Advanced Uses of Pulse Oximetry for Monitoring Mechanically Ventilated Patients

Gerardo Tusman, MD,* Stephan H. Bohm, MD,† and Fernando Suarez-Sipmann, PhD‡§

Pulse oximetry is an undisputable standard of care in clinical monitoring. It combines a spectrometer to detect hypoxemia with a plethysmograph for the diagnosis, monitoring, and follow-up of cardiovascular diseases. These pulse oximetry capabilities are extremely useful for assessing the respiratory and circulatory status and for monitoring of mechanically ventilated patients. On the one hand, the key spectrography-derived function of pulse oximetry is to evaluate a patient's gas exchange that results from a particular ventilatory treatment by continuously and noninvasively measuring arterial hemoglobin saturation (Spo_2). This information helps to maintain patients above the hypoxemic levels, leading to appropriate ventilator settings and inspired oxygen fractions. However, whenever higher than normal oxygen fractions are used, Spo_2 can mask existing oxygenation defects in ventilated patients. This limitation, resulting from the S shape of the oxyhemoglobin saturation curve, can be overcome by reducing the oxygen fraction delivered to the patient in a controlled and stepwise manner. This results in a Spo_2/Fio_2 diagram, which allows a rough characterization of a patient's gas exchange, shunt, and the amount of lung area with a low ventilation/perfusion ratio without the need of blood sampling. On the other hand, the photoplethysmography-derived oximeter function has barely been exploited for the purpose of monitoring hemodynamics in mechanically ventilated patients. The analysis of the photoplethysmography contour provides useful real-time and noninvasive information about the interaction of heart and lungs during positive pressure ventilation. These hemodynamic monitoring capabilities are related to both the assessment of preload dependency—mainly by analyzing the breath-by-breath variation of the photoplethysmographic signals—and the analysis of arterial impedance, which examines the changes in the plethysmographic amplitude, contour, and derived indexes. In this article, we present and describe these extended monitoring capabilities and propose a more holistic monitoring concept that takes advantage of these advanced uses of pulse oximetry in the monitoring of ventilated patients. Today's monitors need to be improved if such novel functionalities were to be offered for clinical use. Future developments and clinical evaluations are needed to establish the true potential of these advanced monitoring uses of pulse oximetry. (Anesth Analg 2016;XXX:00–00)

Pulse oximetry—one of the most used monitoring technologies in medicine—consists of an optical spectrometer[1] and plethysmograph.[2] Most publications on pulse oximetry in the fields of anesthesia, critical care, emergency, and respiratory medicine mainly describe its function as a spectrometer for the measurement of arterial hemoglobin saturation (Spo_2).[3–6] The noninvasive and real-time monitoring of Spo_2 is one of the most important breakthroughs in medicine.[7] Its reliability and accuracy to reflect real arterial hemoglobin saturation (Sao_2) has been solidly validated.[1,8–10] This technology allows early diagnosis and treatment of hypoxemia, substantially reducing the incidence and severity of hypoxemic episodes by a factor of 1.5 to 3 when compared with patients without oximeters.[11–13] Thus, pulse oximetry has become the standard of care for monitoring patients under mechanical ventilation.[14]

Beyond this important function, there are numerous reports highlighting the use of pulse oximeters as a plethysmograph.[2,15–21] Plethysmographic analysis of the peripheral pulse wave provides relevant hemodynamic information that has been used for the diagnosis and follow-up of chronic cardiovascular diseases. However, this optical feature of oximeters has not been developed further to allow for a real-time monitoring of acute hemodynamic events in critically ill patients.

Nowadays, clinicians are facing an overwhelming amount of readily available information, and, therefore, it is not surprising that many interesting aspects of pulse oximetry remain hidden below the detection level of routine clinical management. This is also, in part, due to the fact that most monitoring devices focus on, and thus only display, information related to oxygen saturation. We believe that the 2 basic functionalities of pulse oximetry, spectrometry and plethysmography, are largely underused in critical care practice. The intention of this special article is to introduce the clinical potential of the wide range of optical features of pulse oximetry and to discuss further uses for the advanced monitoring of patients under mechanical ventilation.

PULSE OXIMETERS AS SPECTROMETER

Pulse oximetry is particularly useful during mechanical ventilation, because its main objective is to support and ensure the

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Accepted for publication January 25, 2016.

Funding: None.

The authors declare no conflicts of interest.

Reprints will not be available from the authors.

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DOI: 10.1213/ANE.0000000000001283

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maintenance of an optimal gas exchange.[3–6] The principal use of pulse oximetry in mechanically ventilated patients is the detection of hypoxemia—that is, a decrease in the arterial oxygen content—because hemoglobin is the principal carrier of oxygen in blood. Oximeters do not measure Pao_2, and for this reason, they can diagnose directly neither arterial hypoxia nor hyperoxia, defined as a partial pressure of oxygen in the arterial blood (Pao_2) <60 mm Hg or >120 mm Hg, respectively.[22] What oximeters essentially do is "estimate" arterial hypoxia any time Spo_2 falls <90% based on the oxyhemoglobin dissociation curve, the well-described and predictable relationship between hemoglobin saturation and Pao_2.

