SHOCK

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INTRODUCTION TO SHOCK

DEFINITION OF SHOCK

- The state in which profound and widespread reduction of effective tissue perfusion leads first to reversible and then, if prolonged, irreversible tissue cellular injury
- Effective tissue perfusion may be reduced by a global reduction of systemic perfusion (CO) or by increased ineffective tissue perfusion resulting from a maldistribution of blood flow or a defect of substrate at the cellular level
- Shock is the clinical syndrome that results from inadequate tissue perfusion

DETERMINANTS OF EFFECTIVE TISSUE PERFUSION IN SHOCK

CARDIOVASCULAR PERFORMANCE

- Cardiac function
  - Preload
  - Afterload
  - Contractility
  - Heart rate
- Venous return
  - Right atrial pressure
  - Mean circulatory pressure

DISTRIBUTION OF CARDIAC OUTPUT

- Intrinsic regulatory systems
  - Local tissue factors
- Extrinsic regulatory systems
  - Sympathetic
  - Adrenal activity
- Anatomic vascular disease
- Exogenous vasoactive agents
  - Inotropes
  - Vasopressors
  - Vasodilators

DETERMINANTS OF EFFECTIVE TISSUE PERFUSION IN SHOCK

HEMODYNAMIC CHANGES

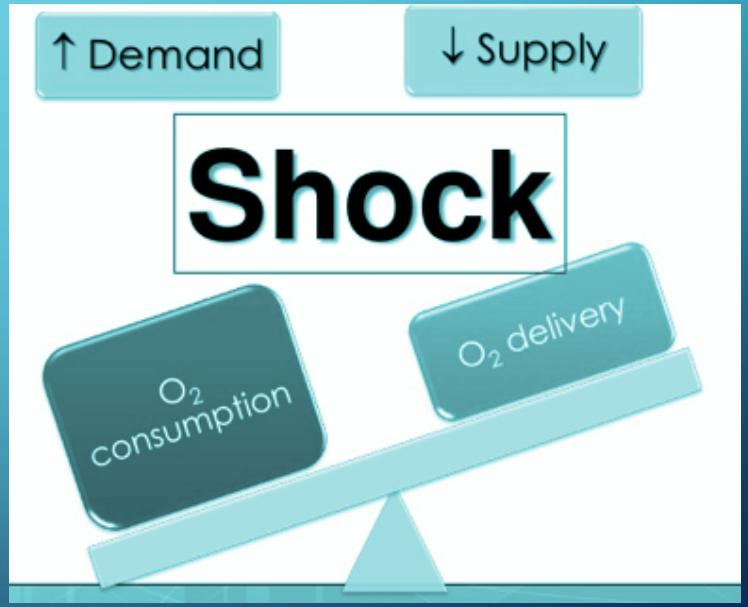
HYPOTENSION

Usually present, but not required

MEAN ARTERIAL PRESSURE

MAP = CO X SVR

(Decrease in one or both)



