CHAPTER 18

The Cardiovascular System: The Heart: Part A
The Pulmonary and Systemic Circuits

• Heart is transport system; two side-by-side pumps
  – *Right side* receives oxygen-poor blood from tissues
    • Pumps to lungs to get rid of CO\textsubscript{2}, pick up O\textsubscript{2}, via **pulmonary circuit**
  – *Left side* receives oxygenated blood from lungs
    • Pumps to body tissues via **systemic circuit**
Figure 18.1 The systemic and pulmonary circuits.

Capillary beds of lungs where gas exchange occurs

Pulmonary Circuit
- Pulmonary arteries
- Pulmonary veins
- Aorta and branches

Systemic Circuit
- Venae cavae
- Left atrium
- Left ventricle
- Right atrium
- Right ventricle

Heart

Oxygen-rich, CO$_2$-poor blood
Oxygen-poor, CO$_2$-rich blood

Capillary beds of all body tissues where gas exchange occurs
Heart Anatomy

• Approximately size of fist
• Location:
  – In *mediastinum* between second rib and fifth intercostal space
  – On superior surface of diaphragm
  – Two-thirds of heart to left of midsternal line
  – Anterior to vertebral column, posterior to sternum

Animation: Rotatable heart
Heart Anatomy

- **Base** (posterior surface) leans toward right shoulder
- **Apex** points toward left hip
- **Apical impulse** palpated between fifth and sixth ribs, just below left nipple
Figure 18.2a Location of the heart in the mediastinum.

- Midsternal line
- 2nd rib
- Diaphragm
- Sternum
- Location of apical impulse

(a)
Figure 18.2c Location of the heart in the mediastinum.

- Superior vena cava
- Pulmonary trunk
- Aorta
- Parietal pleura (cut)
- Left lung
- Pericardium (cut)
- Apex of heart
- Diaphragm
Coverings of the Heart: Pericardium

- Double-walled sac
- Superficial **fibrous pericardium**
  - Protects, anchors to surrounding structures, and prevents overfilling
Pericardium

- Deep two-layered **serous pericardium**
  - **Parietal layer** lines internal surface of fibrous pericardium
  - **Visceral layer (epicardium)** on external surface of heart
  - Two layers separated by fluid-filled **pericardial cavity** (decreases friction)
Figure 18.3 The pericardial layers and layers of the heart wall.
Homeostatic Imbalance

• *Pericarditis*
  – Inflammation of pericardium
  – Roughens membrane surfaces \(\rightarrow\) **pericardial friction rub** (creaking sound) heard with stethoscope
  – *Cardiac tamponade*
    • Excess fluid sometimes compresses heart \(\rightarrow\) limited pumping ability
Layers of the Heart Wall

• Three layers of heart wall:
  – Epicardium
  – Myocardium
  – Endocardium

• Epicardium
  – Visceral layer of serous pericardium
Layers of the Heart Wall

• Myocardium
  – Spiral bundles of contractile cardiac muscle cells
  – Cardiac skeleton: crisscrossing, interlacing layer of connective tissue
    • Anchors cardiac muscle fibers
    • Supports great vessels and valves
    • Limits spread of action potentials to specific paths
Layers of the Heart Wall

• Endocardium continuous with endothelial lining of blood vessels
  – Lines heart chambers; covers cardiac skeleton of valves
Figure 18.3 The pericardial layers and layers of the heart wall.

- Pericardium
- Myocardium
- Pulmonary trunk
- Fibrous pericardium
- Parietal layer of serous pericardium
- Pericardial cavity
- Epicardium (visceral layer of serous pericardium)
- Myocardium
- Endocardium
- Heart chamber
- Heart wall
Figure 18.4 The circular and spiral arrangement of cardiac muscle bundles in the myocardium of the heart.
Chambers

- Four chambers:
  - Two superior atria
  - Two inferior ventricles

- **Interatrial septum** – separates atria
  - **Fossa ovalis** – remnant of foramen ovale of fetal heart

- **Interventricular septum** – separates ventricles
Figure 18.5e Gross anatomy of the heart.

Superior vena cava
Right pulmonary artery
Pulmonary trunk
Right atrium
Right pulmonary veins
Fossa ovalis
Pectinate muscles
Tricuspid valve
Right ventricle
Chordae tendineae
Trabeculae carneae
Inferior vena cava

Aorta
Left pulmonary artery
Left atrium
Left pulmonary veins
Mitral (bicuspid) valve
Aortic valve
Pulmonary valve
Left ventricle
Papillary muscle
Interventricular septum
Epicardium
Myocardium
Endocardium

(e) Frontal section
Atria: The Receiving Chambers

- Small, thin-walled
- Contribute little to propulsion of blood
- Three veins empty into right atrium:
  - Superior vena cava, inferior vena cava, coronary sinus
- Four pulmonary veins empty into left atrium
Ventricles: The Discharging Chambers

- Most of the volume of heart
- **Right ventricle** - most of anterior surface
- **Left ventricle** – posteroinferior surface
- **Trabeculae carneae** – irregular ridges of muscle on walls
- **Papillary muscles** – anchor **chordae tendineae**
Ventricles: The Discharging Chambers

- Thicker walls than atria
- Actual pumps of heart
- Right ventricle
  - Pumps blood into **pulmonary trunk**
- Left ventricle
  - Pumps blood into **aorta** (largest artery in body)
Figure 18.5b Gross anatomy of the heart.

- Brachiocephalic trunk
- Superior vena cava
- Right pulmonary artery
- Ascending aorta
- Pulmonary trunk
- Right pulmonary veins
- Right atrium
  - Right coronary artery (in coronary sulcus)
  - Anterior cardiac vein
  - Right ventricle
  - Right marginal artery
  - Small cardiac vein
  - Inferior vena cava
- Left common carotid artery
- Left subclavian artery
- Aortic arch
- Ligamentum arteriosum
- Left pulmonary artery
- Left pulmonary veins
- Auricle of left atrium
- Circumflex artery
- Left coronary artery (in coronary sulcus)
- Left ventricle
  - Great cardiac vein
  - Anterior interventricular artery (in anterior interventricular sulcus)
  - Apex

(b) Anterior view
Figure 18.5a Gross anatomy of the heart.

- Aortic arch (fat covered)
- Pulmonary trunk
- Auricle of right atrium
- Auricle of left atrium
- Anterior interventricular artery
- Right ventricle
- Apex of heart (left ventricle)

(a) Anterior aspect (pericardium removed)
Figure 18.5f  Gross anatomy of the heart.

