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Volume 1

Textbook of Anesthesia for Postgraduates

2nd Edition

TK Agasti

Foreword
Atul Prabhakar Kulkarni

JAYPEE

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CHAPTER 4

Oxygen and Carbon Dioxide

JAYPEE BROTHERS

HISTORY

William Harvey of London (1538–1657) had first described circulation of blood in his book, named "deMotu Cordis". He was a student of Galileo. He had first observed the difference of color between arterial and venous blood. But, he was not able to give any reason regarding this difference in color and was unable to draw any firm conclusion about the function of lungs. Then, in-between 1665 and 1675, Robert Hooke, Robert Boyle, Richard Lower, and John Mayow of Oxford had made a understanding that certain components of air is absorbed by lungs and this process is necessary for both the maintenance of life and burning of substances. At that time, "phlogiston theory" was invoked for nearly 100 years. According to this theory, "phlogiston" was supposed to be a part of all the combustible substances and was liberated during their combustion or during an animal's respiration. When it is accumulated in excess as a result of combustion or by respiration in a closed container, then it prevents further chemical activity of life, causing the stoppage of combustion or respiration and death.

After that, *Joseph Priestly* had first prepared oxygen as gas from potassium nitrate in 1771, and was first given the credit for discovering oxygen. But, instead of oxygen, he called this gas as "dephlogisticated air". Because, he observed that this new gas was better than air for supporting respiration and combustion. He also supposed this, because it contained less "phlogiston". So, he called it as "dephlogisticated air".

Then, *Lavoisier* had first clarified the use of oxygen in the process of respiration and combustion of substances. Simultaneously, he also clarified the production of carbon dioxide during combustion of substances and respiration. He subsequently demonstrated that oxygen is absorbed by lungs and after metabolism in our body, it is eliminated as CO_2 and water. Later, he and Laplace had coined the term *oxygen* (oxy = acid, gene = producer) in 1779. They were also the first to compare the heat produced during respiration in animal with that produced during the combustion of carbon and showed the relationship between the oxygen used and carbon dioxide produced. But, later *Lavoisier* was executed by guillotine during French revolution.

Then, Von Liebig had showed, in 1851, that carbohydrates and fats were the substrates of metabolism in cell and not carbon itself. In 1850, French chemist, Boussingault also had first discovered that at a temperature of about 1,000°C, barium monoxide absorbs oxygen from air forming barium dioxide and again releases it at a more higher temperature. Then, this process made charmed and was patented by his pupils, named *Brin brother*, in 1880. After that, they had formed a company and their company eventually, then, became the *British oxygen*.

PROPERTIES OF OXYGEN

Oxygen is a tasteless, colorless, and odorless gas. The concentration of O_2 in atmospheric dry air at 760 mm Hg pressure is 20.95%. The molecular weight of oxygen is 32. In atmosphere, mainly O^{16} isotope of oxygen is present, but a small amount of O^{17} and O^{18} isotopes of it is also found. In one atmospheric pressure (760 mm Hg) and at –183°C temperature, the oxygen becomes liquid (boiling point of liquid oxygen) and in same atmospheric pressure (760 mm Hg) at –218°C temperature, it becomes solid (melting point of solid oxygen). The critical temperature of oxygen is –118°C. So, at temperature greater than this, oxygen exists as gas and cannot be liquefied by raising the pressure. The relative density (air = 1) of oxygen is 1.105. The solubility of O_2 in plasma at 37°C temperature is 0.003 mL/100 mL of blood.

The O_2 cannot be ignited itself, but it aids combustion of substances. So, substances often burn more vigorously or even explode in the presence of oxygen. Hence, explosion can occur due to the presence of grease on the valves of oxygen cylinder. Due to some unpaired electrons, in the

outermost orbit of its atom, the oxygen molecules are attracted to the region of high-magnetic field and is so said it as the paramagnetic gas. Hence, this property of O_2 is utilized in some oxygen analyzers to measure the concentration of it in a mixture of gases.

PREPARATION OF OXYGEN

Commercially in large scale, the oxygen is prepared or manufactured by *fractional distillation of liquid air*. When this liquid air is gradually heated, then the more volatile nitrogen gas (boiling point at 760 mm Hg pressure is –196°C) is separated first, followed by oxygen (boiling point at 760 mm Hg pressure is –183°C). After that, gradually the other components of air are also separated, as required, one after another.

STORAGE OF OXYGEN

Oxygen is usually stored at high pressure as gas in cylinders or as liquid in insulated tanks. For large hospitals, the storage of O_2, as liquid form in an insulated tank, is much more economical. Tank with huge capacity of liquid oxygen is usually insulated by a high-vacuum shell to maintain the inside temperature of tank in-between –175 and –150°C. But, still there is continuous evaporation of some liquid O_2 present within the tank. This is due to the continuous absorption of small amount of heat from the surrounding, in spite of this vacuum insulation. So, if no oxygen is drawn off for long time from the tank, then this accumulated gas (O_2) within the tank should be vented out to the atmosphere. All the O_2 tanks or cylinders are guarded with a pressure regulator valve maintaining a constant supply of oxygen at constant pressure, from the tank or the cylinder into the hospital pipeline system.

The medical grade O_2 contains 99–99.5% pure form of it. In most of the small hospitals, the O_2 is stored in more than two separate banks of cylinders, consisting of the H type of cylinders. They are connected by manifolds and valves. The number of cylinders in one bank and the number of banks in one hospital depend on the anticipated daily demand of O_2 by this hospital. Only one bank is utilized at one time. The valves in manifold connection reduce the cylinder pressure to the given pressure of pipeline and supply gases at target points at this set pressure. A bank automatically switches to another bank when all the cylinders of a bank are emptied. In large hospitals, where the liquid O_2 is used in tanks, an additional smaller O_2 supply system of compressed O_2 cylinders or a bank of cylinders should be there, which can be used as reserve in emergency. Most of the anesthetic machines accommodate one or two E type of O_2 cylinder, in addition to pipeline connection, to guard against the hospital pipeline failure. Anesthesiologists must ensure the supply of O_2 from these cylinders, directly attached to anesthetic machine, before starting of any operation.

OXYGEN TRANSPORT IN BODY

The anesthetists are mainly concerned about the transport and delivery of O_2 from the air to the tissues at cellular level. The delivery of O_2 from the air to the cells at mitochondrial level is a continuous process and is occurred in four phases. These are: (1) the mechanical act of breathing by which air with O_2 reaches the alveoli, (2) the exchange of gas in alveoli by which O_2 passes from the air into the blood, (3) the oxygenated blood, then, is transported to the tissues by circulation, and finally (4) at the level of tissue, from capillaries O_2 enters into the cell and mitochondria.

