LV function and haemodynamics in the critically ill

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19th March 2025 (25mins 15:45-16:10)

No relevant disclosures

Overview

- Echo tools for assessing LV & haemodynamics
- Echo in shock, pearls and pitfalls
- Clinical context / management

- Nepean ICU
- 1838 patients
- 5yr retrospective review
- **NEDA database** + **ANZICS CORE database**
- Cluster analysis

Unpublished data …

2.1 Variable correlation

Correlations of these variables and the results are shown below.

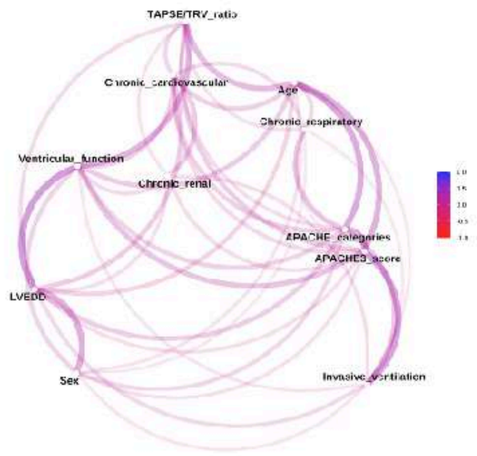


Figure 1. Correlation network. *The figure shows the correlation between the variables. Strong correlation is indicated by darker and thicker connecting lines. Blue = positive correlation, red = negative correlation.*

Conclusion *Most of the variables were weakly correlated with each other (r = 0.08 [0.02, 0.16]*

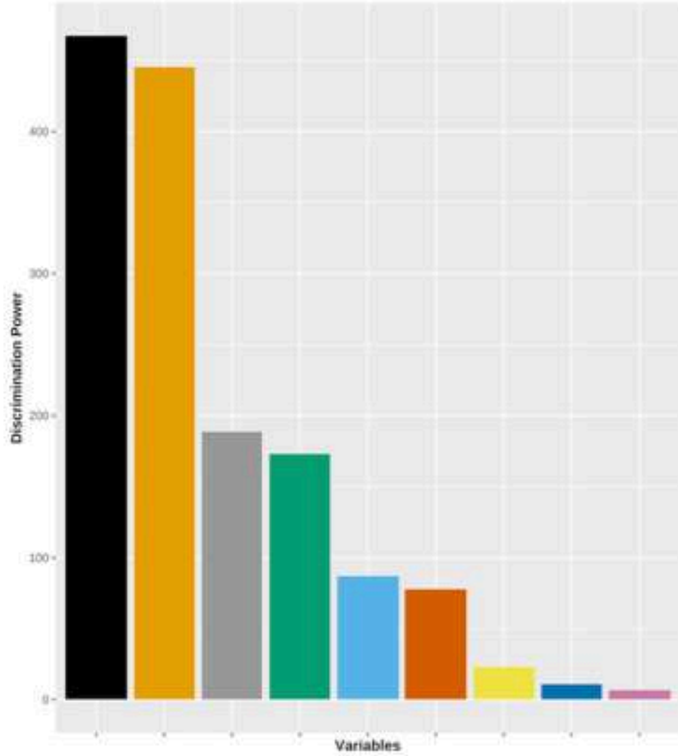


Figure 3. Discrimninative power of selected variables.

Conclusion *Among the 11 variables, only 9 are deemed relevant based for clustering. The most discriminative variables are* ***Ventricular function*** *and* ***Age****, followed by* ***TAPSE/TRV ratio*** *and* ***APACHE3 score*** *which shows moderate discrimination powers.* ***APACHE categories*** *and* ***LVEDD*** *also offer relatively good discriminative power.* ***Chronic cardiovascular, Invasive ventilation*** *and* ***Sex*** *offer less discriminative power. Histories of* ***Chronic respiratory*** *and* ***Chronic renal*** *are not relevant in discriminating the clusters, hence were excluded.*

Echo to help differentiate and manage ....

SHOCK

Intensive Care Med (2024) 50:1668–1680
https://doi.org/10.1007/s00134-024-07590-6

ORIGINAL

The use of echocardiography in the management of shock in critical care: a prospective, multi-centre, observational study

Luke Flower[1,2,3*], Alicia Waite[4,5,6], Adam Boulton[7], Marcus Peck[8], Waqas Akhtar[9], Andrew J. Boyle[10,11], Sandeep Gudibande[12,13], Thomas E. Ingram[14], Brian Johnston[5,6], Sarah Marsh[15], Ashley Miller[16], Amy Nash[17], Olusegun Olusanya[18], Prashant Parulekar[19], Daniel Wagstaff[20], Jonathan Wilkinson[21], Alastair G. Proudfoot[18,22] on behalf of the NEAT ECHO Collaborators[23]

178 critical care units in UK (255 total)
>1000 patients with shock followed for 72hours
over a 1 week period
>1000 pts
(Image storage & documentation low)

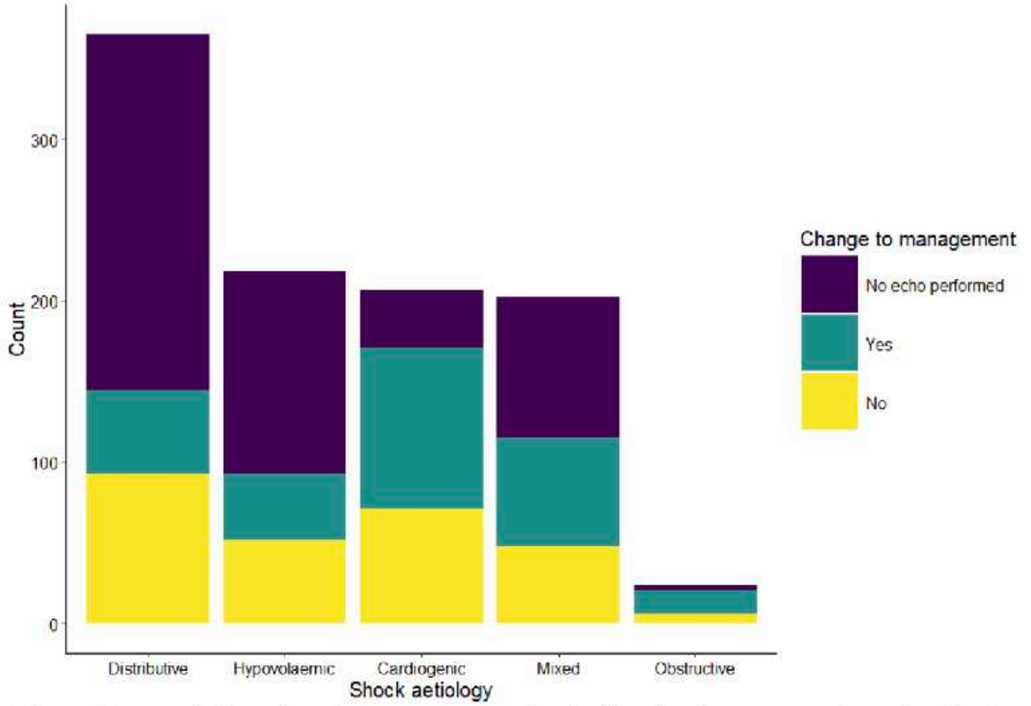


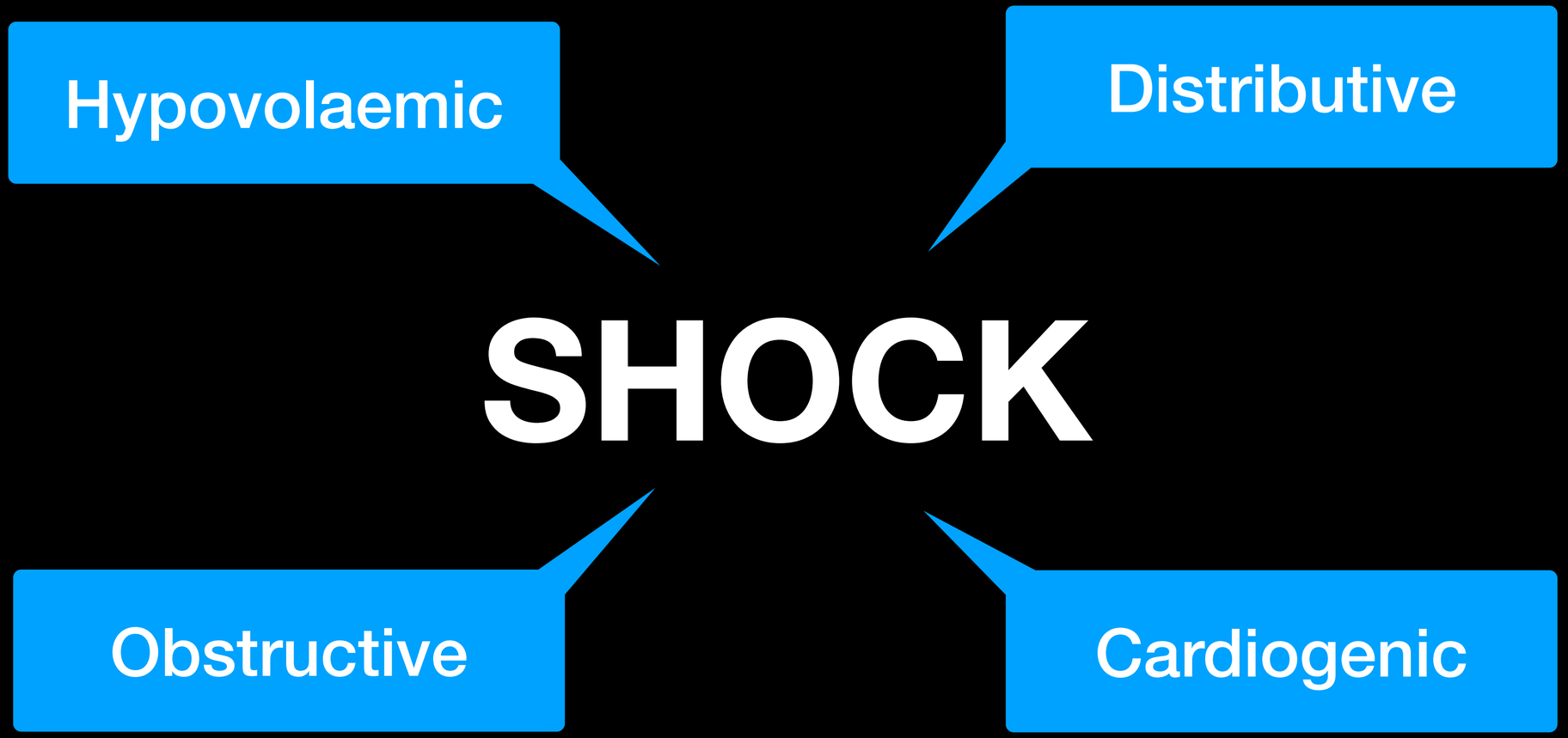
Fig. 1 Shock aetiology and impact of echocardiography on management. A stacked bar chart demonstrating the number of patients with each shock aetiology (count), the proportion of those patients that received an echocardiogram that altered their management (green), the proportion of patients that had an echocardiogram that did not alter their management (yellow) and the proportion of patients that did not receive an echocardiogram (purple)

Echocardiography was reported to either reduce diagnostic uncertainty or change management in 291 (54%)

Conclusion: Use of echocardiography in the assessment of patients with shock remains heterogenous. When echocardiography is used, it improves diagnostic certainty or changes management in most patients. Future research should explore barriers to increasing use of echocardiography in assessing patients presenting with shock.

ECHO AUSTRALIA

Echo to help differentiate and manage ....



Oxygen delivery is dependent on oxygen in blood and …

Cardiac output

which is made up from …

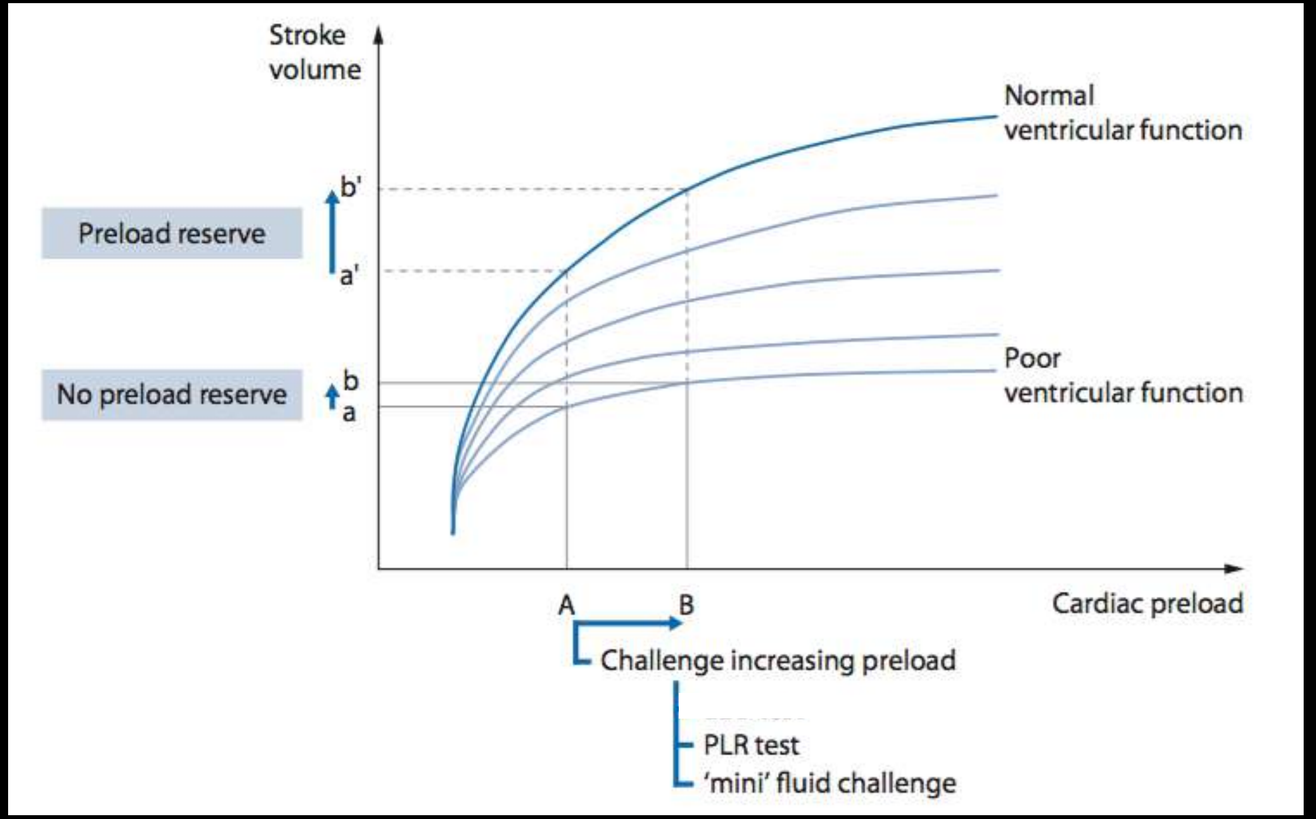
Preload + Contractility + Afterload

Preload = initial stretch of cardiac muscle fibres just before contraction

Contractility = ability of the ventricle to generate pressure and eject blood.

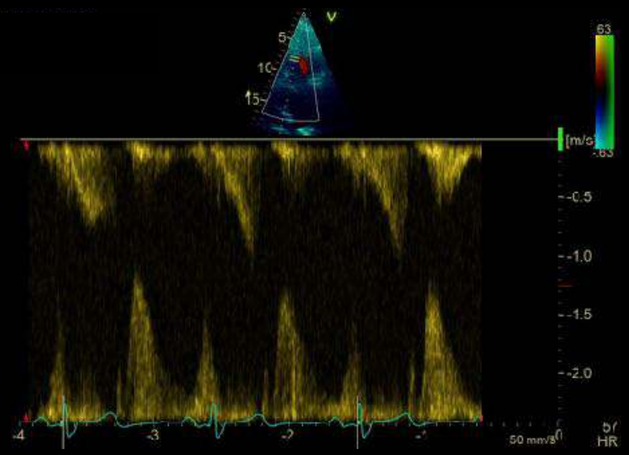
Afterload = pressure the LV must overcome to eject blood

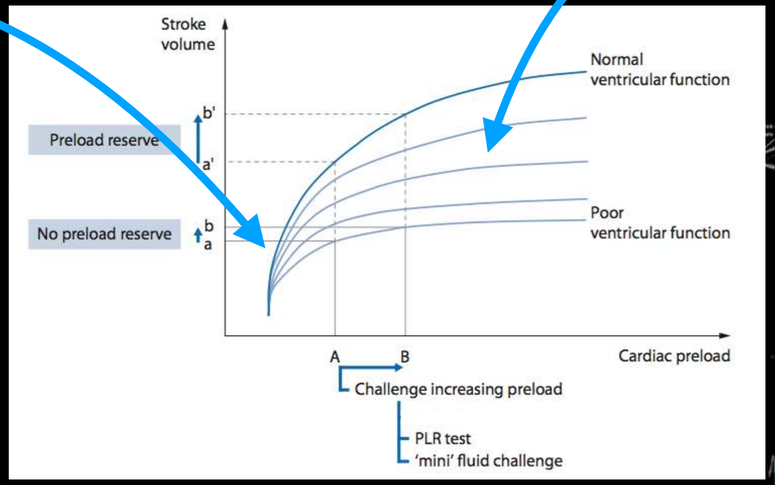
Preload

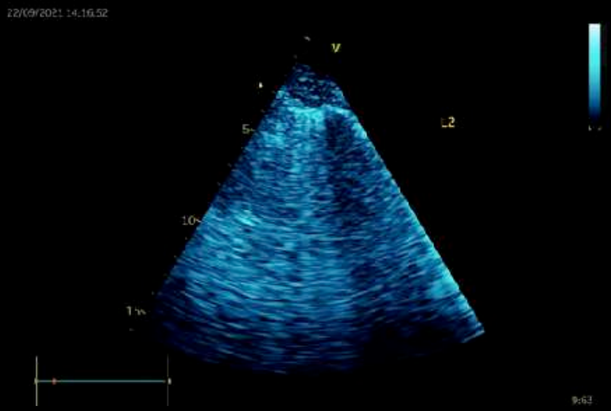

Stroke volume
Normal ventricular function
b'
Preload reserve
a'
No preload reserve
b
a
Poor ventricular function
A
B
Cardiac preload
Challenge increasing preload
PLR test
'mini' fluid challenge

