شوک کاردیوژنیک و انسدادی

دکتر محمودرضا زینت زاده

استادیار طب اورژانس دانشگاه علوم پزشکی تهران

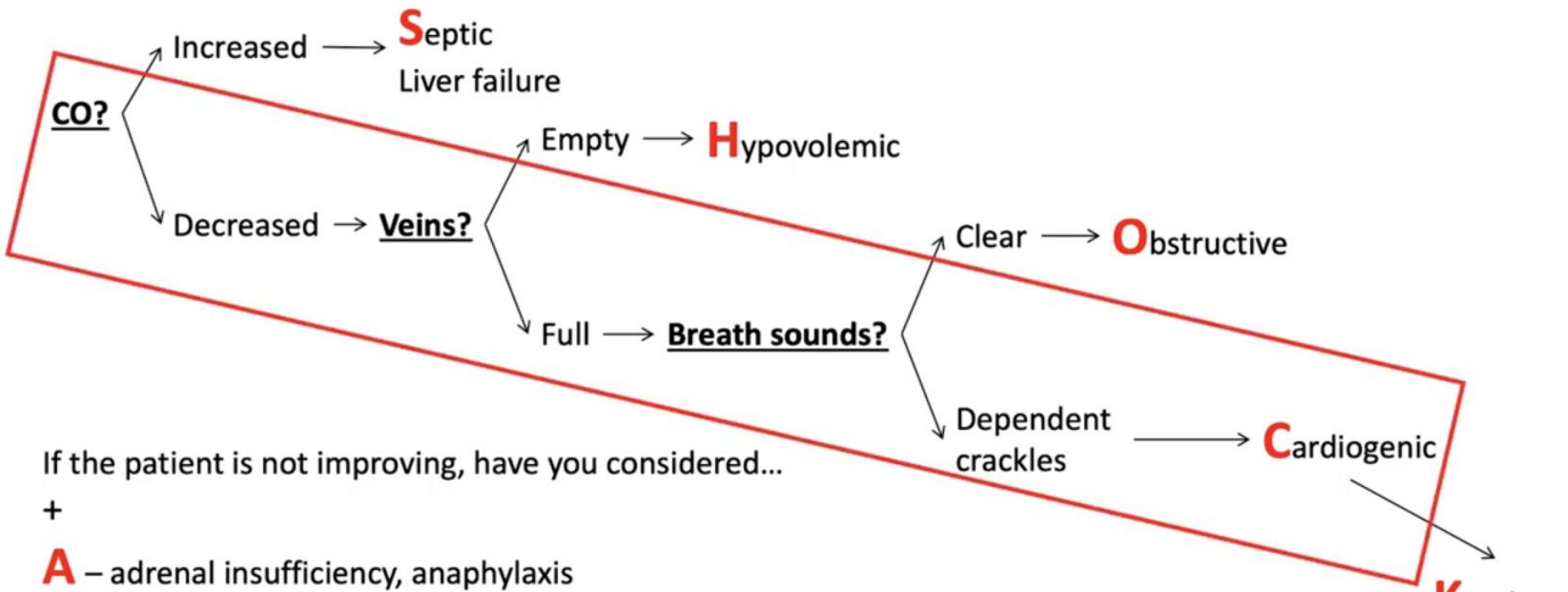
Case

- 50-year-old man with DM and HTN presents with anterior STEMI
- Taken emergently to the cath lab, stents placed in LAD
- Transferred to MICU for post-procedure monitoring
- Initial vitals: BP 75/50 (MAP 58), HR 110, SpO_2 85% on RA
- Physical exam: lethargic, elevated JVP, increased work of breathing, bilateral crackles, 2+ bilateral lower extremity edema, and cool extremities

Objectives

1. Define cardiogenic shock.
2. Describe the pathophysiologic abnormalities and clinical manifestations of cardiogenic shock based on the Forrester classification system.
3. Describe the treatment principles for a patient in cardiogenic shock, including inotropes, afterload reduction, and diuresis.

Physical Exam-Based Approach to Shock



If the patient is not improving, have you considered…

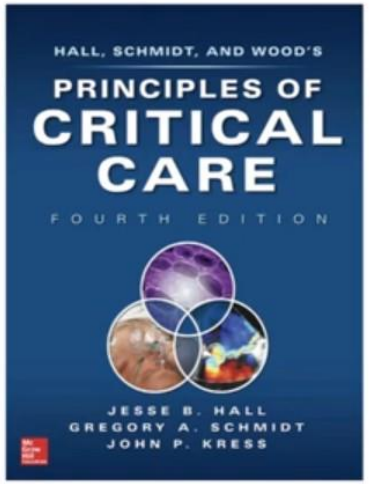
+

A – adrenal insufficiency, anaphylaxis

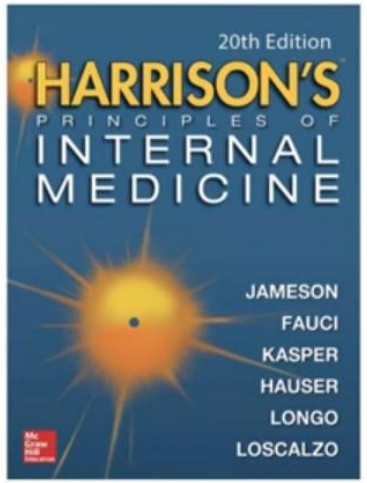
WE – Weird electrolytes/vitamins (hypoCa, thiamine), "we did it" (sedatives, vasoactive drugs)

Adapted with permission from Jesse B. Hall, Gregory A. Schmidt, John P. Kress: Principles of Critical Care, 4th Edition, "Chapter 33: Shock", Figure 33-1 by Keith R. Walley.

Cardiogenic Shock



"Circulatory failure due to pump dysfunction"

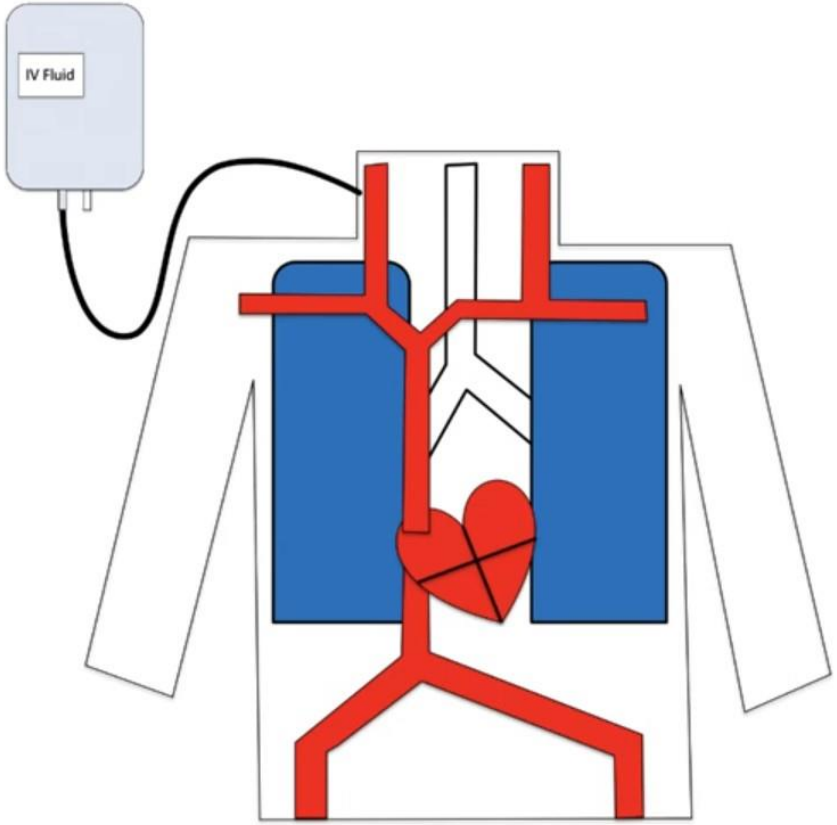


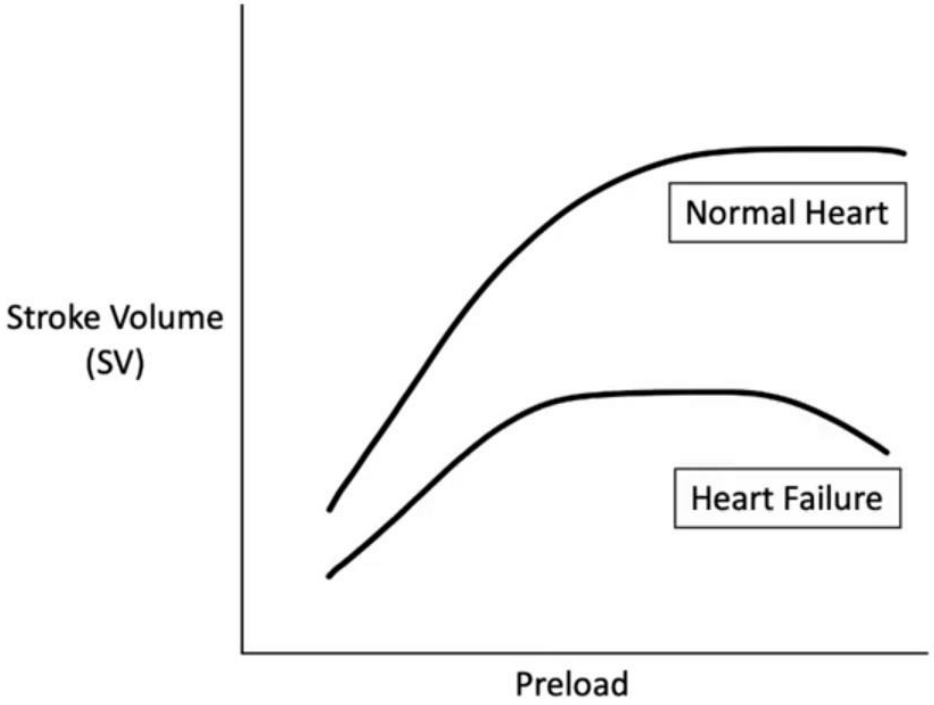
"Low cardiac output state resulting in life-threatening end-organ hypoperfusion and hypoxia"

→

- Persistent hypotension
- Unresponsive to volume replacement *or* worsens with volume replacement
- Elevated lactate

Frank-Starling Curve





Etiology of Cardiogenic Shock

MAP = CO x SVR

↓

SV x HR

↓

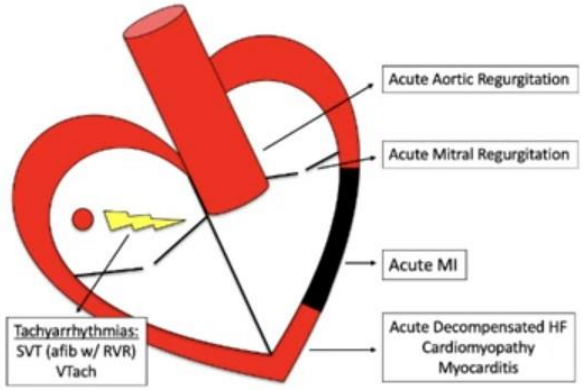
Dysfunctional:

Myocardium

Valves

Electrical System

Etiology of Cardiogenic Shock



MAP = CO x SVR

SV x HR

Dysfunctional:

Myocardium

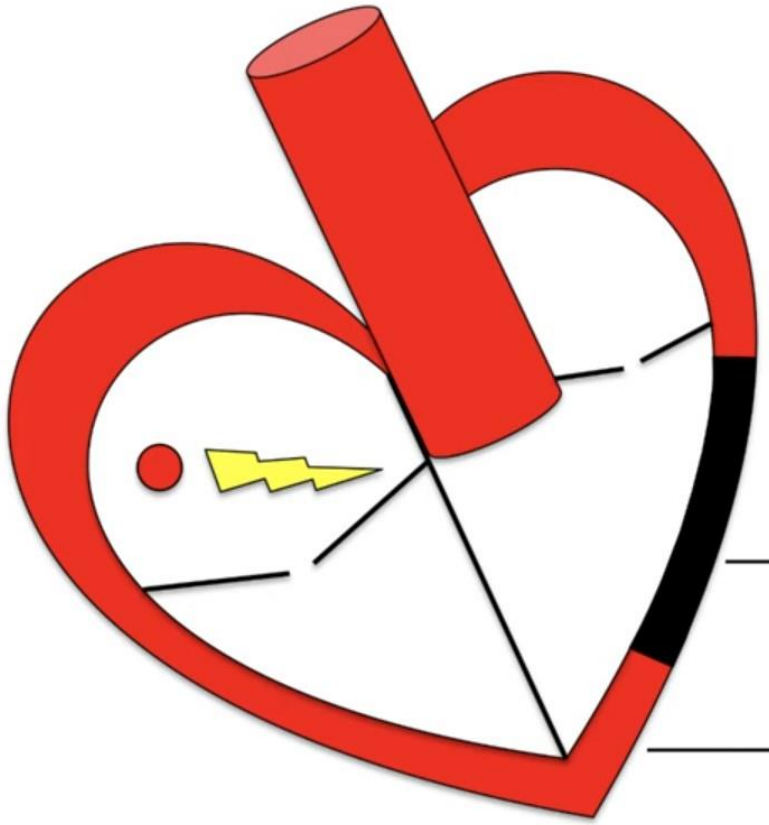
Valves

Electrical System

Heart Block

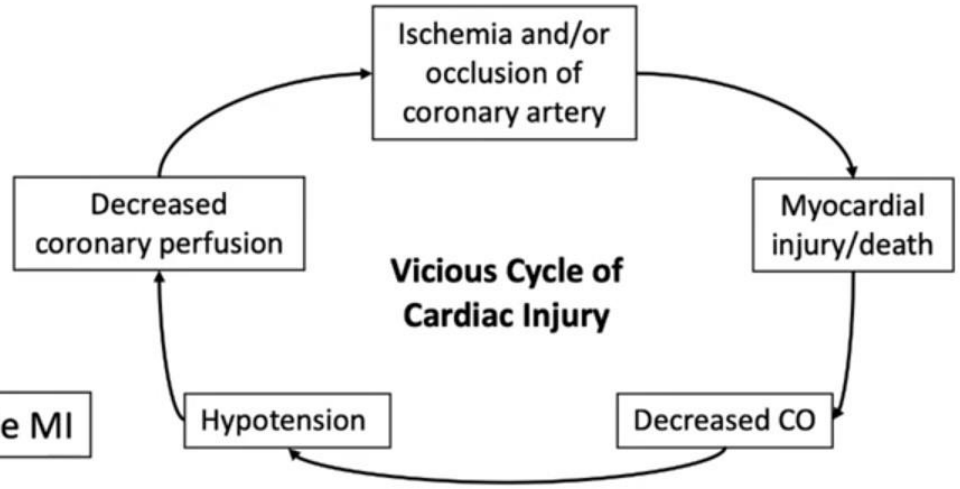
Drug Overdoses (BB, CCB)

Myocardial Dysfunction

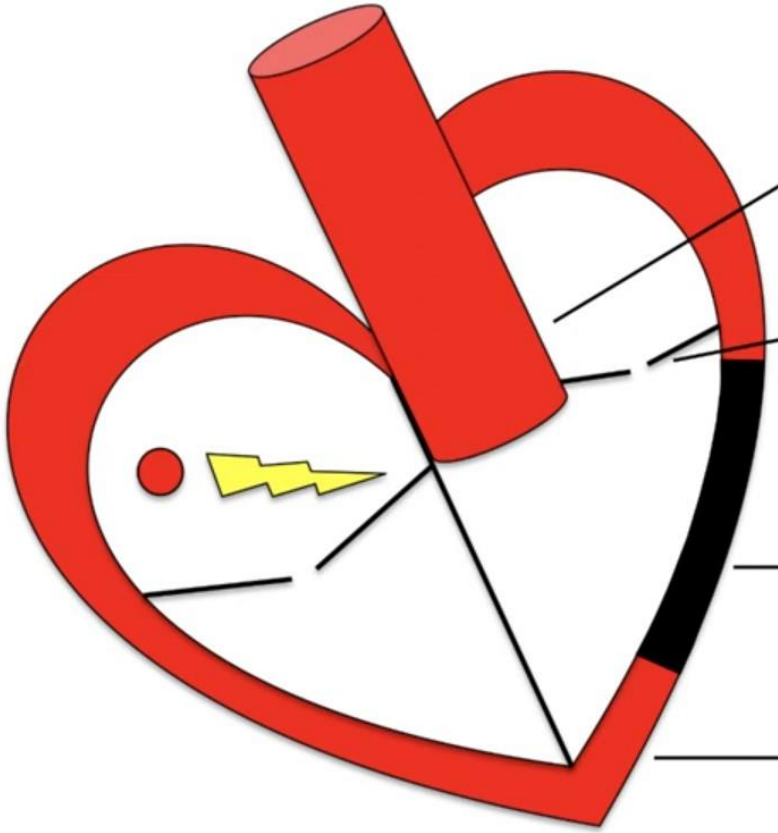


Acute MI

Acute Decompensated HF
Cardiomyopathy
Myocarditis



Valvular Dysfunction



Acute Aortic Regurgitation

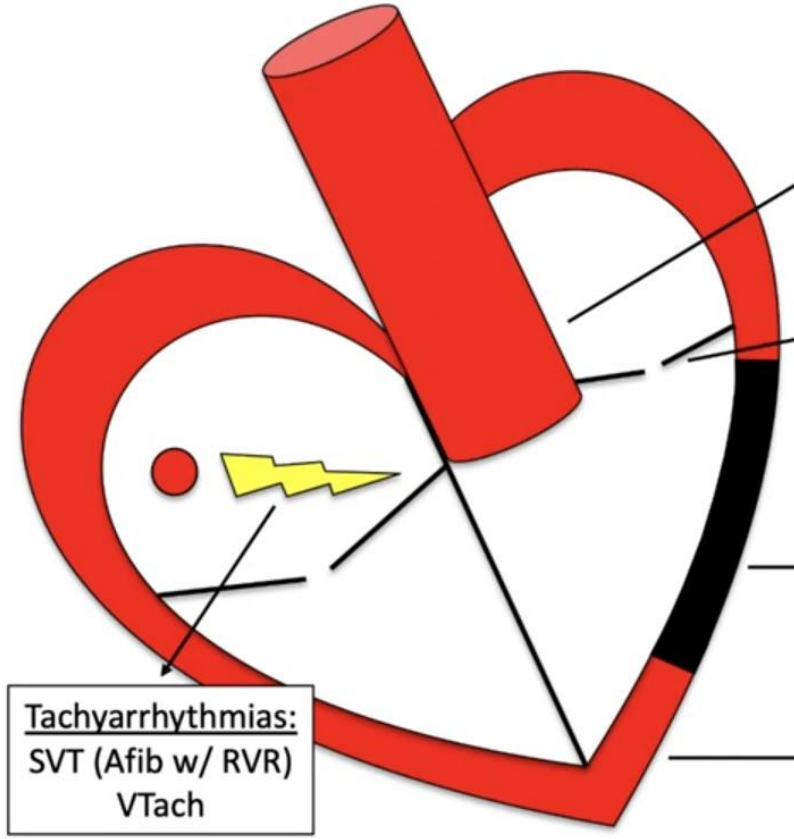
Acute Mitral Regurgitation

Acute MI

Acute Decompensated HF
Cardiomyopathy
Myocarditis

Reflux of blood decreases effective SV and CO

Electrical System Dysfunction



Acute Aortic Regurgitation

Acute Mitral Regurgitation

Acute MI

Acute Decompensated HF
Cardiomyopathy
Myocarditis

<u>Tachyarrhythmias:</u>
SVT (Afib w/ RVR)
VTach

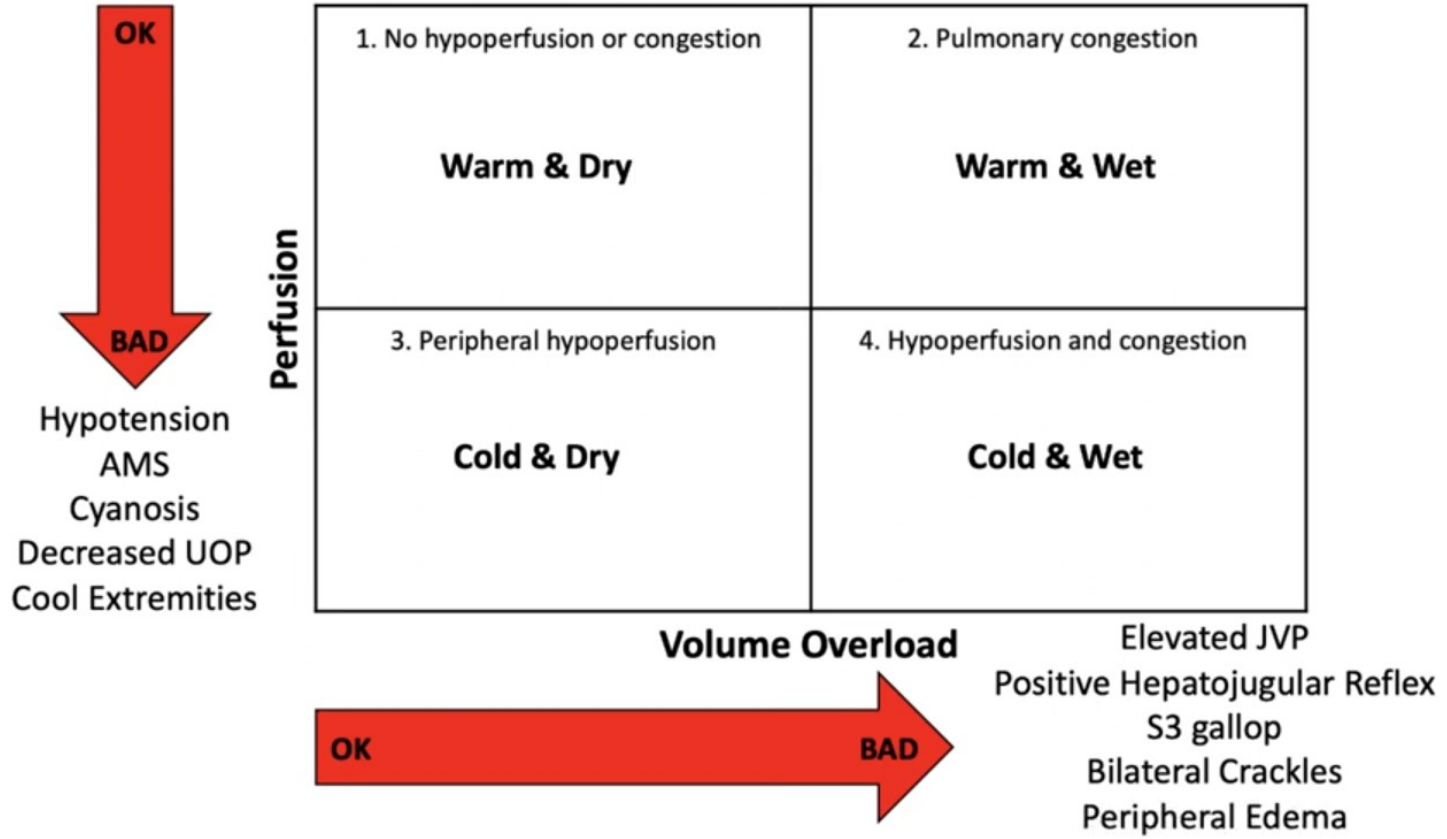
Increased HR prevents adequate diastolic filling

