Cardiovascular System: Heart

Heart:
- Roughly size of human fist (~ 250 – 350 grams)
- Located in the mediastinum (medial cavity of thorax)
- “Double pump” composed of cardiac muscle

Point of maximum intensity
2/3 of heart mass lies left of mid-sternal line

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figures 18.1 / 18.4
Heart:

**Pericardial sac:** Double-walled sac enclosing the heart

**Fibrous pericardium:**
- Protects heart
- Anchors heart
- Prevents overfilling

**Parietal pericardium**

**Pericardial cavity:**
- Contains serous fluid (friction-free environment)

**Visceral pericardium**

**Pericarditis:**
Inflammation of the pericardial sac

---

Heart:

**Heart layers:**
- Anchors cardiac fibers
- Reinforces heart structures
- Directs electrical signals

**Epicardium:**
- Often infiltrated with fat

**Myocardium:**
- Contains fibrous skeleton

**Endocardium**

---

Cardiovascular System – Heart

Cardiac Tamponade:
Compression of heart due to fluid / blood build up in pericardial cavity

Mariëb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.2

Mariëb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figures 18.2 / 18.3
Heart – Chambers, Vessels & Valves

Atria:
- Receiving chambers
- Small, thin-walled

Ventricles:
- Discharging chambers
- Large, thick-walled

Ligamentum arteriosum:
Remnant of fetal duct between aorta and pulmonary trunk (ductus arteriosus)

Pectinate muscles:
Muscle bundles; assist in atrial contraction

Fossa ovalis:
Shallow depression; remnants of hole between atria in fetal heart

Auricles: ('little ears')
Increase atrial volume

Papillary muscle:
Cone-like muscle; assists in valve closure

Trabeculae carneae:
Muscle ridges; assist in maintaining momentum

Superior vena cava
Returns blood above diaphragm

Inferior vena cava
Returns blood below diaphragm

Aorta
Carries blood to body

Pulmonary veins (4)
Returns blood from lungs

Pulmonary trunk
Carries blood to lungs
Side-by-side pumps of the heart serve separate circuits:

**Pulmonary circuit** (short, low pressure loop):
- Right atrium:
  - O₂-poor; CO₂-rich
- Right ventricle
- Pulmonary valve
- Pulmonary artery
- Lung
- O₂-rich; CO₂-poor
- Left atrium
- Left ventricle

**Systemic circuit** (long, high pressure loop):
- Vena cava
- Right atrium
- Right ventricle
- Tricuspid valve
- Pulmonary valve
- Pulmonary artery
- Lung
- Pulmonary vein
- Left atrium
- Left ventricle
- Mitral valve
- Aorta

In steady state, cardiac output from each ventricle must be equal as well as the venous return to each atrium.

Blood in the atria / ventricles provides little nourishment to heart tissue.

How does the heart itself get nourishment?

**Answer:** Coronary circulation

**Angina pectoris:**
Thoracic pain caused by fleeting deficiency in blood delivery

**Anterior interventricular artery**
- (feeds anterior ventricular walls)

**Posterior interventricular artery**
- (feeds posterior ventricular walls)

**Circumflex artery**
- (feeds left atrium and posterior wall of left ventricle)

**Anterior interventricular artery**
- (feeds interventricular septum and anterior ventricular walls)

**Left coronary artery**

1/200 body's mass; 1/20 body's blood supply

**Myocardial infarction:**
Myocardial cell death resulting from prolonged coronary blockage

**Heart attack**

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.7
Blood in the atria / ventricles provides little nourishment to heart tissue.

How does the heart itself get nourishment?

Answer: Coronary circulation

Coronary circulation delivery limited to when heart is relaxed...

Great cardiac vein

Small cardiac vein

Middle cardiac vein

Coronary sinus

(empties blood back into right atrium)

Blood flows through the heart in a single direction due to the presence of valves.

