Master Course in Heart Failure 25 BAKU

Baku Marriott Hotel Boulevard
30th May - 1st June

Advanced echo in shock states

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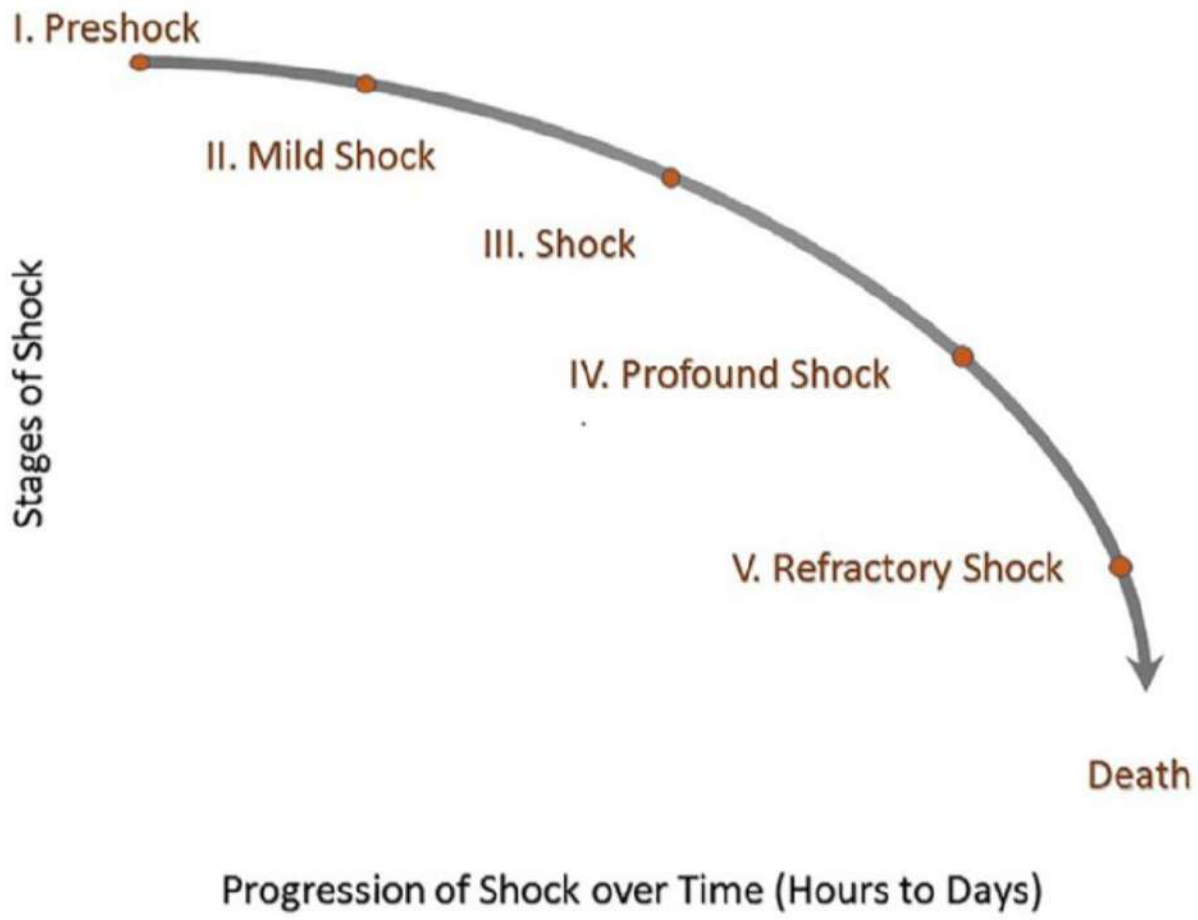
Outline

- The paradigm of perfusion and congestion
- Left and right heart perfusion by Doppler
- Concept of fluid tolerance
- Take home messages

Shock is a continuum



I. **Preshock**-Worsening Heart Failure: Diuretics/Inodilators

II. **Mild Shock**-Hypotension: Inotropes/Vasopressors+/-IABP, Impella CP

III. **Shock**-Lactic Acidosis, Tissue Hypoperfusion: Tandem Heart, Impella 5.0, CentriMag, RotaFlow, ECMO

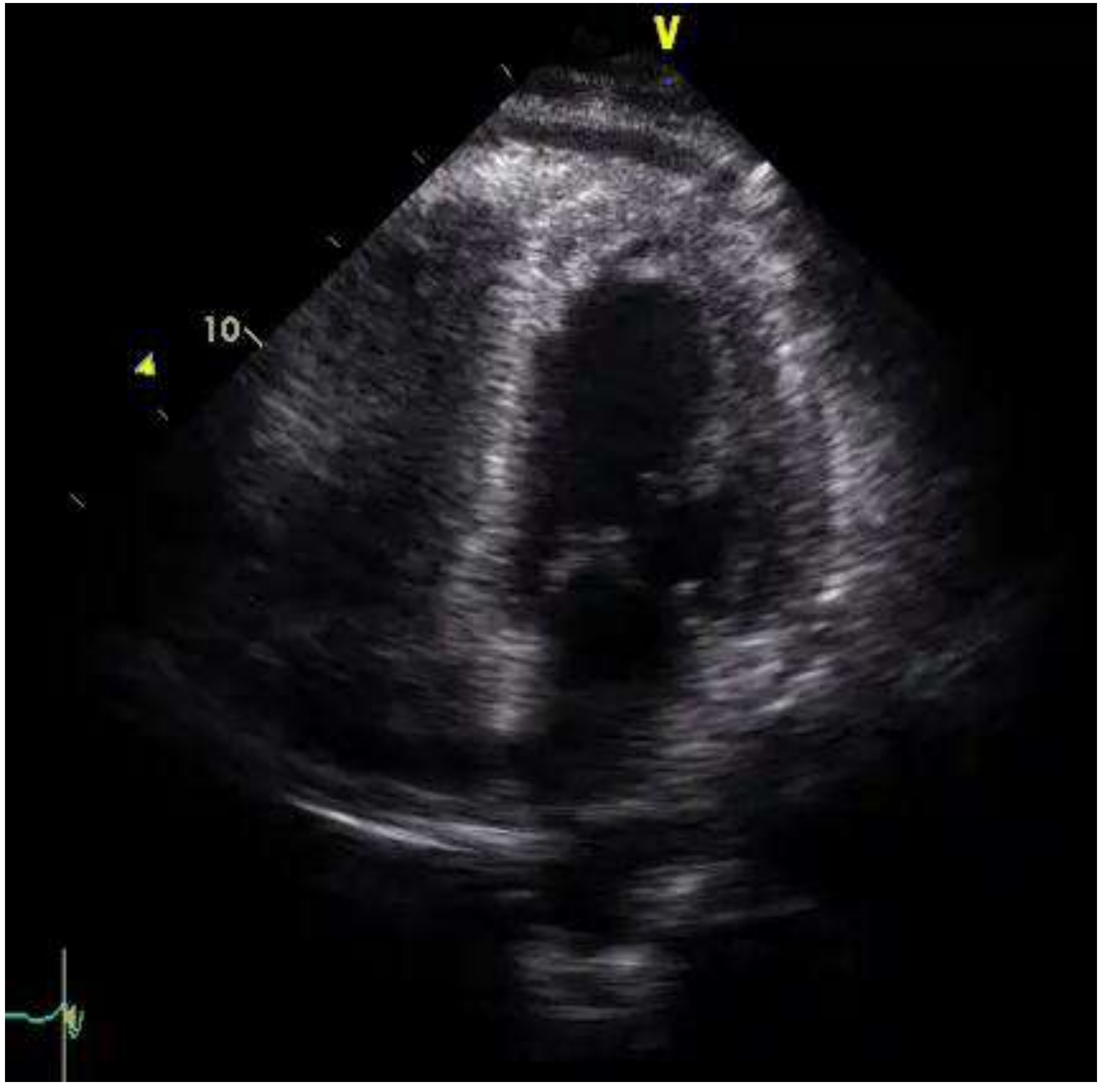
IV. **Profound Shock**-End Organ Failure: CentriMag, RotaFlow, ECMO

V. **Refractory Shock**-Vasodilation, Multiorgan Failure, Refractory Hypotension: Assess for futility of Care

Bellumkonda L. et al. Am J Card 2018

55 years old male → Referred from another center

- Fever, raised inflammatory markers
- Warm, sweaty
- BE -8 and Lactate 6 mmol/L
- SpO2 88% on O2 15 L/min
- BP 75/45 mmHg
- Troponin mildly raised



LV systolic function

EF is not invalidated in haemodynamically unstable patients

Limitations

Dependent on loading conditions (preload, afterload)

Affected by tachycardia

Dependent on LV end-diastolic volume (too big or too small)

Low reproducibility

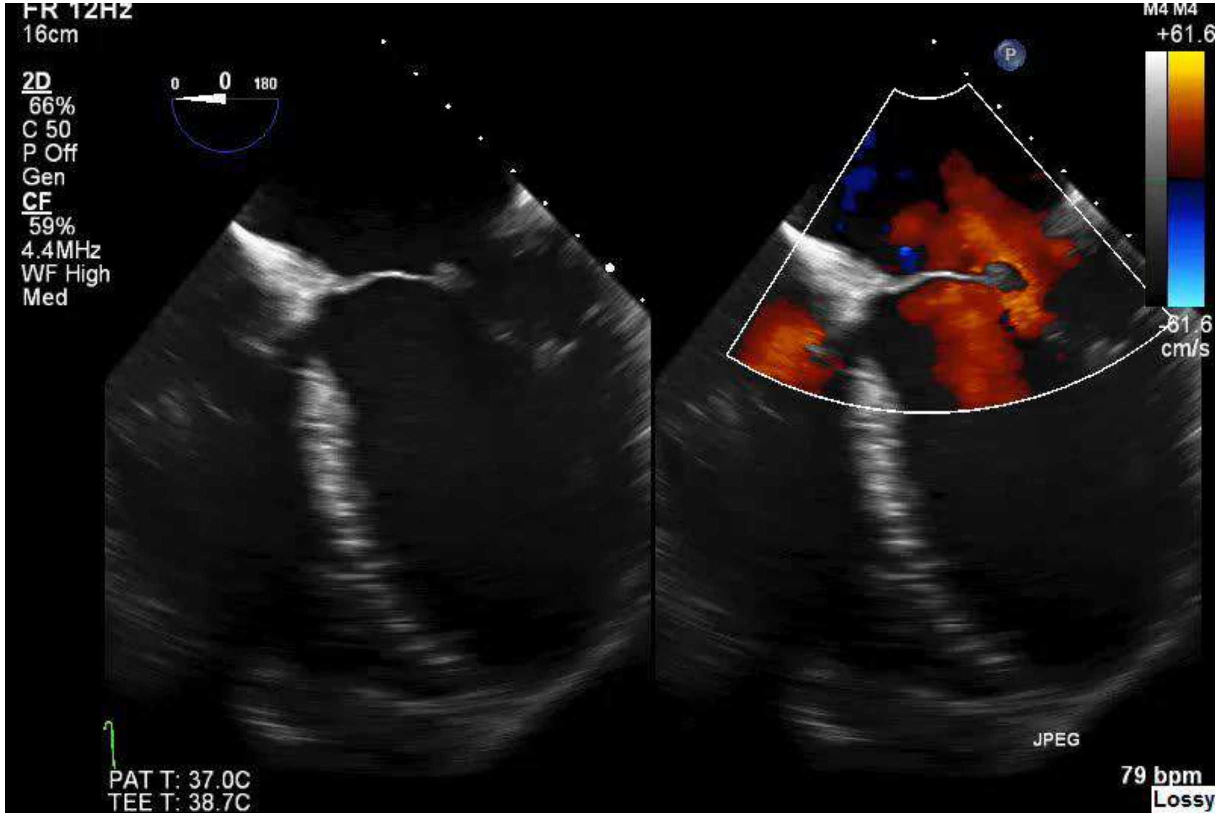
Overestimating in MR and intrventricular shunt

Geometric assumptions

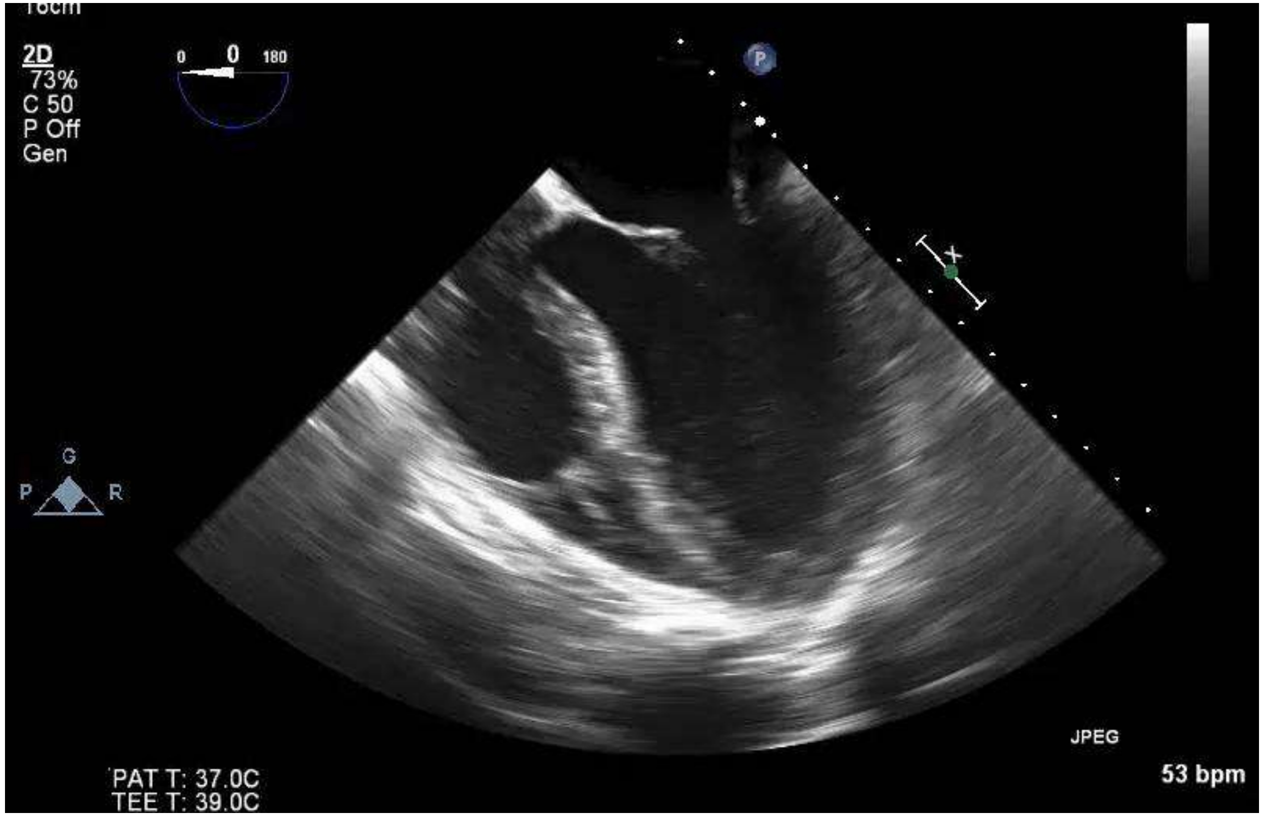
Low sensitivity in detecting subtle LV systolic impairament

Why EF can be misleading in shock?

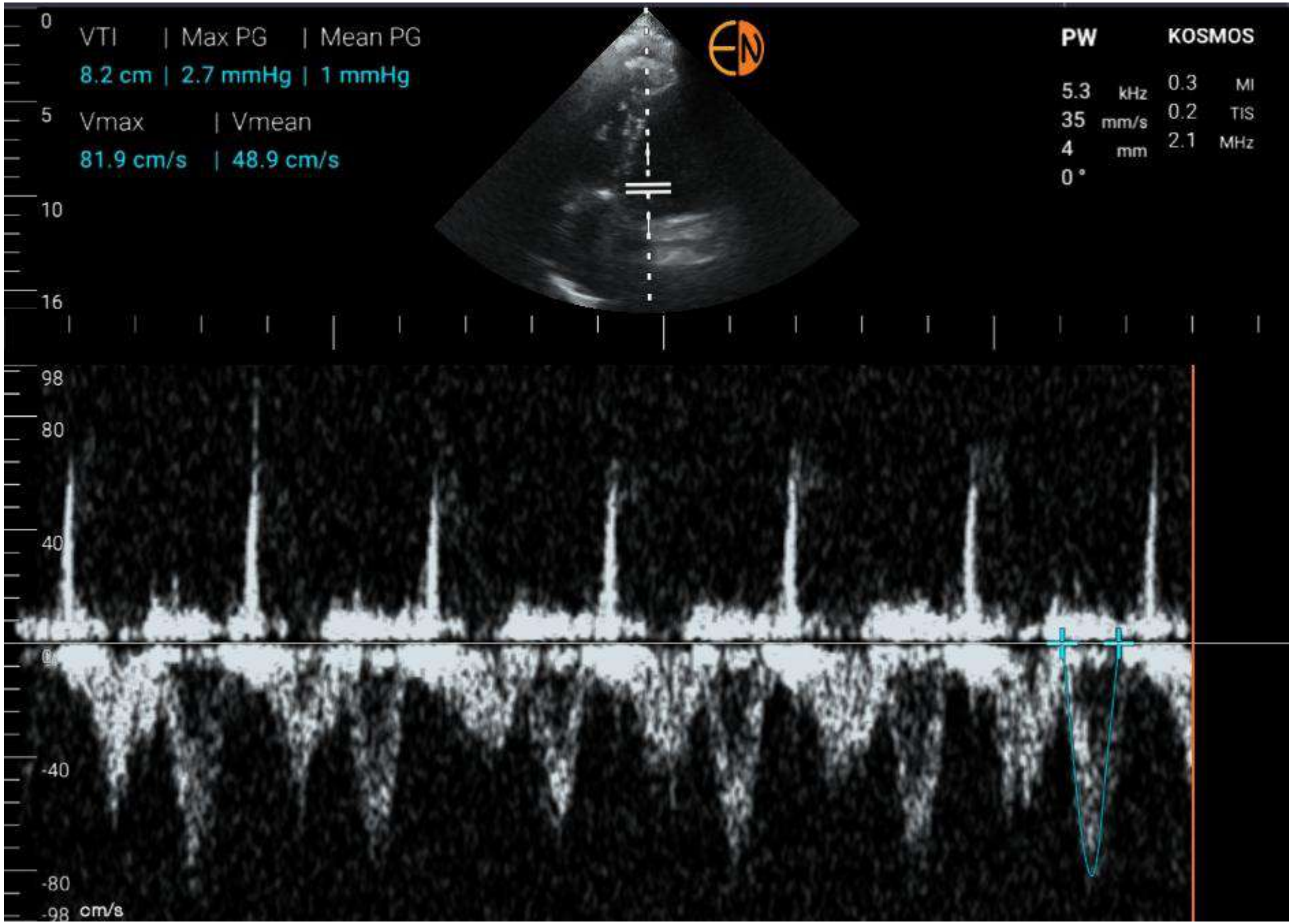
Good EF and in Cardiogenic Shock



Low EF and stable haemodynamics



LVOT VTI (Velocity Time Integral)



SV 27 ml
CO 2.9 L/min
CI 1.46 L/min/m²

Normal values:

- **LVOT diameter = 1.8-2.2 cm**
- **LVOT VTI (dynamic) = 18-22 cm**

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Pitfalls

LVOT obstruction

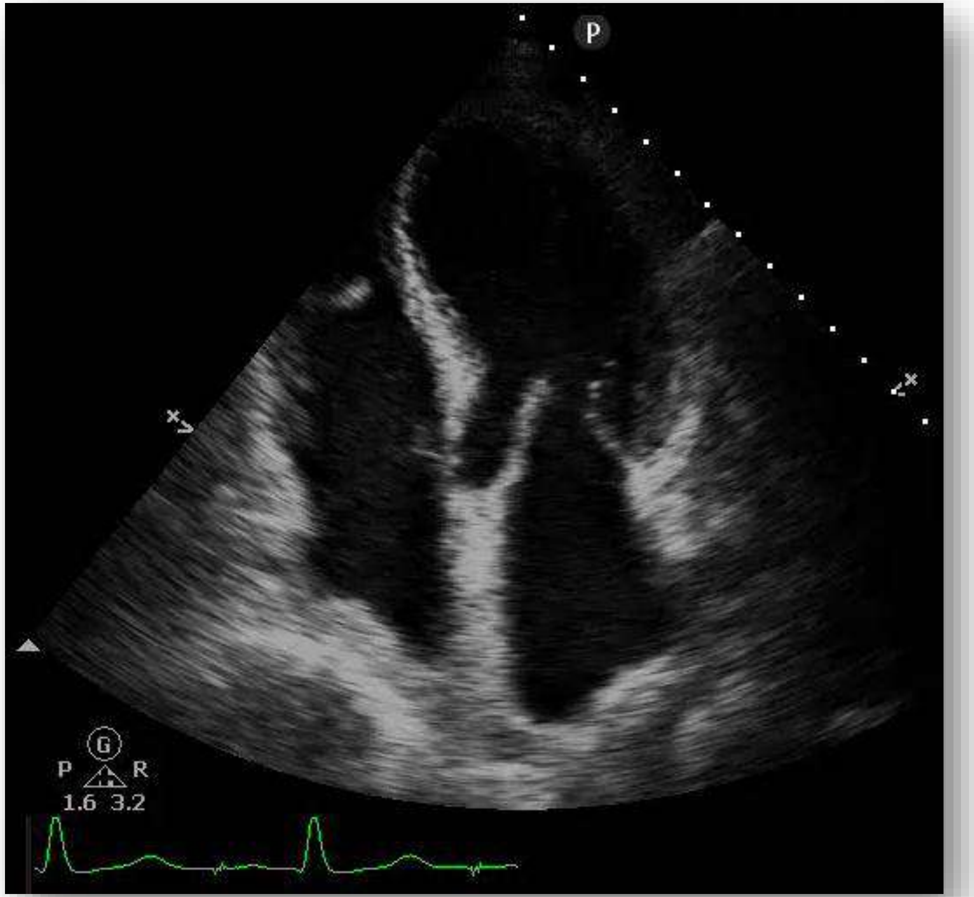
Dynamic

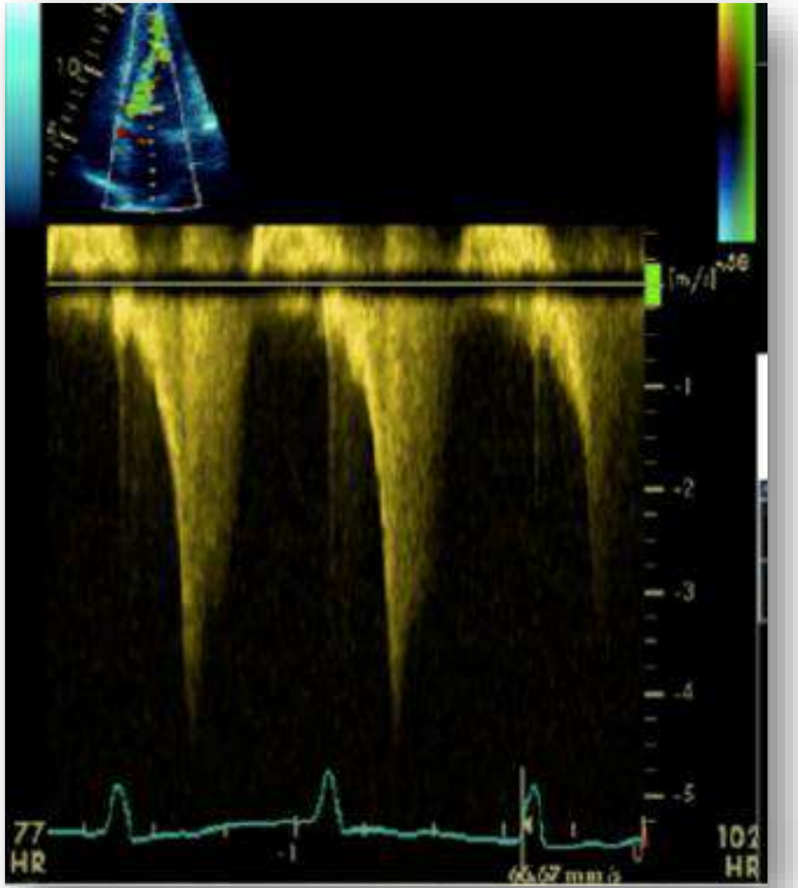
- SAM: severe hypovolaemia, HCM
- Hyperdynamic basal segments: Takotsubo cardiomyopathy or anterior MI with hyperdynamic basal segments

Why?

