

Chapter 21: The Cardiovascular System: Blood Vessels and Hemodynamics

Chapter Objectives

STRUCTURE AND FUNCTION OF BLOOD VESSELS

1. Identify the different types of blood vessels in the body.
2. Describe the functional properties of arteries.
3. Explain how the sympathetic nervous system affects blood vessel diameter
4. Explain how blood bypasses capillary beds.
5. Distinguish among the different types of capillaries and where they are found.

CAPILLARY EXCHANGE

6. List the three ways substances enter and leave capillaries.
7. Describe the substances that use diffusion as a capillary exchange method.
8. Describe transcytosis and the types of substances to be transported.
9. Discuss the importance of bulk flow, the pressures involved in bulk flow, and the pressure changes that occur for bulk flow to happen.

HEMODYNAMICS: PHYSIOLOGY OF CIRCULATION

10. Explain the factors that regulate the velocity of blood flow.
11. Describe the three factors that contribute to the return of venous blood to the heart.
12. Explain how blood pressure changes throughout the cardiovascular system.
13. Explain the factors that determine the systemic vascular resistance.

CONTROL OF BLOOD PRESSURE AND BLOOD FLOW

14. Describe the structure and function of the cardiovascular center.
15. Identify the sources of input to the cardiovascular center.
16. Identify the outcome of sympathetic and parasympathetic output from the cardiovascular center.
17. Describe the role of baroreceptors in the aortic and carotid sinus reflexes and the purpose of these reflexes.
18. Examine the effects of renin-angiotensin-aldosterone, epinephrine, norepinephrine, ADH and ANP on blood pressure.

SHOCK AND HOMEOSTASIS

19. Define shock and its general consequences.
20. Discuss the four causes of shock.
21. Describe the characteristic signs and symptoms of shock.

Chapter Lecture Notes

Blood Vessels

Blood vessels form a closed system of tubes that carry blood away from the heart, transport it to the tissues of the body, and then return it to the heart (Table 21.1 & Fig 21.6)

Types of blood vessels

Arteries

Arterioles

Capillaries

Venules

Veins

Functions of Blood Vessels

Arteries

Carry blood to the tissues

The functional properties of arteries are

elasticity - allows arteries to accept blood under great pressure from the contraction of the ventricles and to send it on through the system

contractility - allows arteries to increase or decrease lumen size and to limit bleeding from wounds

due to the smooth muscle in the tunica media

Smooth muscle in blood vessels is innervated by the sympathetic nervous system

increase in stimulation causes muscle contraction or vasoconstriction

injury to artery or arteriole causes muscle contraction reducing blood loss (vasospasm)

decrease in stimulation or presence of certain chemicals causes vasodilation

nitric oxide, K⁺, H⁺ and lactic acid cause vasodilation

Arterioles

Connect arteries to capillaries

Arteriole walls have fewer layers of smooth muscle than arteries

Metarterioles form branches into capillary beds (Fig 21.3)

to bypass a capillary bed, a precapillary sphincter, a band of smooth muscle around the metarteriole, contracts, vasoconstricting the metarteriole & blood flows out of the bed and into a thoroughfare channel

vasomotion - intermittent contraction & relaxation of precapillary sphincters that allow filling of capillary beds 5-10 times/minute

Capillaries

Site of substance exchange between the blood and body tissues

Venules

connect capillaries to larger veins

very porous endothelium allows for escape of many phagocytic white blood cells

Veins

convey blood from the venules back to the heart

contain valves to prevent the backflow of blood

Capillaries

Found near every cell in the body but more extensive in highly active tissue (muscles, liver, kidneys & brain) (Fig 21.1)

entire capillary bed fills with blood when tissue is active

not found in epithelia, cornea and lens of eye & cartilage

Capillary walls are composed of only a single layer of cells (endothelium) and a basement membrane

Types of capillaries (Fig 21.4)

Continuous capillaries

nearly complete basement membrane

intercellular clefts - gaps between neighboring cells

Locations: skeletal & smooth muscle, connective tissue and lungs

Fenestrated capillaries

basement membrane mostly intact

endothelial cells have fenestrations – holes or pores through the plasma membrane

Locations: kidneys, small intestine, choroid plexuses, ciliary process & endocrine glands

Sinusoids

incomplete basement membrane

very large fenestrations

large intercellular clefts

allow large structures like proteins or blood cells to enter or exit the bloodstream

Locations: liver, bone marrow, spleen, anterior pituitary, & parathyroid gland

Capillary Exchange

Movement of materials in & out of a capillary (Fig 21.7)

Diffusion (most important method)

