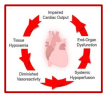

Cardiogenic Shock: Follow the Clues

Clinical Recognition in 2025

Updated Signs, Symptoms, and Diagnostic Phenotypes RV failure and beyond!

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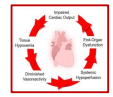
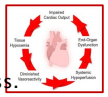
Disclosures



- ✓Barbara McLean is a paid speaker/consultant for BD
- ✓The opinions expressed here are her own and do not represent anyone except her experience and subject matter expertise

2

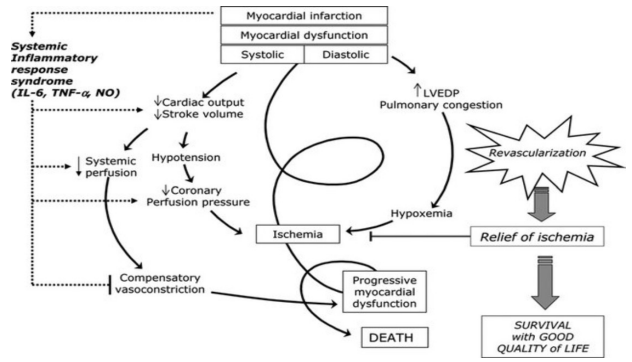
What is Shock?



- ✓Inadequate perfusion to meet tissue demands. A progressive process.
- ✓A systemic reduction in tissue perfusion → decreased tissue O2 delivery DaO2).
  - A shift to less–efficient anaerobic metabolism, leading to lactic and metabolic acidosis
  - Cardiogenic Shock
    - State of critical end–organ hypoperfusion due to primary cardiac dysfunction
    - Reduced cardiac output →inadequate tissue perfusion despite adequate volume (veno?)
    - Usually due to acute MI, severe heart failure, arrhythmia, or mechanical complications/obstruction
- ✓Eventually:
  - Cellular edema, leakage of cells' contents
  - Inadequate regulation of intracellular pH
  - Cell death, organ failure, cardiac arrest, and death
- ✓Initially, effects are reversible. RECOGNIZE and TREAT!

Referenced above:
Hemorrhagic Shock: Hooper; Armstrong. NCBI Bookshelf September 26, 2002

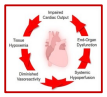
3



den Uil CA, Lagrand WK et al. Management of Cardiogenic Shock: Focus on Tissue Perfusion Curr Probl Cardiol 2009;34:330–349. 0146–2806

4

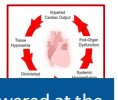
Stages of Shock
A progressive process: Intervene early



- ✓1st. Compensated Shock: Cardiac output (HR xSV) and systemic vascular resistance (peripheral vasoconstriction) work to keep BP within normal.
  - On exam: Tachycardia; decreased pulses & cool extremities in cold shock; flushing and bounding pulses in warm shock; oliguria; labs may show mild lactic acidosis
- ✓2nd Hypotensive (Uncompensated) Shock: Compensatory mechanisms are overwhelmed.
  - On exam: As above, plus hypotension, altered mental status, increased lactic acidosis and metabolic
  - Generally quick progression to cardiac arrest.
- ✓3rd Irreversible Shock: Irreversible organ damage, cardiac arrest

5

Classic Measurements Cardiac Shock



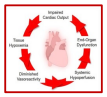
- ✓Monitoring measurements
  - CVP and PaD
    - Index of preload
  - Cardiac Output/Stroke Volume
    - Index of preload and contractility
  - Mixed Venous O2 saturation (SvO2), Base deficit., AG
    - Index of tissue perfusion
    - Metabolic acidosis
  - Systemic Vascular Resistance
    - Index of LV afterload
  - ↑SVR, HR ↓ SvO_2

Four questions must be answered at the bedside

1. Is there evidence of volume/venous load?
   1. Na+, Osmolarity, BNP, CVP, Weight
2. Is volume in the right compartment? Systolic pressure, stroke volume, EF, Weight, GFR, UO
3. Are the tissues perfused? $ScvO_2$, PvO_2, base deficit, Lactate
4. What is the compensation? HR, RR, blood gas, $PvCO_2$

6

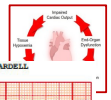
Our Patient

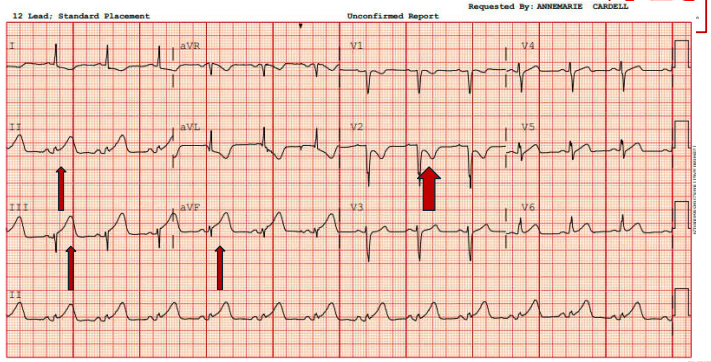


- ✓Admitted on 5/31/2025
  - 59 y/o male w/ PMHx of CAD with prior STEMI w/ LAD stent x 1, HFrEF (35–40%--> now 25–30%), HTN and DM2 who presented via EMS following episode of severe CP while driving.
  - Found to have inferior STEMI, taken emergently to cath lab with finding of embolic occlusions of distal OM1/2/3.
  - Now s/p balloon only angioplasty of distal OM3 with TIMI 1–2. Unable to intervene on OM1 or OM2 due to small calliber, will medically manage.
- ✓Admitted to CVICU for STEMI management.

7

5-31 Admit ECG: What Do you Think?





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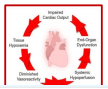
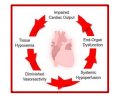
And the labs: What do you think?



What are the basic clues?

9

Cardiac Cath Report



6-1

CONCLUSIONS
Partially successful PTCA balloon only angioplasty of distal OM3 with TIMI 0 initially and TIMI 1 to 2 of distal small caliber OM3 due to embolization. There is also distal embolic occlusion of small caliber terminal ends of OM1 and OM2 best suited for tirofiban for 18 hours followed by anticoagulation plus plavix upon discharge
ï¿½
Right radial approach
Dominance left
Left main: Large caliber . No significant angiographic disease
Left anterior descending LAD: Large caliber. Patent stent
First Diagonal D1: medium caliber. No significant angiographic disease.
Left circumflex LCX:Large caliber. No significant angiographic disease.
First Obtuse Marginal OM1:Large caliber. Distal embolic occlusion small caliber for medical management
Second Obtuse Marginal OM2:Large to medium caliber. Distal embolic occlusion small caliber for medical management
Third Obtuse Marginal OM3:Medium caliber. Distal embolic occlusion small caliber partial success with balloon only angioplasty.
Right Coronary Artery RCA:small caliber Non dominant
LVEDP 42 mmHg

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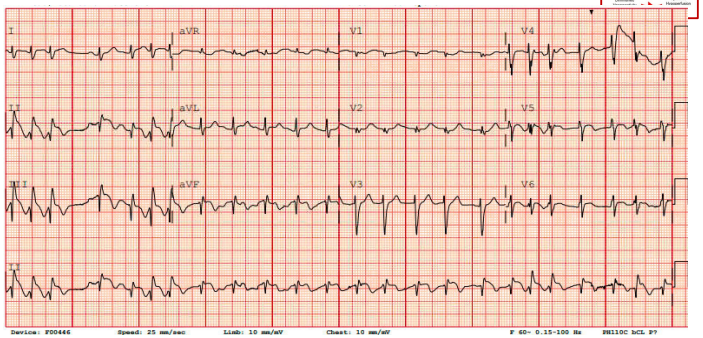
June 3 Emergent ECG: What do you think?



