Chapter 20 (2)

The Heart

Describe the component and function of the intrinsic cardiac conduction system  ///  Trace an impulse through the conduction system of the heart

Identify the area of the CNS concerned with cardiovascular regulation.  ///  Describe factors which regulate heart rate, including the autonomic nervous system and baroreceptors ///  Cardiac rhythms & arrhythmias

Diagram and describe a typical ECG / EKG pattern and relate it to pressure changes and heart sounds
Learning Objectives

• Describe the component and function of the intrinsic cardiac conduction system

• Trace an impulse through the conduction system of the heart
The heart beats every 0.8 seconds (cardiac cycle) or 75 beats per minute.

With each beat the heart pumps 70 ml of blood (stroke volume) out of both the left and right ventricles.

Cardiac output = 5.25 L per minute = 70 ml/beat x 75 beats/min

This rhythmic action is regulated by a pacemaker and the intrinsic conduction system.
Intrinsic Cardiac Conduction System

- Start at preceding cardiac cycle

- At start of new cycle ventricles must be relaxed which allows atrioventricular valves to be open

- With new cardiac cycle - atria depolarization allows blood in atria to move “down” into ventricles

- Action potentials from atria must be “delayed” at atrioventricular node

- The action potential is also blocked from leaking into the superior ventricular region
Intrinsic Cardiac Conduction System

- **Note:** this allows ventricles to fill with blood & prevents depolarization in superior region of ventricles.

- After delay, action potential at atrioventricular node is transmitted to the apex of heart.

- Now ventricles must start to depolarize at apex of heart so blood can be pushed “up” into aorta and pulmonary truck.
• The ICCS coordinates the heartbeat rhythm

• Conduction system creates a cyclical electrical “wave” that passes through heart

• These events are known as the Cardiac Cycle // repeats every 0.8 sec

• Cardiac cycle maintained by an internal pacemaker and by a nerve like conduction pathways (ICCS) which run through the myocardium between the AV note to the apex and beyond
Cardiac cycle maintained by an internal pacemaker and by a nerve like conduction pathways which run through the myocardium between the AV note to the apex and beyond.
(a) Anterior view of frontal section

1. SINOATRIAL (SA) NODE
2. ATRIOVENTRICULAR (AV) NODE
3. ATRIOVENTRICULAR (AV) BUNDLE (BUNDLE OF HIS)
4. RIGHT AND LEFT BUNDLE BRANCHES
5. PURKINJE FIBERS

Left atrium
Left ventricle
Right atrium
Right ventricle
Frontal plane
Nodal Tissue and Nerve Supply of the Heart

Sinoatrial Node = Pacemaker (leak sodium fastest!)

Atrioventricular Node = Secondary Pacemaker (leak sodium more slower than SA note)

The nodal rate of depolarization is “modified” by the autonomic nervous system

  • Sympathetic NS / increase rate
  • Parasympathetic NS / decrease rate

Note: any myocardiocyte isolated from heart will spontaneously depolarize
Cardiac Conduction System

• Generates and conducts rhythmic electrical signals in the following order

• sinoatrial (SA) node
  – modified cardiocytes
  – initiates each heartbeat and determines heart rate
  – signals spread throughout atria
  – pacemaker in right atrium near base of superior vena cava

• atrioventricular (AV) node
  – located near the right AV valve at lower end of interatrial septum
  – electrical gateway to the ventricles
  – fibrous skeleton acts as an insulator to prevent currents from getting to the ventricles from any other route
Cardiac Conduction System

- atrioventricular (AV) bundle (bundle of His)
  - bundle forks into right and left bundle branches
  - these branches pass through interventricular septum toward apex

- Purkinje fibers
  - Nerve like processes spread throughout ventricular myocardium
  - From end of Purkinje Fibers // signal pass from cell to cell through gap junctions
Nodal Tissue and Nerve Supply of the Heart

Some special myocardiocytes are not able to maintain a resting membrane potential; these areas allow sodium to leak into the cytoplasm of these cells.

Those areas with the highest rate of ion leakage spontaneously reach threshold and cause an action potential (nodal potential).

Sinoatrial Node (SA node) and Atrioventricular Node (AV node) “leak sodium ions”.

These nodal cells do not maintain a “static” resting potential; both leak sodium ions.

