Cardiovascular Physiology
Circulatory System
Pressure Gradient

Diagram showing the mean systemic blood pressure (mm Hg) across different blood vessels: Aorta, Arteries, Arterioles, Capillaries, Venules, Veins, and Venae cavae. The graph illustrates the decrease in pressure from the aorta to the venae cavae.
Flow Rate??

Flow rate (Q) = 12 cm³/min

\[ \text{Flow rate (Q)} = \frac{12 \text{ cm}^3}{\text{min}} \]

The narrower the vessel, the faster the velocity of flow.

\[
\begin{array}{c|c}
\text{Velocity (v)} & \text{Flow rate (Q)} \\
\hline
\text{At point X} & \frac{12 \text{ cm}^3/\text{min}}{1 \text{ cm}^2} \quad \text{At point Y} & \frac{12 \text{ cm}^3/\text{min}}{12 \text{ cm}^2} \\
\text{v = 12 cm/min} & \text{v = 1 cm/min} \\
\end{array}
\]
Anatomy

(a) The heart lies in the center of the thorax.

(b) The heart is on the ventral side of the thoracic cavity, sandwiched between the lungs.
Vessels that carry well-oxygenated blood are red; those with less well-oxygenated blood are blue.
The heart is encased within a membranous fluid-filled sac, the pericardium.

The ventricles occupy the bulk of the heart. The arteries and veins all attach to the base of the heart.
One-way flow through the heart is ensured by two sets of valves.

Myocardial muscle cells are branched, have a single nucleus, and are attached to each other by specialized junctions known as intercalated disks.
During ventricular contraction, the **AV valves** remain closed to prevent blood flow backward into the atria.

The **semilunar valves** prevent blood that has entered the arteries from flowing back into the ventricles during ventricular relaxation.
Cardiac Muscle

(a) The spiral arrangement of ventricular muscle allows ventricular contraction to squeeze the blood upward from the apex of the heart.

(b) Intercalated disks contain desmosomes that transfer force from cell to cell and gap junctions that allow electrical signals to pass rapidly from cell to cell.

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EC Coupling

1. Action potential enters from adjacent cell.
2. Voltage-gated $\text{Ca}^{2+}$ channels open. $\text{Ca}^{2+}$ enters cell.
3. $\text{Ca}^{2+}$ induces $\text{Ca}^{2+}$ release through ryanodine receptor-channels (RyR).
4. Local release causes $\text{Ca}^{2+}$ spark.
5. Summed $\text{Ca}^{2+}$ sparks create a $\text{Ca}^{2+}$ signal.
6. $\text{Ca}^{2+}$ ions bind to troponin to initiate contraction.
7. Relaxation occurs when $\text{Ca}^{2+}$ unbinds from troponin.
8. $\text{Ca}^{2+}$ is pumped back into the sarcoplasmic reticulum for storage.
9. $\text{Ca}^{2+}$ is exchanged with $\text{Na}^+$. 
10. $\text{Na}^+$ gradient is maintained by the $\text{Na}^+-$K$^+$-ATPase.
Modulation of Contraction

Epinephrine from adrenal medulla
Norepinephrine from sympathetic neurons
bind to
β₁ receptors on myocardial contractile cell
that activate
CAMP second messenger system
resulting in phosphorylation of

Voltage-gated Ca²⁺ channels
Open time increases
↑ Ca²⁺ entry from ECF

Phospholamban
↑ Ca²⁺-ATPase activity of sarcoplasmic reticulum (SR)

↑ Ca²⁺ stores in SR
Ca²⁺ released through Ca²⁺-induced Ca²⁺ release
More forceful contraction

Ca²⁺ removed from cytosol faster
Time of Ca-troponin binding shorter
Shorter duration of contraction

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**Action Potentials**

![Diagram showing the phases of an action potential](image)

### Table: Membrane Channels

<table>
<thead>
<tr>
<th>Phase</th>
<th>Membrane channels</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Na⁺ channels open</td>
</tr>
<tr>
<td>1</td>
<td>Na⁺ channels close</td>
</tr>
<tr>
<td>2</td>
<td>Ca²⁺ channels open; fast K⁺ channels close</td>
</tr>
<tr>
<td>3</td>
<td>Ca²⁺ channels close; slow K⁺ channels open</td>
</tr>
<tr>
<td>4</td>
<td>Resting potential</td>
</tr>
</tbody>
</table>

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Skeletal vs Cardiac

(a) Skeletal muscle fast-twitch fiber: The refractory period (yellow) is very short compared with the amount of time required for the development of tension.

(b) Skeletal muscles that are stimulated repeatedly will exhibit summation and tetanus (action potentials not shown).

(c) Cardiac muscle fiber: The refractory period lasts almost as long as the entire muscle twitch.

(d) Long refractory period in a cardiac muscle prevents tetanus.
Autorhythmic Cells

(a) The pacemaker potential gradually becomes less negative until it reaches threshold, triggering an action potential.

(b) Ion movements during an action and pacemaker potential

(c) State of various ion channels

Membrane potential (mV)

Time

Pacemaker potential
Action potential

Threshold

Net Na⁺ in
Ca²⁺ in
K⁺ out

Lots of Ca²⁺ channels open

Some Ca²⁺ channels open, If channels close

If channels open

Ca²⁺ channels close, K⁺ channels open

K⁺ channels close
Modulation by Nervous Sys.

(a) Sympathetic stimulation and epinephrine depolarize the autorhythmic cell and speed up the depolarization rate, increasing the heart rate.

