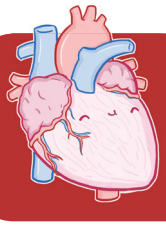

NOTES
CARDIOVASCULAR ANATOMY & PHYSIOLOGY

CARDIOVASCULAR ANATOMY & PHYSIOLOGY

osms.it/cardiovascular-anatomy-physiology

CARDIOVASCULAR SYSTEM

- *Cardia-, cardi-, cardio-*
  - Heart, which pumps blood
- *Vascular:* blood vessels (carry blood to body, return it to heart)
- Delivers oxygen, nutrients to organs, tissues
- Removes waste (carbon dioxide, other cellular respiration by-products) from organs, tissues

MORPHOLOGY

- *Size:* about size of person's first (correlated with person's size)
- *Shape:* blunt cone-shaped
- *Position:* slightly shifted to left side
- Location
  - Lies in mediastinum in thoracic cavity
  - Sits on top of diaphragm (main breathing muscle)
  - Behind sternum (breast bone)
  - In front of vertebral column
  - Between lungs
  - Enclosed, protected by ribs
  - Right, left sides separated by muscular septum

Heart wall layers

- *Epicardium:* covers surface of heart, great vessels (AKA visceral pericardium)
- *Myocardium:* muscular middle layer
  - *Cardiac muscle cells:* striated branching cells with many mitochondria, intercalated disks for synchronous contraction
  - *Cardiac myocytes:* striated, branching cells with fibrous cardiac skeleton

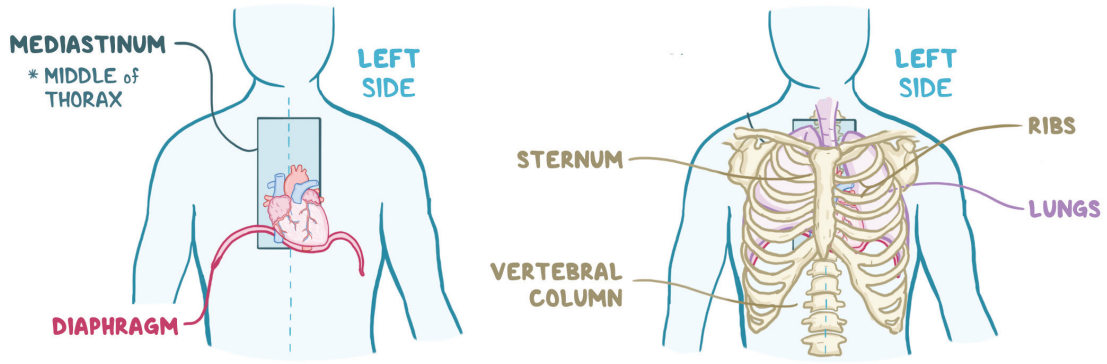

Figure 14.1 Heart location relative to other thoracic structures.

(supports muscle tissue, crisscrossing connective tissue collagen fibers); coronary vessels (lie on outside of heart, penetrate into myocardium to bring blood to that layer)

- *Endocardium:* innermost layer
  - Made of thin epithelial layer, underlying connective tissue
  - Lines heart chamber, valve
- *Pericardium:* double-layered sac surrounding heart
  - *Fibrous pericardium:* outer layer; tough fibrous connective tissue anchors heart within mediastinum
  - *Serous pericardium:* simple squamous epithelium layer
  - *Parietal pericardium:* lines fibrous pericardium
  - *Visceral pericardium (epicardium):* covers outer surface of heart
  - Cells of parietal, visceral pericardium secrete protein-rich fluid (pericardial fluid) → fills space between layers (lubricant for heart, prevents friction)

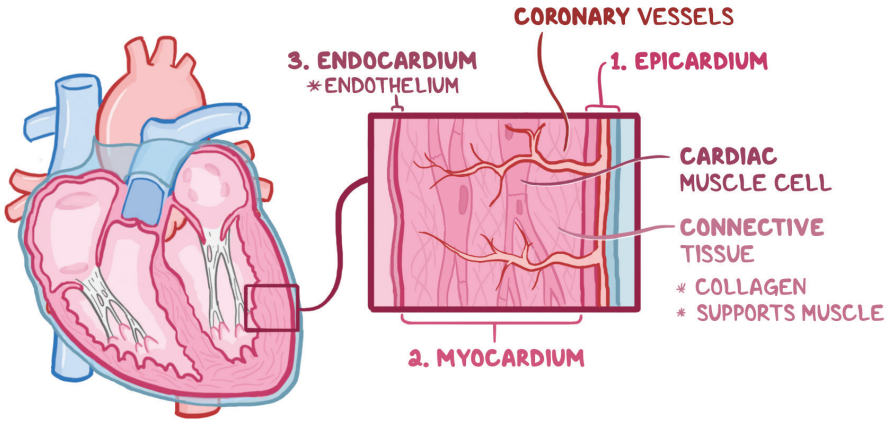

Figure 14.2 Heart wall layers, from superficial to deep.

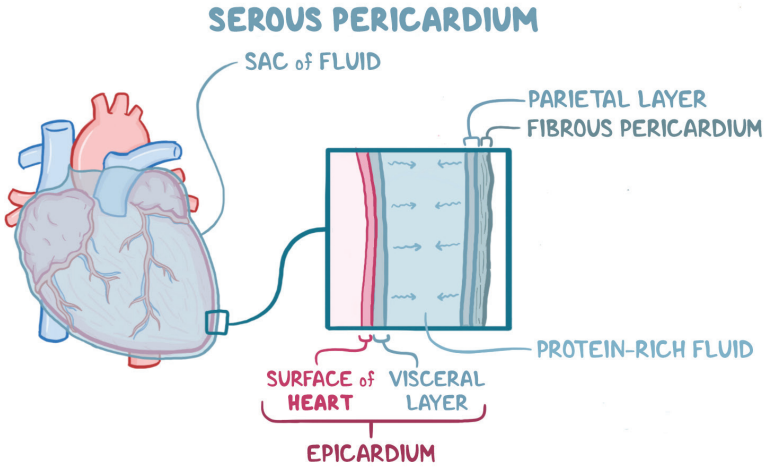

Figure 14.3 Layers of the pericardium (the double-layered sac surrounding the heart.)

Atrioventricular valves

- Separate atria from ventricles
- Tricuspid valve
  - Three cusps with chordae tendinae (tether valve to papillary muscle)
  - Prevents blood backflow into right atrium (right ventricle contracts → papillary muscles contract, keep chordae tendineae taut)
- Bicuspid / mitral valve
  - *Two cusps:* anterior, posterior leaflet
  - Both have chordae tendineae tethered to papillary muscles in left ventricle
  - Prevents blood backflow back into left atrium

Semilunar valves

- Located where two major arteries leave ventricles
- Pulmonary valve
  - Three half-moon shaped cusps
  - Prevents blood backflow into right ventricle
- Aortic valve
  - Three cusps
  - Prevents blood backflow into left ventricle

Blood flow physiology

- Deoxygenated blood enters right side of heart via superior, inferior vena cava (veins)
- Coronary sinus (tiny right atrium opening) collects blood from coronary vessels → right atrium → tricuspid valve → right ventricle → pulmonary valve → pulmonary trunk → pulmonary arteries → pulmonary arterioles → pulmonary capillaries → alveoli
- Blood collects oxygen from alveoli, removes carbon dioxide
- Oxygenated blood travels through pulmonary venules → pulmonary veins → left atrium → bicuspid/mitral valve → left ventricle → aortic valve → aorta → organs, tissues
- Deoxygenated blood returns to heart

SYSTEMIC VS. PULMONARY CIRCULATION

- Pulmonary, systemic circulation both pump same amount of blood

Pulmonary circulation

- Low pressure system
- Right side of heart pumps deoxygenated blood through pulmonary circulation to collect oxygen
  - Right atrium → right ventricle → pulmonary arteries → lungs

Systemic circulation

- High pressure system
- Left side of heart pumps oxygenated blood to systemic circulation
  - Pulmonary veins → left atrium → left ventricle → aorta → body
  - Left ventricle three times thicker than right ventricle (↑ systemic circulation resistance)

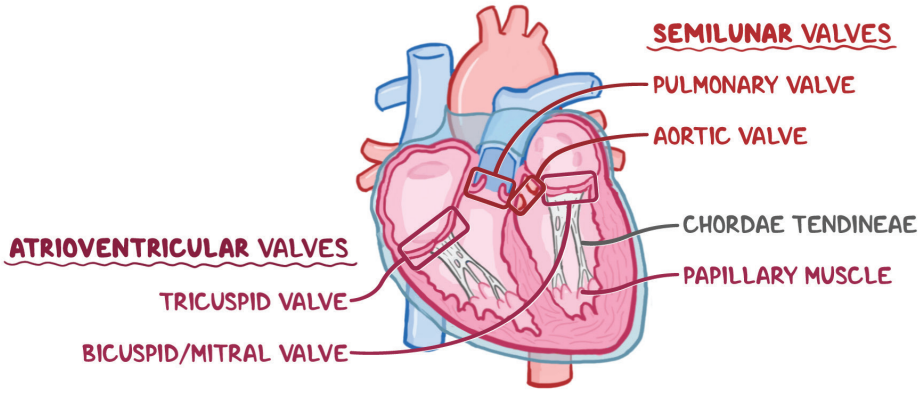

Figure 14.4 The four heart valves. The chordae tendineae and papillary muscles attached to the atrioventricular valves prevent blood backflow into the atria.

