Chapter 9
Cardiac Physiology
by Dr. Jay M. Templin
Circulatory System

Heart

Blood Vessels

Blood
Pulmonary and Systemic Circulation
Location of the Heart

RA = Right atrium  LA = Left atrium
RV = Right ventricle  LV = Left ventricle
Anatomy of the Heart: Chambers

Superior vena cava (from head)
Right pulmonary artery
Right pulmonary vein
Pulmonary semilunar valve
Right atrium
Right atrioventricular (AV) valve
Inferior vena cava (from body)
Right ventricle

Aorta
Left pulmonary artery
Left pulmonary vein
Left atrium
Left atrioventricular (AV) valve
Aortic semilunar valve
Left ventricle
Interventricular septum

Arrows indicate direction of the blood flow.
Red = O₂-rich blood
Blue = O₂-poor blood
Anatomy of the Heart: Blood Vessels

- Superior vena cava (from head)
- Right pulmonary artery
- Right pulmonary vein
- Pulmonary semilunar valve
- Right atrium
- Right atrioventricular (AV) valve
- Inferior vena cava (from body)
- Right ventricle
- Aorta
- Left pulmonary artery
- Left pulmonary vein
- Left atrium
- Left atrioventricular (AV) valve
- Aortic semilunar valve
- Left ventricle
- Interventricular septum

Arrows indicate direction of the blood flow.

- Red = O2-rich blood
- Blue = O2-poor blood
Blood Flow in the Heart

High pressure, High resistance

Low pressure, Low resistance
Heart Valve Action

When pressure is greater behind the valve, it opens.

Valve opened

When pressure is greater in front of the valve, it closes. Note that when pressure is greater in front of the valve, it does not open in the opposite direction; that is, it is a one-way valve.

Valve closed; does not open in opposite direction
Right atrium
Right AV valve
Chordae tendineae
Septum
Direction of backflow of blood
Papillary muscle
Right ventricle
Transverse Heart Structure

- Tricuspid valve
- Mitral valve
- Semilunar valve
- Right AV valve
- Left AV valve
- Aortic valve
- Pulmonary semilunar valve
- Ventricular myocardium
- Fibrous rings

(Rear)

(Front)
Valve System

Notice:

Valves are found between atria and ventricles or between ventricles and arteries (aorta and pulmonary vein)

No valves between atria and veins (pulmonary vein and vena cava) WHY?

http://www.smm.org/heart/heart/pumping-f.htm

MOVIE
Tissue Structure in the Heart

Endocardium - Epithelial tissue

Myocardium - Cardiac muscle tissue

Epicardium - Connective tissue

Pericardial sac and Pericardial fluid
Cell Structure in the Myocardium

- Plasma membranes of adjacent cardiac muscle fibers
- Desmosome
- Action potential
- Gap junction
Cells in Myocardium

**Contractile cells**

**Pacemaker cells**

1) Sinoatrial node

2) Atrioventricular node

3) Atrioventricular bundle

4) Purkinje cells
Function of Pacemaker Cells

![Diagram showing the function of pacemaker cells](http://www.interactivephysiology.com/demo/systems/buildframes.html?cardio/actnpot/01)
Pacemaker Cells

Cell A

Cell B

Membrane potential (mV)

Time (msec)
Whole train will go **70 mph**
(heart rate set by SA node, the fastest autorhythmic tissue).

Train will go **50 mph**
(the next fastest autorhythmic tissue, the AV node, will set the heart rate).
Complete Heart Block

First part of train will go **70 mph**; last part will go **30 mph**
(atria will be driven by SA node; ventricles will assume own, much slower rhythm).