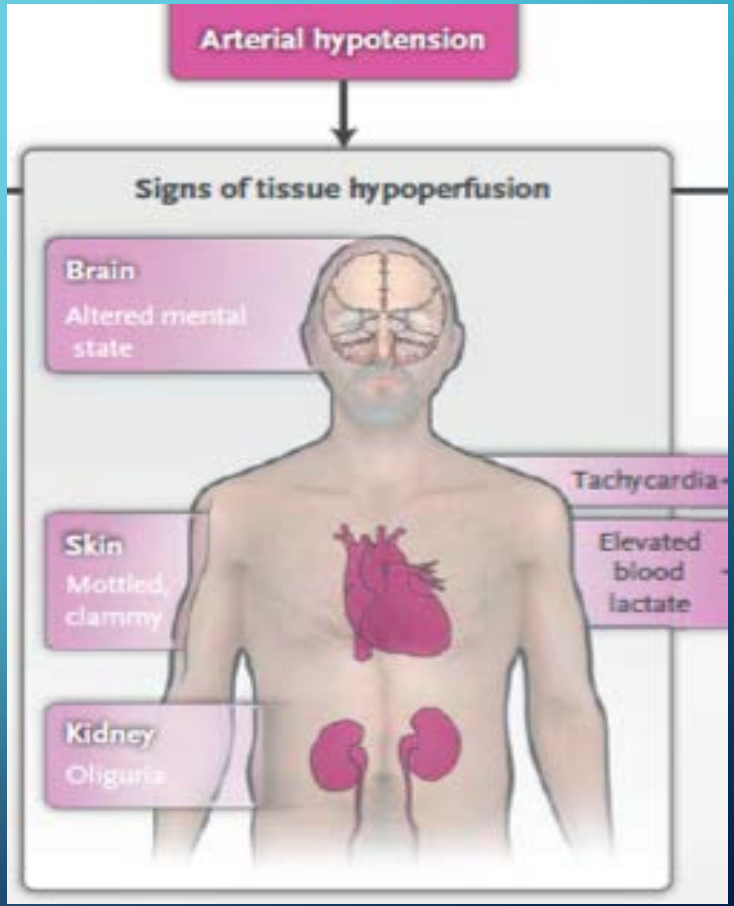
SIGNS

CLINICAL SIGNS

- Tissue Hypoperfusion
  - Brain
  - Skin
  - Kidney

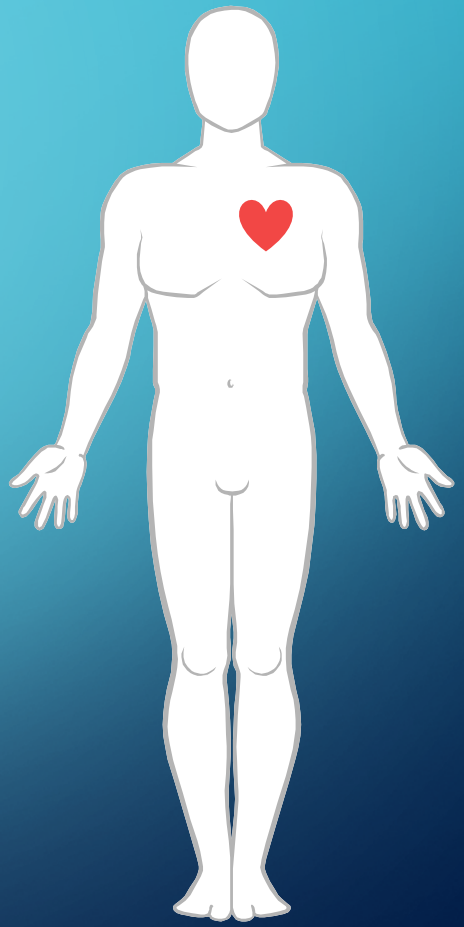
BIOCHEMICAL SIGNS

- Elevated lactate levels
- Abnormal cellular oxygen metabolism

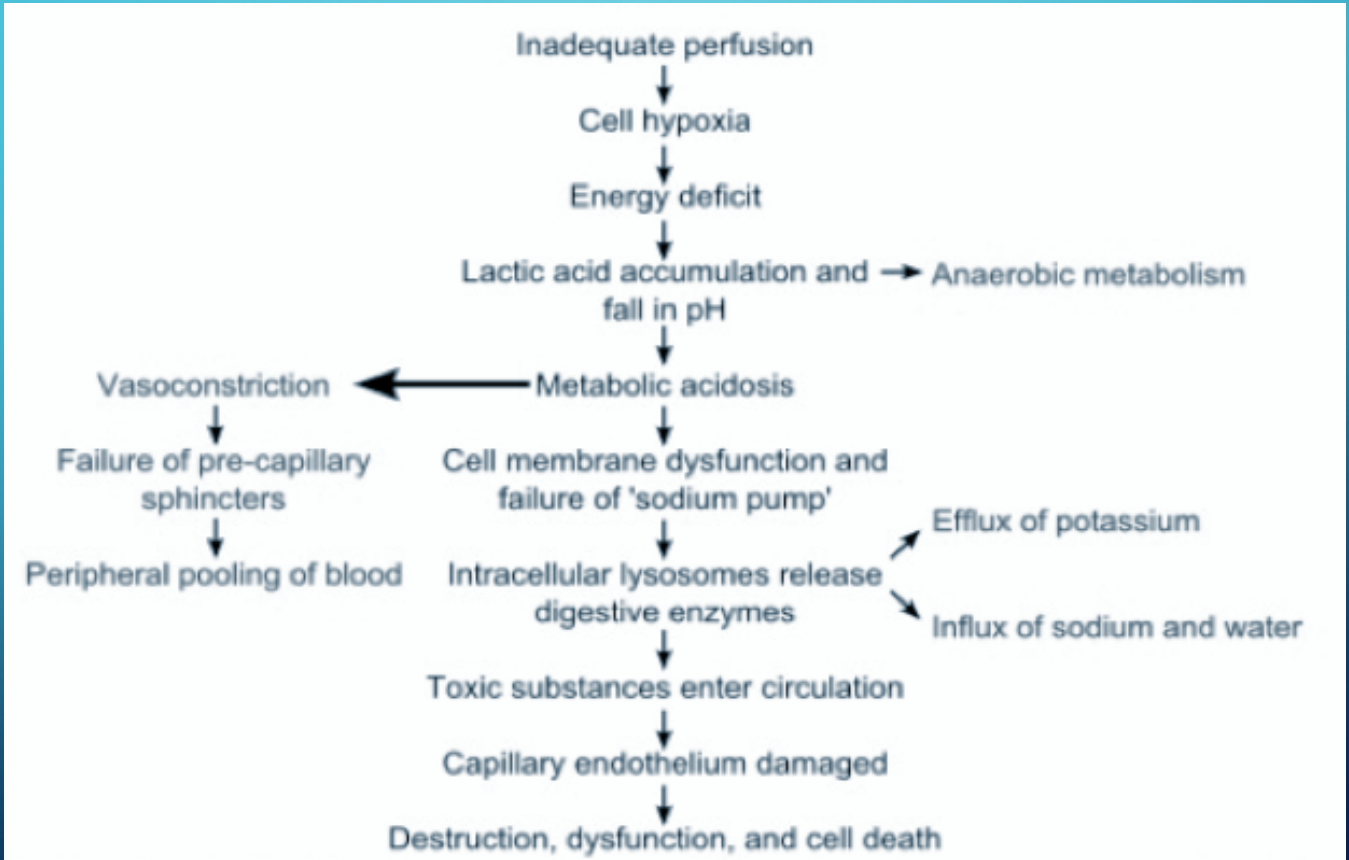


EFFECTS OF SHOCK ON ORGANS

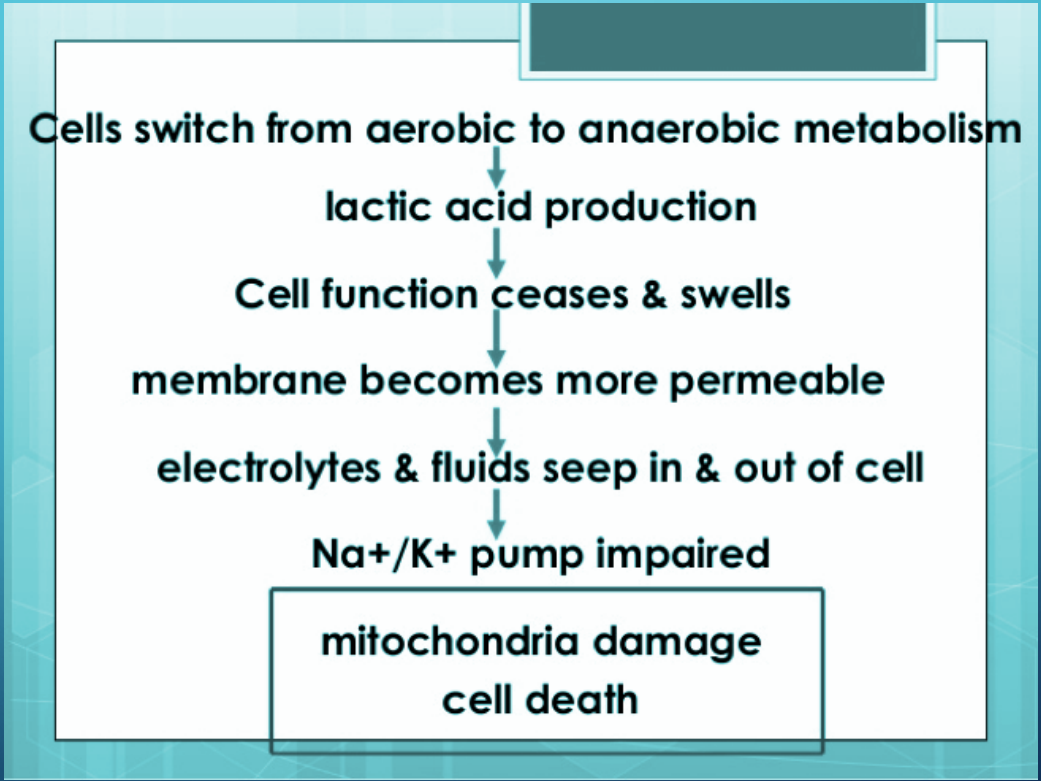
- Heart – ↓ CO / hypotension / myocardial depressants
- Lung - ↓gas exchange / tachypnoea / pulmonary edema
- Endocrine – ADH → ↑ reabsorption of water
- CNS – perfusion ↓ – drowsy
- Blood - Coagulation abnormalities – DIC
- Renal - ↓ GFR - ↓ urine output
- GIT – mucosal ischaemia – bleeding & hepatic - ↑ enzyme levels



PATHOPHYSIOLOGY OF SHOCK



PATHOPHYSIOLOGY OF SHOCK



CLASSIFICATIONS OF SHOCK

Hypovolemic, Distributive, Extracardiac Obstructive, Cardiogenic

CLASSIFICATIONS OF SHOCK

Hypovolemic

- Decreased circulating blood volume in relation to the total vascular capacity
- Characterized by a reduction of diastolic filling pressures and volumes

Distributive

- Loss of vasomotor control resulting in arteriolar and venular dilation
- Characterized by increased Cardiac output with decreased SVR