- LVOT shape is distorted and no longer a cylinder
- High velocity flow across the LVOT (not linear) → aliasing on PWD

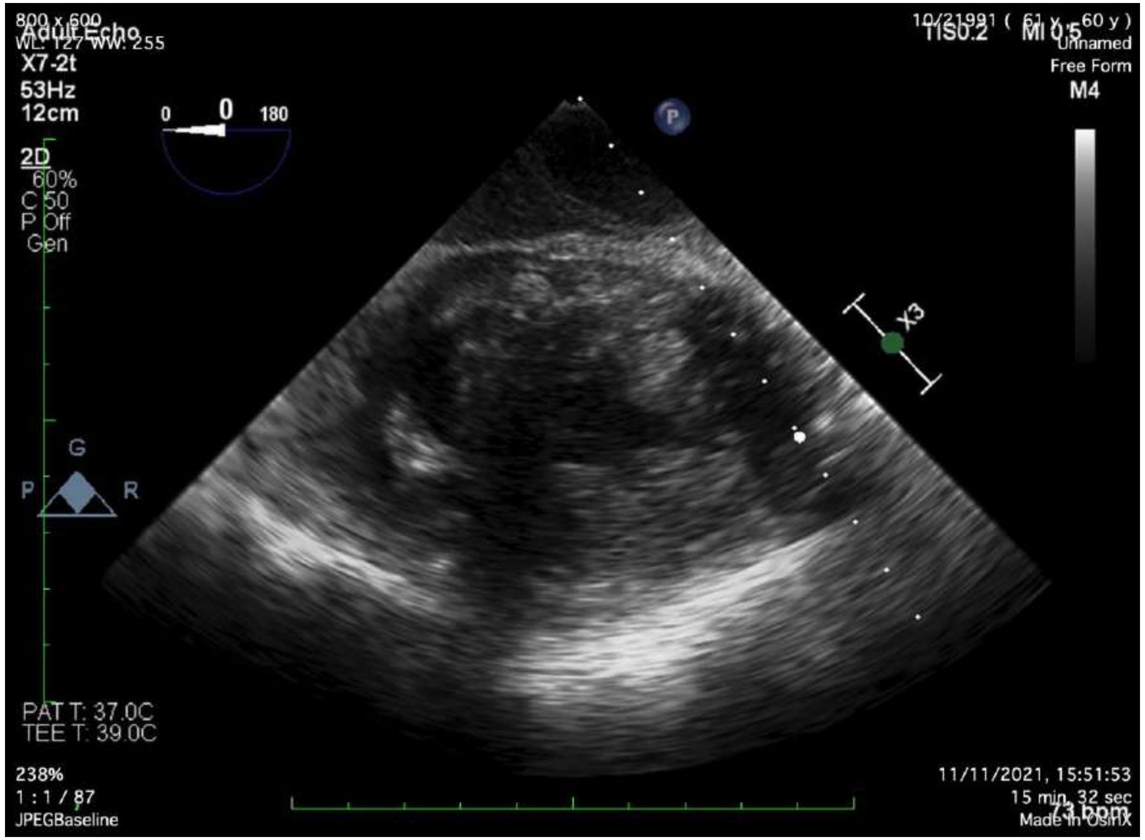
Dynamic LVOT Obstruction

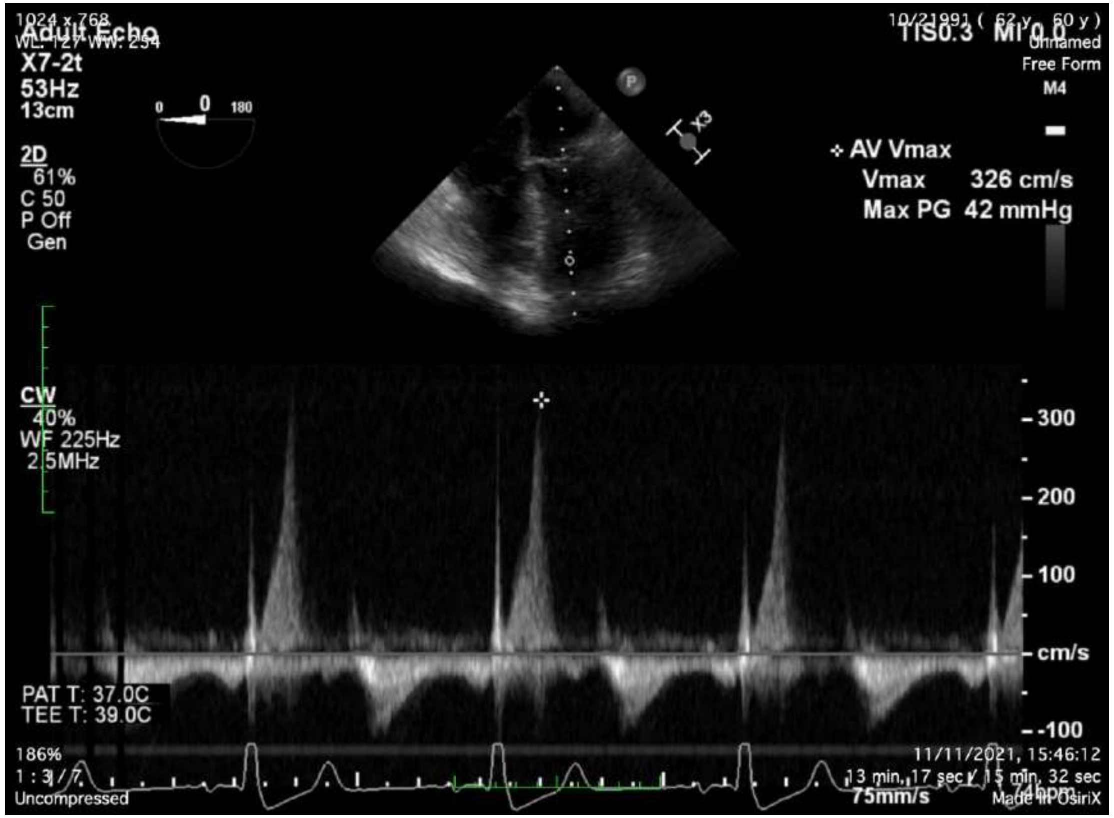




Predisposing factors: Tachycardia, hypovolemia, sepsis, severe anemia, inotropes, mitral valve surgery, LVH, small LV cavity

Dynamic LVOT Obstruction





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VTI is just a number!

Clinical context is the key factor

- LVOT VTI 20 cm in a patient with sepsis is not normal
- LVOT VTI 20 cm in moderate to severe AR is not normal
- LVOT 14 cm could be normal in patient with large LVOT diameter
- LVOT VTI could be from LVF or RVF or both!

VTI is just a number!

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- LVOT VTI 20 cm in a patient with sepsis is not normal
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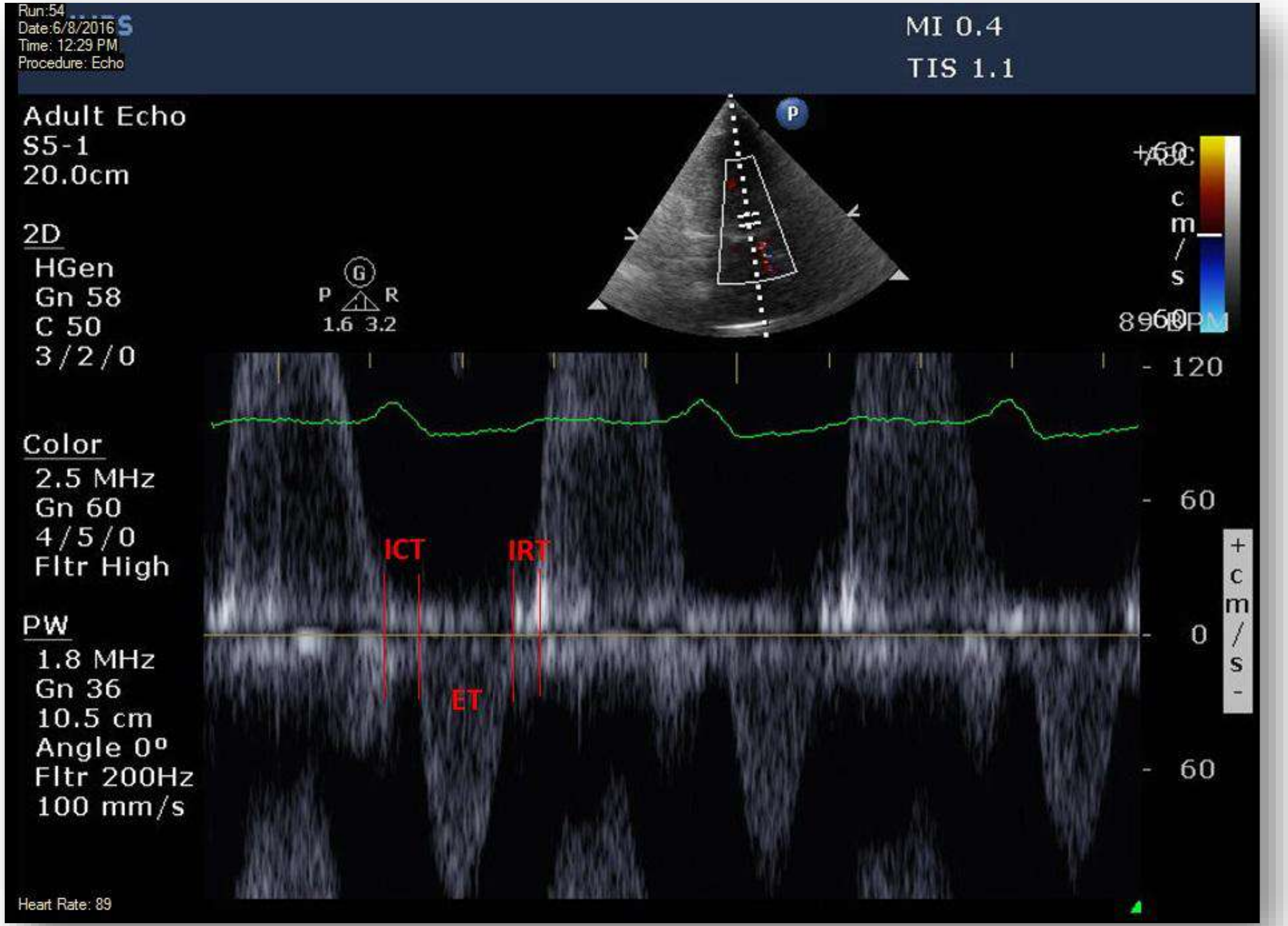
- LVOT 14 cm could be normal in patient with large LVOT diameter
- **LVOT VTI could be from LVF or RVF or both!**

Total Isovolumetric Time (t-IVT)



Normal < 12 s/min

t-IVT = 60 - (t-FT + t-ET)

Published by Elsevier Science Inc.
ISSN 0735-1097/03/$30.0
PII S0735-1097(02)02665-

Limitation of Cardiac Output by Total Isovolumic Time During Pharmacologic Stress in Patients With Dilated Cardiomyopathy

Activation-Mediated Effects of Left Bundle Branch Block and Coronary Artery Disease

Alison M. Duncan, MB, BS, Darrel P. Francis, MB, Michael Y. Henein, PhD, FACC,
Derek G. Gibson, FRCP
London, United Kingdom

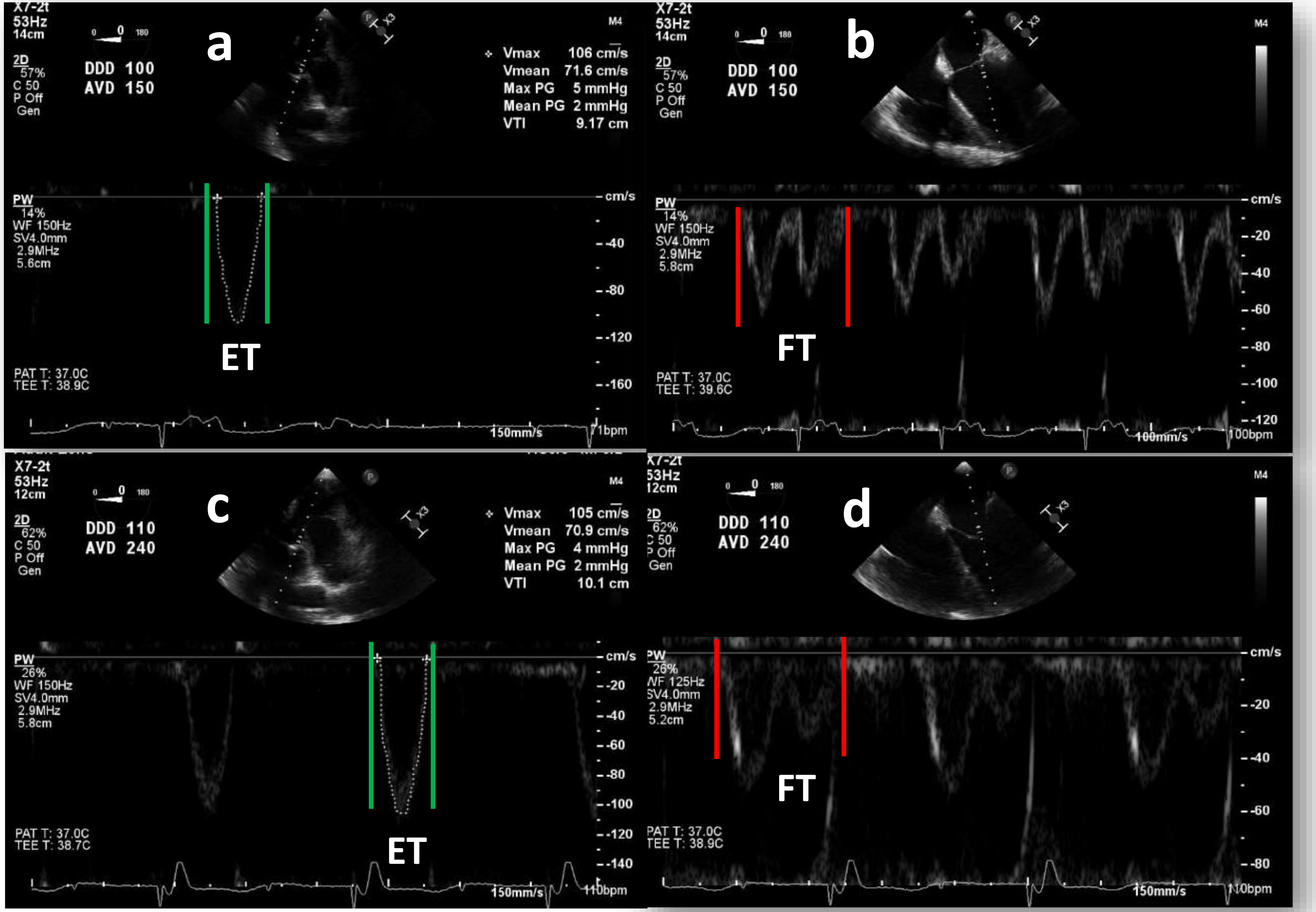
"In patients with dilated cardiomyopathy, t-IVT has a strong inverse relationship with cardiac output during dobutamine stress and is the major determinant of cardiac output"

Duncan A et al, JACC Vol. 41, No. 1, 2003

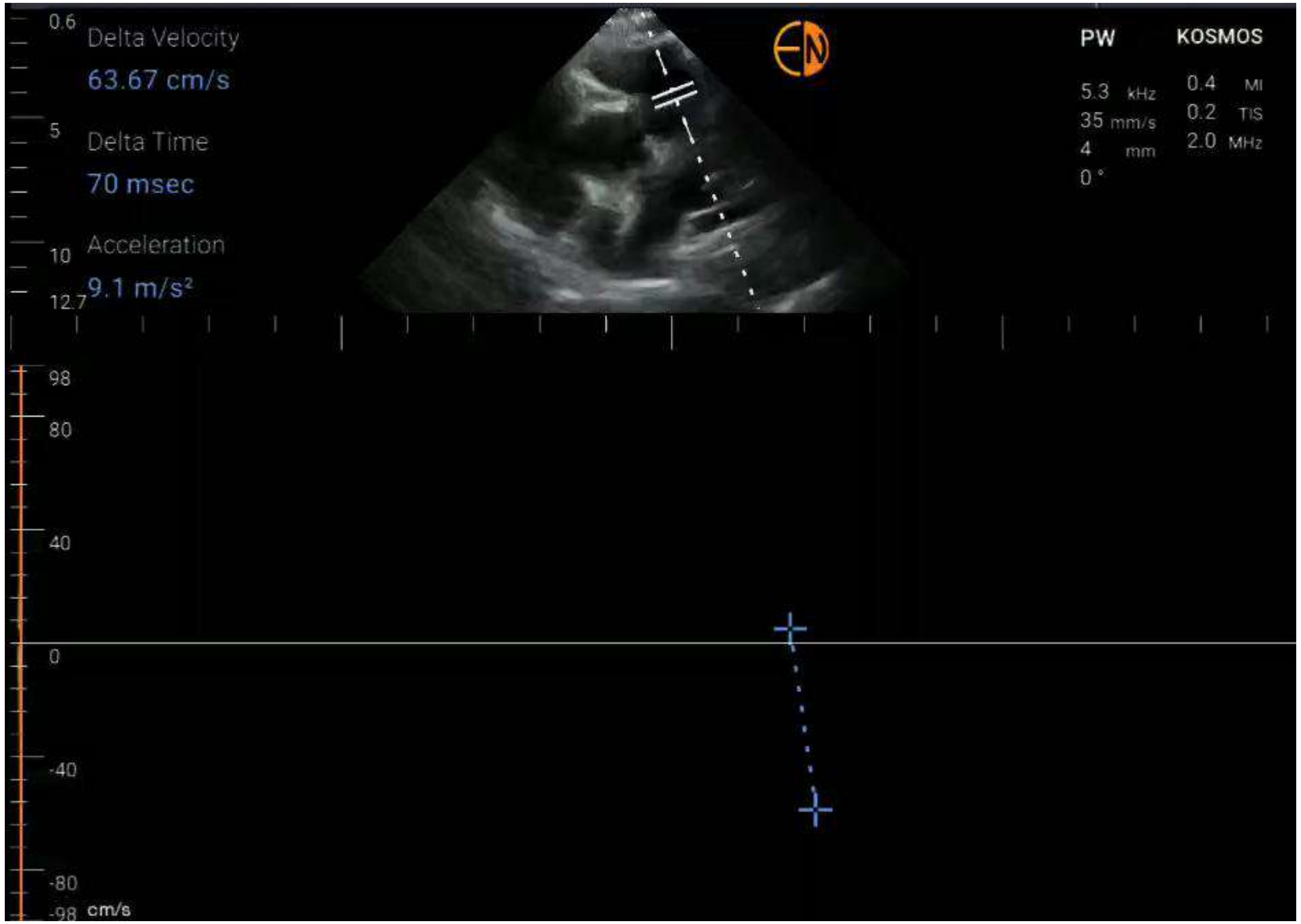
Post redo CABG: Echo-guided pacing optimisation

