Main Idea: The function of the circulatory system is to maintain adequate blood flow to all tissues.

Clinical Application Questions
1. A patient was found to have a blood pressure recording of 125/77:
   a. What is the systolic pressure? 125 mm Hg
   b. What is the diastolic pressure? 77 mm Hg
   c. What is the pulse pressure? 48 mm Hg (125 – 77 = 48 mm Hg)
   d. What is the mean arterial pressure? 93 mm Hg [77 + 1/3(48) = 77 + 16 = 93 mm Hg]
   e. Would any sound be heard when the pressure in an external cuff around the arm was 130 mm Hg? No; no blood would be able to get through the brachial artery, so no sound would be heard.
   f. Would any sound be heard when cuff pressure was 118 mm Hg? Yes
   g. Would any sound be heard when cuff pressure was 75 mm Hg? No; blood would flow continuously through the brachial artery in smooth, laminar fashion, so no sound would be heard.

2. A classmate who has been standing still for several hours working on a laboratory experiment suddenly faints. What is the probable explanation? What would you do if the person next to him tried to get him up?

   The classmate apparently fainted because of insufficient blood flow to the brain as a result of pooling of blood in the lower extremities brought about by standing still for a prolonged time. When a person faints and assumes a horizontal position, the pooled blood will quickly be returned to the heart, improving cardiac output and blood flow to his brain. Trying to get the person up would be counter productive, so the classmate trying to get him up should be advised to let him remain lying down until he recovers on his own.

3. In a person that has a sudden decrease in blood pressure, describe the short-term mechanisms that will be activated in response to a sudden decrease in blood pressure.

   A sudden decrease in blood pressure is detected by baroreceptors in the carotid sinus and aortic arch. Baroreceptors decrease their rate of impulses that are being sent to the medulla oblongata. The medulla oblongata responds by:
   a. Decreased parasympathetic activity (vagus nerve).
   b. Increased sympathetic activity which:
      i. Increases heart rate and force of contraction - higher cardiac output
      ii. Causes vasoconstriction of arterioles.
      iii. Stimulates adrenal glands to release the hormones epinephrine and norepinephrine which enhances heart rate, contractility, and vasoconstriction.
   The result is an increase of blood pressure to normal levels.
4. Ima Fan loves to go to movies. After sitting in a movie for several hours she often develops edema in her legs and feet. Explain how this occurs (Hint: recall from your study of osmosis that a column of liquid has weight).

The weight of the blood produces pressure that increases the blood pressure in her lower limbs. Consequently, more fluid moves out of capillaries because of increased blood pressure than moves back into the capillaries by osmosis. The fluid accumulates in tissues as edema. In addition, because she is sitting, skeletal muscles cannot assist in moving (pumping) blood out of her legs.

5. After a long leisurely lunch at a restaurant, sometimes elderly people faint when they stand up to leave the restaurant. Explain how this happens (Hint: assume that a homeostatic mechanism is not working as well as when they were younger).

Sitting has resulted in a shift of fluid from the elderly person’s blood into the tissues because of the increased blood pressure in her legs. This causes a reduction in blood volume and blood pressure. Also, because of the demands of digestion, blood is rerouted to the digestive organs. Consequently, there is reduced blood flow toward the brain. When the elderly person stands up, there is an even greater tendency for blood to remain in the lower limbs because of gravity. Venous return decreases, which causes cardiac output to decrease (Frank-Starling law). Decreased cardiac output results in a decrease in blood pressure. Normally, the baroreceptor reflexes correct the decrease in blood pressure by increasing heart rate and stroke volume. However, if this mechanism does not respond rapidly enough as can happen in the elderly, blood pressure does not increase, blood delivery to the brain is impaired, and the person faints.

6. Buster Hart has a myocardial infarct (heart attack) and his blood pressure drops. Explain why his blood pressure drops and describe the neural mechanisms that would attempt to compensate for the decreased blood pressure. In Buster’s case, his blood pressure was abnormally low following the myocardial infarct. Gradually (within a few days), however, it returned to normal. Explain how the long term mechanisms would compensate for this. Hint – think about hormonal effects.

The myocardial infarct damages the heart and reduces its ability to pump blood and thus will have a drop in blood pressure. With a decrease in cardiac output there is a decrease in blood pressure and blood oxygen and an increase in carbon dioxide levels. **Neural mechanisms:** The baroreceptor reflexes and the chemoreceptor reflexes compensate by increasing heart rate, stroke volume, and vasoconstriction (peripheral resistance), all of which increase blood pressure. **Hormonal effects:** In the days following the myocardial infarct, long term mechanisms compensate for the still low blood pressure. The rennin-angiotensin-aldosterone mechanism increases vasoconstriction and reduces fluid loss in the kidneys. With an increase in blood volume, blood pressure increases.