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Is this RV failure?

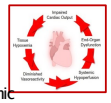
Hemodynamic Monitoring	GHS 7B 6/4/2025								
	2135	2145	2200	2215	2230	2245	2300	2315	2330
Arterial Line BP (mmHg)		130/57	135/90	114/49	118/50	112/47	124/46	119/46	121/45
Arterial Line MAP (mmHg)		74	103	66	68	64	64	64	64
CVP (mmHg)		24	25	16	15	14	11	12	12
Waveform adequate?									
Transducer Level to RA									
Transducer Zeroed									
Type of Hemodynamic monitoring									

Lasix 40 times one
6/3 Lasix 120 times one
6/3 Lasix 140 times one
6/4 Lasix 140 times one
6/5 Lasix 140 times one 6/6

FAIL!!!!

12

Clinical utility of CVP in RV failure



In **right ventricular (RV) failure**, **central venous pressure (CVP)** becomes a key hemodynamic marker, reflecting the **pressure in the right atrium** and indirectly the **RV's ability to handle venous return.**

Prominent **JVD, hepatojugular reflux, peripheral edema**
Worsening renal function (venous congestion) **Worsening Liver Function**

Clinical Significance	Interpretation
CVP > 12–15 mmHg	Suggests significant RV dysfunction or volume overload
High CVP with Low MAP/CO	Indicates RV cannot generate forward flow despite volume
CVP Trending Up	May suggest worsening RV performance, fluid overload, or increased afterload (e.g., PE, PHTN)
CVP + Echo Together	More informative—check RV size/function, IVC dynamics

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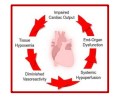
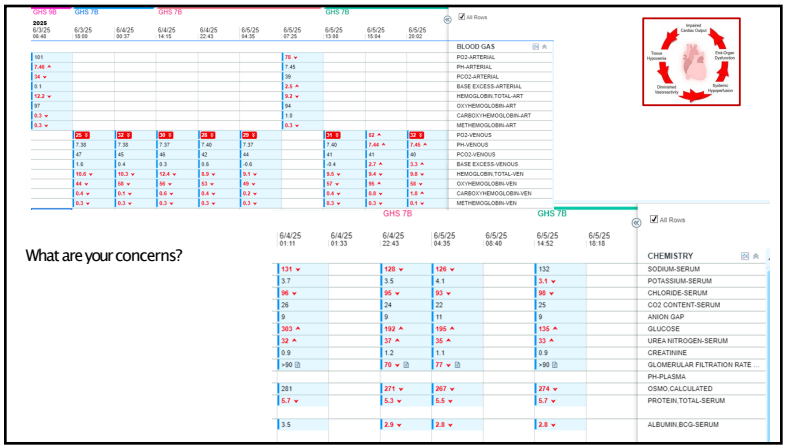
Bedside Clues: Is this CS? Is that all there is?



✓Bedside Clues: All may suggest RV is under stress

- Rising JVP/CVP
- Drop in BP (systole most reliable)
- More inotropic support
- Failure to respond to diuretics

14



15

	1937	1945	1952	2000
Cardiac				
Heart Block Type				
Ectopy and Ectopy Frequency	PVC; Frequent			PVC; Frequent
Heart Sounds				S1; S2
Remote Telemetry Patient				Telemetry M…
Telemetry Box Number				BSM
Cardiac Device				
Hemodynamic Monitoring				Yes
Hemodynamic Monitoring				
Arterial Line BP (mmHg)	108/51	93/50	95/57	100/55
Arterial Line MAP (mmHg)	68	65	68	69
CVP (mmHg)				2
Waveform adequate?	Yes			Yes
Transducer Level to RA	Yes			Yes
Transducer Zeroed	Yes			Yes
Type of Hemodynamic monitoring	PA Catheter/…			PA Catheter/…
Pulmonary Artery Catheter/Swan Ganz				
PA Insertion Site	Left Femoral			Left Femoral
PA Placement in CM	84			84
Transducer Level to RA	Yes			Yes
Transducer Zeroed	Yes			Yes
Waveform adequate?	No (Comment)			No (Comment)
PAP Systolic (mmHg)				
PAP Diastolic (mmHg)				
PAP Mean (mmHg)				
PCWP (mmHg)				
Continuous Cardiac Output (L/min) (Swan-Ganz)	5.1			5.2
SV (Stroke Volume) (mL/beat) (Swan-Ganz)	58			54
CI (Cardiac Index) (L/min/BSA) (Swan-Ganz)	2.9			3

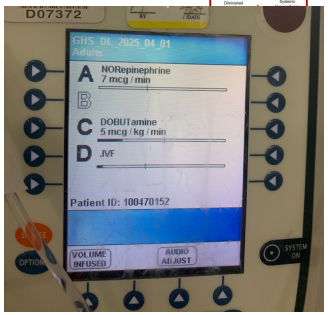
	54
	3
	31
SVR	-2969.23

What was the problem here?

16

Cath Report Pressures

- ✓RIGHT HEART ASSESSMENT
- ✓on Norepinephrine and Dobutamine infusion
- ✓Thermal CO: 2.76 Thermal CI: 1.58
- ✓Fick CO: 2.94 Fick CI: 1.68
- ✓PVR: 272 SVR: 1305
- ✓**LVEDP 30 mm Hg**
- ✓**PCWP 30 mm Hg**
- ✓**PA 63/30 (mean 40) mmHg**
- ✓**RV 60/20 mm Hg**
- ✓**RA 22 mm Hg**



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Classic vs. Modern Recognition of Cardiogenic Shock

- ✓Classic:
  - SBP <90 mmHg for >30 min,
  - cold/clammy skin
  - Oliguria
  - altered mentation.
- ✓Modern:
  - SCAI shock stage classification
  - lactate ≥2.0 mmol/L
  - dynamic echo markers.
- ✓New focus: early detection in pre–shock phase (SCAI B)!!!!

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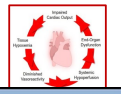
Peripheral Clues to Tissue Hypoxia



- ✓Cool extremities, mottled skin, capillary refill >3 sec
- ✓Low urine output (<0.5 mL/kg/hr), increasing creatinine (0.3 ml/kg/hr)
- ✓Altered mentation, drowsiness, anxiety or restlessness
- ✓Check SpO_2 waveform quality: low amplitude = low perfusion

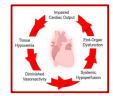
Add MORE!

- ✓PvO2 <30 mmHg
- ✓CVP> 12
- ✓PaD> 20

Peripheral Circulation	Volume Status: Wet	Volume Status: Dry
Cold	↓CI ↑SVRI ↑PCWP *Classical Cardiogenic shock*	↓CI ↑SVRI ↔PCWP *Euvolemic Cardiogenic shock*
Warm	↓CI ↓/↔SVRI ↑PCWP *Vasodilatory Cardiogenic shock or Mixed shock*	↑CI ↓SVRI ↓PCWP *Vasodilatory shock Not cardiogenic shock*

19

How to recognize early and Then what…..?



