TRANSPORT OF OXYGEN AND CARBON DIOXIDE IN BLOOD
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O2 TRANSPORT
REQUIREMENTS FOR OXYGEN TRANSPORT SYSTEM

Match O2 supply with demand
MOVEMENT OF O$_2$ DOWN CONCENTRATION GRADIENT

Dry inspired air

Humidified bronchial air

Alveolar air

Mixed venous blood

Systemic arterial blood
OXYGEN CASCADE

• Oxygen moves down the concentration gradient from relatively high levels in air to that in the cell.

• The PO2 reaches the lowest level (4-20 mmHg) in the mitochondria.

• This decrease in PO2 from air to the mitochondrion is known as the OXYGEN CASCADE.
KEY STEPS IN OXYGEN CASCADE

• Uptake in the lungs
• Carrying capacity of blood
• Delivery to capillaries
• Delivery to interstitium
• Delivery to individual cells
• Cellular use of oxygen
DETERMINANTS OF PaO₂

• Inspired O₂ concentration & barometric pressure
• Alveolar ventilation
• V/Q distribution & matching
• O₂ diffusion from alveoli to pul capillaries
Oxygen Transport

Carried in bld in 2 forms:

1. by red blood cells
   ✓ Bound to Hb
   ✓ 97-98%

2. Dissolved O2 in plasma
   ✓ Obeys Henry’s law
     \[ PO2 \times \alpha = O2 \text{ conc in sol} \]
     \[ \alpha = \text{Solubility Coefficient (0.003mL/100mL/mmHg at 37C)} \]
   ✓ Low capacity to carry O2
Hemoglobin

- Fe porphyrin compound
- Normal adult = HbA = $\alpha_2\beta_2$
- Hb F= $\alpha_2\gamma_2$
- The $\gamma$ chains ↑ hb affinity to O2
- Each gm of Hb can carry up to 1.34ml of O₂, theoretically up to 1.39 ml/gm

Molecular weight of hemoglobin is 64,000
CHEMICAL BINDING OF HEMOGLOBIN & OXYGEN

• Hemoglobin combines **reversibly** with O₂

• Association and dissociation of Hb & O₂ occurs within milliseconds
  – Critically fast reaction important for O₂ exchange
  – Very loose coordination bonds between Fe²⁺ and O₂, easily reversible

• Oxygen carried in molecular state (O₂) not ionic O²⁻
Oxygen Saturation & Capacity

- Up to four oxygen molecules can bind to one hemoglobin (Hb)
- Ratio of oxygen bound to Hb compared to total amount that can be bound is Oxygen Saturation
- Maximal amount of O2 bound to Hb is defined as the Oxygen Capacity
O2 Content in blood (CaO2)

- 97-98% Carried in Combination With Hb
- 2-3% Dissolved in Plasma

O2 CONTENT -

The sum of O2 carried on Hb and dissolved in plasma

\[ CaO2 \text{ (ml/dL)} = (SaO2 \times Hb \times 1.34) + (PO2 \times 0.003) \]

- O2 content in 100 ml blood (in normal adult with Hb 15 gm/dl) ~ 20 ml/dl
  
  (19.4 ml as OxyHb + 0.3 ml in plasma)
If the PAO2 is ↑ed significantly (by breathing 100% oxygen) then a small amount of extra oxygen will dissolve in the plasma (at a rate of 0.003 ml O2/100ml of blood /mmHg PO2) but there will normally be no significant increase in the amount carried by haemoglobin
Venous O2 content (CvO2)

\[
CvO2 = (SvO2 \times Hb \times 1.34) + (PvO2 \times 0.003) - (\text{normally-15ml/dl})
\]

- mixed venous saturation (SvO2) measured in the pul A represents the pooled venous saturation from all organs.
- SvO2 influenced by changes in both DO2 and VO2
- Normally, the SvO2 is about 75%, however, clinically an SvO2 of about 65% is acceptable
Arterial-Venous Difference

• The arterial-venous oxygen content difference is the difference between the CaO2 and the CvO2.
• The normal \( C(a-v)O_2 \): 5 vol%.