Superior vena cava

Ascending aorta (cut open)

Pulmonary trunk

Aortic valve

Pulmonary valve

Interventricular septum (cut)

Left ventricle

Papillary muscles

Right ventricle anterior wall (retracted)

Trabeculae carneae

Opening to right atrium

Chordae tendineae

Right ventricle

(f) Photograph; view similar to (e)
Heart Valves

• Ensure unidirectional blood flow through heart
• Open and close in response to pressure changes
• Two atrioventricular (AV) valves
  – Prevent backflow into atria when ventricles contract
  – Tricuspid valve (right AV valve)
  – Mitral valve (left AV valve, bicuspid valve)
  – Chordae tendineae anchor cusps to papillary muscles
    • Hold valve flaps in closed position
Blood returning to the heart fills atria, pressing against the AV valves. The increased pressure forces AV valves open.

As ventricles fill, AV valve flaps hang limply into ventricles.

Atria contract, forcing additional blood into ventricles.

(a) AV valves open; atrial pressure greater than ventricular pressure

Ventricles contract, forcing blood against AV valve cusps.

AV valves close.

Papillary muscles contract and chordae tendineae tighten, preventing valve flaps from everting into atria.

(b) AV valves closed; atrial pressure less than ventricular pressure
Heart Valves

- **Two semilunar (SL) valves**
  - Prevent backflow into ventricles when ventricles relax
  - Open and close in response to pressure changes
  - Aortic semilunar valve
  - Pulmonary semilunar valve
As ventricles contract and intraventricular pressure rises, blood is pushed up against semilunar valves, forcing them open.

(a) Semilunar valves open

As ventricles relax and intraventricular pressure falls, blood flows back from arteries, filling the cusps of semilunar valves and forcing them to close.

(b) Semilunar valves closed
Figure 18.6a Heart valves.

- Pulmonary valve
- Aortic valve
- Area of cutaway
- Mitral valve
- Tricuspid valve
- Myocardium
- Mitral (left atrioventricular) valve
- Tricuspid (right atrioventricular) valve
- Aortic valve
- Pulmonary valve
- Cardiac skeleton
- Anterior
Figure 18.6c Heart valves.

- Pulmonary valve
- Aortic valve
- Area of cutaway
- Mitral valve
- Tricuspid valve

Chordae tendineae attached to tricuspid valve flap

(c) Papillary muscle
Figure 18.6d Heart valves.

- Pulmonary valve
- Aortic valve
- Area of cutaway
- Mitral valve
- Tricuspid valve

- Opening of inferior vena cava
- Tricuspid valve
- Myocardium of right ventricle
- Mitral valve
- Chordae tendineae
- Interventricular septum
- Papillary muscles
- Myocardium of left ventricle
Homeostatic Imbalance

• Two conditions severely weaken heart:
  – Incompetent valve
    • Blood backflows so heart repumps same blood over and over
  – Valvular stenosis
    • Stiff flaps – constrict opening → heart must exert more force to pump blood

• Valve replaced with mechanical, animal, or cadaver valve
Pathway of Blood Through the Heart

- **Pulmonary circuit**
  - Right atrium $\rightarrow$ tricuspid valve $\rightarrow$ right ventricle
  - Right ventricle $\rightarrow$ pulmonary semilunar valve $\rightarrow$ pulmonary trunk $\rightarrow$ pulmonary arteries $\rightarrow$ lungs
  - Lungs $\rightarrow$ pulmonary veins $\rightarrow$ left atrium
Pathway of Blood Through the Heart

- **Systemic circuit**
  - Left atrium $\rightarrow$ mitral valve $\rightarrow$ left ventricle
  - Left ventricle $\rightarrow$ aortic semilunar valve $\rightarrow$ aorta
  - Aorta $\rightarrow$ systemic circulation
Figure 18.9  The heart is a double pump, each side supplying its own circuit. Both sides of the heart pump at the same time, but let’s follow one spurt of blood all the way through the system.

Oxygen-rich blood returns from the body tissues back to the heart.

Oxygen-poor blood is carried in two pulmonary arteries to the lungs (pulmonary circuit) to be oxygenated.

Oxygen-rich blood is delivered to the body tissues (systemic circuit).

Oxygen-rich blood returns to the heart via the four pulmonary veins.

© 2013 Pearson Education, Inc.
Figure 18.9  The heart is a double pump, each side supplying its own circuit.
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

Superior vena cava (SVC)
Inferior vena cava (IVC)
Coronary sinus

- Oxygen-poor blood
- Oxygen-rich blood

SVC
Coronary sinus
Right atrium

IVC

© 2013 Pearson Education, Inc.
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

Oxygen-poor blood

Oxygen-rich blood

Superior vena cava (SVC)
Inferior vena cava (IVC)
Coronary sinus

Right atrium

Tricuspid valve

Right ventricle
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

- Superior vena cava (SVC)
- Inferior vena cava (IVC)
- Coronary sinus
- Right atrium
- Tricuspid valve
- Right ventricle
- Pulmonary trunk
- Pulmonary arteries
- Pulmonary trunk
- Pulmonary semilunar valve

Oxygen-poor blood
Oxygen-rich blood
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

Oxygen-poor blood is carried in two pulmonary arteries to the lungs (pulmonary circuit) to be oxygenated.

Superior vena cava (SVC)
Inferior vena cava (IVC)
Coronary sinus

Right atrium
Tricuspid valve
Right ventricle
Pulmonary semilunar valve
Pulmonary trunk

Oxygen-rich blood

SVC
IVC
Coronary sinus
Right atrium
Tricuspid valve
Right ventricle
Pulmonary semilunar valve
Pulmonary trunk

Oxygen-poor blood

To lungs

Pulmonary capillaries
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

- Pulmonary veins
- Four pulmonary veins

Oxygen-poor blood
Oxygen-rich blood
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

Blood Flow Through the Heart

- Oxygen-poor blood
- Oxygen-rich blood

Pulmonary veins

Left atrium

Left atrium

Four pulmonary veins
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

Oxygen-poor blood
Oxygen-rich blood

- Pulmonary veins
- Left atrium
- Left ventricle
- Mitral valve
- Left ventricle
- Mitral valve
- Four pulmonary veins

© 2013 Pearson Education, Inc.
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

- Oxygen-poor blood
- Oxygen-rich blood

Aorta
Aortic semilunar valve
Aortic Semilunar valve
Left ventricle
Mitral valve Left ventricle
Left atrium
Pulmonary veins
Four pulmonary veins
Figure 18.9 The heart is a double pump, each side supplying its own circuit.

