Dynamics of Pulmonary Ventilation

Neural Factors
- Medulla (respiratory center), pons, subcortical region & motor cortex
- Inspiratory neurons activate diaphragm and intercostals
  a) Limited by influence of expiratory neurons
    Stretch receptors
- Neural center in the hypothalamus integrates input from ascending & descending neurons:
  a) Influences duration and intensity of respiratory cycle

Humoral Factors
- Peripheral chemoreceptors located in:
  a) Aorta and carotid arteries
    Stimulated by ↓PO2
    Stimulated also by exercise
- During exercise:
  a) ↑PCO2
  b) ↑Temperature
  c) ↑acidity
  d) ↑potassium concentrations

Plasma PCO₂ & H⁺ Concentrations
- PCO₂ in arterial blood provides the most important respiratory stimulus at rest
  a) Ventilation increases to decrease PCO₂
- ↓blood pH reflects CO₂ retention or lactate accumulation (exercise)
- Hyperventilation & breath-holding
  a) Sport-specific application: swimming & divers

Ventilation During Exercise
- Chemical Control:
  a) Combination of factors
    Fluctuations in PO₂, PCO₂

Figure 14.1

Ventilatory Control

Figure 14.3

Metabolic production of CO₂ & H⁺
**Ventilation During Exercise**

- **Nonchemical Control:**
  
  a) **Neurogenic Factors** – responsible for rapid response to increase ventilation
     1. Cortical influence – activated in anticipation
     2. Peripheral influence – sensory input from limbs
  
  b) Temperature has little influence on respiratory rate during exercise
     ✓ Ventilation fluctuation too rapid to reflect changes in core temperature

- **Integrated Regulation During Exercise**

  • Phase I (beginning of exercise): neurogenic stimuli from cortex increases respiration
  
  • Phase II: after about 20 seconds $V_E$ rises exponentially to reach steady state
    a) Central command
    b) Peripheral chemoreceptors
  
  • Phase III: fine tuning of steady-state ventilation through peripheral sensory feedback mechanisms

**In Recovery**

- An abrupt decline in ventilation reflects removal of central command and input from receptors in active muscle

- Slower recovery phase from gradual metabolic, chemical and thermal adjustments

**Pulmonary Ventilation During Exercise**

1. Ventilation ($V_E$) in Steady-State Exercise:
   
   a) During light to moderate exercise:
      ✓ Ventilation increases linearly with $O_2$ consumption and $CO_2$ production
      ✓ At lower intensities, ventilation ↑ primarily due to ↑ TV
      ✓ At higher intensities, primarily ↑ breathing rate

2. Ventilation in Non-Steady-State Exercise:
   
   a) $V_E$ rises sharply and the ventilatory equivalent rises as high as 35–40

**Ventilatory Threshold ($V_T$)**

- The point at which pulmonary ventilation ↑ disproportionately with $O_2$ consumption during exercise
  
  a) pulmonary ventilation no longer tightly associated with $O_2$ demand at the cellular level

- Excess ventilation results from:
  
  a) CO$_2$ increased output from buffering of lactate
  
  Lactate + NaHCO$_3$ $\rightarrow$ Na lactate + H$_2$CO$_3$ $\rightarrow$ H$_2$O + CO$_2$
  
  b) ↑ nonmetabolic $CO_2$ stimulates ventilation
Other factors affecting ventilation

- Energy Cost of Breathing:
  - a) 3 to 5% of total O₂ consumption during light to moderate exercise
  - b) 8 to 15% during maximal exercise
  - c) Respiratory muscles at max ~ 15% of total blood flow

Does $V_E$ Limit Aerobic Power & Endurance?

- Ventilation in healthy individuals is not the limiting factor in exercise
  - a) Breathing reserve even at maximal exercise

Acid-Base Regulation

- General terms:
  - a) Acids: dissociate $H^+$ in solution
  - b) Bases: accept $H^+$ to form $OH^-$ ions
  - c) Buffering: minimize changes in pH or $[H^+]$

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Regulation of internal pH

- Chemical buffers:
  a) Sodium bicarbonate, phosphate, certain proteins

- Ventilatory buffer:
  a) Direct stimulation of respiratory centers & expiration of excess CO₂

- Renal buffer:
  a) Long-term maintenance

Blood pH & Blood lactate relationship