- ✓Early warning signs before full collapse:
  - Narrowing pulse pressure
  - Rising lactate (even before hypotension)
  - Increased venous congestion (CVP, JVD, hepatic waveform)
- ✓Hemodynamic Signatures and Right-Sided Findings
  - Recognizing biventricular or right-sided shock variants:
    - Elevated central venous pressure with clear lungs (e.g., RV infarct, PE)
    - Signs: prominent JVD, pulsatile hepatomegaly, hypotension with low pulmonary congestion
  - Low cardiac index (<2.2 L/min/m^2), elevated PCWP
  - Narrow pulse pressure, early fatigue

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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

Sinha SS, Morrow DA, Kapur NK, et al. 2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock. *JACC*. Published online on March 17, 2025. doi: 10.1016/j.jacc.2025.02.018

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Key Testing Clues

- ✓Non–invasive surrogates increasingly used: IVC dynamics, VExUS score
- ✓Lactate ≥2.0 mmol/L: key marker of tissue hypoperfusion.
- ✓Lactate clearance: dynamic target of therapy.
- ✓Troponin +BNP/NT–proBNP: myocardial stress and volume status.
  - Emerging: sST2, Galectin–3 (fibrosis and myocardial stress)
- ✓Early differentiation critical for mechanical support decisions (e.g., Impella vs RVAD)
- ✓Pv–a CO2 difference
- ✓ Advanced Bedside Echo:
  - Focused Cardiac Ultrasound (FoCUS) is now central:
    - LVOT VTI (stroke volume estimate)
    - RV/LV size and function comparison
    - Pericardial tamponade, valvular disruption

2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock: A Report of the American College of Cardiology Solution Set Oversight Committee**Publication**: JACCVolume 85, Number 16
Baran, D. A., Grines, C. L., Bailey, S., et al. (2019). **SCAI clinical expert consensus statement on the classification of cardiogenic shock**. *Catheterization and Cardiovascular Interventions*, 94(1), 29-37. https://doi.org/10.1002/ccd.28329
van Diepen, S., Katz, J. N., Albert, N. M., et al. (2017). **Contemporary management of cardiogenic shock: A scientific statement from the American Heart Association**. *Circulation*, 136(16), e232-e268. https://doi.org/10.1161/CIR.0000000000000525
Jentzer, J. C., van Diepen, S., Murphree, D. H., et al. (2019). **Early shock phenotypes and short-term outcomes in cardiogenic shock**. *Journal of the American College of Cardiology*, 73(4), 397-408. https://doi.org/10.1016/j.jacc.2018.10.085

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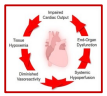
BNP or NT pro BNP/troponin



- ✓Cardiac strain (stress on the heart), two key biomarkers, B-type natriuretic peptide (BNP) or its precursor N-terminal pro-B-type natriuretic peptide (NT-proBNP)
  - BNP and NT-proBNP ↑ with increased pressure or volume overload within the heart chambers, indicating the heart is working harder to pump blood
  - BNP has a shorter half-life compared to NT-proBNP
  - BNP is cleared by receptor-mediated mechanisms and enzymatic degradation,
  - NT-proBNP is primarily cleared by renal excretion
- ✓The combination of high BNP/NT-proBNP (reflecting strain) and elevated troponin (reflecting injury) signal a severe heart failure exacerbation

2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock: A Report of the American College of Cardiology Solution Set Oversight Committee**Publication**. JACCVolume 85, Number 16
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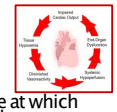
Measuring lactate clearance: a clinical approach



- ✓In clinical settings, measuring lactate clearance involves tracking the rate at which lactate levels decrease over time, which provides valuable insights into the effectiveness of resuscitation and tissue perfusion
  - 1. Obtain serial lactate measurements
  - Collect blood samples at regular intervals, typically every 2-6 hours, to establish a trend of lactate levels.
  - Arterial blood samples are considered the gold standard for lactate measurement, while venous samples are an alternative, albeit potentially less accurate.
  - Point-of-care lactate testing devices offer rapid and convenient measurements at the bedside.
- ✓2. Calculate the lactate clearance rate
  - Use the formula: Lactate clearance =[(initial lactate −subsequent lactate) / initial lactate] x 100.
  - A positive value indicates a decrease in lactate, while a negative value signifies an increase

Efgan MG, Bora ES, et al. Prognostic Significance of Lactate Clearance in Cardiogenic Pulmonary Edema in the Emergency Department. *Medicina (Kaunas)*. 2024;60(9):1502. Published 2024 Sep 14. doi:10.3390/medicina60091502

Thevathasan T, Gregers E, et al. Lactate and lactate clearance as predictors of one-year survival in extracorporeal cardiopulmonary resuscitation – An international, multicentre cohort study, Resuscitation, Volume 198, 2024, 110149,ISSN 0300-9572,

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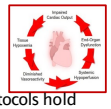
Measuring lactate clearance: a clinical approach



✓3. Interpret the lactate clearance rate
- Continuous lactate monitoring devices and lactate–guided resuscitation protocols hold promise for improving outcomes in critically ill patients.
- Adequate clearance: Generally, a lactate clearance rate of >20% over 6 hours indicates a positive response to treatment and restoration of tissue perfusion.
- Poor clearance: A lactate clearance rate of <10% over 6 hours is associated with poor outcomes and may necessitate adjustments in treatment strategies.
- Intermediate clearance: A rate between 10–20% may warrant further evaluation and careful monitoring.

✓4. Integrate lactate clearance with clinical data
- Interpret lactate clearance in conjunction with other clinical indicators such as vital signs, hemodynamic parameters, and organ function assessments and base deficit

✓Important considerations
- Lactate clearance should not be used as the sole determinant of patient management. Consider the patient's overall clinical picture and differentiate between causes of elevated lactate.
- In patients with liver disease, lactate clearance may be less reliable as a prognostic marker.

REF: Seong et al. Prognostic value of lactate levels and lactate clearance in sepsis and septic shock with initial Hyperlactatemia. Lee et al. Medicine(2021) 100:7

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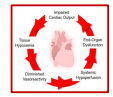
1 Clinical Case



✓68–year–old male presents to the emergency department with acute onset of severe chest pain, shortness of breath, and altered mental status

✓Blood pressure is 80/40 mmhg, heart rate is 120 bpm, exhibits signs of cold, mottled extremities.

✓Initial arterial analysis reveals a lactate level of 7.2 mmol/L.

✓Based on these findings and other clinical signs diagnosed as cardiogenic shock secondary to acute myocardial infarction

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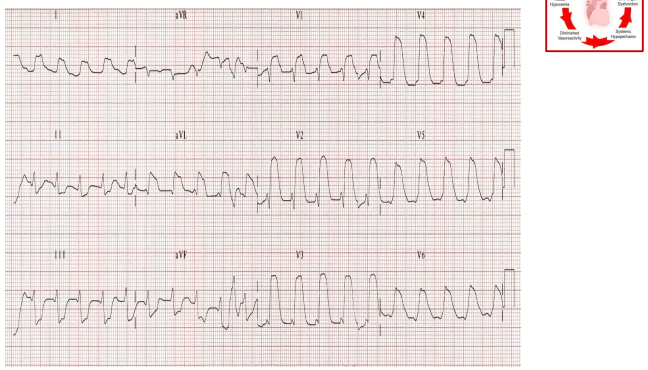

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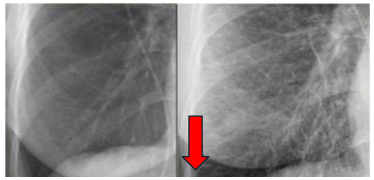
More advanced congestion: Kerley B lines

- ✓ **Stage II - Interstitial edema**
- ✓ Stage II of CHF is characterised by fluid leakage into the interlobular and peribronchial interstitium
- ✓ When fluid **leaks into the peripheral interlobular septa** it is seen as Kerley B or septal lines
- ✓ Kerley-B lines are seen as peripheral short 1-2 cm horizontal lines near the costophrenic angles.
- ✓ These lines run perpendicular to the pleura



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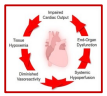
1 Clinical case

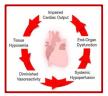


- ✓Immediate Interventions:
- ✓Intubated with mechanical ventilation
- ✓initiated on norepinephrine infusion to support blood pressure
- ✓An intra-aortic balloon pump (IABP) is inserted to augment coronary blood flow.
- ✓Lactate Monitoring: Arterial lactate levels are serially monitored at 0, 6, 12, and 24 hours after the initiation of therapy.
  - 0 hours: Lactate 7.2 mmol/L
  - 6 hours: Lactate 6.1 mmol/L – *6-hour lactate clearance calculated at approx. 15%*
  - 12 hours: Lactate 4.8 mmol/L – *12-hour lactate clearance calculated at approx. 33%*
  - 24 hours: Lactate 1.8 mmol/L – *24-hour lactate clearance calculated at approx. 75%*

Headed in right direction? Almost there!