Cusp:
Flap of endocardium reinforced by connective tissue core

Pulmonary semilunar valve

Right atrioventricular valve (tricuspid valve)

Aortic semilunar valve

Left atrioventricular valve (bicuspid valve) (mitral valve)
Heart – Chambers, Vessels & Valves

Valves open / close based on pressure differences

Atrioventricular valves
(prevent backflow into atria)

Papillary muscle

Semilunar valves
(prevent backflow into ventricles)

(collagen cords)

Chordae tendineae

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figures 18.9 / 18.10

Cardiovascular System – Heart

Heart – Chambers, Vessels & Valves

Pathophysiology:

Valvular Regurgitation:
Valve does not close properly; blood regurgitated

Causes:
• Congenially deformed valve
• Post-inflammatory scarring
• Infective endocarditis
• Rupture of cord / muscle

Valvular stenosis:
Valve flaps become stiff; opening constricted

Causes:
• Congenially deformed valve
• Post-inflammatory scarring
• Calcification of valve

Aortic semilunar valve (pig)

Treatment = Valve replacement
Heart – Chambers, Vessels & Valves

Heart designed to create complex flow patterns
(direct / maintain blood momentum)

1) Chambers arranged in loop pattern
2) Delivery vessels curved
3) Grooves / ridges within chambers

Randall et al. (Animal Physiology, 5th ed.) – Figures 12.4 / 12.10

Muscle Fiber Anatomy

• Striated, branched cells (~85 – 100 µm)
• Single nucleus (sometimes two…)
• Large [mitochondria] (~15x skeletal muscle)
  • High fatigue resistance
• Electrical synapses (intercalated discs)

Contractile cell:

Desmosome

AP

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.11

Functional syncytium:
The entire myocardium behaves as a single coordinated unit

Less elaborate T-tubule system and sarcoplasmic reticulum compared to skeletal muscle
Cardiac Electrophysiology

**Conducting cells:** Cardiac cells specialized to quickly spread action potentials across myocardium
- Weak force generators

**Intrinsic Conduction System:**

**Sinoatrial node:** (SA node)
- Located in right atrial wall
- Initiates action potentials (APs)
  - Pacemaker (~ 80 beats / min)

**Atrioventricular node:** (AV Node)
- Connects atria to ventricles
- Slowed conduction velocity
- Ventricular filling

**Normal sinus rhythm:**
1) AP originates at SA node
2) SA node fires at 60 – 100 beats / min
3) Correct myocardial activation sequence

**Cardiac Electrophysiology**

The concepts applied to cardiac APs are the same concepts as applied to APs in nerves / skeletal muscle

**Review:**
- Membrane potential determined by relative conductances / concentrations of permeable ions
- Ions flow down electrochemical gradient toward equilibrium potential (Nernst equation)
- Membrane potential expressed in mV; inside cell expressed relative to outside
- Resting membrane potential determined primarily by K⁺ ions (leaky K⁺ gates at rest)
- Na⁺ / K⁺ pumps maintain gradients across membranes
- Changes in membrane potential caused by flow of ions into / out of cell
- **Threshold potential** represents the point at which a depolarization even becomes self-sustaining (voltage-gated channels)
Cardiac Electrophysiology

APs of Atria, Ventricles & Purkinje System:

**Phases of the Action Potential:**

**Phase 0 – Upstroke**
- Period of rapid depolarization
  - Na⁺ enters via VG channels (↑ g_{Na})

**Phase 1 – Initial repolarization**
- Brief period of repolarization
  - Na⁺ channels close (↓ g_{Na})
  - K⁺ exits via VG channels (↑ g_{K})
  - Ca²⁺ enters via VG channels (↑ g_{Ca})
  - Steep electrochemical gradient

**Phase 2 – Plateau**
- Stable, depolarized membrane potential
  - K⁺ exits via VG channels (↑ g_{K})
  - Ca²⁺ enters via VG channels (↑ g_{Ca})
- Ca²⁺ entry initiates release of more Ca²⁺ from intracellular stores
  - (excitation-contraction coupling)

\( g_x \) = conductance

VG = voltage-gated

Net current = 0
Cardiac Electrophysiology

APs of Atria, Ventricles & Purkinje System:

Phases of the Action Potential:

Phase 3 – Repolarization
- Period of rapid repolarization
  - Ca²⁺ channels close (↓ g_{Ca})
  - K⁺ exits via VG channels (↑ g_K)

Phase 4 – Resting membrane potential
- Membrane potential stabilizes
  - All VG channels closed
  - K⁺ exits via “leaky” channels
  - Na⁺ / K⁺ pumps restore [gradients]

Changes in RMP (due to [gradient] issues) directly affect responsiveness of heart
- # of VG Na⁺ channels available to respond decreases as RMP becomes more (+)
  - Cardiac dysrhythmia = irregular heartbeat

Refractory Periods:

Absolute refractory period (ARP)
- Na⁺ channels closed (reset at ~ -50 mV)

Relative refractory period (RRP)
- Greater than normal stimulus required to generate AP (some Na⁺ channels recovered)

Supranormal period (SNP)
- Cell is more excitable than normal due
  - Full Na⁺ channel recovery
  - Potential closer to threshold than at rest
Cardiac Electrophysiology

APs of the Sinoatrial Node:

**Pacemaker of the Heart:**
1) Exhibits automaticity (spontaneous AP generation)
2) Unstable resting membrane potential
3) No sustained plateau

**Phase 0 – Upstroke**
- Slower than other cardiac tissue
- $\text{Ca}^{2+}$ enters via VG channels ($\uparrow g_{\text{Ca}}$)

**Phase 1 / Phase 2**
- Absent

**Phase 3 – Repolarization**
- Similar to other cardiac cells

**Phase 4 – Spontaneous depolarization**
- Accounts for automaticity of SA node
- $\text{Na}^+$ enters via VG channels ($\uparrow g_{\text{Na}}$)
  - Open via repolarization event
- Once threshold reached, VG $\text{Ca}^{2+}$ channels open (return to Phase 0)
- Rate of depolarization sets heart rate

Other myocardial cells also have the capacity for spontaneous phase 4 depolarization; these are called **latent pacemakers**

<table>
<thead>
<tr>
<th>Location</th>
<th>Intrinsic firing rate (impulses / min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinoatrial node</td>
<td>70 – 80</td>
</tr>
<tr>
<td>Atrioventricular node</td>
<td>40 – 60</td>
</tr>
<tr>
<td>Bundle of His</td>
<td>40</td>
</tr>
<tr>
<td>Purkinje fibers</td>
<td>15 – 20</td>
</tr>
</tbody>
</table>

**Overdrive suppression:**
Latent pacemakers own capacity to spontaneously depolarize is suppressed by the SA node.

**Ectopic pacemaker:** Latent pacemaker takes over and becomes the pacemaker
1) SA node firing rate decreases (e.g., damage / drug suppression)
2) Intrinsic rate of latent pacemakers increases
3) Blockage in normal conduction pathway (e.g., disease)
Cardiac Electrophysiology

Conduction velocity (speed at which APs propagate in tissues) differs among myocardial tissues.

<table>
<thead>
<tr>
<th>Conduction Velocity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atria</td>
</tr>
<tr>
<td>AV node</td>
</tr>
<tr>
<td>Only connection:</td>
</tr>
<tr>
<td>atria → ventricles</td>
</tr>
<tr>
<td>His-Purkinje</td>
</tr>
<tr>
<td>Ventricle</td>
</tr>
</tbody>
</table>

Costanzo (Physiology, 4th ed.) – Figure 4.14

Electrocardiogram (ECG or EKG): Graphical recording of electrical currents generated and transmitted through heart.

Duration corresponds to atrial conduction rate

Correlates with conduction time through AV node

PR interval

Correlates with the plateau of the ventricular AP

GT interval

P wave
Depolarization of the atria

Q wave

R wave

Repolarization of the atria (hidden)

S wave

QRS wave
Depolarization of the ventricles

T wave
Repolarization of the ventricles

Randall et al. (Animal Physiology, 5th ed.) – Figure12.8
Cardiac Electrophysiology

**Cycle length:**
Time between one R wave and the next
Heart rate = 60 / cycle length (beats / min)

**Junctional Rhythm:**
SA node nonfunctional

**Heart block:**
Poor conduction at AV node

**Fibrillation:**
Out-of-phase contractions

The autonomic nervous system can directly affect the heart rate; these effects are called **chronotropic effects**

**Positive chronotropic effects:**
(increase heart rate)
- Under sympathetic control
  - NE
    - β₁ receptors
    - Leads to ↑ *g* Na⁺ cells reach threshold more rapidly
    - Pharmacology: β-blockers (e.g., propranolol)

**Negative chronotropic effects:**
(decrease heart rate)
- Under parasympathetic control
  - ACh
    - Muscarinic receptors
    - Leads to ↓ *g* Na⁺ cells reach threshold less rapidly
    - Leads to ↑ *g* K⁺ cells hyperpolarized during repolarization stage (further from threshold)

Recall: spontaneous depolarization = VG Na⁺ channels

Costanzo (Physiology, 4th ed.) – Figure 4.16
Cardiac Electrophysiology

The autonomic nervous system can also directly conduction velocity at the AV node; these effects are called dromotropic effects.

