Shock Part 1

Medical Practice Improvement

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Disclosures

- I have no disclosures

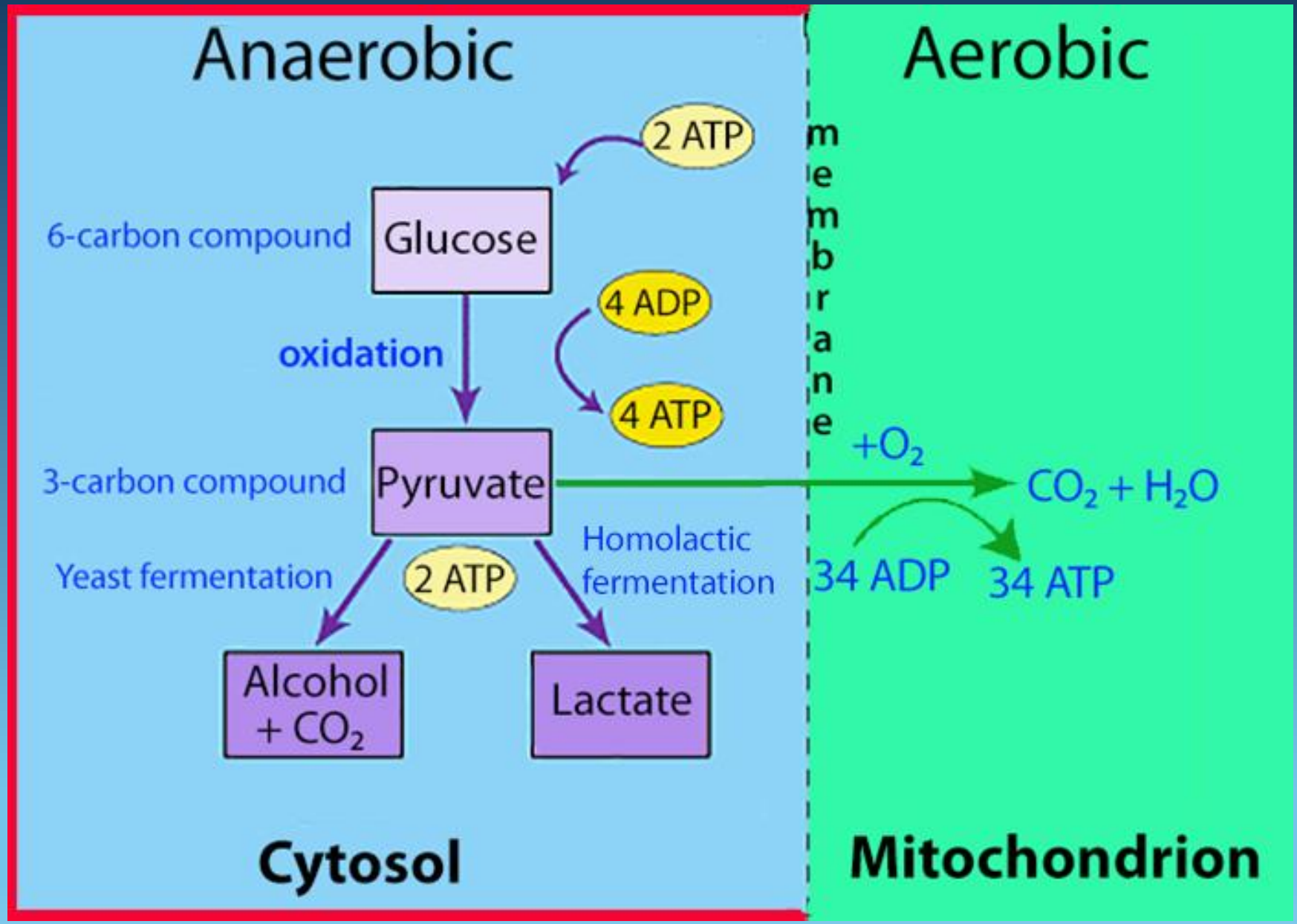
Learning Objectives

- 1. Introduction to shock
- 2. Look at oxygen consumption and delivery mismatch
- 3. Discuss the Classes of Shock
- 4. Cellular responses to shock

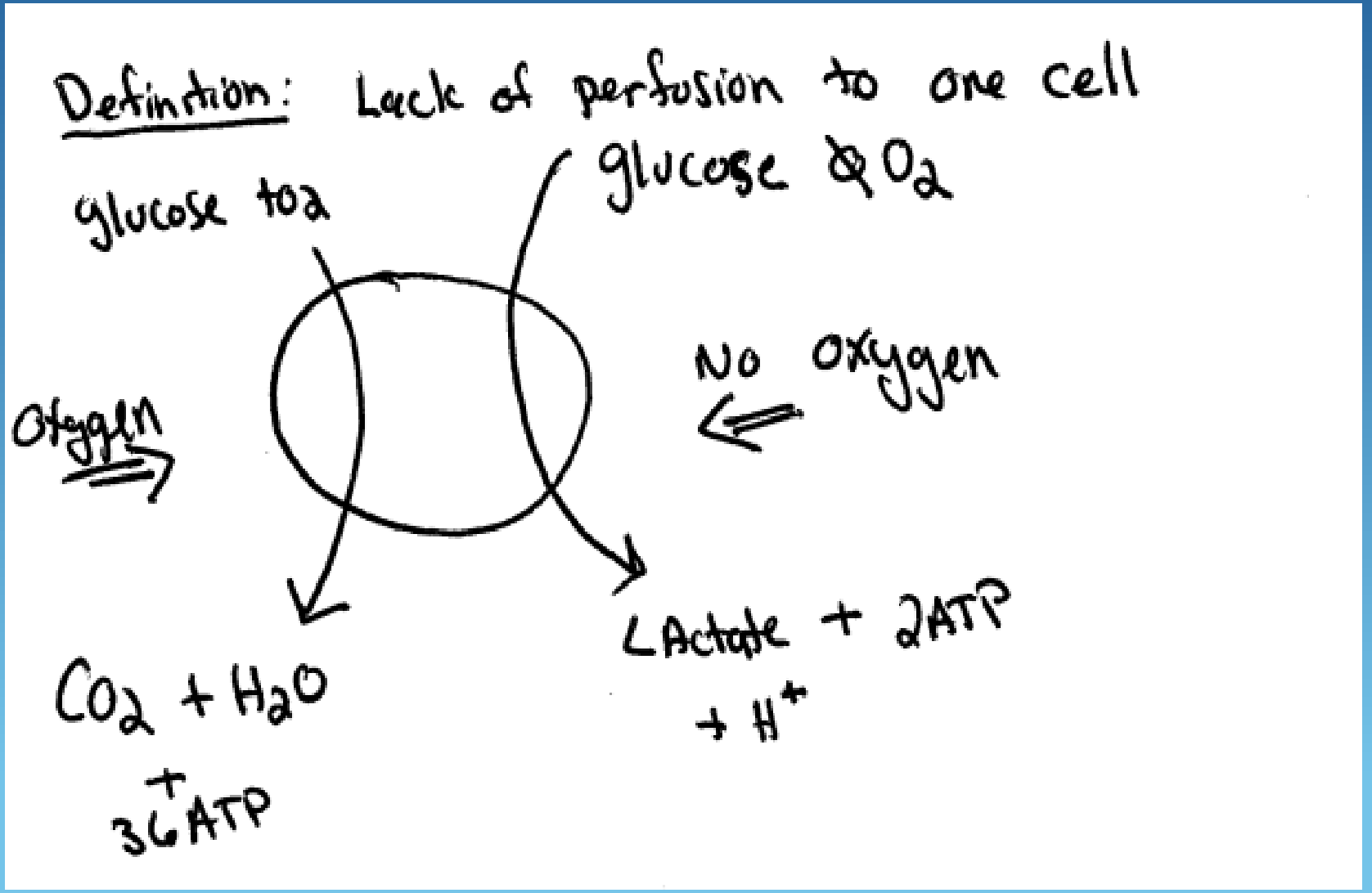
What is Shock?