Improving the Monitoring of Gas Exchange Abnormalities with Pulse Oximetry

A known limitation of the dissociation curve is revealed any time supplemental oxygen is administered to a patient, as fully saturated hemoglobin cannot detect arterial hyperoxia due to the flattening of the upper part of the curve. This is important because the presence of hyperoxia by no means implies that gas exchange is normal. Here, we provide a simple example: an acute respiratory distress syndrome patient with an Spo_2 of 97% and a Pao_2 of 190 mm Hg ventilated with pure oxygen presents with arterial hyperoxia while suffering from a significant impairment of gas exchange if Pao_2/Fio_2 is taken into account. Therefore, the oxyhemoglobin curve and the Spo_2 values lose sensitivity and specificity for monitoring gas exchange during oxygen therapy. Ignoring this important fact can lead to gross misinterpretations of the actual status of gas exchange.

Luckily there are ways to overcome the masking effect of oxygen therapy on Spo_2. One ingenious approach has been described by Sapsford and Jones[23] who proposed the use of a Spo_2/Fio_2 diagram. This method noninvasively determines an individual's oxyhemoglobin curve using Spo_2 instead of Sao_2 and replacing Pao_2 by Fio_2 (Fig. 1). First, a decremental Fio_2 titration is performed from 100% to 21%, in 10% steps, marking at each Fio_2 level the corresponding Spo_2 reading with a dot in the Spo_2/Fio_2 chart. The line connecting these dots creates the modified patient-specific "oxyhemoglobin curve" (Fig. 1A, red line). Thereafter, this individual curve is compared with the theoretical normal one (Fig. 1A, black line). The smart twist introduced by this concept temporarily suppresses the effect of supplemental oxygen to discover the real arterial oxygenation. (Fig. 1)

These authors described this method as a tool for distinguishing shunting from low ventilation/perfusion ratio (V/Q) areas.[23,24] Any time the curve is shifted downward compared with the normal reference, a shunt condition is present, whereas any right-hand displacement indicates a low V/Q problem (Fig. 1A). However, as expected, most mechanically ventilated patients show both gas exchange defects at the same time: shunt induced by atelectasis and low V/Q zones created by small airway closure.[25–28]

The Fio_2/Spo_2 diagram has been tested and validated in several publications. Sapsford and Jones[23] found a good fit between the diagrams constructed in volunteers and in patients with the results obtained by mathematically modeling gas exchange. Later, the same group successfully tested the Fio_2/Spo_2 diagram in different patient populations and clinical scenarios.[24,29,30] We emphasize that this method provides an early, fast, and readable available bedside estimate of shunt and low V/Q.

It should be noted that this concept can be used for monitoring ventilatory treatments. For example, in a mechanically ventilated patient with Spo_2 of 92% breathing room air, the effect of a particular change in the ventilator settings on arterial oxygenation can be evaluated within a few breaths. If this new ventilatory pattern results in a positive clinical effect on arterial oxygenation, Spo_2 must increase despite the low Fio_2.

Recently, we used the same principle to assess the effect of a lung recruitment maneuver in 20 anesthetized morbidly obese patients.[31] Figure 1B shows the Spo_2/Fio_2 diagram obtained in those patients performed under general anesthesia. Standard lung-protective ventilation resulted in an estimated shunt of about 25% as described by such a

