ECRONICON OPEN ACCESS

EC PULMONOLOGY AND RESPIRATORY MEDICINE

Perspective

The Misguided Clinical Hegemony of Pulse Oximetry and the Beside Assessment of Oxygen Delivery and Pulse Oximetry Desaturations

Fidel Davila*

Quality care Institute, USA

***Corresponding Author:** Fidel Davila, Quality care Institute, USA.

Received: May 03, 2017; **Published:** May 06, 2017

The introduction of Pulse Oximetry (PO) into routine clinical practice in the late 1980's was an important advancement in patient care and safety. Subsequent technology improvements and decreased costs resulted in the PO-measured Oxy-Hemoglobin saturation percent, i.e., S_pO_2 %, becoming the '5th vital sign.'

Yet, the assessment and management of S_pO_2's in patients has become clinically misguided because of its universal 'acceptable' level of S_pO_2 > 92% and a 'critically low' level of S_pO_2 < 88%. These levels are nonsensical when one assesses S_pO_2's contribution to the delivery of oxygen (DO_2). Also, generally the therapeutic responses to S_pO_2 desaturations address only the desaturations themselves and not their etiologies. Not addressing etiologies may lead to worsening clinical situations that could have been avoided.

This perspective delineates S_pO_2's contribution to Oxy-Hemoglobin concentrations and DO_2 and how S_pO_2's clinical effect is depended on the Hemoglobin concentration. It also addresses the bedside assessment of DO_2 and low S_pO_2's. Finally, it suggests the current critical low S_pO_2 level of < 88% is misapplied clinical research findings.

First, What PO Doesn't Measure!

As important as to what PO measures is what PO doesn't measure. Pulse oximetry uses a lightwave technology limited to measuring the percent of oxyhemoglobin in relation to oxygenated hemoglobin (OxyHb) plus deoxygenated hemoglobin (deOxyHb).

$$S_pO_2\% = [OxyHb/(OxyHb + deOxyHb)] \times 100 \text{ (equation 1)}$$

The PO lightwaves does not measure other 'abnormal' hemoglobins such as carboxyhemoglobin (COHb), sulfhemoglobin (SulfHb), methemoglobin (MetHb) and the like. Thus, high levels of abnormal hemoglobins will not effect the S_pO_2 readings even though a patient may be experiencing tissue hypoxia due to DO_2 insufficiency from low Oxy-Hemoglobin levels.

If an abnormal hemoglobin is suspected, a 'Co-Oximetry' device should be used to measure arterial blood gas levels. This device will measure the percent of Oxy-Hemoglobin in relation to all hemoglobins and not just deOxy-Hemogloblin. For example, it is not uncommon for Co-Oximetry results on a smoke inhalation patient to show a very high arterial blood oxygen tension (P_aO_2) due to oxygen supplementation and very low arterial Oxy-Hemoglobin (S_aO_2) concentration due to the presence of high levels of COHb -- all the while the S_pO_2 is in the 'normal' range!

The Critical Importance of the Delivery of Oxygen to Cellular Life

The basic units of life are cells and the human body is composed of over 250 different types of cells that number approximately 1 x 10^{16} and are arranged into tissues. Except for red blood cells, each cell requires oxygen and nutrients to produce energy, i.e., adenosine

triphosphate (ATP), through oxidative phosphorylation for sustained life and function. Since, neither oxygen nor ATP are stored in cells and ATP cannot be transferred between cells, the continuous DO_2 to cells is required for continuous the ATP production needed for sustained cellular life and function.

Without oxygen, non-life sustaining cellular anaerobic energy production and its associated lactic acid production ensues and processes that lead to the cell's death begin. Depending on the tissue type, these processes lead to cell death in a few minutes, e.g., gastrointestinal gut lining, to a few hours, e.g., brain and heart, to many hours, e.g., skeletal muscle. If cells are to survive, the DO_2 and energy production through oxidative phosphorylation must be re-established before the processes that lead to cell death become irreversible or the cells are so damaged that they are unable to heal or heal enough to function normally.

