TRANSPORT OF OXYGEN AND CARBON DIOXIDE IN BLOOD
CONTENTS

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• OXYGEN DELIVERY DURING CRITICAL ILLNESS
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O2 TRANSPORT
REQUIREMENTS FOR OXYGEN TRANSPORT SYSTEM

Match O2 supply with demand
MOVEMENT OF O\textsubscript{2} DOWN CONCENTRATION GRADIENT

Dry inspired air

Po\textsubscript{2} 160
Pco\textsubscript{2} 0

Humidified bronchial air

Po\textsubscript{2} 150
Pco\textsubscript{2} 0

Alveolar air

Pa\textsubscript{O2} 100
Pa\textsubscript{CO2} 40

Alveolus

Mixed venous blood

Po\textsubscript{vO2} 40
Po\textsubscript{vCO2} 46

Systemic arterial blood

Po\textsubscript{aO2} 100
Po\textsubscript{aCO2} 40

Pulmonary Capillary

O\textsubscript{2}, CO\textsubscript{2}
OXYGEN CASCADE

• Oxygen moves down the concentration gradient from a relatively high level 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
     \[ \text{PO}_2 \times \alpha = \text{O2 conc in sol} \]
     \[ \alpha = \text{Solubility Coefficient (0.003mL/100mL/mmHg at 37C)} \]
   ✓ Low capacity to carry O2
Hemoglobin

- Fe porphyrin compound
- **Normal adult = HbA = α2β2**
- Hb F= α2γ2
- The γ 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

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

- 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)

\[
CvO2 = (SvO2 \times Hb \times 1.34) + (PvO2 \times 0.003)
\]

- (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 $\Delta C(a-v)O_2 : 5$ vol\%.

<table>
<thead>
<tr>
<th>Factors that increase the C(a-v)O2:</th>
<th>Factors that decrease the C(a-v)O2:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• decreased cardiac output</td>
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</tr>
<tr>
<td>• increased O2 consumption</td>
<td>• skeletal relaxation (drugs)</td>
</tr>
<tr>
<td>• exercise</td>
<td>• peripheral shunting</td>
</tr>
<tr>
<td>• seizures</td>
<td>• poisons</td>
</tr>
<tr>
<td>• shivering</td>
<td>• decreased temp</td>
</tr>
<tr>
<td>• increased temp</td>
<td></td>
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</tbody>
</table>
O2 DELIVERY

DO2 (ml/min) = Q x CaO2 x 10

DO2 = Q x Hb x SaO2 x 1.34 x 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)

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

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

- The oxygen extraction ratio (O₂ ER) 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
  - \( O₂ ER = \frac{VO₂}{DO₂} \)
  - When \( SaO₂ \sim 1 \):
    \[ O₂ ER \sim SaO₂ - SvO₂ \]
  - Normally \sim 25\% but ↑ to 70-80\% during maximal exercise in well trained athletes
Factors that affect $O_2ER$

**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, DO2 >> VO2

- When oxygen consumption is high (exercise) the ↑ed O2 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 = $1.4 - 2.3$ mmHg
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 up to PO₂ of 60mmHg but above that curve becomes flatter

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

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

Plateau:
- haemoglobin highly saturated with $O_2$
- favour the loading of $O_2$ in lung

Steep slope:
- small drop of $O_2$ partial pressure leads to a rapid decrease in % saturation of haemoglobin
- favour the release of $O_2$ in tissue cells
Steep Portion of Curve

• “Dissociation Portion” of curve.
• Between 10 and 60 mm Hg.
• Small increases in $\text{PO}_2$ yield large increases in $\text{SO}_2$.
• At the tissue capillary, blood comes in contact with reduced tissue $\text{PO}_2$ 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>PO$_2$</th>
<th>SO$_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>27</td>
<td>50</td>
</tr>
<tr>
<td>40</td>
<td>75</td>
</tr>
<tr>
<td>60</td>
<td>90</td>
</tr>
<tr>
<td>250</td>
<td>100</td>
</tr>
</tbody>
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<tr>
<th>PO$_2$</th>
<th>SO$_2$</th>
</tr>
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<tbody>
<tr>
<td>40</td>
<td>70</td>
</tr>
<tr>
<td>50</td>
<td>80</td>
</tr>
<tr>
<td>60</td>
<td>90</td>
</tr>
</tbody>
</table>
The partial pressure of oxygen in the blood at which the haemoglobin is 50% saturated, is known as the P50.