Premature Beat (Extrasystole)

Train will be driven by ectopic focus, which is now going faster than the SA node
(the whole heart will be driven more rapidly by an abnormal pacemaker).
1) Atrial excitation should be completed before ventricular excitation

AV nodal delay ~100 msec
2) Each chamber should contract as a unit.
3) Each pairs of atria or ventricles should contract at the same time.
Interatrial pathway

SA node

AV node

Internodal pathway

Purkinje fibers

Bundle of His

Right atrium

Left atrium

Right ventricle

Left ventricle
Action Potential in Cardiac Muscle

- Plateau phase of action potential
- Threshold potential

http://www.interactivephysiology.com/demo/systems/buildframes.html?cardio/actnpot/01
Action potential in cardiac contractile cell

Travels down T tubules

Entry of small amount of \( \text{Ca}^{2+} \) from ECF

Release of large amount of \( \text{Ca}^{2+} \) from sarcoplasmic reticulum

Cytosolic \( \text{Ca}^{2+} \)

Troponin-tropomyosin complex in thin filaments pulled aside

Cross-bridge cycling between thick and thin filaments

Thin filaments slide inward between thick filaments

Contraction
Refractory Period in Cardiac Muscle

No summation of contractions in cardiac muscles WHY?
Electrocardiogram (ECG)

ECG reflect the flow of electrical currents during heart contraction
Electrocardiogram (ECG)
Single Lead Recording
Electrocardiogram (ECG)

- **P wave** = Atrial depolarization
- **PR segment** = AV nodal delay
- **QRS complex** = Ventricular depolarization (atria repolarizing simultaneously)
- **ST segment** = Time during which ventricles are contracting and emptying
- **T wave** = Ventricular repolarization
- **TP interval** = Time during which ventricles are relaxing and filling

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200 msec
NORMAL RATE AND RHYTHM

ABNORMALITIES IN RATE

Tachycardia

Bradycardia-appositive of Tachycardia
ABNORMALITIES IN RHYTHM

Extrasystole (premature beat)

Ventricular fibrillation

Complete heart block
Myocardial infarction
Cardiac Cycle: Systole vs. Diastole

1) Ventricular filling

2) Isovolumetric ventricular contraction

3) Ventricular ejection

4) Isovolumetric ventricular relaxation
Passive filling during ventricular and atrial diastole

Atrial contraction

Right atrium

Left atrium

Right ventricle

Left ventricle

Ventricular filling
Isovolumetric ventricular contraction
Dicrotic Notch

DN represents the closure of the aortic valve during relaxation of ventricles

http://www.interactivephysiology.com/demo/systems/systems/cardio/index.html
Changes in Volume

Stroke volume ~70 mL

http://www.interactivephysiology.com/demo/systems/systems/cardio/index.html
Cardiac Cycle

http://www.interactivephysiology.com/demo/systems/systems/cardio/index.html
Heart Sounds

http://www.interactivephysiology.com/demo/systems/systems/cardio/index.html
Laminar vs. Turbulent Flow

Laminar flow (does not create any sound)

Turbulent flow (can be heard)
Cardiac Output at Rest

**Cardiac Output:** VOLUME of blood pumped by each ventricle per minute

Cardiac Output = Heart Rate X Stroke Volume

\[ (CO) = (HR) \times (SV) \]

IF: Average heart rate = 70 beats/min
Stroke volume = 70 mL

Cardiac Output at Rest ~ 5 L/min
Cardiac Output during Exercise

Cardiac Output during Exercise ~ 25 L/min

How does the organism regulate cardiac output?

- Parasympathetic activity
  - Innervate atria via vagus nerve

- Sympathetic activity (and epinephrine)
  - Innervate atria and ventricles
Regulation of Cardiac Output

**Sympathetic**
- Increase excitability
  - Increase rate of depolarization
  - Decrease AV nodal delay
- Increase heart rate
- Increase contraction of atrium and ventricles
- Increase secretion of **adrenaline** from adrenal medulla

**Parasympathetic**
- Decrease excitability
  - Decrease rate of depolarization
  - Decrease AV nodal delay
- Decrease heart rate
- Decrease contraction of atrium ONLY
- No effect on adrenaline secretion
Sympathetic Regulation of Heart Rate

Decrease K+ permeability

Threshold potential
Parasympathetic Regulation of Heart Rate

- Increase K+ permeability

![Graph showing membrane potential over time with threshold potential indicated.]
Right atrium Left atrium