Blood Flow Through the Heart

- **Systemic circuit**: Oxygen-rich blood is delivered to the body tissues.
- **Pulmonary circuit**: Oxygen-poor blood returns to the heart.

Key Labels:
- **Aorta**
- **Aortic semilunar valve**
- **Mitral valve**
- **Left ventricle**
- **Left atrium**
- **Pulmonary veins**

© 2013 Pearson Education, Inc.
Figure 18.9 The heart is a double pump, each side supplying its own circuit. Both sides of the heart pump at the same time, but let’s follow one spurt of blood all the way through the system.

Oxygen-rich blood returns to the heart via the four pulmonary veins. Oxygen-poor blood returns from the body tissues back to the heart.

Oxygen-rich blood is delivered to the body tissues (systemic circuit).

Oxygen-poor blood is carried in two pulmonary arteries to the lungs (pulmonary circuit) to be oxygenated.

Oxygen-poor blood returns to the heart via the four pulmonary veins.
Pathway of Blood Through the Heart

- Equal volumes of blood pumped to pulmonary and systemic circuits
- Pulmonary circuit short, low-pressure circulation
- Systemic circuit long, high-friction circulation
- Anatomy of ventricles reflects differences
  - Left ventricle walls 3X thicker than right
    - Pumps with greater pressure
Figure 18.10  Anatomical differences between the right and left ventricles.
Coronary Circulation

• Functional blood supply to heart muscle itself
  – Delivered when heart relaxed
  – Left ventricle receives most blood supply

• Arterial supply varies among individuals

• Contains many anastomoses (junctions)
  – Provide additional routes for blood delivery
  – Cannot compensate for coronary artery occlusion
Coronary Circulation: Arteries

- Arteries arise from base of aorta
- **Left coronary artery** branches → **anterior interventricular artery** and **circumflex artery**
  - Supplies interventricular septum, anterior ventricular walls, left atrium, and posterior wall of left ventricle
- **Right coronary artery** branches → **right marginal artery** and **posterior interventricular artery**
  - Supplies right atrium and most of right ventricle
The major coronary arteries

(a) The major coronary arteries
Coronary Circulation: Veins

• **Cardiac veins** collect blood from capillary beds

• **Coronary sinus** empties into right atrium; formed by merging cardiac veins
  – **Great cardiac vein** of anterior interventricular sulcus
  – **Middle cardiac vein** in posterior interventricular sulcus
  – **Small cardiac vein** from inferior margin

• Several **anterior cardiac veins** empty directly into right atrium anteriorly
Figure 18.11b Coronary circulation.

Superior vena cava

Anterior cardiac veins

Great cardiac vein

Coronary sinus

Small cardiac vein

Middle cardiac vein

(b) The major cardiac veins
Figure 18.5d  Gross anatomy of the heart.

(d) Posterior surface view

- Aorta
- Left pulmonary artery
- Left pulmonary veins
- Auricle of left atrium
- Left atrium
- Great cardiac vein
- Posterior vein of left ventricle
- Left ventricle
- Apex
- Superior vena cava
- Right pulmonary artery
- Right pulmonary veins
- Right atrium
- Inferior vena cava
- Coronary sinus
- Right coronary artery (in coronary sulcus)
- Posterior interventricular artery (in posterior interventricular sulcus)
- Middle cardiac vein
- Right ventricle
Homeostatic Imbalances

• **Angina pectoris**
  – Thoracic pain caused by fleeting deficiency in blood delivery to myocardium
  – Cells weakened

• **Myocardial infarction** (heart attack)
  – Prolonged coronary blockage
  – Areas of cell death repaired with noncontractile scar tissue
Microscopic Anatomy of Cardiac Muscle

- Cardiac muscle cells striated, short, branched, fat, interconnected, 1 (perhaps 2) central nuclei
- Connective tissue matrix (endomysium) connects to cardiac skeleton
  - Contains numerous capillaries
- T tubules wide, less numerous; SR simpler than in skeletal muscle
- Numerous large mitochondria (25–35% of cell volume)
Figure 18.12a  Microscopic anatomy of cardiac muscle.

- **Nucleus**
- **Intercalated discs**
- **Cardiac muscle cell**
- **Gap junctions**
- **Desmosomes**
Microscopic Anatomy of Cardiac Muscle

• **Intercalated discs** - junctions between cells - anchor cardiac cells
  – *Desmosomes* prevent cells from separating during contraction
  – *Gap junctions* allow ions to pass from cell to cell; electrically couple adjacent cells
  • Allows heart to be **functional syncytium**
    – Behaves as single coordinated unit
Figure 18.12b Microscopic anatomy of cardiac muscle.
Cardiac Muscle Contraction

• Three differences from skeletal muscle:
  – ~1% of cells have automaticity (autorhythmicity)
    • Do not need nervous system stimulation
    • Can depolarize entire heart
  – All cardiomyocytes contract as unit, or none do
  – Long absolute refractory period (250 ms)
    • Prevents tetanic contractions
Cardiac Muscle Contraction

• Three similarities with skeletal muscle:
  – Depolarization opens few voltage-gated fast Na\(^+\) channels in sarcolemma →
    • Reversal of membrane potential from \(-90\) mV to \(+30\) mV
    • Brief; Na channels close rapidly
  – Depolarization wave down T tubules → SR to release Ca\(^{2+}\) →
  – Excitation-contraction coupling occurs
    • Ca\(^{2+}\) binds troponin → filaments slide
Cardiac Muscle Contraction

• More differences
  – Depolarization wave also opens slow $\text{Ca}^{2+}$ channels in sarcolemma $\rightarrow$ SR to release its $\text{Ca}^{2+}$
  – $\text{Ca}^{2+}$ surge prolongs the depolarization phase (plateau)
Cardiac Muscle Contraction

