Alternative route to the diagnosis

Multimodality imaging

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- And
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CSANZ New Zealand
Annual Scientific Meeting

Energy Events Centre,
Rotorua | 23-25 May 2025

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Disclosures

- None

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Case: Mr T 32yo Male

- Presentation with acute Left sided weakness and altered sensation
  - Onset over ~1-2hours.
  - Followed by gradual improvement – No objective signs at time of exam

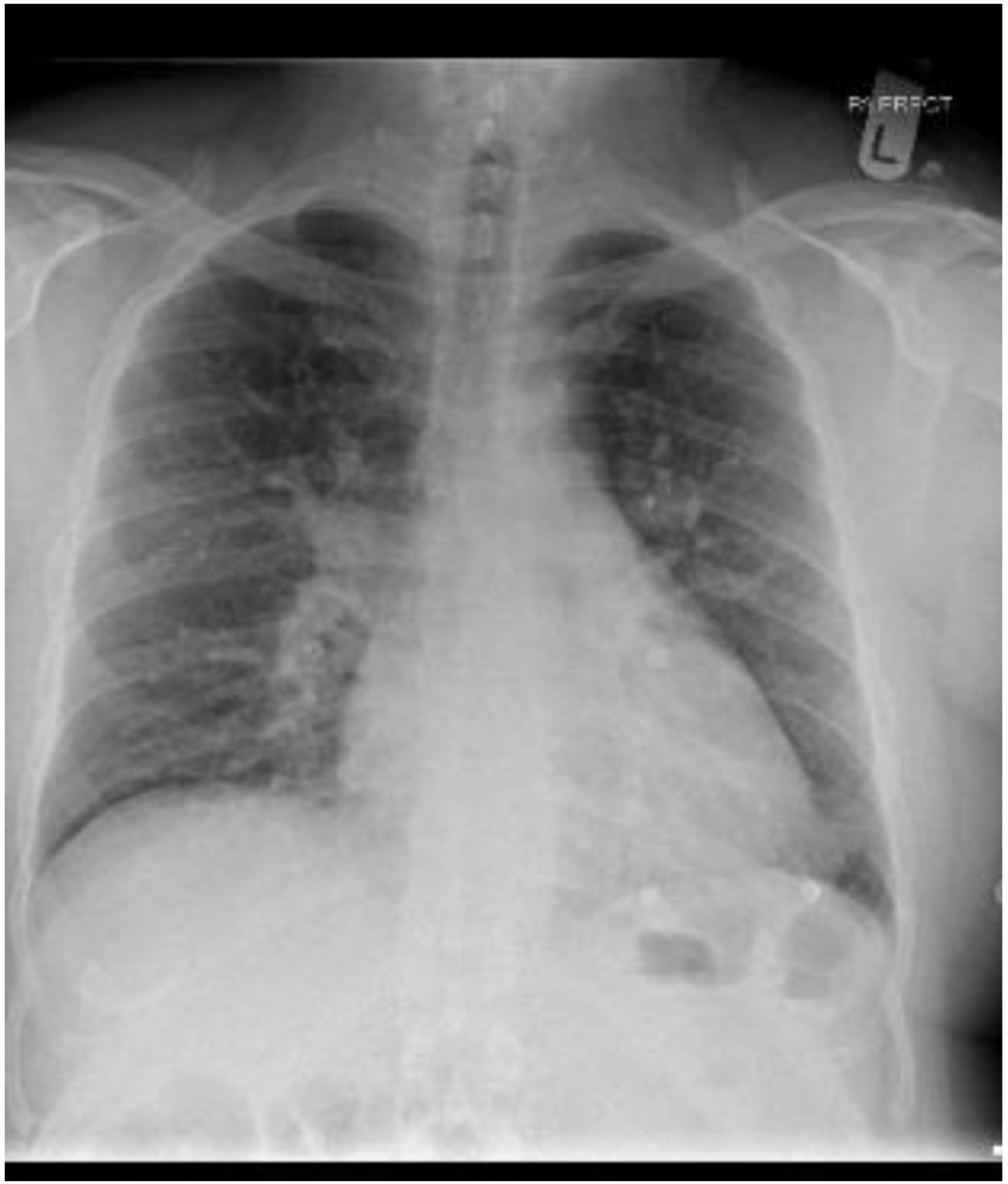
Background

- Previously well
- Murmur detected in childhood – thought to be benign (no investigations performed at the time)
- Very active– played rugby up until 7 years ago stopping due to family commitments
- On no regular medications

Initial Investigations

- Hb 189, WCC 6.2, neut 4.1
- Na 138, K 4.4, Cr 92, eGFR > 90
- CRP 8

2/04/24

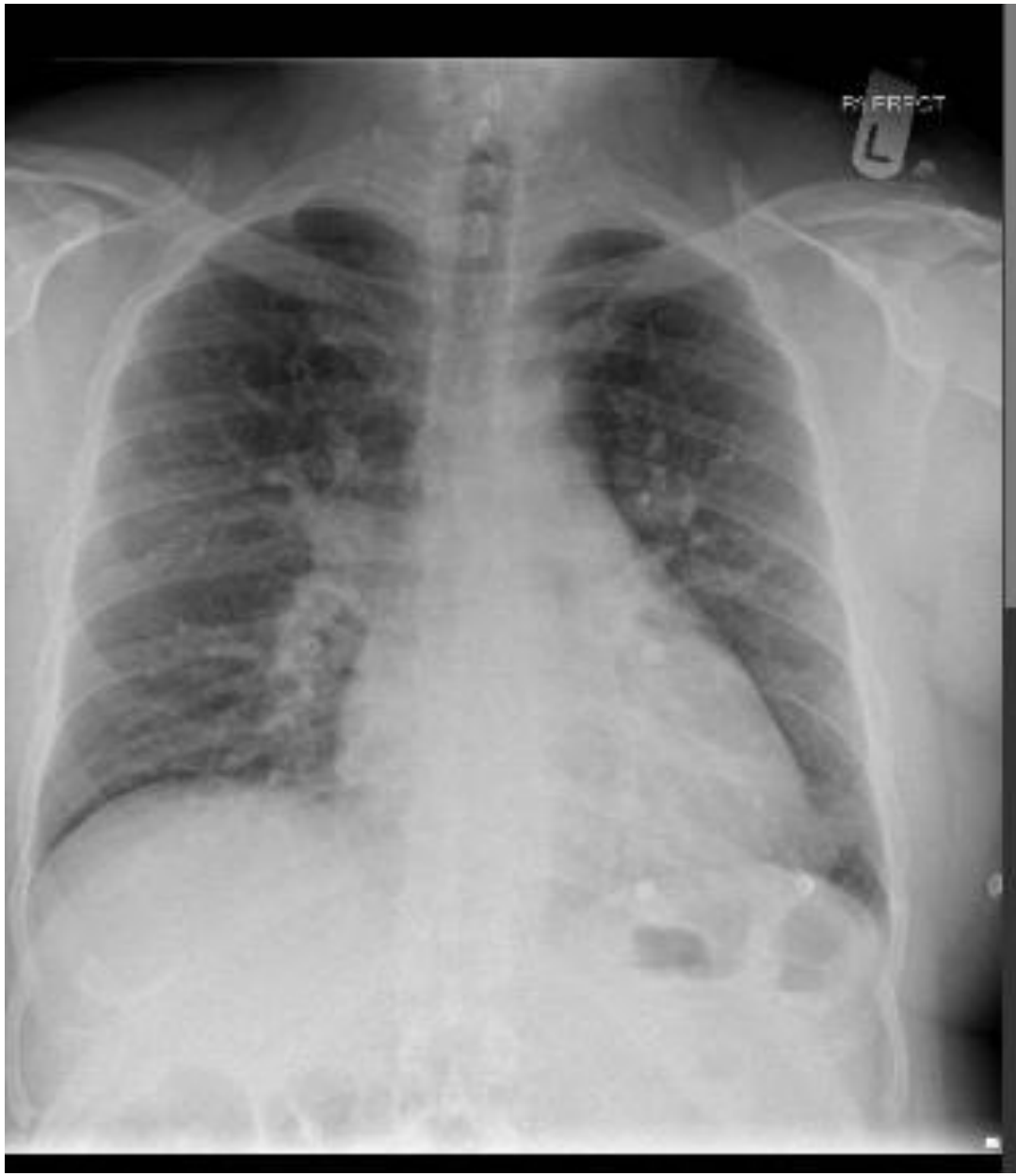


Report:
Moderate cardiomegaly, Ct ratio is 0.56cm.
Prominent hilar regions bilaterally.
Difficult to exclude some pulmonary enlargement with associated interstitial oedema.
No focal airspace consolidation, pleural effusion or focal lung lesion.

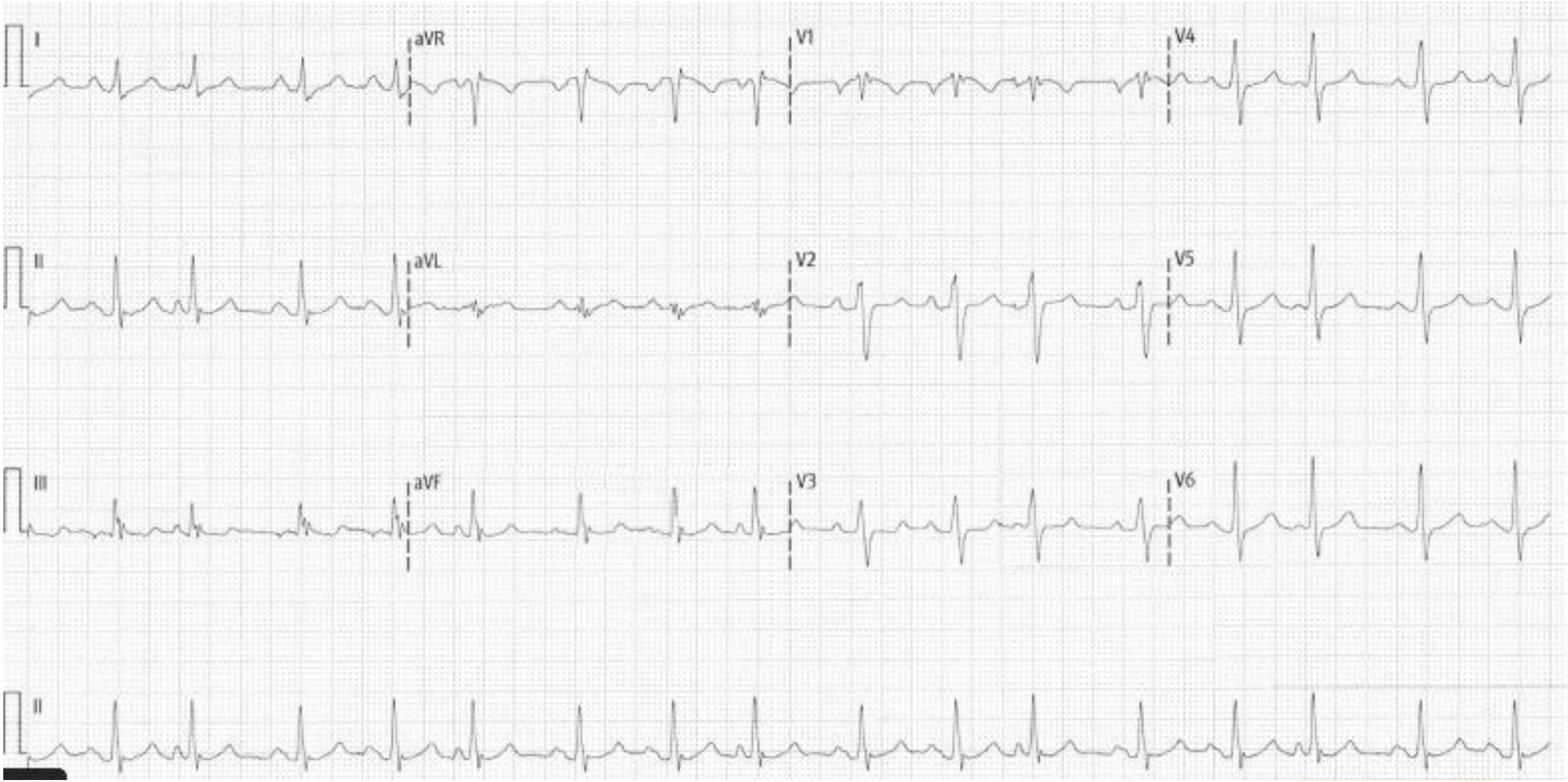
CONCLUSION:

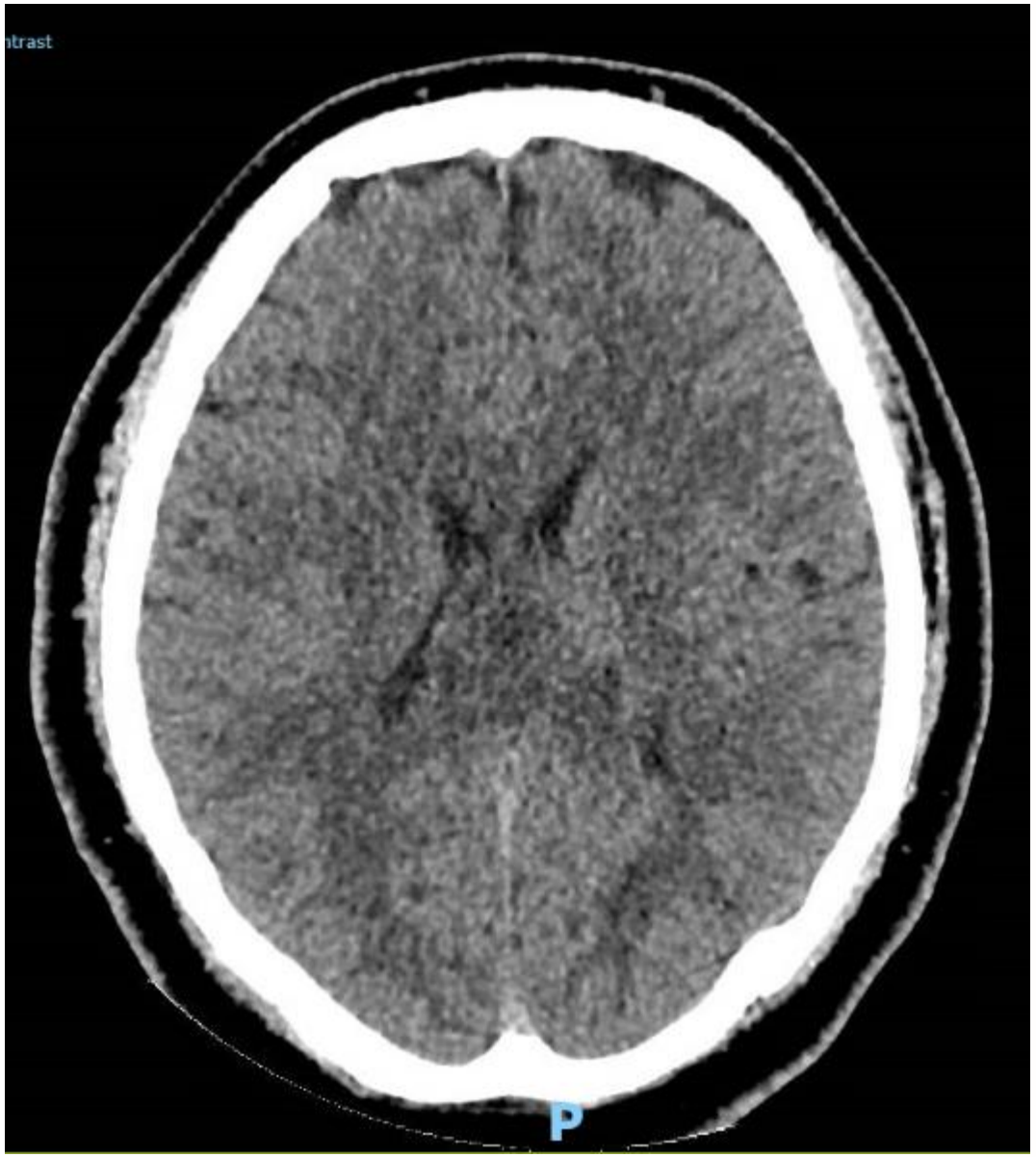
Cardiomegaly with prominent pulmonary arteries. There may be a degree central pulmonary congestion associated with this.

2/04/24



2019


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II


ntrast
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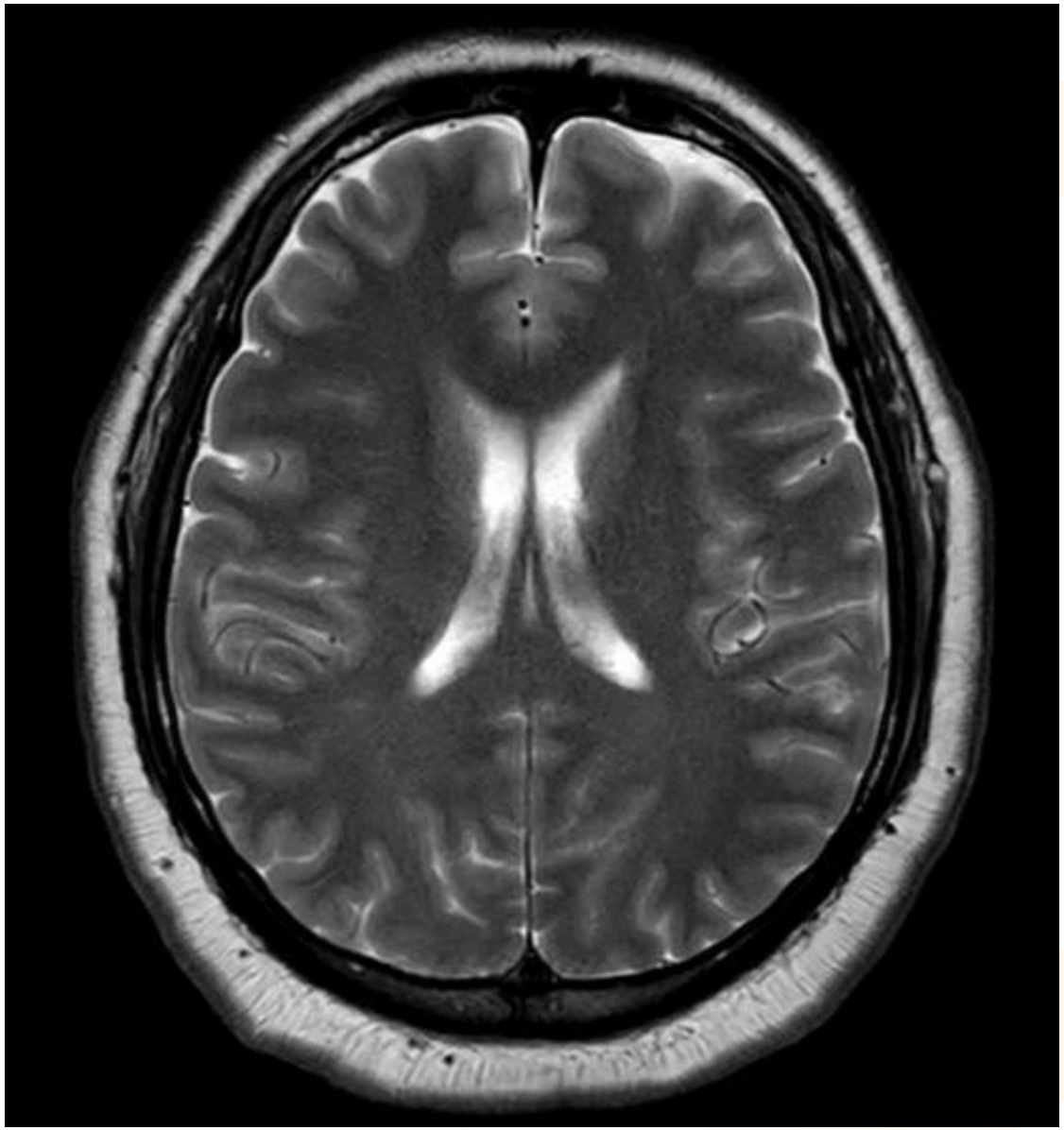
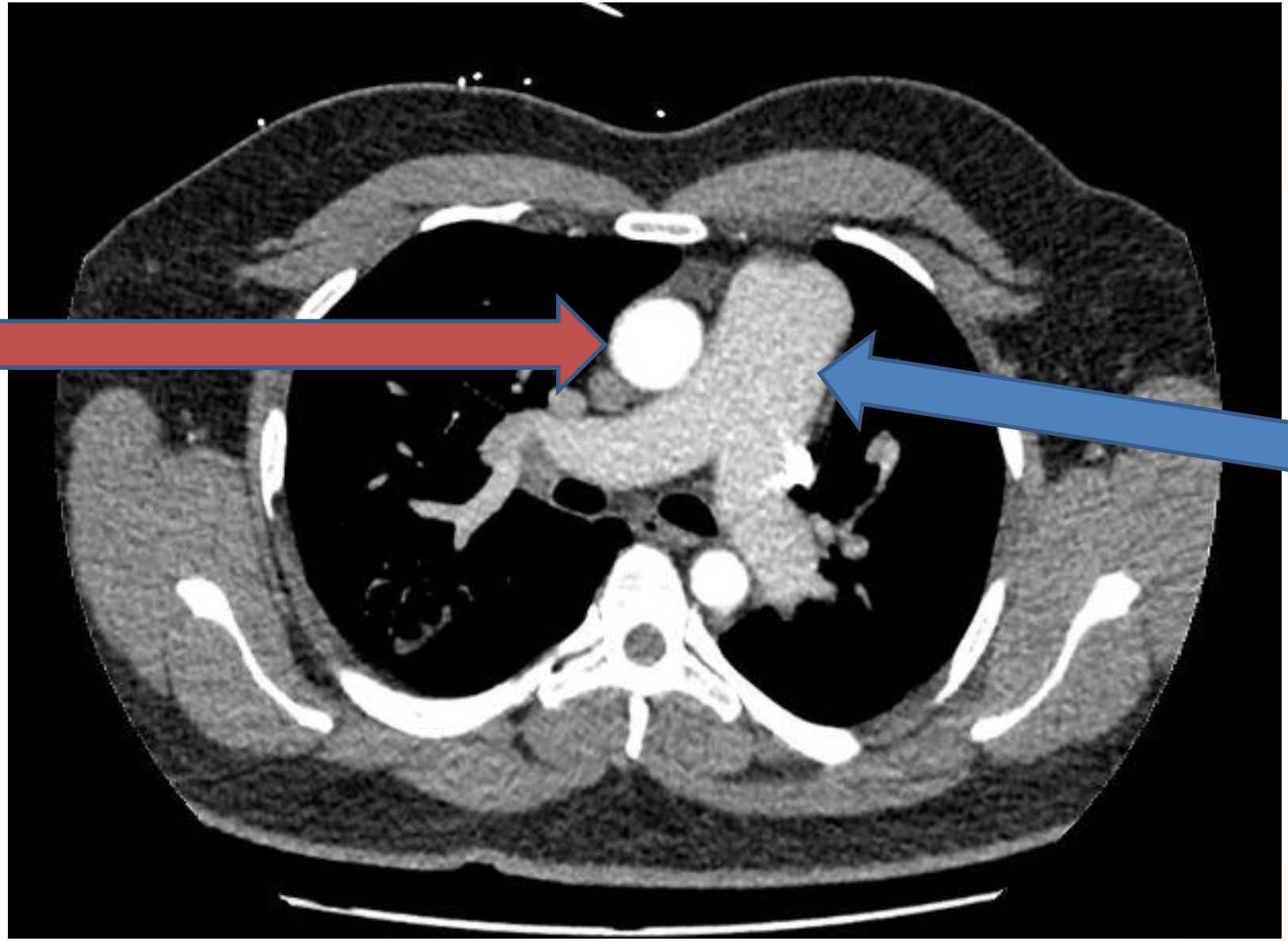


