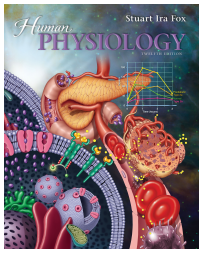
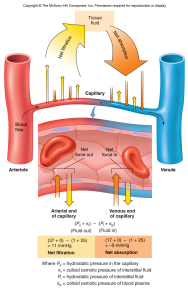


Chapter 14

Cardiac Output, Blood Flow, and Blood Pressure

Lecture PowerPoint

I. Cardiac Output



Cardiac Output

- Volume of blood pumped each minute by each ventricle:

cardiac output = stroke volume X heart rate
(ml/minute) (ml/beat) (beats/min)

– Average heart rate = 70 bpm
– Average stroke volume = 70−80 ml/beat
– Average cardiac output = 5,500 ml/minute

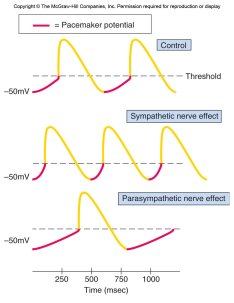
Regulation of Cardiac Rate

- Spontaneous depolarization occurs at SA node when HCN channels open, allowing Na^+ in.
- Sympathetic norepinephrine (vagus nerve) and adrenal epinephrine keep HCN channels open, increasing heart rate.

Regulation of Cardiac Rate

- Parasympathetic acetylcholine opens K^+ channels, slowing heart rate.
- Controlled by cardiac center of medulla oblongata

Regulation of Cardiac Rate



Regulation of Stroke Volume

- Regulated by three variables:

1. End diastolic volume (EDV): volume of blood in the ventricles at the end of diastole
   - Sometimes called preload
   - Stroke volume increases with increased EDV.

Regulation of Stroke Volume

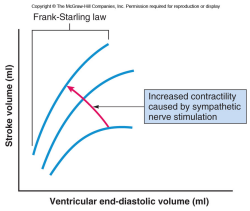
- Regulated by three variables:

2. Total peripheral resistance: Frictional resistance in the arteries
   - Inversely related to stroke volume

3. Contractility: strength of ventricular contraction
   - Stroke volume increases with contractility.

Frank-Starling Law

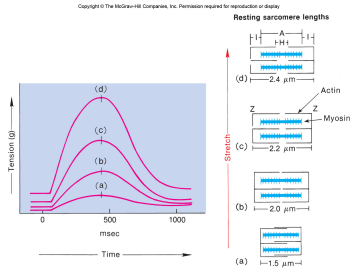
- Increased EDV results in increased contractility and thus increased stroke volume.



Intrinsic Control of Contraction Strength

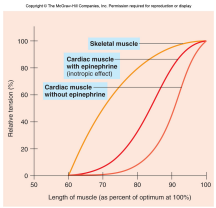
- Due to myocardial stretch
  - Increased EDV stretches the myocardium, which increases contraction strength.
    - Due to increased myosin and actin overlap and increased sensitivity to Ca^{2+} in cardiac muscle cells

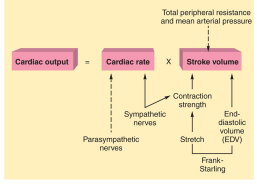
Intrinsic Control of Contraction Strength



Extrinsic Control of Contractility

- Sympathetic norepinephrine and adrenal epinephrine can increase contractility by making more Ca^{2+} available to sarcomeres.

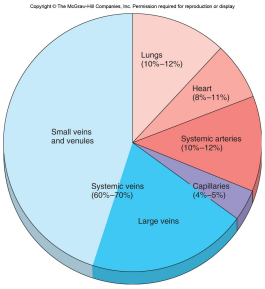




Venous Return

- End diastolic volume is controlled by factors that affect venous return:
  - Total blood volume
  - Venous pressure
    - Veins are compliant = stretch at a given pressure. They hold more blood than arteries but maintain lower pressure.

