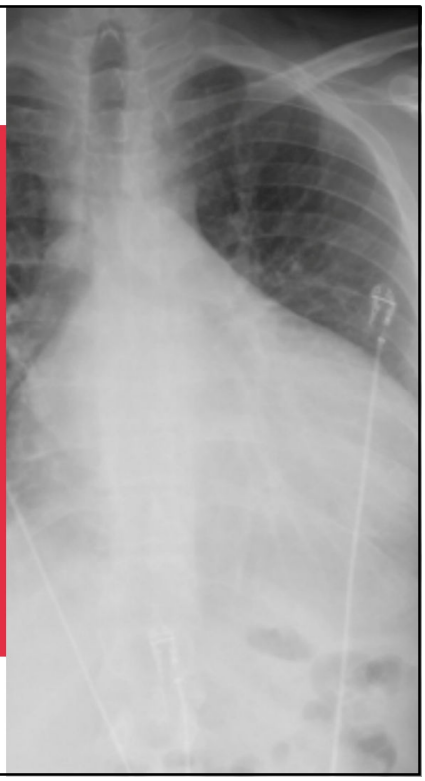
Cardiogenic Shock: Fatal Errors and Assumptions to Avoid

Emergency Cardiology Symposium 2025

Rohit Menon, M.D.



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No Financial Disclosures

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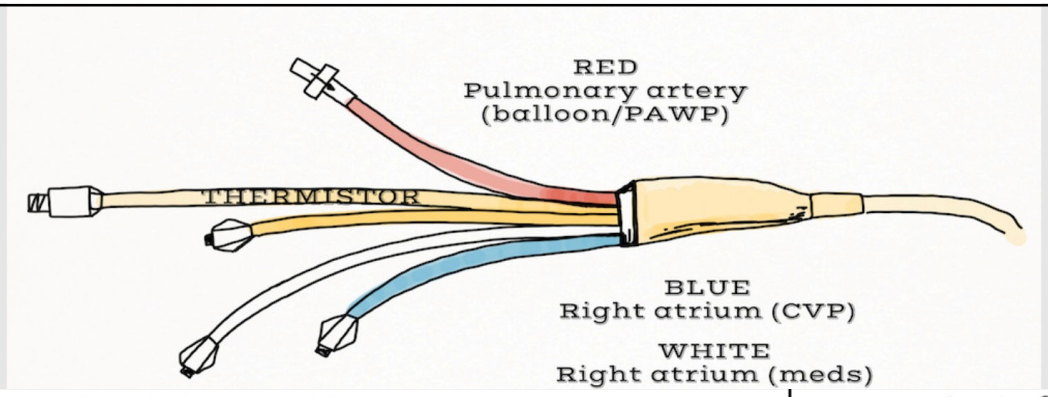
Recognizing and Managing Cardiogenic Shock is Easy

3



Cardiac Index (Thermodilution)	CI < 2 L/m/m^2
Cardiac Index (Fick)	CI < 2 L/m/m^2
Mixed Venous Saturation (MvO2)	MvO2 <60%
Cardiac Power (CPO)	CPO < 0.6 W
Pulmonary Artery Pulsatility Index (PAPi)	PAPi < 0.9
Central Venous Pressure (CVP)	CVP >12 mmHg
Pulmonary Artery Occlusion Pressure (PAOP)	PAOP > 15 mmHg

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Recognizing and Managing Cardiogenic Shock is NOT Easy

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Recognizing and Managing Cardiogenic Shock is NOT Easy

Especially in the Emergency Department

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Agenda

1. Errors and Assumptions of Recognition
2. Errors and Assumptions of Management

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Agenda

Errors and Assumptions of Recognition

1. Assumptions re: Vitals and Physical Exam
2. Misinterpreting Labs
3. Fatal assumptions re: Etiology

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Agenda

Errors and Assumptions of Management

1. Consider RV Failure
2.
3.
4.
5.

9

Agenda

Errors and Assumptions of Management

1. Consider RV Failure
2. Avoid beta blockers and calcium channel blockers
3.
4.
5.

10

Agenda

Errors and Assumptions of Management

1. Consider RV Failure
2. Avoid beta blockers and calcium channel blockers
3. Do not be afraid of Inotropes
4.
5.

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Agenda

Errors and Assumptions of Management

1. Consider RV Failure
2. Avoid beta blockers and calcium channel blockers
3. Do not be afraid of Inotropes
4. Avoid volume overload
5.

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Agenda

Errors and Assumptions of Management

1. Consider RV Failure
2. Avoid beta blockers and calcium channel blockers
3. Do not be afraid of Inotropes
4. Avoid volume overload
5. Know when to transfer / activate cath lab

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Agenda

Management

1. A: Airway
2.
3.
4.
5.

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Agenda

Management

1. A: Airway
2. E: Etiology treatment
3.
4.
5.

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Agenda

Management

1. A: Airway
2. E: Etiology treatment
3. I: Inotropy (and vasoactives)
4.
5.

16

Agenda

Management

1. A: Airway
2. E: Etiology treatment
3. I: Inotropy (and vasoactives)
4. O: Offloading the heart
5.

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Agenda

Management

1. A: Airway
2. E: Etiology treatment
3. I: Inotropy (and vasoactives)
4. O: Offloading the heart
5. U: Upgrade Support

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ERRORS AND ASSUMPTIONS OF RECOGNITION

Understanding Cardiogenic Shock

19

Definition of Cardiogenic Shock?

- **End Organ Injury**
- **Systemic Hypotension**
- **Reduced Cardiac Output**

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Definition of Cardiogenic Shock?

- End Organ Injury
- ~~Systemic Hypotension~~
- ~~Reduced Cardiac Output~~

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Definition of Cardiogenic Shock

- End Organ Injury
- Insufficient Cardiac Output

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Definition of Cardiogenic Shock

CO = Stroke Volume x Heart Rate

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Definition of Cardiogenic Shock

2025 ACC Definition (March 2025)

A cardiac disorder that results in both clinical and biochemical evidence of sustained tissue hypoperfusion irrespective of underlying blood pressure

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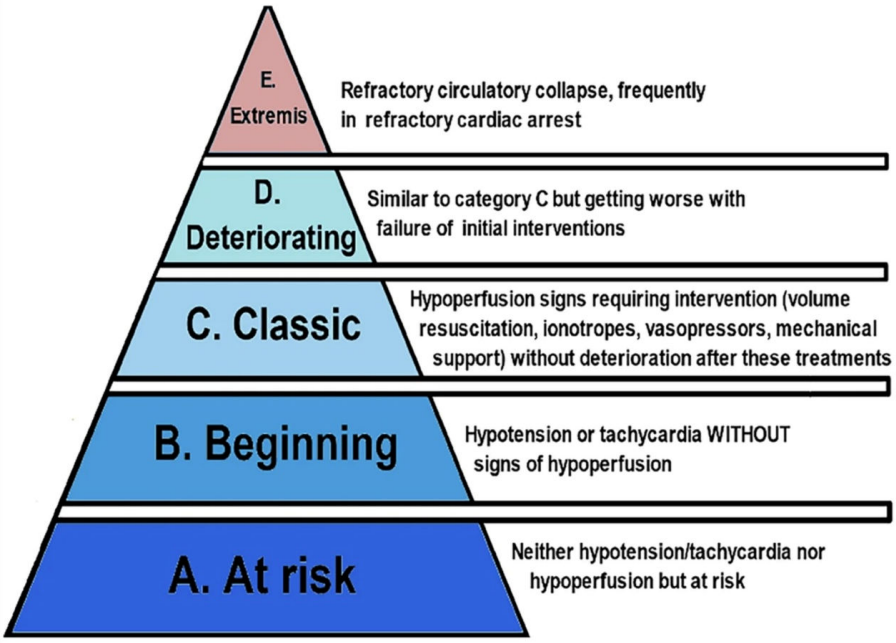
SCAI Classification of Cardiogenic Shock



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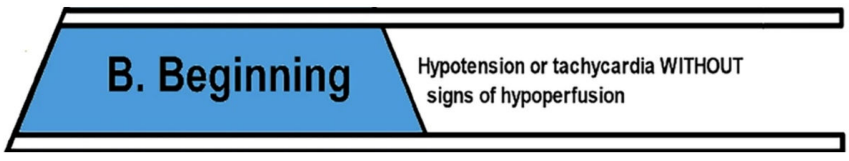
SCAI Classification of Cardiogenic Shock



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Traditional Patient Population for CS

- Age > 75
- Asian American / Pacific Islander
- CAD, HTN, HLD, Obesity
- Recent MI

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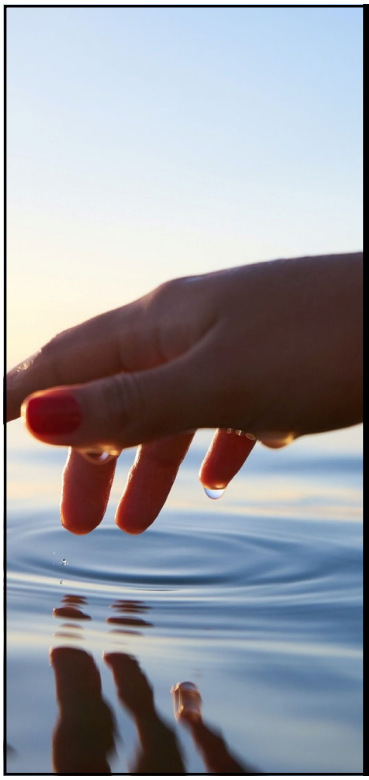

Traditional Physical Exam

- Congestion
- Impaired Perfusion

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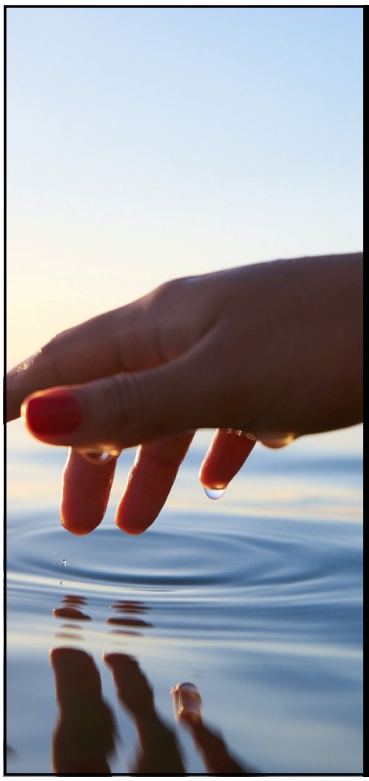

Traditional Physical Exam

- Congestion
  - JVD
  - Crackles
  - Peripheral Edema
  - Hepatomegaly

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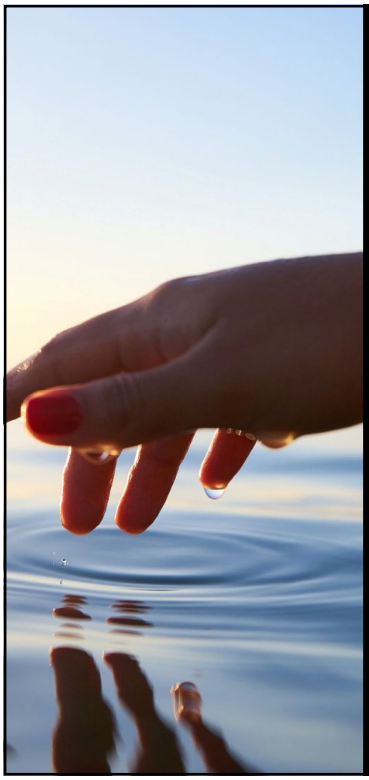

Traditional Physical Exam

- Impaired Perfusion
  - Mottled Skin
  - Cold Extremities
  - Delayed Cap Refill
  - Narrow Pulse Pressure
  - Tachycardia
  - Altered Mental Status
  - Oliguria

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ERRORS AND ASSUMPTIONS OF RECOGNITION

Fatal Assumption: Patients in CS are always fluid overloaded and cold

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Exceptions to the Rule

- **Euvolemic Cardiogenic Shock**
- **Normotensive Cardiogenic Shock**
- **RV Shock**
- **High Output Heart Failure**

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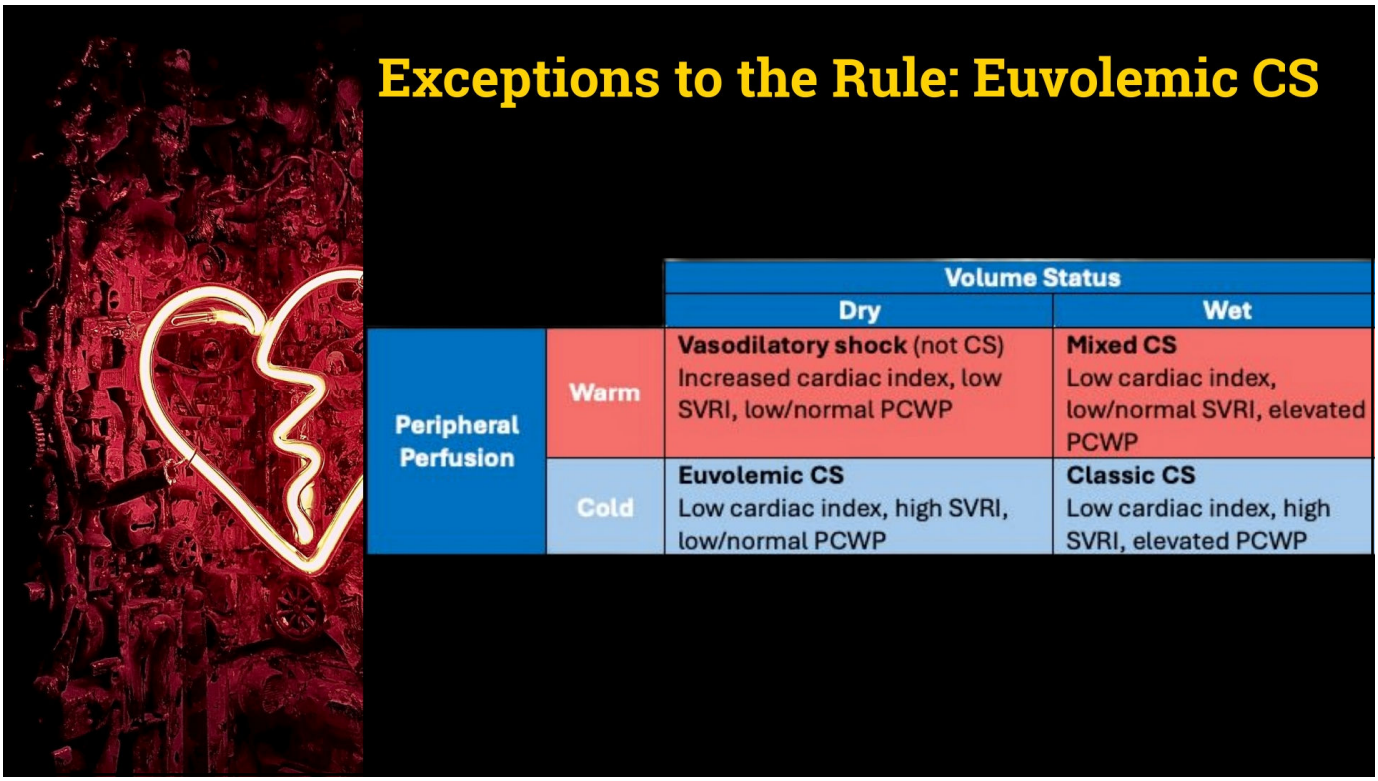
Exceptions to the Rule: Euvolemic CS
Volume Status
Dry
Wet
Peripheral Perfusion
Warm
Vasodilatory shock (not CS)
Increased cardiac index, low SVRI, low/normal PCWP
Mixed CS
Low cardiac index, low/normal SVRI, elevated PCWP
Cold
Euvolemic CS
Low cardiac index, high SVRI, low/normal PCWP
Classic CS
Low cardiac index, high SVRI, elevated PCWP

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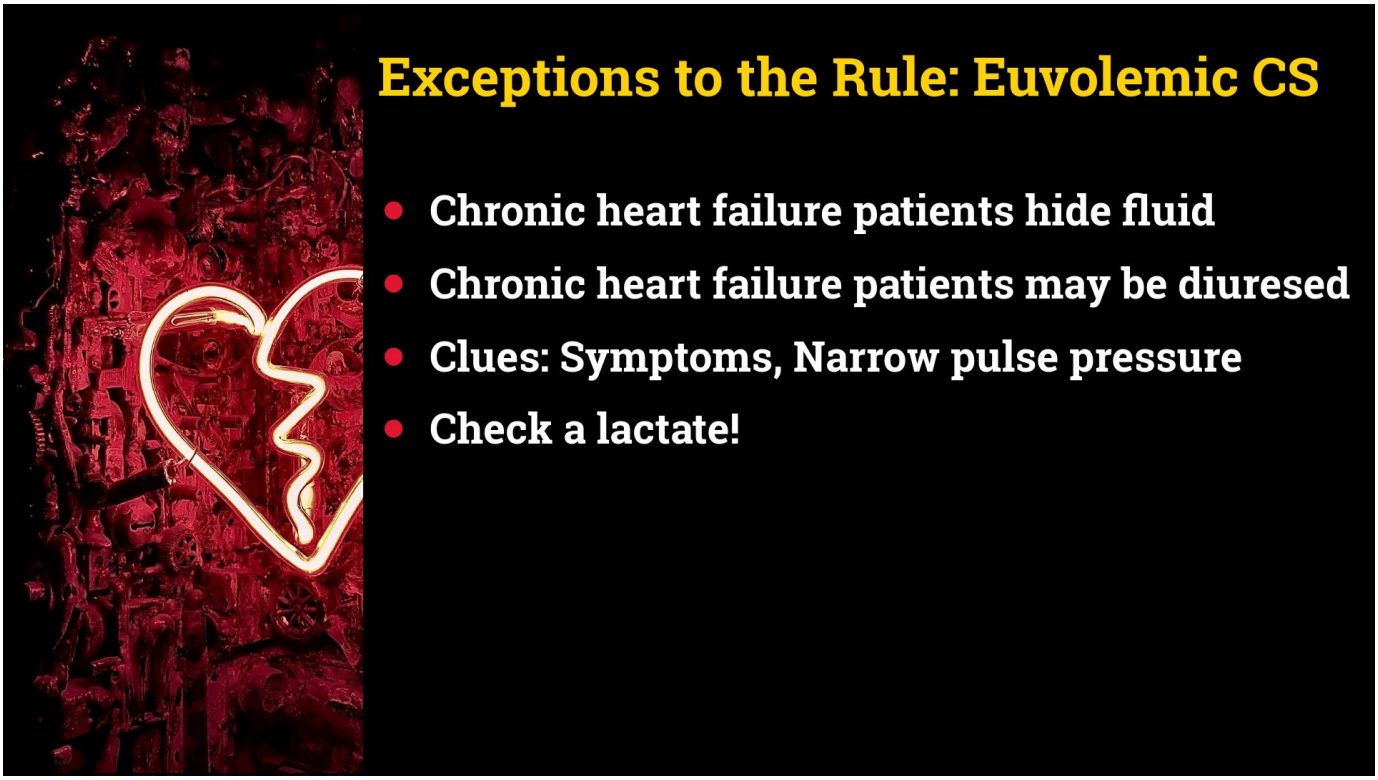
Exceptions to the Rule: Euvolemic CS
Chronic heart failure patients hide fluid
Chronic heart failure patients may be diuresed
Clues: Symptoms, Narrow pulse pressure
Check a lactate!