• More differences
  – Action potential and contractile phase last much longer than a neuron.
    • Allow blood ejection from heart
  – Repolarization result of inactivation of Ca\(^{2+}\) channels and opening of voltage-gated K\(^{+}\) channels
    • Ca\(^{2+}\) pumped back to SR and extracellularly
<table>
<thead>
<tr>
<th>Property/component</th>
<th>Striated muscle</th>
<th>Smooth (nonstriated) muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visible banding pattern</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Myosin thick filaments and actin thin filaments</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Tropomyosin and troponin</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Transverse tubules</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Sarcoplasmic reticulum</td>
<td>Well developed</td>
<td>Very little</td>
</tr>
<tr>
<td>Mechanism of contraction</td>
<td>Sliding of thick and thin filaments past each other</td>
<td>Sliding of thick and thin filaments past each other</td>
</tr>
<tr>
<td>Innervation</td>
<td>Somatic nerves</td>
<td>Autonomic nerves</td>
</tr>
<tr>
<td>Initiation of contraction*</td>
<td>Neurogenic</td>
<td>Myogenic</td>
</tr>
<tr>
<td>Source of Ca(^{2+}) for activation†</td>
<td>SR</td>
<td>ECF and SR</td>
</tr>
<tr>
<td>Gap junctions between fibers?</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Speed of contraction</td>
<td>Fast or slow depending on fiber type</td>
<td>Slow</td>
</tr>
<tr>
<td>Clear-cut relationship between length and tension</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>
**Figure 18.13** The action potential of contractile cardiac muscle cells.

1. **Depolarization** is due to Na\(^+\) influx through fast voltage-gated Na\(^+\) channels. A positive feedback cycle rapidly opens many Na\(^+\) channels, reversing the membrane potential. Channel inactivation ends this phase.

2. **Plateau phase** is due to Ca\(^{2+}\) influx through slow Ca\(^{2+}\) channels. This keeps the cell depolarized because few K\(^+\) channels are open.

3. **Repolarization** is due to Ca\(^{2+}\) channels inactivating and K\(^+\) channels opening. This allows K\(^+\) efflux, which brings the membrane potential back to its resting voltage.
Figure 18.13 The action potential of contractile cardiac muscle cells.

1 Depolarization is due to Na\(^+\) influx through fast voltage-gated Na\(^+\) channels. A positive feedback cycle rapidly opens many Na\(^+\) channels, reversing the membrane potential. Channel inactivation ends this phase.
Depolarization is due to Na\(^+\) influx through fast voltage-gated Na\(^+\) channels. A positive feedback cycle rapidly opens many Na\(^+\) channels, reversing the membrane potential. Channel inactivation ends this phase.

Plateau phase is due to Ca\(^{2+}\) influx through slow Ca\(^{2+}\) channels. This keeps the cell depolarized because few K\(^+\) channels are open.
Figure 18.13 The action potential of contractile cardiac muscle cells.

Depolarization is due to Na$^+$ influx through fast voltage-gated Na$^+$ channels. A positive feedback cycle rapidly opens many Na$^+$ channels, reversing the membrane potential. Channel inactivation ends this phase.

Plateau phase is due to Ca$^{2+}$ influx through slow Ca$^{2+}$ channels. This keeps the cell depolarized because few K$^+$ channels are open.

Repolarization is due to Ca$^{2+}$ channels inactivating and K$^+$ channels opening. This allows K$^+$ efflux, which brings the membrane potential back to its resting voltage.
Energy Requirements

• Cardiac muscle
  – Has many mitochondria
    • Great dependence on aerobic respiration
    • Little anaerobic respiration ability
  – Readily switches fuel source for respiration
    • Even uses lactic acid from skeletal muscles
Homeostatic Imbalance

• Ischemic cells $\rightarrow$ anaerobic respiration $\rightarrow$ lactic acid $\rightarrow$
  – High $H^+$ concentration $\rightarrow$ high $Ca^{2+}$ concentration
    • $\rightarrow$ Mitochondrial damage $\rightarrow$ decreased ATP production
    • $\rightarrow$ Gap junctions close $\rightarrow$ fatal arrhythmias
Heart Physiology: Electrical Events

- Heart depolarizes and contracts without nervous system stimulation
  - Rhythm can be altered by autonomic nervous system
Pacemaker (Autorhythmic) Cells

• Have unstable resting membrane potentials (pacemaker potentials or prepotentials) due to opening of slow Na\(^+\) channels
  – Continuously depolarize
• At threshold, Ca\(^{2+}\) channels open
• Explosive Ca\(^{2+}\) influx produces the rising phase of the action potential
• Repolarization results from inactivation of Ca\(^{2+}\) channels and opening of voltage-gated K\(^+\) channels
Action Potential Initiation by Pacemaker Cells

- **Three parts of action potential:**
  - **Pacemaker potential**
    - Repolarization closes $K^+$ channels and opens slow $Na^+$ channels → ion imbalance →
  - **Depolarization**
    - $Ca^{2+}$ channels open → huge influx → rising phase of action potential
  - **Repolarization**
    - $K^+$ channels open → efflux of $K^+$
Figure 18.14 Pacemaker and action potentials of pacemaker cells in the heart.

1. **Pacemaker potential** This slow depolarization is due to both opening of Na\(^+\) channels and closing of K\(^+\) channels. Notice that the membrane potential is never a flat line.

2. **Depolarization** The action potential begins when the pacemaker potential reaches threshold. Depolarization is due to Ca\(^{2+}\) influx through Ca\(^{2+}\) channels.

3. **Repolarization** is due to Ca\(^{2+}\) channels inactivating and K\(^+\) channels opening. This allows K\(^+\) efflux, which brings the membrane potential back to its most negative voltage.
Sequence of Excitation

• Cardiac pacemaker cells pass impulses, in order, across heart in ~220 ms
  – Sinoatrial node ➔
  – Atrioventricular node ➔
  – Atrioventricular bundle ➔
  – Right and left bundle branches ➔
  – Subendocardial conducting network (Purkinje fibers)
Heart Physiology: Sequence of Excitation

• Sinoatrial (SA) node
  – **Pacemaker** of heart in right atrial wall
    • Depolarizes faster than rest of myocardium
  – Generates impulses about 75X/minute (**sinus rhythm**)
    • Inherent rate of 100X/minute tempered by extrinsic factors
• Impulse spreads across atria, and to AV node
Heart Physiology: Sequence of Excitation

• Atrioventricular (AV) node
  – In inferior interatrial septum
  – Delays impulses approximately 0.1 second
    • Because fibers are smaller diameter, have fewer gap junctions
    • Allows atrial contraction prior to ventricular contraction
  – Inherent rate of 50X/minute in absence of SA node input
• Atrioventricular (AV) bundle (bundle of His)
  – In superior interventricular septum
  – Only electrical connection between atria and ventricles
    • Atria and ventricles not connected via gap junctions
Heart Physiology: Sequence of Excitation

• Right and left bundle branches
  – Two pathways in interventricular septum
  – Carry impulses toward apex of heart
Heart Physiology: Sequence of Excitation

• Subendocardial conducting network
  – Complete pathway through interventricular septum into apex and ventricular walls
  – More elaborate on left side of heart
  – AV bundle and subendocardial conducting network depolarize 30X/minute in absence of AV node input

• Ventricular contraction immediately follows from apex toward atria
The sinoatrial (SA) node (pacemaker) generates impulses.