Thus, the DO_2 is critical to the survival of cells and to the patient in toto. Its assessment should be part of basic clinical patient care.

The Delivery of Oxygen and Its Major Components

The DO_2 to the body's cells requires: 1) red blood cells with 'oxygen bindable' hemoglobin; 2) ventilation system to oxygenate the hemoglobin in red blood cells transiting through the pulmonary capillaries; 3) the cardio-vascular system to transport the oxygen carrying hemoglobin in red blood cells to the tissue capillary level; and 4) the diffusion of oxygen into cells.

For the body, the DO_2 is calculated as follows:

$$DO_2 = \text{Cardiac Output (CO)} \times \text{Arterial Oxygen Content } (C_aO_2) \times 10^* \quad \text{(equation 2)}$$

*Converts the results from liters/min to dl/min which is the clinical convention.

The C_aO_2 is calculated as follows:

$$C_aO_2 = (1.39^* \times Hb \times S_{a/p}O_2) + (P_aO_2 \times 0.003)^{\#} \quad \text{(equation 3)}$$

*The Constant '1.39' is related to the oxygen carrying capacity of Hb; Hb = hemoglobin in gm/dl; $S_{a/p}O_2$ = ratio of arterial blood (a) or PO (p) oxygen saturated Hb in the absence of abnormal Hb's; P_aO_2 = arterial blood partial pressure of oxygen in mmHg; #The amount of oxygen dissolve in plasma (P_aO_2 x 0.003) is negligible and can be ignored clinically.

Thus, the DO_2 relationship can be shorten as follows:

$$DO_2 \cong CO \times (Hb \times S_{a/p}O_2) \times 13.9 \quad \text{(equation 4)}$$

$$DO_2 \cong CO \times \text{Oxy-Hb} \times 13.9 \quad \text{(equation 5)}$$

Thus, the major components of DO_2 are: 1) Cardiac Output and 2) Oxy-Hemoglobin concentration.

The Relationship of Hb Concentrations and S_pO_2

Red blood cell transfusions in acute non-bleeding medical situations are generally not recommended until hemoglobin concentrations fall below 7 to 8 gm/dl [1]. This suggests that Oxy-Hemoglobin concentrations of 8 gm/dl or less are at or near critical levels that require intervention to maintain DO_2.

Thus, when assessing S_pO_2 results it is key to evaluate the corresponding Oxy-Hemoglobin concentration to see if it is above or below the critical 8 gm/dl level. As can be seen (Table 1), when the Hb = 15 gm/dl, a 'critical' Oxy-Hemoglobin level of less than 8 gm/dl for DO_2 is not reach until a S_pO_2 is less than 55%! In contrast, when the Hb = 8 gm/dl a 'critical' Oxy-Hemoglobin level for DO_2 is present even with a S_pO_2 of 99%!

In other words, if the Hb is 8 gm/dl or less the 'acceptable' levels of S_pO_2 > 92% always produces an unacceptable Oxy-Hemoglobin for DO_2. And as long as Hb is > 9 gm/dl the 'critical' level of S_pO_2 of 88% still produces an acceptable Oxy-Hemoglobin for DO_2 (Table 1).

$S_{a/p}O_2$%	Hb = 15	Hb = 12	Hb = 10	Hb = 9	Hb = 8
99%	14.85	11.88	9.9	8.91	7.92
95%	14.25	11.4	9.5	8.55	7.60
'Normal' 92%	13.80	11.04	9.20	8.28	7.36
90%	13.50	10.80	9.00	8.10	7.20
'Critical' 88%	13.20	10.56	8.80	7.92	7.04
85%	12.75	10.20	8.50	7.65	6.80
80%	12.00	9.60	8.00	7.20	6.40
75%	11.25	9.00	7.50	6.75	6.00
70%	10.50	8.40	7.00	6.30	5.60
60%	9.00	7.20	6.00	5.40	4.80
50%	7.50	6.00	5.00	4.50	4.00

Table 1: *Total Blood Oxy-Hemoglobin Levels < 8 gm/dl. For Various $S_{a/p}O_2$% and Hemoglobin Concentrations.*

S_a/pO_2% = percent oxygen saturation of hemoglobin measured in arterial blood gas ('a')

or by pulse oximetry ('p'); Hb = Hemoglobin Concentration (gm/dl)

Thus, the 'acceptable' and 'critical' S_pO_2 levels are dependent on the Hb concentration and therapeutic decisions based solely on S_pO_2 results are clinically misguided. What is needed is the bedside assessment of DO_2 and if found adequate, the determination of the cause(s) of low S_pO_2 's before they adversely effect the DO_2.