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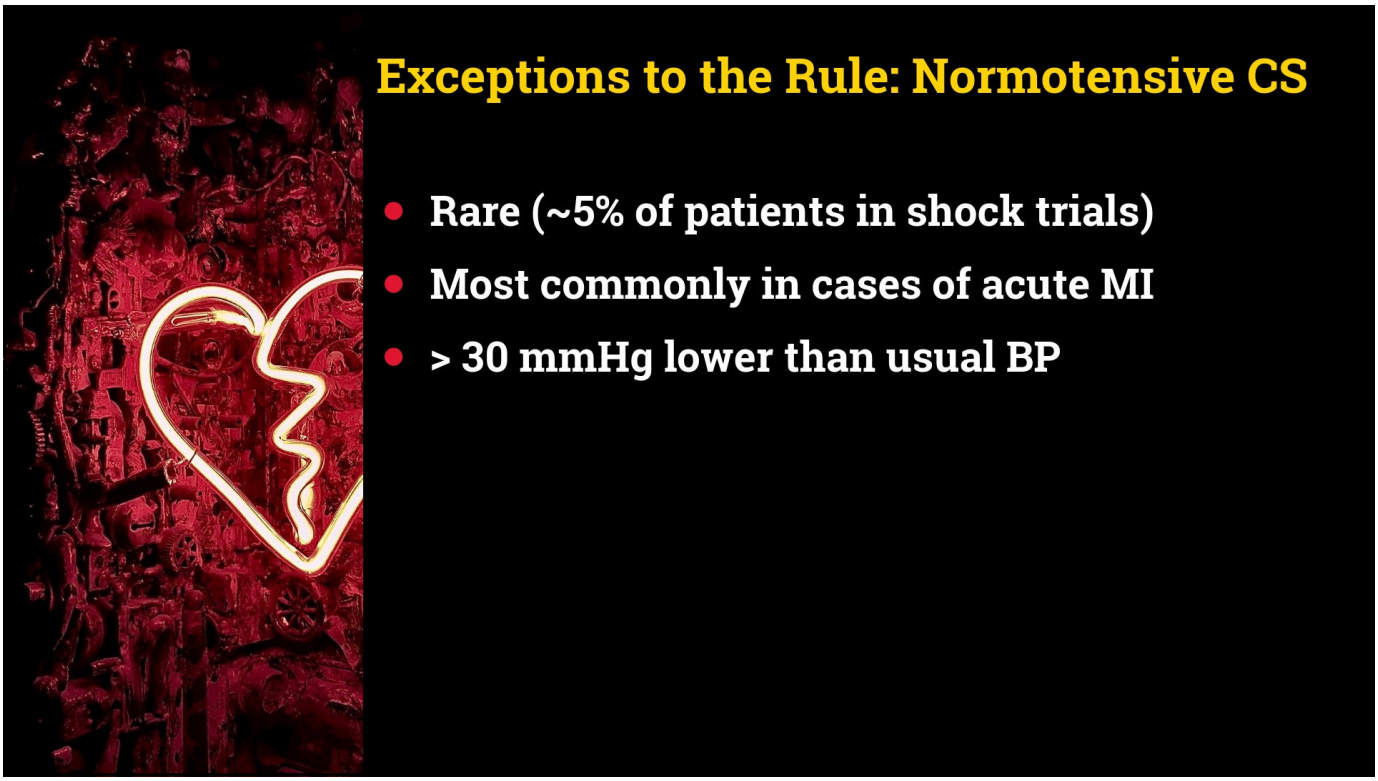
Exceptions to the Rule: Normotensive CS
Rare (~5% of patients in shock trials)
Most commonly in cases of acute MI
> 30 mmHg lower than usual BP

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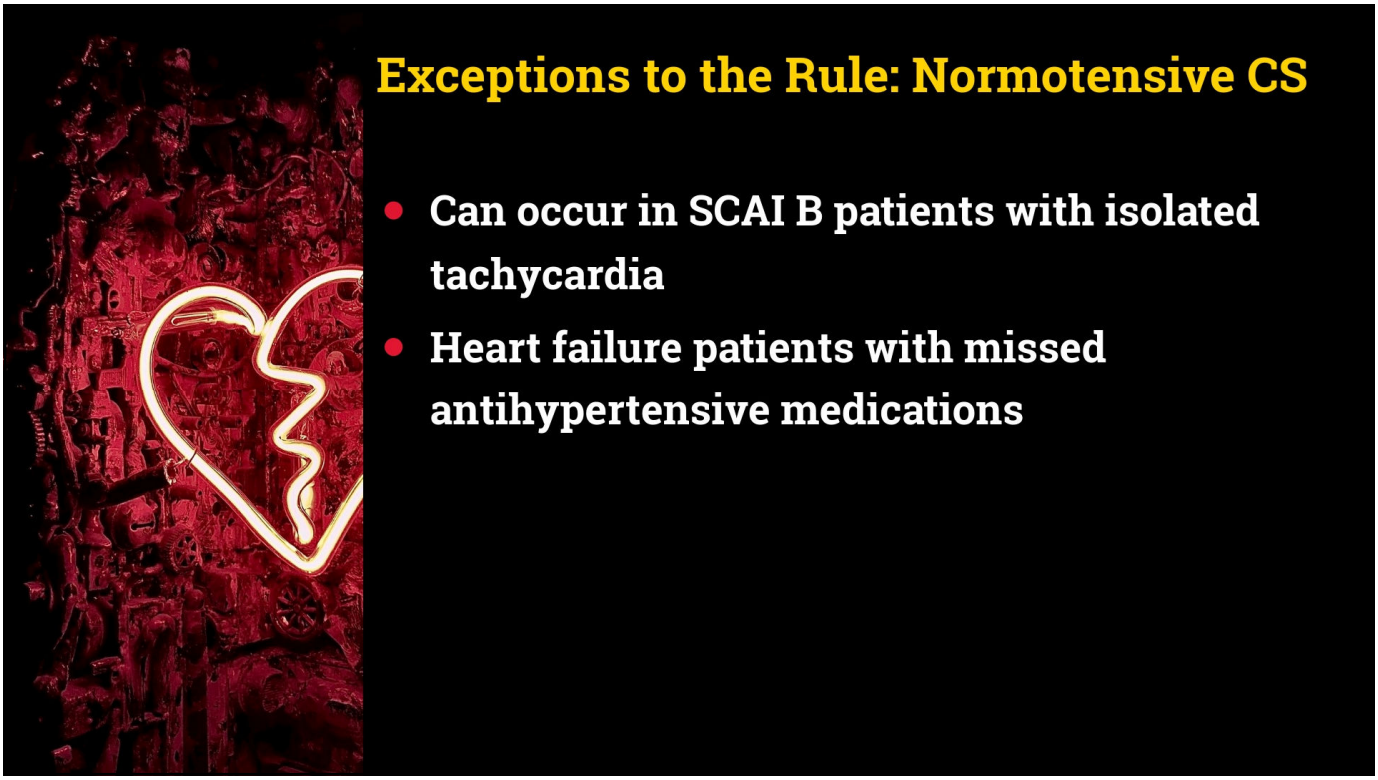
Exceptions to the Rule: Normotensive CS
Can occur in SCAI B patients with isolated tachycardia
Heart failure patients with missed antihypertensive medications

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Exceptions to the Rule: RV Shock

- JVD, Ascites, Hepatomegaly
- May not have crackles, exertional dyspnea, lower extremity edema
- Usually hypotensive, clammy, cool extremities

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Exceptions to the Rule: High Output Heart Failure

- Hyperthyroid
- Sepsis
- Severe malnutrition (Beriberi)

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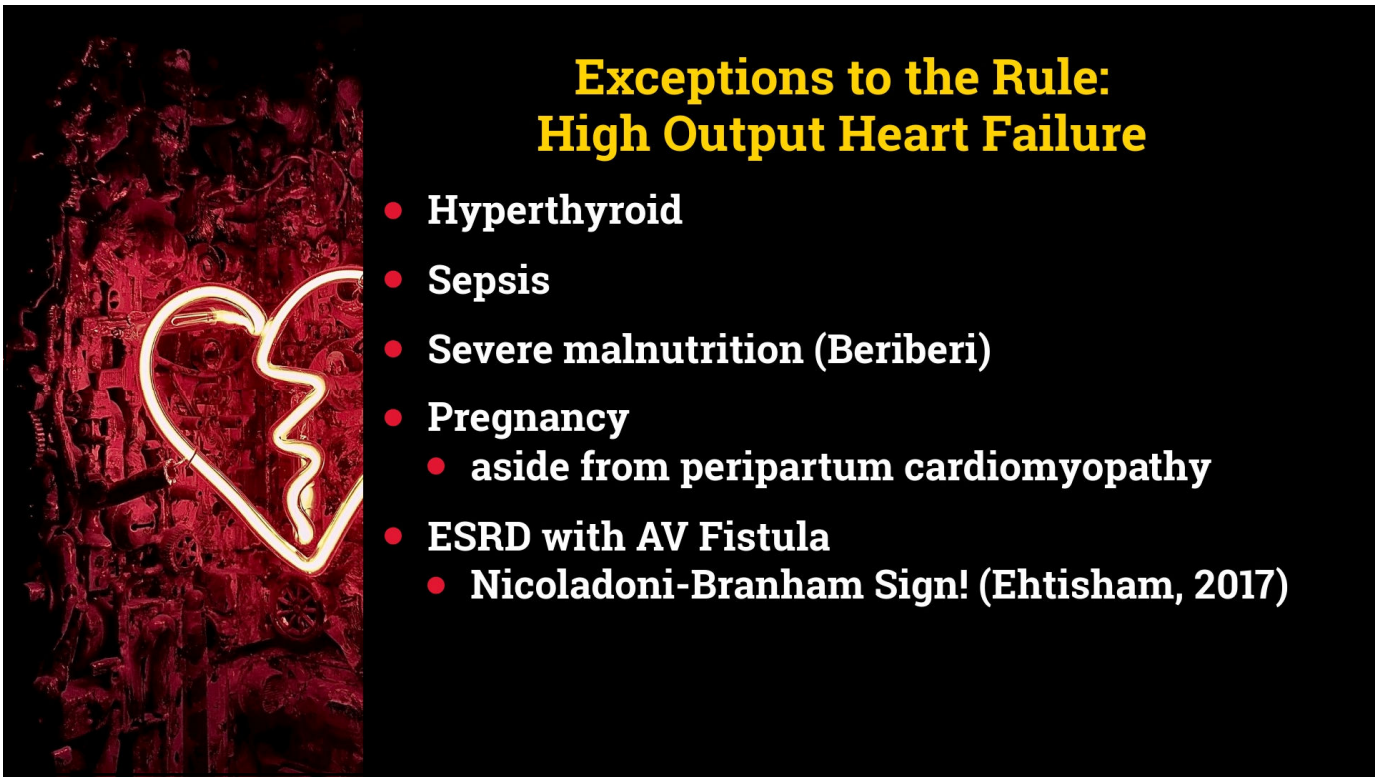
Exceptions to the Rule:
High Output Heart Failure
Hyperthyroid
Sepsis
Severe malnutrition (Beriberi)
Pregnancy
aside from peripartum cardiomyopathy
ESRD with AV Fistula
Nicoladoni-Branham Sign! (Ehtisham, 2017)

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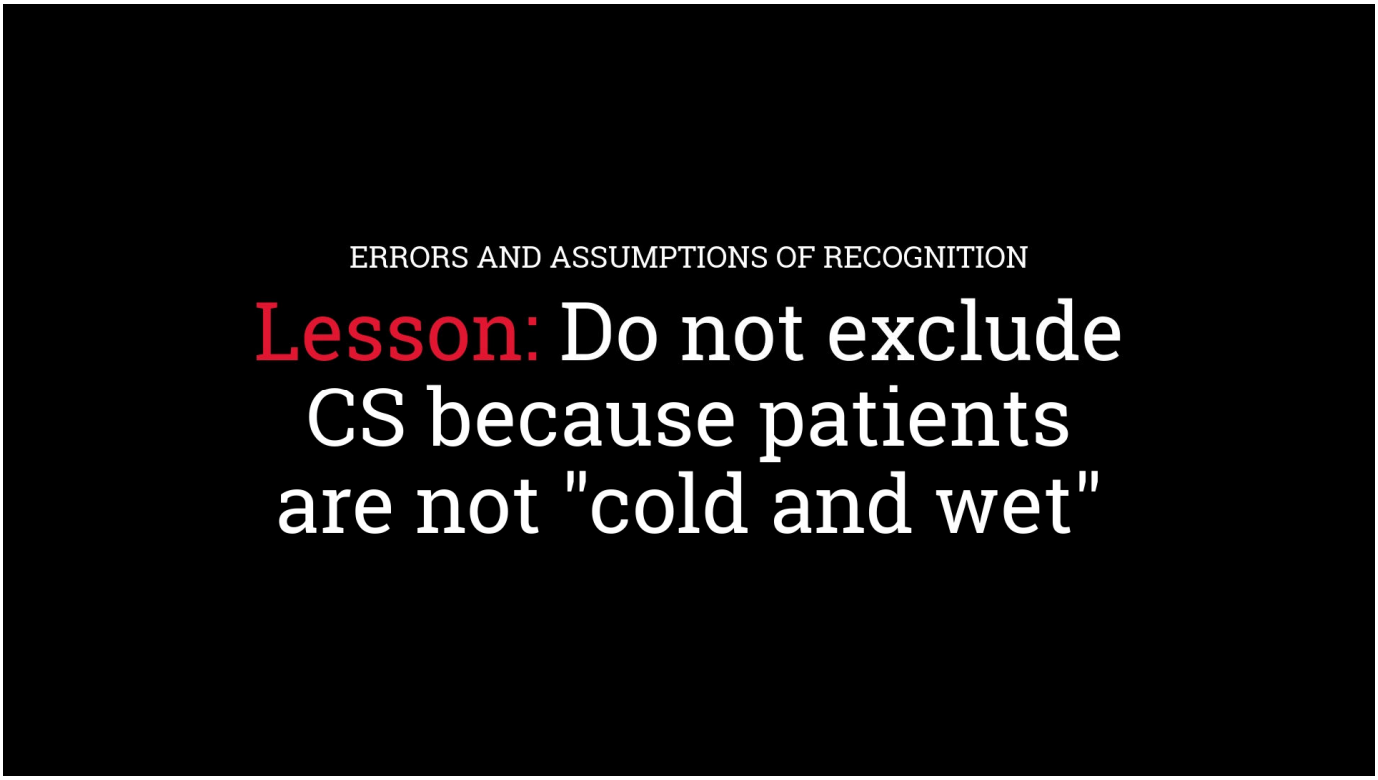
ERRORS AND ASSUMPTIONS OF RECOGNITION
Lesson: Do not exclude CS because patients are not "cold and wet"

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ERRORS AND ASSUMPTIONS OF RECOGNITION

Fatal Assumptions: AKI means patients are dry. High lactate = sepsis.

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Lab Testing in Cardiogenic Shock

- CBC
- Chemistry
- LFTs
- BNP
- Troponin
- Lactate
- SvO2%

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Lab Testing in Cardiogenic Shock

- No perfect lab test
- Labs can easily be misinterpreted as sepsis or other forms of shock

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Lab Testing in Cardiogenic Shock

- CBC
  - WBC can be a marker of systemic inflammation and stress

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Lab Testing in Cardiogenic Shock

- CBC
  - Total Leukocyte Count in CICU Patients (Smith, 2024)
    - Retrospective cohort study n = ~12,000
    - Subgroup Analysis of CS Patients (~1400)
      - n = 20 WBC < 4
      - n = 524 WBC 4-11
      - n = 701 WBC 11-22
      - n = 150 WBC > 22

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Lab Testing in Cardiogenic Shock

- CBC
  - Total Leukocyte Count in CICU Patients (Smith, 2024)
    - Retrospective cohort study n = ~12,000
    - Subgroup Analysis of CS Patients (~1400)
      - n = 20 WBC < 4
      - n = 524 WBC 4-11
      - n = 701 WBC 11-22
      - n = 150 WBC > 22

High WBC does not rule out CS

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Lab Testing in Cardiogenic Shock

- Chemistry
  - Creatinine should be used as a trend
  - High Cr does not mean prerenal!
  - Can be skewed by patients with CKD

Cr ≥2× upper limit of normal

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Lab Testing in Cardiogenic Shock

- LFTs
  - Jänti et al. 2017
    - n = 178
    - ALT was abnormal in 58% of patients

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Lab Testing in Cardiogenic Shock

- LFTs
  - Jänti et al. 2017
    - n = 178
    - ALT was abnormal in 58% of patients
  - Kapur et al. 2022 retrospective analysis
    - n= 3455 hospital admissions with CS
    - SCAI B stratified as ALT 200-500
    - SCAI C stratified as ALT > 500

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Lab Testing in Cardiogenic Shock

- LFTs
  - Jänti et al. 2017
    - n = 178
    - ALT was abnormal in 58% of patients

ALT >200 U/L or >3× upper limit of normal

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Lab Testing in Cardiogenic Shock

- BNP
  - Very little correlation
  - Brett, 2004
    - BNP level <350 pg/mL NPV 95%
    - Unclear what average BMI was

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Lab Testing in Cardiogenic Shock

- Troponin
  - Can be present as demand ischemia
  - Can point towards AMI or right heart strain

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Lab Testing in Cardiogenic Shock

- Lactate
  - Associated with mortality increase

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Lab Testing in Cardiogenic Shock

- Lactate
  - Associated with mortality increase
    - Kapur et al 2022 retrospective analysis
      - n= 3455 hospital admissions with CS
      - SCAI B stratified as Lactate 2-5
      - SCAI C stratified as Lactate 5-10

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Lab Testing in Cardiogenic Shock

- Lactate
  - Associated with mortality increase

Lactic acid >2 mmol/L
(pH < 7.2 without a known cause)

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Lab Testing in Cardiogenic Shock

- SvO2 (Mixed venous oxygen saturation)
  - Controversial
  - Normal 70-80%
  - < 65% supposed to imply impaired tissue oxygenation

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Lab Testing in Cardiogenic Shock

- RIJ CVC 20 cm terminating in the SVC
  - ScvO2 - not a true mixed gas, not as accurate
- Theory: cvO2 > 80% associated Sepsis

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Re: cvO2% (EMCrit, Dr. Farkas)

Numerous factors affect the cvO2%

	Increase cvO2%	Decrease cvO2%
Cardiac Output	High cardiac output	Low cardiac output
Hemoglobin	Hemoconcentration	Anemia
Oxygenation	Hyperoxia	Hypoxemia
Mental status	Sedation, analgesia	Stress, anxiety, pain
Temperature	Hypothermia	Hyperthermia
Muscular activity	Paralysis	Shivering, agitation, elevated work of breathing
Microvascular & cellular behavior	Cells fail to use oxygen (e.g. mitochondrial dysfunction in sepsis or ischemia-reperfusion)	Effective microvascular matching of flow with oxygen demand, effective cellular utilization of oxygen

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Re: cvO2% (EMCrit, Dr. Farkas)

Variation in other parameters creates a complicated relationship between cvO2 & CO

Scenario	HgB	Arterial oxygen saturation	PaO2	VO2	CO	cvO2
first example: different patients have the same cvO2% despite widely varying cardiac output						
Normal	14 mg/dL	99%	100 mm	250 ml/min	4 ml/min	69%
Patient in cardiogenic shock, just intubated & paralyzed	14 mg/dL	99%	600 mm	175 ml/min	2.3 L/min	68%
Septic shock with mild anemia and increased metabolism (VO2)	7 mg/dL	95%	60 mm	275 ml/min	10 L/min	68%
second example: cvO2% provides *misleading* information about the cardiac output						
Another septic shock patient with less marked increase in CO	7 mg/dL	93%	50 mm	275 ml/min	6 L/min	46%
Patient with cardiogenic shock, induced hypothermia and paralysis causing low VO2	15 mg/dL	99%	400 mm	150 ml/min	2.5 L/min	75%

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Re: cvO2% (EMCrit, Dr. Farkas)

Patient in cardiogenic shock, just intubated & paralyzed	14 mg/dL	99%	600 mm	175 ml/min	2.3 L/min	68%

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Lab Testing in Cardiogenic Shock

- Anecdotally
  - Very low saturation suggest CS
  - Best use: trend in response to interventions

"A central venous catheter can be helpful to measure central venous pressure and obtain a central venous oxygen saturation."

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ERRORS AND ASSUMPTIONS OF RECOGNITION

Lessons: Do not assume AKI means dry. Check the heart before bolusing fluid.

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ERRORS AND ASSUMPTIONS OF RECOGNITION

Lessons: Interpret labs in the clinical context of the patient and to trend during interventions

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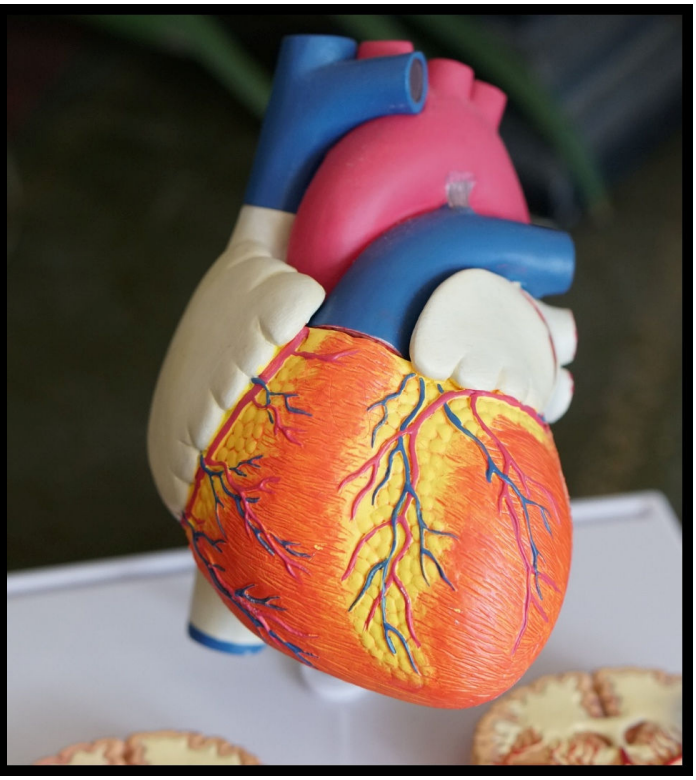
ERRORS AND ASSUMPTIONS OF RECOGNITION

Fatal Assumption: CS is due to new or chronic heart failure



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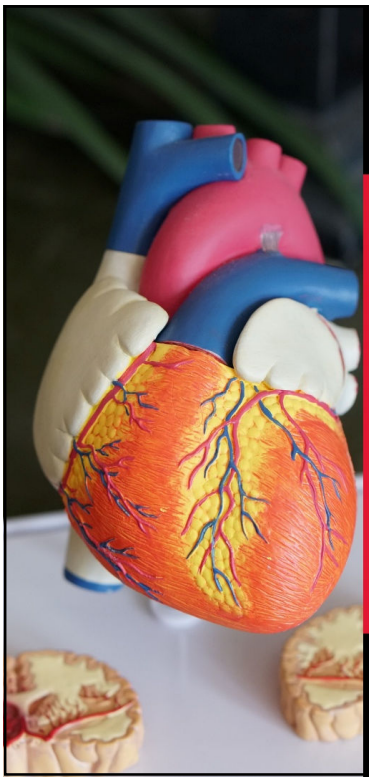

Causes of Acute Cardiogenic Shock

- Conduction System
- Ventricles
- Pericardium
- Valves

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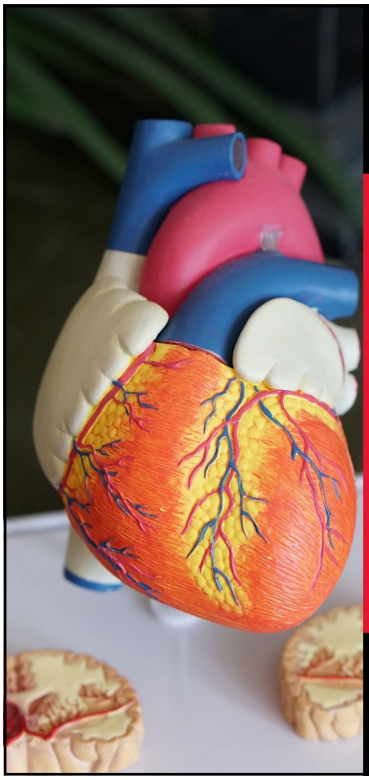

Causes of Acute Cardiogenic Shock

- Conduction
  - Ventricular Arrhythmia
  - Unstable Supraventricular Rhythm
  - Bradycardia

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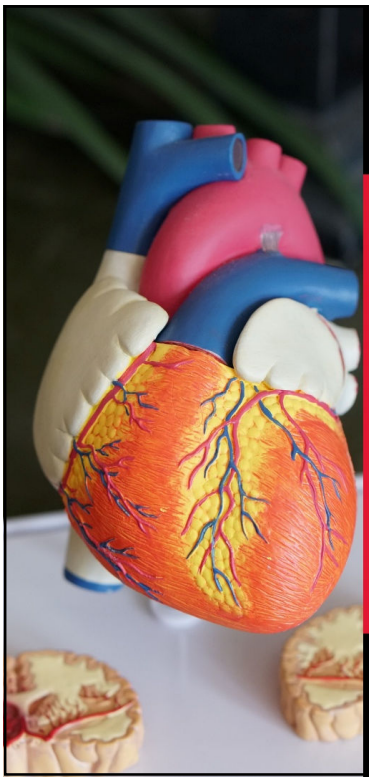

Causes of Acute Cardiogenic Shock

- ~~Conduction System~~
- Ventricles
- Pericardium
- Valves

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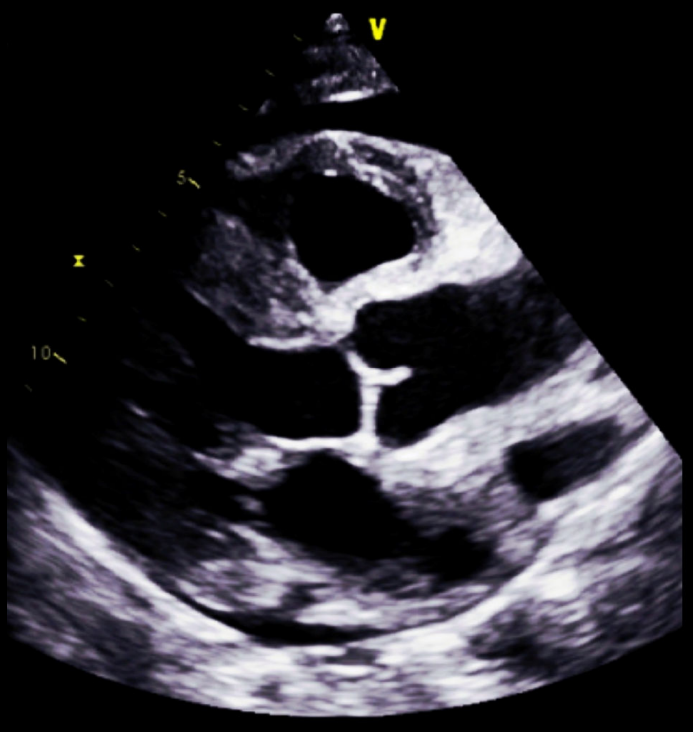
Remember to POCUS!



