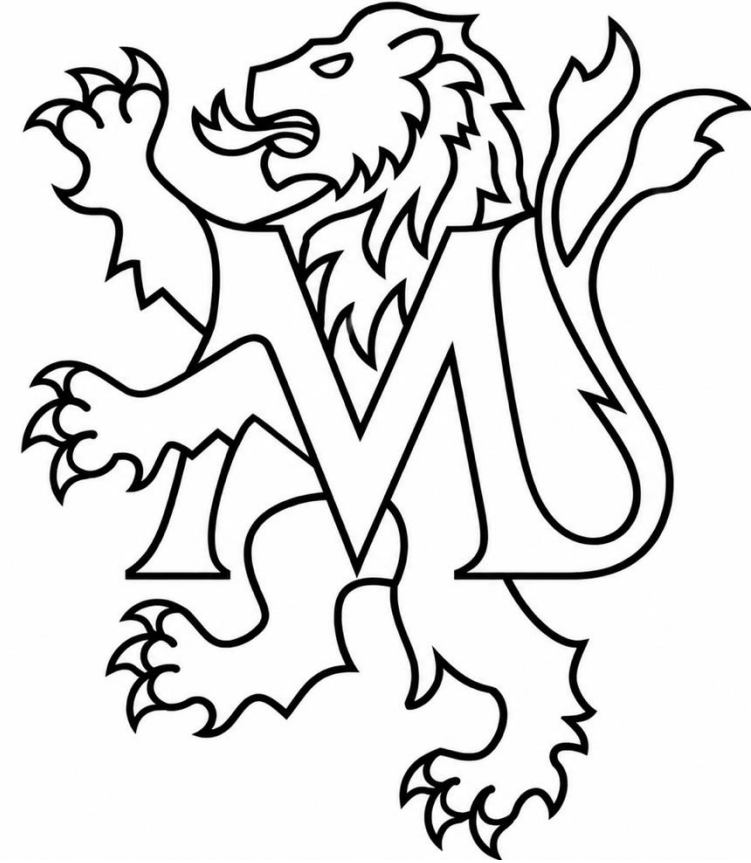


Cardiovascular System:

The heart is a pump

It creates pressure that moves blood through the body.

M-CATS Club Meeting



Two questions about the pump

What does it do, and what makes it work?

What does the pump do?

- Moves blood through adult and fetal routes
- Delivers oxygen and other materials
- Supports pressure, flow, and fluid exchange

What makes the pump work?

- Valves and a coordinated electrical rhythm
- Filling and myocardial contraction
- Autonomic control and the RAAS response

Need to know before the session

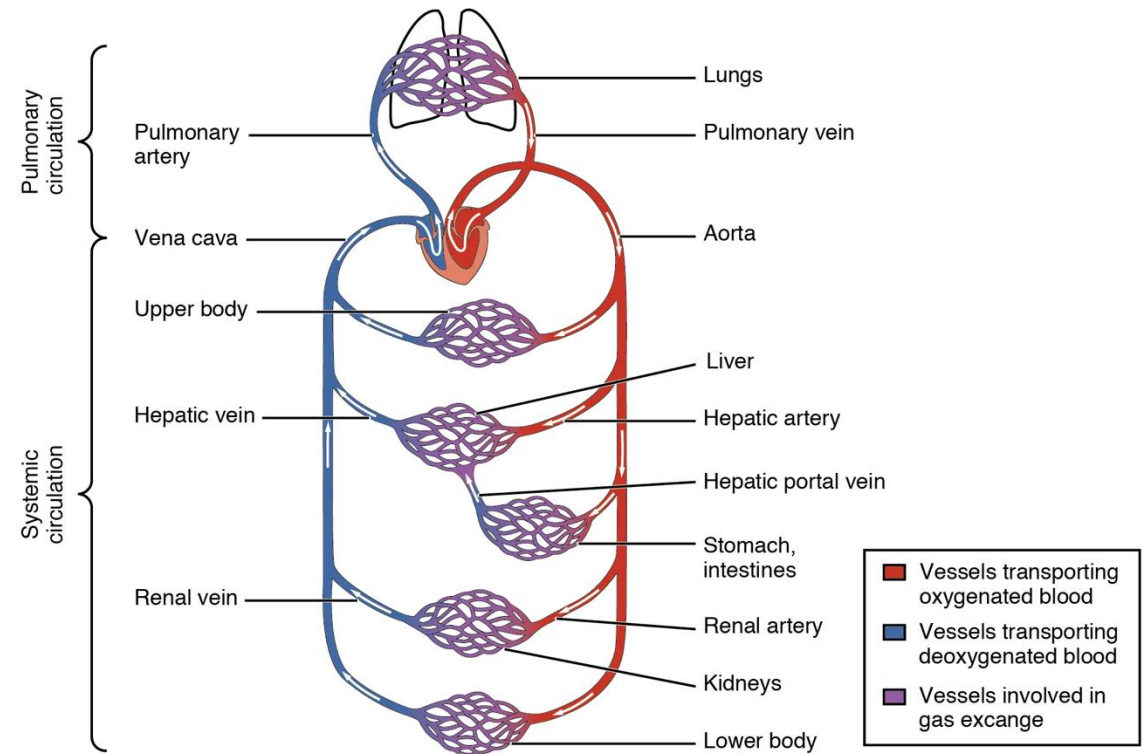
The heart creates a pressure gradient.

- Pressure differences drive blood flow.
- The right heart supplies the lungs. The left heart supplies the body.
- Cardiac output = heart rate $\times$ stroke volume.
- Electrical activation precedes contraction.
- Filling, autonomic input, and circulating hormones adjust the system.