Transport of Oxygen from Air to Alveoli

The mechanical act of breathing which transports the air with O_2 from the environment to alveoli is fully discussed in separate chapter. But, here, we will discuss only how the tension of oxygen (PO_2) in air falls gradually during its transport from environment to alveoli. When the O_2 reaches from atmosphere to the mitochondria of a cell through alveoli, then the tension of O_2 (PO_2) drops in steps. This is called as the *oxygen cascade*. The PO_2 drops in stages from 159 mm Hg in dry air of atmosphere to a very low level of 2 mm Hg in mitochondria. When the level of PO_2 in mitochondria falls below about 1–2 mm Hg, which is called as the *Pasteur point*, then the aerobic metabolism inside the cells stops. The partial pressure or tension of O_2 in inspired air (PiO_2) is about 20 kPa (kilopascal) or 159 mm Hg (1 kPa = 7.6 mm Hg). It is influenced by the barometric or atmospheric pressure (P_B) and the fractional concentration of oxygen (FiO_2) in air. The normal barometric pressure at sea level is known as one atmospheric pressure and is equivalent to 760 mm Hg, and in this condition, the normal concentration of oxygen or FiO_2 in air is 21% or 0.21. So, the equation of PiO_2 is: $PiO_2 = P_B \times FiO_2 = 760 \times 21/100 = 159$ mm Hg.

Then, the inspired air is saturated and diluted by water vapor after mixing with it, while it is passing through air passages to reach alveoli. So, it causes the reduction of PO_2, when it reaches the alveoli. The partial pressure of water vapor at 1 atmospheric pressure and body temperature (37°C) is 47 mm Hg. So, when the inspired air, saturated with water vapor, reaches the alveoli, then the PiO_2 comes down to 149 mm Hg, which is calculated by the following way: $PiO_2 (Sat) = (P_B - PH_2O) \times FiO_2 = (760 - 47) \times 21/100 = 149.73$ mm Hg ≈ 149 mm Hg **(Flowchart 1)**.

Then, the inspired gas (air), containing oxygen and water vapor, is further diluted by addition of CO_2 and removal of

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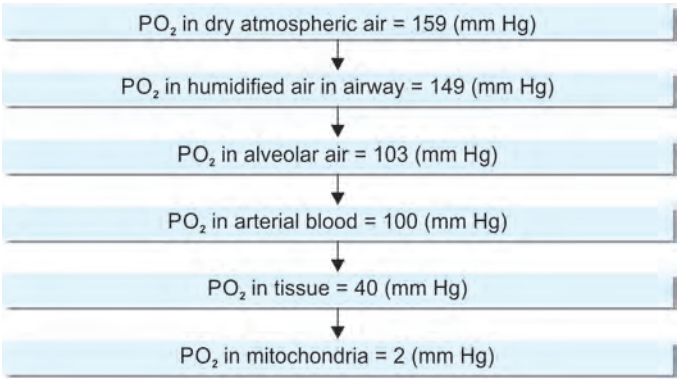
Flowchart 1: Oxygen cascade.



(PO_2: partial pressure of oxygen)

O_2 in alveolus. So, the *final alveolar oxygen tension* in alveoli (P_AO_2) can be estimated by *"alveolar gas equation"*. This alveolar gas equation is: $P_AO_2 = [FiO_2 (P_B - 47) - PaCO_2] \times F = [21/100 (760 - 47) - 46] \times F = 103.73\ F \approx 103$ (while it is taken as 103 mm Hg and F is ignored). Here, P_AO_2 = alveolar O_2 tension, FiO_2 = in air fractional inspired O_2 concentration, P_B = barometric pressure, 47 is the vapor pressure of water at body temperature (37°C), $PaCO_2$ is the arterial CO_2 tension, and F is the respiratory exchange ratio. On the other way, the *final alveolar oxygen tension* also can be calculated by deducting the CO_2 tension in alveoli (which is equivalent to $PaCO_2$) from the inspired O_2 and water vapor tension in alveoli, i.e., 149 – 46 = 103 mm Hg.

From the above equation, it is estimated that the normal value for P_AO_2 is 103 mm Hg at sea level. This normal P_AO_2 will decrease with (1) increasing altitude or decreasing P_B, (2) increasing $PACO_2$, or (3) with decreasing FiO_2. So, a hypercapnic patient at high altitude would have a substantially lowered P_AO_2.

Exchange of Gases in Alveoli

The oxygen tension (PO_2) in blood, entering at the arterial end of pulmonary capillaries, coming from the right side of heart through pulmonary artery, is 40 mm Hg [oxygen saturation (SpO_2) is 75%]. Whereas, the PO_2 in alveolar air is 103 mm Hg. Thus, this pressure gradient helps the O_2 to enter into blood from alveolar air across the alveolar membrane. In the blood, O_2 first dissolves in plasma and then finally unites with hemoglobin (Hb) for its carriage to tissues. Thus, at the venous end of pulmonary capillaries, the PO_2 in blood is 103 mm Hg. In peripheral tissues, the tension of O_2 at the arterial side of capillary blood is about 100 mm Hg. The O_2 tension **(Table 1)** in the tissue is near about 40 mm Hg. When the arterial blood passes through the tissues, it carries about 20 mL of O_2 as oxyhemoglobin and about 0.3 mL of O_2 as physical solution in plasma per 100 mL of blood.

TABLE 1: Partial pressure of gases in alveoli during normal ventilation and hypoventilation with breathing air.

	Normal ventilation with $PaCO_2$ = 40 mm Hg	***Hypoventilation with $PaCO_2$ = 100 mm Hg***
O_2	103 mm Hg	43 mm Hg
N_2	570 mm Hg	570 mm Hg
CO_2	40 mm Hg	100 mm Hg
H_2O	47 mm Hg	47 mm Hg
Total	760 mm Hg	760 mm Hg

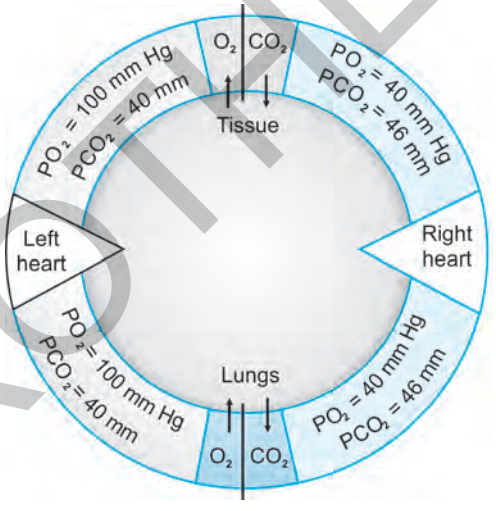


Fig. 1: Transport of O_2 in blood.