(b) Parasympathetic stimulation hyperpolarizes the membrane potential of the autorhythmic cell and slows depolarization, slowing down the heart rate.
Depolarizations of autorhythmic cells rapidly spread to adjacent contractile cells through gap junctions.
Electrical Conduction

(a) The conducting system of the heart

(b) SA node depolarizes.

(c) Electrical activity goes rapidly to AV node via internodal pathways.

(d) Depolarization spreads more slowly across atria. Conduction slows through AV node.

(e) Depolarization moves rapidly through ventricular conducting system to the apex of the heart.

(f) Depolarization wave spreads upward from the apex.
Einthoven Triangle

Electrodes are attached to the skin surface.

A lead consists of two electrodes, one positive and one negative.
Leads

• 1. Left arm (+); Right arm (-); Left leg (gr)**
• 2. Left arm (gr); Right arm (-); Left leg (+)
• 3. Left arm (-); Right arm (gr); Left leg (+)
Electrical events of the cardiac cycle

- **P wave**: atrial depolarization
- **QO or PR segment**: conduction through AV node and A-V bundle
- **T wave**: ventricular repolarization
- **ST segment**: ventricles contract
- **R wave**: repolarization
- **S wave**: atria contract
ECG v. Myocardial AP

(a) The electrocardiogram represents the summed electrical activity of all cells recorded from the surface of the body.

(b) The ventricular action potential is recorded from a single cell using an intracellular electrode. Notice that the voltage change is much greater when recorded intracellularly.
Mechanical Events

1. Late diastole—both sets of chambers are relaxed and ventricles fill passively.

5. Isovolumic ventricular relaxation—as ventricles relax, pressure in ventricles falls, blood flows back into cups of semilunar valves and snaps them closed.

2. Atrial systole—atrial contraction forces a small amount of additional blood into ventricles.

4. Ventricular ejection—as ventricular pressure rises and exceeds pressure in the arteries, the semilunar valves open and blood is ejected.

3. Isovolumic ventricular contraction—first phase of ventricular contraction pushes AV valves closed but does not create enough pressure to open semilunar valves.
Cardiac Output

• $\text{EDV-ESV} = \text{Stroke Volume}$
  (End Diastolic Volume-End Systolic Volume)

• Cardiac Output $= \text{HR} \times \text{Stroke Volume}$
  (Heart Rate)
Reflex Control

Cardiovascular control center in medulla oblongata

- Sympathetic neurons (NE)
  - $\beta_1$ receptors of autorhythmic cells
    - $\uparrow$ Na$^+$ and Ca$^{2+}$ influx
    - $\uparrow$ Rate of depolarization
    - $\uparrow$ Heart rate

- Parasympathetic neurons (ACh)
  - Muscarinic receptors of autorhythmic cells
    - $\uparrow$ K$^+$ efflux; $\uparrow$ Ca$^{2+}$ influx
    - Hyperpolarizes cell and $\downarrow$ rate of depolarization
    - $\downarrow$ Heart rate

KEY
- Integrating center
- Efferent path
- Effector
- Tissue response

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Cardiac Output Factors

Cardiac Output

is a function of

Heart rate
determined by

Rate of depolarization in autorhythmic cells

is slowed by
Parasympathetic innervation

is made faster by
Sympathetic innervation
Epinephrine from adrenal medulla

Stroke volume
determined by

Force of contraction in ventricular myocardium

is influenced by
Contractility
Length-tension relationship of muscle fibers
which varies with
Venous return
aided by
Skeletal muscle pump
Respiratory pump

Which step(s) is (are) controlled by ACh? By norepinephrine? Which tissue(s) has (have) muscarinic receptors? β₁ receptors?
Elastic Recoil in Arteries

(a) Ventricular contraction

1. Ventricle contracts.
2. Semilunar valve opens.
3. Aorta and arteries expand and store pressure in elastic walls.

(b) Ventricular relaxation

1. Isovolumic ventricular relaxation
2. Semilunar valve shuts.
3. Elastic recoil of arteries sends blood forward into rest of circulatory system.

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Review of Pressures

- Systolic pressure
- Diastolic pressure
- Mean pressure
- Pulse pressure

Pressure (mm Hg)

Left ventricle, Arteries, Arterioles, Capillaries, Venules, veins, Right atrium

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Blood Pressure Cuff

(a) Cuff pressure > 120 mm Hg

When the cuff is inflated so that it stops arterial blood flow, no sound can be heard through a stethoscope placed over the brachial artery distal to the cuff.

(b) Cuff pressure between 80 and 120 mm Hg

Korotkoff sounds are created by pulsatile blood flow through the compressed artery.

(c) Cuff pressure < 80 mm Hg

Blood flow is silent when the artery is no longer compressed.
Physiological Controls of BP

**KEY**
- Stimulus
- Receptor
- Afferent pathway
- Integrating center
- Efferent pathway
- Effector
- Tissue response
- Systemic response

**Blood pressure**

1. **Firing of baroreceptors in carotid arteries and aorta**
2. **Sensory neurons**
3. **Cardiovascular control center in medulla oblongata**

**Sympathetic output**
- less NE released
- α receptor
- Arteriolar smooth muscle
- Vasodilation
- **Peripheral resistance**

**Parasympathetic output**
- more ACh on muscarinic receptor
- β₁ receptor
- Ventricular myocardium
- Force of contraction
- **Cardiac output**

- SA node
- Heart rate
- **Blood pressure**

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MAP

- Mean Arterial Pressure
- MAP = Diastolic P + [Systolic Pressure - Diastolic Pressure](1/3)