NOTE: Presentations extensively modified for use in MCB 244 & 246 at the University of Illinois by Drs. Kwast & Brown (2015-2016)

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Chapter 20 Learning Objectives

1. Describe the anatomy of the heart and vasculature including major blood vessels, chambers, and valves.

2. Describe excitation-contraction coupling in cardiac tissue including electrical and ionic events.

3. Describe the physical events during the cardiac cycle including atrial and ventricular systole and diastole.

4. Define cardiac output, describe factors that affect stroke volume, heart rate and contractility and how these change during physical activity.
Chapter 20 Introduction to Cardiovascular System

Organization:

**Pulmonary circuit:**
right ventricle → lungs → left atrium

**Systemic circuit:**
left ventricle → body → right atrium

**Arteries** = away from heart
**Veins** = toward heart

**Capillaries** = exchange vessels in between (gases, nutrients, wastes)

Figure 20–1 An Overview of the Cardiovascular System.
Heart Anatomy: Thoracic Location

- Heart is fist sized, < 1 lb., beats, ~100,000 times/day moving 8,000 liters of blood per day

- Lies left of midline, between 2nd rib and 5th intercostal space, posterior to sternum, in the pericardial cavity of the mediastinum (the region between the two pleural cavities, which also contains the great vessels, thymus, esophagus & trachea).
Heart Anatomy: Thoracic Location

The heart is surrounded by the **pericardial sac**, which consists of dense network of collagen fibers that stabilizes the position of the heart (and major vessels) within the **mediastinum**.

Figure 20–2b: The Location of the Heart in the Thoracic Cavity:

A superior view of the organs in the mediastinum: portions of the lungs have been removed to reveal blood vessels and airways. The heart is situated in the anterior part of the mediastinum, immediately posterior to the sternum.
Heart Anatomy: Pericardium

- The serous membrane lines the pericardium and is comprised of the **visceral and parietal pericardia**. Pericardial fluid (15 - 50 ml) lies between the layers, acting as a lubricant to reduce friction during contractions.

- **Pericarditis** = inflammation of pericardium, usually due to infection

- **Cardiac tamponade** = buildup of fluid in pericardial space that restricts heart movement
Superficial Anatomy of the Heart

- The 2 atria:
  - Superior, thin-walled
  - Smooth posterior walls internally, pectinate muscles (ridges) anteriorly
  - Each has an expandable flap called an auricle (atrial appendage)

- The 2 ventricles:
  - Inferior, thick-walled
  - Lined with trabeculae carneae (muscular ridges)
  - Left ventricle 3x thicker, 5x more friction than right
  - Lt – round; rt. - crescent

- Coronary sulcus: divides atria & ventricles
- Interventricular sulci (anterior & posterior) separate lt. & rt. ventricles

Figure 20–3b
Superficial Anatomy of the Heart

Figure 20–3a The Superficial Anatomy of the Heart
Anatomy of the Heart Wall

- **Epicardium** (thin, outer layer)
  - *Visceral pericardium*: serous membrane with loose CT

- **Myocardium** (thick, middle layer)
  - Concentric layers of cardiac muscle tissue with CT attached to vessels and nerves

- **Endocardium** (thin, inner layer)
  - Simple squamous epithelium
  - Continuous with blood vessel endothelium

- Atrial myocardium wraps around great vessels; ventricular divided in 2
Anatomy of Cardiac Muscle

- Muscle cells = cardio[myo]cytes
  - actin & myosin sliding filaments; small w/ single nucleus
  - rich in mitochondria
  - Cells connected by **intercalated discs** = **desmosomes + gap junctions**
- Propagate action potential and convey timing & force of contraction
- Contractions are all or none; longer contraction phase than skeletal
Comparison of Cardiac and Skeletal Muscle Characteristics

