Page 1. Introduction
- There are two basic mechanisms for regulating blood pressure:
  1. short-term mechanisms, which regulate blood vessel diameter, heart rate and contractility
  2. long-term mechanisms, which regulate blood volume

Page 2. Goals
- To compare and contrast the short-term mechanisms that respond to rising blood pressure with the short-term mechanisms that respond to falling blood pressure.
- To understand the process of long-term regulation of low blood pressure.
- To describe the long-term and short-term effects of increased osmolarity on blood pressure.

Page 3. Short-Term Regulation of Rising Blood Pressure
- Short-term Regulation of Rising Blood Pressure
  - Rising blood pressure
  - Stretching of arterial walls
  - Stimulation of baroreceptors in carotid sinus, aortic arch, and other large arteries of the neck and thorax
  - Increased impulses to the brain

- Label this diagram:

Page 4. Effect of Baroreceptors
- Increased impulses to brain from baroreceptors
- Increased parasympathetic activity and decreased sympathetic activity
- Reduction of heart rate and increase in arterial diameter
- Lower blood pressure

Page 5. Increased Parasympathetic Activity

Interactive Physiology
Page 6. Decreased Sympathetic Activity
- Effect of Decreased Sympathetic Activity on Arteries and Blood Pressure:
  - Decreased activity of vasomotor fibers (sympathetic nerve fibers)
  - Relaxation of vascular smooth muscle
  - Increased arterial diameter
  - Lower blood pressure

Page 7. Recap: Regulation of Rising Blood Pressure
Recap: Short-term regulation of Rising Blood Pressure
- Rising blood pressure
- Stretching of baroreceptors
- Increased impulses to the brain
- Increased parasympathetic activity
- Decreased sympathetic activity
- Slowing of heart rate
- Increased arterial pressure
- Reduction of blood pressure
- Take notes on the diagram above as the animation proceeds.

Page 8. Short-term Regulation of Falling Blood Pressure
- Short-term Regulation of Falling Blood Pressure:
  - Falling blood pressure
  - Baroreceptors inhibited
  - Decreased impulses to the brain
  - Decreased parasympathetic activity, increased sympathetic activity
  - Three effects:
    1. Heart: increased heart rate and increased contractility
    2. Vessels: increased vasoconstriction
    3. Adrenal gland: release of epinephrine and norepinephrine which enhance heart rate, contractility, and vasoconstriction
  - Increased blood pressure

Page 9. Sympathetic Activity on Heart and Blood Pressure
- Effect of Increased Sympathetic Activity on Heart and Blood Pressure:
• Increased activity of sympathetic cardiac nerves
• Decreased activity of vagus (parasympathetic) nerve
• Increased heart rate and contractility
• Higher cardiac output
• Increased blood pressure

Page 10. Vasomotor Fibers
• Effect of Increased Sympathetic Activity on Arteries and Blood Pressure:
  • Increased activity of vasomotor fibers (sympathetic nerve fibers)
  • Constriction of vascular smooth muscle
  • Decreased arterial diameter
  • Increased blood pressure

Page 11. Sympathetic Activity on Adrenal Gland and Blood Pressure
• Effect of Increased Sympathetic Activity on Adrenal Glands and Blood Pressure:
  • Increased sympathetic impulses to adrenal glands
  • Release of epinephrine and norepinephrine to bloodstream
  • Hormones increase heart rate, contractility and vasoconstriction. Effect is slower-acting and more prolonged than nervous system control.
  • Increased blood pressure

Page 12. Recap: Regulation of Falling Blood Pressure
• Recap: Regulation of Falling Blood Pressure
  • Falling blood pressure
  • Baroreceptors inhibited
  • Decreased impulses to the brain
  • Decreased parasympathetic activity
  • Increased sympathetic activity
  • Increased heart rate and contractility
  • Increased vasoconstriction
  • Release of epinephrine and norepinephrine from adrenal gland
  • Increased blood pressure

• Take notes on this diagram as the animation proceeds:

** Now is a good time to go to quiz questions 1, 2, and 3:
  • Click the Quiz button on the left side of the screen.
  • After answering question 3, click the Back to Topic button on the left side of the screen.
  • To get back to where you left off, click on the scrolling page list at the top of the screen and choose "13. Introduction: Long-Term Regulation of Low BP".

Page 13. Introduction: Long-Term Regulation of Low BP
• Long-term regulation of blood pressure is primarily accomplished by altering blood volume.
• The loss of blood through hemorrhage, accident, or donating a pint of blood will lower blood pressure and trigger processes to restore blood volume and therefore blood pressure back to normal.
• Long-term regulatory processes promote the conservation of body fluids via renal mechanisms and stimulate intake of water to normalize blood volume and blood pressures.