?Stroke vs TIA

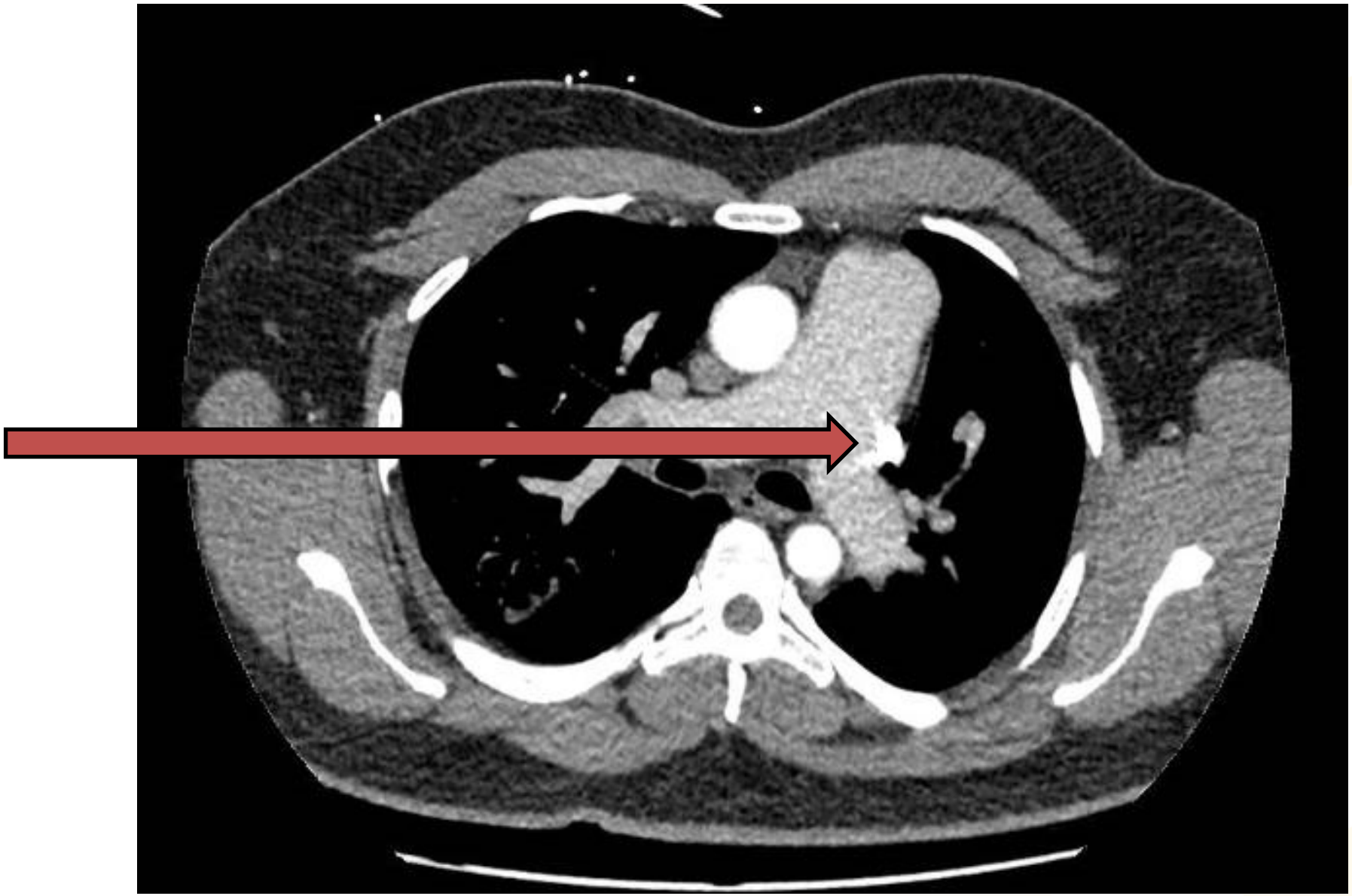
- CT head – Normal
- MRI head – Normal apart from a right cerebellar cavernoma (no acute stroke)
- CT pulmonary angiogram: Requested because of relatively low saturations

