Respiratory System #2

The goal of these lectures is to discuss basic respiratory physiology. This lecture will discuss gas transport, control, hypoxia and non-respiratory functions of lungs.

The sections for this lecture are:
- Transport of gases: PO2, PCO2, H+ conc, t°C, DPG, Hb saturation, Bhor, Haldane, respiratory acidosis / alkalosis
- Neural control of ventilation: Rhythmical breathing, PO2, PCO2, and H+ conc. Exercise, other ventilatory responses
- Hypoxia and non-respiratory functions of lungs: Hypoxia and acclimatization to high altitude, Non-respiratory functions of the lungs

FROM YOUR FACEBOOK SITE

"Look out, it's a vicious circle!"

Dr. Advis Class Help
(a Facebook site developed and run by undergraduate students)
Course Outline

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<th>Silverthorn</th>
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<td>REVIEW #1 material from topic #01 – #06</td>
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<td>EXAM #1 material from topic #01 – #06 (33%)</td>
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<td>Basic Physiology of the Renal System</td>
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<td></td>
<td>REVIEW #2 material from topic #01 – #09</td>
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<td></td>
<td>EXAM #2 material from topic #01 – #09 (33%)</td>
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<td></td>
<td>REVIEW #3 material from topic #01 – #12</td>
<td>1 - 26</td>
</tr>
<tr>
<td></td>
<td>EXAM #3 material from topic #01 – #12 (33%)</td>
<td>1 - 26</td>
</tr>
</tbody>
</table>

(all tests are cumulative)

PCO2
PO2
Pa
inputs

chemo & baroreceptors

(PCO2, pH, Po2)

where we would like to be at the end of the cardiovascular and respiratory sections, by the end of this week

neural CV center

heart

baroreceptor mechanism (e.g. carotid sinus)

periphery

(lateral control)

lung

(local control)

output

extrinsic

intrinsic

where we would like to be at the end of the cardiovascular and respiratory sections, by the end of this week
Respiratory System ("lectures")

Introduction (lecture #11)
- Structure / function, gas laws, lungs / chest wall relations, pressures / forces

Lung mechanics (lecture #11)
- Ventilation, inspiration / expiration, compliance / resistance
- Lung volume / capacities, alveolar ventilation / dead space
- Partial pressures of gases and their diffusion in liquids
- Alveolar gas pressures and alveolar - blood exchange
- Matching alveolar ventilation and alveolar blood flow
- Gas exchange in tissues

Transport of O2, CO2 & H ions in blood (lecture #12)
- Hemoglobin (Hb), effect of PO2 on Hb saturation
- Blood PCO2, H+ conc, t°C, DPG on Hb saturation
- Carbamino compounds and carbonic anhydrase
- Total blood carbon dioxide and the Haldane effect
- Respiratory acidosis and respiratory alkalosis

Control of respiration (lecture #13)
- Neural generation of rhythmic breathing
- Control of ventilation by PO2, PCO2, and H+ conc
- Control of ventilation during exercise
- Other ventilatory responses

Hypoxia and non-respiratory functions of lungs (lecture #12)
- Hypoxia and acclimatization to high altitude
- Non-respiratory functions of the lungs

Transport of gases ("Hb role")

Hemoglobin (Hb), effect of PO2 on Hb saturation

Blood PCO2, H+ conc, t°C, DPG on Hb saturation

Carbamino compounds and carbonic anhydrase

Total blood carbon dioxide and the Haldane effect

Respiratory acidosis and respiratory alkalosis
Transport of gases ("Hb role")

<table>
<thead>
<tr>
<th>Hemoglobin (Hb), effect of PO2 on Hb saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood PCO2, H+ conc, t°C, DPG on Hb saturation</td>
</tr>
<tr>
<td>Carbamino compounds and carbonic anhydrase</td>
</tr>
<tr>
<td>Total blood carbon dioxide and the Haldane effect</td>
</tr>
<tr>
<td>Respiratory acidosis and respiratory alkalosis</td>
</tr>
</tbody>
</table>

**Effect of Added Hemoglobin on Oxygen Distribution**

- **(a)** Initial state: no O2 in solution
- **(b)** Oxygen dissolves
- **(c)** At equilibrium, PO2 in air and water is equal. Low O2 solubility means concentrations are not equal.