7. Explain the effect of respiratory activity on venous return to the heart.

As a result of respiratory activity, particularly inhalation, the pressure within the chest cavity averages 5 mm Hg less than atmospheric pressure. As the venous system returns blood to the heart from the lower regions of the body, it travels through the chest cavity, where it is exposed to the subatmospheric pressure. This pressure differences squeezes blood from the lower veins to the chest veins, promoting increased venous return. This mechanism of facilitating
venous return in called the respiratory pump and greatly enhances venous return during exercise.

8. Explain the effect of cardiac vacuum (“suction”) on venous return to the heart.

The extent of cardiac filling does not depend entirely on factors affecting the veins. The heart plays a role in its own filling. During ventricular relaxation, the AV valves are drawn downward, enlarging the atrial cavities. As a result, the atrial pressure transiently drops (creates a vacuum) below 0 mmHg, thus increasing the vein-to-atria pressure gradient so that venous return in enhanced. In addition, the rapid expansion of the ventricular chambers during ventricular relaxation creates a transient negative pressure (vacuum) in the ventricles so that blood is pushed in from the atria and veins; that is, the negative ventricular pressure increases vein-to-atria-to-ventricle pressure gradient, further enhancing venous return. Thus, the heart functions as a vacuum (“suction”) pump to facilitate cardiac filling.

9. Explain the effect of skeletal muscle activity on venous return to the heart.

Many of the large veins in the extremities lie between skeletal muscles, so muscle contraction compresses the veins. This external venous compression increases venous pressure, in effect squeezing fluid in the veins forward toward the heart. This pumping action, known as the skeletal muscle pump, is one way the blood in veins is returned to the heart during exercise. Increased muscular activity pushes more blood out of the veins and into the heart. Veins contain one way valves that prevent blood from being squeezed away from the heart. Increased sympathetic activity that accompanies exercise causes venous vasoconstriction further enhancing venous return to the heart.

10. Explain how each of the following antihypertensive drugs would lower arterial blood pressure: Note, for this section you are not expected to remember the drug names in parenthesis, but instead, you are expected to know how the antihypertensive drugs work given the following descriptions.

a. Drugs that block α1-adrenergic receptors (for example, phentolamine).

   Because activation of α1-adrenergic receptors in vascular smooth muscle brings about vasoconstriction, blockage of α1-adrenergic receptors reduces vasoconstrictor activity, thereby lowering the total peripheral resistance and arterial blood pressure.

b. Drugs that block β1-adrenergic receptors (for example, metoprolol).

   Because activation of β1-adrenergic receptors, which are found primarily in the heart, increase the rate and strength of cardiac contraction, drugs that block β1-adrenergic receptors reduce cardiac output and thus arterial blood pressure by decreasing the rate and strength of the heartbeat.

c. Drugs that directly relax arteriolar smooth muscle (for example, hydralazine).

   Drugs that directly relax arteriolar smooth muscle by decreasing the phosphorylation of smooth muscle myosin light chain thereby lower arterial blood pressure by promoting arteriolar vasodilation and reducing total peripheral resistance.
d. Diuretic drugs that increase urinary output (for example, furosemide).

Diuretic drugs reduce the plasma volume, thereby lowering arterial blood pressure, by increasing urinary output. Salt and water that normally would have been retained in the plasma are excreted in the urine.

e. Drugs that block release of norepinephrine from sympathetic endings (for example, guanethidine).

Because sympathetic activity promotes generalized arteriolar vasoconstriction, thereby increasing total peripheral resistance and arterial blood pressure, drugs that block the release of norepinephrine from sympathetic endings lower blood pressure by preventing this vasoconstrictor effect.

f. Drugs that act on the brain to reduce sympathetic output (for example, clonidine).

Similarly, drugs that act on the brain to reduce sympathetic output lower blood pressure by preventing the effect of sympathetic activity on promoting arteriolar vasoconstriction and the resultant increase in total peripheral resistance and arterial blood pressure.

g. Drugs that block calcium channels of smooth muscle cells in blood vessels (for example, verapamil).

Drugs that block calcium channels reduce the entry of calcium into the vascular smooth muscle cells from the ECF in response to excitatory input. Because the level of contractile activity in vascular smooth muscle cells depends on their cytosolic calcium concentration, drugs that block calcium channels reduce the contractile activity of these cells by reducing calcium entry and lowering their cytosolic calcium concentration. Total peripheral resistance and, accordingly, arterial blood pressure are decreased as a result of reduced arteriolar contractile activity.

h. Drugs that interfere with the production of angiotensin II (for example, captopril, an ACE inhibitor).

