Ultra-High-Yield Summary — Cardiovascular Physiology (Exam Edition)

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By Topics: Arterial BP • Regulation • Special Circulation • Hemorrhage & Shock • Arterioles

1. Arterial Blood Pressure (ABP)

Definitions

• Arterial blood pressure (ABP): Lateral pressure by blood on arterial walls.

• Systolic blood pressure (SBP): Maximum pressure during ventricular systole (90–140 mmHg, average 120 mmHg).

• Diastolic blood pressure (DBP): Minimum pressure just before the next ventricular contraction (60–90 mmHg, average 80 mmHg).

• Pulse pressure (PP): PP = SBP − DBP = 120 − 80 = 40 mmHg.

• Mean arterial pressure (MAP): MAP = DBP + ■ PP = 80 + ■(40) = 93 mmHg.

Causes of systolic and diastolic pressure

• SBP depends mainly on stroke volume and aortic distensibility.

• During systole: about 1/3 of stroke volume leaves the arteries, and 2/3 distends the elastic arteries → raises SBP.

• DBP is maintained by elastic recoil of large arteries during diastole, which keeps blood flowing when the heart is relaxed.

Importance (functions) of arterial blood pressure

1) Maintains tissue perfusion (blood flow) to all organs, including those above heart level.

2) Produces capillary hydrostatic pressure → main force for tissue fluid formation.

3) Diastolic blood pressure:

• Maintains blood flow to tissues during ventricular diastole (continuous, not intermittent flow).

• Essential for normal coronary blood flow.

• Prevents blood stasis in arteries, reducing the energy required by the heart.

Pulse pressure

• PP = SBP − DBP.

• Increased PP in: hyperdynamic circulation (aortic regurgitation, arteriosclerosis, severe anemia, hyperthyroidism, pregnancy).

• Decreased PP in: hemorrhage.

Physiological variation of ABP

1) Age:

• Newborn: 80/40 mmHg

• 4 years: 100/65 mmHg

• 20 years: 120/80 mmHg

• 60 years: ~150/90 mmHg (due to decreased arterial elasticity)

2) Sex:

• Adult male > adult female.

• After menopause: female BP becomes higher (loss of estrogen protection).

3) Body build: BP usually higher in obese persons.

4) Race: BP in Orientals < Europeans/Americans (genetic, diet, stress, etc.).

5) Sleep: Quiet sleep → ↓ ABP (↑ parasympathetic activity).

6) Emotions: Anger, fear, stress → ↑ ABP (sympathetic overactivity).

7) Meals: After meals, ABP increases due to increased venous return and cardiac output.

8) Gravity: Standing → ↑ BP below heart, ↓ BP above heart by 0.77 mmHg/cm blood column.

9) Exercise:

• Isometric: SBP and DBP both rise sharply, return quickly to normal.

• Isotonic: SBP rises, DBP stays same or decreases (VD in skeletal muscle vessels).

10) Respiration (respiratory pressure waves):

• ABP slightly changes during different phases of inspiration and expiration due to changes in venous return and intrathoracic pressure.

2. Factors Determining and Maintaining ABP

Key equation

• ABP = Cardiac output (COP) × Total peripheral resistance (TPR)

• COP = Heart rate (HR) × Stroke volume (SV)

→ Therefore: ABP = HR × SV × TPR

1) Stroke volume (SV)

• If HR is constant:

• ↑ SV → ↑ SBP more than DBP → ↑ PP.

• SV is mainly determined by cardiac contractility and venous return.

2) Heart rate (HR)

• If SV is constant:

• Changes in HR mainly affect DBP.

• Fast HR → less time in diastole → ↑ DBP less, SBP almost constant → ↓ PP.

3) Total peripheral resistance (TPR)

• Located mainly in the arterioles.

• ↑ TPR → ↑ DBP more than SBP → ↓ PP.

• Determined by:

a) Arteriolar diameter:

• Vasoconstriction → ↑ TPR → ↑ ABP (especially DBP).

• Vasodilation → ↓ TPR → ↓ ABP.

b) Blood viscosity:

• ↑ Hematocrit (e.g. chronic hypoxia) → ↑ viscosity → ↑ TPR → ↑ DBP → ↓ PP.

