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MICROCIRCULATORY FAILURE – SHOCK

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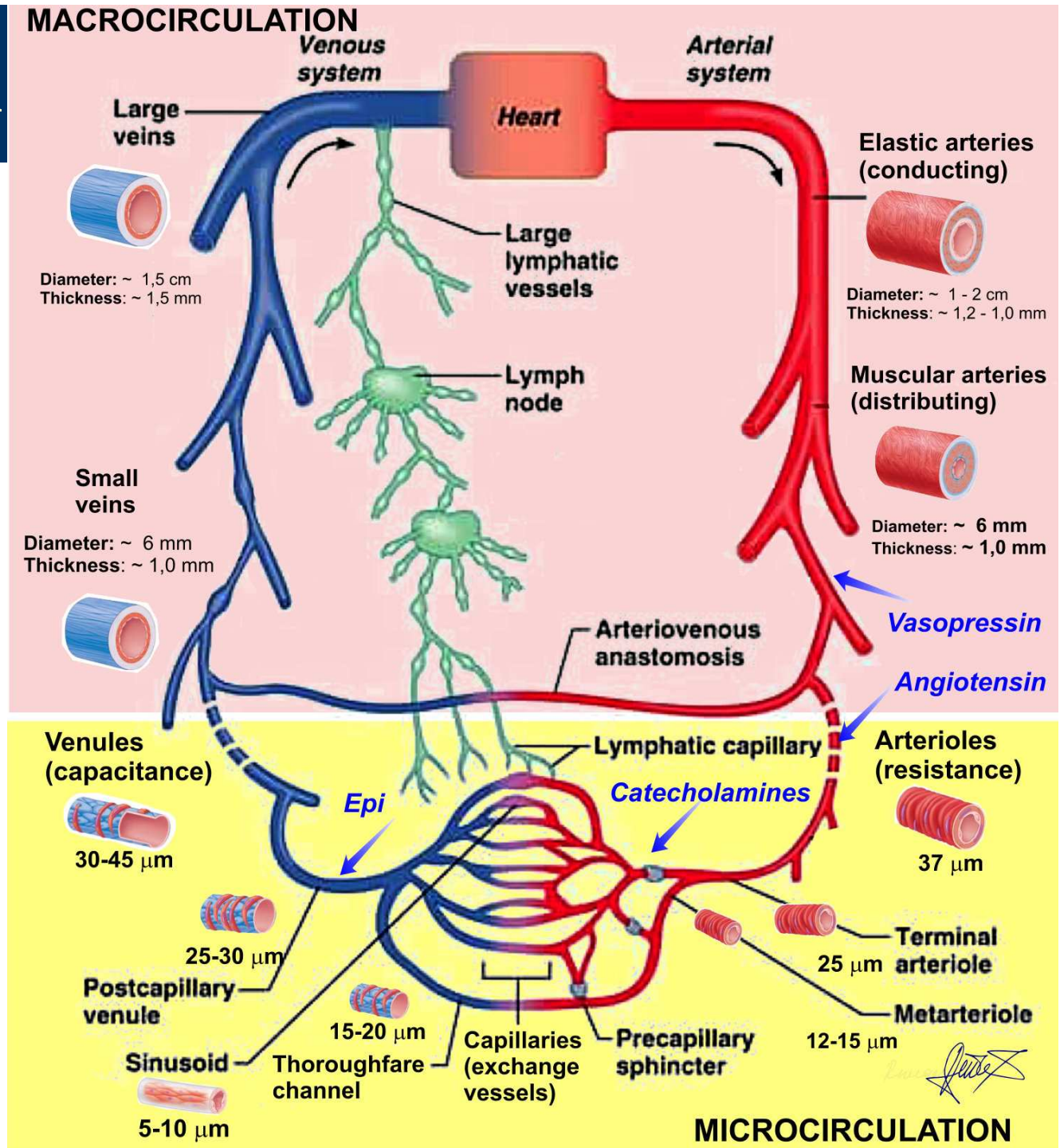
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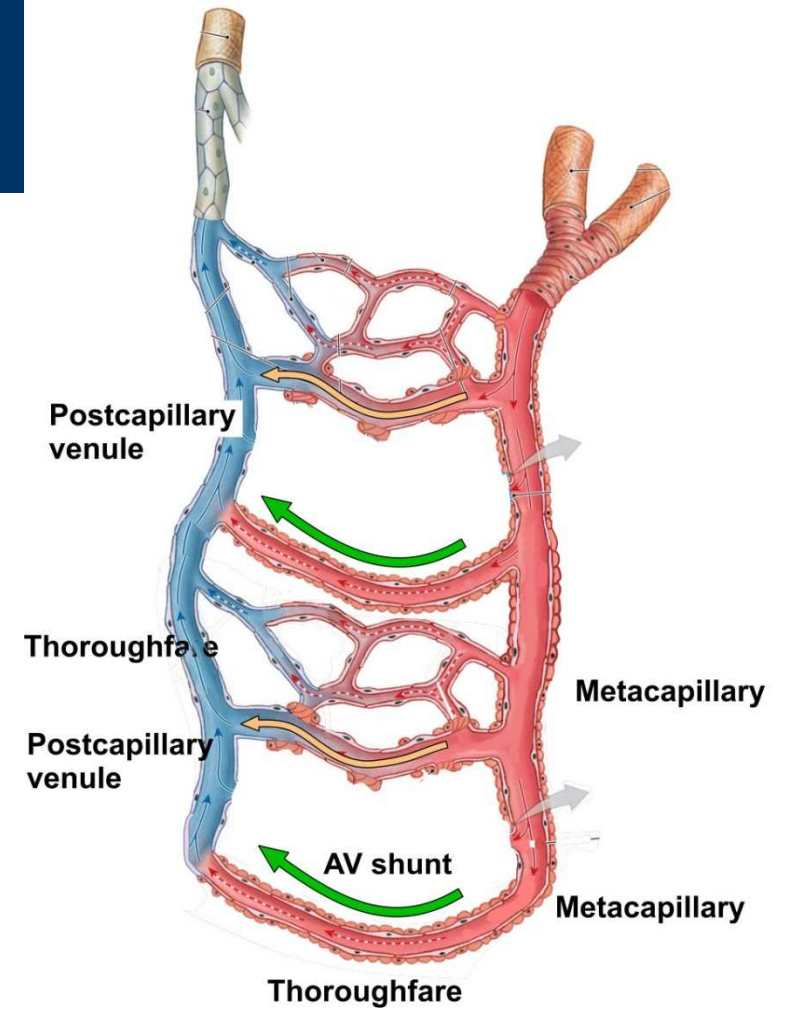
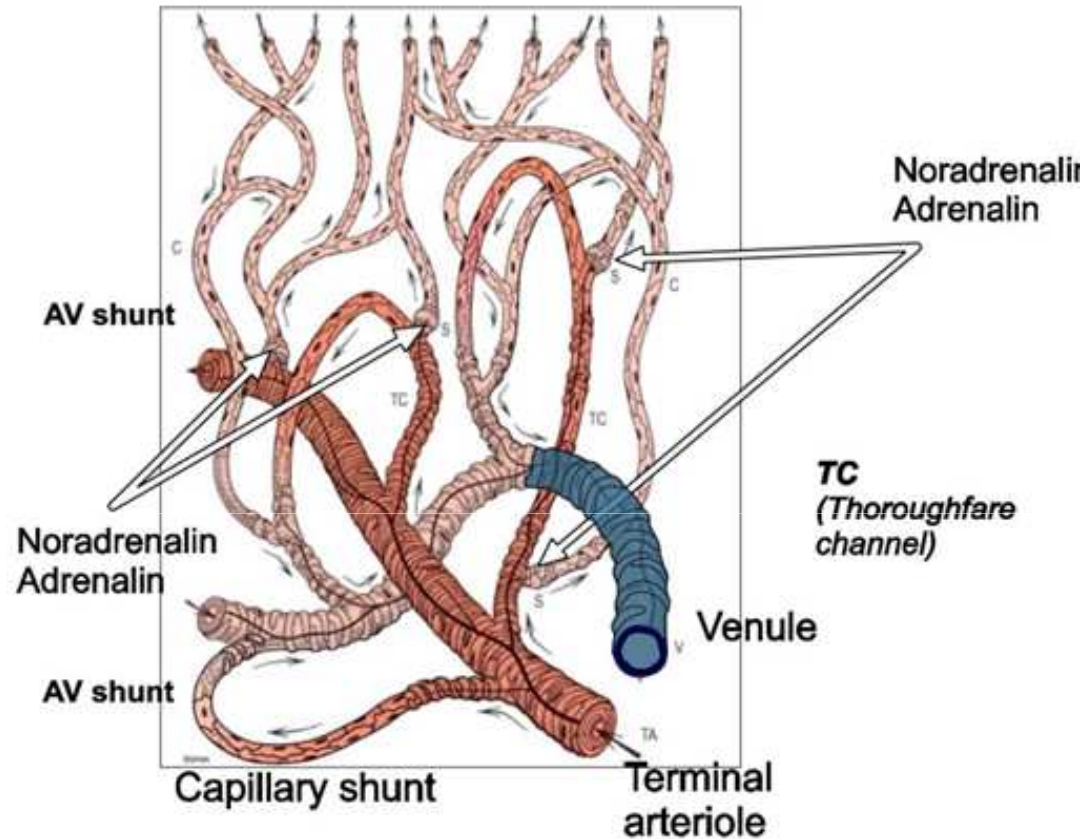
General considerations

Microcirculation

- To maintain heart pumping and circulation (SV 75 mlx HR 80b /min x) **most of blood bypasses capillary system** (venous blood carries ~50% of O₂) to venous return.
- In any moment only a portion (1/4) of blood circulated though **capillaries** to release oxygen and load metabolic wastings + CO₂
- Central dogma of circulation says to **maintain circuit to work in order to keep at least critical perfusion in sentinel tissues.** These tissues and vascular beds are monitored (tension pressure, PO₂, pH) to feed data back to spinal and brainstem and hypothalamic vegetative centra.