CO 2.6 L/min
tIVT 16 s/min

CO 3.2 L/min
tIVT 13.9 s/min



RVOT (PWD)



Valuable insights into RV function, PVR and ventriculo-arterial coupling

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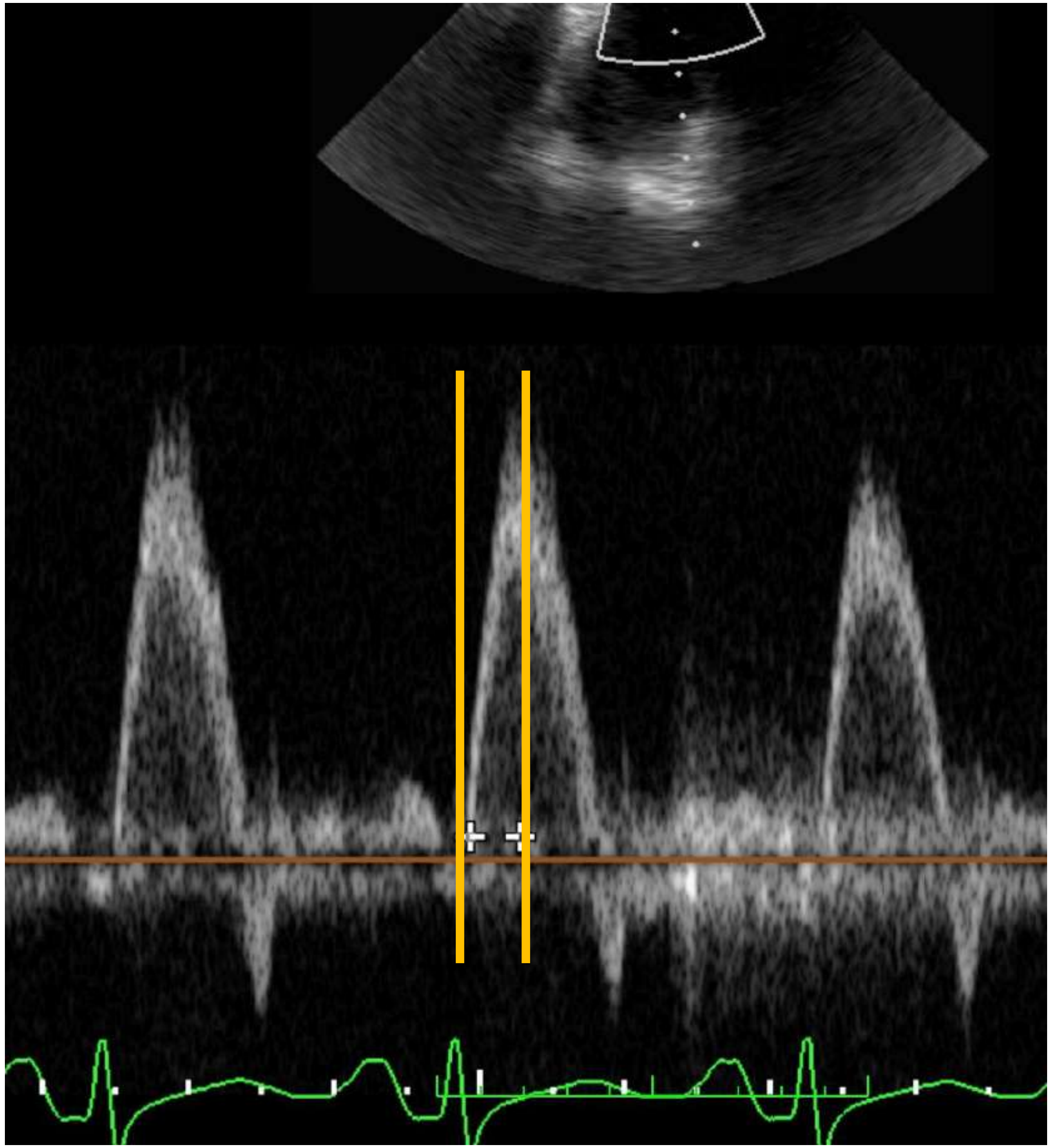
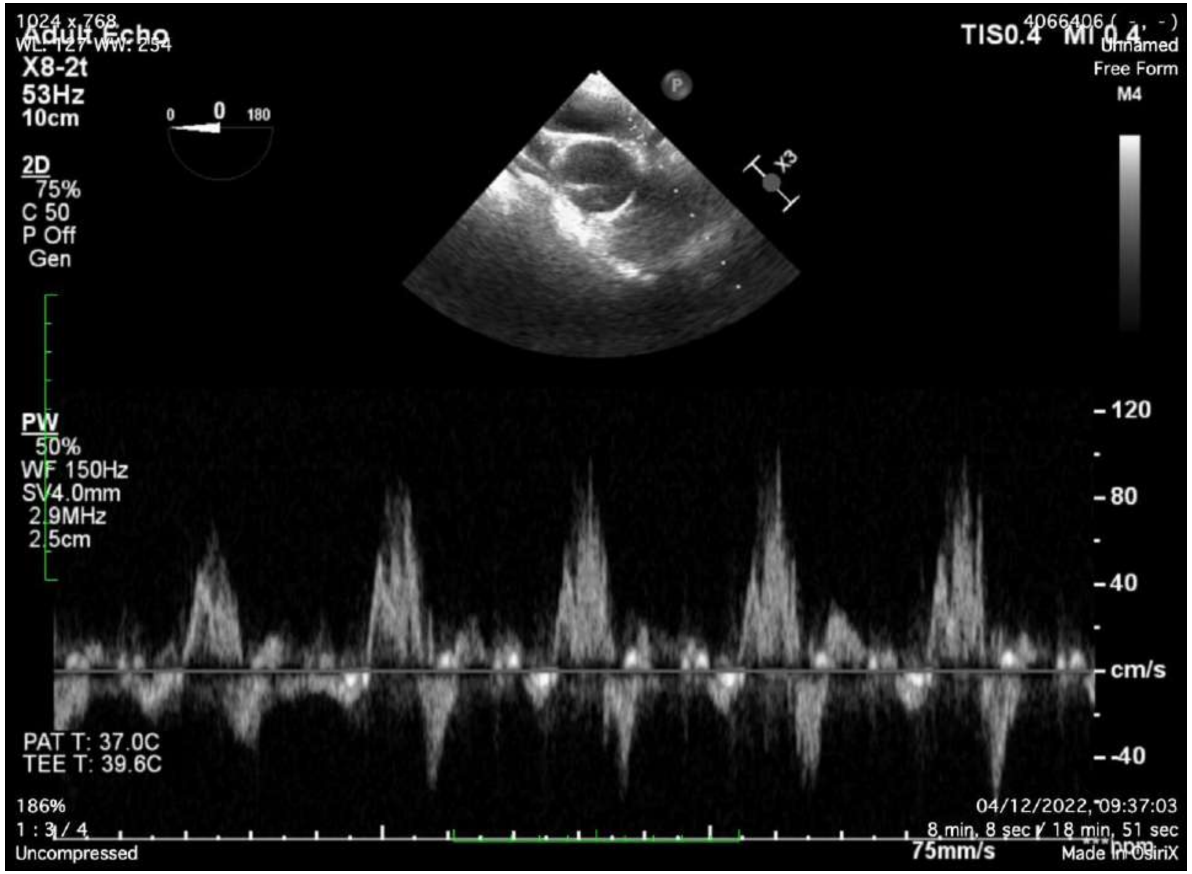
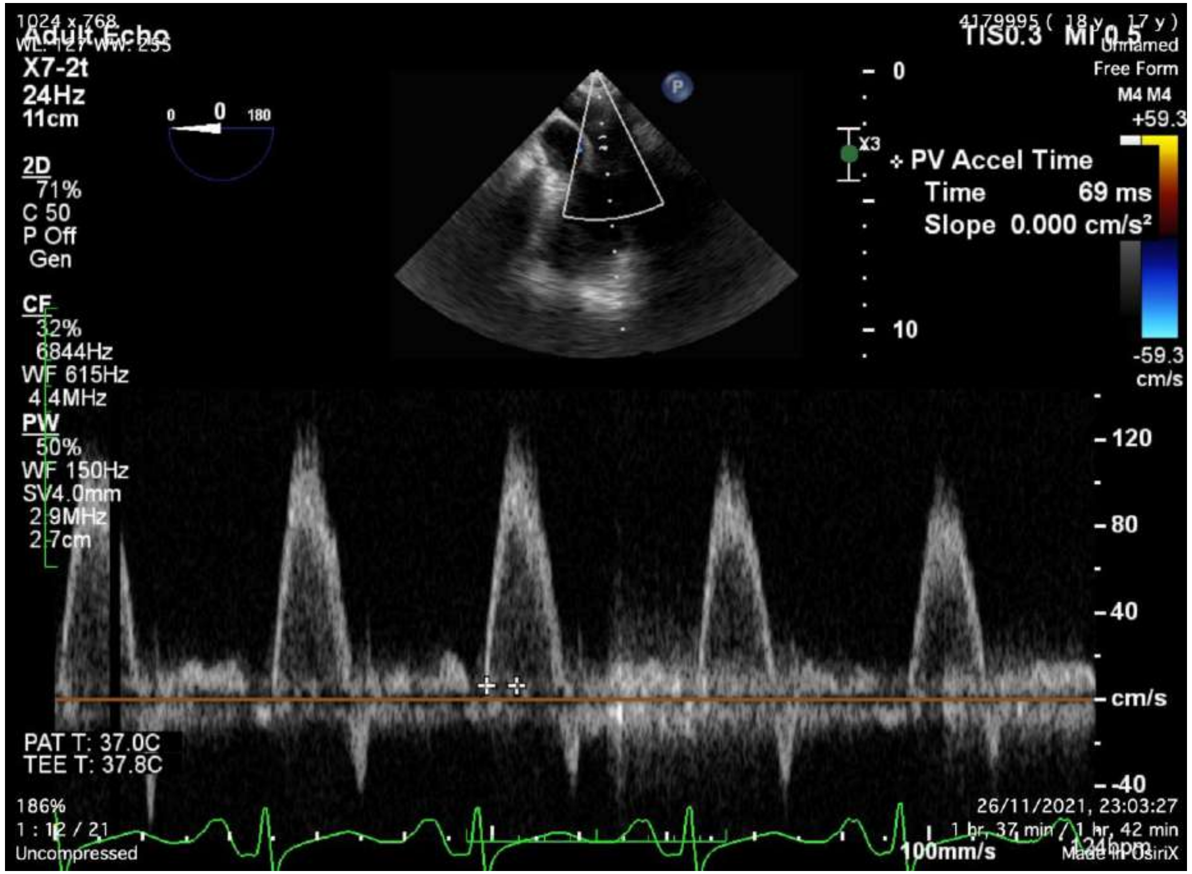
$$mPAP = 90 - (0.65 \times AT)$$



Debastani, 1987



Before pulmonary vasodilator



After pulmonary vasodilator

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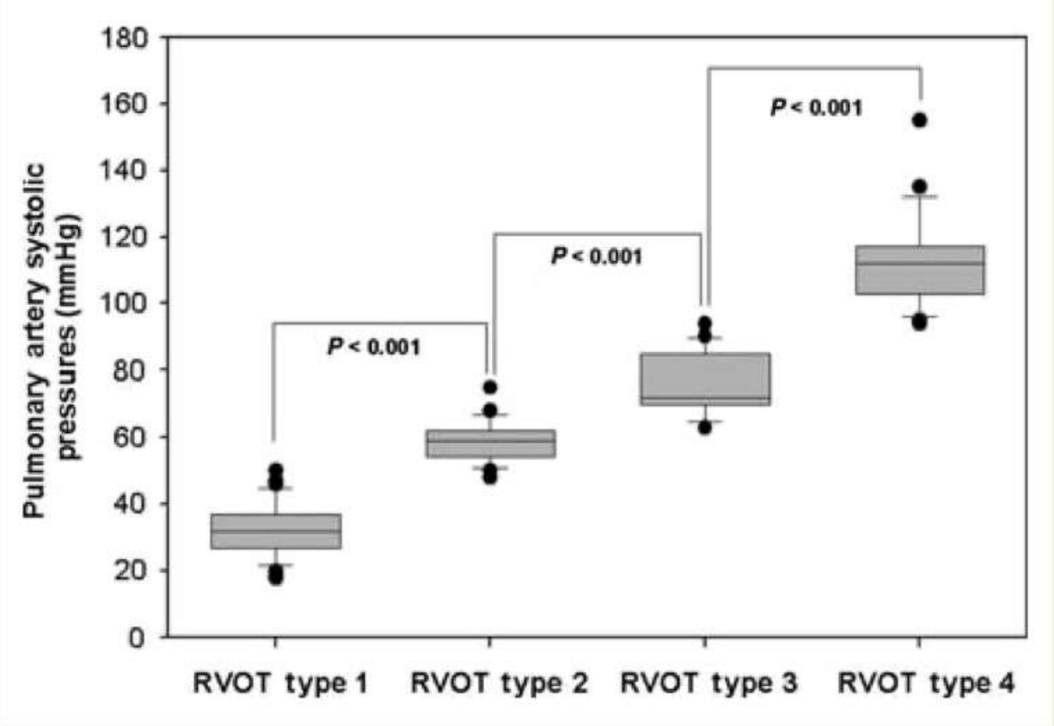
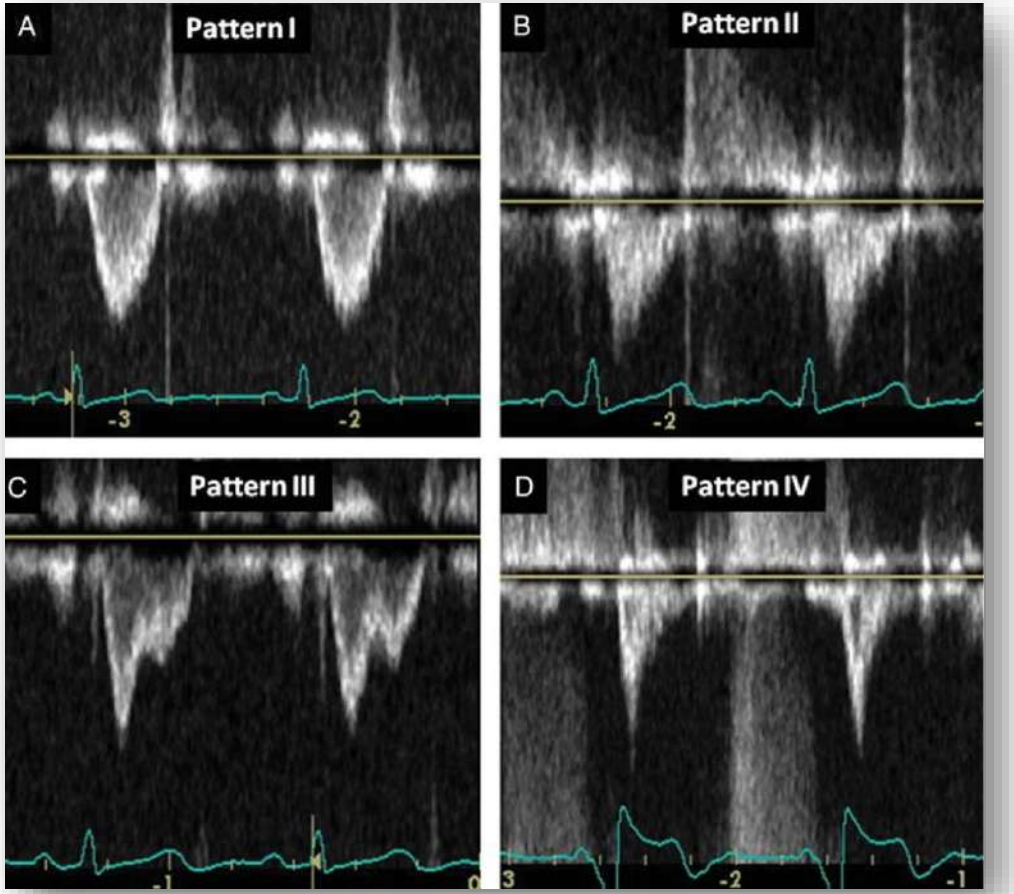
RVOT





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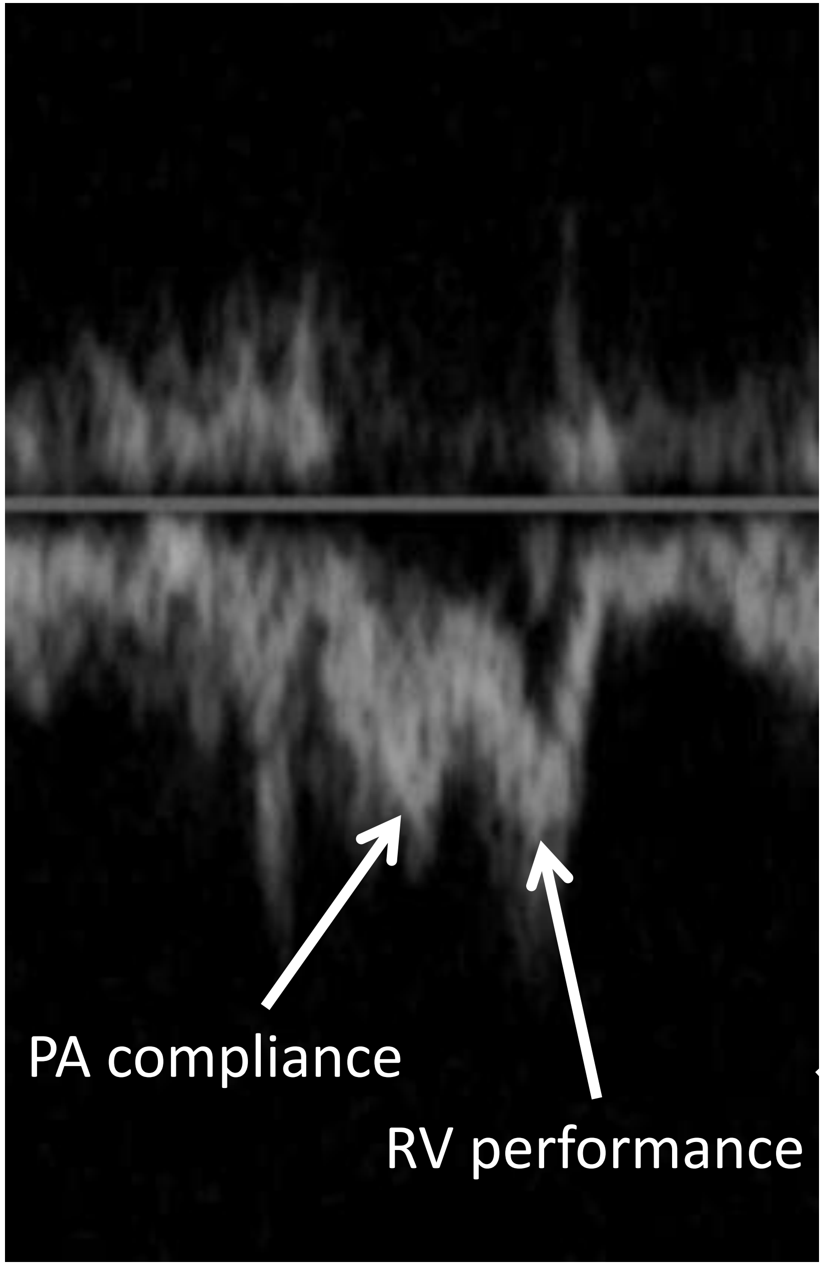
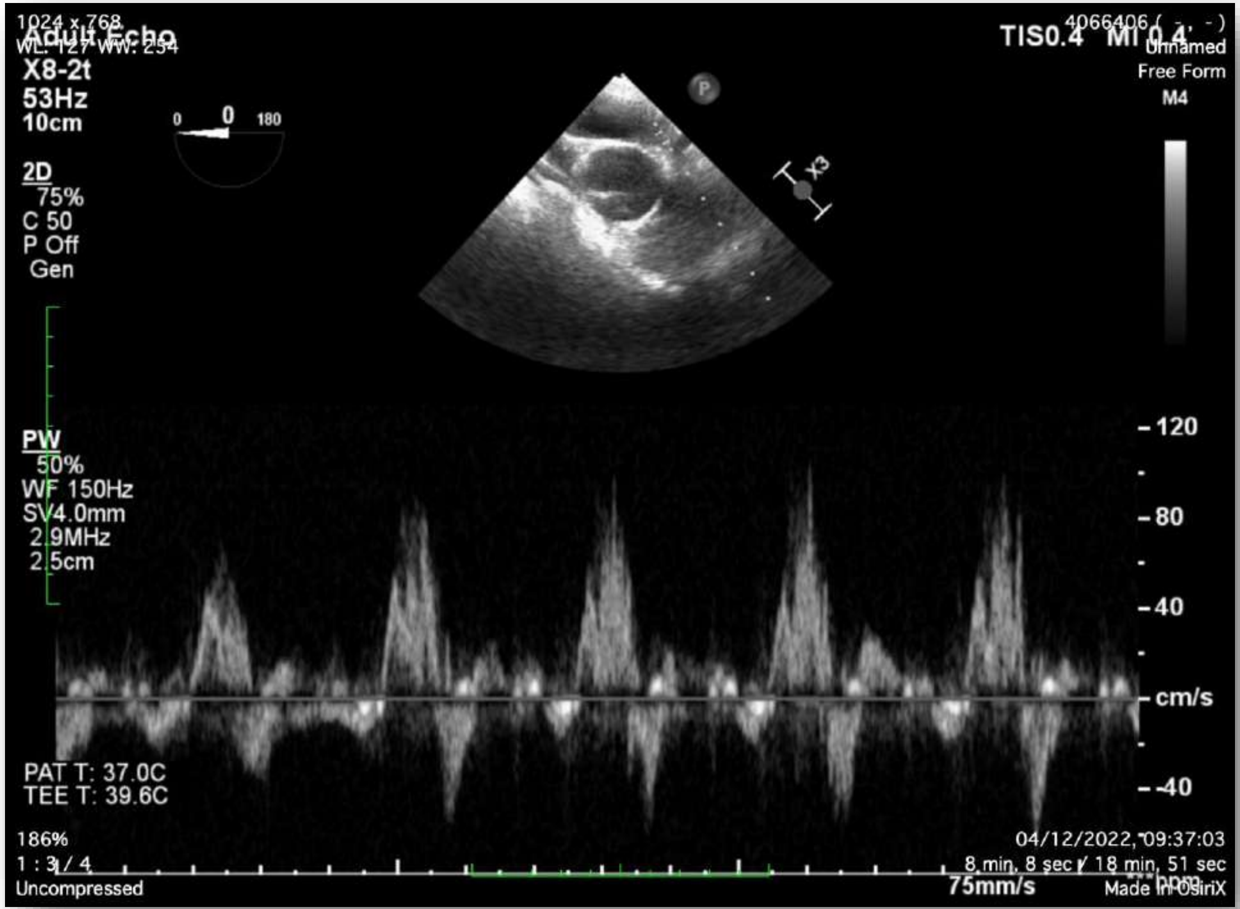
Lopez-Candales 2012

