A Case of Disparity Between Pulse Oximetry Measurements and Blood Gas Analysis Values

Jeffrey M Haynes RRT RPFT

Case Summary

A 79-year-old man was admitted to the intensive care unit because of shortness of breath, dizziness, and confusion. He had a history of T cell prolymphocytic leukemia, which was not responding to therapy with fludarabine. His admission serum leukocyte count was 886,000 cells/μL and his vital signs were: temperature 36.9°C, respiratory rate 28 breaths/min, heart rate 91 beats/min, and blood pressure 160/91 mm Hg. Chest auscultation revealed bibasilar rales. A chest computed tomogram (CT) revealed a diffuse ground-glass appearance of the lung fields and no evidence of pulmonary emboli. While receiving supplemental oxygen via a nonrebreather mask, his arterial oxygen saturation (measured via pulse oximetry [S_{pO_2}]) was 93%. Table 1 shows the concurrent arterial blood gas and hemoximetry data.

Question

What are the possible causes for the discrepancy between S_{pO_2} and the oxygen saturation measured via hemoximetry (oxyhemoglobin) in this case?

Answers

1. Spuriously high S_{pO_2} value
2. Inadvertent venous blood sampling
3. Excessive time delay in sample analysis
4. Consumption of oxygen by leukocytes in the blood sample ("leukocyte larceny")

Discussion

It is unlikely that an inaccurate S_{pO_2} reading explains the disparity between the S_{pO_2} measurement and the oxyhemoglobin value in this case. A common cause of spuriously high S_{pO_2} readings is the presence of above-normal carboxyhemoglobin,[1] but carboxyhemoglobin was only 0.6% (see Table 1). Other factors that can create spurious S_{pO_2} readings,[2,3] such as intravascular dyes, dark skin pigmentation, motion artifact, nail polish, optical shunting, and poor peripheral perfusion, were not present. Also, recent work by Fluck et al[4] refutes the idea that ambient light sources significantly affect pulse oximeter performance.

Inadvertent venous blood sampling is certainly a plausible explanation for the S_{pO_2}-oxyhemoglobin disparity in this case. It is recommended that pulse oximetry be used to help determine whether a hypoxemic blood specimen is in fact arterial, if there is clinical doubt. Inadvertent venous blood sampling is an important pre-analytic error against which all blood gas phlebotomists must be vigilant. Clearly, unrecognized inadvertent venous sampling can subject the patient to substantial morbidity and expenses from unnecessary interventions. Obviously, this does not occur only with pure venous samples; any amount of venous blood in an arterial sample results in a false reading. Therefore the ideal arterial puncture site is devoid of large adjacent veins.

Table 1. Arterial Blood Gas Values

pH	7.39
P_{aCO_2} (mm Hg)	43.6
P_{aO_2} (mm Hg)	49.5
HCO_3^- (mmol/L)	25.9
BE (mmol/L)	1.3
Hb (g/dL)	9.8
O_2Hb (%)	83.6
COHb (%)	0.6
MetHb (%)	0.1
RHb (%)	15.7
C_{aO_2} (vol %)	11.5

HCO_3^- = bicarbonate
BE = base excess
Hb = hemoglobin
O_2Hb = oxyhemoglobin
COHb = carboxyhemoglobin
MetHb = methemoglobin
RHb = reduced hemoglobin
C_{aO_2} = oxygen content in arterial blood

Jeffrey M Haynes RRT RPFT is affiliated with the Department of Respiratory Therapy, St Joseph Hospital, Nashua, New Hampshire.

Correspondence: Jeffrey M Haynes RRT RPFT, Department of Respiratory Therapy, St Joseph Hospital, 172 Kinsley Street, Nashua NH 03060. E-mail: jhaynes@sjh-nh.org.

Ultimately, the S_{pO_2}-oxyhemoglobin disparity in this case was judged not to be due to inadvertent venous sampling. The respiratory therapist who drew the sample was very experienced and had high confidence that the sample was arterial because of the brisk, pulsatile filling of the syringe with blood. In addition, subsequent blood gas samples generated the same S_{pO_2}-oxyhemoglobin disparity.