<table>
<thead>
<tr>
<th>Feature</th>
<th>Cardiac Muscle Cells</th>
<th>Skeletal Muscle Fibers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Size</td>
<td>10–20 ( \mu m \times 50–100 \mu m )</td>
<td>100 ( \mu m \times ) up to 40 cm</td>
</tr>
<tr>
<td>Nuclei</td>
<td>Typically 1 (rarely 2–5)</td>
<td>Multiple (hundreds)</td>
</tr>
<tr>
<td>Contractile proteins</td>
<td>Sarcomeres along myofibrils</td>
<td>Sarcomeres along myofibrils</td>
</tr>
<tr>
<td>Internal membranes</td>
<td>Short T tubules; no triads formed with sarcoplasmic reticulum</td>
<td>Long T tubules form triads with cisternae of the sarcoplasmic reticulum</td>
</tr>
<tr>
<td>Mitochondria</td>
<td>Abundant (25% of cell volume)</td>
<td>Much less abundant</td>
</tr>
<tr>
<td>Inclusions</td>
<td>Myoglobin, lipids, glycogen</td>
<td>Little myoglobin, few lipids, but extensive glycogen reserves</td>
</tr>
<tr>
<td>Blood supply</td>
<td>Very extensive</td>
<td>More extensive than in most connective tissues, but sparse compared with supply to cardiac muscle cells</td>
</tr>
<tr>
<td>Metabolism (resting)</td>
<td>Not applicable</td>
<td>Aerobic, primarily lipid-based</td>
</tr>
<tr>
<td>Metabolism (active)</td>
<td>Aerobic, primarily using lipids and carbohydrates</td>
<td>Anaerobic, through breakdown of glycogen reserves</td>
</tr>
<tr>
<td>Contractions</td>
<td>Twitches with brief relaxation periods; long refractory period prevents tetanic contractions</td>
<td>Usually sustained contractions</td>
</tr>
<tr>
<td>Stimulus for contraction</td>
<td>Autorhythmicity of pacemaker cells generates action potentials</td>
<td>Activity of somatic motor neuron generates action potentials in sarcolemma</td>
</tr>
<tr>
<td>Trigger for contraction</td>
<td>Calcium entry from the ECF and calcium release from the sarcoplasmic reticulum</td>
<td>Calcium release from the sarcoplasmic reticulum</td>
</tr>
<tr>
<td>Intercellular connections</td>
<td>Branching network with plasma membranes locked together at intercalated discs; connective tissue fibers tie adjacent layers together</td>
<td>Adjacent fibers tied together by connective tissue fibers</td>
</tr>
</tbody>
</table>
Internal Anatomy of the Heart: Septa & Atrioventricular Valves

- **Interatrial septum**: separates atria
- **Interventricular septum**: separates ventricles
- **Atrioventricular (AV) valves**:
  - Connect rt. atrium to rt. ventricle (*tricuspid*) and lt. atrium to lt. ventricle (*bicuspidd or mitral*)
  - Permit one-way blood flow: atria → ventricles
  - Cusps attached to *chordae tendineae* from *papillary muscles* on ventricle wall
  - Papillary muscles prevent cusps from swinging into atria; during ventricular contraction pressure closes valves

Figure 20–6a&b
Internal Anatomy of the Heart: Right Atrium

- **Superior vena cava**
  - Receives blood from head, neck, upper limbs, & chest

- **Inferior vena cava**
  - Receives blood from trunk, viscera, and lower limbs

- **Coronary sinus**
  - Cardiac veins return blood to coronary sinus, which opens into rt. atrium

- **Foramen ovale**
  - Before birth, is an opening through interatrial septum
  - Connects the two atria (~25% flow); seals off at birth, forming **fossa ovalis**

- **The ductus arteriosus** (not shown) is the predominate pulmonary bypass in the fetus (connects the pulmonary trunk to the aorta); closes at birth leaving the **ligamentum arteriosum** (shown above)
  - The ductus arteriosus and foramen ovale reduce pulmonary blood flow by 92 – 95%

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Figure 20–6a

A diagrammatic frontal section through the heart, showing major landmarks and the path of blood flow (marked by arrows) through the atria, ventricles, and associated vessels.
Internal Anatomy of the Heart: Right Ventricle

- Free edges attach to **chordae tendineae** from **papillary muscles** of ventricle
- Right atrioventricular (AV) Valve (**tricuspid**)
  - Opening between rt. atrium & rt. ventricle
- **Trabeculae carneae**
  - Muscular ridges on internal surface of right (and left) ventricle
  - Includes moderator band: ridge contains part of conducting system
    - Helps coordinate contractions of cardiac muscle cells
Internal Anatomy of the Heart: Right Ventricle into Pulmonary Trunk