Venous Return



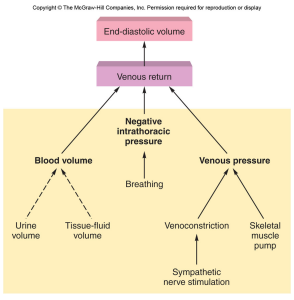
Factors in Venous Return

1. Highest pressure in venules vs. lowest pressure in veins closest to heart
2. Sympathetic nerve activity to stimulate smooth muscles and lower compliance

Factors in Venous Return

3. Skeletal muscle pumps
4. Pressure difference between abdominal and thoracic cavities
5. Blood volume

Factors in Venous Return

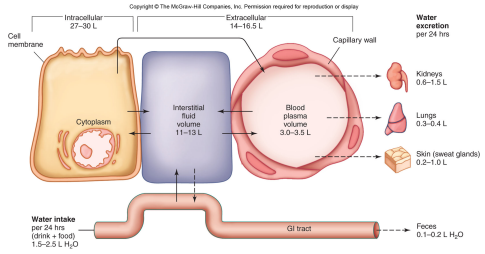


II. Blood Volume

Body Water Distribution

- 2/3 of our body water is found in the cells.
- Of the remaining, 80% exists in interstitial spaces and 20% is in the blood plasma.
- Osmotic forces control the movement of water between the interstitial spaces and the capillaries, affecting blood volume.
- Urine formation and water intake (drinking) also play a role in blood volume.

Body Water Distribution



Tissue/Capillary Fluid Exchange

- Net filtration pressure is the hydrostatic pressure of the blood in the capillaries minus the hydrostatic pressure of the fluid outside the capillaries:

  =36 mmHg at arteriole end

  =16 mmHg at venule end

Tissue/Capillary Fluid Exchange

- Colloid osmotic pressure: due to proteins dissolved in fluid
  - Blood plasma has higher colloid osmotic pressure than interstitial fluid. This difference is called oncotic pressure.
  - Oncotic pressure = 25 mmHg
  - This favors the movement of fluid into the capillaries.

Tissue/Capillary Fluid Exchange

- Starling Forces: combination of hydrostatic pressure and oncotic pressure that predicts movement of fluid across capillary membranes
  - Fluid movement is proportional to:

    $(p_c + \pi_i) - (p_i + \pi_p)$

    fluid out fluid in

  p_c = Hydrostatic pressure in capillary

  $\pi_{i\ s}$ = Colloid osmotic pressure of interstitial fluid

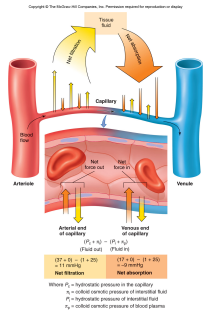
  $p_{i\ s}$ = Hydrostatic pressure of interstitial fluid

  π_p = Colloid osmotic pressure of blood plasma

Tissue/Capillary Fluid Exchange

- Starling Forces predict the movement of fluid into the capillaries at the arteriole end and out of the capillaries at the venule end.

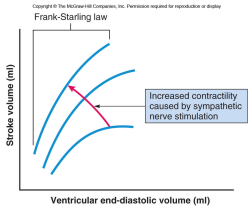
Tissue/Capillary Fluid Exchange



Edema

- Excessive accumulations of interstitial fluids
- May be the result of:
  - High arterial blood pressure
  - Venous obstruction
  - Leakage of plasma proteins into interstitial space
  - Myxedema (excessive production of mucin in extracellular spaces caused by hypothyroidism)
  - Decreased plasma protein concentration
  - Obstruction of lymphatic drainage

Edema



Regulation of Blood Volume by Kidneys

- The formation of urine begins with filtration of fluid through capillaries in the kidneys called glomeruli.
  - 180 L of filtrate is moved across the glomeruli per day, yet only about 1.5 L is actually removed as urine. The rest is reabsorbed into the blood.
  - The amount of fluid reabsorbed is controlled by several hormones in response to the body's needs.

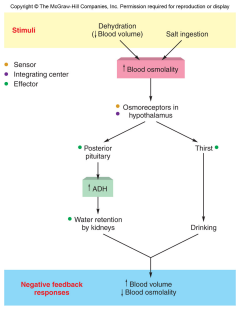
Regulation of Blood Volume by Kidneys

- Antidiuretic hormone (ADH): produced by hypothalamus and released when osmoreceptors there detect increased plasma osmolarity.
  - Plasma osmolarity can increase due to excessive salt intake or dehydration.
  - Increased plasma osmolarity also increases thirst.
  - ADH stimulates water reabsorption.