RVOT




Adult Echo
X8-2t
53Hz
10cm
2D
75%
C 50
P Off
Gen
PW
50%
WF 150Hz
SV4.0mm
2.9MHz
2.5cm
TIS0.4 MI 0.4
Unnamed
Free Form
M4
120
80
40
cm/s
-40
PAT T: 37.0C
TEE T: 39.6C
186%
Uncompressed
04/12/2022, 09:37:03
75mm/s
Made In OsiriX

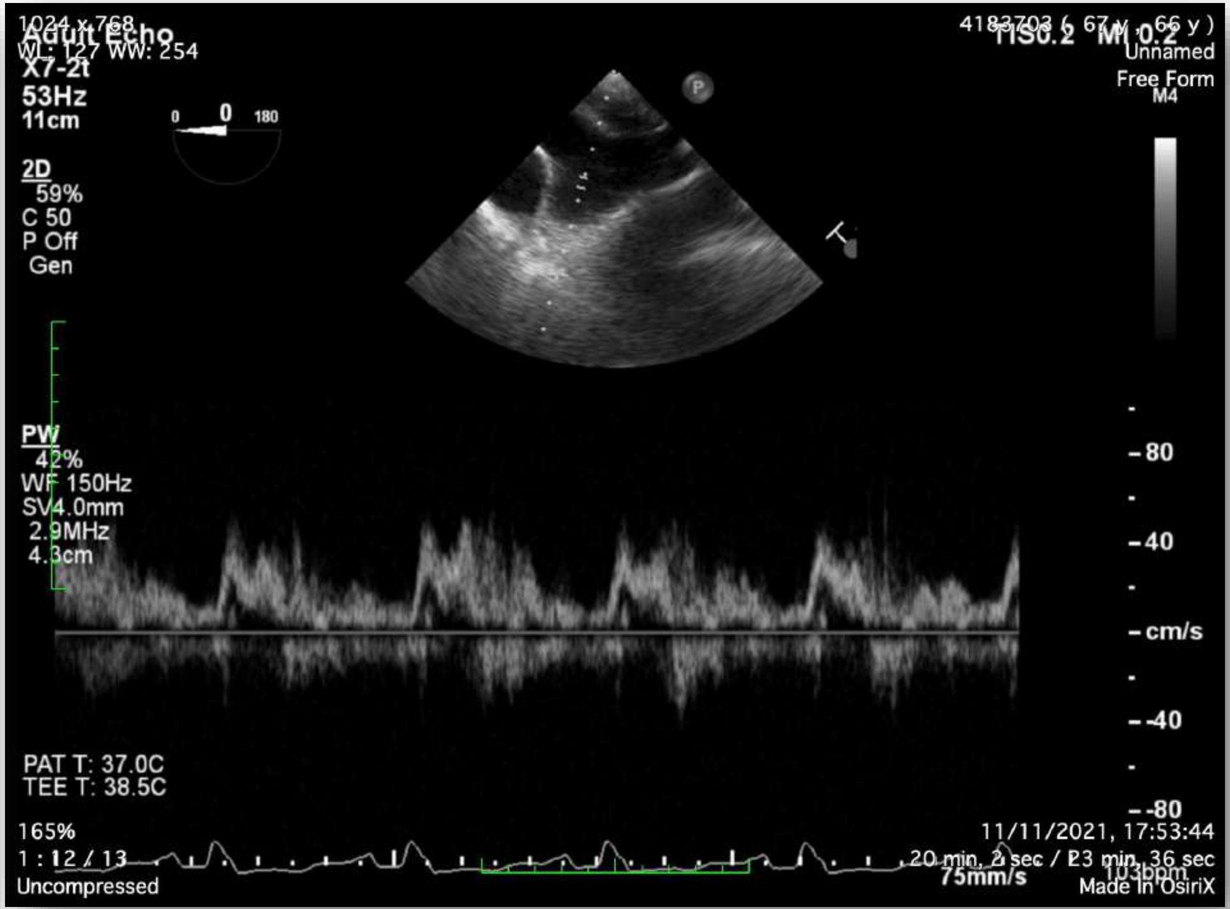

Adult Echo
X7-2t
53Hz
11cm
2D
59%
C 50
P Off
Gen
PW
42%
WF 150Hz
SV4.0mm
2.9MHz
4.3cm
TIS0.2 MI 0.2
Unnamed
Free Form
M4
80
40
cm/s
-40
-80
PAT T: 37.0C
TEE T: 38.5C
165%
Uncompressed
11/11/2021, 17:53:44
75mm/s
Made In OsiriX

FIGURE 5 Association Between Type of RVOT Flow Profile and Clinical Outcomes

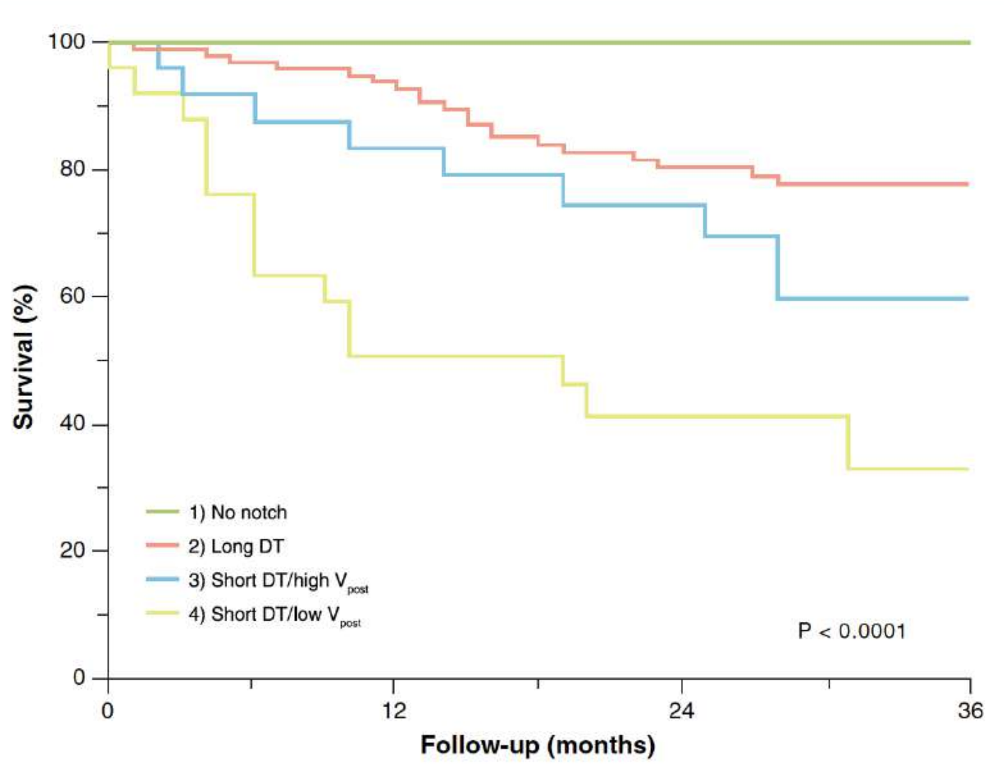
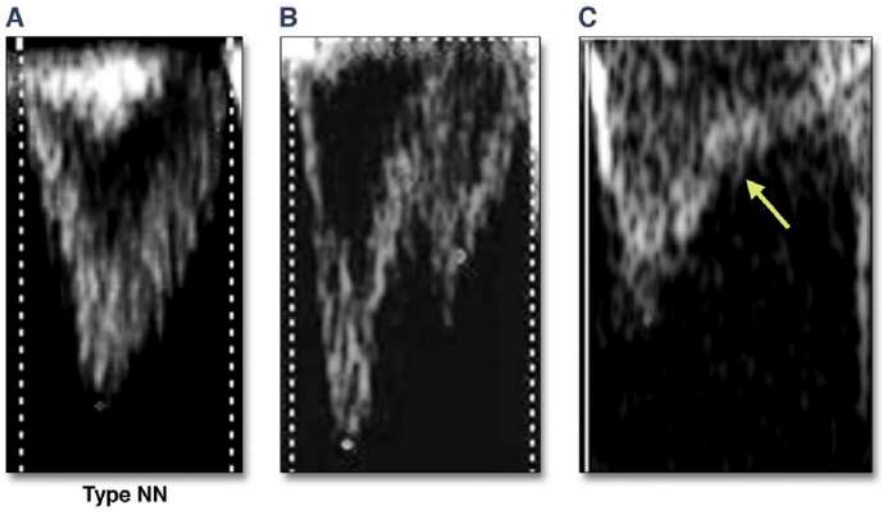


FIGURE 1 Representative RVOT Flow Systolic Profiles



(A) Pattern without mid-systolic deceleration and notching (no notch [NN] pattern), whereas there is shortening of acceleration time, the flow pattern maintains a parabolic curve. **(B)** The pattern demonstrates mid-systolic deceleration and notching separating 2 distinct acceleration flows. **(C)** The pattern of rectilinear mid-systolic deceleration with an inflection point **(arrow)** and no post-notching acceleration flow. RVOT = right ventricular outflow tract.

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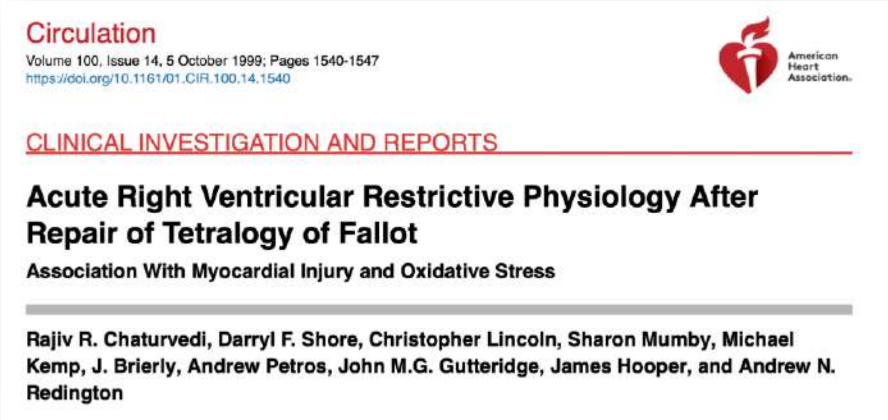
Takahama et al, JACC Imaging, 2017


Circulation
Volume 100, Issue 14, 5 October 1999; Pages 1540-1547
https://doi.org/10.1161/01.CIR.100.14.1540

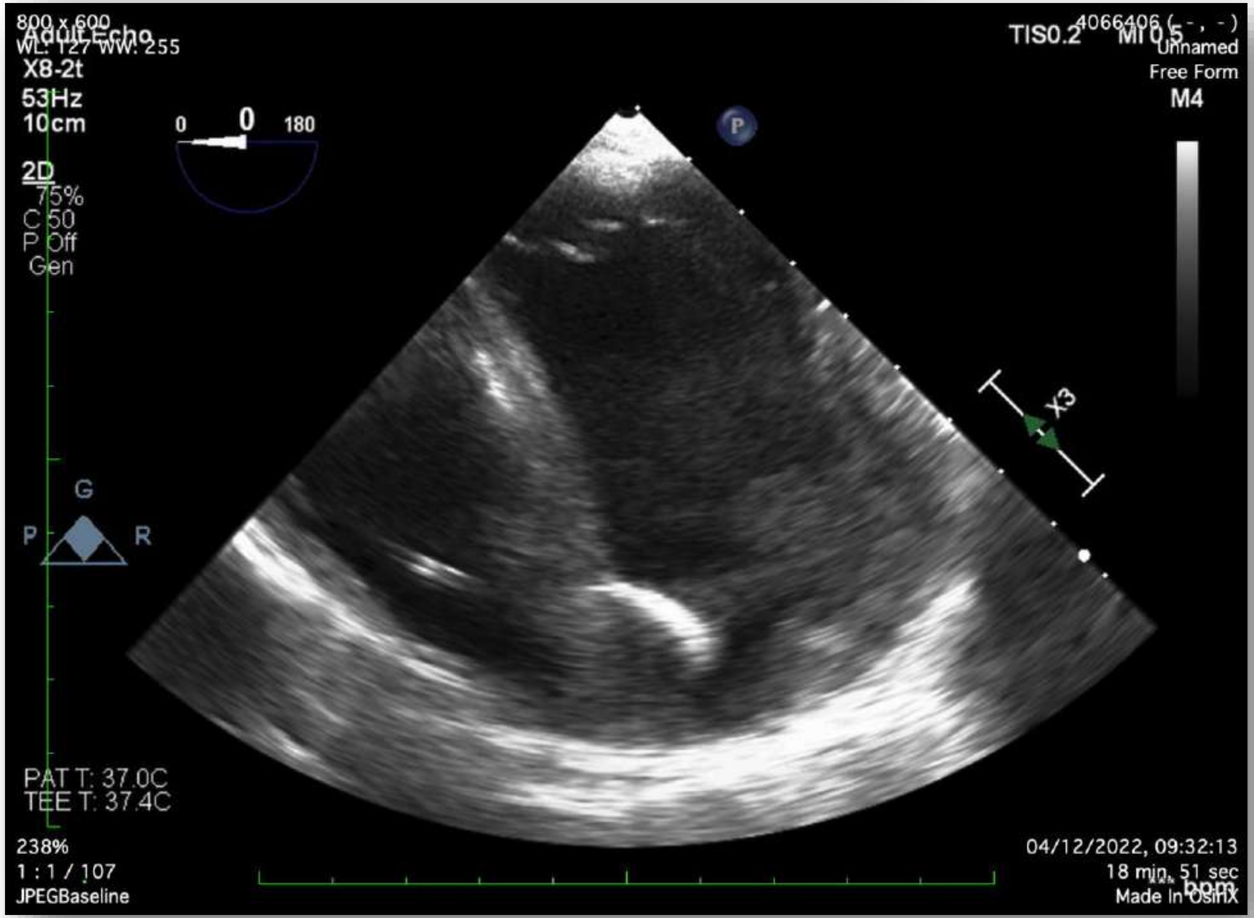
CLINICAL INVESTIGATION AND REPORTS

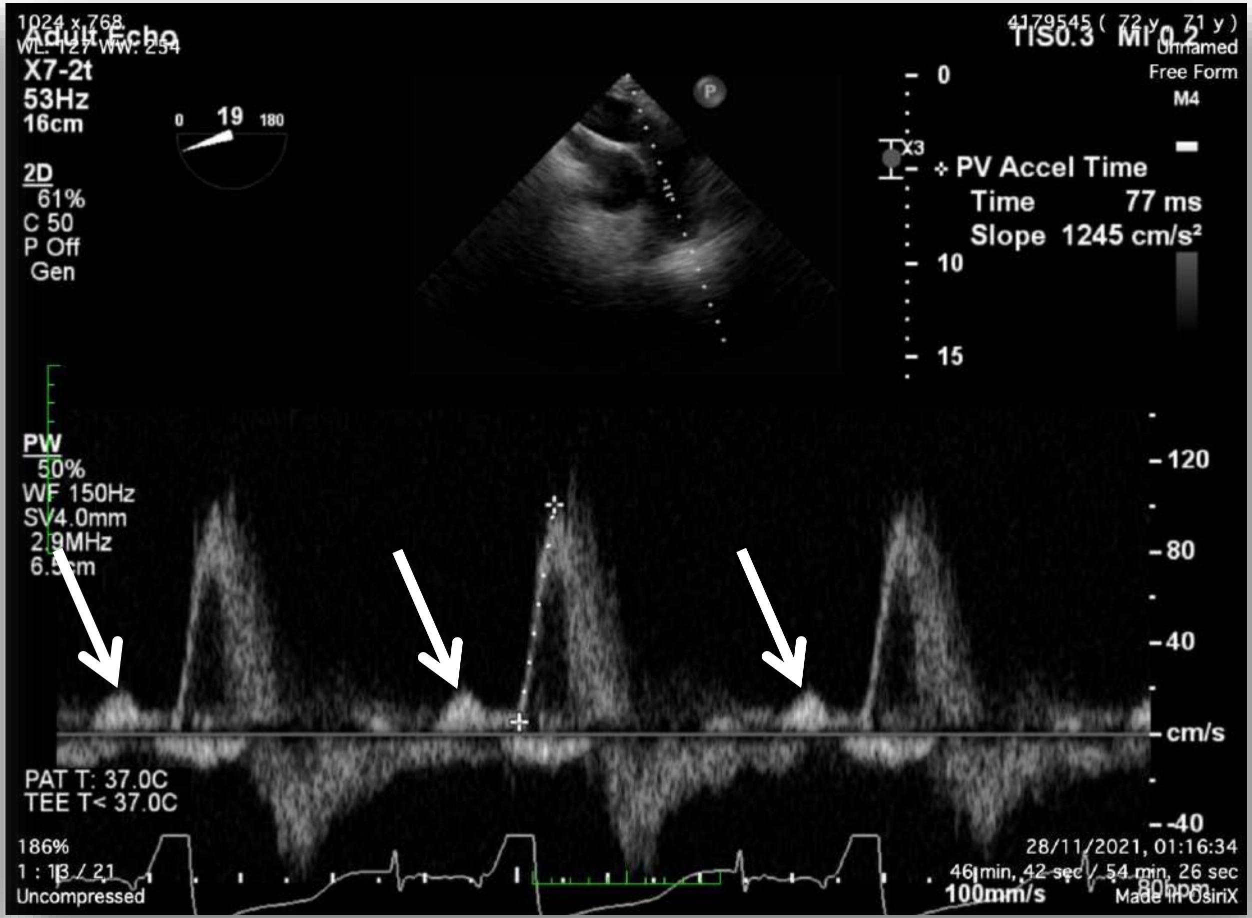
Acute Right Ventricular Restrictive Physiology After Repair of Tetralogy of Fallot

Association With Myocardial Injury and Oxidative Stress

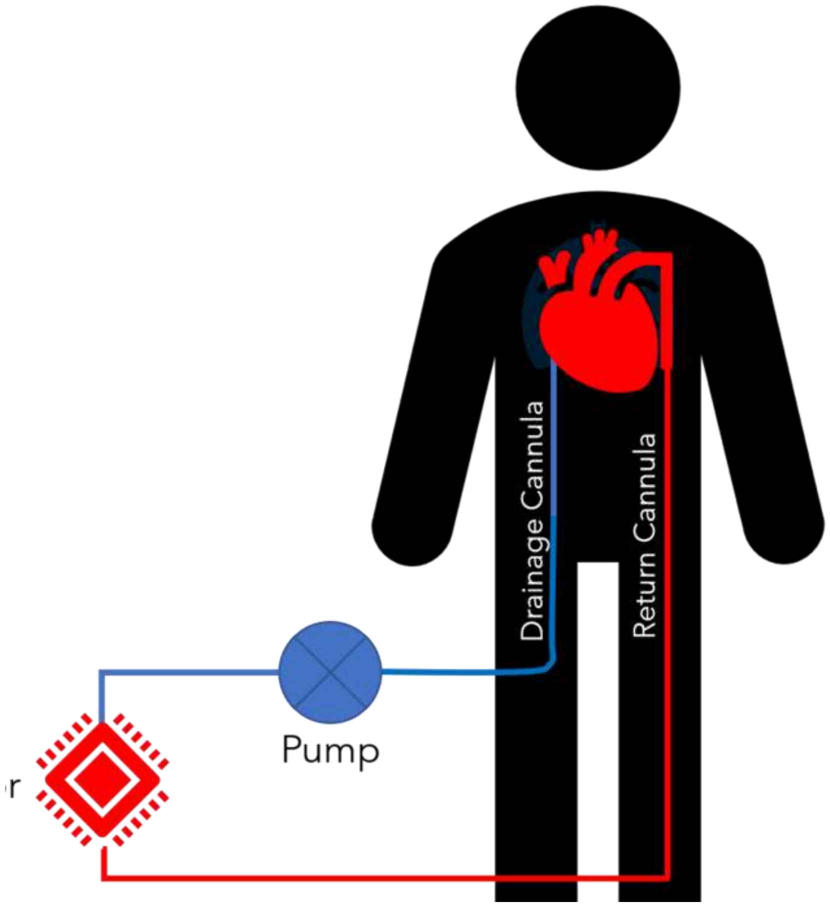
Rajiv R. Chaturvedi, Darryl F. Shore, Christopher Lincoln, Sharon Mumby, Michael Kemp, J. Brierly, Andrew Petros, John M.G. Gutteridge, James Hooper, and Andrew N. Redington