Microcirculation



Capillary perfusion is tightly regulated according to the momentary metabolic and systemic needs. Certain parts of circulation, e.g. lower extremities are in special regime as governed by antigravity needs. Muscle require blood during work, skin for thermoregulation. Internal organs and brain possess special suited „organ autoregulation“ which may differ each other.

General considerations - shock

- **Definition:** state characterized by spontaneously little compensable systemic hypotension, generalized organ hypoperfusion resulting in tissue hypoxia, shift to anaerobic metabolism, fail to clean-up accumulated metabolic wastings, dystrophy and death of cells leading to multiple organ failing
 - Hypotension introduces shock mostly, but these are not synonymous; hypotension, collapse or syncope are not necessarily associated with shock
 - Hypotension is not always only initial requirement for shock to develop. Local
 - Ratio: Metabolic demands \leftrightarrow blood supply + oxygen supply
- Whatever is the reason of hypotension and tissue hypoperfusion **general survival mechanisms of centralisation of circulation** are implied. Selective vasoconstriction in extremities and torso including pulmonary, renal and splanchnic circulation may open the blind circuit
- shock in individual organs – pulmonary embolus blocked blood supply to the lung (shock lung) or an embolus blocked blood supply to the stomach (shock stomach)

Symptoms of shock

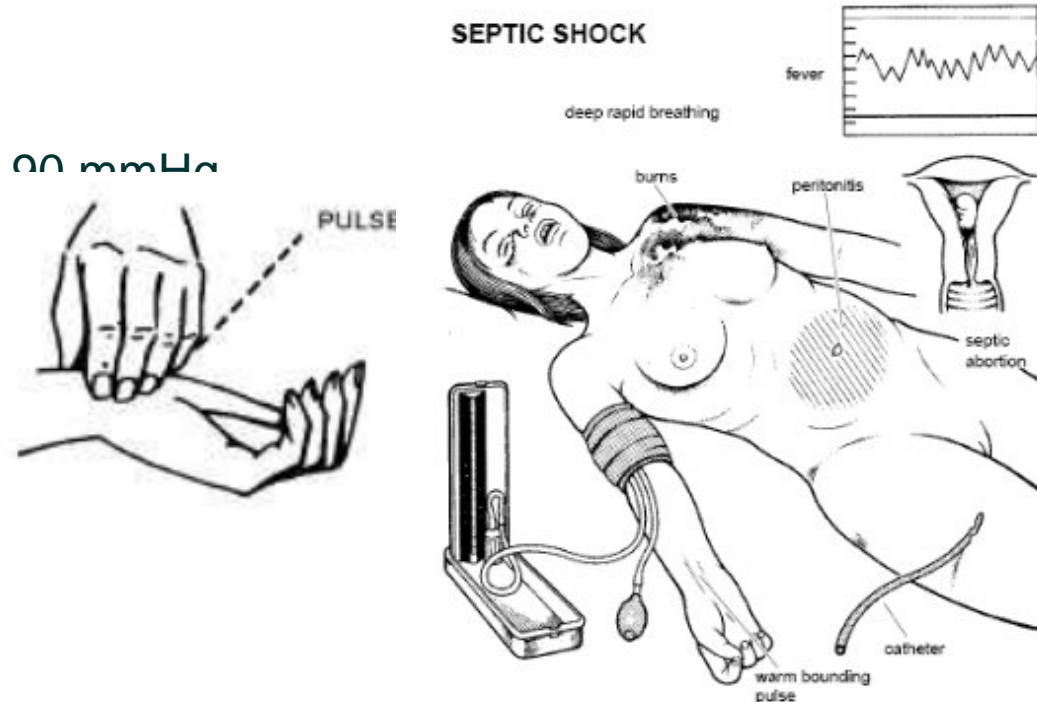
Early in situ manifestations

- Lethargy, Drowsiness, Dizziness,
- Weakness, Altered sensations
- Skin is cool, pale or clammy
- Hyperventilation; rapid and shallow
- Dilated pupils, Thirst, vomiting
- Pulse is rapid and weak
- Prolonged capillary refill time
- Pale, dry mucous membranes



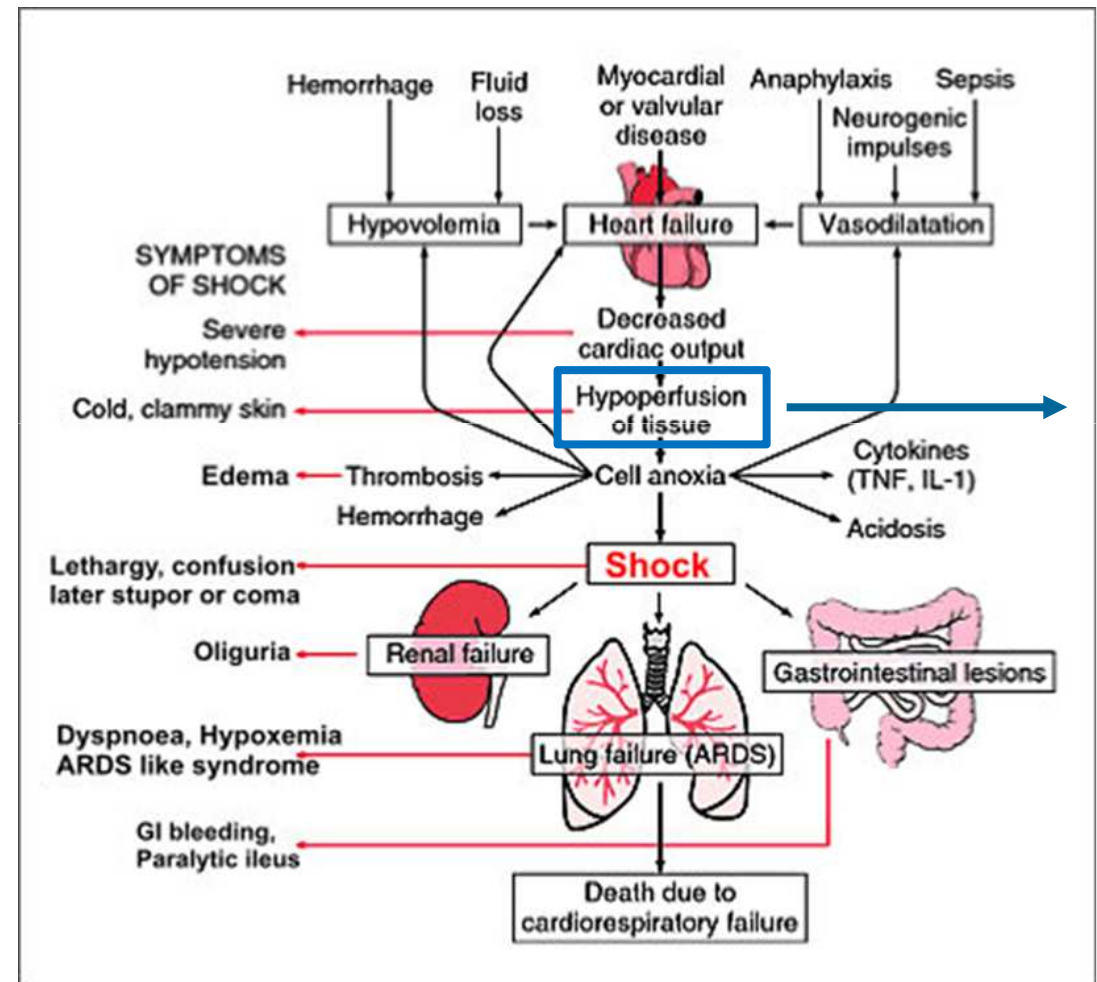
Later Measurable manifestations

- $\downarrow\downarrow$ Blood pressure (SBP is usually < 90 mmHg or $\Delta 30$ mmHg; 50 mmHg in hyp)
- \downarrow Hemoglobin & hematocrit
- Oliguria (\downarrow Urination)
- Electrocardiogram (AMI, other)
- Arterial blood gas
 - Pulmonary artery wedge
- Pressure
 - Cardiac output, Cardiac index
 - Central venous pressure



Compensatory mechanisms in shock

- Inadequate systemic oxygen → (+) O₂ chemoreceptors, H⁺ receptors (acidosis); Hypoperfusion → baroreceptors (aortic, carotic, liver, heart cavities)
- **Vegetative nervous sy.:** (+) sympathetic NS (norepinephrin from nerve endings), sympathetic-stimulated release of epinephrin from supraren medulla, (-) decrease in baseline vagal tone (involves baroreceptors in carotid arch, aortic arch, left atrium, and pulmonary vessels)
- **Major endocrine responses**
 - Epinephrine and dopaminereleased from the adrenal glands
 - HPA axis; release of cortisol
 - ADH (vassopressin) – reabsorption of water in collecting tubuli; small muscular arterial vasoconstriction.
 - RAA system: triggered by sympathetic + kidney hypotension; Angiotensin II → arteriolar constriction; Aldosteron → water + NaCl.