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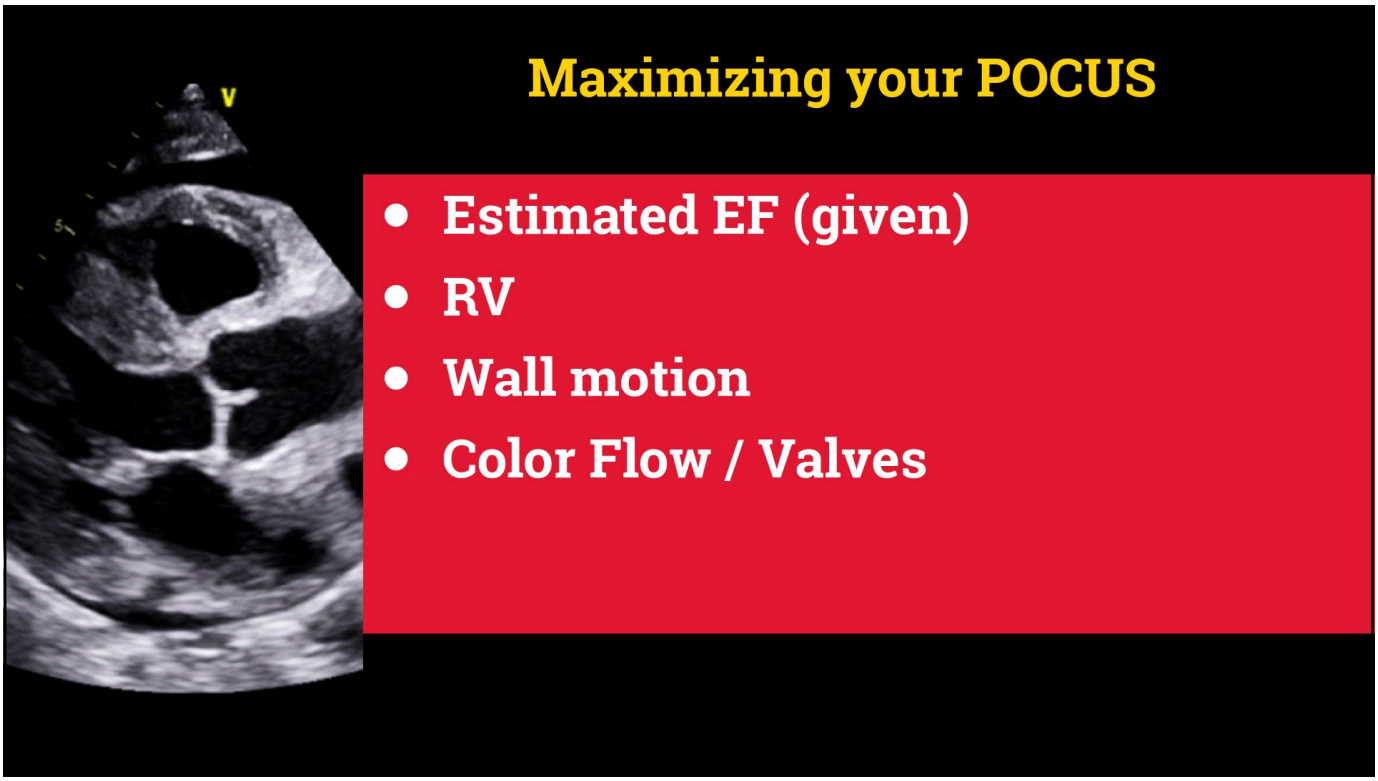
Maximizing your POCUS
Estimated EF (given)
RV
Wall motion
Color Flow / Valves

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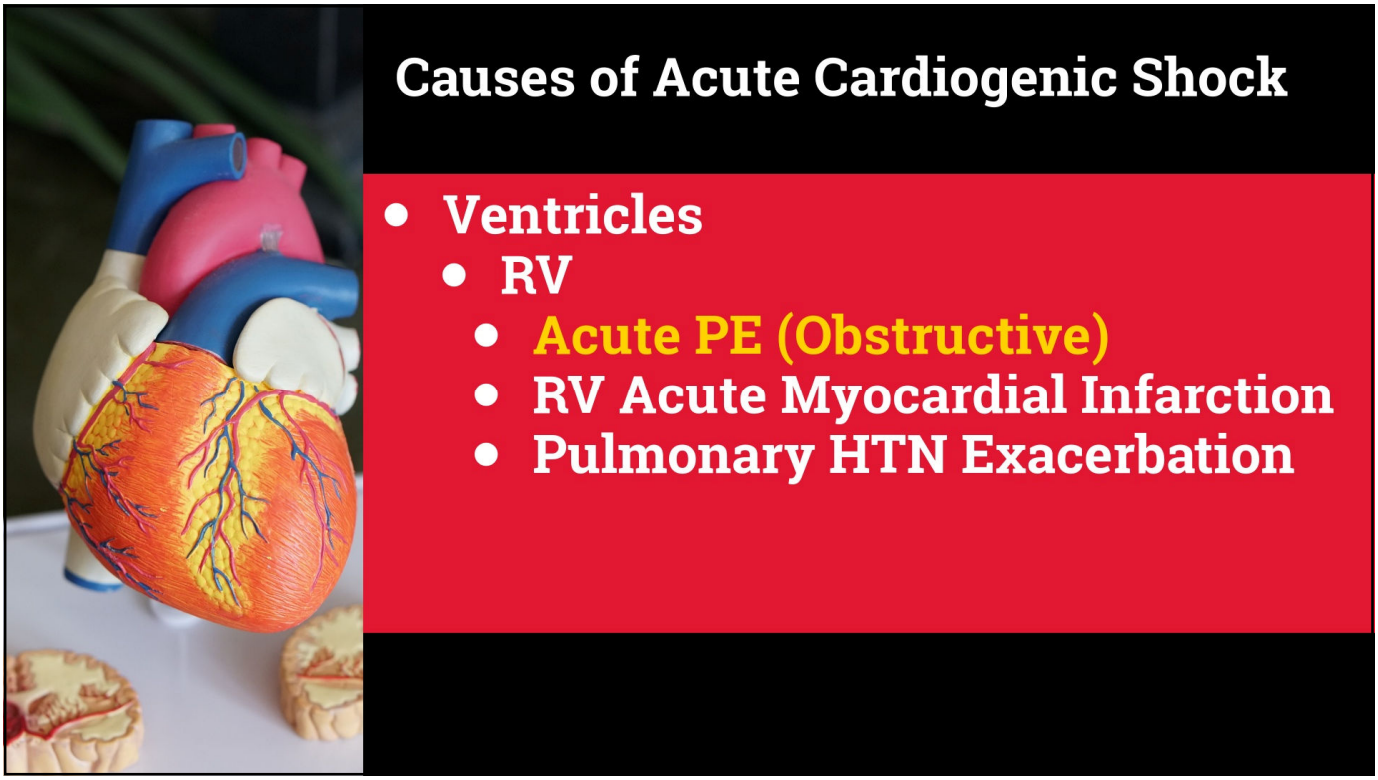
Causes of Acute Cardiogenic Shock
Ventricles
RV
Acute PE (Obstructive)
RV Acute Myocardial Infarction
Pulmonary HTN Exacerbation

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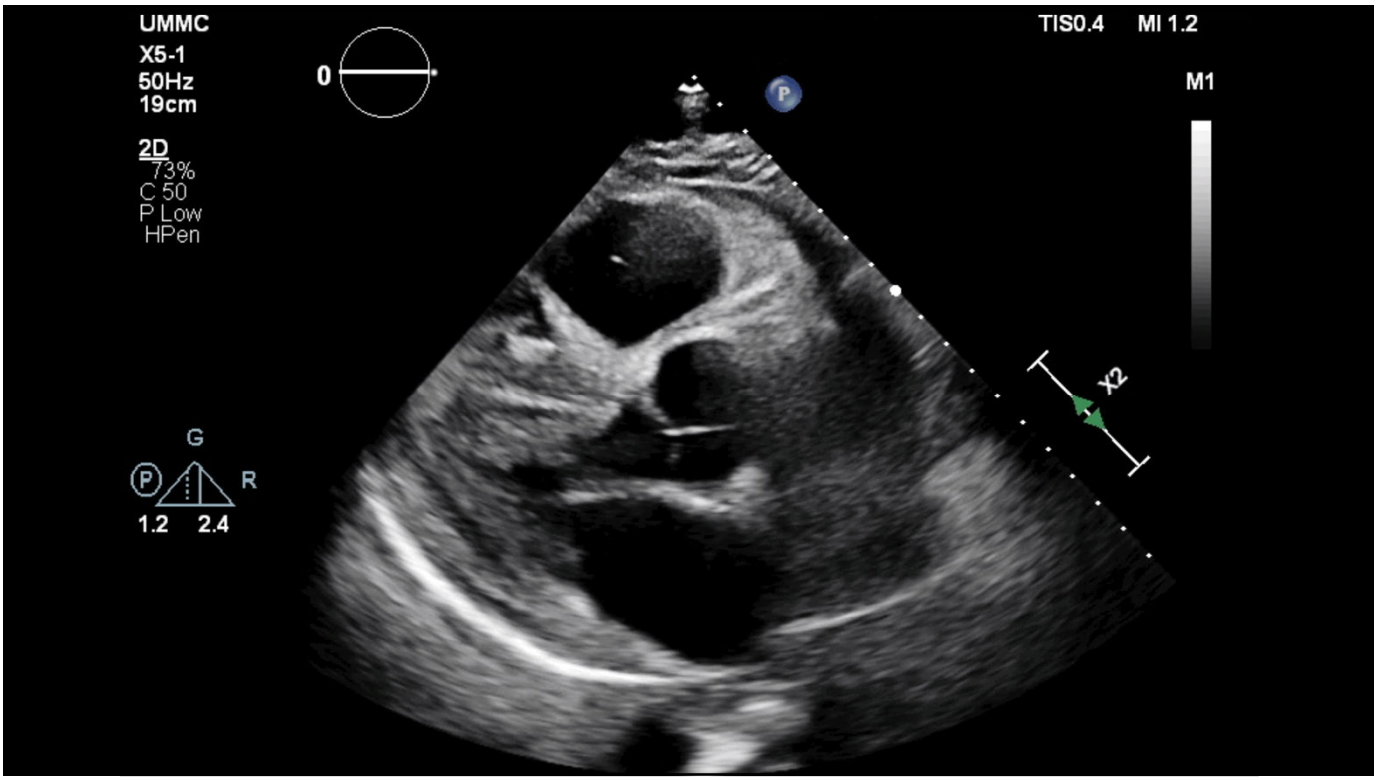
UMMC
X5-1
50Hz
19cm
2D
73%
C 50
P Low
HPen
TIS0.4 MI 1.2
M1
X2
G
R
1.2 2.4

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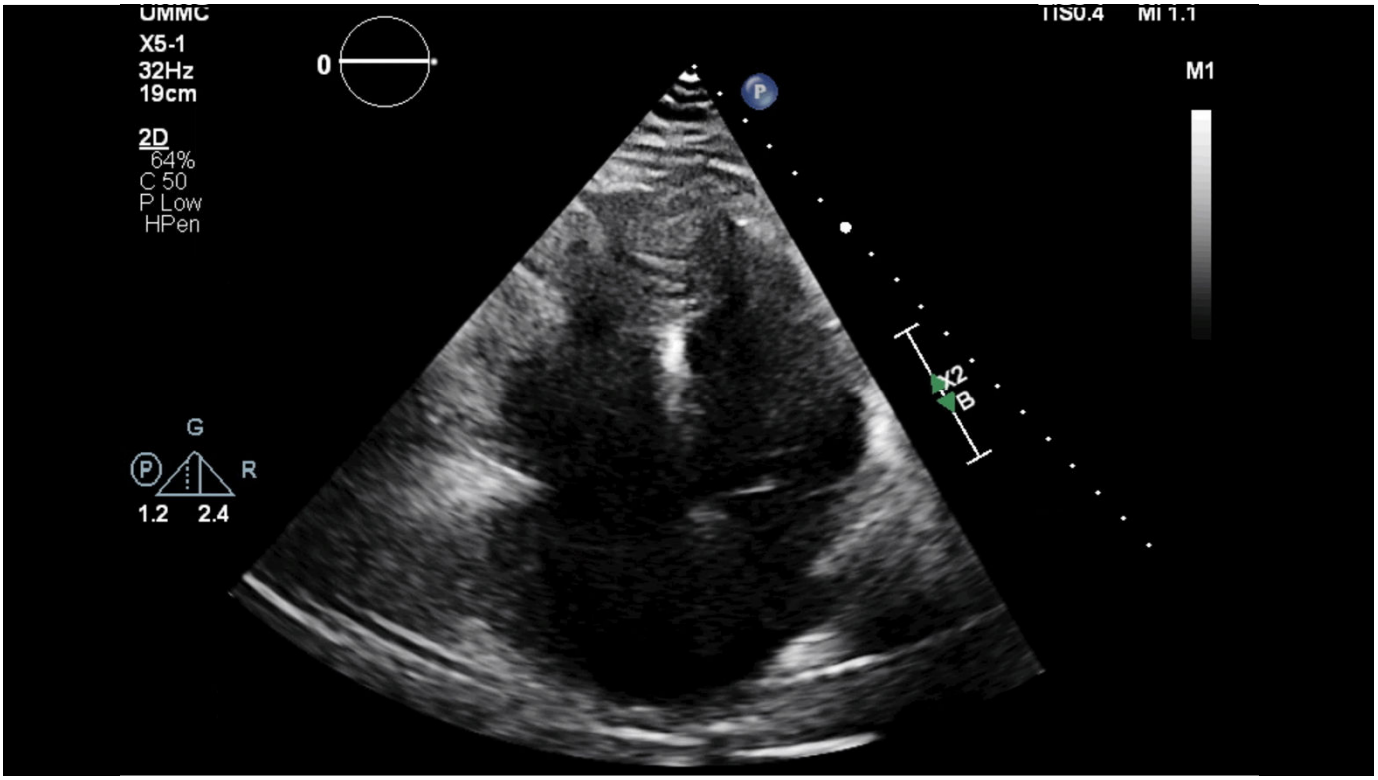
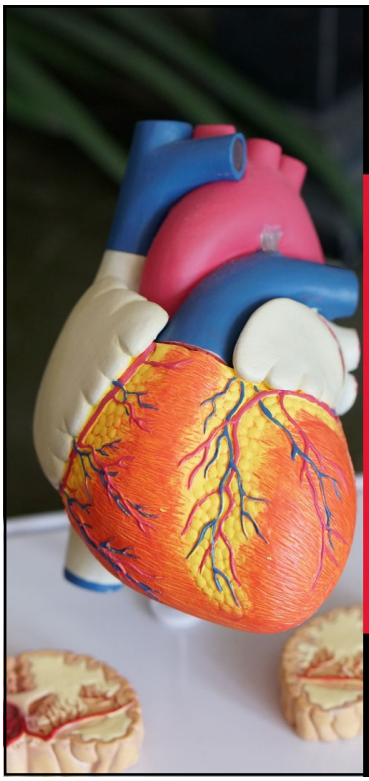

UMMC
X5-1
32Hz
19cm
2D
64%
C 50
P Low
HPen
TIS0.4 MI 1.1
M1
X2
B
G
R
1.2 2.4

72



Causes of Acute Cardiogenic Shock

- Ventricles
  - LV
    - Acute Myocardial Infarction
    - Myocarditis

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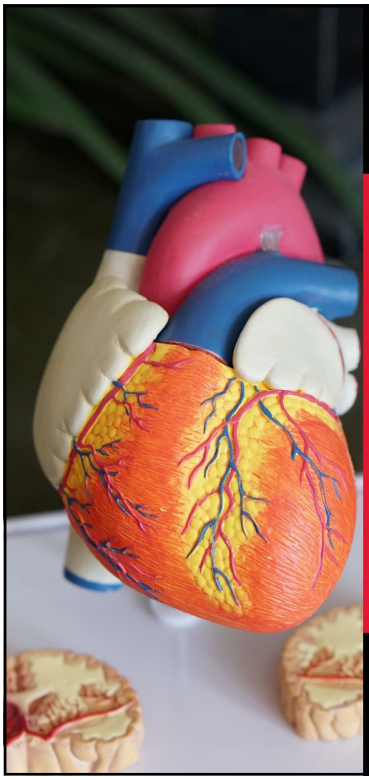

Causes of Acute Cardiogenic Shock

- Ventricles
  - LV
    - Stress Cardiomyopathy
    - Peripartum Cardiomyopathy
    - Septal or Free Wall Rupture

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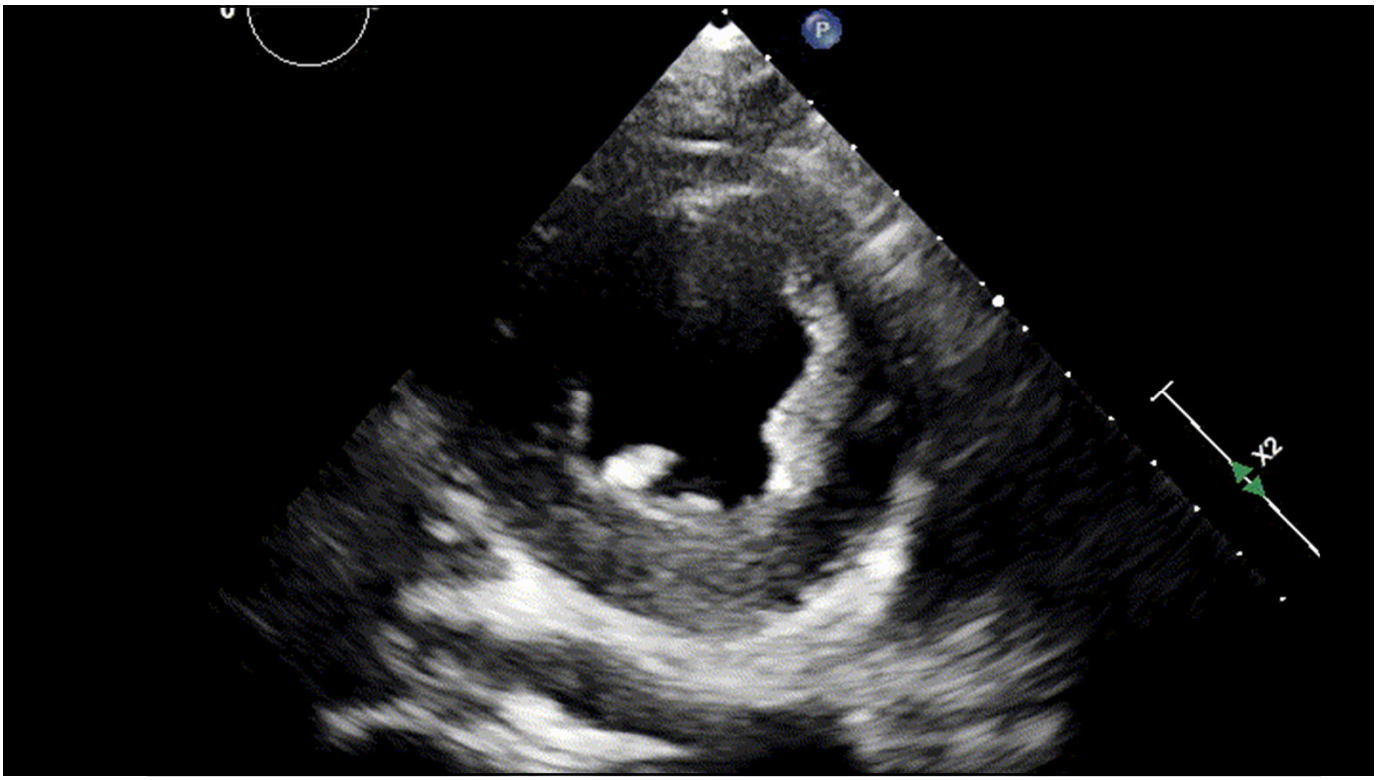
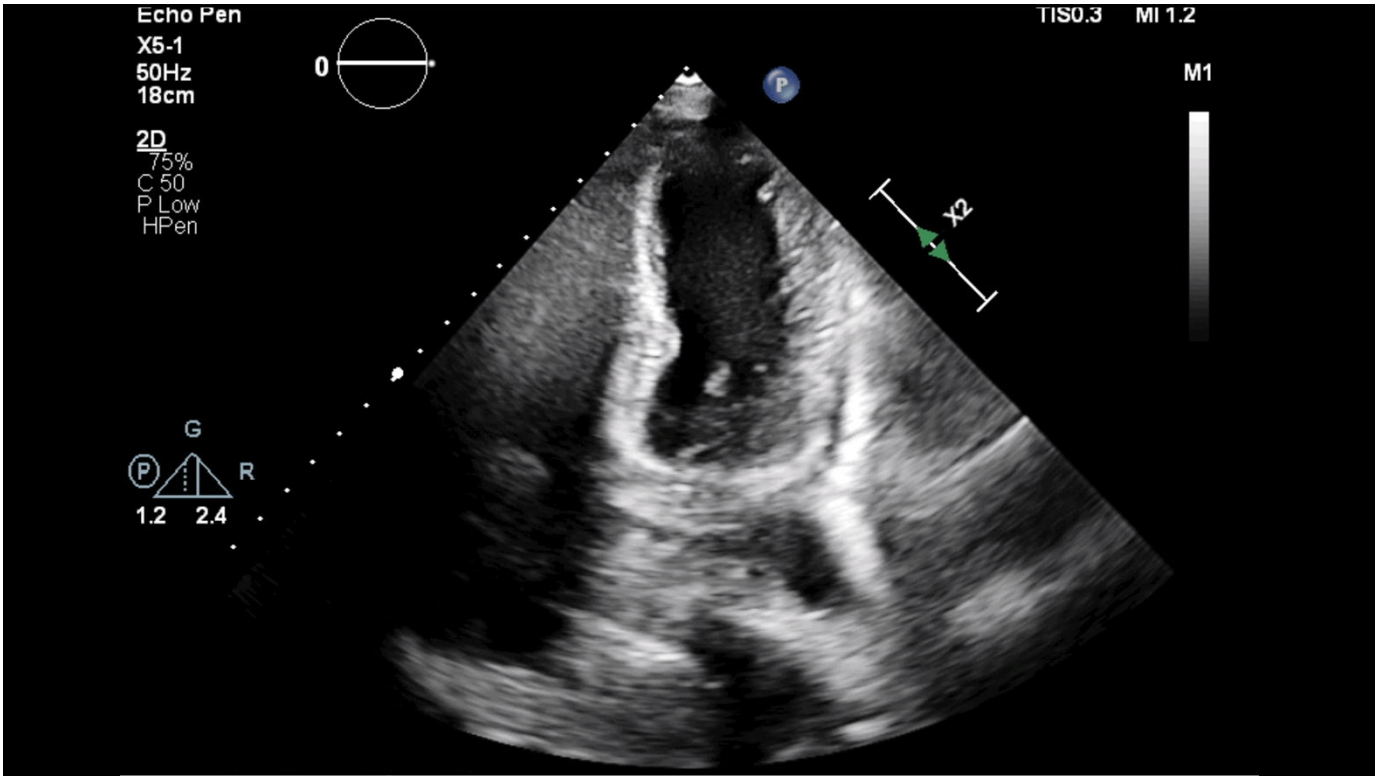
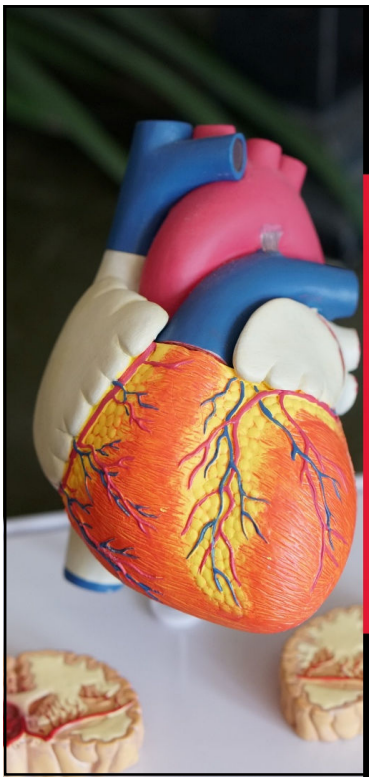