30

2 Clinical Case



- ✓A 72-year-old female with a history of hypertension, type 2 diabetes mellitus, and chronic kidney disease (CKD) presents to the emergency department with acute onset of severe dyspnea, fatigue, and chest discomfort. Her vital signs are: BP 85/50 mmHg, HR 110 bpm, RR 28 breaths/min, and SpO2 88% on room air. Physical examination reveals cool and mottled extremities, elevated jugular venous pressure, and bilateral crackles on lung auscultation. An initial electrocardiogram (ECG) shows ST-segment elevations anterior-lateral wall
- ✓Clinical Diagnosis: Based on the clinical presentation and ECG findings, cardiogenic shock (AMI) is suspected in the patient.

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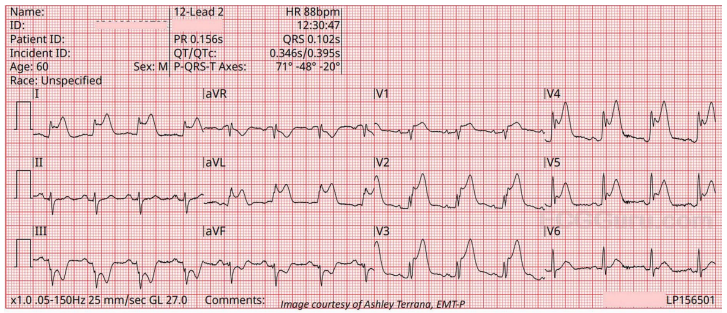
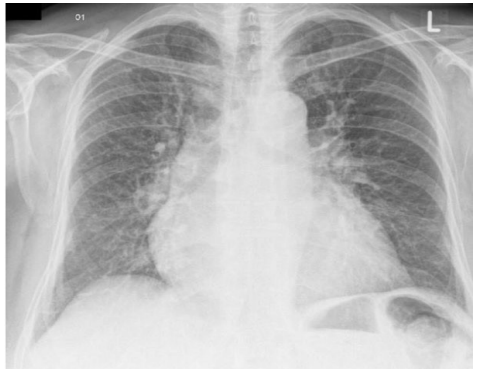

Name:
ID:
Patient ID:
Incident ID:
Age: 60
Sex: M
Race: Unspecified
12-Lead 2
HR 88bpm
12:30:47
PR 0.156s
QRS 0.102s
QT/QTc:
0.346s/0.395s
P-QRS-T Axes:
71° -48° -20°
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
x1.0 .05-150Hz 25 mm/sec GL 27.0
Comments:
Image courtesy of Ashley Terrana, EMT-P
LP156501

32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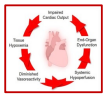
33

2 Clinical Case



- ✓Initial assessment and biomarker evaluation
  - Biomarker Results: Initial blood tests reveal:
    - Cardiac Troponin I (cTnI): Significantly elevated at 10.5 ng/mL(hs Tnt 10,500) (upper limit of normal <0.04 ng/mL(hsTnT 20)), which confirms myocardial injury.
    - NT-proBNP: Significantly elevated at 8,500 pg/mL (normal <125 pg/mL for age <75), indicating significant myocardial stress and left ventricular dysfunction.
  - Lactate: 5.8 mmol/L, indicating tissue hypoperfusion.
- ✓Renal Function: Creatinine 2.1 mg/dL, reflecting pre-existing CKD and potentially exacerbated by the shock state
- ✓GFR 15 ml

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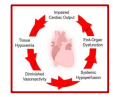
2 Clinical Case



- ✓Initial Resuscitation: The patient receives intravenous fluids, oxygen supplementation, and inotropic support with dobutamine.
- ✓Revascularization: Given the ECG changes and high troponin levels, the patient undergoes emergent coronary angiography, which reveals a complete occlusion of the left main coronary artery. A percutaneous coronary intervention (PCI) is performed to restore blood flow.
- ✓Lactate Clearance Monitoring: Serial lactate measurements are initiated.
  - 6 hours post-PCI: Lactate 4.2 mmol/L (28% clearance).
  - 12 hours post-PCI: Lactate 2.5 mmol/L (57% clearance).
- ✓Lactate Clearance: The improving lactate clearance demonstrated a positive response to the resuscitation and revascularization efforts, suggesting improved tissue perfusion and a better prognosis

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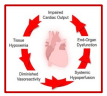
2 Clinical Case



- ✓Biomarker Monitoring: Troponin and NT-proBNP levels are also serially monitored.
  - Troponin: While remaining elevated for several days, the cTnI levels begin to trend downward after revascularization, signifying successful reperfusion and reduction in ongoing myocardial damage.
  - NT-proBNP: NT-proBNP levels gradually decrease in parallel with clinical improvement and resolution of the shock state.
- ✓Interpretation and outcome
  - Troponin: The initial massive troponin elevation confirms the diagnosis of AMI as the cause of CS. The subsequent downward trend after successful revascularization provided evidence of reduced ongoing myocardial injury.
  - NT-proBNP: The high NT-proBNP indicated significant cardiac dysfunction and hemodynamic stress. The subsequent decline mirrored the improvement in heart function and volume status following treatment.
- ✓Overall Outcome: The patient's condition gradually stabilizes in the intensive care unit. She is eventually weaned off inotropic support and mechanical ventilation. Following an extended hospital stay, she is discharged with optimized medical therapy for heart failure and secondary prevention of cardiovascular events

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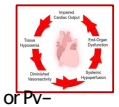
Measuring Pv-aCO2



- ✓Why measure Pv–aCO2? veno–arterial carbon dioxide difference (Pv–aCO2), or Pv–aCO2 gap
  - valuable parameter in critical care circulatory and cardiogenic
  - offers additional insights into tissue perfusion beyond traditional markers like lactate and central venous oxygen saturation (ScvO2)
- ✓1. Indicator of tissue perfusion and adequacy of cardiac output
  - The Pv–aCO2 gap reflects the balance between carbon dioxide production in the tissues and its removal by blood flow
  - An elevated Pv–aCO2 gap (generally >6 mmHg) can indicate inadequate tissue perfusion, or "stagnant dysoxia," due to insufficient cardiac output, : inefficient blood flow to remove CO2
  - inverse relationship between cardiac output and the Pv–aCO2 gap demonstrated in experimental and clinical settings.
- ✓2. Complementary to other markers
  - Pv–aCO2 can be particularly useful when other global oxygenation parameters, such as ScvO2, appear normal but tissue hypoperfusion persists (BD > -4)
  - provides more sensitive and rapid indicator of tissue perfusion changes compared to lactate
    - a slower clearance kinetics dependent on liver function.
  - elevated Pv–aCO2 indicates microcirculatory dysfunction and tissue hypoperfusion

Hørsdal OK. Can utilization of the venous-to-arterial carbon dioxide difference improve patient outcomes in cardiogenic shock? A narrative review. *Am Heart J Plus*. 2025;50:100504. Published 2025 Jan 30. doi:10.1016/j.ahjo.2025.100504