Substances such as O₂, CO₂, glucose, amino acids, hormones, and others diffuse down their concentration gradients

all plasma solutes except large proteins pass freely across

through lipid bilayer, fenestrations or intercellular clefts

blood brain barrier does not allow diffusion of water-soluble materials (nonfenestrated epithelium with tight junctions)

Transcytosis

passage of material across endothelium in tiny vesicles by endocytosis and exocytosis

large, lipid-insoluble molecules such as insulin or maternal antibodies passing through placental circulation to fetus

Bulk Flow

Movement of large amount of dissolved or suspended material in same direction

Movement is in response to pressure

from area of high pressure to area of low

Faster rate of movement than diffusion or osmosis

Most important for regulation of relative volumes of blood & interstitial fluid

Filtration - movement of material into interstitial fluid

promoted by blood hydrostatic pressure & interstitial fluid osmotic pressure

Blood hydrostatic pressure (BHP) – Amount of force exerted on the walls of a blood vessel due to contraction of the heart and the amount of blood within the vessel

At arteriole end is 35 mmHg and at venule end is 26 mmHg

Interstitial fluid osmotic pressure (IFOP) – Amount of “pull” exerted by solutes in the interstitial fluid

assume value is 1 mmHg

Reabsorption - movement from interstitial fluid into capillaries

promoted by blood colloid osmotic pressure and interstitial fluid hydrostatic pressure

Blood colloid osmotic pressure (BCOP) – Amount of “pull” exerted by solutes, mostly large proteins that are not able to leave the capillaries, within the capillaries

BCOP = 26 mmHg

Interstitial fluid hydrostatic pressure (IFHP) – the amount of back pressure “pushing” fluid back into the capillaries

because fluid is drained away from the capillaries almost as soon as it leaves, the

IFHP is close to zero, so assume a value of zero

The balance between filtration and reabsorption determines whether fluids leave or enter capillaries, it depends on a balance of the pressures

Net Filtration Pressure (NFP)

$$\text{NFP} = (\text{BHP} + \text{IFOP}) - (\text{BCOP} + \text{IFHP})$$

At the arterial end of a capillary bed, the BHP is high and the NFP is 10 mm Hg of outward pressure (net filtration)

At the venule end of a capillary bed, the BHP has dropped and the NFP is 8-9 mm Hg of inward pressure (net reabsorption)

About 85% of the filtered fluid is returned to the capillary

Starling's law of the capillaries states that the volume of fluid & solutes reabsorbed is almost as large as the volume filtered

escaping fluid and plasma proteins are collected by lymphatic capillaries (3 liters/day)

Hemodynamics – Blood Flow

Blood flow - The volume that flows through any tissue in a given period of time

The velocity of blood flow is inversely related to the total cross-sectional area of all blood vessels

blood flows most slowly where total cross-sectional area is greatest

Blood flow velocity decreases from the aorta to arteries to capillaries and increases as it returns to the heart (Fig 21.11)

flow in aorta is 40 cm/sec while flow in capillaries is 0.1 cm/sec

slow rate in capillaries allows for exchange

Circulation time is time it takes a drop of blood to travel from right atrium back to right atrium

Volume of blood flowing back to the heart from the systemic veins depends on

pressure difference from venules (16 mm Hg) to right atrium (0 mm Hg) – siphon effect

Respiratory pump

decreased thoracic pressure and increased abdominal pressure during inhalation, moves

blood into thoracic veins and the right atrium

Skeletal muscle pump (Fig 21.9)

contraction of muscles & presence of valves

Hemodynamics – Blood Pressure

Blood pressure – force exerted by blood on walls of a vessel

caused by contraction of the ventricles

highest in aorta

120 mm Hg during systole & 80 during diastole

Pressure falls steadily in systemic circulation with distance from left ventricle (Fig 21.8)

35 mm Hg entering the capillaries

0 mm Hg entering the right atrium

Factors that affect blood pressure (Fig 21.10)

Cardiac output – heart rate and force of contraction

Blood volume

If decrease in blood volume is over 10%, BP drops

Water retention increases blood pressure

Elasticity of arteries

Vascular resistance - opposition to blood flow as a result of friction between blood and the

walls of the blood vessels

average blood vessel radius

smaller vessels offer more resistance to blood flow

can cause moment to moment fluctuations in pressure by changing the size of blood

vessels

blood viscosity (thickness)

ratio of red blood cells to plasma volume

increases in viscosity increase resistance

dehydration or polycythemia

total blood vessel length

the longer the vessel, the greater the resistance to flow

200 miles of blood vessels for every pound of fat

obesity causes high blood pressure

Systemic vascular resistance (also known as total peripheral resistance) refers to all of the

vascular resistances offered by systemic blood vessels;

most resistance is in arterioles, capillaries, and venules due to their small diameters