Compensatory mechanisms & outcomes in shock

- **Compensary systems:**
 - **Hematologic** (hemorrhagic shock, bleeding ← clotting system)
 - **Circulatory** (General sympathetic response: hear rate, myocardial inotropy, selective vasoconstriction) +, redistribution: to the brain, heart, kidneys away from skin, muscle, and GIT
 - **Renal system:** increase in renin → angiotensin I → angiotensin II (vasoconstriction) → aldosteron → reabsorption of NaCl +H₂O; Water and sodium conservation and vasoconstriction
 - **Neuroendocrine** hypotension (baroreceptors in liver, heart) → ADH (hypothalamus) → vasoconstrivtion + reabsorption of H₂O (collecting tubulus)
- **Cellular responses to decreased systemic oxygen delivery**
 - ATP depletion → ion pump dysfunction; anaeronic metablism
 - Production of lactate → acidosis → vasoconstriction
 - Cellular edema; hydrolysis of cellular membranes and cellular death
- **Goal is to maintain cerebral and cardiac perfusion →**
 - Vasodilation of brain vessels
 - Vasoconstriction of splanchnic, musculoskeletal, and renal blood flow
 - Leads to systemic metabolic lactic acidosis that overcomes the body's compensatory mechanisms
- Endothelial inflammation and disruption
- Lactic acidosis, cummulation of reactive oxygen species, release of cytokines

Pathogenesis - the cellular and tissue changes I

Organ, tissue, cellular changes may follow different patterns:

- **Hypoxic cell injury** (most common; typical for cardiogenic/hypovolemic, neurogenic shock) any tissue, particularly: brain, heart, lungs, kidneys, adrenals, and gastrointestinal tract.
- **Septic shock** → *disseminated intravascular coagulation* → deposition of fibrin-rich microthrombi → consumption of platelets and coagulation factors → petechial hemorrhages on serosa + skin; brain, heart, lungs, kidney, adrenal glands, and gastrointestinal tract.

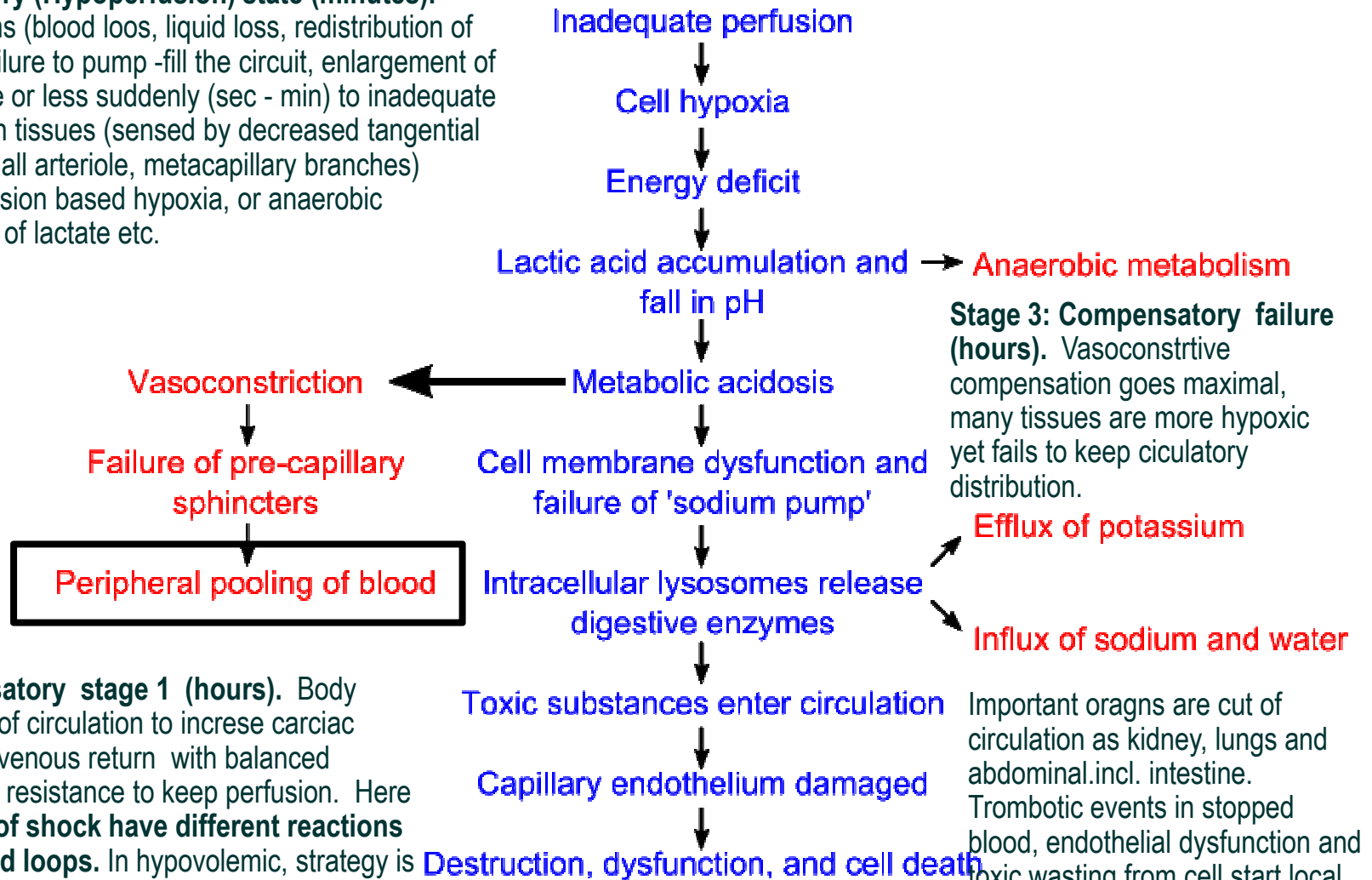
Typical organ changes:

- **Adrenal gland** (in all types of shock); conversion of vacuolate cells to metabolically active (utilize lipids for the synthesis of cortisol)
- **Shock kidneys** - (hypovolemic, cardiogenic shock); acute tubular necrosis
- **Shock lungs** - (septic shock; seldom in hypovolemic); *diffuse alveolar damage* shock lung.
- **Shock bowels** - (trauma, hypovolemia, septic, neurogenic shock; perforating necrosis)

Local cellular and tissue changes II

Stage 1: Introductory (Hypoperfusion) state (minutes).

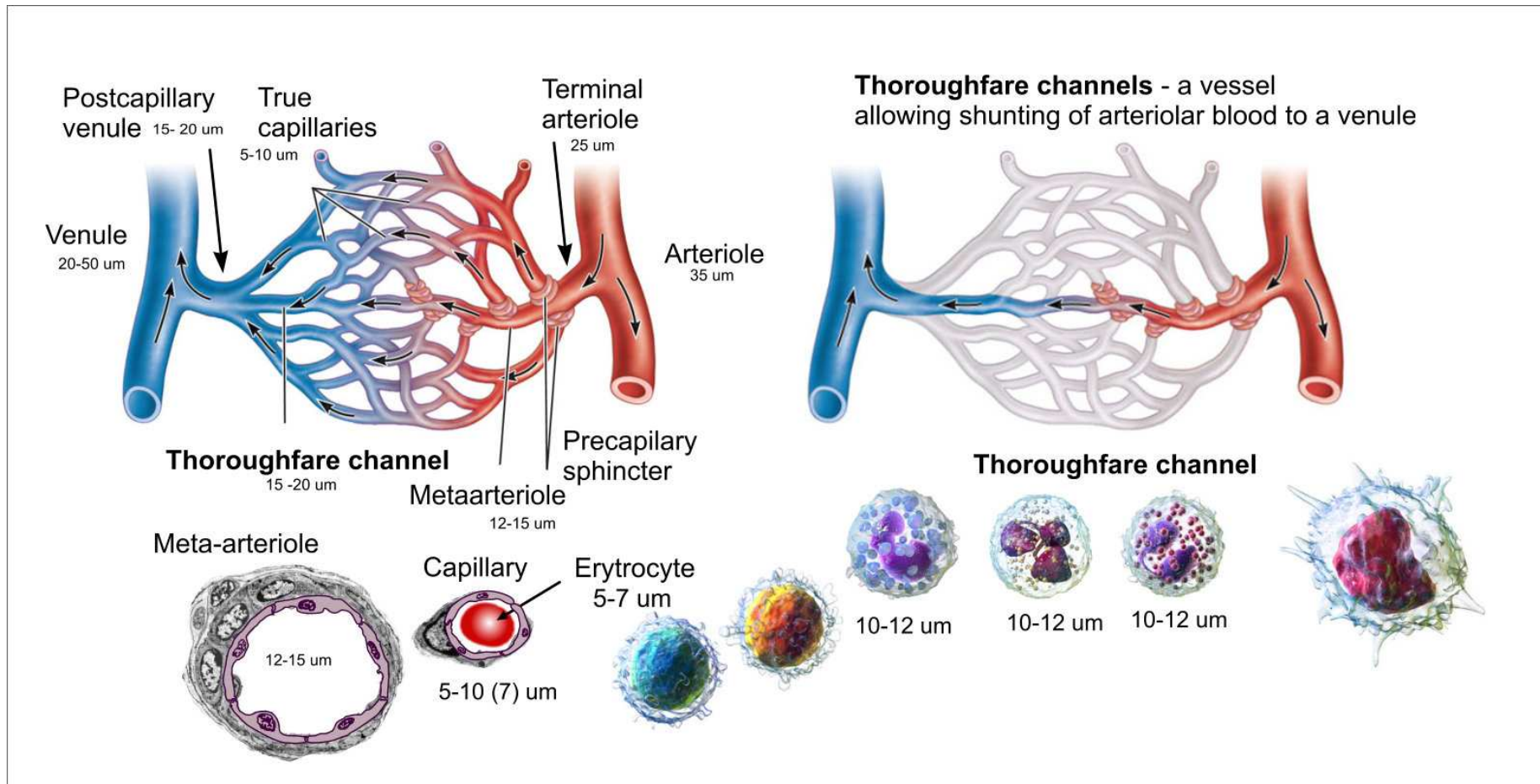
Different mechanisms (blood loss, liquid loss, redistribution of fluid out of circuit, failure to pump -fill the circuit, enlargement of circulation) lead more or less suddenly (sec - min) to inadequate perfusion pressure in tissues (sensed by decreased tangential filling pressure in small arteriole, metacapillary branches) leading to hypoperfusion based hypoxia, or anaerobic metabolism, release of lactate etc.