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<thead>
<tr>
<th>Factors that increase the ( C(a-v)O_2 ):</th>
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<tbody>
<tr>
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<td>• exercise</td>
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<tr>
<td>• skeletal relaxation (drugs)</td>
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<td>• peripheral shunting</td>
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<tr>
<td>• poisons</td>
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<td>• decreased temp</td>
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O2 DELIVERY

\[ \text{DO2 (ml/min)} = Q \times \text{CaO2} \times 10 \]

\[ \text{DO2} = Q \times \text{Hb} \times \text{SaO2} \times 1.34 \times 10 \]

(multiplier of 10 is used to convert CaO2 from ml/dl to ml/L)

N- 900-1,100 ml/min

• Decreased oxygen delivery occurs when there is:
  – ↓ed cardiac output
  – ↓ed hemoglobin concentration
  – ↓ed blood oxygenation
O2 CONSUMPTION

• The amount of oxygen extracted by the peripheral tissues during the period of one minute is called oxygen consumption or $V_{O2}$. (N- 200-300ml/min)

$$V_{O2} = Q \times (CaO2 - CvO2) \times 10$$
$$= Q \times 1.34 \times Hb \times (SaO2-SvO2) \times 10$$

• O2 consumption is commonly indexed by the patients body surface area (BSA) and calculated by:
  – $V_{O2} / BSA$
  – Normal $V_{O2}$ index is between 110 – 160ml/min/m$^2$
OXYGEN EXTRACTION RATIO

- The oxygen extraction ratio (O₂ER) is the amount of oxygen extracted by the peripheral tissues divided by the amount of O₂ delivered to the peripheral cells.
- Index of efficiency of O₂ transport
- aka: Oxygen coefficient ratio & Oxygen utilization ratio
  - \( O₂ER = \frac{VO₂}{DO₂} \)
  - When \( SaO₂ \sim 1 \):
    \[ O₂ER \sim SaO₂-SvO₂ \]
  - Normally \( \sim 25\% \) but ↑ to 70-80% during maximal exercise in well trained athletes
Factors that affect O$_2$ER

**Increased with:**
- Decreased CO
- Increased VO$_2$
  - Exercise
  - Seizures
  - Shivering
  - Hyperthermia
- Anemia
- Low PaO$_2$

**Decreased with:**
- Increased Cardiac Output
- Skeletal Muscle Relaxation
- Peripheral Shunting
- Certain Poisons
- Hypothermia
- Increased Hemoglobin
- Increased PaO$_2$
• In general, $DO_2 \gg VO_2$
• When oxygen consumption is high (exercise) the ↑ed $O_2$ requirement is usually provided by an ↑ed $CO$
• Alternatively, if oxygen delivery falls relative to oxygen consumption the tissues extract more oxygen from the hb (the saturation of mixed venous blood falls below 70%) (a-b)

A reduction below point 'c' in figure cannot be compensated for by an increased oxygen extraction and results in anaerobic metabolism and lactic acidosis.
O2 DIFFUSION FROM INTERSTITIUM TO CELLS

Intracellular PO2 < Interstitial fluid PO2
• O2 constantly utilized by the cells
• Cellular metabolic rate determines overall O2 consumption

N PcO2 ~ 5-40 mm Hg (average 23 mmHg)
N intracellular req for optimal maintenance of metabolic pathways ~ 3 mm Hg
Pasteur point –

- critical mitochondrial PO$_2$ below which aerobic metabolism cannot occur
- 0.15 – 0.3 kPa = 1.4 – 2.3mmHg
Oxygen Dissociation Curve

The relationship between the partial pressure of oxygen and the saturation of oxygen.
OXYGEN DISSOCIATION CURVE

- Sigmoid Shaped

- The amount of oxygen that is saturated on the hemoglobin (SO₂) is dependent on the amount dissolved (PO₂).