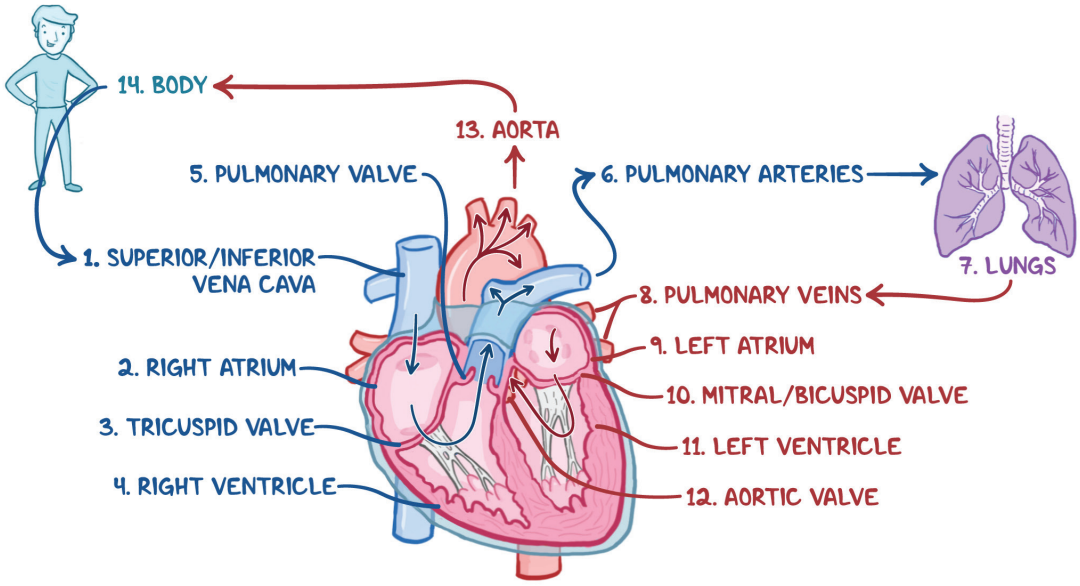

Figure 14.5 Blood flow physiology starting with the superior and inferior vena cavae bringing deoxygenated blood from the body to the right atrium of the heart.

VENTRICULAR SYSTOLE VS. DIASTOLE

Systole

- Ventricular contraction/atrial relaxation
- Occurs during S1 sound
  - Aortic, pulmonic valves open → blood pushed into aorta, pulmonary arteries
- Systolic blood pressure
  - Arterial pressure when ventricles squeeze out blood under high pressure
  - Peripheral pulse felt

Diastole

- Ventricular relaxation/atrial contraction
- Occurs during S2 sound
  - Tricuspid, mitral valves open → blood fills ventricles
- Diastolic blood pressure
  - Ventricles fill with more blood (lower pressure)

BLOOD DISTRIBUTION

- *Average adult:* 5L/1.32gal total blood volume (not cardiac output)
- 10% of total volume (approx. 500ml/0.13gal) in pulmonary arteries, capillaries, pulmonic circulatory veins
- 5% of total volume (250ml/0.07gal) in one of four heart chambers
- 15% (750ml/0.2gal) in systemic arteries
  - 15% to brain
  - 5% nourishes heart
  - 25% to kidneys
  - 25% to GI organs
  - 25% to skeletal muscles
  - 5% to skin
- 5% (250ml/0.07gal) in systemic capillaries
- 65% (3.25L/0.86gal) in systemic veins
- Numbers can change (e.g. exercise)

BLOOD FLOW TERMINOLOGY

Preload

- Amount of blood in left ventricle before contraction
- Determined by filling pressure (end diastolic pressure)
- "Volume work" of heart

Afterload

- Resistance (load) left ventricle needs to push against to eject blood during contraction
- "Tension work" of heart
- Components include
  - Amount of blood in systemic circulation

- Degree of arterial vessel wall constriction (for left side of heart, main afterload source is systemic arterial resistance; for right side of heart, main afterload source is pulmonary arterial pressure)

Stroke volume (SV)

- Blood volume (in liters) pumped by heart per contraction
- Determined by amount of blood filling ventricle, compliance of ventricular myocardium

Cardiac output (CO)

- Blood volume pumped by heart per minute (L/min)
- CO = SV * heart rate
- Example
  - SV = 70mL ejected per contraction
  - HR = 70bpm
  - CO = 70 * 70 = 4900mL/min = 4.9L/min

Venous return

- Blood-flow from veins back to atria

Ejection fraction (EF)

- Percentage of blood leaving heart during each contraction
- EF = (stroke volumeend diastolic volume) * 100

Frank–Starling Mechanism

- Ventricular contraction strength related to amount of ventricular myocardial stretch
- Maximum contraction force achieved when myocardial actin, myosin fibers are stretched about 2–2.5 times normal resting length

BLOOD VESSEL LAYERS ("TUNICS")

Tunica intima (interna)

- Innermost layer

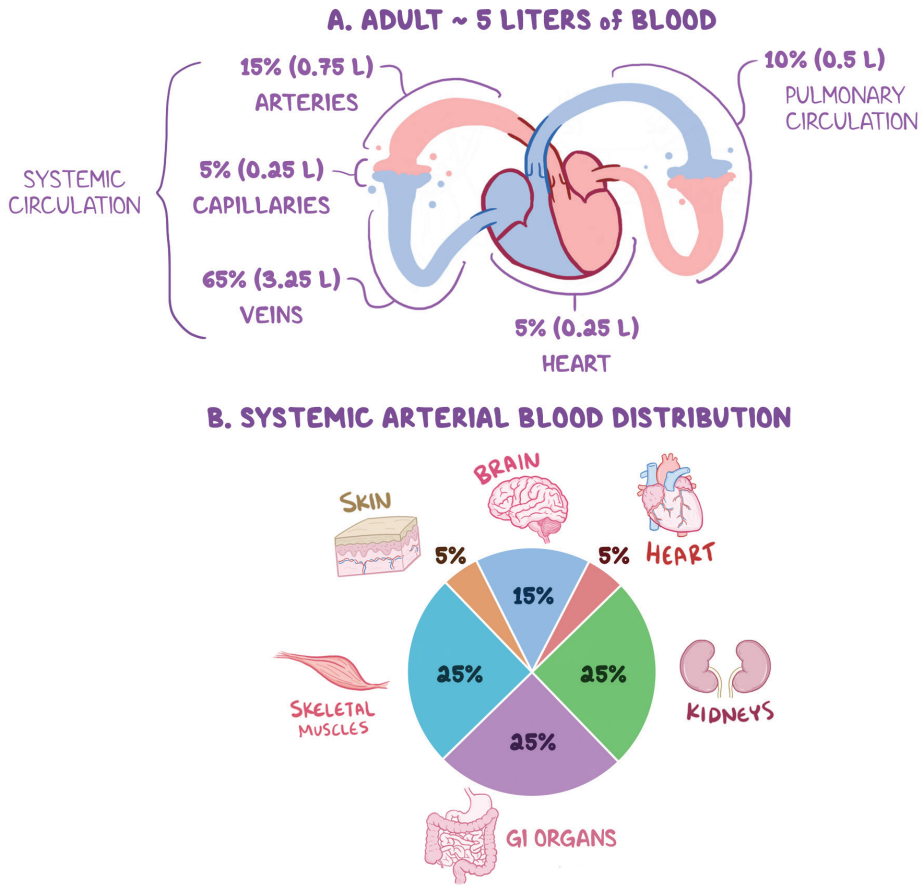

Figure 14.6 **A:** Total blood volume distribution in an average adult. **B:** Systemic arterial blood distribution.

- Endothelial cells create slick surface for smooth blood flow
- Receives nutrients from blood in lumen
- Only one cell thick
  - Larger vessels may have subendothelial basement membrane layer (supports endothelial cells)

Tunica media

- Middle layer
- Mostly made of smooth muscle cells, elastin protein sheets
- Receives nutrients from blood in lumen

Tunica externa

- Outermost layer
- Made of loosely woven fibers of collagen, elastic
  - Protects, reinforces blood vessel; anchors it in place
- Vaso vasorum ("vessels of the vessels")
  - Tunica externa blood vessels are very large, need own blood supply

ARTERIES

Key features

- High pressure, thicker than veins, no valves

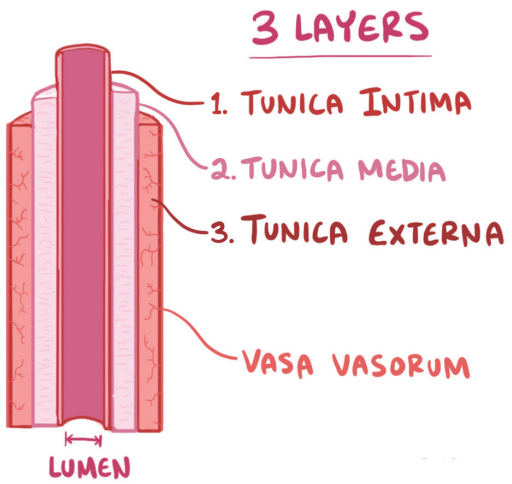

Figure 14.7 The three layers, or "tunics," of a blood vessel.