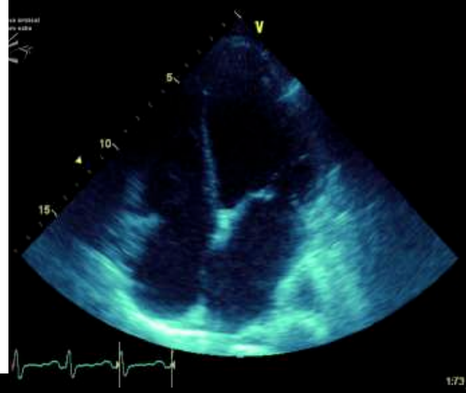
Preload

Static markers



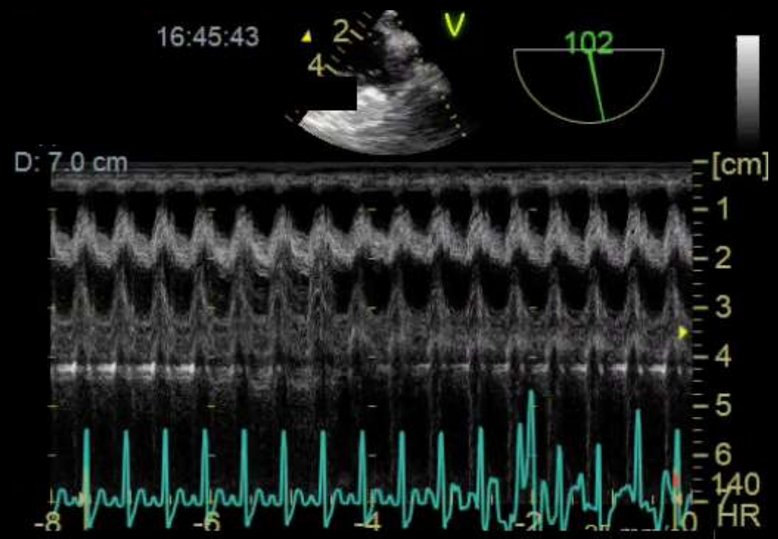


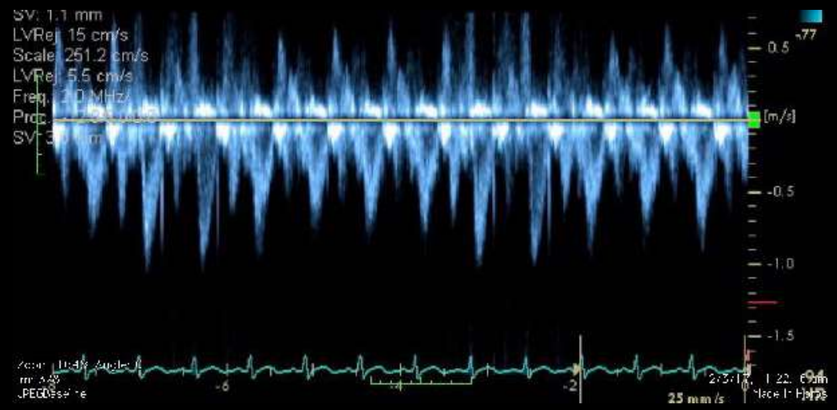


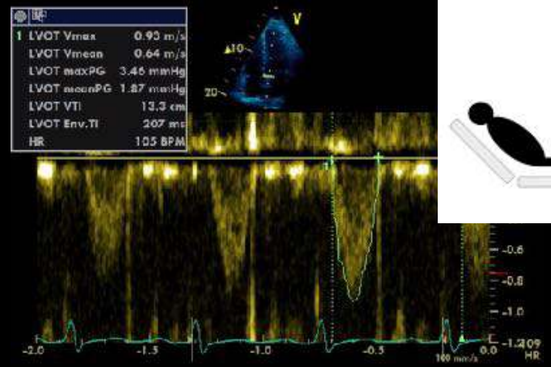


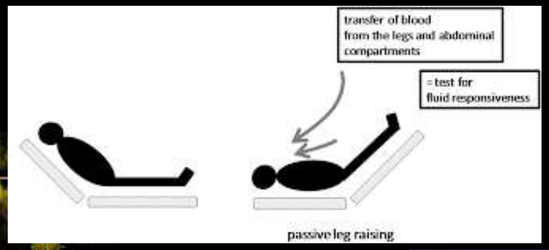
Preload

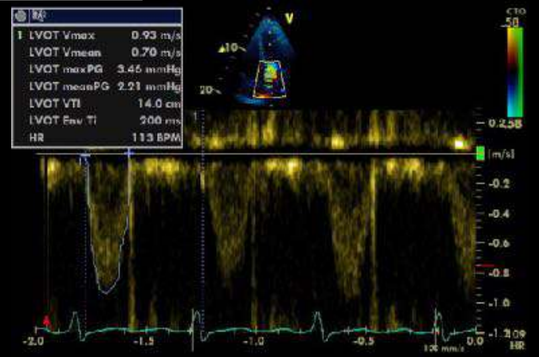
Dynamic markers

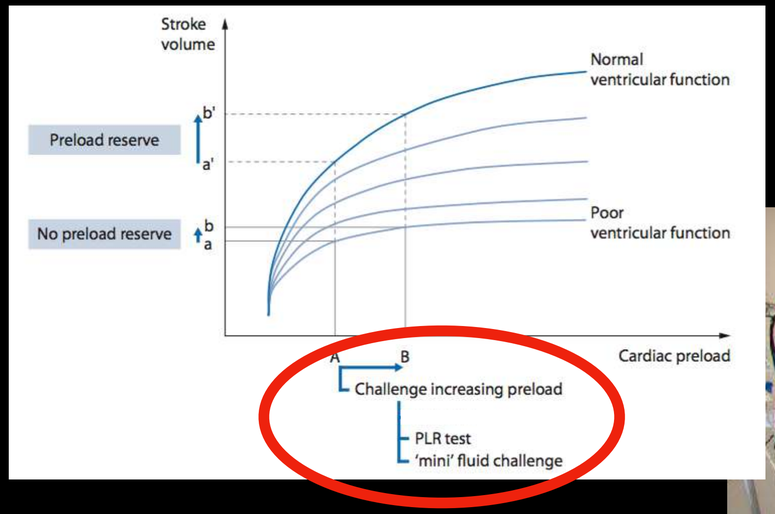




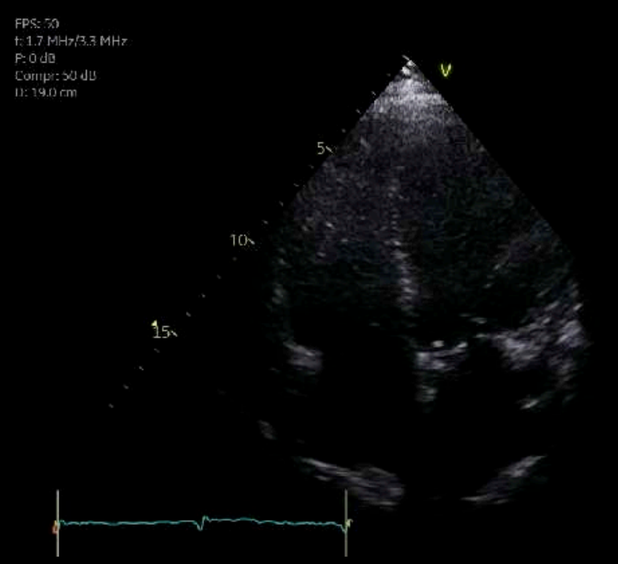


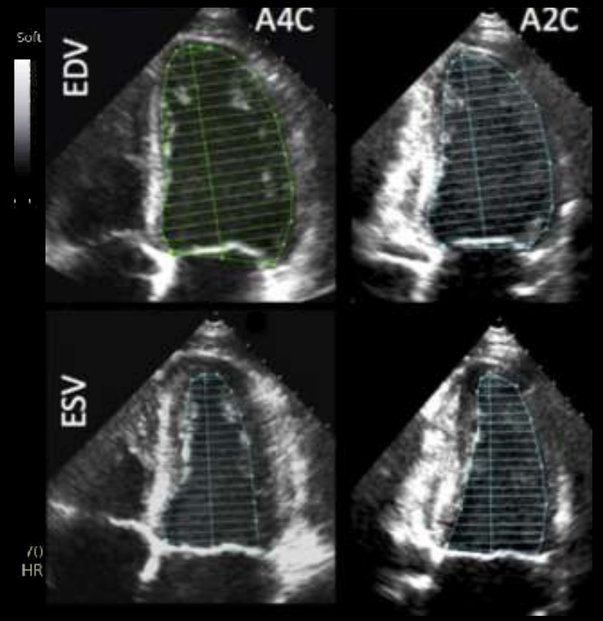


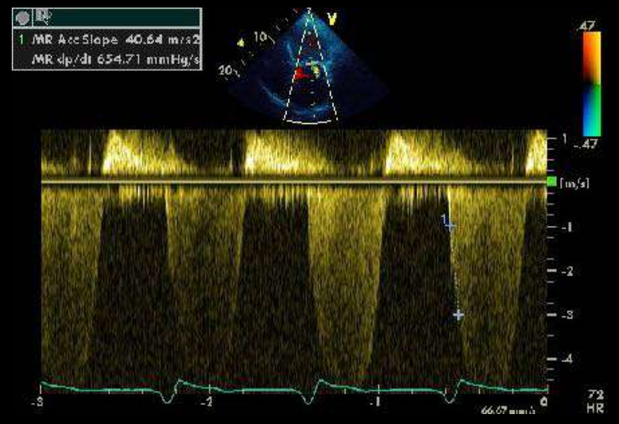


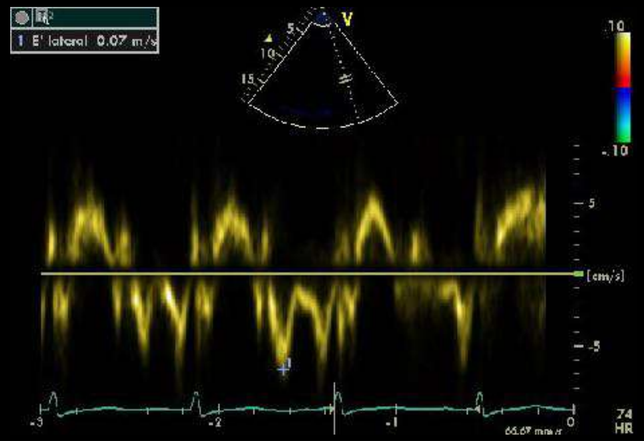


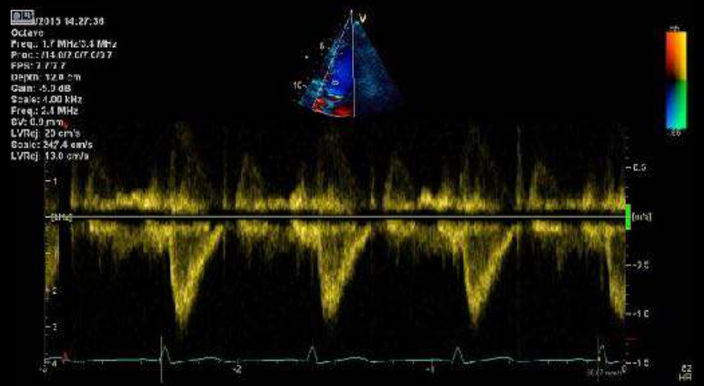
Contractility



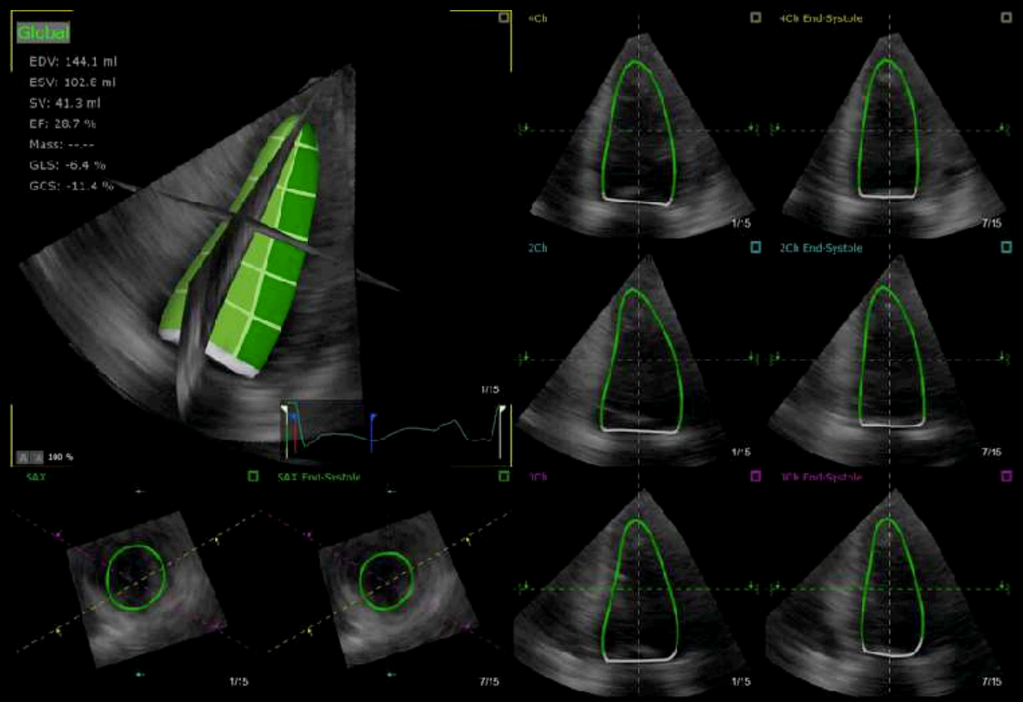


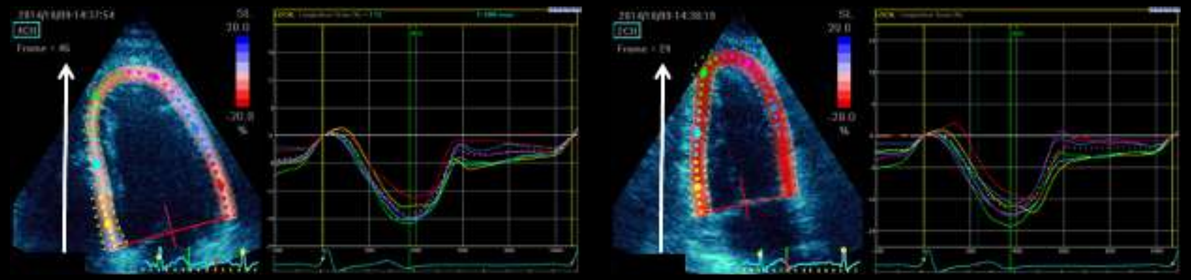


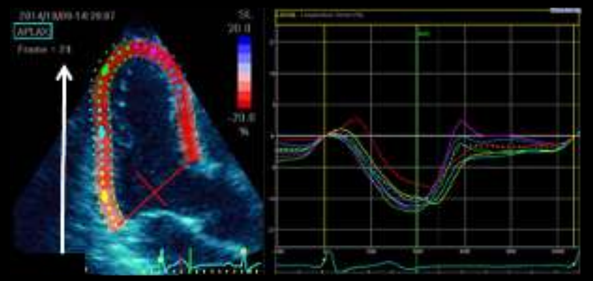


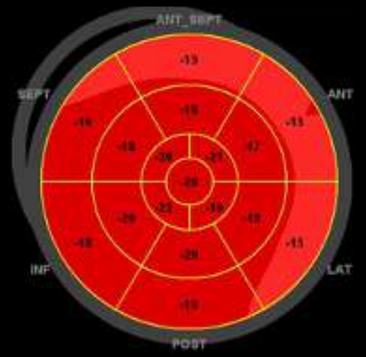


Contractility

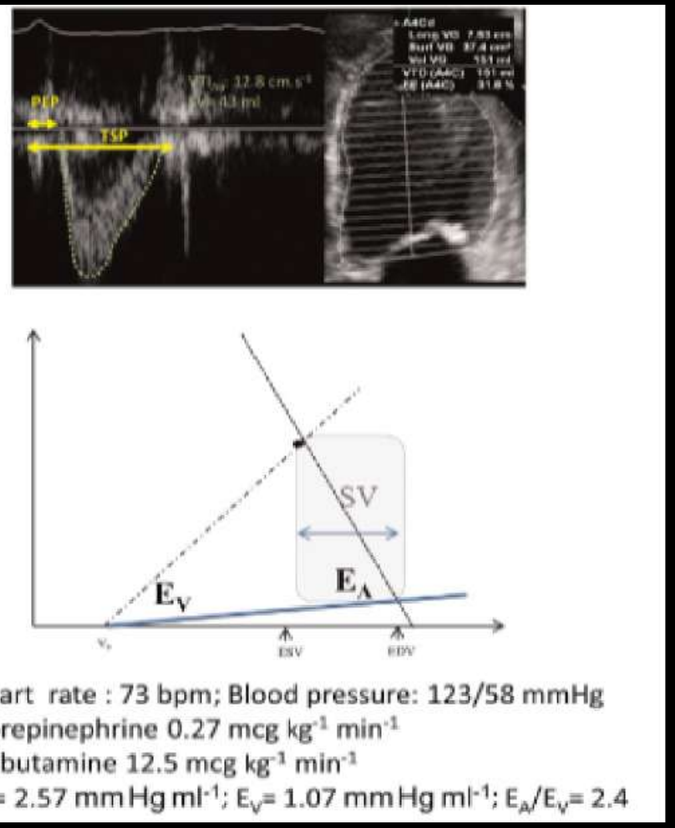








Afterload



Heart rate : 73 bpm; Blood pressure: 123/58 mmHg
Norepinephrine 0.27 mcg kg^{-1} min^{-1}
Dobutamine 12.5 mcg kg^{-1} min^{-1}
E_A= 2.57 mm Hg ml^{-1}; E_V= 1.07 mm Hg ml^{-1}; E_A/E_V= 2.4

ESC European Society of Cardiology
European Journal of Heart Failure (2022) **24**, 600–602
doi:10.1002/ejhf.2456
VIEWPOINT

How to calculate ventricular–arterial coupling?

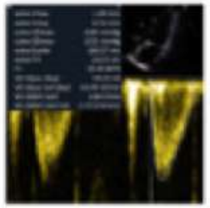
Hannes Holm[1,2*], Martin Magnusson[1,2,3,4], Amra Jujić[1,2], Erwan Bozec[5], and Nicolas Girerd[5]

How to measure VAC in routine practice

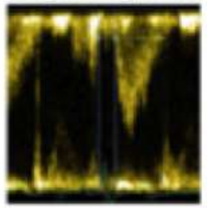
Echocardiography

EF

Stroke volume (SV)

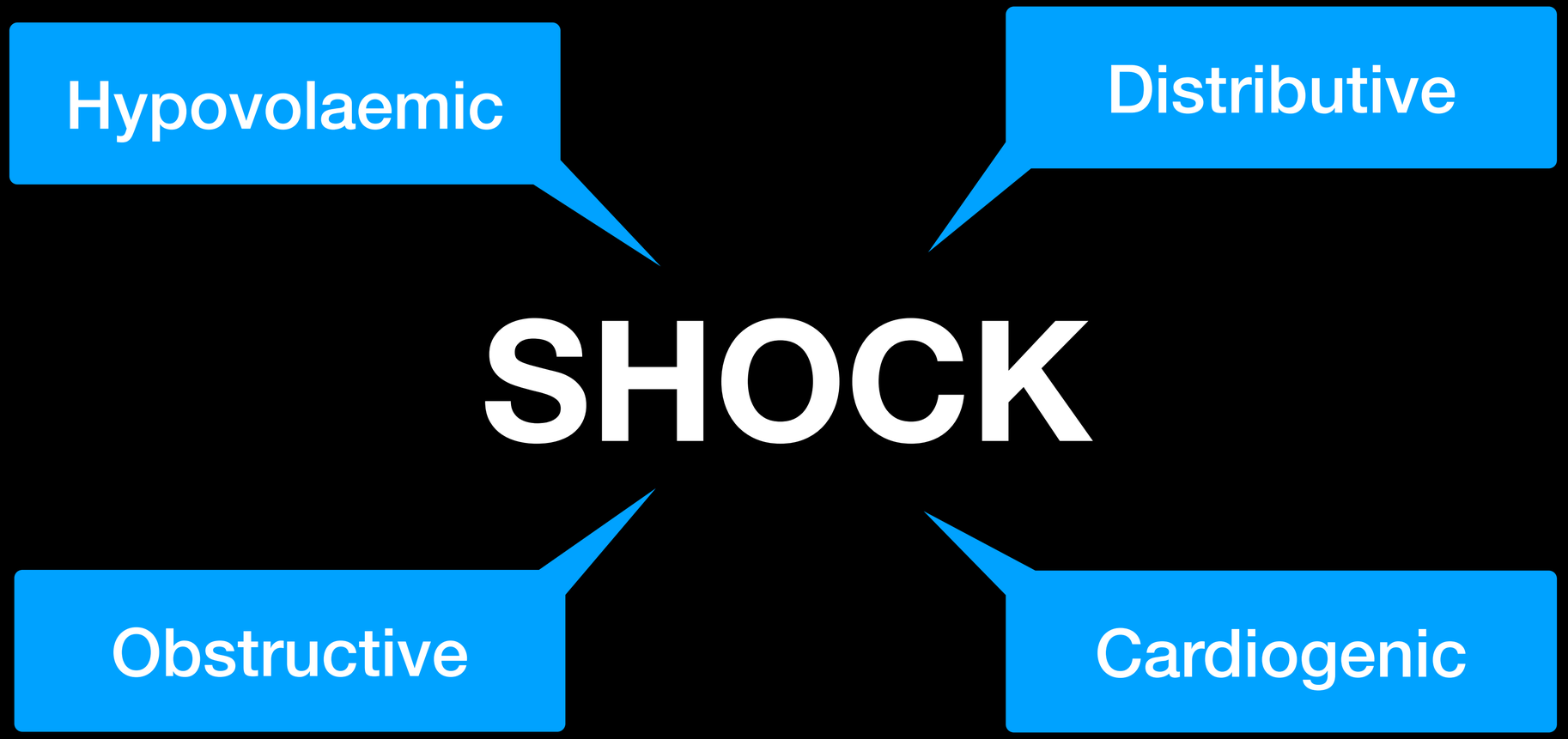


pre-ejection period to total systolic period (tNd)

