Circulatory Shock for medical student

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Outline

- Definition of shock
- Pathophysiology
- Classification of shock
- Clinical features
- Investigation
- Management
- Summary

The personification of cholera, 19th century engraving from Barcelona

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Definition of shock

"The clinical expression (syndrome) of *circulatory failure* (profound & widespread reduction of effective tissue perfusion) that results in *inadequate cellular oxygen utilization* (mismatch O_2 delivery and consumption)"

Shock is mostly found with hypotension but not excluding normotensive state

Critical Care Medicine: Principles of Diagnosis and Management in the Adult, 5th Edition.
Cecconi M, et al. Intensive Care Med. 2014 Dec;40(12):1795-815.
Vincent JL, De Backer D. N Engl J Med. 2013 Oct 31;369(18):1726-34.

Oxygen delivery

- **Oxygen is vital to most organisms**
- **Effective oxygen delivery is needed to keep organs intact**

5 important equations/relations

1) **Oxygen delivery (DO_2) = Cardiac output (CO) x Oxygen content in arterial blood (CaO_2)**
2) **CO = Stroke volume (SV) x Heart rate (HR)**
3) **CaO_2 = (1.34 x Hb x Oxygen saturation (SaO_2)) + (0.003 x arterial oxygen tension PaO_2)**
4) **Stroke volume α Preload, Contractility, 1/afterload**
5) **Mean arterial pressure (MAP) – Central venous pressure (CVP) = CO x Systemic vascular resistance (SVR)**

Normal average values: CO 5-6 L/min, HR 60-100/min, SV 60-100 ml/beat, SaO2 95-100%, PaO_2 75-100 mmHg
DO_2 ≈ 1,000 mL/min

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Oxygen delivery & consumption

- **Tissue hypoxia (shock) is from either disruption of oxygen *delivery* (DO_2) or inability/maldistribution of oxygen *consumption* (VO_2) (or both)**

Normally, **DO_2** is >> **VO_2**; ($\frac{DO_2}{VO_2}$ = 5) by effective Oxygen extraction ratio (ERO_2) ≈ 20%
Thus, VO_2 is independent of DO_2 in most physiologic state

In critical oxygen delivery threshold **(DO_{2crit})** (≈ $\frac{DO_2}{VO_2}$ = 2) ERO_2 is maximally increased, VO_2 becomes ***linearly dependent*** of DO_2 & anerobic metabolism ensues

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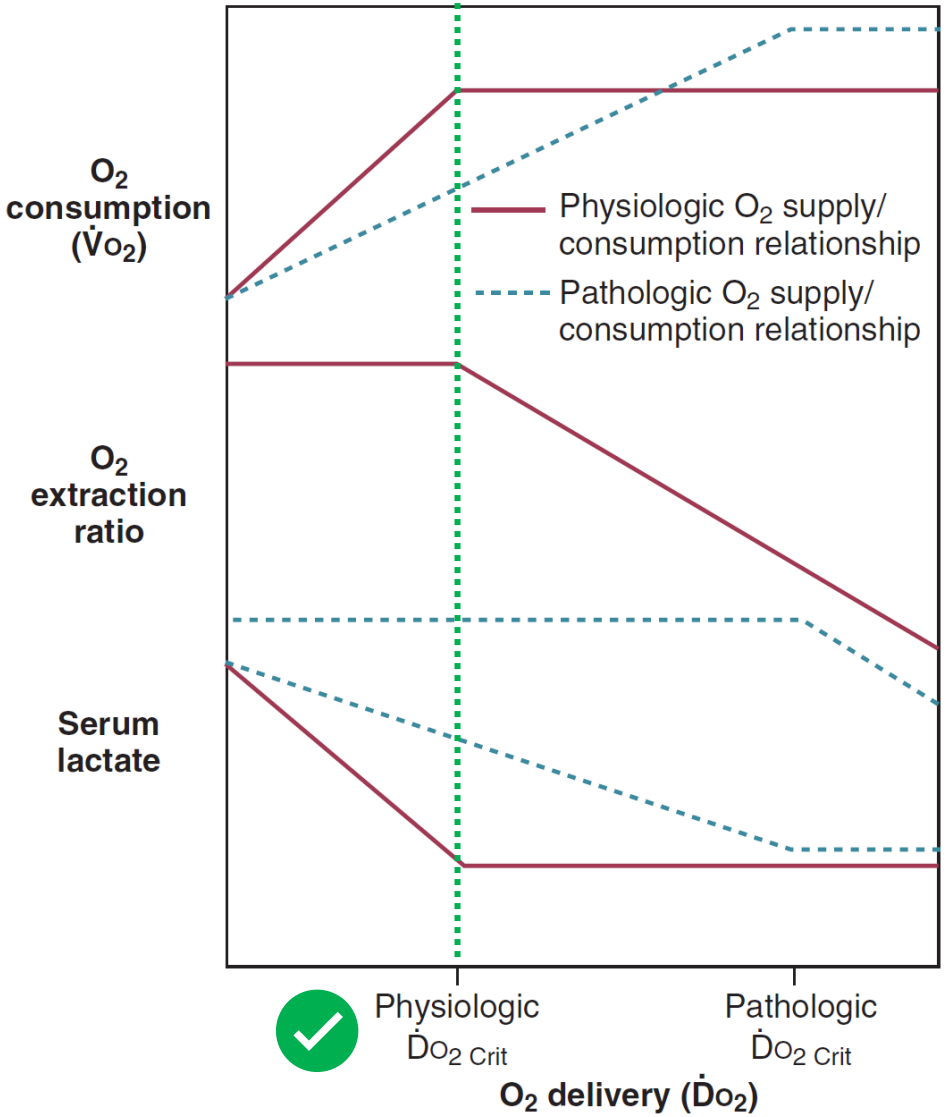
DO_2 & VO_2 relation



- DO_2 & VO_2 relation is a biphasic phase
- If DO_{2crit} is **reached** VO_2 decreased **linearly** with decreasing DO_2
- **Lactate** (marker of anaerobic metabolism) ↑ after the threshold
- **ERO_2** is maxed out at threshold

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Oxygen delivery & consumption

- Mixed venous oxygen saturation **(SvO_2)** measured from pulmonary artery (normally 65-75%)
- SvO_2 represent tissue **O_2 extraction**
- If ↑Tissue demand (consumption) or ↓ Delivery, **SvO_2↓ (<60%)**
- If **abnormal utilization** (extraction defect) or abnormal microvascular distribution, **SvO_2 ↑↑ (80%)**
- Central venous O2 saturation **($ScvO_2$)** > SvO2 ≈ 5% can be easily measured from SVC (central venous catheter)

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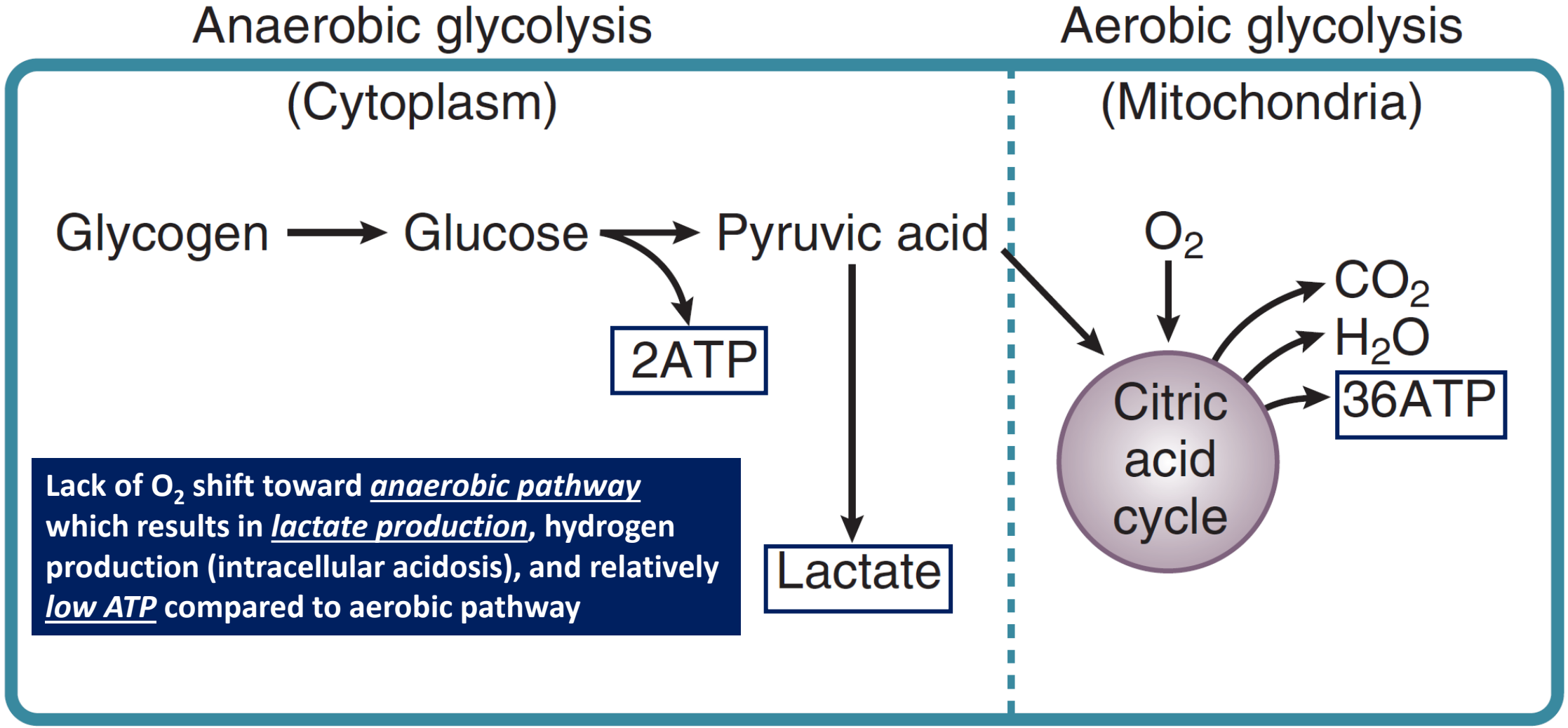
Basic cellular metabolism



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Lactate: marker of perfusion?

- Lactate is produced from **tissues** (muscle, etc.) and **cleared by liver** (mainly) and kidney
- **Liver** & kidney convert **lactate to glucose** (gluconeogenesis) then redistributed to tissue **(Cori cycle)**
- Normal, serum lactate (arterial) is ≈ 1 mmol/L
- Hyperlactemia (**>2** (1.5) mmol/L) can be interpretated as ***↑production*** or **↓clearance** or both
- **Lactic acidosis** = serum lactate **>4** mmol/L + metabolic **acidosis**

Hyperlactemia can be used as a marker for ↓ tissue perfusion

Brenner and Rector's the Kidney, 12th edition.
Kraut JA, Madias NE. N Engl J Med. 2014 Dec 11;371(24):2309-19.