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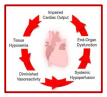
Measuring Pv-a CO2



- ✓3. Guiding resuscitation efforts
  - An elevated Pv–aCO2
    - optimize hemodynamic management by increasing cardiac output or improving microcirculatory blood flow
  - Monitoring the trend of the Pv–aCO2 gap
    - guide therapeutic interventions and predict patient prognosis
- ✓4. Assessment of microcirculatory dysfunction
  - macrocirculatory parameters may be normal
    - microcirculatory dysfunction and tissue hypoperfusion best predictors of adequate cardiac output

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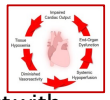
3 Clinical Case



- ✓A 68-year–old male presents to the emergency department with acute onset of severe dyspnea, hypotension (BP 80/50 mmHg), and signs of peripheral hypoperfusion, including cool and mottled extremities
- ✓initial arterial blood gas
  - significant metabolic acidosis BD -9, pH 7.21 with a lactate level of 6.5 mmol/L
- ✓ ECG shows widespread ST-segment elevations, confirming acute myocardial infarction (AMI) as the likely cause of the cardiogenic shock

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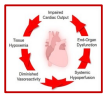
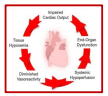
3 Clinical Case



- ✓Clinical Diagnosis: Cardiogenic shock secondary to AMI is diagnosed, and the patient is promptly resuscitated with fluids and initiated on vasopressors to maintain a mean arterial pressure (MAP) above 65 mmHg.
- ✓Need for Further Assessment: Despite achieving the MAP target, the patient's lactate levels remain elevated at 5.0 mmol/L after 3 hours, suggesting ongoing tissue hypoperfusion.
- ✓Pv–aCO2 Measurement: To gain a more comprehensive understanding of tissue perfusion, a central venous catheter is inserted, and simultaneous arterial and central venous blood gas samples are obtained.
  - Arterial PCO2 (PaCO2): 40 mmHg
  - Central Venous PCO2 (PcvCO2): 59 mmHg
  - Calculated Pv–aCO2: 59 mmHg – 40 mmHg = 19 mmHg
  - GAP > 10 indicates shock

40

3 Clinical Case



- ✓Interpretation and intervention
  - Elevated Pv–aCO2: The Pv–aCO2 value of 19 mmHg is elevated, exceeding the normal range (typically 2–10 mmHg)
  - Suggests that despite maintaining a target MAP and potentially adequate global oxygen delivery
    - mismatch between oxygen demand and supply at the tissue level
    - Impaired metabolism and anerobic production
- ✓Adjusting Therapy: Based on the elevated Pv–aCO2, the medical team considers interventions aimed at improving cardiac output and tissue perfusion.
- ✓Optimizing inotropic support to enhance myocardial contractility.
- ✓Considering mechanical circulatory support (e.g., intra–aortic balloon pump, impella or extracorporeal membrane oxygenation) to augment CO if medical therapy proves insufficient.
- ✓Further assessment of microcirculation→identify potential areas of impaired flow

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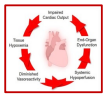
Clinical Red Flags



Traditional Signs

Hypotension (SBP <90)

Cool/mottled skin

Dyspnea, crackles

Tachycardia

Oliguria

Recent Additions / Emphasis
Rising lactate with normal BP ("pre-shock")

Lactate clearance

SCAI B phenotype: narrow pulse pressure, fatigue

Elevated CVP +clear lungs → **RV shock**

Blunted heart rate variability (monitoring insight)

Rising creatinine **with preserved volume**

proBNP/BNP/Troponin

Pv-aC02

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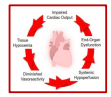
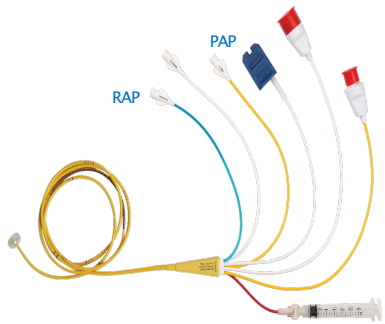
But really you need a PAC





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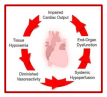
Components of the PA waveform



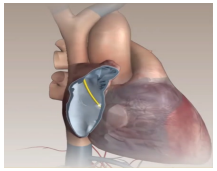
Systole
- Measured at the peak of the wave
- Should be almost straight up

Diastole
- Measured just prior to the upstroke of systole (end of QRS)
- Higher than RV diastolic pressure

Dicrotic notch
- Indicates pulmonic valve closure
- Aids in differentiation from RV waveform

✓**RAP/CVP: right atrial pressure**
- Estimated RV preload (right ventricular end diastolic volume)
- RAP/CVP measures the pressure related to the compliance of RV when **filling** with volume

✓**PCWP: wedge pressure**
- Estimated LV preload (Left ventricular end diastolic volume)
- PAWP measures the pressure related to the RETROGRADE column of blood which fills the LV from the LA and PV



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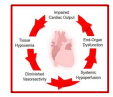
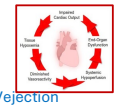
The right pressures



	Systolic	Diastolic	Filling pressures
Right ventricle	15-25	0-8	CVP 2-6
Pulmonary artery	15-25	8-15	PAD 8-15
Left ventricle	90-140	5-15	PAOP 6-10
Aorta	100-150	60-90	
Pulmonary vascular resistance			100-250
Systemic vascular resistance			800-1200
PA pulse pressure			8-15
Systemic pulse pressure			30-40

45

Did you also know….?



- RV filling is different than LV filling

RAP/CVP

- Significantly lower than comparable left-sided
- RV isovolumetric contraction time is short
- Filling of RV
  - RV filling normally starts before and finishes after LV filling
  - RV isovolumic relaxation time is shorter

Compare filling pressures
RA/CVP should ALWAYS be <PAOP /PAD

- RV ejection is different that LV ejection

PAS

- RV systolic pressure rapidly exceeds the low pulmonary resistance
- Two characteristics of RV
  - Distensibility of its free wall
  - Compliance and the increase of volume without significant changes in the wall surface area
  - The right ventricle pumps into a high-compliance, low-resistance pulmonary circuit

Compare EF
RV 40-60%
LV 50-75%

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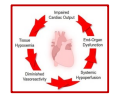
NOTHING can replace the PAC !! (many have tried)
It is a Right Heart Catheter



✓Important information required to assess adequacy of right ventricular function

- Pulmonary pressures informing RV function and pulmonary resistance
- Predicting the load on the LV (PCWP and PAD)
- Continuous cardiac output measurements
- Allows for right heart "volumetric"
  - RVEDV, RVEF, and RVSV
  - RVSW and RVSWI
- Continuous SvO2 measurements
  - Global oxygen utilization

Type of information	Clinical use	Alternatives [42]
Pulmonary artery pressures	Acute and chronic pulmonary hypertensionEssential to assess right ventricular function	Echocardiography (usually intermittent)
Pulmonary artery balloon-occluded pressure (PAOP)	Risk of lung edema (heart failure and/or hypervolemia)Guide for fluid challenge (risk assessment)	Echocardiography (intermittent and imprecise)
Cardiac output	Optimization (in conjunction with Hb and SaO_2	Multiple – all less reliable than PAC
Mixed venous oxygen saturation(SvO_2)	Assessment of the balance between oxygen delivery and oxygen consumption	Central venous oxygen saturation **($ScvO_2$) - poor** substitute

Arias-Ortiz J & Vincent J. The pulmonary artery catheter. *Current Opinion in Critical Care*, 2023,29 (3), 231-235. doi:10.1097/MCC.0000000000001040.