P
X2

75


Echo Pen
X5-1
50Hz
18cm
2D
75%
C 50
P Low
HPen
0
TIS0.3
MI 1.2
M1
P
X2
G
P
R
1.2
2.4

76



Causes of Acute Cardiogenic Shock

- **Pericardial**
  - **Tamponade (Obstructive)**

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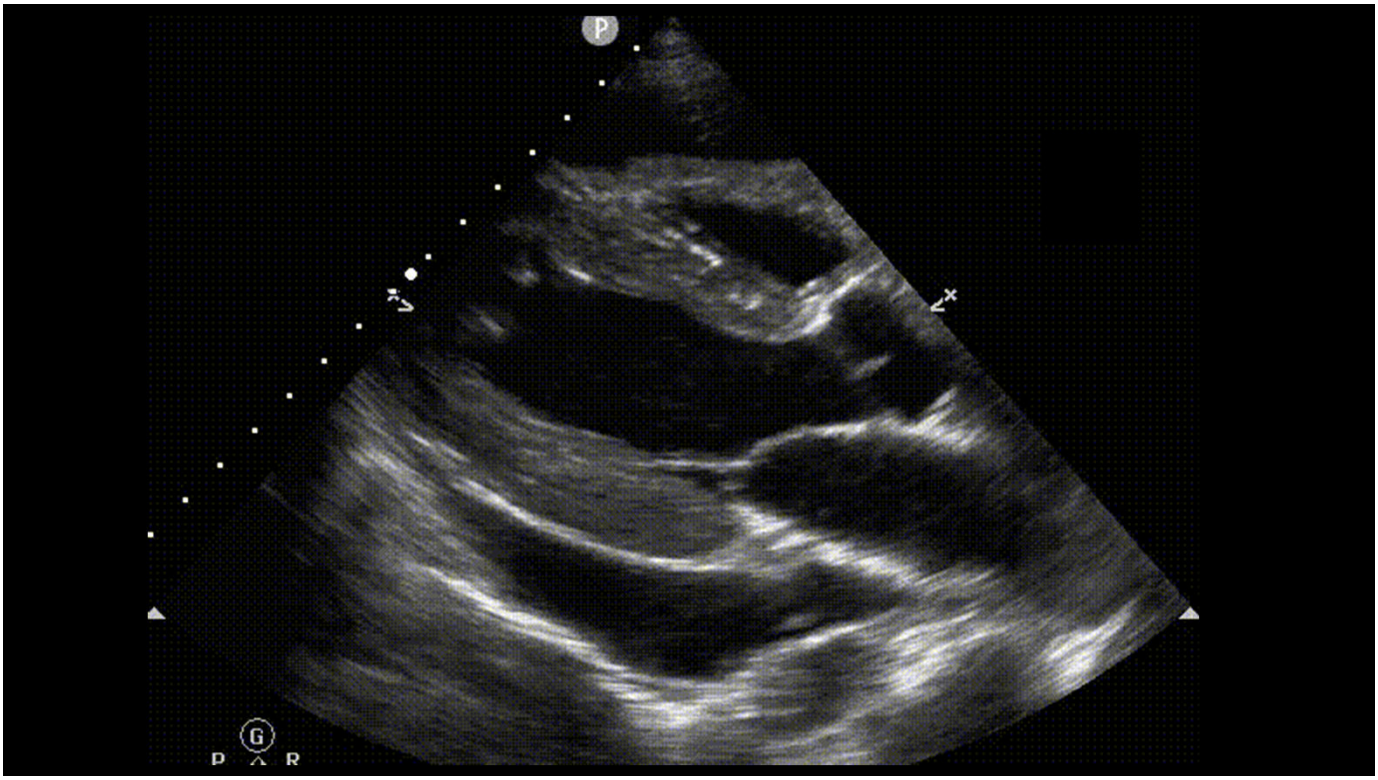

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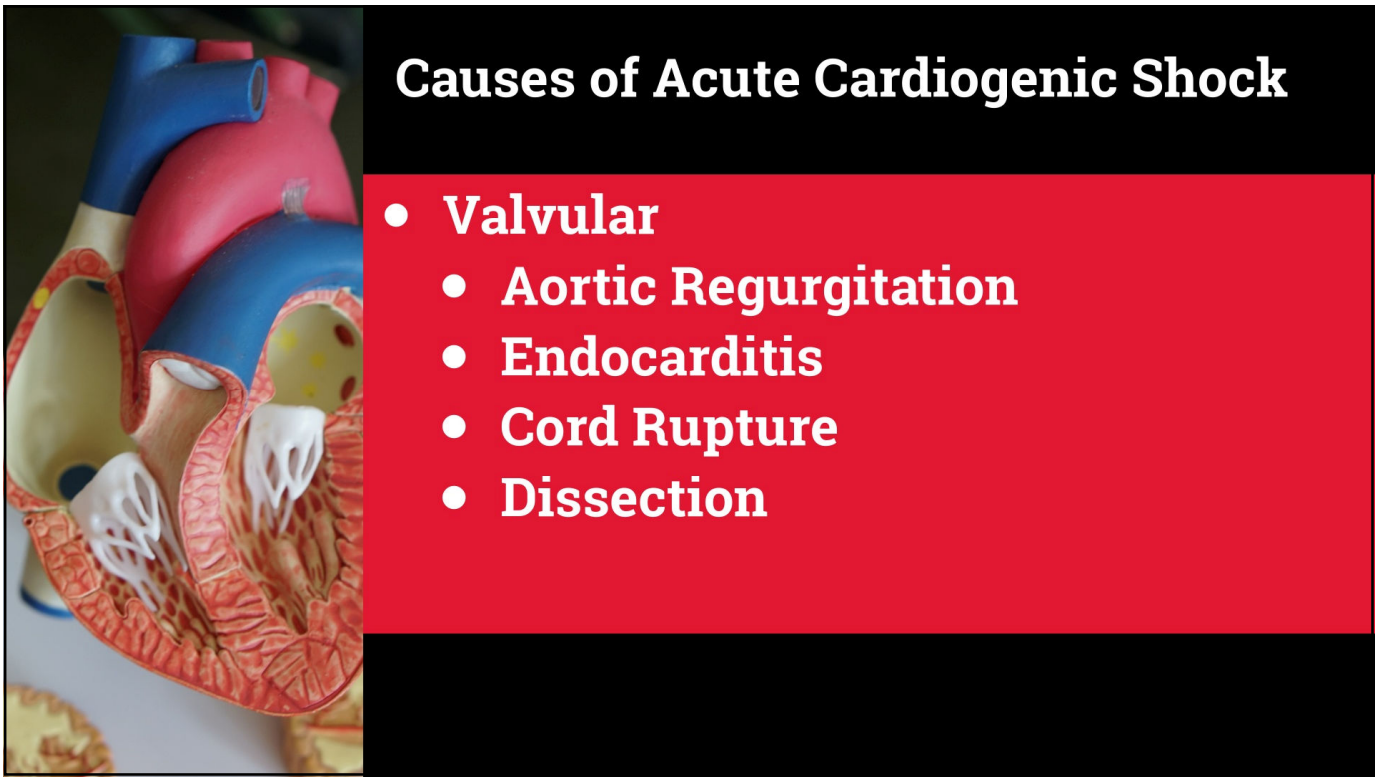
Causes of Acute Cardiogenic Shock
Valvular
Aortic Regurgitation
Endocarditis
Cord Rupture
Dissection

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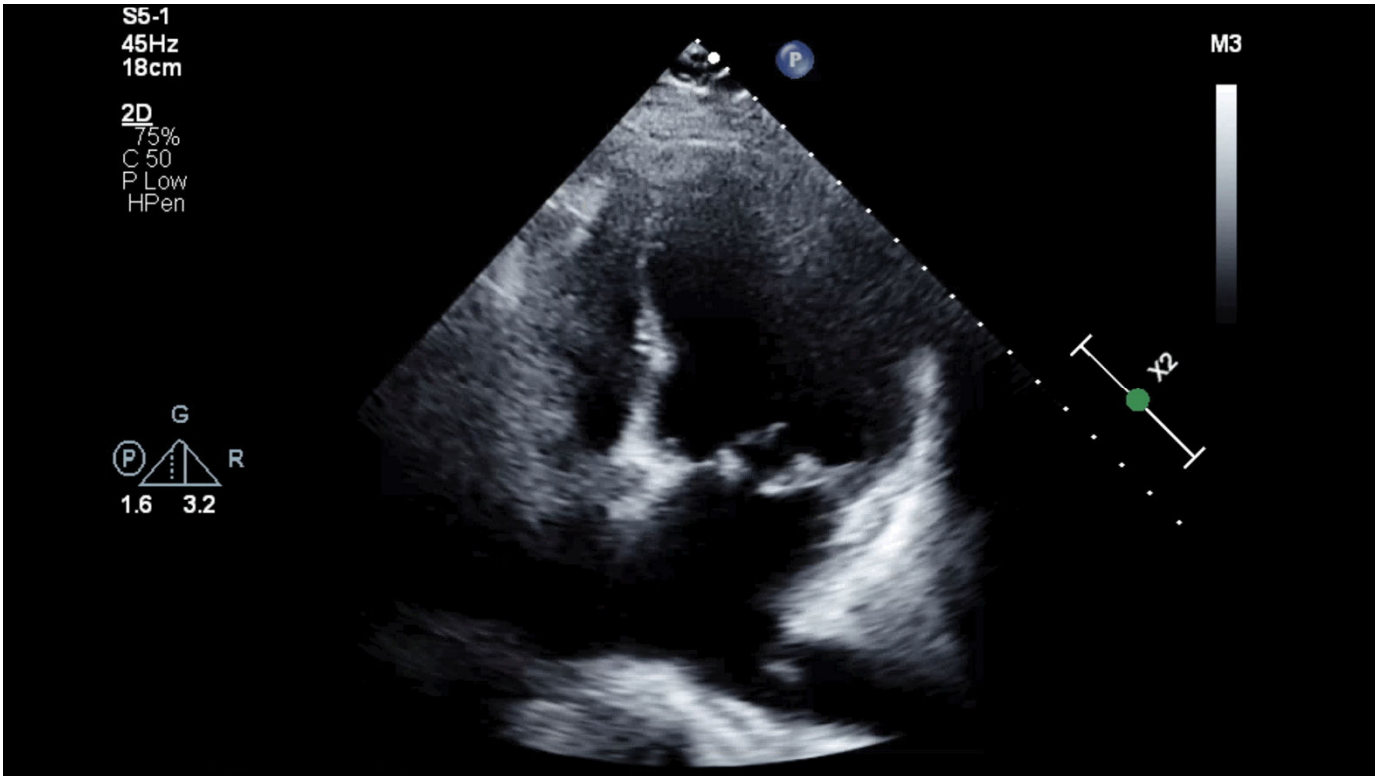
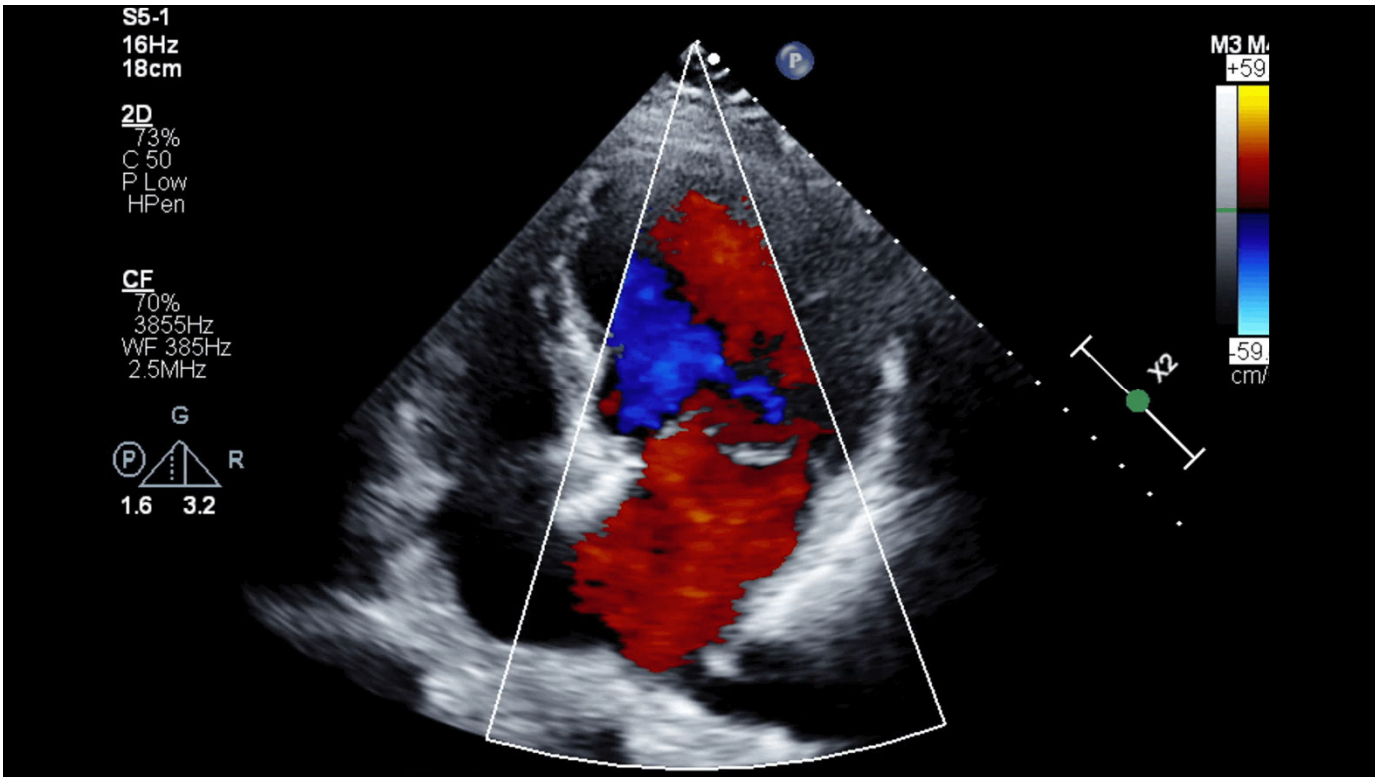
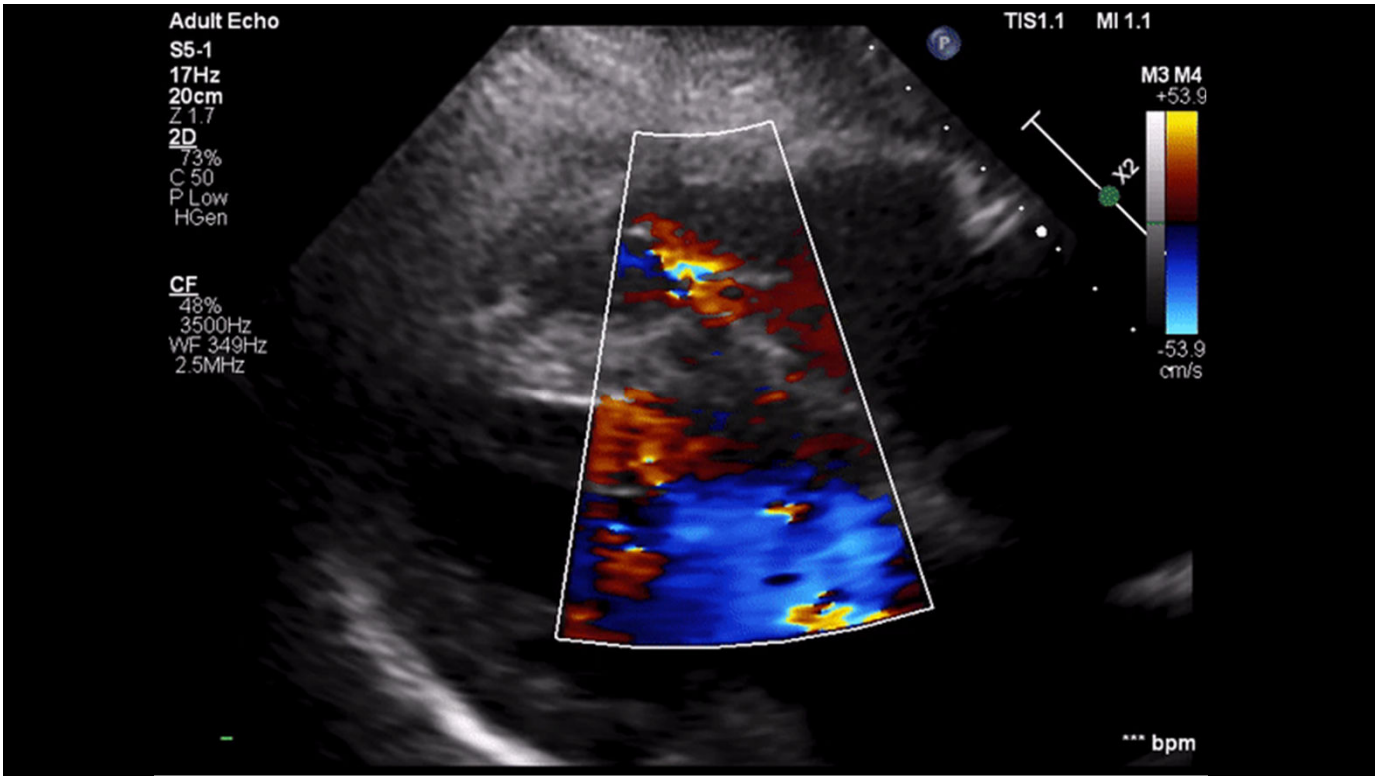

S5-1
45Hz
18cm
2D
75%
C 50
P Low
HPen
M3
X2
G
P
R
1.6
3.2

80


S5-1
16Hz
18cm
2D
73%
C 50
P Low
HPen
CF
70%
3855Hz
WF 385Hz
2.5MHz
G
R
1.6
3.2
M3 M
+59
-59
cm/
X2

81


Adult Echo
S5-1
17Hz
20cm
Z 1.7
2D
73%
C 50
P Low
HGen
CF
48%
3500Hz
WF 349Hz
2.5MHz
TIS1.1
MI 1.1
M3 M4
+53.9
-53.9
cm/s
X2
*** bpm

82

ERRORS AND ASSUMPTIONS OF RECOGNITION

Lesson: Use POCUS to help evaluate the etiology!