Circulatory compensation

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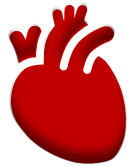
Shock

↓

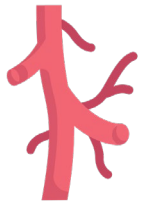
Chemoreceptor & Baroreceptor

↓

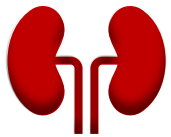
Through multiple mediators
Sympathetic, Angiotensin II, Cortisol, Vasopressin, etc.



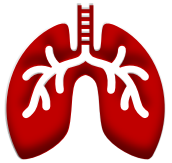
Contractility↑
Heart rate↑
(↑CO & SV)



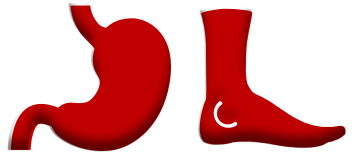
Vasoconstriction↑
(↑SVR)
Venoconstriction↑
(↑Venous return)



Na reabsorption↑
Water reabsorption↑
(↑ Venous return)



↑MV
(↑O_2↓CO_2)



↓Perfusion
(↑Vital organ perfusion)

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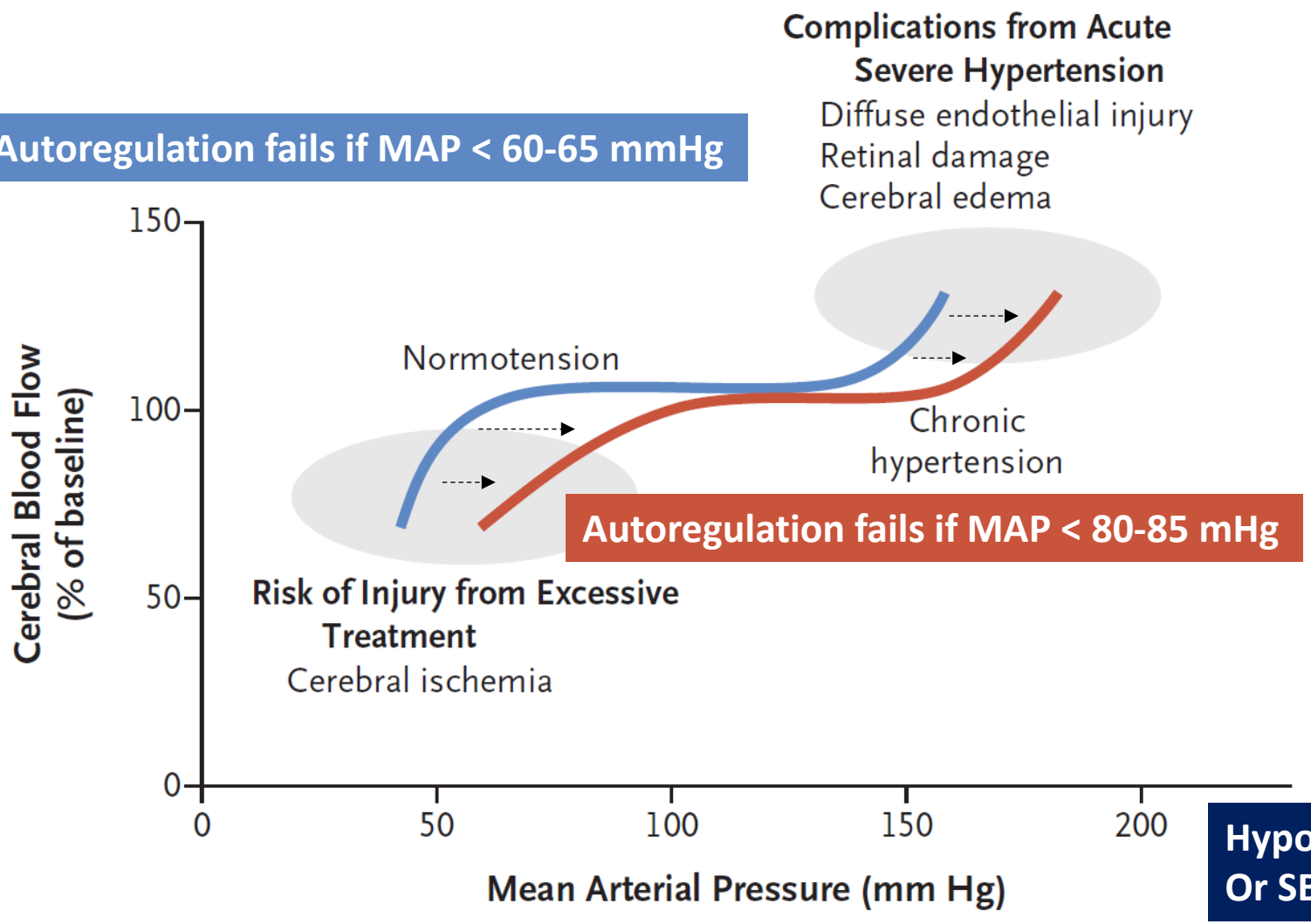
Cecconi M, et al. Intensive Care Med. 2014 Dec;40(12):1795-815.
Vincent JL, De Backer D. N Engl J Med. 2013 Oct 31;369(18):1726-34.

Autoregulation: local organ regulation



Autoregulation of blood flow

- Organs (i.e., **brain/kidney**) try to maintain **constant blood flow** (perfusion) despite wide range of systemic blood pressure
- ***Local*** vasodilation if ↓BP
- ***Local*** vasoconstriction if ↑BP

In ***chronic*** hypertension, graph shifts to the ***right*** to prevent organ injury from intermittent high BP but is ***more prone to hypoperfusion***

Hypotension = MAP <65 mmHg or <90/60 mmHg
Or SBP drop > 30% or ↓Δ40 mmHg

Peixoto AJ. N Engl J Med. 2019 Nov 7;381(19):1843-1852.
Cecconi M, et al. Intensive Care Med. 2014 Dec;40(12):1795-815.

Shock Classification

- Shock is a status/syndrome not a diagnosis
- From a pathophysiology view, shock can be classified into 4 groups (based on primary initiating cause which can guide diagnosis and treatment)

1. Hypovolemic shock
2. Cardiogenic shock
3. Obstructive (extra-cardiac) shock
4. Distributive shock

Vincent JL, De Backer D. N Engl J Med. 2013 Oct 31;369(18):1726-34.
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Hypovolemic shock

- **Defined by ↓CO from ↓Preload (seen by ↓CVP/JVP)**
- **Compensated by ↑SVR, ↑Contractility, ↑HR**
- **Primary insult = effective circulatory volume loss**
- If Volume loss = blood = hemorrhagic shock
- Depends on ***volume*** loss & ***rate*** of loss + baseline condition
- **Causes**
  - **Hemorrhage:** Massive GI bleeding, Retroperitoneal hemorrhage, Post-partum hemorrhage, Trauma
  - **Non-hemorrhage:** Severe diarrhea (cholera, etc.), Severe Burn/heatstroke, Dengue shock syndrome

CVP; central venous pressure, JVP; jugular (internal) venous pressure

Vincent JL, De Backer D. N Engl J Med. 2013 Oct 31;369(18):1726-34.
Harrison's Principles of Internal Medicine, 22nd Edition.
Cannon JW. N Engl J Med. 2018 Jan 25;378(4):370-379.

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Hemorrhagic shock: grading

Table 2. Classification of Hemorrhagic Shock.*

Shock Class	Blood Loss†	Heart Rate	Blood Pressure	Pulse Pressure	Respiratory Rate	Mental Status
	ml (%)	*beats/min*			*breaths/min*	
I	<750 (15)	<100	Normal	Normal	14–20	Slightly anxious
II	750–1500 (15–30)	100–120	Normal	Narrowed	20–30	Mildly anxious
III	1500–2000 (30–40)	120–140	Decreased	Narrowed	30–40	Anxious, confused
IV	>2000 (>40)	>140	Decreased	Narrowed	>35	Confused, lethargic

†Degree of blood loss (ml) is based on male weight 75 kg

Shock grade 1-2, BP might be appearing normal (cryptic shock) but orthostatic hypotension can be demonstrated

Cannon JW. N Engl J Med. 2018 Jan 25;378(4):370-379.

Cardiogenic shock

- **Definition: cardiac disorder that results in both clinical and biochemical evidence of sustained tissue hypoperfusion**
- **SBP <90 mmHg for >30 minutes** or a need for inotropes, vasopressors, or mechanical circulatory support to maintain BP ≥90 mmHg **and evidence of systemic hypoperfusion** (lactate >2 mmol/L) **+ Cardiac index ≤2.2** L/min/m^2 (↓CO)
- **Typically: ↑SVR**, ↑HR (if no bradyarrhythmia), ↑CVP, ↑PCWP
- Example: **Acute MI** (>40% of myocardium), Acute severe **valvular** insufficiency, Fulminant myocarditis, Severe **arrhythmia**
- Diagnosis by **clinical, echocardiogram, ECG, cardiac enzymes**