83% accuracy rate for “clinical evaluation as a predictor of hemodynamic state”

Classifications:

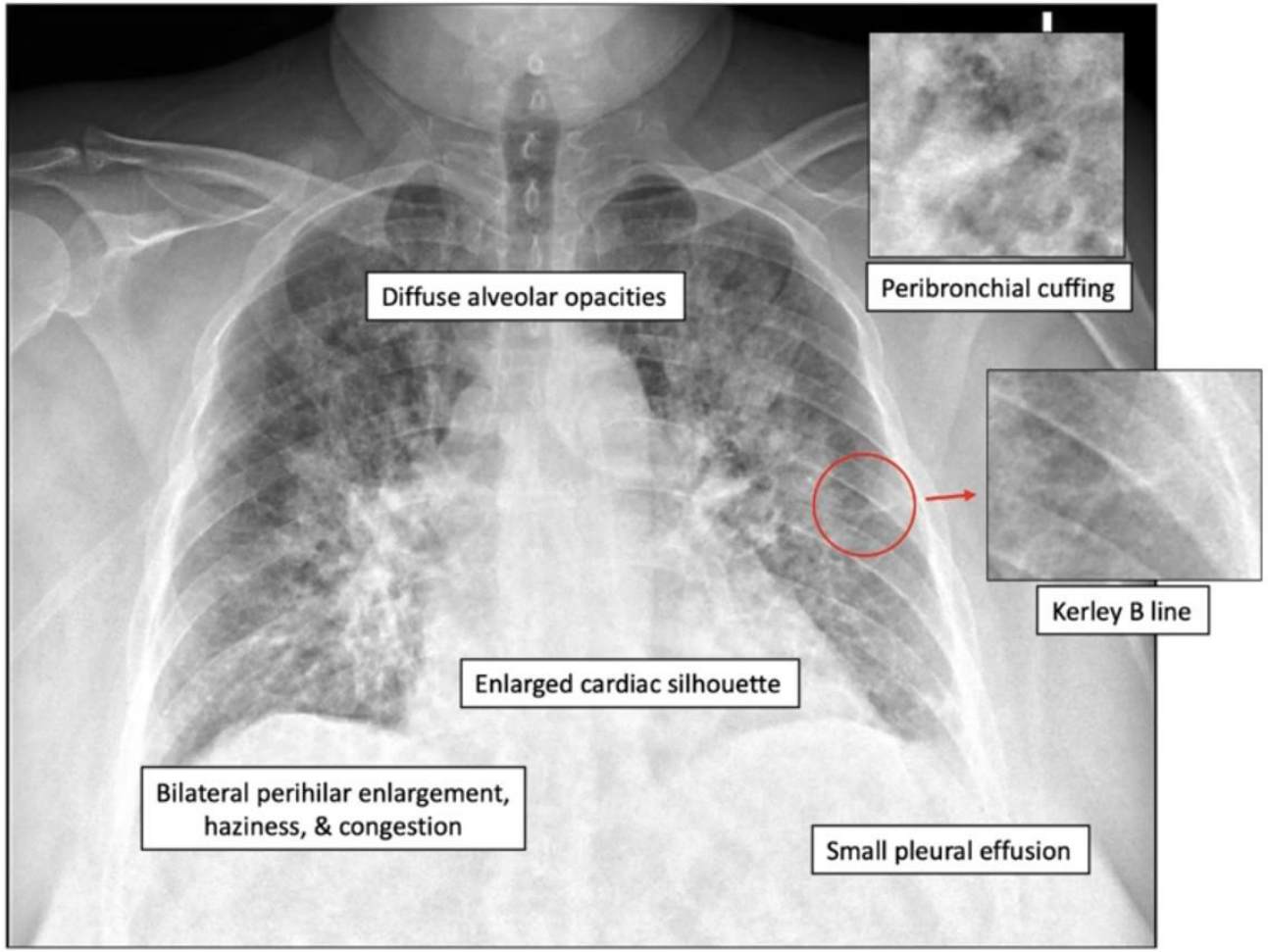
1. No peripheral hypoperfusion or pulmonary congestion
2. Pulmonary congestion
3. Peripheral hypoperfusion
4. Peripheral hypoperfusion and pulmonary congestion