What does the pump do?

It moves blood through the pulmonary and systemic circuits.

- Right ventricle pumps through the lungs.
- Left ventricle pumps through the body.
- Both sides have the same average output in steady state.



The path through the heart

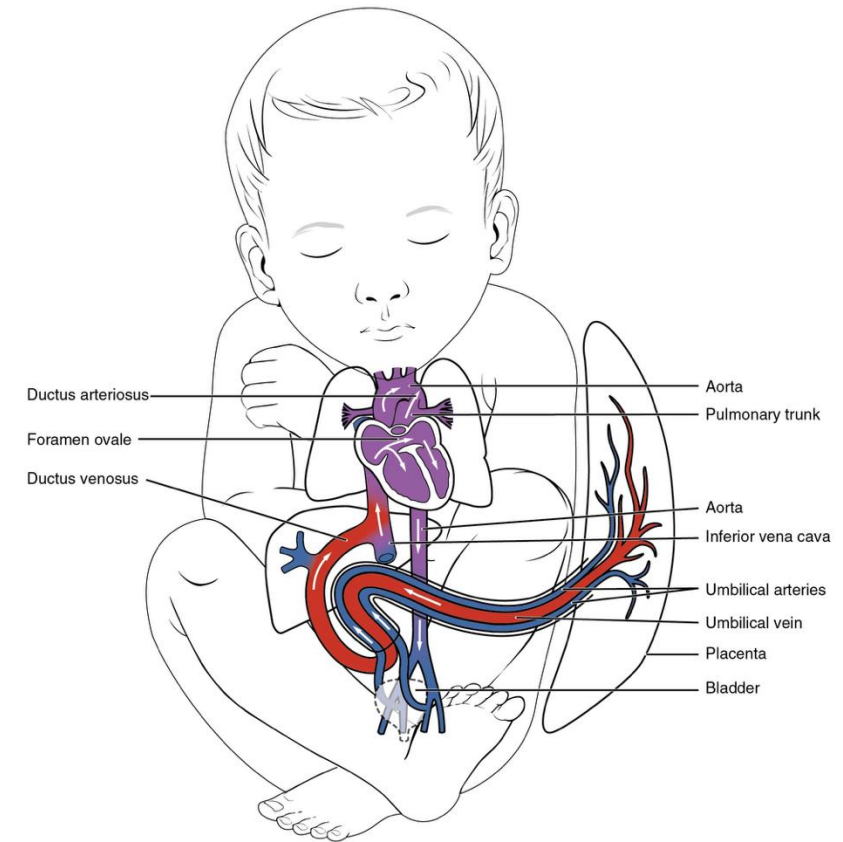
Every valve sits between two parts of the flow path.

- Vena cava → right atrium → tricuspid → right ventricle
- Pulmonary valve → pulmonary arteries → lungs
- Pulmonary veins → left atrium → mitral → left ventricle
- Aortic valve → aorta → tissues → systemic veins

Fetal circulation

The placenta provides gas exchange before birth.

- One umbilical vein brings relatively oxygen-rich blood to the fetus.
- Two umbilical arteries return blood to the placenta.
- High pulmonary resistance limits flow through fetal lungs.



The three fetal shunts

Shunts divert part of the flow around the liver or lungs.

Shunt	Connection	Main bypass
Ductus venosus	Umbilical vein to IVC	Part of liver flow
Foramen ovale	Right atrium to left atrium	Pulmonary circuit
Ductus arteriosus	Pulmonary trunk to aorta	Pulmonary circuit

The transition at birth

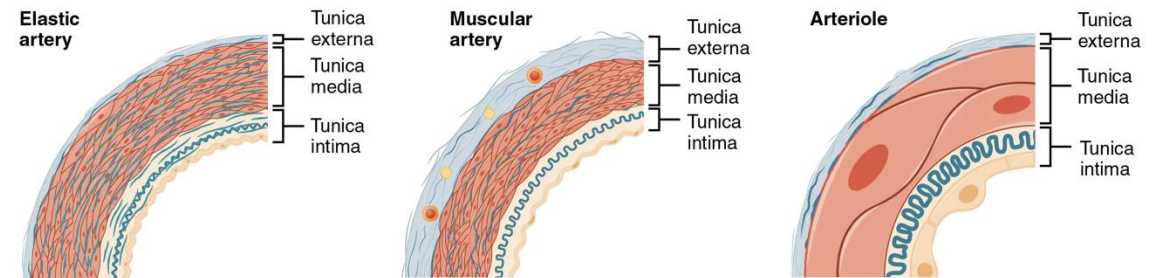
Breathing and loss of placental flow change pressure gradients.

- Lung expansion lowers pulmonary vascular resistance.
- More pulmonary return raises left atrial pressure relative to right atrial pressure.
- The foramen ovale flap closes functionally.
- Higher O_2 promote ductus arteriosus constriction.
- Cord clamping removes placental flow and makes the ductus venosus unnecessary.

Arteries, capillaries, and veins

Vessel structure fits its function.

- Arteries tolerate pressure and provide elastic recoil.
- Arterioles regulate resistance and local flow.
- Thin capillaries allow exchange.
- Compliant veins hold much of the blood volume.



Pressure, resistance, and flow

Flow depends on a pressure difference.

- $Q = \Delta P / R$
- ΔP is the upstream pressure minus downstream pressure.
- At fixed R , a greater ΔP increases flow.
- At fixed ΔP , greater R decreases flow.

Poiseuille's relationship

Resistance varies inversely with radius to the fourth power.