Pulmonary Circuit

- **Conus arteriosus** (superior end of right ventricle) leads to *pulmonary trunk*
- Pulmonary trunk divides into *left* and *right pulmonary arteries*
- Blood flows from right ventricle to *pulmonary trunk* through *pulmonary valve*
- Pulmonary valve has three semilunar cusps
Anatomy of the Heart: Left Atrium and Ventricle

**The Left Atrium**
- *Lt. & rt. pulmonary veins* deliver oxygenated blood to left atrium
- Passes to left ventricle through *left AV (bicuspid or mitral) valve*

**The Left Ventricle**
- Holds same volume as right but thicker and more powerful; no moderator band

**Systemic Circulation**
Blood leaves left ventricle through *aortic valve* into *ascending aorta (aortic arch)*, which then becomes the *descending aorta*
Internal Anatomy of the Heart: Comparison of Right & Left Ventricles

- Right ventricle wall is thinner and develops less pressure than left ventricle (*why?--lungs are close to heart and pulmonary vessels are short and wide*)
- Left ventricle must produce 4 - 6x more pressure than that of right
- When left ventricle contracts, distance between base & apex decreases as well as diameter
- Contraction and bulging of the round left ventricle into the crescent-shaped right ventricle helps eject blood from right ventricle as well (see 20-7b)
Anatomy of Heart Valves

- **Semilunar Valves**
  - Pulmonary and aortic tricuspid valves
  - Prevent backflow from pulmonary trunk and aorta into right & left ventricles, respectively
  - Have no chordae tendineae or muscles

- **Aortic Sinuses**
  - Sacs at base of ascending aorta that prevent valve cusps from sticking to aorta
  - Origin of right and left coronary arteries

- **Valvular Heart Disease (VHD)**
  - Valve function deteriorates
  - e.g., *Rheumatic fever* — childhood reaction to streptococcal infection, chronic *carditis*
  - *Heart Murmur* — leaky valve = valvular incompetence (e.g., mitral valve prolapse – cusps don’t close properly)
  - Leak → ↑ work (work = vol. x press.)
Connective Tissue and the Cardiac Fibrous Skeleton

• **Myocardial Connective Tissues**
  • Physically support cardiac muscle fibers
  • Distribute forces of contraction
  • Add strength and prevent overexpansion of heart
  • Elastic fibers return heart to original shape after contraction

• **The Cardiac (Fibrous) Skeleton**
  • Four bands around heart valves and bases of pulmonary trunk and aorta
  • Stabilize valves
  • Electrically insulate ventricular cells from atrial cells

from Gray’s Anatomy
Coronary Circulation

- Heart is <1% body mass but requires 5% of blood circulation for function
- Coronary blood flow may ↑ 9x during vigorous activity
- Coronary Arteries (lt. & rt.) originate at aortic sinuses and then branch out
- Cardiac veins return deoxygenated coronary blood into right atrium
- High blood pressure & elastic rebound forces blood through coronary arteries between ventricular contractions
- **Arterial anastomoses** = interconnections between arteries (helps stabilize coronary blood flow despite pressure differences between lt. and rt. coronary arteries)
Coronary Circulation

- **Right Coronary Artery** -- supplies blood to rt. atrium, portions of *both* ventricles and cells of *sinoatrial* (SA) & *atrioventricular* (AV) nodes
  - gives rise to *marginal arteries* (surface of right ventricle)
  - Supplies *posterior interventricular artery* (bottom figure)

- **Left Coronary Artery** -- supplies blood to lt. atrium, lt. ventricle and interventricular septum
  - gives rise to *circumflex artery* and *anterior interventricular artery*

- **Cardiac Veins**: small veins drain into great cardiac vein which drains into the coronary sinus and eventually into the rt. atrium (at base of the inferior vena cava)
Coronary Circulation and Disease

- **Coronary Artery Disease (CAD)**
  - partial or complete block of coronary circulation, results in coronary ischemia, typically due to coronary thrombosis
  - Can lead to *myocardial infarction* (heart attack): heart tissue denied oxygen.
  - Common symptom of CAD: *angina pectoris* = pain in the chest as a result of the ischemia
  - **CAD treatments** include drugs that block sympathetic stimulation (e.g., *propranolol*) vasodilators (e.g., *nitroglycerin*) and calcium channel blockers