83

Agenda

Errors and Assumptions of Recognition

1. Assumptions re: Vitals and Physical Exam
2. Misinterpreting Labs
3. Fatal assumptions re: Etiology

84

SUMMARY

Recognizing Cardiogenic Shock

85

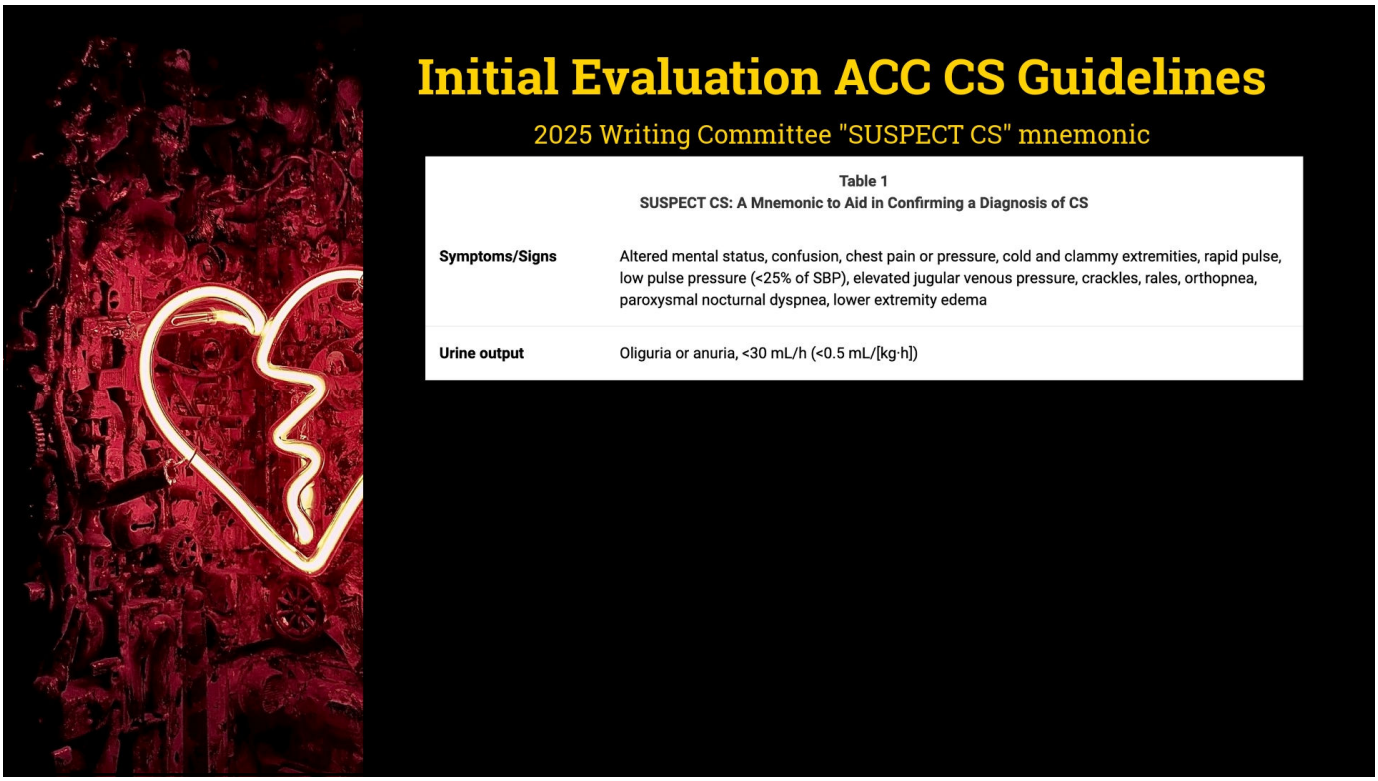
Initial Evaluation ACC CS Guidelines

2025 Writing Committee "SUSPECT CS" mnemonic

Table 1
SUSPECT CS: A Mnemonic to Aid in Confirming a Diagnosis of CS

Symptoms/Signs	Altered mental status, confusion, chest pain or pressure, cold and clammy extremities, rapid pulse, low pulse pressure (<25% of SBP), elevated jugular venous pressure, crackles, rales, orthopnea, paroxysmal nocturnal dyspnea, lower extremity edema
Urine output	Oliguria or anuria, <30 mL/h (<0.5 mL/[kg·h])
Sustained hypotension	SBP <90 mm Hg, MAP <65 mm Hg for >30 min or a >30-mm Hg decrease from baseline, or the need for pharmacological or mechanical support to maintain SBP >90 mm Hg
Perfusion	Evaluate markers of end-organ malperfusion, including lactic acid >2 mmol/L, ALT >200 U/L or >3× upper limit of normal, creatinine ≥2× upper limit of normal, pH <7.2, metabolic acidosis without another known cause
ECG/Echocardiogram	Evaluate acute ischemia, including ECG and sonographic evidence of STEMI (regional wall motion abnormalities); evidence of LV or RV dilation and systolic dysfunction; valvular pathology
Congestion	Presence or absence of congestion based on physical signs and hemodynamics; elucidation of ventricular involvement (LV vs RV vs BiV)
Triage	Appropriate triage/shock team activation or possible transfer to a higher level of care

86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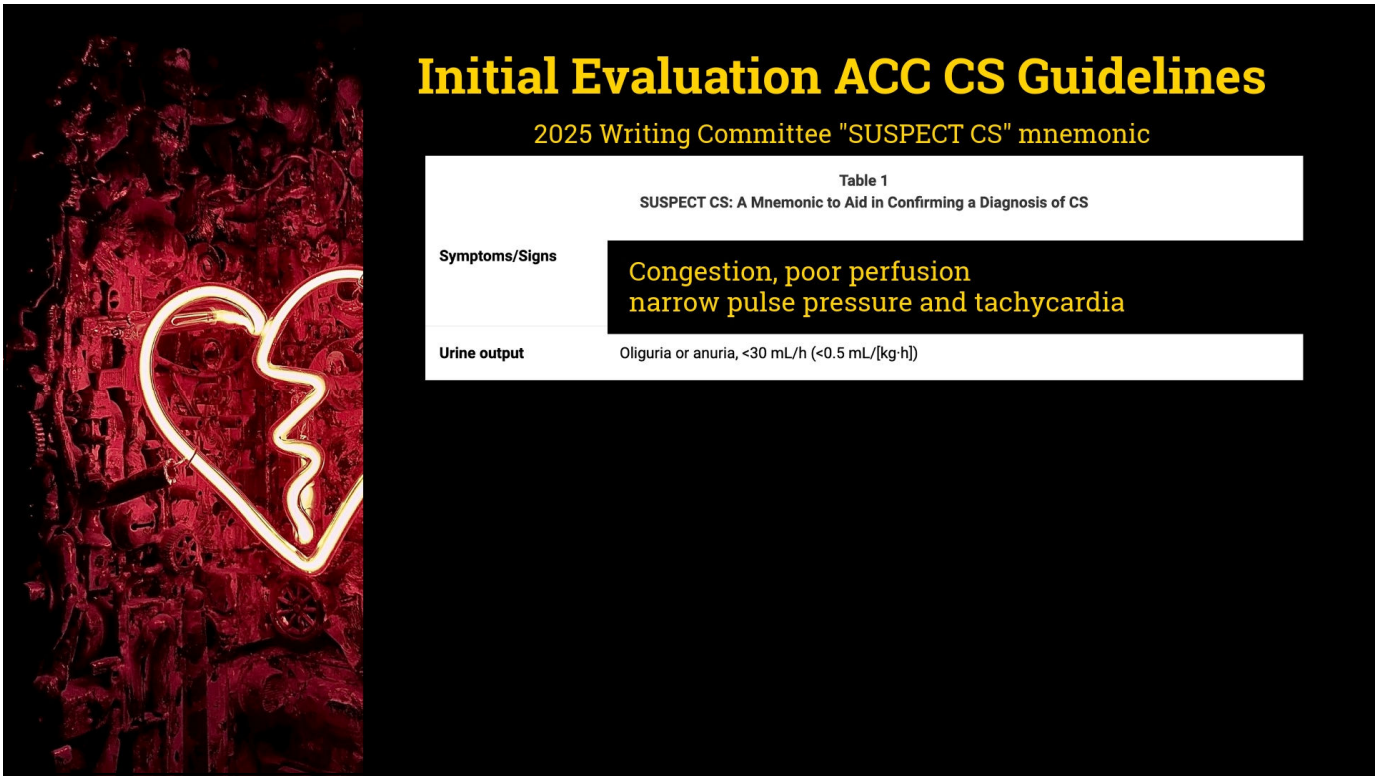
Urine output
Oliguria or anuria, <30 mL/h (<0.5 mL/[kg·h])

87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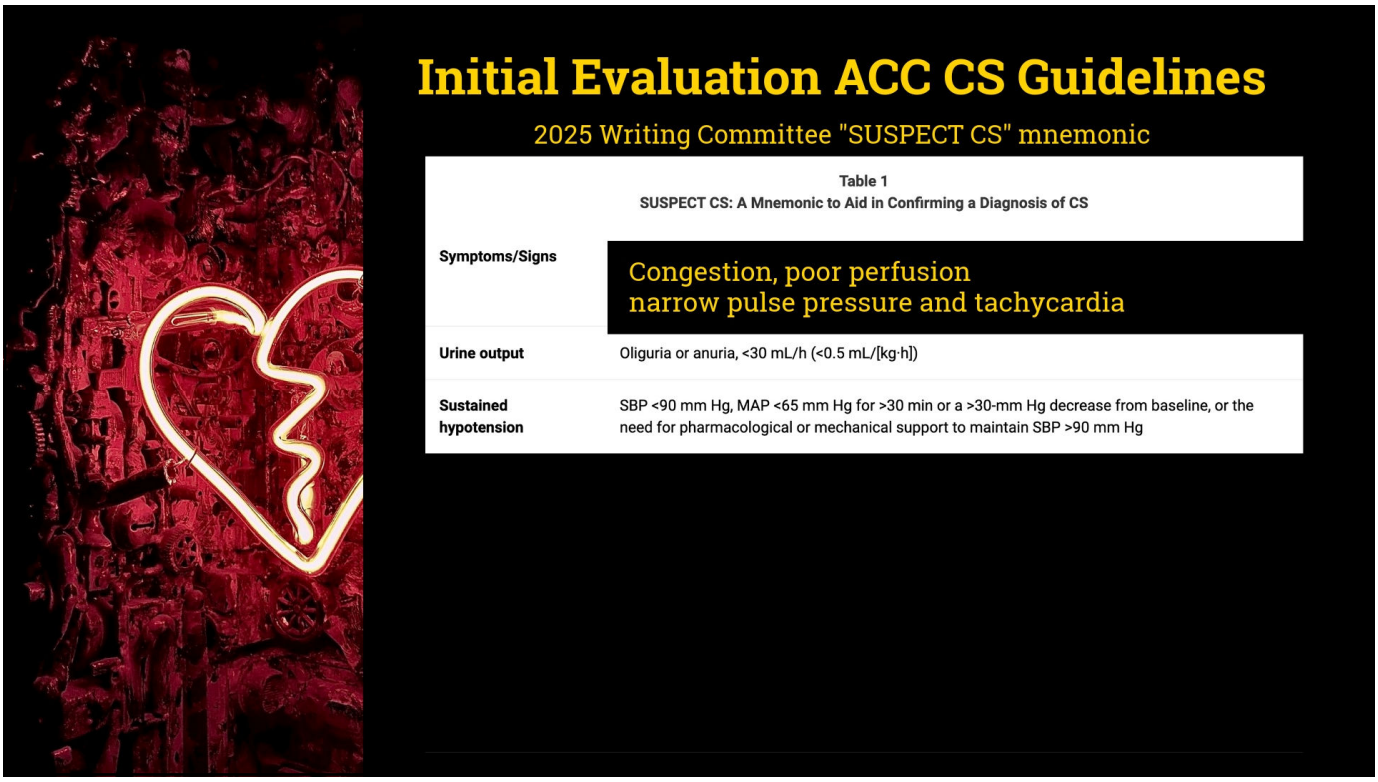
88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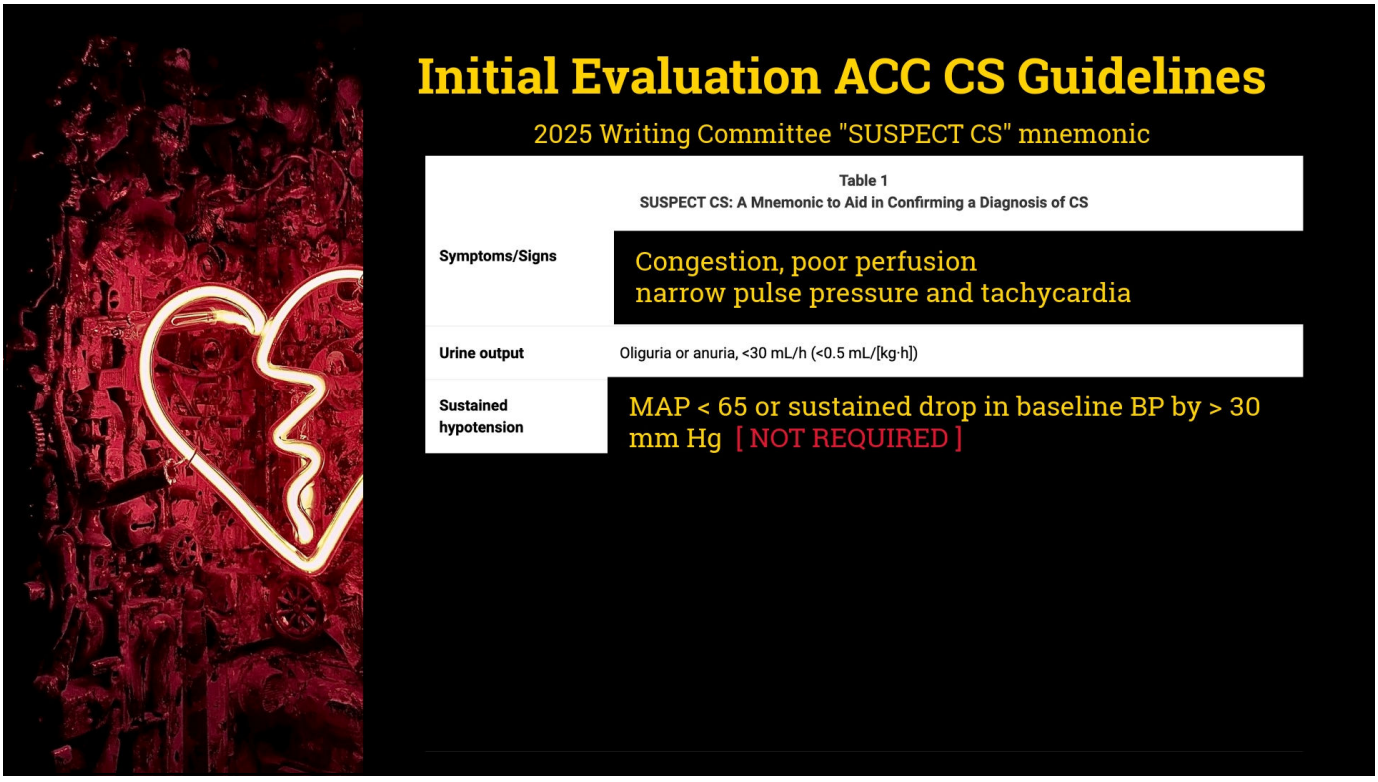
89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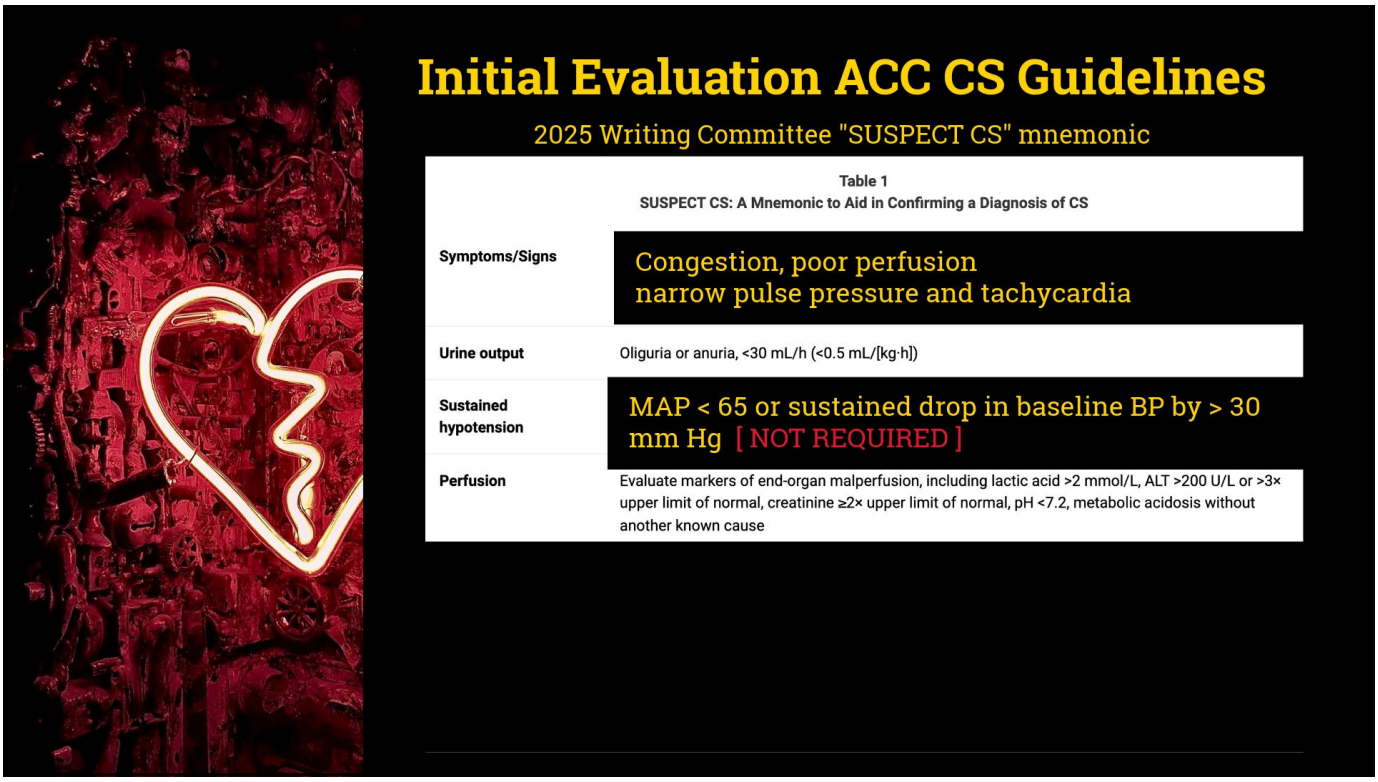
Sustained hypotension
MAP < 65 or sustained drop in baseline BP by > 30 mm Hg [NOT REQUIRED]

90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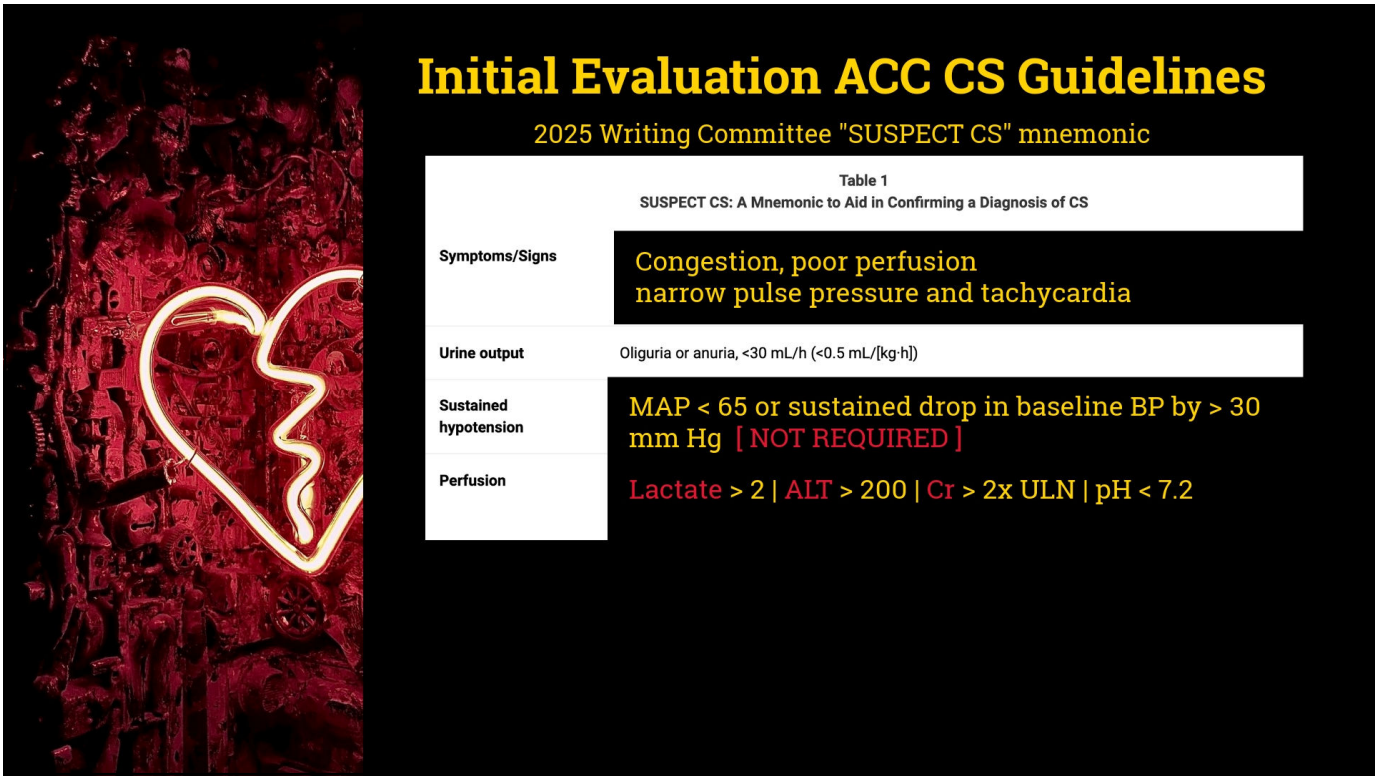
91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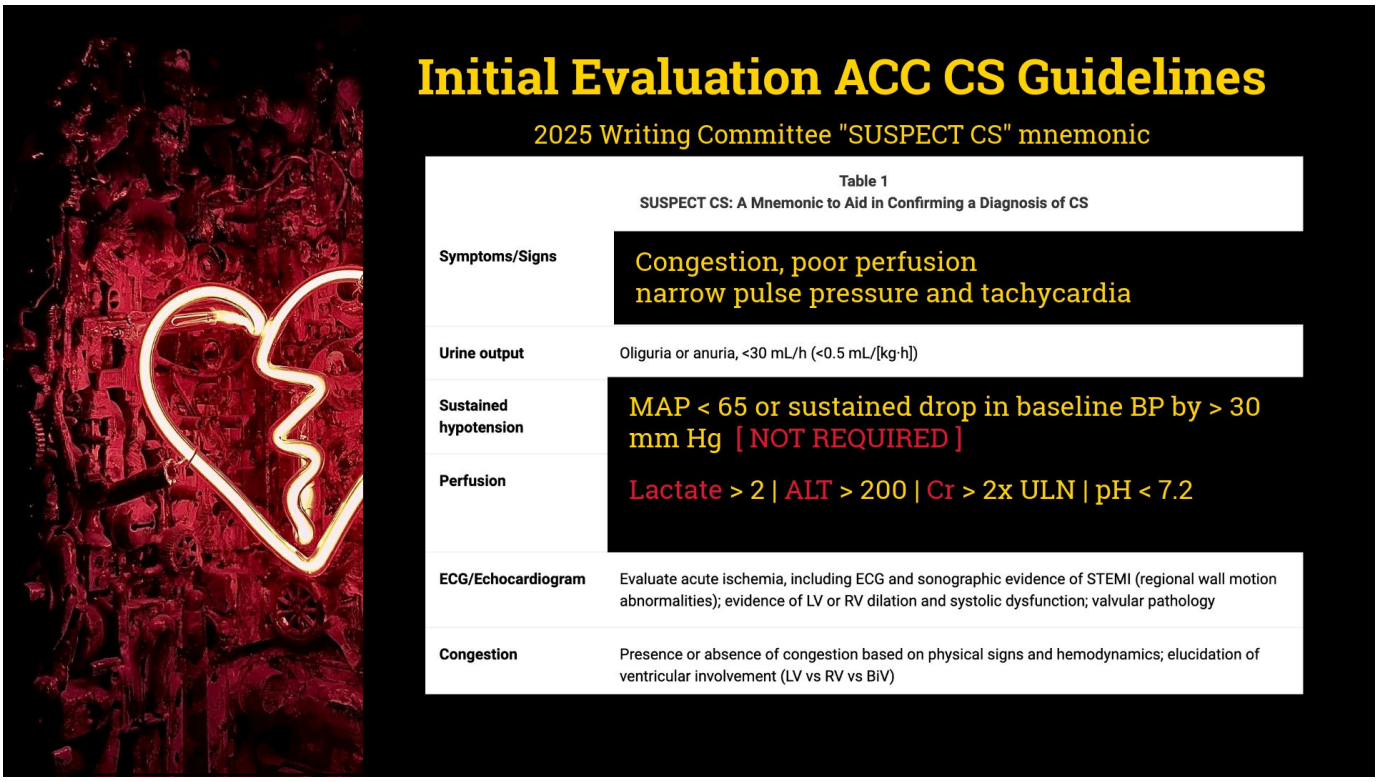
Sustained hypotension
MAP < 65 or sustained drop in baseline BP by > 30 mm Hg [NOT REQUIRED]
Perfusion
Lactate > 2 | ALT > 200 | Cr > 2x ULN | pH < 7.2