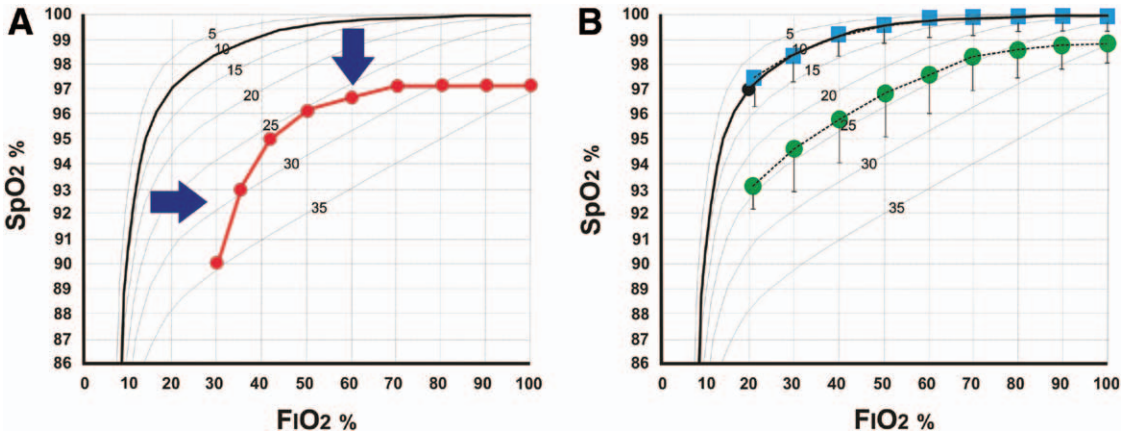

Figure 1. The clinical use of the Spo_2/Fio_2 diagram. A, The Spo_2/Fio_2 diagram detects shunt any time the curve is shifted downward and/or low V/Q areas if the curve is shifted toward the right (arrows). The red line belongs to a COPD patient undergoing anesthesia and mechanical ventilation, whereas the black line represents the normal oxyhemoglobin reference curve. Fine oblique lines indicating estimated shunt values in % are iso-shunt-lines. B, The curve defined by the green circles belongs to 20 morbidly obese patients after anesthesia induction and undergoing a protective ventilation strategy using 8 cm H_2O of PEEP. The estimated shunt was approximately 25%. The curve defined by the blue squares describes the oxygenation condition after a lung recruitment maneuver using exactly the same ventilator setting but at a PEEP of 16 cm H_2O of PEEP. Values are presented as mean ± SD (unpublished data from Tusman et al.[31]). COPD = chronic obstructive pulmonary disease; PEEP = positive end-expiratory pressure; V/Q = ventilation/perfusion ratio.

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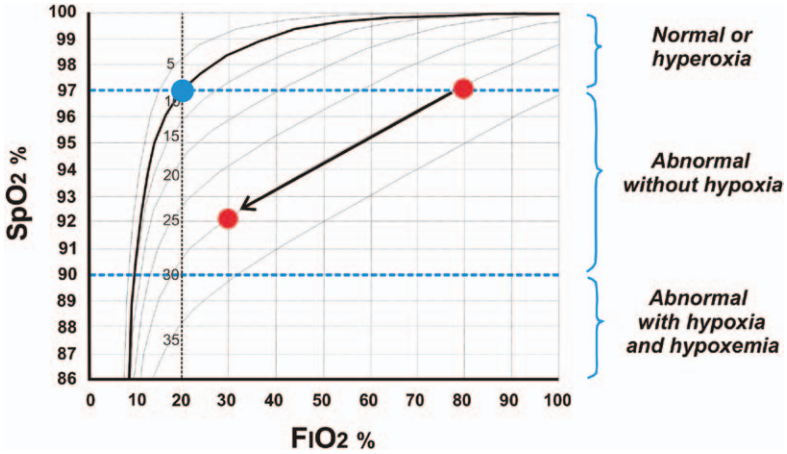

Figure 2. Interpretation of the Spo_2/Fio_2 diagram. The Spo_2/Fio_2 diagram defines 3 conditions. Breathing room air values ≥97% represent healthy lungs or conditions of hyperoxia if high Fio_2 is used. Values between 91% and 96% describe an abnormal oxygenation without arterial hypoxia, whereas values ≤90% define hypoxemias and the oxyhemoglobin curve suggests hypoxia (modified from the study by Sapsford and Jones[23]). The blue dot at 97% and air represents a normal pulmonary condition with a physiologically low shunt. The red dot at 97% and Fio_2 80% appears to be "normal"; however, when Fio_2 is reduced to 30%, Spo_2 decreases to 92% revealing an estimated shunt of 25%. Fine oblique lines indicating estimated shunt values in % are iso-shunt-lines.

diagram (green dots). Later on, after lung recruitment and optimal positive end-expiratory pressure (PEEP), the Spo_2/Fio_2 diagram "normalized" recovering normal shunt values (blue dots).

In summary, the Spo_2/Fio_2 diagram allows a simple, dynamic, and noninvasive characterization of the status of gas exchange that is surely more complete than a "static photograph" given by single Spo_2, Sao_2, or Pao_2 readings obtained at constant Fio_2. Figure 2 summarizes the oxygenation conditions according to the Spo_2 and the applied Fio_2, taking an Spo_2 ≥97% as the normal theoretical value when breathing air.[23,24,31] This method provides a simple estimate of shunt, requiring neither arterial blood samples nor a pulmonary artery catheter. Unfortunately, this tool has not been adopted in operating rooms and intensive care units despite its intriguing simplicity and clinical relevance (Fig. 2).

Characterizing the Status of Gas Exchange During Mechanical Ventilation

The flow chart in Figure 3 proposes a diagnostic sequence to be used before taking the decision to start invasive or noninvasive mechanical ventilation. This guide is based on the principles suggested by Sapsford and Jones[23] and consists of the following 3 simple steps (Fig. 3).