- $R = 8\eta L / (\pi r^4)$
- Longer length or greater viscosity raises resistance.
- Doubling radius lowers resistance to 1/16.
- At fixed ΔP , doubling radius increases flow 16-fold.

Cross-sectional area and speed

Capillaries have a large total area and slow average blood speed.

- $Q = vA$
- A is the total cross-sectional area of the vessel group.
- At the same total flow, greater total area means lower speed.
- Slow transit supports exchange with tissues.

Blood pressure

MAP reflects average driving pressure over a cardiac cycle.

- Pulse pressure = systolic – diastolic pressure
- $MAP \approx \text{diastolic} + \frac{1}{3}(\text{pulse pressure})$
- For 120/80 mmHg: pulse pressure = 40, $MAP \approx 93$ mmHg
- $MAP - \text{right atrial pressure} \approx CO \times SVR$

Hydrostatic and oncotic pressure

The direction depends on which side of the capillary wall creates the force.

Force	What creates it?	Favors
Capillary hydrostatic P_c	Pressure inside capillary	Fluid out
Interstitial hydrostatic P_i	Pressure outside capillary	Fluid in
Capillary oncotic π_c	Plasma proteins	Fluid in
Interstitial oncotic π_i	Interstitial proteins	Fluid out

Net filtration

The balance of forces determines the tendency for fluid movement.

- $J_v = K_f [(P_c - P_i) - \sigma(\pi_c - \pi_i)]$
- Positive J_v means net filtration out of the capillary.
- K_f reflects permeability and exchange area.
- σ reflects how effectively the wall retains proteins.
- Lymphatic drainage returns excess filtered fluid and proteins.

Why edema develops

Edema occurs when fluid entry into tissue exceeds removal.

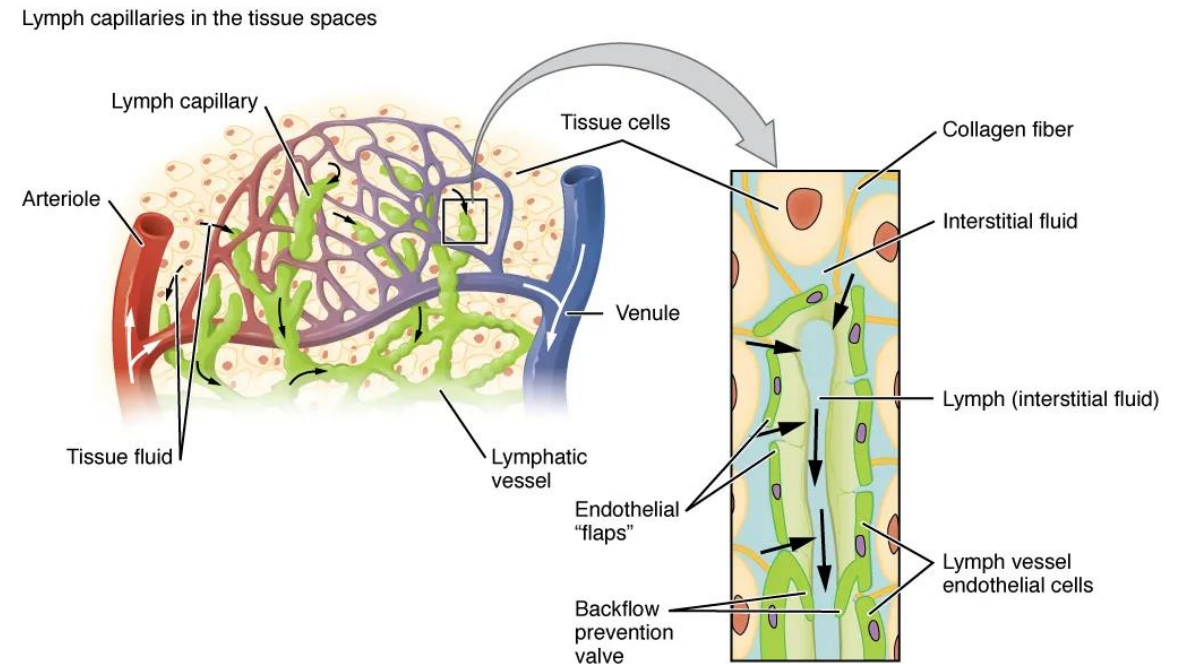
- Greater capillary hydrostatic pressure increases outward filtration.
- Lower plasma albumin weakens the inward oncotic force.
- Greater permeability can allow more fluid and protein to escape.
- Impaired lymph drainage reduces fluid removal.



Lymphatic return

Lymphatic vessels reconnect tissue fluid with the circulation.