**Positive dromotropic effects:**
- (increase conduction velocity)
  - Under sympathetic control
    - Leads to ↑ \(g_{Ca}\) and ↓ \(g_{K}\); cells depolarize more rapidly following threshold

**Negative dromotropic effects:**
- (decrease conduction velocity)
  - Under parasympathetic control
    - Leads to ↓ \(g_{Ca}\) and ↑ \(g_{K}\); cells depolarize more slowly following threshold

Cardiac Muscle Contraction

The basic contractile machinery between cardiac and smooth muscle is similar.

Contraction occurs according to sliding filament model.
Cardiac Muscle Contraction

Excitation-contraction coupling translates the action potential into the production of tension.

Cardiac AP initiated in membrane
• Spreads to cell interior via T-tubules
• Triggers L-type Ca²⁺ channels in membrane

Ca²⁺ enters cell (plateau phase)
• Triggers Ca²⁺-induced Ca²⁺ release from SR

Ca²⁺ floods cytoplasm

Cross-bridging cycling initiated
• Ca²⁺ binds to troponin C; cross-bridging occurs

Mechanisms for changing tension production:
1) ↑ Ca²⁺ in SR → ↑ Ca²⁺ release to cytoplasm
2) ↑ Ca²⁺ inward current → ↑ Ca²⁺ in cytoplasm

The magnitude of the tension developed is proportional to the intracellular [Ca²⁺].

Relaxation

Tension

Cardiovascular System – Heart

Inotropism:
Intrinsic ability of myocardial cells to develop force at a given length

Positive inotropic effects: (increase contractility)
• Under sympathetic control

Mechanisms of action:
1) Phosphorylation of Ca²⁺ channels in sarcolemma
   • ↑ Ca²⁺ enters during plateau / released from SR
2) Phosphorylation of phospholamban (regulates Ca²⁺ ATPase activity)
   • ↑ uptake / storage of Ca²⁺ in SR
   • Faster relaxation time
   • Increased peak tension during subsequent ‘beats’

Positive inotropic effects, also triggered by circulating catecholamines

β₁ receptors

Cardiac tissue

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.12

Similar to Costanzo (Physiology, 4th ed.) – Figure 4.18
Cardiac Muscle Contraction

The autonomic nervous system can directly affect heart contractility; these effects are called **inotropic effects**

**Positive inotropic effects:** (increase contractility)

Cardiac glycosides are a class of drugs that act as positive inotropic agents

Used extensively for the treatment of congestive heart failure

Costanzo (Physiology, 4th ed.) – Figure 4.20

Cardiac glycosides inhibit Na\(^+\)-K\(^+\) ATPase

1)  Intracellular [Na\(^+\)] increases

2)  Change in Na\(^+\) gradient slows down Ca\(^{2+}\)-Na\(^+\) exchanger

3)  Intracellular [Ca\(^{2+}\)] increases

4)  Intracellular [Ca\(^{2+}\)] increases

5)  \[Ca^{2+}\] = \(\uparrow\) tension development

Cardiac Muscle Contraction

The autonomic nervous system can directly affect heart contractility; these effects are called **inotropic effects**

**Negative inotropic effects:** (decrease contractility)

- Under parasympathetic control

  - ACh decreases inward Ca\(^{2+}\) current during plateau
  - ACh increases outward K\(^+\) current (shorten plateau phase)

  Both \(\downarrow\) Ca\(^{2+}\) entering cell and thus the amount of Ca\(^{2+}\) available for tension development

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.12
Cardiovascular System – Heart

Cardiac Muscle Contraction

Changes in heart rate also produce changes in cardiac contractility

Example:

Increase in heart rate = Increase in cardiac contractility

1) ↑ heart rate = ↑ APs per unit time = ↑ total amount of Ca\(^{2+}\) entering cell per unit time

AND

2) ↑ Ca\(^{2+}\) entering cell per unit time = ↑ accumulation of Ca\(^{2+}\) in SR for future release

Positive staircase effect

As heart rate increases, the tension developed on each beat increases stepwise to a maximal value

* Sympathetic input will enhance response

Costanzo (Physiology, 4th ed.) – Figure 4.19

Cardiovascular System – Heart

Cardiac Muscle Contraction

The maximum tension that can be developed by a myocardial cell depends on its resting length (similar to skeletal muscle)