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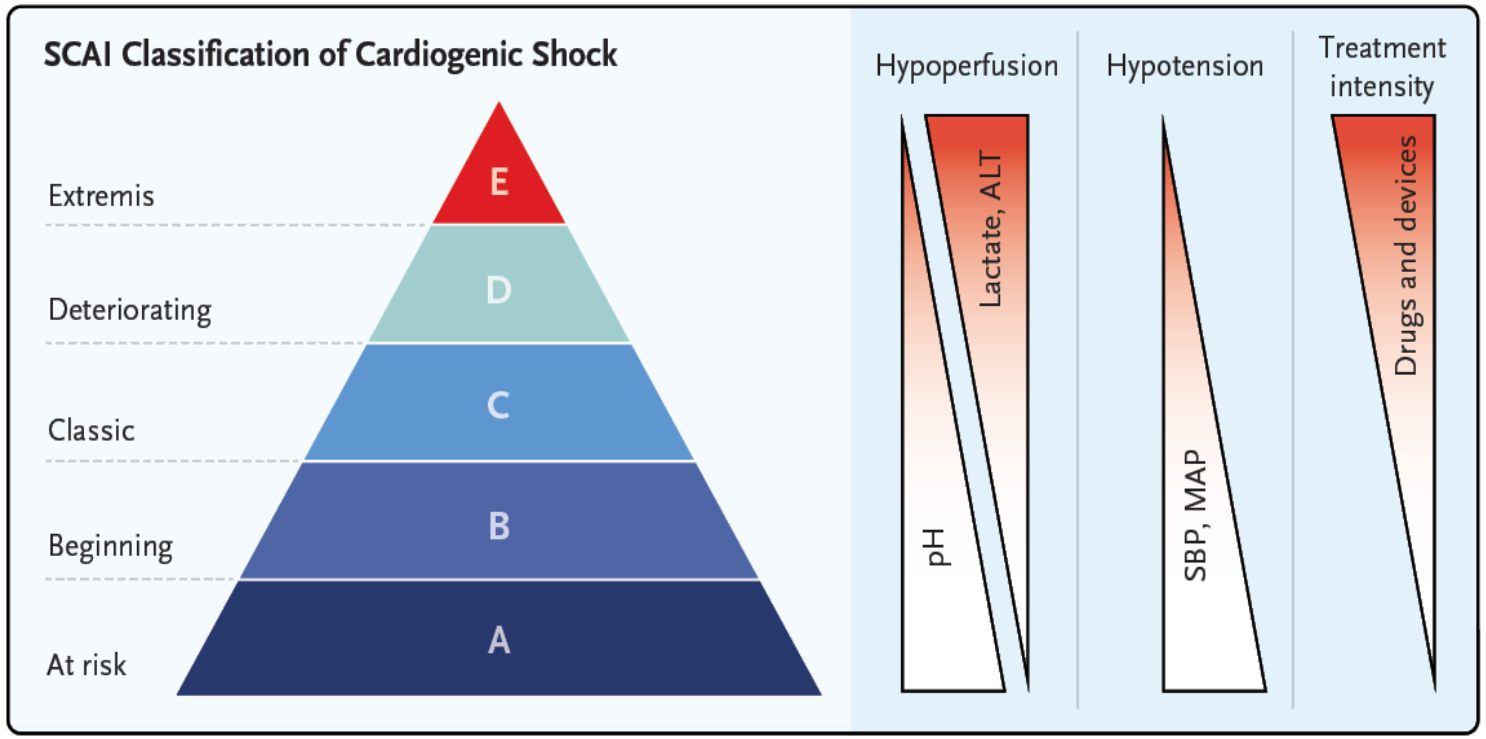
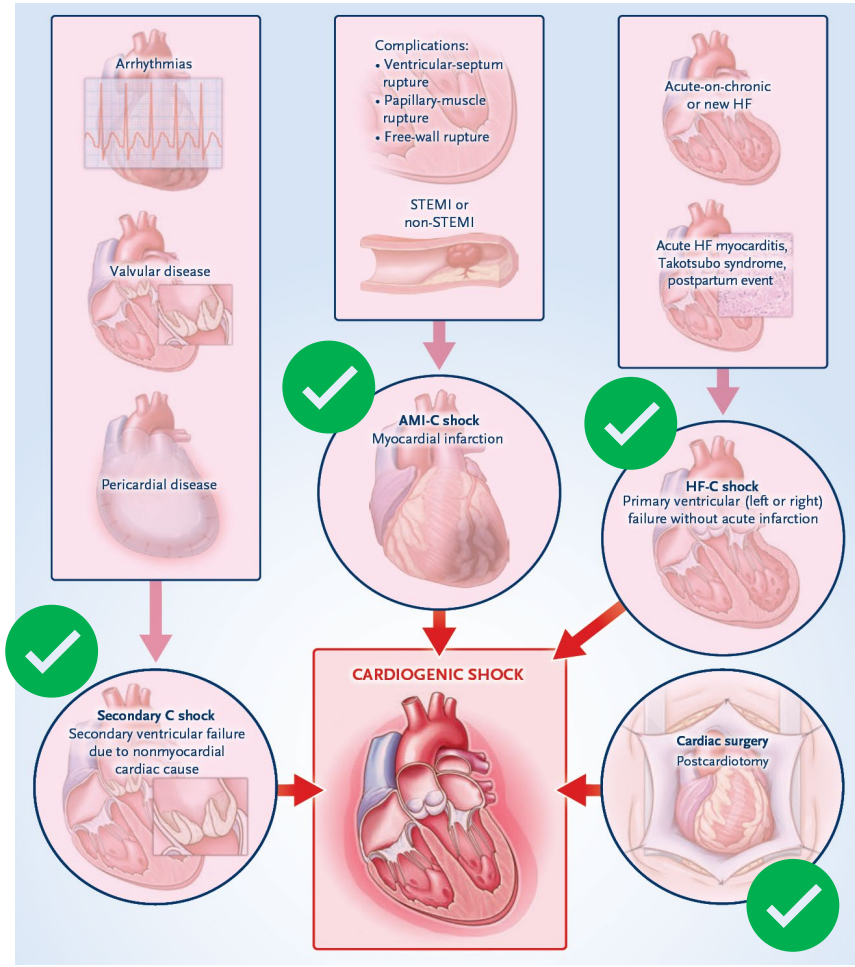
Tehrani BN, et al. JACC Heart Fail. 2020 Nov;8(11):879-891.
Thiele H, Hassager C. N Engl J Med. 2026 Jan 1;394(1):62-77.

Cardiogenic shock grading/causes



At risk = no clinical but at risk for development
Beginning = normal BP (relatively↓ but tachycardia)
Classic = hypotension + tissue hypoperfusion (lactate >2 mmol/L)
Deteriorating = Escalating vasopressor/mechanical device
Extremis = lactate >8 mmol/L + maximum hemodynamic support



4 main causes

Tehrani BN, et al. JACC Heart Fail. 2020 Nov;8(11):879-891.
Thiele H, Hassager C. N Engl J Med. 2026 Jan 1;394(1):62-77.
Waksman R, et al. Circulation. 2023 Oct 3;148(14):1113-1126.

Obstructive shock

- ***Extracardiac*** **cause limiting/obstructing flow in cardiovascular circuit**
- **Divided into**
  - **1) ↓Diastolic filling (↓preload):** tension pneumothorax, cardiac tamponade
  - **2) ↓Systolic contraction (↑afterload):** massive pulmonary embolism, aortic dissection
- **Profile: ↓CO, ↑SVR, ↑CVP, PCWP varied**

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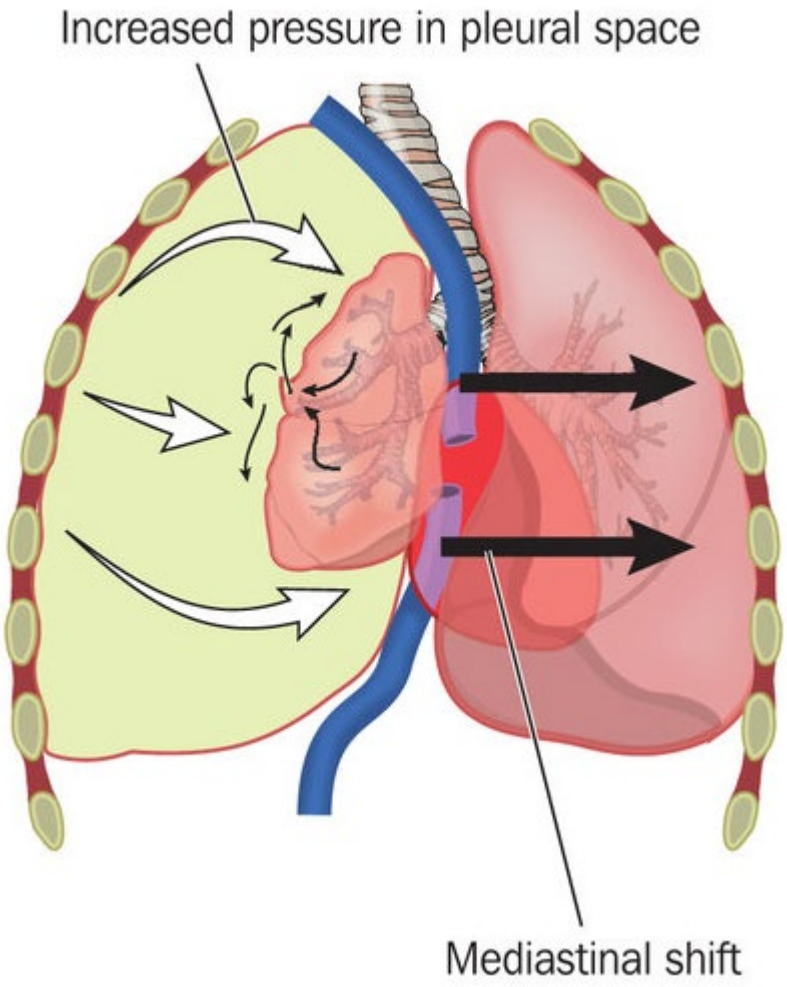
Tension pneumothorax



- **Can be spontaneous/traumatic**
- **Large air in pleural space pressures on mediastinum affecting *large vein* + heart**
- **↓Venous return but JVP↑**
- **Hyper-aeration (↑hyper-resonance on percussion)**
- **Clinical diagnosis** (confirm by CXR, lung ultrasound)