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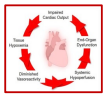
The waveforms



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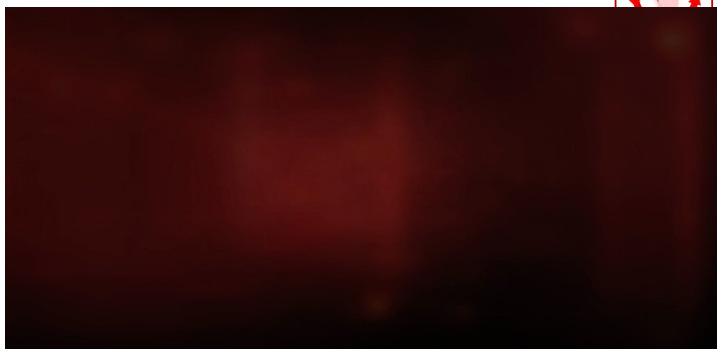
Remember the Float



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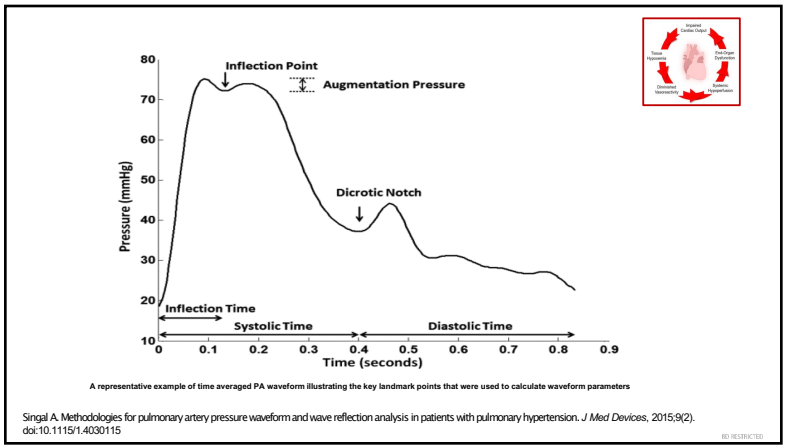

A representative example of time averaged PA waveform illustrating the key landmark points that were used to calculate waveform parameters

Singal A. Methodologies for pulmonary artery pressure waveform and wave reflection analysis in patients with pulmonary hypertension. *J Med Devices*, 2015;9(2). doi:10.1115/1.4030115

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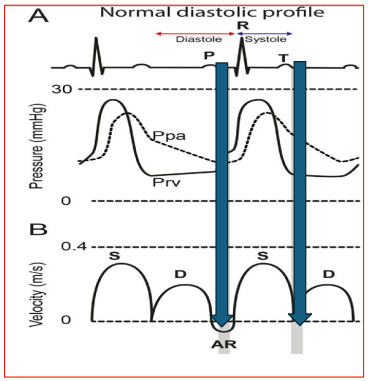
Waveform dynamics

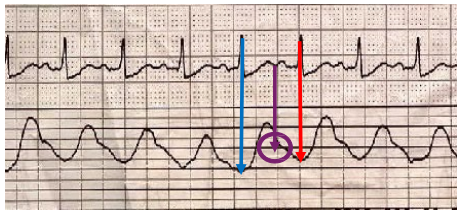


Grønlykke L, et al. "Preliminary experience using diastolic right ventricular pressure gradient monitoring in cardiac surgery." *Journ of Cardiothor & Vasc Anesth*, 34.8 (2020): 2116–2125.

51

PAP waveform

- Characteristics
  - Rapid up stroke and down stroke
  - Dicrotic notch (closure of pulmonic valve)
  - Smooth runoff
- End systolic wave occurs at the Twave
- End diastolic wave occurs after the QRS



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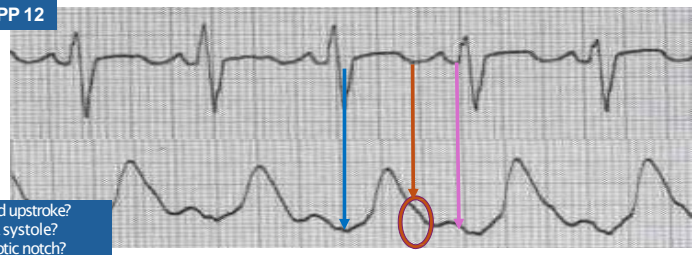
PA waveform

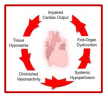
PaS 22
PaD 10
PA PP 12



Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

53

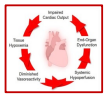
Does this look right to you?



Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

54

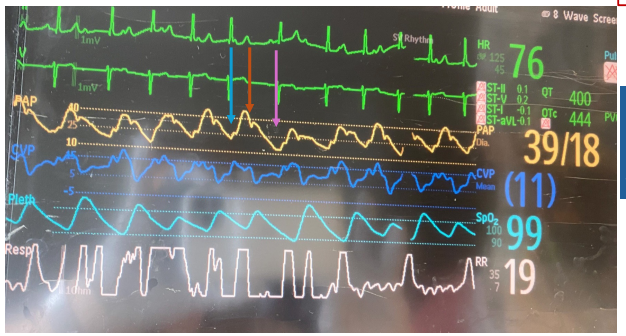
Does this look right to you?



Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

55

Does this look right to you?



Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

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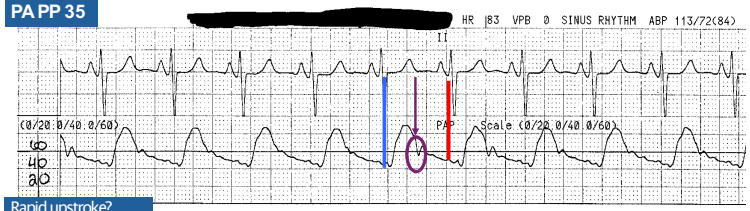
PA waveform

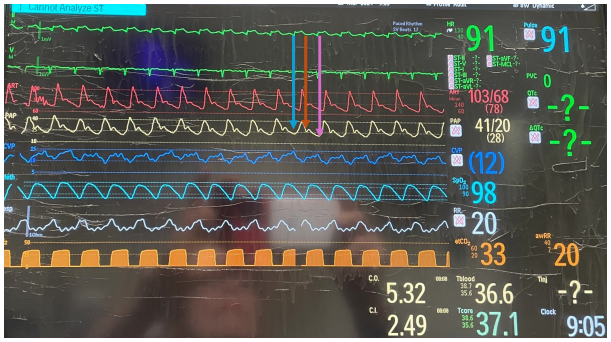
PaS 75
PaD 40
PA PP 35

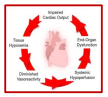


Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

57

Does this look right to you?

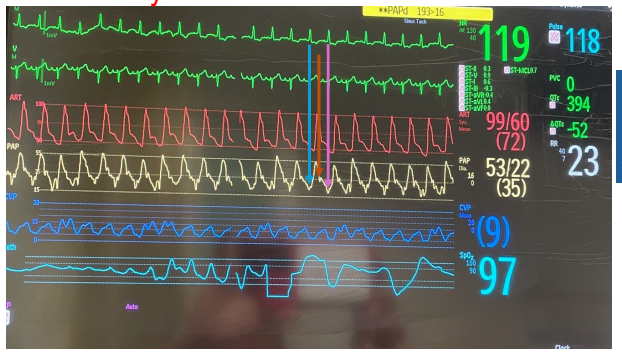


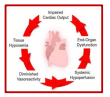


Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

58

What do you think?





Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

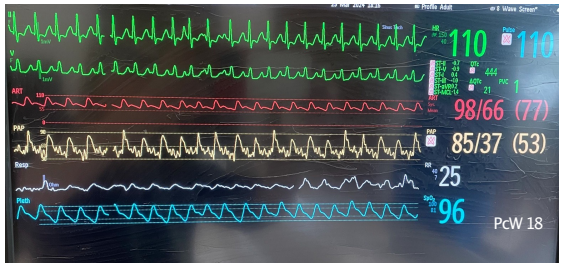
59

What do you think?

Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

60

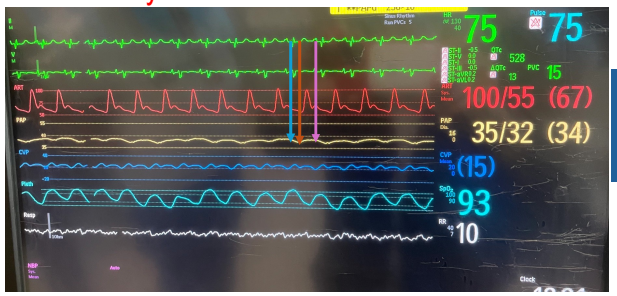
What do you think?

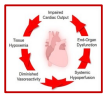


Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
Values OK?

61

What do you think?





Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

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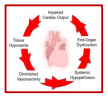
PAC repositioned!



Rapid upstroke?
Peak systole?
Dicrotic notch?
End diastole?
PA PP
Values OK?

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Would you trust this?

64

Evaluating the PA waveform

Wouldn't it be nice to have a direct RV pressure measure?

Even though PA and RA pressures may reflect an issue, indirect!

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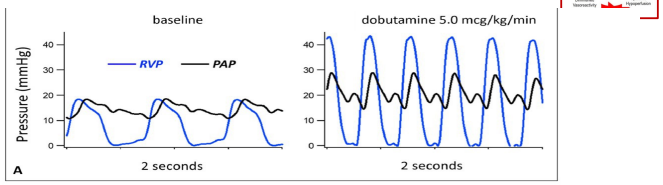
Evaluating the RVP waveform



Heerdt PM, et al. "Right ventricular pressure waveform analysis—clinical relevance and future directions." *Jour Cardiothor & Vasc Anesth*, 38.10 (2024): 2433-2445.

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Another sign to turn right

MAP – CVP = Organ Perfusion Pressure

Advanced PAC

Advanced Arterial based CO

Right-sided shock
CO = 2.0, MAP = 60, **CVP = 23**
Organ perfusion pressure = 37
(low MAP *and* high CVP)

- Organ perfusion pressure severely reduced
  - Renal failure
  - Liver failure
  - Bowel edema, translocation
    - further ↓ SBP
  - Ascites

Early onset multisystem organ failure

Left-sided shock
CO = 2.0, MAP = 60, **CVP = 8**
Organ perfusion pressure = 52
(low MAP *but* low CVP)

- Relatively preserved end organ perfusion

Hrymak C, Strumpher J, Jacobsohn E. Acute right ventricle failure in the intensive care unit: assessment and management. *Can Journal of Cardiology*, 2017, 33(1):61-71.

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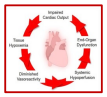
The right turn



Measure	Normal	RV dysfunction	Severe RV dysfunction	Consider?
RAP/CVP (mmHg) SP/MV*	<5 SP / <8 MV	>10 SP / >13 MV	>15 mmHg	Method of ventilator management? Reduce MawP? Pulmonary dilation (NO, Flowlan) RV inotropic (Dobutamine, Milrinone)
PAS/PAD	15–25/8–15	↑↑	↑↑↑	
RAP/PaOP	0.5 or less	>0.63	>0.86	
PAPI	>2	<2	<1.5	MCS

Read more about it!
Manek G, et al. Hemodynamic indices in pulmonary hypertension: a narrative review. *Cardiovasc Diagn Ther*. 2022 Oct;12(5):693-707. doi: 10.21037/cdt-22-244. PMID: 36329964; PMCID: PMC9622402.

*SP: spontaneous ventilation
MV: mechanical ventilation

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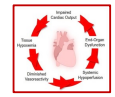
SCAI Shock Classification (Stages A–E)



- ✓Stage A: at risk:
  - large MI, no hypoperfusion.
- ✓Stage B: beginning :
  - Hypotension or rising lactate, no clear hypoperfusion
- ✓Stage C: classic shock :
  - hypotension +hypoperfusion +elevated lactate.
- ✓Stage D: deteriorating
  - Escalating support, worsening acidosis/lactate
- ✓Stage E: extremis
  - Refractory shock, impending arrest or ongoing CPR

Baran, D. A., Grines, C. L., Bailey, S., et al. (2019). **SCAI clinical expert consensus statement on the classification of cardiogenic shock**. *Catheterization and Cardiovascular Interventions*, 94(1), 29–37. https://doi.org/10.1002/ccd.28329

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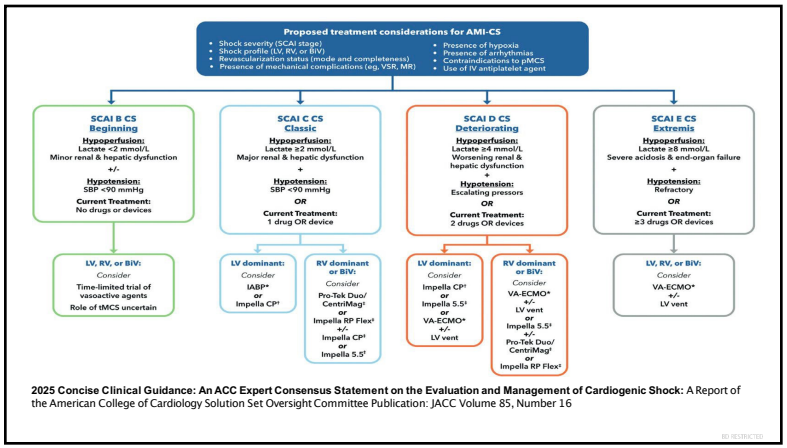

2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock: A Report of the American College of Cardiology Solution Set Oversight Committee Publication: JACC Volume 85, Number 16

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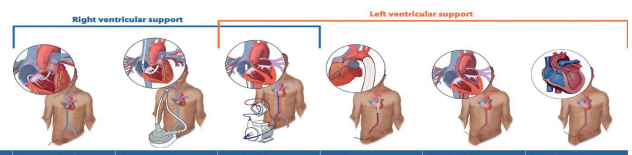

	Impella RP Flex	RA-PA pVAD	VA-ECMO	IABP	Impella CP	Impella 5.5
Max flow	3.0 - 4.0 L/min	4.0 - 5.0 L/min	5.0 - 7.0 L/min	0.5 - 1.0 L/min	3.0 - 4.3 L/min	5.0 - 6.0 L/min
Max pump speed	33,000 rpm	7,500 rpm	6,000 rpm	NA	46,000 rpm	33,000 rpm
Mechanism	Axial flow continuous pump (RA-to-PA)	Centrifugal flow continuous pump (RA-to-PA)	Centrifugal flow continuous pump (RA-to-AO)	Balloon inflation-deflation (AO)	Axial flow continuous pump (LV-to-AO)	Axial flow continuous pump (LV-to-AO)
Sheath size	23 F venous peel-away	29 or 31 F venous (inflow)	15-24 F arterial 19-25 F venous	7-8 F arterial	14 F arterial peel-away	23 F arterial peel-away
Typical insertion/ placement	Internal jugular vein	Internal jugular vein	Femoral vein (drain) Femoral artery (return)	Femoral artery or Axillary artery	Femoral artery or Axillary artery	Axillary artery
Direct LV unloading	-	-	-	-	+++	+++
Direct RV unloading	+	+	+	-	-	-
Afterload	-	-	↑↑↑	↓↓	↓	↓
Coronary perfusion	-	-	↑↑	↑↑	↑↑	↑↑

