Section 02:
The Cardiovascular System

Chapter 15 – The Cardiovascular System
Chapter 16 – Cardiovascular Regulation and Integration
Chapter 17 – Functional Capacity of the Cardiovascular System

HPHE 6710 Exercise Physiology II
Dr. Cheatham

Major Functions of Cardiovascular System

• The cardiovascular system has five main functions:
  – Delivery
    • The CV systems delivers oxygen and nutrients to every cell in the body.
  – Removal
    • The CV system helps remove carbon dioxide and waste materials from the body.
  – Transport
    • The CV system transports hormones from endocrine glands to their target receptors.
  – Maintenance
    • The CV system helps maintain such things as pH and temperature.
  – Prevention
    • The CV system helps prevent dehydration and infection by invading organisms.
Review of CV Variables

- Heart Rate (HR)
  - How many times the heart contracts every minute
- Stroke Volume (SV)
  - The amount of blood ejected by the heart during each contraction (systole)
  - End-Diastolic Volume
    - The amount of blood in the left ventricle at the end of diastole (relaxation)
  - End-Systolic Volume
    - The amount of blood in the left ventricle after systole (contraction)
  - Ejection Fraction
    - The proportion of EDV that is ejected during systole
    - $SV = EDV - ESV$ or $Q/HR$

Review of CV Variables

- Cardiac Output (Q)
  - The volume of blood pumped by the heart every minute
    - $Q = HR \times SV$
- Arteriovenous Oxygen Difference (a-\(\text{O}_2\) diff)
  - The oxygen difference between arterial and venous blood
  - Index of $O_2$ extraction by muscles/tissues/cells
- Blood Pressure
  - The amount of force exerted on the walls of the arteries by the blood (SBP and DBP)
    - $MABP = Q \times TPR$
    - $MABP = DBP + (0.33 \times (SBP-DBP))$
Review of CV Variables

• The Fick Equation

\[ \text{VO}_2 = Q \times \text{a-VO}_2 \text{ difference} \]

Chapter 15

The Cardiovascular System
Chapter Objectives

- Identify the different anatomical regions of the heart
- Understand the circulation system of the human body
- Understand the determination of blood pressure and the blood pressure response during exercise
- Understand the circulation within the myocardium

CV System Components

- Heart
  - Pump
- Arteries, Arterioles
  - Distribution system
- Capillaries
  - Exchange vessels
- Veins
  - Collection and return system
CV System Components

- The Heart
  - Myocardium
  - Striated lattice-like network
  - Functions as a unit
The Heart (cont’d)

— Functions of right side
  • Receive blood returning from body
  • Pump blood to lungs for gas exchange

— Functions of left side
  • Receive oxygenated blood from lungs
  • Pump blood into systemic circulation
CV System Components

- The Arterial System
  - Aorta → Arteries → Arterioles
  - Vessels have endothelial tissue, smooth muscle, and connective tissue.
  - Blood Pressure
    - Systolic Blood Pressure
      - Provides an estimate of the work of the heart
      - \[ \text{RPP} = \frac{(HR \times \text{SBP})}{1000} \]
    - Diastolic Blood Pressure
      - Indicates peripheral resistance
    - Mean Arterial Blood Pressure

CV System Components

- The Arterial System (cont’d)
  - Blood Pressure (cont’d)
    - Cardiac Output and Total Peripheral Resistance
      - \[ Q = \frac{\text{MABP}}{\text{TPR}} \]
      - \[ \text{MABP} = Q \times \text{TPR} \]
      - Together, MABP and Q estimate the change in total resistance to blood flow in the transition from rest to exercise
      - Resistance to peripheral blood flow DECREASES dramatically from rest to exercise
        » Increase in Q and an increase in SBP with little change in DBP (also due to vasodilation in active muscle beds)
CV System Components

- Capillaries
  - Microscopic vessels 7 – 10 μm in diameter
  - Contain 6% of total blood volume
  - Walls contain one layer of epithelial cells
  - Skeletal muscles have a dense capillary network.
  - Myocardium has an even denser network.
  - Blood flow in capillaries
    - Pre-capillary sphincters regulate flow.
    - Capillaries open and flow increases during exercise.
The Venous System

- Venules → Veins → vena cava

Venous Return

- One-way valves prevent back flow.
- Veins serve a capacitance role.
  - At rest, ~65% of blood is on the venous side of the system.