As the O_2 tension in tissue level is much lower, so the oxygen present as physical solution in plasma first passes from plasma to the tissues by diffusion. As a result, the O_2 tension in arterial plasma falls. So, the oxyhemoglobin in red blood cell (RBC), being exposed to low O_2 tension in plasma, dissociates and releases O_2 form Hb. Then, this O_2 from RBC enters into plasma and is carried as physically dissolved solution. Again, this physically dissolved O_2 leaves the bloodstream and enters the tissues. Thus, the portion of O_2 dissolved as physical solution in plasma remains always constant and this is measured as PO_2 in blood. About 30% of O_2 is liberated from blood to supply the tissues, when the tissue cells are in resting phase. This dissociation of oxygen from oxyhemoglobin in RBC depends upon: (1) the plasma O_2 tension, (2) the plasma CO_2 tension, (3) plasma H^+ concentration, (4) plasma electrolyte content, and (5) the temperature of blood and tissues **(Fig. 1)**.

The CO_2 tension and its content in tissues are much higher than that of its capillary blood. Due to this difference of pressure (tension), CO_2 diffuses from tissues into the blood of capillaries in tissues. Thus, CO_2 tension in tissue capillary blood rises, which also subsequently favors

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the dissociation of O_2 from oxyhemoglobin into the blood of tissue capillary and enters into tissues. As the increase in PCO_2 in tissue capillary blood favors the dissociation of O_2 from Hb, similarly the increase in PO_2 in tissues favors the dissociation of CO_2 from tissues. Hence, as a result of supply of O_2 to tissues from tissue capillary blood, the O_2 tension and O_2 content of tissue capillary blood fall.

Thus, at the venous end of capillaries in tissues, the O_2 tension of blood is about 40 mm Hg and O_2 content is about 14–15 mL as oxyhemoglobin and about 0.15 mL as physical solution per 100 mL of blood. Subsequently, when this systemic venous blood with PO_2 of about 40 mm Hg passes through the pulmonary capillaries into lungs, wherein the alveoli PO_2 is about 103 mm Hg, then O_2 again enters from alveoli into the systemic venous blood during passing through the pulmonary artery and capillaries. Therefore, O_2 tension (PO_2) rises and more oxy-Hb is formed in the red cells. Simultaneously, CO_2 diffuses out from the systemic venous blood of pulmonary artery into the alveolar air. CO_2 tension and H^+ concentration in the venous blood fall which also favor the entry of O_2 in the pulmonary capillary. The entry of O_2 in pulmonary capillary blood from alveoli also favors the exit of CO_2 from pulmonary capillary blood to alveoli.

Carbon dioxide is much more soluble in plasma than O_2. Hence, as a large amount of CO_2 remains in plasma as solution and diffuses out easily from pulmonary capillary into alveoli, so *hypercapnia (↑ in $PaCO_2$) is rarely a problem in pulmonary fibrosis*, which is discussed in Chapter 3. *It does occur only when the alveolar ventilation is severely inadequate and there is much V_A/Q inequality. If, due to any cause, the $PaCO_2$ increases, other than the decrease in ventilation, then by increasing ventilation the extra CO_2 can easily be expired out. But, it accumulates when the ventilation is severely compromised. It is to be noted that in normal condition, the difference between P_ACO_2 and $PaCO_2$ is very small and alveolar ventilation (V_A) has a great impact on $PaCO_2$ and PaO_2. Doubling the V_A results in half of $PaCO_2$ and reducing the V_A to half results in a doubling of $PaCO_2$.* Therefore, relationship between V_A and $PaCO_2$ is exponential. Similarly, if this change is entered into "alveolar gas equation", then also we will get the relationship between V_A and PaO_2. It can also be seen that hypoxemia develops rapidly than hypercarbia as V_A decreases. But, it can readily be corrected by a small increase in FiO_2 from 0.21 to 0.3. *In summary, we can say that decrease in V_A readily produces hypoxemia than hypercarbia. But, increase in V_A readily corrects hypercarbia than hypoxia. Increase in inspired oxygen concentration (FiO_2) only readily corrects hypoxemia, but not hypercapnia.*

Transport of Oxygen by Blood

Oxygen from higher pressure in the alveolar air enters into the blood of pulmonary capillary, where PO_2 is low than blood. Hence, O_2 gets dissolved in plasma. As more and more O_2 gets dissolved, PO_2 in plasma will increase and then O_2 will enter inside the RBCs. In RBCs, this O_2 will combine with Hb to form HbO_2 (oxyhemoglobin). Thus, O_2 remains in blood as physical solution and also as oxyhemoglobin.

The amount of O_2 transported or delivered to the tissues by blood depends on—(1) the *blood flow (cardiac output)* and (2) the quantity of O_2 carried by blood (O_2 content). Again, the O_2 content carried by blood depends on—(1) the *rate of diffusion of O_2* or the oxygenation of blood in lungs (PaO_2 and SaO_2 where SaO_2 is the percentage of available binding sites of O_2 on Hb) and (2) *Hb content* **(Box 1)**. On the other hand, oxygenation of blood or diffusion of O_2 in blood depends on the inspired and subsequently alveolar O_2 tension. The alveolar O_2 tension (P_AO_2), on the other hand, determines the arterial O_2 tension (PaO_2). The *alveolar and arterial O_2 tension difference (A-a) or gradient* is the simplest way of quantifying the pulmonary diffusion of O_2 from alveolar air into pulmonary capillary blood. Other determining factors of PaO_2 are *R-L shunt and pulmonary diffusion barriers*. We should consider these two factors during determination of PaO_2 only in pathological condition. *Oxygen is transported by blood: (1) as simple physical solution by dissolving oxygen in plasma, and (2) as oxyhemoglobin (HbO_2) combining with Hb.*

Carriage of O_2 by Simple Physical Solution Dissolving in Plasma

A very small proportion of total O_2 content of blood is carried as physically dissolved solution, i.e., 0.3 mL/100 mL of blood/100 mm Hg pressure (0.003 mL/100 mL of blood/1 mm Hg pressure). *But, this small quantity of O_2 is very important, because it alone reflects the tension of O_2 in blood (PO_2). On the other hand, the amount of O_2 present as HbO_2 does not determine PO_2. For example, in case of anemia, the HbO_2 is less, but the dissolved O_2 and PO_2 in arterial blood are normal.* This dissolved O_2 in plasma remains in equilibrium

BOX 1: Factors influencing the amount of O_2 delivered to tissues.