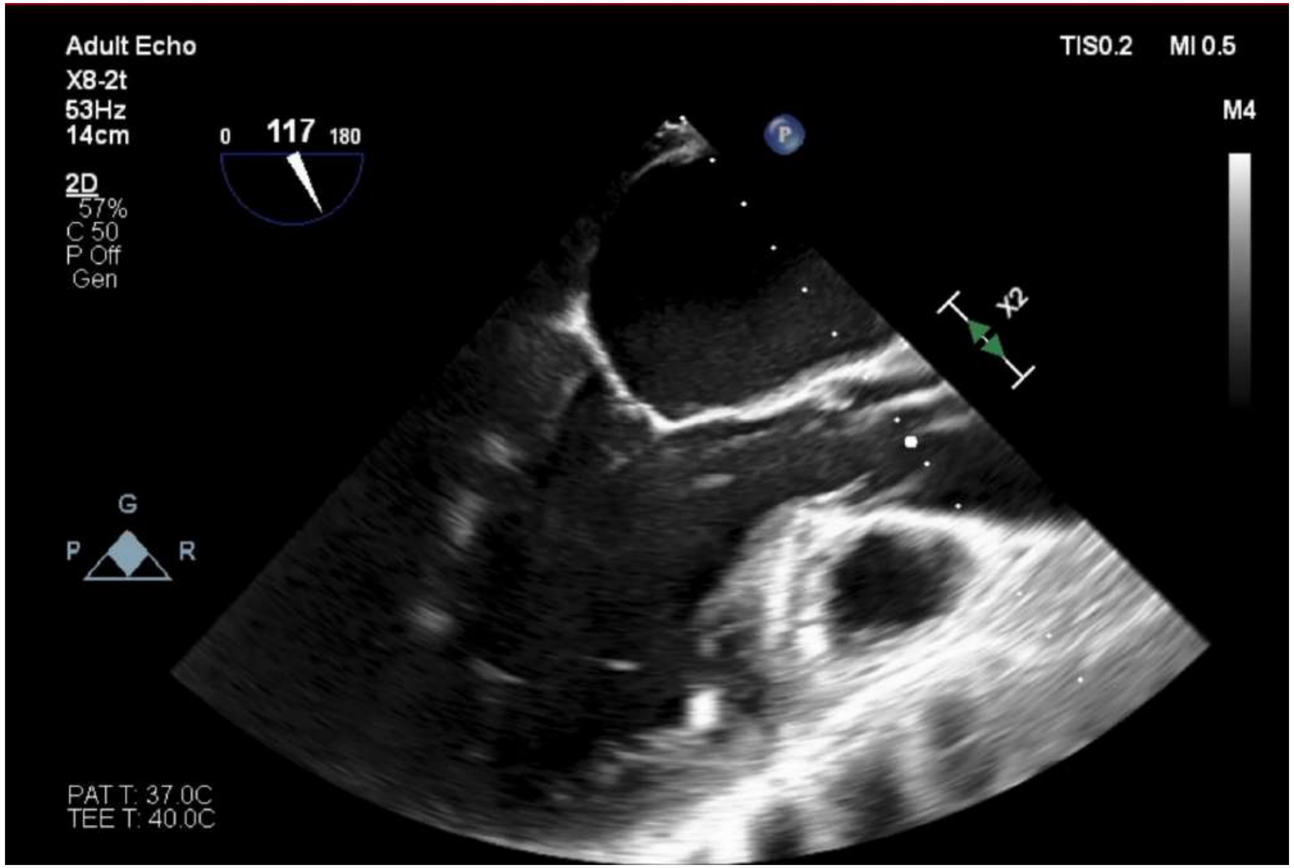
- Pre-systolic A wave (antegrade PA flow)
- Frequently seen in critically ill
- Can persist even after normalisation of PVR





Impella CP and V-A ECMO (ECPELLA)





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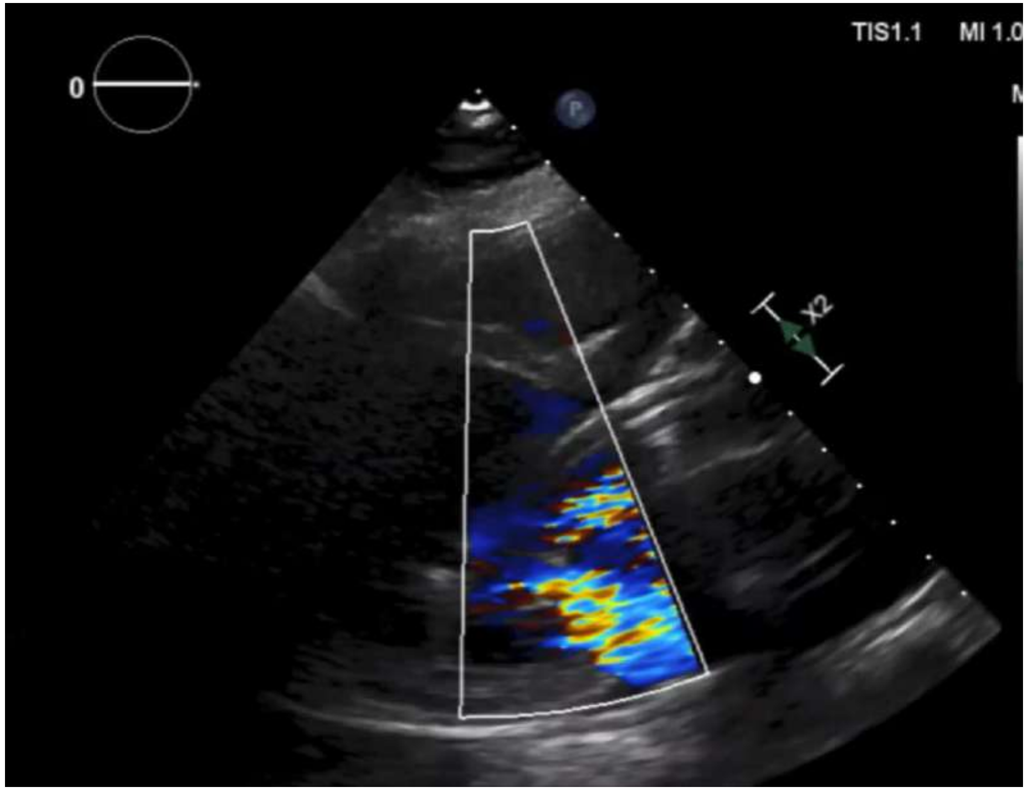
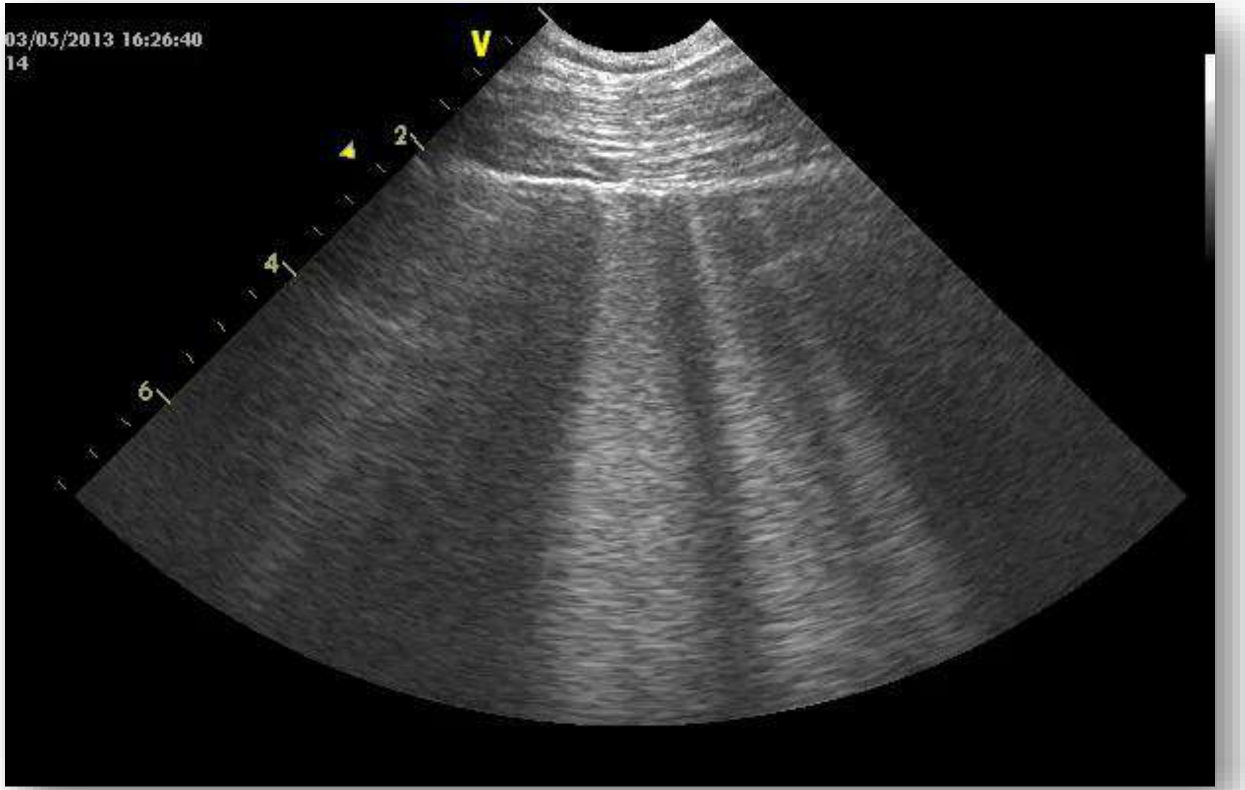
MR and worsening oxygenation





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Heart-lung interactions

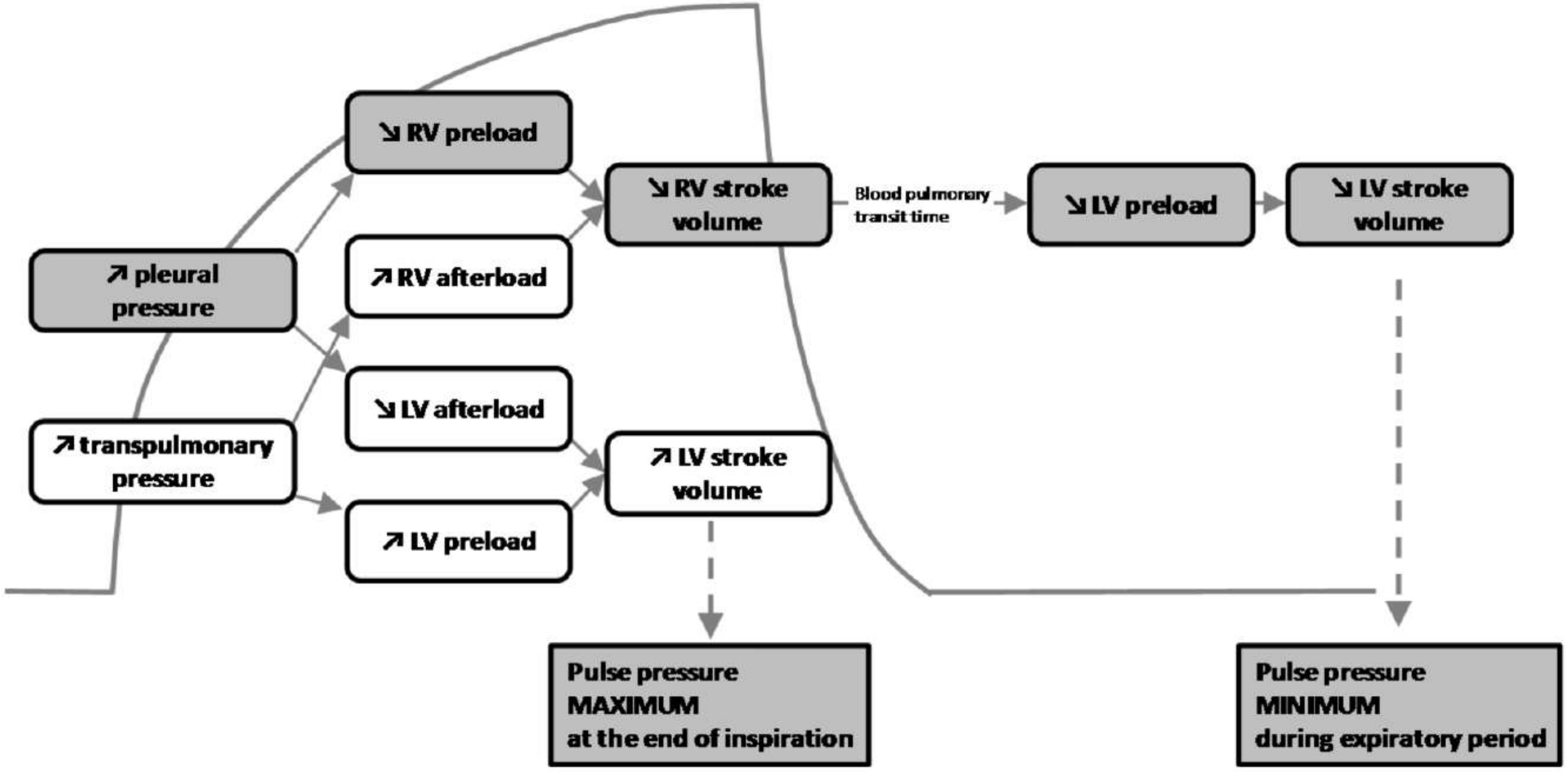
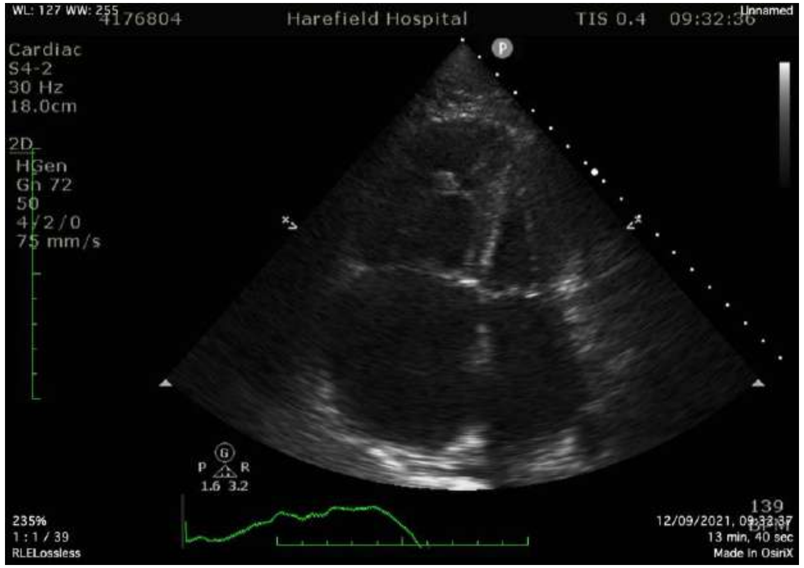


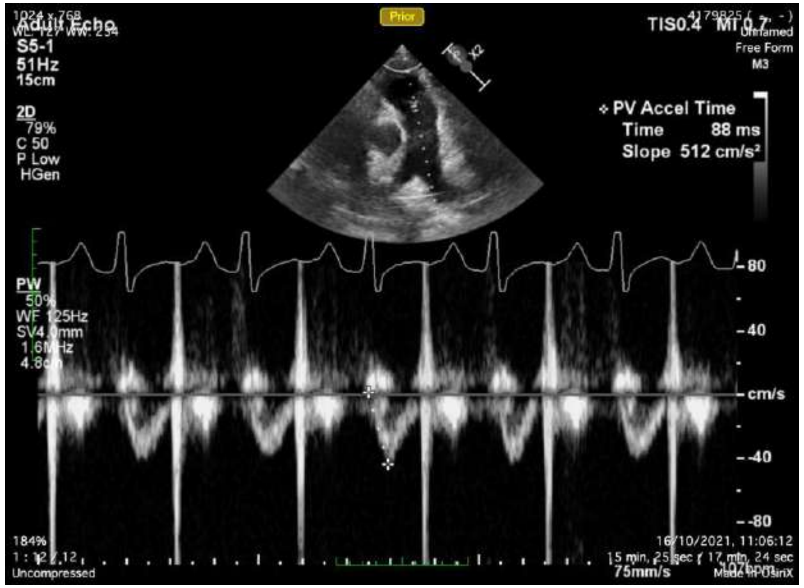
Figure 2 Heart-lung interactions. Hemodynamic effects of mechanical ventilation. The cyclic changes in left ventricular (LV) stroke volume are mainly related to the expiratory decrease in LV preload due to the inspiratory decrease in right ventricular (RV) filling. Reproduced with permission from Critical Care/Current Science Ltd [24].

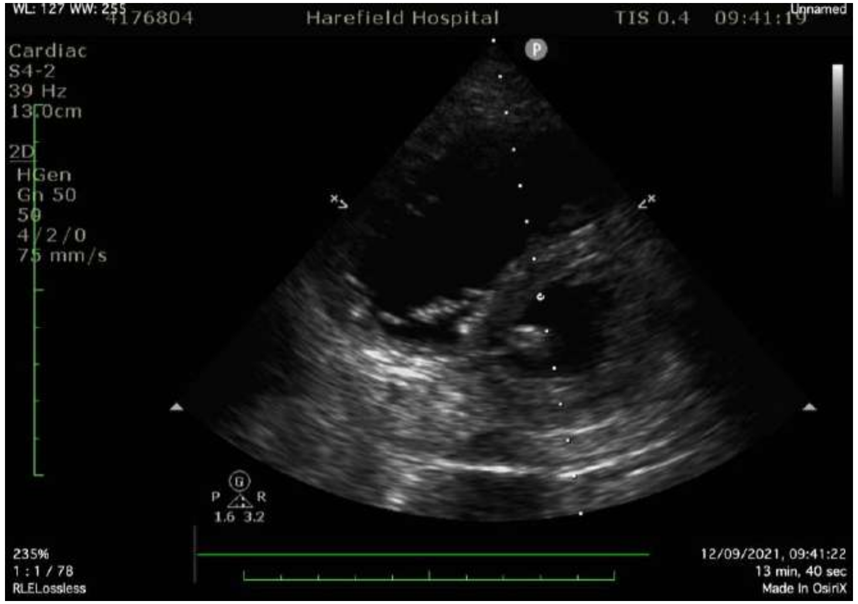
Marik et al. Annals of Intensive Care 2011

