Chapter 9

Cardiovascular System
Function and exercise responses

Cardiac Cycle

- Mechanical changes
  - Pressure and volume/heartbeat
- Cardiac Cycle
  - Repeating pattern of contraction/relaxation
  - Includes all of the events that occur between consecutive heart beats

Systolic interval = 1/3 total cardiac cycle
Diastolic interval = 2/3 total cardiac cycle

- HR = 74 bpm
- Cardiac cycle takes 0.81 s to complete
- What is the length of the systolic and diastolic intervals

The Cardiac Cycle

Systole
- Contraction phase
- Relaxation phase

Diastole

Fig 9.3

Cardiac Cycle

1. Atrial systole
2. Isovolumetric contraction
3. Rapid ventricular ejection
4. Reduced ventricular ejection
5. Isovolumetric relaxation
6. Rapid ventricular filling
7. Reduced ventricular filling

Cardiac Cycle

- Atrial depolarization
  - P wave
- (1) Atrial systole
- Pressure
  - "a" wave
  - Small ↑ venous pressure
- Volume ↑
  - Atria
  - Ventricles (25%)
  - End diastolic volume (EDV)
Cardiac Cycle

(2) isovolumic contraction
- Initial phase
- Insufficient pressure to open aortic and pulmonary valves
- Ventricular volume unchanged
- Pressure builds
  - C wave-av valves bulge

(3) rapid ventricular ejection
- Pressure
  - Sharp increase
  - Aortic valve opens
- Volume
  - Vent decreases

(4) reduced ventricular ejection
- Reduced pressure drop
- Reduced volume drop

(5) isovolumetric relaxation
- Vent pr > atrial pr
- AV valves closed
- Vent pr < atrial pr
- Mitral valve opens

(6) Rapid ventricular filling (diastole)
- Contraction has ended
- T wave-ventricles have repolarized
- All chambers relaxed
- Aortic and pulmonary valves closed
  - Note drop in aortic pressure
  - No blood leaving ventricles
  - Rapid inflow into ventricles (75% filled)
  - Note AV valves are open

(7) Reduced ventricular filling (diastole)
- Ventricular-filling period (VFP)

Useful Equations

Cardiac Output = Heart Rate X Stroke Volume
Stroke Volume = End-Diastolic Volume - End-Systolic Volume
Ejection Fraction = Stroke Volume / End-Diastolic Volume
In the steady state:
Cardiac Output = Venous Return
Cardiac Volumes and Cardiac Output

Representative Values for 70 kg Resting Man

- End - Diastolic Volume: 120 ml
- End - Systolic Volume: 50 ml
- Stroke Volume: 70 ml

Stroke Volume: 70 ml
\[ \times \text{Heart Rate} \quad 70 \text{ min}^{-1} \]

Cardiac Output: 4,900 ml min\(^{-1}\)

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Cardiac Output

The amount of blood pumped by the heart each minute
- Product of heart rate and stroke volume

\[ Q = HR \times SV \]

- Heart rate = number of beats per minute
- Stroke volume = amount of blood ejected in each beat

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Cardiac Output and Venous Return

1. Cardiac output = volume pumped by each ventricle per minute.
2. In steady state conditions, the heart pumps blood at the same rate at which blood enters the heart, i.e., cardiac output = venous return.
3. If cardiac output does not equal venous return, the volume of blood in cardiac chambers will change. Such non-steady state conditions can exist only briefly, but have important effects on cardiac mechanical function.