Forrester Classification



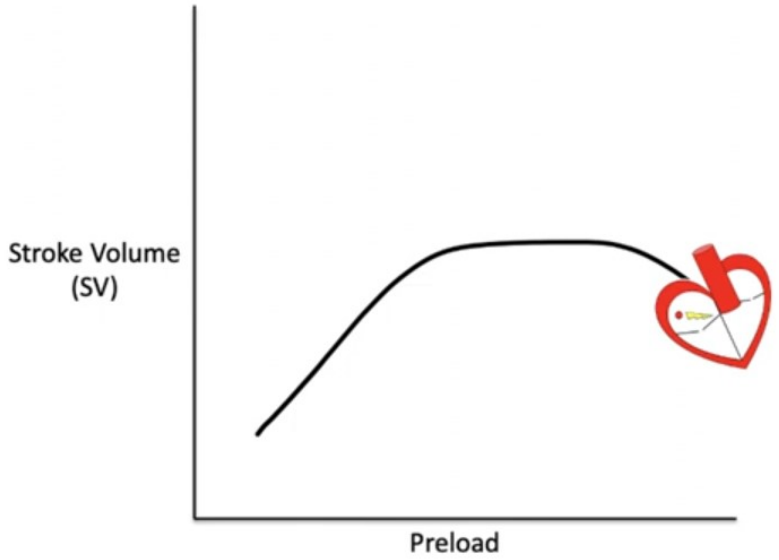
Imaging

- Chest x-ray



Management of Cardiogenic Shock

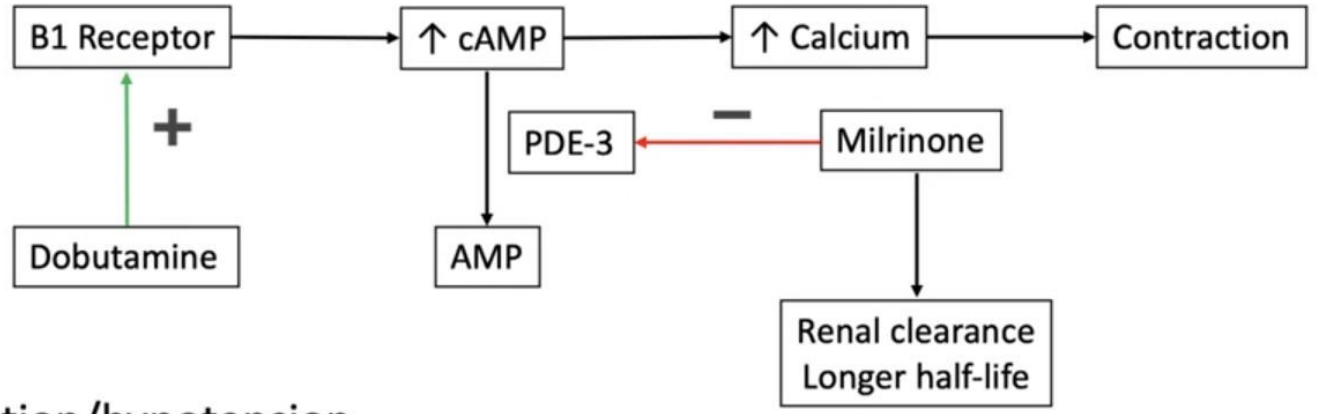
MAP = CO x SVR



1. Increase CO
2. Decrease SVR
3. Remove Volume

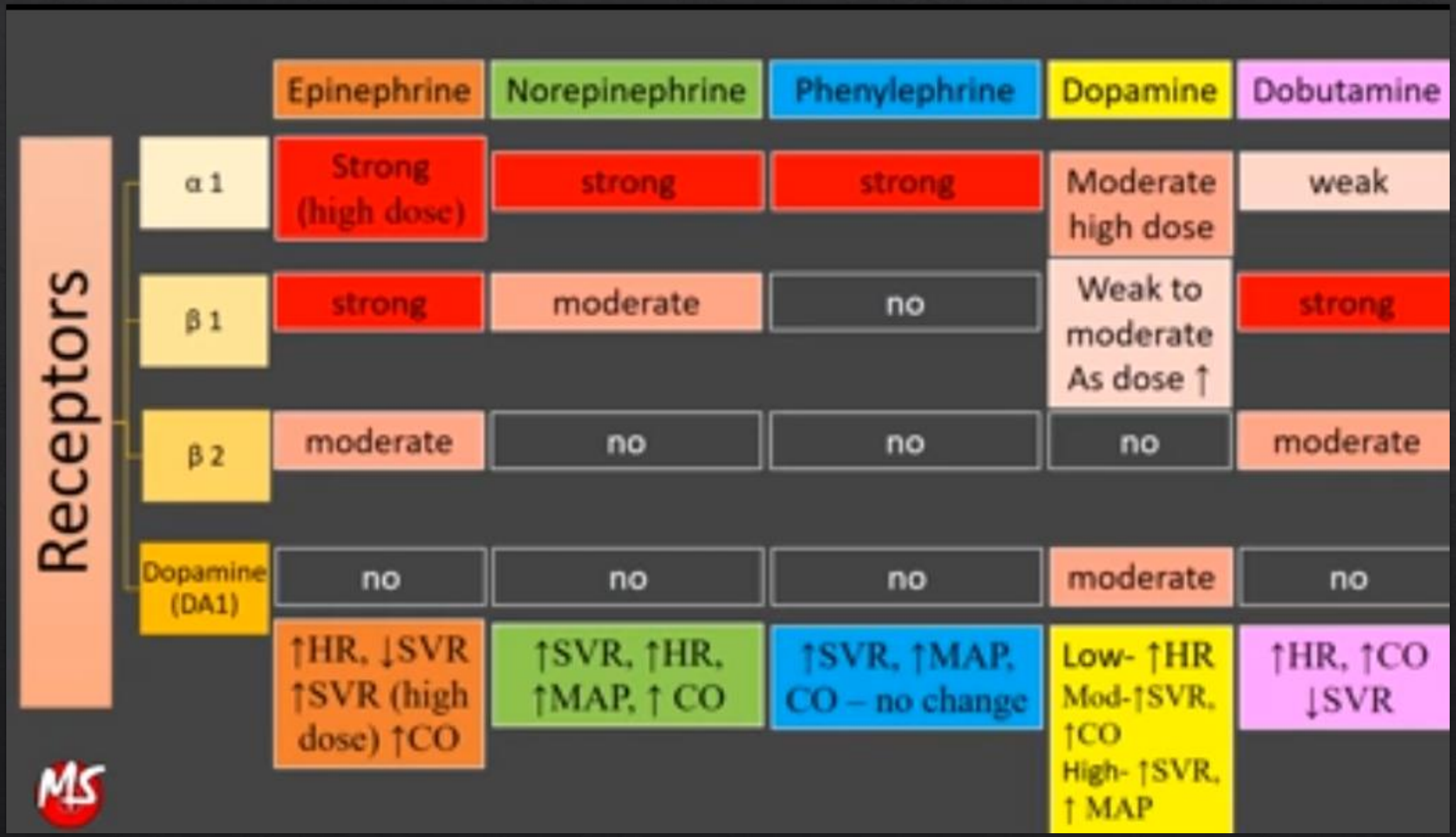
Increase CO

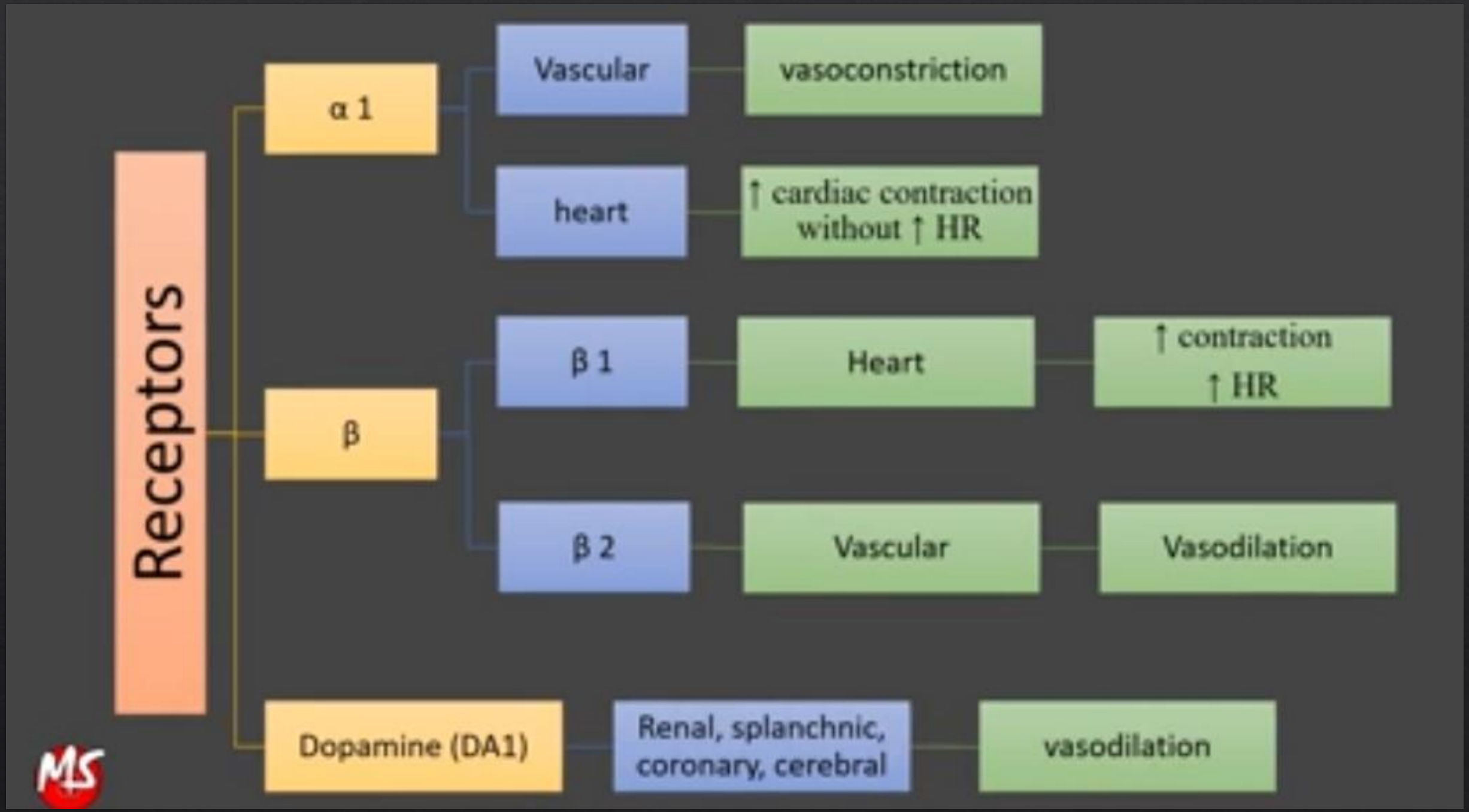
- Inotropes
  - Dobutamine
  - Milrinone



- Adverse effects:
  - Systemic vasodilation/hypotension
  - Increased incidence of arrhythmias
  - Increased myocardial oxygen demand

Drug	α1	β1	β2	V1
Norepinephrine	++++	++	+	None
Epinephrine	++++	+++	++	None


Receptors
Epinephrine
Norepinephrine
Phenylephrine
Dopamine
Dobutamine
α 1
Strong (high dose)
strong
strong
Moderate high dose
weak
β 1
strong
moderate
no
Weak to moderate As dose ↑
strong
β 2
moderate
no
no
no
moderate
Dopamine (DA1)
no
no
no
moderate
no
↑HR, ↓SVR ↑SVR (high dose) ↑CO
↑SVR, ↑HR, ↑MAP, ↑ CO
↑SVR, ↑MAP, CO – no change
Low- ↑HR Mod-↑SVR, ↑CO High- ↑SVR, ↑ MAP
↑HR, ↑CO ↓SVR


Receptors
α 1
Vascular
vasoconstriction
heart
↑ cardiac contraction without ↑ HR
β
β 1
Heart
↑ contraction
↑ HR
β 2
Vascular
Vasodilation
Dopamine (DA1)
Renal, splanchnic, coronary, cerebral
vasodilation

Vasopressors and Inotropes
(Summary)

Phenylephrine

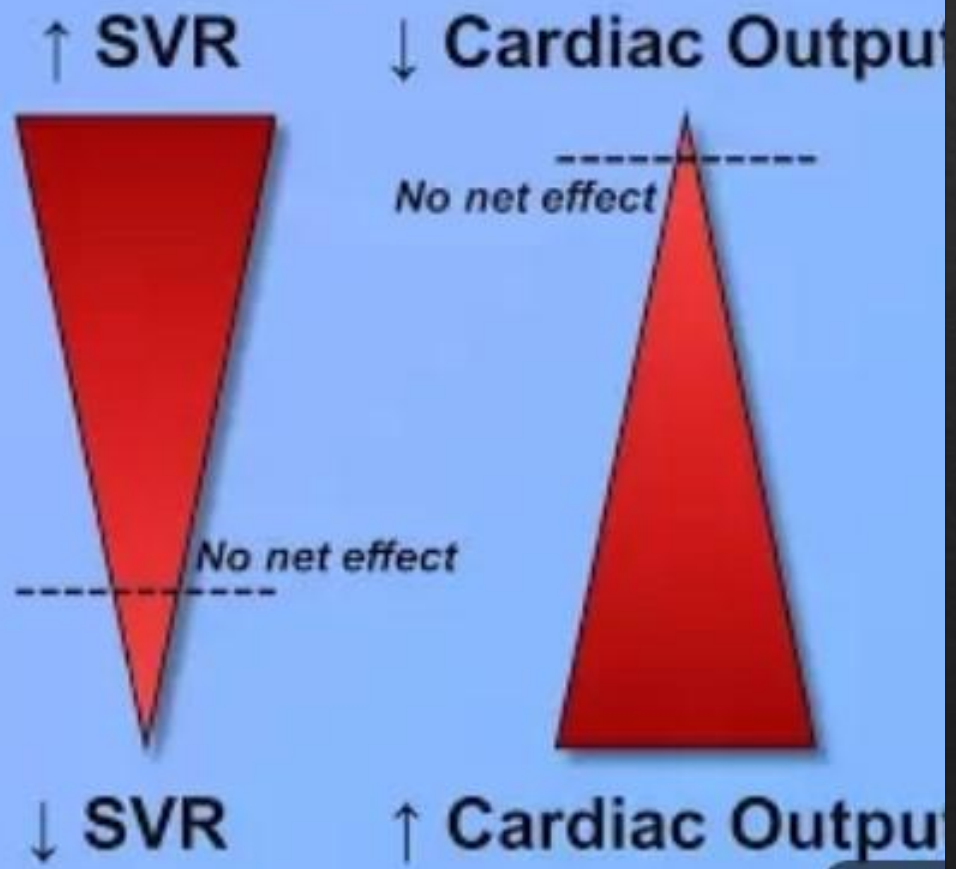
Norepinephrine

Epinephrine

Dopamine < **High Dose (> ~10µg/kg/min)**
Low Dose (< ~10µg/kg/min)

Dobutamine

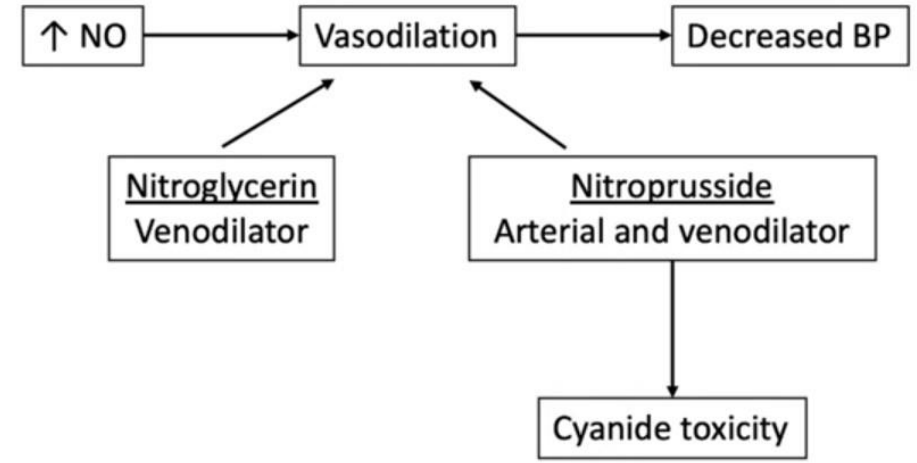
Milrinone



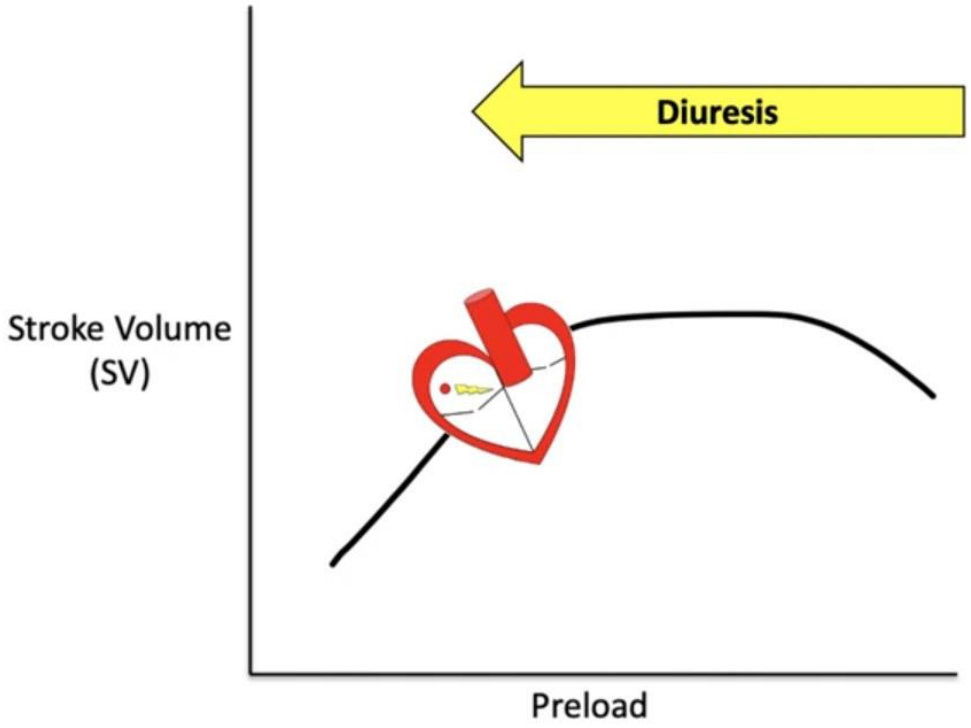
No Beta Blockers or Calcium Channel Blockers!