- **Plaques** can be removed surgically via catheter (laser or “roto-rooter”) or via balloon angioplasty; stents (wire mesh) used to keep artery open

- **Coronary bypass surgery** - use healthy veins (from legs) to create anatomoses around blockages; most people have 4 major coronary arteries, hence “quadruple bypass surgery”

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The Conducting System

Heartbeat – single cardiac cycle

- Two Types of Cardiac Muscle Cells
  - 1. Conducting System - control and coordinate heartbeat
  - 2. Contractile Cells - produce contractions that propel blood

- 1% of myocardial cells are auto-rhythmic: depolarize without neural or endocrine stimulation

- Each heartbeat begins with the action potential generated by pacemaker cells in the *sinoatrial (SA)* node

- Depolarization transmitted to other myocardial cells through conducting system & intercalated discs

- The entire heart contracts in series: atria first followed by ventricles

- Electrocardiogram (ECG or EKG) – electrical events in cardiac cycle

Fig 20–11
The Conducting System

- **Sinoatrial (SA) node**: located in wall of right atrium near superior vena cava
- **Atrioventricular (AV) node**: located at the junction between atria and ventricles
- **Conducting cells**: interconnect the 2 nodes and distribute electrical impulses throughout the myocardium
  - Includes the **AV bundle, bundle branches** and **Purkinje fibers**
- Cells of SA and AV nodes cannot maintain resting membrane potential & spontaneously drift towards threshold potential
  - Called **prepotential** or **pacemaker potential**
- **SA node**: 80-100 action potentials/min; **AV node**: 40-60
Impulse Conduction Through Heart

1. SA node activity and atrial activation begin.

   Time = 0

2. Stimulus spreads across the atrial surfaces and reaches the AV node.

   Elapsed time = 50 msec

3. There is a 100-msec delay at the AV node. Atrial contraction begins.

   Elapsed time = 150 msec

4. The impulse travels along the interventricular septum within the AV bundle and the bundle branches to the Purkinje fibers and, via the moderator band, to the papillary muscles of the right ventricle.

   Elapsed time = 175 msec

5. The impulse is distributed by Purkinje fibers and relayed throughout the ventricular myocardium. Atrial contraction is completed, and ventricular contraction begins.

   Elapsed time = 225 msec

Fig 20–12 Impulse Conduction

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The Conducting System

• Overall heart rate set by SA node
• Resting heart rate (sinus rhythm) ~75 bpm set by SA node (80-100) + parasympathetic stimulation (Vagus)
• Max rate 230 bpm set by AV node max; inefficient pumping above 180 bpm
• Abnormal Pacemaker Function
  • Bradycardia: abnormally slow heart rate (<60 bpm)
  • Tachycardia: abnormally fast heart rate (>100 bpm)
  • Ectopic pacemaker
    • Abnormal cells partially or completely bypass conducting system; disrupts timing of ventricular contractions
• Allometry “fact”?: average of 800 million beats lifespan
Electrocardiogram

- A recording of electrical events in the heart
- Obtained by electrodes at specific body locations
- Abnormal patterns used to diagnose damage

- **P wave** = Atria depolarize
- **QRS complex** = Ventricles depolarize
- **T wave** = Ventricles repolarize

*Time Intervals Between ECG Waves*

- **P–R interval**
  - From start of atrial depolarization to start of QRS complex
- **Q–T interval**
  - From ventricular depolarization to ventricular repolarization
ECG Diagnostics

- P-R segment longer than ~200ms = damage to AV node or conducting cells
- Total heart block = damaged AV node
  - No impulses transmitted through, atria and ventricles beat independently (atria fast, ventricles slow)
- Large QRS = enlarged heart (e.g., ventricular hypertrophy)
- Q-T interval longer than ~380ms = coronary ischemia or myocardial damage
- Cardiac arrhythmias = abnormal patterns of cardiac activity
  - **Fibrillation** = rapid, irregular, out of phase contractions due to activity in areas other than SA node: defibrillation to stop all activity so SA node can resume control
- Holter monitor can record continuously over 24h period
Premature Atrial Contractions (PACs) often occur in healthy individuals. In a PAC, the normal atrial rhythm is momentarily interrupted by a "surprise" atrial contraction. Stress, caffeine, and various drugs may increase the incidence of PACs, presumably by increasing the permeabilities of the SA pacemakers. The impulse spreads along the conduction pathway, and a normal ventricular contraction follows the atrial beat.