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Cardiac Output and Venous Return

4. For the normal heart, cardiac output is largely determined by events in the peripheral circulation.
5. In other words, venous return drives cardiac output.
Venous Return

- Factors that regulate venous return during exercise
  - Venoconstriction
    - Reduces volume capacity of veins
  - Muscle pump
    - Muscle contraction compresses veins
  - Respiratory pump
    - Rhythmic pattern of breathing compresses veins

Cardiac Performance (Q)

- How does cardiac function adjust to changes in venous return?
  - Preload or end-diastolic volume (pressure)
    - Is the extent to which the heart chambers are stretched when they fill with blood - SV
  - Contractility
    - Strength of cardiac contraction
  - Heart Rate
    - Speed at which heart beats

Preload

- EDV
  - The amount of blood in the ventricle at the start of systole and
  - Compliance - how easy it is to stretch
- If EDP is increased (EDV)
  - The force of the next contraction increases
  - The stroke volume is increased

Frank-Starling Mechanism

The Frank-Starling mechanism showing that a change in left ventricular end-diastolic volume results in a change in stroke volume, such as occurs when moving from the supine to standing position.
Preload
- Factors that stretch the myocardium
  - Total blood volume
  - Body position
  - Venous tone
  - Atrial contribution to ventricular filling
  - Skeletal muscle pump
  - Intrathoracic pressure
  - Intra-abdominal pressure

Afterload
- Resistance to ventricular emptying
- Increased blood pressure causes LV to pump against higher load
- Smaller ejection fraction
- Increase ESV
- Decreased cardiac performance
- Hypertension – chronic afterload stress
- Not generally of concern during dynamic exercise

Contractility
- A change in cardiac mechanical function (force and velocity of contraction) that is NOT due to a change in preload

Contractility
- Inotropic state
  - Change in the ability of cardiac muscle to generate force without any change in length
  - Ventricular performance at constant conditions of loading and HR
  - Greater or lesser force of contraction at a given end-diastolic volume
  - Contractility changes independent of preload or afterload

Contractility
- Increased contractility – due to altered intracellular calcium
  - Higher contraction strength increases SV at a given EDV – positive inotropic agents
    - norepinephrine
  - Less contraction strength decreases SV at a given EDV – negative inotropic agents
    - Pharmacologic depressants
Contractility

Factors affecting cardiac contractility
- Increasing contractility
  - Sympathetic stimulation - NE
  - Circulating catecholamines – Epi, NE
  - Inotropic agents – digitalis

- Decreasing contractility
  - Loss of myocardium
  - Hypoxia
  - Hypercapnia
  - Acidosis

Regulation of Heart Rate

- Decrease in HR
  - Parasympathetic nervous system
    - Via vagus nerve
    - Slows HR by inhibiting SA node

- Increase in HR
  - Sympathetic nervous system
    - Via cardiac accelerator nerves
    - Increases HR by stimulating SA node

Nervous System Regulation of Heart Rate

Heart Rate

- Rest
  - Controlled by parasympathetic nervous system
  - Chronotropic
    - Increase or decrease in heart rate

- Exercise
  - Primarily controlled by sympathetic nervous system
  - Major determinant of Q (moderate to max)
  - Changes little with training, more with age
Regulation of Stroke Volume

- End-diastolic volume (EDV)
  - Volume of blood in the ventricles at the end of diastole ("preload")

- Average aortic blood pressure
  - Pressure the heart must pump against to eject blood ("afterload")

- Strength of the ventricular contraction
  - "Contractility"

Afterload

- Hypertension
  - High peripheral resistance to blood flow
  - Myocardial hypertrophy
    - Due to increased connective tissue
    - Increases metabolic requirements
    - Increases load on heart muscle
    - Little increase in heart’s functional capacity

- Static-dynamic exercise
  - Shoveling snow
  - Increases intrathoracic pressure
  - Impedes blood flow with near maximal muscle contraction
  - Creates large afterload
  - Results in death of those with CHD, unaccustomed to exercise

- Weight lifting – high muscle tension
  - Increases afterload
  - Decreases SV
  - Increases EDV
  - Stimulates FS mechanism which increases SV at a higher pressure than normal

- Weight lifting
  - Valsalva maneuver-expire against closed glottis
  - Increases intrathoracic pressure
  - Brachial arterial pressures-400 mmHg
Summary of factors affecting CO:

- HR
- Sympathetic stimulation
- Vasopressin
- CVP
- Arterial pressure
- ECP
- Compliance
- Tissue health (O₂, pH, etc.)
- Sympathetic stimulation
- ESV
- SV