1. The impulses pause (0.1 s) at the atrioventricular (AV) node.

2. The atrioventricular (AV) bundle connects the atria to the ventricles.

3. The bundle branches conduct the impulses through the interventricular septum.

4. The subendocardial conducting network depolarizes the contractile cells of both ventricles.

(a) Anatomy of the intrinsic conduction system showing the sequence of electrical excitation
Figure 18.15b Intrinsic cardiac conduction system and action potential succession during one heartbeat.

Comparison of action potential shape at various locations

(b) Comparison of action potential shape at various locations
Homeostatic Imbalances

• Defects in intrinsic conduction system may cause
  – **Arrhythmias** - irregular heart rhythms
  – Uncoordinated atrial and ventricular contractions
  – **Fibrillation** - rapid, irregular contractions; useless for pumping blood → circulation ceases → brain death
  • Defibrillation to treat
Homeostatic Imbalances

• Defective SA node may cause
  – **Ectopic focus** - abnormal pacemaker
  – AV node may take over; sets junctional rhythm (40–60 beats/min)

• **Extrasystole** (premature contraction)
  – Ectopic focus sets high rate
  – Can be from excessive caffeine or nicotine
Homeostatic Imbalance

• To reach ventricles, impulse must pass through AV node
• Defective AV node may cause
  – Heart block
    • Few (partial) or no (total) impulses reach ventricles
      – Ventricles beat at intrinsic rate – too slow for life
  – Artificial pacemaker to treat
Extrinsic Innervation of the Heart

• Heartbeat modified by ANS via cardiac centers in medulla oblongata
  – Sympathetic $\rightarrow$ $\uparrow$ rate and force
  – Parasympathetic $\rightarrow$ $\downarrow$ rate
  – **Cardioacceleratory center** – sympathetic – affects SA, AV nodes, heart muscle, coronary arteries
  – **Cardioinhibitory center** – parasympathetic – inhibits SA and AV nodes via vagus nerves
The vagus nerve (parasympathetic) decreases heart rate.

Sympathetic cardiac nerves increase heart rate and force of contraction.

Parasympathetic fibers  Sympathetic fibers  Interneurons
Electrocardiography

• Electrocardiogram (ECG or EKG)
  – Composite of all action potentials generated by nodal and contractile cells at given time

• Three waves:
  – **P wave** – depolarization SA node $\rightarrow$ atria
  – **QRS complex** - ventricular depolarization and atrial repolarization
  – **T wave** - ventricular repolarization
Figure 18.17 An electrocardiogram (ECG) tracing.
**Figure 18.18** The sequence of depolarization and repolarization of the heart related to the deflection waves of an ECG tracing.

1. **Atrial depolarization**, initiated by the SA node, causes the P wave.
   - Depolarization: SA node → AV node → Ventricular depolarization begins at apex, causing the QRS complex. Atrial repolarization occurs.

2. With atrial depolarization complete, the impulse is delayed at the AV node.
   - Depolarization: AV node

3. Ventricular depolarization begins at apex, causing the QRS complex. Atrial repolarization occurs.
   - Depolarization: Ventricular depolarization begins at apex

4. Ventricular depolarization is complete.
   - Depolarization: Ventricular depolarization is complete

5. Ventricular repolarization begins at apex, causing the T wave.
   - Repolarization: Ventricular repolarization begins at apex

6. Ventricular repolarization is complete.
   - Repolarization: Ventricular repolarization is complete

**Depolarization**  **Repolarization**
Normal sinus rhythm.

Junctional rhythm. The SA node is nonfunctional, P waves are absent, and the AV node paces the heart at 40–60 beats/min.

Second-degree heart block. Some P waves are not conducted through the AV node; hence more P than QRS waves are seen. In this tracing, the ratio of P waves to QRS waves is mostly 2:1.

Ventricular fibrillation. These chaotic, grossly irregular ECG deflections are seen in acute heart attack and electrical shock.
Electrocardiography

- **P-R interval**
  - Beginning of atrial excitation to beginning of ventricular excitation

- **S-T segment**
  - Entire ventricular myocardium depolarized

- **Q-T interval**
  - Beginning of ventricular depolarization through ventricular repolarization
Heart Sounds

• Two sounds (lub-dup) associated with closing of heart valves
  – First as AV valves close; beginning of systole
  – Second as SL valves close; beginning of ventricular diastole
  – Pause indicates heart relaxation

• Heart murmurs - abnormal heart sounds; usually indicate incompetent or stenotic valves
Cardiac cycle related to sounds & values:
Aortic valve sounds heard in 2nd intercostal space at right sternal margin

Pulmonary valve sounds heard in 2nd intercostal space at left sternal margin

Mitral valve sounds heard over heart apex (in 5th intercostal space) in line with middle of clavicle

Tricuspid valve sounds typically heard in right sternal margin of 5th intercostal space
Mechanical Events: The Cardiac Cycle

- **Cardiac cycle**
  - Blood flow through heart during one complete heartbeat: atrial systole and diastole followed by ventricular systole and diastole
  - **Systole**—contraction
  - **Diastole**—relaxation
  - Series of pressure and blood volume changes
Phases of the Cardiac Cycle

• 1. Ventricular filling—takes place in mid-to-late diastole
  – AV valves are open; pressure low
  – 80% of blood passively flows into ventricles
  – Atrial systole occurs, delivering remaining 20%
  – **End diastolic volume (EDV):** volume of blood in each ventricle at end of ventricular diastole
Phases of the Cardiac Cycle