4) Elasticity of large arteries

• Normal elasticity:

• Prevents excessive rise in SBP (arteries stretch).

• Elastic recoil maintains DBP during diastole.

• Atherosclerosis (↓ elasticity):

• SBP ↑ markedly.

• DBP ↓.

• Pulse pressure becomes wide (↑ PP).

5) Blood volume & circulatory capacity

• Changes in blood volume and venous capacity affect venous return and COP.

• Mainly influence SBP.

3. Regulation of Arterial Blood Pressure

Three time scales:

1) Rapidly acting mechanisms (neural, seconds to minutes)

2) Intermediately acting mechanisms (minutes to hours)

3) Slowly acting mechanisms (renal/hormonal, hours to days)

A. Rapidly acting mechanisms (Neural)

Medullary cardiovascular centers

1) Pressor area (Vasomotor center – VMC)

• Location: ventrolateral medulla.

• Sends sympathetic fibers to vessels and heart.

Components:

a) Vasoconstrictor center (VCC):

• Continuous discharge at rest → sympathetic vasoconstrictor tone.

• Stimulation → arteriolar vasoconstriction → ↑ TPR → ↑ ABP.

• Venoconstriction → ↑ venous return → ↑ COP → ↑ ABP.

b) Cardiac stimulatory center (CSC):

• Low tonic discharge at rest.

• Stimulation:

– ↑ HR

– ↑ force of contraction → ↑ SV

– → ↑ COP → ↑ ABP

2) Depressor area

• Location: central & dorsal to pressor area.

Components:

a) Vasodilator center (VDC):

• Inhibits VCC → vasodilation → ↓ TPR → ↓ ABP.

b) Cardiac inhibitory center (CIC):

• Vagal fibers to SA node (vagal tone).

• ↓ HR → ↓ COP → ↓ ABP.

• There is reciprocal innervation between pressor and depressor areas.

• Dominant centers at rest: VCC & CIC.

B. Afferent influences on cardiovascular centers

I. Baroreceptors (pressure receptors, mechanoreceptors)

1) Arterial baroreceptors (high-pressure receptors)

• Location: Carotid sinus & aortic arch.

• Nerve: Buffer nerves (glossopharyngeal & vagus).

• Stimulus: Stretch due to ABP changes.

• Discharge range: 60–180 mmHg.

– Below 60 mmHg → no discharge.

– At 180 mmHg → maximal discharge.

Functions:

1. Continuous inhibitory impulses at rest.

2. Buffering action: oppose sudden changes in ABP.

• If ABP ↑:

– ↑ firing → inhibition of pressor area & stimulation of depressor area:

a) Arteriolar vasodilation → ↓ TPR → ABP returns toward normal.

b) Venodilation → ↓ VR → ↓ COP → ↓ ABP.

c) Reflex bradycardia → ↓ HR → ↓ COP → ↓ ABP.

Marey's reflex (Marey's law)

• "A rise in ABP leads to a fall in heart rate, and vice versa, if other factors are constant."

• Initiated by arterial baroreceptors.

Clinical points:

• Carotid sinus syndrome: hypersensitive baroreceptors → slight pressure on neck → marked ↓ ABP & syncope.

• Essential hypertension: resetting of baroreceptors to a higher BP level.

2) Atrial baroreceptors (low-pressure / volume receptors)

• Location: walls of both atria near venous openings.

• Nerve: Vagus.

• Types:

– Type A: discharge during atrial systole.

– Type B: discharge late in diastole (atrial filling).

• Stimulus: ↑ central venous pressure (CVP) → also called volume receptors.

Functions:

a) ↑ VR → ↑ CVP → ↑ atrial receptor discharge →

– Inhibition of VCC →

• Arteriolar vasodilation → ↓ TPR → ↓ ABP.

• Venodilation → ↓ VR, ↓ CVP, ↓ COP → ↓ ABP.

b) Inhibition of ADH & aldosterone + ↑ ANP →

– ↑ Na+ and water excretion → ↓ blood volume → ↓ CVP → ↓ VR → ↓ COP → ↓ ABP.

Bainbridge reflex

• ↑ VR → atrial stretch → reflex tachycardia via vagus to prevent pooling of blood in atria.