- Amount of O₂ carried by Hb rises rapidly upto PO₂ of 60mmHg but above that curve becomes flatter

- When Hb takes up small amount of O₂ – relaxed state favours – additional uptake

- Combination Of 1\textsuperscript{st} Heme with O₂ increases affinity of 2\textsuperscript{nd} Heme and so on
Significance of the S-shape curve

% saturation of haemoglobin

100%

partial pressure of O₂ (mmHg)

Plateau:
- haemoglobin highly saturated with O₂
- favour the loading of O₂ in lung

Steep slope:
- small drop of O₂ partial pressure leads to a rapid decrease in % saturation of haemoglobin
- favour the release of O₂ in tissue cells
Steep Portion of Curve

• “Dissociation Portion” of curve.
• Between 10 and 60 mm Hg.
• Small increases in PO$_2$ yield large increases in SO$_2$.
• At the tissue capillary, blood comes in contact with reduced tissue PO$_2$ and oxygen diffuses from the capillary to the tissue.
Flat Portion of Curve

- “Association Portion” of curve.
- Greater than 60 mm Hg.
- Large increases in PO$_2$ yield small increases in SO$_2$.
- At the pulmonary capillary, blood comes in contact with increased alveolar PO$_2$ and oxygen diffuses from the alveolus to the capillary. As the PO$_2$ rises, oxygen binds with the hemoglobin (increasing SO$_2$).
- Very little rise in oxygen saturation above 100 mm Hg of PaO$_2$. 
Rules of Thumb of the Oxyhemoglobin Curve

<table>
<thead>
<tr>
<th>$\text{PO}_2$</th>
<th>$\text{SO}_2$</th>
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<tbody>
<tr>
<td>27</td>
<td>50</td>
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<td>40</td>
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<tr>
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<td>90</td>
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<tr>
<td>250</td>
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<td>40</td>
<td>70</td>
</tr>
<tr>
<td>50</td>
<td>80</td>
</tr>
<tr>
<td>60</td>
<td>90</td>
</tr>
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P \textsubscript{50}

- The partial pressure of oxygen in the blood at which the haemoglobin is 50% saturated, is known as the P\textsubscript{50}.
- The P\textsubscript{50} is a conventional measure of haemoglobin affinity for oxygen
- Normal P\textsubscript{50} value is 26.7 mm Hg
- As P\textsubscript{50} increases/decreases, we say the “curve has shifted”.
  - P\textsubscript{50} less than 27: Shift to the left.
  - P\textsubscript{50} greater than 27: Shift to the right.
Factors affecting Dissociation

BLOOD TEMPERATURE
• increased blood temperature
• reduces haemoglobin affinity for O₂

BLOOD Ph
• lowering of blood pH (making blood more acidic)
• caused by presence of H⁺ ions from lactic acid or carbonic acid
• reduces affinity of Hb for O₂

CARBON DIOXIDE CONCENTRATION
• the higher CO₂ concentration in tissue
• the less the affinity of Hb for O₂
LEFT SHIFT
INCREASED AFFINITY

Acute alkalosis
Decreased PCO₂
Decreased temperature
Low levels of 2,3 DPG
Carboxyhemoglobin
Methemoglobin
Abnormal hemoglobin

RIGHT SHIFT
DECREASED AFFINITY

Acute acidosis
High CO₂
Increased temperature
High levels of 2,3 DPG
Abnormal hemoglobin

Decreasing P½₀

Increasing P½₀
Bohr Effect

• By Christian Bohr in 1904

• The effect of CO$_2$ on the OHDC is known as the **Bohr Effect**

• High P$_{CO_2}$ levels and low pH decrease affinity of hemoglobin for oxygen (a right-ward shift).

• This occurs at the tissues where a high level of P$_{CO_2}$ and acidemia contribute to the unloading of oxygen.
Bohr effect – the effect of $[\text{CO}_2]$ on haemoglobin

Lower $[\text{CO}_2]$ e.g. in lung

- curve shift to the left
- haemoglobin has a higher affinity to $\text{O}_2$

Higher $[\text{CO}_2]$ e.g. tissue cells

- curve shift to the right
- haemoglobin has a lower affinity to $\text{O}_2$
pH & pO$_2$: BOHR EFFECT

The graph shows the relationship between percent Hb saturation and $P_O_2$ (mm Hg) at different pH levels. The pH levels indicated are 7.2, 7.4, and 7.6.
IMPLICATIONS OF BOHR EFFECT

• Enhance oxygenation of blood in lungs and to enhance release of O₂ in the tissues

• In lungs, CO₂ diffuses out of the blood (H⁺ conc ↓ due to ↓ in H₂CO₃ conc) → Shift of O₂-Hb curve to left → ↑ O₂ bound to Hb → ↑ O₂ transport to tissues.

