Welcome to

University of Buea

HSC 205

2011 2012

Physiology by Dr. Salah A. Martin

Faculty of Health Sciences

Health Science Courses

Heart Anatomy

- Approximately the size of your fist
- Location
  - Superior surface of diaphragm
  - Left of the midline
  - Anterior to the vertebral column, posterior to the sternum

Heart Anatomy

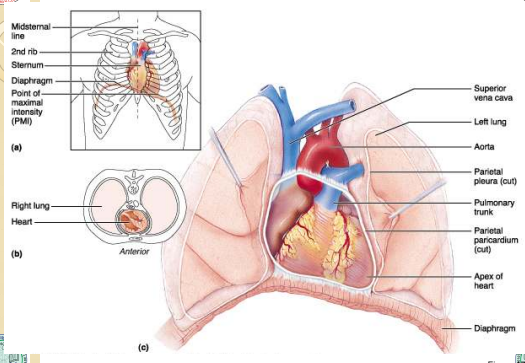

Figure 17.1

Heart Covering

- Pericardial physiology
  - Protects and anchors heart
  - Prevents overfilling

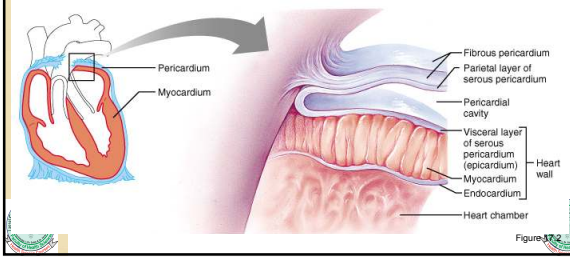

Figure 17.2

Heart Covering

- Pericardial anatomy
  - Fibrous pericardium
  - Serous pericardium (separated by pericardial cavity)
  - Epicardium (visceral layer)

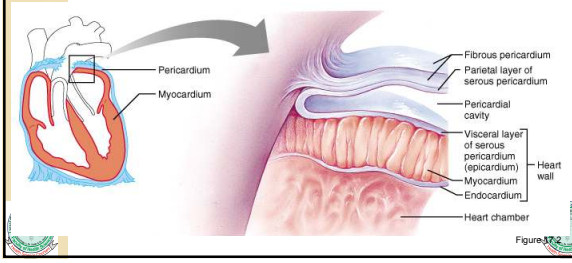

Figure 17.2

Heart Wall

- Epicardium – visceral layer of the serous pericardium
- Myocardium – cardiac muscle layer forming the bulk of the heart
- Fibrous skeleton of the heart – crisscrossing, interlacing layer of connective tissue
- Endocardium – endothelial layer of the inner myocardial surface

External Heart: Major Vessels of the Heart (Anterior View)

- Returning blood to the heart
  - Superior and inferior venae cavae
  - Right and left pulmonary veins
- Conveying blood away from the heart
  - Pulmonary trunk, which splits into right and left pulmonary arteries
  - Ascending aorta (three branches) – brachiocephalic, left common carotid, and subclavian arteries

External Heart: Vessels that Supply/Drain the Heart (Anterior View)

- Arteries – right and left coronary (in atrioventricular groove), marginal, circumflex, and anterior interventricular
- Veins – small cardiac vein, anterior cardiac veins, and great cardiac vein

External Heart: Anterior View

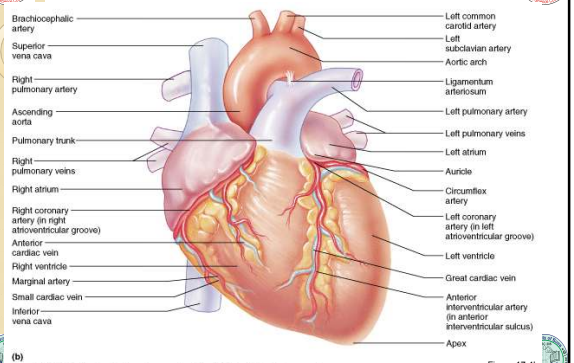

Figure 17.4b

External Heart: Major Vessels of the Heart (Posterior View)

- Returning blood to the heart
  - Right and left pulmonary veins
  - Superior and inferior venae cavae
- Conveying blood away from the heart
  - Aorta
  - Right and left pulmonary arteries

External Heart: Vessels that Supply/Drain the Heart (Posterior View)