II. Chemoreceptors

1) Peripheral chemoreceptors

• Location: Carotid bodies & aortic bodies.

• Nerve: Buffer nerves.

• Stimuli:

– Hypoxia (main)

– Hypercapnia (↑ CO2)

– Acidosis (↑ H+)

• Functions:

– Stimulate pressor area → mild ↑ ABP.

– Mainly: stimulate respiratory center → ↑ ventilation.

2) Pulmonary chemo-reflex

• Distension of pulmonary vessels (embolism, congestion) → reflex hypotension, bradycardia, apnea.

3) Coronary chemo-reflex (Bezold–Jarisch reflex)

• Location: coronary vessels.

• Afferent: Vagus.

• Stimuli: chemicals released during MI, serotonin injections, etc.

• Response: ↓ ABP, ↓ HR, apnea.

C. Intermediate mechanisms

1) Capillary fluid shift

• ↑ ABP → ↑ capillary hydrostatic pressure → more filtration → ↓ blood volume → ↓ CVP → ↓ VR → ↓ COP → ABP returns toward normal.

• ↓ ABP → fluid shifts from interstitial space to plasma → ↑ blood volume.

2) Stress relaxation and reverse stress relaxation

• Vessels adjust their diameter over minutes to hours.

• After blood transfusion: initial ↑ BP, then vessels relax and BP returns near normal.

• After blood loss: vessels constrict (reverse stress relaxation) to maintain BP (limited ~15% blood loss).

3) Thirst sensation

• Decreased blood volume / hypovolemia and Ang II stimulate thirst center in hypothalamus → ↑ water intake → ↑ blood volume → help restore ABP.

D. Slowly acting mechanisms (Renal)

1) Pressure diuresis

• ↑ ABP → ↑ renal filtration → ↑ urine output → ↓ blood volume → ↓ ABP.

• ↓ ABP (shock, hemorrhage) → ↓ urine formation → conservation of volume.

2) Renin–angiotensin–aldosterone system (RAAS)

• ↓ ABP or ↓ renal blood flow → renal ischemia → juxtaglomerular cells release renin.

• Renin converts angiotensinogen → Ang I → Ang II (in lungs via ACE).

Effects of Angiotensin II:

1. Strong arteriolar vasoconstriction (50× norepinephrine) → ↑ TPR → ↑ ABP.
2. Stimulates aldosterone secretion → Na+ and water retention → ↑ blood volume, ↑ COP, ↑ ABP.
3. Stimulates ADH secretion → water retention, vasoconstriction → ↑ ABP.
4. Enhances norepinephrine release from sympathetic terminals.
5. Stimulates thirst → ↑ water intake → ↑ blood volume → ↑ ABP.
6. Promotes Na+ and water reabsorption in kidney.

4. Arterioles

Definition and characteristics

• Terminal branches of arterial system.

• Low elastic elements, thick smooth muscle layer.

• High resistance to blood flow.

• Sympathetic innervation (vasoconstrictor tone).

• Sensitive to blood-borne chemicals.

• Endothelium produces vasoactive substances.

• Only site where arterioles can be directly seen: retina.

Functions of arterioles

1. Determine total peripheral resistance → determine DBP.
2. Control blood flow to tissues by changing diameter (VC/VD).

Regulation of arteriolar diameter

I) Local regulation

1) O2 tension

• ↓ PO2 → direct vasodilation of arteriolar smooth muscle in most tissues (except pulmonary vessels, which constrict with hypoxia).

• ↑ PO2 (low metabolic activity) → vasoconstriction.

• In heart: ↓ PO2 = most potent vasodilator.

• In CNS: ↑ PCO2 = most potent vasodilator.

2) Metabolites

• Increased metabolism → ↓ PO2, ↑ PCO2, ↑ H+, ↑ metabolites (adenosine, K+, etc.) → vasodilation.

Hyperemia

a) Active hyperemia: ↑ blood flow due to ↑ metabolic activity (e.g. exercising muscle).

b) Reactive hyperemia: transient ↑ blood flow after release of occlusion due to accumulated metabolites.

3) Autoregulation (intrinsic mechanism)

• Ability of tissues to keep blood flow relatively constant despite changes in perfusion pressure.

• Well developed in kidneys, liver, myocardium, brain.