Types

- "Elastic" arteries (conducting arteries)
  - Lots of elastin in tunica externa, media
  - Stretchy; allows arteries to expand, recoil during systole, diastole
  - Absorbs pressure
  - Largest arteries closest to heart (aorta, main branches of aorta, pulmonary arteries) have most elastic in walls
- Muscular arteries (distributing arteries)
  - Carry blood to organs, distant body parts
  - Thick muscular layer
- Arterioles (smallest arteries)
  - Artery branches when they reach organs, tissues
  - Major systemic vascular resistance regulators
  - Bulky tunica media (thick smooth muscle layer)
  - Regulate blood flow to organs, tissues
  - Contract (vasoconstriction) in response to hormones/autonomic nervous system, ↓ blood/↑ systemic resistance
  - Vasodilate (relax) ↑ blood flow to organs/tissues, ↓ systemic resistance
  - Ability to contract/dilate provides thermoregulation

VEINS

Key features

- Low pressure
- Cannot tolerate high pressure but are distensible → adapts to different volumes, pressures
- Have valves (folds in tunica interna) to resist gravity, keep blood flowing unidirectionally heart

Types

- *Venules:* small veins that connect to capillaries

CAPILLARIES

- Only one cell thick (flat endothelial cells)
- Oxygen, carbon dioxide, nutrients, metabolic waste easily exchanged between tissues; circulation through capillary wall by diffusion

- Fluid moves out of vessel, into interstitial space (space between blood vessels, cells)
  - Water-soluble substances (ions) cross capillary wall through clefts, between endothelial cells, through large pores in fenestrated capillary walls
  - Lipid-soluble molecules (oxygen, carbon dioxide) dissolve, diffuse across endothelial cell membranes

BULK FLOW

- Passive water, nutrient movement across capillary wall down concentration gradient

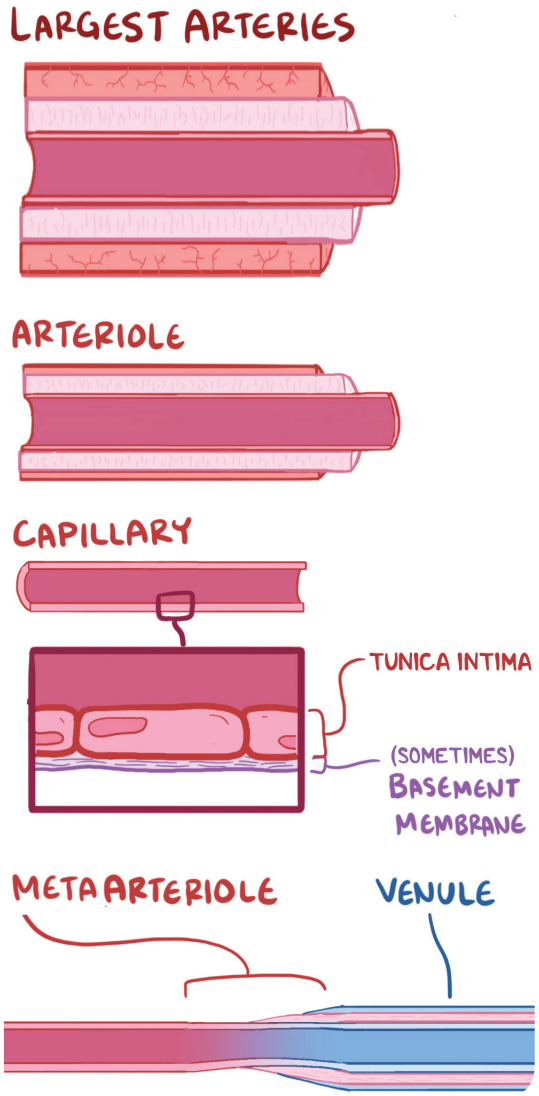

Figure 14.8 Key features of different blood vessel types.

Key features

- Moves large amounts of water, substances in same direction through fenestrated capillaries
- Material movement
- Faster transport method
- Regulates blood, interstitial volume
- Filtration, reabsorption
- Continuous fluid mixing between plasma, interstitial fluid

Types

- *Filtration:* bulk flow when moving from blood to interstitium
- *Reabsorption:* bulk flow when moving from interstitium to blood

Other characteristics

- *Kidney:* major site of bulk flow where waste products are filtered out, nutrients reabsorbed
- Fluid filters out of capillaries into interstitial space (net filtration) at arteriolar end, reabsorbed (net reabsorption) at venous end
  - Hydrostatic interstitial fluid pressure draws fluid into capillary
  - Hydrostatic capillary pressure pushes fluid out of capillary
  - Colloid interstitial fluid pressure pushes fluid out of capillary
  - Colloid capillary pressure draws fluid into capillary

MICROCIRCULATION

- *Microcirculation:* arterioles + capillaries + venules
- Arteriole blood flow through capillary bed, to venule (nutrient, waste, fluid exchange)
  - Capillary beds composed of vascular shunt (vessel connects arteriole, venule to capillaries), actual capillaries
  - Terminal arteriole → metarteriole → thoroughfare channel → postcapillary venule
  - *Precapillary sphincter:* valve regulates blood flow into capillary
  - Various chemicals, hormones, vasomotor nerve fibers regulate amount of blood entering capillary bed

LYMPHATIC ANATOMY & PHYSIOLOGY

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LYMPHATIC SYSTEM

Function

- Fluid balance
  - Returns leaked interstitial fluid, plasma proteins to blood, heart via lymphatic vessels
  - *Lymph:* name of interstitial fluid when in lymph vessels
  - *Lymphedema:* lymph dysfunctional/absent (lymph node removal in cancer) → edema forms
- Immunity
- Fat absorption

Lymphatic capillaries

- Collect interstitial fluid leaked by capillaries
- Found in all tissues (except bone, teeth, marrow)
  - Microscopic dead-ended vessels unlike blood capillaries, helps fluid remain inside
  - Usually found next to blood capillaries
- Lymph moves via breathing, muscle contractions, arterial pulsation in tight tissues
- Carries particles away from inflammation sites/injury towards bloodstream, stopping first through lymph nodes that filter out harmful substances
- Overlapping endothelial cells create valves; prevent backflow, infectious spread
- *Lacteals:* specialized lymphatic capillaries found in small intestine villi
  - Carry absorbed fats into blood
  - *Chyle:* fat-containing lymph

Larger lymphatics

- Capillaries → collecting vessels → trunks → ducts → angle of jugular, subclavian veins; right lymphatic duct empties into right angle, thoracic into left
- Collecting vessels have more valves, more anastomoses than veins
  - Superficial collecting vessels follow veins
  - Deep collecting vessels follow arteries
- Lymphatic trunks
  - *Paired:* lumbar, bronchomediastinal, subclavian, jugular
  - *Singular:* intestinal

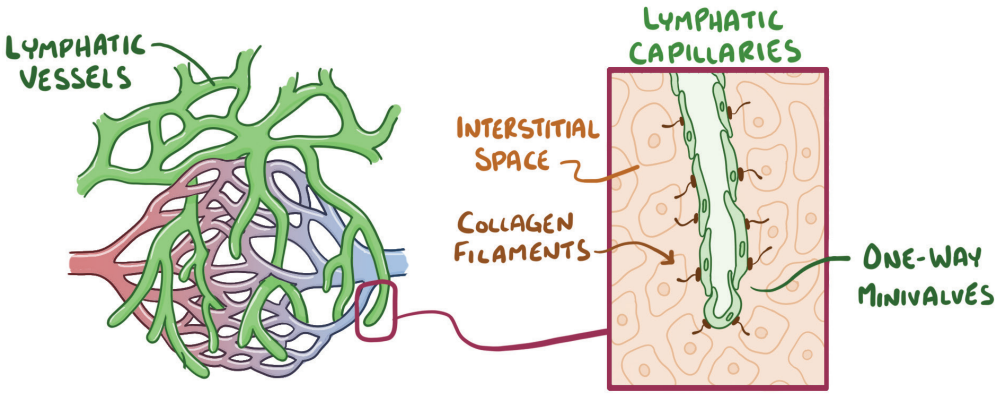

Figure 14.9 Lymphatic vessels collect interstitial fluid (which is then called lymph) and return it to the veins. Lymphatic capillaries have minivalves that open when pressure in the interstitial space is higher than in the capillary and shut when pressure in the interstitial space is lower.