- Amount of O_2 delivered to tissues depends on—blood flow and O_2 content in blood
- O_2 content in blood depends on—Hb concentration and rate of diffusion of O_2
- Rate of diffusion of O_2 through alveolar wall depends on—(A-a) O_2 tension difference
- (A-a) O_2 tension difference depends on—R-L shunt and pulmonary diffusion barrier

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with HbO_2. Hence, first when O_2 enters into bloodstream, it dissolves as physical solution in plasma. Then, from this plasma, the O_2 enters into RBC to attach with Hb. At tissue level, this plasma part of dissolved O_2 is first transferred to tissue cells and its position is then rapidly taken over (replaced) by O_2 liberated from Hb. In this way, the dissolved O_2 in plasma is responsible for movement of O_2. This dissolved O_2 in plasma assumes its practical importance in case of hyperbaric O_2 therapy. Here, the dissolved amount of O_2 in plasma can be increased by giving O_2 at a very high pressure (2 or 3 times of atmospheric pressure or 2–3 × 760 mm Hg) in the treatment of gas gangrene, acute CO poisoning, etc.

Carriage of O_2 by Attaching with Hb (as HbO_2)

Other than physical solution, O_2 is also carried by combining with Hb, which is present in the red cells. This combination of O_2 with Hb is called oxyhemoglobin, which is a loose and reversible compound. O_2 is quickly combined with Hb in the capillary of alveoli, where its tension is high and then is equally quickly dissociated from Hb in the capillary of tissues where its tension is low.

Hemoglobin is a chromoprotein and has four subunits. Each subunit **(Box 2)** contains one structure of heme, consisting of four pyrrole rings with one iron (Fe^{2+}) atom. One heme structure is attached with one α- or β-polypeptide chain of globin (a globin molecule consists of four polypeptide chains: 2α and 2β). So, a hemoglobin molecule contains four atoms of iron in ferrous form and its molecular weight is 68,000. Each atom of iron can reversibly combine with one molecule of O_2. As one Hb molecule contains four heme units and four iron atoms, so it reacts with four molecules of O_2 with formation of oxy-Hb (Hb_4O_8). Under physiological conditions, the reaction of Hb with O_2 takes place in four separate but simultaneous reactions: $Hb_4 + O_2 \rightarrow Hb_4O_2 + O_2 \rightarrow Hb_4O_4 + O_2 \rightarrow Hb_4O_6 + O_2 \rightarrow Hb_4O_8$. The attachment of first O_2 molecule is difficult, but then there is a conformational change in Hb molecule and subsequent O_2 molecules can attach easily. This is called as heme–heme reaction. Amount of O_2 present as HbO_2 depends not only on the amount of Hb, but also on the partial pressure of O_2 to which the blood is exposed and the other factors, such as PCO_2, $[H^+]$, temperature, and 2,3-diphosphoglycerate (2,3-DPG).

Oxygen content: The total oxygen content (in milliliter) in each 100 mL of blood is the total quantity of O_2 attached with Hb in red cells in 100 mL of blood plus the total quantity of O_2 dissolved in plasma in 100 mL of blood, that means the amount of O_2 transported per 100 ml of blood which is shown in **Table 2**. Average Hb level in an adult male is about 15 g/100 mL and 1 g of fully oxygenated Hb contains maximal 1.39 mL of O_2 (in some book, it is written that 1 g of fully oxygenated Hb contains maximum 1.34 mL of O_2). At an arterial PO_2 of 100 mm Hg, Hb is 100% saturated. On the other hand, oxygen content as physically dissolved solution in plasma is 0.3 mL/100 mL of blood/100 mm Hg PaO_2 (0.003 mL/100 mL of blood/1 mm Hg pressure).

So, the total O_2 content in 100 mL of arterial blood at PO_2 of 100 mm Hg pressure is: as oxyhemoglobin + as physical solution.

$$= 1.39 \times Hb \times \% \text{ of saturation} + 0.003 \times PaO_2$$
$$= 1.39 \times 15 \times 1 + 0.003 \times 100$$
$$= 20.85 + 0.3$$
$$= 21.15 \text{ mL/100 mL}$$

Sometimes, this value is taken as average 20 mL/100 mL of arterial blood.

In the venous blood, PO_2 is 40 mm Hg and Hb is 75% saturated.

BOX 2: Gradient of CO_2 and O_2 at tissue and pulmonary level.

At lungs:

A. Venous PCO_2 = 46 mm Hg
Alveolar PCO_2 = 40 mm Hg
Gradient of PCO_2 = 6 mm Hg

B. Venous PO_2 = 40 mm Hg
Alveolar PO_2 = 103 mm Hg
Gradient of PO_2 = 63 mm Hg

At tissue:

C. Arterial PO_2 = 100 mm Hg
Tissue PO_2 = 40 mm Hg
Gradient of PO_2 = 60 mm Hg

D. Arterial PCO_2 = 40 mm Hg
Tissue PCO_2 = 46 mm Hg
Gradient of PCO_2 = 6 mm Hg

(PCO_2: partial pressure of carbon dioxide; PO_2: partial pressure of oxygen)

TABLE 2: Transport of O_2 through blood.

Transport of O_2 by arterial blood	• *As physical solution:* 0.3 mL/100 mL of blood at PaO_2 of 100 mm Hg • *As oxyhemoglobin:* 20.85 mL/100 mL of blood at PaO_2 of 100 mm Hg • *Total:* 21.15 mL/100 mL of blood at PaO_2 of 100 mm Hg
Transport of O_2 by venous blood	• *As physical solution:* 0.12 mL/100 mL of blood at P_VO_2 of 40 mm Hg • *As oxyhemoglobin:* 15.63 mL/100 mL of blood at P_VO_2 of 40 mm Hg • *Total:* 15.75 mL/100 mL of blood at P_VO_2 of 40 mm Hg

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TABLE 3: Distribution of the amount of O_2 in arterial and venous blood.

Oxygen	*Arterial blood*	*Venous blood*
Amount of O_2 as physical solution in plasma	0.3 mL/100 mL	0.13 mL/100 mL
Tension of O_2	100 mm Hg	40 mm Hg
Amount of O_2 combined with Hb	21 mL/100 mL	15 mL/100 mL
Saturation of O_2	100%	75%

(Hb: hemoglobin)

So, O_2 content in 100 mL of venous blood at 40 mm Hg pressure.

$= 1.39 \times Hb \times \%$ of saturation $+ 0.003 \times P_VO_2$
$= 1.39 \times 15 \times 0.75 + 0.003 \times 40$
$= 15.63 + 0.12$
$= 15.75$ mL/100 mL

Therefore, tissue extracts only 21.15 – 15.75 = 5.4 mL (average 5 mL) of O_2 from every 100 mL of arterial blood, while it is passing through the tissue capillaries. Similarly, every 100 mL of blood while passing through lungs takes about 5.4 (average 5 mL) mL of O_2 **(Table 3)**.