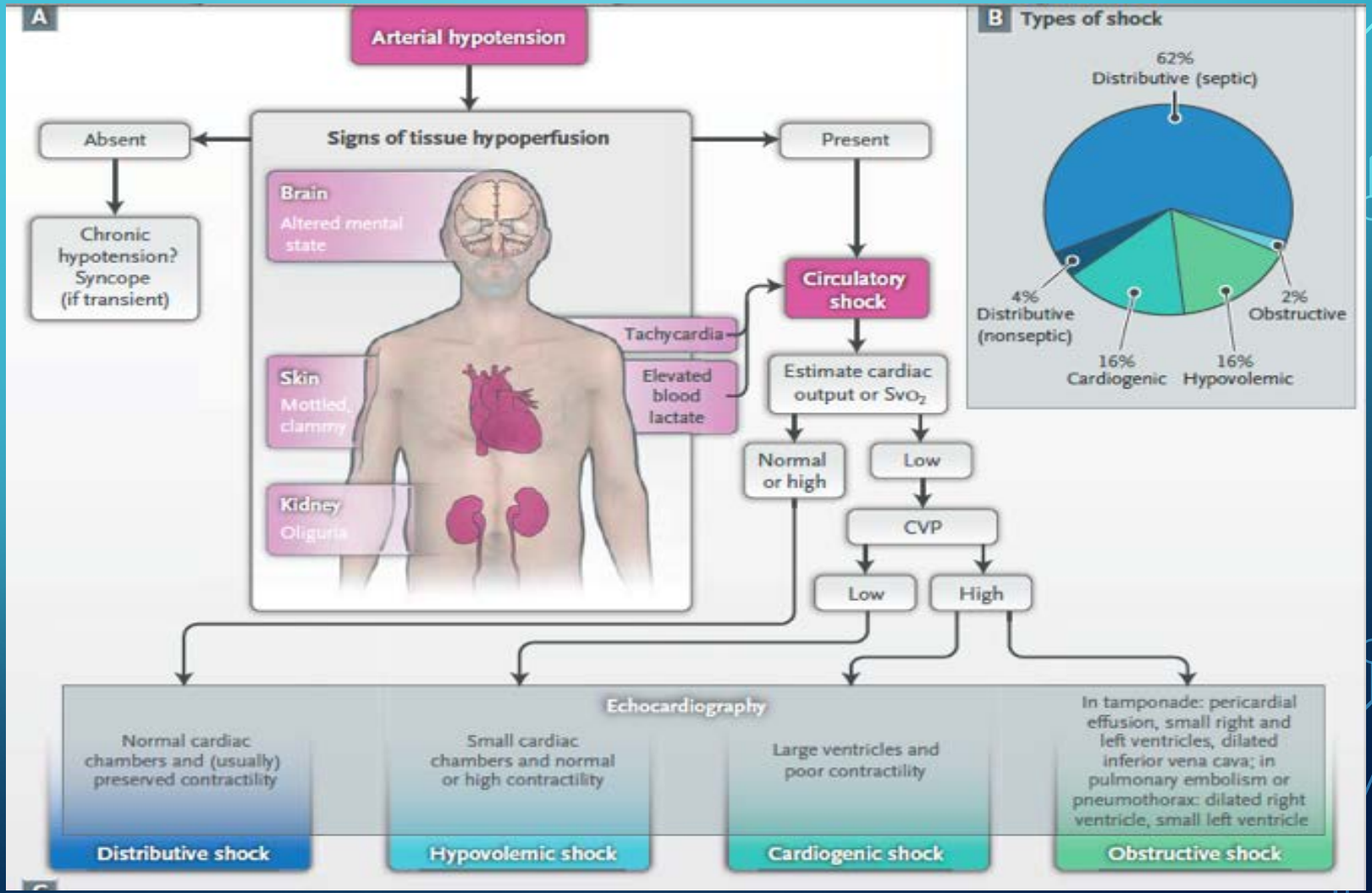
CLASSIFICATIONS OF SHOCK

Extracardiac Obstructive

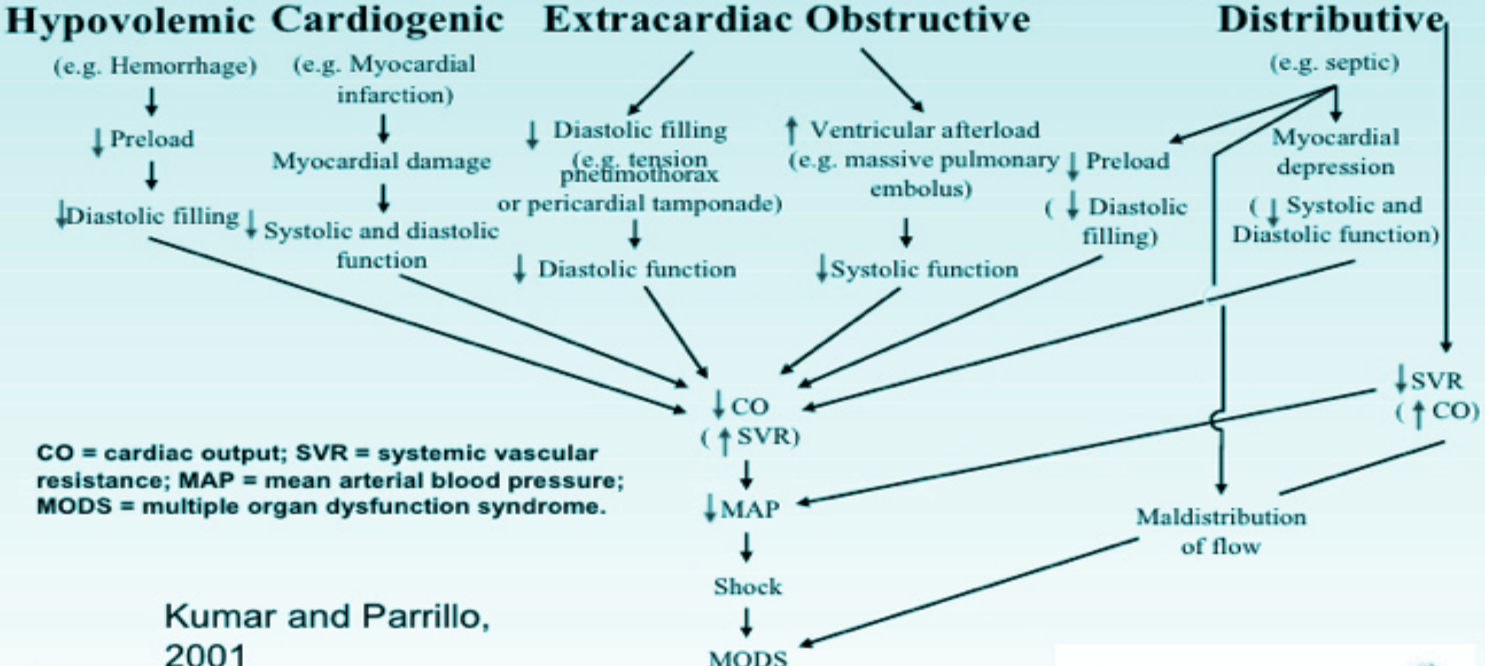
- Obstruction to flow in the cardiovascular circuit
- Characterized by either impairment of diastolic filling or excessive afterload

Cardiogenic

- Related to pump failure owing to loss of myocardial contractility and functional myocardium
- Structural and mechanical failure of the cardiac anatomy
- Characterized by elevations of diastolic filling pressures and volumes


A
Arterial hypotension
Absent
Chronic hypotension? Syncope (if transient)
Signs of tissue hypoperfusion
Brain
Altered mental state
Skin
Mottled, clammy
Kidney
Oliguria
Tachycardia
Elevated blood lactate
Present
Circulatory shock
Estimate cardiac output or SvO2
Normal or high
Low
CVP
Low
High
B Types of shock
62% Distributive (septic)
4% Distributive (nonseptic)
2% Obstructive
16% Cardiogenic
16% Hypovolemic
Echocardiography
Normal cardiac chambers and (usually) preserved contractility
Distributive shock
Small cardiac chambers and normal or high contractility
Hypovolemic shock
Large ventricles and poor contractility
Cardiogenic shock
In tamponade: pericardial effusion, small right and left ventricles, dilated inferior vena cava; in pulmonary embolism or pneumothorax: dilated right ventricle, small left ventricle
Obstructive shock

Classification of Shock



CO = cardiac output; SVR = systemic vascular resistance; MAP = mean arterial blood pressure; MODS = multiple organ dysfunction syndrome.

Kumar and Parrillo, 2001

Society of Critical Care Medicine
The Intensive Care Professionals

HEMODYNAMIC PROFILES IN SHOCK

Diagnosis	CO	SVR	PWP	CVP	$S\bar{V}o_2$
Cardiogenic shock					
Caused by myocardial dysfunction	↓↓	↑	↑↑	↑↑	↓
Caused by a mechanical defect					
Acute ventricular septal defect	LVCO ↓↓ RVCO > LVCO	↑	nl or ↑	↑↑	↑ or ↑↑
Acute mitral regurgitation	Forward CO ↓↓	↑	↑↑	↑ or ↑↑	↓
Right ventricular infarction	↓↓	↑	nl or ↑	↑↑	↓
Extracardiac obstructive shock					
Pericardial tamponade	↓ or ↓↓	↑	↑↑	↑↑	↓
Massive pulmonary emboli	↓↓	↑	nl or ↓	↑↑	↓
Hypovolemic shock	↓↓	↑	↓↓	↓↓	↓
Distributive shock					
Septic shock	↑↑ or nl, rarely ↓	↓ or ↓↓	↓ or nl	↓ or nl	↑ or ↑↑
Anaphylaxis	↑↑ or nl, rarely ↓	↓ or ↓↓	↓ or nl	↓ or nl	↑ or ↑↑

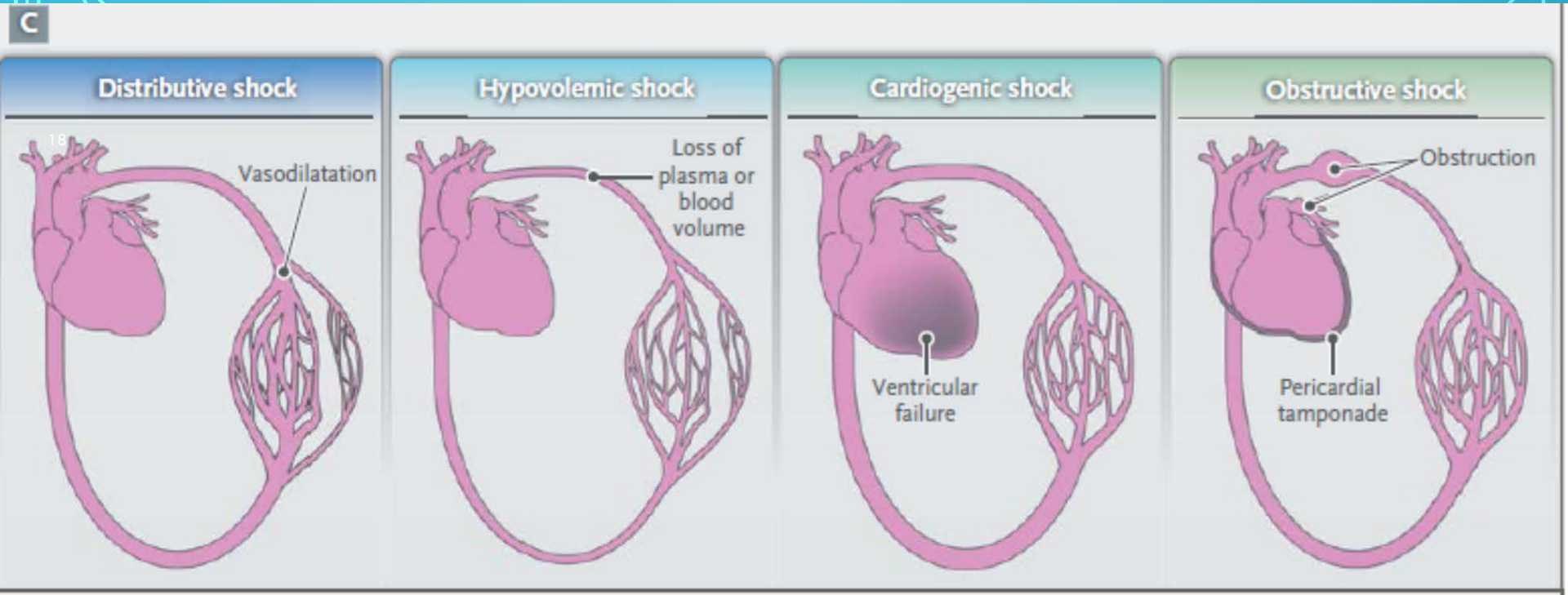


Figure 1. Initial Assessment of Shock States.