- Defined as the lack of perfusion to one cell
  - Disruption of cellular respiration
  - No oxygen delivery
  - Then no Krebs cycle
  - Then you get Lactate and a proton H+ as waste product net 2 ATP
  - If there is oxygen, then it goes to Krebs cycle and the waste products are CO2 and H2O and 36 ATP

Anaerobic metabolism



What is Shock?



What is the Point of Resuscitation?

- Restore Oxygen delivery to the tissues
  - Lactate washout
  - Lactate may go up initially once perfusion restored
    - Metabolized in liver
    - Takes 4-6 hours to metabolize
  - Base deficit will correct in real time
    - Protons are used up in the Krebs cycle and electron transport chain to make H2O

There are Only 5 Causes for Hypotension

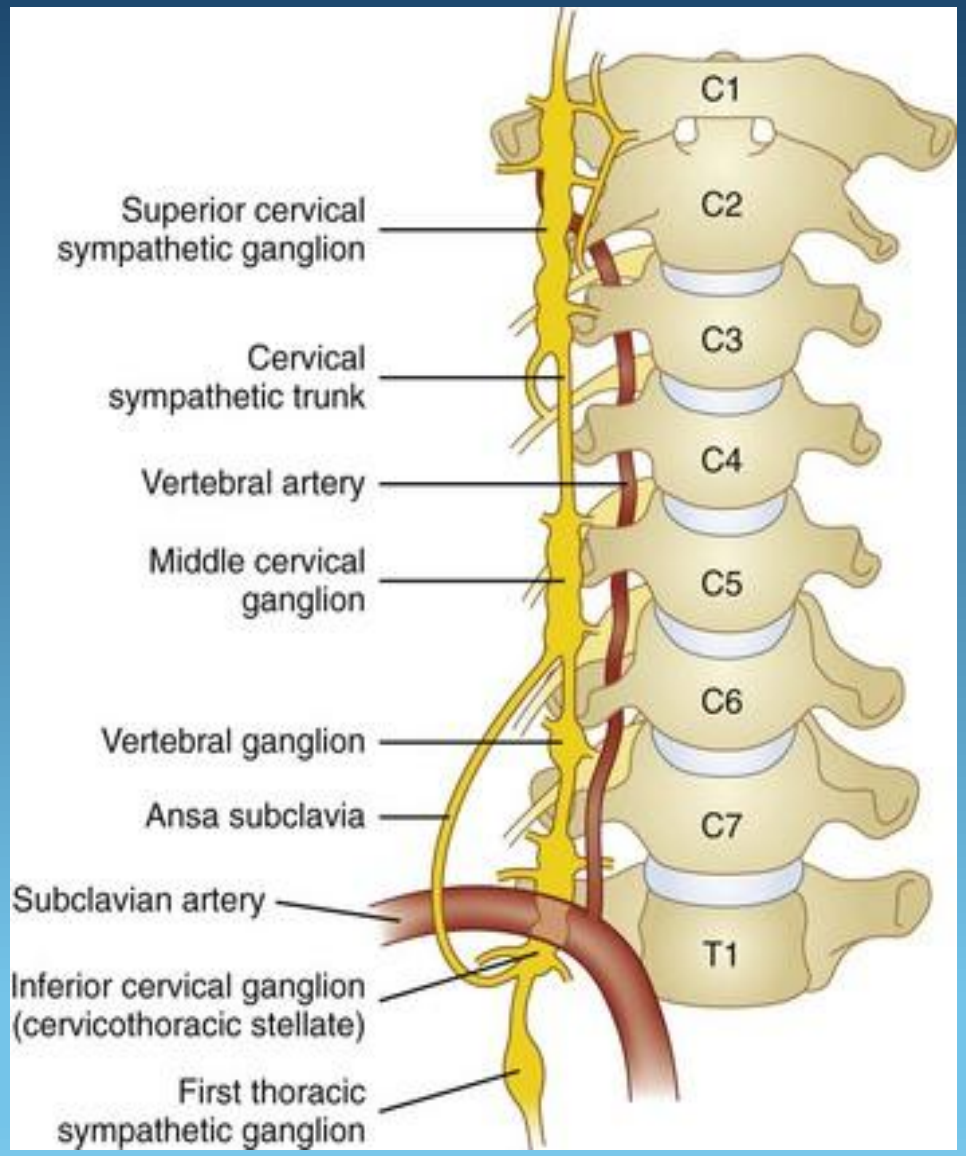
- Classes of Shock
  - Neurogenic Shock
  - Cardiogenic Shock
  - Obstructive Shock
  - Distributive Shock
    - Anaphylactic
    - Endocrine → adrenal insufficiency
  - Hypovolemic
    - Hemorrhagic