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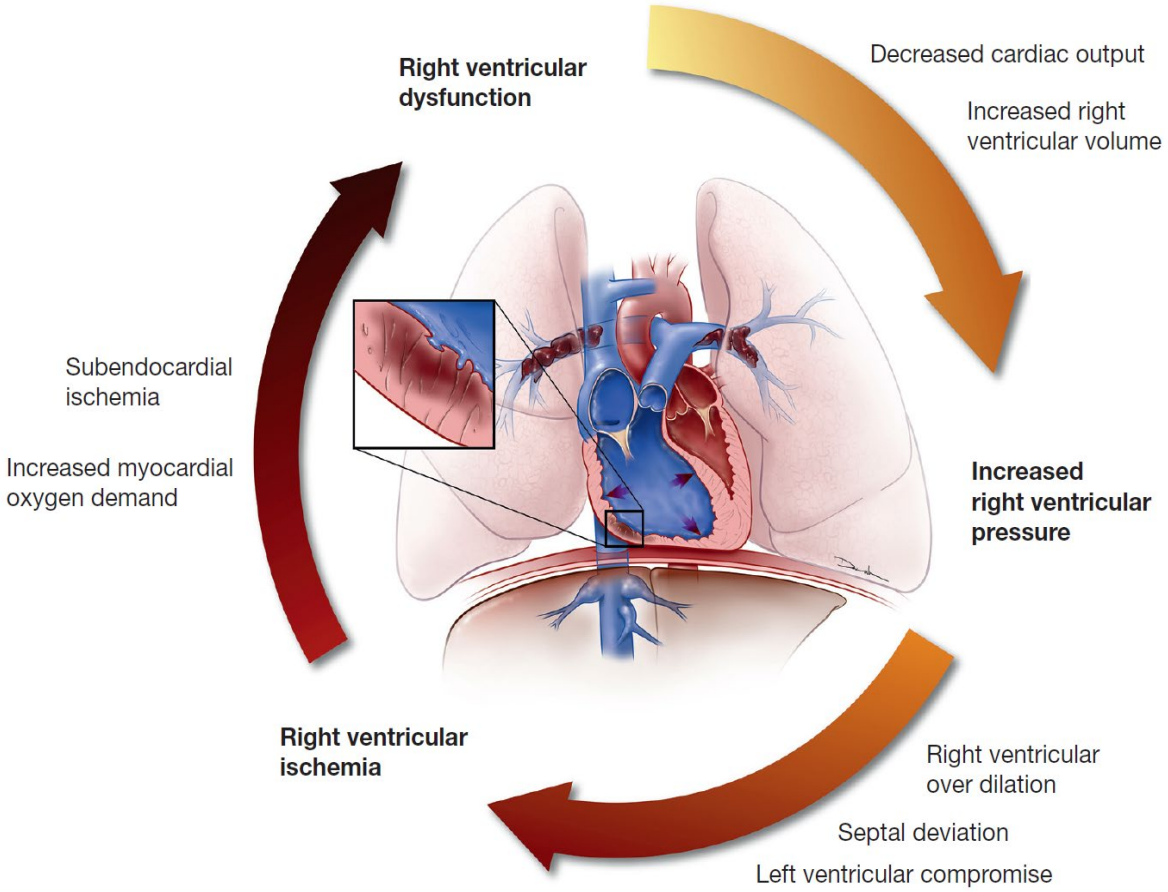
Massive pulmonary embolism

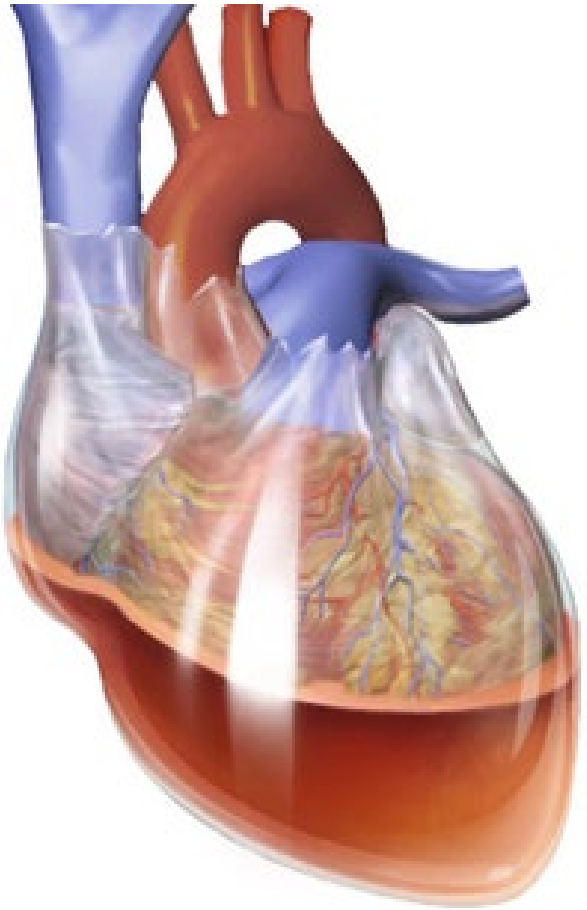


- **Abrupt ↑of pulmonary resistance & pressure (from large clot + reflex)**
- **RV failure + distend compress left heart**
- **Ischemic process of RV**
- **↓CO, ↑SVR, ↑CVP, ↓PCWP**
- **Diagnosed by echocardiogram** (if massive), CTPA

CTPA; Computed Tomography Pulmonary Angiography

McGuire WC, et al. Chest. 2024 Dec;166(6):1532-1545.

Cardiac tamponade



- Depend on **rate** and volume of **pericardial effusion**
- **Equalization + increase of left & right end diastolic pressure** (equal to increased pericardial pressure)
- ↓Cardiac cavity, **↓Venous return**
- **Muffled heart sound, pulsus paradoxus, ↑JVP**
- Causes: trauma, TB, malignancy
- Diagnosis by **echocardiogram**

Wang TKM, et al. J Am Coll Cardiol. 2025 Dec 23;86(25):2691-2719.

Distributive shock (a.k.a. hyperdynamic shock)

- **Loss of *systemic* peripheral vascular resistance (SVR ↓)**
- **Cardiac output (SV & HR)↑** (SV maybe↓ initially)
- CVP & PCWP ↔ or ↓initially
- O_2 delivery may look adequate (on a macro-scale) but microvascular is dysfunctional (↓O_2 extraction, AV-shunt, mal-distribution) result in **hypoxia despite adequate cardiac output**
- $ScvO_2$ (marker of tissue O_2 extraction) is **intact or ↑** in contrast to other types of shock
- Prototype: **septic shock, anaphylactic shock**, adrenal shock, neurogenic shock

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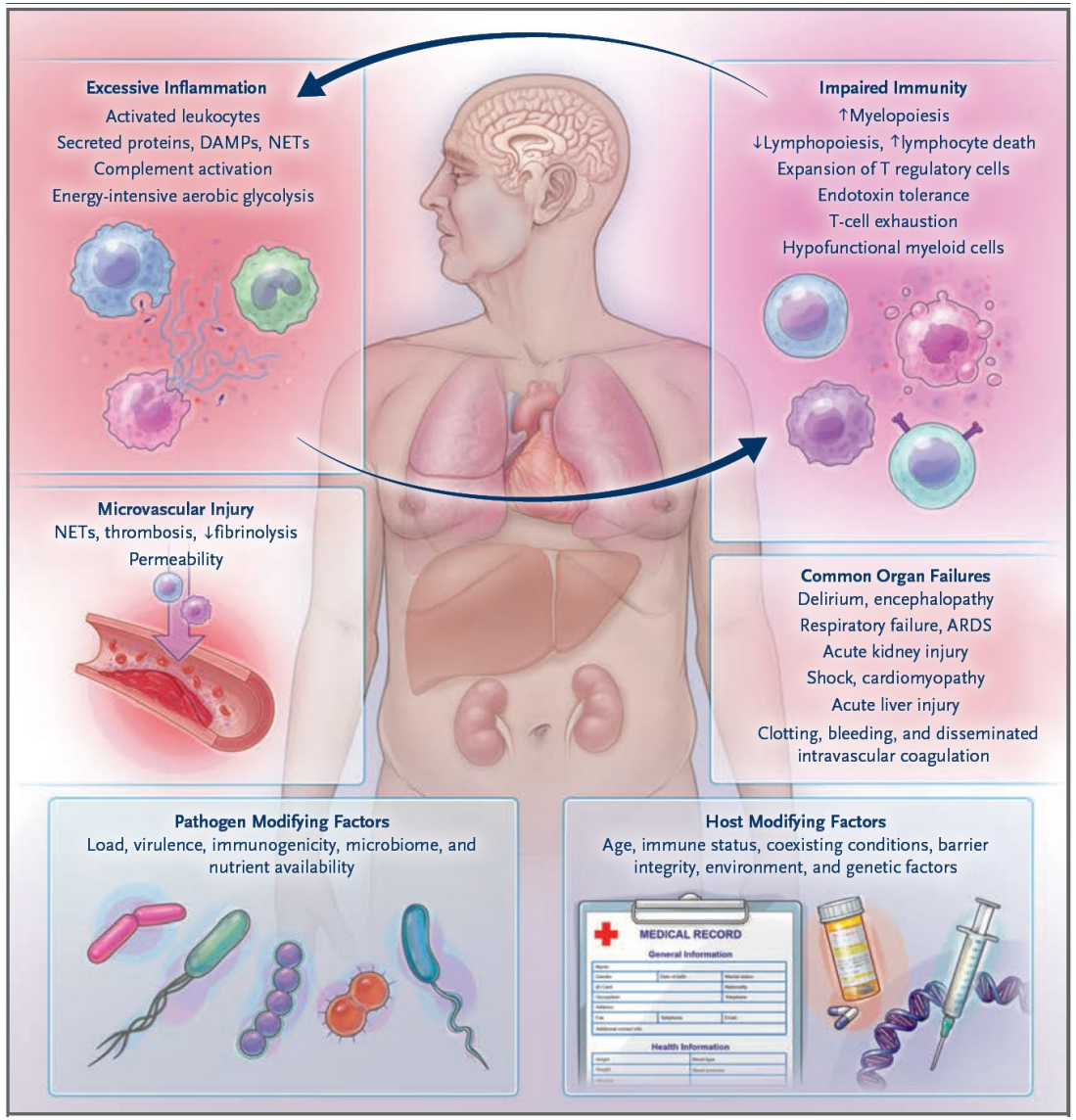
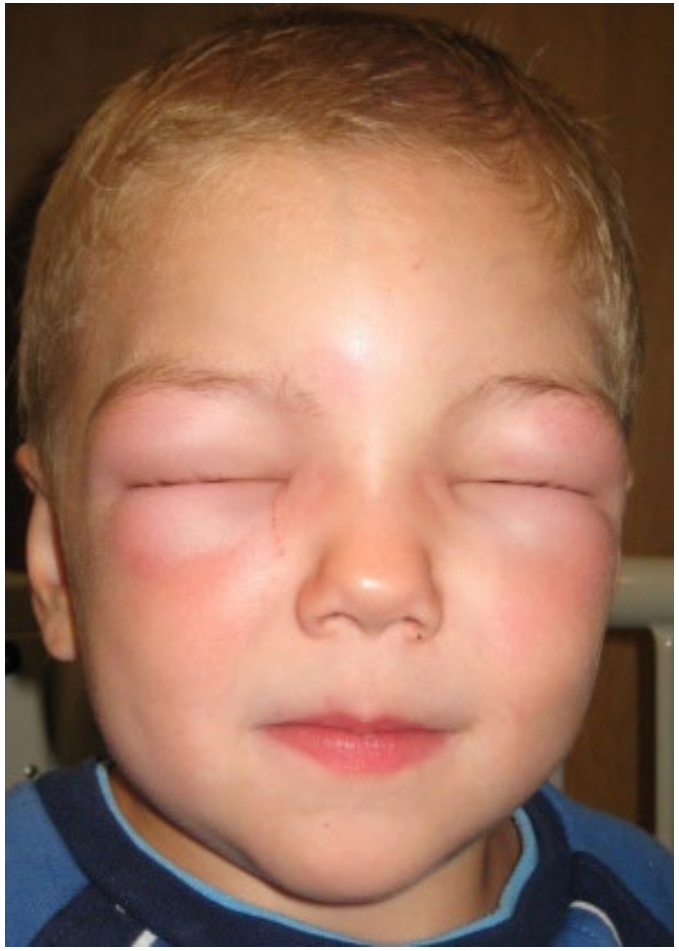
Septic shock: Sepsis-3 criteria



- **Sepsis** is defined as "life-threatening ***organ dysfunction*** caused by a ***dysregulated*** host response to ***infection***"
- **Septic shock** = Sepsis with ***persisting*** **hypotension** requiring ***vasopressors*** to maintain MAP ≥65 mmHg and having ***lactate*** **>2 mmol/L** despite ***adequate volume*** resuscitation

Meyer NJ, Prescott HC. N Engl J Med. 2024 Dec 5;391(22):2133-2146.
The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016 Feb 23;315(8):801-10.