- Arteries – right coronary artery (in atrioventricular groove) and the posterior interventricular artery (in interventricular groove)
- Veins – great cardiac vein, posterior vein to left ventricle, coronary sinus, and middle cardiac vein

External Heart: Posterior View

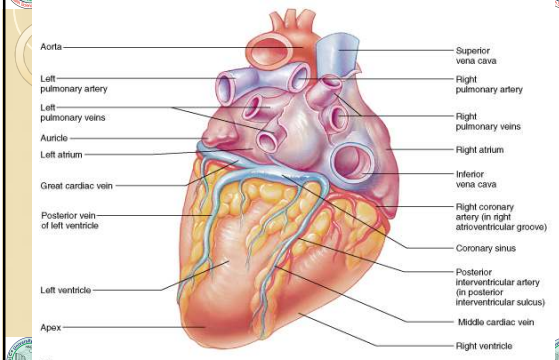

Figure 17.4d

Gross Anatomy of Heart: Frontal Section

- Frontal section showing interior chambers and valves
- Major vessels leading to and from the heart

Gross Anatomy of Heart: Frontal Section

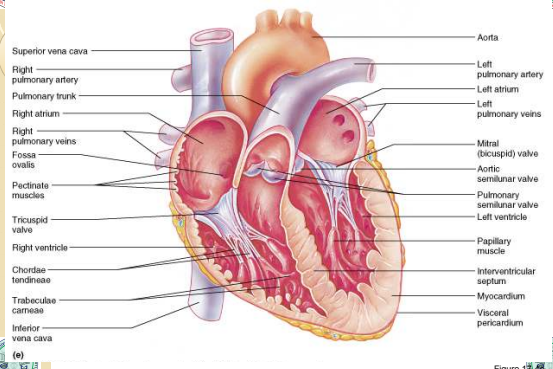

Figure 17.4e

Atria of the Heart

- Atria are the receiving chambers of the heart
- Each atrium has a protruding auricle
- Pectinate muscles mark atrial walls
- Blood enters right atria from superior and inferior venae cavae and coronary sinus
- Blood enters left atria from pulmonary veins

Ventricles of the Heart

- Ventricles are the discharging chambers of the heart
- Papillary muscles and trabeculae carneae muscles mark ventricular walls
- Right ventricle pumps blood into the pulmonary trunk
- Left ventricle pumps blood into the aorta

Pathway of Blood through the Heart and Lungs

- Right atrium → tricuspid valve → right ventricle
- Right ventricle → pulmonary semilunar valve → pulmonary arteries → lungs
- Lungs → pulmonary veins → left atrium
- Left atrium → bicuspid valve → left ventricle
- Left ventricle → aortic semilunar valve → aorta
- Aorta → systemic circulation

Pathway of Blood through the Heart and Lungs

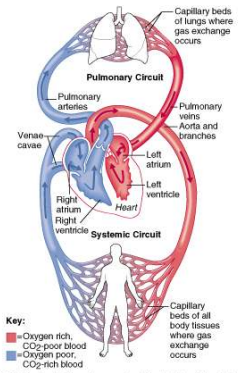

Figure 17.5

Coronary Circulation

- Coronary circulation is the functional blood supply to the heart
- Collateral routes insure blood delivery to heart even if major vessels are occluded

Coronary Circulation

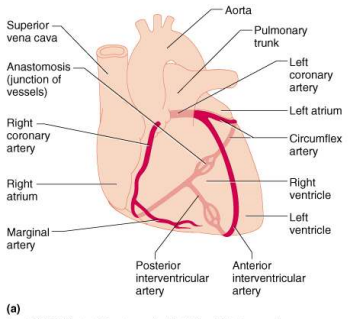

Figure 17.7a

Coronary Circulation

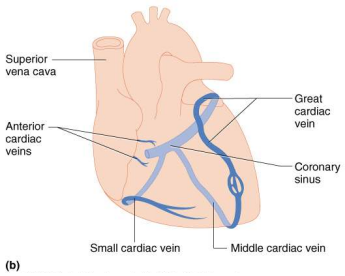

Figure 17.7b

Heart Valves

- Heart valves insure unidirectional blood flow through the heart
- Atrioventricular (AV) valves lie between the atria and the ventricles
- AV valves prevent backflow into the atria when ventricles contract
- Chordae tendineae anchor AV valves to papillary muscles