First, baseline Spo_2 during spontaneous breathing of air in the supine position is determined. This step will assess the patient's oxygenation status according to Figure 2. This is an important step because the aging process, smoking, obesity, or pulmonary diseases will all lower baseline Spo_2 and can confound clinicians during and after ventilator treatment. For example, in an elderly patient breathing air, an Spo_2 of 93% could erroneously be considered abnormal after surgery if the anesthesiologist omitted to take a reference value, which, in this case, would have been 93%. In this respect, a preoperative baseline Spo_2 ≤96% breathing air in the supine position has recently been shown to be a strong predictor of an increased risk to develop postoperative pulmonary complications.[32]

Second, the Spo_2 response to an increase in Fio_2 to 1 immediately after intubation also provides relevant information. The question then becomes as follows: Is the patient's hemoglobin able to saturate to its maximum or not? If full saturation is not reached at Fio_2 1, an oxygenation problem should be suspected, as shown in Figures 1 and 2.

Third, the reduction of Fio_2 from pure oxygen to room air either stepwise following the Spo_2/Fio_2 diagram or abruptly in 1 step ("air test") while observing the effect on Spo_2 will identify patients with a shunt problem. This can help decide which specific treatments such as PEEP, recruitment maneuvers, bronchodilators, etc. to apply. Finally, any time, clinicians suspect a deterioration of gas exchange during the course of a ventilatory treatment, and the decremental Fio_2 maneuver can be repeated for diagnostic and clinical decision-making purposes.

ADVANCED USES OF THE PULSE OXIMETER AS A PLETHYSMOGRAPH

The origin and meaning of the photoplethysmography (PPG) waveform has not been well understood for many decades. The ventricular-vascular interaction has provided the theoretical basis for a better understanding and interpretation of the PPG waveform.[33–35] It represents the volume of blood versus time curve measured in a tissue during 1 cardiac cycle.[36–41] This flow wave has a systolic-forward and a diastolic-backward component, similar to pulse pressure and Doppler waveforms[33–35,40–42] (Fig. 4). Changes in aortic wall elasticity and vascular tone alter PPG, arterial pulse pressure, and Doppler waveform morphology in a predictable and similar manner.[33,40–42] These facts support the notion that all waves represent the same vascular phenomenon, confirming that PPG has the potential to characterize the status of the vascular system in a simple and noninvasive manner (Fig. 4).

Role of PPG for Monitoring Mechanical Ventilation

Most of the areas of research around PPG have dealt with the prevention, diagnosis, follow-up, and ambulatory treatment of chronic cardiovascular diseases.[17–21] Although clinicians involved in the care of mechanically ventilated patients use oximeters all the time, the clinical application of PPG for hemodynamic monitoring is only minimally used, if at all.

The link between PPG and mechanical ventilation has its foundation in the physiologic principles governing cardiopulmonary interactions. Hemodynamic impairment during mechanical ventilation is most frequently related to the volemic status and to fast changes in vascular tone.

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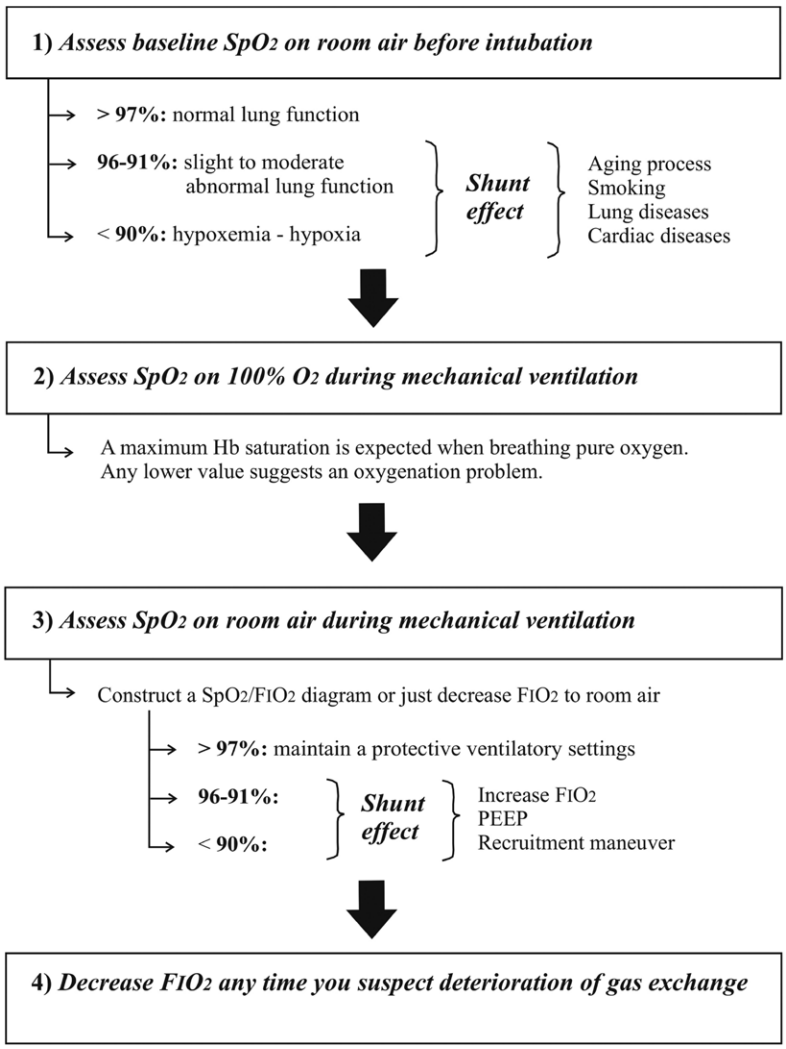

