Circulatory System

Heart (pump)

Blood Vessels
(passageway and distribution channels)

Blood (transport medium)
Pulmonary and Systemic Circulation
Location of the Heart

- Sternum
- Ribs
- 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
Arrows indicate direction of the blood flow.

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

© 2001 Brooks/Cole - Thomson Learning

O₂-rich blood
O₂-poor blood
The Heart is a dual pump

The heart is divided into left and right halves separated by the septum.

Left and right halves behave as two separate pumps.
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.

= O₂-rich blood
= O₂-poor blood

© 2001 Brooks/Cole - Thomson Learning
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
Valve System in the Heart

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

© 2001 Brooks/Cole - Thomson Learning
Transverse Heart Structure

(Rear)
- Right AV valve
- Tricuspid valve
- Fibrous rings
- Left AV valve
- Mitral OR bicuspic valve

(Front)
- Aortic valve
- Semilunar valve
- Ventricular myocardium
- Pulmonary semilunar valve Semilunar valve
Right atrium

Direction of backflow of blood

Right AV valve

Chordae tendineae

Septum

Papillary muscle

Right ventricle
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) because atrial pressure is similar to venous pressure.

MOVIE!
Tissue Structure in the Heart

Endocardium—Epithelial layer that covers the entire circulatory system

Myocardium—Cardiac muscle tissue

Epicardium—Connective tissue covering the heart

Pericardial sac and Pericardial fluid
Cells in Myocardium

**Contractile cells**

**Pacemaker cells**

1) Sinoatrial node

2) Atrioventricular node

3) Atrioventricular bundle

4) Purkinje cells
There are no gap junctions between atrial and ventricular cardiac cells.
The heart beats because pacemaker cells generate a depolarizing potential that can contract cardiac muscle cells.

The heart is **autorhythmic**
Cells in Myocardium

Contractile cells

Pacemaker cells

1) Sinoatrial node
2) Atrioventricular node
3) Atrioventricular bundle
4) Purkinje cells

http://www.interactivephysiology.com/demo/systems/buildframes.html?cardio/actnpot/01
Generation of Pacemaker Potential

Pacemaker potential is calcium dependent!

http://www.interactivephysiology.com/demo/systems/buildframes.html?cardio/actnpot/01
Generation of Action Potentials in Pacemaker cells

Between spikes: Leak $K^+$ current decreases, leak $Na^+$ current remains constant

T-type voltage-gated $Ca^{+2}$ channels open

Membrane potential reaches threshold-activation of L-type voltage-gated $Ca^{+2}$ channels

Membrane depolarization opens voltage-gated $K^+$ channels=repolarization

http://www.interactivephysiology.com/demo/systems/buildframes.html?cardio/actnpot/01
Pacemaker Cells

Pacemaker cells are located in:
1) Sinoatrial node (SA node)
2) Atrioventricular node (AV node)
3) Bundle of His (atrioventricular node)
4) Purkinje cells

Pacemaker cells generate action potentials at various rates
Pacemaker cells have different pacemaking rates

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).
Spreading of Cardiac Excitation

- Sinoatrial (SA) node
- Right atrium
- Internodal pathway
- Right branch of bundle of His
- Right ventricle
- Left atrium
- Atrioventricular (AV) node
- Left branch of bundle of His
- Left ventricle
- Purkinje fibers
- Inneratrial pathway
1) Atrial excitation should be completed before ventricular excitation. 

**WHY?**

Delay b/ SA & AV nodes = 30 msec.
2) Each chamber should contract as a unit.
3) Each pairs of atria or ventricles should contract at the same time

AV delay=100 msec
Interatrial pathway

Right atrium

SA node

Internodal pathway

AV node

Right ventricle

Purkinje fibers

Left atrium

Bundle of His

Right ventricle

Left ventricle
Cardiac muscle cells have a very hyperpolarized resting membrane potential (-90 mV) WHY?