Sinha SS, Morrow DA, Kapur NK, et al. 2025 Concise Clinical Guidance: An ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock. *JACC*. Published online on March 17, 2025. doi: 10.1016/j.jacc.2025.02.018

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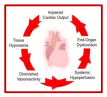
Why Early Recognition Matters



- ✓SCAI B–C patients can be rescued before full collapse.
- ✓Time to MCS (Impella, ECMO) matters—don't delay
- ✓Helps guide vasopressor and inotrope choice (e.g., norepinephrine vs dobutamine)
- ✓Multidisciplinary shock teams rely on accurate staging
- ✓New biomarkers add to the classic clues
- ✓Apply today! Save a heart and a life!
- ✓**Key Points**
  - **LV MCS** focuses on *forward flow augmentation and LV unloading*
  - **RV MCS** focuses on *unloading systemic venous congestion and restoring LV filling*
  - **VA-ECMO** supports both ventricles but increases LV afterload; in RV failure it may increase RV stress unless unloading is provided.
  - **Combination strategies** (e.g., ECpella =ECMO +Impella) may be required in biventricular shock

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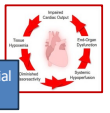
Comparison of MCS in Right vs. Left Ventricular Failure



Feature	LV failure: Systemic arterial measures	RV failure: pulmonary arterial measures
Hemodynamic problem	↓ LV contractility →↓ CO/CI (<2.2 L/min/m²), ↑ LVEDP/PCWP, pulmonary congestion	↓ RV contractility →systemic venous congestion, ↑ CVP, impaired LV filling (septal shift), ↓ forward flow
Primary targets	Augment systemic cardiac output, reduce LV filling pressures	Unload RV, reduce venous congestion, maintain LV preload, restore RV-PA coupling
Most used MCS	– **IABP** (afterload reduction, modest support) – **Impella CP/5.5** (direct LV unloading, ↑ CO) – **VA-ECMO** (full cardiopulmonary support, but ↑ LV afterload unless vented)	– **Impella RP** (percutaneous RV axial pump, unloads RV →PA) – **ProtekDuo cannula + CentriMag** (percutaneous RVAD via IJ, returns to PA) – **Surgical RVAD** (centrifugal or axial flow, bridge to recovery/transplant) – **VA-ECMO** (biventricular support, but increases RV afterload)
Adjunct/Bridge	Durable LVAD for advanced HF; bridge to transplant	Durable **BiVAD** or LVAD +temporary RVAD if RV recovery unlikely
Limitations	IABP: limited efficacy in severe LV failure Impella: vascular complications, hemolysis VA-ECMO: ↑ LV afterload unless vented (Impella/IABP combo)	RV Impella: limited to 14 days; not ideal with severe pulmonary HTN ProtekDuo: requires large-bore IJ access VA-ECMO: may worsen RV dilation unless LV vented
Weaning signals	↑ LV stroke volume, ↓ PCWP, improved MAP without support	↓ CVP (<12-15), improved RV stroke work index, normalization of RV-PA coupling (TAPSE/PASP > 0.31)

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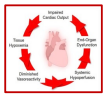
ESICM New Updates in 2025: How to diagnose Circulatory Shock



Skin Perfusion Index
Fluid Responsiveness
Arterial pressure targets
New CO monitoring devices
Echocardiography

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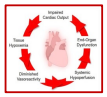
How should we define shock? ungraded

Highlights from Jan Bakker



Shock is defined as a life-threatening circulatory failure characterized by decreased tissue perfusion, created by inadequate oxygen delivery and/or oxygen utilization failure to meet cellular metabolic demands.

Not hypotension
Not singular lactate elevation
Clinical Sign:
- Monitoring skin perfusion using CRT and skin temperature and mottling

Clinical Measures:
- ScVO2 or SvO2
- P V-aCO2 Gap
- Gap/C a-v O2 difference (Gap)

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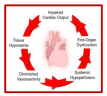
Fluid Therapy ungraded

Highlights from Antonio Messina



Fluid responsiveness must be assessed after intial fluid resuscitation

- Responsiveness disappears quickly
- Fluid overload is profoundly deleterious
- Measure efficacy of fluid administration
  - PLR for patients on MV
  - 300-500 mL given over 5-10 mins
  - evaluate with SV, PP, BPS, NOT MaP
- Diagnostic methodologies
  - PAC, PiCO, Dynamic variables (PPV, SVV)

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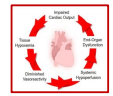
Fluid Therapy ungraded

Highlights from Xavier Monnet



Fluid responsiveness must be assessed after intial fluid resuscitation

- Measure efficacy of fluid administration
  - End Occulsion test
- Diagnostic methodologies
  - IVC compression is now of limited evaluation

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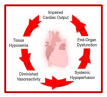
Cardiac Output Monitoring ungraded

Highlights from Xavier Monnet



Fluid responsiveness must be assessed after initial fluid resuscitation before more fluid is administered

Addition of low dose vasopressors AND if not responsive

CO should be monitored

- evaluated in conjunction with tissue oxygenation parameters
- Transpulmonary thermal dilution: no suspected/measured RV failure
- PA catheter: suspected/defined RV failure, ARDS measuring PA pressure
  - may be considered for any persistent shock

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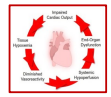
Arterial Pressure Monitoring ungraded

Highlights from Xavier Monnet



Should always be monitored in patients with Shock when not responsive to initial therapy and have not responded to low dose vasopressor therapy

Arterial pressure should be adapted to patient's condition

NO one target, always adjust to level of CVP and perfusion pressure

Measure and monitor CVP if central catheter is present

Consider MaP ↑ and CVP↓

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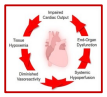
Echocardiography ungraded

Highlights from Michelle Chew



First line imaging modality to assess type of shock and hemodynamic status

Perform as soon as possible in any patient in shock

Serialization should be performed even if utilizing methods to monitor CO

Diagnostic importance in definition and evaluation between RV and LV failure leads to better intervention and therapeutic manipulation

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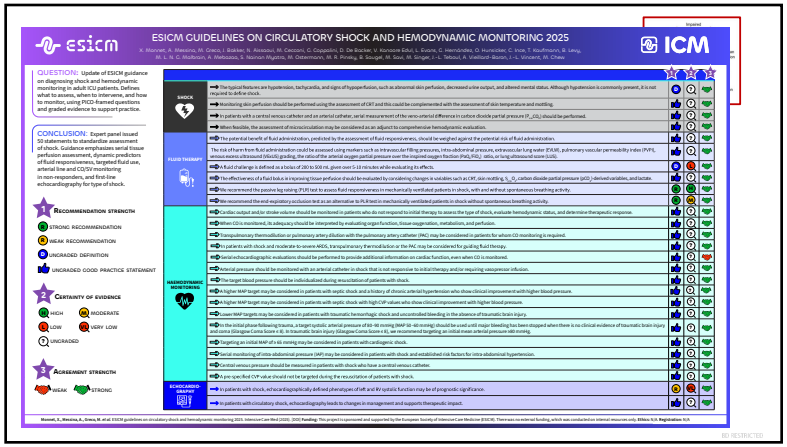

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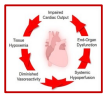

Follow the Clues

Thank you!

bmclean1@gmh.edu
404-626-2843

You tube: BarbaraMcLeanCriticalCare

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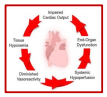

Barbara MacLean is a paid consultant of Becton Dickinson

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