**TABLE 13-7 Oxygen Content of Systemic Arterial Blood at Sea Level**

<table>
<thead>
<tr>
<th>Component</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 liter (L) arterial blood contains</td>
<td></td>
</tr>
<tr>
<td>3 ml</td>
<td>O2 physically dissolved (1.5%)</td>
</tr>
<tr>
<td>197 ml</td>
<td>O2 bound to hemoglobin (98.5%)</td>
</tr>
<tr>
<td>Total 200 ml</td>
<td>O2</td>
</tr>
<tr>
<td>Cardiac output = 5 L/min</td>
<td></td>
</tr>
<tr>
<td>O2 carried to tissues/min = 5 L/min × 200 ml O2/L</td>
<td></td>
</tr>
<tr>
<td>= 1000 ml O2/min</td>
<td></td>
</tr>
</tbody>
</table>
Transport of gases ("Hb role")

- Hemoglobin (Hb), effect of PO2 on Hb saturation
- Blood PCO2, H+ conc, t°C, DPG on Hb saturation
- Carbamino compounds and carbonic anhydrase
- Total blood carbon dioxide and the Haldane effect
- Respiratory acidosis and respiratory alkalosis

Transport of gases ("Hb role")

- Oxygen-Hemoglobin Dissociation Curve

<table>
<thead>
<tr>
<th>Systemic arterial $P_O_2$</th>
<th>Systemic venous $P_O_2$</th>
<th>Percent hemoglobin saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>40</td>
<td>98%</td>
</tr>
<tr>
<td>40</td>
<td>80</td>
<td>75%</td>
</tr>
</tbody>
</table>
Transport of gases ("O2 diffusion")

- Hemoglobin (Hb), effect of PO2 on Hb saturation
- Blood PCO2, H+ conc, t°C, DPG on Hb saturation
- Carbamino compounds and carbonic anhydrase
- Total blood carbon dioxide and the Haldane effect
- Respiratory acidosis and respiratory alkalosis

Oxygen Movement in Lungs and Tissues

- In pulmonary capillaries
- In tissue capillaries
Transport of gases ("O2 diffusion")

Hemoglobin (Hb), effect of PO2 on Hb saturation

Blood PCO2, H+ conc., t°C, DPG on Hb saturation

Carbamino compounds and carbonic anhydrase

Total blood carbon dioxide and the Haldane effect

Respiratory acidosis and respiratory alkalosis

\[
\text{pH} = pK + \log \left( \frac{\text{HCO}_3^-}{\text{H}_2\text{CO}_3} \right)
\]

\[
\text{pH} = pK + \log \left( \frac{\text{HCO}_3^-}{\text{PCO}_2} \right)
\]

In the lungs

Direct effect

Haldane effect

CO2 unloading

Carbamino effect

BOHR effect

Hemoglobin's affinity for O2

pH = pK + log \left( \frac{\text{HCO}_3^-}{\text{H}_2\text{CO}_3} \right)

pH = pK + log \left( \frac{\text{HCO}_3^-}{\text{PCO}_2} \right)
In the lungs

Hemoglobin (Hb), effect of PO2 on Hb saturation

Blood PCO2, H+ conc, t°C, DPG on Hb saturation

Carbamino compounds and carbonic anhydrase

Total blood carbon dioxide and the Haldane effect

Respiratory acidosis and respiratory alkalosis

Transport of gases ("CO2 diffusion")

Hemoglobin (Hb), effect of PO2 on Hb saturation

Blood PCO2, H+ conc, t°C, DPG on Hb saturation

Carbamino compounds and carbonic anhydrase

Total blood carbon dioxide and the Haldane effect

Respiratory acidosis and respiratory alkalosis

\[
\text{pH} = pK + \log \frac{\text{HCO}_3^-}{\text{H}_2\text{CO}_3}
\]

\[
\text{pH} = pK + \log \frac{\text{HCO}_3^-}{\text{PCO}_2}
\]
Effect of PO2 on Hb saturation

Blood PCO2, H+ conc, t°C, DPG on Hb saturation

Carbamino compounds and carbonic anhydrase

Total blood carbon dioxide and the Haldane effect

Respiratory acidosis and respiratory alkalosis

Hemoglobin (Hb) saturation

Transport of gases ("O2-Hb")
Transport of gases ("O2-Hb")