Paroxysmal Atrial Tachycardia (PAT) In paroxysmal (par-ok-SIZ-mal) atrial tachy-cardia, or PAT, a premature atrial contraction triggers a flurry of atrial activity. The ventricles are still able to keep pace, and the heart rate jumps to about 180 beats per minute.

Atrial Fibrillation (AF) During atrial fibrillation (fib-ri-LA-shun), the impulses move over the atrial surface at rates of perhaps 500 beats per minute. The atrial wall quivers instead of producing an organized contraction. The ventricular rate cannot follow the atrial rate and may remain within normal limits. Even though the atria are now nonfunctional, their contribution to ventricular end-diastolic volume (the maximum amount of blood the ventricles can hold at the end of atrial contraction) is so small that the condition may go unnoticed in older individuals.

Premature Ventricular Contractions (PVCs) Premature ventricular contractions (PVCs) occur when a Purkinje cell or ventricular myocardial cell depolarizes to threshold and triggers a premature contraction. Single PVCs are common and not dangerous. The cell responsible is called an ectopic pacemaker. The frequency of PVCs can be increased by exposure to epinephrine, to other stimulatory drugs, or to ionic changes that depolarize cardiac muscle cell membranes.

Ventricular Tachycardia (VT) Ventricular tachycardia is defined as four or more PVCs without intervening normal beats. It is also known as VT or V-tach. Multiple PVCs and VT may indicate that serious cardiac problems exist.

Ventricular Fibrillation (VF) Ventricular fibrillation (VF) is responsible for the condition known as cardiac arrest. VF is rapidly fatal, because the ventricles quiver and stop pumping blood.
Cardiac Muscle Cell Action Potential

Once threshold (~-75mV) reached, AP proceeds rapidly in 3 steps:

1. **Rapid Depolarization**
   - Cause: Na⁺ entry
   - Duration: 3–5 msec
   - Ends with: Closure of voltage-gated fast sodium channels

2. **The Plateau**
   - Cause: Ca²⁺ entry
   - Duration: ~175 msec
   - Ends with: Closure of slow calcium channels

3. **Repolarization**
   - Cause: K⁺ loss
   - Duration: 75 msec
   - Ends with: Closure of slow potassium channels

![Diagram of Action Potential](image)

Figure 20–15 The Action Potential in Skeletal and Cardiac Muscle

- **a** Events in an action potential in a ventricular muscle cell.
- **b** Action potentials and twitch contractions in a skeletal muscle (above) and cardiac muscle (below). The shaded areas indicate the durations of the absolute (blue) and relative (beige) refractory periods.
The Cardiac Action Potential

• **Refractory Period**
  • Period when AP cannot be elicited with a second stimulus OR requires > than normal stimulus
  • *Absolute* – no AP no matter what the stimulus because Na⁺ channels are either open and/or inactivated
  • *Relative* – can elicit a 2\(^{nd}\) AP with stronger-than-normal stimulus
• Length of cardiac action potential in ventricular cell
  • 250–300 ms:
    • 30 times longer than skeletal muscle fiber
    • long refractory period prevents summation and tetany
Role of Calcium Ions and Energetics for Cardiac Contraction

- 20% of Ca$^{2+}$ for contraction enters through slow, voltage-sensitive calcium channels in plasma membrane (extracellular sources)
- Influx triggers release of calcium ion reserves from sarcoplasmic reticulum (SR)
- As slow calcium channels close, intracellular Ca$^{2+}$ is pumped into SR or out of cell
- Energy for Cardiac Contractions
  - From mitochondrial breakdown of fatty acids and glucose
  - Oxygen from circulating hemoglobin and internal myoglobin
The Cardiac Cycle

Fig 20–16: Phases of the Cardiac Cycle

- **Systole** = contraction
- **Diastole** = relaxation

**Systole**

- **Atrial systole begins:** Atrial contraction forces a small amount of additional blood into relaxed ventricles.
- **Atrial systole ends, atrial diastole begins:**