Neurogenic Shock

- Loss of Sympathetic tone from spinal cord injury above the level of T2

Symptoms

- Loss of sympathetic chain ganglion
- Hypotension and Bradycardia
- Paralysis
- Loss of sympathetic tone



Neurogenic Shock

- Diagnosis of exclusion
- Every Hypotensive patient for trauma has hypovolemic shock until proven otherwise
- Treatment is fluid 1-2 liters then if unresponsive
- Dopamine or Levaphed
- Try not to use pure alpha agonist as it can exacerbate bradycardia

Vasopressor	Initial Dose	Usual Maintenance Dose Range	Alpha-1	Beta-1	Beta-2	V1	V2	DA
Norepinephrine	5-15 mcg/min	2-80 mcg/min	++++	++	++	0	0	0
Epinephrine	1-15 mcg/min	1-40 mcg/min	+++	+++	+++	0	0	0
Phenylephrine	40-160 mcg/min	20-400 mcg/min	++++	0	0	0	0	0
Vasopressin	0.03 units/min	0.01-0.04 units/min	0	0	0	+++	+++	0
Dopamine	2-5 mcg/kg/min	0.5-2 mcg/kg/min	0	+	0	0	0	++
		5-10 mcg/kg/min	+	++	0	0	0	++
		10-20 mcg/kg/min	++	++	0	0	0	++

Receptors

Receptor	Primary Location	Normal Physiologic Action	**Agonist Effects**	**Agonist Side Effects**	**Antagonist Effects**	**Antagonist Side Effects**	Antagonizes / Opposes
α1	Vascular smooth muscle, pupils, bladder sphincter	Vasoconstriction, mydriasis, ↑ urinary retention	↑ BP, vasoconstriction, pupil dilation	Hypertension, reflex bradycardia, ischemia	Vasodilation, ↓ BP, improved urine flow	Orthostatic hypotension, dizziness	Opposes **β2**
α2	CNS (presynaptic), pancreas	↓ NE release, ↓ insulin, ↓ sympathetic tone	↓ BP, sedation, ↓ HR	Bradycardia, sedation, dry mouth	↑ NE release, ↑ BP	Hypertension, anxiety	Opposes **β1, β2**
β1	Heart, kidneys	↑ HR, ↑ contractility, ↑ renin	↑ HR, ↑ CO, ↑ BP	Tachycardia, arrhythmias	↓ HR, ↓ CO, ↓ BP	Bradycardia, heart block	Opposed by **α2**
β2	Lungs, uterus, skeletal muscle vessels	Bronchodilation, vasodilation, uterine relaxation	Bronchodilation, ↓ uterine tone	Tremor, tachycardia, hypokalemia	Bronchoconstriction, uterine contraction	Bronchospasm	Opposed by **α1**
V1	Vascular smooth muscle	Vasoconstriction	↑ SVR, ↑ BP	Ischemia, ↓ organ perfusion	Vasodilation	Hypotension	Opposes **β2**
V2	Renal collecting ducts	Water reabsorption (ADH effect)	↑ free water retention	Hyponatremia, fluid overload	Diuresis	Dehydration, hypernatremia	Functional opposite of diuretics

Key Teaching Relationships (High-Yield)

Major Functional Oppositions

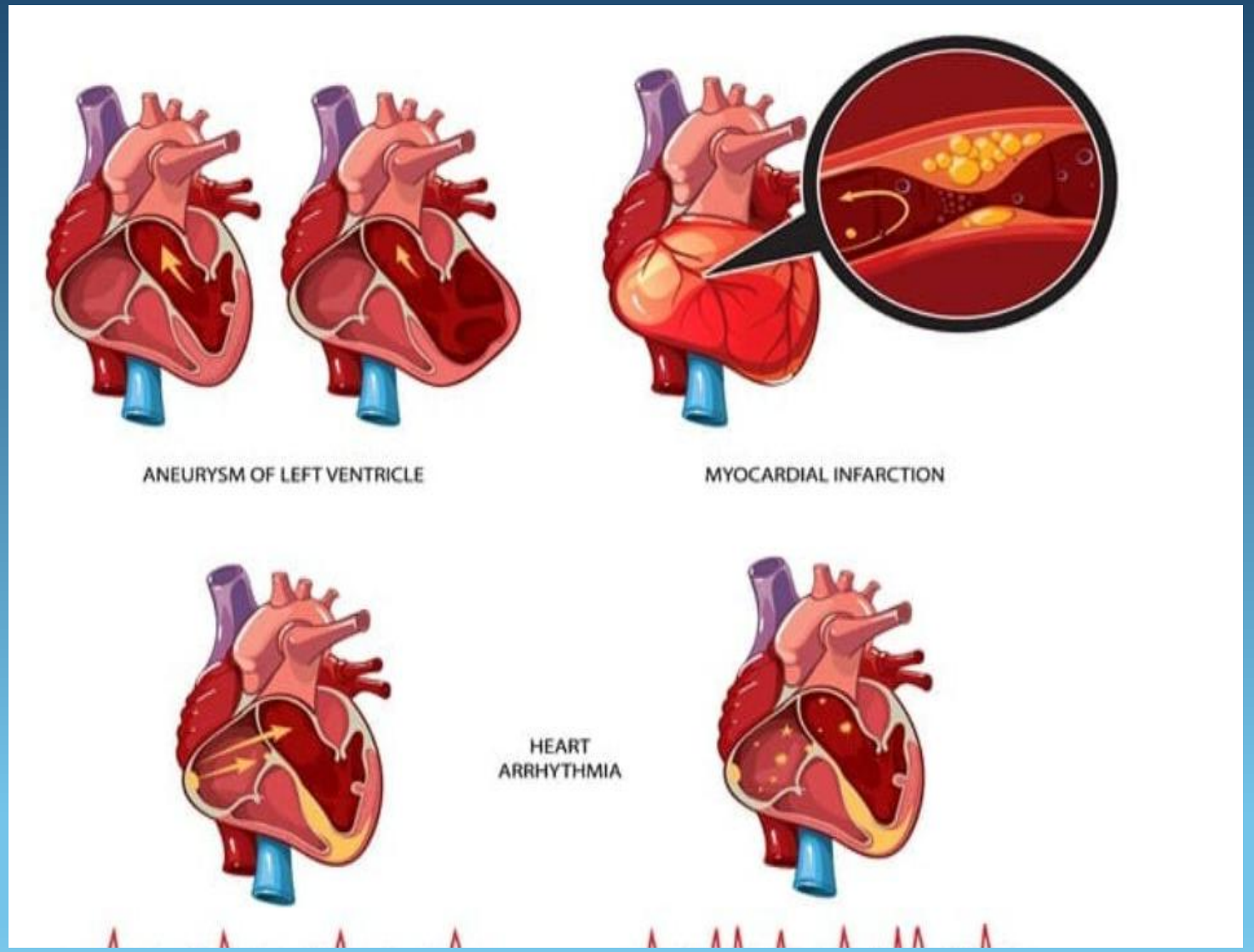
- **α1 vs β2**
  - α1 → vasoconstriction
  - β2 → vasodilation & bronchodilation
- **α2 vs β1**
  - α2 → ↓ sympathetic output
  - β1 → ↑ cardiac stimulation
- **V1 vs β2**
  - V1 → vasoconstriction
  - β2 → vasodilation
- **V2 vs Diuretics**
  - V2 → water retention
  - Diuretics → water excretion