Figure 3. Algorithm to determine arterial oxygenation using Spo_2 during mechanical ventilation. The algorithm addresses oxygenation problems caused by V/Q mismatch—the most common cause of hypoxemia assuming no hypoventilation, Fio_2 <21%, or diffusion problems. V/Q = ventilation/perfusion ratio.

Hypo- and hypervolemia as well as hypotension due to prolonged vasodilation are associated with high morbidity and mortality.[43–45] Therefore, it is of great clinical relevance to be able to determine the volemia state and vascular tone of the patient to choose the right therapeutic intervention. However, this has been a difficult undertaking at the bedside of mechanically ventilated patients, even with standard invasive monitoring methods. Due to its noninvasiveness, PPG may play a potential role as first-line monitoring of these 2 important hemodynamic parameters. The reader is asked to follow the explanations and descriptions given in Tables 1 and 2 while reading through the next 2 sections.

PPG for Monitoring Preload Dependency

The assessment of fluid responsiveness (i.e., the identification of preload-dependent patients in whom IV fluids will increase cardiac index) constitutes the basis of many goal-directed fluid therapy protocols aimed at both optimizing volemia and avoiding fluid overload in mechanically ventilated patients. Table 1 shows the PPG-derived parameters related to the assessment of preload dependency based on the changes in the morphology of the PPG waveform.[46]

As PPG shares the same physiologic principles with arterial pulse pressure, it has been suggested that variations in the PPG waveform during mechanical breaths correspond to the delta up and down, as described for the pulse pressure waveform. Partridge[47] was the first to describe in 1987 shifts in PPG baseline in synchrony with breathing. Murray and Foster[48] described the same phenomenon. Shamir et al.[49] studied the role of PPG waveform variations in anesthetized patients after removing 10% of blood volume and after replacing it by colloids. The authors found a good correlation between changes in PPG and arterial systolic pulse pressure variation ($r = 0.85$; $P = 0.0009$). Natalini et al.[50] showed that the ventilation-induced pulse variations of both arterial and PPG waveforms were similar. Taking these data into account, a PPG variation of 9% was the threshold value for predicting fluid responsiveness, which corresponded to a pulse pressure variation of >13% (sensitivity 100%, specificity 75%, and area under the receiver operating characteristic curve 0.90). Cannesson et al.[51] also obtained a good correlation and agreement between the arterial pulse pressure variation and the PPG signals in mechanically ventilated patients ($r^2 = 0.83$, $P < 0.001$). The same authors demonstrated that a change of >13% in the plethysmographic waveform amplitude can predict fluid responsiveness with a sensitivity of 80% and a specificity of 90% during general anesthesia.[52] This group also validated the plethysmographic variability index for clinical use[53,54] (Table 1).

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Another parameter related to the diagnosis of hypovolemia is the left ventricular ejection time (LVET) or the time between the onset of the systolic upstroke and the dicrotic notch. LVET measured by PPG was highly correlated with the one determined by Doppler from the aortic flow ($r = 0.89$, $P < 0.05$).[55] Using PPG to calculate LVET, Geeraerts et al.[56] demonstrated that LVET decreased proportionally to a simulated hypovolemic condition in healthy volunteers. They found that central LVET measured by a strain-gauge transducer in the carotid artery was similar to the LVET measured by PPG in the periphery. Middleton et al.[57] obtained similar results in an elegant model of hypovolemia in volunteers donating blood. Shortening of PPG-derived LVET and prolongation of pulse transit time were observed in 81% and 91% of 43 subjects, respectively.