92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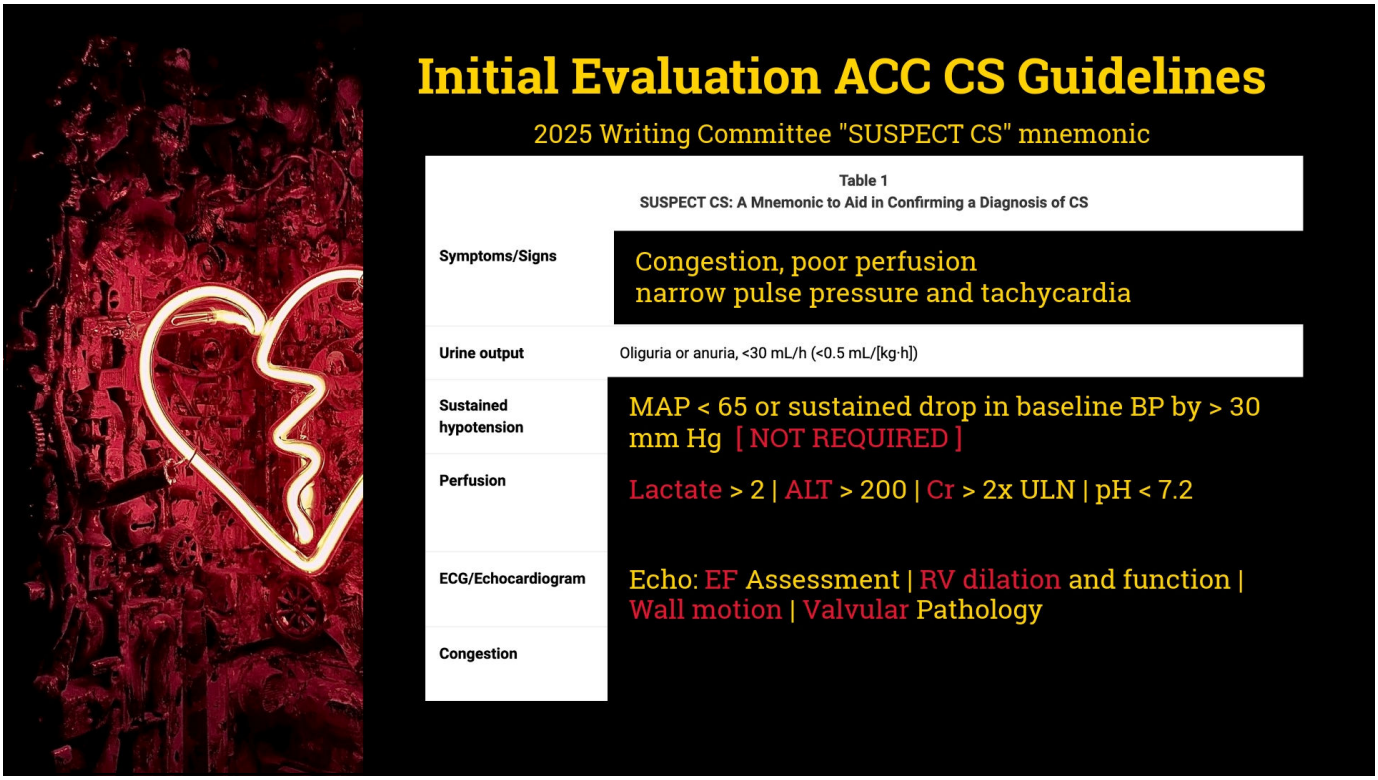
Presence or absence of congestion based on physical signs and hemodynamics; elucidation of ventricular involvement (LV vs RV vs BiV)

93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MAP < 65 or sustained drop in baseline BP by > 30 mm Hg [NOT REQUIRED]
Perfusion
Lactate > 2 | ALT > 200 | Cr > 2x ULN | pH < 7.2
ECG/Echocardiogram
Echo: EF Assessment | RV dilation and function | Wall motion | Valvular Pathology
Congestion

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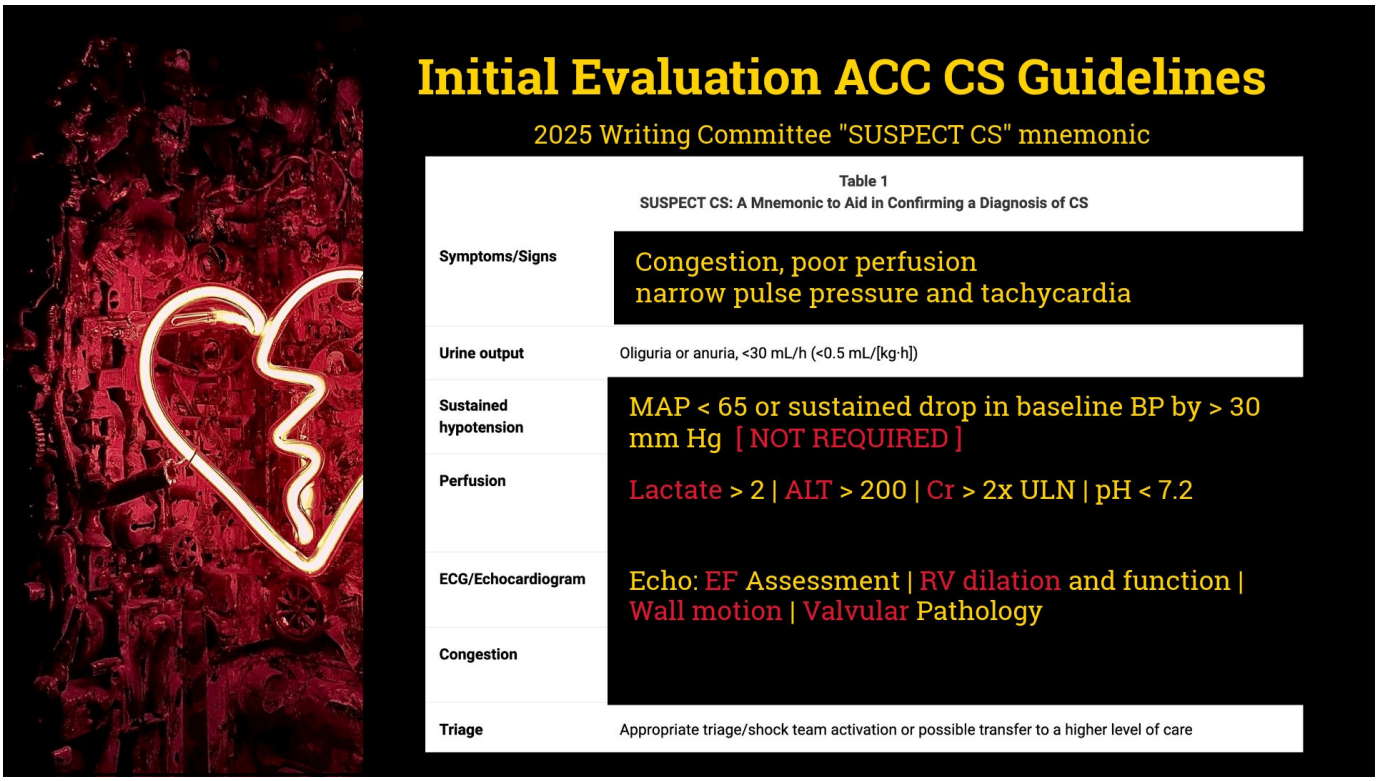
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ECG/Echocardiogram
Echo: EF Assessment | RV dilation and function | Wall motion | Valvular Pathology
Congestion
Triage
Appropriate triage/shock team activation or possible transfer to a higher level of care

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Management of CS

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Agenda

Management

1. A: Airway
2.
3.
4.
5.

97

Agenda

Management

1. A: Airway
2. E: Etiology treatment
3.
4.
5.

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Agenda

Management

1. A: Airway
2. E: Etiology treatment
3. I: Inotropy (and vasoactives)
4.
5.

99

Agenda

Management

1. A: Airway
2. E: Etiology treatment
3. I: Inotropy (and vasoactives)
4. O: Offloading the heart
5.

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Agenda

Management

1. A: Airway
2. E: Etiology treatment
3. I: Inotropy (and vasoactives)
4. O: Offloading the heart
5. U: Upgrade Support

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Agenda

Management

1. **A: Admit**
2.
3.
4.
5.

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Agenda

Management

1. **A: Admit**
2. **E: Exit the ED**
3.
4.
5.

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Agenda

Management

1. A: Admit
2. E: Exit the ED
3. I: ICU Page Sent
4.
5.

105

Agenda

Management

1. A: Admit
2. E: Exit the ED
3. I: ICU Page Sent
4. O: Off my hands, signing out!
5.

106

Agenda

Management

1. A: Admit
2. E: Exit the ED
3. I: ICU Page Sent
4. O: Off my hands, signing out!
5. U: Unavailable for questions

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ERRORS AND ASSUMPTIONS OF MANAGEMENT

Fatal Error: Ignoring RV failure

108

Airway

Oxygenation and Ventilation

- 50-88% of CS patients require respiratory support (Alviar et al 2020)

109

Airway

Oxygenation and Ventilation

- 50-88% of CS patients require respiratory support (Alviar et al 2020)
- Positive Pressure Ventilation (PPV) reduces preload and afterload

110

Airway

Oxygenation and Ventilation

- 50-88% of CS patients require respiratory support (Alviar et al 2020)
- Positive Pressure Ventilation (PPV) reduces preload and afterload
- Median duration of mechanical ventilation 3-3.3 days

111

Airway

Oxygenation and Ventilation

- Consider MV in patients with:
  - Refractory hypoxia or hypercarbia

112

Airway

Oxygenation and Ventilation

- Consider MV in patients with:
  - Refractory hypoxia or hypercarbia
  - Increased work of breathing on NIMV

113

Airway

Oxygenation and Ventilation

- Consider MV in patients with:
  - Refractory hypoxia or hypercarbia
  - Increased work of breathing on NIMV
  - Patients who require mechanical cardiac support

114

Airway

Oxygenation and Ventilation

- Intubation is still high risk
  - Optimize pH, fluid status, BP
  - Sedation: no best recommendation

115

Airway

Oxygenation and Ventilation

- Intubation is still high risk
  - Optimize pH, fluid status, BP
  - Sedation: no best recommendation
  - No best mode of ventilation
    - Initial AC TV 6-8 mL/kg PEEP 5-10

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Airway

Oxygenation and Ventilation

- Cardiogenic shock with RV failure is much trickier.....

117

Airway

Oxygenation and Ventilation

- Cardiogenic shock with RV failure is much trickier.....
- Avoid intubation.

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Airway

Oxygenation and Ventilation

- Cardiogenic shock with RV failure is much trickier.....
- Avoid intubation.
  - > 35% Mortality
  - Consider High Flow Nasal Cannula
  - Consider early VA-ECMO

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Airway

Oxygenation and Ventilation

- PPV / PEEP decreases RV preload, increases RV afterload

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Airway

Oxygenation and Ventilation

- PPV / PEEP decreases RV preload, increases RV afterload
- Intubating RV failure (tamponade, PE)
  - MAP > 60 with pressors hanging
  - Avoid hypercapnia
  - Titrate up RR to normal pCO2
  - Initial PEEP 3-5

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ERRORS AND ASSUMPTIONS OF MANAGEMENT

Lesson: Determine whether there is RV failure before intubating CS

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Agenda

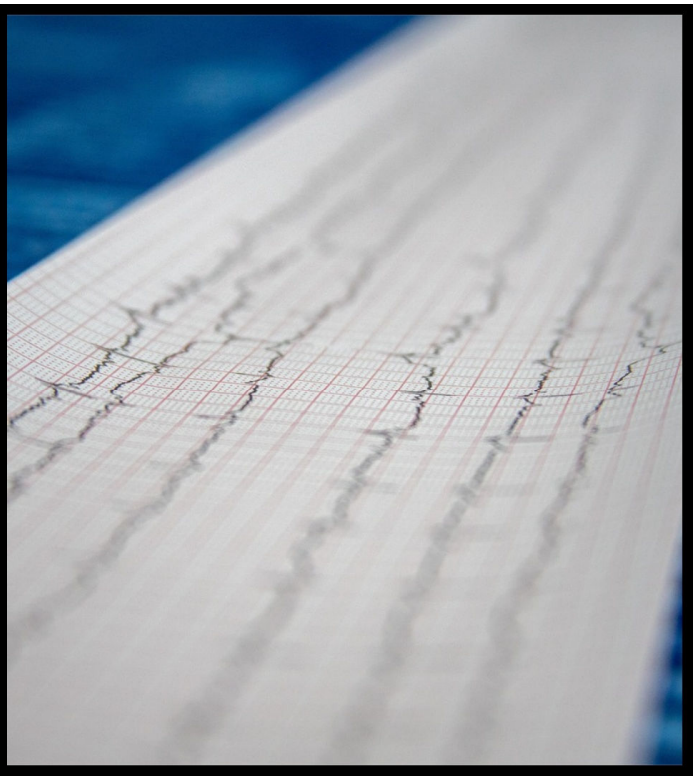
Management

- ✔ A: Airway
- ✔ E: Etiology treatment
- ✔ I: Inotropy (and vasoactives)
- ✔ O: Offloading the heart
- ✔ U: Upgrade Support

123

ERRORS AND ASSUMPTIONS OF MANAGEMENT

Fatal Error: Precipitating CS with IV beta blockers and calcium channel blockers

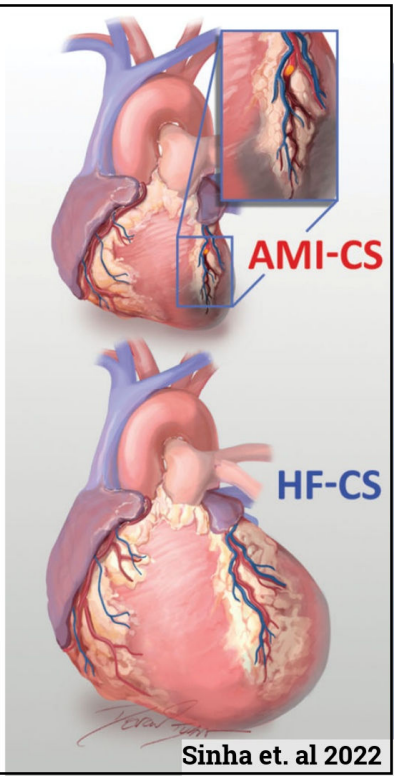


124

Etiology

Treat underlying etiology

- Acute Myocardial Infarction
- Heart Failure

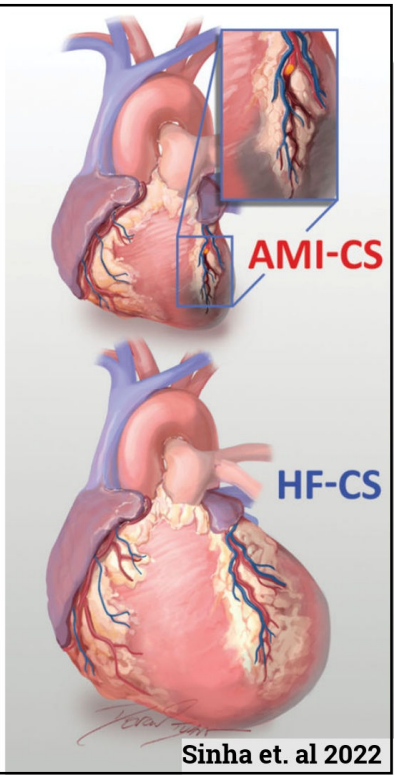


125

Etiology

Treat underlying etiology

- Acute Myocardial Infarction
  - STEMI / NSTEMI
    - acute bradyarrhythmias
    - acute tachyarrhythmias
    - postcardiac arrest
    - complications of MI
  - Cath Lab vs. Cardiac Surgery

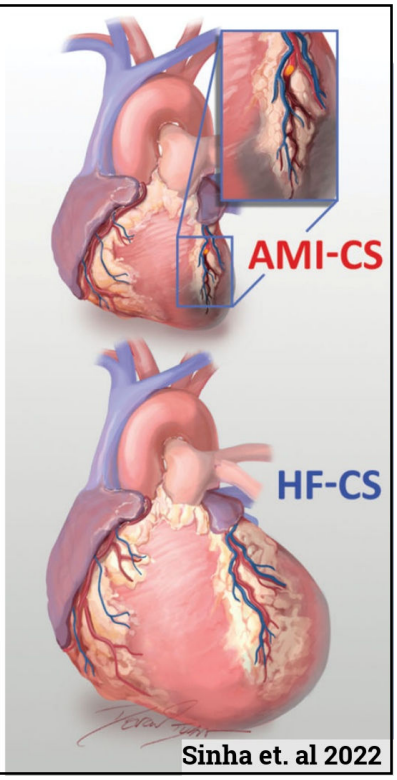


126

Etiology

Treat underlying etiology

- Heart Failure
  - Also Includes:
    - acute myocarditis
    - takotsubo | peripartum | tachycardia-related | hypertrophic cardiomyopathy
    - infiltrative diseases
  - Addressing congestion (diuresis)
  - Needs advanced heart failure therapies



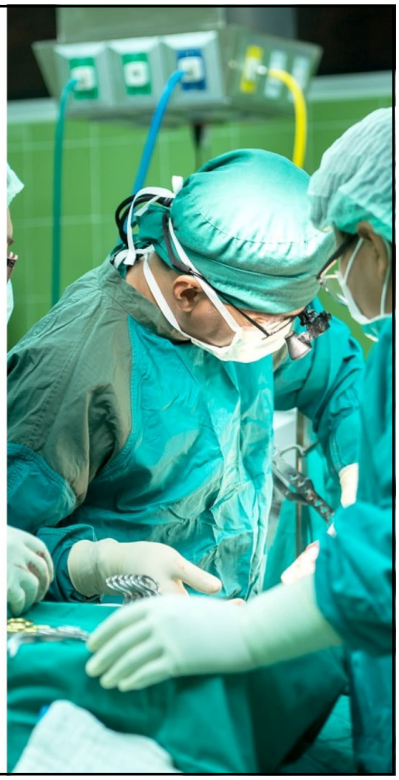
Sinha et. al 2022

127

Etiology

Treat underlying etiology

- PE - thrombolysis, thrombectomy
- Structural / Valvular CS - cardiac surg
- Tamponade - pericardiocentesis

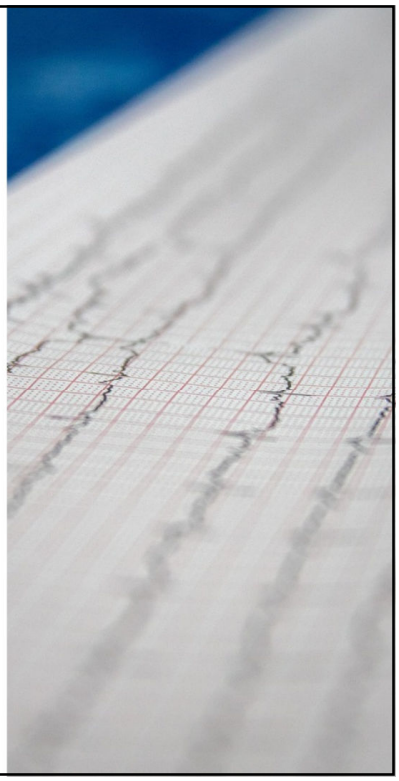


128

Etiology

Treat underlying etiology

- Arrhythmia poses special challenges

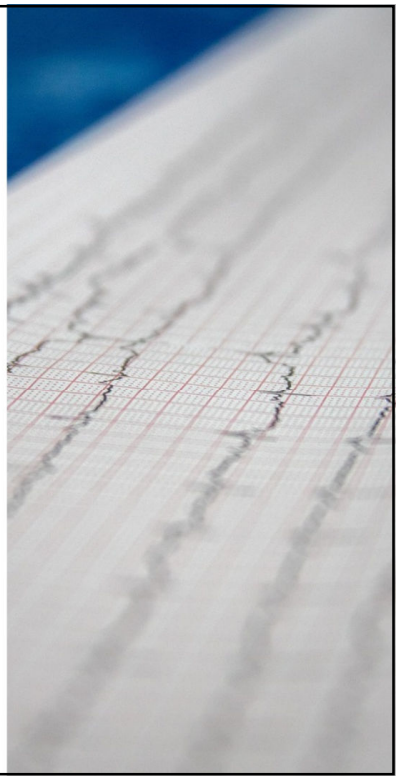


129

Etiology

Treat underlying etiology

- Arrhythmia is bad for the RV
- Mg > 2.2, K> 4
- Many antiarrhythmics are negative chronotropes

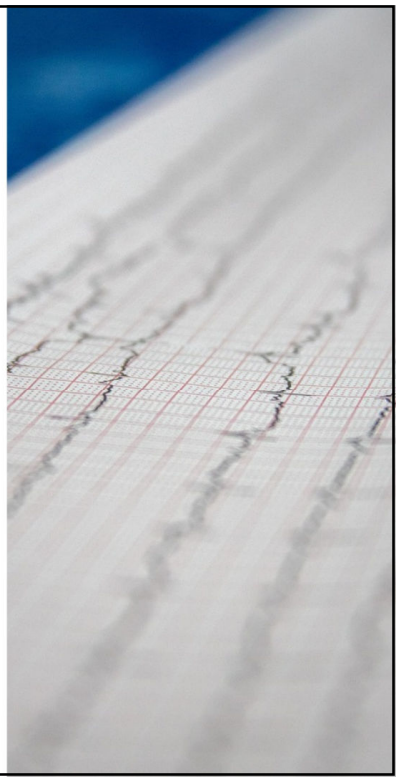


130

Etiology

Treat underlying etiology

- Arrhythmia is bad for the RV
- Mg > 2.2, K> 4
- Many antiarrhythmics are negative chronotropes
- Avoid IV beta blockers
- Avoid IV calcium channel blockers

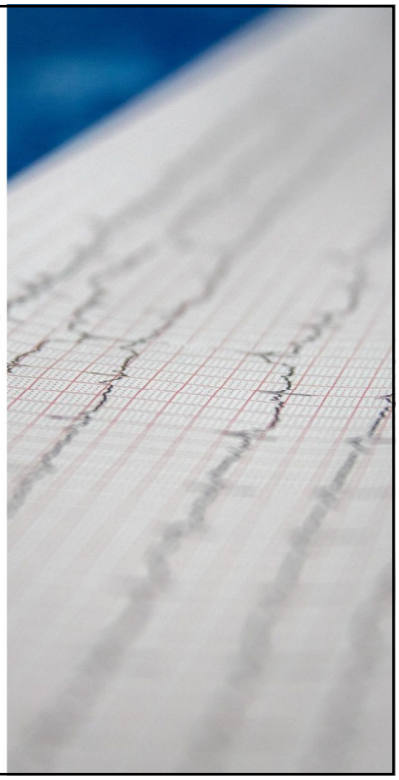


131

Etiology

Treat underlying etiology

- If arrhythmia is causing instability: Shock!
- Make extra sure it is not sinus tachycardia!