Shown is an algorithm for the initial assessment of a patient in shock (Panel A), relative frequencies of the main types of shock (Panel B), and schematic representations of the four main types of shock (Panel C). The algorithm starts with the most common presentation (i.e., arterial hypotension), but hypotension is sometimes minimal or absent. CVP denotes central venous pressure, and SvO_2 mixed venous oxygen saturation.

CARDIOGENIC SHOCK

Statistics, Risk Factors, Pathophysiology, Diagnosis, Parameters, Causes, Management

CARDIOGENIC SHOCK

STATISTICS

- Most commonly due to ischemic myocardial injury with a total of 40% of the myocardium nonfunctional
- Cardiogenic shock from myocardial infarction has a mortality of 60-90%
- Diagnosed after documentation of myocardial dysfunction and exclusion of correction of factors such as hypovolemia, hypoxia and acidosis
- Occurs in 5-8% of STEMI patients and 2.5% of NSTEMI patients
- The only way to prevent development is early reperfusion

CARDIOGENIC SHOCK

RISK FACTORS IN CONTEXT OF MI

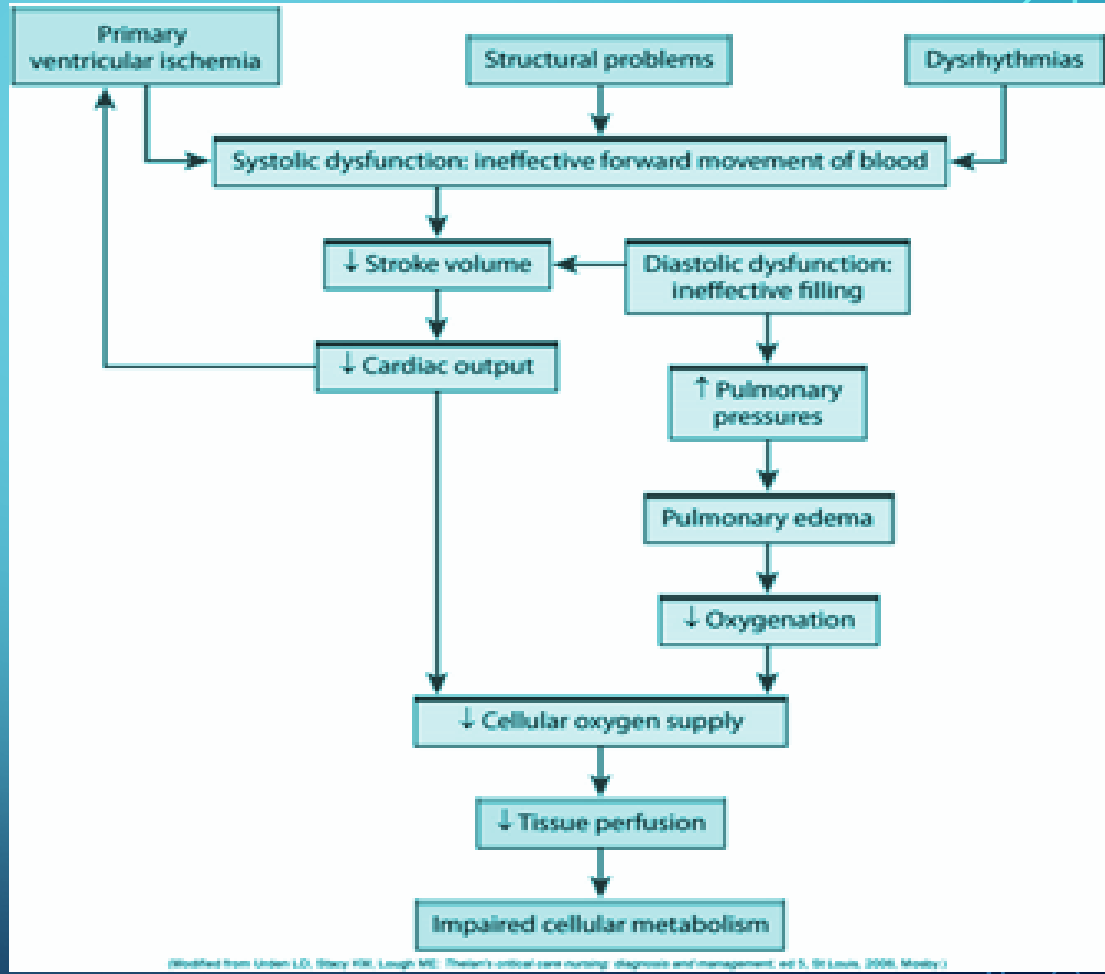
- Older age
- Anterior MI
- Hypertension
- Diabetes mellitus
- Multivessel coronary artery disease
- Prior MI or angina
- Prior diagnosis of heart failure
- STEMI
- Left bundle branch block

There may be clues to impending shock: Heart rate is higher and blood pressure lower on hospital presentation among patients who develop CS after admission

CARDIOGENIC SHOCK

PATHOPHYSIOLOGY

Systolic Dysfunction and Diastolic Dysfunction can both lead to the development of cardiogenic shock



CARDIOGENIC SHOCK

DIAGNOSIS

- Clinical signs
  - Hypotension
  - Oliguria
  - Clouded Sensorium
  - Cool and mottled extremities
- Hemodynamic criteria
  - SBP < 90 mmHg for > 30 minutes
  - Cardiac Index <2.2 L/min/m2
  - PAOP >15mmHg

Early recognition and intervention to interrupt the devastating 'cardiogenic shock spiral' are critical to survival

CAUSES OF CARDIOGENIC SHOCK: PUMP FAILURE

Cardiomyopathies

Dilated cardiomyopathy

Myocardial Infarction

- Large infarction
- Small infarction with pre-existing left ventricular dysfunction
- Infarct extension
- Reinfarction
- Infarct expansion

Arrhythmias

Both tachycardic and bradycardic

Obstructive/Extracardiac

Pulmonary embolus

Tension pneumothorax

Pericardial tamponade

Mechanical

Valvular stenosis or insufficiencies

Acute MR secondary to papillary muscle rupture

VSD

Free wall rupture

Pericardial tamponade

OTHER CAUSES OF CARDIOGENIC SHOCK

MORE CONDITIONS

- End-stage cardiomyopathy
- Myocarditis
- Myocardial contusion
- Prolonged cardiopulmonary bypass
- Septic Shock with severe myocardial depression
- Left ventricular outflow tract obstruction (AS, HOCM)
- Obstruction to left ventricular filling (MS, left atrial myxoma)
- Acute MR (chordal rupture)
- Acute aortic insufficiency

CARDIOGENIC SHOCK

OTHER ETIOLOGIES

- AMI most common etiology, must also consider other cardiac causes and extracardiac causes

- Consideration of these non-ischemic etiologies serve as a reminder that while CS is typically accompanied by acutely reduced ejection fraction, CS is a physiological condition of depressed CO ill-defined in anatomical terms and may occur with only moderately impaired ejection fraction.