72 y/o, pneumonia, ARDS
Profound shock on increasing PEEP



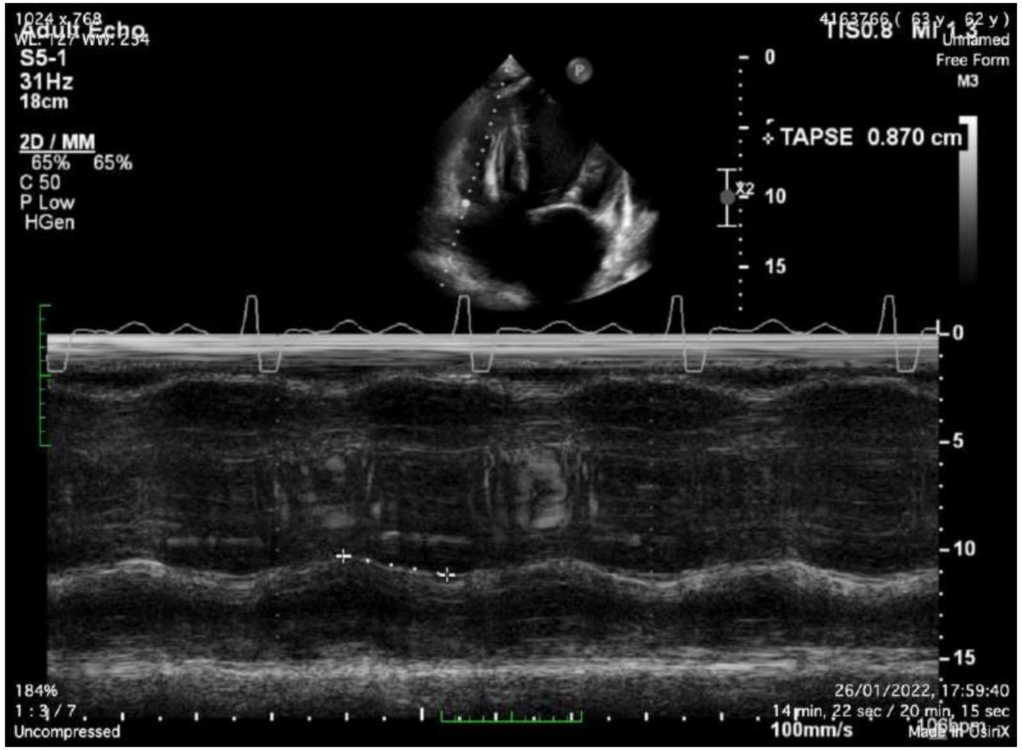
Hypoxaemia
Hypercapnoea

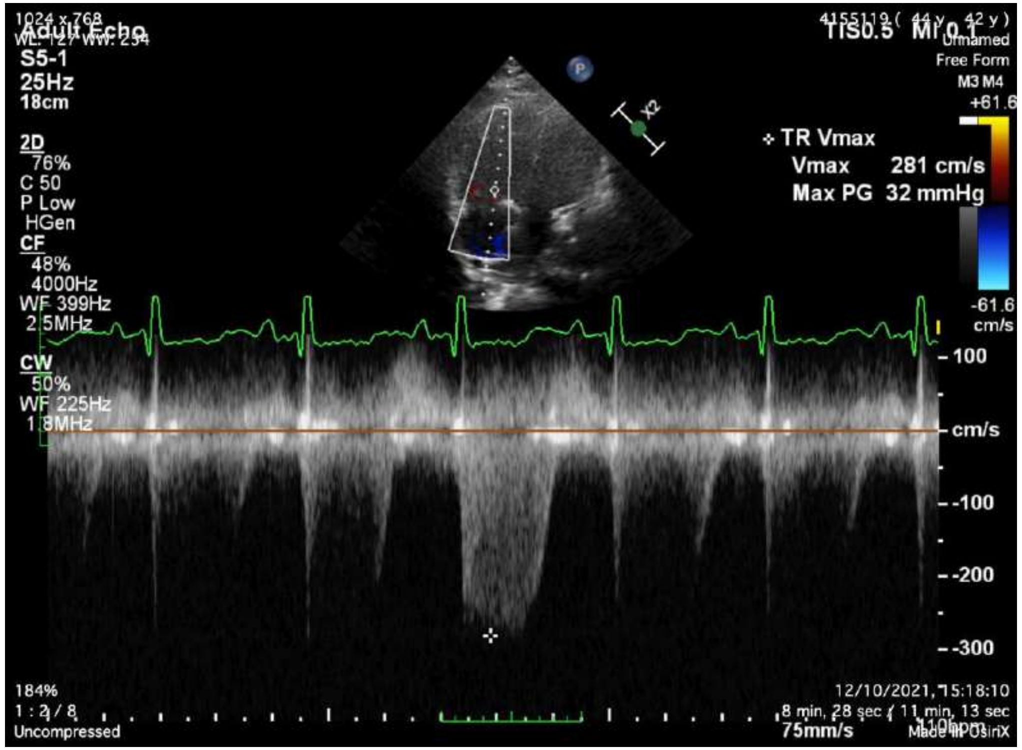




PEEP 8 → 12 cmH2O

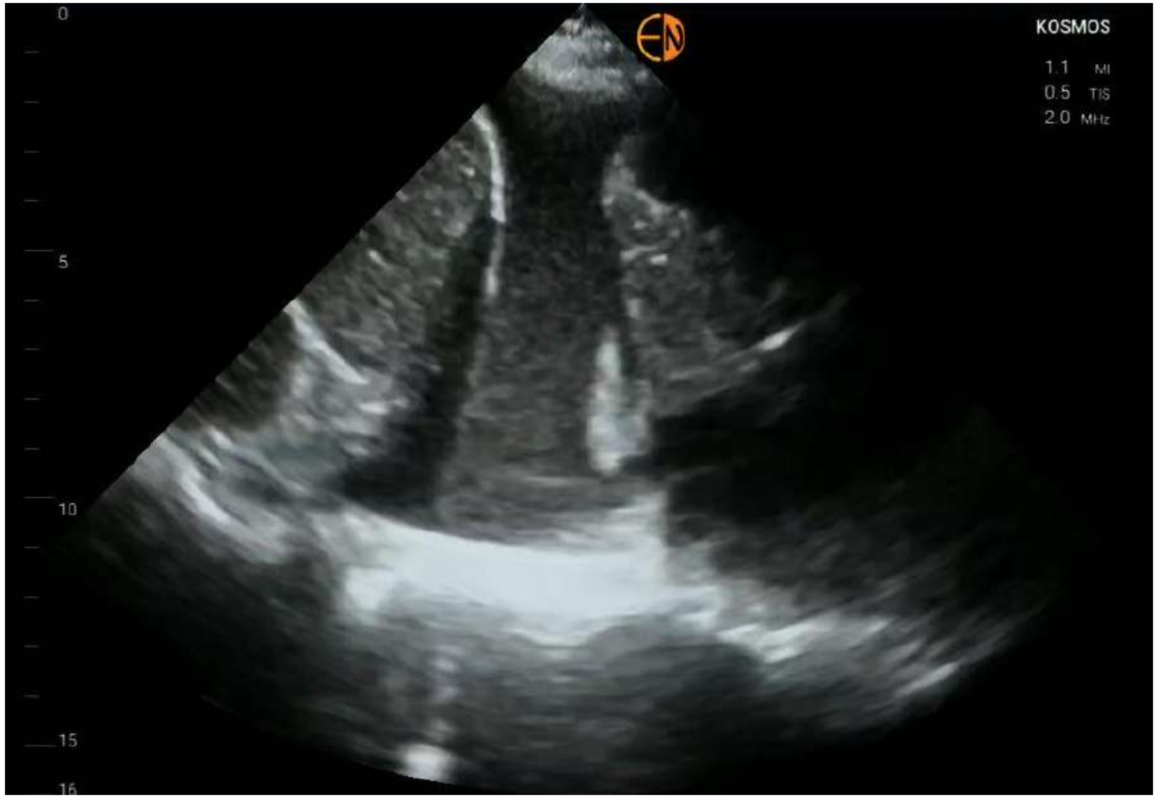
72 y/o, pneumonia, ARDS
Profound shock on increasing PEEP



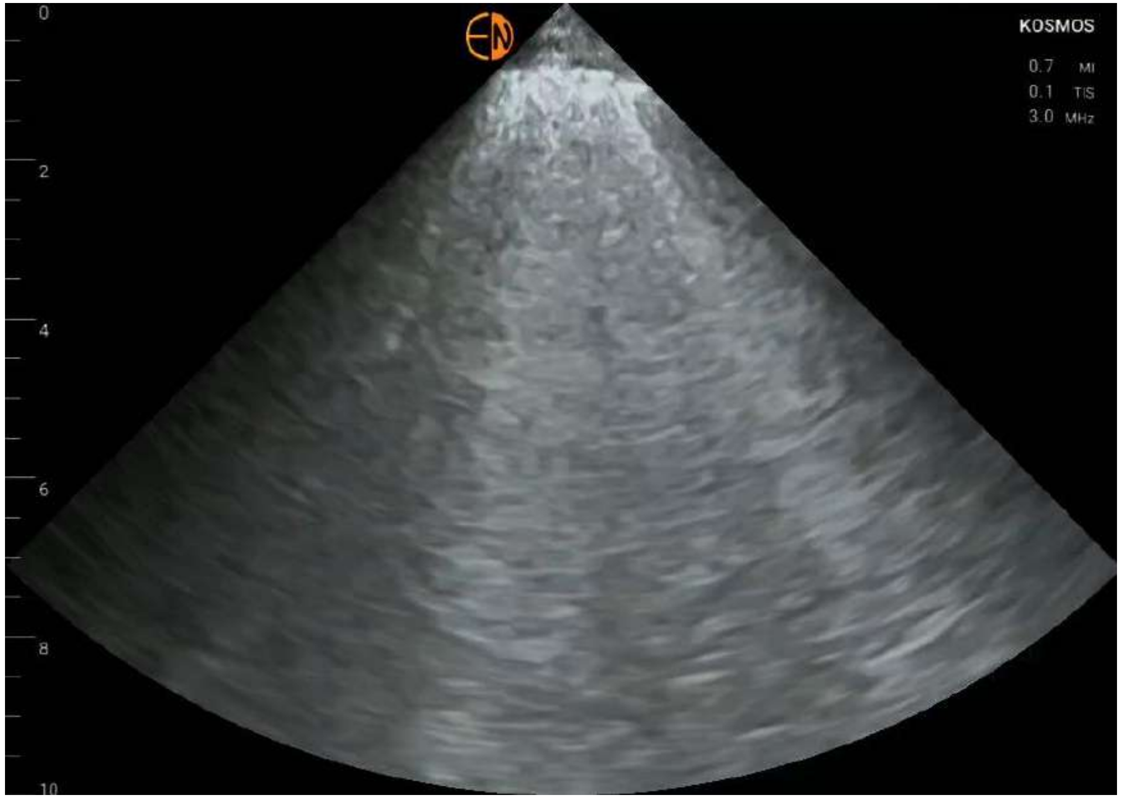


TAPSE 8 mm / PASP 42 mmHg = 0.2 → RV-PA uncoupling <0.31

SpO2 = 88% on O2 15 L/min



Rt. Sided consolidation



Bilateral coalescent B lines

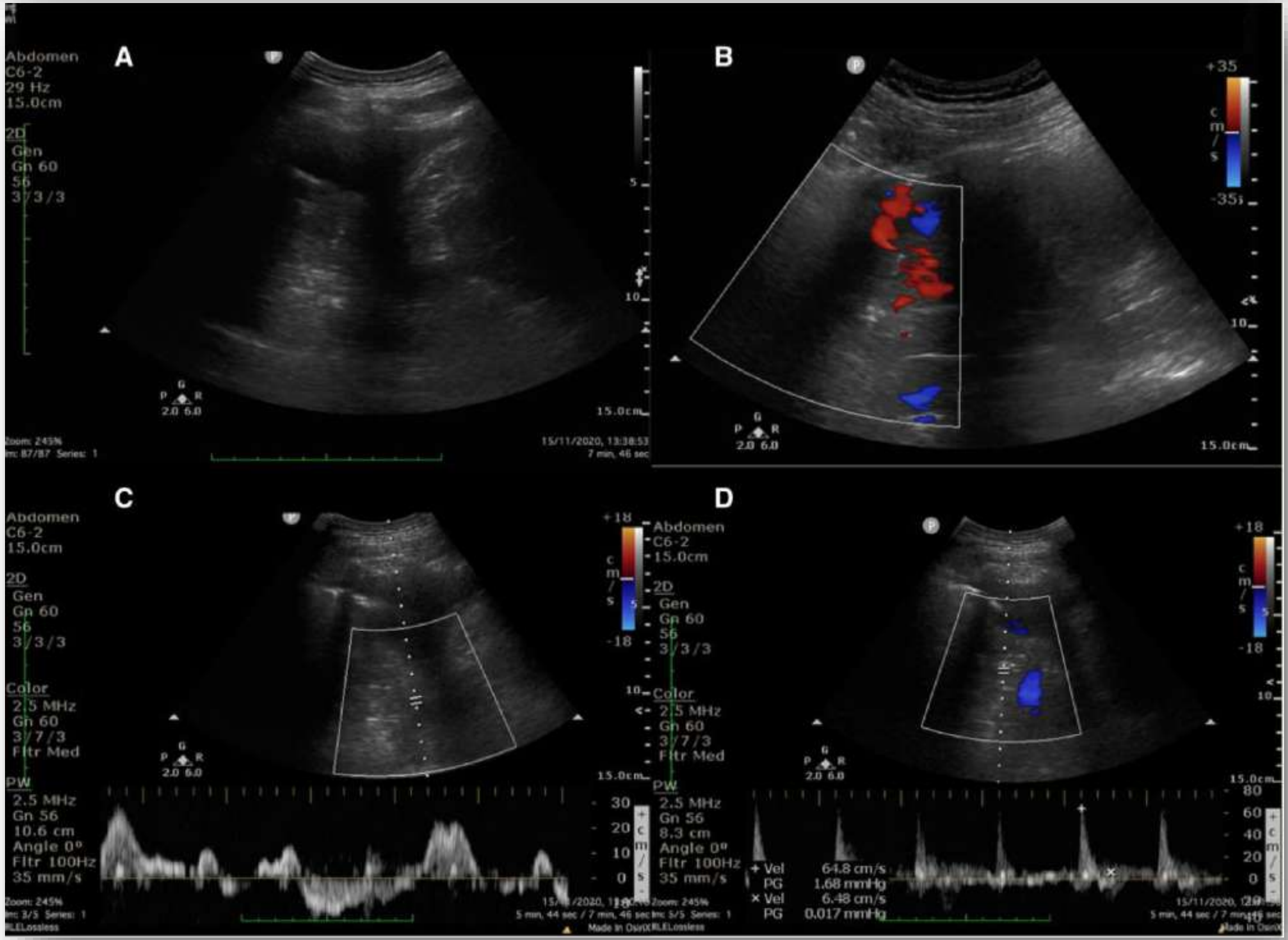
JOURNAL ARTICLE

Doppler demonstration of ventilation–perfusion mismatching due to intrapulmonary shunting

Hatem Soliman-Aboumarie

European Heart Journal - Cardiovascular Imaging, Volume 23, Issue 2, February 2022, Page e89, https://doi.org/10.1093/ehjci/jeab256

Published: 02 December 2021 **Article history** ▾



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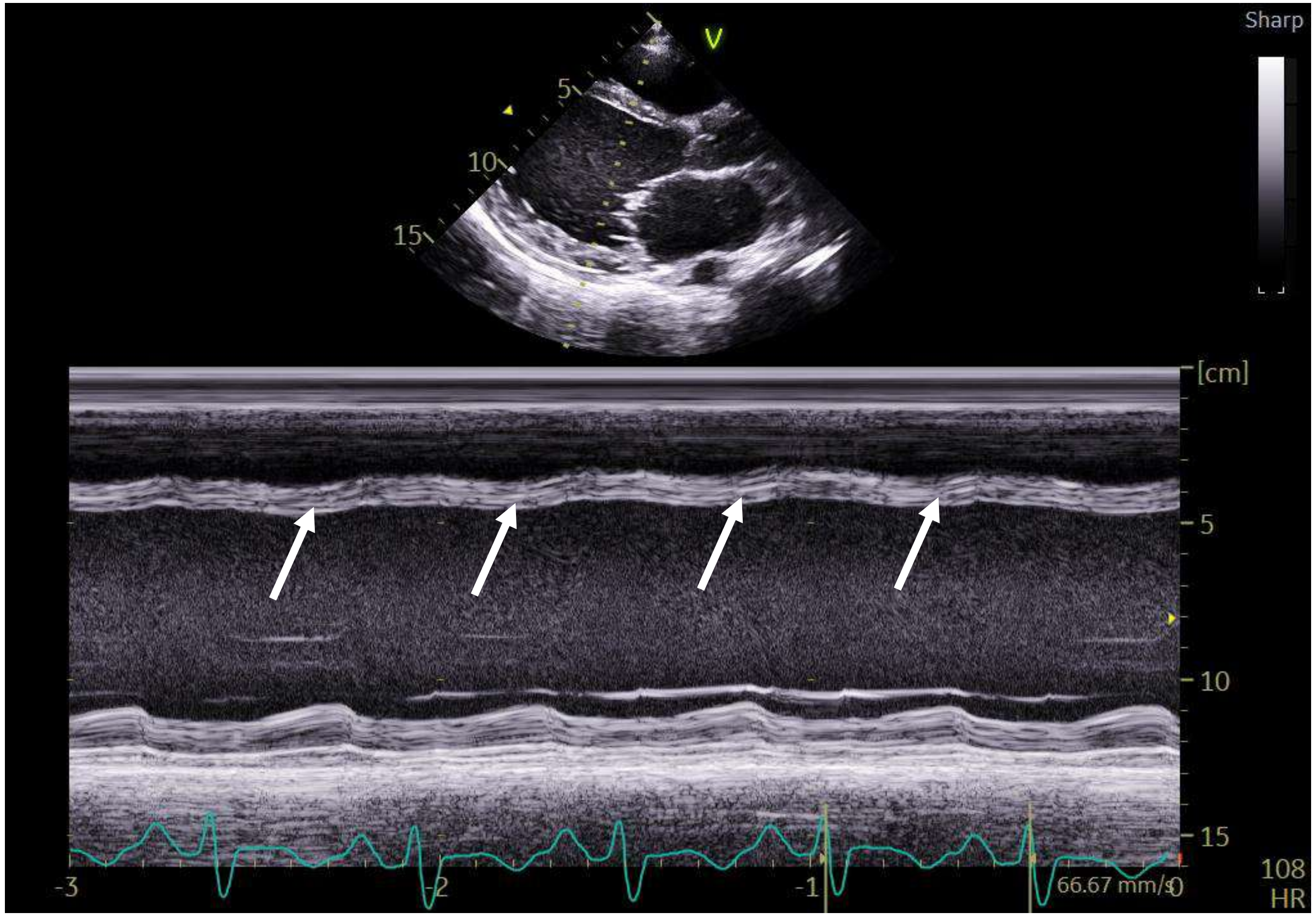
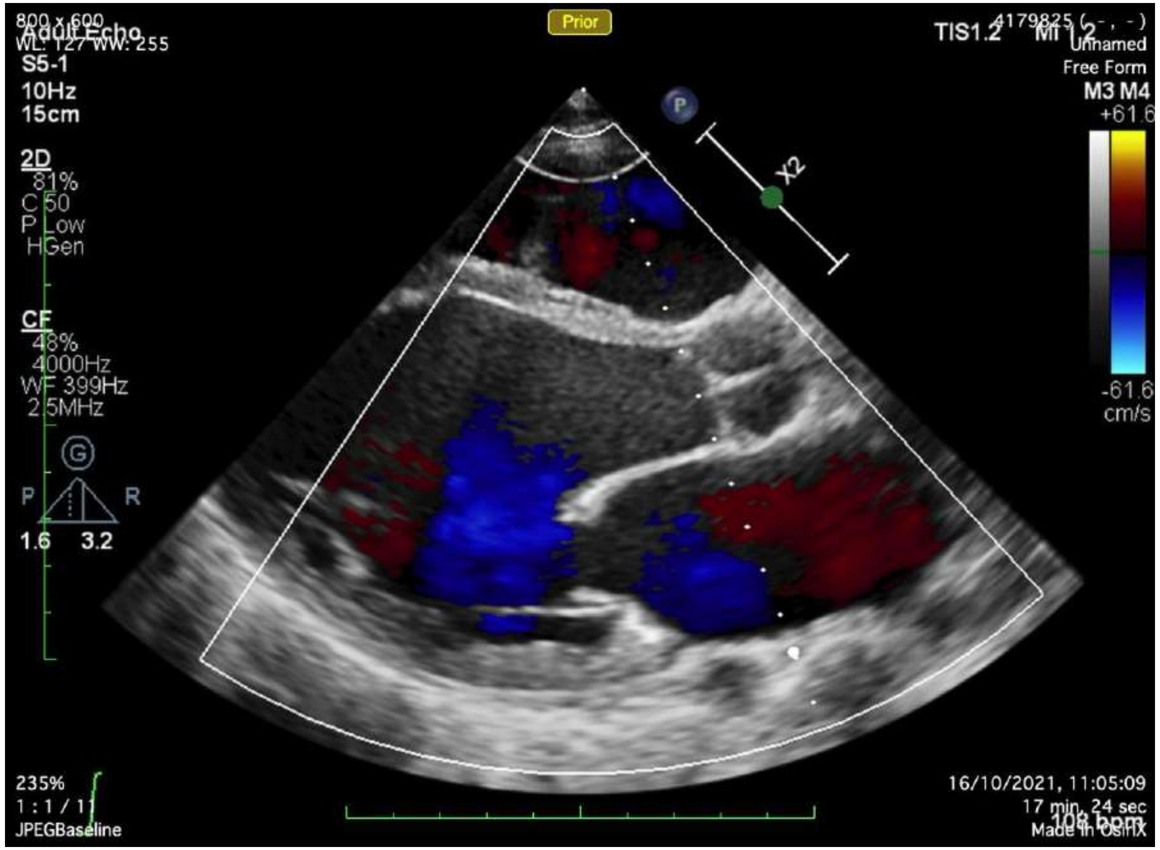
45 y/o suspected ARDS, 10 yr post MVR for rupture cord