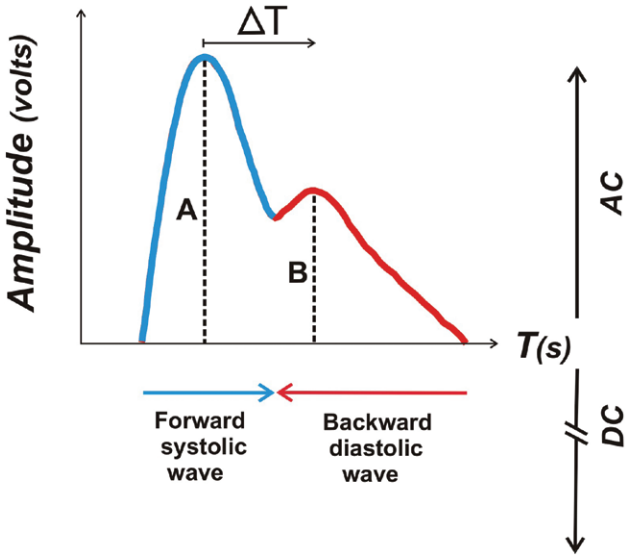

Figure 4. The photoplethysmographic (PPG) waveform. The PPG is formed by a large forward systolic wave (blue) and a small backward diastolic wave (red). It is determined by (1) the pulsatile or alternant component (AC) of the light absorption of the arterial blood, (2) the continuous component (DC) due to the absorption of light within the examined tissues plus the venous blood volume, which varies slowly but which is not represented in the PPG. The time delay between peaks (ΔT) is related to the stiffness of the arterial tree. The ratio of the amplitudes of the B and A waves and the ratio between the AC and the DC components are related to systemic vascular resistance and local perfusion.

The preejection period, that is, the time interval between the R wave on the electrocardiogram (ECG) and the beginning of the upstroke of the radial artery pulse pressure, has been shown to be another useful parameter for predicting fluid responsiveness in mechanically ventilated patients.[58] Feissel et al.[59] demonstrated, in septic ventilated patients, a good correlation between the variation in the preejection period measured by PPG and cardiac index after a fluid challenge ($r^2 = 0.70$, $P < 0.001$). This parameter accurately identified responders defined as a change in cardiac index >15% (area under the curve 0.94) (Table 1).

PPG for Monitoring Left Ventricle Impedance

Pulse wave velocity and vascular tone are factors greatly affecting left ventricular outflow impedance,[33] which changes dynamically during the course of ventilatory treatment. Any time the vascular tree becomes stiffer due to vasoconstriction, the faster pulse wave returns to the LV early in systole, thereby increasing outflow impedance. The opposite situation is observed during vasodilation.[20] Such interaction between the left ventricle and the vasculature

Table 1. Photoplethysmographic-Derived Parameters Related to Preload Assessment

PPG-derived parameters		Formula	Ref.	Clinical meaning	Clinical use
Parameters related to preload dependency	Plethysmographic waveform shape	—	46	Changes in PPG wave features such as height, width, slopes, and AUC are related to the volemic state.	Analysis of changes in the PPG contour has the potential to further increase the sensitivity and specificity of PPG for the diagnosis of preload dependency.
	Plethysmographic waveform amplitude index (POP)	POP = [(POPmax − POPmin) (POPmax + POPmin)/2]	49–52	Delta *up* and *down* as described for the pulse pressure signal during mechanical are also found in the PPG signal. POPmax and POPmin were defined as maximum and minimum pulse oximetry plethysmograph waveform amplitude, respectively.	Respiratory variations in the amplitude of the pulse oximetry waveform (POP) are related to respiratory variations in pulse pressure. Both indices are sensitive to changes in preload.
	PVI	PVI = (PImax − PImin) PImax × 100%	53,54	As above	As above
	LVET	—	55–57	Time between the onset of the upstroke to the catacrotic incision. This parameter comes from the analysis of the aortic blood flow profile, which is strongly related to LV filling and stroke volume (volemia-dependent variable).	PPG-derived LVET was correlated with Doppler measurements. It decreases proportional to the lack of intravascular fluid.
	PEP	$\Delta PEP = [(PEP_E - PEP_I)\ (PEP_E + PEP_I)/2]$	58,59	Time interval between the R wave of the ECG and the upstroke of the PPG. Delta PEP is measured during expiration ($_E$) and inspiration ($_I$)	The respiratory changes in the preejection period are a dynamic index to predict increases in cardiac output after IV fluid infusion.

AUC = area under the curve; ECG = electrocardiogram; LV = left ventricular; LVET = left ventricular ejection time; PEP = preejection period; PI = perfusion index; POP = pulse oximetry amplitude index; PPG = photoplethysmography; PVI = plethysmographic variability index.