Decrease SVR

- Decrease SVR = afterload reduction
  - The lower the afterload, the less pressure a failing heart must pump against
- Afterload reduction agents:
  - Nitroprusside
  - Nitroglycerin



Remove Volume



Furosemide:

- Loop diuretic
- Administer IV BID

- Enrollment: 308 patients
- Compared intermittent vs. continuous, low-dose (home dose) vs. high-dose (2.5x home dose) furosemide in adults with acute decompensated HF
- Outcomes:
  - No difference between intermittent and continuous dosing
  - High-dose associated with greater diuresis and symptom improvement

Acute Cardiogenic Pulmonary Edema

The "L-M-N-O-P's"

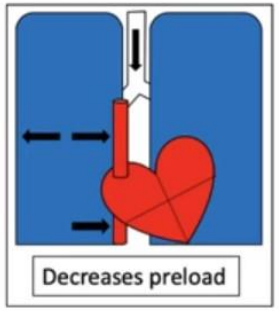
Lasix (Furosemide) – reduce preload, remove fluid

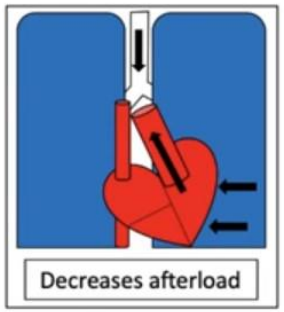
Morphine – pulmonary vasodilator, decrease dyspnea

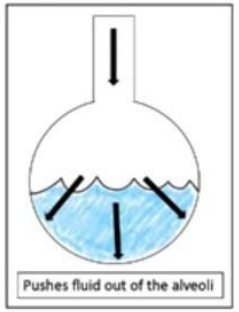
Nitrates – reduce preload, pulmonary vasodilator

Oxygen – treat acute hypoxemic respiratory failure

Positive pressure ventilation







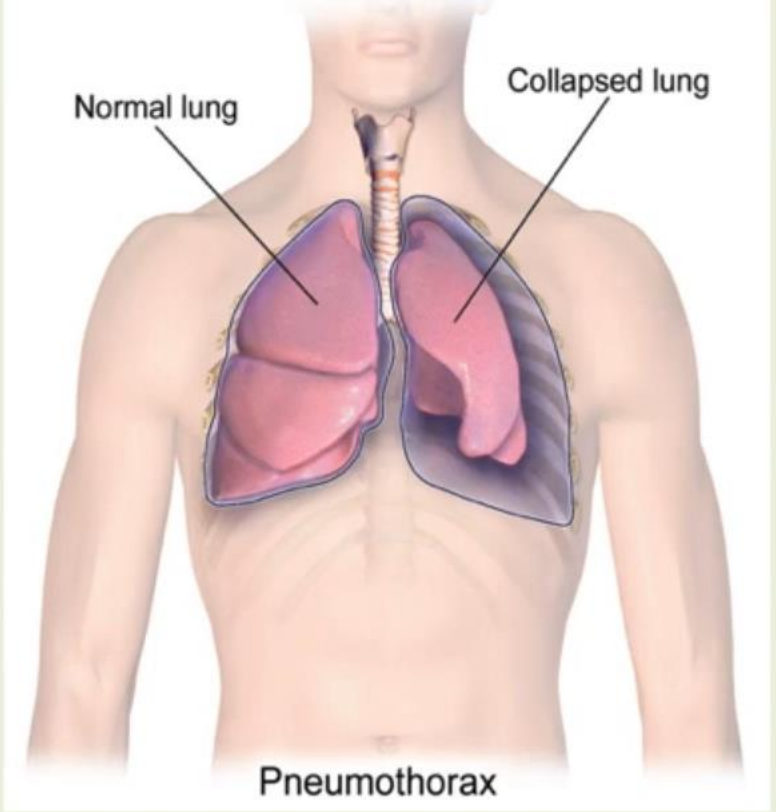
Obstructive Shock

- Very similar to cardiogenic shock
- Is associated with obstruction of great vessels or heart itself

Obstructive shock is defined as inability of heart to produce adequate cardiac output despite of normal intravascular volume and myocardial function

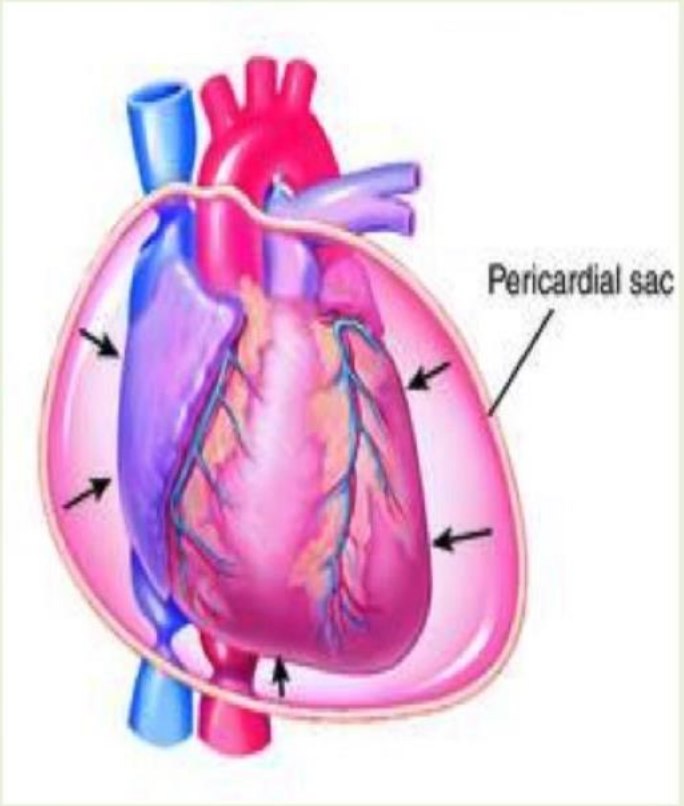
CAUSES:

- **Tension pneumothorax**



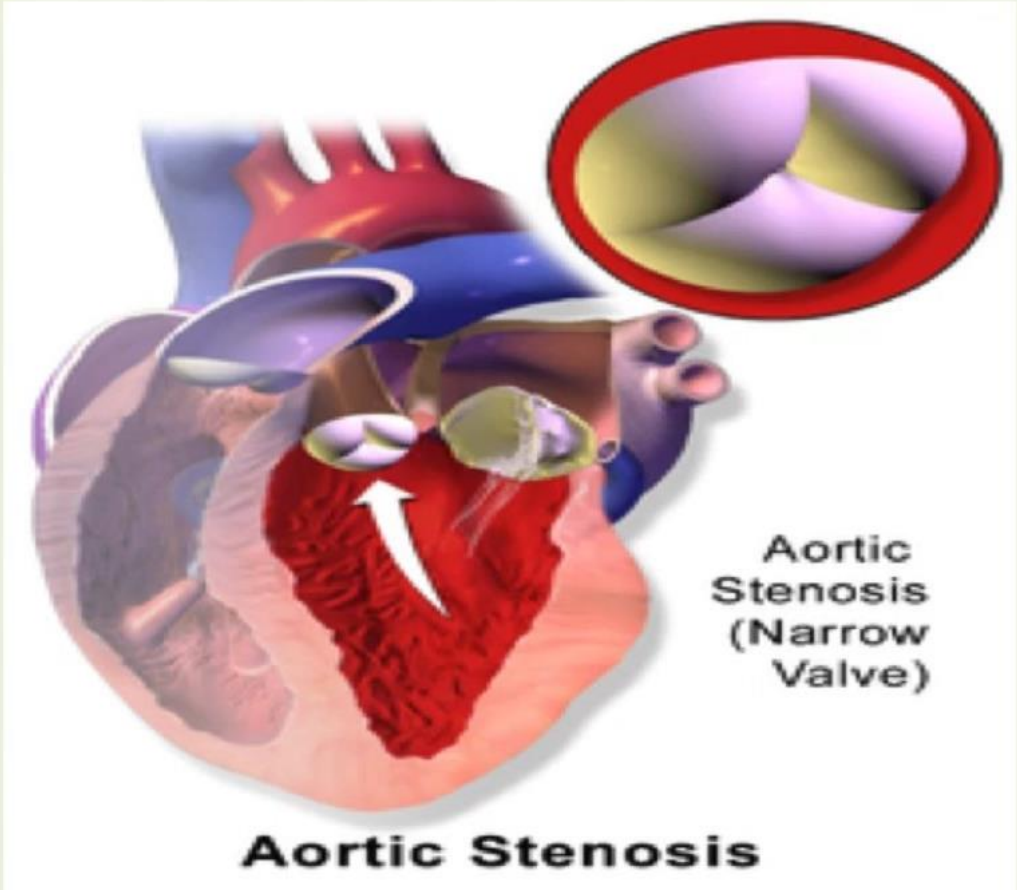
CAUSES:

- **Tension pneumothorax**
- **Cardiac tamponade**



CAUSES:

- **Tension pneumothorax**
- **Cardiac tamponade**
- **Aortic stenosis**



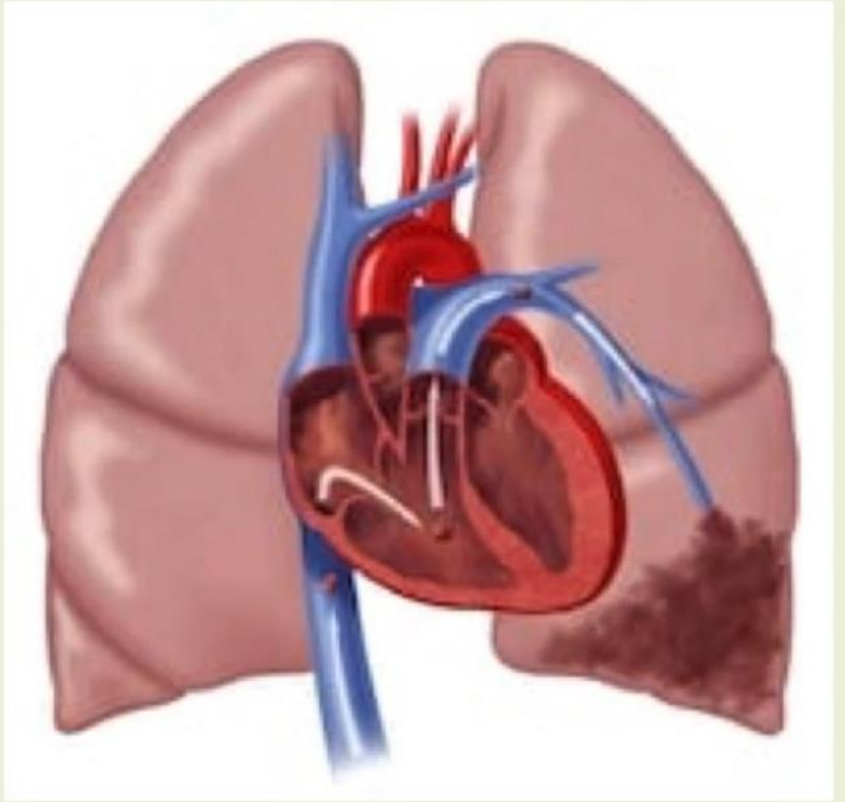
Aortic Stenosis

CAUSES:

- Tension pneumothorax
- Cardiac tamponade
- Aortic stenosis
- Constructive pericarditis

CAUSES:

- Tension pneumothorax
- Cardiac tamponade
- Aortic stenosis
- Constructive pericarditis
- Pulmonary embolism



SYMPTOMS :

✓ Back up of blood into lungs and right side of the heart

- Difficulty breathing
- Coughing
- Pulmonary edema
- Cardiomegaly
- Jugular Vein Distension (JVD)
- Swelling of total body

DIAGNOSIS :

- Lactate levels
- ABG
- Echo
- Chest X-ray

SYMPTOMS :

✓ Back up of blood into lungs and right side of the heart

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- Swelling of total body

DIAGNOSIS :

- Lactate levels
- ABG
- Echo
- Chest X-ray

TREATMENT :

- Treatment of the underlying cause
- O2 delivery

Cardiac tamponade

- The distinction between a pericardial effusion and cardiac tamponade . The pericardium resists sudden stretching, and in acute tamponade the cardiac silhouette may appear normal in size. As a result of the noncompliance of the pericardium, a small amount of fluid (usually less than 200 mL) is all that is necessary to produce tamponade.
- With chronic distention, however, large volumes of pericardial fluid may accumulate with little to no effect on cardiac physiology.
- The volume of the effusion alone, therefore, does not dictate the clinical course as much as the acuity of its development. uses of acute pericardial effusion include trauma, ischemic myocardial rupture and aortic dissection
- **Which side? Right (thinner muscle – less pressure)**
- **Which chamber? Right atrium (thinner muscles – less pressure)**
- **At which phase of cardiac cycle? Diastole (less pressure)**

Cardiac tamponade

- Causes of cardiac tamponade:
  - Penetrating chest trauma.
  - Ischemic myocardial rupture.
  - Aortic dissection.

Massive pulmonary embolism

- Cardiac output is restricted either by **mechanical obstruction** of the pulmonary arterial tree or by **pulmonary arterial vasoconstriction** induced by the release of secondary mediators.

Tension pneumothorax

Mechanism:

- Air enters into the pleural cavity (with ball valve mechanism)
- Air accumulates with increase in the pleural pressure
- It causes collapse of the epsilateral lung and mediastinal shift.
- It causes twist to the great vessels carrying the venous return and the cardiac output.
- Decreasing the cardiac output to the systemic circulation.

Obstructive shock (tension pneumothorax):

- **Classical manifestations of shock:** hypotension, tachycardia, weak thready pulse, tachypnea, altered mental status, oliguria, pale cold sweaty skin.
- Congested neck veins – high central venous pressure (CVP).
- Normal heart sounds but displaced.
- **Decreased air entry** and **tympanic note on percussion** on affected side – **tracheal shift** to the contralateral side.

Emergency measures: ABCDE – **consider needle decompression during B assessment**

Specific investigations:

- Confirmation after release by chest X ray or CT chest.

Specific treatment:

- After needle decompression, we can insert intercostal tube with under water seal.

Obstructive shock (cardiac tamponade):

- **Classical manifestations of shock:** hypotension, tachycardia, weak thready pulse, tachypnea, altered mental status, oliguria, pale cold sweaty skin.
- Congested neck veins – high central venous pressure (CVP).
- **Distant heart sounds.**

Emergency measures: ABCDE – **pericardiocentesis during C assessment**.

Specific investigations:

- Echocardiography is diagnostic (diastolic collapse of right atrium and ventricle).

Specific treatment:

- Pericardiocentesis (echocardiography guided, fluoroscopy guided or blind).
- Surgical intervention if needed (penetrating injury, aortic dissection).

Obstructive shock (massive pulmonary embolism)

- **Classical manifestations of shock:** hypotension, tachycardia, weak thready pulse, tachypnea, altered mental status, oliguria, pale cold sweaty skin.
- Congested neck veins – high central venous pressure (CVP).
- Some wheezes on lung fields (histamine release).
- Reversed and splitted second heart sound.
- Signs of DVT in any limb.

Emergency measures: ABCDE.

Specific investigations:

- Echocardiography may be helpful (right heart dilatation and increased pulmonary artery pressure)
- CT pulmonary angiography (most diagnostic).
- D-dimer.
- Doppler on venous system of limb.

Obstructive shock (massive pulmonary embolism).

Specific treatment:

- Thrombolytic therapy (streptokinase – urokinase – tissue plasminogen activator).
- Embolectomy.

Obstructive Shock: Pathogenesis, complications and clinical findings

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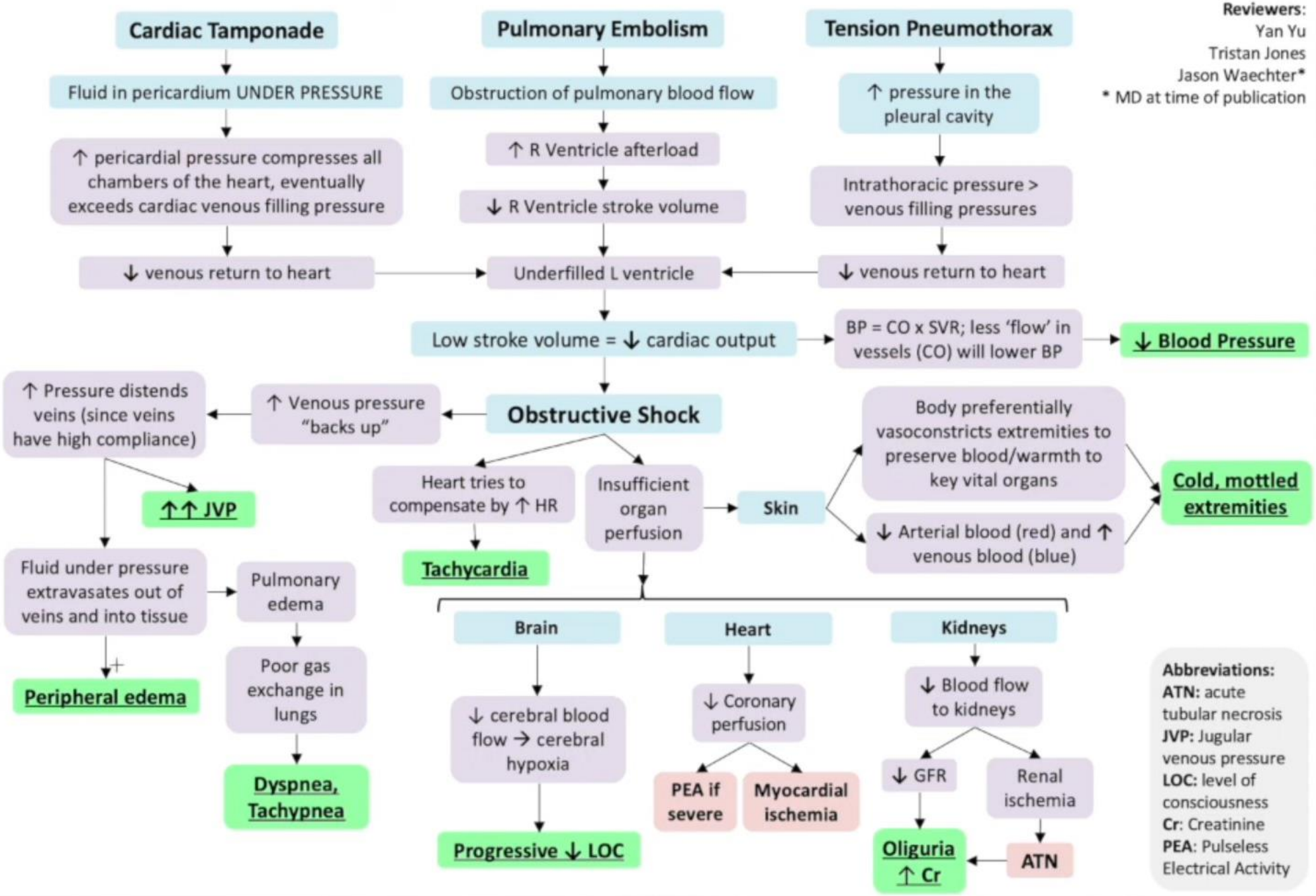
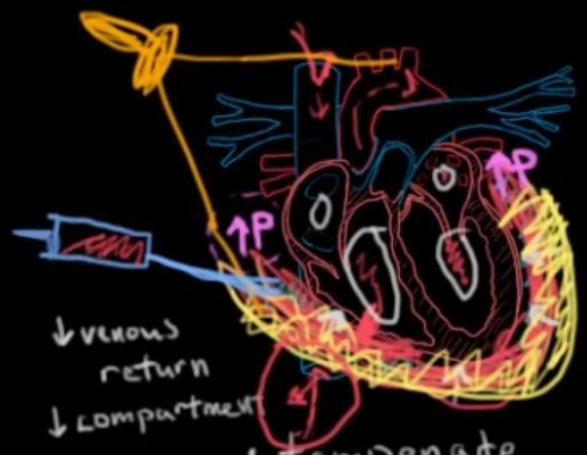
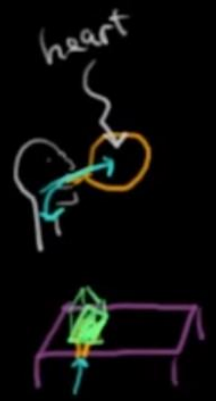


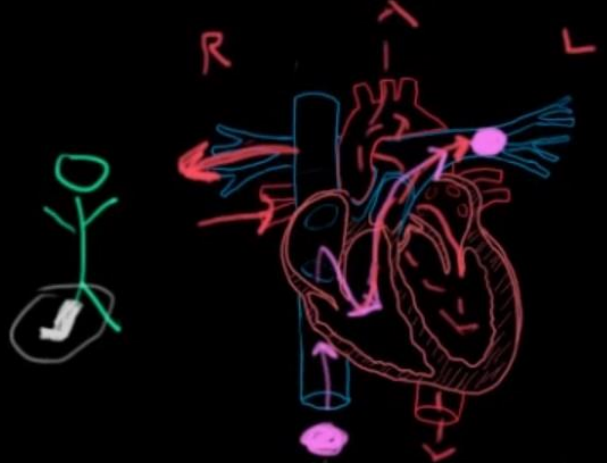
Abbreviations:
ATN: acute tubular necrosis
JVP: Jugular venous pressure
LOC: level of consciousness
Cr: Creatinine
PEA: Pulseless Electrical Activity





- tamponade
- constrive pericarditis

- tension pneumothorax



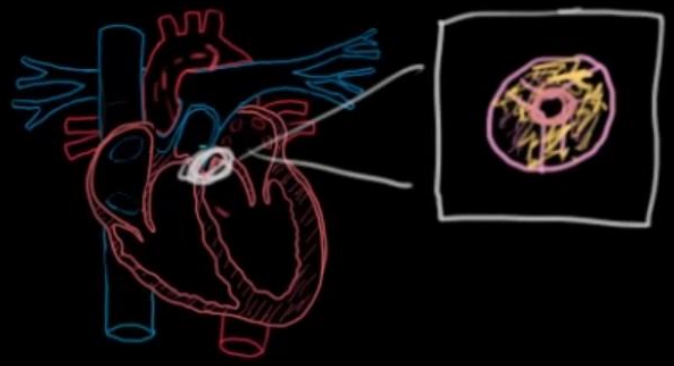
pulmonary embolism

LABS

- serum lactate
- ABG
- obstruction?