CV System Components
Blood Pressure Response to Exercise

• Resistance exercise
  – Straining compresses vessels.
  – Peripheral resistance increases.
  – Blood pressure increases in an attempt to perfuse tissues.

• Steady-Rate (Aerobic Exercise)
  – Systolic pressure increases with increases in workload.
    • There is a linear relationship between workload and systolic BP.
  – Diastolic pressure remains fairly constant.
Blood Pressure Response to Exercise

The Heart’s Blood Supply
Chapter 16

Cardiovascular Regulation and Integration

Chapter Objectives

• Understand the control of the cardiovascular system during rest and exercise
• Understand the electrical activity of the heart
• Understand the cardiac cycle
• Review the nervous system
• Understand control of the cardiovascular system
• Understand how blood flow is increased from rest to exercise
Overview

• The vascular system (heart and blood vessels) demonstrates exceptional capacity for expansion
  – Vessels can conduct a blood volume three to four times the pumping capacity of the heart
• Complex mechanisms continually interact to:
  – Maintain systemic blood pressure
  – Deliver adequate blood flow to tissues

Intrinsic Regulation of HR

• Cardiac muscle has an inherent rhythm.
• The sinoatrial node
  – Would generate a rate ~ 100 BPM
  – Described as pacemaker
The Heart’s Electrical Activity

- Cardiac Cycle
  - Relaxation Period
    - Isovolumetric Relaxation
  - Ventricular Filling
    - Rapid Ventricular Filling
    - Atrial Systole
  - Ventricular Systole
    - Isovolumetric Contraction
    - Rapid Ejection
    - Reduced Ejection

The Cardiac Cycle
Extrinsic Regulation of HR and Circulation

- Autonomic Nervous System Review

Nervous System

- Central Nervous System
  - Brain
  - Spinal Cord

- Peripheral Nervous System
  - Sensory (Afferent)
  - Motor (Efferent)

  Autonomic
  - Sympathetic
  - Parasympathetic

  Somatic

Extrinsic Regulation of HR and Circulation (cont’d)
Extrinsic Regulation of HR and Circulation

- ANS Review (cont’d)
  - ANS Receptors
    - Sympathetic Nervous System
      - Pre-ganglionic = ACH
      - Post-ganglionic = NE, EPI (sometimes ACH)
    - Parasympathetic Nervous System
      - Pre-ganglionic = ACH
      - Post-ganglionic = ACH
    - Two main classifications of receptors
      - Adrenergic
        » Bind NE, EPI
      - Cholinergic
        » Bind ACH

Extrinsic Regulation of HR and Circulation

- Adrenergic Receptors:
  - Alpha (α) adrenergic
    - Alpha 1 (NE>EPI)
      - Works via G-Protein system
      - Mainly excitatory effects like smooth muscle contraction
      - Most prevalent in peripheral vasculature
    - Alpha 2 (EPI>NE)
      - Works via G-Protein system
      - Most prevalent pre-synaptically in the SNS and often results in a down regulation of the sympathetic response
Extrinsic Regulation of HR and Circulation

• Adrenergic Receptors:
  – Beta (β) adrenergic
    • Beta 1 (NE>EPI)
      – Works via G-Protein system
      – Mainly stimulatory cardiac effects (increase HR, contractility)
    • Beta 2 (EPI>NE)
      – Works via G-Protein system
      – More widespread throughout the body
      – Mainly causes relaxation of blood vessels and airways

Extrinsic Regulation of HR and Circulation

• Cholinergic Receptors:
  – Nicotinic
    • Opens ion channels to allow for the influx of sodium or calcium
    • Involved in muscular (somatic) contraction
  – Muscarinic
    • Works via G-Protein system
    • Mediate the majority of the effects of the parasympathetic nervous system
**Extrinsic Regulation of HR and Circulation**

- Sympathetic and Parasympathetic Neural Input
  - Sympathetic Influence
    - Releases the catecholamines epinephrine and norepinephrine
    - Two primary effects at heart:
      - Chronotropic: Increase in HR
      - Inotropic: Increase myocardial contractility
  - Effects on circulation:
    - Adrenergic fibers
    - Primarily causes vasoconstriction of small arteries, arterioles, and pre-capillary sphincters
    - Also veins (venoconstriction)
Extrinsic Regulation of HR and Circulation

• Sympathetic and Parasympathetic Neural Input (cont’d)
  – Parasympathetic Influence
    • Release acetylcholine
    • Primary effect at heart:
      – Decrease in HR
      – No inotropic effect (i.e. no effect on myocardial contractility)