Mechanisms:

a) Myogenic theory:

• ↑ pressure → stretch vascular smooth muscle → reflex contraction → ↑ resistance → normalizes flow.

b) Metabolic theory:

• ↓ perfusion → accumulation of metabolites → vasodilation → ↑ blood flow → washout of metabolites.

II) Systemic (central) regulation – Neuronal

A. Sympathetic vasoconstrictor fibers

• Originate from lateral horn (T1–L2) → generalized partial vasoconstriction (sympathetic tone).

• ↑ sympathetic activity → vasoconstriction → ↑ TPR → ↑ ABP.

• ↓ sympathetic activity → vasodilation → ↓ TPR → ↓ ABP.

B. Sympathetic vasodilator fibers

1) Coronary vessels: indirect VD via increased metabolic activity of heart (metabolic mechanism).

2) Skeletal muscle vessels: sympathetic vasodilator fibers (cholinergic) activated from cortex–hypothalamus pathway, especially before and during exercise.

C. Splanchnic area

• Rich in β-adrenergic receptors → vasodilation under certain conditions.

D. Sweat glands

• Sympathetic cholinergic vasodilator fibers to skin vessels; controlled by heat loss center in anterior hypothalamus.

E. Parasympathetic vasodilator fibers

• Direct vasodilator fibers to genital organs (sacral outflow).

• Parasympathetic fibers to salivary glands → indirect VD via ↑ metabolic activity and secretion.

Antidromic vasodilator impulses

• Pain receptor stimulation → impulses travel along sensory fibers → at branches can travel antidromically → release of substance P at arterioles → local vasodilation and flare in inflammation.

III) Hormonal regulation

1) Antidiuretic hormone (ADH, vasopressin)

• Secreted from posterior pituitary.

• Acts on V1 receptors (vascular smooth muscle) → ↑ Ca2+ → vasoconstriction → ↑ ABP.

• Acts on V2 receptors (kidney) → ↑ cAMP → ↑ water reabsorption → ↑ ECF volume → ↑ ABP.

• Important in acute hemorrhage.

2) Angiotensin II

• See RAAS section above – powerful vasoconstrictor and volume regulator.

3) Catecholamines

• Noradrenaline: potent vasoconstrictor (α-receptors).

• Adrenaline: in small doses → vasodilation in skeletal muscle and liver (β2-receptors).

4) Circulating vasodilator substances

a) Kinins (e.g. bradykinin):

• Vasodilation (via NO/EDRF release) → ↓ ABP.

• Increase capillary permeability.

• Contraction of some smooth muscles (e.g. bronchi).

• Chemotaxis for WBCs.

• Stimulate pain receptors.

b) Atrial natriuretic peptide (ANP):

• Secreted mainly from atria when atrial pressure, Na+, blood volume, or venous return is increased.

• Opposite actions of Ang II:

1. Vasodilation → ↓ BP.

2. Natriuresis → ↑ Na+ excretion.

3. ↓ Aldosterone secretion.

4. ↓ ADH secretion.

5. ↓ Renin release → ↓ Ang II formation.

IV) Endothelial regulation

1) Nitric oxide (NO, Endothelium-derived relaxing factor – EDRF)

• Produced from arginine by NO synthase in endothelial cells.

• Increases cGMP in smooth muscle → vasodilation.

• Released in response to bradykinin, substance P, VIP, etc.

• Deficiency (endothelial damage) → loss of vasodilator effect → tendency to vasoconstriction.

2) Prostacyclin vs Thromboxane A2

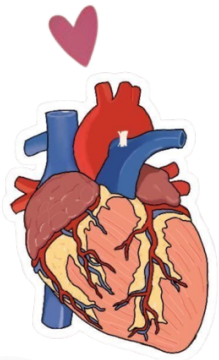
• Both from arachidonic acid via cyclooxygenase.

• Prostacyclin (PGI2): from endothelium → vasodilation + inhibits platelet aggregation.

• Thromboxane A2: from platelets → vasoconstriction + promotes platelet aggregation.

• Aspirin irreversibly inhibits cyclooxygenase in platelets → ↓ TXA2 → vasodilation + ↓ aggregation → used to prevent thrombosis.