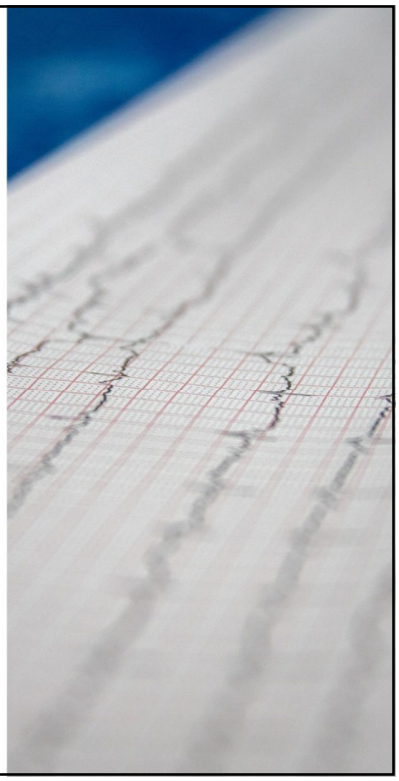


132

Etiology

Treat underlying etiology

- Atrial fibrillation with RVR
  - Make sure it is not compensatory!
  - Amiodarone
  - Digoxin

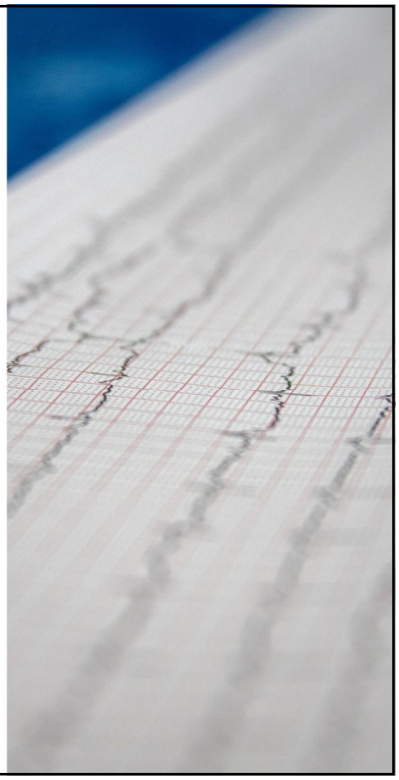


133

Etiology

Treat underlying etiology

- Decrease vasoactives and consider mechanical cardiac support



134

ERRORS AND ASSUMPTIONS OF MANAGEMENT

Lesson: Avoid IV BB and CCBs. Shock if there is an unstable arrhythmia

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Agenda

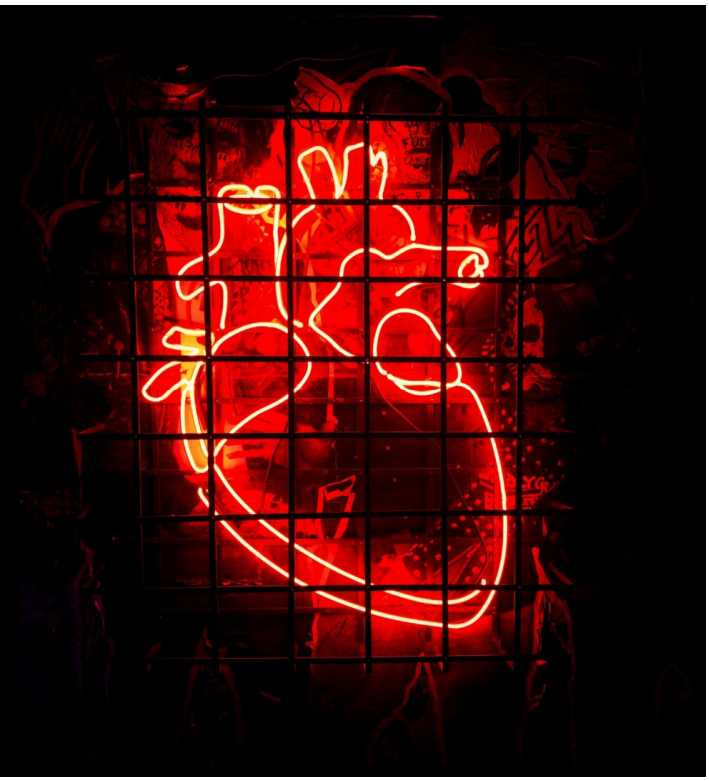
Management

- A: Airway
- E: Etiology treatment
- I: Inotropy (and vasoactives)
- O: Offloading the heart
- U: Upgrade Support

136

ERRORS AND ASSUMPTIONS OF MANAGEMENT

Fatal Error: Do not be afraid of Inotropes

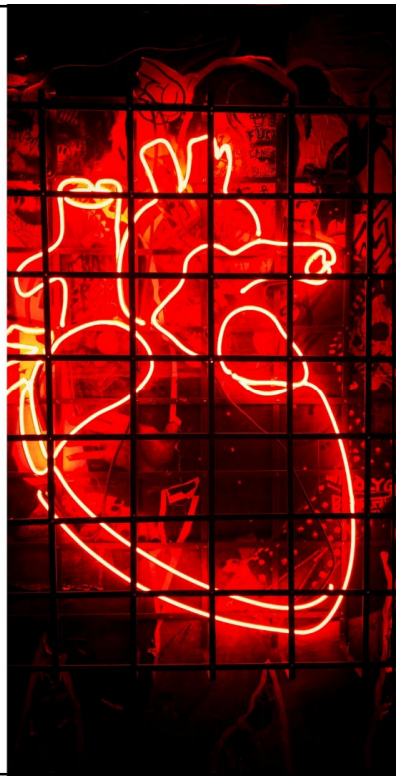


137

Inotropes and vasoactives

Pressors

- **Have pressors ready before inotropes**

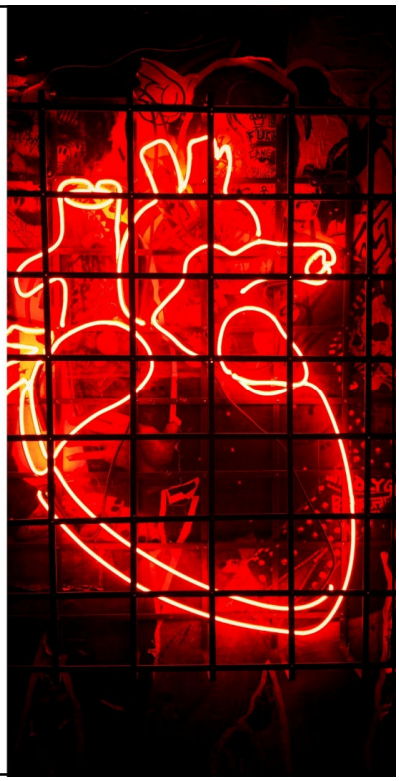


138

Inotropes and vasoactives

Pressors

- Have pressors ready before inotropes
- No trials for MAP goals in CS

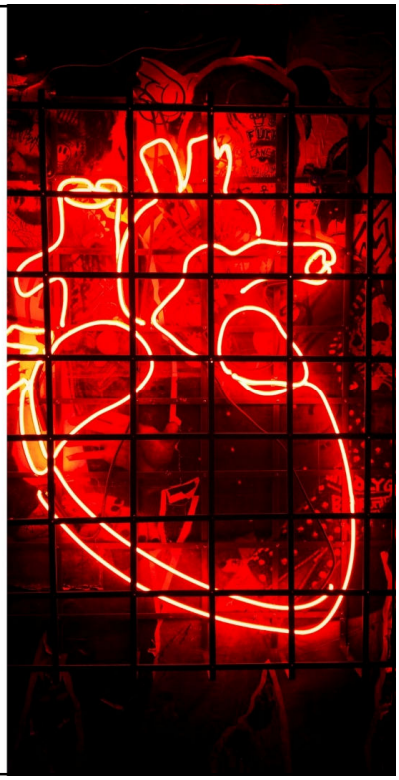


139

Inotropes and vasoactives

Pressors

- Have pressors ready before inotropes
- No trials for MAP goals in CS
- Systolic BP > 90, MAP 60-65



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(Shahriar Lahouti, 2021)

Figure 8. Pulmonary vascular properties of commonly used vasoactive agents in management of acute right heart failure

Drug	Target	SVR	PVR	PVR/SVR	HR Contractility	Dose
Inopressors						
☐ Norepinephrine	ααaβ	↑↑	↑	↔	↑	0.2–1.0 mcg/kg/min
☐ Epinephrine*	αβββ	↑↑	↓	↓	↑↑	LD: 5-8mcg/kg/min HD: >8mcg/kg/min
Pure Vasopressors						
☐ Vasopressin	V1, V2	↑↑↑	↓	↓↓	↓	0.01- 0.06 U/min (37)
Inodilators						
☐ Dobutamine**	αββββ	↓	↓	↓	↑↑↑	2-20 mcg/kg/min
☐ Milrinone**	cAMP	↓↓	↓↓	↓	↑↑↑	0.375-0.75 µg/kg/min

Sh.Lahouti @RECAPEM

*At low dose β-agonist effects predominates, with up-titration there's increasing α-agonist effects. Unlike dobutamine, milrinone; epinephrine at low dose poses a taste of α-agonist effects which will prevent hypotension.
**Despite favorable effect on PVR/SVR ratio, dobutamine and milrinine cause systemic hypotension if used alone, without a vasopressor, especially if left heart failure present.

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Vasoactive Medication Summary

(Shahriar Lahouti, 2021)

rug	Target	SVR	PVR	PVR/SVR	HR Contractility	Dose
Inopressors						
Norepinephrine	ααaβ	↑↑	↑	↔	↑	0.2–1.0 mcg/kg/min
Epinephrine*	αβββ	↑↑	↓	↓	↑↑	LD: 5-8mcg/kg/min HD: >8mcg/kg/min
Pure Vasopressors						
Vasopressin	V1, V2	↑↑↑	↓	↓↓	↓	0.01- 0.06 U/min (37)

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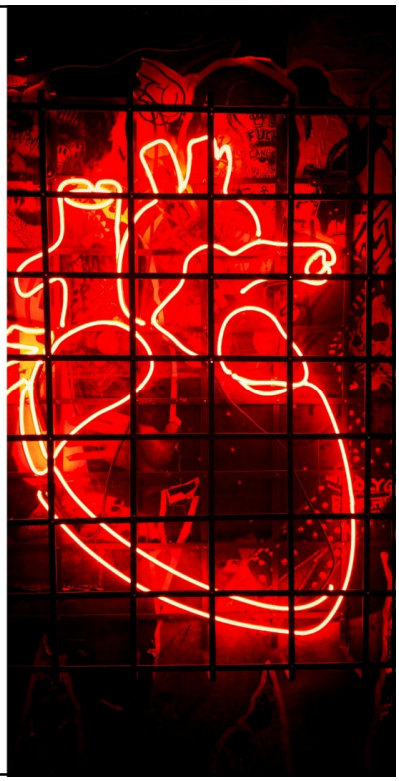
Inotropes and vasoactives

Pressors

- Uhlig 2020 Cochrane analysis: No convincing data supporting any specific inotropic or vasodilating therapy to reduce mortality in hemodynamically unstable patients with CS"



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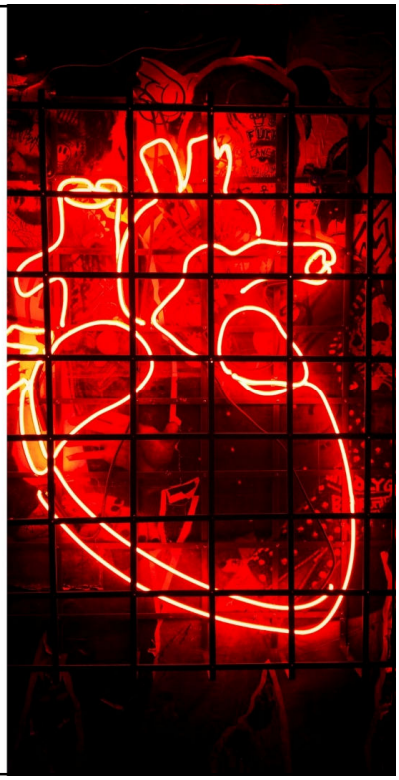
Inotropes and vasoactives

Pressors

- Uhlig 2020 Cochrane analysis: No convincing data supporting any specific inotropic or vasodilating therapy to reduce mortality in hemodynamically unstable patients with CS"

"Although there is no clear consensus regarding the choice of first-line vasoactive agent, the writing committee agrees that norepinephrine is a reasonable first choice for most patients with CS who are hypotensive."

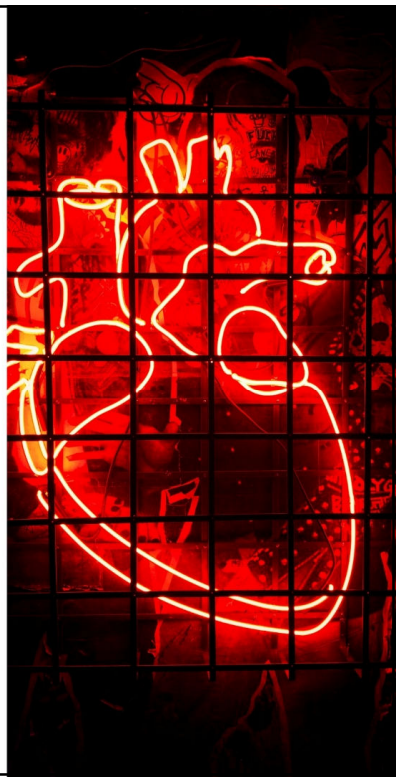


144

Inotropes and vasoactives

Caveat

- Increasing doses of norepinephrine to "fix the MAP" without inotropic support can be detrimental

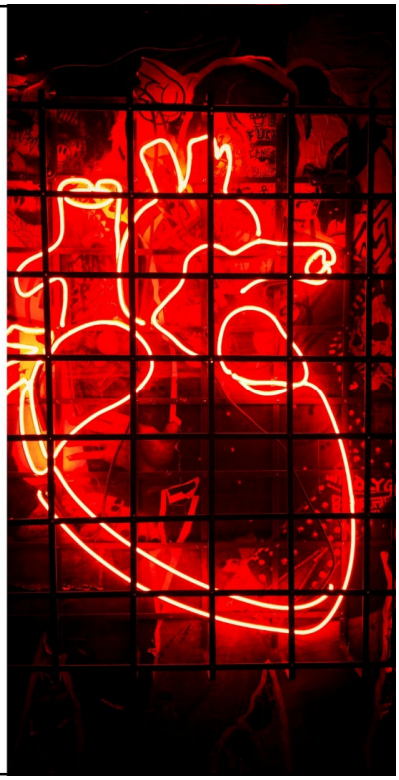


145

Inotropes and vasoactives

Caveat

- Increasing doses of norepinephrine to "fix the MAP" without inotropic support can be detrimental
  - Increased tachycardia
  - Increased afterload

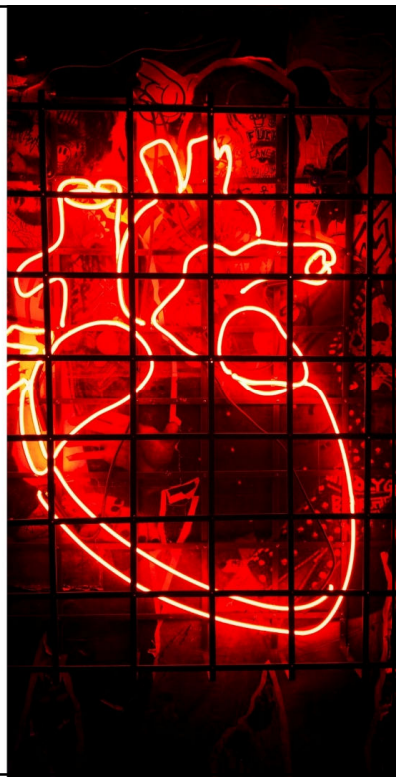


146

Inotropes and vasoactives

Inotropes - Which one?

- Dobutamine
  - Time of Onset: 1 to 2 minutes
  - Half life 2 minutes

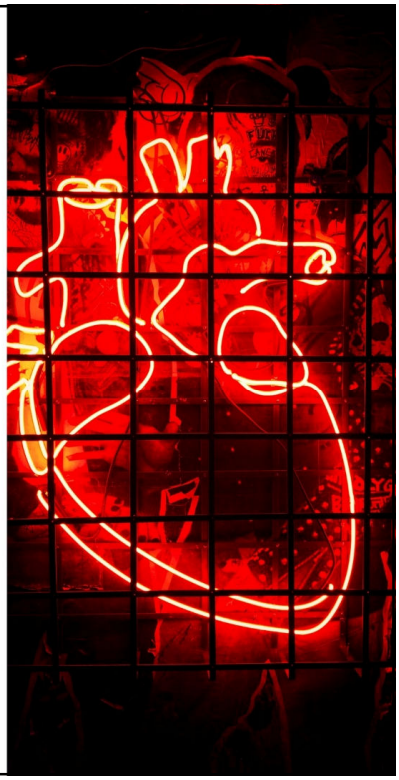


147

Inotropes and vasoactives

Inotropes - Which one?

- Dobutamine
  - Time of Onset: 1 to 2 minutes
  - Half life 2 minutes
  - May be less effective with beta blockers on board
  - Initiate at 2.5 mcg/kg/min to max 5

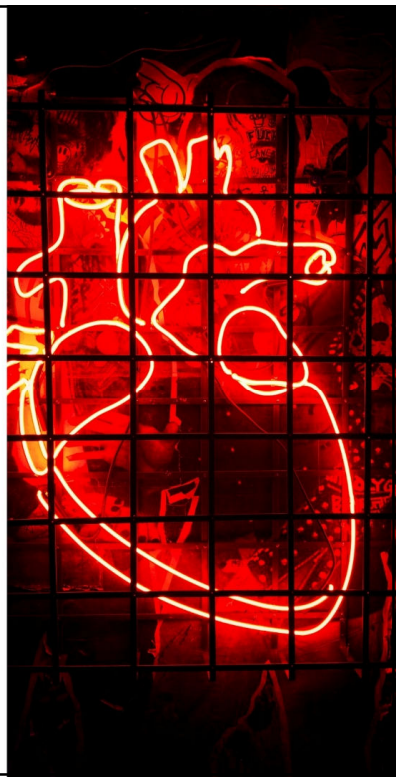


148

Inotropes and vasoactives

Inotropes - Which one?

- Milrinone
  - Time of Onset: 5 to 15 minutes
  - Half life 2 to 4 hours



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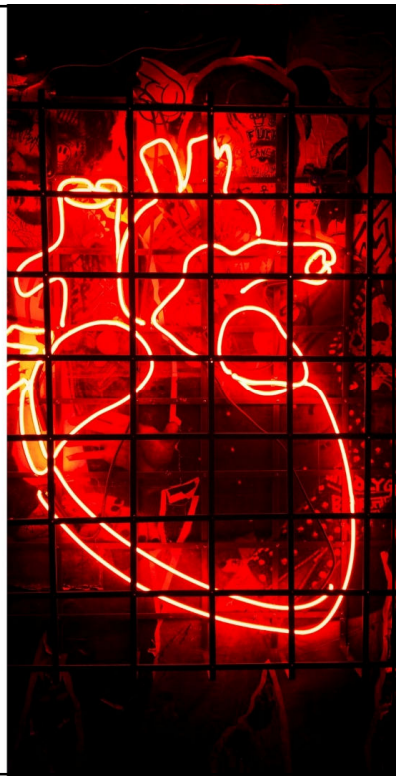
Inotropes and vasoactives

Inotropes - Which one?

- Milrinone
  - Time of Onset: 5 to 15 minutes
  - Half life 2 to 4 hours
  - Renal clearance, avoid in AKI
  - Initiate at 0.125 mcg/mg/min



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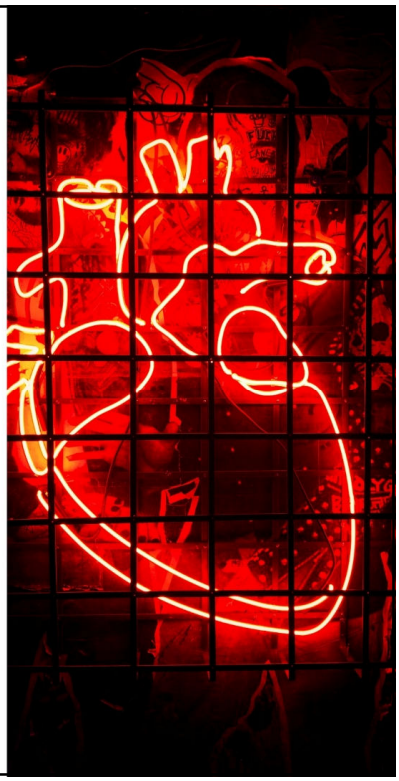
Inotropes and vasoactives

Inotropes - Which one?