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Table 2. Photoplethysmographic-Derived Parameters Related to Systemic Vascular Impedance

PPG-derived parameters		Formula	Ref.	Clinical meaning	Clinical use
Parameters related to systemic vascular impedance	SI	$SI = h/\Delta T_{PPG}$	60,61	Time between peaks of forward and backward waves (ΔT) along the vascular tree (proportional to patient's height = h) is related to the vascular system's elasticity (Fig. 3).	This index, which is related to the chronological age, is highly predictive of outcome in patients with cardiovascular diseases.
	AI	AI = (b − c − d − e)/a	63,64	Index derived from second derivative reflecting the stiffness of the vascular tree.	Useful in some patients when the shape of the PPG and the position of the notch are not clearly discernable. These ratios were related to the aging process, aortic stiffness, and arterial pressure.
	PPG amplitude	—	48,65–70	Vasodilation increases PPG amplitude while vasoconstriction decreases it.	A simple change in the height of the PPG helps diagnose changes in vascular tone. The autoscale function of device must be disabled to be able to observe the real changes in PPG amplitude.
	RI	RI = B/A × 100	20,73–74	Ratio between the heights of the backward and the forward waves, which reflecting systemic vascular resistance (Fig. 3).	This index was used by several authors to detect and quantify the effect of vasodilators on the vascular tone.
	Peripheral PI	PI = AC/DC	71,72	Relationship between the pulsatile (alternant = AC) and the nonpulsatile (continuous = DC) components of red light absorption (Fig. 3). This is an index closely related to the amount of local perfusion.	A cutoff value of 1.4 detected abnormal perfusion due to vasoconstriction. This index is helpful when the dicrotic notch is not easily identified in the PPG.

AC = alternant component; AI = aging index; DC = continuous component; PI = perfusion index; PPG = photoplethysmography; RI = reflection index; SI = stiffness index.

can be monitored by PPG-derived parameters as described in Table 2.

On the one hand, pulse wave velocity is represented by the time between peak-to-peak forward and backward waves that can be clearly determined by the PPG-derived stiffness index[60,61] (Fig. 4). The stiffness index depends on the presence and detection of the dicrotic notch, which is not well defined or even absent in some patients. In those latter cases, the dicrotic notch may yet be identifiable in the first and second derivatives of the PPG wave. Although this index was initially created for the study of the aging process,[60–62] we believe that this concept can be easily applied also in ventilated patients for the beat-by-beat evaluation of LV impedance. The same is true for the indices derived from the second derivative such as the aging index described by Takazawa et al.[63] and Imanaga et al.[64]

On the other hand, the PPG contour provides valuable online qualitative information about the vascular tone, which, in clinical practice, is rarely assessed simply by observing the PPG amplitude.[48] Vasodilation increases the amplitude of the PPG, as observed during hyperthermia or vasodilator infusion, whereas vasoconstriction decreases PPG amplitude as described during tracheal intubation, painful stimuli, or hypothermia.[65–68] Other features of the PPG waveform have also been related to the changes in vascular impedance. Awad et al.[69] found that the PPG width was directly proportional to systemic vascular resistance and its best indicator among other PPG features in anesthetized patients. Lee et al.,[70] using a multivariate approach instead of a single PPG-dependent variable, could accurately discriminate a low systemic vascular resistance with a sensitivity of 85% and specificity of 86%.

There are PPG-derived indices, such as the reflection index and the peripheral perfusion index, that are indirectly related to the vascular impedance. The reflection index represents the degree of wave reflection based on the ratio of the heights of the backward and the forward wave. The perfusion index is calculated from the pulsatile (alternant) and nonpulsatile (continuous) components of light absorption. The clinical value of these indices has been clearly demonstrated in different kinds of patients and clinical scenarios[71–74] (Table 2).

The position of the "dicrotic notch," or the incision between forward and backward waves, has been extensively studied in hypertension, diabetes mellitus, and atherosclerosis,[75–77] and it has been established that, with vasoconstriction, the notch position moves toward the left into the systolic wave. Based on this, Dawber et al.[77] classified the PPG into 4 categories for the diagnosis of arterial hypertension. However, this classification is incomplete because it includes only vasoconstriction but not vasodilation. As can be expected, arterial vasodilation also has a clear effect on the PPG contour, which is characterized by a decrease in the height of the backward wave.[65,78,79]

Because of the predictable nature of its changes, PPG contour analysis could be used to describe and classify the changes in vascular impedance in mechanically ventilated patients. Figure 5 presents a proposal for such a classification that includes all the described features of PPG that have been used to assess vascular impedance. Such classification, based on the PPG shape, will allow clinicians to perform a real-time and noninvasive diagnosis, treatment, and assessment of vasoconstriction and vasodilation in mechanically ventilated patients at the bedside. Even though many publications

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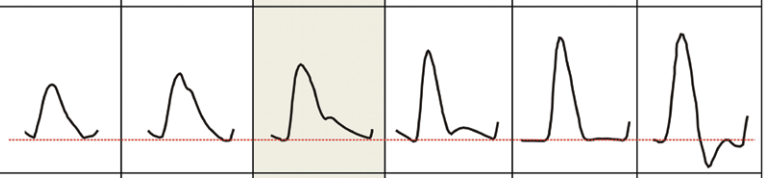
Vascular tone	Vasoconstriction		Normal	Vasodilation		
	severe	*moderate*		*slight*	*moderate*	*severe*
PPG waveform shape						
Amplitude	↓↓↓	↓↓	=	↑	↑↑	↑↑↑
Notch position	↑↑↑	↑↑	=	↓	↓↓	↓↓↓
RI	↑↑↑	↑↑	=	↓	↓↓	↓↓↓
PI	↓↓↓	↓↓	=	↑	↑↑	↑↑↑
SI	↓↓↓	↓↓	=	↑	↑↑	↑↑↑