**Hemoglobin (Hb), effect of PO2 on Hb saturation**

**Blood PCO2, H+ conc., t°C, DPG on Hb saturation**

**Carbamino compounds and carbonic anhydrase**

**Total blood carbon dioxide and the Haldane effect**

**Respiratory acidosis and respiratory alkalosis**

---

**TABLE 13-8 Effects of Various Factors on Hemoglobin**

The affinity of hemoglobin for oxygen is decreased by:

1. Increased hydrogen ion concentration
2. Increased $P_{CO_2}$
3. Increased temperature
4. Increased DPG concentration

The affinity of hemoglobin for both hydrogen ions and carbon dioxide is decreased by increased $P_{O_2}$; that is, deoxyhemoglobin has a greater affinity for hydrogen ions and carbon dioxide than does oxyhemoglobin.
Transport of gases ("O2-Hb")

Hemoglobin (Hb), effect of PO2 on Hb saturation

Blood PCO2, H+ conc, t°C, DPG on Hb saturation

Carbamino compounds and carbonic anhydrase

Total blood carbon dioxide and the Haldane effect

Respiratory acidosis and respiratory alkalosis

\[ pH = pK + \log \frac{HCO_3^-}{H_2CO_3} \]

\[ pH = pK + \log \frac{HCO_3^-}{PCO_2} \]

In the tissue

\[ [H^+] \uparrow \]

In the tissue
Hemoglobin (Hb), effect of PO2 on Hb saturation
Blood PCO2, H+ conc, t°C, DPG on Hb saturation
Carbamino compounds and carbonic anhydrase
Total blood carbon dioxide and the Haldane effect
Respiratory acidosis and respiratory alkalosis
Transport of gases ("CO2 diffusion")

Hemoglobin (Hb), effect of PO2 on Hb saturation

Blood PCO2, H+ conc, t°C, DPG on Hb saturation

Carbamino compounds and carbonic anhydrase

Total blood carbon dioxide and the Haldane effect

Respiratory acidosis and respiratory alkalosis

<table>
<thead>
<tr>
<th>CO2</th>
<th>SYSTEMIC ARTERIAL BLOOD</th>
<th>SYSTEMIC VENOUS BLOOD</th>
</tr>
</thead>
<tbody>
<tr>
<td>FORM</td>
<td>CO2 volume</td>
<td>% of total CO2 in blood</td>
</tr>
<tr>
<td></td>
<td>(mL/liter of blood)</td>
<td></td>
</tr>
<tr>
<td>Dissolved in blood</td>
<td>27</td>
<td>5.5</td>
</tr>
<tr>
<td>Dissolved as bicarbonate</td>
<td>439</td>
<td>88.6</td>
</tr>
<tr>
<td>Bound to hemoglobin</td>
<td>24</td>
<td>4.9</td>
</tr>
<tr>
<td>Total</td>
<td>480</td>
<td>100.0</td>
</tr>
</tbody>
</table>
Transport of gases (“dysfunctions”)

- Hemoglobin (Hb), effect of PO2 on Hb saturation
- Blood PCO2, H+ conc, t°C, DPG on Hb saturation
- Carbamino compounds and carbonic anhydrase
- Total blood carbon dioxide and the Haldane effect
- Respiratory acidosis and respiratory alkalosis

\[
pH = pK + \log \frac{HCO_3^-}{PCO_2}
\]

Control of respiration (“structures”)

- Neural generation of rhythmic breathing
  - Stimulatory
  - Inhibitory

- Control of ventilation by PO2, PCO2, and H+ conc
- Control of ventilation during exercise

- PONS pneumotaxic apneustic
  - Medulla inspiratory center is tonically active
  - Other inputs
  - Expiratory center is active only when stimulated
  - Spinal cord inspirational muscles expiratory muscles
  - Control of respiration means control of amplitude and frequency
  - Lung stretch receptors
Control of respiration (“structures”)

- Neural generation of rhythmical breathing

KEY
- Stimuli
- Sensory receptors
- Afferent neurons
- Integrating centers
- Different neurons
- Effector neurons
- Effectors

Emotions and voluntary control
- CO₂
- O₂ and pH

Higher brain centers
- Medullary chemoreceptors
- Carotid and aortic chemoreceptors