SA node

AV node

Right ventricle Left ventricle

Sympathetic

Decrease AV nodal delay

Parasympathetic

Increase AV nodal delay
Regulation of Cardiac Output

Heart rate

Parasympathetic activity
- Innervate atria via vagus nerve

Sympathetic activity (and epinephrine)
+ Innervate atria and ventricles
Regulation of Stroke Volume

Stroke volume

Extrinsic control

Strength of cardiac contraction

Intrinsic control

End-diastolic volume

Sympathetic activity (and epinephrine)

Intrinsic control

Venous return

Regulation factors:
- Extrinsic control:
  - Sympathetic activity (and epinephrine)

- Intrinsic control:
  - End-diastolic volume
  - Venous return

Cardiac factors:
- Strength of cardiac contraction
Frank-Starling Law

The heart will pump out all the blood returned to it

Increase in stroke volume at same end-diastolic volume

Frank-Starling curve on sympathetic stimulation

Normal Frank-Starling curve
Effect of Sympathetic Stimulation on Stroke Volume

End-diastolic volume
135 ml

Stroke volume
70 ml

End-systolic volume
65 ml

Normal SV
SV following sympathetic stimulation:

Due to increase Ca$^{2+}$ influx during muscle fiber depolarization
SV following sympathetic stimulation and increase end-diastolic volume

End-diastolic volume
175 ml

Stroke volume
140 ml

End-systolic volume
35 ml
Heart rate is determined by the balance between sympathetic and parasympathetic stimulation.

Notice that at rest parasympathetic activation have the dominant effect.
Heart Failure
Inability of the heart to contract

![Diagram showing normal and failing hearts with respect to stroke volume and end-diastolic volume.]

- Normal stroke volume
- Decrease in stroke volume
- Stroke volume with uncompensated heart failure
- Normal end-diastolic volume
- Failing heart

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Heart Failure

Caused by:

1) Damage heart muscle

2) Excess pumping against increased load (for example after hypertension or damaged valves)
Area of cardiac muscle deprived of blood supply if coronary vessel is blocked at point A:

- Right coronary artery
- Right ventricle

Area of cardiac muscle deprived of blood supply if coronary vessel is blocked at point B:

- Left coronary artery
- Left ventricle
Coronary Blood Flow
Supply heart muscle with $O_2$ and nutrients
Coronary Blood Flow

Aortic pressure

Blood flow in left coronary artery

Systole

Diastole
CARDIAC MYOPATHIES: DAMAGE TO CARDICA MUSCLE FIBERS

Myocardial infarction
Compensatory Measures Against Heart Failure

Increased blood volume by reducing water and Na⁺ secretion
Compensatory Measures Against Heart Failure: Sympathetic stimulation

- Normal end-diastolic volume
- Normal stroke volume

- Normal heart
- Failing heart with sympathetic stimulation
- Failing heart without sympathetic stimulation

Increase in end-diastolic volume
Interdependence of Blood Flow and Oxygen Need

Metabolic activity of cardiac muscle cells ↑ oxygen need

↑ Adenosine

Vasodilation of coronary vessels

↑ Blood flow to cardiac muscle cells

↑ Oxygen available to meet ↑ oxygen need
Factors that Affect Blood Flow

Vascular Spams
Atherosclerosis
Thromboembolism
Atherosclerotic Plaque

- Collagen-rich smooth muscle cap of plaque
- Normal blood vessel wall
- Lipid-rich core of plaque
- Endothelium

Plaque
Formation of Atherosclerotic Plaque

Blood vessel damage

Inflammatory response

Deposition and oxidation of low-density lipoprotein (bad cholesterol) (Prevented by Vit E, Vit C, beta-carotene)

Recruitment of macrophages and fibroblasts leading to formation of collagen cap

Ca²⁺ precipitation and hardening of blood vessels
Formation of Thrombus

Rupture of collagen cap

Platelet aggregation and formation of blood clot (thrombus)
Thrombembolism

Leading cause of strokes (brain) and myocardial ischemia (heart muscle)
Consequences of Thrombembolism

Angina Pectoris or Heart Pain due to narrowing of coronary blood vessels

Heart Attack occurs when blood vessels are completely plugged