Correlation between the levels of SpO_2 and PaO_2

Sir,

Sarkar *et al*. in 2017, in their recent article,[1] have very nicely elucidated various mechanisms of hypoxemia, and I would like to congratulate them for this endeavor.

In continuation, I would like to offer the following hypothesis which correlates well between the levels of hemoglobin saturation (SpO_2) and partial pressure of oxygen in the arterial blood (PaO_2).

We keep on looking arterial blood gas (ABG) reports and keenly watch multi-monitors in Intensive Care Units (ICUs) and wards. As standard teaching, we have a certain image and interpretation of high and low SpO_2 and PaO_2 reports.[2]

The sigmoid shape of the oxy–hemoglobin (Hb) dissociation curve reflects the cooperative interaction between Hb and oxygen (O_2) molecules. The oxy–Hb dissociation curve is initially steep and then flattens out (sigmoid shape). The most important aspect of the curve is that as the oximeter reading falls below 90%, the PaO_2 drops very rapidly and O_2 delivery to the tissues is reduced and leads to irreversible brain damage and cardiac arrest.

The understanding of sigmoidal-shaped oxy–Hb dissociation curve comes very handy in these situations [Figure 1]. O_2 saturation varies with the PaO_2 in a nonlinear relationship and is affected by temperature, pH, 2,3 diphosphoglycerate, and $PaCO_2$ (partial pressure of carbon dioxide in the arterial blood).[3] Above 90 mmHg of PaO_2, the curve becomes almost flat, and there is a small rise in SpO_2 in spite of big increments in PaO_2. The flat upper part acts as a buffer in the sense that the PaO_2 can drop to about 60 mmHg and yet the Hb will still remain highly saturated (90%) with O_2. The steep lower part also has big advantage in that if the tissues require more O_2, substantial amounts of O_2 can be removed from Hb without greater drops in PaO_2.[4] For example, Hb would be still 50% saturated although PaO_2 has dropped to 26.6 mmHg (P50).

From last 30 years, while working in critical care wards, I always used to wonder if any formula can be devised which, while waiting for ABG results, can rapidly help a clinician to reach to a PaO_2 level just by looking at SpO_2 values, and I have come up with certain observations/calculations. For the first 10% reduction in SpO_2 from 100% to 90%, decrease PaO_2 by 4 mmHg for every single percent reduction in SpO_2 with a resultant PaO_2 falling from 100 to 60 mmHg [Table 1]. For the next

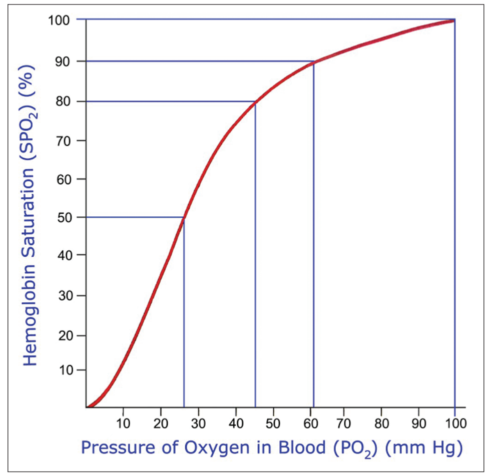

Figure 1: Oxy–hemoglobin dissociation curve

Table 1: Calculation for PaO_2 assessment

SpO_2 (on monitor)	Calculation for PaO_2	Resultant PaO_2 range
100%-90%	Decrease PaO_2 by 4 mmHg for every single percent reduction in SpO_2	100-60 mmHg
90%-80%	Decrease PaO_2 by 1.5 mmHg for every single percent reduction in SpO_2	60-45 mmHg
<80%	Divide SpO_2 by 2 to reach to a PaO_2 level	40 mmHg and downward

10% reduction in SpO_2 from 90% to 80%, decrease PaO_2 by 1.5 mmHg for each percent reduction in SpO_2 which will result in PaO_2 falling from 60 to 45 mmHg. Finally, for SpO_2 levels below 80%, divide it by 2, that is half the value of SpO_2, and we get the requisite PaO_2 level.

This hypothesis can have some pitfalls, for example, cyanide poisoning and certain hemoglobinopathies, but still, a fair and working assessment may be drawn from this calculation. Two proven measurements further support this hypothesis:

i. As per classical teaching, at mixed venous point, the SpO_2 of deoxygenated blood returning to the heart is taken as 75% with a saturation of 40 mmHg.[1,3] With the present calculation, O_2 saturation would come out to be 75/2 = 37.5 mmHg, which, in clinical parlance, is not much further away from 40 mmHg
ii. The hypothesis is further supported by the value of P50, which is 26.6 mmHg, and again, which is almost half of 50%.

This formula, which is not an exact mathematically proven entity, can be of extreme help to ICU residents and consultants. For example, if the monitor is showing a SpO_2 of 70%, we can almost consider a value of PaO_2 to be around 35 mmHg and take appropriate measures for the patient.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Arun Madan

Department of Pulmonary Medicine, NDMC Medical College, Hindu Rao Hospital, New Delhi, India

E-mail: drarunmadan@gmail.com

REFERENCES

1. Sarkar M, Niranjan N, Banyal PK. Mechanisms of hypoxemia. Lung India 2017;34:47-60.
2. Leitch AG. Functions of the lung. In: Seaton A, Seaton D, Leitch AG, editors. Crofton & Douglas's Respiratory Diseases. 5th ed., Vol. 1, Ch. 2. Oxford: Blackwell Science; 2000. p. 26-62.
3. West JB. Gas transport by the blood. In: Respiratory Physiology-The Essentials. 9th ed., Ch. 6. Baltimore: Lippincott Williams & Wilkins; 2012. p. 77-94.
4. Wagner PD, Powell FL, West JB. Ventilation, blood flow and gas exchange. In: Mason RJ, Broaddus VC, Martin TR, King TE Jr., Schraufnagel DE, Murray JF, *et al.*, editors. Textbook of Respiratory Medicine. 5th ed., Vol. 1, Ch. 4. Philadelphia: Saunders Elsevier; 2010. p. 53-88.

Access this article online

Quick Response Code:

Website:
www.lungindia.com

DOI:
10.4103/lungindia.lungindia_106_17

How to cite this article: Madan A. Correlation between the levels of SpO_2 and PaO_2. Lung India 2017;34:307-8.