Another pre-analytic error that can cause S_{pO_2}-oxyhemoglobin disparity is delay of blood sample analysis. Though erythrocytes derive much of their energy from anaerobic sources, other blood cells can consume considerable amounts of oxygen in vitro, which can lower the measured oxygen tension and content.

It is recommended that blood gas samples stored at room temperature be analyzed within 30 min of procurement.[5] Others have recommended that samples should be placed in ice to reduce oxygen consumption if the specimen cannot be analyzed within 10 min of procurement.[6] That type of pre-analytic procedural error did not occur in this case. The blood gas sample was analyzed within 3 min of procurement.

Ultimately, it was concluded that the cause of the S_{pO_2}-oxyhemoglobin disparity in this case was from oxygen consumption by leukocytes in the blood sample—a phenomenon known as "leukocyte larceny"; between the time the sample is drawn and the time the blood is analyzed, aerobic cells consume oxygen in vitro, thereby lowering the oxyhemoglobin saturation measurement. Extreme leukocytosis (886,000 cells/μL in this case) can be associated with rapid in vitro oxygen consumption, even if the sample is immediately cooled and analyzed within 10 min.[7] Fox et al[8] demonstrated 2-min declines in oxygen tension, ranging from 13 to 72 mm Hg, in leukemia patients, with leukocyte counts ranging from 55,000 cells/μL to 490,000 cells/μL. Placing a sample on ice and adding potassium cyanide can be effective in halting the consumption of oxygen by leukocytes.[8,9] Another strategy is to use plasma to measure the oxygen tension.[10]

Though extreme leukocytosis can spuriously lower measured oxygen indices, one must not lose sight of the fact that leukocyte larceny and gas exchange dysfunction often coexist. Patients with leukemia are susceptible to respiratory dysfunction from chemotherapy agents, malignancy, and pneumonia. In addition, patients with lymphocytic leukemia and those who undergo bone marrow transplantation can develop pulmonary veno-occlusive disease.[11]

REFERENCES

1. Hampson NB. Pulse oximetry in severe carbon monoxide poisoning. Chest 1998;114(4):1036–1041.
2. American Association for Respiratory Care. AARC Clinical Practice Guideline: Pulse oximetry. Respir Care 1991;36(12):1406–1409.
3. Shapiro RS, Kacmarek RM. Monitoring of the mechanically ventilated patient. In: Marini JJ, Slutsky AS, editors. Physiological basis of ventilatory support. (Lung Biology in Health and Disease, Vol 118, Claude Lenfant, Executive Editor) New York: Marcel Dekker; 1998:709–782.
4. Fluck RR Jr, Schroeder C, Frani G, Kropf B, Engbretson B. Does ambient light affect the accuracy of pulse oximetry? Respir Care 2003;48(7):677–680.
5. American Association for Respiratory Care. AARC Clinical Practice Guideline: Blood gas analysis and hemoximetry: 2001 revision & update. Respir Care 2001;46(5):498–505.
6. Shapiro BA, Harrison RA, Walton JR. Clinical application of blood gases, 3rd ed. Chicago: Year Book Medical Publishers; 1982:156–157.
7. Shohat M, Schonfeld T, Zaizoz R, Cohen IJ, Nitzan M. Determination of blood gases in children with extreme leukocytosis. Crit Care Med 1988;16(8):787–788.
8. Fox MJ, Brody JS, Weintraub LR. Leukocyte larceny: a cause of spurious hypoxemia. Am J Med 1979;67(5):742–746.
9. Rello J, Benito S, Triginer C, Net A. False hypoxemia induced by leukocytosis (letter). Crit Care Med 1989;17(9):970.
10. Charan NB, Marks M, Carvalho P. Use of plasma for arterial blood gas analysis in leukemia. Chest 1994;105(3):954–955.
11. Williams LM, Fussell S, Veith RW, Nelson S, Mason CM. Pulmonary veno-occlusive disease in an adult following bone marrow transplantation: case report and review of the literature. Chest 1996; 109(5):1388–1391.