Anaphylactic shock



- **Mast cell & basophil degranulation of** mediators **(histamine**, leukotrienes, prostaglandins, etc.) **activated by IgE & *allergens*** (direct activation = anaphylactoid)
- **Angioedema, urticaria, GI** (N/V, abdominal pain, diarrhea)**, bronchospasm immediately** after contact with allergens
- **Clinical diagnosis** (±confirm with elevated serum tryptase)

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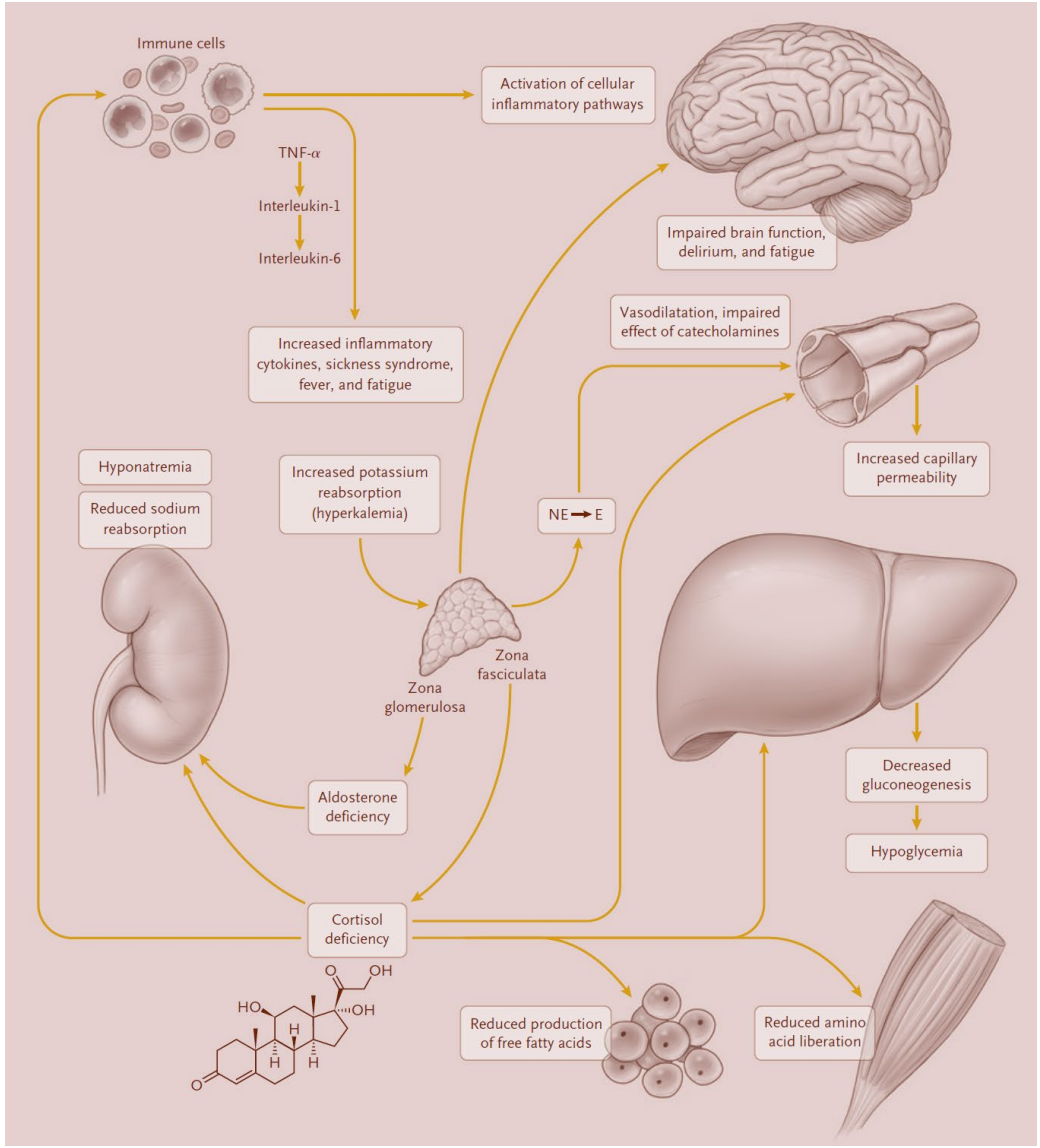
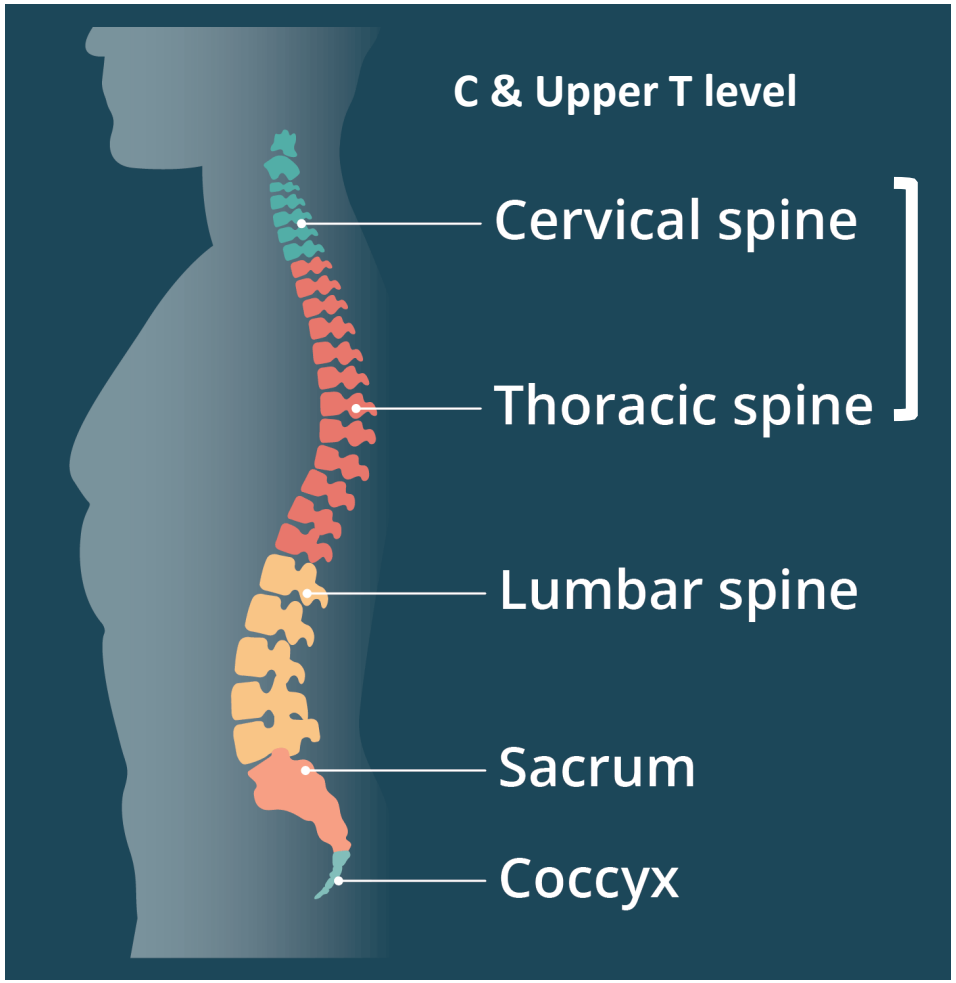
Adrenal shock (adrenal crisis)



- **Lack of cortisol during acute stress (infection)**
- Fatigue, fever, **hypoglycemia**, **shock unresponsive to vasopressor**, hypercalcemia, hyponatremia, hyperkalemia
- Causes: Addison disease, infection, malignancy, abrupt withdraw of chronic steroid use
- Diagnosis by **stress cortisol level**

Rushworth RL, et al. N Engl J Med. 2019 Aug 29;381(9):852-861.

Neurogenic shock



- Spinal cord injury in **C & Upper T level**
- **↓Sympathetic flow &** unopposed parasympathic
- Typical with **relative bradycardia** (HR <100/min)
- **Neurological deficit**
- Trauma, myelitis, anesthesia
- Clinical diagnosis

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Mixed (combined) shock

- **Common scenario in clinical practice due to**

1. Phase of shock & treatment (early distributive shock has component of hypovolemic shock but after adequate volume, distributive shock profile dominates or in late/severe sepsis cardiac suppression is common)

2. Concurrent illness (Septic shock may precipitate acute MI with cardiogenic shock or relative adrenal insufficiency)

3. Baseline/Underlying disease (End-stage cardiac disease is prone to stress which may result in combination profile)

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Shock classification summary

Characteristics	Hypovolemic	Cardiogenic	Obstructive	Distributive
Cardiac output	↓	↓	↓	↑
Contractility	↑	↓	↓	↑
Preload	↓	↑	↓	↔, ↓ (early)*
CVP	↓	↑	↑	↔, ↓ (early)*
PCWP	↓	↑	↑(Tamponade), ↓(PE, Pulmonary HT)	↔, ↓ (early)*
SVR	↑	↑	↑	↓
$ScvO_2$	↓	↓ (except VSD)	↓	↔, ↑
CRT	↑	↑	↑	↔
Cold extremities	↑	↑	↑	↔
Pulse pressure	↓	↓	↓	↑

CRT; capillary refill time, VSD; ventricular septal defect. *In early phase of distributive shock, hemodynamic profile is similar to hypovolemia.