- Dobutamine vs. Milrinone
  - DOREMI Trial
    - Single center double blinded RCT
    - n= 192 patients



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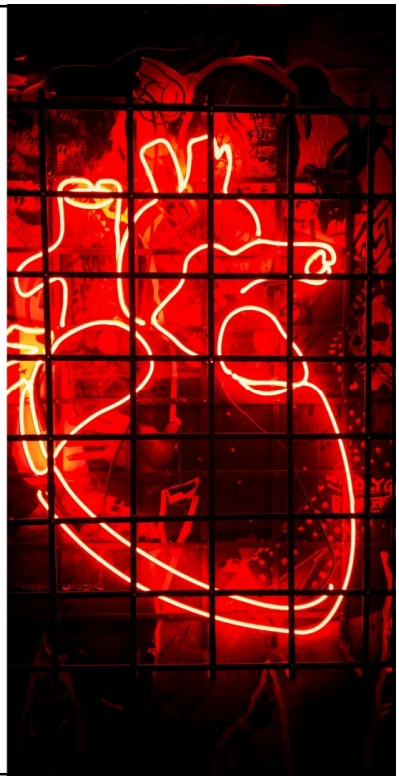
Inotropes and vasoactives

Inotropes - Which one?

- Dobutamine vs. Milrinone
  - No significant difference:
    - in-hospital death, resuscitated arrest
    - receipt transplant or MCS
    - non-fatal MI, CVA
    - renal replacement therapy



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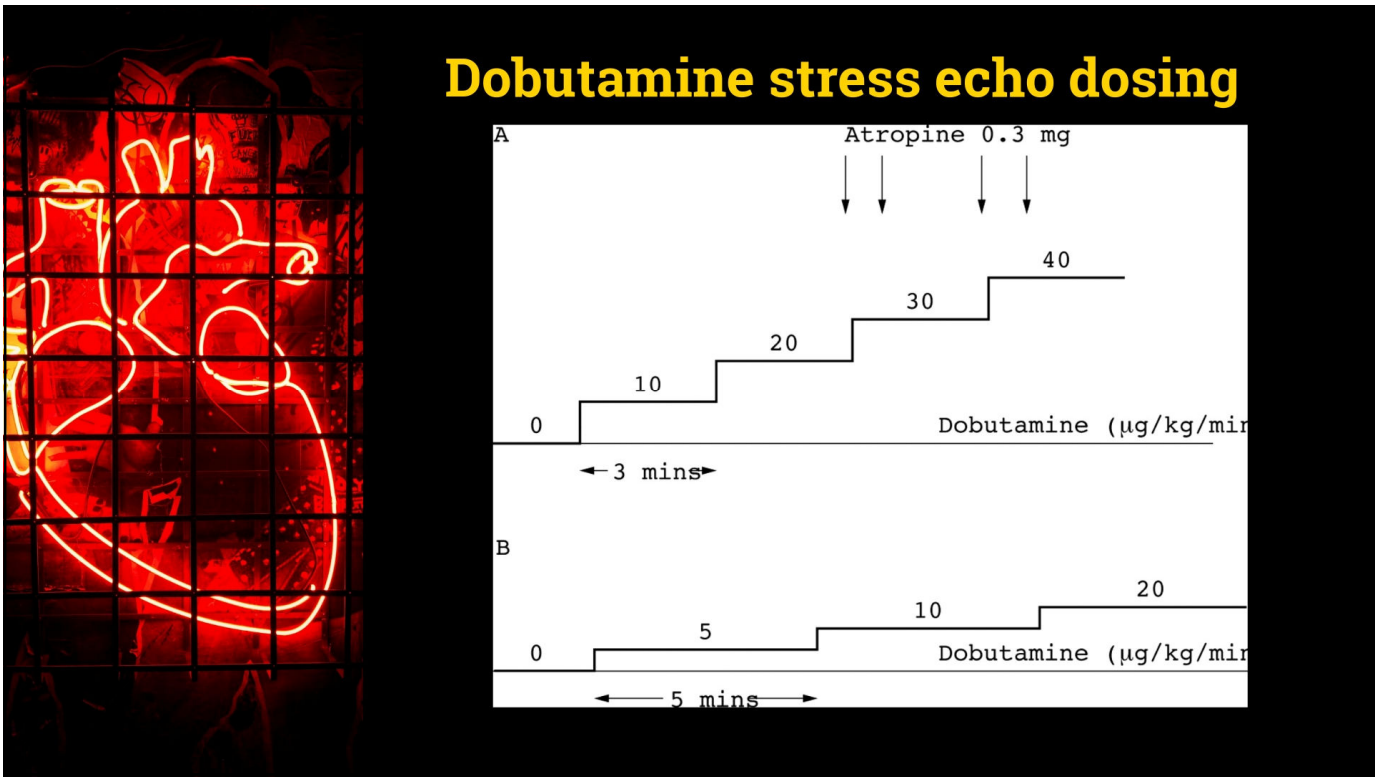

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ERRORS AND ASSUMPTIONS OF MANAGEMENT

Lesson: Do not be afraid of inotropes

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Agenda

Management

- ✔ A: Airway
- ✔ E: Etiology treatment
- ✔ I: Inotropy (and vasoactives)
- ✔ O: Offloading the heart
- ✔ U: Upgrade Support

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ERRORS AND ASSUMPTIONS OF MANAGEMENT

Fatal Error: Weak furosemide dosing (in HF-CS)

156

Offload the Heart

Furosemide dosing

- 20 mg IV is rarely enough
  - 1 - 2.5x PO mg dose in IV mg

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Offload the Heart

Furosemide dosing

- 20 mg IV is rarely enough
  - 1 - 2.5x PO mg dose in IV mg
  - If not effective, trial 30 minutes after inotropic support

158

Offload the Heart

Furosemide dosing

- 20 mg IV is rarely enough
  - 1 - 2.5x PO mg dose in IV mg
  - If not effective, trial 30 minutes after inotropic support
  - Check and replete Mg and K to avoid arrhythmia

159

Offload the Heart

Renal replacement therapy

- Early nephrology involvement
- Refractory metabolic acidosis (pH < 7.2)

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Offload the Heart

Renal replacement therapy

- Refractory hyperkalemia (>5.5)
- Inability to achieve negative fluid balance despite infusion, augmentation

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ERRORS AND ASSUMPTIONS OF MANAGEMENT

Lesson: Aggressive diuresis with volume overloaded patients

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Agenda

Management

- ✔ A: Airway
- ✔ E: Etiology treatment
- ✔ I: Inotropy (and vasoactives)
- ✔ O: Offloading the heart
- ✔ U: Upgrade Support

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ERRORS AND ASSUMPTIONS OF MANAGEMENT

Fatal Error: Not calling for transfer early / knowing to activate the cath lab

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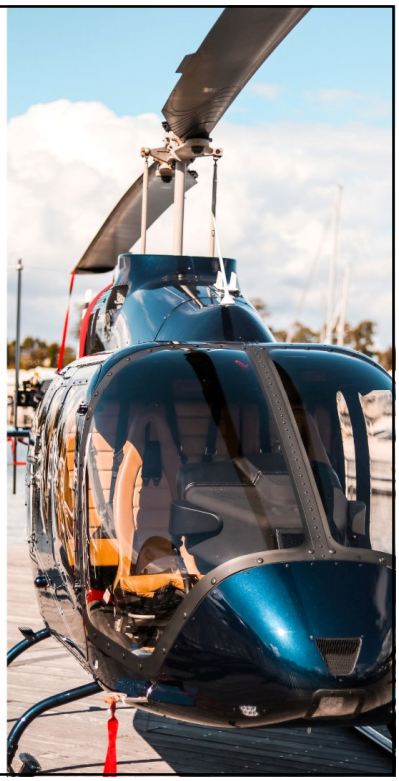
Upgrade Support

Mechanical Cardiac Support

- Indication
  - CS from AMI
  - CS refractory to inotropes
  - CS due to acute myocarditis



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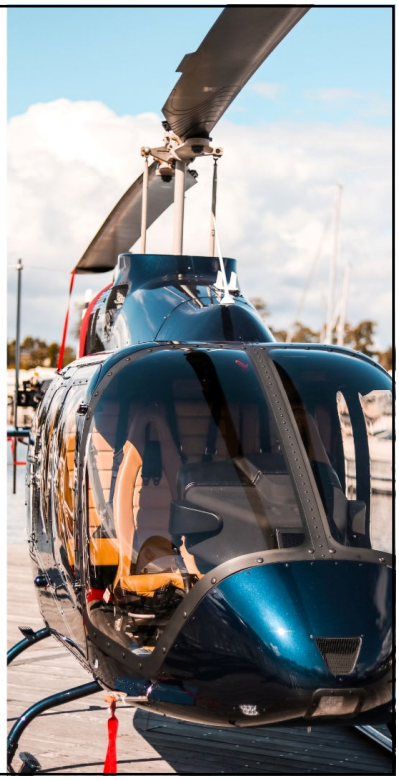
Upgrade Support

Mechanical Cardiac Support

- Indication
  - CS in cardiac transplant rejection
  - CS with biventricular failure
  - Arrhythmogenic storm



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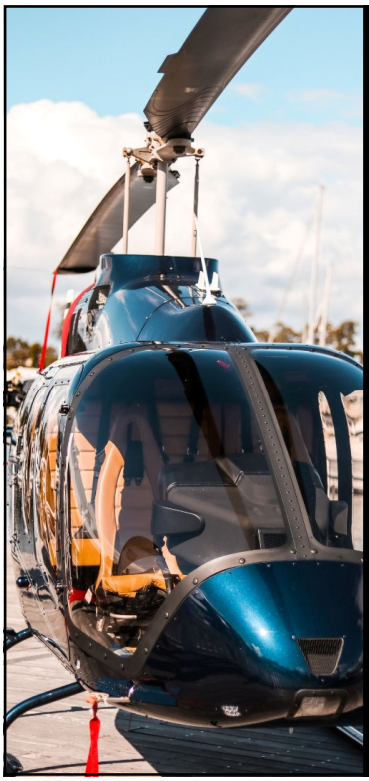
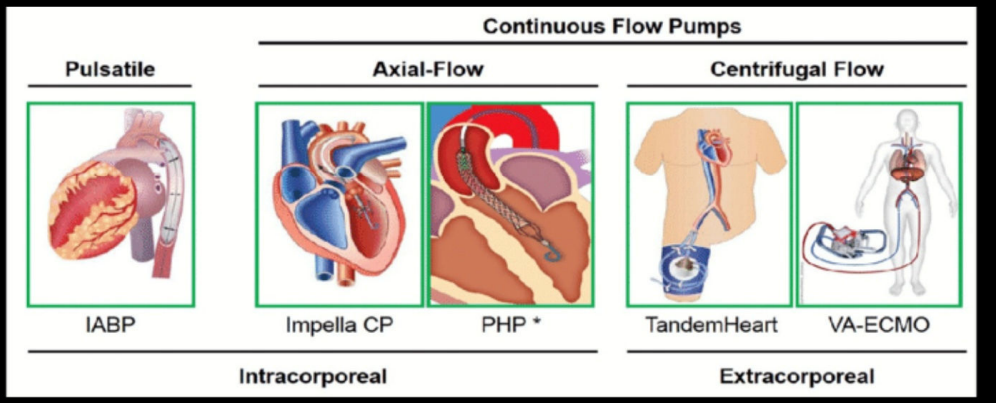


Examples of MCS



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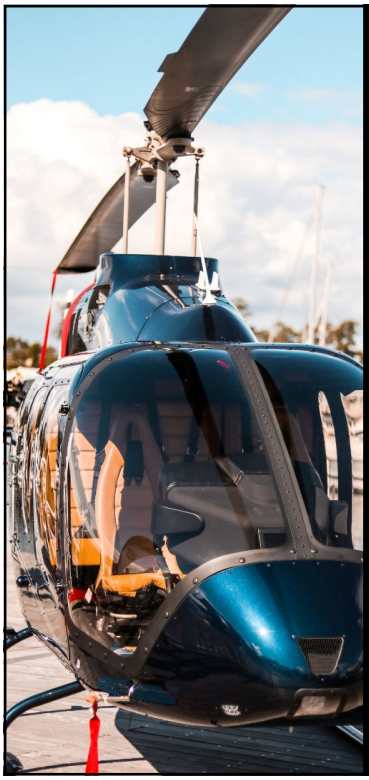
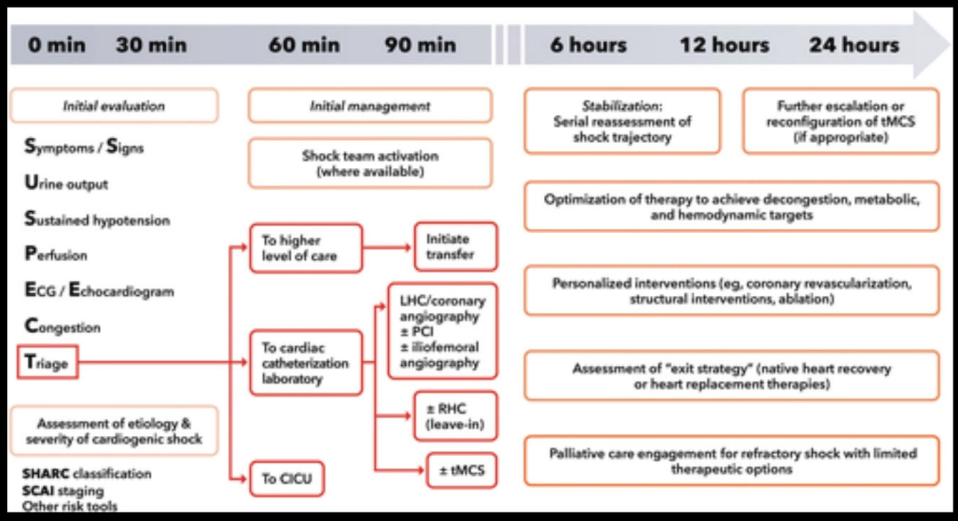


2025 ACC CS Guidelines



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Agenda

Management

- A: Airway
- E: Etiology treatment
- I: Inotropy (and vasoactives)
- O: Offloading the heart
- U: Upgrade Support

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ERRORS AND ASSUMPTIONS OF MANAGEMENT

Lesson: Ask for help early!

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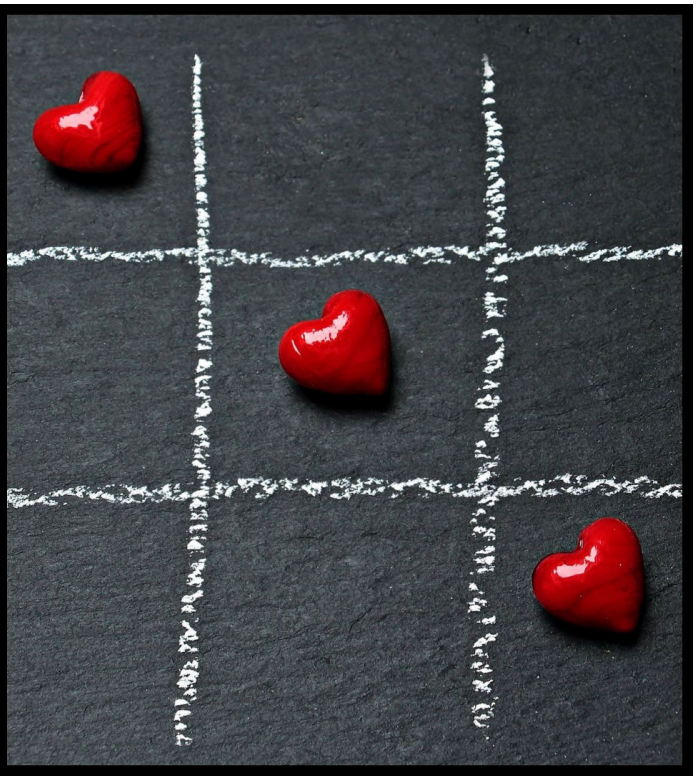
Conclusion



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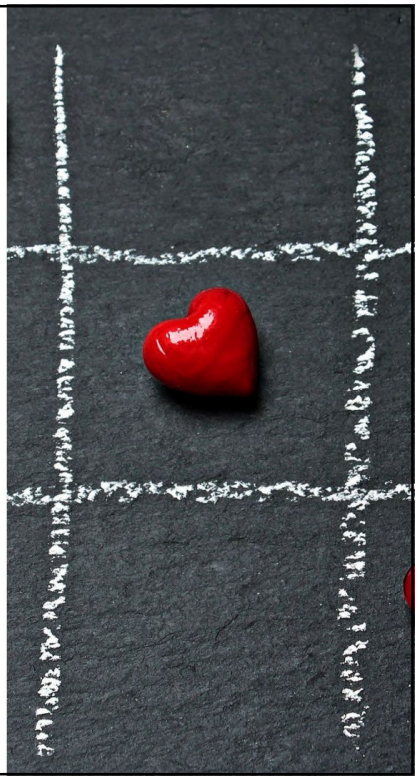
Conclusion - Salient Points

- **Have a high suspicion for CS despite physical exam and labs**
-
-
-



172

Conclusion - Salient Points

- Have a high suspicion for CS despite physical exam and labs
- Utilize POCUS to assist with determining etiology
-
-
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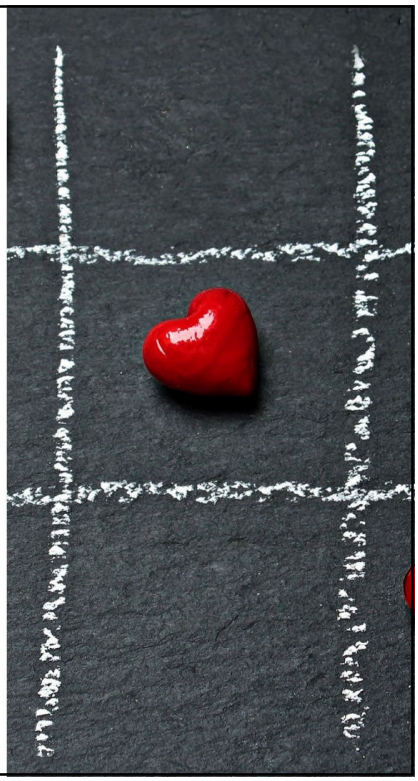

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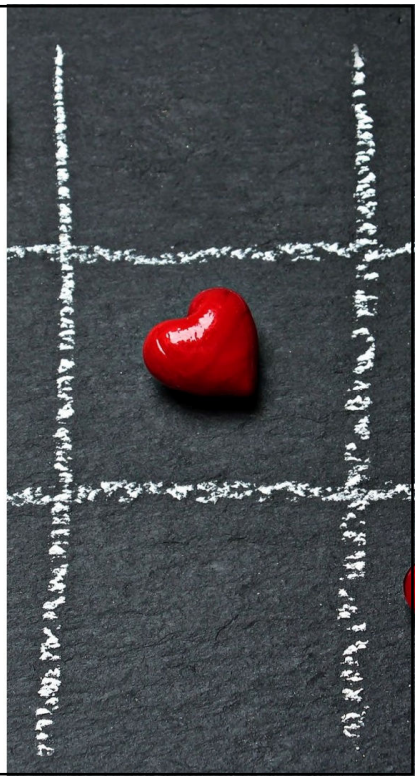
Conclusion - Salient Points

- Have a high suspicion for CS despite physical exam and labs
- Utilize POCUS to assist with determining etiology
- Respect RV failure
-
-



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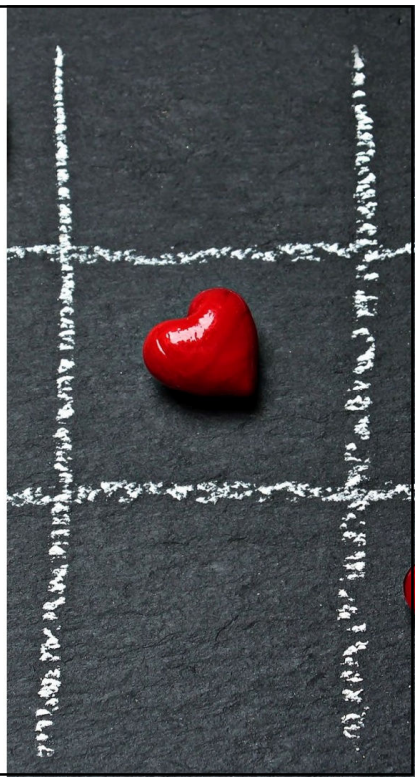
Conclusion - Salient Points

- Have a high suspicion for CS despite physical exam and labs
- Utilize POCUS to assist with determining etiology
- Respect RV failure
- Avoid IV beta blockers and calcium channel blockers
-



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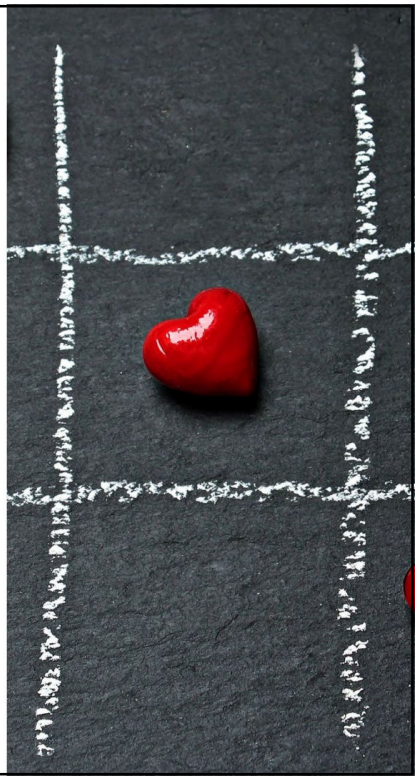
Conclusion - Salient Points

- Have a high suspicion for CS despite physical exam and labs
- Utilize POCUS to assist with determining etiology
- Respect RV failure
- Avoid IV beta blockers and calcium channel blockers
- Do not be afraid of inotropes, furosemide and calling for help



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Conclusion - Thank you!

Questions?

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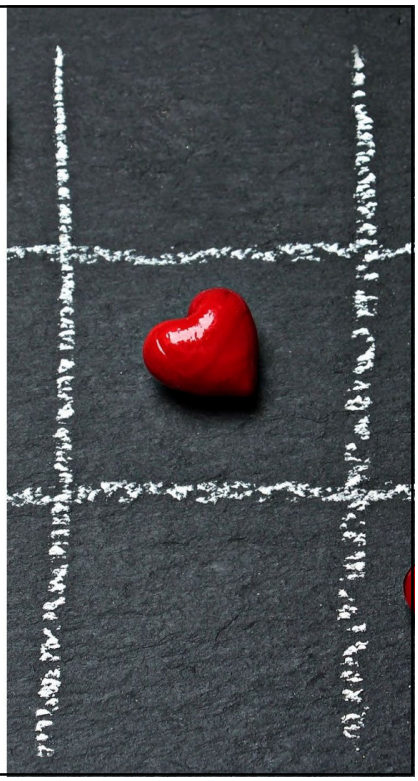

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