3) Endothelium-derived hyperpolarizing factor (EDHF)



• Opens K+ channels in VSMC membranes → hyperpolarization.

• Hyperpolarization → ↓ Ca2+ entry → ↓ intracellular Ca2+ → vasodilation.

4) Endothelins (ETs)

• 3 isoforms, 21 amino acid peptides.

• Synthesized by endothelial cells in response to Ang II, trauma, hypoxia.

• Receptors:

– ETA: vasoconstriction

– ETB: vasodilation

• ET-1: potent vasoconstrictor acting mainly via ETA receptors → ↑ intracellular Ca2+ → vasoconstriction.

5. Special Circulation

A. Coronary circulation

• Coronary arteries: left and right; considered functionally end arteries (limited anastomosis).

• Capillaries: approximately one capillary per muscle fiber; in hypertrophy capillary number does not increase significantly → limiting factor → heart failure.

• Venous drainage:

– Superficial system: drains ~60% from left ventricle directly to right atrium.

– Deep system: drains rest of heart directly into chambers.

Coronary blood flow

• At rest: ~80 ml/100 g/min, total ~250 ml/min (≈5% of COP).

• During exercise: ↑ 3–5 times; in athletes up to 600 ml/100 g/min.

• Oxygen extraction at rest: ~75% (arterial 19 ml O2/dl → venous 5 ml O2/dl).

→ Therefore, increased O2 demand during exercise must be met mainly by increased coronary blood flow (not extraction).

Regulation of coronary blood flow

1) Chemical (metabolic) – main mechanism

• Increased cardiac work → hypoxia, ↑ CO2, H+, K+, lactate, adenosine.

• Hypoxia is the most effective vasodilator directly and via adenosine release.

2) Mechanical

• Coronary flow follows aortic pressure but is mainly during diastole:

– Systole (especially isometric contraction): compression of coronary vessels → ↓ flow.

– Early diastole (isometric relaxation): high aortic pressure + relaxed myocardium → maximal coronary flow.

• Tachycardia shortens diastole → ↓ coronary perfusion.

3) Neural

• Sympathetic:

– Indirect: ↑ HR & contractility → ↑ metabolism → ↑ vasodilator metabolites → coronary VD (dominant).

– Direct: α-mediated VC and β-mediated VD; α effect dominates but is overridden by metabolic VD.

• Parasympathetic (vagal):

– Indirect: ↓ HR & contractility → ↓ metabolism → ↓ flow (dominant).

– Direct: ACh can cause VD.

– Net effect: usually slight decrease in flow.

4) Hormonal and chemical

• VC: ADH, Ang II, endothelin, thromboxane A2.

• VD: thyroxine, adrenaline (β), NO, alcohol, ADP, caffeine, nitrites.

B. Cerebral circulation

• Arterial supply: internal carotid + vertebral arteries forming Circle of Willis.

• Cerebral arteries are considered end arteries (anastomoses insufficient during large artery occlusion).

• Capillaries: tight junctions + astrocyte end-feet → blood–brain barrier (BBB).

Blood–brain barrier

• Restricts entry of proteins, bile pigments, H+ ions, dopamine, many drugs.

• O2, CO2, lipid-soluble substances (anesthetics, alcohol) pass easily.

Cerebral blood flow (CBF)

• Normal: ~54 ml/100 g/min.

• Adult brain ~1400 g → total ~750 ml/min (≈15% of COP).

Regulation of CBF

1) Autoregulation (60–140 mmHg)

• Maintains nearly constant flow despite moderate changes in ABP.

• Metabolic theory: ↓ BP → ↓ CBF → hypoxia, ↑ CO_2, ↑ H^+ → vasodilation → restore CBF.

• Myogenic theory: stretch of vessels → reflex VC.

2) Circulating vasoactive substances

• VC: Ang II, endothelin, ADH, thromboxane A2.

• VD: NO (EDRF), ANP, ACh.

3) ABP and COP

• Severe fall in COP or ABP (shock, hemorrhage) → ↓ CBF → brain damage.

• CBF interruption for 10 s → loss of consciousness; >3 min → irreversible damage.

4) Effective perfusion pressure (EPP)

• EPP = arterial pressure – venous pressure at brain level.

• CBF ∝ EPP.