arterioles control BP by changing diameter

Control of Blood Pressure & Flow

Nervous control of blood pressure and flow is regulated by the cardiovascular center (CV) - a

group of neurons in the medulla that regulates heart rate, contractility, and blood vessel

diameter (Fig 21.12 & 21.13)

input from

higher brain regions such as cerebral cortex, limbic system & hypothalamus

anticipation of competition

increase in body temperature

Proprioceptors

input during physical activity

Baroreceptors

changes in pressure within blood vessels

Chemoreceptors

monitor concentration of chemicals in the blood

output from the CV flows along sympathetic and parasympathetic fibers

Parasympathetic impulses along vagus nerves decrease heart rate

Sympathetic impulses along cardioaccelerator nerves increase heart rate and contractility

The sympathetic division also continually sends impulses to smooth muscle in blood vessel walls via vasomotor nerves. The result is a moderate state of tonic contraction or vasoconstriction, called vasomotor tone

Baroreceptor reflexes controlled by the CV (Fig 21.14)

carotid sinus reflex

swellings in internal carotid artery wall

glossopharyngeal nerve to cardiovascular center in medulla

maintains normal BP in the brain

aortic reflex

receptors in wall of ascending aorta

vagus nerve to cardiovascular center

maintains general systemic BP

Hormonal Control of Blood Pressure (Table 21.2)

Renin-angiotensin-aldosterone system (Fig 18.16)

release of renin by kidneys results in conversion of a protein made by the liver,

angiotensinogen, into angiotensin II by the lungs

Angiotensin II stimulates systemic vasoconstriction

Angiotensin II stimulates the adrenal gland to release aldosterone

Aldosterone influences kidney function during urine production, decreasing the

amount of urine production and increasing the amount of H₂O & Na⁺

retained by the blood

Net effect of the Renin-Angiotensin-Aldosterone System is to increase blood volume and

blood pressure

Epinephrine & norepinephrine

increases heart rate & force of contraction

causes vasoconstriction in skin & abdominal organs

vasodilation in cardiac & skeletal muscle

increases blood pressure

ADH (anti diuretic hormone)

causes vasoconstriction

promotes retention of water by the blood during urine production

increases blood volume and pressure

ANP (atrial natriuretic peptide) lowers BP

causes vasodilation

loss of salt and water in the urine

decreases blood volume and pressure

Shock

Shock is the condition in which cardiac output (CO) cannot deliver enough oxygen and nutrients to meet the needs of body cells.

There are three stages of shock.

Nonprogressive or compensated

If the cause is removed the body's negative feedback mechanisms alone can usually restore homeostasis.

Progressive or Decompensated

Shock becomes steadily worse. Recovery from progressive shock is possible but there will be some damage to tissues.

Irreversible shock

In this condition body cells have been too long without adequate oxygen and nutrients.

All forms of known therapy are unable to save a person's life at this point even though cardiac output and arterial pressure may appear to be restored to normal.

There are four main types of shock

Hypovolumic - decreased blood volume (Fig 21.16)

may be caused by acute hemorrhage (internal or external bleeding) or excessive fluid loss
(as occurs in excessive vomiting, diarrhea, sweating, urine production,
dehydration and burns)

Cardiogenic (cardiac shock, power failure syndrome) - decreased cardiac output

may be caused by cardiac abnormalities that decrease the ability of the heart to pump
blood (for example, myocardial infarction, severe valve dysfunction, heart
arrhythmias)

85% of people who develop cardiogenic shock do not survive

Vascular

Neurogenic - occurs without any loss of blood

vascular capacity increases so much that even the normal amount of blood cannot
adequately fill the circulatory system

may be caused by sudden loss of vasomotor tone throughout the body causing
especially massive dilation of veins (venous pooling) causing reduction of
venous return to the heart (may occur in deep general anesthesia, spinal
anesthesia, and brain damage)

Anaphylactic (an allergic reaction)

allergic reaction of anaphylactic shock causes massive release of histamine from
basophils and mast cells dilates arteries and veins reducing venous return

Septic (blood poisoning, toxic shock syndrome)

widely disseminated, blood borne bacterial infection causing extensive tissue damage
important in hospital setting because this type of shock, more often than any other
type of shock besides cardiogenic, causes patient death in the hospital

Obstructive

may be caused by blockage of blood flow through part of the circulation

blockage causes backup of blood and increases pressure behind the blockage leading to leaking of plasma and proteins out of capillaries (loss of blood volume) into surrounding tissues (for example, pulmonary emboli)

Signs and symptoms of shock

rapid resting heart rate (tachycardia)

weak, rapid pulse

hypotension

clammy, cool pale skin

sweating

altered mental state

decreased urine formation

thirst

acidosis

nausea