Heart Valves

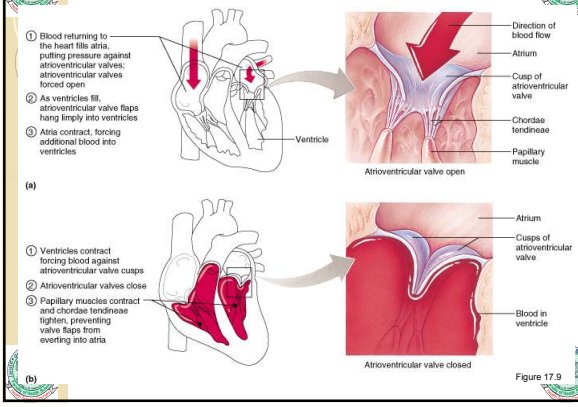

Figure 17.9

Heart Valves

- Aortic semilunar valve lies between the left ventricle and the aorta
- Pulmonary semilunar valve lies between the right ventricle and pulmonary trunk
- Semilunar valves prevent backflow of blood into the ventricles

Heart Valves

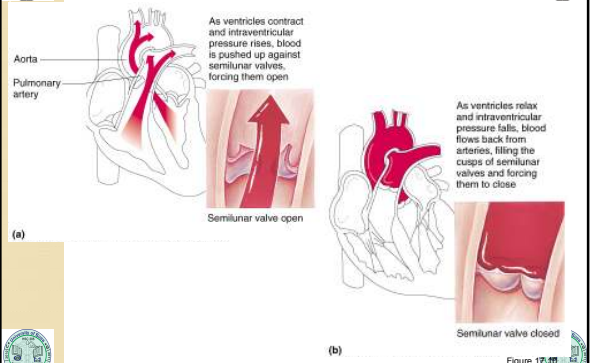

Figure 17.16

Microscopic Heart Muscle Anatomy

- Cardiac muscle is striated, short, fat, branched, and interconnected
- Connective tissue endomysium acts as both tendon and insertion
- Intercalated discs anchor cardiac cells together and allow free passage of ions
- Heart muscle behaves as a functional syncytium

Microscopic Heart Muscle Anatomy

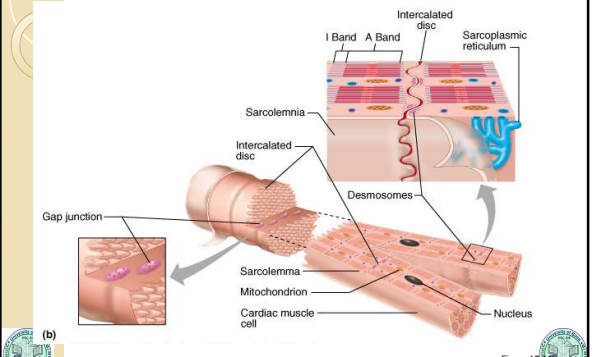

Figure 17.17b

Cardiac Muscle Contraction

- Heart muscle:
  - Is stimulated by nerves and self-excitable (automaticity)
  - Contracts as a unit
  - Has a long (250 ms) absolute refractory period
- Cardiac muscle contraction is similar to skeletal muscle contraction

Heart Physiology: Intrinsic Conduction System

- Autorhythmic cells:
  - Initiate action potentials
  - Have unstable resting potentials called *pacemaker potentials*
  - Use calcium influx (rather than sodium) for rising phase of the action potential

Heart Physiology: Intrinsic Conduction System

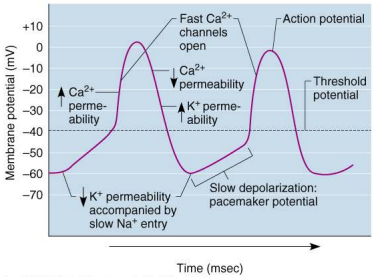

Figure 17.18

Heart Physiology: Sequence of Excitation

- Sinoatrial (SA) node generates impulses about 75 times/minute
- Atrioventricular (AV) node delays the impulse approximately 0.1 second
- Impulse passes from atria to ventricles via the atrioventricular bundle (bundle of His)

Heart Physiology: Sequence of Excitation

- AV bundle splits into two pathways in the interventricular septum (bundle branches)
  - Bundle branches carry the impulse toward the apex of the heart
  - Purkinje fibers carry the impulse to the heart apex and ventricular walls