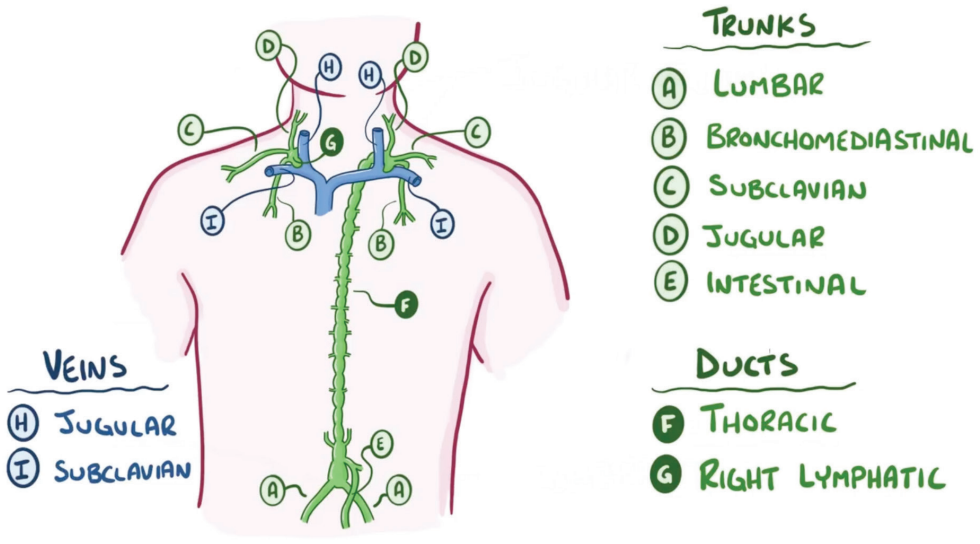

Figure 14.10 Lymphatic system structures and their locations in the body.

- Ducts
  - Upper right lymphatic drains right arm; right thorax; right side of head, neck
  - Thoracic duct drains into cisterna chyli (a dilation created to gather all lymph drained from body area that's not covered by upper right lymphatic duct)

LYMPHOID CELLS

- *Lymphocytes:* T subtype activate immune response; B subtype → plasma cells, produce antibodies
- *Macrophages:* important in T cell activation, phagocytosis
- *Dendrocytes:* return to nodes from inflammation sites to present antigens
- *Reticular cells:* similar to fibroblasts; create mesh to contain other immune cells

LYMPHOID TISSUES

- Reticular connective tissue
- *Composition:* macrophage-embedded reticular fibers
- Loose
  - Diffuse lymphoid tissue
  - Venules enter, filters blood
  - Found in all organs
- Dense
  - Follicles/nodules
  - Mostly contain germinal centers
  - Found in larger organs (lymph nodes)/ individually (mucosa)

LYMPHOID ORGANS

Spleen

- Largest lymphoid tissue in body
- Located below left side of diaphragm
- Blood supplied by splenic artery; blood leaves spleen via splenic vein
  - Capsules with projections into organ, form splenic trabeculae
- Function
  - Macrophages remove foreign particles, pathogens from blood
  - Red blood cell turnover
  - Compound storage (e.g. iron)
  - Platelet/monocyte storage
  - *Blood reservoir:* stores about 300mL/0.08gal
  - Fetal erythrocyte production
- Histology
  - *White pulp:* lymphocyte, macrophage islands that surround central arteries
  - *Red pulp:* composed mostly of red blood cells, macrophages; macrophages remove old red blood cells, platelets; splenic cords (reticular tissues running between venous sinusoids)

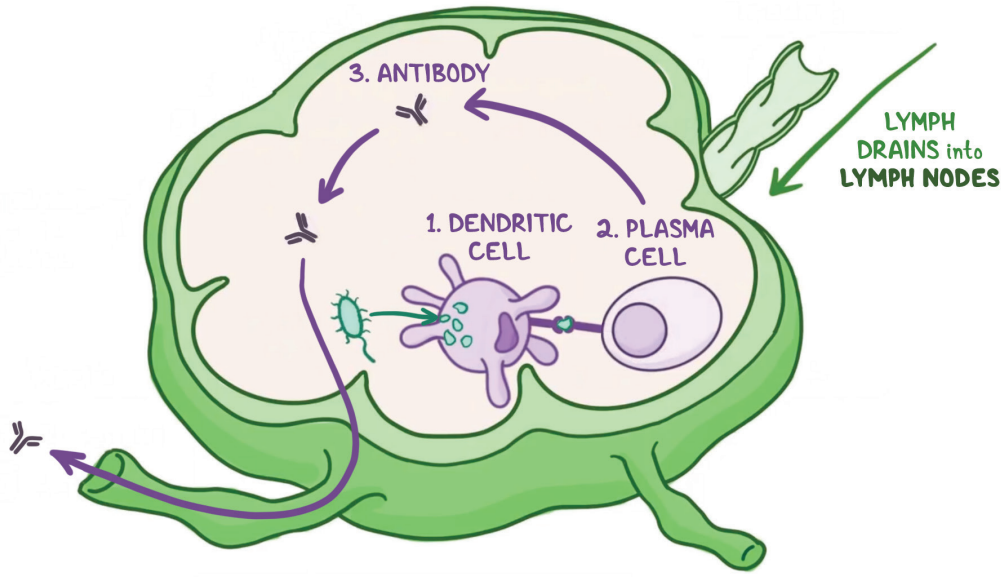

Figure 14.11 In lymph nodes, dendritic cells present pieces of pathogens they come across to B cells. If a dendritic cell presents something foreign to a B cell, the B cell turns into a plasma cell and starts secreting antibodies, which flow into the lymph and exit the lymph node.

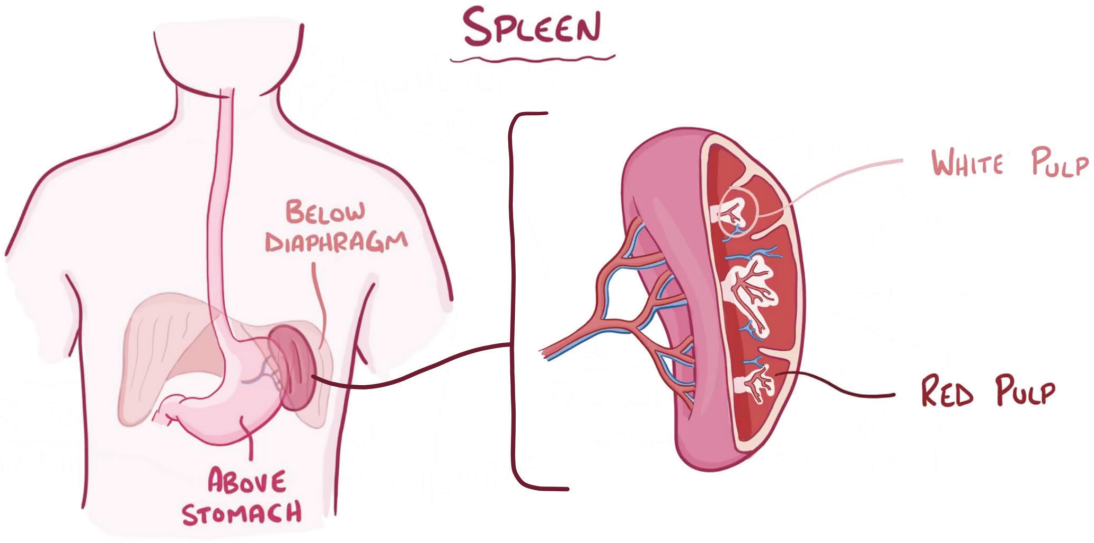

Figure 14.12 Spleen location, histology.

Lymph nodes

- Hundreds scattered throughout body, often grouped along lymphatic vessels
  - Superficial, deep
  - Many found in inguinal, axillary, cervical regions
- Function
  - Lymph filtration, immune system activation
- Kidney-shaped formations
  - Built like tiny spleens, 1–25cm/0.4–9.8in long
  - Covered by capsule with trabeculae, extend inward; trabeculae divide nodes sectionally
- Cortex
  - Subcapsular sinus, lymphoid follicle, germinal center

- Medulla
  - Medullary cord, medullary sinus
- Lymph flows through afferent lymphatic vessels → enters node through hilum → subcapsular sinus → cortex → medullary sinus → exiting via efferent lymphatic vessels in hilum
  - Fewer efferent vessels than afferent vessels, slows traffic down → allows node to filter lymphatic fluid
- Swollen painful nodes indicate inflammation, painless nodes may indicate cancer

Thymus

- Located between sternum, aorta in mediastinum
- Two lobes, many lobules composed of cortex, medulla
  - *Cortex:* T lymphocyte maturation site (immature T lymphocytes move from bone marrow to thymus for maturation)
  - *Medulla:* contains some mature T lymphocytes, macrophages, cell-clusters called thymic corpuscles (corpuscles contain special T lymphocytes thought to be involved in preventing autoimmune disease)
- Lymphocyte production site in fetal life
  - Active in neonatal, early life; atrophies with age

Bone marrow

- *B cells:* made, mature in bone marrow
- *T cells:* made in bone marrow, mature in thymus

Mucosa-associated lymphoid tissue (MALT)