Treat

- relieve obstruction



aortic stenosis

شوک در اطفال

دکتر محمودرضا زینت زاده

استادیار طب اورژانس دانشگاه علوم پزشکی تهران

case

Introduction

You enter the room of a 3-month-old girl who was brought to the emergency department with a history of vomiting and diarrhea with poor PO intake.

General Assessment

You see an infant who appears listless. She is lying on the bed and does not respond to her parents. She is breathing rapidly without retractions or respiratory distress. Her color appears mottled.

5A	What is your initial impression of the child's condition based on your general assessment?
5B	Does this infant require immediate intervention? If so, what intervention is indicated?

Initial impression

This infant has a ***worrisome clinical picture with tachypnea and decreased response to her parents and her environment***. Based on the history of vomiting and diarrhea, she likely had a viral gastroenteritis and now has hypovolemia secondary to fluid loss. ***The infant's appearance with decreased responsiveness suggests that this infant is in shock***.

Shock

Shock is an acute process characterized by the body's inability to deliver adequate oxygen to meet the metabolic demands of vital organs and tissues. Insufficient oxygen at the tissue level is unable to support normal aerobic cellular metabolism, resulting in a shift to less-efficient anaerobic metabolism.

Shock

- As shock progresses, increases in tissue oxygen extraction are unable to compensate for this deficiency in oxygen delivery, leading to progressive clinical deterioration and lactic acidosis.
- If inadequate tissue perfusion persists, adverse vascular, inflammatory, metabolic, cellular, endocrine, and systemic responses worsen physiologic instability.

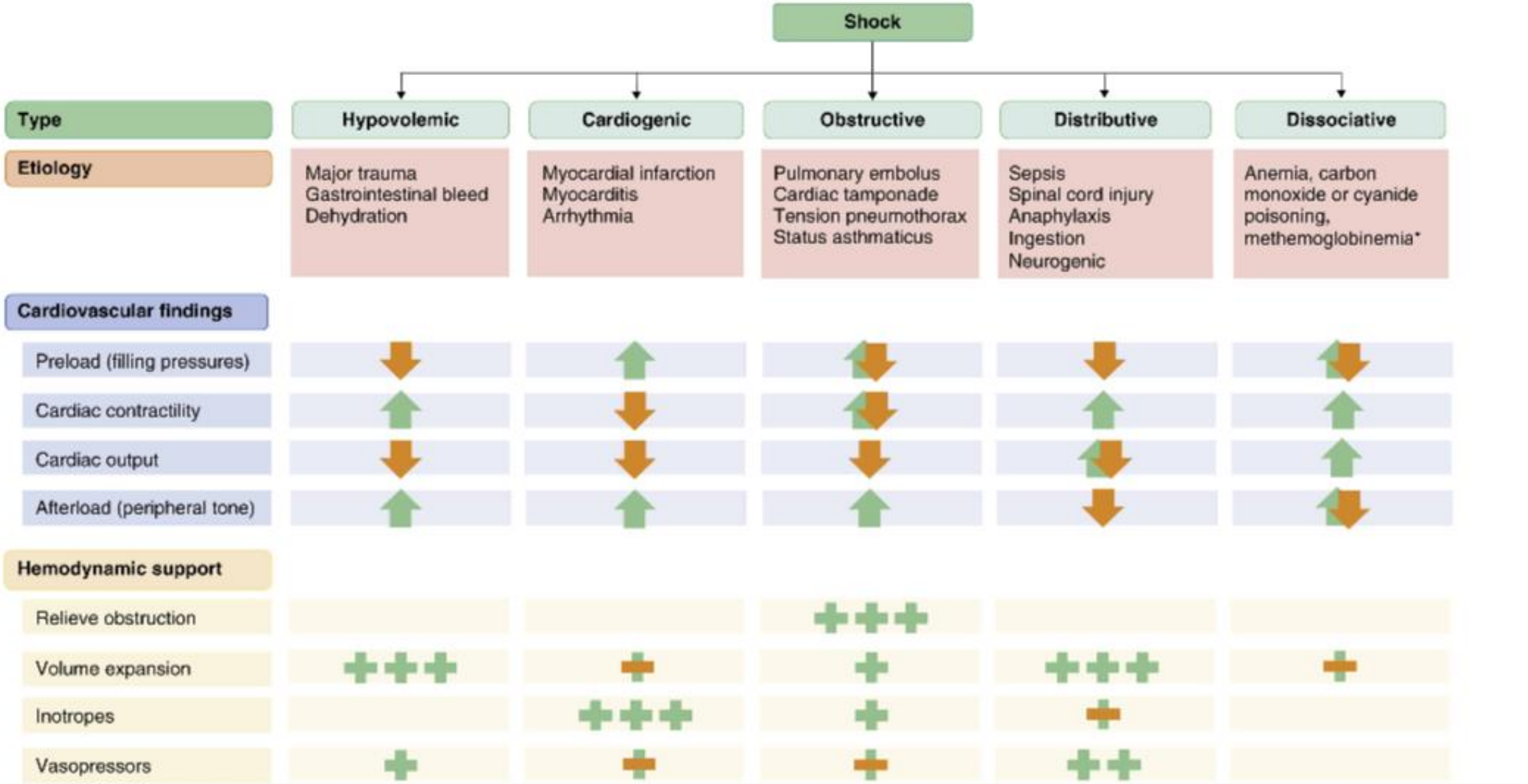


Fig. 85.1

The causes, cardiovascular findings, and hemodynamic support for different types of shock. Under cardiovascular findings, *bidirectional arrows* indicate variation in findings among patients with the particular type. Under hemodynamic support, the number of + *signs* indicates the importance of therapy. A combined + *and* − indicates that the intervention could help some patients but must be used with caution. *Treat specific etiology transfusion or specific antidotes.

Immediate intervention?

Does this infant require immediate intervention?
Yes.

If so, what intervention is indicated?

Based on your general assessment, it is appropriate to ***provide oxygen*** and call for help to ***obtain vascular access and get assistance in providing care for this infant.***

This infant appears to have an open airway and is breathing adequately, so ventilation is not needed. To obtain more objective data about the infant's condition, you should ***rapidly proceed to the primary assessment*** and ***place the infant on a cardiac monitor and pulse oximeter***. Once vascular access is established, ***begin rapid fluid resuscitation***.

Primary Assessment

You administer high-flow oxygen and proceed with your primary assessment. The child's heart rate is 210/min, respiratory rate is 50/min, blood pressure is 60/43 mm Hg, and axillary temperature is 97°F (36.1°C). The pulse oximeter is not picking up the pulse consistently—when a reading is obtained it is 99% to 100%. You palpate weak brachial and femoral pulses, but you cannot palpate distal pulses. Heart sounds are normal. The extremities are cool and mottled below the elbows and knees. Capillary refill time in the foot is >5 seconds. Auscultation reveals clear lungs with good distal air entry bilaterally. During the exam the child moans occasionally but otherwise has little response to verbal or painful stimulation.

5C	How would you categorize this infant's condition? Is the infant hypotensive?
5D	What decisions and actions are appropriate at this time?
5E	What is the definition of shock?
5F	What elements of the secondary assessment would you like to know?

Table 67-1 Normal Vital Signs According to Age

AGE	HEART RATE (beats/min)	BLOOD PRESSURE (mm Hg)	RESPIRATORY RATE (breaths/min)
Premature	120-170*	55-75/35-45†	40-70‡
0-3 mo	100-150*	65-85/45-55	35-55
3-6 mo	90-120	70-90/50-65	30-45
6-12 mo	80-120	80-100/55-65	25-40
1-3 yr	70-110	90-105/55-70	20-30
3-6 yr	65-110	95-110/60-75	20-25
6-12 yr	60-95	100-120/60-75	14-22
12+ yr	55-85	110-135/65-85	12-18

Table 4-1 Lower Limit of Normal Systolic Blood Pressure by Age

Age	Lower Limit of Normal Systolic Blood Pressure
Term neonate (0 to 28 days)	More than 60 mm Hg or strong central pulse
Infant (1 to 12 months)	More than 70 mm Hg or strong central pulse
Child 1 to 10 years	More than 70 + (2 × age in years)
Child 10 years or older	More than 90 mm Hg

PALS Pearl

Infants and children are capable of more effective vasoconstriction than adults are. As a result, a previously healthy infant or child is able to maintain a normal blood pressure and organ perfusion for a longer time in the presence of shock.

Recognition of Shock Flowchart

Clinical Signs		Hypovolemic Shock	Distributive Shock	Cardiogenic Shock	Obstructive Shock
A	Patency	Airway open and maintainable/not maintainable			
B	Respiratory rate	Increased			
	Respiratory effort	Normal to increased		Labored	
	Breath sounds	Normal	Normal (± crackles)	Crackles, grunting	
C	Systolic blood pressure	**Compensated Shock → Hypotensive Shock**			
	Pulse pressure	Narrow	Variable	Narrow	
	Heart rate	Increased			
	Peripheral pulse quality	Weak	Bounding or weak	Weak	
	Skin	Pale, cool	Warm or cool	Pale, cool	
	Capillary refill	Delayed	Variable	Delayed	
	Urine output	Decreased			
D	Level of consciousness	Irritable early Lethargic late			
E	Temperature	Variable			

Appropriate decision?

Since this infant is in hypotensive shock, you must ***quickly establish vascular access*** to enable administration of one or more isotonic crystalloid fluid boluses. Brief attempts to gain peripheral venous access are appropriate. But ***if they are not rapidly successful, then place an IO needle*** because the infant is in hypotensive shock.

Secondary assessment

The infant most likely has a viral gastroenteritis based on the history, but you should ***carefully look for the presence of signs*** **suggesting a different cause**, such as sepsis. Look for the ***presence of a skin rash or bruising***. ***Assess the abdomen*** (ie, is the abdomen distended and tender?) for signs of a potential surgical problem, such as an acute abdomen.

Critical bedside laboratory test: BS

Secondary assessment

Symptom

Allergy

Medications

Past medical history

Last meal

Events

Fluid, which, how much?

As soon as the needle is placed, you should deliver ***20 mL/kg of isotonic crystalloid, such as normal saline***. Remember to use a 3-way stopcock or pressure bag to rapidly deliver the fluid since it will generally not flow rapidly through a small peripheral IV or IO needle.

