auto-PEEP and RWV. Statistical analysis was done using a data analysis system (NCSS Statistical Software; Kaysville, UT).

**Results**

**Clinical Characteristics**

A total of 26 patients (mean age of 56 ± 18 years) requiring ICU admission for exacerbation of obstructive lung disease, either asthma or emphysema, were enrolled in the study. Fifty percent of the study population were men, 61% were African American, 35% were white, and 4% were Asian. Forty-six percent had COPD, 54% had asthma, 46% required mechanical ventilation, and one patient was managed with noninvasive ventilation. Sixty-nine percent of the patients had an abnormal pulsus paradoxus (> 10 mm Hg), and 50% of these patients were on mechanical ventilation. The mean pulsus paradoxus in patients with an elevated pulsus was 23 ± 12 mm Hg. In patients with a physiologic pulsus (≤ 10 mm Hg), the mean pulsus was 7 ± 2 mm Hg. The auto-PEEP levels were measured in patients undergoing mechanical ventilation (46% of the study population). The mean level of auto-PEEP in these patients was 15 ± 14 cm H2O.

In patients admitted to the VMICU with significant asthma or COPD, we documented a variation in the baseline of the pulse oximetry tracing, defined as the RWV. The degree of RWV correlated with the severity of obstructive lung disease. In patients with an elevated pulsus paradoxus (Fig 4), there was a significantly larger RWV with each breath than was seen in patients with a physiologic pulsus paradoxus (≤ 10 mm Hg) or an auto-PEEP level of ≤ 5 cm of H2O (p < 0.0001). An RWV of ≥ 6 mm strongly correlated with a pulsus paradoxus of > 10 mm Hg. The mean RWV in patients with an elevated pulsus paradoxus was 12.9 ± 6.7 (95% confidence interval: 9.4, 16.0). In patients with a physiologic pulsus paradoxus, the mean RWV was 4.5 ± 1.7 (95% confidence interval: 3.1, 5.9). The intraindividual variability in the measurement of pulsus paradoxus was 1.35 mm Hg. Linear regression reveals a very strong correlation between the measured pulsus paradoxus and the measured RWV (p < 0.0001; R² = 0.879; Fig 4) Clinical improvement, as judged by a decrease in pulsus paradoxus, correlated with a return toward baseline of the pulse oximetry tracing or RWV. In patients studied during the follow-up period who were asked to breathe deeply, an increase of their pulsus was noted, as was a marked increase in the RWV, which, after several normal breaths, returned to baseline. The provocation of a pulsus paradoxus by deep breathing probably indicates a continued abnormal airway function. The positive predictive value of RWV for pulsus paradoxus is 94%, with 90% sensitivity and 100% specificity. The negative predictive value is 100%.

There also appears to be a relationship between the measured pulsus paradoxus and the degree of auto-PEEP, as has been suggested in several case reports by Conacher and McMahon, as well as by arterial waveform variations described by Perel et al.

![Figure 4](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21906/)

**Figure 4.** Left, A: a box plot of RWV in patients with physiologic pulsus paradoxus vs patients with elevated pulsus paradoxus. The mean RWV in patients with an elevated pulsus paradoxus was 12.9 mm, and the RWV in patients with physiologic pulsus paradoxus was 4.5 mm. The top and bottom of each box represent the upper and lower quartiles, respectively. The line in the middle of each box is the median, and the spread lines represent the upper and lower adjacent values. The outlier (the solid circle) was not included in the analysis. Right, B: a partial residual plot of RWV vs pulsus paradoxus revealing a strong correlation between increasing RWV and pulsus paradoxus, respectively: R² = 0.879 (p < 0.0001) vs R² = 0.76 (p < 0.001), excluding outlier.
and Pizov et al. The degree of measured auto-PEEP correlates with the degree of the RWV on the pulse oximetry tracing (Fig 5). The absolute value of the RWV in mm × 1.5 approximates the measurable value of auto-PEEP in cm H₂O in our study patients.

In addition, in patients with greater auto-PEEP or pulsus paradoxus, there was a diminution in the height of each pulse wave during inspiration. In patients with significant auto-PEEP and pulsus paradoxus, ECG changes were also noted, with a decrease in R-wave amplitude with respiratory variation.

A change in the RWV of the pulse oximetry tracing with pulsus paradoxus was confirmed in the normal individuals—breathing through an expiratory resistance exerciser and monitored using a pulse oximeter (504 Series; Criticare Systems; Waukesha, WI) with a waveform printout—as well as in the study patients using the Nellcor oximeters. We could not reproduce the magnitude of variability in the pulse oximetry tracing, or as large a pulsus paradoxus as is seen in patients with significant cardiopulmonary disease. The trends, however, were the same. With the patient breathing very rapidly through a resistance exerciser for 1 min, a pulsus paradoxus developed along with a RWV in the pulse oximetry baseline tracing in an oscillating fashion with the respiratory cycle.

**DISCUSSION**

Noninvasive continuous monitoring of the pulse oximetry respiratory waveform allows for continuous and simple recognition of pulsus paradoxus, which correlates with airways obstruction. This provides clinicians who are caring for critically ill patients with obstructive airway disease objective data that was not easily obtainable previously. The RWV also allows clinicians to recognize auto-PEEP.