Common Drugs and the Effects

Drug	Action
Phenylephrine	α1 agonist
Clonidine	α2 agonist
Metoprolol	β1 antagonist
Albuterol	β2 agonist
Vasopressin	V1 + V2 agonist
Tolvaptan	V2 antagonist

Cardiogenic Shock

- Loss of cardiac function
- MI, ruptured Cordie tendinea, cardiac failure
- Hypotension
- Tachycardia
- Hypoxia
- Patients can die quickly
- Impedes pre- load



Chemical Augmentation

- Keep cardiac out put up for oxygen delivery to tissues
- Increase stroke volume
  - Norepi
  - Epi
  - Dobutamine
  - Dopamine
  - Milrinone

Goals of Mechanical Augmentation

- Cardiac output
- ↓ LV workload & oxygen demand
- Maintain coronary & end-organ perfusion
- Bridge to:
  - Recovery
  - Decision
  - LVAD
  - Transplant

Spectrum of Mechanical Support

Least → Most Support

- IABP
- Impella (2.5 / CP / 5.0 / 5.5)
- Durable LVAD

Support escalates as:

- Hemodynamics worsen
- Pharmacologic therapy fails

Intra-Aortic Balloon Pump (IABP)

Mechanism

- Balloon inflates in diastole → ↑ coronary perfusion
- Deflates in systole → ↓ afterload

Hemodynamic Effects

- Modest ↑ CO (~0.5 L/min)
- ↓ LV afterload
- ↑ diastolic pressure

IABP: Pros, Cons, Use

Advantages

- Quick to place
- Low complication rate
- Familiar technology

Limitations

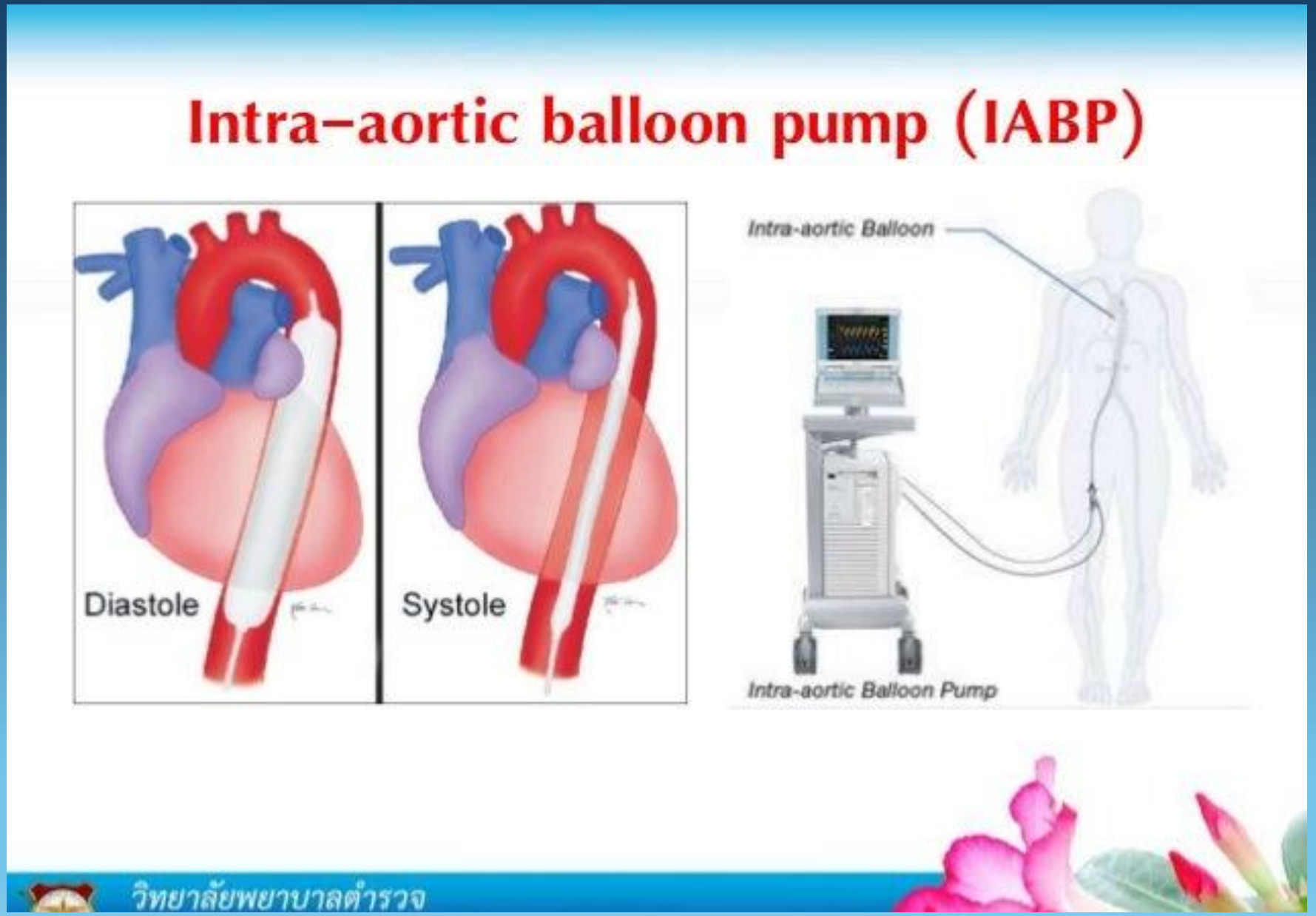
- Minimal CO augmentation
- No direct LV unloading
- Limited benefit in severe shock