Sarcomere length of ~2.2 μm = \(L_{\text{max}}\) for cardiac muscle

Overlap of actin / myosin optimal for cross-bridge formation

Myocardium cells maintain a ‘working length’ of ~1.9 μm

Additional Length-dependent Mechanisms:

1) Increasing muscle length increases Ca\(^{2+}\)-sensitivity of troponin C
2) Increasing muscle length increases Ca\(^{2+}\) release from SR

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 9.22
Cardiac Muscle Contraction

Cardiac output = Heart rate \times \text{Stroke volume}

(ml / min) \times (beats / min) = (ml / beat)

1) Cardiac Output: Total volume of blood ejected by each ventricle per unit time (usually one minute)

Cardiac output = Heart rate \times \text{Stroke volume}

Heart rate = 75 \text{ beats / min}
End diastolic volume = 140 \text{ ml}
End systolic volume = 70 \text{ ml}

Calculate:
Stroke volume: 70 \text{ ml}
Ejection fraction: 0.50
Cardiac output: 5250 \text{ ml / min}

2) Stroke Volume: Volume of blood ejected by each ventricle per heart beat

\text{Stroke volume} = \text{End diastolic volume} - \text{End systolic volume}

(ml) - (ml) = (ml)

3) Ejection Fraction: Fraction of the end diastolic volume ejected in each stroke volume

\text{Ejection fraction} = \frac{\text{Stroke volume (ml)}}{\text{End diastolic volume (ml)}}

Frank-Starling Law of the Heart:
The volume of blood ejected by the ventricle depends on the volume present in the ventricle at the end of diastole

Sarcomere length = \sim 1.9 \mu m
Sarcomere length = \sim 2.2 \mu m

Preload:
The resting length from which cardiac muscle contracts

* Cardiac muscle normally 'held' on the ascending limb of the length-tension curve; much 'stiffer' than skeletal muscle

Costanzo (Physiology, 4th ed.) – Figure 4.21
Cardiovascular System – Heart

Cardiac Muscle Contraction

**Frank-Starling Law of the Heart:**
The volume of blood ejected by the ventricle depends on the volume present in the ventricle at the end of diastole.

The relationship ensures that the volume the heart ejects in systole equals the volume it receives in venous return.

- **Positive inotropic effect** = \( \uparrow \) ejection fraction
- **Negative inotropic effect** = \( \downarrow \) ejection fraction

Cardiovascular System – Heart

**A ventricular pressure-volume loop allows for the function of a ventricle to be observed for a single heart beat.**

**Isovolumetric contraction (1 → 2):**
- Ventricle activates (systole)
- No change in blood volume
- \( P_{aorta} > P_{ventricle} > P_{atrium} \)
- Aortic / mitral valves closed

**Ventricular ejection (2 → 3):**
- Aortic valve opens
- \( P_{ventricle} > P_{aorta} \)
- Blood rapidly ejected (\( \downarrow \) volume)

**Systolic pressure-volume curve**

Atrium 'tops off' blood in ventricle
Ventricle relaxed (late diastole)
Cardiac Muscle Contraction

A ventricular pressure-volume loop allows for the function of a ventricle to be observed for a single heart beat.

**Isovolumetric relaxation (3 → 4)**
- Ventricles relaxes (early diastole)
  - no change
  - in blood volume
  - ↓ pressure

**Ventricular filling (4 → 1)**
- Mitral valve opens
- Ventricle refills
- Aortic / mitral valves closed
- \( P_{aorta} > P_{ven} \)
- \( P_{ven} > P_{atrium} \)
- Slight pressure increase due to passive filling of compliant ventricle

**Increased preload**
- Increased end diastolic volume
- Increased stroke volume
  (Frank-Starling Law)

**Increased afterload**
- Pressure in the vessel leaving the heart (e.g., aorta) that must be overcome to eject blood
  - Increased internal pressure
  - Decreased stroke volume

**Increased contractility**
- Increased tension / pressure
- Decreased end systolic volume
- Increased stroke volume
Cardiovascular System – Heart

Cardiac Muscle Contraction

The stroke work is defined as the work the heart performs on each beat:

\[ \text{Work} = \text{force} \times \text{distance} \]

Cardiac minute work:
Work performed by the heart during a unit time (e.g., minute)

\[ \begin{align*}
\text{Force} &= \text{aortic pressure} \\
\text{Distance} &= \text{cardiac output}
\end{align*} \]

Increases in cardiac output or increases in aortic pressure will increase work of the heart.