Cardiocytes to reach threshold first (SA node) will depolarize and set the rate of depolarization for all the other myocardiocytes in the heart.
SA Node Potentials

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(b) Pacemaker potentials (green) and action potentials (black) in autorhythmic fibers of SA node
Impulse Conduction to Myocardium

- Signal from SA node stimulates right and left atria to contract almost simultaneously
  - Reaches AV node in 50 msec
- Signal slows down or is delayed at AV node
  - This allows atria to complete their contraction plus allows complete filling of the ventricles
  - Thin cardiocytes at AV node have fewer gap junctions // helps explain the delay at AV node
  - Delays signal 100 msec
Impulse Conduction to Myocardium

• signals travel very quickly through AV bundle and Purkinje fibers
  – entire ventricular myocardium depolarizes and contracts in near unison
  – papillary muscles contract an instant earlier than the rest///
    tightening slack in chordae tendineae

• ventricular systole progresses up from the apex of the heart
  – spiral arrangement of cardiocytes twists ventricles slightly
  – like someone wringing out a towel
Cardiac Conduction System

1. SA node fires.
2. Excitation spreads through atrial myocardium.
3. AV node fires.
4. Excitation spreads down AV bundle.
5. Purkinje fibers distribute excitation through ventricular myocardium.

Right atrium
Sinoatrial node (pacemaker)
Atrioventricular node
Atrioventricular bundle
Purkinje fibers
Left atrium
Purkinje fibers
Bundle branches
Learning Objectives

• Identify the area of the CNS concerned with cardiovascular regulation.

• Describe factors which regulate heart rate, including the autonomic nervous system and baroreceptors

• Cardiac rhythms & arrhythmias
INPUT TO CARDIOVASCULAR CENTER
- From higher brain centers: cerebral cortex, limbic system, and hypothalamus
- From sensory receptors:
  - Proprioceptors—monitor movements
  - Chemoreceptors—monitor blood chemistry
  - Baroreceptors—monitor blood pressure

OUTPUT TO HEART
- Increased rate of spontaneous depolarization in SA node (and AV node) increases heart rate
- Increased contractility of atria and ventricles increases stroke volume
- Decreased rate of spontaneous depolarization in SA node (and AV node) decreases heart rate
Regulation (Inputs) to Cardiac Center

• Cardiac centers located in the medulla oblongata (reticular formation)
  – receive input from many sources and integrate them into ‘decisions’ to either speed up or slow down the heart

• Higher brain centers can affect heart rate
  • cerebral cortex, limbic system, hypothalamus // sensory or emotional stimuli // biofeedback // meditation

  • Heart rate may start to increase even “before the event starts!” // anticipation of muscular activity
Regulation (Inputs) to Cardiac Center

• Medulla also receives input from muscles & joints

• Proprioceptors in the muscles and joints
  – inform cardiac center about changes in activity
Regulation (Inputs) to Cardiac Center

- Baroreceptors send signal to cardiac center

  • pressure sensors (called sinuses) located: in aortic arch & internal carotid arteries

  • blood pressure decreases // cardiac center increases heart rate // more blood pumped into vessels and blood pressure increases

  • if blood pressure increases /// cardiac center decreases heart rate /// less blood pumped into vessels and blood pressure decreases
Blood Pressure & Heart Function

Person rises from bed

Blood pressure rises to normal; homeostasis is restored

Cardiac center accelerates heartbeat

Baroreceptors above heart respond to drop in blood pressure

Baroreceptors send signals to cardiac center of brainstem

Blood drains from upper body, creating homeostatic imbalance
Chemoreceptors (three locations)

- aortic arch
- carotid arteries
- medulla oblongata

- sensitive to blood pH, CO$_2$ and O$_2$ levels

- *Chemoreceptors are more important in respiratory control than cardiac control*

- if CO$_2$ accumulates in blood or CSF (hypercapnia), reacts with water and causes increase in H$^+$ levels
• Hypercapnia (high CO2) and acidosis stimulate the cardiac center to increase heart rate

• also respond to hypoxemia
  – oxygen deficiency in the blood
  – usually slows down the heart

• Note: chemoreflexes and baroreflexes
  – responses to fluctuation in blood chemistry
  – both negative feedback loops
Nerve Supply to Heart – Sympathetic Nerves

- Sympathetic nerves
  - sympathetic pathway to the heart originates in the lower cervical to upper thoracic segments of the spinal cord
  - continues to adjacent sympathetic chain ganglia
  - some pass through cardiac plexus in mediastinum
  - continue as cardiac nerves to the heart
Nerve Supply to Heart – Sympathetic Nerves