• 2. Ventricular systole
  – Atria relax; ventricles begin to contract
  – Rising ventricular pressure → closing of AV valves
  – Isovolumetric contraction phase (all valves are closed)
  – In ejection phase, ventricular pressure exceeds pressure in large arteries, forcing SL valves open
  – End systolic volume (ESV): volume of blood remaining in each ventricle after systole
Phases of the Cardiac Cycle

3. **Isovolumetric relaxation** - early diastole
   - Ventricles relax; atria relaxed and filling
   - Backflow of blood in aorta and pulmonary trunk closes SL valves
     - Causes *dicrotic notch* (brief rise in aortic pressure as blood rebounds off closed valve)
     - Ventricles totally closed chambers
   - When atrial pressure exceeds that in ventricles → AV valves open; cycle begins again at step 1
Figure 18.21 Summary of events during the cardiac cycle.
Cardiac Output (CO)

- Volume of blood pumped by each ventricle in one minute
- \( \text{CO} = \text{heart rate (HR)} \times \text{stroke volume (SV)} \)
  - HR = number of beats per minute
  - SV = volume of blood pumped out by one ventricle with each beat
- Normal – 5.25 L/min
Cardiac Output (CO)

• At rest
  – CO (ml/min) = HR (75 beats/min) × SV (70 ml/beat)
    = 5.25 L/min
  – CO increases if either/both SV or HR increased
  – Maximal CO is 4–5 times resting CO in nonathletic people
  – Maximal CO may reach 35 L/min in trained athletes
  – Cardiac reserve - difference between resting and maximal CO
Regulation of Stroke Volume

- \( SV = EDV - ESV \)
  - EDV affected by length of ventricular diastole and venous pressure
  - ESV affected by arterial BP and force of ventricular contraction
- Three main factors affect SV:
  - Preload
  - Contractility
  - Afterload
Regulation of Stroke Volume

• Preload: degree of stretch of cardiac muscle cells before they contract (Frank-Starling law of heart)
  – Cardiac muscle exhibits a length-tension relationship
  – At rest, cardiac muscle cells shorter than optimal length
  – Most important factor stretching cardiac muscle is venous return – amount of blood returning to heart
    • Slow heartbeat and exercise increase venous return
    • Increased venous return distends (stretches) ventricles and increases contraction force
Regulation of Stroke Volume

• **Contractility**—contractile strength at given muscle length, independent of muscle stretch and EDV

• Increased by
  - Sympathetic stimulation $\rightarrow$ increased $\text{Ca}^{2+}$ influx $\rightarrow$ more cross bridges
  - Positive inotropic agents
    • Thyroxine, glucagon, epinephrine, digitalis, high extracellular $\text{Ca}^{2+}$

• Decreased by negative inotropic agents
  - Acidosis, increased extracellular $\text{K}^{+}$, calcium channel blockers
Figure 18.23 Norepinephrine increases heart contractility via a cyclic AMP second messenger system.

1. Norepinephrine binds to β1-adrenergic receptors.
2. G protein (Gₛ) activates adenylate cyclase.
3. ATP is converted to cAMP.
4. cAMP activates protein kinase, causing phosphorylation.
5. Phosphorylation of SR Ca²⁺ channels increases intracellular Ca²⁺ release.
6. Phosphorylation of SR Ca²⁺ pumps speeds Ca²⁺ removal and relaxation.
7. Enhanced actin-myosin interaction.
8. SR Ca²⁺ channel opens, allowing Ca²⁺ uptake.
9. Cardiac muscle force and velocity increase.
Regulation of Stroke Volume

• **Afterload** - pressure ventricles must overcome to eject blood

• Hypertension increases afterload, resulting in increased ESV and reduced SV
Regulation of Heart Rate

- Positive chronotropic factors increase heart rate
- Negative chronotropic factors decrease heart rate
Autonomic Nervous System Regulation

- Sympathetic nervous system activated by emotional or physical stressors
  - Norepinephrine causes pacemaker to fire more rapidly (and increases contractility)
    - Binds to $\beta_1$-adrenergic receptors $\rightarrow$ ↑ HR
    - ↑ contractility; faster relaxation
      - Offsets lower EDV due to decreased fill time
Autonomic Nervous System Regulation

• Parasympathetic nervous system opposes sympathetic effects
  – Acetylcholine hyperpolarizes pacemaker cells by opening K⁺ channels → slower HR
  – Little to no effect on contractility

• Heart at rest exhibits **vagal tone**
  – Parasympathetic dominant influence
• Atrial (Bainbridge) reflex - sympathetic reflex initiated by increased venous return, hence increased atrial filling
  – Stretch of atrial walls stimulates SA node $\rightarrow \uparrow$ HR
  – Also stimulates atrial stretch receptors, activating sympathetic reflexes
Figure 18.22 Factors involved in determining cardiac output.

Exercise (by sympathetic activity, skeletal muscle and respiratory pumps; see Chapter 19)

- Heart rate (allows more time for ventricular filling)

Bloodborne epinephrine, thyroxine, excess Ca2+

- Exercise, fright, anxiety

- Venous return

- EDV (preload)

- Contractility

- ESV

- Sympathetic activity

- Parasympathetic activity

- Stroke volume

- Heart rate

- Cardiac output

Initial stimulus

Physiological response

Result
Chemical Regulation of Heart Rate

• Hormones
  – Epinephrine from adrenal medulla increases heart rate and contractility
  – Thyroxine increases heart rate; enhances effects of norepinephrine and epinephrine

• Intra- and extracellular ion concentrations (e.g., Ca\(^{2+}\) and K\(^{+}\)) must be maintained for normal heart function
Homeostatic Imbalance

- Hypocalcemia $\rightarrow$ depresses heart
- Hypercalcemia $\rightarrow$ increased HR and contractility
- Hyperkalemia $\rightarrow$ alters electrical activity $\rightarrow$ heart block and cardiac arrest
- Hypokalemia $\rightarrow$ feeble heartbeat; arrhythmias
Other Factors that Influence Heart Rate

- **Age**
  - Fetus has fastest HR
- **Gender**
  - Females faster than males
- **Exercise**
  - Increases HR
- **Body temperature**
  - Increases with increased temperature
Homeostatic Imbalances

- **Tachycardia** - abnormally fast heart rate (>100 beats/min)
  - If persistent, may lead to fibrillation
- **Bradycardia** - heart rate slower than 60 beats/min
  - May result in grossly inadequate blood circulation in nonathletes
  - May be desirable result of endurance training
Homeostatic Imbalance

• **Congestive heart failure (CHF)**
  – Progressive condition; CO is so low that blood circulation inadequate to meet tissue needs
  – Reflects weakened myocardium caused by
    • Coronary atherosclerosis—clogged arteries
    • Persistent high blood pressure
    • Multiple myocardial infarcts
    • Dilated cardiomyopathy (DCM)
Homeostatic Imbalance

- Pulmonary congestion
  - Left side fails → blood backs up in lungs
- Peripheral congestion
  - Right side fails → blood pools in body organs → edema
- Failure of either side ultimately weakens other
- Treat by removing fluid, reducing afterload, increasing contractility
Developmental Aspects of the Heart

- Embryonic heart chambers
  - Sinus venosus
  - Atrium
  - Ventricle
  - Bulbus cordis
Figure 18.24 Development of the human heart.