Regulation of Blood Volume by Kidneys

- Increased water intake and decreased urine formation increase blood volume.
- Blood becomes dilute, and ADH is no longer released.
- Stretch receptors in left atrium, carotid sinus, and aortic arch also inhibit ADH release.

Regulation of Blood Volume by Kidneys



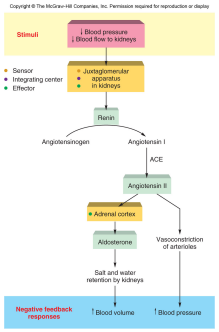
Regulation of Blood Volume by Kidneys

- Aldosterone: secreted by adrenal cortex *indirectly* when blood volume and pressure are reduced
  - Stimulates reabsorption of salt in kidneys
  - Regulated by renin-angiotensin-aldosterone system

Regulation of Blood Volume by Kidneys

- Renin-angiotensin-aldosterone system: When blood pressure is low, cells in the kidneys (juxtaglomerular apparatus) secrete the enzyme renin:
  - Angiotensinogen is converted to angiotensin I.
  - Angiotensin I is converted to angiotensin II by ACE enzyme.

Regulation of Blood Volume by Kidneys



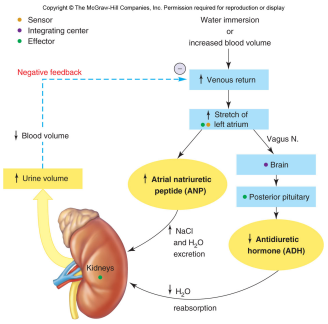
Regulation of Blood Volume by Kidneys

- Angiotensin II has many effects that result in a raise in blood pressure:
  - Vasoconstriction of small arteries and arterioles
  - Stimulates thirst center in hypothalamus
  - Stimulates production of aldosterone in adrenal cortex

Regulation of Blood Volume by Kidneys

- Atrial natriuretic peptide: produced by the atria of the heart when stretch is detected
  - Promotes salt and water excretion in urine in response to increased blood volume
  - Inhibits ADH secretion

Regulation of Blood Volume by Kidneys



III. Vascular Resistance to Blood Flow

Blood Flow to the Organs

- Cardiac output is distributed unequally to different organs due to unequal resistance to blood flow through the organs.

Blood Flow to the Organs

Table 14.3 | Estimated Distribution of the Cardiac Output at Rest

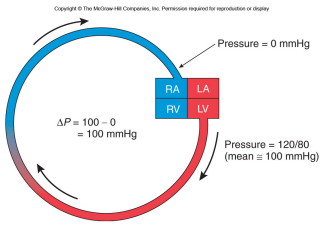
Organs	Blood Flow	
	Milliliters per Minute	Percent Total
Gastrointestinal tract and liver	1,400	24
Kidneys	1,100	19
Brain	750	13
Heart	250	4
Skeletal muscles	1,200	21
Skin	500	9
Other organs	600	10
Total organs	5,800	100

Source: From O. L. Wade and J. M. Bishop, *Cardiac Output and Regional Blood Flow*. Copyright © 1962 Blackwell Science, Ltd. Used with permission.

Physical Laws Regulating Blood Flow

- Blood flows from a region of higher pressure to a region of lower pressure.
- The rate of blood flow is proportional to the differences in pressure.

Physical Laws Regulating Blood Flow



Physical Laws Regulating Blood Flow

- The rate of blood flow is also inversely proportional to the frictional resistance to blood flow within the vessels.

$$\text{blood flow} = \frac{\Delta P}{\text{resistance}}$$

ΔP = pressure difference between the two ends of the tube

Physical Laws Regulating Blood Flow

- Resistance is measured as:

$$\text{resistance} = \frac{L\eta}{r^4}$$

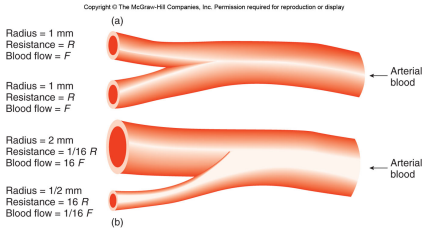
L = length of the vessel
η = viscosity of the blood
r = radius of the blood vessel

Physical Laws Regulating Blood Flow

- Poiseuille's Law adds in physical constraints:

$$\text{blood flow} = \frac{\Delta P r^4(\pi)}{\eta L(8)}$$

Physical Laws Regulating Blood Flow



Physical Laws Regulating Blood Flow

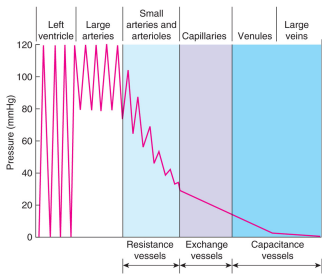
– Vessel length (L) and blood viscosity (η) do not vary normally.