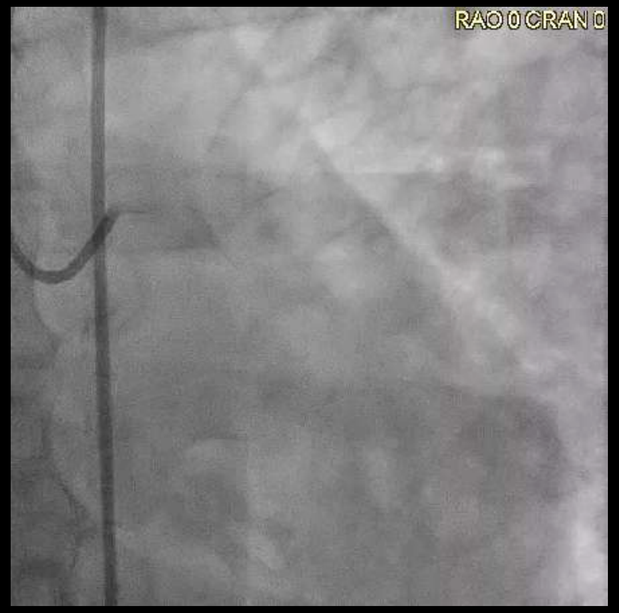


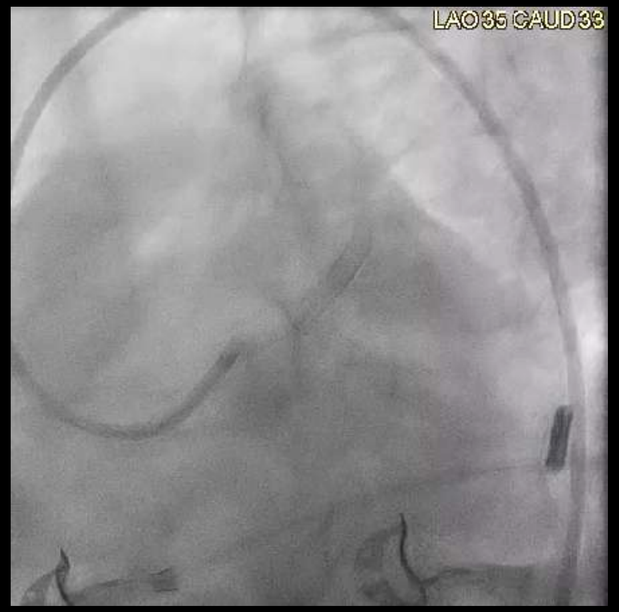
+

Blood pressure measurement

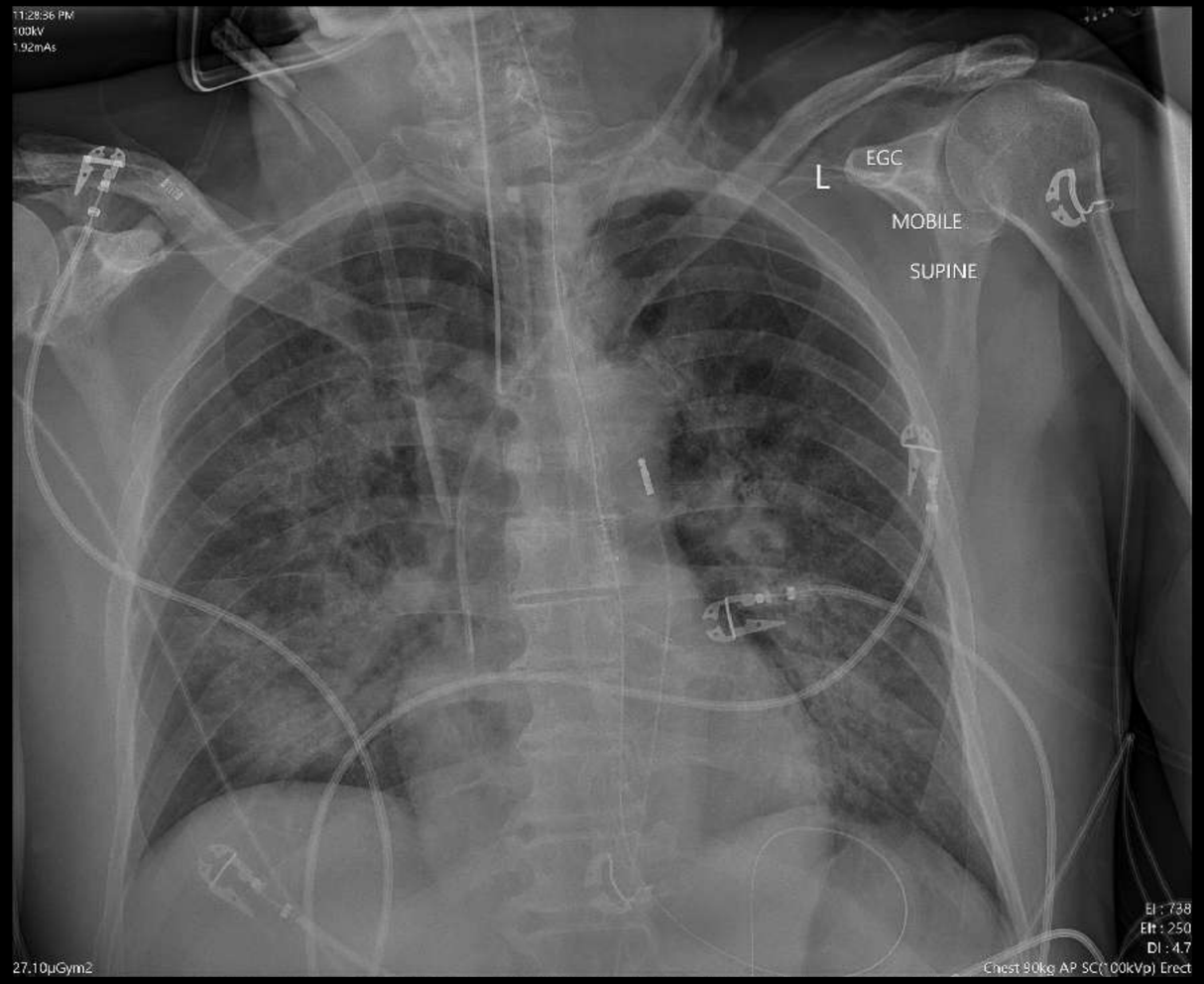

Hypovolaemic
Distributive
SHOCK
Obstructive
Cardiogenic

1st cardiogenic shock pt

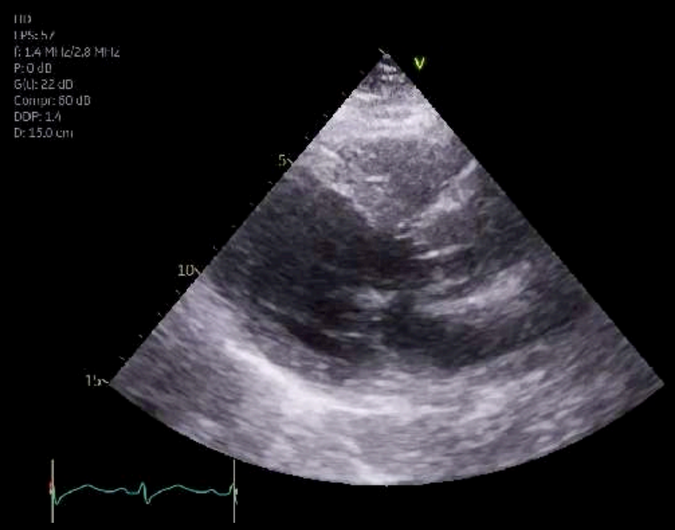


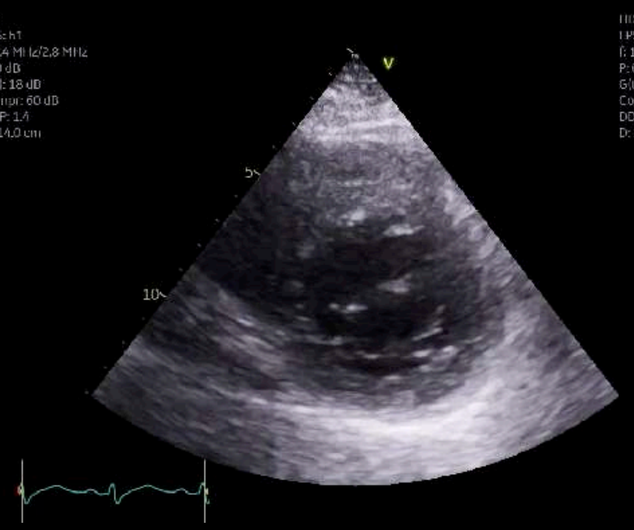


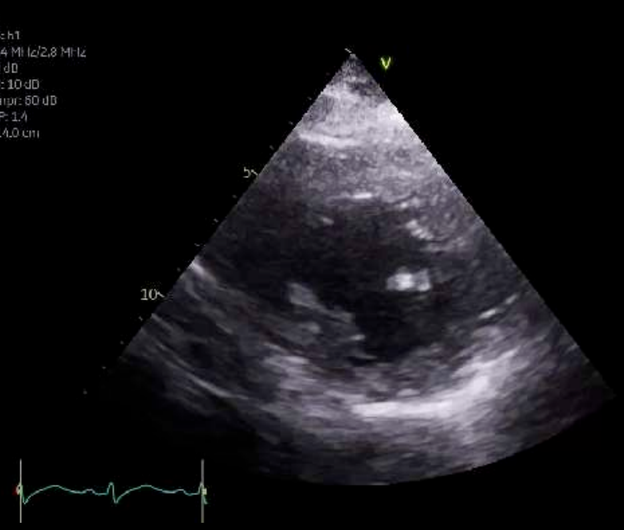
L main 95%
Cx 90%
Stent x3
Cardiogenic shock
IABP inserted
Dobutamine 5mcg/kg/min



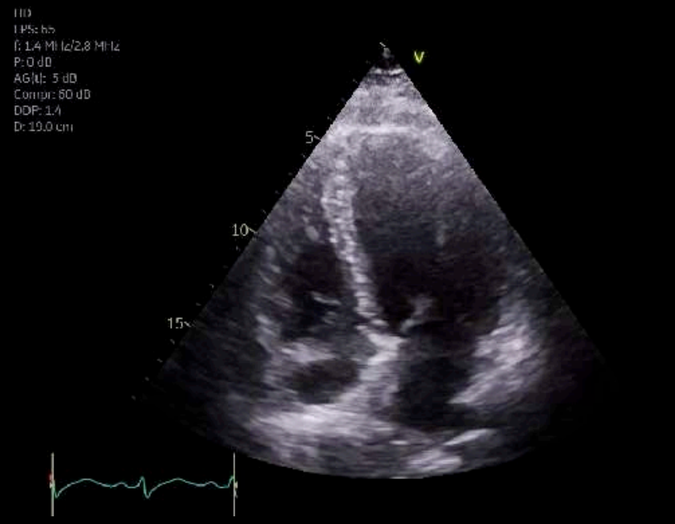
1st cardiogenic shock pt

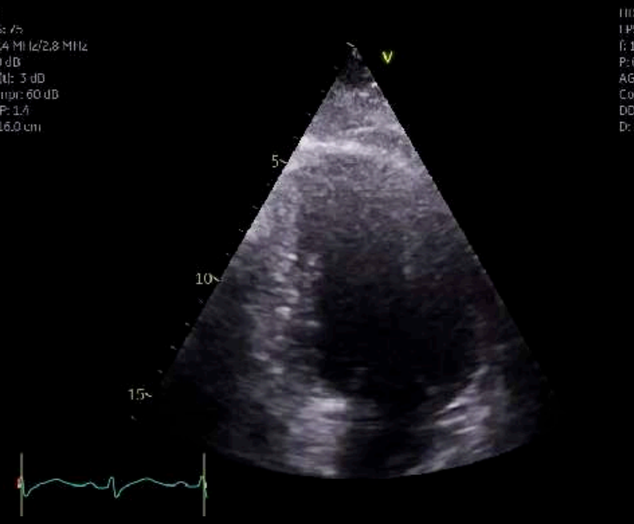






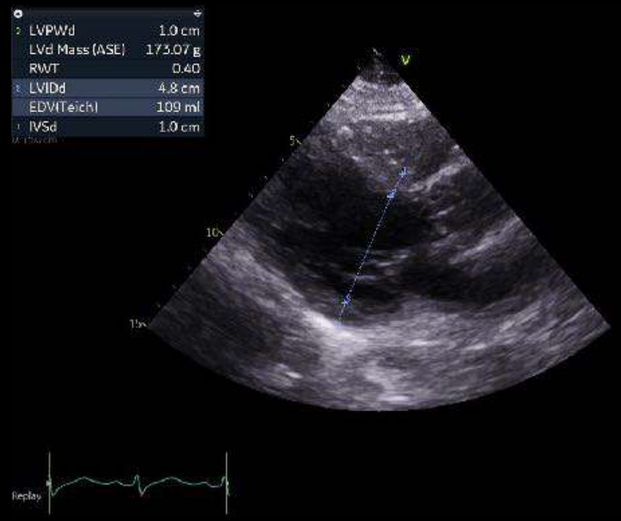
L main 95%
Cx 90%
Stent x3
Cardiogenic shock

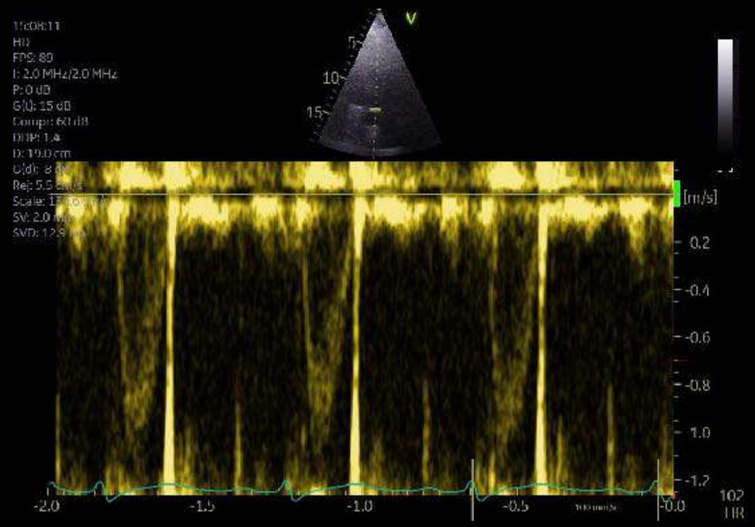




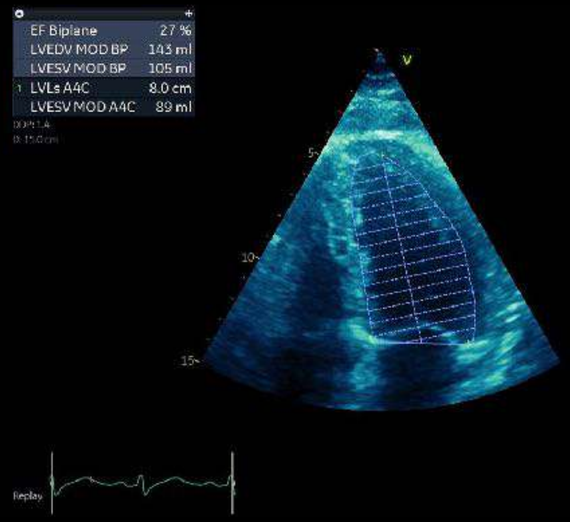
1st cardiogenic shock pt

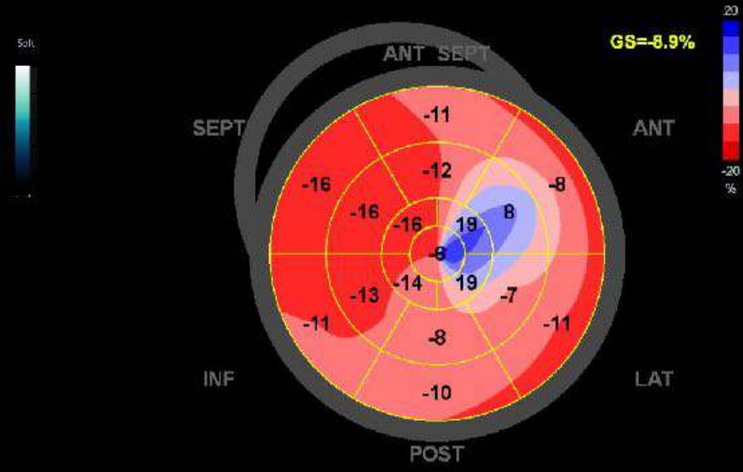
EF ~30% LVIDd 48mm LVEDV 140ml SV 39ml



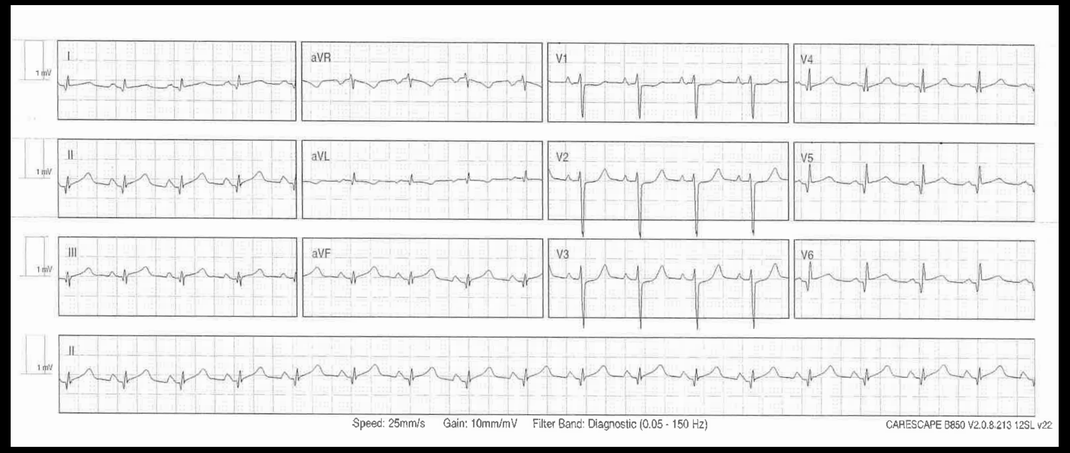


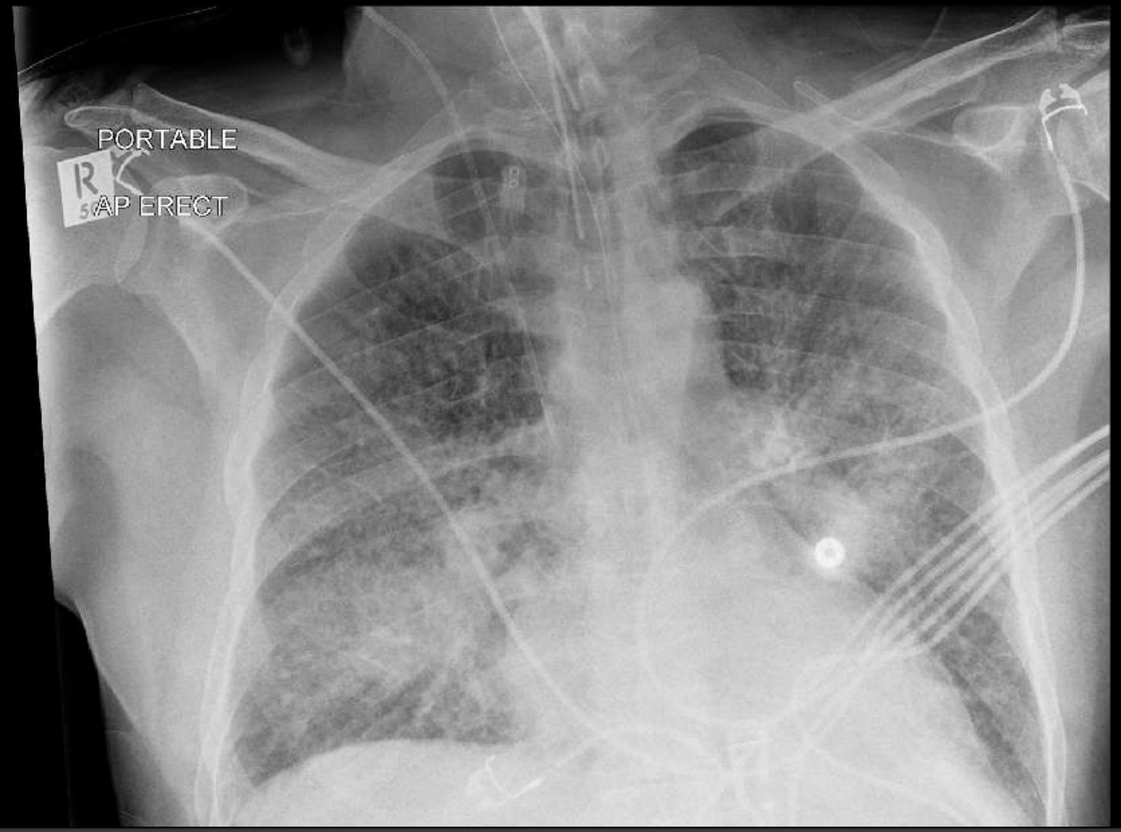
LVOT Diam	2.0 cm	Av	2.0
LVOT Trace			
LVOT Vmax	1.00 m/s	Av	1.00
LVOT Vmean	0.72 m/s	Av	0.72
LVOT maxPG	4.04 mmHg	Av	4.04
LVOT meanPG	2.33 mmHg	Av	2.33
LVOT Env.Ti	166 ms	Av	166
LVOT VTI	11.8 cm	Av	11.8
HR	102 BPM	Av	102
LVSV Dopp	39 ml		39
LVCO Dopp	3.98 l/min		3.98