(a) Day 20: Endothelial tubes begin to fuse.
(b) Day 22: Heart starts pumping.
(c) Day 24: Heart continues to elongate and starts to bend.
(d) Day 28: Bending continues as ventricle moves caudally and atrium moves cranially.
(e) Day 35: Bending is complete.
Developmental Aspects of the Heart

• Fetal heart structures that bypass pulmonary circulation
  – **Foramen ovale** connects two atria
    • Remnant is **fossa ovalis** in adult
  – **Ductus arteriosus** connects pulmonary trunk to aorta
    • Remnant - **ligamentum arteriosum** in adult
  – Close at or shortly after birth
Developmental Aspects of the Heart

• Congenital heart defects
  – Most common birth defects; treated with surgery
  – Most are one of two types:
    • Mixing of oxygen-poor and oxygen-rich blood, e.g., septal defects, patent ductus arteriosus
    • Narrowed valves or vessels $\rightarrow$ increased workload on heart, e.g., coarctation of aorta
  – Tetralogy of Fallot
    • Both types of disorders present
Fetus Heart: Before & After Birth

- Ductus venosus
- Foramen ovale
- Ductus arteriosus
Sounds

Normal


Ventricular septal defect
Indomethacin

In humans, postnatal indomethacin can cause closure of the ductus arteriosus, and is used therapeutically when this structure remains patent in preterm neonates (Heymann et al., ’76).

Ductal constriction can also occur in utero after maternal indomethacin administration (Moise et al., ’88).

In addition, infants exposed to prenatal indomethacin were more likely to require surgical ligation of their PDA due to either a lack of response to postnatal indomethacin or a reopening of the duct after initial closure.
The human heart beats more than 3.5 billion times in an average lifetime.

The human embryonic heart begins beating approximately 21 days after conception.

The human heart begins beating at a rate near the mother’s, about 75-80 BPM.

The embryonic heart rate (EHR) then accelerates linearly for the first month of beating, peaking at 165-185 BPM during the early 7th week. This acceleration is approximately 3.3 BPM per day, or about 10 BPM every three days (increase of 100 BPM in the first month).

After peaking at about 9.2 weeks after the normal menstrual period (LMP), it decelerates to about 150 BPM (+/-25 BPM) during the 15th week after the LMP.
HEART RATE & AGE

n = 9043

Embryonic age in days from LMP (± 6 days) = EHR x 0.3 + 6 days
Before 9.2 LMP weeks

Terry J. DuBose, M.S., RDMS; Director Diagnostic Medical Sonography Program
Congenital Heart Diseases: Neonate & Young Infant

- Significant congenital heart disease (CHD) may be diagnosed at virtually any age.

- Some conditions always are discovered in neonates; others rarely are identified during infancy.
Table 1. Mendelian Gene Syndromes Associated with Congenital Heart Anomalies*