– Mean arterial pressure and vessel radius (r) are therefore the most important factors in blood flow.

Physical Laws Regulating Blood Flow

– Vasoconstriction of arterioles provides the greatest resistance to blood flow and can redirect flow to/from particular organs.

Physical Laws Regulating Blood Flow



Total Peripheral Resistance

- The sum of all vascular resistance in systemic circulation
  - Blood flow to organs runs parallel to each other, so a change in resistance within one organ does not affect another.
  - Vasodilation in a large organ may decrease total peripheral resistance and mean arterial pressure.
  - Increased cardiac output and vasoconstriction elsewhere make up for this.

Extrinsic Regulation of Blood Flow

- Autonomic and endocrine control of blood flow
  - Sympathetic nerves:
    - Increases total peripheral resistance through release of norepinephrine onto smooth muscles of arterioles in the viscera and skin to stimulate vasoconstriction.
    - Acetylcholine is released onto skeletal muscles, resulting in increased vasodilation to these tissues (due in part to adrenal epinephrine).

Extrinsic Control of Blood Flow

Table 14.4 | Extrinsic Control of Vascular Resistance and Blood Flow

Extrinsic Agent	Effect	Comments
Sympathetic nerves		
Alpha-adrenergic	Vasoconstriction	Vasoconstriction is the dominant effect of sympathetic nerve stimulation on the vascular system, and it occurs throughout the body.
Beta-adrenergic	Vasodilation	There is some activity in arterioles in skeletal muscles and in coronary vessels, but effects are masked by dominant alpha-receptor-mediated constriction.
Cholinergic	Vasodilation	Effects are localized to arterioles in skeletal muscles and are produced only during defense (flight-or-flight) reactions.
Parasympathetic nerves	Vasodilation	Effects are restricted primarily to the gastrointestinal tract, external genitalia, and salivary glands and have little effect on total peripheral resistance.
Angiotensin II	Vasoconstriction	A powerful vasoconstrictor produced as a result of secretion of renin from the kidneys; it may function to help maintain adequate filtration pressure in the kidneys when systemic blood flow and pressure are reduced.
ADH (vasopressin)	Vasoconstriction	Although the effects of this hormone on vascular resistance and blood pressure in anesthetized animals are well documented, the importance of these effects in conscious humans is controversial.
Histamine	Vasodilation	Histamine promotes localized vasodilation during inflammation and allergic reactions.
Bradykinins	Vasodilation	Bradykinins are polypeptides secreted by sweat glands and by the endothelium of blood vessels; they promote local vasodilation.
Prostaglandins	Vasodilation or vasoconstriction	Prostaglandins are cyclic fatty acids that can be produced by most tissues, including blood vessel walls. Prostaglandin I_2 is a vasodilator, whereas thromboxane A_2 is a vasoconstrictor. The physiological significance of these effects is presently controversial.

Extrinsic Regulation of Blood Flow

– Parasympathetic nerves:
  - Acetylcholine stimulates vasodilation.
  - Limited to digestive tract, external genitalia, and salivary glands
  - Less important in controlling total peripheral resistance due to limited influence

Extrinsic Regulation of Blood Flow

– Paracrine control: Molecules produced by one tissue control another tissue within the same organ.

  - Example: The tunica intima produces signals to influence smooth muscle activity in the tunica media.

Extrinsic Regulation of Blood Flow

- Smooth muscle relaxation influenced by bradykinin, nitric oxide, and prostaglandin I_2 to produce vasodilation
- Endothelin-1 stimulates smooth muscle contraction to produce vasoconstriction and raise total peripheral resistance.

Intrinsic Regulation of Blood Flow

- Used by some organs (brain and kidneys) to promote constant blood flow when there is fluctuation of blood pressure; also called autoregulation.
  - Myogenic control mechanisms: Vascular smooth muscle responds to changes in arterial blood pressure.