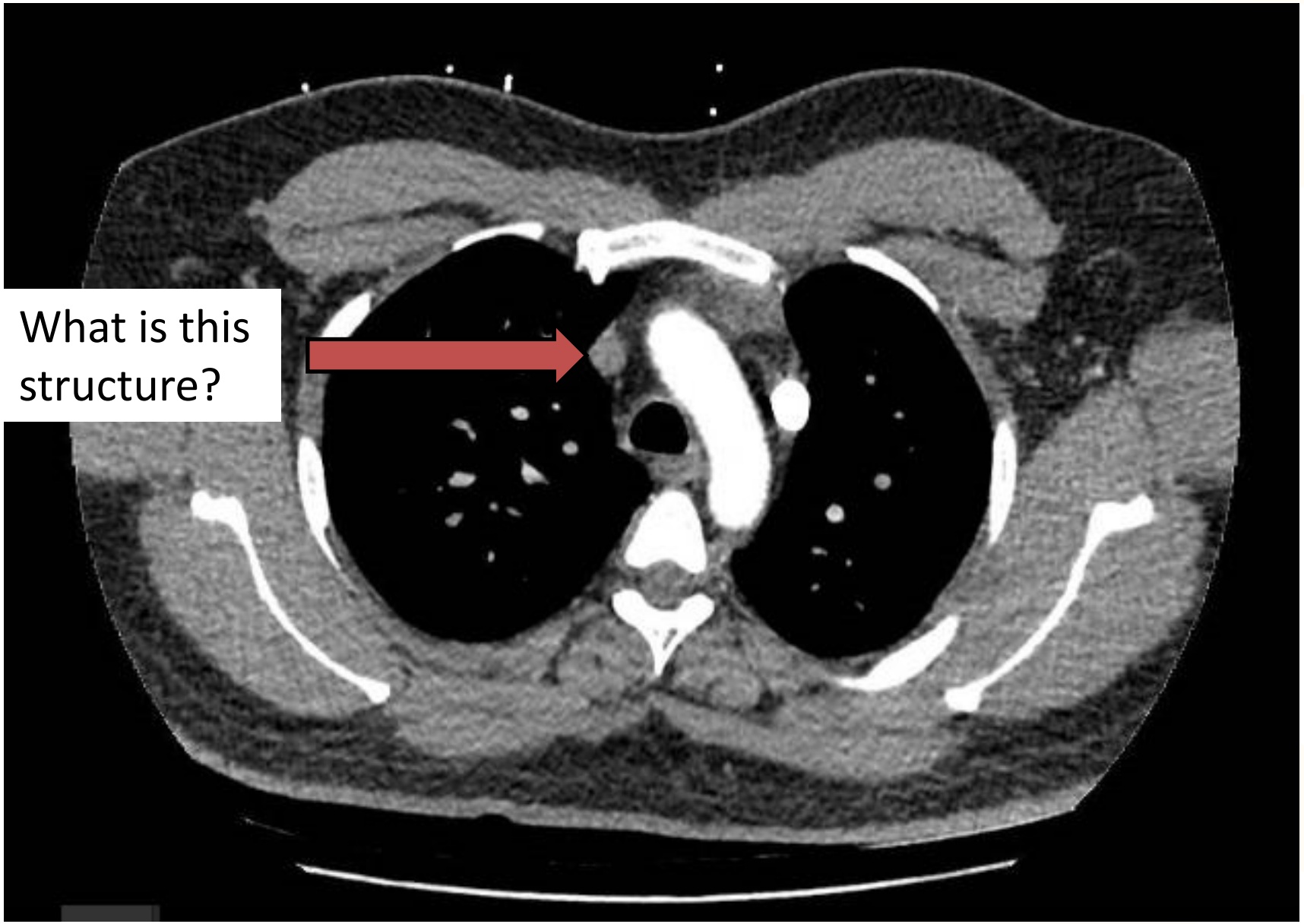
CTPA

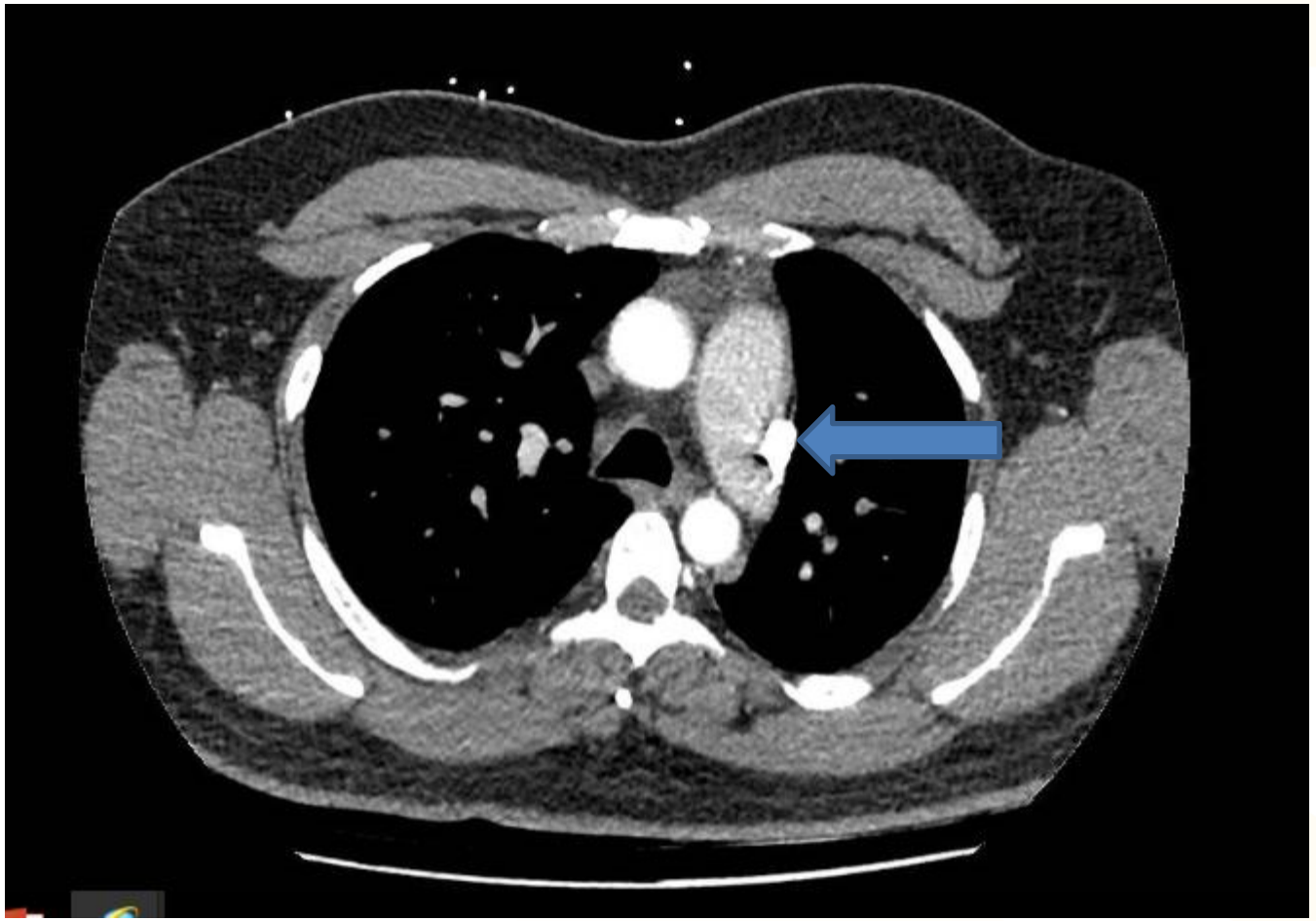


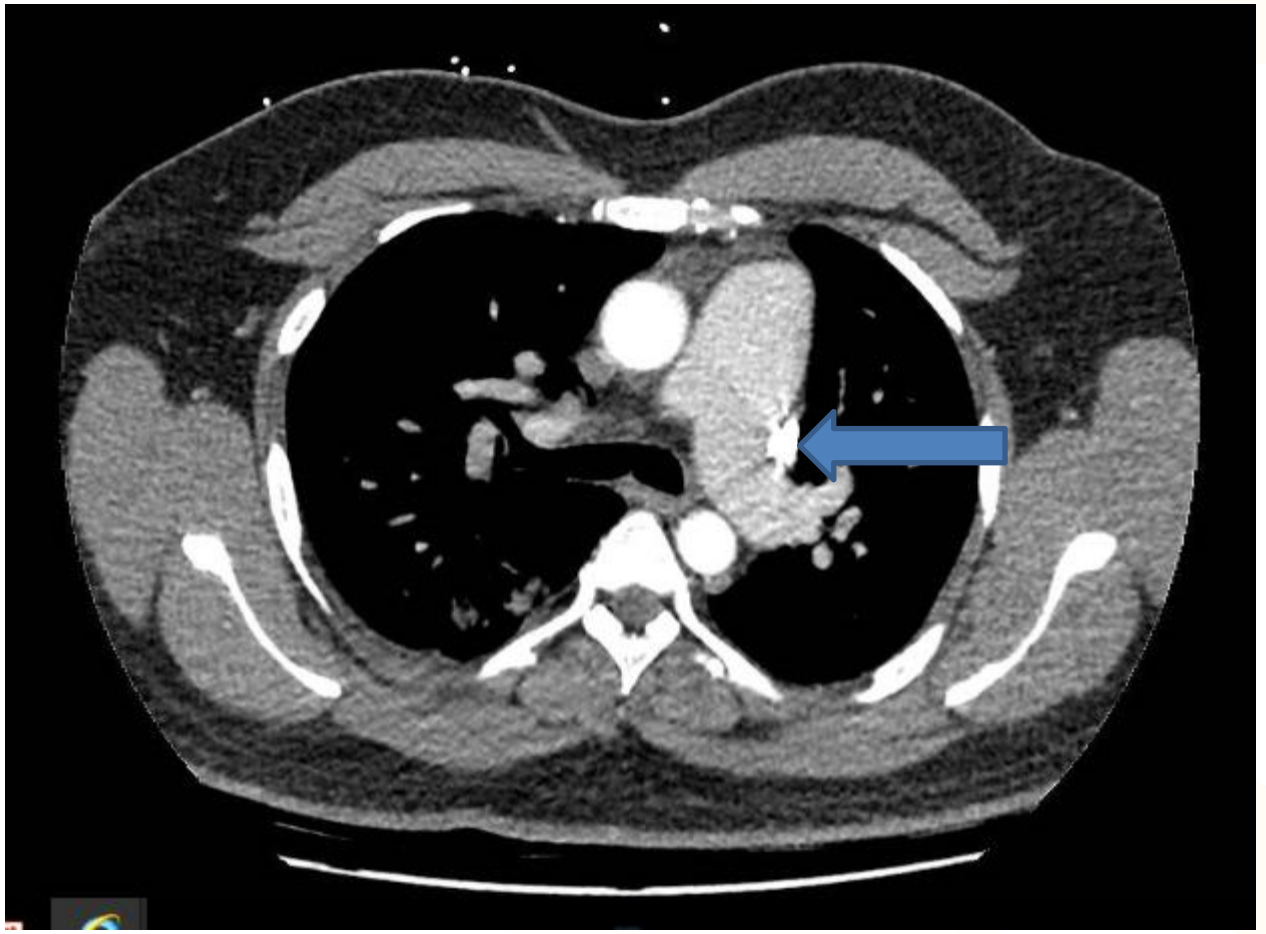
CTPA

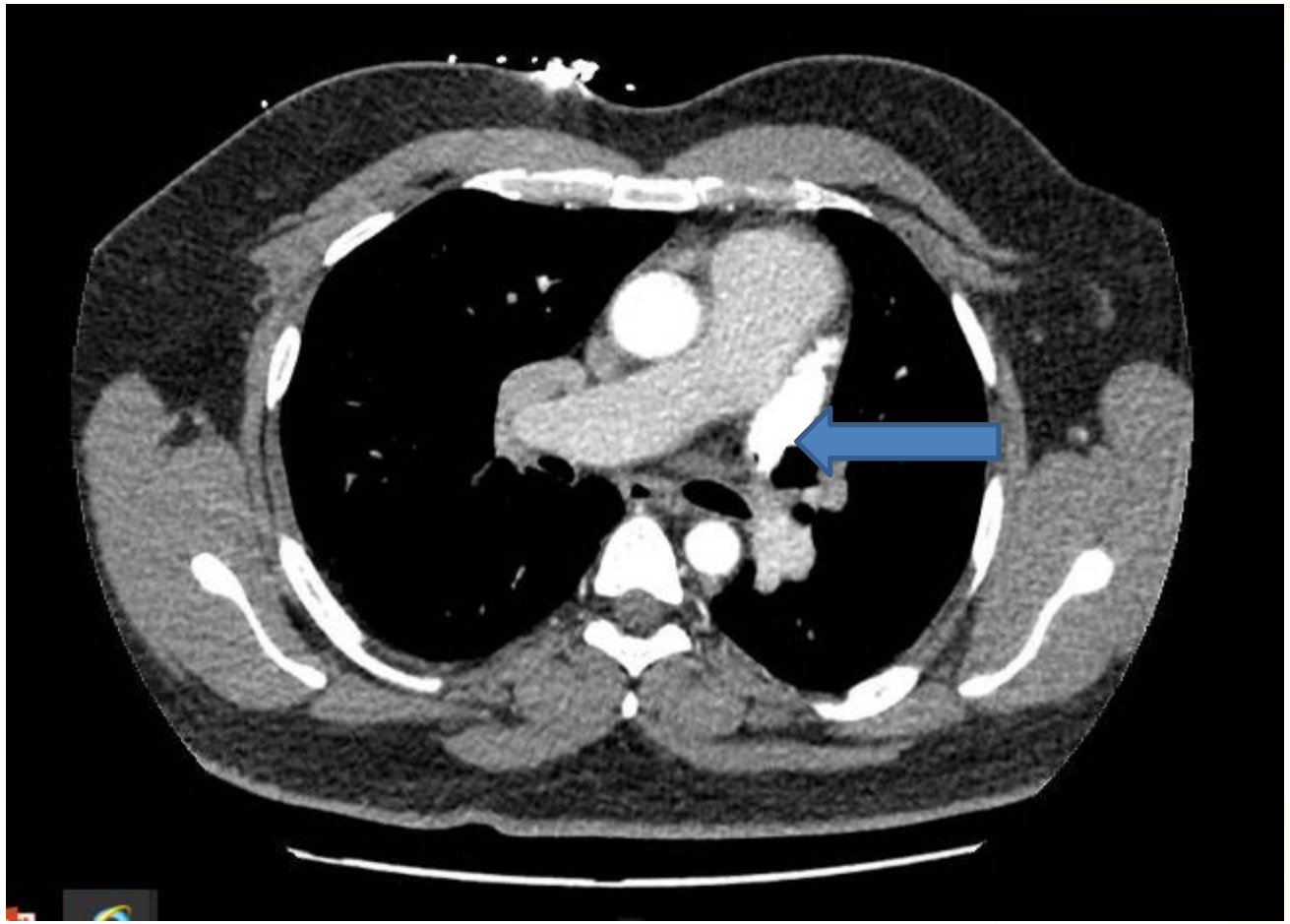
What is this structure?