**Diastole**

- **Ventricular diastole—early:** As ventricles relax, pressure in ventricles drops; blood flows back against cusps of semilunar valves and forces them closed. Blood flows into the relaxed atria.
- **Ventricular diastole—late:** All chambers are relaxed. Ventrices fill passively.
- **Ventricular systole—first phase:** Ventricular contraction pushes AV valves closed but does not create enough pressure to open semilunar valves.
- **Ventricular systole—second phase:** As ventricular pressure rises and exceeds pressure in the arteries, the semilunar valves open and blood is ejected.
8 Steps in the Cardiac Cycle

1. **Atrial systole begins**
   - Atrial contraction begins
   - Rt. & Lt. AV valves remain open

2. **Atria “top off” ventricles**
   - Filling ventricles

3. **Atrial systole ends**
   - AV valves close
   - Ventricles contain max vol. known as end-diastolic volume (EDV)

4. **Ventricular systole begins**
   - Isovolumetric ventricular contraction
   - Pressure in ventricles rises with AV valves shut
The Cardiac Cycle

5. **Ventricular ejection**
   - Semilunar valves open
   - Blood flows into pulmonary and aortic trunks
   - Stroke volume (SV) = 60% of end-diastolic volume

6. **Ventricular pressure falls**
   - Semilunar valves close
   - Ventricles contain end-systolic volume (ESV), about 40% of end-diastolic volume

7. **Ventricular diastole**
   - Ventricular pressure is higher than atrial pressure
   - All heart valves are closed
   - Ventricles relax (isovolumetric relaxation)
The Cardiac Cycle

8. AV valves open & passive atrial filling begins.
   - When ventricular pressure falls below atrial pressure, the AV valves open
   - Blood then flows from the atria into the ventricles while both are in diastole
The Cardiac Cycle: Sounds & Auscultation

**S₁** = “lubb” – AV valves closing at start of ventricular systole

**S₂** = “dubb” – semilunar valves closing at start of ventricular diastole

**S₃** = blood flowing into ventricles as AV valves open

**S₄** = atrial contraction

*Murmur* = sound produced by regurgitation through valve

By determining where a sound originates and when it occurs in cardiac cycle, cardiologists can determine if valve problems are due to incompetence (leak) or stenosis (narrowing)
Cardiodynamics

Cardiodynamics = movement & force generated by cardiac contractions

- **End-diastolic volume (EDV)** – amount of blood in ventricles at end of diastole
- **End-systolic volume (ESV)** – amount of blood in ventricles at end of systole
- **Stroke volume (SV)** – amount of blood ejected in single beat (ml/beat)
  - SV = EDV – ESV
- **Ejection fraction**
  - The percentage of EDV represented by SV
- **Cardiac output (CO)** (ml/min)
  - The volume pumped by ventricle in 1 minute
  - CO = HR x SV (heart rate [beats/min] times stroke volume)
    - (e.g., 75 bpm x 80 ml/beat = 6000 ml/min)

Fig 20-19
Figure 20–20 Factors Affecting Cardiac Output

Cardiodynamics

Factors Affecting Heart Rate (HR)
- Autonomic innervation
- Hormones

Factors Affecting Stroke Volume (SV)
- End-diastolic volume
- End-systolic volume

Heart Rate (HR)

Stroke Volume (SV) = EDV - ESV

Cardiac Output (CO) = HR × SV
Adjusting Cardiodynamics: Innervation

- **Autonomic innervation**
  - Both sympathetic (NE) and parasympathetic (ACh) innervation of SA node, AV node and atrial myocardium
  - Sympathetic dominates in ventricles
  - Cardiac centers of medulla oblongata monitor blood pressure (baroreceptors) arterial O$_2$ and CO$_2$ levels (chemoreceptors)
    - **cardioacceleratory center** controls sympathetic neurons (increases heart rate – *positive chronotropic effect*)
    - **cardioinhibitory center** controls parasympathetic neurons (slows heart rate – *negative chronotropic effect*)

- **Autonomic tone**
  - Dual innervation maintains resting tone by releasing ACh (and NE)
  - At rest, parasympathetic tone reduces SA node inherent rate (80-100 bpm) to ~70 bpm
Adjusting Cardiodynamics: Innervation

Figure 20–21 Autonomic Innervation of the Heart
Adjusting Cardiodynamics: Innervation

Effects of Sympathetic & Parasympathetic Stimulation on Pacemaker Cells

ACh (parasympathetic stimulation) opens K⁺ channels thereby slowing the rate of spontaneous depolarization and slightly extending the duration of repolarization.

