

Topic 21: CARDIAC PHYSIOLOGY – ANATOMY REVIEW/ELECTRICAL ACTIVITY

- I Introduction: review of anatomy
 - A Circulatory System consists of 3 components
 - 1 Heart = pump
 - 2 Blood vessels = passageways
 - 3 Blood = transport medium

 - B Circulatory System has two separate loops (heart is a dual pump)
 - 1 Pulmonary = right half -- receives blood from elsewhere, sends blood to lungs
 - 2 Systemic = left half – receives blood from lungs, sends everywhere else
 - 3 Separated by muscle wall called septum; each half is a separate pump
 - 4 Each half divided into two chambers
 - a upper chamber (atrium) receives blood returning to heart and transfers it to the
 - b lower chamber, (ventricle) which pumps the blood out of the heart
 - 3 Veins
 - a venae cavae brings blood from systemic circulation
 - b pulmonary veins brings blood from lungs to heart
 - 4 Arteries
 - a aorta carries blood from heart to systemic circulation
 - b pulmonary artery carries blood from heart to lungs
 - 5 Blood flow in circulatory systems
 - a pulmonary: all blood flows through lungs
 - b systemic: blood divided up among different body systems; one drop of blood visits only one tissue/trip, not all tissues/trip
 - c both sides of heart simultaneously pump equal amounts of blood
 - i pulmonary circulation is low pressure, low resistance
 - ii systemic circulation is high pressure, high resistance
 - iii the left side of heart performs more work & the muscle on the left is much thicker

 - C One way blood flow in heart; requires valves to prevent backflow
 - 1 Open and close passively because of pressure difference
 - a forward pressure gradient open valves
 - b backward pressure gradient closes valves
 - 2 Two atrioventricular (AV) valves, between atrium and ventricle of each side
 - 3 One aortic valve (left ventricle and aorta)
 - 4 One pulmonary valve (right ventricle and pulmonary artery)
 - 5 No valves between veins and atria (backflow not a problem because atrial pressure is not higher than venous pressure)

- D Heart wall structure : three layers
- 1 endocardium (inner layer; continuous with endothelium that lines entire circulatory system)
 - 2 myocardium (middle layer composed of cardiac muscle)
 - 3 epicardium (outer layer; thin membrane covering the heart)
- E Myocardial structure
- 1 cardiac muscle fibers interlaced & arranged spirally around circumference of the heart; during ventricular contraction, diameter of chambers reduced, and apex is pulled upwards; wrings blood out of chambers
 - 2 Interconnections of cardiac muscle cells
 - a adjacent cells joined end to end
 - b joining point called intercalated discs composed of
 - i gap junctions (so APs run from muscle cell to muscle cell)
 - ii desmosomes (very sturdy connections between cells)
 - c No gap junctions between atrial and ventricular cells

II Electrical Activity

- A Two types of cardiac cells
- 1 99% are **contractile cells** which do mechanical work of pumping
 - 2 1 % are **autorhythmic cells** (pacemakers) which are specialized for initiating and conducting action potentials
- B Characteristics of cardiac action potentials in **contractile cells**
- 1 When AP reaches a contractile cardiac cell, Na^+ channels open and Na^+ rushes into cell. This results in depolarization of membrane. Na^+ channels close around +30 mv (just like neurons).
 - 2 Depolarization of membrane causes opening of slow Ca^{++} channels, and Ca^{++} moves slowly into cell. This creates the plateau phase of the AP that lasts about 250 msec (specific to heart muscle!). Additionally, during this time, the K^+ channels are closed – so repolarization does not yet occur.
 - 4 Repolarization eventually occurs as Ca^{++} channels close, and K^+ channels open and K^+ moves out of cell.

- C Characteristics of cardiac muscle contraction (again, **contractile cells**)
- 1 AP moves across cell membrane, and down T-tubules
 - 2 Voltage-gated Ca^{++} channels open on cell surface
 - 3 Ca^{++} activates Ca^{++} channels on ER
 - 4 Ca^{++} floods into the cytosol (contributes to plateau phase described above, as well as to prolonged contraction of cardiac muscle-- about 3 times longer than skeletal muscle contraction)
 - 5 Ca^{++} binds to troponin, and tropomyosin then moves away from binding sites on actin, allowing ...
 - 6 cross bridge cycling just as in skeletal muscle.
 - 7 Ca^{++} eventually removed by active transport, and muscle relaxes.

- D Pacemaker activity: **autorhythmic cells**
- 1 Molecular basis
 - a Initial phase of slow depolarization caused inward leak of Na^+ through “funny channels” -- Inside of cell slowly becomes more positive & drifts toward threshold
 - b Eventually a transient voltage gated Ca^{++} channel (T-type Ca^{++} channel) opens, and Ca^{++} begins to leak into the cell, causing it to become more positive and get closer to threshold
 - c At threshold, another, long lasting, voltage gated Ca^{++} channel (L-type Ca^{++} channel) opens up and Ca^{++} rushes in, depolarizing the membrane rapidly
 - f Membrane repolarizes by exit of K^+ from cell as K^+ channels are opened.
 - 2 Autorhythmic regions of heart
 - a sinoatrial node (**SA node**) in right atrial wall near opening of superior vena cava. AP per min: 70 to 80. This is the pacemaker of the heart under normal conditions & sets AP pace of whole heart
 - b atrioventricular node (**AV node**) at base of right atrium near the septum, just above junction of atria and ventricles. Spreads AP to

ventricles from right atrium.