Central pattern generator
- Medullary oblongata
- Pons
- Dorsal respiratory group
- Ventral respiratory group

Somatic motor neurons (inspiration)
- Scalene and sternocleidomastoid muscles
- External intercostals
- Diaphragm

Somatic motor neurons (expiration)
- Somatic muscles

Inspiration
Expiration
Control of respiration (“baroreceptors”)

• Neural generation of rhythmical breathing

• Control of ventilation by PO₂, PCO₂, and H⁺ conc

• Control of ventilation during exercise

Control of respiration (“chemoreceptors”)

• Neural generation of rhythmical breathing

• Control of ventilation by PO₂, PCO₂, and H⁺ conc

• Control of ventilation during exercise

TABLE 13–9 Major Stimuli for the Central and Peripheral Chemoreceptors

Peripheral chemoreceptors—that is, carotid bodies and aortic bodies—respond to changes in the arterial blood. They are stimulated by:

1. Decreased $P_{O_2}$ (hypoxia)
2. Increased hydrogen ion concentration (metabolic acidosis)
3. Increased $P_{CO_2}$ (respiratory acidosis)

Central chemoreceptors—that is, located in the medulla oblongata—respond to changes in the brain extracellular fluid. They are stimulated by increased $P_{CO_2}$ via associated changes in hydrogen ion concentration. (See Equation 13–11.)
Control of respiration (‘ventilation’)

- Neural generation of rhythmical breathing
- Control of ventilation by PO2, PCO2, and H+ conc
- Control of ventilation during exercise

Chemicals That Stimulate Ventilation

1. **Arterial $P_{O_2}$**
2. **Production of non-CO2 acids**
3. **Arterial $P_{CO_2}$**
4. **Brain extracellular fluid $P_{CO_2}$**
5. **Brain extracellular fluid $[H^+]$**
6. **Firing of medullary inspiratory neurons**
7. **Firing of neurons to diaphragm and inspiratory intercostals**
8. **Diaphragm and inspiratory intercostals contractions**
9. **Ventilation**
Control of respiration ("ventilation")

- Neural generation of rhythmical breathing
- Control of ventilation by PO2, PCO2, and H+ conc
- Control of ventilation during exercise
Control of respiration ("ventilation")

- Neural generation of rhythmical breathing
- Control of ventilation by PO2, PCO2, and H+ conc
- Control of ventilation during exercise

Factors That Increase and Decrease Ventilation

1. Neural generation of rhythmical breathing
2. Control of ventilation by PO2, PCO2, and H+ conc
3. Control of ventilation during exercise
Control of respiration

- Neural generation of rhythmical breathing
  - stimulate inspiratory center
  - inhibit inspiratory center
  - peripheral mechanoreceptors (skin, joints, etc.)
  - upper respiratory tract (swallowing, diving reflex)

- Control of ventilation by PO2, PCO2, and H+ conc
  - increase baroreceptor activity
  - decreases inspiration & venous return
  - decrease baroreceptor activity
  - increases inspiration & venous return
  - increase PCO2 → increases Va
  - decrease PCO2 → decrease Va
  - increase H → increase Va
  - decrease H → decrease Va
  - increase Po2 → decrease Va
  - decrease Po2 → increase Va

- Control of ventilation during exercise
  - periphereal mechanoreceptors (skin, joints, etc..)
  - Low PO2
  - High PCO2

Control of respiration (‘ventilation’)

- Increase baroreceptor activity increases inspiration & venous return
- Decrease baroreceptor activity decreases inspiration & venous return
- Increase PCO2 increases Va
- Decrease PCO2 decreases Va
- Increase H increases Va
- Decrease H decreases Va
- Increase Po2 decreases Va
- Decrease Po2 increases Va
Control of respiration (‘‘high PCO2’’)

- Neural generation of rhythmic breathing
- Control of ventilation by PO2, PCO2, and H+ conc
- Control of ventilation during exercise

KEY
- Stimulus
- Receptor
- Systemic response

Plasma PCO2
- ↑PCO2 in CSF
- ▲Arterial PCO2
- ↑CO2 → ↑H+ + ↑HCO3− in CSF
- ↑CO2 → ↑H+ + ↑HCO3− in plasma