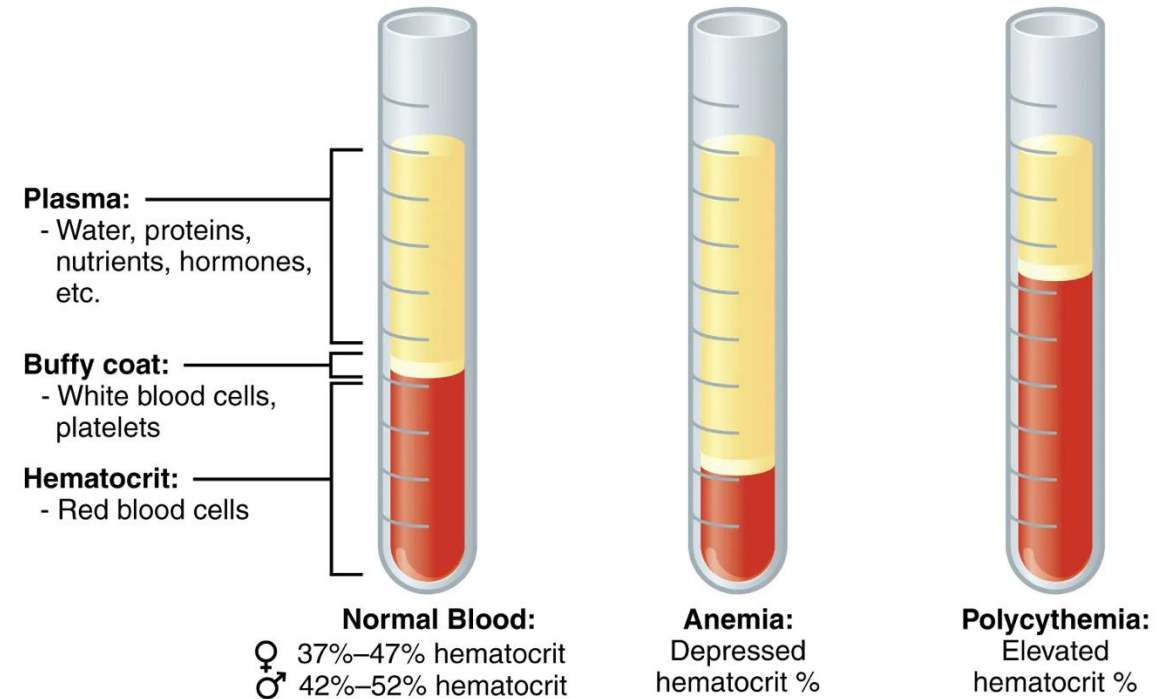
- Collect excess interstitial fluid and proteins.
- Return lymph to veins near the subclavian–jugular junctions.
- Lymph nodes support immune surveillance.



Blood composition

Plasma carries dissolved substances. Cells perform specialized work.

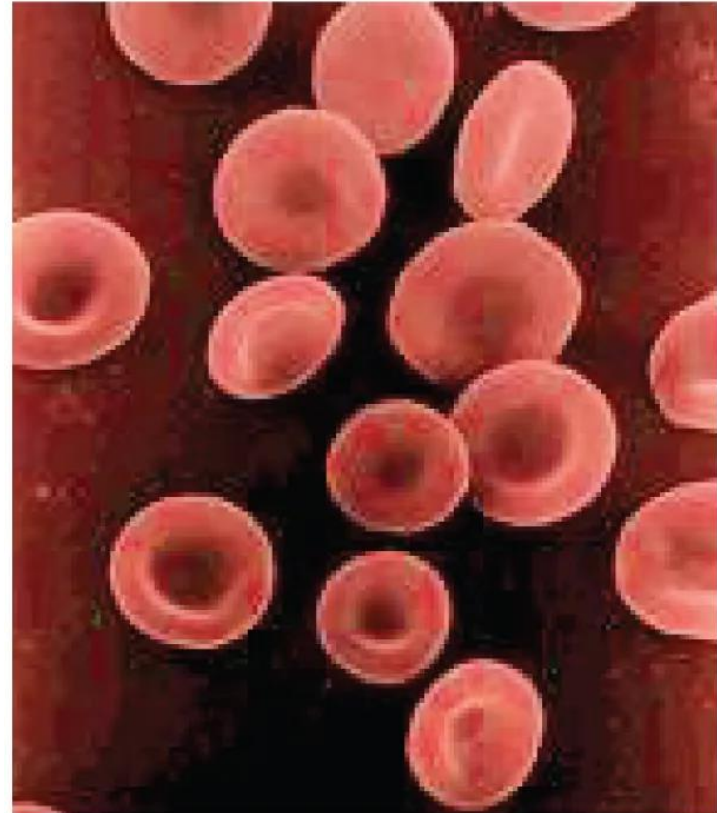
- Plasma: water, proteins, electrolytes, and dissolved solutes
- Erythrocytes: gas transport
- Leukocytes: immune defense
- Platelets: fragments involved in hemostasis
- Hematocrit: the fraction of blood volume occupied by RBCs



Red blood cells

Hemoglobin gives blood a large oxygen-carrying capacity.

- Mature human RBCs lack nuclei and mitochondria.
- Their biconcave shape supports diffusion and flexibility.
- They obtain ATP through glycolysis.
- Renal erythropoietin promotes RBC production in marrow.