Extrinsic Regulation of HR and Circulation

• Central Command: Input from Higher Centers
  – Coordinates neural activity to regulate flow to match demands
  – Essentially a feed-forward mechanism which coordinates the rapid adjustment of the heart and blood vessels to optimize tissue perfusion and maintain central blood pressure.
  – Effect is mostly due to parasympathetic nervous system withdrawal.
Extrinsic Regulation of HR and Circulation

- Peripheral Input
  - Chemoreceptors
    - Monitor metabolites, blood gases
    - Group IV afferents
  - Mechanoreceptors
    - Monitor movement and pressure
    - Group III afferents (GTO, muscle spindles)
  - Baroreceptors
    - Monitor blood pressure in arteries
    - Role is to help maintain BP or avoid an excessive rise in BP
    - Thought to work via a set-point
Extrinsic Regulation of HR and Circulation

- Baroreceptors (cont’d)
  - Thinking question:
    - So, if baroreceptors are responsible for maintaining BP and work via a set-point, how are we able to increase our BP during exercise?
Extrinsic Regulation of HR and Circulation

Extrinsic Regulation of HR and Circulation

Extrinsic Regulation of HR and Circulation

Extrinsic Regulation of HR and Circulation

Extrinsic Regulation of HR and Circulation

Extrinsic Regulation of HR and Circulation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Activation</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-exercise “anticipatory” response</td>
<td>Activation of central command from the motor cortex and higher areas of the brain causes an increase in sympathetic outflow and reciprocal inhibition of parasympathetic activity.</td>
<td>Acceleration of heart rate; increased myocardial contractility; vasodilation in skeletal and heart muscle (cholinergic fibers); vasoconstriction in other areas, especially skin, gut, spleen, liver, and kidneys (adrenergic fibers); increase in arterial blood pressure.</td>
</tr>
<tr>
<td>Exercise</td>
<td>Parasympathetic withdrawal at onset and during low-intensity exercise; progressive sympathetic stimulation in more intense exercise; reflex feedback from peripheral mechanical and chemical receptors that monitor muscle action; alterations in local metabolic conditions due to hypoxia, lactic acid, pH, PO2, P02, H+K+, Mg2+, and temperature cause autoregulatory vasodilation in active muscle.</td>
<td>Further dilation of muscle vasculature. Concomitant constriction of vasculature in inactive tissues to maintain adequate perfusion pressure throughout arterial system. Action of the muscle pump and visceral vasoconstriction combine to facilitate venous return and maintain central blood volume.</td>
</tr>
<tr>
<td></td>
<td>Continued sympathetic adrenergic outflow in conjunction with epinephrine and norepinephrine from the adrenal medulla.</td>
<td></td>
</tr>
</tbody>
</table>
Distribution of Blood

• Physical Factors Affecting Blood Flow
  – The volume of flow in any vessel relates to:
    • Directly to the pressure gradient between two vessels
    • Inversely to the resistance encountered to fluid flow

Poiseuille’s Equation

\[ F = \frac{(P1 - P2) \pi R^4}{8LN} \]

• Effect of Exercise
  – At the start of exercise
    • Dilation of local arterioles
    • Vessels to non-active tissues constrict
  – Factors within active muscle (during exercise)
    • At rest, only 1 of every 30 – 40 capillaries is open in skeletal muscle.
    • During exercise, capillaries open and increase perfusion and O₂ delivery.
    • Vasodilation mediated by
      – Temp – pH
      – CO₂ – Adenosine
      – NO – K⁺
      – MG⁺
**Nitric Oxide**

- Produced and released by vascular endothelium
- NO spreads through cell membranes to muscle within vessel walls, causing relaxation.
- Net result is vasodilation.
**Distribution of Blood**

- **Hormonal Factors**
  - Adrenal medulla releases
    - Epinephrine (mostly)
    - Norepinephrine
  - Cause vasoconstriction
    - Except in coronary arteries and skeletal muscles
  - Minor role during exercise

**Distribution of Blood**

- A few other things the book barely mentions:
  - Improving venous return
    - Venoconstriction
    - Muscle Pump
    - Respiratory Pump
• So, let’s put this together and explain how blood flow/delivery is augmented from rest to exercise

Chapter 17

Functional Capacity of the Cardiovascular System
Chapter Objectives

- Review the cardiovascular responses to acute exercise
- Understand the factors that influence cardiac performance
- Understand how cardiac output distribution changes from rest to exercise
- Understand how oxygen transport (and utilization) changes from rest to exercise
- Understand the cardiovascular adaptations to endurance-type training