Action potential in cardiac muscle cell is generated by the flow of sodium and calcium ions

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 Ca\(^{2+}\) from ECF

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

Cytosolic 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
Cardiac muscle contraction is long lasting because:

1) it relies mainly on calcium release from intracellular stores, and
2) slow removal from intracellular space
Skeletal Muscle Contraction
Summation and Tetanus

http://harveyproject.science.wayne.edu/development/muscle/twitch~1.htm
Cardiac muscles have a long refractory period which prevents tetanus. No summation of cardiac muscle contractions. Why?
Electrocardiogram (ECG)

ECG reflect the flow of electrical currents during a heart contraction that spread through body fluids.

The shape of the ECG wave depends on the orientation of the recording electrodes.
Electrocardiogram (ECG)
The shape of the ECG wave is correlated with specific cardiac events

- 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
P wave: atrial depolarization
PR: AV nodal delay
QRS complex: ventricular depolarization, atrial repolarization
ST: ventricular contraction and emptying
T wave: ventricular repolarization
TP: ventricular relaxation and filling
Abnormal ECG

1) Normal heart rate (70 beats/min): time between two consecutive QRS complexes

2) Abnormal heart rate: tachycardia, bradycardia

3) Abnormal heart rhythm: arrhythmia, extrasystole or premature heart beat, atrial fibrillation, ventricular fibrillation, heart block

4) Cardiac myopathies: acute myocardial infarction caused by myocardial ischemia
NORMAL RATE AND RHYTHM

ABNORMALITIES IN RATE

Tachycardia

Bradycardia-opposite of Tachycardia
ABNORMALITIES IN RHYTHM

- Extrasystole (premature beat)
- Ventricular fibrillation: uncontrolled depolarization of ventricles
- Complete heart block: ventricles fail to be stimulated
CARDIAC MYOPATHIES: DAMAGE TO CARDIAC MUSCLE FIBERS

Myocardial infarction

http://www.skillstat.com/ECG_Sim_demo.html
Cardiac Cycle: Systole vs. Diastole

1) Ventricular filling (ventricular diastole)

2) Isovolumetric ventricular contraction (ventricular systole)

3) Ventricular ejection

4) Isovolumetric ventricular relaxation

- Electrocardiogram
- Aortic pressure (mm Hg)
- Left ventricular pressure (mm Hg)
- Left atrial pressure (mm Hg)
- End-diastolic volume (ml)
- End-systolic volume
- Heart sounds
Passive filling during ventricular and atrial diastole

Atrial contraction

Ventricular filling

Right atrium

Right ventricle

Left atrium

Left ventricle
Isovolumetric ventricular contraction
Ventricular ejection

Isovolumetric ventricular relaxation

Ventricular emptying
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: amount of blood pumped out by each ventricle during a heart beat

**Stroke volume** ≈ 70 mL
Cardiac Cycle

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

Heart sounds are caused by vibrations within the walls of ventricles and arteries when valves close.
Abnormal Heart Sounds
Are caused by turbulent flow

Laminar flow (does not create any sound)

Turbulent flow (can be heard)
Turbulent Flow

Cause by:

1) **Stenotic (stiff) valves:** valves that can not open completely. Generate whistling sound

2) **Insufficient valves:** valves that can not close completely. Generate gurgling sound

3) **Rheumatic fever:** autoimmune disease causing stenotic or insufficient valves
Changes in Volume

Stroke volume: amount of blood pumped out by each ventricle during a heart beat

**Stroke volume** ~70 mL
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 ~ 5 L/min (at rest)

Cardiac reserve ~ 20-25 L/min (physical exercise)
Regulation of Cardiac Output
Stimulation of the SA node by the autonomic NS regulates heart rate
(at rest parasympathetic activity prevails)

Heart rate

Parasympathetic activity

Innervate atria via vagus nerve (X cranial nerve)

ACh, mAChR

Sympathetic activity (and epinephrine)

Innervate atria and ventricles

NA, β1
Regulation of Cardiac Output

**Sympathetic**

- Increase excitability
  - increase rate of depolarization in SN node
  - 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 in SA node
  - increase AV nodal delay
- Decrease heart rate
- Decrease contraction of atrium ONLY
- No effect on adrenaline secretion
Generation of Action Potentials in Pacemaker cells