• In tissue capillaries, ↑ CO₂ and ↑ H⁺ → greater release of O₂ due to less avid binding of O₂ to Hb.
DOUBLE BOHR EFFECT

• Reciprocal changes in acid - base balance that occur in maternal & fetal blood in transit through the placenta

FETAL BLOOD               MATERNAL BLOOD

➢ Loss of CO$_2$          Gain of CO$_2$

➢ Rise in pH              Fall in pH

➢ Leftward shift of ODC   Rightward shift of ODC
Oxygen dissociation curve: Foetal VS Maternal

→ Foetal haemoglobin has higher affinity to $O_2$ so as obtain $O_2$ from maternal blood in the placenta.
ROLE OF 2,3 DPG (diphosphoglycerate)

2,3 DPG is an organic phosphate normally found in the RBC. Produced during Anaerobic glycolysis in RBCs.
2,3 DPG

- Tendency to bind to β chains of Hb and thereby decrease the affinity of Hemoglobin for oxygen.
- \( \text{HbO}_2 + 2,3 \text{ DPG} \rightarrow \text{Hb-2,3 DPG} + \text{O}_2 \)
- It promotes a rightward shift and enhances oxygen unloading at the tissues.
- This shift is longer in duration than that due to \([H^+], P_{CO_2}\) or temperature.
  - A doubling of DPG will result in a 10 torr increase in \(P_{50}\).
2,3 DPG

• The levels increase with
  – Cellular hypoxia.
  – Anemia
  – Hypoxemia secondary to COPD
  – Congenital Heart Disease
  – Ascent to high altitudes

• The levels decrease with
  – Septic Shock
  – Acidemia
  – Stored blood
    • No DPG after 2 weeks of storage.
EFFECTS OF 2,3-BPG ON STORED BLOOD

• In banked blood, the 2,3-BPG level falls and the ability of this blood to release $O_2$ to the tissues is reduced.

• less if blood is stored in citrate–phosphate–dextrose solution than acid–citrate–dextrose solution.
Effects of anemia & CO on the oxyhemoglobin dissociation curve

Anemia
- ↓OCC of blood & O₂ content;
- SaO₂ remains normal

Carbon Monoxide [CO]
- affinity of Hb for CO is 250 fold relative to O₂, competes with O₂ binding
- L shift- interfere with O₂ unloading at tissues
- severe tissue hypoxia
- sigmoidal HbO₂ curve becomes hyperbolic

CHANGE THE SHAPE OF OHDC
Oxygen dissociation curve: Haemoglobin VS Myoglobin

Myoglobin has an increased affinity for O₂ (binds O₂ at lower PO₂)

Myoglobin stores O₂ in muscles and release it only when the O₂ partial pressure is very low.
O2 DELIVERY DURING EXERCISE

• During strenuous exercise VO2 may \( \uparrow \) to 20 times N

• Blood also remains in the capillary for \(<1/2\) N time due to \( \uparrow \) C.O.

O2 Sat not affected

• Blood fully sat in first \( 1/3 \) of N time available to pass through pul circulation
• Diffusion capacity ↑ upto 3 fold since:

1. Additional capillaries open up → ↑ no of capillaries participating in diffusion process
2. Dilatation of both alveoli and capillaries → ↓ alveolo-capillary distance
3. Improved V/Q ratio in upper part of lungs due to ↑ blood flow to upper part of lungs
Shift of O2-Hb dissociation curve to right because of:

1. ↑ CO2 released from exercising muscles
2. ↑ H+ ions → ↓ pH
3. ↑ Temp
4. Release of phosphates → ↑ 2,3 - DPG
Percent $O_2$ saturation of hemoglobin