Best For

- Mild cardiogenic shock
- Mechanical complications
- Bridge therapy

Balloon pump

Inflates with diastole to augment coronary blood flow
Heart perfuses during diastole only
Can be 1:1 all the way to 4:1 (prior to removal)



Impella: What It Is?

- **Percutaneous Axial-Flow LV Assist Device**
- Actively pumps blood:
  - From LV → Ascending aorta
- Direct LV unloading
- Provides forward flow independent of rhythm

Impella Hemodynamic Impact

- ↑ Cardiac output (2.5–5.5 L/min)
- ↓ LVEDP
- ↓ myocardial oxygen consumption
- Improves coronary perfusion
- **Key Advantage:** True ventricular unloading

- Drawback need to anticoagulated and causes hemolytic anemia

Impella Variants

Device	Max Flow	Access
Impella 2.5	~2.5 L/min	Femoral
Impella CP	~3.5–4.0 L/min	Femoral
Impella 5.0 / 5.5	~5.0–5.5 L/min	Axillary / Surgical

Impella: Risks & Considerations

Complications

- Hemolysis
- Vascular injury
- Limb ischemia
- Device migration

Clinical Use

- Moderate–severe cardiogenic shock
- High-risk PCI
- Bridge to LVAD or recovery

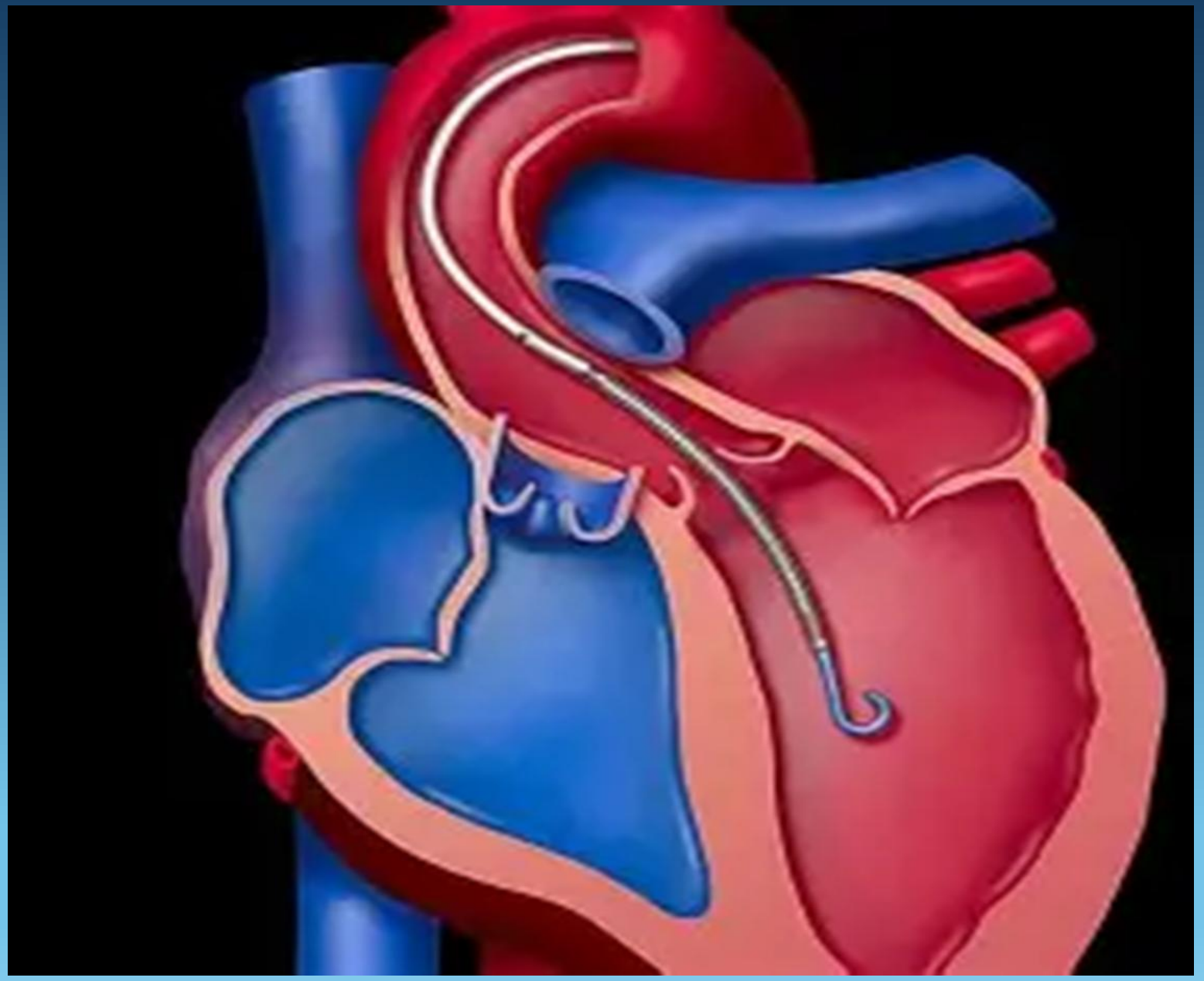
How the Impella works

Impella works via a **micro-axial rotary pump** (an Archimedes-screw–style impeller).