Stimulates central chemoreceptor
Stimulates peripheral chemoreceptor

Ventilation

Plasma PO2 < 60 mm Hg

↓Plasma PO2
↓Plasma PCO2

Negative feedback
Control of respiration

Factors That Stimulate Ventilation

- Neural generation of rhythmical breathing
- Control of ventilation by PO2, PCO2, and H+ conc
- Control of ventilation during exercise

![Diagram of respiratory control systems](image-url)
Control of respiration ("exercise")

- Neural generation of rhythmic breathing
- Control of ventilation by PO2, PCO2, and H+ conc
- Control of ventilation during exercise

Hypoxia ("dysfunctions")

- Hypoxia and acclimatization to high altitude

**Table 18-1**: Normal Blood Values in Pulmonary Medicine

<table>
<thead>
<tr>
<th>Arterial</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>FiO2</td>
<td>95 mm Hg (85–100)</td>
</tr>
<tr>
<td>FiCO2</td>
<td>40 mm Hg (25–40)</td>
</tr>
<tr>
<td>pH</td>
<td>7.4 (7.38–7.42)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 18-2: Classification of hypoxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute hypoxia</td>
</tr>
<tr>
<td>Chronic hypoxia</td>
</tr>
<tr>
<td>Hypothalamic hypoxia</td>
</tr>
<tr>
<td>Hypoxic hypoxia</td>
</tr>
</tbody>
</table>

- Normal lung: PO2 normal
- Emphysema: PO2 normal or low
- Fibrotic lung diseases: thickened alveolar membrane slows gas exchange. Loss of lung compliance may decrease alveolar ventilation.
- Pulmonary edema: fluid in interstitial space increases diffusion distance. Arterial Po2 may be normal due to higher CO2 solubility in water.
- Asthma: increased airway resistance decreases alveolar ventilation.
### TABLE 18-1

<table>
<thead>
<tr>
<th>Normal Blood Values in Pulmonary Medicine</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ARTERIAL</strong></td>
</tr>
<tr>
<td>$P_{O_2}$ 95 mm Hg (85–100)</td>
</tr>
<tr>
<td>$P_{CO_2}$ 40 mm Hg (35–45)</td>
</tr>
<tr>
<td>pH 7.4 (7.38–7.42)</td>
</tr>
</tbody>
</table>

### TABLE 18-2

<table>
<thead>
<tr>
<th>TYPE</th>
<th>DEFINITION</th>
<th>TYPICAL CAUSES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxic hypoxia</td>
<td>Low arterial $P_{O_2}$</td>
<td>High altitude; alveolar hypoventilation; decreased lung diffusion capacity; abnormal ventilation-perfusion ratio</td>
</tr>
<tr>
<td>Anemic hypoxia</td>
<td>Decreased total amount of $O_2$ bound to hemoglobin</td>
<td>Blood loss; anemia (low Hb) or altered HbO$_2$ binding; carbon monoxide poisoning</td>
</tr>
<tr>
<td>Ischemic hypoxia</td>
<td>Reduced blood flow</td>
<td>Heart failure (whole-body hypoxia); shock (peripheral hypoxia); thrombosis (hypoxia in a single organ)</td>
</tr>
<tr>
<td>Histotoxic hypoxia</td>
<td>Failure of cells to use $O_2$ because cells have been poisoned</td>
<td>Cyanide and other metabolic poisons</td>
</tr>
</tbody>
</table>

---

### (a) Normal lung

- $P_{O_2}$ normal
- $P_{O_2}$ normal

### (b) Emphysema: destruction of alveoli reduces surface area for gas exchange.

- $P_{O_2}$ normal or low
- $P_{O_2}$ low

### (c) Fibrotic lung disease: thickened alveolar membrane slows gas exchange. Loss of lung compliance may decrease alveolar ventilation.

- $P_{O_2}$ normal or low
- $P_{O_2}$ low

### (d) Pulmonary edema: fluid in interstitial space increases diffusion distance. Arterial $P_{CO_2}$ may be normal due to higher $CO_2$ solubility in water.