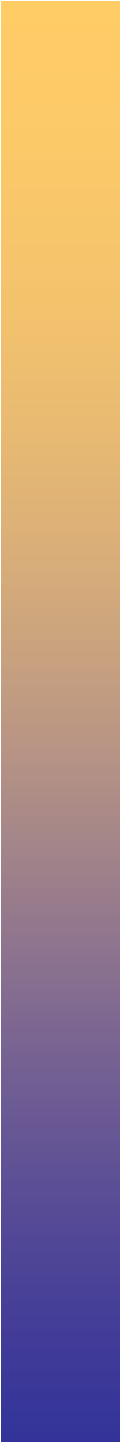


Stage 2: Compensatory stage 1 (hours). Body uses centralisation of circulation to increase cardiac output, to increase venous return with balanced systemic peripheral resistance to keep perfusion. Here **different variants of shock have different reactions of benefit and blind loops**. In hypovolemic, strategy is to keep volume in circuit with minima to capillaries.

Important organs are cut off from circulation as kidney, lungs and abdominal.incl. intestine. Thrombotic events in stopped blood, endothelial dysfunction and toxic wasting from cell start local proces of necrosis, toxins are released into cicaluation. MODS is comming.

Microcirculation





Clinical types of shock

Types of shock

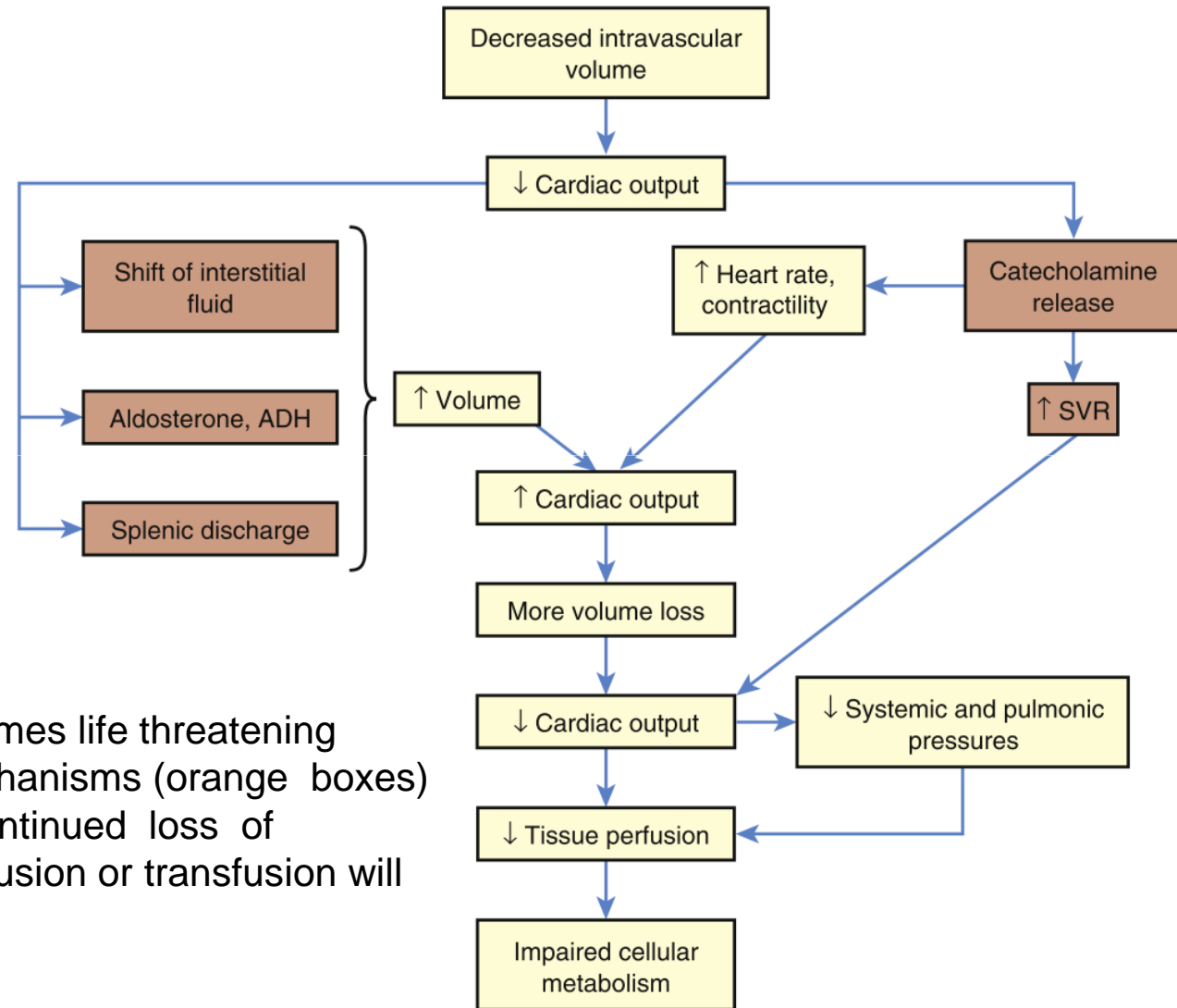
- **Hypovolemic** – loss of adequate volume of fluid in circulating blood
- **Cardiogenic** – failure of heart as a pump; systolic or diastolic dysfunction
- **Obstructive** – disability to pump a blood
- **Distributive (Vasogenic)** – extensive inappropriate vasodilation or redistribution of blood volume to unused vessels
 - **Septic** (Gram (-) endotoxic); toxic vasoparalysis
 - **Anaphylactic** (hypersensitive response)
 - **Neurogenic** (failure of reflexive vascular pressure regulation due to spinal cord injury)
 - **Psychogenic** (failure of reflexive vascular pressure regulation due to psychogenic reactions, fainting)
 - **Endocrine** – suprarenal failure (Waterhouse- fridrichsen sy.) - combination of septic +

Types of shock - Hypovolemic Shock

- Def: untrated progressive hypotension due to loos of blood volume or blood stasis in abdominal area
- Etio: trauma, polytrauma, hemorrhage, Diarrhea, Vomiting,
 - Non-hemorrhagic: Vomiting, Diarrhea, Peritonitis (loss of solutes; bowel obstruction, acute pancreatitis – peforation); Burns (leak of plasma), Neglect, Environmental (dehydration)
 - Hemorrhagic: traumatic, non-traumatic GI bleed, Trauma, Massive hemoptysis, AAA rupture
 - Ectopic pregnancy, post-partum bleeding
- Sy: typical is hypotension; centralisation of circulation, paleness, tachycardia, fainting, drowsiness,
- Compensation: 3-4 systems

Hypovolemic shock

Centralisation of circulation is done by **sympathetic vasoconstriction** via NA action on alfa receptors in resistant vessels and precapillary sphincters..



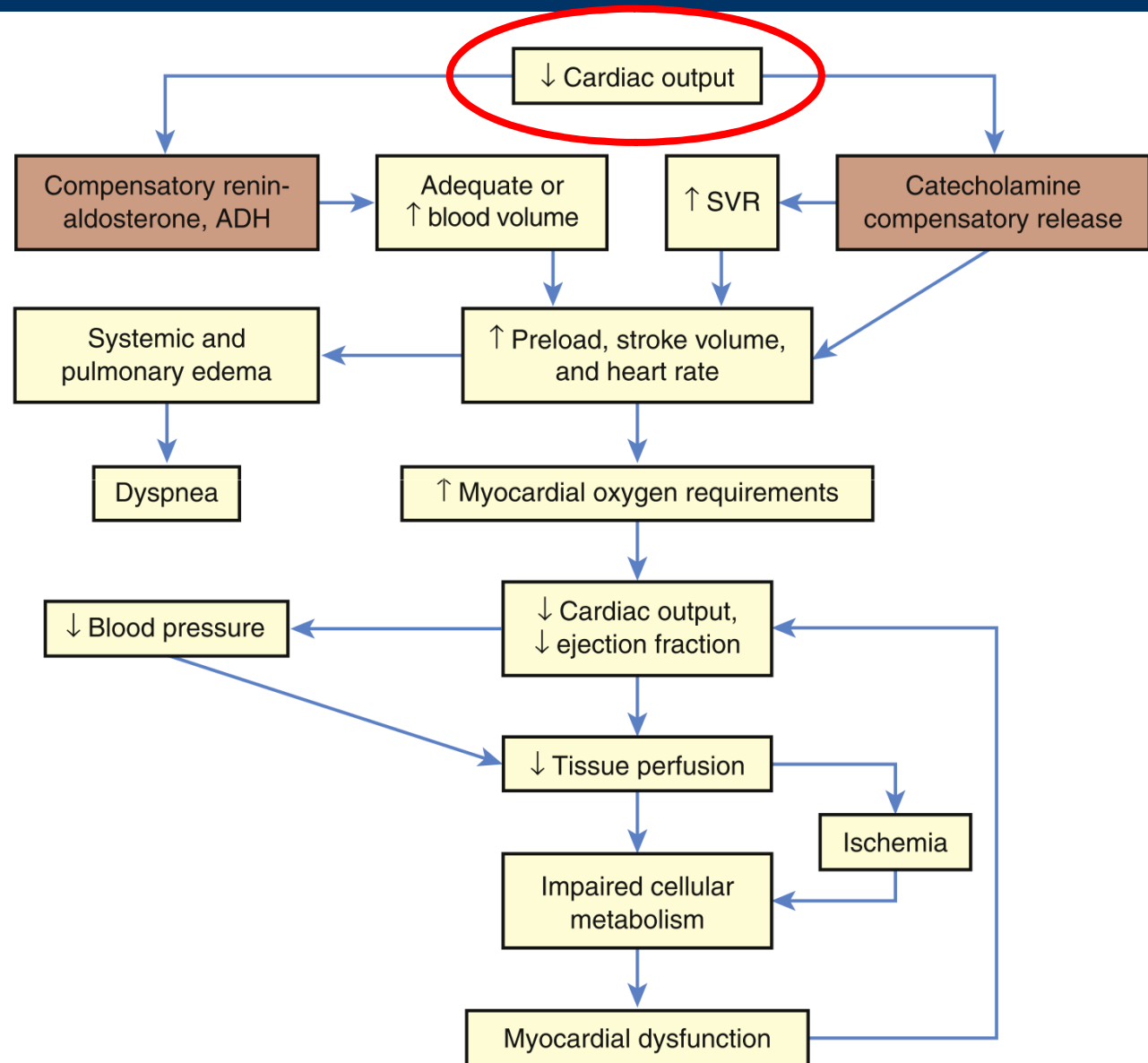
Hypovolemic shock becomes life threatening when compensatory mechanisms (orange boxes) are overwhelmed by continued loss of intravascular volume. Inflation or transfusion will be necessary