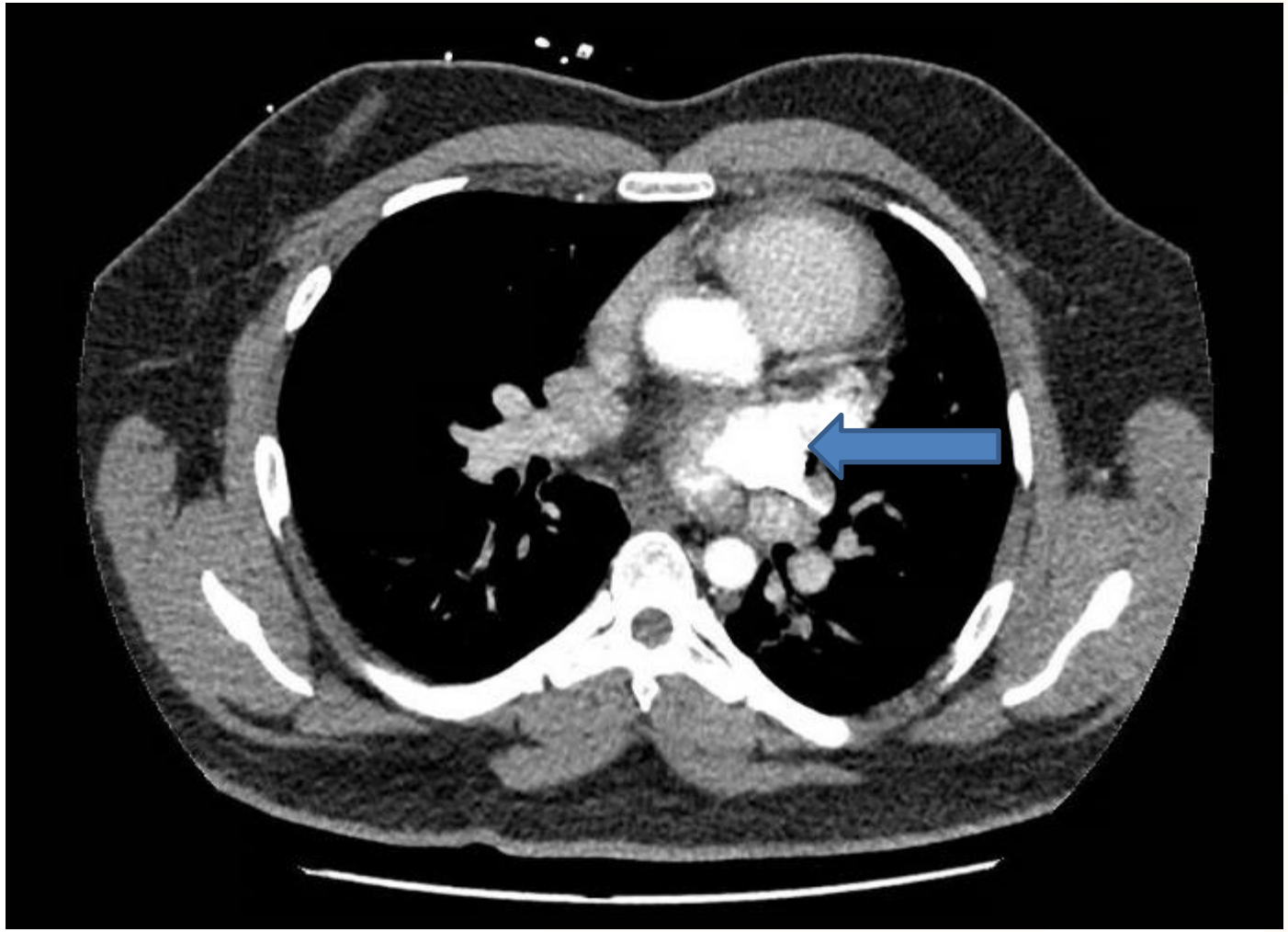


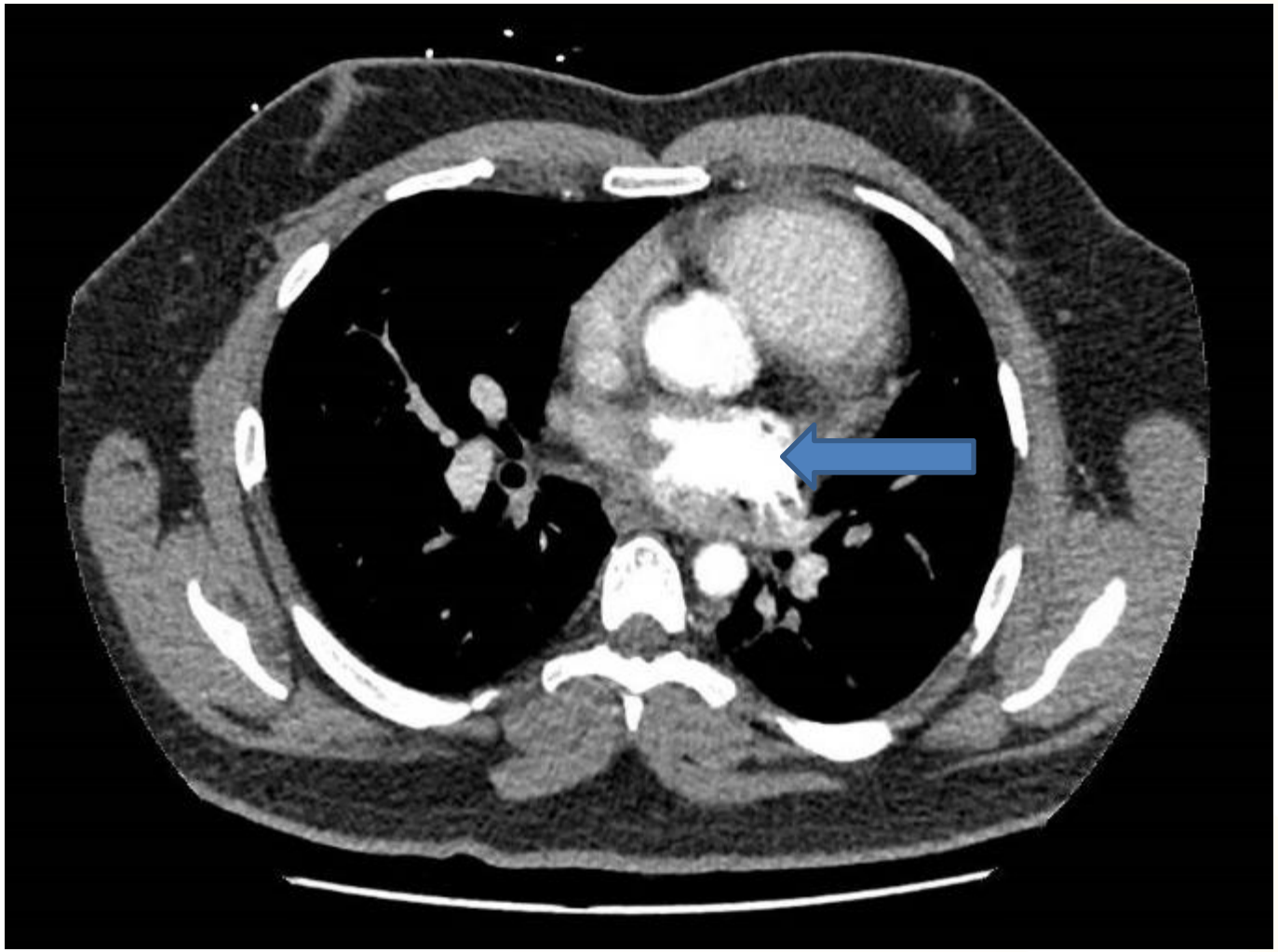

What is this structure?