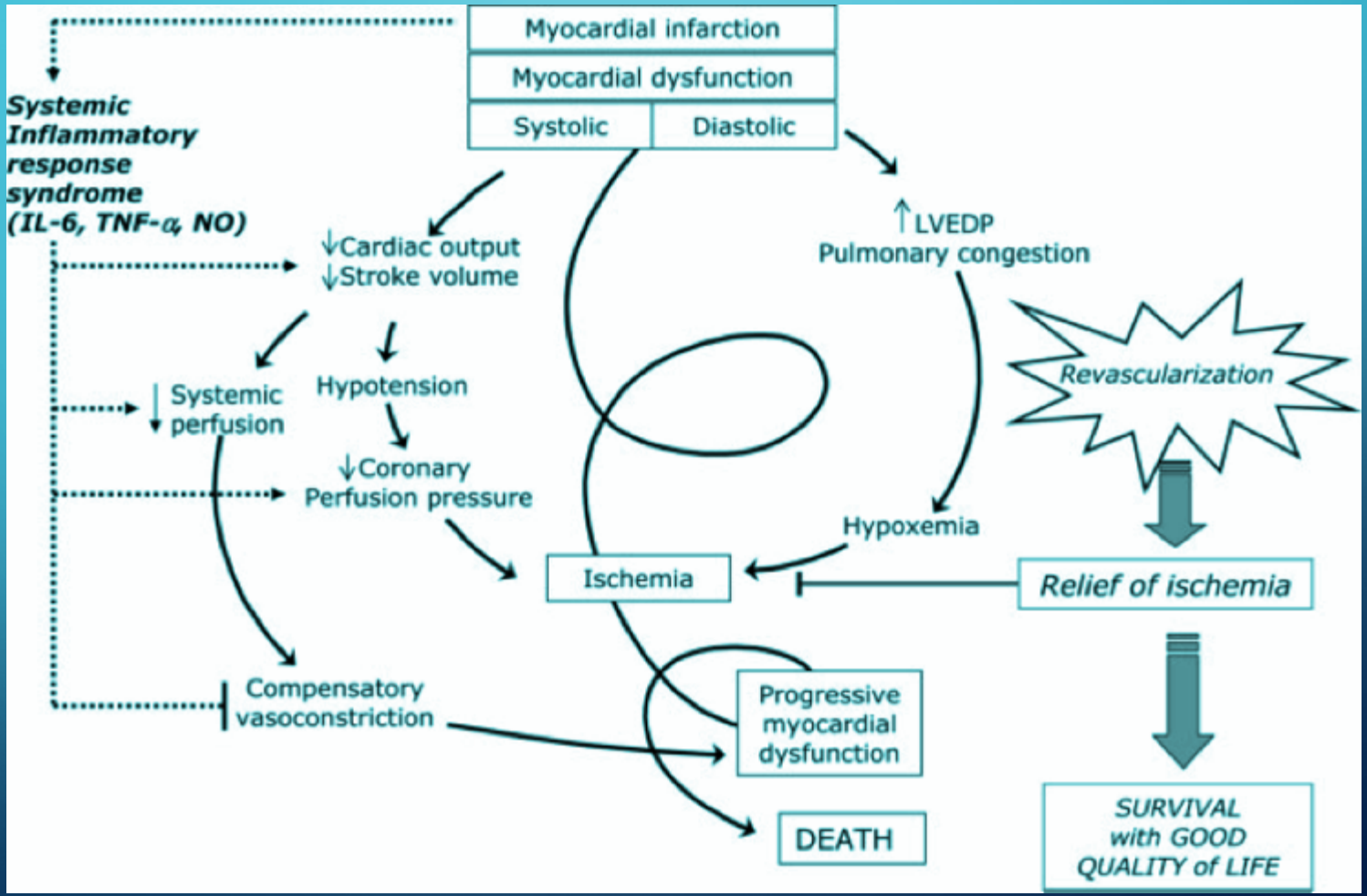
CARDIOGENIC SHOCK

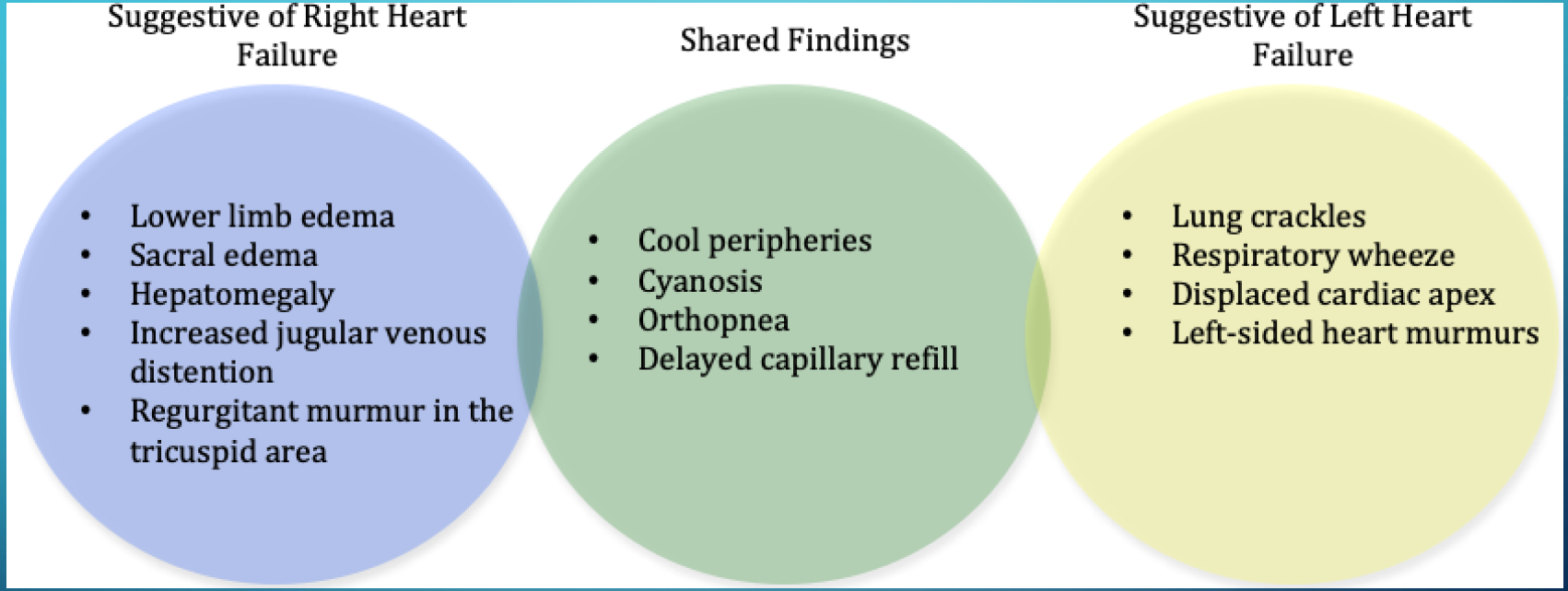
- Local and systemic release of catecholamines transiently increase myocardial chronotropy and inotropy at the cost of increased rates of arrhythmia and peripheral vasoconstriction, exacerbating afterload and myocardial perfusion mismatch.
- The systemic inflammatory response system, in contrast, causes inappropriate vasodilation, capillary leak, microvascular dysfunction and hypoperfusion of end organs, most gravely in the intestinal tract, which predisposes to gut translocation and sepsis

CARDIOGENIC SHOCK

OTHER ETIOLOGIES

- Tumour necrosis factor-α and interleukin 6 (SIRS cascade), are further proinflammatory and cardio-depressive.
- Neurohormonal activation of the renin-angiotensin system results in increased salt and water retention adding to the burden of preload and decompensated heart failure.
- The ability to interrupt these processes and prevent or reverse the extension of myocardial injury before unsalvageable damage is central to the hypothesis that CS is treatable.
- The goal of therapies is thus to rescue, support and optimise the remaining viable myocardium


Myocardial infarction
Myocardial dysfunction
Systolic
Diastolic
Systemic Inflammatory response syndrome (IL-6, TNF-α, NO)
↓Cardiac output
↓Stroke volume
↑LVEDP
Pulmonary congestion
Revascularization
↓ Systemic perfusion
Hypotension
↓Coronary Perfusion pressure
Hypoxemia
Ischemia
Relief of ischemia
Compensatory vasoconstriction
Progressive myocardial dysfunction
DEATH
SURVIVAL with GOOD QUALITY of LIFE



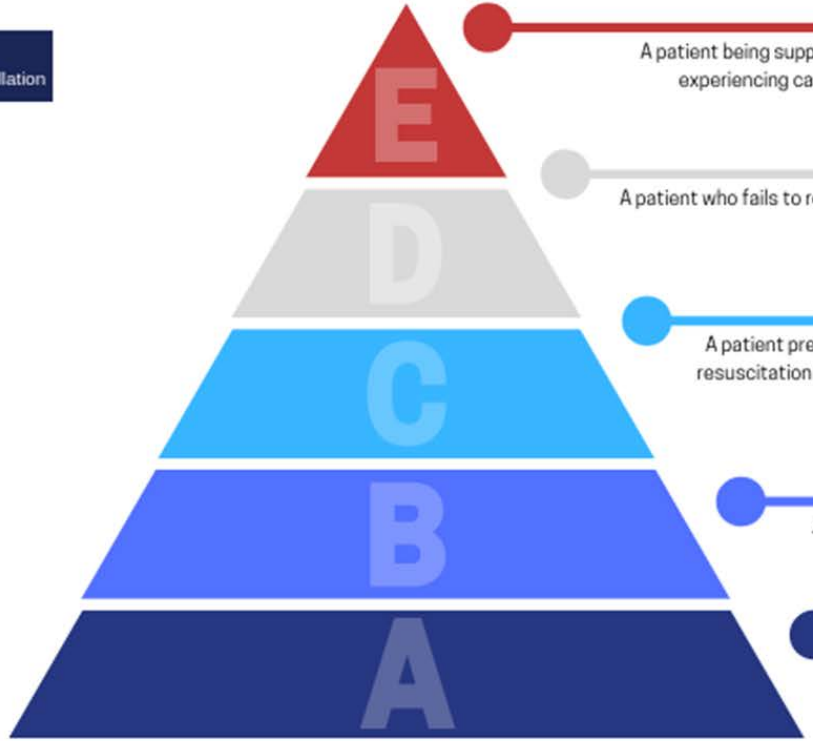
Cyrus Vahdatpour. Journal of the American Heart Association. Cardiogenic Shock, Volume: 8, Issue: 8, DOI: (10.1161/JAHA.119.011991)

SCAI
Society for Cardiovascular Angiography & Interventions

SCAI Stages of Cardiogenic Shock

Adapted from the SCAI Clinical Expert Consensus Statement on the Classification of Cardiogenic Shock
Endorsed by ACC, AHA, SCCM, and STS

Arrest (A) Modifier:
CPR, including defibrillation



EXTREMIS

A patient being supported by multiple interventions who may be experiencing cardiac arrest with ongoing CPR and/or ECMO.