Page 14. Loss of Blood

Interactive Physiology
• When there is a loss of blood, blood pressure and blood volume decrease.

Page 15. Kidney Juxtaglomerular Cells
• Juxtaglomerular cells in the kidney monitor alterations in the blood pressure. If blood pressure falls too low, these specialized cells release the enzyme renin into the bloodstream.

Page 16. Renin-Angiotensin Mechanism: Step 1
• The renin/angiotensin mechanism consists of a series of steps aimed at increasing blood volume and blood pressure.
• Step 1: Catalyzing Formation of Angiotensin I: As renin travels through the bloodstream, it binds to an inactive plasma protein, angiotensinogen, activating it into angiotensin I.
• Label this diagram:

Page 17. Step 2: Conversion of Angiotensin I
• Step 2: Converting Angiotensin I to Angiotensin II: As angiotensin I passes through the lung capillaries, an enzyme in the lungs converts angiotensin I to angiotensin II.
• Label this diagram:

Page 18. Step 3: Angiotensin II in the Bloodstream
• Step 3: Angiotensin II Stimulates Aldosterone Release: Angiotensin II continues through the bloodstream until it reaches the adrenal gland.

Page 19. Release of Aldosterone
• Step 3: Angiotensin II Stimulates Aldosterone Release: Here it stimulates the cells of the adrenal cortex to release the hormone aldosterone.
Page 20.  Angiotensin II as a Vasoconstrictor
  • A secondary effect is that angiotensin II is a vasoconstrictor and therefore raises blood pressure in the body’s arterioles.

Page 21.  Aldosterone Mechanism
  • Long-Term Regulation: Aldosterone Mechanism: The target organ for aldosterone is the kidney. Here aldosterone promotes increased reabsorption of sodium from the kidney tubules.

Page 22.  Distal Convoluted Tubule
  • Long-Term Regulation: Aldosterone Mechanism:
    • Each distal convoluted tubule winds through the kidney and eventually empties its contents into a urine-collecting duct.
    • The peritubular capillaries absorb solutes and water from the tubule cells as these substances are reclaimed from the filtrate.
    • Label the diagram of the kidney tubules and associated blood vessels on the top of the next page.
Page 23. Sodium Reabsorption

- Aldosterone stimulates the cells of the distal convoluted tubule to increase the active transport of sodium ions out of the tubule into the interstitial fluid, accelerating sodium reabsorption.

- Label this diagram:
**Page 24. Water Reabsorption**
- As sodium moves into the bloodstream, water follows. The reabsorbed water increases the blood volume and therefore the blood pressure.
- Label this diagram:

**Page 25. Increase in Osmolarity**
- Dehydration due to sweating, diarrhea, or excessive urine flow will cause an increase in osmolarity of the blood and a decrease in blood volume and blood pressure.

**Page 26. Long-Term Effect of Osmolarity on BP**
- As increased osmolarity is detected there is both a short and long-term effect. For the long-term effect, the hypothalamus sends a signal to the posterior pituitary to release antidiuretic hormone (ADH).

**Page 27. Antidiuretic Hormone**
- ADH increases water reabsorption in the kidney.

**Page 28. ADH in Distal Convoluted Tubule**
- ADH promotes the reabsorption of water from the kidney by stimulating an increase in the number of water channels in the distal convoluted tubules and collecting tubules (ducts).
- These channels aid in the movement of water back into the capillaries, decreasing the osmolarity of the blood volume and therefore blood pressure.
- Label the diagram on the top of the next page.
Page 29. Short-Term Effect of Osmolarity on BP
• A short-term effect of increased osmolarity is the excitation of the thirst center in the hypothalamus. The thirst center stimulates the individual to drink more water and thus hydrate the blood and extracellular fluid, restoring blood volume and therefore blood pressure.

Page 30. Other Chemicals That Influence BP
• There are many other chemicals which influence blood flow and blood vessel diameter. Most of them act by influencing blood vessel diameter.

Page 31. Summary
• In the short-term, rising blood pressure stimulates increased parasympathetic activity, which leads to reduced heart rate, vasodilation and lower blood pressure.
• Falling blood pressure stimulates increased sympathetic activity, which leads to increased heart rate, contractility, vasoconstriction, and blood pressure.
• Long-term blood pressure regulation involves renal regulation of blood volume via the renin-angiotensin mechanism and aldosterone mechanism.
• Increased blood osmolarity stimulates release of antidiuretic hormone (ADH), which promotes reabsorption of water, and excites the thirst center, resulting in increased blood volume and blood pressure.