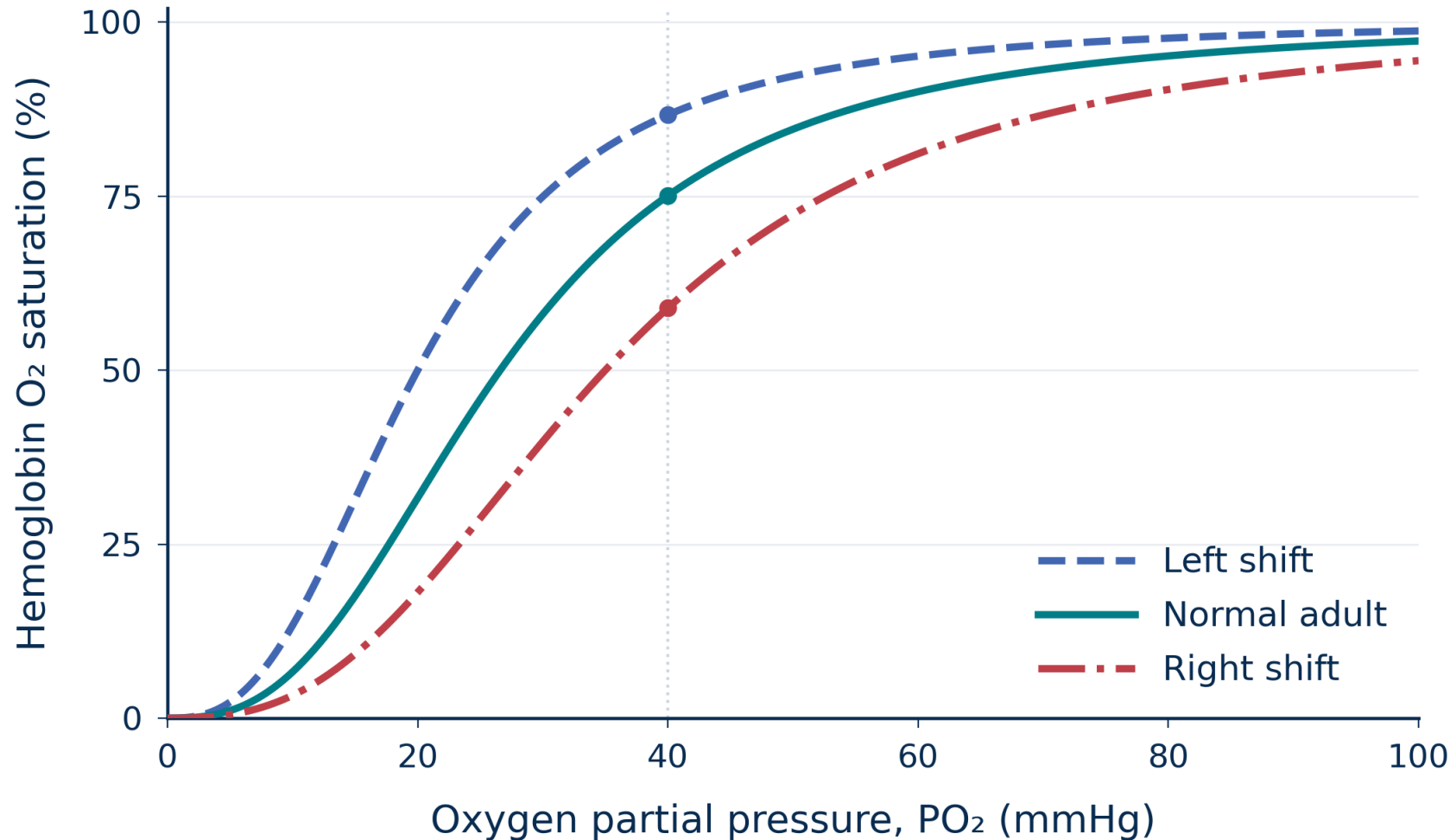
Oxygen transport

Hemoglobin saturation and oxygen content describe different things.

- Most blood oxygen binds to hemoglobin.
- Each hemoglobin can bind four O_2 molecules.
- Cooperative binding produces a sigmoidal saturation curve.
- Less hemoglobin lowers content even at the same saturation.

Hemoglobin oxygen affinity

At the same PO_2 , a left shift means more O_2 stays bound.



LEFT SHIFT

Higher affinity · lower P_{50}

Less unloading at a given PO_2

- ↓ CO_2 and ↓ H^+ (↑ pH)
- ↓ Temperature
- ↓ 2,3-BPG

RIGHT SHIFT

Lower affinity · higher P_{50}

More unloading at a given PO_2

- ↑ CO_2 and ↑ H^+ (↓ pH)
- ↑ Temperature
- ↑ 2,3-BPG

P_{50} = PO_2 at 50% saturation. A higher P_{50} means lower O_2 affinity.

ABO and Rh blood groups

RBC antigens determine which antibodies can react with them.

- A cells carry A antigen. B cells carry B antigen.
- Type O cells carry neither A nor B antigen.
- Type AB cells carry both antigens.
- Rh status usually refers to the D antigen.

Hemostasis

Platelets form a plug. Fibrin reinforces it.

- Vascular spasm reduces local blood loss.
- Platelets adhere, activate, and recruit additional platelets.
- The coagulation cascade generates thrombin.
- Thrombin converts fibrinogen to fibrin.
- Plasmin participates in later clot breakdown.

What makes the pump work?

A coordinated rhythm drives contraction against an opposing load.

Valves keep pressure-driven flow moving forward.

Electrical activation coordinates the contraction.

