Anemia and Oxygen Delivery

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KEYWORDS

• Anemia • Hematocrit • Hemoglobin • Perfusion • Viscosity

KEY POINTS

- Tissue oxygenation requires both adequate oxygen delivery and effective microvascular perfusion.
- The effects of perioperative anemia cannot be accurately assessed until normovolemia is established.
- The oxygen debt that occurs in severe anemia is caused in part by compromised microvascular blood flow associated with reductions in blood viscosity.
- Blood transfusion can improve tissue oxygenation by normalizing blood viscosity and restoring capillary perfusion as well as by augmenting oxygen carrying capacity.

INTRODUCTION

Oxygen delivery involves the unidirectional transport of oxygen from the atmosphere to the interior of the mitochondria within all of the body's cells. Red blood cells (RBCs) play a dominant role in the convective transport of oxygen from the lungs to the microvasculature. Anemia is a common comorbidity in surgical patients that can complicate perioperative care by compromising oxygen delivery and leading to tissue hypoxia. This article focuses on the effects of anemia on oxygen delivery. Quantitative aspects of global oxygen delivery and the role of hemoglobin (Hb) in gas transport are reviewed. Key concepts regarding microvascular blood flow and its impact on regional oxygen delivery are discussed. Physiologic effects of anemia are summarized. In addition, clinical assessment and management of anemia in the perioperative period are touched on from the perspective of microvascular function.

DETERMINANTS OF TISSUE OXYGENATION

Global oxygen delivery can be described quantitatively by the following familiar equations:

$$Do_2 = CO \times Cao_2 \quad (1)$$

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where Do_2 is whole-body oxygen delivery, CO is cardiac output, and Cao_2 is the oxygen content within arterial blood. Under physiologic condition, 1 g of saturated Hb binds approximately 1.34 mL of oxygen. Cao_2 can therefore be calculated as:

$$Cao_2 \text{ (in mL/dL)} = [Hb](g/dL) \times 1.34 \text{ (mL/g)} \times Sao_2 + (0.003 \times Pao_2) \quad (2)$$

where [Hb](g/dL) is the blood Hb concentration in grams per deciliter, Sao_2 is the percent saturation of Hb, 0.003 is the solubility coefficient of oxygen in plasma, and Pao_2 is the partial pressure of oxygen in the arterial blood. For animals breathing room air, dissolved oxygen accounts for approximately 2% of Cao_2, and its contribution is often disregarded. Thus, Cao_2 can be approximated as:

$$Cao_2 \text{ (mL/dL)} = [Hb](g/dL) \times 1.39 \text{ (mL/g)} \times Sao_2 \quad (3)$$

However, for animals receiving oxygen supplementation, dissolved oxygen can make up a significant proportion of overall delivered oxygen. This point is discussed in more detail later.

The amount of oxygen consumed by the body is expressed as:

$$Vo_2 = CO \times (Cao_2 - Cvo_2) \quad (4)$$

where Vo_2 is total oxygen consumption, and Cvo_2 is the oxygen content within the mixed venous blood of the main pulmonary artery. The fraction of delivered oxygen that is used by the body is the oxygen extraction ratio (o_2ER), expressed as:

$$o_2ER = Vo_2/Do_2 \quad (5)$$

Rearranging Equation 5 and substituting yields the following:

$$Vo_2 = CO \times [Hb](g/dL) \times 1.34 \times Sao_2 \times o_2ER \quad (6)$$

Equation 6 expresses the relationship between oxygen consumption and the parameters that determine oxygen delivery. For a given rate of Vo_2, a decrease in any of the parameters on the right of Equation 6 must be matched by a reciprocal change in 1 or more of the other parameters. Likewise, an increase in oxygen demand (Vo_2) can only be met by a proportional increase in 1 or more of the terms on the opposite side of the equation. Although Equation 6 refers to whole-body Vo_2, it is easy to appreciate how this basic relationship can be used to describe the balance between Vo_2 and Do_2 on regional, organ-specific, or even microvascular scales, by substituting tissue-specific blood flow for CO.

Normal o_2ER is approximately 25%; that is, the amount of oxygen delivered to the body exceeds overall tissue requirements by a factor of approximately 4.[1] A prominent exception is the myocardium, which consumes approximately 50% of delivered oxygen. The excess of Do_2 in relation to Vo_2 represents the physiologic reserve in Do_2 capacity, and is an evolutionary adaptation that ensures adequate oxygen supply to tissues despite moment-to-moment fluctuations in Do_2. As such, the relationship between Do_2 and Vo_2 is biphasic (**Fig. 1**). The upper portion of the curve represents the supply-independent region, where physiologic reserves and compensatory responses maintain Vo_2 despite a decrease in Do_2. However, below a threshold value of Do_2, termed the anaerobic threshold, compensatory mechanisms become exhausted, and Vo_2 becomes limited by Do_2 (supply dependency). The value of Do_2 corresponding with the anaerobic threshold is the Do_2crit, representing the critical level of oxygen delivery below which tissue hypoxia develops.