Intrinsic Regulation of Blood Flow

- Metabolic control mechanisms: Vasodilation is controlled by changes in:
  - Decreased oxygen concentrations due to increased metabolism
  - Increased carbon dioxide concentrations
  - Decreased tissue pH (due to CO_2, lactic oxide, etc.)
  - Release of K^+ and paracrine signals

IV. Blood Flow to the Heart and Skeletal Muscles

Aerobic Requirements of the Heart

- The coronary arteries supply blood to a massive number of capillaries (2,500–4,000 per cubic mm tissue).
  - Unlike most organs, blood flow is restricted during systole. Cardiac tissue therefore has myoglobin to store oxygen during diastole to be released in systole.

Aerobic Requirements of the Heart

- Cardiac tissue also has lots of mitochondria and respiratory enzymes, thus is metabolically very active.
- During exercise, the coronary arteries increase blood flow from 80 ml to 400 ml/ minute/100 g tissue.

Regulation of Coronary Blood Flow

- Norepinephrine from sympathetic nerve fibers stimulates vasoconstriction, raising vascular resistance at rest.
- Adrenal epinephrine stimulates vasodilation and thus vascular resistance during exercise.
- Vasodilation is enhanced by intrinsic metabolic control mechanisms

Regulation of Blood Flow Through Skeletal Muscles

- Arterioles have high vascular resistance at rest.
  - Even at rest, skeletal muscles still receive 20−25% of the body's blood supply.

Regulation of Blood Flow Through Skeletal Muscles

- Vasodilation is stimulated by both adrenal epinephrine and sympathetic acetylcholine.
- Intrinsic metabolic controls enhance vasodilation during exercise

Regulation of Blood Flow Through Skeletal Muscles

Table 14.5 | Changes in Skeletal Muscle Blood Flow Under Conditions of Rest and Exercise

Condition	Blood Flow (ml/min)	Mechanism
Rest	1,000	High adrenergic sympathetic stimulation of vascular alpha receptors, causing vasoconstriction
Beginning exercise	Increased	Dilation of arterioles in skeletal muscles due to cholinergic sympathetic nerve activity and stimulation of beta-adrenergic receptors by the hormone epinephrine
Heavy exercise	20,000	Fall in alpha-adrenergic activity
		Increased cholinergic sympathetic activity
		Increased metabolic rate of exercising muscles, producing intrinsic vasodilation

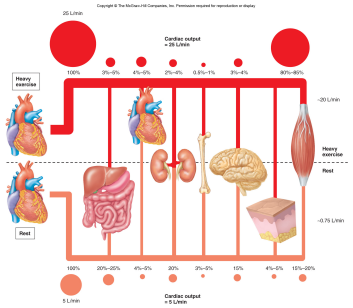
Circulatory Changes During Exercise

- Vascular resistance to skeletal and cardiac muscles decreases due to:
  - Increased cardiac output
  - Metabolic vasodilation
  - Diversion of blood away from viscera and skin
- Blood flow to brain increases a small amount with moderate exercise and decreases a small amount during intense exercise.

Circulatory Changes During Exercise

- Cardiac output can increase 5X due to increased cardiac rate.
- Stroke volume can increase some due to increased venous return.

Circulatory Changes During Exercise



Circulatory Changes During Exercise

Table 14.7 | Cardiovascular Changes During Moderate Exercise

Variable	Change	Mechanisms
Cardiac output	Increased	Increased cardiac rate and stroke volume
Cardiac rate	Increased	Increased sympathetic nerve activity; decreased activity of the vagus nerve
Stroke volume	Increased	Increased myocardial contractility due to stimulation by sympathoadrenal system; decreased total peripheral resistance
Total peripheral resistance	Decreased	Vasodilation of arterioles in skeletal muscles (and in skin when thermoregulatory adjustments are needed)
Arterial blood pressure	Increased	Increased systolic and pulse pressure due primarily to increased cardiac output; diastolic pressure rises less due to decreased total peripheral resistance
End-diastolic volume	Unchanged	Decreased filling time at high cardiac rates is compensated for by increased venous pressure, increased activity of the skeletal muscle pump, and decreased intrathoracic pressure aiding the venous return
Blood flow to heart and muscles	Increased	Increased muscle metabolism produces intrinsic vasodilation; aided by increased cardiac output and increased vascular resistance in visceral organs
Blood flow to visceral organs	Decreased	Vasoconstriction in digestive tract, liver, and kidneys due to sympathetic nerve stimulation
Blood flow to skin	Increased	Metabolic heat produced by exercising muscles produces reflex (involving hypothalamus) that reduces sympathetic constriction of arteriovenous shunts and arterioles
Blood flow to brain	Unchanged	Autoregulation of cerebral vessels, which maintains constant cerebral blood flow despite increased arterial blood pressure