Overview

- In this chapter, we are going to focus on factors that influence cardiac performance (or the functional capacity of the CV system)
- Cardiac performance is best represented by cardiac output
Review – CV Responses During Acute Exercise

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![Graph showing oxygen uptake vs. power output.](image1)

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Review – CV Responses During Acute Exercise

![Graph showing heart rate changes during exercise and recovery.](image2)
Review – CV Responses During Acute Exercise

![Graph showing stroke volume response during acute exercise](image1.png)

Review – CV Responses During Acute Exercise

![Graph showing cardiac output response during acute exercise](image2.png)
**Review – CV Responses During Acute Exercise**

![Graph showing arteriovenous oxygen difference during acute exercise](image)

**Measuring Cardiac Output**

- Direct Fick Method

![Diagram illustrating Fick's method for measuring cardiac output](image)
Measuring Cardiac Output

- Indicator Dilution Method
  - \( Q = \frac{\text{Amount of Dye Injected}}{\text{Ave. Dye Concentration in blood for duration of curve}} \times \text{duration of curve} \)

- CO\(_2\) Rebreathing Method
  - \( Q = \frac{\text{VCO}_2}{\text{v-aCO}_2 \text{ difference}} \times 100 \)

Cardiac Output at Rest

- Untrained individuals
  - Average cardiac output at rest:
    - 5 L/min for males
    - 4 L/min for females
    - Based on a HR of around 70 b/min, SV would be:
      - ~ 70 mL/beat for males
      - ~ 50-60 mL/beat for females
    - Generally, cardiac output and stroke volume are about 25% lower in females than males
**Cardiac Output at Rest**

- Endurance Athletes
  - Characteristics of Q
    - HR ~ 50 BPM
    - SV ~ 100 mL
  - Mechanisms
    - Increased vagal tone w/decreased sympathetic drive
    - Increased blood volume
    - Increased myocardial contractility and compliance of left ventricle
- Thinking question: Why is resting cardiac output not different between untrained and trained individuals?

**Cardiac Output During Exercise**

- Q increases rapidly during transition from rest to exercise.
- Q at max exercise increases up to 4 times.

<table>
<thead>
<tr>
<th></th>
<th>Q</th>
<th>HR</th>
<th>SV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untrained</td>
<td>22 L</td>
<td>195</td>
<td>113 mL</td>
</tr>
<tr>
<td>Trained</td>
<td>35 L</td>
<td>195</td>
<td>179 mL</td>
</tr>
</tbody>
</table>
Cardiac Output During Exercise

TABLE 17.1 * Maximal Values for Oxygen Consumption, Heart Rate, Stroke Volume, and Cardiac Output in Three Groups with Very Low, Normal, and High Aerobic Capacities

<table>
<thead>
<tr>
<th>Group</th>
<th>$\text{VO}_2\text{max}$ (L·min$^{-1}$)</th>
<th>Max Heart Rate (B·min$^{-1}$)</th>
<th>Max Stroke Volume (mL)</th>
<th>Max Cardiac Output (L·min$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral stenosis</td>
<td>1.6</td>
<td>190</td>
<td>50</td>
<td>9.5</td>
</tr>
<tr>
<td>Sedentary</td>
<td>3.2</td>
<td>200</td>
<td>100</td>
<td>20.0</td>
</tr>
<tr>
<td>Athlete</td>
<td>5.2</td>
<td>190</td>
<td>160</td>
<td>30.4</td>
</tr>
</tbody>
</table>

Cardiac Output During Exercise

- Preload (Enhanced Diastolic Filling)
  - Blood volume
  - Venous tone
  - Muscle Pump
  - Respiratory pump
  - Cardiac output
  - Atrial priming

Cardiac Output During Exercise

- Preload (cont’d)
  - Frank-Starling Curve
Cardiac Output During Exercise

• Preload (cont’d)
  – Frank-Starling Curve

Cardiac Output During Exercise

• Afterload
  – The pressure that the heart has to contract against

*Figure 1.* Effects of changes in afterload on Frank-Starling curves. A shift from A to B occurs with increased afterload, and from A to C with decreased afterload.
**Cardiac Output During Exercise**

- Contractility (Greater Systolic Emptying)

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**Cardiac Output During Exercise**

- Contractility (Greater Systolic Emptying) (cont’d)
Cardiac Output During Exercise