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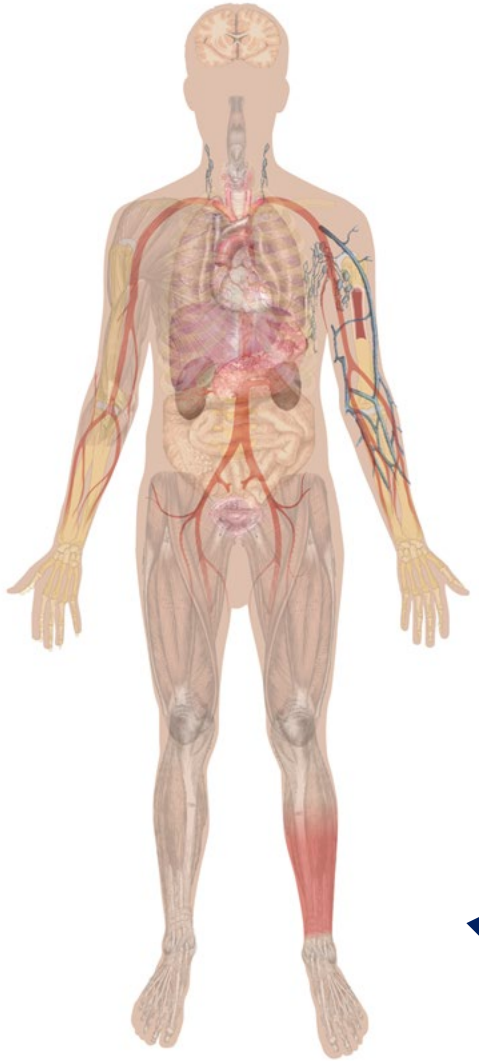
Clinical features: signs of shock



- Agitation, confusion, delirium, drowsiness
- Hypotension, orthostasis, tachycardia, tachypnea, weak pulse
- Oliguria (urine output <500 mL/d or <0.5 mL/kg/hr)
- Cold & clammy skin, delayed capillary refill (except distributive shock)

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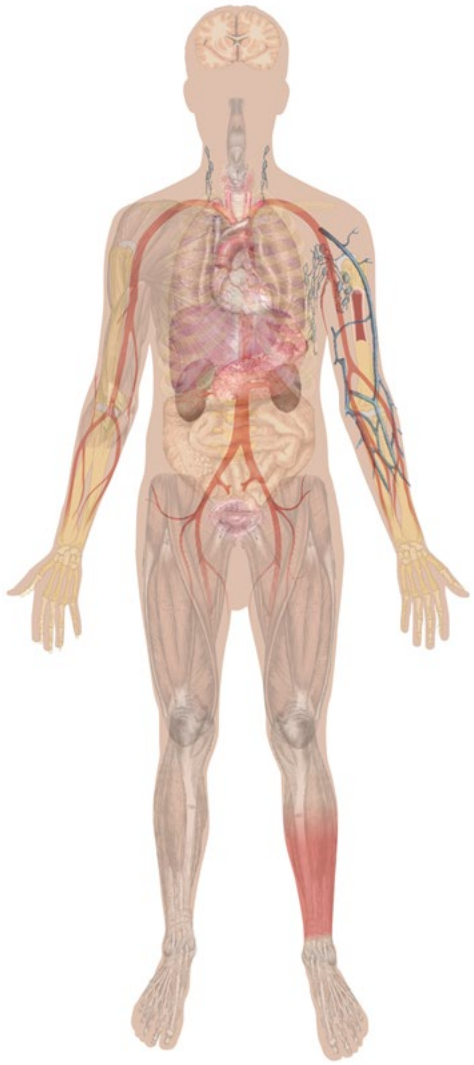
Clinical features: causes of shock



Extracardiac obstruction: lung sound, pulsus paradoxus
Anaphylaxis: urticaria, wheezing, angioedema

Cardiac diseases: PMI shift, murmur, S3 gallop, engorge JVP, etc.

Source of infection: pneumonia, UTI, skin & soft tissue infection, etc.

Source of bleeding, pallor, signs of volume depletion

Vincent JL, De Backer D. N Engl J Med. 2013 Oct 31;369(18):1726-34.
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Investigation: shock assessment

Renal function: BUN, SCr, electrolytes, urine sp.gr., urine Na

Tissue perfusion & O_2 extraction: serum lactate, $ScvO_2$, invasive BP monitoring

Ventilation & oxygenation: O_2 saturation, ABG

O_2 capacity & severity: Hemoglobin, Platelet count, Coagulogram

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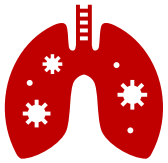
Investigation: causes of shock



Obstructive shock: CXR, CTPA, Echocardiogram, ECG

Cardiogenic shock: ECG, Echocardiogram, cardiac enzymes

Distributive shock: Hemoculture & specimen from suspected source, cortisol level, serum tryptase

Hypovolemic shock: Hemoglobin/hematocrit

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Management: principal

1) Recognize shock early (normotensive is possible)
2) Assess for type of shock present
3) Treatment simultaneous with the evaluation into the etiology
4) Multidisciplinary team
5) Aim of therapy is to restore oxygen delivery
6) Lifesaving interventions specific to causes of shock

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Specific management

Causes	Treatment
Septic shock	Broad-spectrum **Anti-biotics within 1 hr** (as soon as possible) + **Control source** of infection
Anaphylactic shock	**Adrenaline IM**, Systemic glucocorticoids, Anti-histamine
Adrenal shock	High-dose **systemic corticosteroids**
Cardiogenic shock	**Revascularization (if acute MI)**, Cardioversion or defibrillation if tachyarrhythmia, Pacemaker if bradyarrhythmia
Hemorrhagic shock	**Control source of bleeding** + blood transfusion
Pulmonary embolism	**Thrombolytics** or surgical embolectomy
Cardiac tamponade	**Pericardiocentesis**
Tension pneumothorax	**Needle thoracostomy** then intercostal drain (ICD) insertion

Vincent JL, De Backer D. N Engl J Med. 2013 Oct 31;369(18):1726-34.
Harrison's Principles of Internal Medicine, 22nd Edition.
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General/Supportive management

- **A-B-C stabilization (simultaneously)**
  - **Airway:** Airway patency
  - **Breathing:** O_2 support (92-95%), Intubation if respiratory failure
  - **Circulation**

- **Set Goal BP (macro-circulation)**
- **IV resuscitation**
- **Vasopressor or inotrope ± adjunct**
- **Monitor + achieve microcirculation**

Vincent JL, De Backer D. N Engl J Med. 2013 Oct 31;369(18):1726-34.
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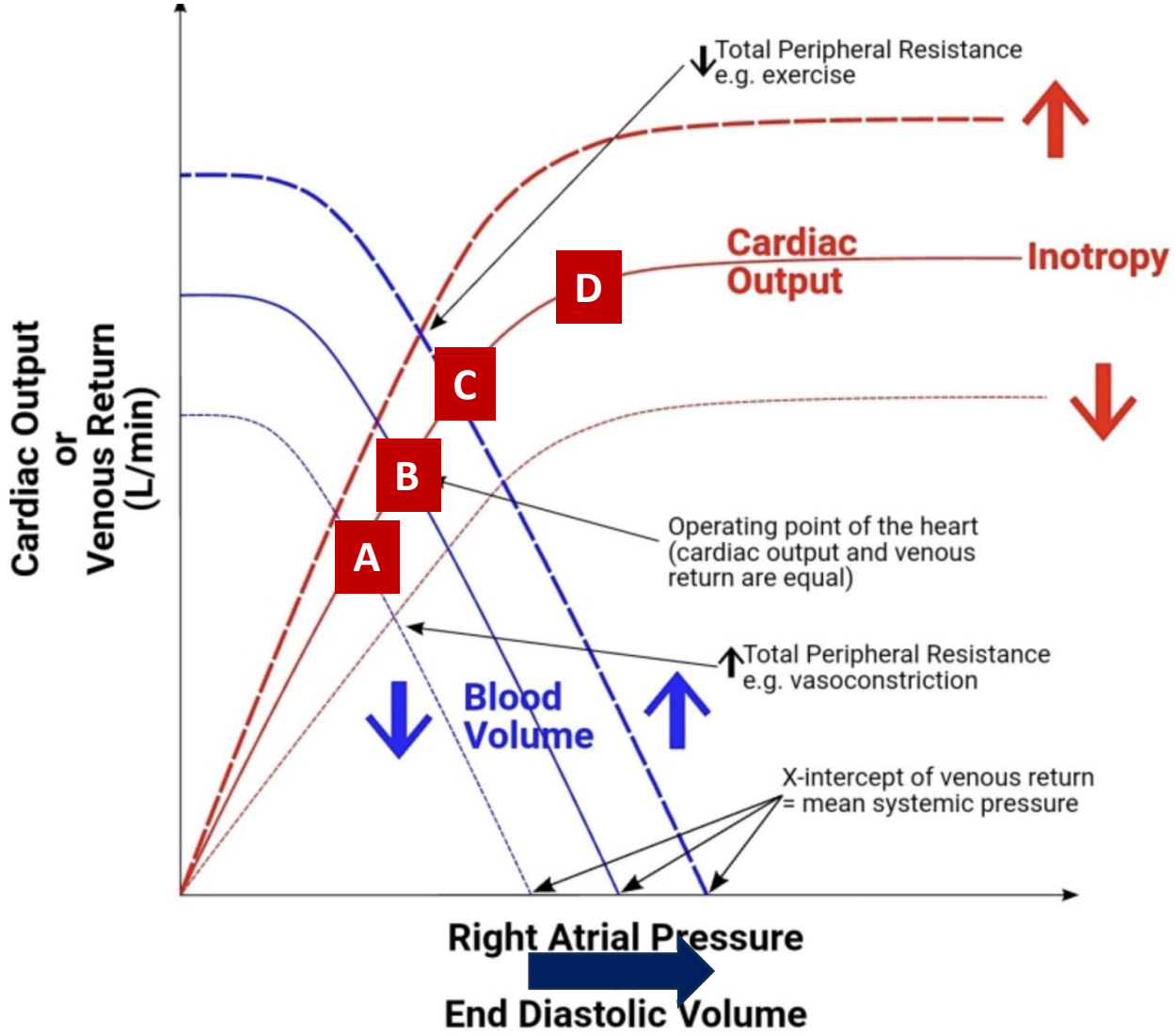
Macro-circulation goal: BP

- **Generally, goal mean arterial pressure *(MAP)* is set to be > *65 mmHg***
- **In some settings, goal BP *maybe* different**
  - Chronic hypertension: MAP 80-85 mmHg
  - Elderly (>65 y): MAP 60-65 mmHg (in septic shock)
  - Trauma (before bleeding control): SBP 80-90 mmHg, MAP 50-60 mmHg
  - Traumatic brain injury: SBP ≥100-110 mmHg
- **Reaching BP goal should be prioritized first (ASAP!)**

Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2026.
Asfar P, et al. N Engl J Med. 2014 Apr 24;370(17):1583-93.
Ramasamy A. Injury. 2026 Apr;57(4):113079.
Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition.