5) Venous obstruction

• Jugular vein thrombosis, Valsalva maneuver → ↑ venous pressure → ↓ EPP → ↓ CBF.

6) Blood viscosity

• ↑ viscosity → ↓ CBF.

7) Intracranial pressure (ICP)

• Brain, CSF, vessels inside rigid skull.

• ↑ ICP compresses vessels → ↓ CBF.

• ↑ cerebral venous pressure → ↑ ICP and ↓ EPP → ↓ CBF.

8) Nervous factors

• Sympathetic VC has limited effect because of strong autoregulation.

9) Brain activity

• Total CBF remains almost constant.

• Regional flow ↑ in active areas and ↓ in inactive areas.

10) Age

• Newborn/children: CBF ≈100 ml/100 g/min (double adult).

• After puberty: decreases to adult levels.

6. Venous Circulation

Functions of veins

1) Drain blood from tissues to heart.

2) Blood reservoir (capacitance vessels) ~65% of blood volume.

3) Venous pump (via muscle contraction, valves).

4) Regulation of cardiac output (via venous return).

Venous pressures

A. Central venous pressure (CVP)

• Pressure in right atrium and large veins near it.

• Value: 0–4 mmHg.

• Determined by balance between venous return and cardiac output.

• Functions:

– Main determinant of venous return.

– Fills the ventricle.

B. Peripheral venous pressure

• 4–7 mmHg higher than CVP due to resistance points along venous system.

Effects of gravity

A. Orthostatic (postural) hypotension

• On standing, 400–600 ml blood pools in lower limb veins → ↓ VR & COP → ↓ ABP → possible syncope.

• Compensated rapidly by arterial baroreflex.

B. Venous pressure gradient

• Standing:

– Veins below heart: ↑ pressure.

– Veins above heart: sub-atmospheric pressure.

• Superior sagittal sinus pressure ~ −10 mmHg (risk of air embolism if opened).

C. Hydrostatic indifferent point (HIP)

• Level where venous pressure is independent of position (~5–7 cm below diaphragm).

Shift of (HIP) down ↓	Shift of (HIP) up ↑
1. Hypovolemia.	1. Hypervolemia.
2. VD.	2. VC.
3. ⇧ Capacitance of lower limbs Veins as in varicose veins.	3. ⇩ Capacitance of lower limbs veins as in swimming Shift HIP to level of right atrium ⇨ constant CVP ⇨ constant VR & COP & ABP in spite of change position. So, no orthostatic hypotension during swimming.

Mechanisms aiding venous return

1) Venomotor tone (sympathetic VC of veins).

2) Muscle pump (skeletal muscle contraction + valves).

3) Thoracic pump (inspiration: ↓ intrathoracic pressure, ↑ intra-abdominal pressure → ↑ VR).

4) Cardiac suction (atrial and ventricular suction during phases of cardiac cycle).

7. Hemorrhage and Shock

I. Hemorrhage

Definition

• Loss of blood from cardiovascular system.

Types

• By site: external, internal, interstitial.

• By onset: acute vs chronic.

• By prognosis:

– <20% blood volume loss → usually compensated.

– >20% → may require rapid transfusion to avoid shock.

Compensatory mechanisms

I) Immediate (minutes)

A) Nervous control

• ↓ ABP & CVP → ↓ baroreceptor and atrial receptor discharge.

• Hypoxia of chemoreceptors.

→ Stimulate pressor area & inhibit depressor area →

1. ↑ Sympathetic to arterioles → VC → ↑ TPR → ↑ ABP (esp. skin & splanchnic).
2. ↑ Sympathetic to veins → venoconstriction → ↑ VR → ↑ COP → ↑ ABP.
3. ↑ Sympathetic to heart → tachycardia + ↑ contractility.
4. Spleen & liver contraction (small role in humans).
5. ↑ Coagulability (fibrinogen) & hyperglycemia.

B) Hormonal

1. Catecholamines: Adrenal medulla → ↑ adrenaline → tachycardia, VC, ↑ glucose, ↑ coagulation.
2. RAAS: ↓ COP → renal ischemia → renin → Ang II → VC + Na+/water retention.
3. ADH: ↓ VR → ↓ CVP → atrial input to hypothalamus → ↑ ADH → VC + water retention.
4. Cortisol: stress hormone → ↑ glucose, stabilizes membranes, prevents cell damage.