- Lymphoid tissue that is associated with mucosal membranes
- *Tonsils:* lymphoid-tissue ring around pharynx
  - Have crypts (epithelial invaginations) which trap bacteria
  - *Palatine:* paired tonsils on each side of pharynx (largest tonsils, most often inflamed)
  - *Lingual:* near base of tongue
  - *Pharyngeal:* near nasal cavity (called adenoid when inflamed)
  - *Tubal:* near Eustachian tube
- *Peyer's patches:* small bowel MALT

Appendix

- Worm-like large bowel extension
- Contains numerous lymphoid follicles
- Fights intestinal infections

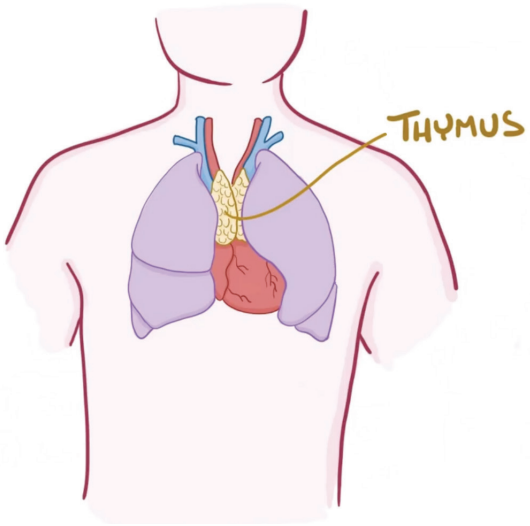

Figure 14.13 Thymus location.

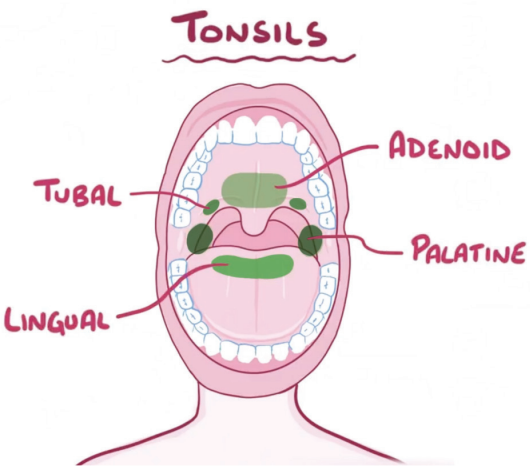

Figure 14.14 Tubal, pharyngeal (adenoid), palatine, and lingual tonsils create a lymphoid-tissue ring around pharynx.

NORMAL HEART SOUNDS

osms.it/normal-heart-sounds

HEART SOUNDS

Causes

- Opening / closing cardiac valves
- *Blood movement:* into chambers, through pathological constrictions, through pathological openings

WHERE ARE THEY HEARD?

- By auscultating specific points individual sounds can be isolated
  - These points are not directly above their respective valves, but are where valve sounds are best heard; however, they generally map a representation of different heart chambers
- Knowing normal heart size, auscultation locations allows for enlarged (diseased) heart detection

Optimal auscultation sites

- *Aortic valve sounds:* 2nd intercostal, right sternal margin
- *Pulmonary valve sounds:* 2nd intercostal space, left sternal margin
- *Tricuspid valve sounds:* 4/5th intercostal, left sternal margin
- *Mitral valve sounds:* 5th intercostal space, midclavicular line (apex)

NORMAL HEART SOUNDS

- Two sounds for each beat
  - Lub (S1), dub (S2)
- Factors affecting intensity
  - Intervening tissue, fluid presence, quantity
  - Mitral valve closure speed (mitral valve contraction strength)

S1 heart sound

- "*Lub*": low-pitched sound
- Marks beginning of systole/end of diastole
- Early ventricular contraction (systole) → ventricular pressure rises above atrial pressure → atrioventricular valves close → S1
- S1: mitral, tricuspid closure
  - Intensity predominantly determined by mitral valve component, loudest at apex
- S1 (lub) louder, more resonant than S2 (dub)
- S1 displays negligible variation during breathing

S2 heart sound

- "*Dub*": higher-pitched sound
- Marks end of systole/beginning of diastole
- S2: semilunar valves (aortic, pulmonic) snap shut at beginning of ventricular relaxation (diastole) → short, sharp sound
- Best heard at Erb's point, 3rd intercostal space on left, medial to midclavicular line
- Splits on expiration
  - During expiration S2 split into earlier aortic component; later, softer pulmonic component (A2 P2). Lower intrathoracic pressure during inspiration → ↑ right ventricular preload → ↑ right ventricular systole duration → delays P2
  - ↓ left ventricular preload during inspiration → shorter ventricular systole, earlier A2
  - A2, P2 splitting during inspiration usually about 40ms
  - A2, P2 intensity roughly proportional to respective systemic. pulmonary circulation pressures
  - P2 best heard over pulmonic area

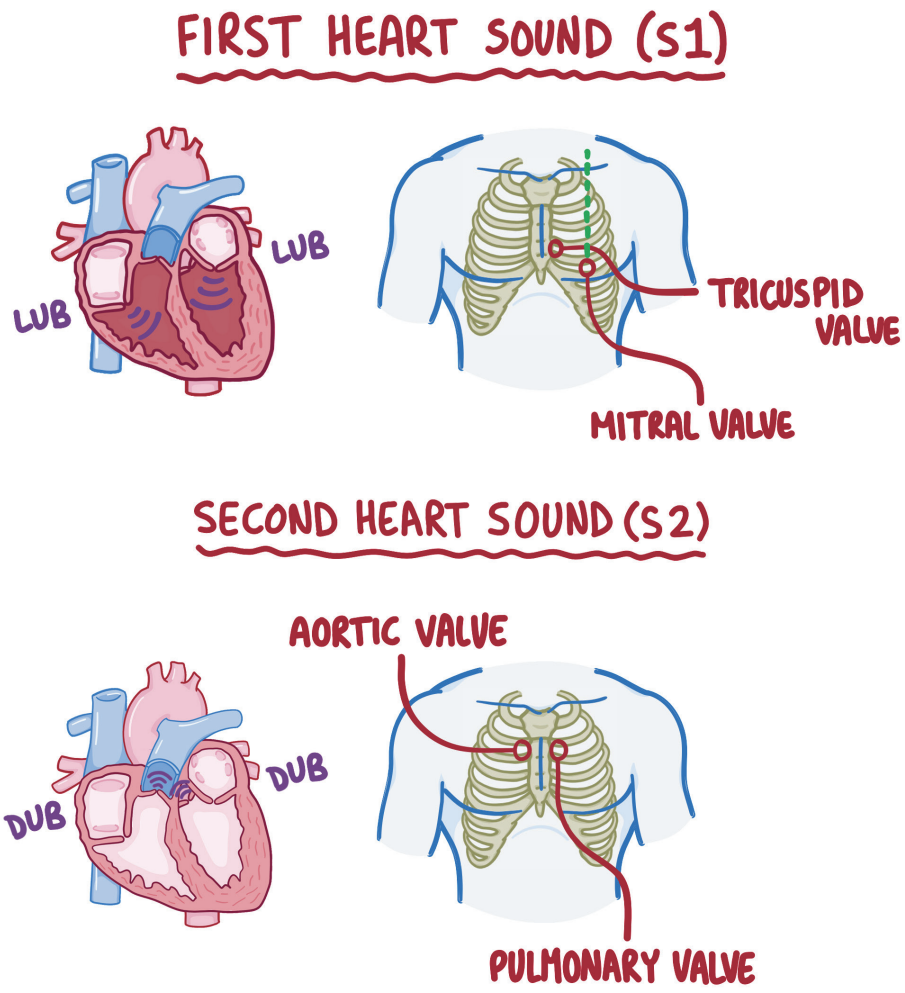

Figure 14.15 Valves that close to produce S1 and S2 sounds and optimal auscultation sites.