Frank-Starling curve

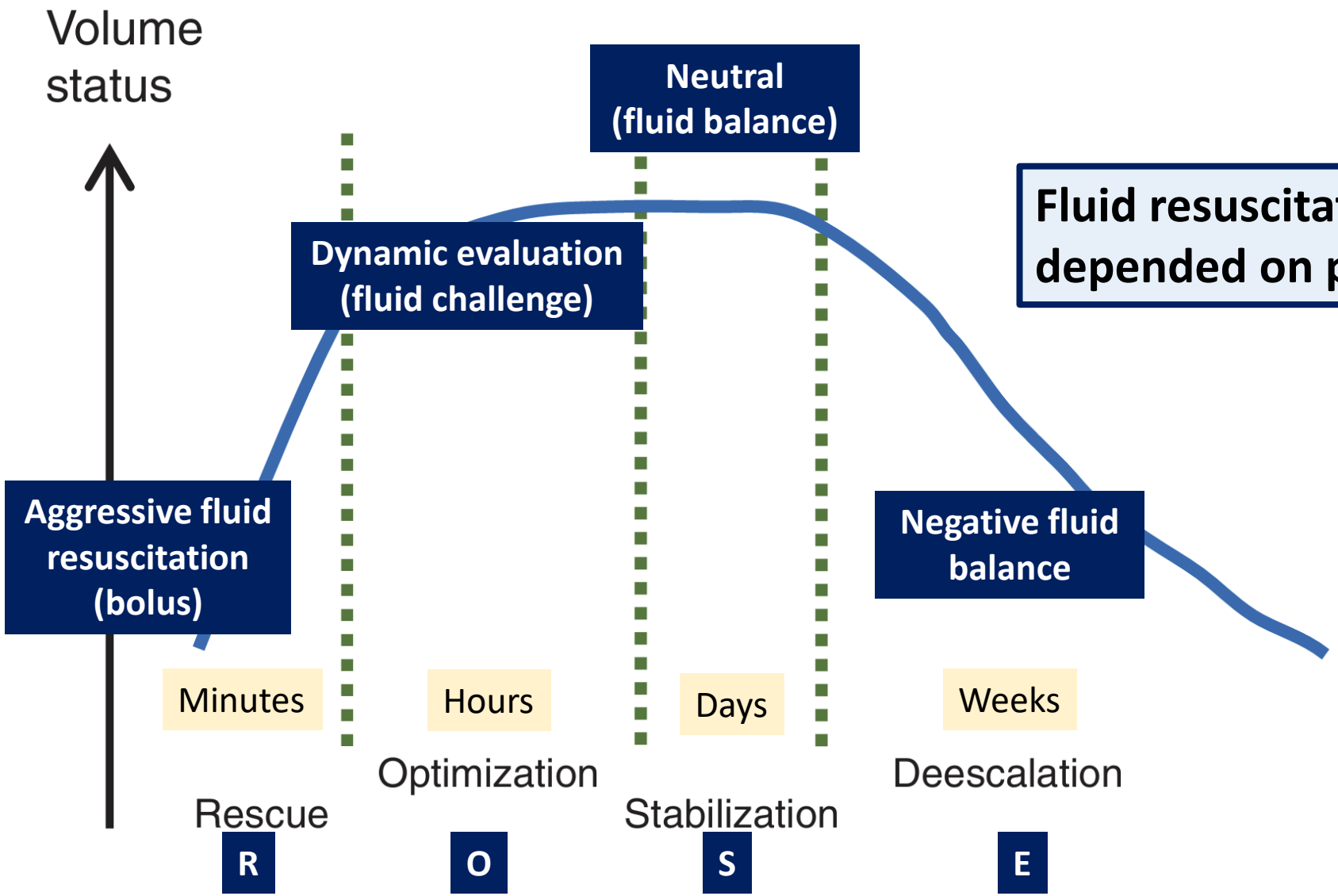


By increasing end diastolic volume **(preload)**; x-axis
Cardiac output (red curve) is increased **(A→B→C)** until plateau is reached (no increase of cardiac output despite increase preload; **point D**)

Goal of fluid resuscitation is to increase cardiac output based on "Frank-Starling curve" at the steep part (not plateau part)

Delicce AV, Makaryus AN. [Updated 2023 Jan 30]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan-.

Fluid resuscitation phases



Fluid resuscitation is depended on phases of illness

Hoste EA, et al. Br J Anaesth. 2014 Nov;113(5):740-7.

Initial fluid resuscitation: amount

- **IV bolus: prototype in septic shock = 30 mL/kg within 3 h**
- **≈300-500 mL in 20-30 min** (large bore needle)
- **But amount can be varied based on causes or patient status** (↓ in trauma prior to stop bleeding, ↑severe hypovolemia from other causes, ↓Obstructive shock)
- **Avoid or very closed observe in cardiogenic shock**

Mayerhöfer T, et al. Nephrol Dial Transplant. 2023 Jun 30;38(7):1603-1612.
European Society of Intensive Care Medicine (ESICM) 2025 clinical practice guideline on fluid therapy in adult critically ill patients.
Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2026.

Initial fluid resuscitation: type

- ***Isotonic*** (equal to extracellular fluid tonicity ≈ 280+ mmol/L) ***crystalloids***
- **Except hemorrhagic shock: Blood transfusion = 1st line**
- **Crystalloids**
  - **Normal saline (0.9%NaCl):** preferred in alkalosis, traumatic brain injury
    - Na 154 mEq/L, Cl 154 mEq/L; pH 5.5
  - **Balanced salt solution (Ringer lactate, Acetar, etc.):** preferred in acidosis, sepsis, hyperchloremia
    - Similar to plasma (pH, chloride, K, Ca/Mg)

Mayerhöfer T, et al. Nephrol Dial Transplant. 2023 Jun 30;38(7):1603-1612.
European Society of Intensive Care Medicine (ESICM) 2025 clinical practice guideline on fluid therapy in adult critically ill patients.
Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2026.

Vasopressor/Inotropes

Drug	Dose	CARDIAC		PERIPHERAL VASCULATURE			Typical Clinical Use
		Heart Rate	Contractility	Vasoconstriction	Vasodilation	Dopaminergic	
Dopamine	1–4 μg/kg/min 5–10 μg/kg/min 11–20 μg/kg/min	1+ 2+ 2+	1–2+ 2+ 2+	0 1–2+ 2–3+	1+ 1+ 1+	4+ 4+ 4+	All shock
Norepinephrine	0.01–0.3 μg/kg/min	2+	2+	4+	0	0	Refractory shock
Dobutamine	1–20 μg/kg/min	1–2+	3+	1+	2+	0	CHF; cardiogenic, obstructive and septic shock
Dopexamine[b]	0.5–6 μg/kg/min	2+	1+	0	3–4+	4+	CHF; cardiogenic shock
Epinephrine	0.05–0.2 μg/kg/min	4+	4+	4+	3+	0	Refractory shock or anaphylactic shock
Phenylephrine	0.1–1 μg/kg/min	0	1+	4+	0	0	Neurogenic or septic shock
Isoproterenol	1–8 μg/min	4+	4+	0	4+	0	Cardiogenic shock (bradyarrhythmia), torsades de pointes, ventricular tachycardia
Vasopressin	0.02–0.04 U/min	0	0	4+	0	0	Vasodilatory (e.g., septic) shock
Milrinone	37.5–75 μg/kg bolus over 10 min; 0.375–0.75 μg/kg/min infusion	1+	3+	0	2+	0	CHF; cardiogenic shock

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Vasopressor/Inotropes: choosing

Conditions	Vasopressors/inotropes
Septic shock	**1st line = Norepinephrine**, 2nd Vasopressin, 3rd Epinephrine
Cardiogenic shock	**Early: Norepinephrine** then dobutamine
Anaphylactic shock	**Epinephrine**
Neurogenic shock	Mostly favor **Norepinephrine**
Hypovolemic shock	**Avoid** unless profound hypotension then rapid wean off

Common (shared) adverse event

Tachyarrhythmia, limb ischemia/gangrene

Unique adverse event

Epinephrine: lactic acidosis

Dobutamine: transient hypotension

Dopamine: prone to arrhythmia > Norepinephrine

Taman M, et al. J Clin Med. 2025 Jan 29;14(3):902.
Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2026.
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Thiele H, Hassager C. N Engl J Med. 2026 Jan 1;394(1):62-77.

Goal: microcirculation/perfusion

Clinical features

↓Capillary refill time, ↓Cold & Clammy skin
↓Mottling skin

Urine output >0.5 mL/kg/h

Improvement in level of consciousness

Laboratory investigation

Serum lactate normalization

$ScvO_2$ >70%

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Adjunctive treatment

Conditions	Adjunctive treatment
Septic shock	**Hydrocortisone** (moderate dose of Norepinephrine)
Cardiogenic shock	**Mechanical Circulatory Support Devices Veno-arterial Extracorporeal Membrane Oxygenation** (VA-ECMO)
Anaphylactic shock	**Systemic corticosteroids**

In refractory shock: correct acidosis, hypocalcemia, corticosteroid insuf.