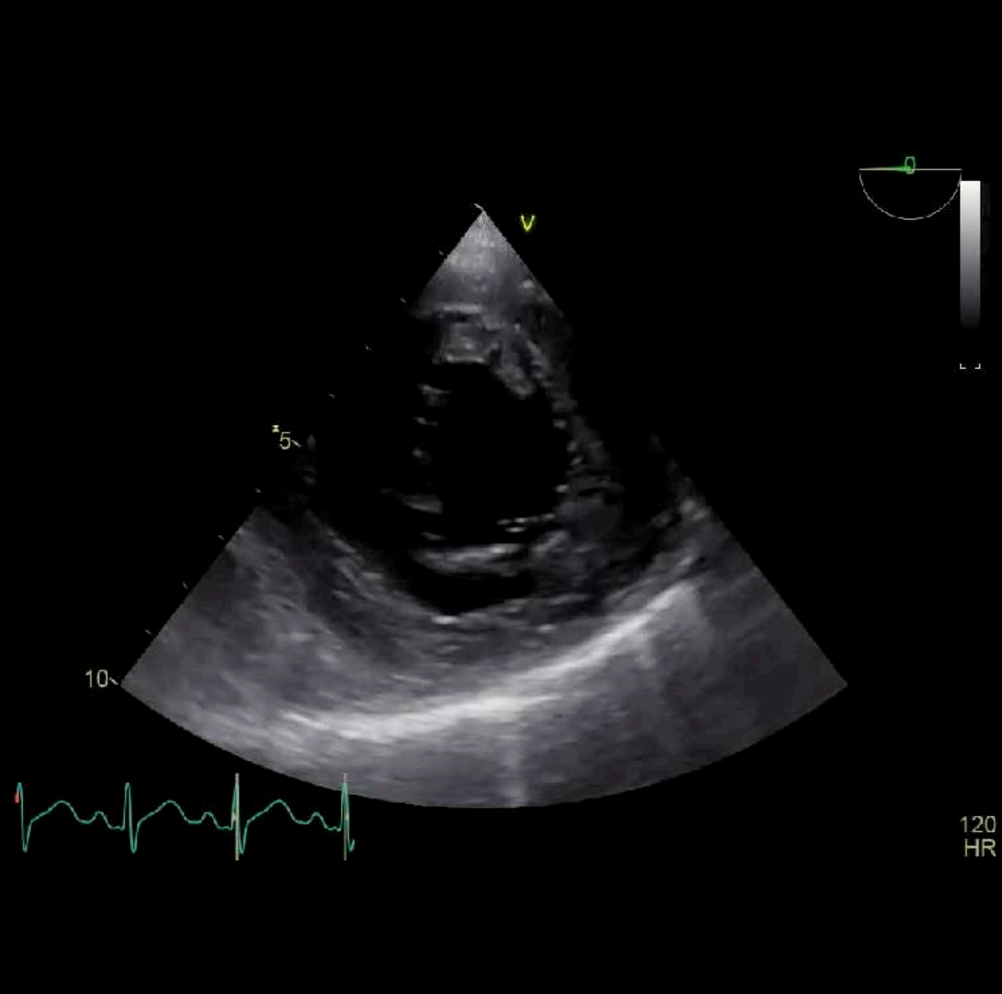
2nd cardiogenic shock pt

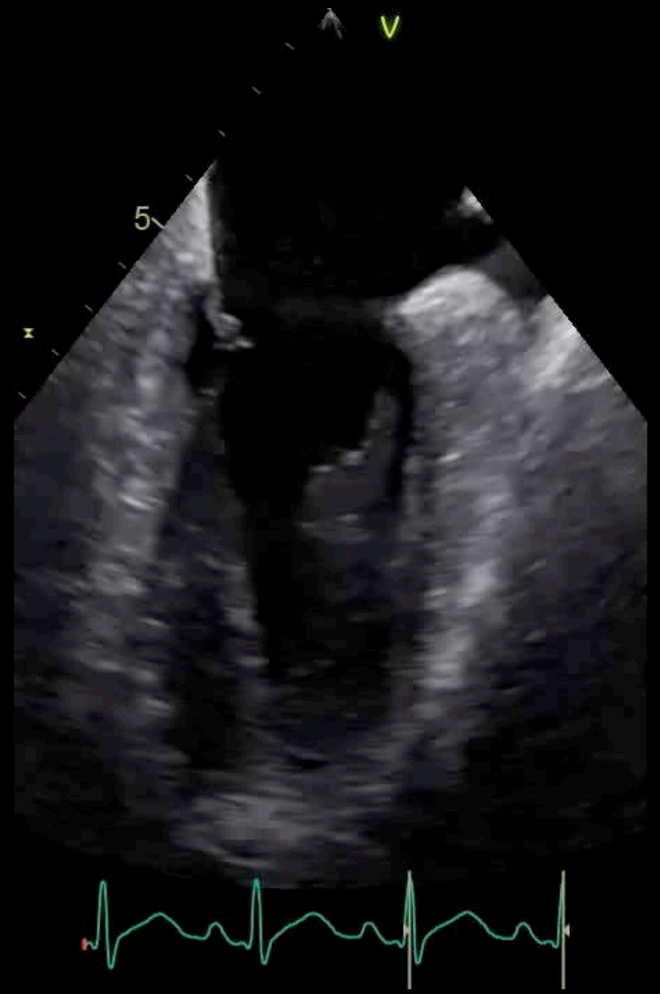


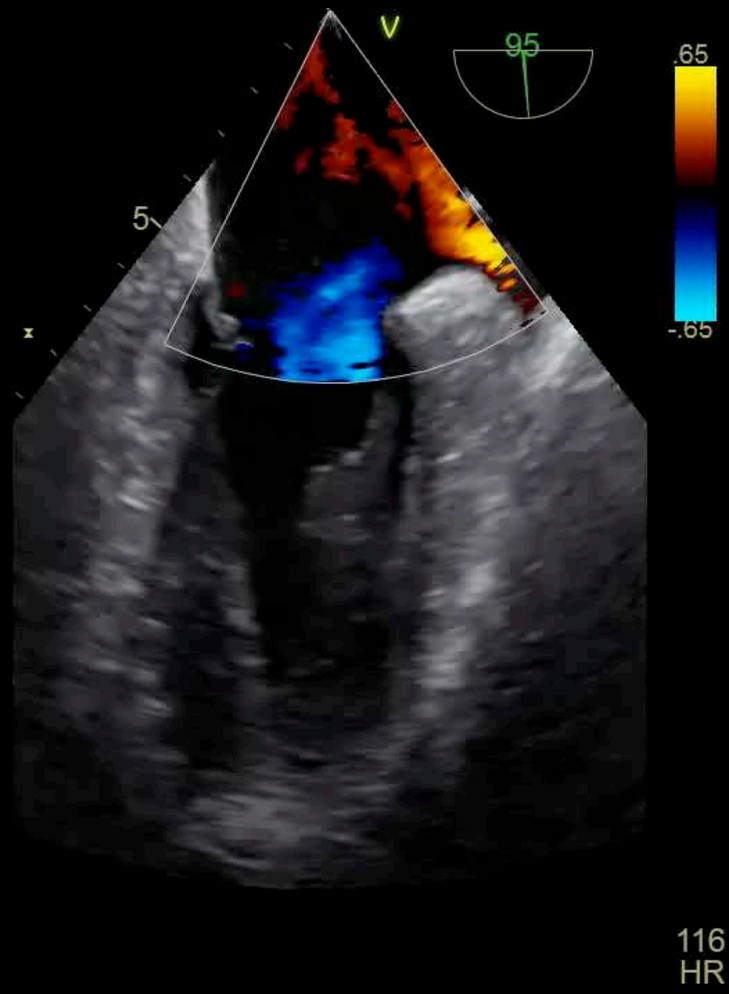


STEMI
100% Cx occlusion
Stented
Cardiogenic shock

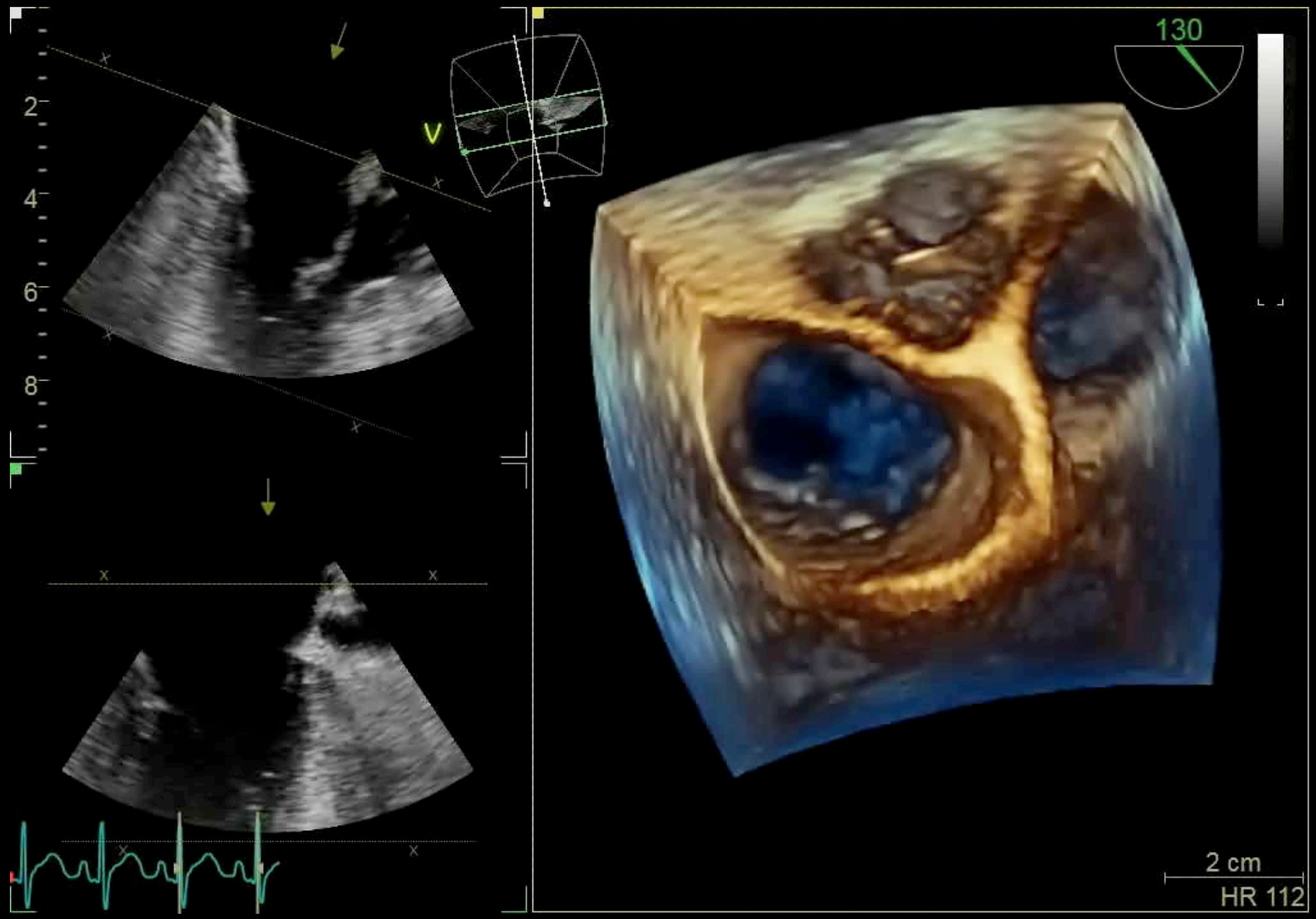
2nd cardiogenic shock pt





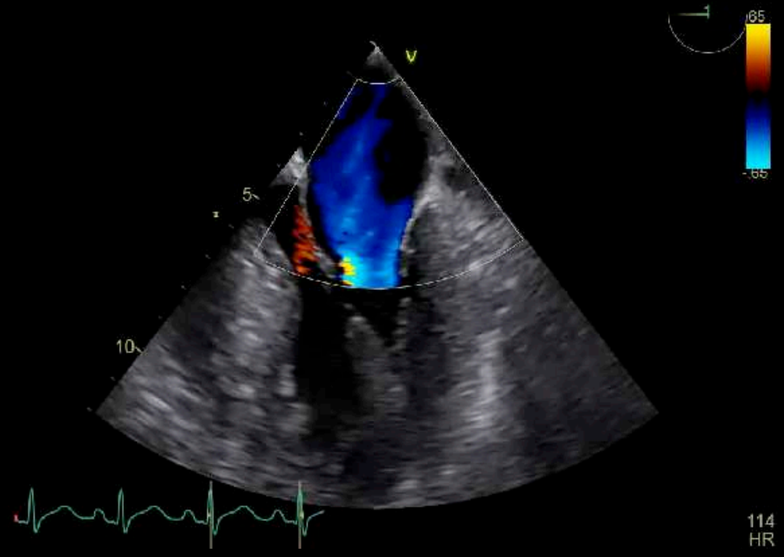


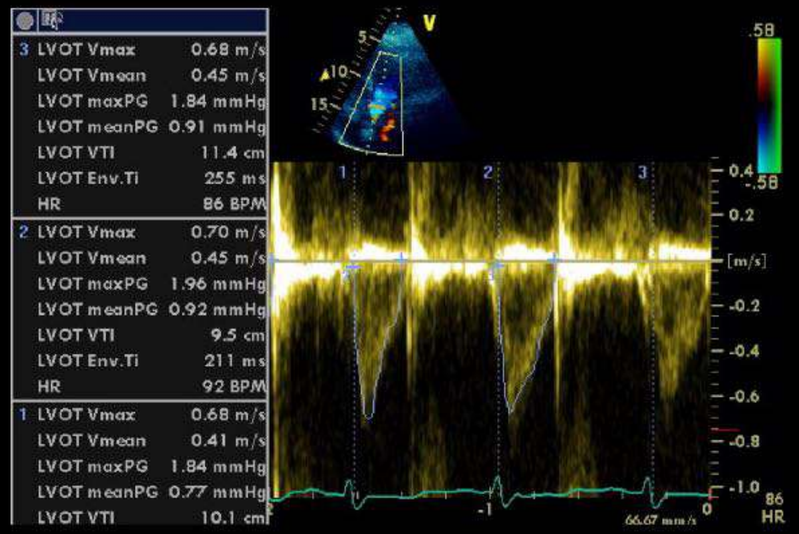
2nd cardiogenic shock pt

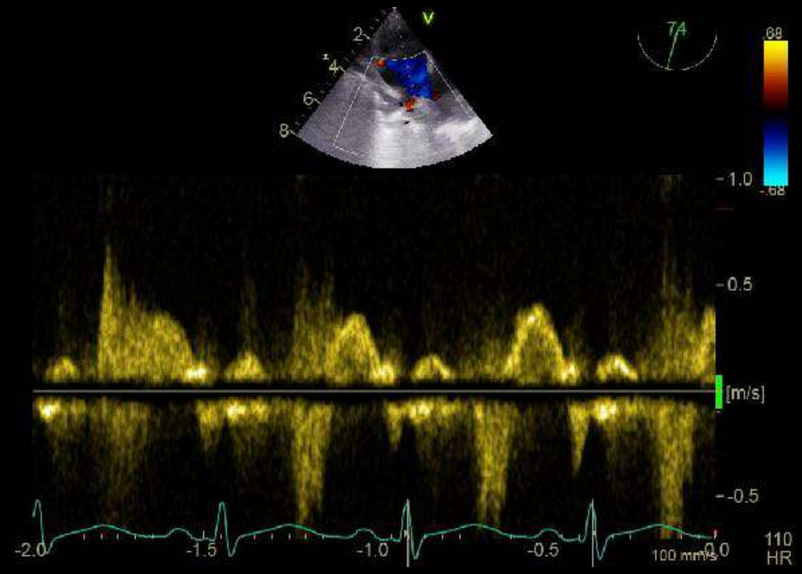


2nd cardiogenic shock pt

EF >75% LVIDd 35mm LVEDV 60ml SV 34ml



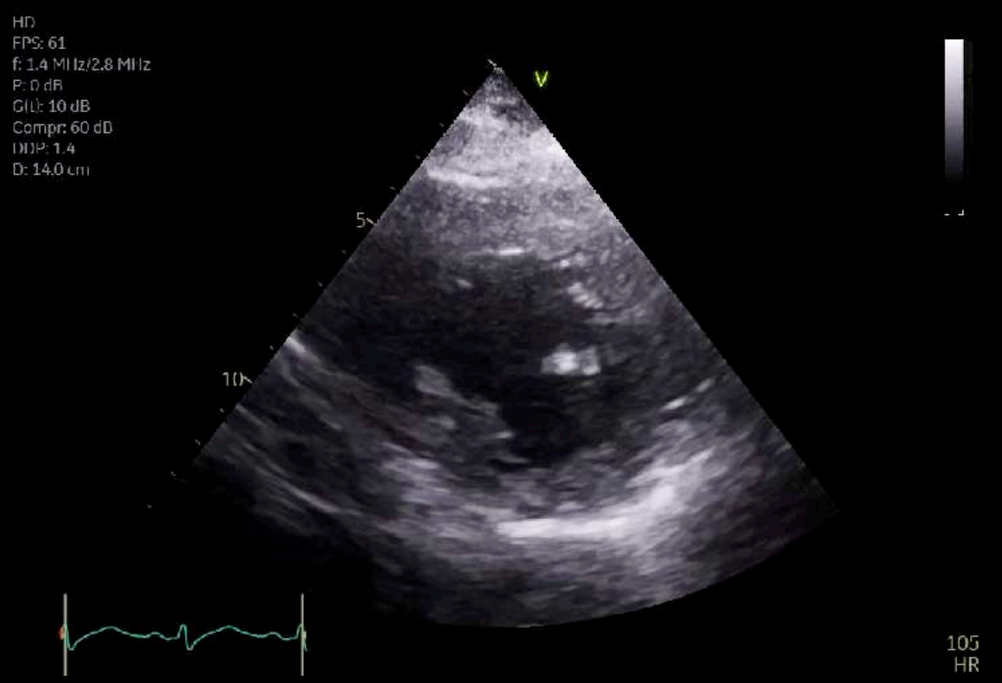




CARDIOGENIC SHOCK PATIENTS

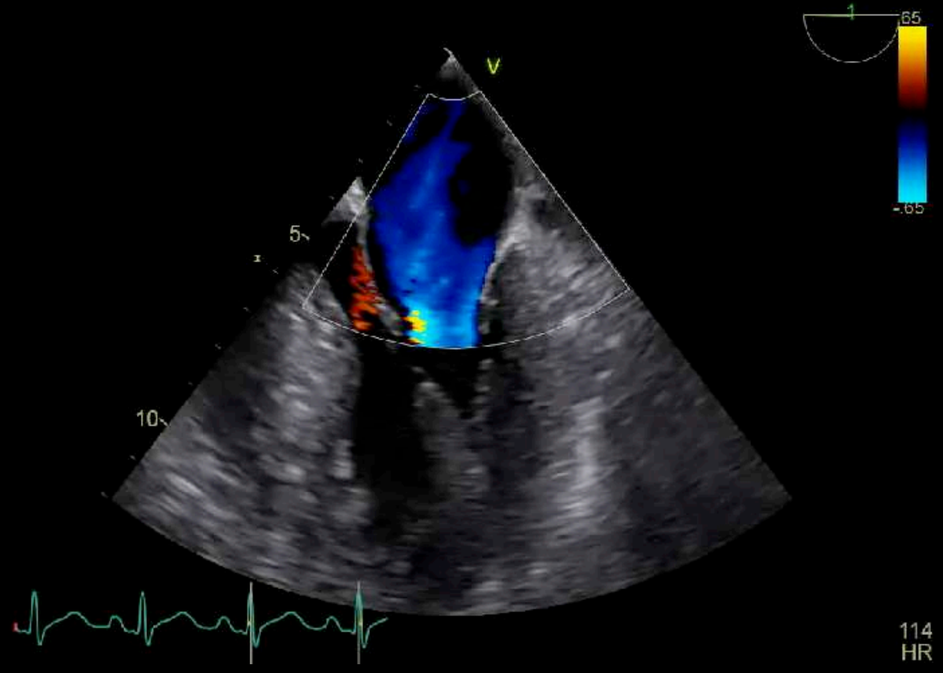
AMI PATIENT

EF ~30% LVIDd 48mm LVEDV 140ml SV 39ml



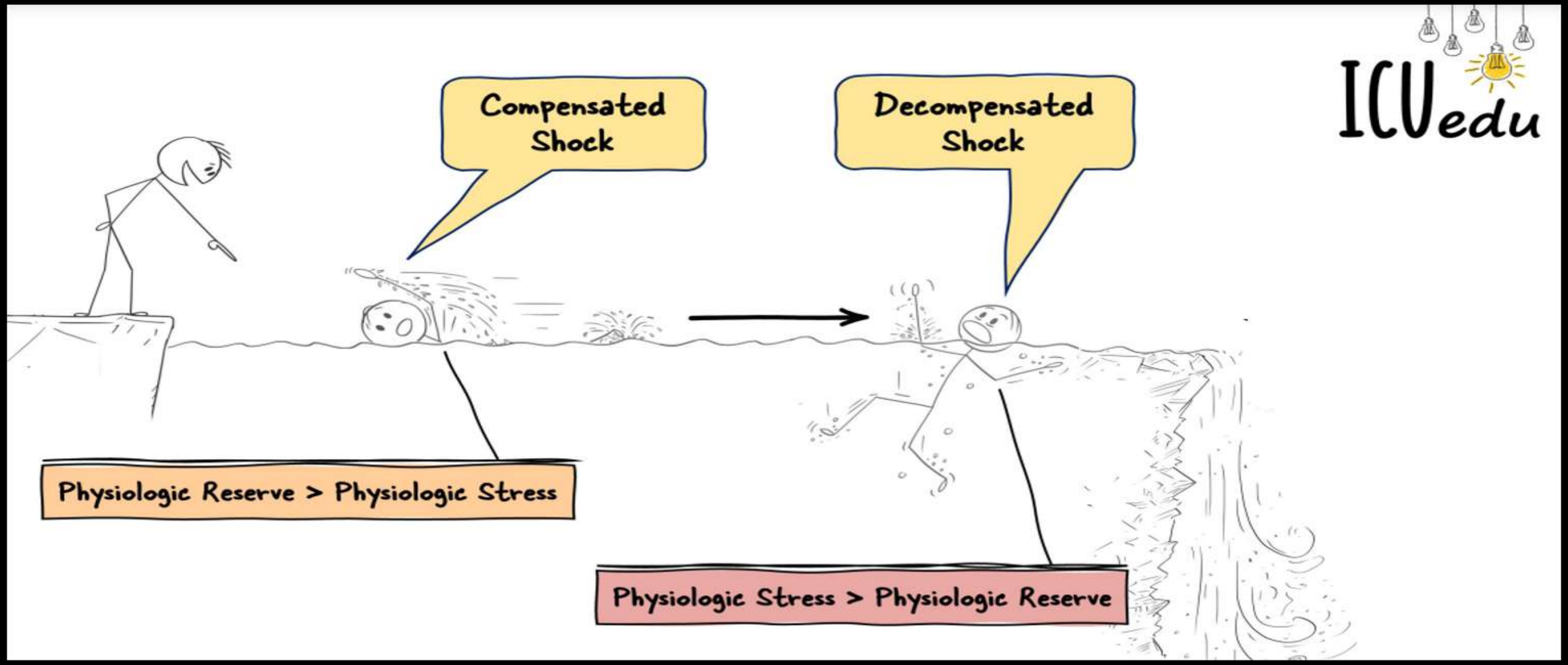
P2 PROLAPSE PATIENT

EF >70% LVIDd 35mm LVEDV 60ml SV 34ml



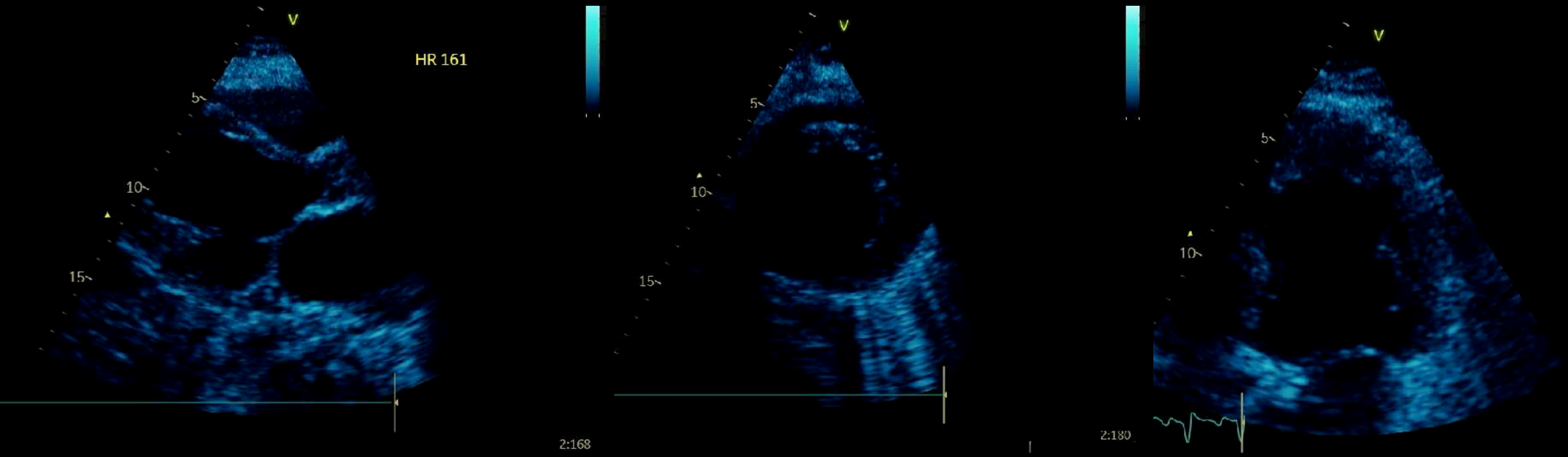
Take home message 1: Don’t take EF in isolation

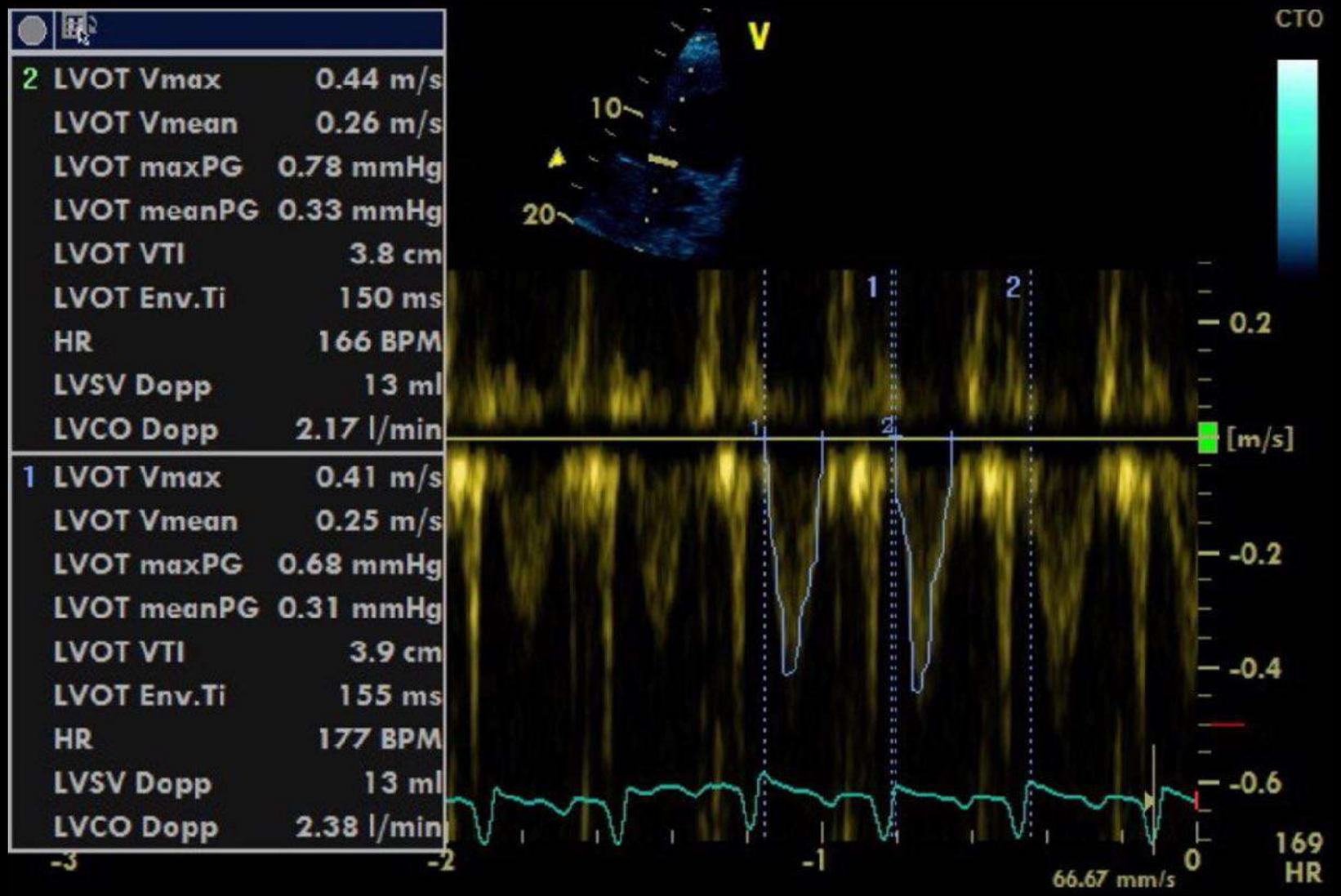
3rd cardiogenic shock pt



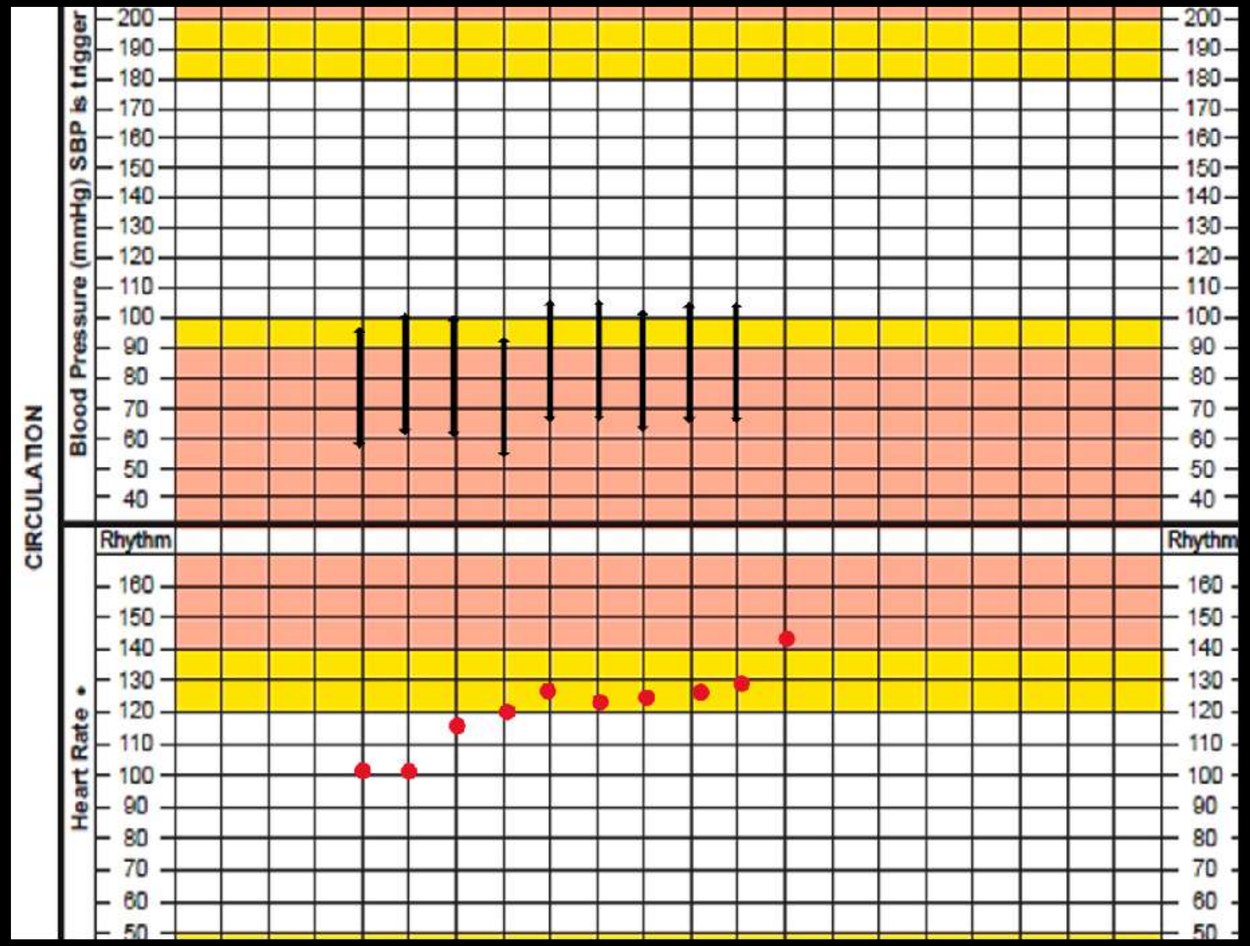
Take home message 2: Identify cardiogenic shock early & call it ‘cardiogenic shock’