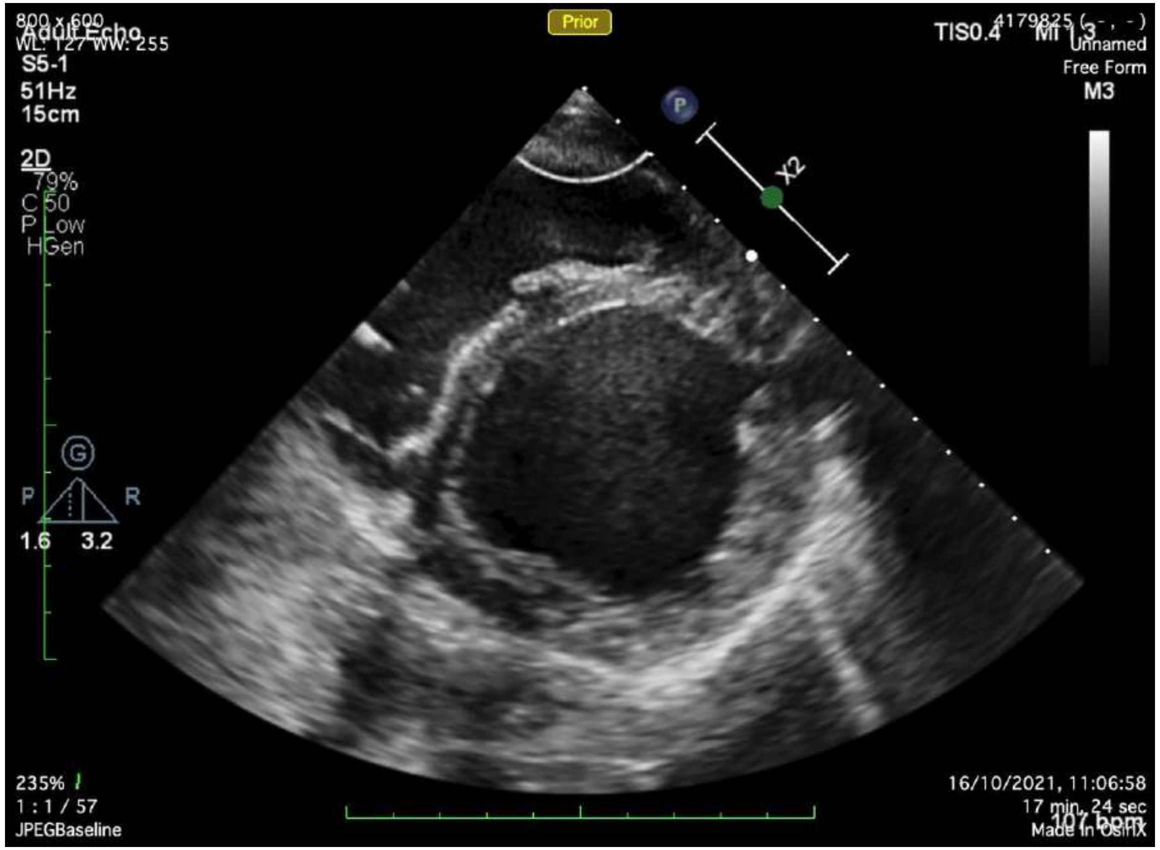


M-mode can provide vital information

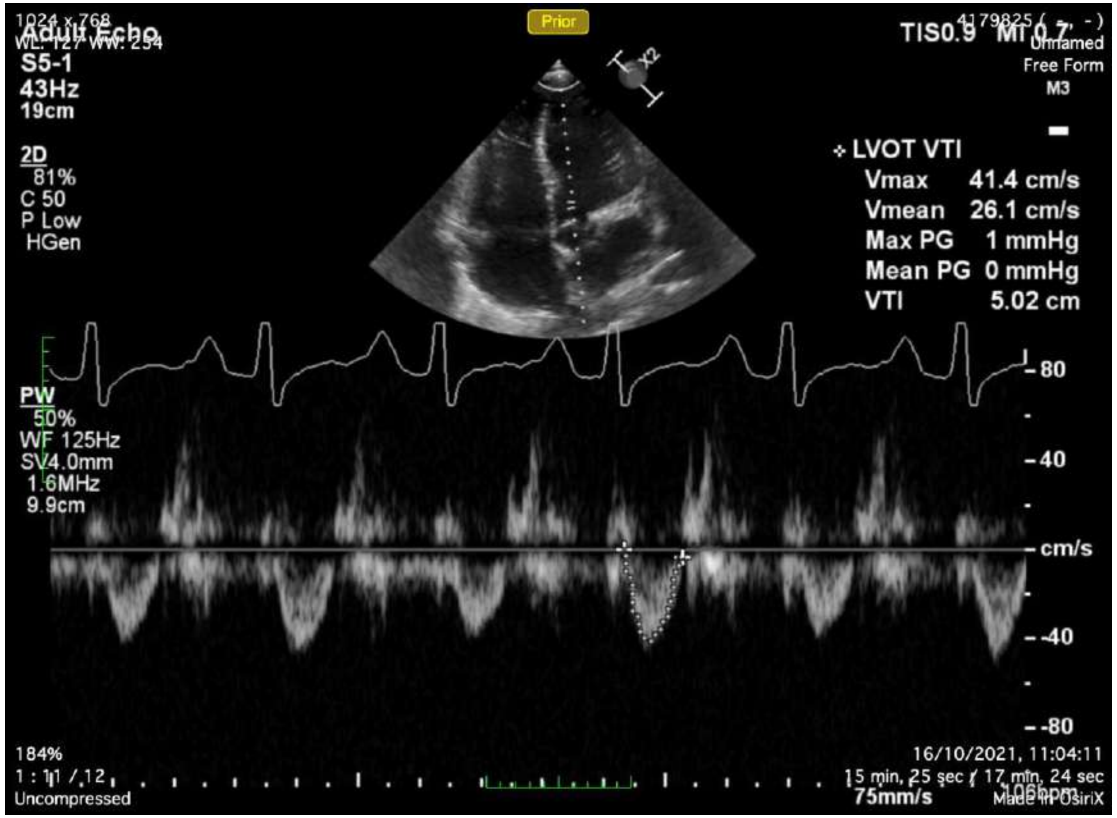
TTE → no MR seen, however, signs of LV volume overload and high LVEDP (? mitral regurgitation)
TOE → paravalvular leakage due to dehiscence of posterior annulus

25 y/o SOB, few wks after flu, worse perfusion

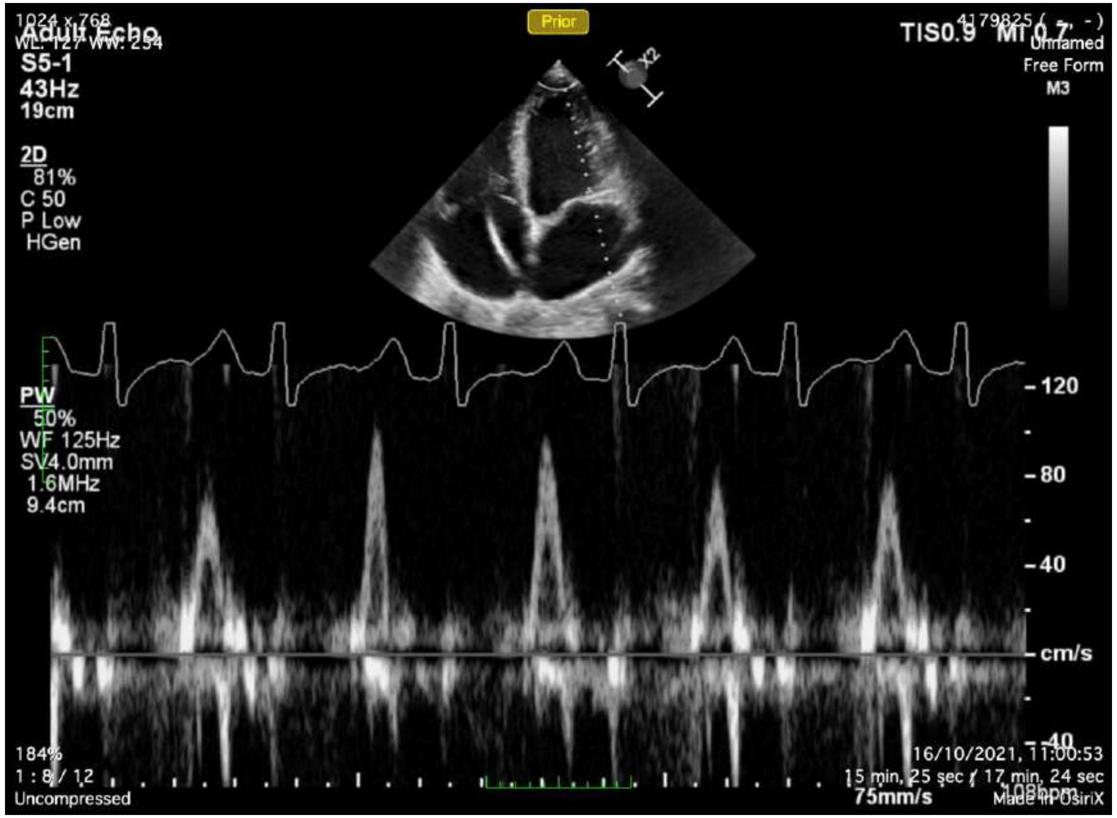




25 y/o SOB, few wks after flu, worse perfusion



LVOT VTI 5 cm



’A’ filler = Suppressed early filling

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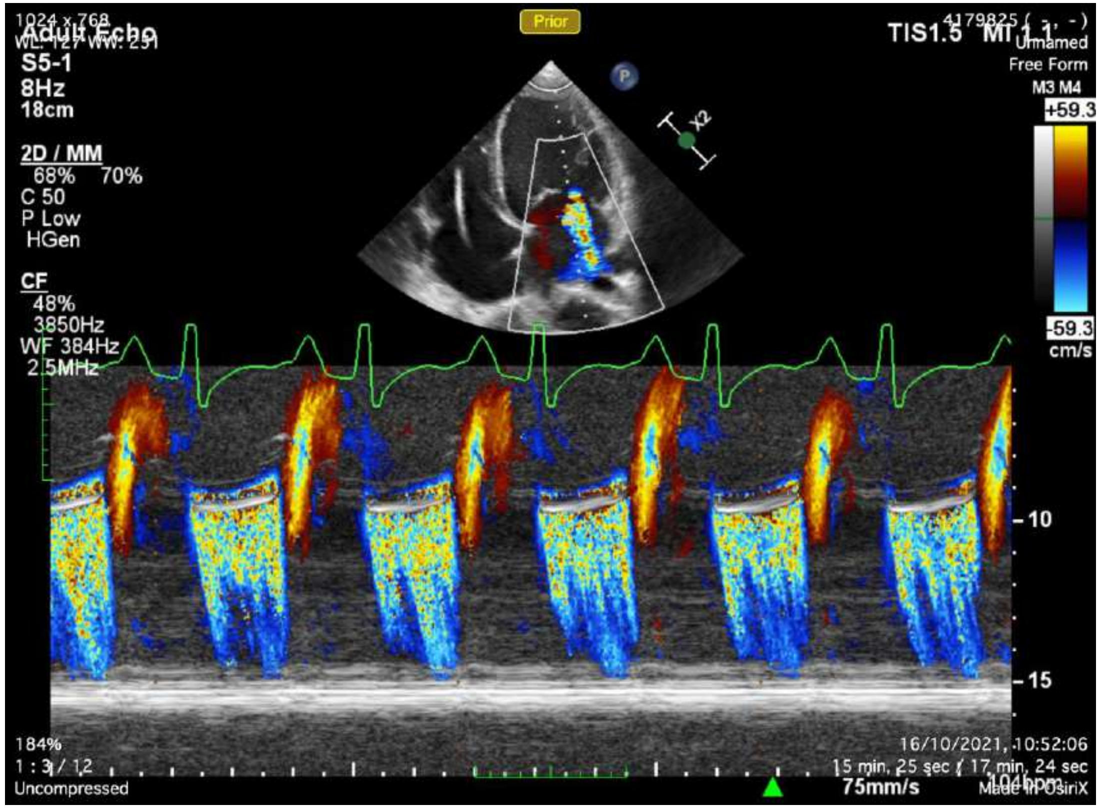
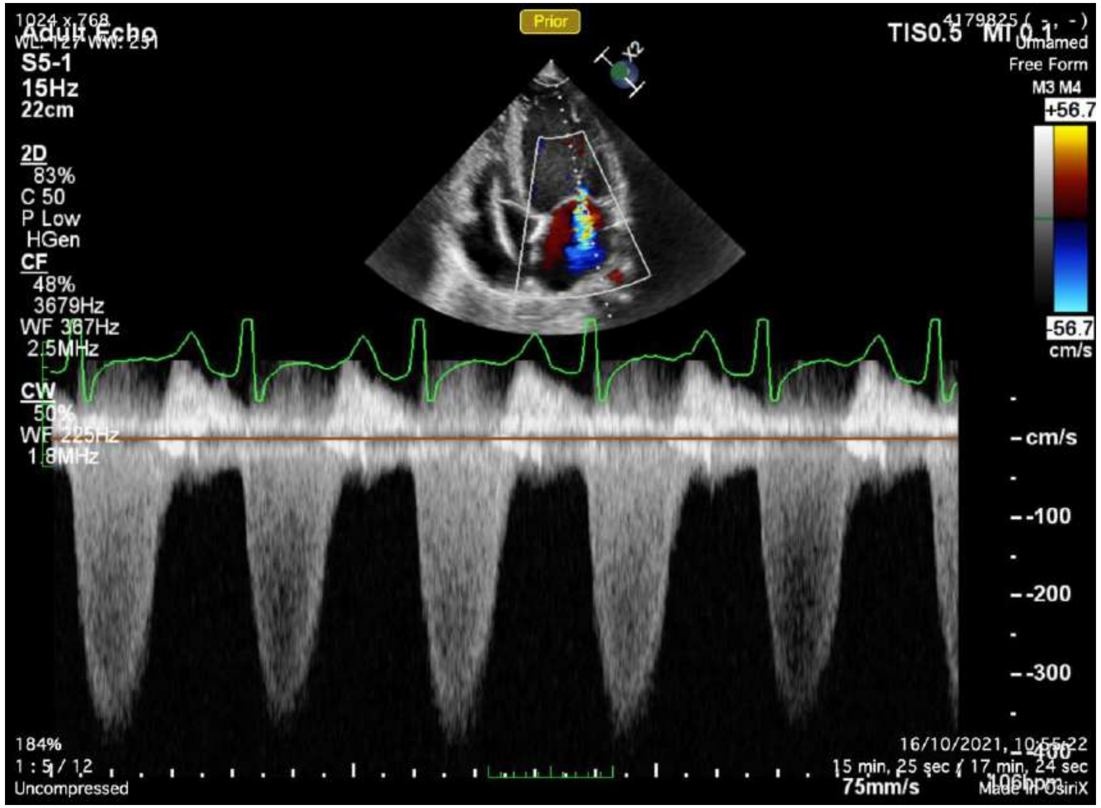
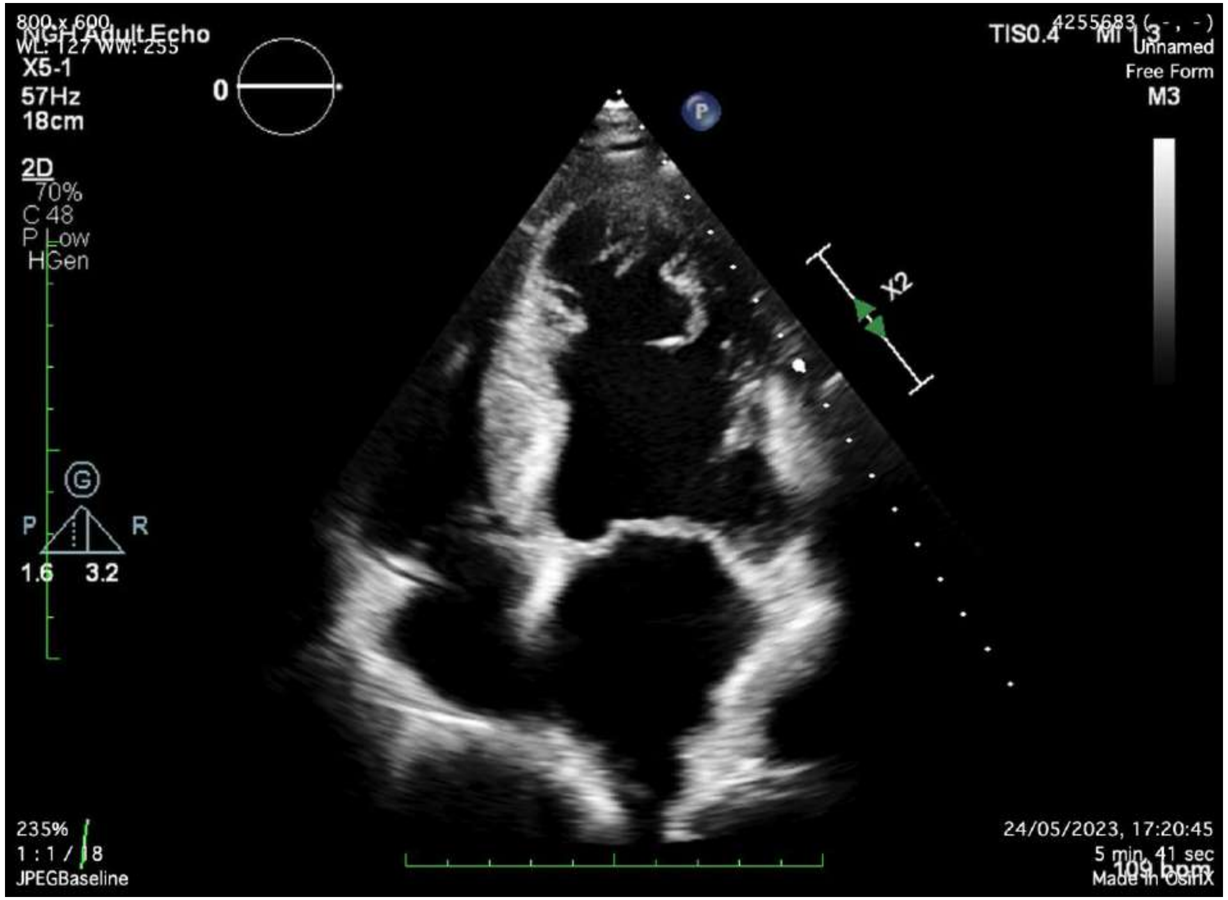
25 y/o SOB, few wks after flu, worse perfusion





Diastolic mitral regurgitation = very high LVEDP

48 y/o, post PCI to LAD, RCA

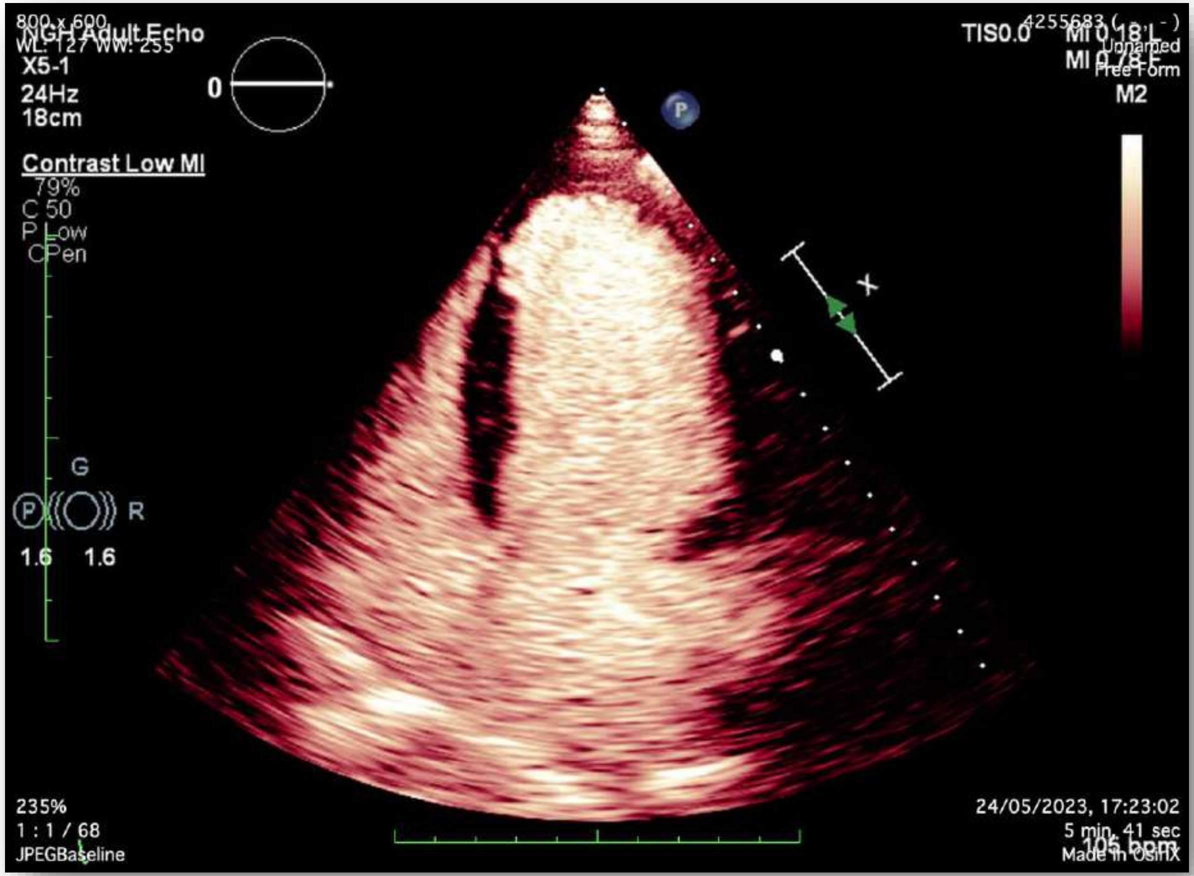


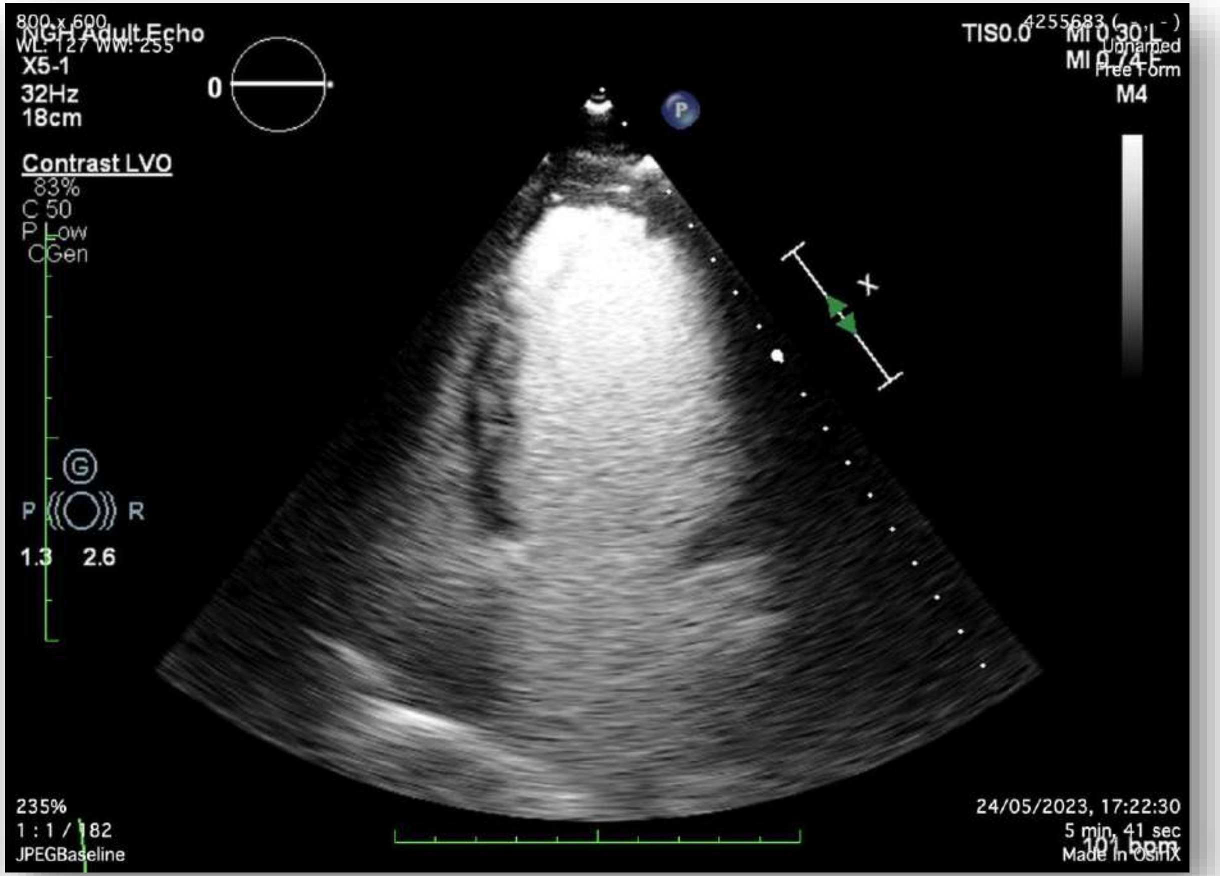
Lactate 7, BE -6, ScVO2 35%, CI 1.7

LVOT VTI 8 cm

Noradrenaline 0.10 mcg/ks/min,
Milrinone 0.2 mcg/kg/min,
Adrenaline 0.08 mcg/kg/min