** Now is a good time to go to quiz questions 4 and 5:
• Click the Quiz button on the left side of the screen.
• Click on the scrolling page list at the top of the screen and choose "4. Blood Volume Chain Reaction".
• Work through quiz questions 4-5.

Notes on Quiz Questions:
Quiz Question #1: Identification
• This question asks you to label structures which are important in blood pressure regulation.

Quiz Question #2: High Blood Pressure
• This question asks you to lower blood pressure by clicking on the appropriate nerve.

Quiz Question #3: Chemical Heart Stabilization
• This question asks you to identify the chemical that will increase blood pressure.

Quiz Question #4: Blood Volume Chain Reaction
• This question asks you to list the proper sequence of events that occurs when blood volume and blood pressure increases or decreases.

Quiz Question #5: Dehydration Chain Reaction
• This question asks you to list the proper sequence of events that occurs when dehydration increases or decreases.
Study Questions on Blood Pressure Regulation:
1. What are baroreceptors?
2. Where are the baroreceptors that sense blood pressure located?
3. What happens to baroreceptors when blood pressure is high?
4. What happens to both parasympathetic activity and sympathetic activity when blood pressure is high?
5. What is the effect of increased parasympathetic activity and decreased sympathetic activity on both heart rate and blood pressure?
6. What is the name of the parasympathetic nerve that decreases heart rate?
7. How does a decrease in heart rate decrease blood pressure?
8. What is a vasomotor fiber?
9. What is the effect of high blood pressure on arteries?
10. How does vasodilation decrease blood pressure?
11. What happens to baroreceptors when the blood pressure is low? What effect does that have on the brain?
12. What are the three effects of an increased sympathetic activity and decreased parasympathetic activity?
13. How does an increase in heart rate increase blood pressure?
14. What is the effect of low blood pressure on arteries?
15. How does vasoconstriction increase blood pressure?
16. What is the effect of sympathetic activity on the adrenal gland?
17. Why are the effects of epinephrine and norepinephrine from the adrenal gland slower-acting and more prolonged than nervous system control?
18. When there is a loss of blood through hemorrhage, accident, or donating a pint of blood, what two long-term regulatory processes will restore blood volume and therefore blood pressure back to normal?
19. What happens to blood volume and blood pressure when there is blood loss?
20. If blood pressure falls too low, what do the juxtaglomerular cells of the kidney release into the bloodstream?
21. Label the diagram on p. 16.
22. What is angiotensinogen?
23. How is angiotensinogen activated?
24. Label the diagram on p. 17.
25. How is angiotensin I converted into Angiotensin II?
26. Place the following steps in the release of aldosterone in order:
   a. An enzyme in the lungs converts angiotensin I to angiotensin II.
   b. Angiotensinogen is activated into angiotensin I.
   c. Angiotensin II stimulates the cells of the adrenal cortex to release the hormone aldosterone.
   d. As renin travels through the bloodstream, it binds to an inactive plasma protein, angiotensinogen.
   e. Angiotensin II continues through the bloodstream until it reaches the adrenal gland.
   f. Angiotensin I passes through the lung capillaries.
27. Label the diagram on p. 19.
28. What happens when Angiotensin II reaches the adrenal gland?
29. What are two effects of angiotensin II?
30. (Page 21.) What is the target organ for aldosterone?
31. (Page 21.) What is the effect of aldosterone?
32. (Page 22.) What is "filtrate" and where is it located within the kidneys? What is its relationship to the blood capillaries?
33. (Page 22.) What is the process of reabsorption within the kidneys?
34. (Page 23.) What happens when aldosterone binds to the cells of the distal convoluted tubule?
35. (Page 23.) Label the diagram on p. 23.
37. (Page 24.) How does aldosterone increase the blood volume and blood pressure?
38. (Page 25.) What effect does dehydration due to sweating, diarrhea, or excessive urine flow have on osmolarity of the blood, blood volume, and blood pressure?
39. (Page 26.) An increased osmolarity of the blood causes the release of what hormone?
40. (Page 27.) What is the effect of ADH?
41. (Page 28.) How does ADH increase water reabsorption in the kidney?
42. (Page 29.) What is the short-term effect of increased osmolarity of the blood on blood pressure?
43. (Summary) When blood volume and blood pressure are increased, do the following increase or decrease?
   a. Renin release from the kidney ________.
   b. Angiotensinogen into Angiotensin I ________.
   c. Angiotensin I into Angiotensin II ________.
   d. Aldosterone release from the adrenal gland ________.
   e. Sodium reabsorption from the filtrate into the blood ________.
   f. Water reabsorption ________.
   g. Blood volume and blood pressure ________.
44. (Summary) When blood volume and blood pressure are decreased, do the following increase or decrease?
   a. Renin release from the kidney ________.
   b. Angiotensinogen into Angiotensin I ________.
   c. Angiotensin I into Angiotensin II ________.
   d. Aldosterone release from the adrenal gland ________.
   e. Sodium reabsorption from the filtrate into the blood ________.
   f. Water reabsorption ________.
   g. As a result blood volume and blood pressure ________.
45. (Summary) When there is an increase in dehydration, are the following increased or decreased?
   a. Body water ________.
   b. Blood volume and blood pressure ________.
   c. Blood osmolarity ________.
   d. ADH release from the pituitary ________.
   e. Water permeability of the kidney tubules ________.
   f. Urine output and blood osmolarity ________.
   g. As a result, blood volume and blood pressure ________.