The Bedside Assessment of Oxygen Delivery

Unfortunately, the bedside measurement of Cardiac Output and Oxy-Hemoglobin concentrations are not readily available. Nevertheless, each can be inferred from bedside assessments.

At the bedside, a low Cardiac Output is suggested by an increased level of general distress, increased heart rate, low blood pressure (or orthostatic changes), slow nail capillary refill, a depressed sensorium and a low large toe temperature [2]. For example, a cold, clammy, cyanotic and confused patient suggests a low DO_2.

Assessing the Oxy-Hemoglobin level at the bedside requires evaluating both the S_pO_2 and the Hb concentration. For the most part, recent Hb concentrations are available in essentially all inpatients. Once the Oxy-Hemoglobin is calculated using the Hb concentration and S_pO_2, its adequacy for DO_2 needs to be determined (Table 1).

Insufficiency in DO_2 is frequently due to a combination of Cardiac Output and Oxy-Hemoglobin problems. In these situations, a moderate decrease in one is compounded by a moderated decrease in the other leading to insufficiency in DO_2 's.

If DO_2 insufficiencies are suspected, a metabolic acidosis on arterial blood pH/gases measurements is supportive of the existence of anaerobic metabolism. Anaerobic metabolism can be confirmed by the presence of an increased serum lactic acid level. In these situations, interventions to improve DO_2 are emergently required to prevent cellular and possible patient death.

The Bedside Assessment of Low S_pO_2's

When a S_pO_2 is low and the assessment of DO_2 is determined to be adequate, the etiology of the low S_pO_2 's should be identified and treated to prevent possible worsening. A physical examination of the lungs is required and a chest radiograph to look for lung abnormalities and/or arterial blood gases to assess alveolar to arterial oxygen pressure gradient ($P_{A-a}O_2$) using a simplified formula (Figure 1) [3] may be necessary.

$$P_AO_2 = (F_iO_2 * (P_{atmos} - P_{H2O})) - (P_aCO_2/RQ)$$

P_{atmos} = barometric pressure; clinically assumed 760 mm Hg; P_{H2O} = water vapor pressure; clinically assumed 47 mm Hg; RQ = respiratory quotient; clinically assumed to be 0.8

$$P_AO_2 = (F_iO_2 * (760 - 47)) - (P_aCO_2 / 0.8)$$

$$P_AO_2 = (100/100) * (F_iO_2 * 713) - (1.25 * P_aCO_2)$$

$$P_AO_2 = [(100 * F_iO_2) * (713/100)] - (P_aCO_2 + ¼ P_aCO_2)$$

$$P_AO_2 = (\%O_2 * 7.\cancel{13}^{\#}) - (P_aCO_2 + ¼ P_aCO_2)$$

The '.13' drop takes into account that on average the P_{atmos} is closer to 740 mm Hg in densely populated US area and leads to a clinically acceptable maximum variance of < 2%.

$$P_AO_2 = (\%O_2 * 7) - (P_aCO_2 + ¼P_aCO_2)$$

$$P_{A-a}O_2 = P_AO_2 - P_aO_2$$

An Approximate $P_{A-a}O_2$ is less than (¼ age + 4)

Figure 1: *Simplified Alveolar to arterial oxygen pressure gradient ($P_{A-a}O_2$) Calculation.*

In most acute clinical situations, S_pO_2 drops are caused by lung ventilation/perfusion (V/Q) imbalances, i.e., lungs areas with low ventilation and persistent perfusion, usually from micro- and macro-atelectasis. Being bedridden, acute and chronic lung diseases and cardiogenic pulmonary edema are the most common clinical conditions with V/Q imbalances.