- Cardiovascular Drift
  - A progressive decrease in SV and increase in HR during steady-state exercise; Q is maintained

Cardiac Output Distribution

- Distribution of cardiac output during rest
- Distribution of cardiac output during strenuous exercise
Cardiac Output Distribution

Cardiac Output and Oxygen Transport

- Rest
  - 200 mL of O₂ per liter of blood
  - Average Q = 5 L/min
  - Therefore, about 1000 mL (1L) of oxygen is available to the body
  - Typical resting VO₂ = 250-300 mL/min

- Exercise
  - O₂ content of arterial blood the same.
  - But, Q increases dramatically
  - Up to 2.5x increase in oxygen delivery
Cardiac Output and Oxygen Transport

• Oxygen Extraction: The $a-vO_2$ difference
  - $O_2$ consumption increases during exercise.
    - Increases $Q$
    - Increases extraction of $O_2$ by tissues
  - $O_2 = Q \times a-vO_2$ difference
  - At Rest:
    - 20 mL $O_2\cdot dL^{-1}$ arterial blood
    - 15 mL $O_2\cdot dL^{-1}$ venous blood
    - 5 mL $a-vO_2$diff
  - During Exercise:
    - 20 mL $O_2\cdot dL^{-1}$ arterial blood
    - 5 – 15 mL $O_2\cdot dL^{-1}$ venous blood
    - Up to a threefold increase in $O_2$ extraction
**Cardiac Output and Oxygen Transport**

- Factors Affecting the Exercise a-VO₂ Difference
  - Redistribution of flow to active tissues during exercise
  - Increased capillary density due to training increases surface area and O₂ extraction
  - Increased number and size of mitochondria
  - Increased oxidative enzymes
  - Vascular and metabolic improvements

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**Exercise Training and Cardiac Performance**

<table>
<thead>
<tr>
<th>Change in Heart</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Decreased resting and submaximal exercise heart rate (bradycardia)</td>
<td>Increased parasympathetic influence</td>
</tr>
<tr>
<td>* Increased stroke volume</td>
<td>Decreased sympathetic influence</td>
</tr>
<tr>
<td>* Altered electrocardiogram: AV blocks, wandering atrial pacemaker, AV nodal pacemaker rhythm, ST segment elevation, PVo, T-wave inversion</td>
<td>Lower intrinsic heart rate</td>
</tr>
<tr>
<td>* Increased prevalence of third and fourth heart sounds</td>
<td>Increased heart rate (rest and submaximal exercise)</td>
</tr>
<tr>
<td>* Improved calcium release and transport</td>
<td>Increased blood volume</td>
</tr>
<tr>
<td></td>
<td>Increased heart size and volume</td>
</tr>
<tr>
<td></td>
<td>Increased cardiac contractility</td>
</tr>
<tr>
<td></td>
<td>Increased ventricular compliance</td>
</tr>
<tr>
<td></td>
<td>Increased ventricular filling pressure</td>
</tr>
<tr>
<td></td>
<td>Increased parasympathetic influence</td>
</tr>
<tr>
<td></td>
<td>More rapid filling of ventricles</td>
</tr>
<tr>
<td></td>
<td>Prolonged P-R interval on ECG</td>
</tr>
<tr>
<td></td>
<td>Thinner chest walls in athletes</td>
</tr>
<tr>
<td></td>
<td>Increased strength of contraction</td>
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</table>
Exercise Training and Cardiac Performance