30 yo in peripheral hospital, In ED with SOB after recent viral infection




2 LVOT Vmax 0.44 m/s
LVOT Vmean 0.26 m/s
LVOT maxPG 0.78 mmHg
LVOT meanPG 0.33 mmHg
LVOT VTI 3.8 cm
LVOT Env.Ti 150 ms
HR 166 BPM
LVSV Dopp 13 ml
LVCO Dopp 2.17 l/min
1 LVOT Vmax 0.41 m/s
LVOT Vmean 0.25 m/s
LVOT maxPG 0.68 mmHg
LVOT meanPG 0.31 mmHg
LVOT VTI 3.9 cm
LVOT Env.Ti 155 ms
HR 177 BPM
LVSV Dopp 13 ml
LVCO Dopp 2.38 l/min
V
10
20
CTO
1
2
0.2
[m/s]
-0.2
-0.4
-0.6
-3
-2
-1
0
66.67 mm/s
169
HR

3rd cardiogenic shock pt



‘Normotensive’

n.b Shock Index
= HR/SBP
> 1 = worry

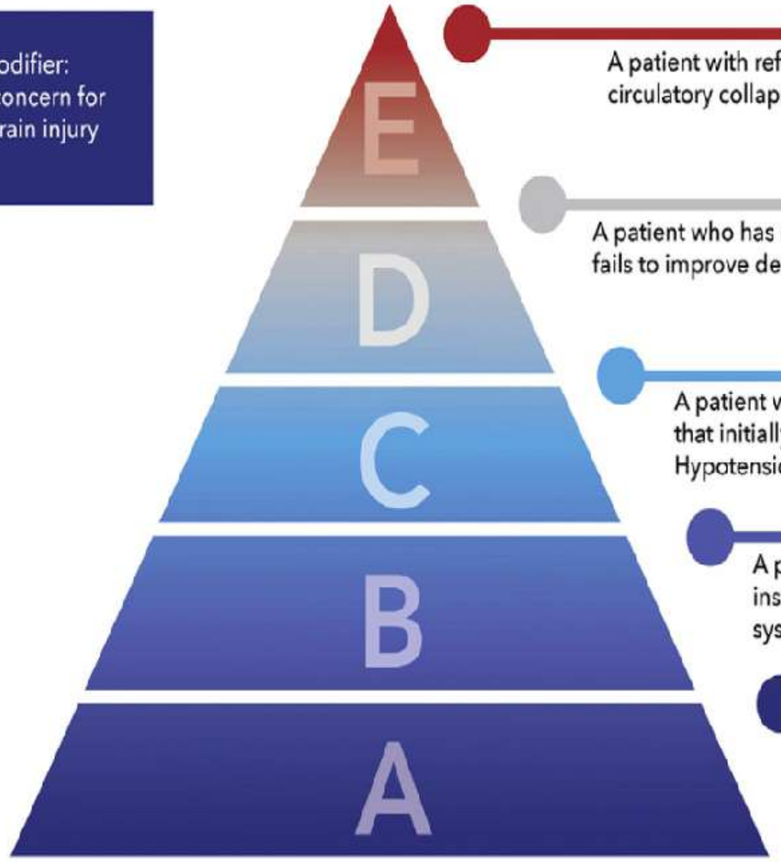
Wei et al. The value of shock index in predicting cardiogenic shock in patients undergoing PCI. BMC Cardiovascular Disorders (2018) 18:188

SCAI Stages of Cardiogenic Shock

SCAI
Society for Cardiovascular Angiography & Interventions

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

(A) Modifier:
CA with concern for anoxic brain injury



EXTREMIS

A patient with refractory shock or actual/impending circulatory collapse.

DETERIORATING

A patient who has clinical evidence of shock that worsens or fails to improve despite escalation of therapy.

CLASSIC

A patient who has clinical evidence of hypoperfusion that initially requires pharmacologic or mechanical support. Hypotension is usually present.

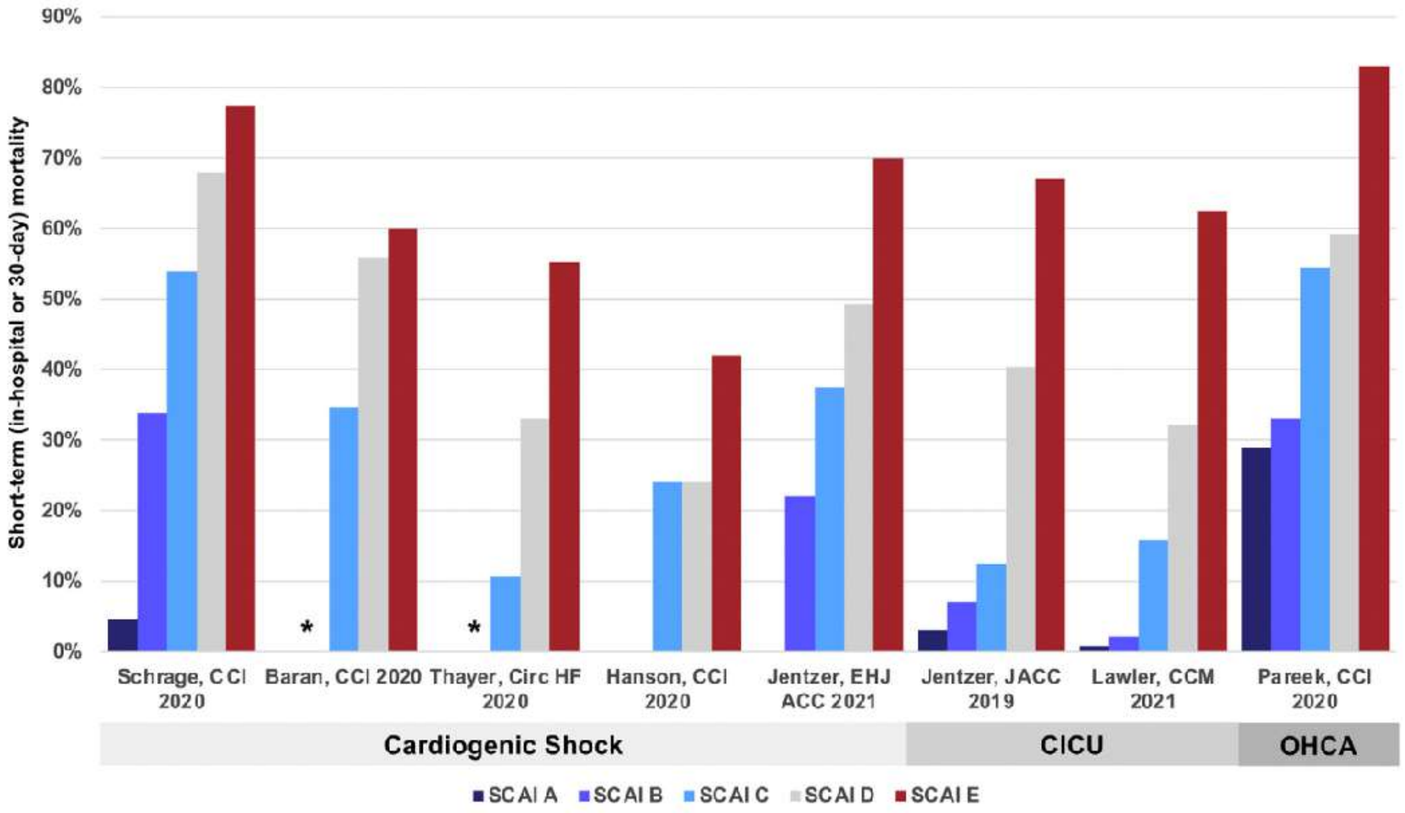
BEGINNING

A patient who has clinical evidence of hemodynamic instability (including hypotension, tachycardia or abnormal systemic hemodynamics) without hypoperfusion.

AT RISK

A hemodynamically stable patient who is NOT experiencing signs or symptoms of CS, but is at risk for its development (i.e. large AMI or decompensated HF).

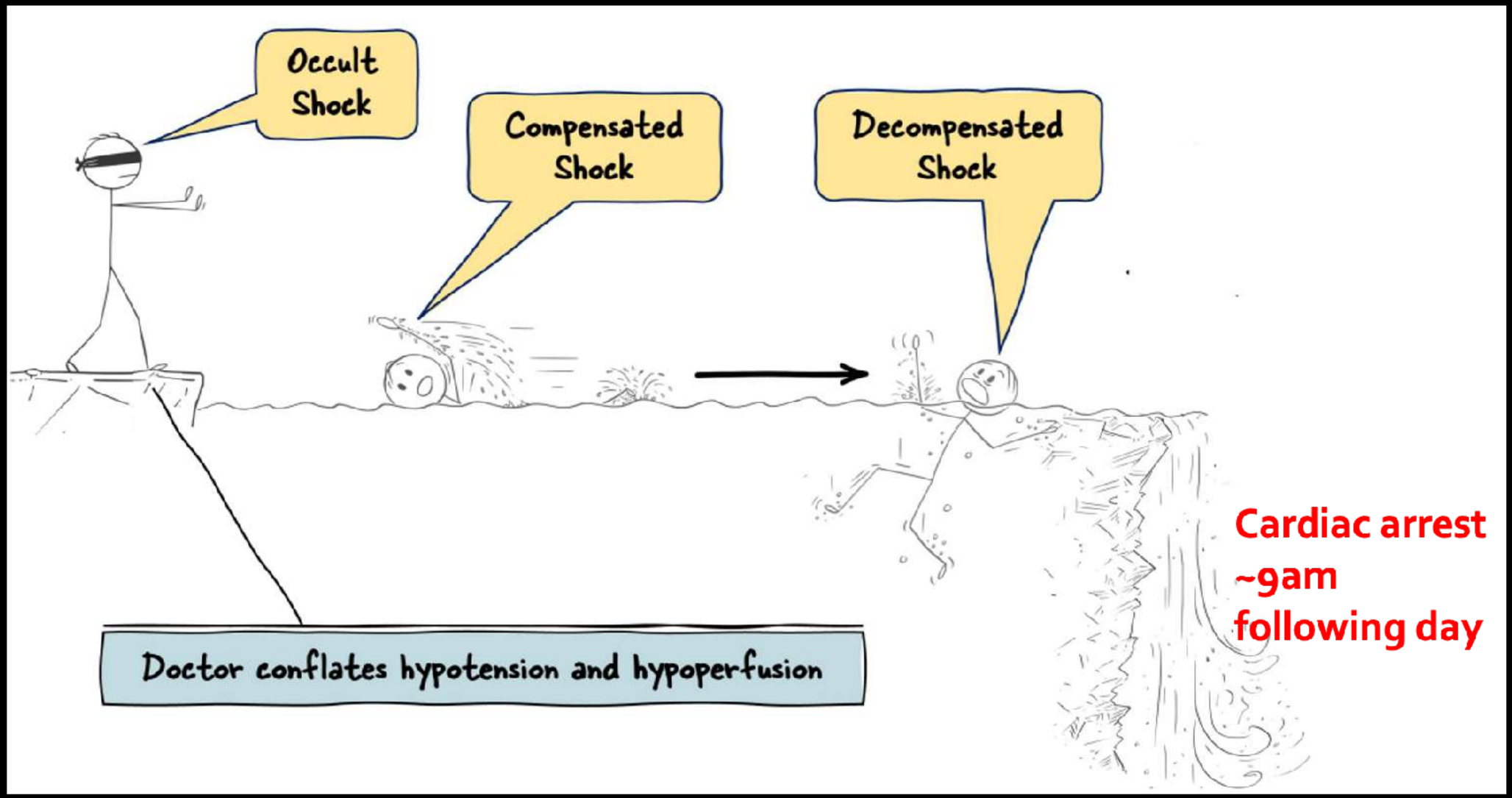
FIGURE 2 Short-Term Mortality as a Function of SCAI SHOCK Stages in Each Study

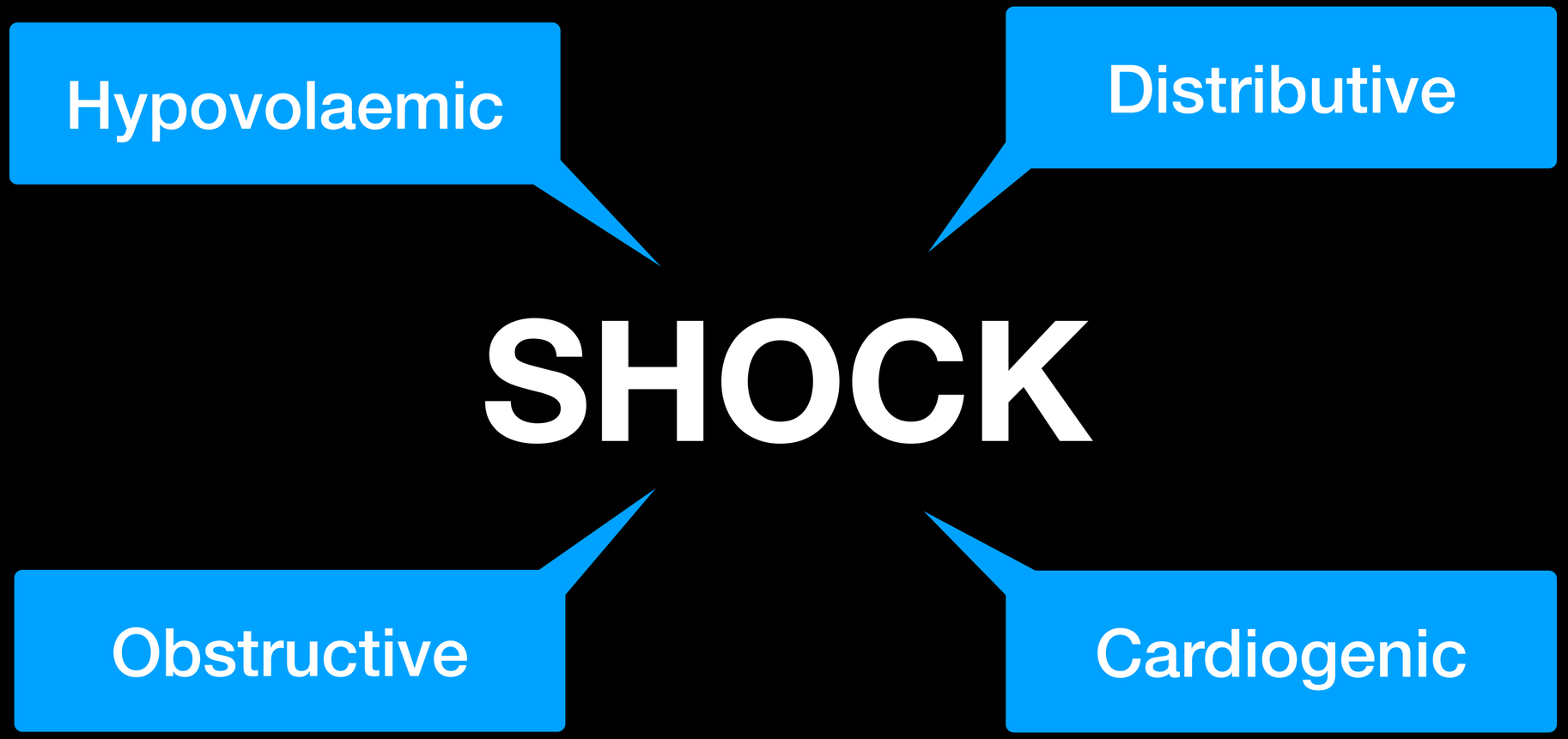


*denotes that no deaths were observed in patients with SCAI stage B in these studies. CICU = cardiac intensive care unit; OHCA = out-of-hospital cardiac arrest; SCAI = Society for Cardiovascular Angiography and Interventions.

Naidu et al, SCAI shock expert consensus update. JACC 2022

6 hours later = hypotension started, lactate raised




Hypovolaemic
Distributive
SHOCK
Obstructive
Cardiogenic

60yo man PMH HOCM

PC: Day 2 in hospital with shortness of breath

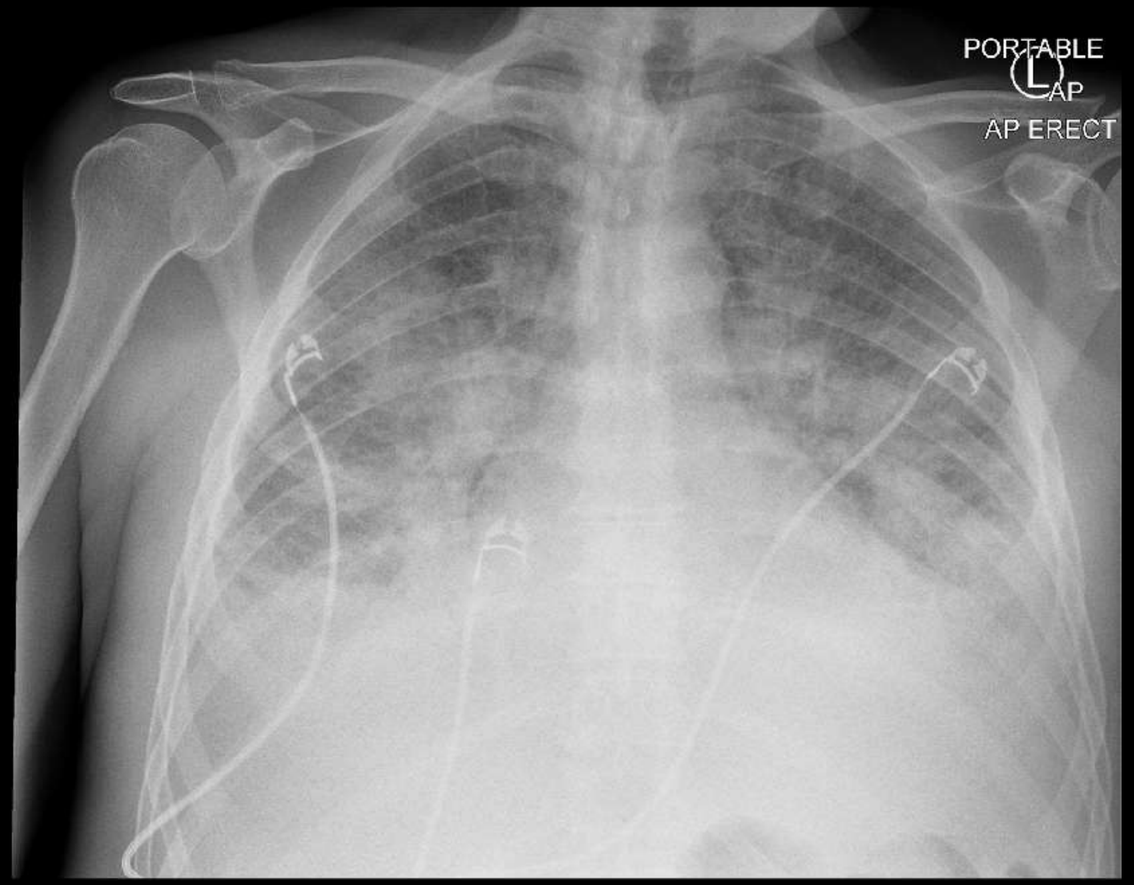
Imp = Acute heart failure => APO

Tmt = Frusemide (180mg in first 12hrs), GTN patch

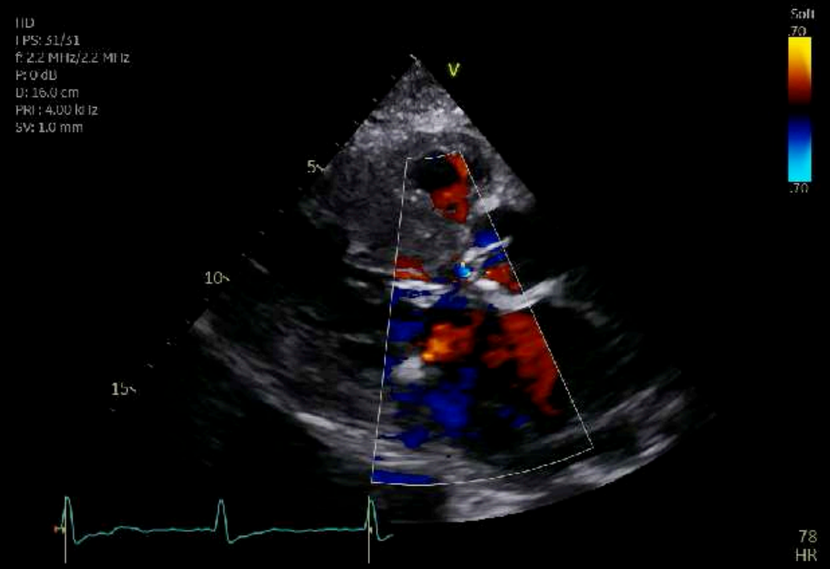
Midnight = MET call ward for hypoxia and hypotension