V. Blood Flow to the Brain and Skin

Cerebral Circulation

- The brain cannot tolerate much variation in blood flow.
- Unless mean arterial pressure becomes very high, there is little sympathetic control of blood flow to the brain.
  - At high pressure, vasoconstriction occurs to protect small vessels from damage and stroke.

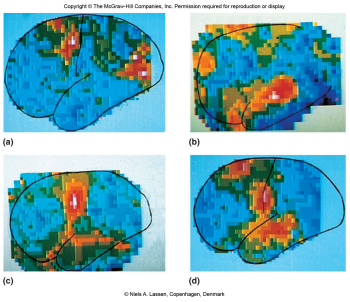
Myogenic Control of Cerebral Blood Flow

- When blood pressure falls, cerebral vessels automatically dilate.
- When blood pressure rises, cerebral vessels automatically constrict.
- Decreased pH of cerebrospinal fluid (buildup of CO_2) also causes arteriole dilation.

Metabolic Control of Cerebral Blood Flow

- The most active regions of the brain must receive increased blood flow due to arteriole sensitivity to metabolic changes.
  - Astrocytes may play a role

Metabolic Control of Cerebral Blood Flow



Cutaneous Blood Flow

- The skin can tolerate the greatest fluctuations in blood flow.
- The skin helps control body temperature in a changing environment by regulating blood flow = thermoregulation.
  - Increased blood flow to capillaries in the skin releases heat when body temperature increases.
  - Sweat is also produced to aid in heat loss.
  - Bradykinins in the sweat glands also stimulate vasodilation in the skin.

Cutaneous Blood Flow

- Vasoconstriction of arterioles keeps heat in the body when ambient temperatures are low.
- This is aided by arteriovenous anastomoses, which shunt blood from arterioles directly to venules.
  - Cold temperatures activate sympathetic vasoconstriction.
  - This is tolerated due to decreased metabolic activity in the skin.

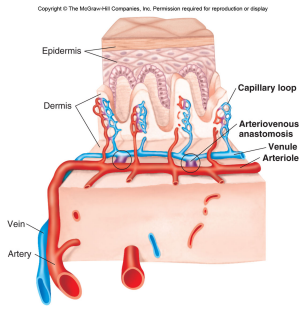
Cutaneous Blood Flow

- At average ambient temperatures, vascular resistance in the skin is high, and blood flow is low.
- Sympathetic stimulation reduces blood flow further.
- With continuous exercise, the need to regulate body temperature overrides this, and vasodilation occurs.

Cutaneous Blood Flow

- May result in lowered total peripheral resistance if not for increased cardiac output
- However, if a person exercises in very hot weather, he or she may experience extreme drops in blood pressure after reduced cardiac output.
- This condition can be very dangerous.

Cutaneous Blood Flow

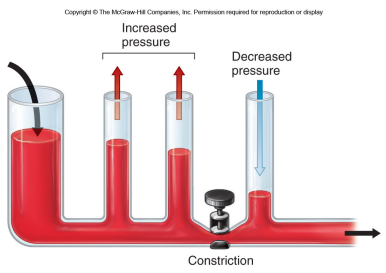


VI. Blood Pressure

Blood Pressure

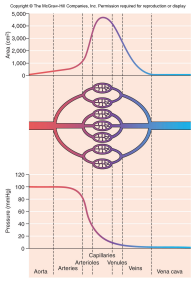
- Affected by blood volume/stroke volume, total peripheral resistance, and cardiac rate
  - Increase in any of these will increase blood pressure.
  - Vasoconstriction of arterioles raises blood pressure upstream in the arteries.

Blood Pressure



Blood Pressure

- Capillary blood pressure is low because of large total cross-sectional area.



Blood Pressure Regulation

- Kidneys can control blood volume and thus stroke volume.
- The sympathoadrenal system stimulates vasoconstriction of arterioles (raising total peripheral resistance) and increased cardiac output.