O_2 capacity of Hb: It is the maximum amount of O_2 that can combine with Hb in 100 mL of blood. If a sample of 100-mL blood contains 14 or 15 g Hb, then the O_2 capacity of that sample of blood is 14 or $15 \times 1.39 = 19.4$ or 20.8 mL.

Saturation of Hb by oxygen (oxygen saturation) at particular PO_2: It means the fraction of Hb in the form of HbO_2 out of total Hb in 100 mL of blood at a particular oxygen tension. This is calculated as follows: Present O_2 content/maximum O_2 capacity that can be content × 100. For example, in a sample of 100-mL blood there is 19 mL of O_2 at PO_2 of 100 mm Hg. But, it can combine with 20 mL of O_2 at maximum. Therefore, O_2 saturation of Hb in that sample is: Present O_2 content / maximum O_2 capacity $\times 100 = 19/20 \times 100 = 95\%$. If in any blood sample, the O_2 content is 20 mL, which is equal to its maximum capacity, then O_2 saturation of Hb is: $20/20 \times 100 = 100\%$ saturated. This is the arterial blood. In venous blood, the O_2 content is 15 mL in 100 mL of blood. Therefore, O_2 saturation in venous blood is: $15/20 \times 100 = 75\%$. On the other way, when blood contains 20 mL of O_2, then this blood is 100% saturated. Therefore, when a sample of blood is 75% saturated, then the O_2 content is: $20/100 \times 75 = 15$ mL.

Oxygen flux: The total amount of O_2 leaving the left ventricle per minute, when breathing air (with 21% O_2) at sea level (760 mm Hg pressure), has been termed as the oxygen flux. The normal oxygen content of arterial blood is 21.15 mL/100 mL or 211.5 mL/L. The normal cardiac output is 5 L/min.

So, oxygen flux = 5×211.5 mL/min = 1057.5 mL/min.

The O_2 content of venous blood is 15.75 mL/100 mL or 157.5 mL/L.

Therefore, the total amount of O_2 leaving the right ventricle = $5 \times 157.5 = 787.5$ mL/min.

Hence, 1057.5 – 787.5 = 270 mL of O_2 is used up by whole body, during cellular metabolism in every minute and this amount of O_2 is taken up from alveolar air in every minute.

Oxygen flux when Hb drops to 4 g/100 mL in anemia: This can be calculated by the following way:

The O_2 content of 100 mL arterial blood with 4 g of Hb/100 mL of blood

$= 1.39 \times 4 \times 1 + 0.003 \times 100$
$= 5.56 + 0.3$
$= 5.86$ mL/100 mL
$= 58.6$ mL/L

Therefore, O_2 flux = 5×58.6 mL/min = 293 mL/min. It indicates there will be no O_2 reserve.

Oxygen Delivery at Tissues

The mechanism of delivery of O_2 at tissue level has been discussed in this chapter before. But, here we will discuss only some extra points. Previously, it was stated that the amount of O_2 delivered to tissues depends on cardiac output and total oxygen content in blood. This overall flow rate of O_2 to tissues is called as O_2 delivery. With a normal cardiac output of 5 L/min and with normal O_2 content in blood (20 mL/100 mL of blood), the normal O_2 delivery to tissues is 1,000 mL/min or 1 L/min.

Cardiac output varies with the size of patient. So, cardiac output is commonly indexed to body surface area (BSA). This is called as the cardiac index (CI) and can be represented as: CI = CO/BSA (L/min/m^2) or CO = CI × BSA. So, O_2 delivery can be expressed as index: therefore O_2 delivery (index) = CO_2 × CO (mL/min) = CO_2 × CI × BSA (mL/min/m^2). In normal adult patient, the O_2 content = 20 mL/100 mL of blood, the CI = 3 L/min/m^2 and BSA = 10 m^2. So, the O_2 delivery index is 600 mL/min/m^2.

LACK OF OXYGEN OR HYPOXIA

The failure to receive adequate quantities of O_2 by tissues is called as *hypoxia*. Whereas, the total lack of O_2 is called as *anoxia* and it is used in more restricted sense. Less oxygen content in blood is termed as *hypoxemia*. The *asphyxia* is a condition, characterized by decreased O_2 and increased CO_2 (asphyxia = hypoxia + hypercapnia). It (asphyxia) is

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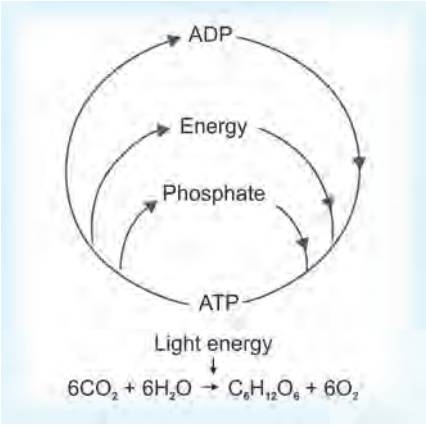


Fig. 2: Mechanism of formation of ATP. (ADP: adenosine diphosphate; ATP: adenosine triphosphate)

discussed in more details, after hypoxia. It occurs mainly due to obstruction of airway such as strangulation and hanging. In conscious patient, there are certain regulatory mechanisms, which prevent the tissue to suffer from lack of oxygen (hypoxia). But, during the course of anesthesia, due to the lack of this regulatory mechanism, the hypoxia has a deleterious effect **(Fig. 2)**.

We get energy from the sun. Earth's atmosphere originally contains no O_2. But, during photosynthesis with the help of CO_2, plant absorbs this light energy in form of glucose and releases O_2 in atmosphere.

$$\text{Light energy} \downarrow$$
$$6CO_2 + 6H_2O \rightarrow C_6H_{12}O_6 + 6O_2$$

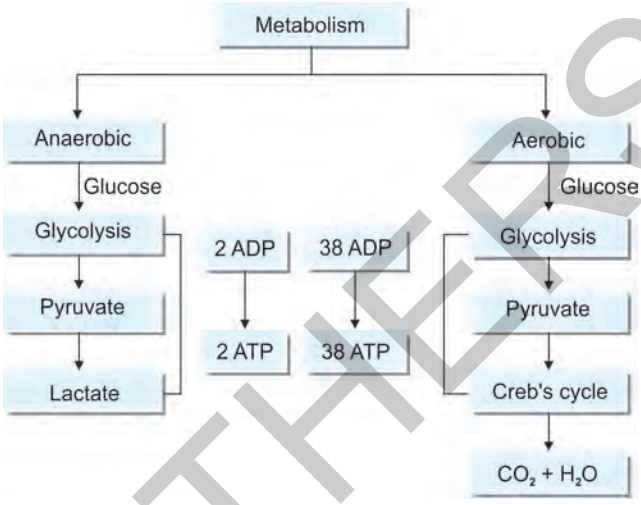
But, within our cell, this reaction is reverse. The solar energy, which is incorporated into glucose, is released by oxidation with the help of oxygen and is used for cellular activity.