The P50 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 P₅₀
Increasing P₅₀

PERCENT SATURATION HEMOGLOBIN

PO₂ (mm Hg)
Bohr Effect

- By Christian Bohr in 1904
- The effect of CO$_2$ on the OHDC is known as the **Bohr Effect**
- High PCO$_2$ levels and low pH decrease affinity of hemoglobin for oxygen (a right-ward shift).
- This occurs at the tissues where a high level of PCO$_2$ and acidemia contribute to the unloading of oxygen.
Bohr effect – the effect of $[\text{CO}_2]$ on haemoglobin

- Higher $[\text{CO}_2]$ e.g. tissue cells
  - curve shift to the right
  - haemoglobin has a lower affinity to $O_2$

- Lower $[\text{CO}_2]$ e.g. in lung
  - curve shift to the left
  - haemoglobin has a higher affinity to $O_2$
pH & pO$_2$: BOHR EFFECT
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

<table>
<thead>
<tr>
<th>FETAL BLOOD</th>
<th>MATERNAL BLOOD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of CO₂</td>
<td>Gain of CO₂</td>
</tr>
<tr>
<td>Rise in pH</td>
<td>Fall in pH</td>
</tr>
<tr>
<td>Leftward shift of ODC</td>
<td>Rightward shift of ODC</td>
</tr>
</tbody>
</table>
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 \( \beta \) 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 \([\text{H}^+]\), \(\text{PCO}_2\) 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
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
The diagram illustrates the relationship between the percent oxygen saturation of hemoglobin and the partial pressure of oxygen ($pO_2$) in blood. The graph shows two curves: one for fully oxygenated blood (at rest) and another for deoxygenated blood (exercising). The right-shift of the curves due to increases in $pCO_2$, temperature, acidity, or BPG is indicated.
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 hypoxaemia
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

\[ \text{O}_2 = 150 \text{mmHg} \]
\[ \text{CO}_2 = 0 \]

\[ \begin{align*}
\text{O}_2 &= 40 \\
\text{CO}_2 &= 45
\end{align*} \]

\[ \begin{align*}
\text{O}_2 &= 100 \\
\text{CO}_2 &= 40
\end{align*} \]

\[ \begin{align*}
\text{O}_2 &= 150 \\
\text{CO}_2 &= 0
\end{align*} \]

\[ V/Q = 0 \quad \text{DECREASING} \quad V_x/Q \]
\[ \text{NORMAL} \quad V/Q \]
\[ V/Q = \alpha \quad \text{INCREASING} \quad V_x/Q \]

(Shunt) \quad \text{(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 BOUND AS HCO3

• Dissolved CO2 in blood reacts with water to form Carbonic Acid
  • \( \text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \)

<table>
<thead>
<tr>
<th>Carbonic acid dissociates into H+ &amp; HCO3</th>
</tr>
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<tbody>
<tr>
<td>( \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^- )</td>
</tr>
</tbody>
</table>

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 illustrating the difference between the content in blood of oxygen and carbon dioxide with change in partial pressure.](image)
Respiring tissue

- CO₂: 7%
- CO₂: 23%
- CO₂: 70%

Capillary blood

- Dissolved CO₂ gas
- CO₂ + plasma protein → Carbaminohemoglobin compounds
- CO₂ + Hb → HbCO₂
- CO₂ + H₂O → CAH → H₂CO₃ → HCO₃⁻ + H⁺
- O₂ + HbO₂ + H⁺ → HbO₂

Key
- Hb: Hemoglobin
- HbCO₂: Carbaminohemoglobin
- HbO₂: Oxyhemoglobin
- HHb: Deoxyhemoglobin
- CAH: Carbonic anhydrase
THANK YOU
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