$pO_2$ (mm Hg)

Fully oxygenated blood

Deoxygenated blood (exercising) (at rest)

Right-shift due to increases in $pCO_2$, temperature, acidity, or BPG
OXYGEN DELIVERY IN CRITICAL ILLNESS

• Tissue hypoxia is due to disordered regional distribution of blood flow
• often caused by capillary microthrombosis after endothelial damage and neutrophil activation rather than by arterial hypopxaemia
OXYGEN STORES

• o2 stores are limited to lung and blood.
• The amount of O2 in the lung is dependent on the FRC and the alveolar concentration of oxygen.
• Breathing 100% oxygen causes a large increase in the total stores as the FRC fills with oxygen.
• This is the reason why pre-oxygenation is so effective.
THE EFFECTS OF ANAESTHESIA

• The normal protective response to hypoxia is reduced by anaesthetic drugs and this effect extends into the post-operative period.

• Following induction of anaesthesia: FRC ↓
• V/Q mismatch is ↑ed
• Atelectasis develops rapidly
• This 'venous admixture' increases from N 1% to around 10% following induction of anaesthesia.
THE EFFECTS OF ANAESTHESIA

• Volatile anaesthetic agents suppress hypoxic pulmonary vasoconstriction.
• Many anaesthetic agents depress CO and therefore ↓ O2 delivery.
• Anaesthesia causes a 15% ↓ in metabolic rate and therefore a reduction in oxygen requirements.
• Artificial ventilation causes a further 6% ↓ in oxygen requirements as the work of breathing is removed.
Pulmonary Shunting

• **PERFUSION WITHOUT VENTILATION.**

• Pulmonary shunt is that portion of the cardiac output that enters the left side of the heart without coming in contact with an alveolus.
  
  – **“True” Shunt** – No contact
    • Anatomic shunts (Thebesian, Pleural, Bronchial)
    • Cardiac anomalies
  
  – **“Shunt-Like”** (Relative) Shunt
    • *Some* ventilation, but not enough to allow for complete equilibration between alveolar gas and perfusion.

• **Shunts are refractory to oxygen therapy.**
Fig 1

\[ O_2 = 150\text{mmHg} \]
\[ CO_2 = 0 \]

\[ O_2 = 40 \]
\[ CO_2 = 45 \]

\[ O_2 = 100 \]
\[ CO_2 = 40 \]

\[ O_2 = 150 \]
\[ CO_2 = 0 \]

\[ V/Q = 0 \]
NORMAL \[ V/Q \]
\[ V/Q = \alpha \]

DECREASING \[ V_A/Q \]

(Shunt)

INCREASING \[ V_A/Q \]

(Dead space)
Venous Admixture

- Venous admixture is the mixing of shunted, non-reoxygenated blood with reoxygenated blood distal to the alveoli
- resulting in a reduction in:
  - $\text{PaO}_2$
  - $\text{SaO}_2$
- Normal Shunt: 3 to 5%
- Shunts above 15% are associated with significant hypoxemia
CO$_2$ TRANSPORT
INTRODUCTION TO PHYSIOLOGY OF CO2 TRANSPORT

• end-product of aerobic metabolism.
  – production averages 200 ml/min in resting adult
  – During exercise this amount may increase 6x
• Produced almost entirely in the mitochondria.
• Importance of CO2 elimination lies in the fact that -Ventilatory control system is more responsive to PaCO2 changes.
• Carbon dioxide is transported in the blood from the tissue to the lungs in 3 ways:
  (i) dissolved in solution;
  (ii) buffered with water as carbonic acid;
  (iii) bound to proteins, particularly haemoglobin.

• Approximately 75% of carbon dioxide is transport in the red blood cell and 25% in the plasma attributable to
  – lack of carbonic anhydrase in plasma
  – plasma plays little role in buffering and combination with plasma proteins is poor.
Dissolved carbon dioxide

- Carbon dioxide is 20 times more soluble than oxygen;
- obeys HENRY’S LAW, which states that the number of molecules in solution is proportional to the partial pressure at the liquid surface.

\[
\text{PCO}_2 \times \alpha = \text{CO}_2 \text{ conc in sol}
\]

\[
\alpha = \text{Solubility Coefficient}
\]

Value dependant upon temp (inversely proportional) \(\rightarrow\) more temp lesser amount of CO2 dissolved.