- Exchange surface normal
- $P_{O_2}$ normal
- Increased diffusion distance
- $P_{O_2}$ low

### (e) Asthma: increased airway resistance decreases airway ventilation.

- Bronchioles constricted
- $P_{O_2}$ low
- $P_{O_2}$ low
### Hypoxia ("dysfunctions")

#### Hypoxia and acclimatization to high altitude

<table>
<thead>
<tr>
<th>TABLE 13-10 Causes of a Decreased Arterial $P_{\text{O}_2}$ (Hypoxic Hypoxia) in Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Hypoventilation may be caused (a) by a defect anywhere along the respiratory control pathway, from the medulla through the respiratory muscles, (b) by severe thoracic cage abnormalities, and (c) by major obstruction of the upper airway. The hypoxemia of hypoventilation is always accompanied by an increased arterial $P_{\text{CO}_2}$.</td>
</tr>
<tr>
<td>2. Diffusion impairment results from thickening of the alveolar membranes or a decrease in their surface area. In turn, it causes failure of equilibration of blood $P_{\text{O}<em>2}$ with alveolar $P</em>{\text{O}<em>2}$. Often it is apparent only during exercise. Arterial $P</em>{\text{CO}_2}$ is either normal, since carbon dioxide diffuses more readily than oxygen, or reduced, if the hypoxemia reflexly stimulates ventilation.</td>
</tr>
<tr>
<td>3. A shunt is (a) an anatomic abnormality of the cardiovascular system that causes mixed venous blood to bypass ventilated alveoli in passing from the right side of the heart to the left side of the heart, or (b) an intrapulmonary defect in which mixed venous blood perfuses unventilated alveoli (ventilation/perfusion $= 0$). Arterial $P_{\text{CO}<em>2}$ generally does not rise since the effect of the shunt on arterial $P</em>{\text{CO}_2}$ is counterbalanced by the increased ventilation reflexly stimulated by the hypoxemia.</td>
</tr>
<tr>
<td>4. Ventilation/perfusion inequality is by far the most common cause of hypoxemia. It occurs in chronic obstructive lung diseases and many other lung diseases. Arterial $P_{\text{CO}_2}$ may be normal or increased, depending upon how much ventilation is reflexly stimulated.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 13-11 Acclimatization to the Hypoxia of High Altitude</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. The peripheral chemoreceptors stimulate ventilation.</td>
</tr>
<tr>
<td>2. Erythropoietin, a hormone secreted by the kidneys, stimulates erythrocyte synthesis, resulting in increased erythrocyte and hemoglobin concentration in blood.</td>
</tr>
<tr>
<td>3. DPG increases and shifts the hemoglobin dissociation curve to the right, facilitating oxygen unloading in the tissues. However, this DPG change is not always adaptive and may be maladaptive. For example, at very high altitudes, a right shift in the curve impairs oxygen loading in the lungs, an effect that outweighs any benefit from facilitation of unloading in the tissues.</td>
</tr>
<tr>
<td>4. Increases in capillary density (due to hypoxia-induced expression of the genes that code for angiogenic factors), mitochondria, and muscle myoglobin occur, all of which increase oxygen transfer.</td>
</tr>
<tr>
<td>5. The peripheral chemoreceptors stimulate an increased loss of sodium and water in the urine. This reduces plasma volume, resulting in a concentration of the erythrocytes and hemoglobin in the blood.</td>
</tr>
</tbody>
</table>
Respiratory System

TOTAL ARTERIAL O₂ CONTENT

Oxygen dissolved in plasma (Pₒ₂ of plasma) helps determine

- Composition of inspired air
- Alveolar ventilation
- Oxygen diffusion between alveoli and blood
- Adequate perfusion of alveoli
- Rate and depth of breathing
- Lung compliance
- Surface area
- Diffusion distance
- Membrane thickness
- Amount of interstitial fluid

Oxygen bound to Hb

- % Saturation of Hb affected by
  - Pₒ₂
  - pH
  - Temperature
  - 2,3-DPG
- Hb content per RBC
- Number of RBCs

Total number of binding sites

- Total number of binding sites
- Number of RBCs
Respiratory System

Respiratory / cardiovascular interaction

integrators compare what it should be with what it actually is and generate an error signal
Respiratory System

integrators compare what it should be with what it actually is and generate an error signal

where we would like to be at the end of the cardiovascular and respiratory sections, by the end of this week
I wonder how will I look after the next section

???

I’D RATHER BE AT THE BEACH