Of note, V/Q-imbalance S_pO_2 drops readily respond to small increases in oxygen supplementation under 50% oxygen. Yet, oxygen supplementation does NOT address the underlying cause(s) of the S_pO_2 drop. In fact, due to resorptive atelectasis, increases in oxygen supplementation may lead to increases in alveolar oxygen level in low V/Q areas of the lung which may lead to worsening atelectasis. This may develop into a vicious cycle of S_pO_2 drops treated with increases in oxygen supplementations.

Treating V/Q imbalances caused by atelectasis requires mobilizing patients esp. with ambulation and sitting in a chair which is not only therapeutic but also preventative. Additionally, frequent Incentive Spirometry and deep breathing exercises are helpful. Oro- or nasotracheal suctioning are effective in inducing coughing which clears secretions and, probably more importantly, result in post-tussive lung

hyperinflation. Chest physiotherapy is indicated only in patients with neurologic defects and unable to cough effectively or in patients with extremely copious secretions and unable to clear them sufficiently.

In contrast to lung V/Q imbalances, lung shunts, i.e., areas of the lung with perfusion but no ventilation, are very poorly responsive to oxygen supplementation and usually require 60% or higher oxygen supplementation to maintain S_pO_2 's > 90%. Diffuse pneumonitis due to trauma or shock or multi-lobar pneumonias are the most common causes of lung shunts.

Of note since oxygen supplementation over 60% leads to minimal S_pO_2 improvements, there are usually no reasons to push the oxygen supplementation to above 60% especially when Oxy-Hemoglobin levels have been deemed adequate for DO_2.

As a rule, positive airway pressures such as continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP) are needed to open collapse or filled gas exchanged airways to improve S_pO_2 's. This positive pressures improve V/Q and result in the decreased need for oxygen supplementation.

A third less common cause of low S_pO_2 's is increased 'venous admixture' from a lower venous Oxy-Hemoglobin levels. Normally, the lungs have small 'fixed shunts' and unoxygenated blood from the venous system mixes, i.e., 'venous admixture', with oxygenated blood to result in the arterial Oxy-Hemoglobin level. In low relative-to-oxygen-requirements DO_2 situations, increased oxygen extraction at the tissues leads to lower than normal returning venous. a.k.a., mixed venous, Oxy-Hemoglobin levels. The subsequent 'venous admixture' then leads to lower arterial Oxy-Hemoglobin levels.

In summary, the treatment of low S_pO_2 's involves treating their causes whether V/Q, shunt and/or worsening venous admixture. All three are commonly present with usually one predominating.

The Source of the Misguided S_pO_2 < 88% 'Critical Level'

The current S_pO_2 's critical level of < 88% most likely comes from two published long term oxygen therapy studies in COPD patients [4,5]. These studies showed that continuous oxygen therapy with S_pO_2's > 88% was beneficial in patients with COPD. Nevertheless, these benefits were not shown for less than 10 - 20 months of continuous oxygen therapy. Using this level as a 'critical level' in all patients with low S_pO_2's for short periods, i.e., minutes, hours, days, weeks and maybe even a few months, is a gross and misguided application of these study findings.

Conclusions

The clinical practices of PO is misguided and unsupported by clinical science. The current S_pO_2's acceptable level of > 92% and critical level of < 88% are nonsensical since it is Oxy-Hemoglobin level and not $S_{a/p}O_2$ that is a major determinant of DO2.

Although low S_pO_2's indicate abnormal clinical situations, they do not automatically mean dire circumstances. Instead low S_pO_2's should be seen as a 'red flag' mandating assessment of the patient's DO_2, i.e., cardiac output and Oxy-Hemoglobin level, and if adequate, the patient's lungs to assess the underlying abnormality (-ies).

In summary, PO improved patient care and safety. Yet, S_pO_2's contribute to DO_2 only in relation to Hb concentration. Only responding to S_pO_2's changes without assessing its effect on DO_2 and assessing and as needing treating it cause(s) is misguided.

Volume 3 Issue 4 May 2017