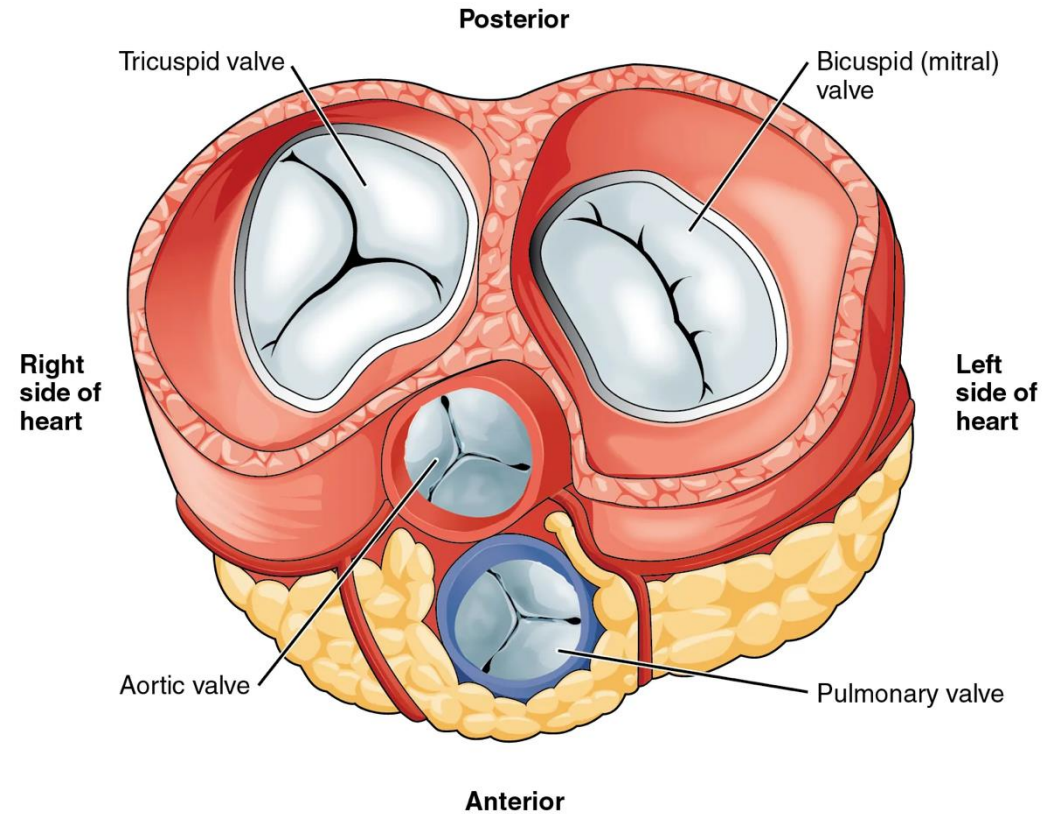
Filling and contractile force determine how much blood leaves.

Neural and hormonal responses adjust the system.

Heart valves

Pressure differences open and close valves.

- AV valves: tricuspid and mitral
- Semilunar valves: pulmonary and aortic
- Forward pressure opens a valve. Reverse pressure closes it.
- Chordae and papillary muscles prevent AV valve prolapse.



Ventricles and coronary blood flow

The left ventricle generates greater pressure.

- The systemic circuit has greater resistance than the pulmonary circuit.
- The left ventricular wall is thicker.
- Coronary vessels supply the myocardium.
- Most left ventricular coronary perfusion occurs during diastole.

The cardiac cycle

Valves change state as ventricular pressure changes.

- Filling: AV valves open, semilunar valves closed
- Isovolumetric contraction: all valves closed, pressure rises
- Ejection: semilunar valves open, ventricular volume falls
- Isovolumetric relaxation: all valves closed, pressure falls

Heart sounds

S1 and S2 mark valve closure.

- S1: AV valves close at the start of ventricular systole.
- S2: semilunar valves close at the end of ejection.
- Most ventricular filling occurs passively.
- Atrial contraction adds the final portion of filling.

Cardiac conduction and pumping

Electrical activation comes first. Contraction follows.

1. At rest

The conduction system is ready.

2. SA node fires

The signal spreads across both atria.

3. AV node delays transmission

Atria finish assisting ventricular filling.

4. His bundle and bundle branches

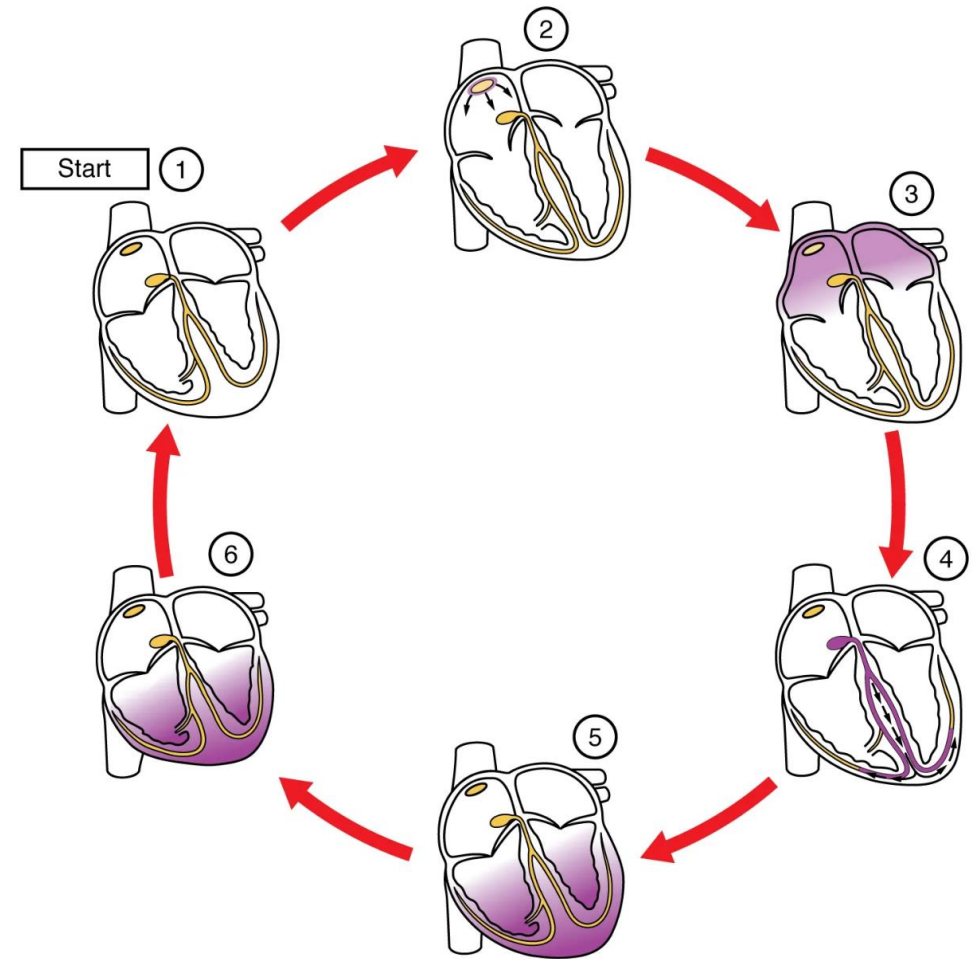
The signal travels down the septum toward the apex.