Baroreceptor Reflex

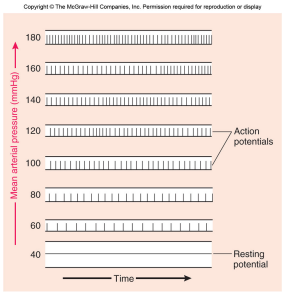
- Activated by changes in blood pressure detected by baroreceptors in the aortic arch and carotid sinuses

Baroreceptor Reflex

– Increased blood pressure stretches these receptors, increasing action potentials to the vasomotor and cardiac control centers in the medulla.

– Most sensitive to drops in blood pressure

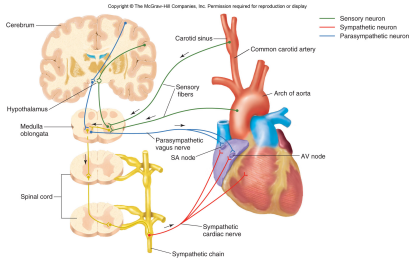
Baroreceptor Reflex



Baroreceptor Reflex

- The vasomotor center controls vasodilation and constriction.
- The cardiac center controls heart rate.

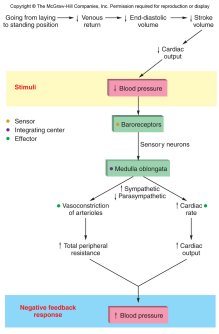
Baroreceptor Reflex



Baroreceptor Reflex

- Fall in blood pressure = ↑ sympathetic and ↓ parasympathetic activity, resulting in increased heart rate and total peripheral resistance
- Rise in BP has the opposite effects.
- Good for quick beat-by-beat regulation

Baroreceptor Reflex



Atrial Stretch Reflexes

- Activated by increased venous return to:
  - Stimulate tachycardia
  - Inhibit ADH release
    - Results in excretion of more urine
  - Stimulate secretion of atrial natriuretic peptide
    - Results in excretion of more salts and water in urine

Blood Pressure Measurement

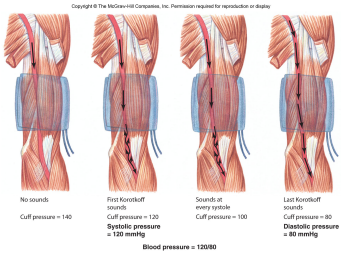
- Measured in mmHg by an instrument called a sphygmomanometer.
  - A blood pressure cuff produces turbulent flow of blood in the brachial artery, which can be heard using a stethoscope; called sounds of Korotkoff.

Blood Pressure Measurement

- The cuff is first inflated to beyond systolic blood pressure to pinch off an artery. As pressure is released, the first sound is heard at systole and a reading can be taken.
- The last Korotkoff sound is heard when the pressure in the cuff reaches diastolic pressure and a second reading can be taken.

Blood Pressure Measurement

- The average blood pressure is 120/80.



Pulse Pressure

- "Taking the pulse" is a measure of heart rate.
- What the health professional feels is increased blood pressure in that artery at systole.
  - The difference between blood pressure at systole and at diastole is the pulse pressure.
    - If your blood pressure is 120/80, your pulse pressure is 40 mmHg.

Mean Arterial Pressure

- The average pressure in the arteries in one cardiac cycle is the mean arterial pressure.
  - This is significant because it is the difference between mean arterial pressure and venous pressure that drives the blood into the capillaries.
  - Calculated as:

    diastolic pressure + 1/3 pulse pressure

VII. Hypertension, Shock, and Congestive Heart Failure

Hypertension

- Hypertension is high blood pressure.
  - 20% of Americans have hypertension.
  - It can increase the risk of cardiac diseases and stroke.
  - Hypertension can be classified as "essential" or "secondary."
    - Secondary hypertension is a symptom of another disease, such as kidney disease.

