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Respiratory Control, Ventilation, and Regulation of \( P_{a}CO_2 \)

M1 – Cardiovascular/Respiratory Sequence

Louis D’Alecy, Ph.D.
Tuesday 11/18/08, 10:00
Respiratory Control
vs.
Control of Breathing (Ventilation)
vs.
Regulation of $P_aCO_2$
(22 slides, 50 minutes)
Apnea, Eupnea, Tachypnea, Hyperpnea
Hyperventilation & Hypoventilation
Eupnea: normal quiet ventilation

- Automatic & cyclic
- Originate in CNS - medulla
- “final common pathway” via phrenic nerves
- Bursts of nerve firing cause inspiration
- Interval between groups determines rate
- Frequency and # of motor units - depth (TV)
- Diaphragm (2/3 supine)+external intercostals
All modifiers of basic rate

1. lung and airways
2. baroreceptors
3. muscle and joints
4. pain
5. chemoreceptors

respiratory center in medulla

Ach-nicotinic respiratory muscles
Control of Respiration

Influences from higher centers

Voluntary

Cycle of inspiration and expiration

Muscles of breathing

Reflexes from:
- Lungs
- Airways
- Cardiovascular system
- Muscles and joints
- Skin

Reflexes from:
- Arterial chemoreceptors
- Central chemoreceptors
CONTROL OF RESPIRATION DURING EXERCISE

RESPIRATORY CENTER

- Temperature
- Epinephrine
- Joint receptors
- Ventilation

Motor cortex

Skeletal muscle

Muscle spindles & Golgi tendon organs
Receptor Modulation of Ventilation

1. Lung and airway receptors
   
   A. stretch receptors - inflation inhibits inspiration  
      Hering-Breuer reflex  
   
   B. irritant receptors - sneeze and cough  
   
   C. juxtapulmonary receptors - stimulated by  
      pulmonary edema - sensation of dyspnea  

2. Baroreceptors - decreased MAP increases ventilation  

3. Muscles and joints - movement increases ventilation  

4. Pain - gasp reflex
Altered Breathing

- **Apnea**: absence of breathing
- **Eupnea**: normal quiet breathing
- **Tachypnea**: rapid breathing
- **Hyperpnea**: increase $V_T$ + or - increased $V_A$
- **Hyperventilation**: excess ventilation that
  - ******* Lowers PaCO2 *******
- **Hypoventilation**: inadequate ventilation
  - ******* Increases PaCO2 *******
\[ \dot{V}_T \equiv \dot{V}_E \]

Minute Ventilation

Alveolar Ventilation

Dead Space Ventilation

\[ \dot{V}_A = \dot{V}_T - \dot{V}_D \quad \text{or} \]

\[ \dot{V}_A = \dot{V}_E - \dot{V}_D \]
Ventilation on $PA_{CO_2}$

Source Undetermined

Hypoventilation  Normal  Hyperventilation
PERIPHERAL CHEMORECEPTORS

- Carotid sinus
- Common carotid artery

Stimulated by:
- Arterial $P_{O_2}$
- Arterial $pH$ (equal to $H^+$)
- Arterial $P_{CO_2}$

Please see: http://www.medicine.mcgill.ca/physio/resp-web/Figures/Figtt20.jpg
PaO₂ leads to ↑ CO₂ Response

Hypoxia increases ventilatory response to CO₂.

Normal slope. Small Increases in PaCO₂ markedly increase ventilation.

Alveolar Ventilation L/min

PaCO₂ mmHg

PaO₂ = 35 mmHg
PaO₂ = 50 mmHg
PaO₂ = 100 mmHg

Increased slope
ACIDOSIS = an increase in the [H+] concentration of blood

**Respiratory Acidosis** - accumulation of CO₂ (hence H+) due to hypoventilation.

**Metabolic Acidosis** - Any accumulation of acid (H+) that does not come from retained CO₂.

From: a) metabolic production of organic acids such as lactic acid or ketone bodies,

b) ingestion of acidic substances or

c) failure of kidneys to excrete acid.
Plasma Acidosis → Ventilation

Minor increases in $[H^+]$ markedly stimulate ventilation.

pH  7.4  →  7.33
PERIPHERAL CHEMORECEPTORS

Response to Acid & CO$_2$

<table>
<thead>
<tr>
<th>$[H^+]$ nmol/L</th>
<th>40</th>
<th>42</th>
<th>44</th>
<th>46</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.4</td>
<td></td>
<td></td>
<td>7.33</td>
</tr>
<tr>
<td>$P_{CO_2}$</td>
<td>40</td>
<td>50</td>
<td>55</td>
<td></td>
</tr>
</tbody>
</table>
The decrease in CSF CO₂ causes CSF alkalosis.
Arterial CO$_2$ is major determinant of ventilation **BUT** this CO$_2$ response can be altered significantly.
Hyperventilation: $\downarrow$ PaCO$_2$

Breathing CO$_2$: $\uparrow$ ventilation

Breathing CO$_2$ increases ventilation.

The higher the ventilation, the lower the PaCO$_2$. 

O₂ Response* Interacts with CO₂

- Insensitive at normal PO₂
- O₂ *response all peripheral
- PₐO₂ not content

Low PO₂ response "fail safe".

Does giving 100% O₂ to a hypercapnic patient suppress ventilation? Let’s ask Dr. Sisson.
What about central chemoreceptors?
No direct effect of blood $H^+$ and $HCO_3^-$.

Central
-No $O_2$ response.
-Slower.
Cerebrospinal fluid

Central chemoreceptors in medulla

↑ Blood CO₂ and CSF H⁺

Insensitive to Blood H⁺ or O₂

Central chemoreceptors respond to

Blood-brain barrier

CO₂ + H₂O → H⁺ + HCO₃⁻

Cerebral blood flow
Summary:

Control of the respiratory system is based on responses to peripheral chemoreceptor stimulation resulting in tight regulation of arterial CO₂ ($P_a CO_2$).
Additional Source Information

for more information see: http://open.umich.edu/wiki/CitationPolicy

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