Cardiac Muscle Contraction

The myocardial O\textsubscript{2} consumption rate correlates directly with the cardiac minute work:

\[ \uparrow \text{cardiac minute work} = \uparrow \text{O}_2 \text{ consumption} \]

However, the largest percentage of O\textsubscript{2} consumption is for pressure work.

Aortic stenosis results in greatly increased O\textsubscript{2} consumption, even though cardiac output reduced.

Mean aortic pressure = 100 mm Hg
Mean pulmonary pressure = 15 mm Hg

Thus,

Law of Laplace:
In a sphere (e.g., heart), pressure correlates directly with tension and wall thickness and correlates inversely with radius:

\[ P = \frac{2HT}{R} \]

What are the ramifications if a person exhibits systemic hypertension?
Recall:
Cardiac output equals the total volume of blood ejected by a ventricle per unit time

The cardiac output can also be measured using the Fick principle (conservation of mass)

In the steady state, the rate of \( O_2 \) consumed by the body must equal the amount of \( O_2 \) leaving the lungs (pulmonary veins) minus the amount of \( O_2 \) returning to the lungs (pulmonary artery)

\[
O_2 \text{ consumption} = CO_{\text{left ventricle}} \times [O_2]_{\text{pulmonary vein}} - CO_{\text{right ventricle}} \times [O_2]_{\text{pulmonary artery}}
\]

Solve for cardiac output:

\[
\text{Cardiac Output} = \frac{O_2 \text{ consumption}}{[O_2]_{\text{pulmonary vein}} - [O_2]_{\text{pulmonary artery}}}
\]

A man has a resting \( O_2 \) consumption of 250 mL \( O_2 \)/min, a femoral arterial \( O_2 \) content of 0.20 mL \( O_2 \)/mL blood, and a pulmonary arterial \( O_2 \) content of 0.15 mL \( O_2 \)/mL blood.

What is his cardiac output?

\[
\text{Cardiac Output} = \frac{250 \text{ mL } O_2 / \text{min}}{0.20 \text{ mL } O_2 / \text{mL blood} - 0.15 \text{ mL } O_2 / \text{mL blood}}
\]

\[
\text{Cardiac Output} = \frac{250 \text{ mL } O_2 / \text{min}}{0.05 \text{ mL } O_2 / \text{mL blood}} = 5000 \text{ mL } / \text{min}
\]
Mechanical / Electrical Overview:

Cardiac Cycle: Mechanical and electrical events during single heart beat

**Phases of the cardiac cycle:**

**Atrial Systole:** (A)
- Preceded by P wave on ECG
- Increased tension in left atrium
- Ventricular volume / pressure increases

**Isovolumetric Ventricular Contraction:** (B)
- Begins during QRS wave on ECG
- Ventricular pressure increases (systole)
- Mitral valve closes (1st heart sound – ’Lub’)
- NO VOLUME CHANGE

**Rapid Ventricular Ejection:** (C)
- Aortic valve opens ($P_{aorta} > P_{ventricle}$)
- Majority of stroke volume ejected
- Aorta pressure increases
- Atrial filling begins

**Reduced Ventricular Ejection:** (D)
- Ventricles begin to repolarize (start of T wave)
- Ventricular / atrial pressure falls (blood still moving out)
- Atrium continues to fill (pressure rising)

**Isovolumic Ventricular Relaxation:** (E)
- Ventricles fully repolarize (T wave complete)
- Left ventricular pressure drops rapidly (diastole)
- Aortic valve closes (2nd heart sound – ’Dub’)
- NO VOLUME CHANGE

**Rapid Ventricular Filling:** (F)
- Mitral valve opens ($P_{atrium} > P_{ventricle}$)
- Ventricular volume increases rapidly
- Little change in ventricular pressure (compliance)
- Aortic pressure decreases (blood carried away)

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.20
Mechanical / Electrical Overview:

**Cardiac Cycle**: Mechanical and electrical events during single heart beat

**Phases of the cardiac cycle**:

Reduced Ventricular filling: (G)
- Longest phase of cardiac cycle
- Final portion of ventricular filling

Increase in heart rate reduces G phase interval; if heart rate too high, ventricular filling compromised

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.20