**Between spikes:** Leak $K^+$ current decreases, leak $Na^+$ current remains constant

T-type voltage-gated $Ca^{+2}$ channels open

Membrane potential reaches threshold-activation of L-type voltage-gated $Ca^{+2}$ channels

Membrane depolarization opens voltage-gated $K^+$ channels=repolarization

http://www.interactivephysiology.com/demo/systems/buildframes.html?cardio/actnpot/01
Parasympathetic Regulation of Heart Rate

- Increase K⁺ permeability
- Lower membrane potential

Threshold potential
Sympathetic Regulation of Heart Rate

- Decrease K⁺ permeability

Threshold potential
Regulation of Cardiac Output

The autonomic NS regulates the rate of depolarization of the pacemaker potential and the conduction velocity of action potentials in the AV pathway (AV nodal delay)
Cardiac Output during Exercise

Cardiac Output during Exercise ~ 25 L/min

How does the organism regulate cardiac output?

Heart rate

Parasympathetic activity
- Innervate atria via vagus nerve

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

SV = amount of blood pumped out by each ventricle during a heart beat

**Extrinsic control**
- Stroke volume
- Strength of cardiac contraction

**Intrinsic control**
- Sympathetic activity (and epinephrine)
- End-diastolic volume
- Venous return
Frank-Starling Law

The heart will pump out all the blood returned to it.

Increased venous return results in increased stroke volume.
Effect of Sympathetic Stimulation on Stroke Volume

Normal SV

End-diastolic volume
135 ml

Stroke volume
70 ml

End-systolic volume
65 ml
SV following sympathetic stimulation:

Due to increase Ca^{2+} influx during muscle fiber depolarization
SV following sympathetic stimulation and increase end-diastolic volume
Heart rate is determined by the balance between sympathetic and parasympathetic stimulation.

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

- Normal heart
- Failing heart

Stroke volume

Normal stroke volume
Decrease in stroke volume
Stroke volume with uncompensated heart failure

Normal end-diastolic volume
End-diastolic volume
Heart Failure

Caused by:

1) Damaged heart muscle

2) Excess pumping against increased load (for example in the presence of a stenotic semilunar valve or increased blood pressure)
Compensatory Measures Against Heart Failure: Sympathetic stimulation

Normal stroke volume

Normal end-diastolic volume

Failing heart with sympathetic stimulation

Failing heart without sympathetic stimulation

Increase in end-diastolic volume
Compensatory Measures Against Heart Failure

Increased blood volume by reducing water and Na⁺ secretion
Coronary Blood Flow
Supply heart muscle with $O_2$ and nutrients
Coronary Blood Flow

Blood supply to heart tissue occurs during diastole because coronary arteries are compressed during systole and entrance to coronary artery is blocked by an open aortic valve.
Interdependence of Blood Flow and Oxygen Need in Cardiac Tissue

Under extraneous conditions, blood supply to the heart increases by vasodilation to allow more blood (and O₂) to flow to cardiac muscle.

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
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

© Brooks/Cole - Thomson Learning
CARDIAC MYOPATHIES: DAMAGE TO CARDIAC MUSCLE FIBERS

Myocardial infarction
Factors that Affect Blood Flow
(leading to myocardial ischemia)


**Atherosclerosis**: occlusion of coronary blood vessels caused by the formation of atherosclerotic plaque

**Thromboembolism**: occlusion of coronary blood vessels caused by formation of a thrombus
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

Ca^{2+} precipitation around the plaque and hardening of blood vessels
Formation of Thrombus

Rupture of collagen cap/or collagen exposure in damaged vessels allows platelets aggregation

Platelet aggregation and formation of blood clot (thrombus)

http://www.sci.sdsu.edu/movies/coronary/
Thromboembolism

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

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

Heart Attack occurs when coronary blood vessels are completely plugged