- Sympathetic nerves
  - fibers terminate in
    - SA and AV nodes
    - in atrial and ventricular myocardium
    - coronary arteries (as well as the aorta, pulmonary trunk)
  - increase heart rate and contraction strength
  - dilates coronary arteries to increase myocardial blood flow
Nerve Supply to Heart - Parasympathetic

• Parasympathetic nerves
  – pathway begins with nuclei of the vagus nerves in the medulla oblongata
  – extend to cardiac plexus and continue to the heart by way of the cardiac nerves
  – fibers of right vagus nerve lead to the SA node
  – fibers of left vagus nerve lead to the AV node
  – little or no vagal stimulation of the myocardium

• parasympathetic stimulation reduces the heart rate // slows heart rate
Cardiac Rhythms / Terminology

• Cycle of events in heart given special names
  – systole – atrial or ventricular contraction
  – diastole – atrial or ventricular relaxation

• Sinus rhythm
  – normal heartbeat triggered by the SA node
  – heart rate benchmark 75 bpm
  – if all ANS fibers cut / heart rate 100 bpm
  – Vagal tone – vagus nerve under normal conditions suppresses the heart rate
Cardiac Rhythms / Terminology

- **Ectopic focus** /// caused by another parts of heart that fires before SA node discharges
  - caused by hypoxia, electrolyte imbalance, caffeine, nicotine, cocaine and other drugs

- **Nodal rhythm** /// if SA node is damaged, heart rate is then set by AV node
  - 40 to 50 bpm /// not great but you may survive with nodal rhythm
Cardiac Rhythms / Terminology

- **Intrinsic ventricular rhythm** // if both SA and AV nodes are not functioning
  - rate set by other myocardioocytes at 20 to 40 bpm
  - this requires artificial pacemaker to sustain life long term

- **Arrhythmia** // any abnormal cardiac rhythm
  - could be failure of nodal potential(s)
  - conduction system to transmit signals
  - bundle branch block

- **Total heart block** // damage to AV node
  - potential fails to pass AV node
Cardiac Rhythms / Terminology

- **Atrial fibrillation** // ectopic foci in atria
  - atria beat **200 - 400 times per minute**
  - may not be fatal / ventricles will still fill with blood passively

- **Ventricular fibrillation** // serious arrhythmia caused by electrical signals reaching different regions at widely different times
  - heart can’t pump blood and no coronary perfusion
  - Will kill quickly if not stopped
  - **defibrillation** - strong electrical shock whose intent is to depolarize the entire myocardium
    - stop the fibrillation
    - hopefully, reset normal SA node to sinus rhythm
• **Premature ventricular contractions (PVCs)** // caused by stimulants, stress or lack of sleep

• **Tachycardia** // Persistent resting adult heart rate above 100 bpm

• **Bradycardia** // Persistent resting adult heart rate below 60 bpm
Learning Objectives

• Diagram and describe a typical ECG (EKG) pattern and relate it to pressure changes and heart sounds
Electrocardiogram (ECG or EKG)

- composite of all action potentials of nodal and myocardial cells
- detected, amplified and recorded by electrodes on arms, legs and chest
ECG Deflections

- **P wave**
  - SA node fires
  - atria depolarize

- **PQ segment**
  - atrial systole
  - atrial systole begins 100 msec after SA signal

- **QRS complex**
  - ventricular depolarization
  - complex shape of spike due to different thickness and shape of the two ventricles

- **ST segment**
  - ventricular systole
  - plateau in myocardial action potential

- **T wave**
  - ventricular repolarization and relaxation

Note: asystole = “flat line” / no contraction of myocardium / requires cardiopulmonary resuscitation (CPR)
Action Potential of Myocardiocyte

1. Voltage-gated Na⁺ channels open.
2. Na⁺ inflow depolarizes the membrane and triggers the opening of still more Na⁺ channels, creating a positive feedback cycle and a rapidly rising membrane voltage.
3. Na⁺ channels close when the cell depolarizes, and the voltage peaks at nearly +30 mV.
4. Ca²⁺ entering through slow Ca²⁺ channels prolongs depolarization of membrane, creating a plateau. Plateau falls slightly because of some K⁺ leakage, but most K⁺ channels remain closed until end of plateau.
5. Ca²⁺ channels close and Ca²⁺ is transported out of cell. K⁺ channels open, and rapid K⁺ outflow returns membrane to its resting potential.
1. Rapid depolarization due to Na\(^+\) inflow when voltage-gated fast Na\(^+\) channels open

2. Plateau (maintained depolarization) due to Ca\(^{2+}\) inflow when voltage-gated slow Ca\(^{2+}\) channels open and K\(^+\) outflow when some K\(^+\) channels open