ABNORMAL HEART SOUNDS

osms.it/abnormal-heart-sounds

ABNORMAL S1

Loud S1

- As left ventricle fills, pressure increases
- As left atrium empties, pressure increases as it empties against increasingly pressure-loaded ventricle; as atrium approaches empty, pressure begins to decrease
- *Differential diagnosis:* short PR interval, mild mitral stenosis, hyperdynamic states
- Short PR interval (< 120ms)
  - Normally atrioventricular valve leaflets drift towards each other before onset of systole
  - Shorter PR interval → less time to drift closure → wider closure distance → louder S1
  - Short PR interval → incomplete ventricular emptying → higher ventricular filling pressure → ventricular pressure crosses critical atrioventricular valve closing threshold while atrial pressures are still high → load snap

- Mild mitral stenosis
  - Significant force required to close stenotic mitral valve → large atrioventricular pressure gradient required
  - Slam shut with increased force, producing loud sound
- Hyperdynamic states
  - Shortened diastole → large amount of ongoing flow across valve during systole → leaflets wide apart, pressure remains high
  - Results in forceful atrioventricular valve closure

Soft S1

- *Differential diagnosis:* long PR intervals, severe mitral stenosis, left bundle branch block, chronic obstructive pulmonary disease (COPD), obesity, pericardial effusion
- Long PR intervals (> 200ms)
  - Atrium empties fully → low pressure → low ventricular pressure required to close atrioventricular valves → valves close when ventricle is in early acceleration phase (low pressures) → soft sound
- Severe mitral stenosis
  - Leaflets too stiff, fixed to change position

Variable S1

- Auscultatory alternans
  - When observed with severe left ventricular dysfunction, correlate of pulsus alternans
- *Differential diagnosis:* atrioventricular dissociation, atrial fibrillation, large pericardial effusion, severe left ventricular dysfunction

Split S1

- S1 usually a single sound
  - Near-simultaneous mitral, tricuspid valve closures; soft intensity of tricuspid valve closure
- Splitting usually from tricuspid valve closure being delayed relative to mitral valve closure
- *Differential diagnosis:* right bundle branch block, left-sided preexcitation, idioventricular rhythm arising from left ventricle

ABNORMAL S2

Split S2

- Physiological S2 splitting
  - *Expiration:* S1 A2P2 (no split)
  - *Inspiration:* S1 A2....P2 (40ms split)
- Wide split
  - *Detection:* splitting during expiration
  - *Expiration:* S1 A2..P2 (slight split)
  - *Inspiration:* S1 A2.....P2 (wide split)
  - *Differential diagnosis:* right bundle branch block, left ventricle preexcitation, pulmonary hypertension, massive pulmonary embolism, severe mitral regurgitation, constrictive pericarditis
- Fixed split
  - Splitting during both expiration, inspiration; does not lengthen during inspiration
  - *Expiration:* S1 A2..P2 (slight split)
  - *Inspiration:* S1 A2..P2 (slight split)
  - *Differential diagnosis:* atrial septal defect, severe right ventricular failure
- Reversed split
  - Split during expiration, but not inspiration
  - *Expiration:* S1 P2....A2 (moderate split)
  - *Inspiration:* S1 P2A2
  - *Differential diagnosis:* left bundle branch block, right ventricle preexcitation, aortic stenosis/AR

Abnormal single S2 variants

- Loud P2
  - *Expiration:* S1 A2P2
  - *Inspiration:* S1 A2....P2!
  - *Diagnosis:* pulmonary hypertension
- Left ventricular outflow obstruction
  - Absent A2
  - *Expiration:* S1 P2
  - *Inspiration:* S1 P2
  - *Diagnosis:* severe aortic valve disease
- Fused A2/P2
  - *Expiration:* S1 A2P2
  - *Inspiration:* S1 A2P2
  - *Differential diagnosis:* ventricular septal defect with Eisenmenger's syndrome, single ventricle

ADDED HEART SOUNDS

S3 heart sound

- S3 (ventricular gallop)
  - Low-pitched early diastolic sound
  - Best heard in mitral/apex region
  - Left lateral decubitus position
- Associated with volume overload conditions
- Early diastolic sound, produced in rapid filling phase → excessive volume filling ventricle in short period → rapid filling → chordae tendineae tensing → S3 sound
- *Children/adolescents:* may be normal
- *Middle aged/elderly person:* usually pathological
  - *Over 40 years old:* indicative of left ventricular failure
- *Auscultatory summary:* S1... S2.S3... S1

S4 heart sound

- *S4 (atrial gallop):* low pitched late diastolic (pre-systolic) sound, best heard in mitral/apex region, left lateral decubitus position
- Associated with hypertension, left ventricular hypertrophy, ischaemic cardiomyopathy
- *Pressure overload:* thought to be caused by atrial contraction into stiff / non-compliant ventricle
- Chronic heart contraction effort against increased pressure → hypertrophy → stiff ventricle (concentric hypertrophy)
- Always pathological
- *Auscultatory summary:* S4.S1...S2...S4.S1

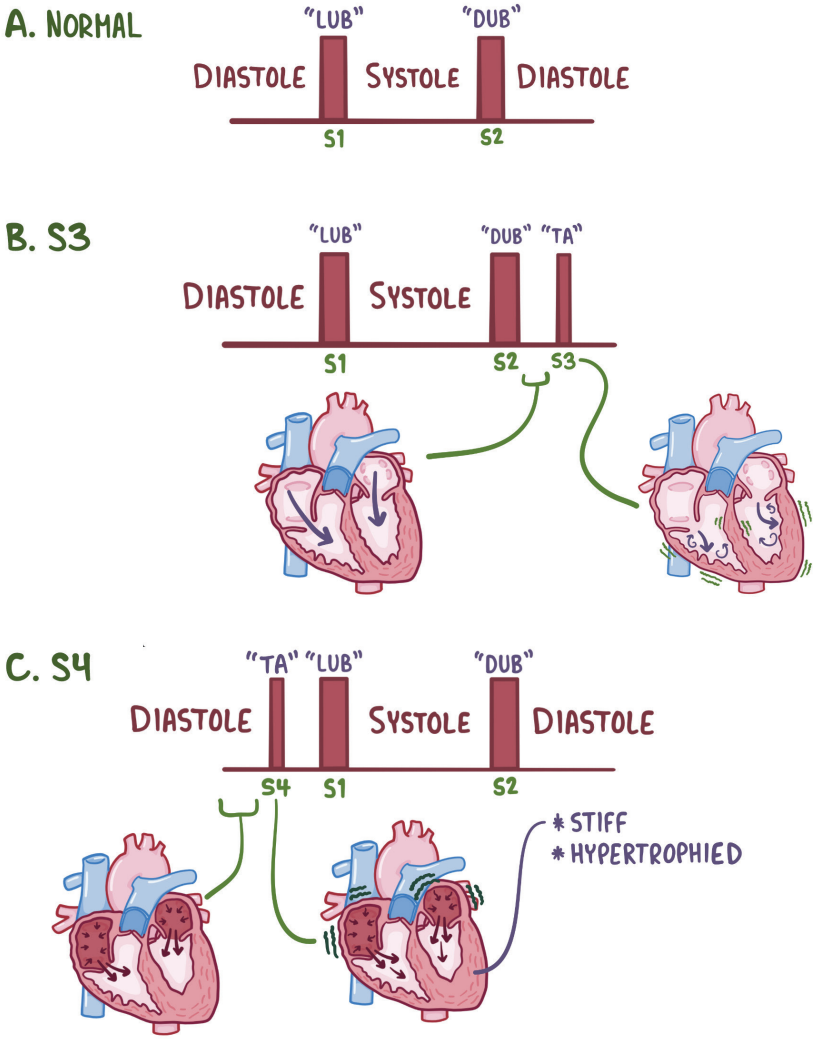

Figure 14.16 Linear representation of **A:** normal (S1, S2), **B:** S3, and **C:** S4 heart sounds.

Summation gallop

- Superimposition of atrial, ventricular gallops during tachycardia
- Heart rate ↑ → diastole shortens more than systole → S3, S4 brought closer together until they merge

HEART MURMURS

Key features

- Blood flow silent when laminar, uninterrupted
- Turbulent flow may generate abnormal sounds (AKA "heart murmurs")
- Murmurs can be auscultated with stethoscope

Causes

- May be normal in young children, some elderly individuals
- ↓ blood viscosity (e.g. anaemia)
- ↓ diameter of vessel, valve, orifice (e.g. valvular stenosis, coarctation of aorta, ventricular septal defect)
- ↑ blood velocity through normal structures (e.g. hyperdynamic states—sepsis, hyperthyroid)
- Regurgitation across incompetent valve (e.g. valvular regurgitation)

Describing heart murmurs

- Specific language used to describe murmurs in diagnostic workup
- *Timing:* refers to timing relative to cardiac cycle
  - *Systolic "flow murmurs":* aortic, pulmonic stenosis; mitral, tricuspid regurgitation; ventricular septal defect; aortic outflow tract obstruction
  - *Diastolic:* aortic, pulmonic regurgitation; mitral, tricuspid stenosis
  - Continuous murmurs are least common, generally seen in children with congenital heart disease (e.g. patent ductus arteriosus, cervical venous hum)
  - Occasionally may have two related murmurs, one systolic, one diastolic; gives impression of continuous murmur (e.g. concurrent aortic stenosis, aortic regurgitation)
- Location
  - Location on chest wall where murmur is best heard
- Radiation
  - Location where murmur is audible despite not lying directly over heart
  - Generally radiate in same direction as turbulent blood is flowing
  - *Aortic stenosis:* carotid arteries
  - *Tricuspid regurgitation:* anterior right thorax
  - *Mitral regurgitation:* left axilla
- Shape
  - How sound intensity changes from onset to completion
  - Shape determined by pattern of pressure gradient driving turbulent flow, loudest segment occurring at time of greatest gradient (moment of highest velocity)
  - *Three basic shapes:* crescendo-decrescendo, uniform (holosystolic when occurring during systole), decrescendo
  - Crescendo-decrescendo, uniform generally systolic; decrescendo murmurs generally diastolic

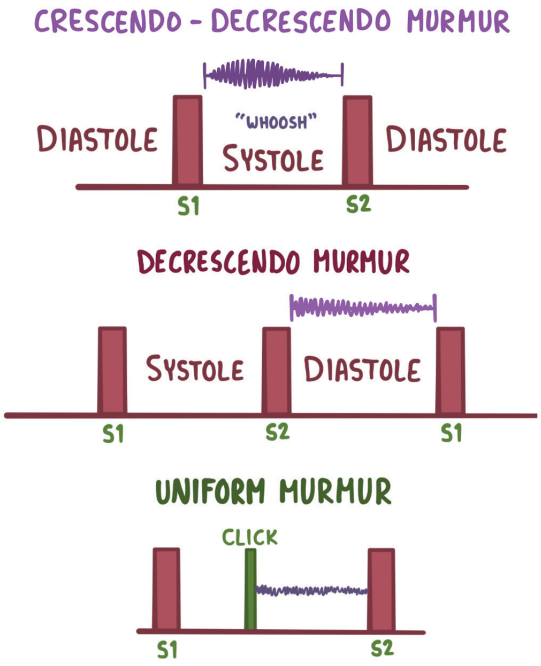

Figure 14.17 Three basic heart murmur shapes: crescendo-decrescendo, decrescendo, uniform/holosystolic.