5. Purkinje fibers distribute the signal

Ventricular muscle depolarizes.

6. Ventricles contract

Contraction progresses from the apex toward the base, ejecting blood.



SA node → atria → AV node → bundle of His → bundle branches → Purkinje fibers

Cardiac muscle

A long refractory period permits relaxation between beats.

- Gap junctions spread electrical activation between cells.
- Calcium entry helps trigger additional calcium release.
- The plateau prolongs the action potential and prevents tetanus.

Cardiac output and ejection fraction

Stroke volume is the amount ejected per beat.

- $SV = EDV - ESV$
- $CO = HR \times SV$
- $EF = SV / EDV \times 100\%$
- Example: EDV 120 mL, ESV 50 mL, HR 70/min
- $SV = 70 \text{ mL/beat}$, $CO = 4.9 \text{ L/min}$, $EF \approx 58\%$

Preload, contractility, and afterload

Stroke volume depends on filling, force, and opposing pressure.

- Greater preload usually increases SV within the normal range.
- Greater contractility lowers ESV and increases SV.
- Greater afterload tends to increase ESV and decrease SV acutely.

The Frank–Starling mechanism

Within the normal range, greater filling produces a larger stroke volume.

- Greater venous return increases EDV and myocardial stretch.
- Length-dependent activation increases force at a given contractile state.
- Greater myofilament Ca^{2+} sensitivity contributes to this response.
- The ventricle ejects more of the blood returning to it.
- This intrinsic response can occur without increased sympathetic input.

Autonomic influence on the heart

Sympathetic and vagal signals adjust the intrinsic rhythm.

Input	Main receptor	Main effects
Sympathetic	β_1	Higher HR, faster AV conduction, stronger contraction
Parasympathetic (vagus)	M ₂	Lower HR and slower AV conduction

Autonomic influence on vessels

Sympathetic control affects resistance and venous return.

- α_1 activation constricts many arterioles and raises resistance.
- Venokonstriction reduces venous storage and supports venous return.
- Circulating epinephrine can activate β_2 receptors and dilate some vascular beds.
- Local metabolic signals can increase flow to active tissue.

The baroreflex

The baroreflex buffers rapid blood pressure changes.

- Higher arterial pressure stretches carotid and aortic baroreceptors.
- More firing reduces sympathetic and increases vagal influence.
- Heart rate falls and vascular tone decreases.
- Lower pressure produces the opposite response.

RAAS activation

The kidney releases renin when perfusion or distal NaCl delivery falls.

- RAAS = renin–angiotensin–aldosterone system
- Reduced renal perfusion pressure stimulates juxtaglomerular cells.
- Low NaCl delivery at the macula densa promotes renin release.
- Renal sympathetic β_1 activation also stimulates renin.
- Renin converts liver-derived angiotensinogen to angiotensin I.
- ACE converts angiotensin I to angiotensin II.

The RAAS response

Angiotensin II supports pressure through vascular and volume effects.

- Angiotensin II causes vasoconstriction.
- It stimulates adrenal cortical aldosterone release.
- Aldosterone increases distal Na^+ reabsorption and K^+ secretion.
- ADH and thirst support water retention and intake.
- Restored perfusion and angiotensin II feedback reduce renin release.

The response to blood loss

Rapid neural compensation and slower volume responses work together.

- Blood loss reduces venous return, preload, SV, and often MAP.
- The baroreflex raises sympathetic drive and lowers vagal drive.
- The kidneys activate RAAS when renal perfusion falls.
- Vasoconstriction supports pressure while salt and water retention support volume.
- Compensation may be incomplete if the loss is severe.

Interactive activity: what changes?

Each prediction needs a mechanism.

- 1. An arteriole narrows while ΔP stays fixed.
- 2. Venous return rises within the normal range.
- 3. Plasma albumin falls while other forces stay fixed.
- 4. Arterial pressure suddenly falls.

Pacing passage: normal function

AV timing supports filling before coordinated ventricular contraction.

- SA node initiates atrial activation. AV node delays transmission.
- His–Purkinje fibers rapidly activate the ventricles.
- P wave: atrial depolarization. QRS: ventricular depolarization. T wave: ventricular repolarization.

Adult measurement	Typical reference
PR interval	120–200 ms
QRS duration	Usually less than 120 ms
Resting CO / LV ejection fraction	Approximately 4–8 L/min / 55–70%

Pacing passage: the device setting

The ventricle activates before atrial contraction finishes.

- A patient has heart failure with reduced ejection fraction and bilateral leg edema.
- Initially: normal ventricular conduction, PR 160 ms, QRS 90 ms.
- An experimental setting stimulates the RV apex 40 ms after P-wave onset.
- The setting interrupts atrial assistance with filling and creates an abnormal activation sequence.
- The device maintains HR at 80 beats/min in both conditions.

Assume no medications block the hormonal responses discussed.

Pacing passage: measured changes

Compare the initial state with sustained premature ventricular pacing.