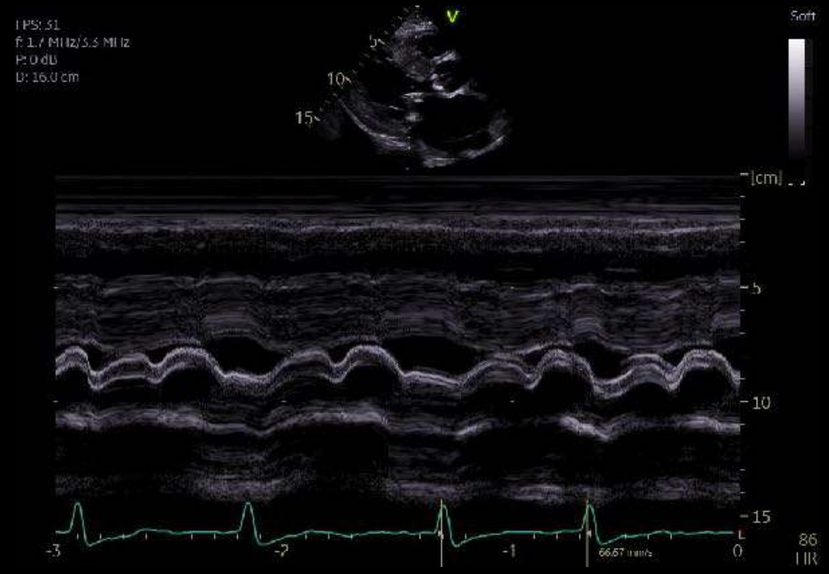
Gm positive cocci in chains in blood (Strep)

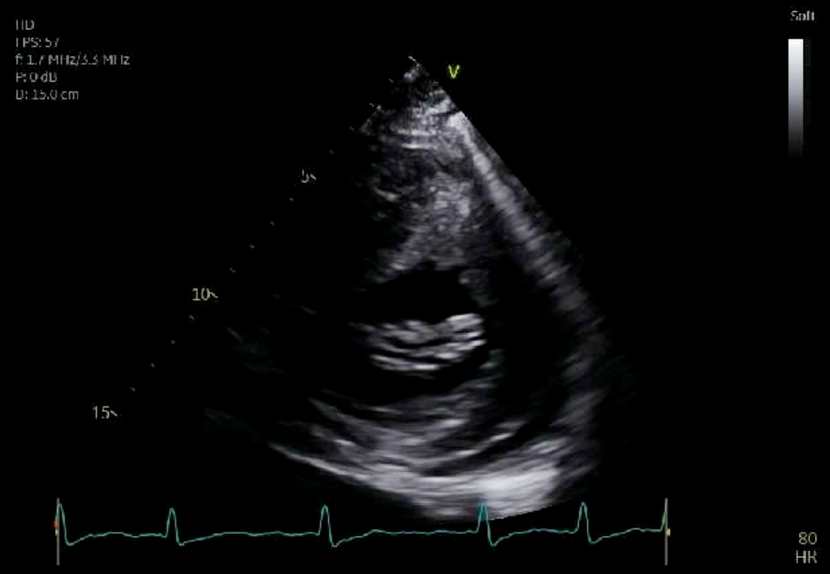
Rapid escalation in catecholamines



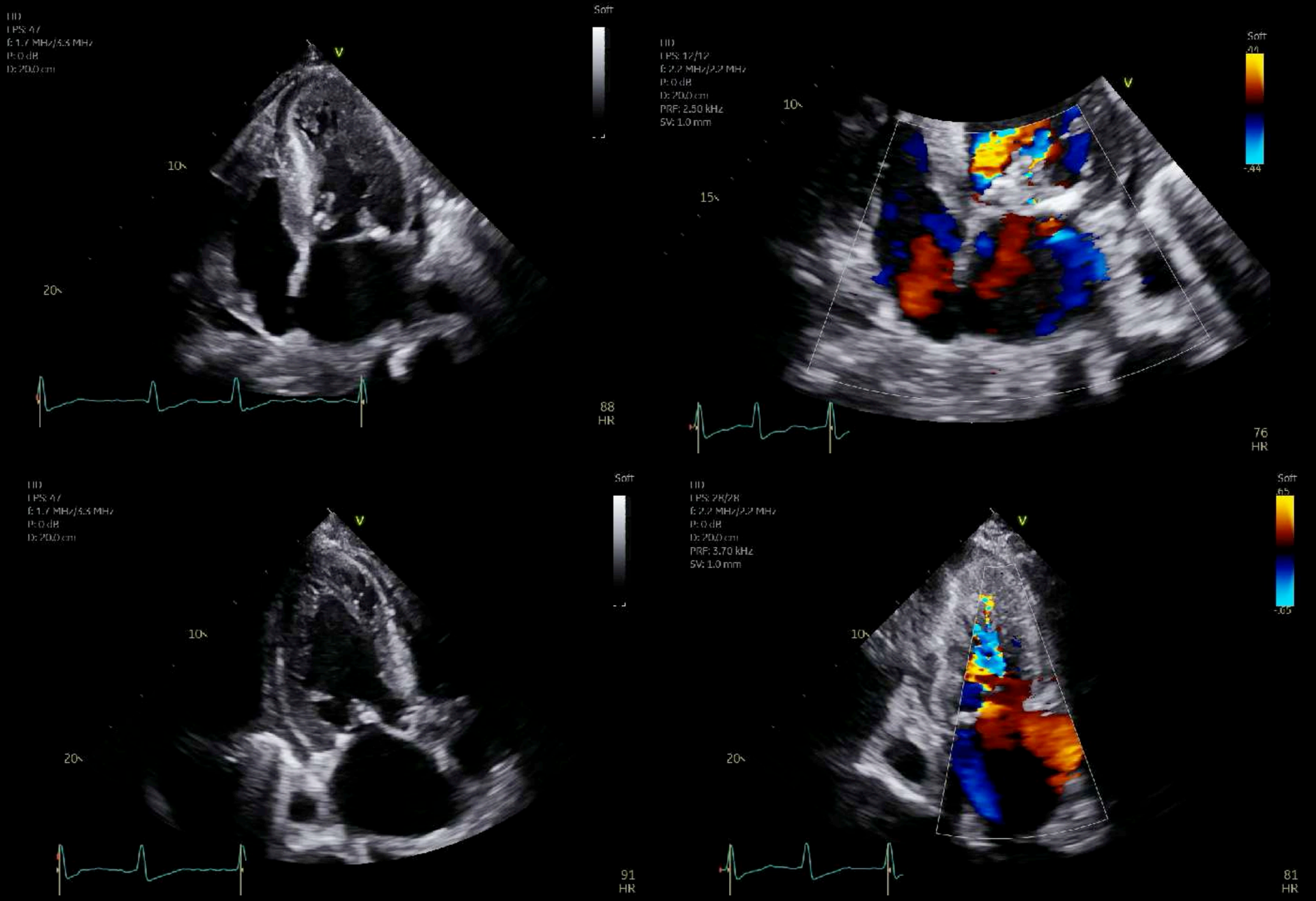
Rapid escalation in catecholamines

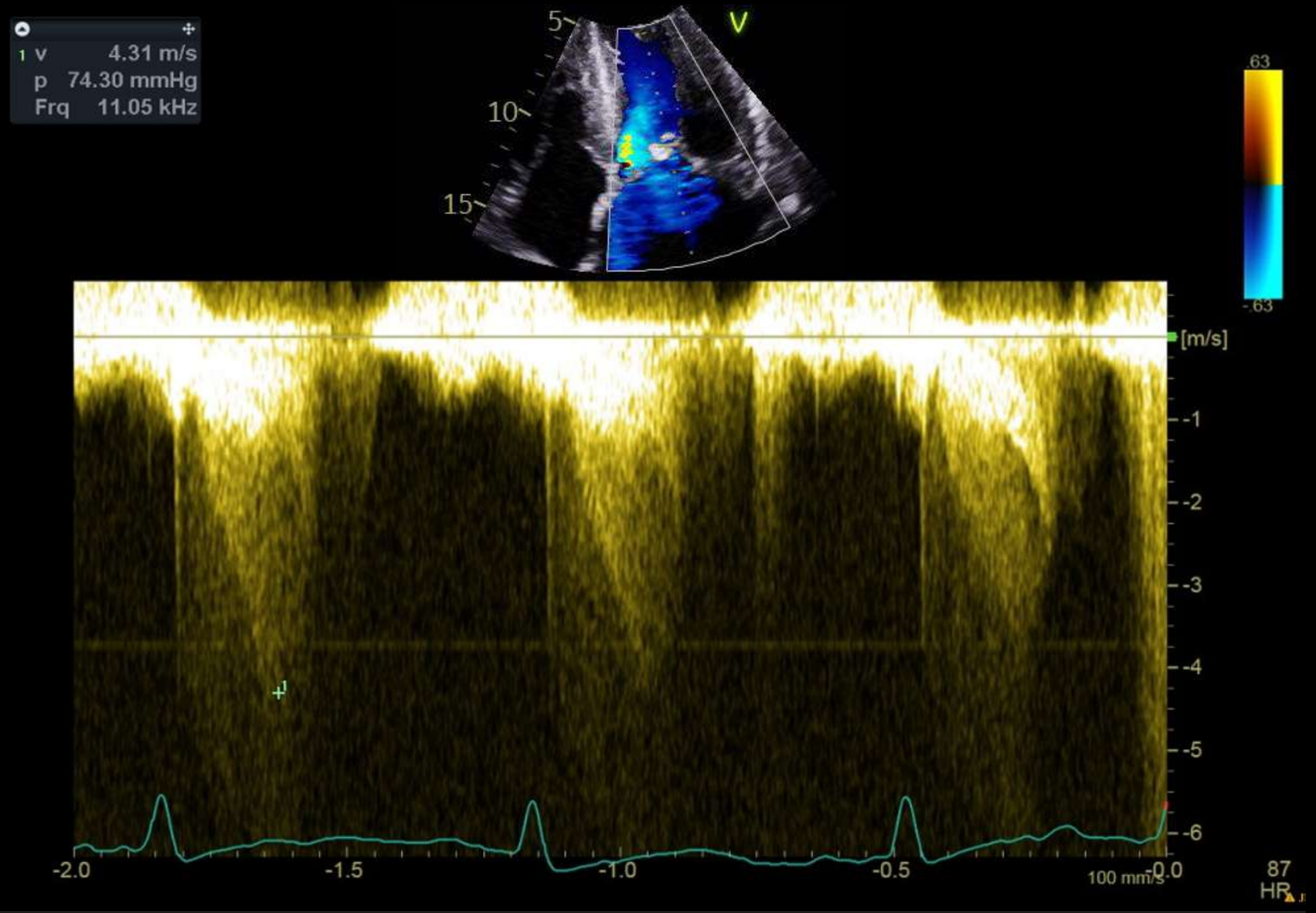




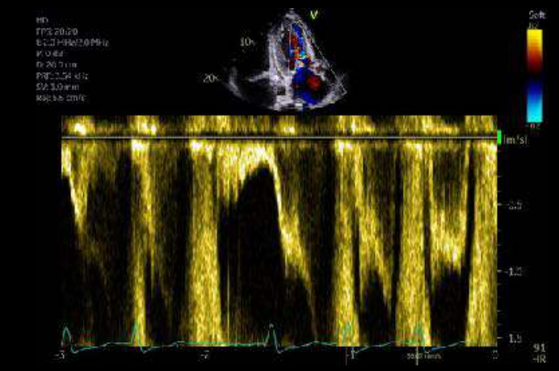


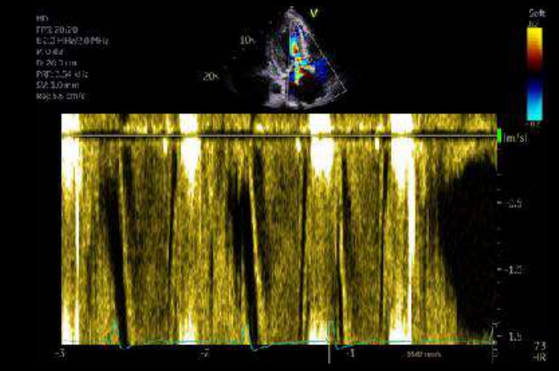
ECHO AUSTRALIA

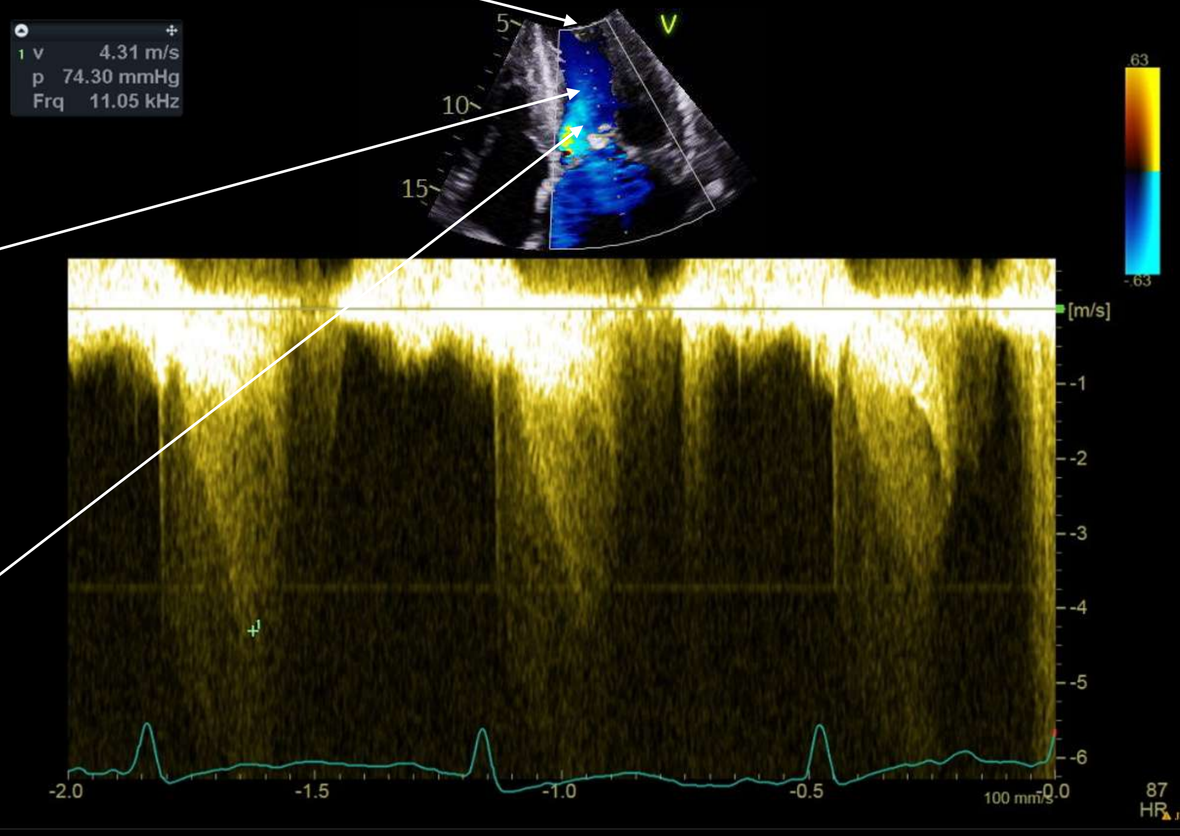




Left ventricle outflow obstruction (LVOT obstruction

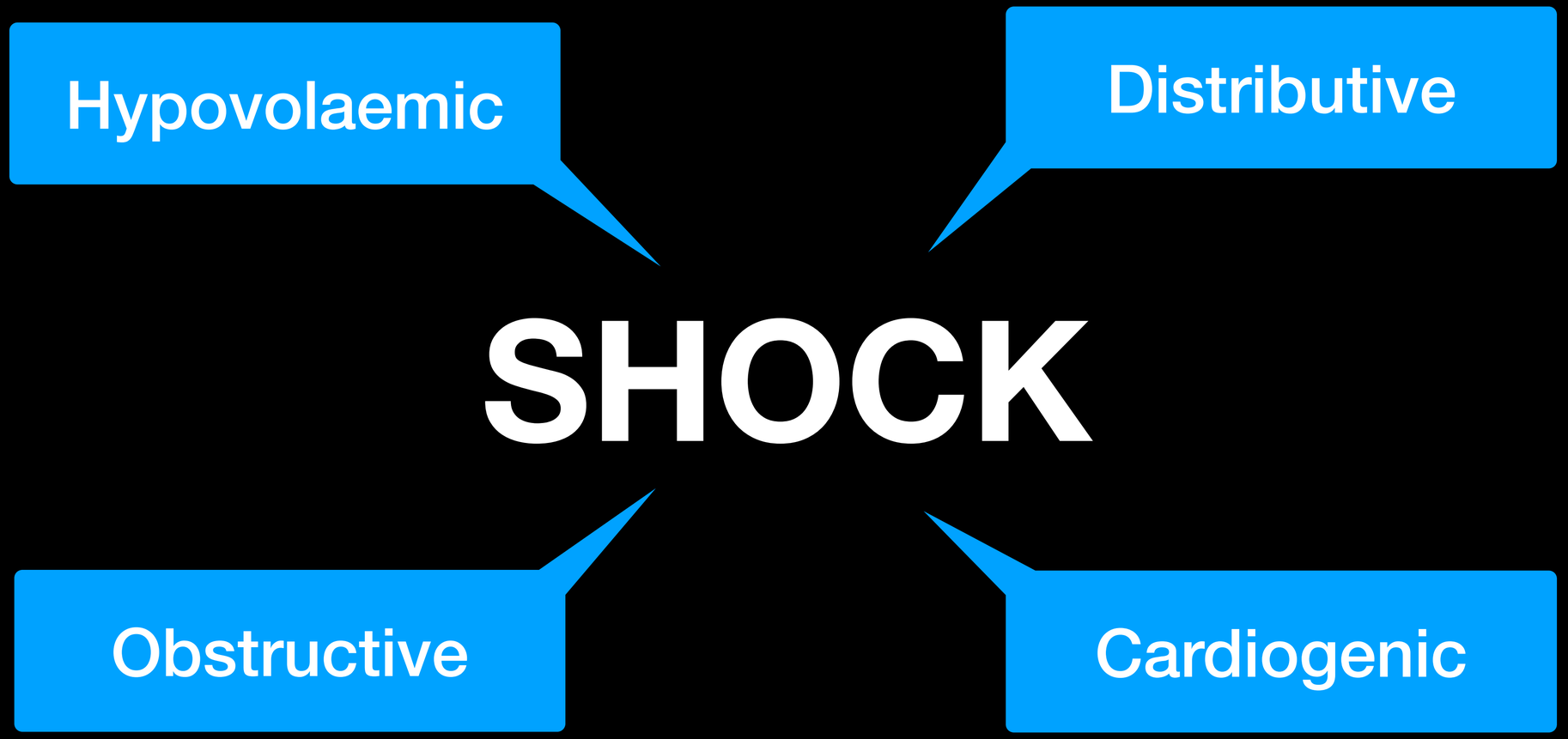






Treatment

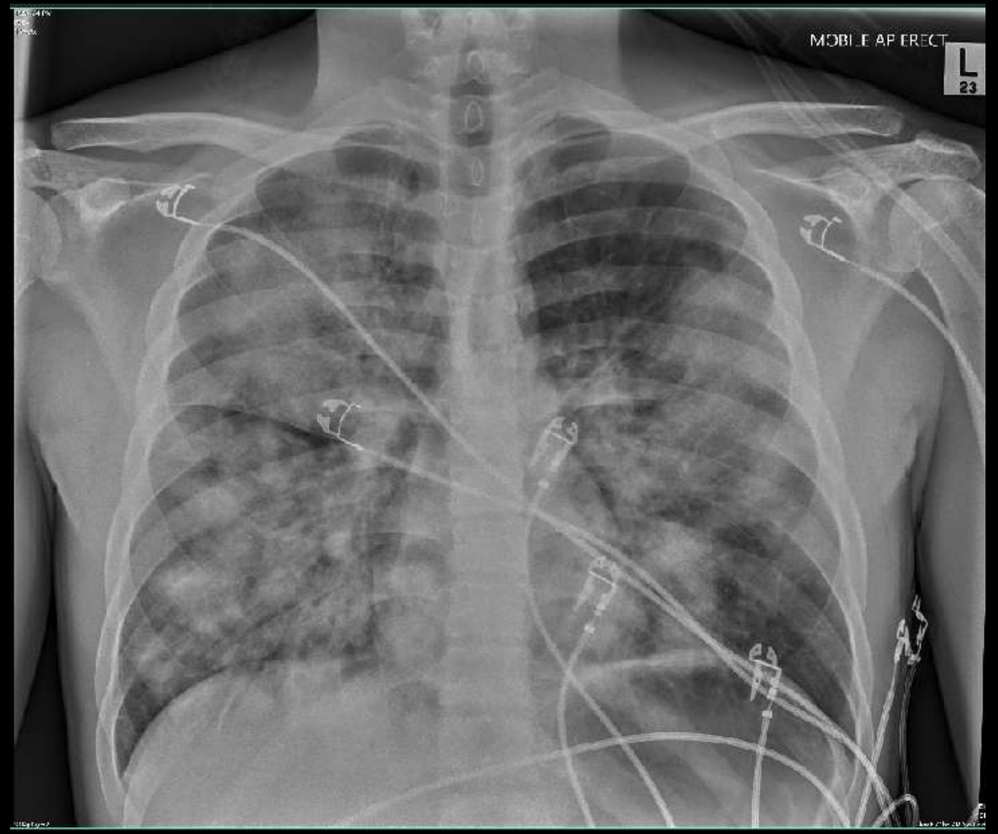
- Stop increasing Noradrenaline
- Fluids ++
- Amiodarone
- Vasopressin
- (avoid b agonists)


