Blood Pressure
1. Can you describe the flow of vessel types through the body starting with leaving the heart?
2. Can you describe the structure and characteristics of arteries?
3. Can you describe the structure and characteristics of veins?
4. Can you contrast the features that are different between arteries and veins? How do these contrasting features lend to the different functions of these vessels?
5. Can you describe the structure and characteristics of capillaries? Can you list and describe the three types of capillaries and give examples of where each type can be found in the body?
6. If the blood loses most of its pressure in the capillaries, how does blood make it back up to the heart in the venous system?
7. Can you list the six factors that influence blood pressure?
8. Can you list and describe the three initial responses to a decrease in blood pressure in the body?
9. Can you describe how myogenic response helps control blood perfusion in capillary beds of organs?
10. Can you describe how vasomotor response helps control blood pressure in the periphery?
11. Do you know what the normal heart rate and blood pressure averages are for humans?
   a. Generally, how do the sympathetic and parasympathetic nervous systems affect these averages?
12. How does the sympathetic nervous system affect the heart’s rate and force of contraction?
13. Can you define the following terms?
   a. Cardiac output (CO)
   b. Stroke volume (SV)
   c. Heart rate (HR)
   d. Blood Pressure
   e. End diastolic volume (EDV)
   f. End systolic volume (ESV)
   g. Peripheral Resistance (PR)
14. Can you write the equation for cardiac output?
   a. If you can write the equation, you know the two factors that affect cardiac output. What are they?
15. Can you write the equation for stroke volume?
16. If you can write the equation, you know the two factors that affect stroke volume. What are they?
17. How does an increase in contractility of the ventricles affect stroke volume? What does this do to the ESV?
18. How would a decrease in ESV affect CO? Can you show this in a drawing?
19. Can you write the equation for blood pressure?
20. If you can write the equation, you know the two factors that affect blood pressure. What are they?
21. Can you explain how ESV, EDV, SV and CO would affect BP?
22. Do you know what must happen to arteries for PR to increase?
   a. How is this accomplished?
23. Can you define “venous return”? How can an increase in venous return affect EDV?
   a. How is an increase in venous return accomplished?
24. Can you describe the “Frank-Starling Law”?
   a. How does this law affect ESV? If you know how it would affect ESV, you can explain how it affects BP.
25. How is the Frank-Starling Law and venous return related?
26. Can you define “blood viscosity”? 
27. Can you explain how blood viscosity influences BP?
28. Can you explain/diagram the mechanism for increasing blood viscosity?
29. Do you know what “blood volume” is and how it affects BP?
30. Can you explain/diagram the mechanism for increasing blood volume?
31. Can you explain the role of angiotensin II in vasoconstriction?
32. Can you define the function of the following, and which tissue/organ it is released from?
   a. Renin
   b. Erythropoietin
   c. Aldosterone
33. Do you know what detects a drop in blood pressure? Where are these structures located?
34. Can you describe/diagram the mechanism for blood clotting?
35. Do you know what the disorder for blood clotting is called? Which factor is usually missing in these individuals?

**Blood Pressure- Chapter 17, 18, 19, 16 & 25**

1. Arteries leave the heart and branch into smaller arteries and finally arterioles that lead blood into capillary beds. Blood enters venules as it leaves the bed and anastomoses into larger and larger veins until it is returned to the heart.
2. They have thicker walls than veins with more tunica media (smooth muscle layer). They also have elastin in their walls to withstand fluctuations of pressure from the heart.
3. Thin walls with very little smooth muscle or elastin. They have large lumens and valves.
4. Arteries have more smooth muscle and can be used as effector organs for regulation of blood pressure by the nervous system. Arteries also have elastin to withstand pressure from the heart. Veins have large lumens to lessen resistance to blood flow as well as valves to prevent backflow of blood.
5. Capillaries are one cell thick and are the smallest blood vessels in the body. They are the site of exchange between blood and the body tissues or air on the lungs. The three types are:
a. Continuous- intracellular clefts are small/thin. Squamous cells form a nearly continuous wall. Only very small molecules such as glucose, oxygen and carbon dioxide can be exchanged. Found in areas of the body where regulation of exchange is needed, such as the brain and skeletal muscle.
b. Fenestrated- intracellular clefts are still small, but cells are porous. Small fenestrations (pores) in the cells allow increased exchange of small molecules such as glucose, oxygen and carbon dioxide. Found in areas where absorption and filtration are necessary such as in the intestines and the kidney.
c. Sinusoidal- clefts and pores are large. Proteins and whole cells can be exchanged. Found in areas of the body where we need exchange of these substances such as bone marrow, liver and spleen.