In uncorrected microcirculation: look for severe anemia and hypoxemia (adequate BP achieved)

Thiele H, Hassager C. N Engl J Med. 2026 Jan 1;394(1):62-77.
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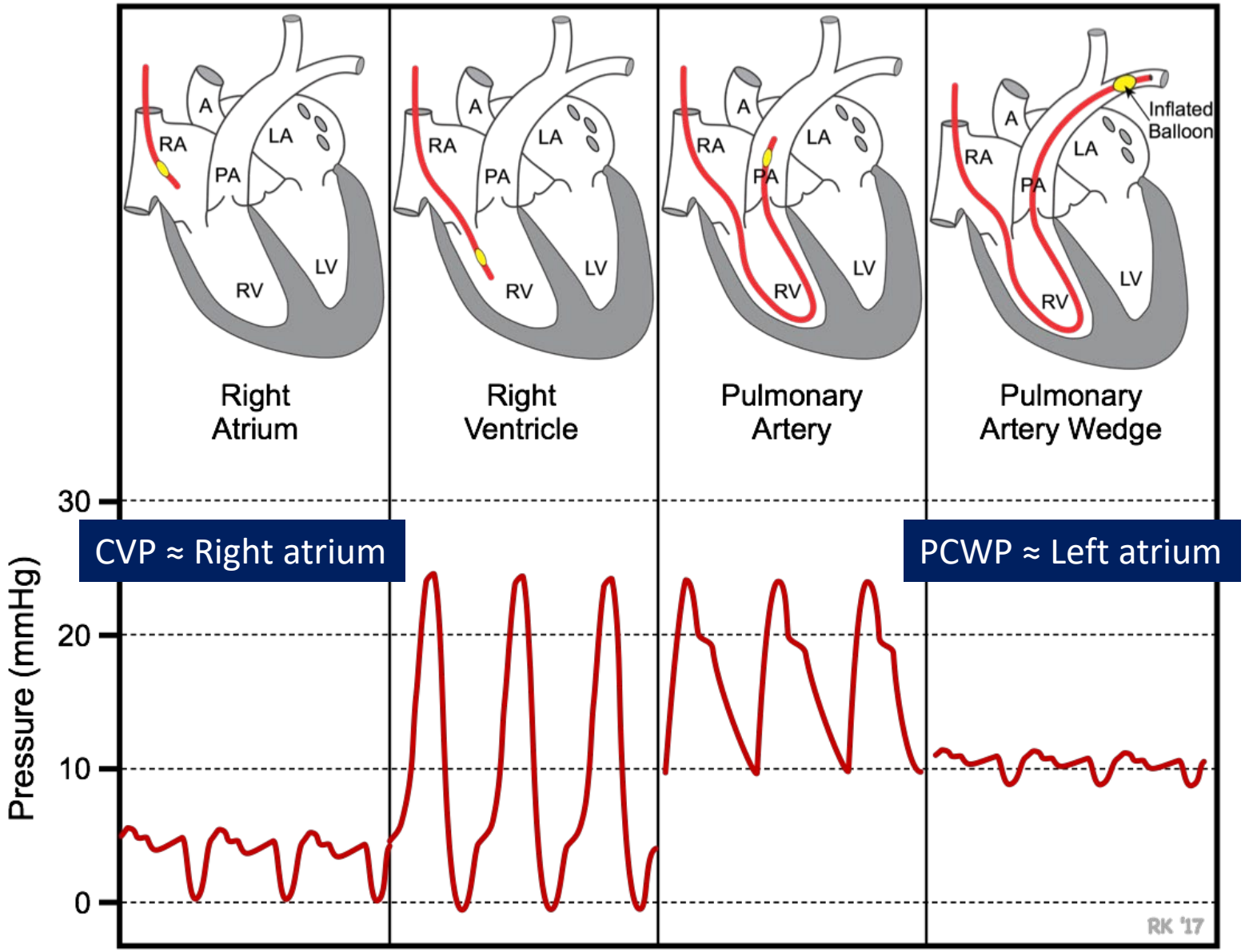
Take home message

- Shock is a life-threatening condition from circulatory failure results in inadequate O_2 supplied to tissue
- Recognition of shock, identification of etiologies, and ***simultaneous*** treatment of specific cause and hemodynamic stabilization are crucial
- Closed monitoring of both clinical & laboratory parameters are necessary
- IV resuscitation & vasopressors are the main tools

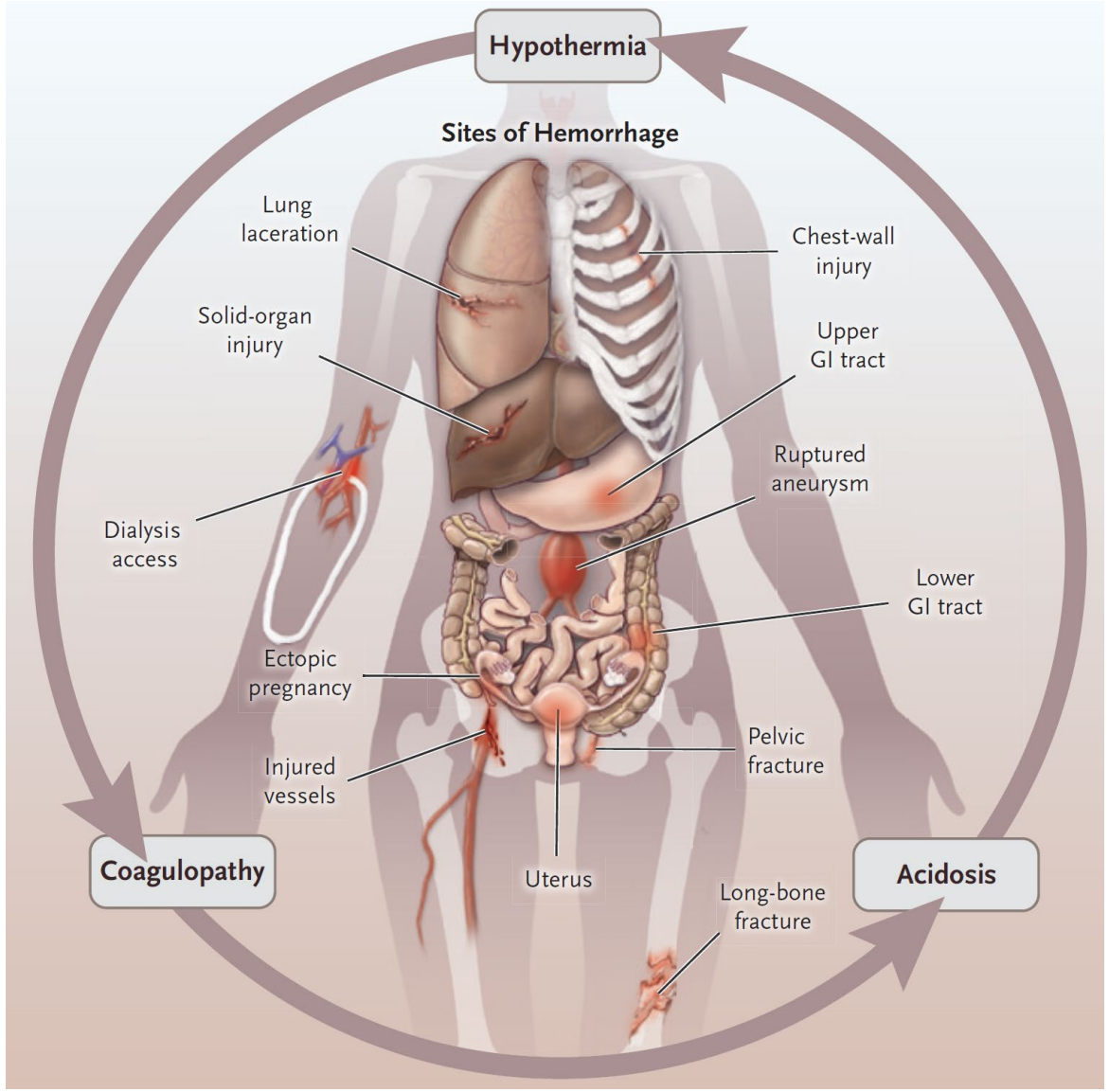
Thank you for your attention

Pulmonary artery catheter: surrogates



Bootsma IT, et al. J Clin Monit Comput. 2022 Feb;36(1):17-31.

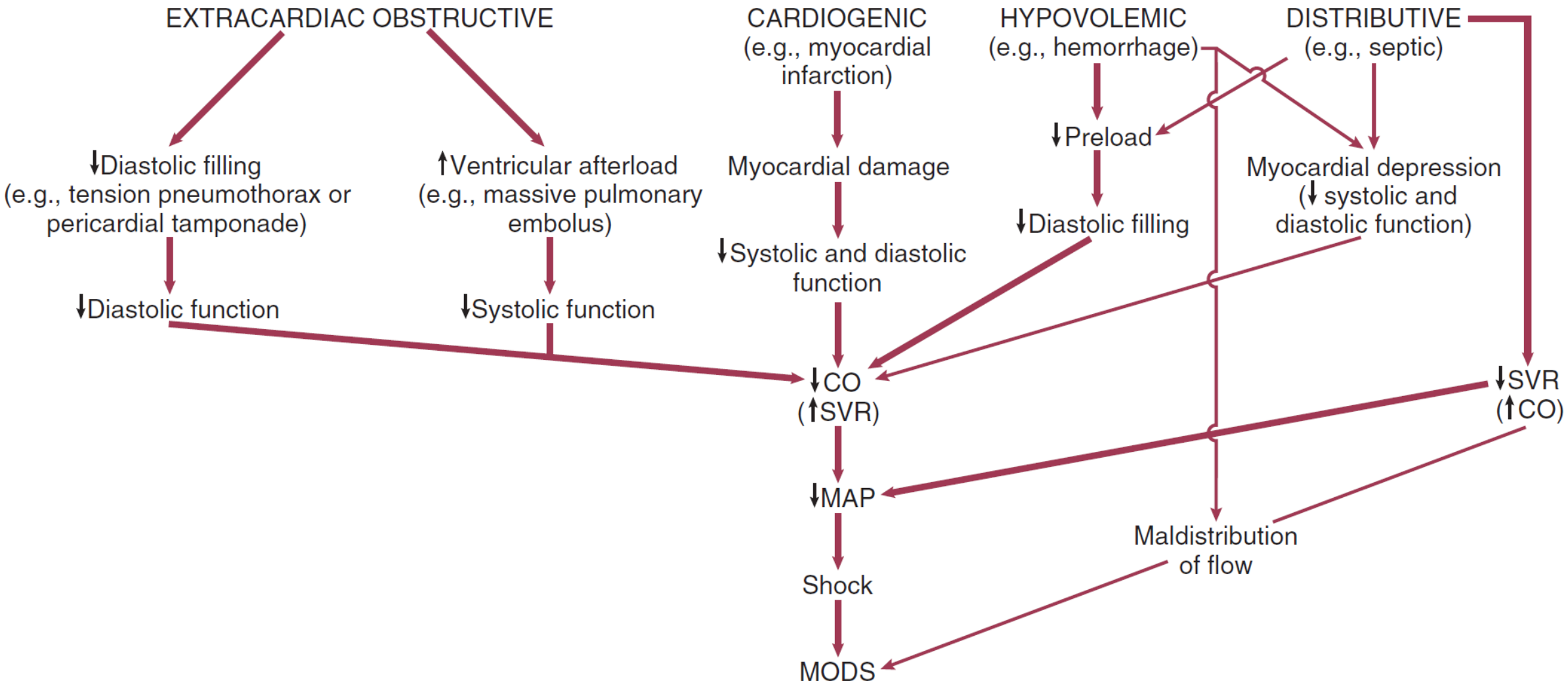
Vicious cycle of hemorrhagic shock



3 main mediators of mortality
- **Hypothermia**
- **Coagulopathy**
- **Metabolic acidosis**

Cannon JW. Hemorrhagic Shock. N Engl J Med. 2018 Jan 25;378(4):370-379.

The interrelationships between different forms of shock



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