Measurement	Initial	Premature pacing
EDV	180 mL	170 mL
ESV	120 mL	130 mL
Mean arterial pressure	90 mmHg	70 mmHg
Local leg venous pressure	10 mmHg	25 mmHg

$$SV = EDV - ESV \quad CO = HR \times SV$$

$$EF = (SV / EDV) \times 100\%$$

Hypothetical teaching data

Pacing passage: capillary model

Use the supplied model and keep the other forces constant.

$$P_c = (P_a + 4P_v) / 5$$

- Use MAP as P_a . Use the measured local venous pressure as P_v .
- Plasma oncotic pressure $\pi_c = 25$ mmHg
- Interstitial oncotic pressure $\pi_i = 3$ mmHg
- Interstitial hydrostatic pressure $P_i = 2$ mmHg

Permeability and K_f stay constant. Protein reflection coefficient $\sigma = 1$.

$$NFP = (P_c - P_i) - (\pi_c - \pi_i)$$

Positive NFP favors outward fluid movement.

Question 1: pump performance

Which set correctly describes premature ventricular pacing?

- A. SV 40 mL/beat, CO 4.8 L/min, EF $\approx$ 24%
- B. SV 40 mL/beat, CO 3.2 L/min, EF $\approx$ 24%
- C. SV 60 mL/beat, CO 4.8 L/min, EF $\approx$ 35%
- D. SV 40 mL/beat, CO 3.2 L/min, EF $\approx$ 76%

Question 2: mechanism

Which explanation best connects the pacing setting to reduced pumping effectiveness?

- A. Premature activation increases filling, but a slower HR reduces CO.
- B. The lead prevents ventricular depolarization, allowing filling without contraction.
- C. Premature activation reduces atrial assistance with filling and disrupts coordinated ventricular contraction.
- D. Frank–Starling prevents SV from changing because HR stays constant.

Question 3: the ECG

Which ECG changes are most likely during this pacing setting?

- A. Longer P-to-QRS interval, wider QRS, longer R–R interval
- B. Shorter P-to-QRS interval, narrower QRS, unchanged R–R interval
- C. Unchanged P-to-QRS interval, wider QRS, shorter R–R interval
- D. Shorter P-to-QRS interval, wider QRS, unchanged R–R interval

Question 4: capillary pressure

What happens to capillary hydrostatic pressure in the supplied model?

- A. It rises from 26 to 34 mmHg because the venous rise outweighs the arterial fall.
- B. It falls from 26 to 14 mmHg because it follows arterial pressure alone.
- C. It rises from 18 to 19 mmHg because arterial and venous pressures contribute equally.
- D. It stays at 26 mmHg because HR and plasma oncotic pressure stay constant.

Question 5: filtration and edema

Which prediction follows from the supplied assumptions?

- A. NFP changes from 2 to -10 mmHg, favoring movement into capillaries.
- B. NFP stays at 2 mmHg because plasma protein concentration is unchanged.
- C. NFP rises from 2 to 10 mmHg, increasing filtration and potentially worsening edema.
- D. NFP rises from 2 to 10 mmHg, but edema also requires albumin to decrease.

Question 6: hormonal responses

If renal perfusion falls while cardiac chamber stretch remains elevated, which response is most consistent?

- A. RAAS and ADH decrease. ANP/BNP increase because total body fluid is elevated.
- B. RAAS and ADH release are favored. ANP/BNP release is also favored by chamber stretch.
- C. RAAS increases. ADH and ANP/BNP decrease because MAP falls.
- D. RAAS decreases. ADH increases and ANP/BNP decrease because HR stays fixed.

Pacing passage: answers 1–3

Output and activation change even though heart rate stays fixed.

- 1. B: SV 40 mL/beat, CO 3.2 L/min, EF $\approx$ 24%.
- Initial CO was 4.8 L/min and EF $\approx$ 33%.
- 2. C: reduced atrial assistance and abnormal ventricular coordination impair pumping.
- 3. D: shorter P-to-QRS timing, wider QRS, unchanged R–R.
- R–R = $60/80 = 0.75$ seconds.

Pacing passage: answers 4–6

Low arterial pressure can coexist with congestion and edema.

- 4. A: P_c rises from 26 to 34 mmHg as venous pressure rises.
- 5. C: NFP rises from 2 to 10 mmHg outward.
- Filtration increases even with unchanged albumin.
- 6. B: low renal perfusion favors RAAS and ADH.
- Chamber stretch also favors ANP/BNP release.
- Excess total fluid can coexist with inadequate effective arterial perfusion.

Why physicians learn this

Flow and oxygen content both matter for tissue oxygen delivery.

- Anemia can reduce oxygen delivery despite normal saturation.
- Reduced cardiac output can limit tissue perfusion.
- Venous congestion and low albumin can promote edema.
- Valve and conduction problems affect how the pump works.

Formula summary

The equation follows from the question you are asking.

- $SV = EDV - ESV$ $CO = HR \times SV$
- $EF = SV / EDV \times 100\%$
- $Q = \Delta P / R$ $R \propto \eta L / r^4$ $Q = vA$
- $\text{Pulse pressure} = SBP - DBP$
- $MAP \approx DBP + \frac{1}{3}(SBP - DBP)$
- $MAP - RAP \approx CO \times SVR$ $O_2 \text{ delivery} = CO \times CaO_2$

Closing review: explain why

A correct explanation connects the structure to the result.

- Fetal shunts divert much of the blood away from the lungs.
- Greater filling can increase SV without sympathetic stimulation.
- Lower albumin favors edema.
- A pressure drop increases sympathetic influence.
- Low renal perfusion can activate RAAS.
- O₂ delivery depends on both flow and oxygen content.