DETERIORATING

A patient who fails to respond to initial interventions. Similar to stage C and getting worse.

CLASSIC

A patient presenting with hypoperfusion requiring intervention beyond volume resuscitation (inotrope, pressor, or mechanical support including ECMO). These patients typically present with relative hypotension.

BEGINNING

A patient who has clinical evidence of relative hypotension or tachycardia without hypoperfusion.

AT RISK

A patient with risk factors for cardiogenic shock who is not currently experiencing signs or symptoms. For example, large acute myocardial infarction, prior infarction, acute and/or acute on chronic heart failure.

Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock. Catheter Cardiovasc Interv. 2019;1–9. https://doi.org/10.1002/ccd.28329
For more information, please visit: www.scai.org/shockdefinition

STAGE A: AT RISK

- A patient who is not currently experiencing signs or symptoms of CS but is at risk for its development.
- These patients may include those with NSTEMI, STEMI, acute or acute on chronic CHF.

Physical Exam	Biochem Markers	Hemodynamics
Normal JVP	Normal Labs	Normotensive
Clear Lungs	Normal renal function	SBP ≥ 100 or normal for pt
Warm/ Well Perfused	Normal lactic acid	If hemodynamics done
Strong distal pulses		CI ≥ 2.5
Normal mentation		CVP < 10
		PA Sat ≥ 65%

STAGE B: BEGINNING

- A patient who has clinical evidence of relative hypotension or tachycardia ***without hypoperfusion.***

Physical Exam	Biochem Markers	Hemodynamics
Elevated JVP	Normal lactate	SBP <90 OR MAP<60 or >30 mm drop from baseline
Rales in Lung fields	Normal renal function	Pulse ≥ 100
Warm/ Well Perfused	Elevated BNP	If hemodynamics done
Strong distal pulses		CI ≥ 2.2
Normal mentation		PA Sat ≥ 65

STAGE C: CLASSIC

- A patient ***with hypoperfusion*** that requires interventions such as inotrope, pressor, or perc. MCS to restore perfusion.
- These patients typically have relative hypotension.

Physical Exam May Include any of:	Biochem Markers: May Include any of	Hemodynamics: May Include any of
Looks unwell, panicked	Lactate ≥ 2	SBP<90 or MAP < 60 or > 30 mm drop from baseline AND drugs/ device used to maintain BP above these
Ashen, mottled, dusky	Creatinine doubling or > 50 % loss of GFR	Hemodynamics CI < 2.2
Volume overload Extensive rales Killip 3 /4	Increased LFT's	PCW > 15
BiPAP or mechanical vent	Increased BNP	RAP / CVP ≥ 0.8
Cold, clammy Urine output < 30 mL/h		PAPI < 1.85
Acute alteration of mental status		CPO ≤ 0.6

STAGE D: DETERIORATING

- Patients similar to C but are getting worse.
- These patients have ***failure to respond*** to initial interventions.

Physical Exam May Include any of:	Biochem Markers: May Include any of	Hemodynamics: May Include any of
Looks unwell, panicked	Lactate ≥ 2	SBP<90 or MAP < 60 or > 30 mm drop from baseline AND drugs/ device used to maintain BP above these
Ashen, mottled, dusky	Creatinine doubling or > 50 % loss of GFR	Hemodynamics CI < 2.2
Volume overload Extensive rales Killip 3 /4	Increased LFT's	PCW > 15
BiPAP or mechanical vent	Increased BNP	RAP / CVP ≥ 0.8
Cold, clammy Urine output < 30 mL/h	**DETERIORATING**	PAPI < 1.85 CPO ≤ 0.6
Acute alteration of mental status		**Requiring multiple pressors OR addition of MCS to maintain perfusion**

STAGE E: EXTREMIS

- Patient in cardiac arrest with ongoing CPR or ECLS placement.
- Alternately, being supported by multiple interventions.

Physical Exam May Include any of:	Biochem Markers: May Include any of	Hemodynamics: May Include any of
"Trying to die"	Lactate ≥ 5	No blood pressure without CPR
Cardiac collapse	Arterial pH ≤ 7.2	PEA or refractory VT/VF
Mechanical Vent	Increased LFT's	Hypotension despite max support
BiPAP or mechanical vent	Increased BNP	
Defibrillated	No time to draw	

SCAI SHOCK STAGE	PHYSICAL EXAM	BIOCHEMICAL MARKERS	HEMODYNAMICS
A	Normal JVP Lung sounds clear Strong distal pulses Normal mentation	Normal renal function Normal lactic acid	Normotensive (SBP>100 or normal for pt.) If hemodynamics done: • Cardiac index >2.5 • CVP <10 • PA Sat >65%
B	Elevated JVP Rales in lung fields Strong distal pulses Normal mentation	Normal lactate Minimal renal function impairment Elevated BNP	SBP <90 OR MAP <60 OR >30mmHg drop Pulse >100 If hemodynamics done: • Cardiac Index >2.2 • PA Sat >65%
C	Ashen, mottled, dusky Volume overload Extensive Rales Killip class 3 or 4 BiPap or mechanical ventilation Acute alteration in mental status	Lactate >2 Creatinine doubling OR >60% drop in GFR Increased LFTs Elevated BNP Urine Output <30mL/h	Drugs/device used to maintain BP above stage B values. • Cardiac Index <2.2 • PCWP >15 • RAP/PCWP >0.8 • PAPI <1.85 • Cardiac Power Output <0.6
D	Any of stage C	Any of stage C AND deteriorating	Any of stage C AND Requiring multiple pressors OR addition of mechanical circulatory support devices to maintain perfusion
E	Near pulselessness Cardiac collapse Mechanical ventilation Defibrillator used	Lactate >5 pH < 7.2	No SBP without resuscitation PEA or Refractory VT/VF Hypotension despite maximal support

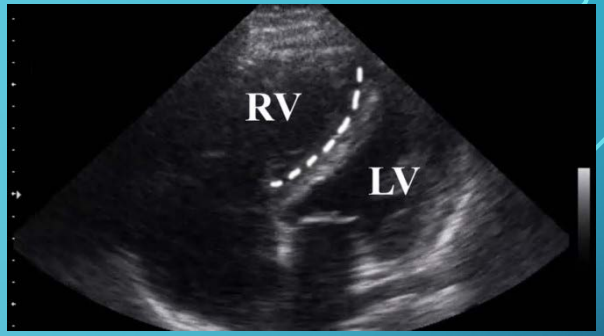
SCAI
Society for Cardiovascular Angiography & Interventions

CARDIOGENIC SHOCK

LEFT VENTRICLE EJECTION FRACTION (LVEF)

- LV ejection fraction (LVEF) may be only moderately depressed in CS
- In the SHOCK (SHould we emergently revascularize Occluded coronaries for Cardiogenic shocK) trial mean LVEF was 30%
- Some patients present with CS despite preservation of LVEF in the absence of severe mitral regurgitation.
- Among patients in shock, however, LVEF remains a prognostic indicator
- Approximately half of all CS patients have small or normal LV size which represents failure of the adaptive mechanism of acute dilation to maintain stroke volume in the early phase of MI. Progressive LV dilation (remodeling) in the chronic phase can be maladaptive

CARDIOGENIC SHOCK



RIGHT VENTRICLE

- RV shock occurs in 5% of cases of cardiogenic shock complicating MI
- Treatment is focused on ensuring adequate right-sided filling pressures to maintain CO and adequate LV preload; however, patients with CS due to RV dysfunction have very high RV end-diastolic pressure, often 20 mm Hg.
- This elevation of RV end-diastolic pressure may result in shifting of the interventricular septum toward the LV cavity, which raises left atrial pressure but impairs LV filling due to the mechanical effect of the septum bowing into the LV. This alteration in geometry also impairs LV systolic function

CARDIOGENIC SHOCK

- Inotropic therapy is indicated for RV failure when CS persists after RV end-diastolic pressure has been optimized. RV end-diastolic pressure of 10 to 15 mm Hg has been associated with higher output than lower or higher pressures but marked variability exists in optimal values.
- iNO helps facilitate forward flow due to decreased pulmonary vascular resistance
- Mortality due to RV shock is equivalent to LV shock

CARDIOGENIC SHOCK

NEUROHORMONAL & INFLAMMATORY MEDIATORS

- Vasopressin and angiotensin II levels increase in the setting of MI and shock → improvement in coronary and peripheral perfusion at the cost of increased afterload → which may further impair myocardial function.
- Activation of the neurohormonal cascade promotes salt and water retention → this may improve perfusion but exacerbates pulmonary edema
- MI can cause the systemic inflammatory response syndrome (SIRS) and suggest that inappropriate vasodilation as part of SIRS results in impaired perfusion of the intestinal tract, which enables transmigration of bacteria and sepsis
- Cytokine levels rise more dramatically over the 24 to 72 hours after MI. Tumor necrosis factor and interleukin-6 have myocardial depressant action.
- TNF-alpha also induces coronary endothelial dysfunction, which may further diminish coronary flow

DIABETES AND CARDIOGENIC SHOCK

- In a large cohort of acute myocardial infarction patients, preexisting diabetes was associated with an increased risk of cardiogenic shock and worse outcomes in those with cardiogenic shock.
- 42.8% (n = 31,135) of patients with acute myocardial infarction and cardiogenic shock having diabetes.