C) Thirst

• Hypovolemia + Ang II → stimulate thirst center → ↑ water intake.

D) Respiration

• Hypoxia + ↓ baroreceptor activity → hyperventilation → ↑ VR & COP and ↑ O2.

Clinical picture after hemorrhage

• Progressive ↓ ABP, rapid weak (thready) pulse, tachypnea, cold pale sweaty skin, intense thirst, irritability, coma in severe cases.

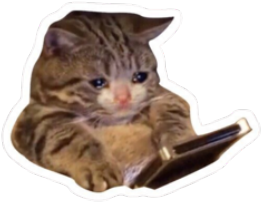
II) Delayed (long-term)

1) Plasma volume restoration (12–72 h)

• ↓ ABP → ↓ capillary hydrostatic pressure → fluid moves from interstitial space to capillaries.

• ↑ ADH → water retention.

• Thirst → water intake.



2) Plasma proteins restoration (3–4 days)

• Liver releases stored proteins and synthesizes new ones.

• Extravascular albumin mobilized into circulation.

3) Cellular elements restoration (4–8 weeks)

• Activation of bone marrow by erythropoietin, leucopoietin, thrombopoietin.

II. Shock

Definition

• Inadequate tissue perfusion due to ↓ COP and ↓ ABP.

Types

1) Low-resistance (distributive, normovolemic) shock

• Severe vasodilation with normal blood volume.

a) Neurogenic shock: emotional (vago-vagal) → VD and bradycardia.

b) Anaphylactic shock: antigen–antibody reaction → histamine, kinins → VD, ↑ capillary permeability.

c) Septic shock: bacterial endotoxins → VMC depression, VD, ↑ capillary permeability.

2) Hypovolemic shock (cold shock)

• Due to blood/plasma/ECF loss: hemorrhage, burns, trauma, dehydration.

3) Cardiogenic shock

• Due to pump failure: MI, severe HF, arrhythmias → ↓ COP.

4) Obstructive shock

• Obstruction of blood flow at central circulation: pulmonary embolism, tension pneumothorax, cardiac tamponade.

Prognosis

A) Reversible (compensated) shock

• Compensatory mechanisms can restore ABP if treated early.

B) Irreversible (refractory) shock

• Severe, prolonged shock (3–5 hours untreated) → progressive fall in COP & ABP in a vicious circle.

Mechanisms leading to death in refractory shock

1) Cardiac depression (ischemia, toxins).

2) Cerebral depression (↓ CBF → VMC failure).

3) Dilatation of precapillary sphincters with persistent venule constriction → pooling, filtration, ↓ VR.

4) Release of toxins (myocardial depressant factor, endotoxins, free radicals).

5) Thrombosis of small vessels.

6) Acidosis (lactic + CO2) → cell damage.

7) Acute respiratory failure (lung damage, ARDS).

8) Acute renal failure (ischemia, tubular necrosis, myoglobin).

Treatment of shock

1) Warm the patient, elevate legs to improve VR.

2) O2 therapy, glucose.

3) Maintain airway, prevent lung infection.

4) Low-resistance shock: antihistamines, sympathomimetics, glucocorticoids, analgesia.

5) Hypovolemic shock: appropriate fluid replacement (blood, plasma, saline).

6) Septic shock: antibiotics.

7) Correct electrolyte and acid-base imbalance; dialysis if renal failure.

8) Always treat the underlying cause.

وكان الإمام الشافعي – رحمه الله – يقول:

"أخي، لن تنال العلم إلا بستةٍ: ذكاءٍ، وحرصٍ، واجتهادٍ، وبلغةٍ، وصحبةِ أستاذٍ، وطولِ زمان."

والصبر في العلم إنما هو صبرٌ على التعب، وصبرٌ على تكرار المذاكرة، وصبرٌ على فهم المسائل، وصبرٌ على مراجعة ما فات، وصبرٌ على ترك ما لا ينفع. فطالب العلم يعلم أن كل دقيقة يبذلها ستعود عليه نورًا في قلبه، ورفعةً في نفسه، ونجاحًا في دنياه وآخرته.