Types of shock - Cardiogenic Shock

- Def.: failure of pumping capability of the heart; is characterized by a decreased pumping ability of the heart causing a shock-like state with inadequate perfusion to the tissues
- Etio: most commonly acute myocardial infarction (AMI), cardiomyopathy;
 - **Intrinsic:** Myocardial injury, paroxysmal tachycardia, Bradycardic arrhythmias (AV block III), valvular defects (rupture), Aortic or mitral stenosis, Acute aortic insufficiency, cardiomyopathy
 - **Extrinsic:** Pericardial tamponade, Tension pneumothorax, Large pulmonary embolus, Myocardial contusion
- Compensation: 2 systems mainly: kidney (RAA), neuroendocrine (ADH)
- Sym:
 - Cool, mottled skin, Tachypnea, Hypotension, Altered mental status
 - Narrowed pulse pressure, rales, murmur
 - SBP (systolic) < 90 mmHg, CI < 2.2 L/m/m²; PCWP > 18 mmHg
 - Lose 40% of LV clinical shock ensues, CO reduction = lactic acidosis, hypoxia

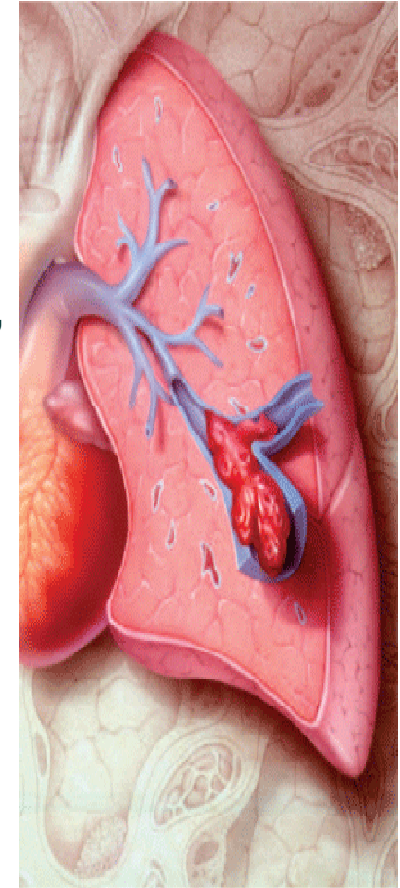
Cardiogenic shock

Centralisation of circulation is done by **sympathetic vasoconstriction** via NA action on alfa receptors in resistant small muscular arteries and precapillary sphincters. Adequate intravascular volume + vasoconstriction are achieved by ADH together with RA-aldosterone.



Types of shock - Obstructive shock

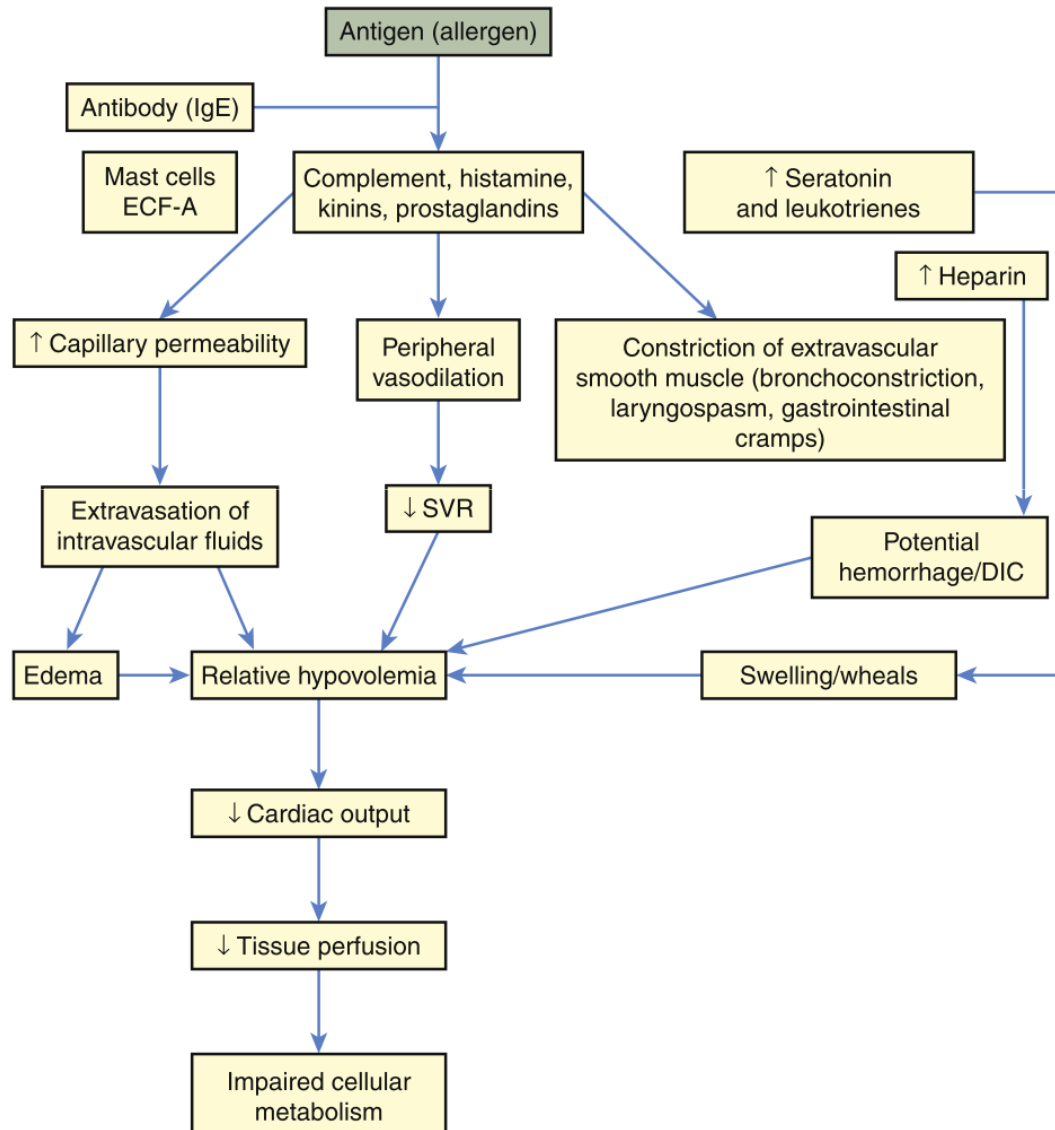
- **Tension pneumothorax** - air trapped in pleural space with 1 way valve, air/pressure builds up
 - Mediastinum shifted impeding venous return, Chest pain, SOB, decreased breath sounds
- **Cardiac tamponade** - blood in pericardial sac prevents venous return to and contraction of heartm, Related to trauma, MI pericarditis,
 - Beck's triad: hypotension, muffled heart sounds,
- **Pulmonary embolism**
 - Virchow triad: hypercoaguability, venous injury, venostasis
 - Signs: tachypnea, tachycardia, hypoxia,
 - Low risk: D-dimer` Higher risk: CT chest or VQ scan
- **Aortic stenosis**
 - Resistance to systolic ejection causes decreased cardiac function
 - Chest pain with syncope, Systolic ejection murmur
 - Diagnosed with echo, Vasodilators (NTG) will drop pressure!



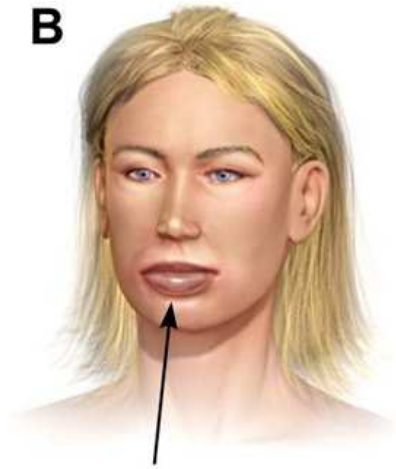
Types of shock - Anaphylactic shock

- Def.: sudden hypotension during anaphylactic reaction (exaggerated systemic hypersensitivity type I); happens in allergic persons after peroral (15 to 30 min after) or parenteral exposure (2-5 min after) to specific allergens.
- Etio: allergens = β -lactam ATB (penicillin), NSAIDs, aspirin, local anesthetics like lidocaine, procaine, articaine, mepivacaine.
- Patg: massive mastocyte degranulation \rightarrow histamine and cytokines (SRS-A) \rightarrow overall precapillary vasoparalysis+ increased capillary permeability \rightarrow leak of volume out of vessels \rightarrow no return \rightarrow hypotension
- Sy.:
 - Early- Pruritus, flushing, urticaria appear, flushing and swelling of face, especially the areas around the eyelids and lips;.
 - Next- Throat fullness, anxiety, chest tightness, shortness of breath and lightheadedness, generalized urticaria (mainly the hands and feet paresthesia, rapid weak pulse, wheezing and difficulty in breathing
 - Late Altered mental status, respiratory distress and circulatory collapse