Am J Med. 2018 Jul;131(7):778-786.e1

CARDIOGENIC SHOCK

DIABETES

- Myocardial ischemia → massive secretion of catecholamines, via the sympathetic system and adrenal glands → increases blood glucose levels by stimulating hepatic glycogenolysis and gluconeogenesis, decreasing peripheral glucose use and insulin sensitivity.
- Tumor necrosis factor, interleukin -1 and interleukin-6 → stimulate the hypothalamus–pituitary axis to produce corticotropin releasing hormone and adrenocorticotropic hormone → that act directly on the adrenal cortex to increase glucocorticoid synthesis,→ which also increases blood glucose level

CARDIOGENIC SHOCK AND DIABETES

- In patients without diabetes, those with glucose concentrations between 110 and 143 mg/dL (6.1 to 8 mmol/L) had a 3.9-fold higher risk of death compared to patients with lower glucose concentrations.
- Glucose values between 144 and 180 mg/dL (8 to 10 mmol/L) were associated with a three-fold higher risk of heart failure or cardiogenic shock.
- Diabetic patients with glucose concentrations ≥180 to 196 mg/dL (10 to 11 mmol/L) also had an increased risk of death compared with normoglycemic diabetic patients (relative risk 1.7), but this relative risk was lower than in non-diabetics.

CARDIOGENIC SHOCK

MORTALITY RATES WITH CARDIOGENIC SHOCK & DIABETES

- In patients with AMI and cardiogenic shock, diabetes was associated with a trend for increased in-hospital mortality (odds ratio, 2.82; 95% confidence interval [CI], 0.90-9.92; P = .08).
- In 73 patients with cardiogenic shock, estimated survival at 1, 3, and 5 years was 25%, 17%, and 17%, respectively, for diabetic patients, and 50%, 44%, and 36%, respectively, for nondiabetic patients (P = .046).
- The association between diabetic patients and increased long-term mortality was stronger in patients with cardiogenic shock than in patients without cardiogenic shock (adjusted relative risk, 2.08; 95% CI, 1.11-3.90; P = .02)

CARDIOGENIC SHOCK

MORTALITY RATES WITH CARDIOGENIC SHOCK & DIABETES

- Over the 15-year study period, the researchers found that revascularization rates increased for patients with and without diabetes (both P <.0001), but rates were lower for those with diabetes. Patients with diabetes more often received only medical therapy compared with those without diabetes (42.3% vs 36.1%; P <.0001).
- Patients with diabetes had higher all-cause in-hospital mortality compared with those without diabetes (5.4% vs 2.5%; P <.0001). In addition, mortality rates decreased more consistently for patients without diabetes and remained ≥2-fold higher in patients with diabetes across all study years.

CARDIOGENIC SHOCK

METHODS & RESULTS

- Baseline characteristics and in-hospital complications to the infarction were prospectively recorded in 6676 patients with MI.
- Ten-year mortality was collected. Diabetes was present in 10.8% of the total population. A total of 443 developed cardiogenic shock with an incidence of 6.2% among nondiabetics and 10.6% among diabetics.
- Age, wall motion index, reinfarction, and the absence of thrombolytic treatment were significant independent predictors of mortality in patients with cardiogenic shock.
- Intriguingly, diabetes was not a significant predictor for short- and long-term mortality in this population.
- The 30-day and 5-year mortality rate was equally poor in both diabetic and nondiabetic patients with cardiogenic shock (diabetics: 30-day 63%, 5-year 91%; nondiabetics: 30-day 62%, 5-year 86%; p 0.05).

CARDIOGENIC SHOCK

	Diabetes	Non-Diabetes
Incidence of Cardiogenic Shock	10.6%	6.2%
30-Day Mortality with CS	63%	62%
5-Year Mortality with CS	91%	86%

CARDIOGENIC SHOCK

CONCLUSIONS

- Cardiogenic shock develops approximately twice as often among diabetics as among nondiabetic patients with acute MI.
- The prognosis of diabetics with cardiogenic shock is similar to the prognosis of nondiabetic patients with cardiogenic shock.

KEY CONSIDERATIONS IN THE DIAGNOSIS AND MANAGEMENT OF CARDIOGENIC SHOCK

- Is this cardiogenic shock?
- What is the severity
- Is it predominantly LV or RV or both?
- What are the support options?

CARDIOGENIC SHOCK

MANAGEMENT

- PA catheter is commonly used
- Vasopressor support often needed
- May need intra-aortic balloon pump
- Hypotension may or may not be present
- Identification of type of cardiogenic shock critical for optimal management

CARDIOGENIC SHOCK

MANAGEMENT FOLLOWING MI

- Most common cause of cardiogenic shock
- Directed therapy for MI
- ASA, heparin, glycoprotein IIb/IIIa inhibitors, revascularization

CARDIOGENIC SHOCK

REVASCULARIZATION

- Most patients who develop cardiogenic shock do so within 48 hrs of admission, with only 10% shocked on arrival.
- Mortality rate is exceedingly high and reaches 70-80% in those treated conservatively.
- Early revascularization is the cornerstone treatment of acute myocardial infarction complicated by cardiogenic shock.
- Revascularization is effective up to 36 hours after the onset of cardiogenic shock and performed within 18 hours after the diagnosis of cardiogenic shock.
- Primary percutaneous coronary intervention is the most efficient therapy to restore coronary flow in the infarct-related artery.

TRIALS

THE SHOCK TRIAL

The Should we emergently revascularize Occluded Coronaries for cardiogenic shock (SHOCK) Trial failed to demonstrate mortality benefit of early revascularization over initial medical stabilisation at 30 days; however, significant mortality benefit with early revascularization was seen at 6 months, 1 year and 6 years.

THE CS (IABP-SHOCK II) TRIAL

In one of the largest randomised trials of patients with CS, the intraaortic balloon counterpulsation in AMI complicated by the CS (IABP-SHOCK II) Trial, 600 patients were randomised to either IABP or medical therapy. No significant difference was seen in the primary end point of 30-day all-cause mortality. Follow-up analysis also showed no difference in mortality at 12 months.

THE IMPRESS TRIAL

The Impella CP vs IABP in AMI complicated by cardiogenic shock (IMPRESS) Trial, showed no difference in the primary end point of 30-day mortality with use of the Impella CP device compared with the IABP

TRIALS

CATHETER BASED VENTRICULAR ASSIST DEVICE REGISTRY ANALYSIS

(PROSPECTIVE)

- Early MCS implantation before starting inotrope/vasopressor support and before PCI independently associated with improved survival rates

DETROIT CARDIOGENIC SHOCK INITIATIVE

(RCT)

- Reporting 76% survival rates
- Improvement on stagnant ≈50% mortality rates over the past 2 decades
- 2018 and ongoing

PA CATHETER MEASUREMENTS

Hemodynamic Parameters	Abbreviations	Normal Values
Mean Arterial Pressure	MAP	70-90 mm Hg
Right Atrial Pressure	RAP	2-6 mm Hg
Central Venous Pressure	CVP	2-6 mm Hg
Pulmonary Artery Systolic Pressure	PAS	20-30 mm Hg
Pulmonary Artery Diastolic Pressure	PAD	6-12 mm Hg
Pulmonary Artery Wedge Pressure	PAWP, Wedge, PAOP	8-12 mm Hg
Cardiac Output	CO	4-8 L/min
Cardiac Index	CI	2.5-4 L/min
Stroke Volume	SV	60-130 ml
Stroke Volume Index	SVI	40-50 ml/m2
Systemic Vascular Resistance	SVR	800-1200 dynes
Systemic Vascular Resistance Index	SVRI	2000-2400 dynes
Pulmonary Vascular Resistance	PVR	150-300 dynes