Hypovolaemic
Distributive
SHOCK
Obstructive
Cardiogenic

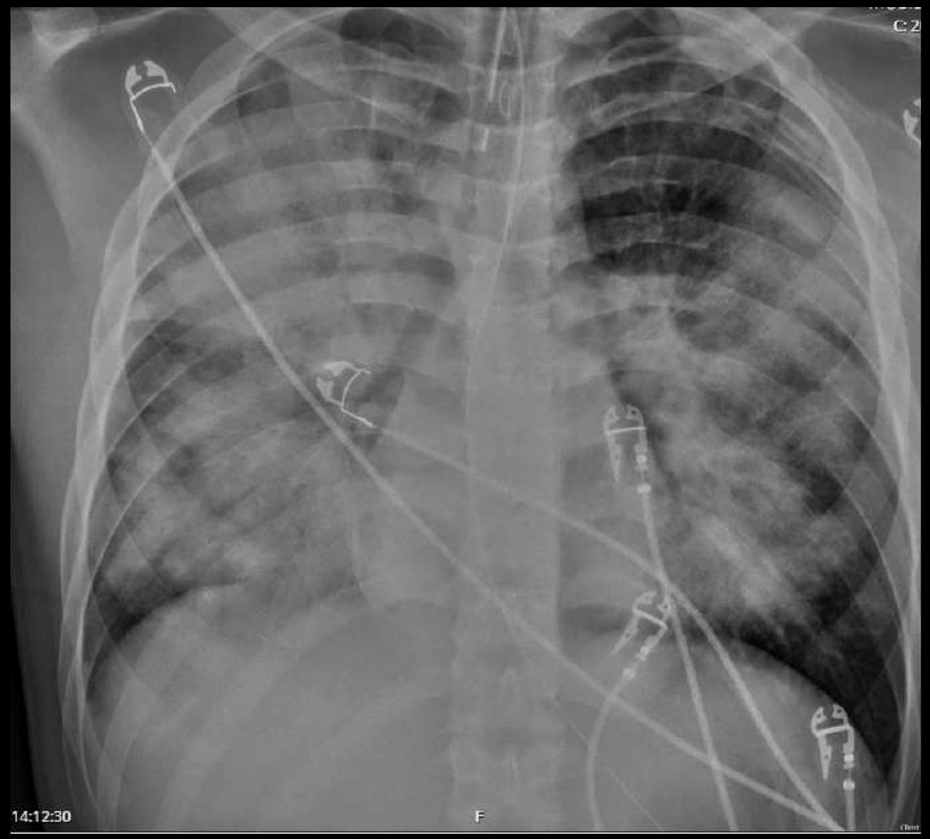
16yo male, unwell 48hrs, No PMH

- Cough
- Vomiting & diarrhoea

ED = HR 128, RR 50,
Sats 87%, SBP 105/-

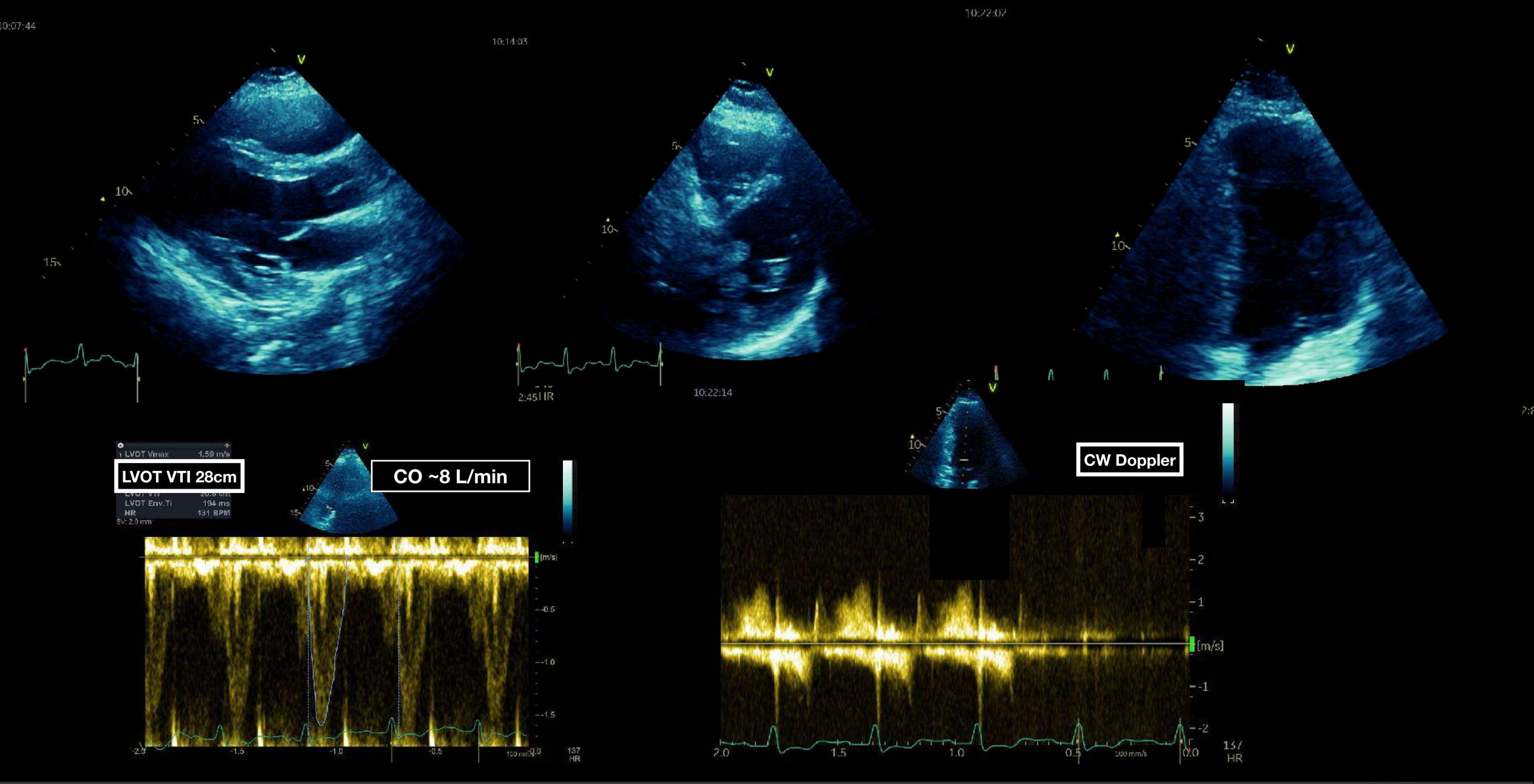


	12:32
Instrument ID	D210EMERGM01
Specimen Type PoCT	Venous
Patient Temperature	37.0 DegC
FiO2 POCT	
pH POCT	7.13 C
pH POCT Corrected	7.13 C
PO2 POCT	30 mmHg
PO2 POCT Corrected	30 mmHg
PCO2 POCT	54.0 mmHg H
PCO2 POCT Corrected	54.0 mmHg H
sO2 POCT	41 %
HCO3 POCT	18 mmol/L L
Base Excess POCT	-11.8 mmol/L C
Sodium POCT	130 mmol/L L
Potassium POCT	3.6 mmol/L
Chloride POCT	93 mmol/L L
Glucose POCT	6.4 mmol/L H
Lactate POCT	11.1 mmol/L C
Ionised Calcium POCT	1.07 mmol/L L
Blood Total Haemoglobin	173 g/L H
Blood Oxyhaemoglobin	39.9 %
Blood Carboxyhaemoglobin	1.1 %
Blood Methaemoglobin	0.9 %

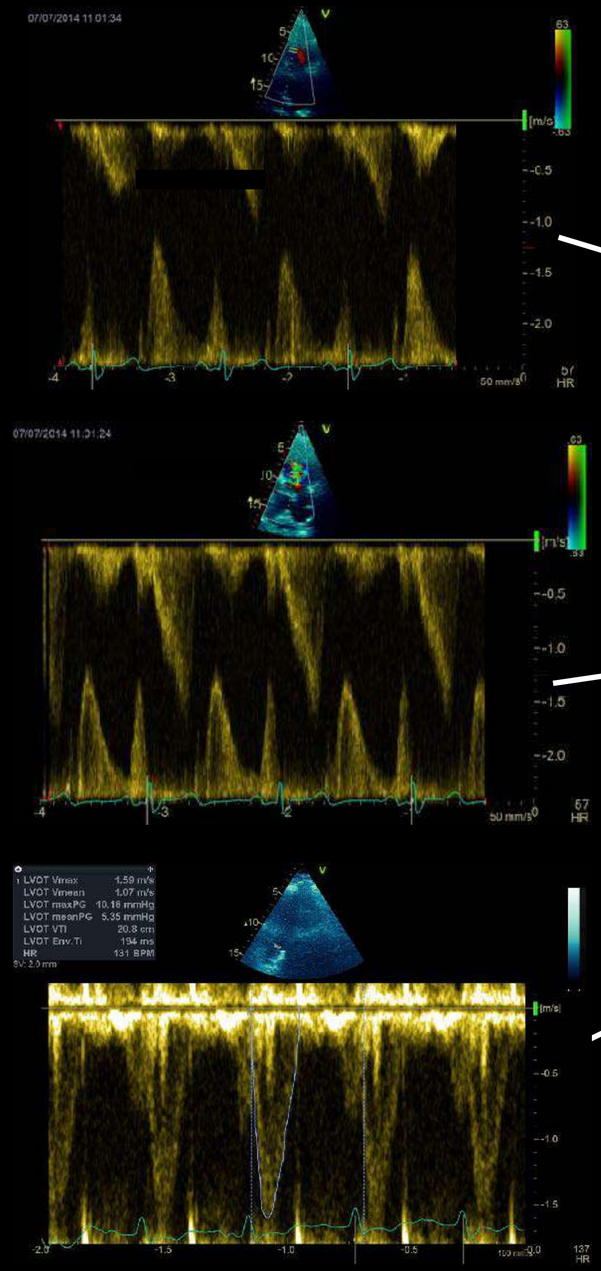


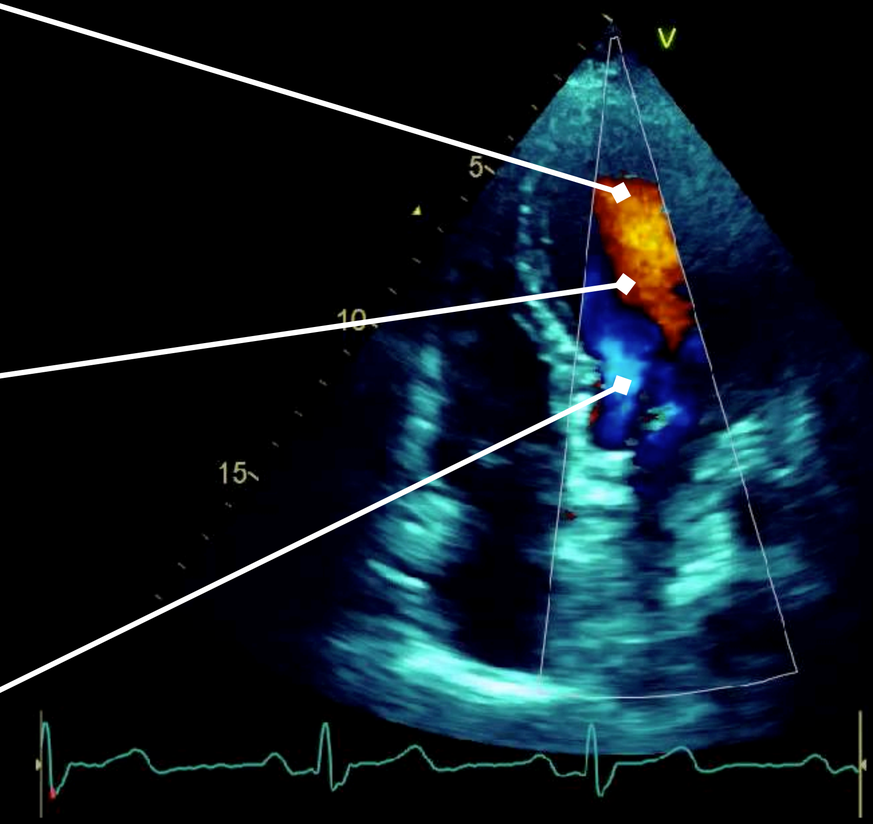
	14:04	13:47	13:16
Instrument ID	D210EMERGM01	D210EMERGM01	D210EMERGM01
Specimen Type PoCT	Arterial	Arterial	Arterial
Patient Temperature	37.0 DegC	37.0 DegC	37.0 DegC
FiO2 POCT	100.0 %	50.0 %	40.0 %
pH POCT	7.02 C	7.14 C	7.18 C
pH POCT Corrected	7.02 C	7.14 C	7.18 C
PO2 POCT	56 mmHg C	113 mmHg H	61 mmHg L
PO2 POCT Corrected	56 mmHg C	113 mmHg H	61 mmHg L
PCO2 POCT	64.0 mmHg C	41.0 mmHg	41.0 mmHg
PCO2 POCT Corrected	64.0 mmHg C	41.0 mmHg	41.0 mmHg
sO2 POCT	80 % L	99 %	90 % L
HCO3 POCT	16 mmol/L L	14 mmol/L L	15 mmol/L L
Base Excess POCT	-15.7 mmol/L C	-14.6 mmol/L C	-12.6 mmol/L C
Sodium POCT	131 mmol/L L	131 mmol/L L	130 mmol/L L
Potassium POCT	4.0 mmol/L	3.3 mmol/L	3.5 mmol/L
Chloride POCT	98 mmol/L	100 mmol/L	98 mmol/L
Glucose POCT	5.3 mmol/L	5.4 mmol/L	5.5 mmol/L H
Lactate POCT	8.4 mmol/L C	7.9 mmol/L C	8.8 mmol/L C
Ionised Calcium POCT	1.01 mmol/L L	0.99 mmol/L L	1.00 mmol/L L
Blood Total Haemoglobin	175 g/L H	171 g/L H	166 g/L
Blood Oxyhaemoglobin	78.6 % L	96.4 %	87.2 % L
Blood Carboxyhaemoglobin	1.5 %	1.6 %	2.5 % H
Blood Methaemoglobin	0.9 %	1.1 %	0.3 % L

2.5L administered already in ED. Concern about fluid overload with ARDS pattern on CXR


LVOT VTI 28cm
CO ~8 L/min
CW Doppler
LVOT Vmax 1.59 m/s
LVOT Env.Ti 194 ms
HR 131 BPM
[m/s]
HR

Left Ventricle Obstruction (LVO)





Treatment

- Likely can tolerate more fluids

218 patients
Septic shock
Echo on admission
French ICU
Looking for LV obstruction

Chauvet et al. Critical Care (2015) 19:262
DOI 10.1186/s13054-015-0980-z

CRITICAL CARE

RESEARCH Open Access

Early dynamic left intraventricular obstruction is associated with hypovolemia and high mortality in septic shock patients

Jean-Louis Chauvet[1], Shari El-Dash[2,3], Olivier Delastre[1], Bernard Bouffandeau[1], Dominique Jusserand[1], Jean-Baptiste Michot[1], Fabrice Bauer[4], Julien Maizel[2,5] and Michel Slama[2,5*]

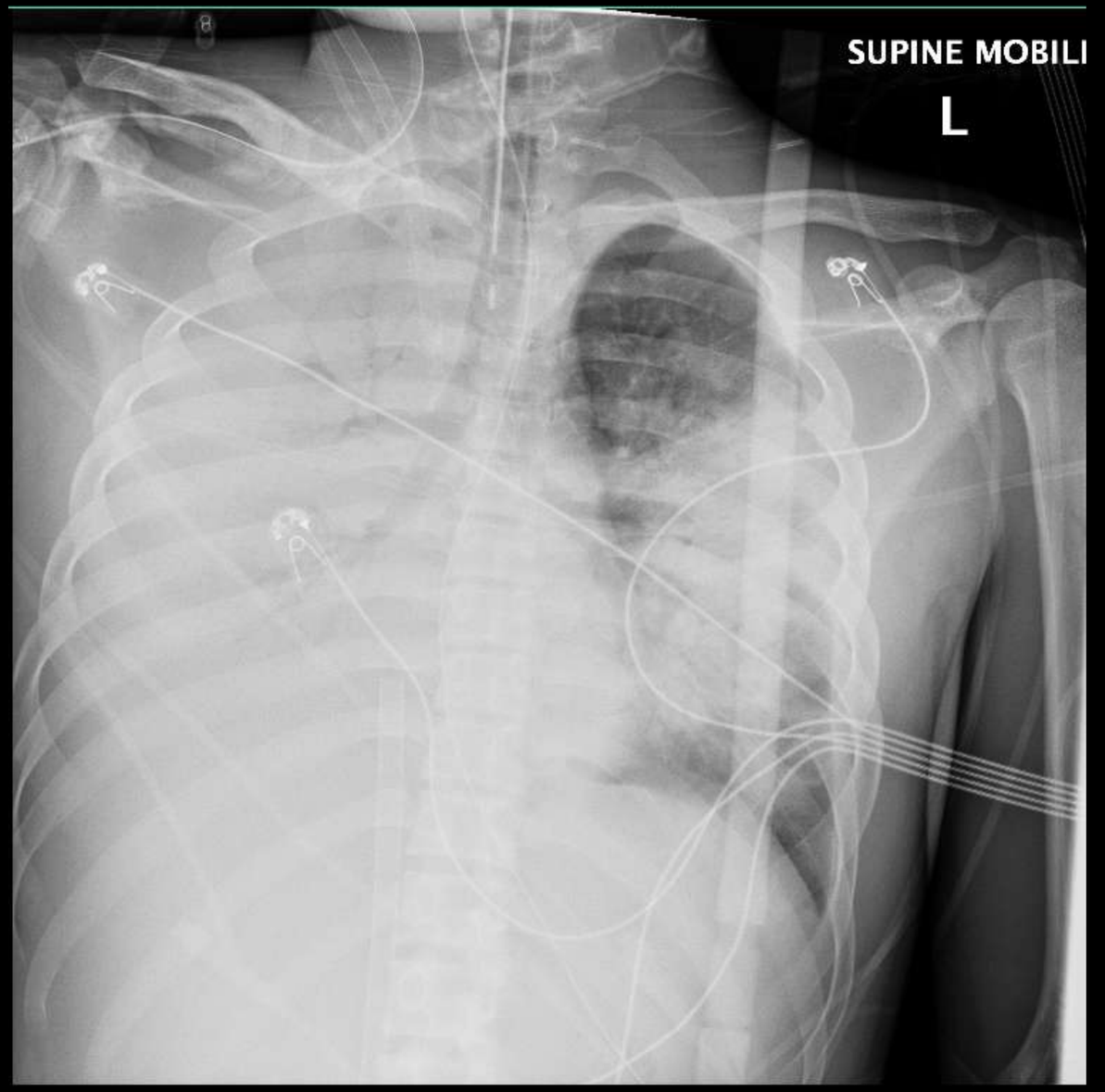
Abstract

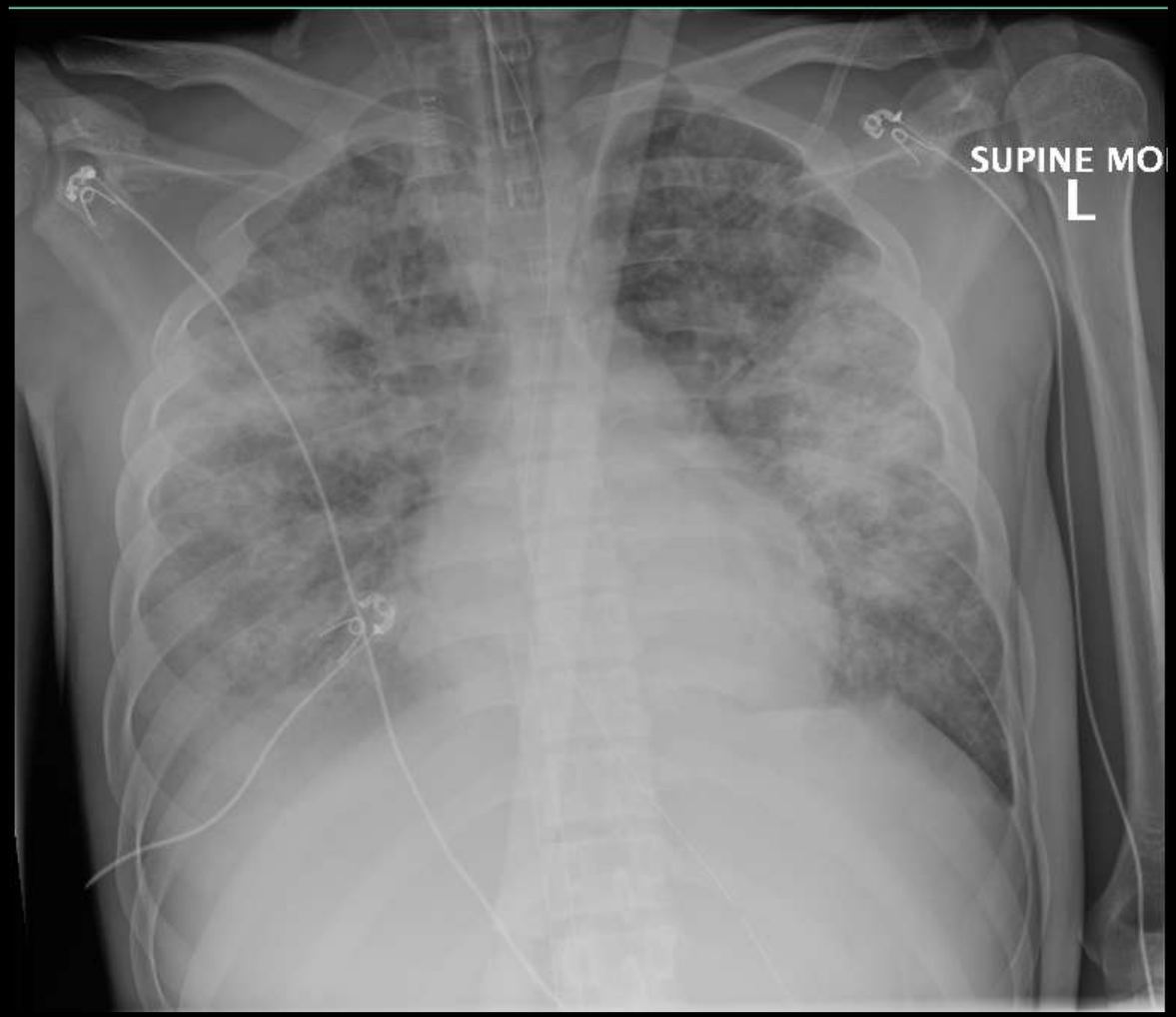
Introduction: Based on previously published case reports demonstrating dynamic left intraventricular obstruction (IVO) triggered by hypovolemia or catecholamines, this study aimed to establish: (1) IVO occurrence in septic shock

Results: During the study period, 218 patients with septic shock were admitted to our ICU. IVO was observed in 47 (22 %) patients. Mortality rate at 28 days was found to be higher in patients with than in patients without IVO (55 % versus 33 %, $p < 0.01$). Small, hypercontractile left ventricles (end-diastolic left ventricular surface 4.7 ± 2.1 cm^2/m^2 and ejection fraction 82 ± 12 %), and frequent pseudohypertrophy were found in these patients. A rise ≥12 % in stroke index was found in 87 % of patients with IVO, with a drop of 47 % in IVO after fluid infusion.

Conclusion: Left IVO is a frequent event in septic shock patients with an important correlation with fluid responsiveness. The mortality rate was found to be higher in these patients in comparison with patients without obstruction.