- The carbon dioxide solubility coefficient is 0.69 ml/L/mm Hg at 37C.
• At rest, contribution of dissolved CO2 to total A-V CO2 conc diff only ~10%. In absolute terms only 0.3 ml of CO2/dL transported in dissolved form

• During heavy exercise contribution of dissolved CO2 can ↑ 7 fold → ~1/3 of total CO2 exchange
**CO2 BOUNDED AS HCO3**

- Dissolved CO2 in blood reacts with water to form Carbonic Acid
  - \[ \text{CO2} + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \]

\[
\text{Carbonic acid dissociates into H+ & HCO3}
\]

\[ \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^- \]

- When conc of these ions inc in RBCs, HCO3 diffuses out

- but H+ can’t easily do this because cell memb is relatively impermeable to cations.

- Thus to maintain electrical neutrality, Cl- ions move into cell from plasma [CHLORIDE SHIFT] Band 3 HCO3/Cl carrier protein in RBC memb
Movement of gases at tissue level

Resp for ~ 70% of CO2 transport
• Most of H+ combine with Hb because reduced Hb is less acidic so better proton acceptor

• This fact that deoxygenation of the blood inc its ability to carry CO2 is known as **HALDANE EFFECT.**

• As a result of the shift of chloride ions into the red cell and the buffering of hydrogen ions onto reduced haemoglobin, the **intercellular osmolarity increases slightly** an →→ water enters causing the cell to swell →→ **an increase in mean corpuscular volume (MCV).**

• Hematocrit of venous blood is 3%>arterial

• Venous RBC are more fragile

• Cl content of RBCs V>A
CO2 BOUND AS CARBAMATE

- 15-25% of total CO2 transport
- CO2 reacts directly with terminal amine group of Hb to form the carbaminoHb (Hgb.CO)
- Reversible RX
- Amount of CO2 bound as carbamate to Hb or plasma proteins depends on:
  1) O2 Sat of Hb
  2) H+ conc
- During passage of blood through muscle & tissues, O2 Sat and H+ conc change considerably, in particular during exercise.
Reduction of Hb (↓ oxygenation of heme)

↓

TISSUES

↑ basicity of Hb

↓

↑ H+ binding to reduced Hb

↓

↑ dissociation of H2CO3

↓

↑ carriage of CO2 as HCO3
Oxygenation of Hb

↑ acidity of Hb

↓ tendency to combine with CO2 to form Hgb.CO

Displacement of CO2 from Hb

↓ H+ binding to Hb

↑ Release of H+ from Hb

↑ formation of H2CO3

↑ release of CO2

LUNGS
CO2 DISSOCIATION CURVE

- Total CO2 carriage in the blood depends on the three blood-gas parameters:
  - PCO2
  - Plasma pH
  - PO2

Carbon dioxide dissociation curves relate PaCO2 to the amount of carbon dioxide carried in blood.
CARBON DIOXIDE DISSOCIATION CURVE

- Carbamino HB is much affected by the state of oxygenation of HB, less so by the PCO2.
- Lower the saturation of Hb with O2, larger the CO2 conc for a given PaCO2.
- CO2 curve is shifted to right by increase in SpO2.

Figure 21.10: Effect of O2 on the carbon dioxide equilibrium curve.
• CO2 content rises throughout the increase in partial pressure.
• O2 content rises more steeply until a point at which the hb is fully saturated. After that, the increase is small because of the small increased amount in solution.
• Consequently, the CO2 curve is more linear than the O2Hb dissociation curve.

• Graph illustrates the difference between the content in blood of oxygen and carbon dioxide with change in partial pressure.
O2 DELIVERY FROM LUNGS TO TISSUES

• Major function of circulation to transport O2 from lungs to peripheral tissues at a rate that satisfies overall oxygen consumption.

• Under normal resting conditions - DO2 >> VO2