Anaphylactic shock



Exanthema + swelling



Swelling



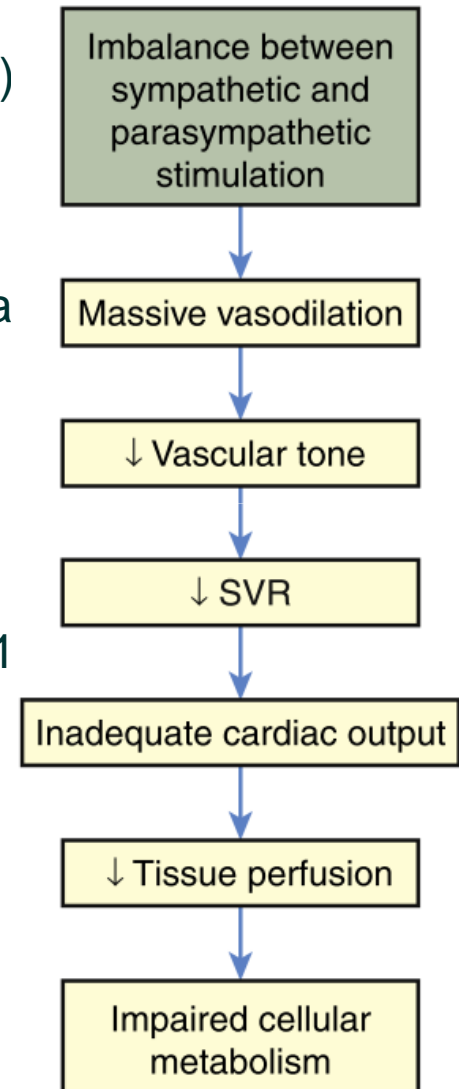
Exanthema + swelling



Laryngospasm in alimentary anaphylaxis

Types of shock - Neurogenic shock

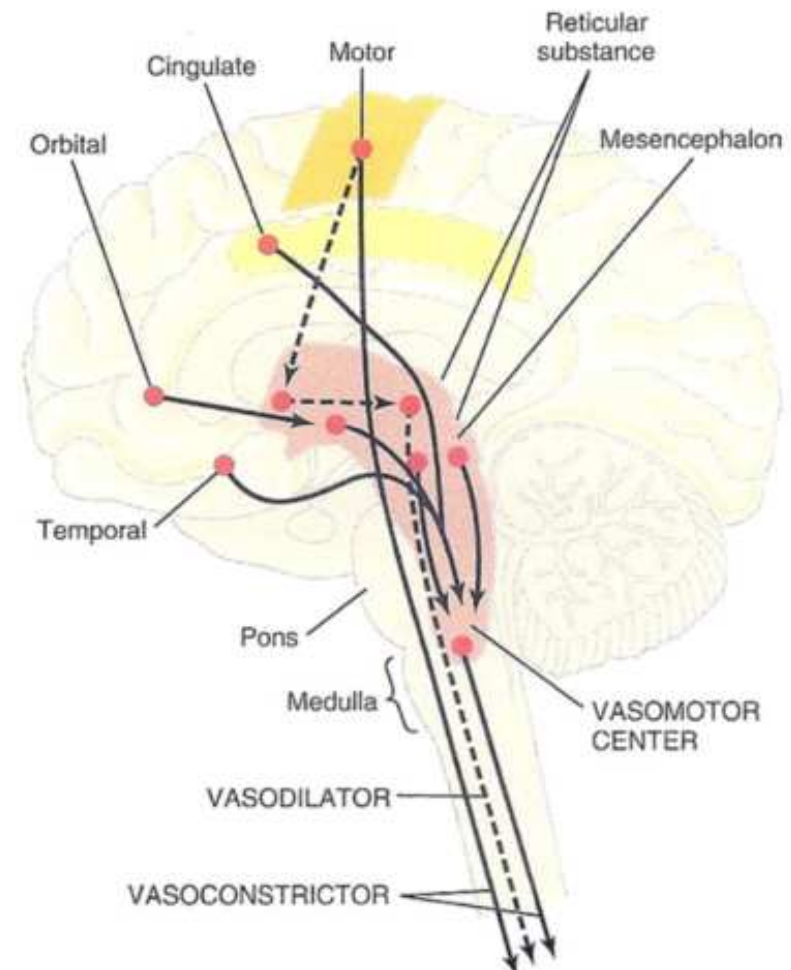
- Def.: failure of baroreflexive regulation of resistive vascular tone by vegetative NS by damage on central or peripheral level) no actual blood loss
 - Neurogenic shock is not identical with spinal shock, even if it may occur concurrently
 - Spinal shock- temporary loss of spinal reflex activity below a total or near total spinal cord
- Etio:
 - damage of bulbar vasomotor centra (lateral medullary sy. involving adrenergic and serotonergic groups),
 - descending pathways (bulbospinal) and intermediolateral spinal cord columns in high-level spinal cord injury above T1 can disrupt the entire sympathetic system
- Sy:
 - Shock usually lasts 1 - 3 weeks; classic signs may not be present; higher injuries → worse paralysis
 - Pooling of blood within the extremities → warmed flushed dry (reddish) skin
 - Vagal overactivity → bradycardia, It is easily exacerbated by hypoxia and vagal maneuvers.



Types of shock - Psychogenic shock

Psychogenic shock (syncope, fainting)

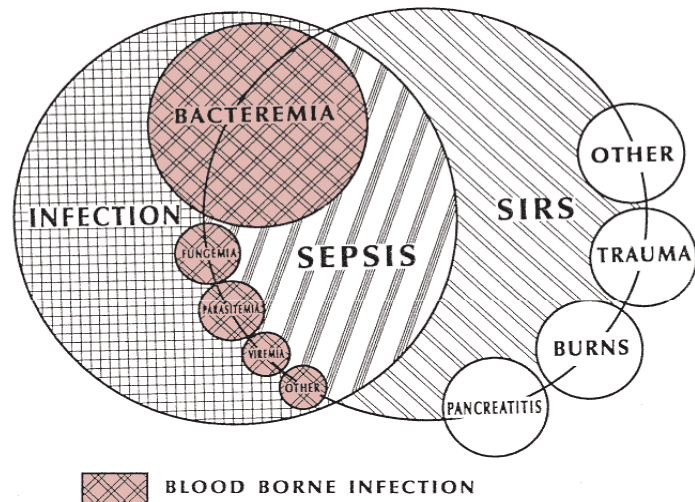
- **Def:** Sudden hypotensive attack caused by central failure of vegetative vasomotor regulation and baroreflexive loops
- **Etio:** pain (e.g. torture, burns, polytrauma, fractures (often after head injury, skull); emotional distress
- Differs from classical syncope as of primary nervous non.epileptogenic origin. Effect similar to neurogenic shock; not real microcirculatory shock state
- **Sy:**
 - Sudden hypotension ← loss of sympathetic activity → bradycardia
 - Nausea, vomiting ← overactivity of vagi
 - Loss of consciousness, “fainting” (syncope)



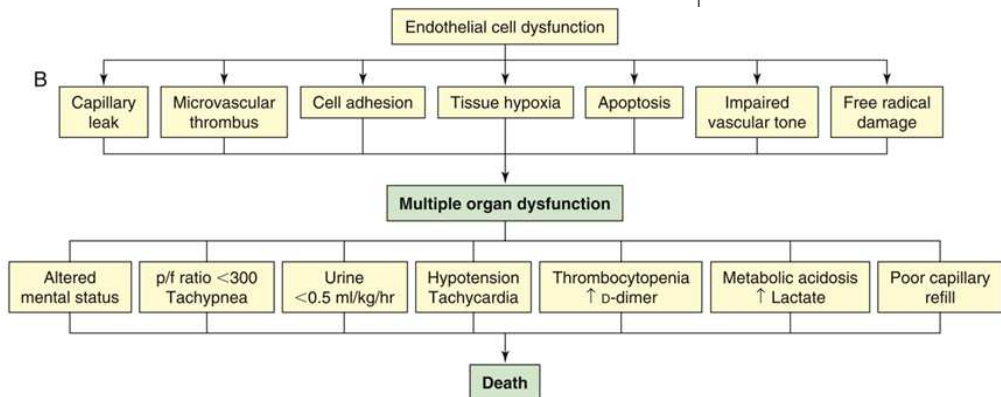
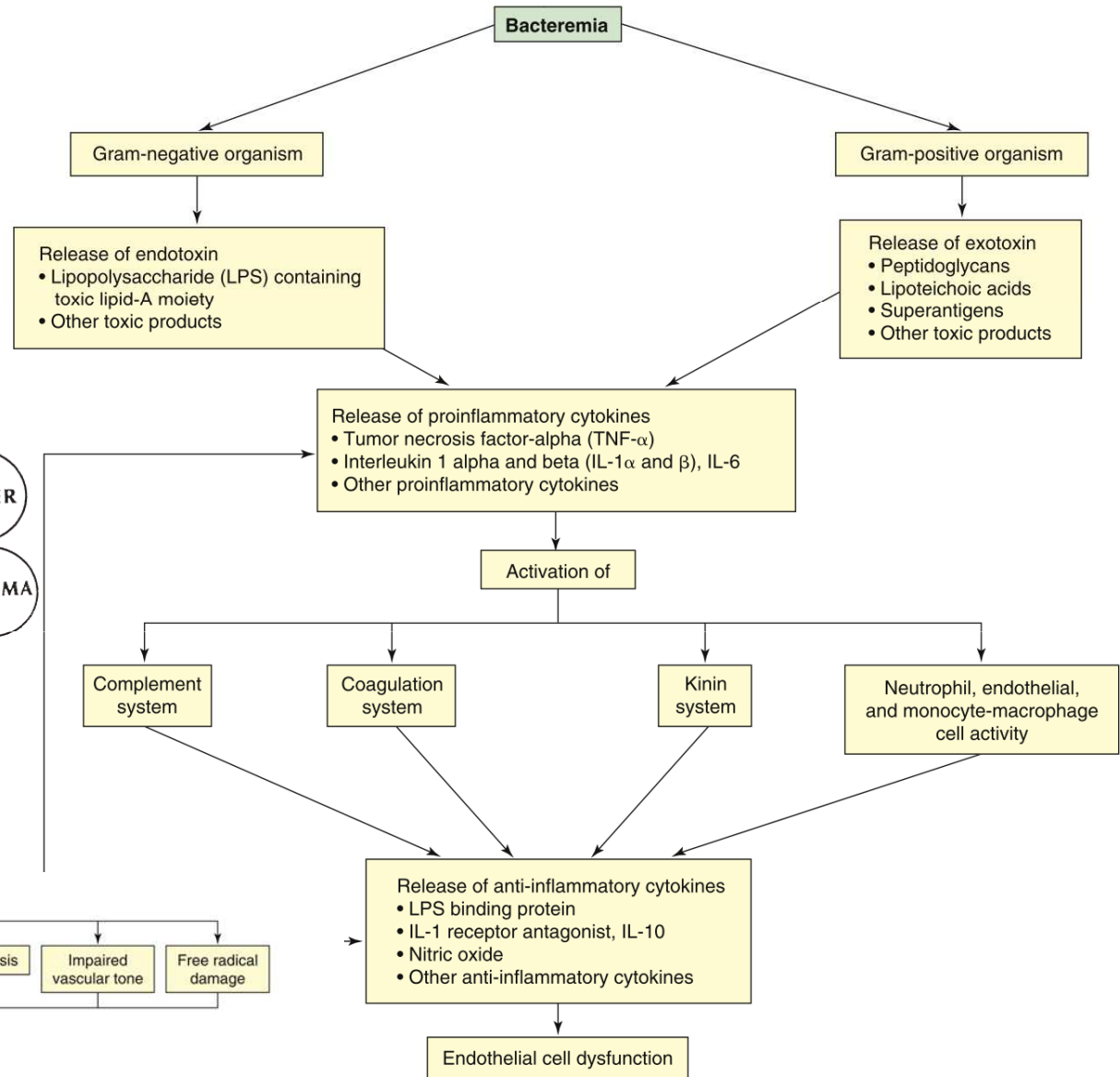
Types of shock - Septic shock