Angiotensin converting enzyme (ACE) inhibitors interfere with the production of angiotensin II and blocks activation of the hormonal pathway that releases aldosterone which promotes salt and water conservation via the rennin-angiotensin-aldosterone system. As a result, more salt and water are lost in the urine, and less fluid is retained in the plasma. The resultant reduction in plasma volume lowers the arterial blood pressure. Angiotensin II is a powerful vasoconstrictor of arterioles which increases total peripheral resistance and increases arterial BP. It also stimulates ADH release and activates the body’s thirst center within the hypothalamus.

i. Drugs that block angiotensin II receptors (for example, losartan).

Drugs that block angiotensin II receptors prevent angiotensin II from causing arteriolar vasoconstriction, thereby decreasing total peripheral resistance, and also reduce the action of the salt- and water-conserving rennin-angiotensin-aldosterone system, thereby decreasing plasma volume. Blocks the angiotensin II stimulation of ADH release. Together these effects lower blood pressure.
Part I
1. increased; decreased; pre; after; increases; increases
2. kidney; blood; blood; hydrostatic; colloid osmotic
3. lymph; lymphatic; edema
4. antidiuretic; aldosterone; cortex; retention
5. Poiseuille’s; sympathetic; aerobically
6. aerobically; high; intrinsic
7. decreased; increased; stroke; output
8. constrict; dilation; sympathetic; anastomoses; constrict
9. carotid sinus; aortic arch; medulla oblongata; rate; resistance; autonomic
10. laminar; turbulent; korotkoff
11. 48; 90; primary; secondary
12. oxygen; venous
13. B
14. C
15. False; Replace “capillaries” with “arterioles”
16. True
17. True
18. False; Replace “pons” with “medulla oblongata”
19. True
20. True
21. True
22. False; Replace “diastole” with “systole”
23. False; MAP = 1/3 pulse pressure + diastolic pressure
24. C
25. D
26. True
27. True
28. False; Replace “greater” with “less”
29. True
30. True
31. False; Osmoreceptors would stop firing ADH levels would decrease
32. True
33. True
34. False: replace “increase” with “decrease”
35. B
36. C
37. E
38. B
39. D
40. C
41. B
42. C
43. A
Part II
1. A and B antigens; AB; People with type AB blood lack both Anti-A and Anti-B antibodies
2. +; +
3. neither Rh group
4. -; +
5. -; +; +; hemolytic disease of the newborn or erythroblastosis fetalis; Anti-Rh antibody therapy (Rhogam) is given to the mother to bind to any fetal Rh antigens, destroying them. Therefore the Rh- mother will not produce antibodies that would attack antigens of a future Rh+ fetus.
6. oxyhemoglobin; 1.5%
7. iron; HbO2; saturated; increase
8. E
9. C
10. True
11. True
12. False; replace type “I” (IDDM) with Type “II” (NIDDM)
13. False; replace “obesity” with “exercise”
14. False; replace “high” with “low” and “lowered” with “raised”
15. True
16. True
17. Through the metarteriole-thoroughfare channel
18. A
19. capillary blood
20. capillary hydrostatic (blood) pressure
21. blood pressure
22. capillary colloid osmotic pressure
23. albumin
24. at the arteriole end
25. It is picked up by lymphatic vessels for return to the blood stream
26. D
27. I
28. I
29. I
30. I
31. D
32. D
33. I
34. D
35. D
36. D
37. I
38. I

Part III
1. A
2. A
3. B
4. A
5. B
6. A
7. Hypertension
8. Orthostatic hypotension
9. Cardiogenic
10. Hypovolemic
11. Varicose veins
12. Baroreceptors
Part IV
1. B
2. A
3. B
4. A
5. A
6. A
7. B
8. A
9. B
10. A
11. B
12. A
13. A
14. E

Part V, A
1. Blood pressure
2. Systolic pressure
3. Diastolic pressure
4. Auscultatory
5. Korotkoff sounds
6. Systolic pressure
7. Diastolic pressure

Part V, B
1. Pulse pressure
2. Increases
3. Increase
4. Increases
5. Pulse
6. Decreases
7. Increases

Part V, C
1. Diffusion
2. Blood pressure
3. Osmosis
4. Blood pressure
5. Osmosis
6. Lymphatic capillaries
7. Edema

Part V, D Local and Nervous Control of Blood Vessels
1. Local control
2. Relaxation
3. Nervous control
4. Vasomotor center
5. Vasomotor tone
6. Nervous control
7. Local control

Part V, E Baroreceptor Reflexes
1. Baroreceptors
2. Vasoconstrict
3. Increases
4. Increase
5. Increase
6. Increase
7. Increase

Part V, F Chemoreceptor Reflexes
1. Chemoreceptors
2. Vasoconstrict
3. Increases
4. Increase
5. Increase
6. Increases

Part V, G Hormonal Mechanisms
1. Epinephrine
2. Renin
3. Angiotensin II
4. Angiotensin II
5. Aldosterone
6. Antidiuretic hormone
7. Atrial natriuretic hormone