**Answers to Questions on Blood Pressure Regulation:**
1. Sensory receptors that detect increased pressure, by increased stretch in arterial walls.
2. In the carotid sinus, aortic arch, and other large arteries of the neck and thorax.
3. Increased blood pressure stretches arterial walls, stimulating the baroreceptors. The result is an increase in sensory signals to the brain.
4. Get increased parasympathetic activity and decreased sympathetic activity.
5. Heart rate decreases and blood pressure decreases.
6. The vagus nerve (cranial nerve X).
7. By lowering cardiac output.
8. A sympathetic nerve fiber that innervates muscular arteries and arterioles.
9. High blood pressure decreases sympathetic activity, sympathetic impulses to vascular smooth muscle decreases and there is a vasodilation of the arteries.
10. When vasodilation occurs, there is an increase in arterial diameter, allowing more space for the blood to be in. The result is less pressure on the walls of the arteries (lowered blood pressure).
11. Baroreceptors are not stimulated. Get decreased impulses to the brain which increase sympathetic activity and decrease parasympathetic activity.
12. 1. Get increased heart rate and increased contractility leading to an increased blood pressure. 2. Get increased vasoconstriction 3. Get release of epinephrine and norepinephrine which enhance heart rate, contractility, and vasoconstriction leading to an increased blood pressure.
13. By increasing cardiac output.
14. Low blood pressure increases sympathetic activity, sympathetic impulses to vascular smooth muscle increases and there is a vasoconstriction of the arteries, causing the blood pressure to rise.
15. When vasoconstriction occurs, there is an decrease in arterial diameter, allowing less space for the blood to be in. The result is more pressure on the walls of the arteries (increased blood pressure).
16. Adrenal gland releases the hormones epinephrine and norepinephrine.
17. Hormones travel in the blood which takes more time than the release and action of neurotransmitters.
18. (1) Renal mechanisms promote conservation of body fluids (2) mechanisms that stimulate intake of water
20. The enzyme renin.
21. From left to right: angiotensinogen, renin, angiotensin I
22. An inactive plasma protein.
23. It binds to renin which activates it into angiotensin I.
24. From left to right: angiotensin I, enzyme in wall of lung capillary, angiotensin II
25. As angiotensin I passes through the lung capillaries, an enzyme in the lungs converts angiotensin I to angiotensin II.
26. 1. d, 2. b, 3. f, 4. a, 5. e, 6. c
27. From left to right: Angiotensin II, adrenal gland, Aldosterone, kidney
28. It causes the release of aldosterone.
29. 1. Causes the release of aldosterone 2. constricts blood vessels, raising the blood pressure
30. The kidney
31. Aldosterone promotes increased reabsorption of sodium from the kidney tubules.
32. Filtrate is the forming urine. It is located within kidney tubules. The distal convoluted tubule is a part of the tubule that filtrate is found in. Blood capillaries are found around the tubules (They are called peritubular capillaries.
33. The movement of materials from the filtrate into the blood capillaries.
34. Sodium ions are reabsorbed from the filtrate within the distal convoluted tubule into the peritubular capillaries.
35. From left to right: Aldosterone, distal convoluted tubule, peritubular capillary, interstitial fluid.
36. From top to bottom: distal convoluted tubule, Aldosterone, interstitial fluid, peritubular capillary
37. Aldosterone causes sodium to move into the bloodstream. The water that follows the sodium increases the blood volume and therefore the blood pressure.
38. It increases the osmolarity of the blood, decreases the blood volume, and decreases the blood pressure.
39. antidiuretic hormone (ADH)
40. ADH increases water reabsorption in the kidney.
41. By stimulating an increase in the number of water channels.
42. The thirst center in the hypothalamus stimulates the individual to drink more water and thus rehydrate the blood and extracellular fluid, restoring blood volume and therefore blood pressure.
43. a-g all decreases
44. a-g all increases
45. a. decrease  b. decrease  c. increase  d. increase  e. increases f. decreases  g. increases