The explanation for this phenomenon is, most likely, technically based. The tracing component that is recorded for clinical purposes represents the AC component, of which the most downward reflection we considered to be the tracing baseline. We believe that the baseline variation represents changes in perfusion or venous congestion, both of which are secondary to changes in pleural pressure. The devices are designed to automatically increase amplification as the pulse signal decreases; therefore, the oximetry display should be relatively insensitive to changes in perfusion. Several clinical studies, however, have used the pulse oximeter to assess the adequacy of peripheral perfusion and cardiac output. Filters have been developed to process what is called an artifact. It is likely that this software has been developed using normal subjects, and therefore it has filter changes that are equivalent to a normal pulsus paradoxus (<10 mmHg). These filters almost certainly vary to some extent between manufacturers. We postulate that the variation in the pulse oximetry tracing baseline occurs in patients with an elevated pulsus paradoxus, because the filters are not likely to have been designed to filter a pulsus paradoxus of >10 mm Hg. This observation may be device specific; however, we have documented this observation in four different monitoring

![Figure 5](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21906/)

**Figure 5.** Left, A: a partial residual plot of pulsus paradoxus vs auto-PEEP showing a strong correlation between the measured pulsus paradoxus and auto-PEEP, respectively: $R^2 = 0.96$ ($p < 0.0001$) vs $R^2 = 0.81$ ($p < 0.001$), excluding outlier. Right, B: a partial residual plot of RWV vs auto-PEEP showing a strong correlation between the measured auto-PEEP and RWV, respectively: $R^2 = 0.96$ ($p < 0.0001$) vs $R^2 = 0.84$ ($p < 0.001$), excluding outlier.
systems. It is likely, however, that the filter is calibrated differently for each monitoring system. This would result in different magnitudes and scales of change for different monitoring systems.

The next question to answer is, what does this variation represent? We postulate that the change in the baseline detected in the pulse oximetry tracing reflects a decrease in cardiac output, and/or an increase in absorbance that results from venous congestion from impaired venous return secondary to increased lung volumes and pleural pressure fluctuations. We know that the normal systolic pressure drop during inspiration is caused by a number of factors. First, the high negative intrapleural pressure draws blood into the vena cava and increases the filling of the right atrium, and thus the right ventricle. Evidence suggests that with increased right ventricular volume, the interventricular septum bulges into the left ventricular outflow tract, decreasing the stroke volume. During inspiration, the highly negative intrapleural pressure increases the pulmonary blood volume and, therefore, momentarily decreases left atrial and left ventricular filling, decreasing the stroke volume. Most authorities feel that an abnormal pulsus paradoxus is usually produced by accentuation of these basic mechanisms. Although this study was not designed to determine what causes the change in the pulse oximetry tracing baseline, we speculate that in severe obstructive lung disease the respiratory variation in cardiac output and/or venous return may be reflected in the RWV of the pulse oximetry tracing.

There are limitations of this study that should be noted. First, as we hypothesize that the RWV represents a venous blood phenomenon, this may not be present, or so exaggerated, in hypervolemic patients. To address this question, the volume status was assessed in all of the study patients; however, only one patient was designated as hypervolemic by the clinical care team. Second, we feel that an analysis using the area under the curve of the baseline during the respiratory cycle of the pulse oximetry tracing would be a more accurate measurement of RWV than an estimate based on the peak of the RWV. Additionally, the measurement of only two values for pulsus paradoxus and auto-PEEP may not be optimal given the intrinsic variability of these measurements. The third important limitation is in our measurement of auto-PEEP using either the occlusion-M technique or programmable methods available on some ventilators. Since this was an observational study, the patients could not be paralyzed unless the clinical care team deemed it necessary, thus making it difficult to interpret the measurable auto-PEEP. The measurement of auto-PEEP in the patients who were mechanically ventilated was therefore based on patients who were not paralyzed. In one patient, the auto-PEEP was measured at 25 cm. Clearly, expiratory muscle activity must have played a strong role here, as it does for many critically ill patients with obstructive airway disease. Although auto-PEEP raises the WOB, it is subject to sedation, ventilator settings, CO₂ responsiveness, and expiratory muscle strength.

The correlation of the RWV with pulsus paradoxus and auto-PEEP therefore depends heavily on the particulars of how the patient is ventilated. This, however, may still be useful, as clinicians often fail to recognize the marked increase in the WOB created by instituting mechanical ventilation without adequate sedation or paralysis, particularly in patients with obstructive airway disease. Similarly, it may alert clinicians to the catastrophic occurrence of the patient with severe obstruction who is intubated and overventilated, and who shortly thereafter becomes hypotensive or arrests secondary to dramatic hyper-inflation and impaired cardiac venous return. Last, one of the most difficult engineering and clinical problems facing the use of pulse oximeters is that of the signal artifact. This is problematic for the reporting of the arterial oxygen saturation, but it may also be problematic for the interpretation of the waveform. The major artifact of concern in this study is a low AC/DC signal resulting from low perfusion, or a large AC/DC signal resulting from a motion artifact. To address this problem, all of the recordings were done with a finger probe with the patient still, and the recording was done when the pulsatile deflections matched the heart rate and the oximeter was recording a saturation. The baseline, which represents the downward deflection of the AC signal, should not be altered by the artifact.

There are two main implications of our study. First, with existing technology we may be able to fill a deficit in present critical care: the ability to assess the severity of obstructive airway disease with an easily identifiable and characteristic alteration in the pulse oximetry tracing that correlates with pulsus paradoxus. There are few objective measures in the ICU for determining the severity of air trapping, and routine peak expiratory flows and forced expiratory volumes often cannot be utilized. Although the only clinical signs that have been shown to correlate well with severe airways obstruction are pulsus paradoxus and sternocleidomastoid muscle retraction, they will be absent in the heavily sedated and paralyzed patient with obstructive airway disease. The RWV, however, should detect such changes. Second, the RWV may be a useful means of recognizing and approximating the WOB or possibly the auto-PEEP in patients both on and off mechanical ventilation.
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