Heart Physiology: Sequence of Excitation

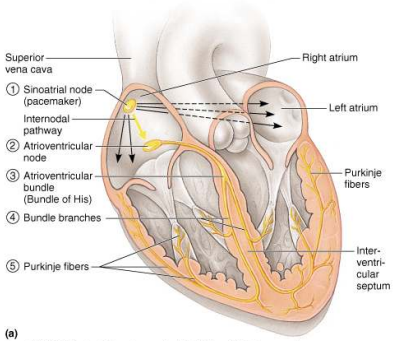

Figure 17.14a

Extrinsic Innervation of the Heart

- The heart is stimulated by the sympathetic cardioacceleratory center
- The heart is inhibited by the parasympathetic cardioinhibitory center

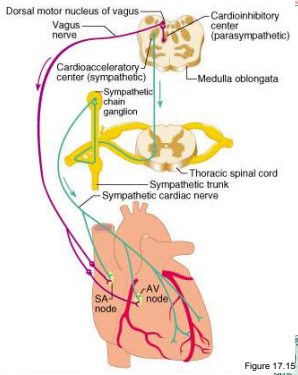

Figure 17.15

Electrocardiography

- Electrical activity is recorded by electrocardiogram (ECG)
- P wave corresponds to depolarization of SA node
- QRS complex corresponds to ventricular depolarization
- T wave corresponds to ventricular repolarization
- Atrial repolarization record is masked by the larger QRS complex

Electrocardiography

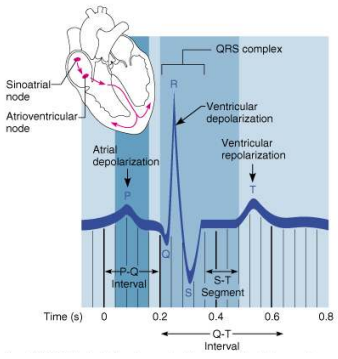

Figure 17.16

Cardiac Cycle

- Cardiac cycle refers to all events associated with blood flow through the heart
  - Systole – contraction of heart muscle
  - Diastole – relaxation of heart muscle

Phases of the Cardiac Cycle

- Ventricular filling – mid-to-late diastole
  - Heart blood pressure is low as blood enters atria and flows into ventricles
  - AV valves are open, then atrial systole occurs

Phases of the Cardiac Cycle

- Ventricular systole
  - Atria relax
  - Rising ventricular pressure results in closing of AV valves
  - Isovolumetric contraction phase
  - Ventricular ejection phase opens semilunar valves

Phases of the Cardiac Cycle

- Isovolumetric relaxation – early diastole
  - Ventricles relax
  - Backflow of blood in aorta and pulmonary trunk closes semilunar valves
- Dicrotic notch – brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves

Phases of the Cardiac Cycle

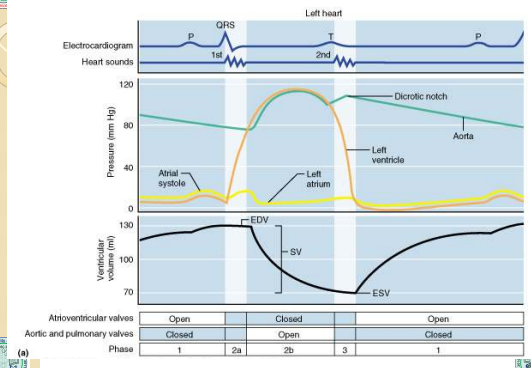

Figure 17.16a

Phases of the Cardiac Cycle

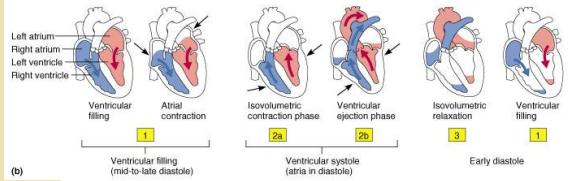

Figure 17.16b

Heart Sounds

- Heart sounds (lub-dup) are associated with closing of heart valves

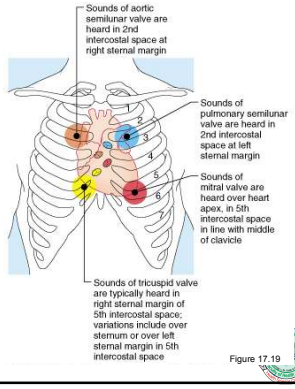

Figure 17.19

Cardiac Output (CO) and Reserve

- CO is the amount of blood pumped by each ventricle in one minute
- CO is the product of heart rate (HR) and stroke volume (SV)
- HR is the number of heart beats per minute
- SV is the amount of blood pumped out by a ventricle with each beat
- Cardiac reserve is the difference between resting and maximal CO