Contrast echo





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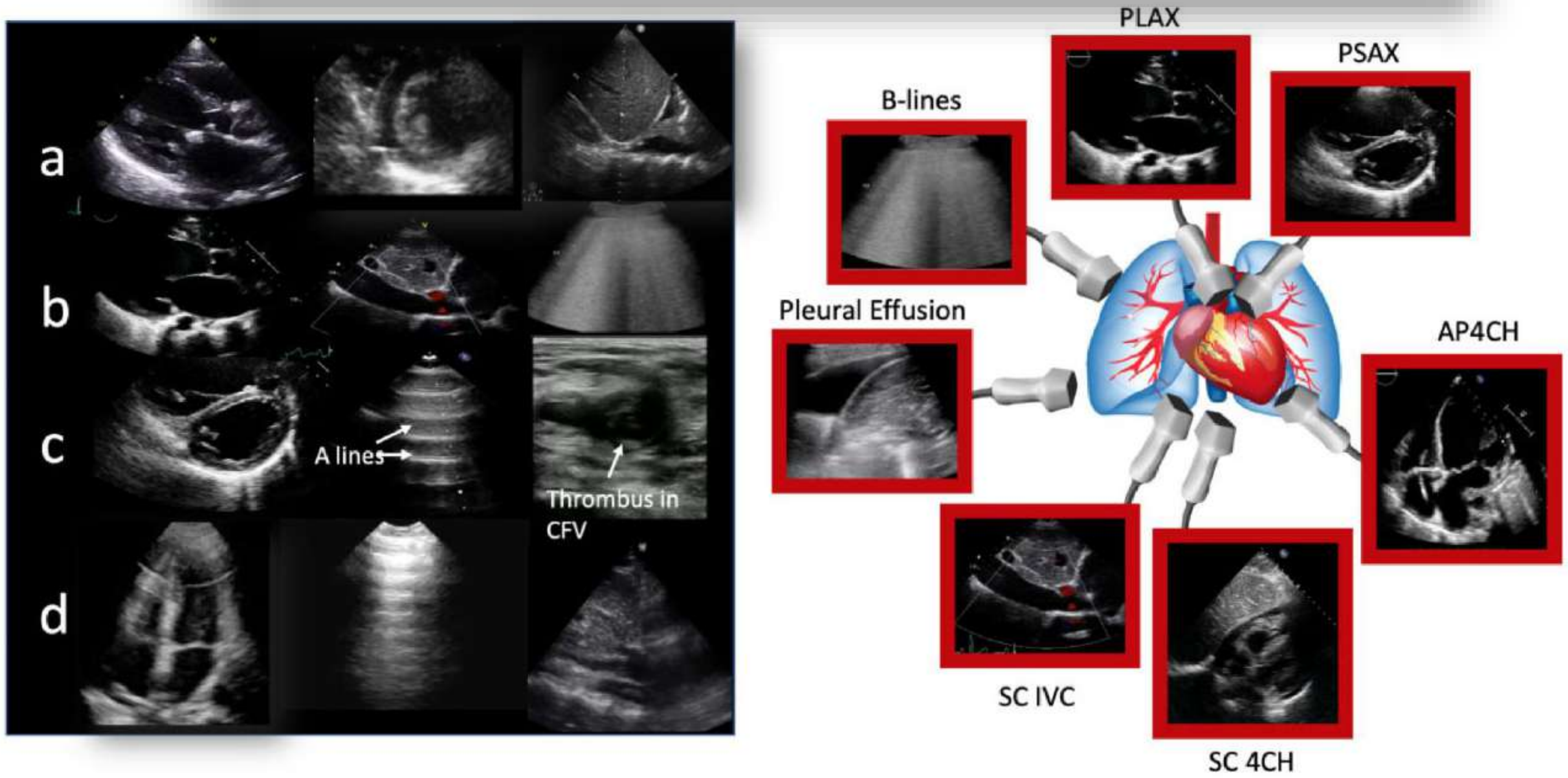
ESC European Society of Cardiology
European Heart Journal - Cardiovascular Imaging (2022) **23**, 150–153
doi:10.1093/ehjci/jeab149

HOW TO

How-to: Focus Cardiac Ultrasound in acute settings

Hatem Soliman-Aboumarie [1*], Ole-A. Breithardt[2], Luna Gargani[3], Paolo Trambaiolo[4], and Aleksandar N. Neskovic[5]



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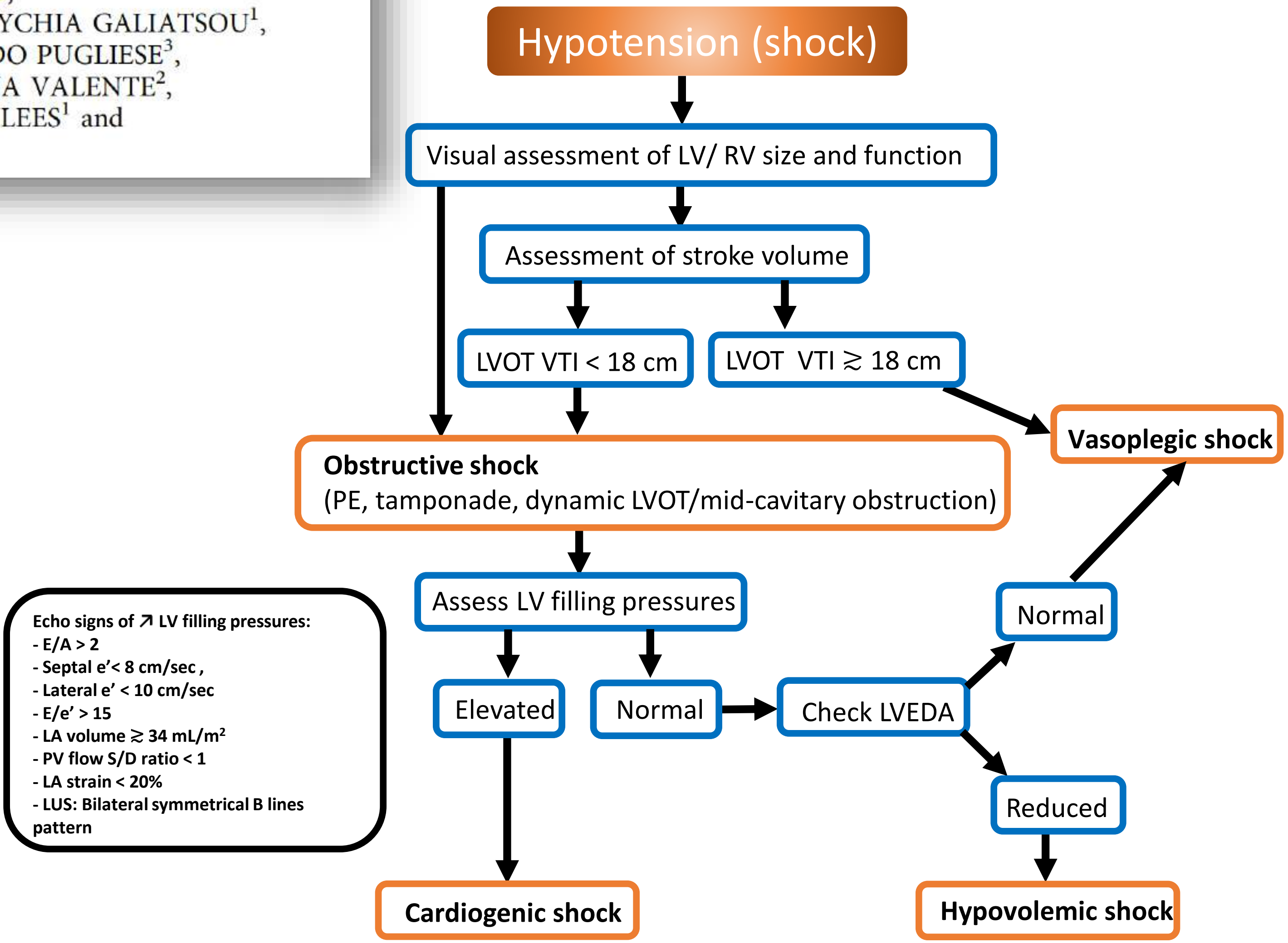
AKADÉMIAI KIADÓ

Echocardiography in the intensive care unit: An essential tool for diagnosis, monitoring and guiding clinical decision-making

IMAGING

HATEM SOLIMAN-ABOUMARIE[1,4†*], MARIA CONCETTA PASTORE[2†], EFTYCHIA GALIATSOU[1], LUNA GARGANI[3], NICOLA RICCARDO PUGLIESE[3], GIULIA ELENA MANDOLI[2], SERAFINA VALENTE[2], ANA HURTADO-DOCE[1], NICHOLAS LEES[1] and MATTEO CAMELI[2]



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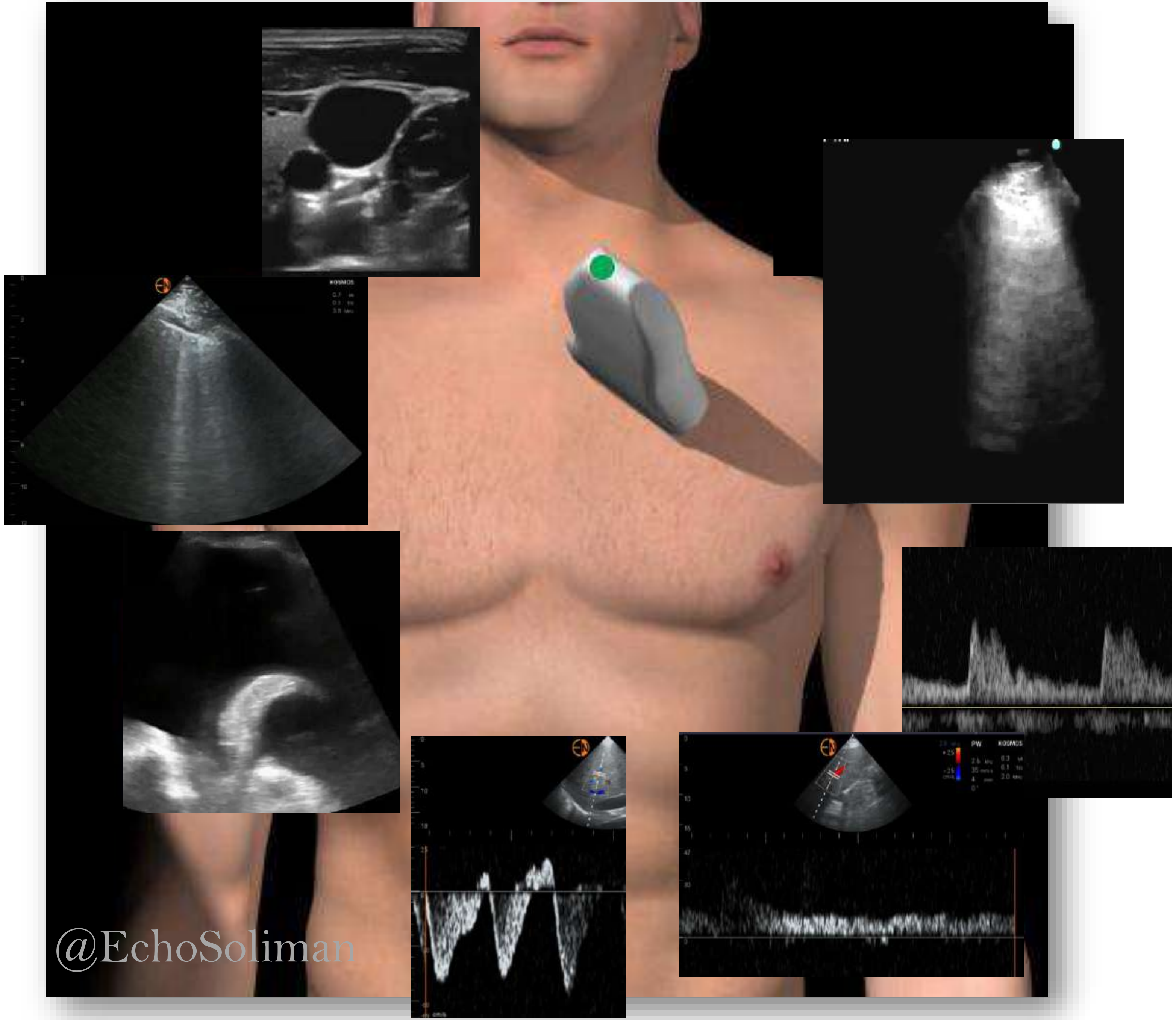
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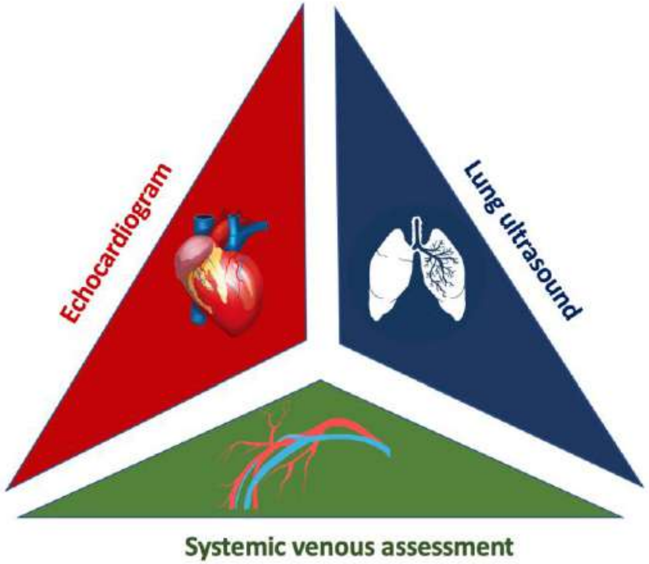
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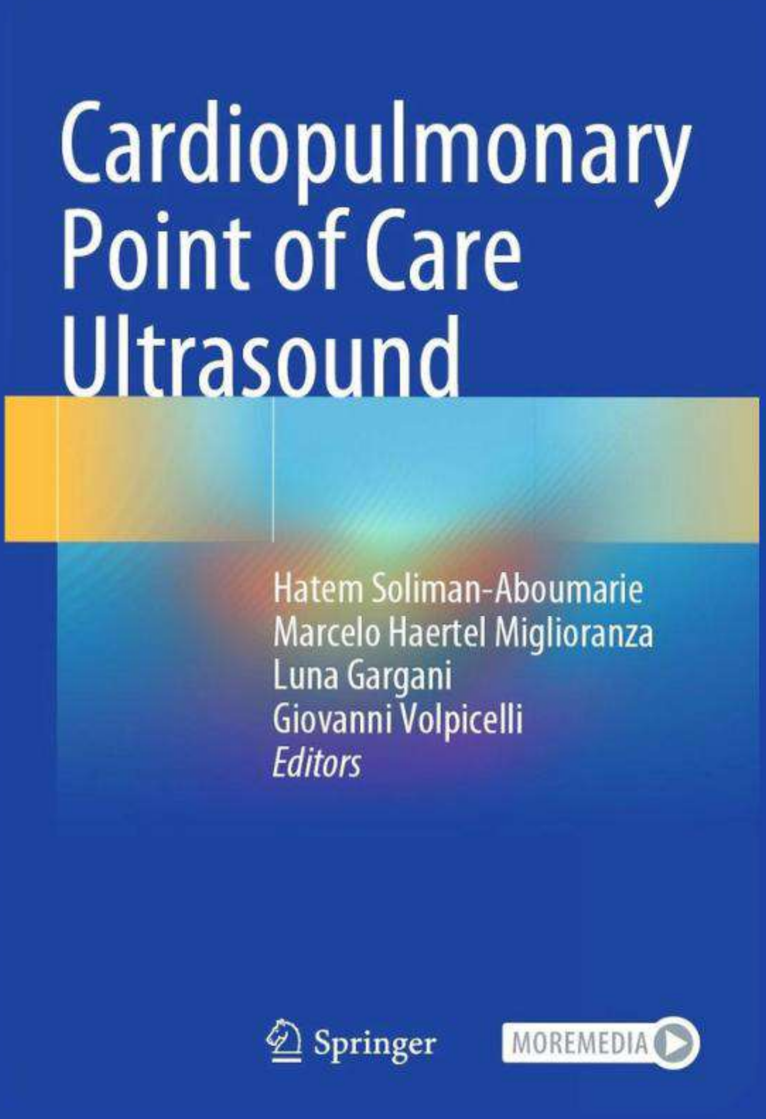

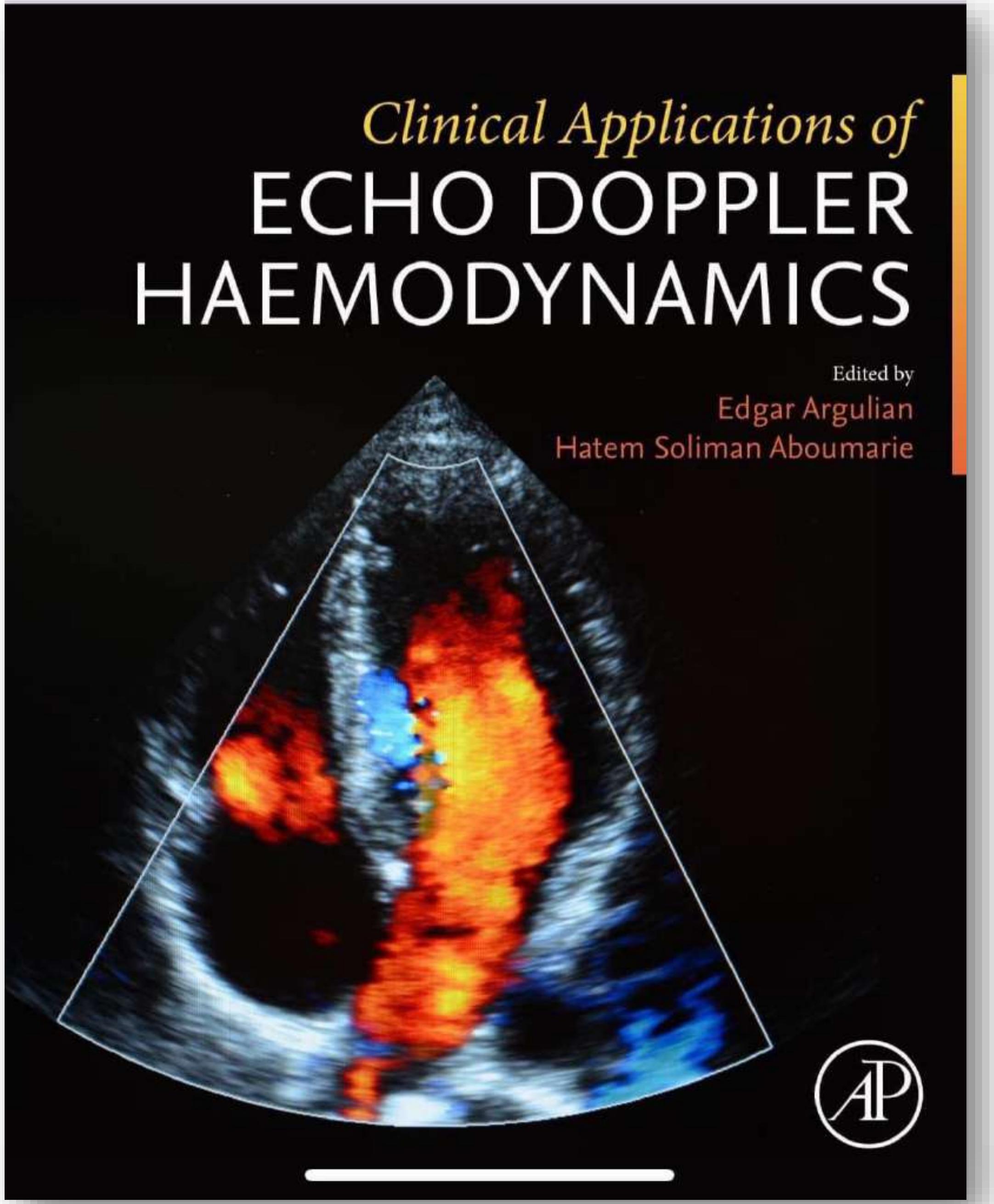
Clinical Applications of
ECHO DOPPLER
HAEMODYNAMICS
Edited by
Edgar Argulian
Hatem Soliman Aboumarie

Take Home Messages

- Echo enables phenotyping of cardiogenic shock
- Multimodal approach (cardiac, lung and VeXUS)
- Functional haemodynamic echo arena is evolving
- Training and accreditation is essential
- Further research and validation is awaiting

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Advanced echo in shock states

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