<table>
<thead>
<tr>
<th>Etiologic Syndrome</th>
<th>Frequency of Cardiac Anomalies</th>
<th>Distinguishing Features</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All (%)</td>
<td>Distinctive or Most Common</td>
</tr>
<tr>
<td>Autosomal Dominant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adams–Oliver syndrome</td>
<td>20</td>
<td>Left-sided obstruction (eg, COA, parachute MVP, TOF (P)PS, TOF/TOF with PA, ASD, VSD)</td>
</tr>
<tr>
<td>Alagille syndrome</td>
<td>95</td>
<td>PDA</td>
</tr>
<tr>
<td>Char syndrome</td>
<td>60</td>
<td>VSD, ASD, PS, TOF</td>
</tr>
<tr>
<td>Cornelia de Lange syndrome</td>
<td>25</td>
<td>ASD ± other CVM, VSD, TA, TOF, PAPVC, conduction defect</td>
</tr>
<tr>
<td>Holt–Oram syndrome</td>
<td>80</td>
<td>PSV, ASV, COA, HCM</td>
</tr>
<tr>
<td>Neurofibromatosis</td>
<td>2</td>
<td>PSV, ASD, AVSD partial, COA, HCM</td>
</tr>
<tr>
<td>Noonan syndrome</td>
<td>85</td>
<td>PDA, ASD, VSD, left-sided obstruction (eg, COA, HLHS)</td>
</tr>
<tr>
<td>Rubinstein–Taybi syndrome</td>
<td>35</td>
<td>VSD, ASD, VSD, left-sided obstruction (eg, COA, HLHS)</td>
</tr>
<tr>
<td>Williams syndrome</td>
<td>60</td>
<td>SVAS, PS, other left-sided obstructions (eg, ASV, MS, COA)</td>
</tr>
<tr>
<td>Autosomal Recessive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ellis–van Creveld syndrome</td>
<td>60</td>
<td>AVSD, common atrium, ASD primum</td>
</tr>
<tr>
<td>Fryns syndrome</td>
<td>50</td>
<td>ASD, VSD, conotruncal (P)PS</td>
</tr>
<tr>
<td>Keute syndrome</td>
<td>70</td>
<td>ASP, VSD, other CVM</td>
</tr>
<tr>
<td>Smith–Lemli–Opitz syndrome</td>
<td>45</td>
<td>ASO, VSD, complete AVSD, TAPVC</td>
</tr>
<tr>
<td>X-linked Recessive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Simpson–Golabi–Behmel syndrome</td>
<td>25</td>
<td>ASD; VSD; rare, variable cardiomyopathy</td>
</tr>
<tr>
<td>Suspected Gene Etiology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardio–facio–cutaneous syndrome</td>
<td>75</td>
<td>ASO, HCM</td>
</tr>
<tr>
<td>Hall–Hittner syndrome (CHARGE association)</td>
<td>80</td>
<td>Conotruncal/arch, assorted CVMs</td>
</tr>
<tr>
<td>Costello syndrome</td>
<td>60</td>
<td>MVP, AV, thickening HCM, arrhythmia (atrial tachycardia)</td>
</tr>
<tr>
<td>PHACES syndrome</td>
<td>100</td>
<td>COA; LAA, A right: double, cervical aortic arch</td>
</tr>
<tr>
<td>Ritscher–Schinzel syndrome</td>
<td>100</td>
<td>TOF, DORV, AVSD</td>
</tr>
<tr>
<td>-------------------------------------------------------</td>
<td>----------------------------------------------------------</td>
<td>---------------------------------------------</td>
</tr>
<tr>
<td>Heterotaxy, L-TGA</td>
<td>1.6</td>
<td>1.4</td>
</tr>
<tr>
<td>Outflow tract defects, total</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td><strong>4.7</strong></td>
<td>3.3</td>
</tr>
<tr>
<td>D-TGA</td>
<td>2.4</td>
<td>2.3</td>
</tr>
<tr>
<td>Double-outlet right ventricle</td>
<td>2.2</td>
<td>0.7</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td>0.6</td>
<td>0.5</td>
</tr>
<tr>
<td>Atrioventricular septal defect</td>
<td></td>
<td></td>
</tr>
<tr>
<td>With Down syndrome</td>
<td>2.4</td>
<td>2.3</td>
</tr>
<tr>
<td>Without Down syndrome</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Ebstein anomaly</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>Total APVC</td>
<td>0.6</td>
<td>0.7</td>
</tr>
<tr>
<td>Right-sided obstruction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peripheral pulmonic stenosis</td>
<td>7</td>
<td>Not available</td>
</tr>
<tr>
<td>Pulmonic stenosis, atresia</td>
<td><strong>5.9</strong></td>
<td>5.4</td>
</tr>
<tr>
<td>Pulmonic atresia/intact septum</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>Tricuspid atresia</td>
<td>0.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Left-sided obstruction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coarctation of the aorta</td>
<td>3.5</td>
<td>1.4</td>
</tr>
<tr>
<td>Hypoplastic left heart</td>
<td>2.1</td>
<td>1.8</td>
</tr>
<tr>
<td>Aortic valve stenosis</td>
<td>0.8</td>
<td>0.8</td>
</tr>
<tr>
<td>Aortic arch atresia or hypoplasia</td>
<td>0.6</td>
<td>Not available</td>
</tr>
<tr>
<td>Septal defects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td><strong>24.9</strong></td>
<td>11.2</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>10</td>
<td>3.2</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>8.1</td>
<td>0.9</td>
</tr>
<tr>
<td>Other major heart defects</td>
<td>9.7</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td><strong>90.2</strong></td>
<td><strong>48.4</strong></td>
</tr>
</tbody>
</table>

APVC = anomalous pulmonary venous connection, TGA = transposition of the great arteries.

Tetralogy of Fallot

This condition results from a single error: the conus septum develops too far anteriorly giving rise to two unequally proportioned vessels—-a large aorta and a smaller stenotic pulmonary trunk.

The four main characteristics of Tetralogy of Fallot are:
(1) pulmonary stenosis
(2) ventricular septal defect (VSD) of the membranous portion (the septum is displaced too far anteriorly to contribute to the septum)
(3) overriding aorta (the aorta straddles the VSD)
Tricuspid Atresia:
Total Correction: mortality less than 3%

Transposition of the great arteries
Total Correction: mortality less than 2%

Pulmonic stenosis
Total Correction: mortality less than 1%

Truncus Arteriosus (various types)
Mortality is > 10%

Hypoplastic left heart syndrome
Mortality ~10%

Septal defects

At the end of the seventh week - final stage of development. Fetus does not use its lungs, most of the blood is diverted to the systemic circulation. Foramen ovale and the septum primum At birth the child will use its lungs for the first time: more blood pulmonary circulation. The pressure increase in the left atrium will force septum primum to wall- two septa fuse

(common atrial septum)
O1 = Ostium primum
S1 = Septum primum
FO = Foramen ovale
S2 = Septum secundum
ECG: Neonate

- Arrhythmias in fetuses and newborns are relatively common, occurring in up to 90% of newborns and in 1% to 3% of pregnancies (NeoReviews Vol.9 No.6 2008 e242, 2008 American Academy of Pediatrics, Fetal and Neonatal Arrhythmias)

- Weak arterial pulses and right heart overload in the electrocardiogram suggested the diagnosis of hypoplasia of the left heart. Impaired coronary perfusion to portions of the right and left ventricular myocardium. Pulmonary vasoconstriction from hypoxia. Myocardial ischemia on the electrocardiogram (The Journal of Pediatrics Volume 81 (2): 243-250)

- SIDS: A prolonged QT interval may be an important cause for SIDS. (Schwartz et al., The New England Journal of Medicine, 1998 338(24):1709-1714.)
MUSCLE CONTRACTION

Na\textsuperscript{+}  \hspace{1cm} K\textsuperscript{+}

- Ca\textsuperscript{2+} PMCA
- Ca\textsuperscript{2+} NCX
- Ca\textsuperscript{2+} Na\textsuperscript{+}/K\textsuperscript{+} Pump
- Ca\textsuperscript{2+} SERCA
- Voltage gated Ca\textsuperscript{2+} channel
- Ryanodine Receptor
- ATP

Myofilaments

Na\textsuperscript{+}  \hspace{1cm} K\textsuperscript{+}
Brugada syndrome: Genetic disease, abnormal ECG sudden cardiac death (Sudden Unexpected Death Syndrome - SUDS). First described in 1992, ventricular fibrillation mutation in Na+ ion channel.
Ion channels

• Recent evidence indicates that between 5 and 15% of SIDS cases carry potentially lethal loss-of-function mutations in cardiac channelopathy genes.


• Morphological changes in the mitochondrial network likely accompany the uncoupling with mitochondrial fission dampening the signals leading to cardiomyocyte death.

(J Bioenerg Biomembr. 2009 Apr;41(2):133-6. Uncouple my heart: the benefits of inefficiency.)
Sonographer: Pediatric echocardiography
Age-Related Changes Affecting the Heart

- Sclerosis and thickening of valve flaps
- Decline in cardiac reserve
- Fibrosis of cardiac muscle
- Atherosclerosis