- **Def.:** sepsis-induced hypotension that persists despite treatment with intravenous fluids- It is the most common cause of distributive shock developing in the severe sepsis characterized by intensive systemic inflammation (SIRS), bacteriemia and septicemia or pyemia.
- **Pat:** *polytopic vasoparalysis*, increased capillary leak, decreased systemic vascular resistance; hypotension due to intravascularly released bacterial endotoxins or exotoxins,
- **Etio:**
 - Most cases caused by **gram-positive bacteria** (pneumococci, streptococci as well as *Gram-positive bacterial* toxins (heat-stable entero-toxins, hemolysins, phospholipases),
 - **Endo-toxin-producing gram-negative bacteria** (lipopolysaccharides LPS, Escherichia coli, Proteus species, Klebsiella pneumoniae).
 - Even if precipitating infection is most commonly by bacteria, **fungal infections** are an increasingly prevalent cause and also *viruses, or parasites*.
- Infection most commonly in sepsis: *lungs* (pneumonia), *brain* (meningitis), *urinary tract* (pyelonephritis), *skin* (necrotizing fasciitis), *or abdominal organs* (appendicitis, diverticulitis, mesenteric necrosis, pancreatitis).

Septic shock



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„Warm“ and „cold“ stage of septic shock

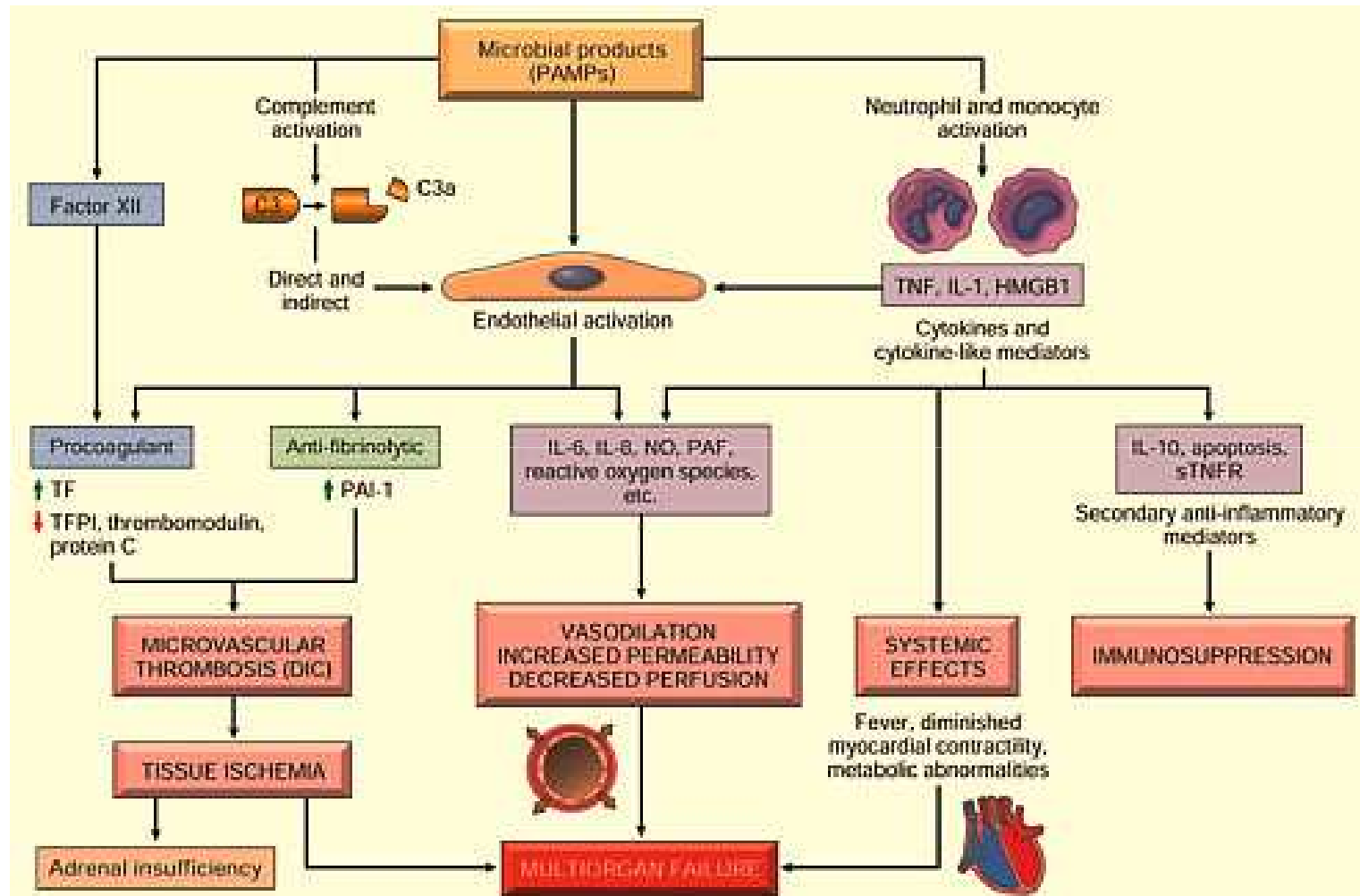
- Septic shock can be broken down into two different types of shock: **warm (or hyperdynamic) shock** and **cold (or hypodynamic) shock**.
- **Warm shock (6-72h)** is characterized by **high cardiac output + low peripheral vascular resistance**. Vasodilation is the effects of histamine, bradykinins, serotonin, and endorphins dramatically decrease total peripheral vascular resistance. It also makes capillaries more permeable causing **leakage and fluid shifting into tissues and physiologic third spaces**.
- **Fever (>39C)** is caused by endogenous pyrogen released by leukocytes attacking gram-negative bacteria. Unusually **high respiratory rate** (32 breaths/min) cause a profound **respiratory alkalosis** that **counterbalances lactic acidemia**. It may actually shift the **patient's pH toward alkalosis**. **Tachypnoea** result from the effect of the bacterial endotoxins on the medullary respiratory center.
- **Profound diuresis** develops due to high osmotic load being handled by the kidneys. This may be result of dead and dying bacteria, phagocytic cells, tissue breakdown (?).
- **Tissue metabolic acidosis** and **hyperventilatory alkalisation** may keep PaO₂ elevated while in this early stage of septic shock. Oxygen saturation of mixed venous blood will most likely of mixed venous blood will most likely be greater than the normal 80%.
- **Activation of the clotting mechanism**, resulting in **coagulopathy**. The complement system contributes to the damage of the vascular endothelium and to neutrophil aggregation, while the Hageman factor accelerates clotting and causes multiple fibrin clots to form. Clots plug up small capillaries, producing **petechiae and altered blood flow, which appears as "creeping" mottling of the legs**. The mottling starts in the feet and works its way up to the knees. When clotting factors are used up in the microcirculation serum levels of clotting Factors V, VIII, and XIII, as well as platelet and fibrinogen decrease.
- Decreased cerebral perfusion produce **impaired mental status**- delayed responses with restlessness and confusion. Many patients are alert and oriented until multisystem failure sets in. The release of endorphins, may be responsible for keeping the patient relatively calm.