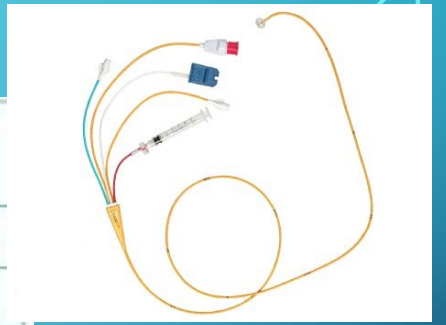


Table 2.Summary of Systemic Vasopressors

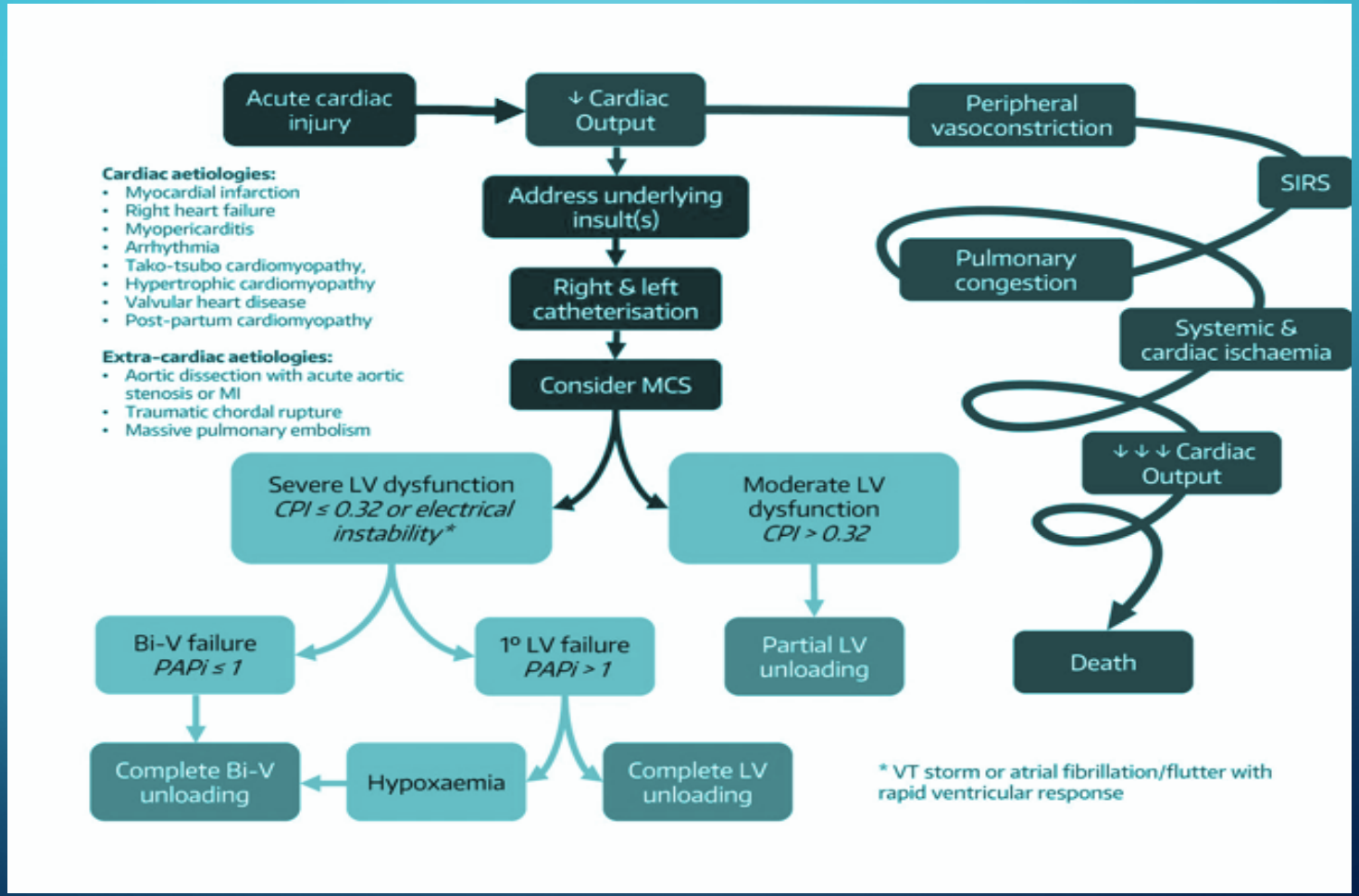
Agents	Mechanism	Effect	Indications	Considerations
Phenylephrine	A1 agonist	Vasoconstriction	Various forms of shock	Caution in cardiac dysfunction as it increases afterload
Norepinephrine	A<B agonist	Inotropy, chronotropy, dromotropy, and vasoconstriction	Most common first line agent in shock	Most benefits demonstrated in septic shock
Epinephrine	A≪B agonist	Inotropy, chronotropy, dromotropy, and vasoconstriction	Commonly used as second line agent or first line in anaphylactic shock	Surviving Sepsis Guidelines has most data for epinephrine as second line agent
Dopamine	Dose dependent A, B, and D agonism	Inotropy, dromotropy, chronotropy, and vasoconstriction (at highest doses)	Second line agent in most forms of shock	SOAP II trial demonstrated more incidence of tachy-arrythmias and increased mortality in CS patients when dopamine was used as first line
Vasopressin	V1 agonist	Vasoconstriction	Second line agent in most forms of shock	On or Off dosing, can cause hyponatremia
Dobutamine	B agonist	Inotropy and mild vasodilation	Commonly used in cardiogenic shock	May contribute to hypotension
Levosimendan	Myofilament Ca^{2+} sensitizer and K^{+} channel modifier	Ionotropy and inodilator	Used in acutely decompensated chronic heart failure	Minimal effect on myocardial oxygen consumption

CS indicates cardiogenic shock; SOAP, Sepsis Occurrence in Acutely Ill Patients.

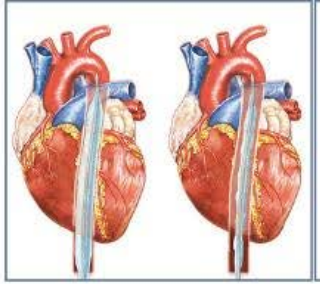
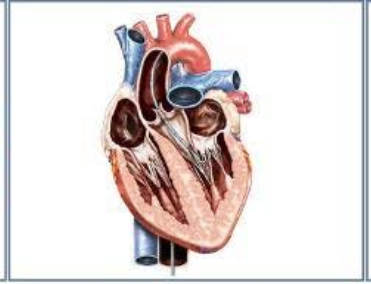
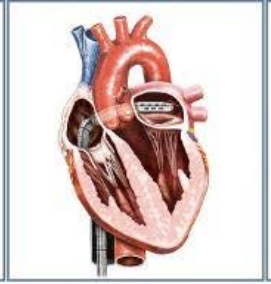
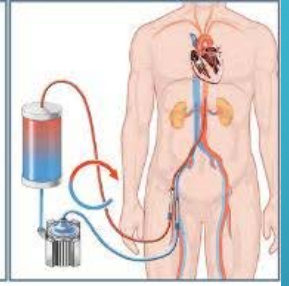
CARDIAC POWER OUTPUT AND INDEX

Univariate and multivariate analysis of the SHOCK Trial registry data identified CPO <0.53 and CPI<0.33 as the strongest independent haemodynamic correlates of in-hospital mortality in CS.

Cardiac power output (CPO) (calculated as mean arterial pressure (MAP) × CO/451) and Cardiac Power Index (CPI) (calculated as MAP × Cardiac Index/451) where MAP = ((systolic blood pressure – diastolic blood pressure) / 3) + diastolic blood pressure



Jones TL, et al. Open Heart 2019;6:e000960. doi:10.1136/openhrt-2018-000960

	IABP	IMPELLA	TANDEMHEART	VA-ECMO
Cardiac Flow	0.3-0.5 L/ min	1-5L/ min (Impella 2.5, Impella CP, Impella 5)	2.5-5 L/ min	3-7 L-min
Mechanism	Aorta	LV → AO	LA → AO	RA → AO
Maximum implant days	Weeks	7 days	14 days	Weeks
Sheath size	7-8 Fr	13-14 Fr Impella 5.0 - 21 Fr	15-17 Fr Arterial 21 Fr Venous	14-16 Fr Arterial 18-21 Fr Venous
Femoral Artery Size	>4 mm	Impella 2.5 & CP - 5-5.5 mm Impella 5 - 8 mm	8 mm	8 mm
Cardiac synchrony or stable rhythm	Yes	No	No	No
Afterload	↓	↓	↑	↑↑↑
MAP	↑	↑↑	↑↑	↑↑
Cardiac Flow	↑	↑↑	↑↑	↑↑
Cardiac Power	↑	↑↑	↑↑	↑↑
LVEDP	↓	↓↓	↓↓	↔
PCWP	↓	↓↓	↓↓	↔
LV Preload	---	↓↓	↓↓	↓
Coronary Perfusion	↑	↑	---	---
Myocardial oxygen demand	↓	↓↓	↔↓	↔

SUMMARY

- Recognize the various forms of shock
- Understand the pathophysiology of shock
- Recognize the physical exam findings associated with cardiogenic shock
- Recognize there are cardiac and non-cardiac causes of cardiogenic shock
- Understand the importance of glycemic control in patients susceptible to the development of cardiogenic shock
- Recognize the various mechanical cardiac support devices available

THANK YOU