- Pitch
  - High pressure gradients → high pitched murmurs (e.g. mitral regurgitation, ventricular septal defect)
  - Large volume of blood-flow across low pressure gradients → low pitched murmurs (e.g. mitral stenosis)
  - If both high pressure, high flow (severe aortic stenosis), both high, low pitches are produced simultaneously → subjectively unpleasant/"harsh" sounding murmur
- Intensity
  - Murmur loudness graded on scale from I–VI
  - Dependent on blood velocity generating murmur; acoustic properties of intervening tissue; hearing; examiner experience; stethoscope used, ambient noise presence
  - *I:* barely audible
  - *II:* faint, but certainly present
  - *III:* easily, immediately heard
  - *IV:* associated with thrill (palpable vibration over involved heart valve)
  - *V:* heard with only edge of stethoscope touching chest wall
  - *VI:* heard without stethoscope (or without it making direct contact with chest wall)
- Quality
  - Subjective, attempt to describe timbre, depends on how many different base frequencies of sound are generated, relative amplitude of various harmonics
  - *Mitral regurgitation:* blowing/musical
  - *Mitral stenosis:* rumbling
  - *Aortic stenosis:* harsh
  - *Aortic regurgitation:* blowing
  - *Still's murmur (benign childhood):* musical
  - *Patent ductus arteriosus:* machine-like

Diagnostic maneuvers (dynamic auscultation)

- Some maneuvers may elicit characteristic intensity/timing changes (changes in hemodynamics during maneuvers)
- *Dynamic auscultation:* listening for subtle changes during physical maneuvers
- Inspiration
  - ↓ intrathoracic pressure → ↑ pulmonary venous return to right heart → ↑ right heart stroke volume → right sided murmurs → ↑ intensity
  - Dilation of pulmonary vascular system → ↓ pulmonary venous return to left side of heart → ↓ left heart stroke volume → left side murmurs → ↓ intensity
- Expiration
  - ↑ intrathoracic pressure → ↓ venous return to right heart → ↓ right ventricle stroke volume → ↓ intensity of right sided murmurs
  - ↑ pulmonary venous return to left side → ↑ left ventricle stroke volume → left sided murmur → ↑ intensity
- Valsalva maneuver
  - Forceful exhalation against closed glottis
  - ↓ venous return to heart → ↓ left ventricular volume → ↓ cardiac output
  - Murmurs of hypertrophic obstructive cardiomyopathy, occasionally mitral valve prolapse → ↑ intensity
  - All other systolic murmurs → ↓ intensity
- Isometric handgrip
  - Squeeze two objects (such as rolled towels) with both hands
  - Do not simultaneously Valsalva
  - If unconscious, simulate by transient arterial occlusion (BP cuffs applied to both upper arms, inflated to 20–40mmHg above systolic blood pressure for 20 seconds)
  - ↑ venous return, ↑ sympathetic tone → ↑ heart rate, systemic venous return → ↑ cardiac output → murmurs from mitral regurgitation, aortic regurgitation, ventricular septal defect → ↑ intensity
  - Murmur from hypertrophic obstructive cardiomyopathy → ↓ intensity
  - Murmur from aortic stenosis → most commonly unchanged
- Leg elevation
  - Lying supine, both legs raised 45°
  - ↑ venous return → ↑ left ventricular volume
  - Murmur from hypertrophic obstructive cardiomyopathy → ↓ intensity
  - Murmurs from aortic stenosis, mitral regurgitation may → ↑ intensity

- Müller's maneuver
  - Nares closed, forcibly suck on incentive spirometer/air-filled syringe for 10 seconds (conceptual opposite of Valsalva)
  - ↓ venous return → ↓ left ventricular volume → ↓ systemic venous resistance murmur from hypertrophic obstructive myopathy → ↑ intensity
  - Murmur from aortic stenosis may → ↓ intensity
- Squatting to standing
  - Abruptly stand up after 30 seconds of squatting
  - ↓ venous return → ↓ left ventricular volume
  - Murmur from hypertrophic obstructive cardiomyopathy → ↑ intensity
  - Murmur from aortic stenosis may → ↓ intensity
- Standing to squatting
  - From standing upright, squat down
  - If unable to squat, examiner can passively bend knees up towards abdomen to mimic maneuver
  - ↑ venous return → ↑ left ventricular volume
  - Murmur from hypertrophic obstructive cardiomyopathy → ↓ intensity
  - Murmur from aortic stenosis may → ↑ intensity
  - Murmur from aortic regurgitation → ↑ intensity

Systolic murmurs

- Aortic stenosis
  - *Aortic valve auscultation site:* 2nd intercostal, right sternal margin
  - S1, closing of mitral valve, during systole → heart contracts against closed stenotic aortic valve → pressure must rise during systole to force open stenotic aortic valve → valve pops open → produces ejection click
  - Followed by ↑ flow as heart contracts more forcefully to empty left ventricle → murmur intensity ↑ as flow across partially open valve ↑
  - Chamber begins to empty → pressure, flow diminish → ↓ murmur intensity
  - Radiates to neck/carotids (murmur occurs in aorta, these are its first branches)
  - *Auscultatory summary:* S1. Ejection click. Crescendo-decrescendo murmur. S2
- Pulmonic stenosis
  - *Pulmonary valve auscultation site:* 2nd intercostal space, left sternal margin
  - S1, closing of tricuspid valve, during systole
  - Heart contracts against closed pulmonic valve → pressure builds during systole, forcing open stenotic pulmonic valve → valve pops open → ejection click
  - Flow rate increases as heart contracts more forcefully to empty right ventricle → murmur gets louder as flow across partially open valve increases → chamber empties → pressure, flow diminishing → ↓ murmur intensity
  - Radiates to neck/carotids, back
  - *Auscultatory summary:* S1. Ejection click. Crescendo-decrescendo murmur. S2
- Mitral regurgitation
  - *Mitral valve auscultation site:* 5th intercostal space, midclavicular line/apex
  - Holo-/pansystolic murmur (occurs for systole duration)
  - Normal S1 as mitral valve closes → in mitral regurgitation, valve cannot completely close → pressure builds in left ventricle (with closed aortic valve) → blood forced back through partially closed mitral valve → murmur occurs along with S1 as long as pressures remain high enough
  - Aortic valve will open to redirect majority of blood → left ventricle continues contracting → continuously raised pressures → blood continuously flowing through partially closed mitral valve (whole of systole)
  - As heart continues to contract, pressure ↑, but atrium becomes more compliant. Even though blood-flow across partially closed valve may ↑, pressure in atrium does not significantly increase
  - Left ventricle pressure notably higher than left atrium → sound does not

change throughout murmur
    - Referred to as "flat" murmur because intensity does not change
    - Radiates to axilla due to direction of regurgitant jet
    - *Auscultatory summary:* S1. Flat murmur. S2
- Tricuspid regurgitation
    - *Tricuspid valve auscultation site:* 4/5th intercostal, left sternal margin
    - Holo-/pansystolic murmur
    - Normal S1 occurs due to tricuspid valve closure → pulmonic valve closed, pressure rises in right ventricle
    - In tricuspid regurgitation, valve cannot completely close → pressure builds in right ventricle → blood forced back out through partially closed tricuspid valve → murmur is continuous as long as pressures remain high enough
    - Pulmonic valve opens to redirect blood → left ventricle maintains contraction (thus raises pressure) → blood continues flowing through partially closed tricuspid valve (through whole systole)
    - Arium becomes more compliant as it fills → atrium pressure does not significantly increase
    - Right ventricle pressure notably higher than that of right atrium → murmur sound does not change throughout murmur
    - Referred to as "flat" murmur (intensity does not change)
    - *Auscultatory summary:* S1. flat murmur. S2
- Mitral valve prolapse
    - *Mitral valve auscultation site:* 5th intercostal space, midclavicular line/apex
    - Mitral valve billows into left atrium → clicking sound (unlike aortic stenosis, not associated with ejection of blood, non-ejection click, mid-late systolic)
    - Ventricle contracts → mitral valve closure → S1 → pressure rises → mitral valve accelerates into left atrium → stops abruptly (chordae tendineae restraint) → rapid tensing → click
    - Often associated with mitral regurgitation → after click murmur of mitral regurgitation may follow
    - *Auscultatory summary:* S1. Mid systolic click with late systolic murmur. S2