Name	15	16	17	18	19	20	21	22	23	Total
Noradrenaline (Norepinephrine) 8 mg in 100 ml 5% G	54	57	77	110	140	100	47	25	37	647
Fentanyl 1000 microgram in 50 ml 5% G										0
Cisatracurium 150 mg	5	5	5	5	5	5	5	5	5	45
Plasmalyte 1000 ml	100	100	500	750	2500	250	100	150		4450
Magnesium sulphate 10 mmol/hr for 1 hrs via Maintenance										0
Fentanyl 1000 microgram in 30 ml 5% G		10	6	6	6	6	6	8	10	58
Vasopressin (Argipressin) 40 unit(s) in 40 ml 5% G		2.4	2.4	2.4	2.4	2.4	2.4	2.4	2.4	19.2
Midazolam 100 mg in 100 5% G		10	6	6	6	6	6	10	10	60
Potassium Acetate 15 mmol/hr for 2 hrs via Maintenance										0
Dobutamine 250 mg in 100 ml 5% G			5.2	5.2	5.2	5.2				20.8
Albumin 4% 1000 ml						500				500
Adrenaline (Epinephrine) 8 mg in 100 ml 5% G				10	20	20	30	30	30	140
Fresh frozen plasma (FFP) 2 unit(s)						271	273			544
Protamine sulfate 100 mg in 45 mL 0.9% S							37.5			37.5
Platelet concentrate - (small),(pooled),(irradiated) 1 unit(s)							263			263
Hourly Totals	159	184.4	601.6	894.6	2684.6	1165.6	496.9	230.4	94.4	6784.5



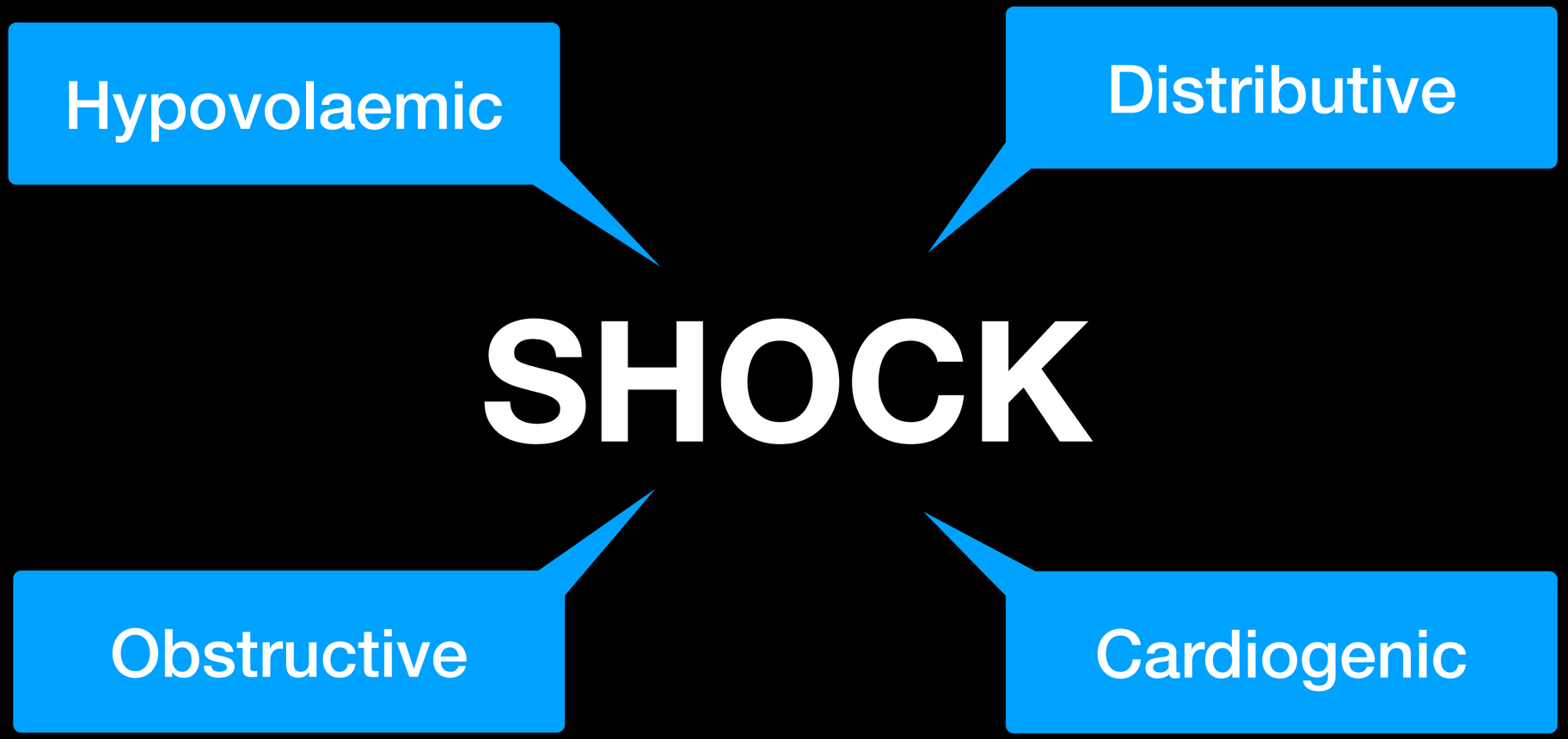


Micro results

- Nepean: hMPV
- SVH: Strep pyogenes

- Over next few days remained on VV ECMO & CRRT; markedly decreased vasoactive dose; tolerating negative fluid balance

- Recovered well.

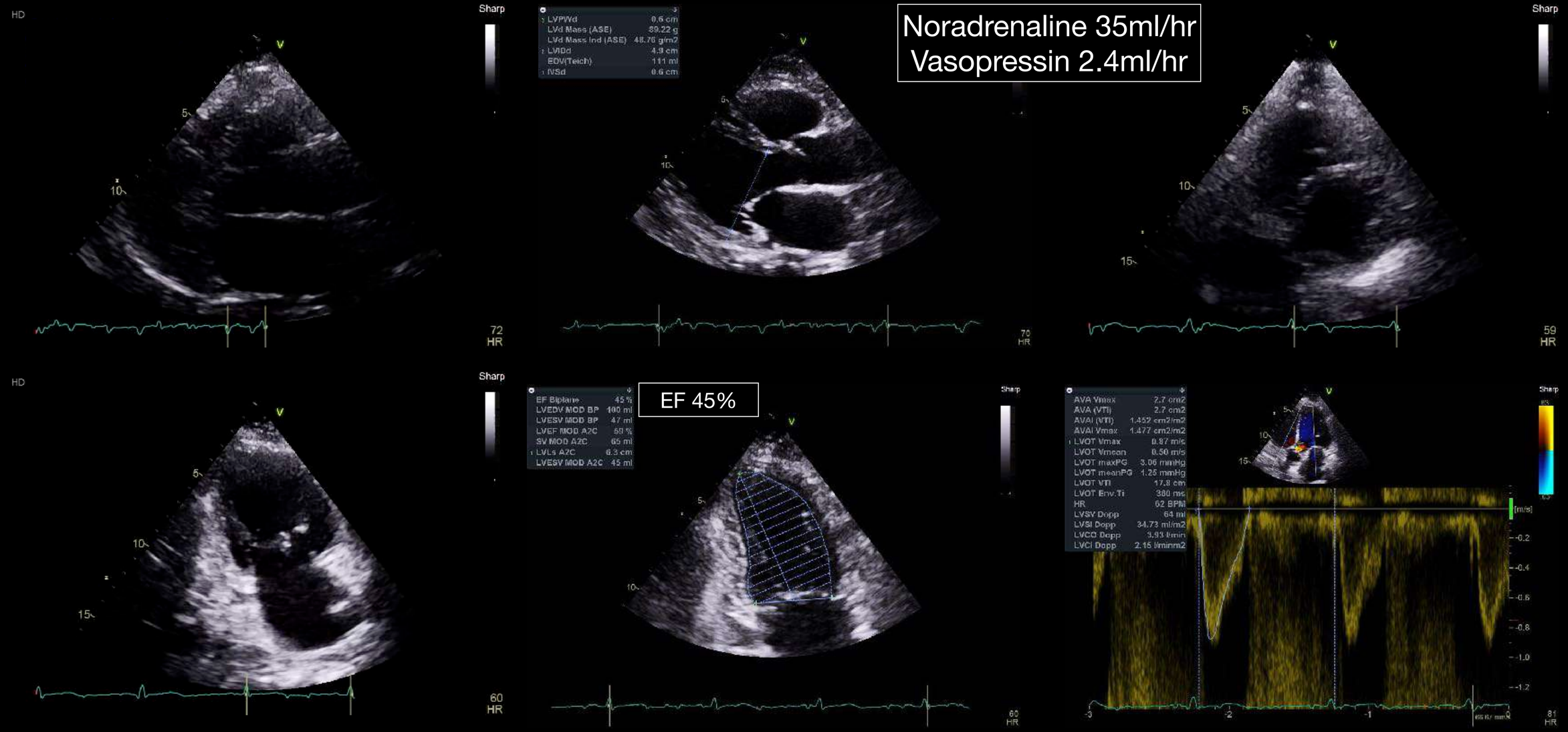

Hypovolaemic
Distributive
SHOCK
Obstructive
Cardiogenic

2nd septic shock patient

62yo female. Septic shock from Grp A Strep bacteraemia. MAP59. Lactate 3

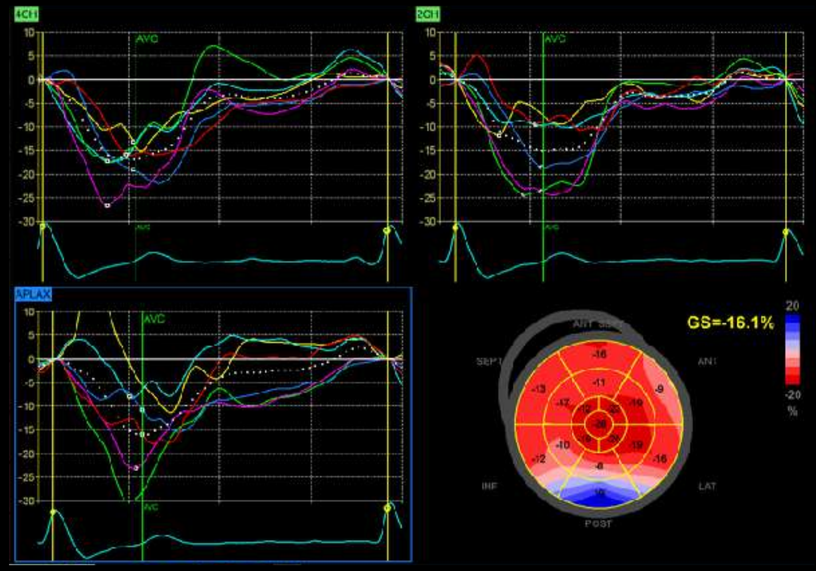
Noradrenaline 35ml/hr
Vasopressin 2.4ml/hr

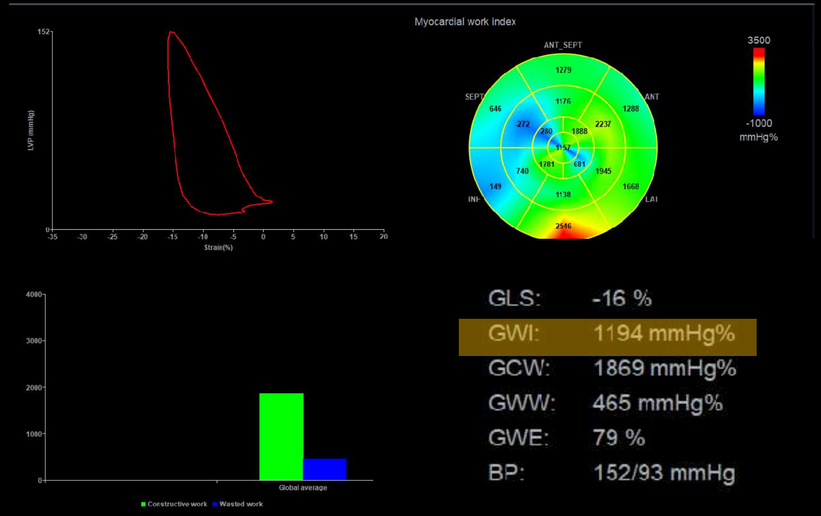
EF 45%



Noradrenaline 35ml/hr
Vasopressin 2.4ml/hr

MAP 59
Lactate 3

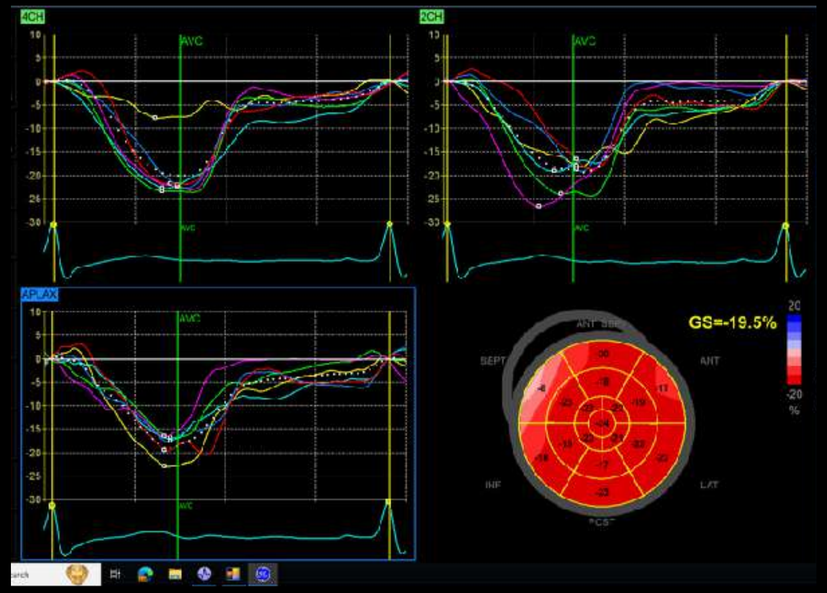


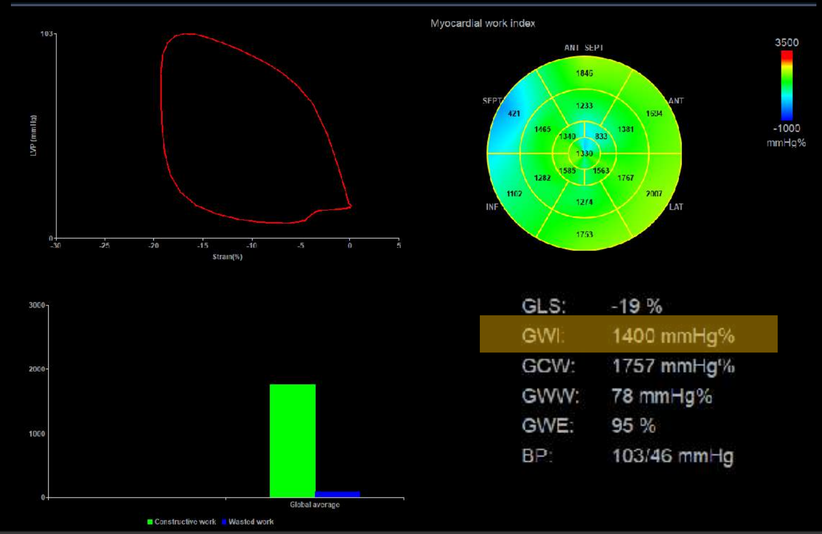


GLS: -16 %
GWI: 1194 mmHg%
GCW: 1869 mmHg%
GWW: 465 mmHg%
GWE: 79 %
BP: 152/93 mmHg

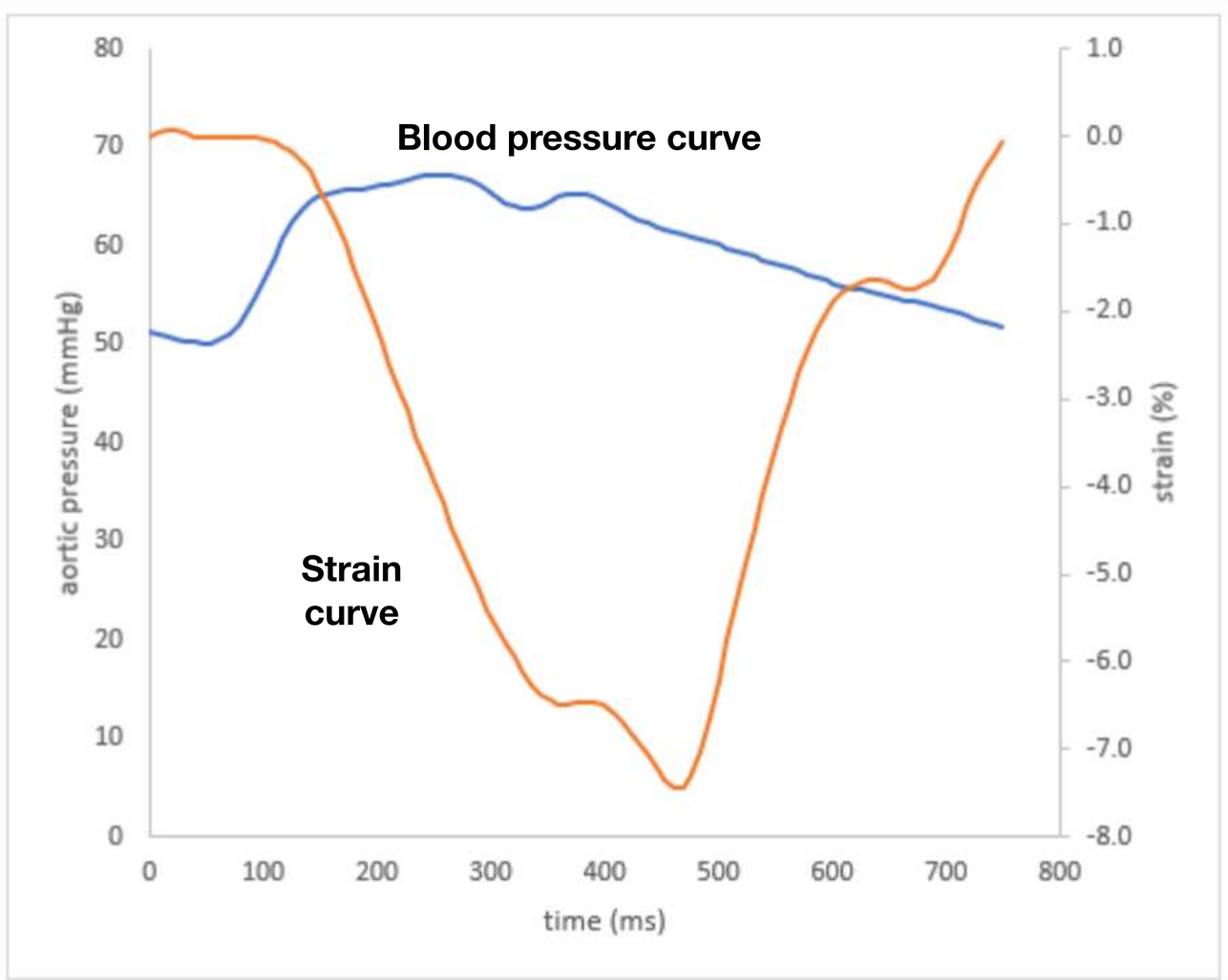
Noradrenaline 15ml/hr
Dobutamine 5mcg/kg/hr

MAP 64
Lactate 2





GLS: -19 %
GWI: 1400 mmHg%
GCW: 1757 mmHg%
GWW: 78 mmHg%
GWE: 95 %
BP: 103/46 mmHg

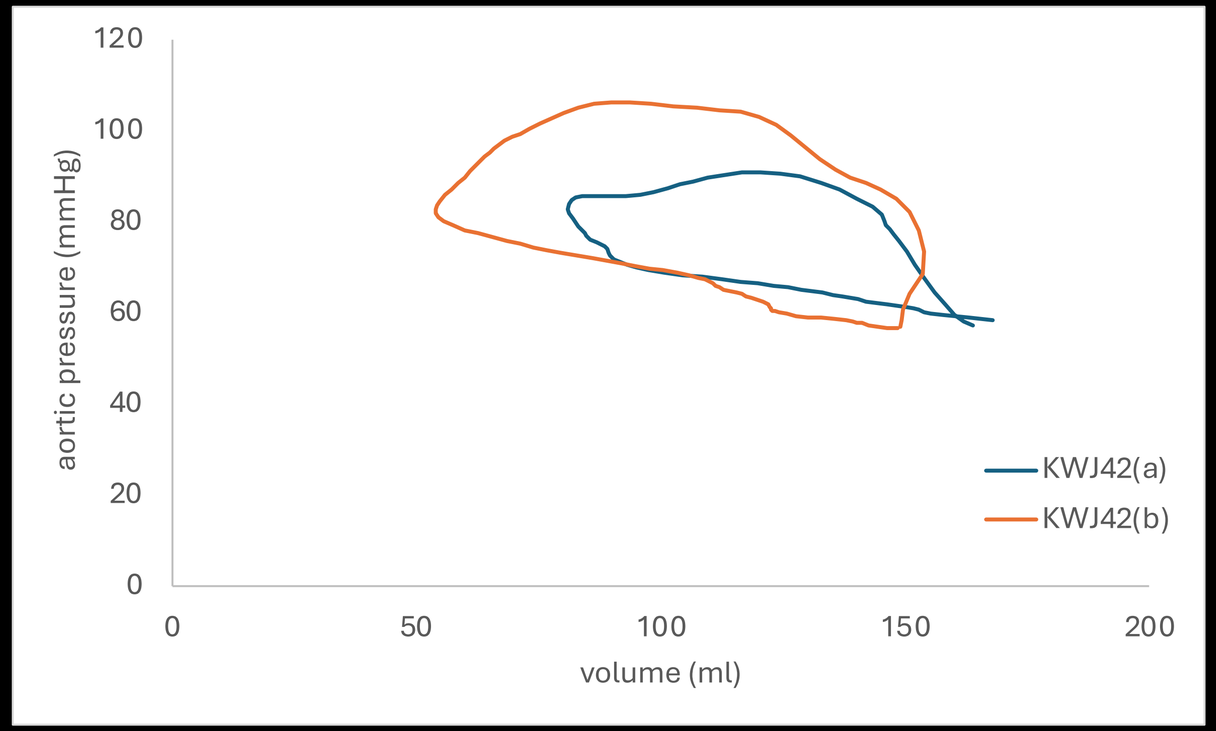


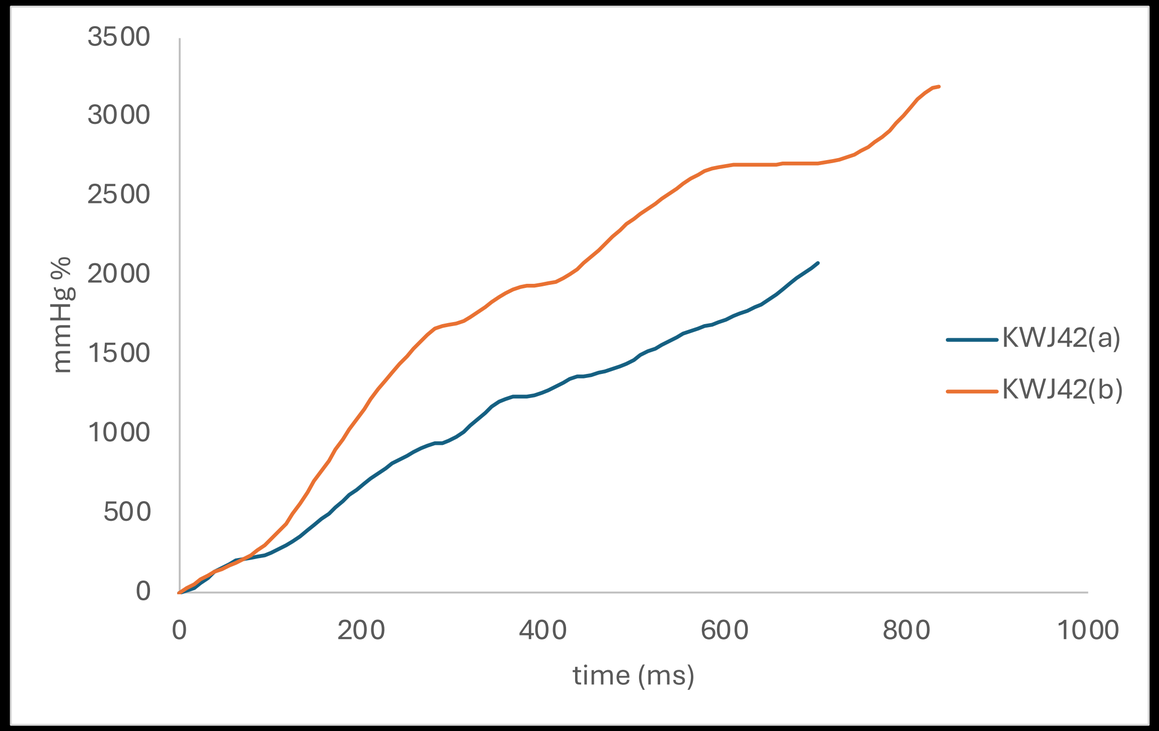
Noradrenaline 35ml/hr
Vasopressin 2.4ml/hr

MAP 59
Lactate 3

Noradrenaline 15ml/hr
Dobutamine 5mcg/kg/hr

MAP 64
Lactate 2





Summary

- LV & haemodynamic assessment key in shock
- Clinical context + echo findings key
- Still more to learn …

Thank you very much for listening

YouTube = Echo at Nepean

sam.orde@health.nsw.gov.au