Secondary Hypertension

Table 14.8 | Blood Pressure Classification in Adults

Blood Pressure Classification	Systolic Blood Pressure		Diastolic Blood Pressure	Drug Therapy
Normal	Under 120 mmHg	and	Under 80 mmHg	No drug therapy
Prehypertension	120–139 mmHg	or	80–89 mmHg	Lifestyle modification;* no antihypertensive drug indicated
Stage 1 Hypertension	140–159 mmHg	or	90–99 mmHg	Lifestyle modification; antihypertensive drugs
Stage 2 Hypertension	160 mmHg or greater	or	100 mmHg or greater	Lifestyle modification; antihypertensive drugs

*Lifestyle modifications include weight reduction; reduction in dietary fat and increased consumption of vegetables and fruit; reduction in dietary sodium (salt); engaging in regular aerobic exercise, such as brisk walking for at least 30 minutes a day, most days of the week; and moderation of alcohol consumption.
Source: From the Seventh Report of the Joint National committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 Report. *Journal of the American Medical Association; 289* (2003): 2560-2572.

Table 14.9 | Possible Causes of Secondary Hypertension

System Involved	Examples	Mechanisms
Kidneys	Kidney disease	Decreased urine formation
	Renal artery disease	Secretion of vasoactive chemicals
Endocrine	Excess catecholamines (tumor of adrenal medulla)	Increased cardiac output and total peripheral resistance
	Excess aldosterone (Conn's syndrome)	Excess salt and water retention by the kidneys
Nervous	Increased intracranial pressure	Activation of sympathoadrenal system
	Damage to vasomotor center	Activation of sympathoadrenal system
Cardiovascular	Complete heart block; patent ductus arteriosus	Increased stroke volume
	Arteriosclerosis of aorta; coarctation of aorta	Decreased distensibility of aorta

Essential Hypertension

- Most people fall in this category.
- The cause is difficult to determine and may involve any of the following:
  - Increased salt intake coupled with decreased kidney filtering ability
  - Increased sympathetic nerve activity, increasing heart rate
  - Responses to paracrine regulators from the endothelium

Dangers of Hypertension

- Vascular damage within organs, especially dangerous in the cerebral vessels and leading to stroke
- Ventricular overload to eject blood, leading to arrhythmias and cardiac arrest
- Contributes to the development of atherosclerosis

Treatments for Hypertension

- Lifestyle modification: limit salt intake; limit smoking and drinking; lose weight; exercise
- K^+ (and possibly calcium) supplements
- Diuretics to increase urine formation
- Beta blockers to decrease cardiac rate
- ACE inhibitors to block angiotensin II production

Circulatory Shock

- Occurs when there is inadequate blood flow to match oxygen usage in the tissues
  - Symptoms result from inadequate blood flow and how our circulatory system changes to compensate.
  - Sometimes shock leads to death.

Circulatory Shock

Table 14.11 | Signs of Shock

	Early Sign	Late Sign
Blood pressure	Decreased pulse pressure	Decreased systolic pressure
	Increased diastolic pressure	
Urine	Decreased Na^+ concentration	Decreased volume
	Increased osmolality	
Blood pH	Increased pH (alkalosis) due to hyperventilation	Decreased pH (acidosis) due to "metabolic" acids
Effects of poor tissue perfusion	Slight restlessness; occasionally warm, dry skin	Cold, clammy skin; "cloudy" senses

Source: From *Principles and Techniques of Critical Care*, Vol. 1, edited by R. F. Wilson. Copyright © 1977 F.A. Davis Company, Philadelphia, PA. Used by permission.

Hypovolemic Shock

- Due to low blood volume from an injury, dehydration, or burns
  - Characterized by decreased cardiac output and blood pressure
  - Blood is diverted to the heart and brain at the expense of other organs.
  - Compensation includes baroreceptor reflex, which lowers blood pressure, raises heart rate, raises peripheral resistance, and produces cold, clammy skin and low urine output.

Septic Shock

- Dangerously low blood pressure due to an infection (sepsis)
  - Bacterial toxins induce NO production, causing widespread vasodilation.
  - Mortality rate is high (50−70%).

Other Causes of Circulatory Shock

- Severe allergic reactions can cause anaphylactic shock due to production of histamine and resulting vasodilation.
- Spinal cord injury or anesthesis can cause neurogenic shock due to loss of sympathetic stimulation.
- Cardiac failure can cause cardiogenic shock due to significant myocardial loss.

Congestive Heart Failure

- Occurs when cardiac output is not sufficient to maintain blood flow required by the body
  - Caused by myocardial infarction, congenital defects, hypertension, aortic valve stenosis, or disturbances in electrolyte levels (K^+ and Ca^{2+})
  - Similar to hypovolemic shock in symptoms