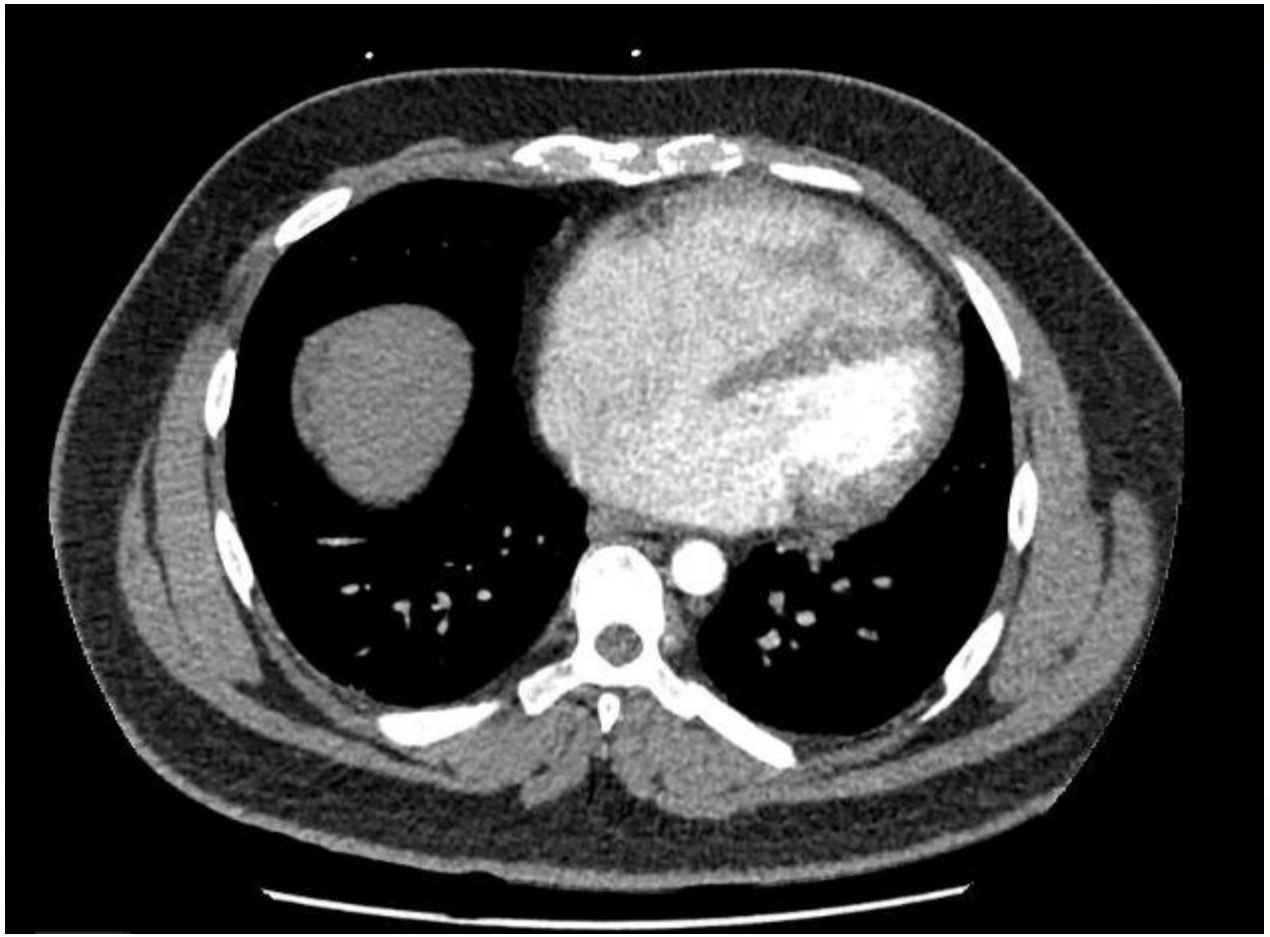




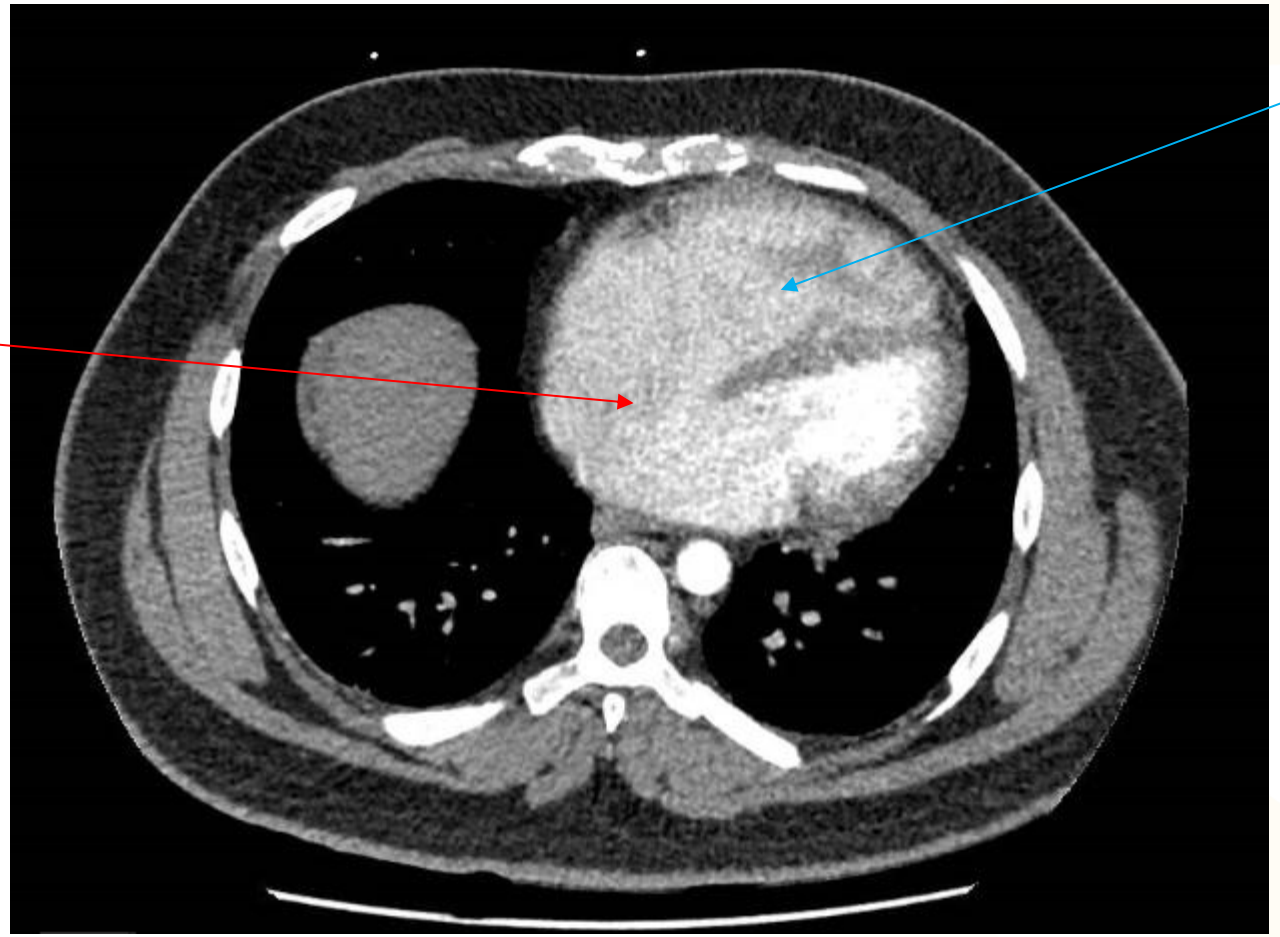








Large ASD



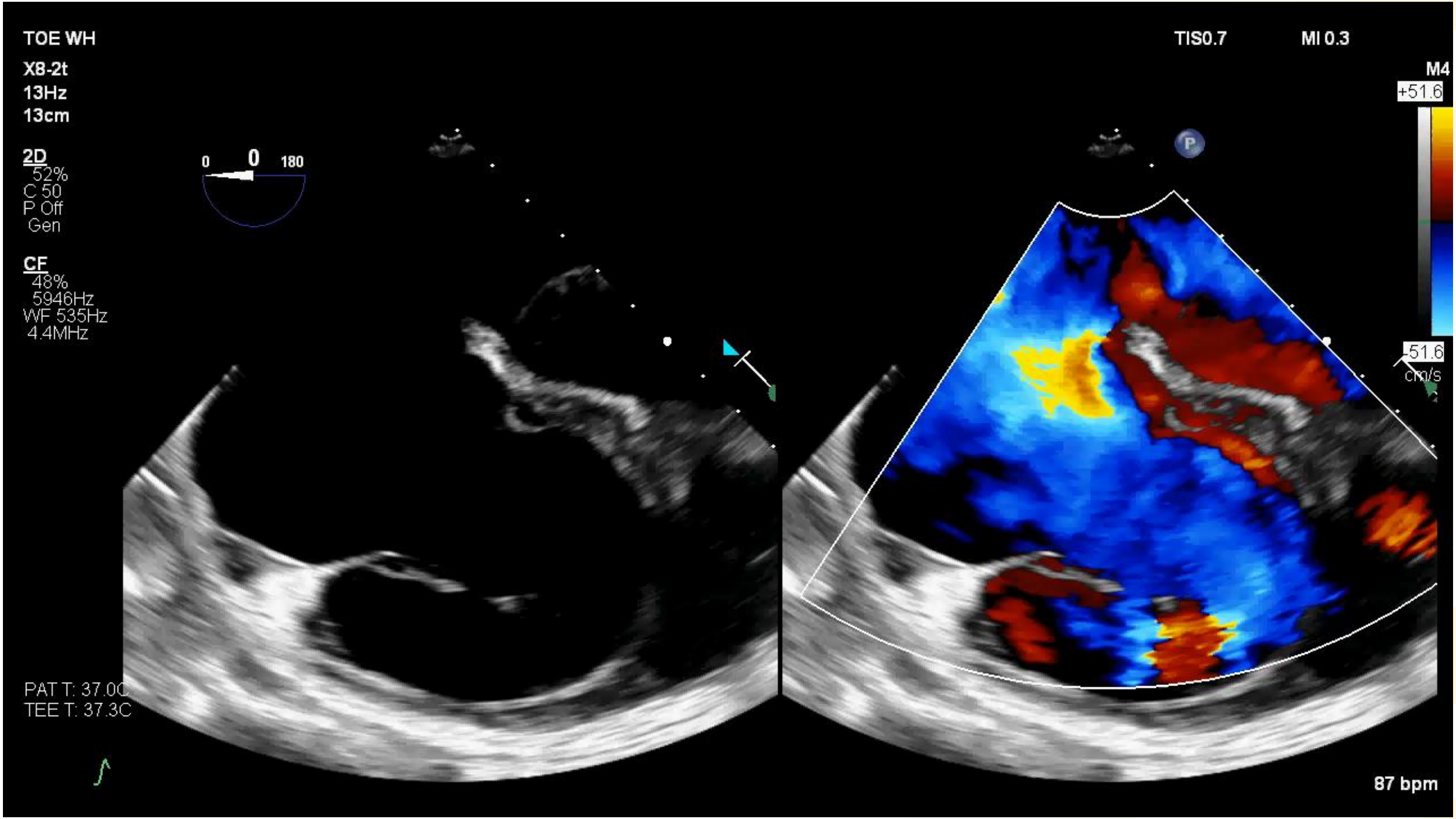
Dilated RV

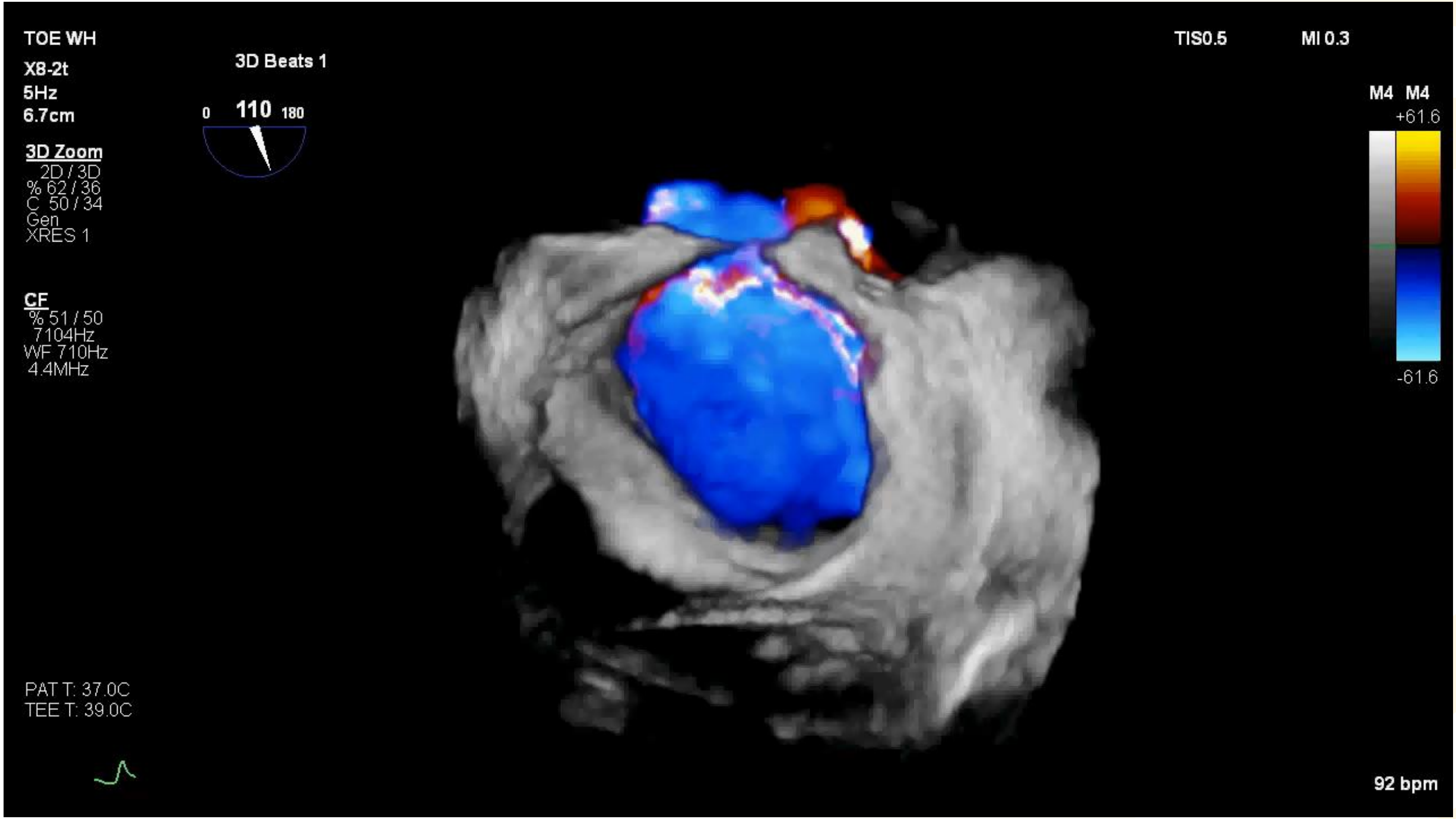

TAULA, ASOSA
18/11/1991, FSU6141
Hutt Valley DHB
03/04/2024 15:51:04
M5Sc
USR Cardiac4
MI 1.4
TIs 0.5
HD
FPS: 61
f: 1.4 MHz/2.8 MHz
D: 18.0 cm
Soft
V
5
10
15
103
HR