Diastolic murmurs

- Aortic regurgitation
    - *Aortic regurgitation auscultation site:* left parasternal border
    - Blood flows back through incompletely closed aortic valve
    - Occurs between S2, S1
    - S2, aortic valve closure → mitral valve opens, heart in diastole → blood enters left ventricle through regurgitant valve, through normal filling via mitral valve
    - Initially, low pressure in ventricle (compared to systemic blood pressure forcing blood through regurgitant valve) → ventricle fills → as pressure mounts, less flow through regurgitant valve → decrescendo murmur
    - Early diastolic decrescendo murmur
    - *Auscultatory summary:* S1. S2. Early diastolic decrescendo murmur. S1
- Pulmonic regurgitation
    - *Pulmonic regurgitation auscultation site:* upper left parasternal border
    - Blood flows back through incompletely closed pulmonic valve
    - Occurs between S2, S1
    - S2 aortic valve closure → tricuspid valve opens, heart in diastole → incomplete pulmonic valve closure → right ventricle fills via incompletely closed pulmonic valve as well as tricuspid valve
    - Initially → low ventricle pressure allows for high flow through regurgitant valve → pressure rises, ↓ flow through regurgitant valve → decrescendo murmur
    - Early diastolic decrescendo murmur
    - *Auscultatory summary:* S1. S2. Early diastolic decrescendo murmur. S1
- Mitral stenosis
    - *Mitral valve auscultation site:* 5th intercostal space, midclavicular line/apex
    - Mitral valve can't open efficiently
    - S2 → aortic valve closure → milliseconds later, mitral valve should open (fill ventricle during diastole), only small opening occurs

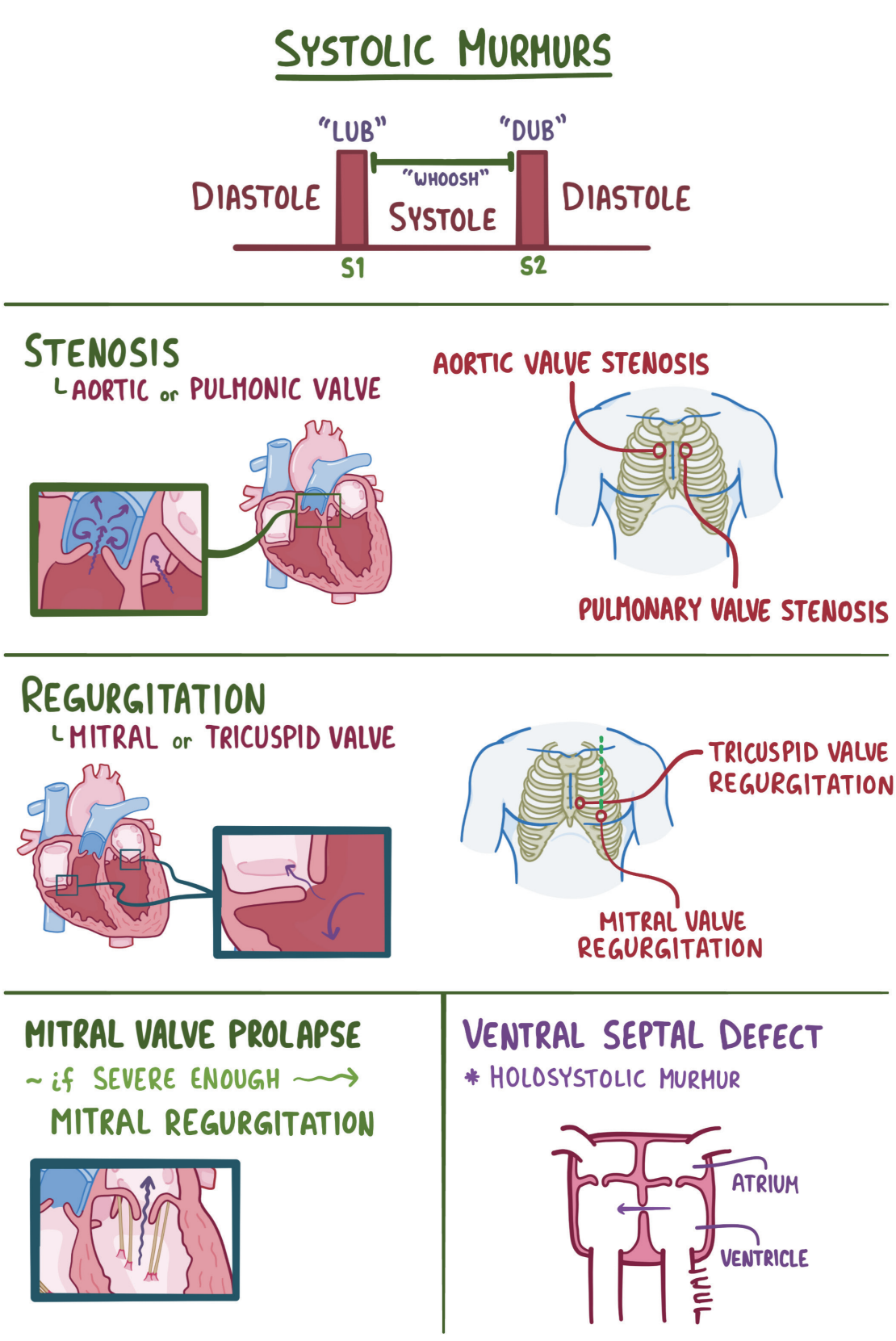

Figure 14.18 Causes of systolic murmurs.

- Beginning of diastole, highest flow of blood comes from left atrium to left ventricle (rapid filling), fills more blood at beginning of diastole (beginning due to highest pressure difference) → most intense phase of murmur
- Aortic valve closure → mitral valve opens, due to stenotic leaflets, they can only open slightly → chordae tendineae snap as limit is reached (similar to ejection snap) → opening snap from stenotic leaflets shooting open (milliseconds after S2) → highest intensity of murmur thereafter → murmur diminishes as pressure equalises
- End of diastole atrium contracts to force remaining blood into left ventricle → atrial kick sound (presystolic accentuation at end of murmur)
- *Auscultatory summary:* S1. S2. Opening snap. Decrescendo mid diastolic rumble. Atrial kick. S1

- Tricuspid stenosis
  - *Tricuspid valve auscultation site:* 4/5th intercostal space, left sternal margin
  - Tricuspid valve can't open efficiently
  - S2 → pulmonic valve closure → milliseconds later, tricuspid valve should open (fill ventricle during diastole), only small opening occurs
  - Beginning of diastole, high flow of blood comes from right atrium to right ventricle (rapid filling), fills more blood at beginning of diastole (due to highest pressure difference) → most intense murmur phase
  - Pulmonic valve closure → tricuspid valve opens (due to stenotic leaflets, they can only open slightly) → chordae tendineae snap as limit is reached (similar to ejection snap) → opening snap from stenotic leaflets shooting open (milliseconds after S2) → highest murmur intensity thereafter → murmur diminishes as pressures equalise
  - End of diastole atrium contracts to force remaining blood into left ventricle → atrial kick sound (presystolic accentuation at end of murmur)
  - *Auscultatory summary:* S1. S2. Opening snap. Decrescendo mid diastolic rumble. Atrial kick. S1

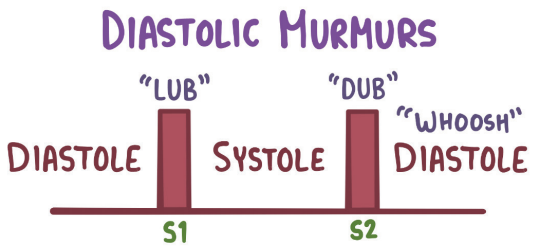

Figure 14.19 Diastolic murmurs are heard as a "whoosh" after S2.

Murmur Identification

- Detect murmur?
  - Yes/no
- Identify phase?
  - *Systolic/diastolic:* S1 -systole- S2 -diastole- S1 (in tachycardia, feel pulse → tapping → ejection phase, therefore S1)
- Which valves normally open/which valves normally closed
  - Systole, aortic and pulmonic, open (mitral and tricuspid, closed)
  - If systolic murmur, either open valves stenotic/closed valves regurgitant (1/4 choice)
  - Diastole, mitral and tricuspid, open (aortic and pulmonic, closed) (1/4 choice)
- To choose between four resultant options auscultate over respective areas, employ maneuvers as required

MISCELLANEOUS HEART SOUNDS

- Mechanical valve clicks
  - Distinctly audible, harsh, metallic sound
- Pericardial knock
  - Sound occasionally heard in constrictive pericarditis; similar in acoustics, timing to S3
- Tumor plop
  - Rare low-pitched early diastolic sound, occasionally heard in atrial myxoma presence
  - Occurs when relatively mobile tumour moves in front of mitral valve during diastole → functional mitral stenosis along with low pitched diastolic rumbling murmur