$$C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O + \text{Energy (heat)}$$

So, O_2 is very essential to maintain life. Some energy also can be obtained in absence of O_2 (anaerobic metabolism), but in terms of adenosine triphosphate (ATP) production, the anaerobic process is 1/19 as less efficient as aerobic process of metabolism. Because, during aerobic metabolism, one glucose molecule produces 38 ATP (recently it is said that one glucose molecule produces 32 ATP molecules), whereas in anaerobic process, only two ATP is produced from one glucose molecule. Again, lactic acid accumulation, as a result of anaerobic metabolism, will lead to metabolic acidosis. Then, when O_2 becomes available, this lactate can further be metabolized to CO_2 and H_2O with further ATP formation.

The hypoxia may result from various causes and according to the causes of hypoxia, it can be classified into five types: (1) hypoxic hypoxia, (2) anemic hypoxia, (3) stagnant hypoxia, (4) histotoxic hypoxia, and (5) conditions where P_{50} is low **(Flowchart 2)**.

Flowchart 2: Difference between aerobic and anaerobic metabolism. (ADP: adenosine diphosphate; ATP: adenosine triphosphate)



Compensatory Changes due to Hypoxia

Compensatory changes take place, if the hypoxia is persistent and gradual. Because, body gets time to fight with deficiency of O_2. These compensatory changes are:

- Hypoxia stimulates respiratory center, leading to hyperventilation, with an aim to raise PaO_2. The role of hyperventilation in raising PaO_2 can be proved by the alveolar gas equation.
- Rise of BP and there is polycythemia with increased Hb concentration, leading to increased O_2 content of blood.
- Increase in 2,3-DPG content in RBCs, leading to increased delivery of O_2 to tissues.

Aim of these changes is to increase the O_2-carrying capacity in blood and also to increase the ability to release more O_2 at tissue level. Hypoxia, produced due to ventilator failure, also leads to accumulation of CO_2 into tissue. But, mild cases of ventilatory failure are not accompanied by hypercapnia ($\uparrow CO_2$). This is due to higher diffusivity of CO_2. This is also true for pulmonary edema or pneumoconiosis. So, initially these cases are pure hypoxias. Whereas, the chronic cases of gross ventilatory failure, e.g., asthma, chronic bronchitis, etc., actually lead to increase in $PaCO_2$ along with hypoxia. The immediate treatment of hypoxia is O_2 therapy. But in chronic cases, where there is hypercapnia ($\uparrow CO_2$), particularly where chronic airway obstruction is the cause, O_2 should be given with great care. Because, getting O_2 the ventilatory stimulation will be withdrawn and more CO_2 will be accumulated. In such situation, both O_2 therapy and assisted ventilation are needed.

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Types of Hypoxia

Hypoxic Hypoxia

Here, the cause of hypoxia is defective oxygenation of blood in the alveoli of lungs. This results in incomplete saturation of hemoglobin by oxygen and less amount of O_2 is available in physically dissolved form, causing low-oxygen tension in arterial blood. Hypoxic hypoxia is produced in the following conditions.

Decreased partial pressure of O_2 in inspired gas ($PiO_2\downarrow$): This is due to:

- Fall in fractional concentration of O_2 in inspired gas (FiO_2). This occurs during rebreathing or when the supplied gas is hypoxic in mixture (contains less O_2).
- Fall in barometric pressure, for example, at high altitude. At sea level, with 1 atmospheric pressure (760 mm Hg), the air contains 20–21% O_2 concentration and alveolar air contains 14% O_2 concentration. The remaining part of alveolar air is filled up by CO_2, N_2, and H_2O vapor. The total pressure, exerted by all these gases in alveoli, is same as the barometric pressure (760 mm Hg). So, the partial pressure of O_2 (PO_2) in alveoli is 14% of 760 mm Hg or about *106 mm Hg*. At an altitude of 5,500 meters or 18,000 feet, the barometric pressure is only 380 mm Hg. So, the partial pressure of O_2 in alveoli at this altitude will be 14% of 380 mm Hg, i.e., only about *50 mm Hg*. At Mount Everest, where barometric pressure is only 236 mm Hg, the alveolar PO_2 is only *40 mm Hg* (after saturated with water vapor at nasopharynx and adding CO_2 in alveoli). Thus, due to diminution of partial pressure of O_2 in alveoli, which is due to the reduction of barometric pressure at high altitude, less amount of O_2 will diffuse from alveoli into blood and as a result, there will be reduced tension of oxygen in blood.

Fink effect or diffusion hypoxia: At the end of anesthesia, which is carried out by N_2O and O_2, when the patient breaths room air, the N_2O will diffuse out from body tissues via venous blood into alveoli. Subsequently, the N_2 now being breathed with room air will fill the alveoli and diffuse back from alveolar air into tissue. Due to the higher solubility of N_2O than N_2, relatively small amount of N_2 will diffuse in from alveolar air into blood and restore tissue PN_2. But, much larger amount of N_2O than N_2 will diffuse out from tissues into alveoli and dilute (reduce) the alveolar O_2 concentration. Thus, it (N_2O) reduces alveolar PO_2 and produces hypoxia which is opposite to the phenomenon, occurring during the induction of anesthesia with N_2O–O_2 mixture. *This is called as diffusion hypoxia and is opposite to second gas effect* **(Fig. 3)**.

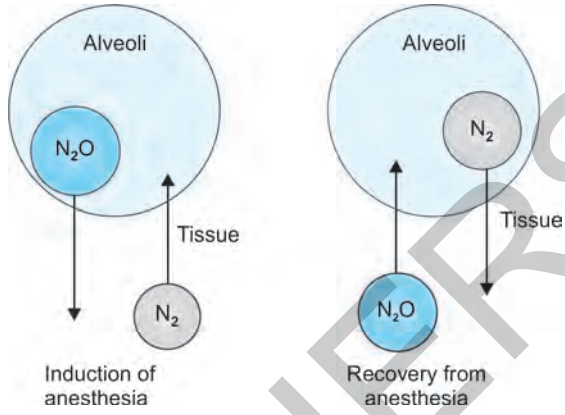

Fig. 3: The comparison between N_2O and N_2 regarding their speed and amount of entry and exit during induction and recovery from anesthesia.