- An **electric motor** spins blades inside the catheter
- This **actively pulls blood** from the LV
- And **pushes it forward** into the ascending aorta
- Flow is **continuous and pressure-generating**

👉 That's **active mechanical pumping**, not fluid entrainment.

- Spins at 35000 RPM



LVAD: Durable Mechanical Support

What Is an LVAD

- Surgically implanted continuous-flow pump
- LV → Aorta
- Long-term circulatory support

Types

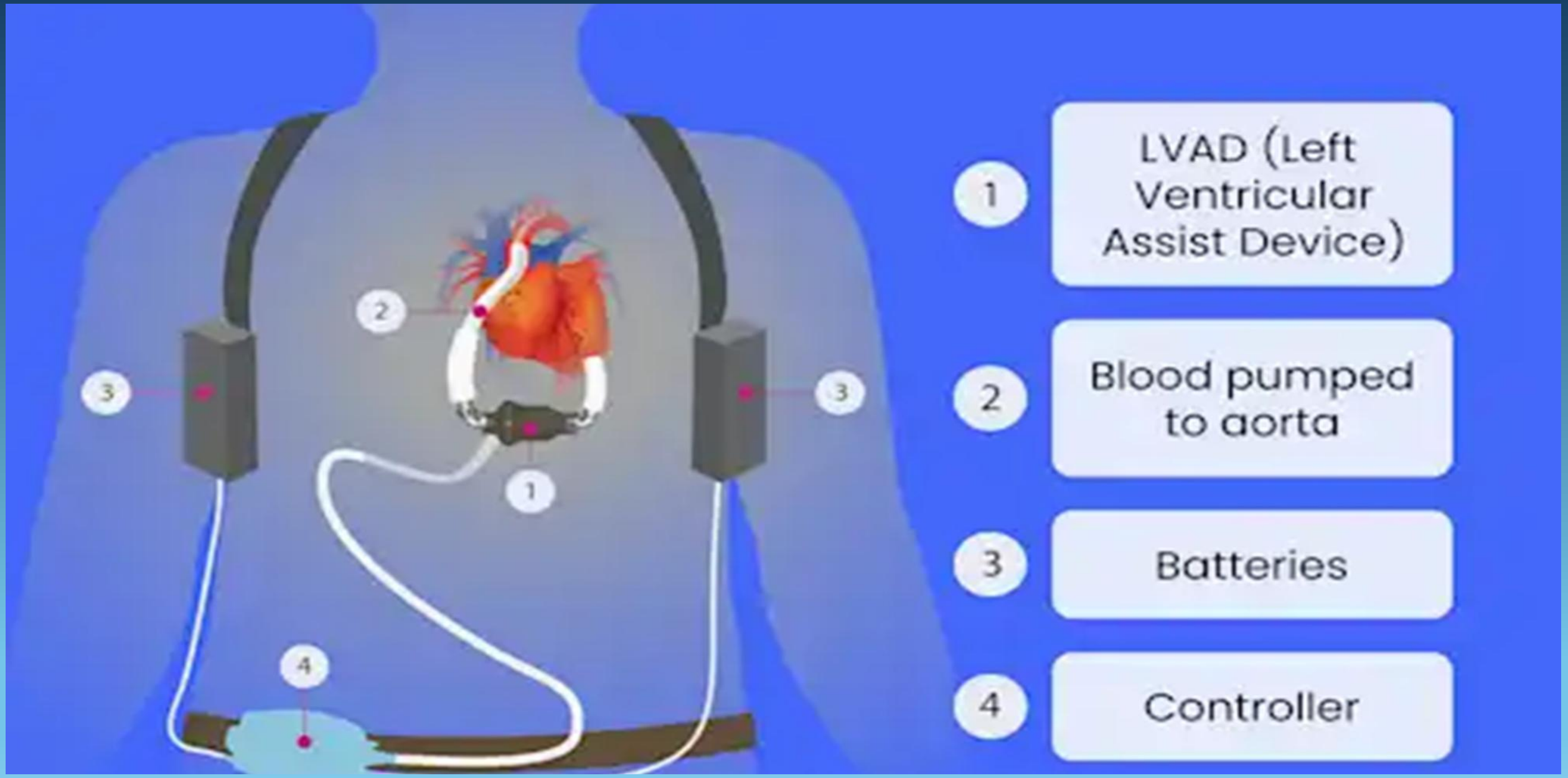
- Bridge to transplant
- Destination therapy

LVAD Hemodynamics & Outcomes

- Provides full cardiac support
- Sustains systemic perfusion
- Allows end-organ recovery
- Long-term survival improvement in advanced HF

LVAD Risks

- Bleeding
- Infection
- Thromboembolism
- Right ventricular failure
- Lifelong anticoagulation