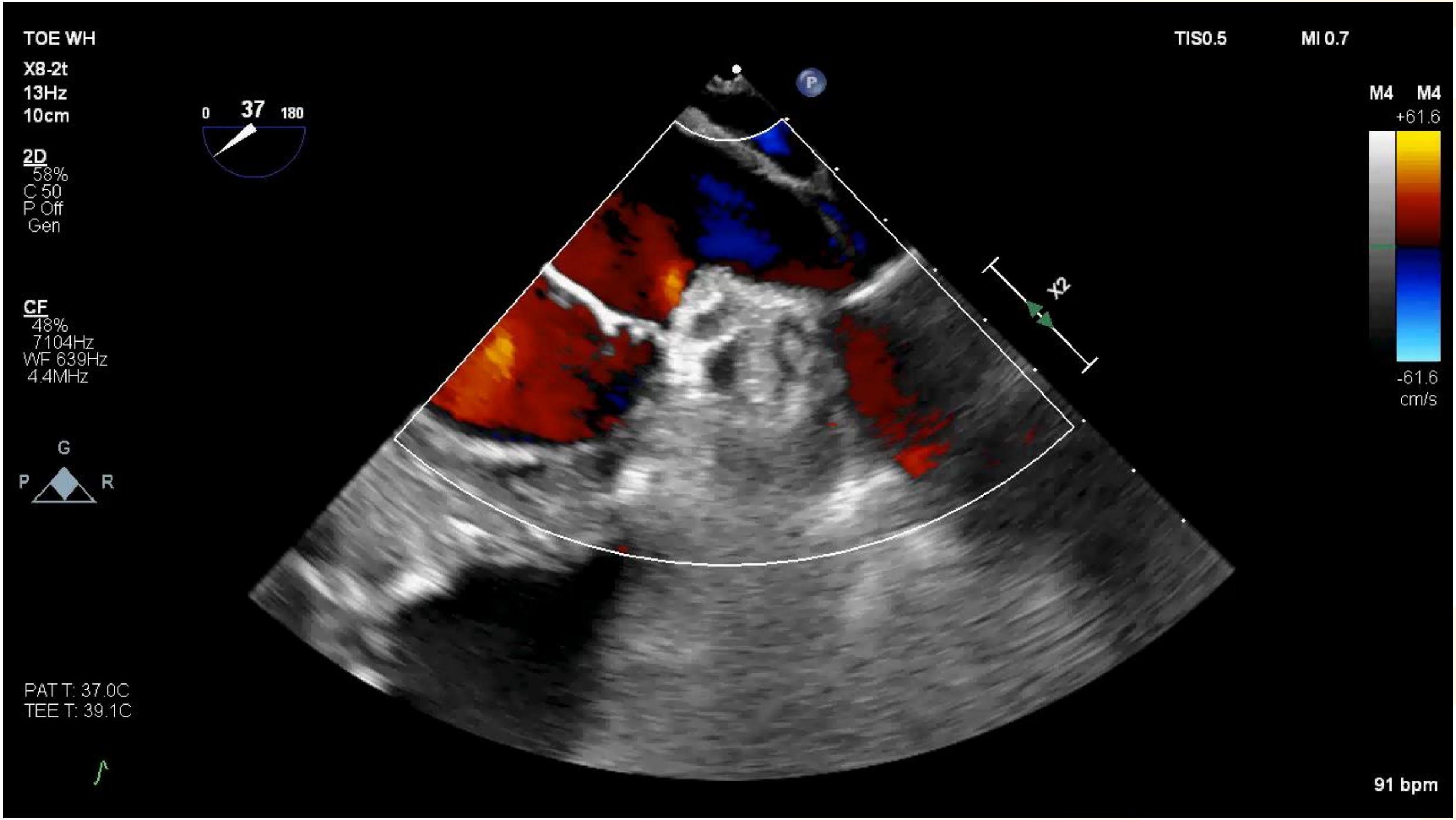

TAULA, ASOSA
18/11/1991, FSU6141
Hutt Valley DHB
03/04/2024 16:00:21
4V
USR Cardiac4
MI 1.0
TIs 0.2
HD
FPS: 57/
f: 1.5 MHz/3.1 MHz
D: 22.0 cm
Soft
V
10
20
94
HR

TTE

- Sub-optimal imaging
- Key features:
  - Normal LV size and function
  - Dilated RV (no clear reason for this – interatrial septum not clearly seen)
  - Significant TR


TOE WH
X8-2t
13Hz
13cm
2D
52%
C 50
P Off
Gen
CF
48%
5946Hz
WF 535Hz
4.4MHz
TIS0.7
MI 0.3
M4
+51.6
-51.6
cm/s
0
0
180
PAT T: 37.0C
TEE T: 37.3C
87 bpm


TOE WH
X8-2t
5Hz
6.7cm
3D Beats 1
0
110
180
3D Zoom
2D / 3D
% 62 / 36
C 50 / 34
Gen
XRES 1
CF
% 51 / 50
7104Hz
WF 710Hz
4.4MHz
TIS0.5
MI 0.3
M4 M4
+61.6
-61.6
PAT T: 37.0C
TEE T: 39.0C
92 bpm

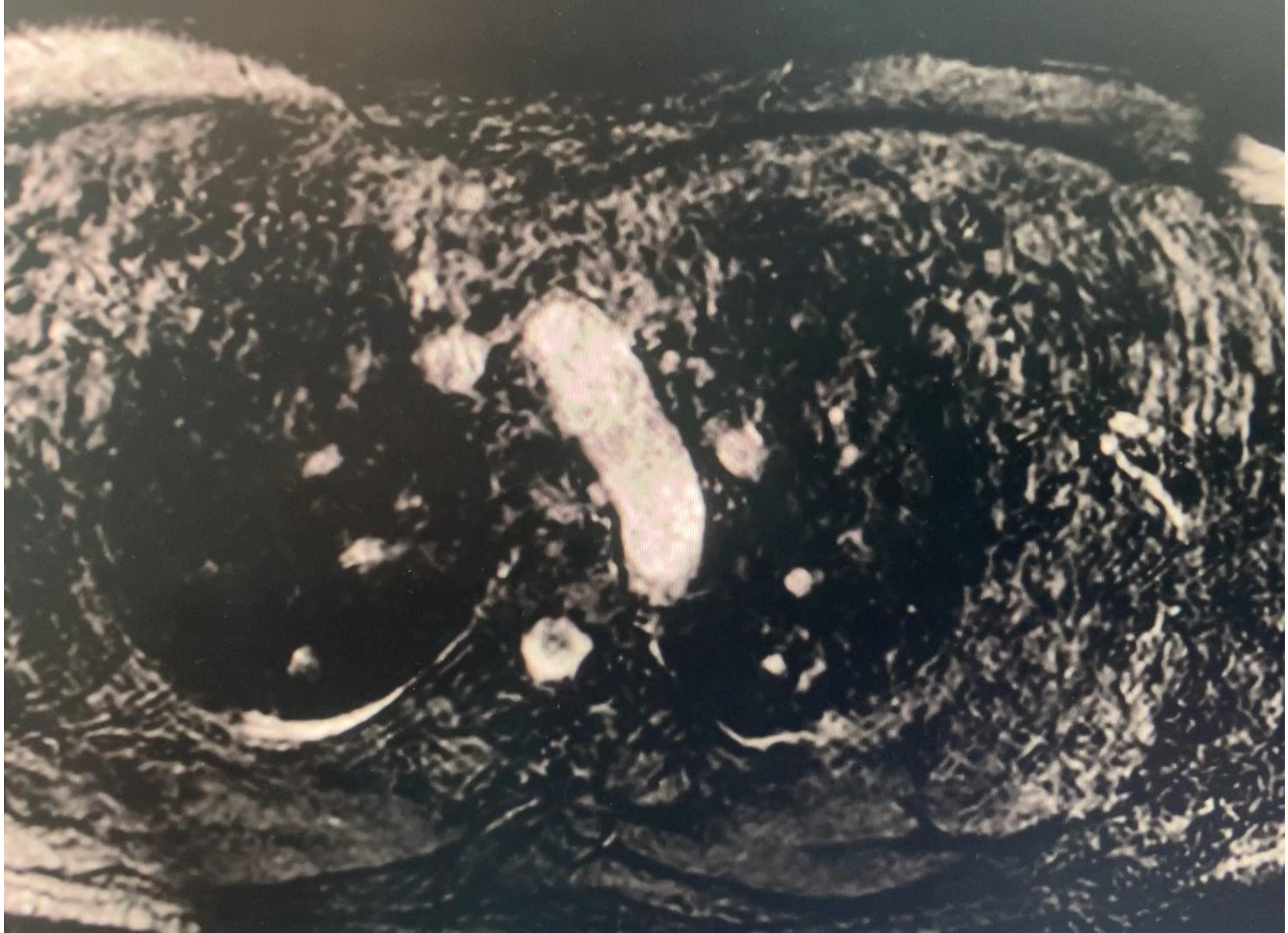

TOE WH
X8-2t
13Hz
10cm
2D
58%
C 50
P Off
Gen
CF
48%
7104Hz
WF 639Hz
4.4MHz
G
P
R
PAT T: 37.0C
TEE T: 39.1C
0
37
180
TIS0.5
MI 0.7
M4
M4
+61.6
-61.6
cm/s
X2
91 bpm

TOE

- Large ASD
- Significant TR directed towards ASD demonstrating some flow from right to left (although predominantly left to right shunting)
- Abnormal vessel entering to LA between the L atrial appendage and the left upper Pulmonary vein

CMR

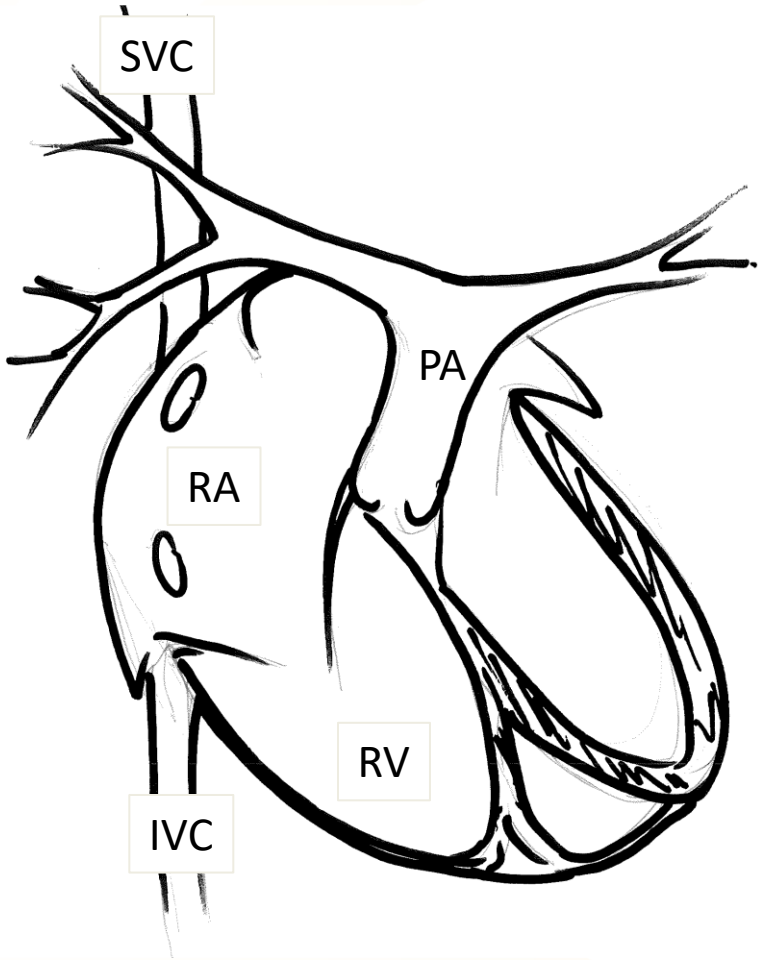
- Sub-optimal imaging due to irregularity of heart rhythm (thought to be AF – no evidence of AF throughout admission).
- Flows performed but likely to be relatively unreliable due to irregularity of heart rhythm (which is one of the main indications for CMR for shunt assessment)
  - SV MPA: 225ml
  - SV prox. aorta: 57ml (large left to right shunt)
- 3D whole heart demonstrating the abnormal connections demonstrated on the CT scan
  - Persistent L SVC – draining into the LA just posterior to the LA appendage