Second gas effect: N_2O is more soluble in blood than N_2. So, during induction with N_2O, the volume of N_2O taken up by blood is much higher than the volume of N_2 entering the alveoli from tissue via blood. Therefore, the alveoli become gradually smaller and the partial concentration of remaining gases in alveoli increases. Now, if another inhalation anesthetic agent (e.g., halothane) is also given with N_2O, then the fractional concentration of this anesthetic gaseous agent will also increase. This phenomenon is called the *second gas effect*, and it increases the speed of induction by this anesthetic gaseous agent. In the early part of induction, the volume of N_2O absorbed from alveoli is in the order of 1 L/min. Although the volume of N_2O in alveoli has decreased, due to high absorption of it from alveoli into blood, but the concentration of it does not reduce to that same extent, because the volume of alveoli is also decreased.

- *Reduced alveolar ventilation:* Arterial O_2 tension (PaO_2) depends on alveolar O_2 tension (P_AO_2) which again depends on alveolar ventilation. The relationship between the alveolar ventilation (V_A) and alveolar PO_2 (P_AO_2) is hyperbolic. It indicates **(Fig. 4)** that a reduction in alveolar ventilation (V_A) of 2 L/min, i.e., from 6 to 4 L/min has little effect on P_AO_2. Whereas, the reduction of ventilation from 4 to 2 L/min has very marked effect on P_AO_2. Again, raising the inspired PO_2 by 64 mm Hg (achieved by increasing the FiO_2 from 0.21 to 0.3, i.e., 21 to 30%) results in a rise in P_AO_2 of same amount, like the reduction of ventilation from 4 to 2 L/min. Increase in O_2 consumption also shifts the V_A and P_AO_2 relationship downward and toward the right. It means that what was previously a perfect adequate alveolar ventilation may be grossly inadequate now, if the oxygen consumption increases. For example, "halothane shake" is a common cause of increased O_2 consumption and hypoxic hypoxia that occurs in the early part of postoperative period.

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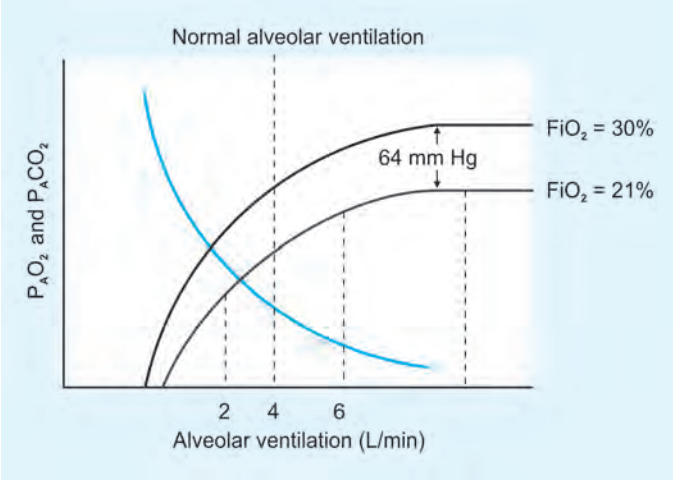

Fig. 4: The effect of the change of alveolar ventilation on alveolar O_2 tension (P_AO_2) drawn in black line and alveolar CO_2 tension (P_ACO_2) drawn in blue line. It also shows the effect of change of FiO_2 from 21 to 30% on P_AO_2. Doubling of V_A results in half of $PaCO_2$ and vice versa. The relationship between V_A and PaO_2 also shows that hypoxemia develops rapidly as V_A decreases. But, it can be readily corrected by a small increase in FiO_2. On the other hand, hyperventilation results in little increase in PaO_2. (FiO_2: fraction of inspired oxygen; $PaCO_2$: arterial CO_2 tension; V_A: alveolar ventilation)

- *Reduced diffusing capacity of O_2 across alveoli:* Due to thickening of alveolar capillary membrane for any cause, there is also impairment of diffusion of O_2 from alveolar gas across the alveolar and pulmonary capillary membrane into pulmonary capillary blood, causing hypoxia. The examples of such type of hypoxia are emphysema, pulmonary edema, pneumonia, interstitial fibrosis, etc.
- *Abnormalities of pulmonary mechanics:* Many abnormalities in pulmonary mechanics such as asthma, emphysema, pneumothorax, collapse, and obstruction in air passages also produce hypoxic hypoxia by reducing the ventilation or decreasing the FiO_2 or changing the V_A/Q ratio.
- *Venous admixture and shunt:* This refers to some conditions when blood passes directly from the venous side of circulation to the arterial side of circulation, i.e., from right side of heart to the left side of heart, without picking up any oxygen from lungs or passing through the zones of lung with low V_A/Q ratio, so that it is less oxygenated than normal. The examples of such direct communication between the right and the left side of heart are true shunt which is responsible for not picking up any oxygen from lungs. This takes place in patent ductus arteriosus (PDA), patent foramen ovale or ventricular septal defect (VSD), etc. with pulmonary hypertension where blood flows from right to left side of heart (true shunt).

Anemic Hypoxia

Here, the problem lies in the carrying of O_2 by blood to tissues, but not in the oxygenation or uptake of O_2 by Hb in alveoli. The characteristic of this anemic hypoxia is that the total O_2 content of arterial blood is reduced, but the partial pressure and the saturation of Hb by O_2 are normal. Therefore, the hemoglobin is fully saturated with O_2, but as the quantity of O_2-carrying Hb is low, so the total amount of O_2 carried by it is below normal. This is called as the anemic hypoxia. Anemic hypoxia will result from following.

Anemia: When the Hb concentration in blood becomes half, then the total O_2 content of blood is also reduced to half. But, the partial pressure of O_2 (which is due to dissolved O in plasma) in blood remains same. Thus, O_2 dissociation curve shifts to the left. But, for compensatory mechanism, anemia causes increase in 2,3-DPG level, therefore shifting again the oxygen dissociation curve to the right at its normal position and favor the unloading of O_2 to tissue. The shifting of O_2 dissociation curve toward left prevents the unloading of O_2 into tissues.