**TABLE 16-1:**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Resting</th>
<th>Upright Submaximal</th>
<th>Upright Maximal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats·min⁻¹)</td>
<td>70</td>
<td>63</td>
<td>150</td>
</tr>
<tr>
<td>Stroke volume (ml·beat⁻¹)</td>
<td>72</td>
<td>80</td>
<td>90</td>
</tr>
<tr>
<td>Cardiac output (litters·min⁻¹)</td>
<td>5.0</td>
<td>5.0</td>
<td>13.5</td>
</tr>
<tr>
<td>Stroke volume (ml·beat⁻¹)</td>
<td>72</td>
<td>80</td>
<td>90</td>
</tr>
<tr>
<td>Cardiac output (litters·min⁻¹)</td>
<td>5.0</td>
<td>5.0</td>
<td>13.5</td>
</tr>
<tr>
<td>O₂ uptake (liters·min⁻¹)</td>
<td>0.280</td>
<td>0.280</td>
<td>1.485</td>
</tr>
<tr>
<td>(ml·kg⁻¹·min⁻¹)</td>
<td>3.7</td>
<td>3.7</td>
<td>19.8</td>
</tr>
<tr>
<td>Work load (kg·kg⁻¹·min⁻¹)</td>
<td>—</td>
<td>600</td>
<td>600</td>
</tr>
<tr>
<td>Systemic arterial systolic BP</td>
<td>120</td>
<td>114</td>
<td>156</td>
</tr>
<tr>
<td>Systemic arterial diastolic BP</td>
<td>75</td>
<td>70</td>
<td>82</td>
</tr>
<tr>
<td>Total peripheral resistance (dyne·cm⁻²)</td>
<td>1250</td>
<td>1250</td>
<td>770</td>
</tr>
<tr>
<td>Blood flow (ml·min⁻¹)</td>
<td>280</td>
<td>250</td>
<td>560</td>
</tr>
<tr>
<td>Corr.</td>
<td>750</td>
<td>740</td>
<td>740</td>
</tr>
<tr>
<td>Viscera</td>
<td>2800</td>
<td>2500</td>
<td>900</td>
</tr>
<tr>
<td>Active muscle</td>
<td>600</td>
<td>550</td>
<td>500</td>
</tr>
<tr>
<td>Skirt</td>
<td>800</td>
<td>800</td>
<td>800</td>
</tr>
<tr>
<td>Total</td>
<td>5000</td>
<td>5000</td>
<td>15,900</td>
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</tbody>
</table>

*Symbols:
- / increase
- / decrease
- / no change
- / not applicable
# Exercise Training and Cardiac Performance

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Pre</th>
<th>Post</th>
<th>Pre</th>
<th>Post</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma volume (liters)</td>
<td>2.8</td>
<td>3.0</td>
<td>44.8</td>
<td>36.2</td>
<td>129</td>
<td>145</td>
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<tr>
<td>Red cell mass (liters)</td>
<td>2.3</td>
<td>2.3</td>
<td>30</td>
<td>24</td>
<td>43</td>
<td>52</td>
</tr>
<tr>
<td>Heart volume (ml)</td>
<td>756</td>
<td>786</td>
<td>1.5</td>
<td>1.6</td>
<td>3.0</td>
<td>2.8</td>
</tr>
<tr>
<td>Pulmonary ventilation (liters·min⁻¹)</td>
<td>10.2</td>
<td>10.3</td>
<td>40.6</td>
<td>42.0</td>
<td>48.2</td>
<td>50.6</td>
</tr>
<tr>
<td>Respiratory rate (breathe·min⁻¹)</td>
<td>12</td>
<td>12</td>
<td>129.3</td>
<td>141.2</td>
<td>124.5</td>
<td>122.0</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>850</td>
<td>855</td>
<td>3.0</td>
<td>3.0</td>
<td>11.0</td>
<td>12.4</td>
</tr>
<tr>
<td>Lung diffusion capacity (D_L) (nl at STPD)</td>
<td>34.1</td>
<td>35.2</td>
<td>7.43</td>
<td>7.43</td>
<td>7.43</td>
<td>7.43</td>
</tr>
<tr>
<td>Pulmonary capillary blood volume (ml)</td>
<td>90.1</td>
<td>97.2</td>
<td>7.41</td>
<td>7.43</td>
<td>7.35</td>
<td>7.29</td>
</tr>
<tr>
<td>Vital capacity (liters)</td>
<td>5.1</td>
<td>5.2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Blood lactic acid (mM)</td>
<td>0.7</td>
<td>0.7</td>
<td>3.9</td>
<td>3.0</td>
<td>11.0</td>
<td>12.4</td>
</tr>
<tr>
<td>Blood pH</td>
<td>7.43</td>
<td>7.43</td>
<td>7.43</td>
<td>7.43</td>
<td>7.35</td>
<td>7.29</td>
</tr>
</tbody>
</table>

*Estimated for a healthy man, age 45, weighing 78 kg. Pre = pretraining; post = posttraining; minus (−) sign usually means a decrease in value with training; plus (+) sign usually means an increase in value with training; zero (0) sign usually means no change in value with training.

A MET is equal to the O₂ cost at rest. One MET is generally equal to 3.5 ml·kg⁻¹·min⁻¹ of body weight per minute of O₂ uptake or 1.2 cal·min⁻¹.

STPD is standard temperature (Fahrenheit), pressure 760 mmHg, and dry.

Source: Data courtesy of W. Haskell.