NE (sympathetic stimulation) binds to β-1 receptors thereby opening Na⁺-Ca²⁺ channels and increasing the rate of depolarization and shortening the duration of repolarization.

Figure 20–22 Autonomic Regulation of Pacemaker Function
Adjusting Cardiodynamics: Reflexes, Hormones & Drugs

- **Atrial or Bainbridge Reflex**
  - Increased venous return activates stretch receptors in right atrium → ↑ sympathetic activity → ↑ HR

- **Hormonal Effects on Heart Rate**
  - Epinephrine (E), Norepinephrine (NE) and Thyroid hormone (thyroxine) ↑ HR and *contractile strength (positive inotropic effect)* by acting on SA node
  - β-1 blockers (hypertensive drugs) block E & NE effects

- **Other Heart Rate Effectors**
  - Caffeine: rapid depolarization of SA node, ↑ HR
  - Nicotine: stimulates sympathetic neurons, ↑ HR
  - Changes in K⁺, Ca²⁺, temperature, etc.
Cardiodynamics: Stroke Volume Adjustments

- **End diastolic volume (EDV)** is affected by
  1. Venous return
  2. Filling time

  \[ \text{↑ EDV} \rightarrow \text{↑ preload (amount of ventricular stretch)} \rightarrow \text{↑ SV} \text{ (Frank-Starling Principle)} \]

- **End systolic volume (ESV)** is affected by
  1. Preload
  2. Contractility = force produced during contraction *(inotropic)*
  3. Afterload = tension the ventricle must produce to open the semilunar valve and eject blood (↑ by any factor that restricts arterial blood flow)

  - Thus it is obvious that \[ \text{↑ ESV} \rightarrow \text{↓ SV} \]
Cardiodynamics: Stroke Volume Adjustments

Factors Affecting Stroke Volume (SV)

- Venous return (VR)
  - ↑VR = ↑EDV
  - ↓VR = ↓EDV

- Filling time (FT)
  - ↑FT = ↑EDV
  - ↓FT = ↓EDV

- Increased by sympathetic stimulation

- Decreased by parasympathetic stimulation

- Increased by E, NE, glucagon, thyroid hormones

- Contractility (Cont)
  - ↑Cont = ↓ESV
  - ↓Cont = ↑ESV

- Increased by vasoconstriction

- Decreased by vasodilation

- Afterload (AL)
  - ↑AL = ↑ESV
  - ↓AL = ↓ESV

- End-diastolic volume (EDV)

- End-systolic volume (ESV)

STROKE VOLUME (SV)

- ↑EDV = ↑SV
- ↓EDV = ↓SV
- ↑ESV = ↓SV
- ↓ESV = ↑SV

Figure 20–23
Cardiodynamics: **Contractility**

- **Sympathetic stimulation – positive inotropic effect**
  - NE released by postganglionic fibers of cardiac nerves AND both E & NE released by suprarenal (adrenal) medullae
  - Increases ejection fraction and decreases ESV

- **Parasympathetic activity – negative inotropic effect**
  - Acetylcholine released by vagus nerve

- **Hormones – can have neg. or pos. inotropic effects**
  - Many pharmaceutical drugs mimic E, NE and thyroxine actions
  - β-1 Receptors Mimetics (e.g., *isoproterenol, dopamine, dobutamine*)
  - β-1 Receptor Blockers (e.g., *propranolol, timolol, metoprolol*)
  - Others affect Ca\(^{2+}\) (e.g., *digitalis* [inhibit SR uptake], *nifedipine* and *verapamil* [channel blockers])
Summary of Factors Affecting Cardiac Output

Factors affecting heart rate (HR)
- Atrial reflex
- Autonomic innervation
- Hormones

Factors affecting stroke volume (SV)
- Venous return
- Preload
- End-diastolic volume
- End-systolic volume
- Filling time
- Contractility
- Changes in peripheral circulation
- Blood volume
- Skeletal muscle activity
- Autonomic innervation
- Hormones

STROKE VOLUME (SV) = EDV – ESV

CARDIAC OUTPUT (CO) = HR x SV

Figure 20–24 A Summary of the Factors Affecting Cardiac Output