*Carbon monoxide (CO) poisoning or carboxyhemoglobinemia **(Fig. 5)**:* As affinity of CO to hemoglobin is 250 times greater than that of O_2, so a small amount of CO will replace a substantial amount of O_2 from Hb and will produce large amount of carboxyhemoglobin. Therefore, little free hemoglobin will be available for carrying O_2. The other changes produced by carboxy-Hb are the shifting of O_2 dissociation curve to the left. The significance of this shifting of O_2 dissociation curve toward left is that it also unfavors the unloading of remaining O_2 to tissues. *The shift in oxygen dissociation curve of Hb is usually presented mathematically as changes in P_{50} value.* The P_{50} value is defined as the PO_2 at which Hb is 50% saturated. The normal value of P_{50} is 27 mm Hg. It means that at 27 mm Hg in O_2 tension, Hb is only 50% saturated. A shift to the left of O_2 dissociation curve lowers the P_{50} value, i.e., it indicates that at 50% saturation of Hb, the O_2 tension is <27 mm Hg. Similarly, a shift to the right of O_2 dissociation curve raises the P_{50} value, which indicates that at 50% saturation of Hb, the O_2 tension is >27 mm Hg. But, the changes in P_{50} have only a modest effect on the uptake of O_2 in the lungs. The main consequence of alterations of P_{50} is on the release of O_2 in tissues. A low P_{50} or shifting of O_2 dissociation curve to the left decreases the O_2 availability to tissues and, therefore, may lead to cellular hypoxia. Similarly, a high P_{50} value or shifting of O_2 dissociation curve to the right increases the O_2 availability to tissues. As the upper part of O_2 dissociation curve is flat, so the changes in PO_2 at this portion have relatively little effect on Hb saturation by oxygen and, therefore, the blood O_2 content. Whereas, over the lower more vertical part of the curve, the changes in PO_2

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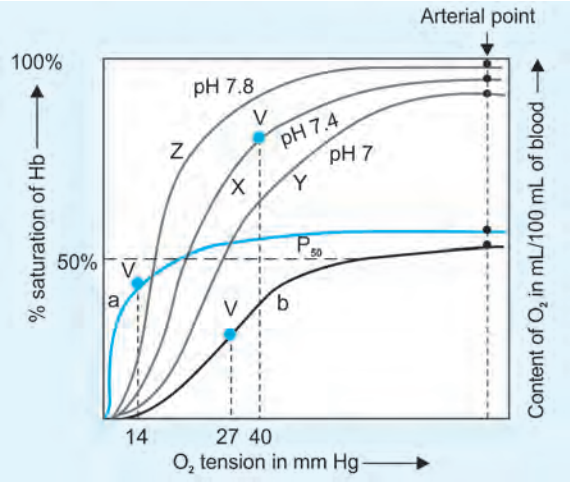

Fig. 5: Different O_2–Hb dissociation curves in different conditions, such as normal, acidosis (pH = 7), and alkalosis (pH = 7.8) (X, Y, and Z curves), CO poisoning (a), and anemia (b). (Hb: hemoglobin)

TABLE 4: Classification and causes of hypoxia.

Hypoxic hypoxia	↓FiO_2, ↓barometric pressure, ↓V_A, diffusion hypoxia, V/Q mismatch, pulmonary diffusion defect, R → L shunt
Anemic hypoxia	Anemia, copoisoning, methemoglobinemia, sulfhemoglobinemia
Stagnant hypoxia	↓cardiac output, MI, heart failure, dehydration
Histotoxic hypoxia	Cyanide poisoning
Low P_{50}	↑ pH, ↓ 2,3-DPG

(DPG: diphosphoglycerate; FiO_2: fraction of inspired oxygen; MI: myocardial infarction)

have a very marked effect on the percentage of saturation of oxy-Hb so the transfer of O_2 from Hb to tissues. These changes in O_2 dissociation curve are also very crucial to the survival of patient, following a massive blood transfusion. Another significance of the shifting of point P_{50} or O_2 dissociation curve is that in order to offload the same amount of O_2 to tissues, the venous PO_2 is also reduced to a much lower level. When the Hb level is 14.4 g/dL, the venous point is 40 mm Hg. In anemia (Hb = 7.2 g/dL), the venous point is 27 mm Hg. In 50% HbCO (Hb = 14.4 g/dL), the venous point is 14 mm Hg. So, although this shift of oxyhemoglobin dissociation curve has little effect on arterial O_2 content, but its effect on venous PO_2 is very significant. At a cerebral venous PO_2 of 14 mm Hg, the subject will be unconscious.

Coal gas has 10% CO as its content. Natural gas also contains trace amount of CO but does not cause CO poisoning. So, coal gas has been replaced by natural gas, which contains about 90% methane with only trace amounts of carbon monoxide. Carboxyhemoglobin level also goes up to 10% in cigarette smoking. Car exhaust and commercial paint removal, containing methylene chloride, cause also severe CO poisoning.

The treatment of CO poisoning consists of hyperbaric O_2 mixed with 5% CO_2, in order to shift the oxyhemoglobin dissociation curve to the right toward its normal position **(Table 4)**.

Methemoglobin and sulfhemoglobin: Methemoglobinemia is another important, but uncommon etiology for the development of cyanosis and anemic hypoxia, which demands prompt diagnosis and treatment. So, for quick evaluation, the history, physical examination, bedside diagnostic techniques, and laboratory confirmation are all important. But, in the absence of significant history, mild cyanosis can easily be missed in dark-skinned individuals during the preanesthetic checkup.

Actually, methemoglobinemia is a condition where the ferrous iron (Fe^{2+}) of hemoglobin complex is oxidized to ferric iron (Fe^{3+}). Usually, under normal conditions, methemoglobin ($HbFe^{3+}OH$) is continuously being formed into RBCs of a normal individual by the process of auto-oxidation. But, a continuous reduction mechanism maintains the methemoglobin levels in blood <1% of total hemoglobin. The most important mechanism for converting the methemoglobin back into normal hemoglobin is the enzymatic reduction by nicotinamide adenine dinucleotide hydrogen (NADH) cytochrome-b5 reductase enzyme. Other minor alternative pathways of methemoglobin reduction utilize the reduced form of nicotinamide adenine dinucleotide phosphate hydrogen (NADPH), which is generated by glucose-6-phosphate dehydrogenase (G6PD) enzyme in the pentose phosphate pathway. Glutathione and ascorbic acid also can reduce the methemoglobin to normal hemoglobin directly, but quantitatively are not very important.

The accumulation of large amounts of chocolate brown, reversibly oxidized methemoglobin in RBCs may be either inherited or acquired. The inherited condition producing methemoglobinemia is also due to two reasons: (1) a dominantly inherited abnormality in the synthesis of a special type of hemoglobin, called hemoglobin M, in which the structural lesion prevents the reduction of methemoglobin to hemoglobin and (2) a recessively inherited deficiency in the synthesis of enzyme methemoglobin reductase.

Thus, in summary, the congenital causes of methemoglobin anemia include—hemoglobin M disease in which there is globin chain mutation defect (autosomal dominant) and deficiency of enzyme such as NADH cytochrome-b5 reductase (autosomal recessive). Hereditary

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