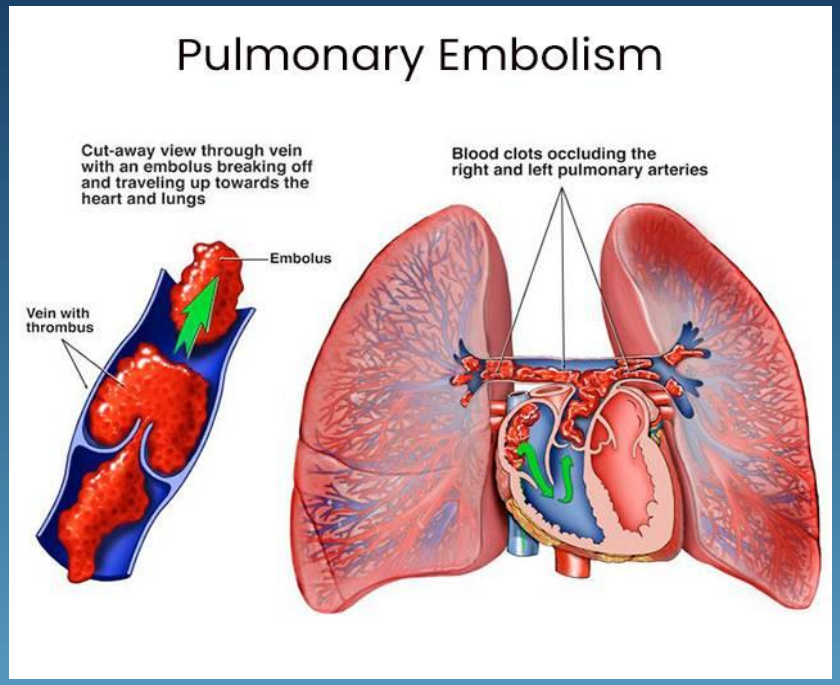
Device Comparison

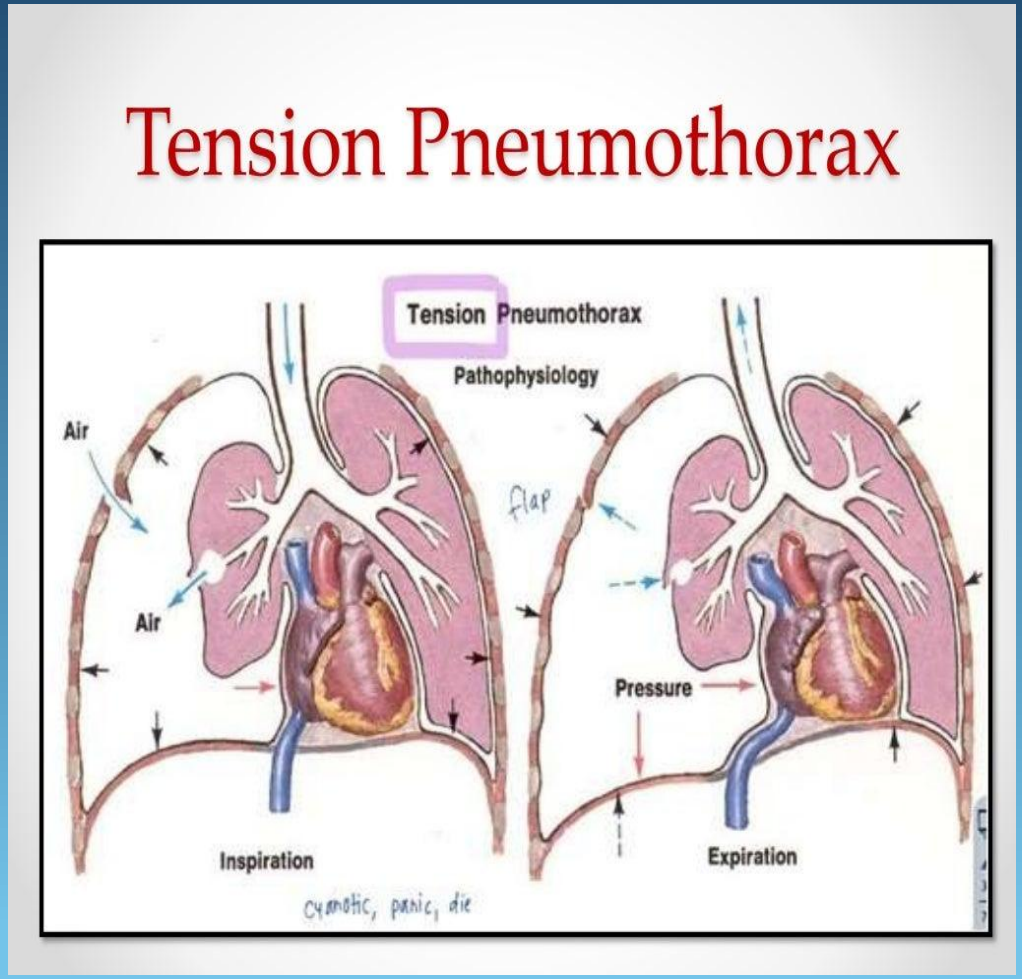
Feature	IABP	Impella	LVAD
Flow Support	Low	Moderate–High	Full
LV Unloading	Minimal	Significant	Complete
Invasiveness	Low	Moderate	High
Duration	Days	Days–Weeks	Months–Years
Shock Severity	Mild	Moderate–Severe	End-Stage

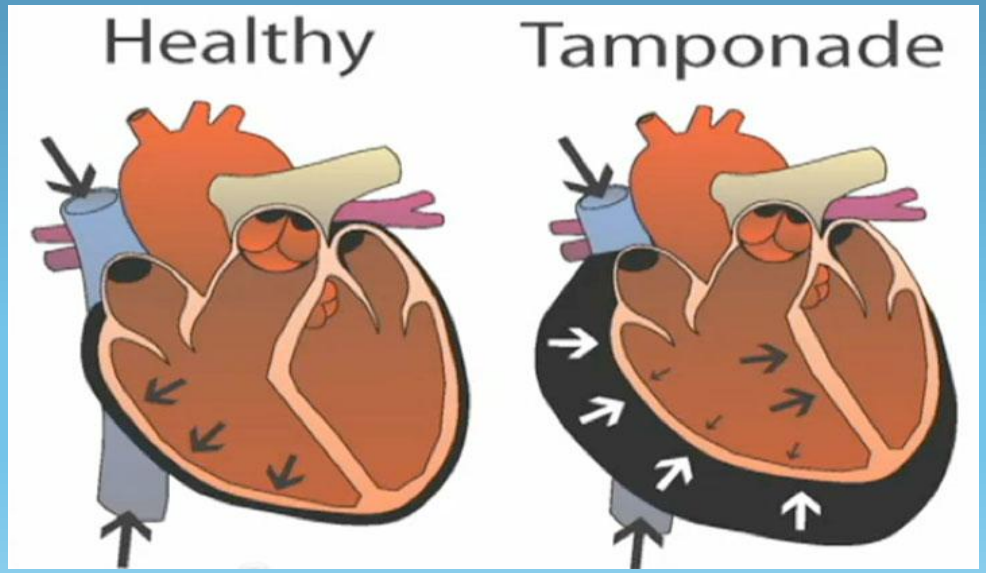
Obstructive Shock

- Failure of cardiac Filling. No Pre-load
- Tension PTX, Cardiac tamponade, Pulmonary embolism
- Most dangerous shock
- Patient die quickly