Figure 5. Proposed classification of vascular tone based on the photoplethysmographic (PPG) contour. Arrows indicate the direction and the number of arrows indicate the magnitude of change. = indicates reference value. PI = perfusion index; RI = reflection index; SI = stiffness index.

strongly support the principles on which this proposed classification is based, its definitive interpretation and validity must be tested and confirmed in future studies (Fig. 5).

In summary, PPG contour analysis provides useful, real-time, and noninvasive information about cardiopulmonary interactions during mechanical ventilation. Its monitoring capabilities are beyond the known concept of preload dependency. Particularly, the assessment of the vascular impedance is clinically relevant because this information remains hidden to most clinicians, thereby limiting diagnostic options and treatment decisions. For example, changes in vascular tone decrease both the sensitivity and specificity of pulse pressure variation to reliably diagnose preload dependency.[80–82] This means that vasoconstriction and vasodilation could falsely suggest a normovolemic state when the patient is in fact hypovolemic and a hypovolemic state when the patient is in fact normovolemic. Vasodilation is a known and common finding in mechanically ventilated patients,[83] and a failure to recognize this condition subjects patients to the known negative consequences of an iatrogenic positive fluid balance.

Limitations and Drawbacks of Pulse Oximetry

In general, pulse oximeters are robust, safe, accurate, reliable, and easy to operate and require no calibration. However, clinicians must be aware about the limitations of this technology and the common drawbacks of most noninvasive measurements. The performance of the advanced pulse oximetry–derived functions described in this article will depend not only on patient-related factors (such as local temperature and blood flow) but also on the specific technical capabilities of pulse oximeters used (such as time response, noises, margin of error, etc.).

Shelley[84] clearly described the desirable characteristics of oximeters to perform a correct pulse oximetry analysis. Newer pulse oximeters should provide at least the following functions: the ability to disable the autogain and autocenter functions, to eliminate certain signal filtering, to change the time scale, to improve the time resolution, and to show red-infrared data and the continuous component of PPG. The access to real and unfiltered raw data could enable new clinical applications and/or improve the standard ones.

Let us illustrate the above with an example. Some commercial oximeters have a time response of Spo_2 between 8 to 12 seconds. An improved time resolution allowing a beat-by-beat calculation of Spo_2 would increase the accuracy and reliability of the Spo_2/Fio_2 diagram because it will provide more data points per unit of time. A better diagram will allow a better analysis. Using standard oximeters, a step change in Fio_2 of 10% will result in no >5 to 8 Spo_2 values per minute in a patient with a heart rate of 80, but in 80 data points if a beat-by-beat calculation of Spo_2 was available.

The sensitivity and specificity of a particular pulse oximeter or one of its features will depend on many technical factors. Manufacturers know these technical factors and the limitations of their devices. Therefore, it is time for them to change the way they analyze and interpret raw data, but, more importantly, they should change the way they present clinically relevant information to the users. Such highly appreciated technical improvements will increase the clinical impact of both current and future pulse oximeters.

Furthermore, as with any noninvasive technology, the performance of oximeter-dependent calculations will be below the accuracy of standard invasive techniques. The main advantage of pulse oximetry resides in its simplicity and availability as a first-line monitoring system not only for mechanically ventilated patients. One of the next steps will be to investigate in more depth the accuracy and reliability of these unusual capabilities of current pulse oximeters.

CONCLUSIONS

Pulse oximetry is an indispensable monitoring modality in respiratory medicine, because it provides real-time, continuous, and noninvasive information on arterial oxygenation. However, the optical spectrometric and plethysmographic capabilities of pulse oximetry are largely underused in the monitoring of patients undergoing mechanical ventilation. The extended clinical applications proposed here are related to the diagnosis of hidden oxygenation deficits using the Spo_2/Fio_2 diagram and to the detection of preload

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dependencies and the monitoring of changes in arterial vascular impedance. Such advanced uses of pulse oximetry will necessarily be less precise than standard invasive techniques but will constitute a valuable first-line monitoring approach for most mechanically ventilated patient in whom more invasive monitoring means are not indicated.

The future challenge will be to identify and better characterize these pulse oximetry functionalities and to further develop them for use in the acute care setting. Many of these potential uses have been described, and this accumulated knowledge should be incorporated into our pulse oximetry devices, providing more holistic and intelligent solutions for the assessment and treatment of oxygenation and hemodynamic problems during positive pressure ventilation.

DISCLOSURES

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