Tertiary studies

- Blood sample to evaluate ***serum electrolytes***
- ***Repeat bedside glucose test***
- Complete ***blood count*** will help determine if the infant is anemic, and the white blood cell count may help identify if the infant is at increased risk of bacterial sepsis (either a high or low white blood cell count); it will also confirm whether the infant has adequate oxygen-carrying capacity (ie, hemoglobin concentration)
- ***Blood and urine cultures*** if sepsis is considered a risk based on the history and examination
- ***Urine analysis***

Tertiary studies

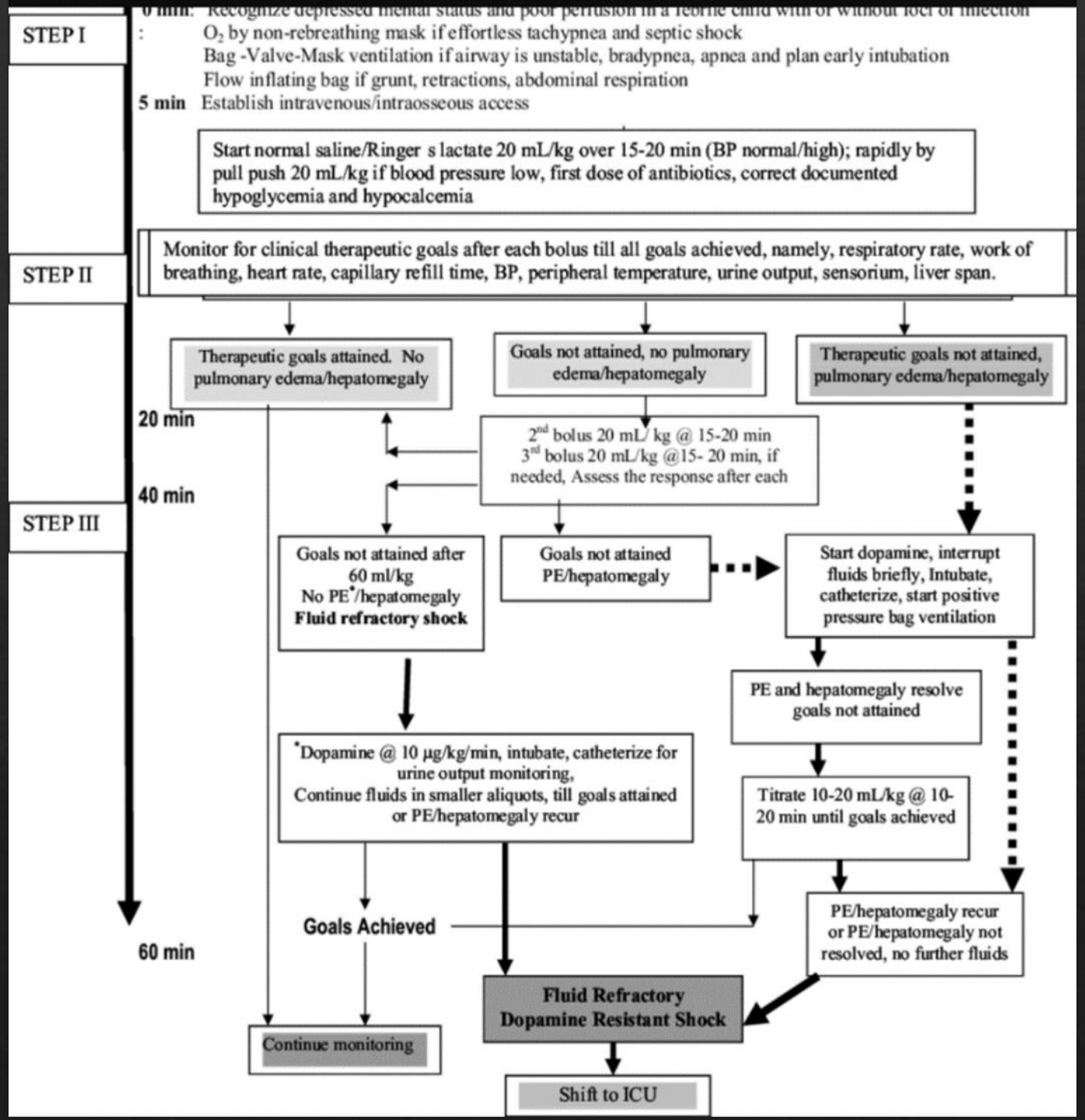
It is helpful to place a bladder catheter both to monitor the volume of urine produced and to obtain a urine sample for analysis. Remember that the *initial* volume of urine in the bladder does not determine the current urine output since you don't know how long the urine has been in the bladder. An indwelling bladder catheter will permit ongoing assessment of urine production, which provides indirect evidence of effectiveness of renal perfusion.

Table 70-13 Cardiovascular Drug Treatment of Shock

DRUG	EFFECT(S)	DOSING RANGE	COMMENT(S)
Dopamine	↑ Cardiac contractility Significant peripheral vasoconstriction at >10 µg/kg/min	3-20 µg/kg/min	↑ Risk of arrhythmias at high doses
Epinephrine	↑ Heart rate and ↑ cardiac contractility Potent vasoconstrictor	0.05-3.0 µg/kg/min	May ↓ renal perfusion at high doses ↑ Myocardial O_2 consumption Risk of arrhythmia at high doses
Dobutamine	↑ Cardiac contractility Peripheral vasodilator	1-10 µg/kg/min	—
Norepinephrine	Potent vasoconstriction No significant effect on cardiac contractility	0.05-1.5 µg/kg/min	↑ Blood pressure secondary to ↑ systemic vascular resistance ↑ Left ventricular afterload
Phenylephrine	Potent vasoconstriction	0.5-2.0 µg/kg/min	Can cause sudden hypertension ↑ O_2 consumption

Table 70-14 Vasodilators/Afterload Reducers

DRUG	EFFECT(S)	DOSING RANGE	COMMENT(S)
Nitroprusside	Vasodilator (mainly arterial)	0.5-4.0 µg/kg/min	Rapid effect Risk of cyanide toxicity with prolonged use (>96 hr)
Nitroglycerin	Vasodilator (mainly venous)	1-20 µg/kg/min	Rapid effect Risk of increased intracranial pressure
Prostaglandin E_1	Vasodilator Maintains an open ductus arteriosus in the newborn with ductal-dependent congenital heart disease	0.01-0.2 µg/kg/min	Can lead to hypotension Risk of apnea
Milrinone	Increased cardiac contractility Improves cardiac diastolic function Peripheral vasodilation	Load 50 µg/kg over 15 min 0.5-1.0 µg/kg/min	Phosphodiesterase inhibitor—slows cyclic adenosine monophosphate breakdown


0 min: Recognize depressed mental status and poor perfusion in a febrile child with or without foci of infection
STEP I
: O_2 by non-rebreathing mask if effortless tachypnea and septic shock
Bag -Valve-Mask ventilation if airway is unstable, bradypnea, apnea and plan early intubation
Flow inflating bag if grunt, retractions, abdominal respiration
5 min Establish intravenous/intraosseous access
Start normal saline/Ringer s lactate 20 mL/kg over 15-20 min (BP normal/high); rapidly by pull push 20 mL/kg if blood pressure low, first dose of antibiotics, correct documented hypoglycemia and hypocalcemia
STEP II
Monitor for clinical therapeutic goals after each bolus till all goals achieved, namely, respiratory rate, work of breathing, heart rate, capillary refill time, BP, peripheral temperature, urine output, sensorium, liver span.
Therapeutic goals attained. No pulmonary edema/hepatomegaly
Goals not attained, no pulmonary edema/hepatomegaly
Therapeutic goals not attained, pulmonary edema/hepatomegaly
20 min
2nd bolus 20 mL/ kg @ 15-20 min
3rd bolus 20 mL/kg @15- 20 min, if needed, Assess the response after each
40 min
STEP III
Goals not attained after 60 ml/kg
No PE*/hepatomegaly
Fluid refractory shock
Goals not attained PE/hepatomegaly
Start dopamine, interrupt fluids briefly, Intubate, catheterize, start positive pressure bag ventilation
PE and hepatomegaly resolve goals not attained
*Dopamine @ 10 µg/kg/min, intubate, catheterize for urine output monitoring,
Continue fluids in smaller aliquots, till goals attained or PE/hepatomegaly recur
Titrate 10-20 mL/kg @ 10-20 min until goals achieved
Goals Achieved
PE/hepatomegaly recur or PE/hepatomegaly not resolved, no further fluids
60 min
Fluid Refractory
Dopamine Resistant Shock
Continue monitoring
Shift to ICU

Management of Shock Flowchart

Management of Shock Flowchart	
• Oxygen • Pulse oximetry • ECG monitor	• IV/IO access • BLS as indicated • Point-of-care glucose testing

Hypovolemic Shock Specific Management for Selected Conditions	
Nonhemorrhagic	**Hemorrhagic**
• 20 mL/kg NS/LR bolus, repeat as needed • Consider colloid	• Control external bleeding • 20 mL/kg NS/LR bolus, repeat 2 or 3x as needed • Transfuse PRBCs as indicated

Distributive Shock Specific Management for Selected Conditions		
Septic	**Anaphylactic**	**Neurogenic**
Management Algorithm: • Septic Shock	• IM epinephrine (or autoinjector) • Fluid boluses (20 mL/kg NS/LR) • Albuterol • Antihistamines, corticosteroids • Epinephrine infusion	• 20 mL/kg NS/LR bolus, repeat PRN • Vasopressor

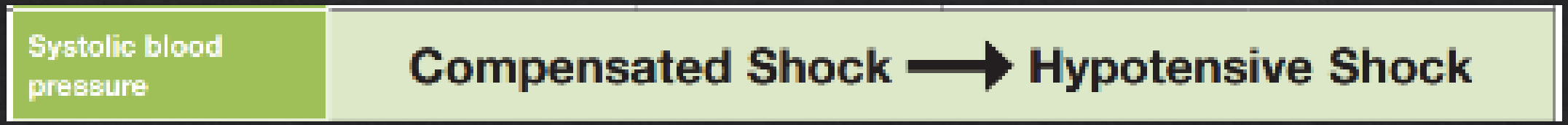
Cardiogenic Shock Specific Management for Selected Conditions	
Bradyarrhythmia/Tachyarrhythmia	**Other (eg, CHD, Myocarditis, Cardiomyopathy, Poisoning)**
Management Algorithms: • Bradycardia • Tachycardia With Poor Perfusion	• 5 to 10 mL/kg NS/LR bolus, repeat PRN • Vasoactive infusion • Consider expert consultation

Obstructive Shock Specific Management for Selected Conditions			
Ductal-Dependent (LV Outflow Obstruction)	**Tension Pneumothorax**	**Cardiac Tamponade**	**Pulmonary Embolism**
• Prostaglandin E_1 • Expert consultation	• Needle decompression • Tube thoracostomy	• Pericardiocentesis • 20 mL/kg NS/LR bolus	• 20 mL/kg NS/LR bolus, repeat PRN • Consider thrombolytics, anticoagulants • Expert consultation

Goals for Resuscitation

- Blood pressure (systolic pressure at least fifth percentile for age: 60 mmHg <1 month of age, 70 mmHg + [2 x age in years] in children 1 month to 10 years of age, 90 mmHg in children 10 years of age or older)
- Quality of central and peripheral pulses (strong, distal pulses equal to central pulses)
- Skin perfusion (warm, with capillary refill <2 seconds)
- Mental status (normal mental status)
- **Urine output (≥1 mL/kg per hour, once effective circulating volume is restored)**
- Clearance of lactate (hope to see down trending and preferably cut in half after initial resuscitation)

Take home message



Thank you