3. Repolarization due to closure of Ca\(^{2+}\) channels and K\(^+\) outflow when additional voltage-gated K\(^+\) channels open

- Depolarization
- Repolarization
- Refractory period
- Contraction

Membrane potential (mV)

Time (0.3 sec)
Electrical Activity of Myocardium

1) atrial depolarization begins
2) atrial depolarization complete (atria contracted)
3) ventricles begin to depolarize at apex; atria repolarize (atria relaxed)
4) ventricular depolarization complete (ventricles contracted)
5) ventricles begin to repolarize at apex
6) ventricular repolarization complete (ventricles relaxed)
(a) Normal electrocardiogram (ECG)
ECGs: Normal and Abnormal

- abnormalities in conduction pathways
- myocardial infarction
- heart enlargement
- electrolyte and hormone imbalances

(a) Sinus rhythm (normal)

(b) Nodal rhythm—no SA node activity
Long P–Q interval

(b) First-degree AV block
(d) Ventricular tachycardia  (e) Ventricular fibrillation
Irregular R–R intervals

No detectable P waves

(c) Atrial fibrillation
Diagnostic Value of ECG

- abnormalities in conduction pathways
- myocardial infarction
- nodal damage
- heart enlargement
- electrolyte and hormone imbalances
New Slides
(a) Anterior view of coronary arteries
Superior vena cava
Right atrium
SMALL CARDIAC
ANTEROIOR CARDIAC
MIDDLE CARDIAC
Right ventricle
Inferior vena cava
Pulmonary trunk
Left auricle
CORONARY SINUS
GREAT CARDIAC
Left ventricle

(b) Anterior view of coronary veins
(b) Pacemaker potentials (green) and action potentials (black) in autorhythmic fibers of SA node
Increased end-diastolic volume (stretches the heart)

- Increased PRELOAD

  - Within limits, cardiac muscle fibers contract more forcefully with stretching (Frank-Starling law of the heart)

Positive inotropic agents such as increased sympathetic stimulation; catecholamines, glucagon, or thyroid hormones in the blood; increased Ca\(^{2+}\) in extracellular fluid

- Increased CONTRACTILITY

  - Positive inotropic agents increase force of contraction at all physiological levels of stretch

Decreased arterial blood pressure during diastole

- Decreased AFTERLOAD

  - Semilunar valves open sooner when blood pressure in aorta and pulmonary artery is lower

Increased STROKE VOLUME

Increased CARDIAC OUTPUT

- Increased HEART RATE

Increased sympathetic stimulation and decreased parasympathetic stimulation

NERVOUS SYSTEM
Cardiovascular center in medulla oblongata receives input from cerebral cortex, limbic system, proprioceptors, baroreceptors, and chemoreceptors

CHEMICALS
Catecholamine or thyroid hormones in the blood; moderate increase in extracellular Ca\(^{2+}\)

OTHER FACTORS
Infants and senior citizens; females; low physical fitness; increased body temperature
(a) Normal electrocardiogram (ECG)

(b) First-degree AV block

(c) Atrial fibrillation

(d) Ventricular tachycardia

(e) Ventricular fibrillation
(a) Normal electrocardiogram (ECG)
(b) First-degree AV block
(d) Ventricular tachycardia
(e) Ventricular fibrillation
(c) Atrial fibrillation

Irregular R–R intervals

No detectable P waves
New Slides
(a) Anterior view of frontal section
(b) Pacemaker potentials (green) and action potentials (black) in autorhythmic fibers of SA node
2 Plateau (maintained depolarization) due to Ca\(^{2+}\) inflow when voltage-gated slow Ca\(^{2+}\) channels open and K\(^+\) outflow when some K\(^+\) channels open

1 Rapid depolarization due to Na\(^+\) inflow when voltage-gated fast Na\(^+\) channels open

3 Repolarization due to closure of Ca\(^{2+}\) channels and K\(^+\) outflow when additional voltage-gated K\(^+\) channels open

Depolarization \(\rightarrow\) Repolarization

Refractory period

Contraction
Key:
- Blue: Atrial contraction
- Yellow: Ventricular contraction
1. Depolarization of atrial contractile fibers produces P wave
2. Atrial systole (contraction)
3. Depolarization of ventricular contractile fibers produces QRS complex
4. Ventricular systole (contraction)
5. Repolarization of ventricular contractile fibers produces T wave
6. Ventricular diastole (relaxation)