Obstructive Shock







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Distributive Shock

- Anaphylactic shock→ hemodynamic collapse.
  - Hypotension
  - Tachycardia
  - Hypoxia
  - From an offending agent

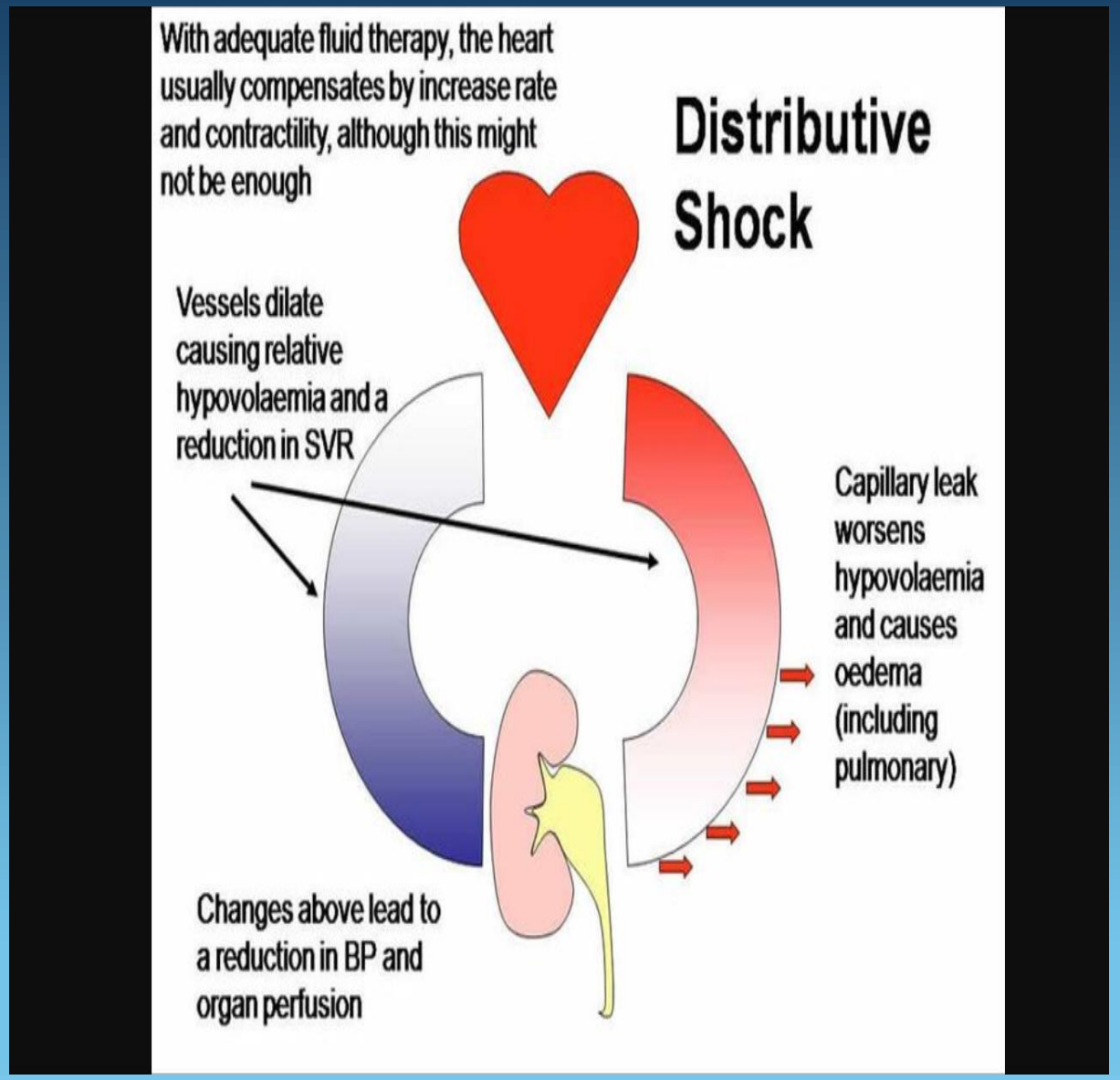
Endocrine → adrenal insufficiency

Distributive shock→ “Septic shock”

From infection

Hypotension and tachycardia

Capillary Leak



Hypovolemic Shock

- Lack of volume
- Decreased cardiac filling
- Tachycardia
- Hypotension
- Subcategory
  - Hemorrhagic

Hypovolemic Shock

Classes of Hypovolemic Shock

	Class I	Class II	Class III	Class IV
Blood Loss	< 750	750-1500	1500-2000	> 2000
% Blood Vol.	< 15%	15 – 30%	30 – 40%	> 40%
Pulse	< 100	**> 100**	> 120	> 140
Blood Pressure	Normal	Normal	**Decreased**	**Decreased**
Pulse Pressure	Normal	**Decreased**	**Decreased**	**Decreased**
Resp. Rate	14 – 20	**20 – 30**	**30 – 40**	**> 40**
UOP	> 30	**20 – 30**	**5 – 15**	**negligible**
Mental Status	sl. Anxious	mildly anx	**confused**	**lethargic**
Fluid	crystalloid	crystalloid	**blood**	**blood**

Treatment of shock

Recognize what type of shock you are treating	Categorize it
Remember :	Patients may have more than one type of shock
Three steps to treating shock	Resuscitation Identify the cause Reverse the underlying physiology

Oxygen Delivery

- DO_2 term for oxygen delivery
- Equation is as follows
- D$O_2 = Cardiac\ Output\ X\ CaO_2\ X\ 10$
- Cardiac Output = CO
- CaO_2 = Oxygen carrying capacity of blood
- $CaO_2 =$ $1.34\ (Hgb\ in\ grams)(SaO_2)$+(0.003 x Pa$O_2$)

Concludes Part I of shock

Part 2 will discuss treatment of the classes of shock

Part 3 will look at monitoring and advance ICU treatment for Shock

Work Cited

- glycolysis with inadequate oxygen delivery waste products - Search Images
- sympathetic chain ganglion - Search Images
- cardiogenic shock - Search Images
- tension pneumothorax - Search Images
- cardiac tamponade - Search Images
- pulmonary embolism - Search Images
- distributive shock - Search Images
- hypovolemic shock - Search Images

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