- c **bundle of His** is a tract of specialized cells that originates in AV node and travels around the tip of the ventricles. Spreads AP to rest of ventricles.
- d **purkinje fibers** are small terminal fibers that extend from the bundle of His. Spreads AP to rest of ventricles.

E Spread of Action Potential

- 1 SA node in right atrium initiates an AP
- 2 AP spreads through both atria via gap junctions between cells. The interatrial pathway extends from SA node to left atrium; the AP moves rapidly through to the left atrium, so that left and right atria contract simultaneously.
- 3 AP spread to AV node from SA node via the internodal pathway. Because atria are connected to ventricles by non-conducting tissue, AP has to go through AV node to get to ventricles.
- 4 AP travels slowly through the AV node, which allows time for complete ventricular filling. The AV nodal delay is about 0.1 second, which is enough time for atria to contract & empty their contents into ventricles.
- 5 AP goes from AV node through the bundle of His and through Purkinje fibers throughout ventricular myocardium. This rapid conduction of AP to all parts of ventricles allows for a coordinated ventricular contraction to eject blood from ventricles

F Electrocardiograms (ECG or EKG): more in the lab!

- 1 What an EKG represents
 - a recording a portion of heart electrical activity that reaches body surface; NOT a direct recording of actual heart electrical activity
 - b recording of overall spread of electrical activity throughout heart, NOT a single AP in the heart.
 - c recording represents comparisons in voltage detected by electrodes on 2 different parts of body surface, NOT the actual potential

- 2 EKG trace
 - a P wave: atrial depolarization
 - b PR segment: AV nodal delay

- c QRS complex: ventricular depolarization (note that atrial repolarization occurs at the same time)
- d ST segment: ventricles are contracting and emptying
- e T wave: ventricular repolarization
- f TP interval: ventricles are relaxing (and passively filling)

Topic 22: CARDIAC PHYSIOLOGY: MECHANICS AND CONTROL

I Mechanical Events in the Cardiac Cycle

A Introduction

- 1 **Systole:** Contraction and emptying
- 2 **Diastole:** Relaxation and filling
- 3 Atria and Ventricles go through separate cycle of systole and diastole

B Events, described from perspective of ventricle

1 **Ventricular diastole: from the end of the T wave to the beginning of the next QRS complex in the EKG.**

- a Atrial pressure higher than ventricular pressure, so AV valve is open and blood flows from atria directly into ventricles; volume in ventricles rises
- b Shortly after the P wave (SA node fires and atria depolarize), the atria contract and squeeze more blood into ventricles; volume in ventricles rises more sharply

2 **Ventricular systole: from the QRS complex to the end of the T wave in the EKG.**

- a Ventricular filling is complete; volume of blood in ventricles is called **end-diastolic volume (EDV)**, about 135 ml/ventricle
- b AV node fires, ventricles depolarize (QRS complex), ventricles begin to contract; ventricular pressure rises, causing the atrioventricular valves to close (first heart sound), but pressure is not high enough to open the semilunar valves so volume in ventricles remains constant (= isovolumetric contraction phase)
- c Finally ventricular pressure exceeds arterial pressure, and the semilunar valves open, allowing blood out of the ventricles; ventricular volume decreases
- d Ventricles repolarize (T-wave) and begin to relax, pressure in arteries higher than pressure in ventricles, so semilunar valve closes (second heart sound); remaining blood in each ventricle is called **end-systolic volume (ESV)**, approx. 65 ml/ventricle
- e When pressure in ventricles is less than pressure in atria, atrioventricular valves open, and ventricular filling (diastole) begins again

3. The **stroke volume (SV)** is equal to **EDV-ESV**.

II Cardiac Output and its Control

A Cardiac output

- 1 Cardiac Output (CO) is volume of blood pumped by each ventricle/minute (**not** volume of blood pumped by whole heart)
- 2 Calculated by heart rate (HR) multiplied by stroke volume (SV); this means that control of cardiac output is accomplished by controlling heart rate and stroke volume.
- 3 At rest for average individual
 - a $HR = 70 \text{ beats/min}$ $SV = 70 \text{ ml/beat}$ so $CO = 70 \text{ beats/min} \times 70 \text{ ml/beat} = 4900 \text{ ml/min}$
 - b Because total blood volume in a person is 5 to 5.5 liters, each half of the heart pumps nearly the whole blood volume each minute at rest

B Control of heart rate

- 1 Parasympathetic
 - a Decreases heart rate by:
 - i decreasing rate of depolarization in SA node
 - ii increasing AV node delay
- 2 Sympathetic
 - a Increases heart rate by
 - i increasing rate of depolarization in SA node
 - ii reducing AV node delay

C Control of stroke volume

- 1 Intrinsic control
 - a Varying length of heart muscle fibers = Starling's Law
 - i longer the fiber at start of contraction, the stronger the contraction
 - ii more blood that is in chamber, the longer the fiber (ie they are stretched) so the stronger the contraction
 - iii Bottom line: when venous return of blood to heart is increased, the blood volume in heart is increased, which increases fiber length, which increases strength of contraction, which allows pumping of larger volume
- 2 Extrinsic control
 - a Sympathetic

- i increases contractile strength of atrial and ventricle cells by increasing Ca^{++} permeability of contractile cells
- ii More Ca^{++} enters; more cross-bridge cycling and stronger contractions