Warm“ and „cold“ stage of septic shock

Cold shock (72h – end)

- Most patient will remain in warm shock for **6 to 72 hours** before entering cold shock (also known as low-output or high-resistance shock). This late and nearly irreversible phase of septic shock is usually **indistinguishable from terminal hypovolemic shock**.
- Two ominous signs of cold shock are a **subnormal temperature** and a **low white blood cell count** (leucopenia (with many immature cells). By the time the patient gets to this stage, his **hypotension** and **hypoperfusion** are profound. His **skin will be cold and mottled** in a more generalized fashion- not just below the knees, as in warm shock. Pulse and respirations will still be rapid because of the continued firing of sympathetic nerves and increased catecholamine levels.
- **Cardiac output decreases, peripheral resistance increases**. Skin is cold with many spots, heart rate and respiratory rate are still increased since sympathetic overstimulation persists. Catecholamines make selective „centralisation of circulation“ vasoconstriction of **the renal, pulmonary, and splanchnic circulations**. Myocardial depressant factor is released from pancreatic cells. More beta endotoxins block pain impulses but further depresses the myocardium.
- In **microcirculation thrombotic process runs**., which lead to irreversible capillary closure perfusion and also may lead to consumptive coagulopathy.
- Cold shock brings **multisystem failure** - pulmonary edema, adult respiratory distress syndrome, liver and kidney failure, even hemorrhaging from disseminated intravascular coagulation. The patient's mental status and reflexes deteriorate because of hypoperfusion and cerebral microemboli.
- ABGs will show **uncompensated hypoxemia, acidemia, and hypoventilation**.

Major pathogenic pathways in septic shock.

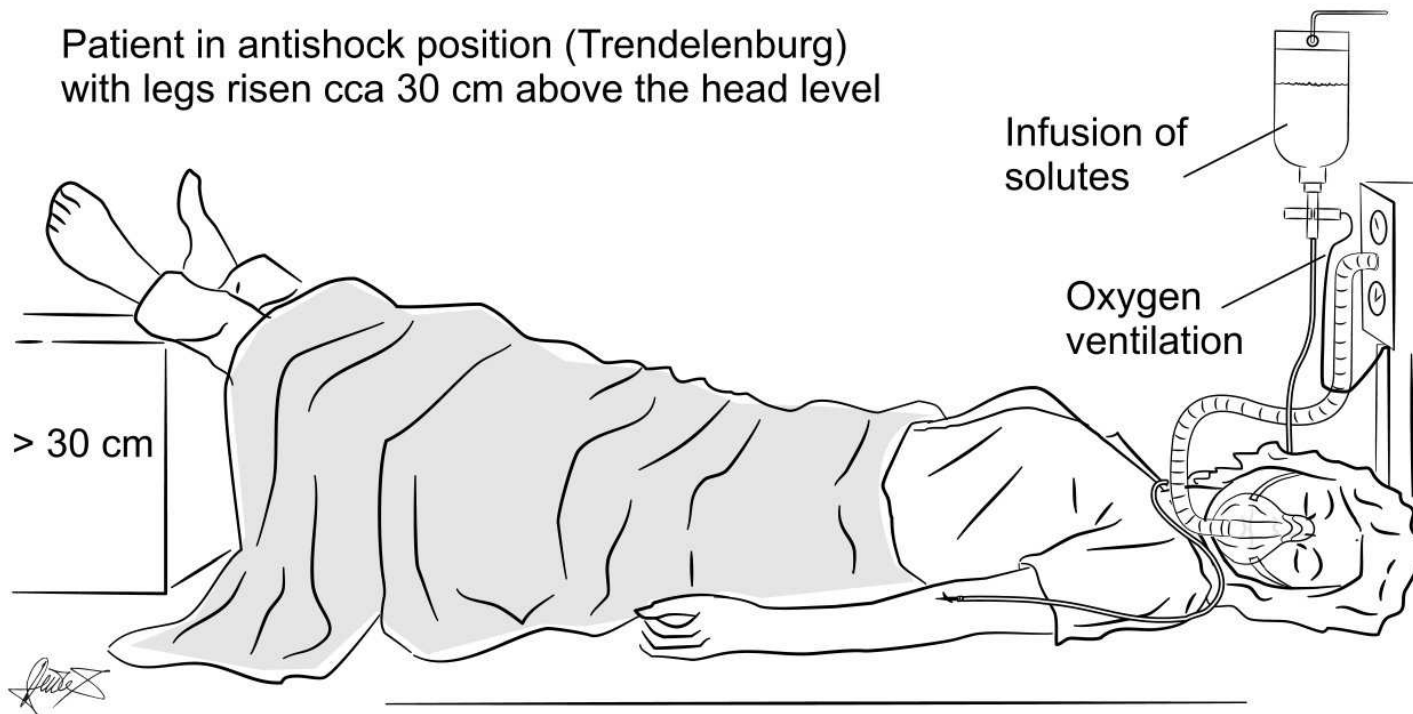


Microbial products (PAMPs, molecular patterns) activate endothelial cells and cellular and humoral elements of the innate immune system, initiating a cascade of events that lead to end-stage multiorgan failure. HMGB1, high mobility group box 1 protein; NO, nitric oxide; PAF, platelet activating factor; PAI-1, plasminogen activator inhibitor 1; TF, tissue factor; TFPI, tissue factor pathway inhibitor.

Shock resuscitation

- **Shock position** - patient is positioned flat with feet at least 30 cm above head level (Trendelenburg position)
- **Ventilation** - medicinal oxygen or room air
- **Catecholamine:** 0,5 ml epinephrine (adrenaline) 1mg/ml (1 in 1000) intramuscular - 0.25 ml for 6-12 years and 0,12 ml for 6 months to 6 years
- **Chlorphenamine** (chlorpheniramine) 10 mg in 1 ml intramuscular or slow i.v. injection
- **Glucocorticosteroide.** Hydrocortisone sodium succi-nate 200 mg by slow i.v. injection
- **Fluids** i.v. (colloids) infused rapidly if shock not responding quickly to adrenaline injection

Patient in antishock position (Trendelenburg)
with legs risen cca 30 cm above the head level



Physiological values for evaluation of shock state

Parameter	Values	Content
Systemic arterial pressure (SAP)	120-130/80 mmHg	pressure in the arterial system
Central venous pressure (CVP)	3–8cm H ₂ O	the blood pressure in the venae cavae, near the right atrium of the heart.
Right ventricular pressure (RVP)	systolic 15–30mmHg diastolic 3–8mmHg	blood pressure within the right ventricle
Pulmonary arterial pressure (PA) systolic 15–30 diastolic 4–12	mean 9 - 18 mmHg	blood pressure found in the pulmonary artery by catheter into the pulmonary artery.
Pulmonary wedge pressure (PWA)	2–15 mmHg 6–12 mm Hg	pressure measured by wedging a pulmonary catheter with an inflated balloon into a small pulmonary arterial branch
Cardiac index (CI)	2.6–4.2 L/min/m ² .	cardiac output (CO) from left ventricle in one minute to body surface area (BSA)
Cardiac output (CO)	4.0–8.0 L/minute	the volume of blood being pumped by the heart left or right ventricle, per unit time.
Systemic vascular resistance (SVR)	9–20 mmHg·min/L	resistance that must be overcome to push blood through the circulatory system and create flow.
Stroke volume (SV)	94 mL (± 15 mL)	volume of blood pumped from the left ventricle per beat.

Differential diagnostics for various shock states

Shock	CVP	RVP	PA	PAWP	CI	CO	SVRI	SVR	SvO ₂
Hypovolemic	↓	↓	↓	↓	↓	↓	↑↑	↑↑	↑
Cardiogenic	↑	↑	↑↑	↑	↓	↓	↑↑	↑↑	↑
Distributive	↓	↓	↓	↓	↑↑	↓	↑↑	↓	↓
Obstructive pulmonary embolia	↑↑	↑↑	↑↑	↓	↓	↓	↑↑	↑↑	—
Obstructive cardiac tamponade	—	—	—	—	↓	↓	↑	↑↑	—

Central venous pressure (CVP), Right ventricular pressure (RVP), Pulmonary arterial pressure (PA)
Mixed venous oxygen saturation (SvO₂)

Repetitorium 1

Repetitorium 2

- In shock resuscitation the goal is to provide some perfusion while not elevating the BP too much. Sometimes this is called permissive hypotension.
- In shock, the pulse pressure (difference between the systolic BP and diastolic BP) narrows or becomes smaller.
- Typical for obstructive shock is that increased pressure in the thoracic cavity prevents blood return from the head to the thoracic cavity resulting in distended neck veins.
- Vital signs in the shock patient shows that the pulse increases to help improve cardiac output and respirations increase to help maximize oxygenation of circulating blood.
- Anaphylaxis causes distributive shock by loss of vascular tone
- Obstructive shock situation is commonly associated with tension pneumothorax
- Tension pneumothorax increases pressure within the chest collapsing low-pressure vessels like the vena cava. This pressure creates an obstruction to adequate blood flow. Cardiac output is significantly lowered and shock develops.
- Torso contains large vessels (e.g. aorta) and organs (e.g. lungs) that can bleed profusely and are common causes of profound shock. One can not bleed too much into head to make shock, on other hand bleeding from extremities is visible and stoppable

Cases

Neurogenic shock

- A 41 yo M presents to the ER after an MVC complaining of decreased sensation below his waist and is now hypotensive, bradycardic, with warm extremities
- Fluid resuscitation
- Keep MAP at 85-90 mm Hg for first 7 days
- Thought to minimize secondary cord injury
- If crystalloid is insufficient use vasopressors
- Search for other causes of hypotension
- For bradycardia
- Atropine
- Methylprednisolone
- Used only for blunt spinal cord injury
- High dose therapy for 23 hours
- Must be started within 8 hours
- Controversial- Risk for infection, GI bleed

Obstructive shock

- A 24 yo M presents to the ED after an MVC c/o chest pain and difficulty breathing. On PE, you note the pt to be tachycardic, hypotensive, hypoxic, and with decreased breath sounds on left
- Corticosteroids
- Methylprednisolone 125 mg IV
- Prednisone 60 mg PO
- Antihistamines
- H1 blocker- Diphenhydramine 25-50 mg IV
- H2 blocker- Ranitidine 50 mg IV
- Bronchodilators
- Albuterol nebulizer
- Atrovent nebulizer
- Magnesium sulfate 2 g IV over 20 minutes
- Glucagon
- For patients taking beta blockers and with refractory hypotension
- 1 mg IV q5 minutes until hypotension resolves