6. Muscular and respiratory pumps.
8. List:
a. Myogenic response- smooth muscle response to change in stretch of the walls of the arterioles.
b. Kidneys- secrete renin and erythropoietin. These mechanisms are described later.
c. Sympathetic nervous system: EPI and NE release cause responses such as increase in heart rate and contractility, vasoconstriction and muscular spasms.
9. If blood pressure drops the arterioles will vasodilate to increase blood flow into the capillary bed and thus raise blood pressure to ensure adequate exchange with the tissues.
If blood pressure rises, the arterioles will vasoconstrict to reduce blood flow into the capillary bed and decrease blood pressure to preserve the integrity of the capillaries.
10. If the vasomotor center of the medulla oblongata is activated due to a DROP in peripheral blood pressure, it causes vasoconstriction of peripheral vessels, which increased resistance to flow of blood and thus increases blood pressure to homeostasis. If the vasomotor center of the medulla is activated due to a RISE in blood pressure then impulses to the peripheral vessels is decreased, allowing vasodilation and thus less resistance to flow resulting in a drop in blood pressure back to homeostasis.
11. Blood pressure (BP) is 120/80 and heart rate (HR) is 70 beats/min.
a. Sympathetic causes these values to rise. Parasympathetic causes these values to drop. These divisions are antagonists to each other.
12. EPI and NE bind to beta adrenergic receptors on the heart and decrease potassium permeability, which raises HR, and causes a G-protein activation and results in opening of calcium channels in the SR and plasma membrane- the more calcium you have the more cross bridges you form = increase in contractility.
13. Definitions:
a. Cardiac output (CO)- amount of blood pumped from the left ventricle in one minute.
b. Stroke volume- amount of blood pumped from the left ventricle in one stroke (contraction).
c. Heart rate- number of beats per minute of the heart.
d. Blood pressure- pressure exerted on the walls of the vessels by blood.
e. End diastolic volume (EDV)- amount of blood in the left ventricle after diastole (ventricular relaxation and the ventricle is filling).
f. End systolic volume- amount of blood remaining in the left ventricle after systole (ventricular contraction and the blood is pumped from the chamber).
g. Peripheral resistance- resistance to flow of blood in the periphery (blood vessels).
14. CO = HR x SV
a. Stroke volume- amount of blood pumped from the left ventricle in one stroke
(contraction).
b. Heart rate- number of beats per minute of the heart.
15. SV = EDV – ESV
16. End diastolic volume (EDV)- amount of blood in the left ventricle after diastole
(ventricular relaxation and the ventricle is filling) and End systolic volume- amount of
blood remaining in the left ventricle after systole (ventricular contraction and the blood is
pumped from the chamber).
17. More blood is pumped out of the left ventricle. ESV is lowered.
18. If ESV is lowered then SV increases because a smaller number is subtracted from EDV.
See lecture ink for drawing.
19. BP = CO x PR.
20. Cardiac output (CO)- amount of blood pumped from the left ventricle in one minute.
Peripheral resistance- resistance to flow of blood in the periphery (blood vessels).
21. If EDV increases and ESV decreases you get a significant rise in SV. If HR and SV
significantly raise then you have a significant raise in CO. IF CO rises, then BP raises.
22. They must vasoconstrict. Sympathetic nervous system- EPI/NE bind to alpha adrenergic
receptors.
23. The amount of blood from venous supply returning to fill the chambers of the heart. If
more blood returns to the heart, more blood fills the left ventricle and EDV raises.
a. Increased respiratory and muscular spasms by the sympathetic nervous system.
Increase HR and SV also increase the amount of blood entering the vessels, and
therefore the amount of blood that will return in them.
24. Increased filling of the ventricle causes the walls to stretch, thus aligning myosin heads
better with actin binding sites. More calcium channels will open due to this stretch as
well and more crossbridges form. Both result in increased contractility and more blood
leaving the left ventricle per pump.
a. More blood leaving = smaller ESV. Smaller ESV = bigger SV = bigger CO =
bigger BP.
25. More venous return = more blood in the left ventricle = Frank Starling Law.
26. Thickness of the blood.
27. Thicker blood exerts more pressure on the walls of the vessels because there will be more
resistance to flow in a thicker fluid.
28. See Lecture Ink.
29. It is the AMOUNT of blood in the vessels. More volume = more resistance to flow =
increased PR = increased BP.
30. See Lecture Ink.
31. It is a powerful vasoconstrictor when it is present in the blood supply, and therefore
causes increased PR = increased BP.
32. Definitions:
a. Renin- from the kidney. Plays a role in production of angiotensin II, ADH
release, increased thirst and aldosterone release.
b. Erythropoietin- released from the kidney. Increases erythropoiesis in the bone
marrow and results in more RBC in the blood.
c. Aldosterone- released from the adrenal cortex. Increases water reabsorption in
the kidney.
33. Baroreceptors in the kidney.
34. See Lecture Ink.
35. Hemophilia is the most common. Factor VIII.