Cardiac Output: Example

- CO (ml/min) = HR (75 beats/min) x SV (70 ml/beat)
- CO = 5250 ml/min (5.25 L/min)

Regulation of Stroke Volume

- SV = end diastolic volume (EDV) minus end systolic volume (ESV)
- EDV = amount of blood collected in a ventricle during diastole
- ESV = amount of blood remaining in a ventricle after contraction

Factors Affecting Stroke Volume

- Preload – amount ventricles are stretched by contained blood
- Contractility – cardiac cell contractile force due to factors other than EDV
- Afterload – back pressure exerted by blood in the large arteries leaving the heart

Frank-Starling Law of the Heart

- Preload, or degree of stretch, of cardiac muscle cells *before* they contract is the critical factor controlling stroke volume
- Slow heartbeat and exercise increase venous return to the heart, increasing stroke volume
- Blood loss and extremely rapid heartbeat decrease stroke volume

Preload and Afterload

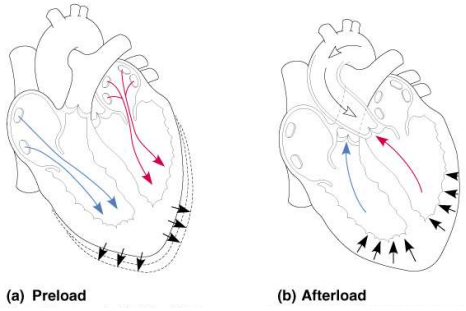

Figure 17.26

Extrinsic Factors Influencing Stroke Volume

- Contractility is the increase in contractile strength, independent of stretch and EDV
- Increase in contractility comes from:
  - Increased sympathetic stimuli
  - Certain hormones
  - Ca^{2+} and some drugs
- Agents/factors that decrease contractility include:
  - Acidosis
  - Increased extracellular potassium
  - Calcium channel blockers

Contractility and Norepinephrine

- Sympathetic stimulation releases norepinephrine and initiates a cyclic AMP second-messenger system

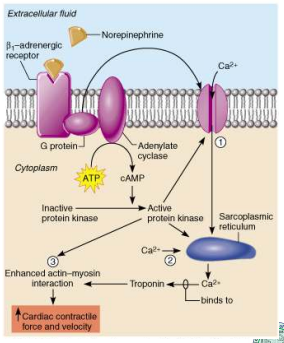

Figure 17.27

Regulation of Heart Rate: Autonomic Nervous System

- Sympathetic nervous system (SNS) stimulation is activated by stress, anxiety, excitement, or exercise
- Parasympathetic nervous system (PNS) stimulation is mediated by acetylcholine and opposes the SNS
- PNS dominates the autonomic stimulation, slowing heart rate and causing vagal tone

Bainbridge Reflex

- Bainbridge (atrial) reflex – a sympathetic reflex initiated by increased blood in the atria
  - Causes stimulation of the SA node
  - Stimulates baroreceptors in the atria, causing increased SNS stimulation

Chemical Regulation of the Heart

- The hormones epinephrine and thyroxine increase heart rate
- Intra- and extracellular ion concentrations must be maintained for normal heart function

Factors Involved in Regulation of Cardiac Output

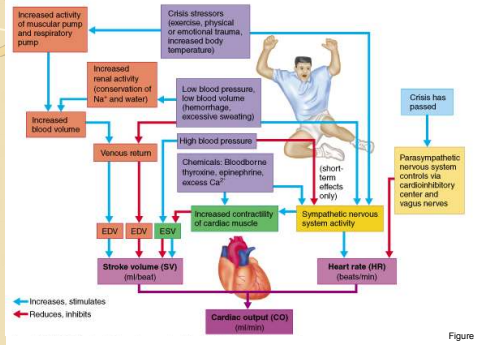

Figure 17.22

Homeostatic Imbalances

- Hypocalcemia – reduced ionic calcium depresses the heart
- Hypercalcemia – dramatically increases heart irritability and leads to spastic contractions
- Hypernatremia – blocks heart contraction by inhibiting ionic calcium transport
- Hyperkalemia – leads to heart block and cardiac arrest

Homeostatic Imbalances

- Tachycardia – heart rate over 100 beats/min
- Bradycardia – heart rate less than 60 beats/min

Congestive Heart Failure (CHF)

- Congestive heart failure (CHF), caused by:
  - Coronary atherosclerosis
  - Increased blood pressure in aorta
  - Successive myocardial infarcts
  - Dilated cardiomyopathy (DCM)