Left and right heart cath – Sats run

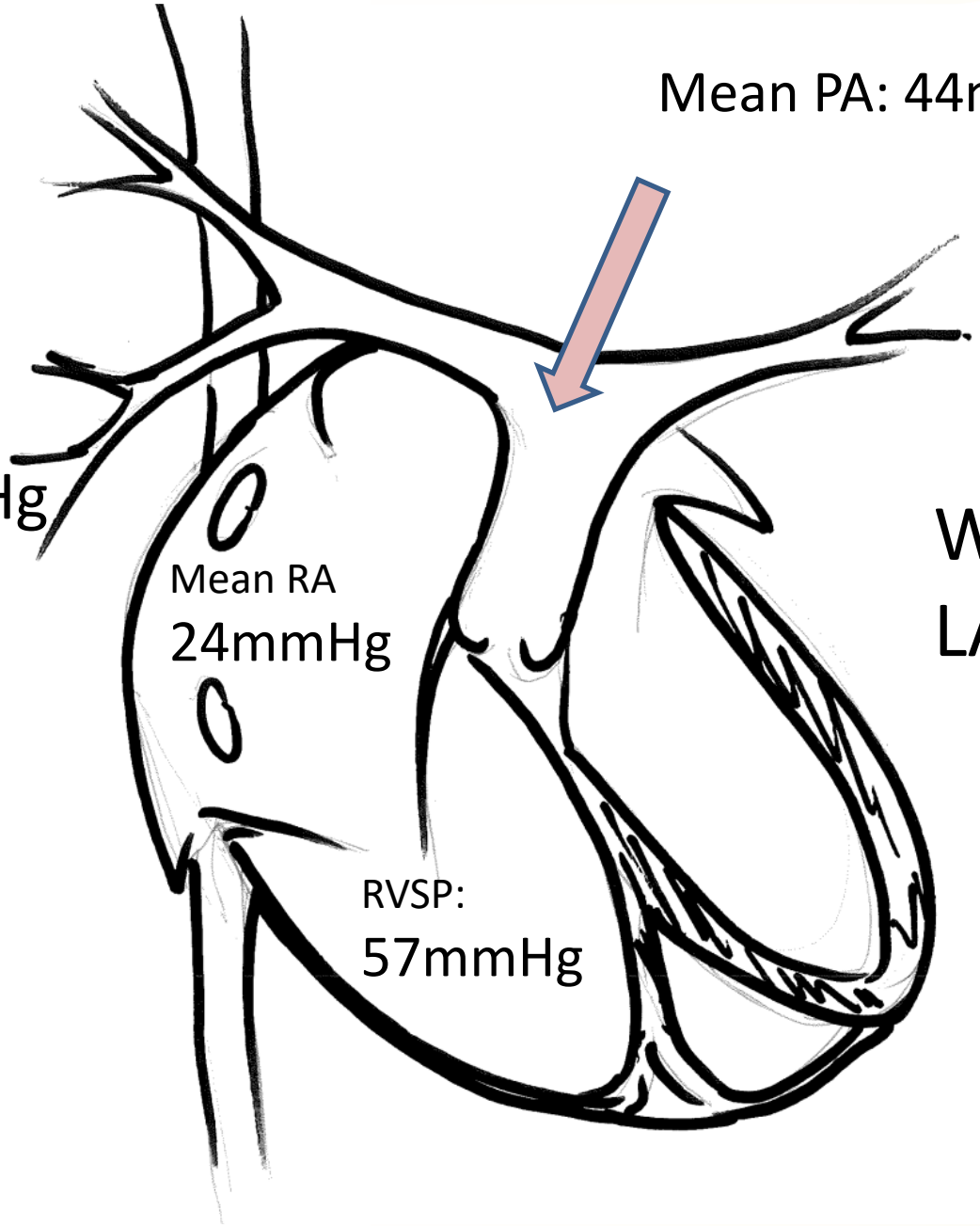
- IVC: 68%
- SVC: 59%
- RA: 86%
- RV: 88%
- PA: 86%
- Rad artery: 89%

- Qp/Qs: 2.5 to 1

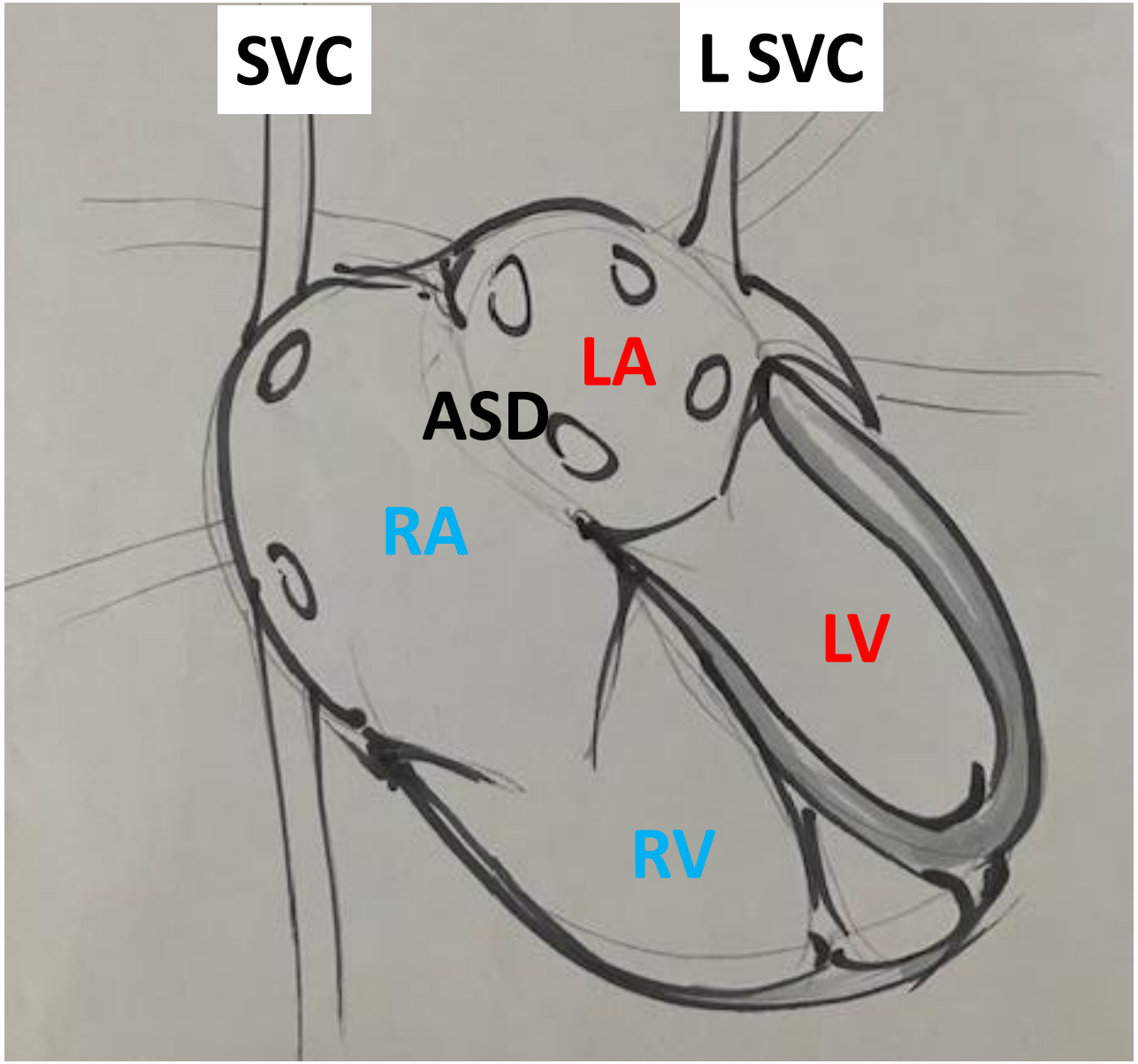


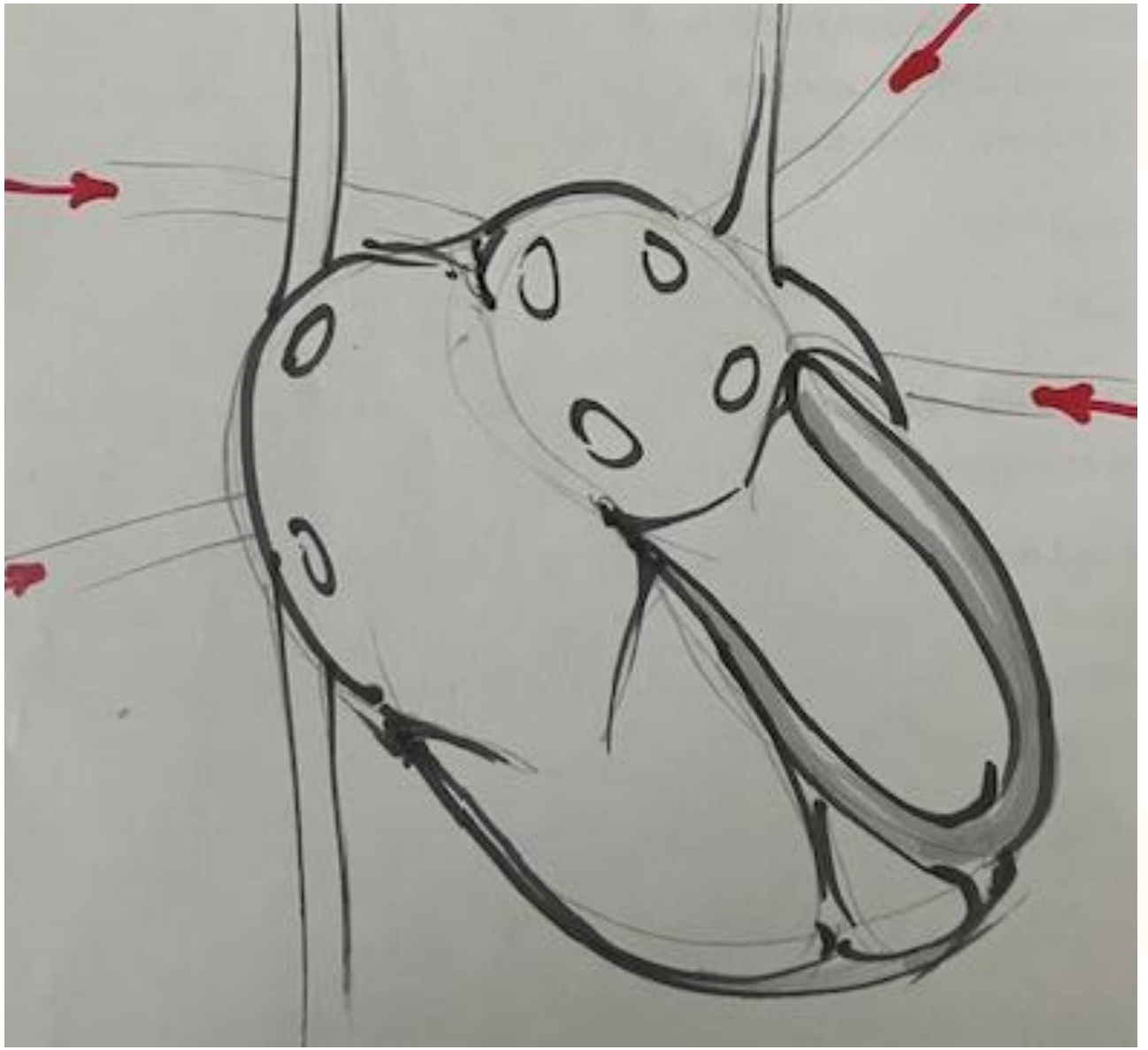
Mean PA: 44mmHg

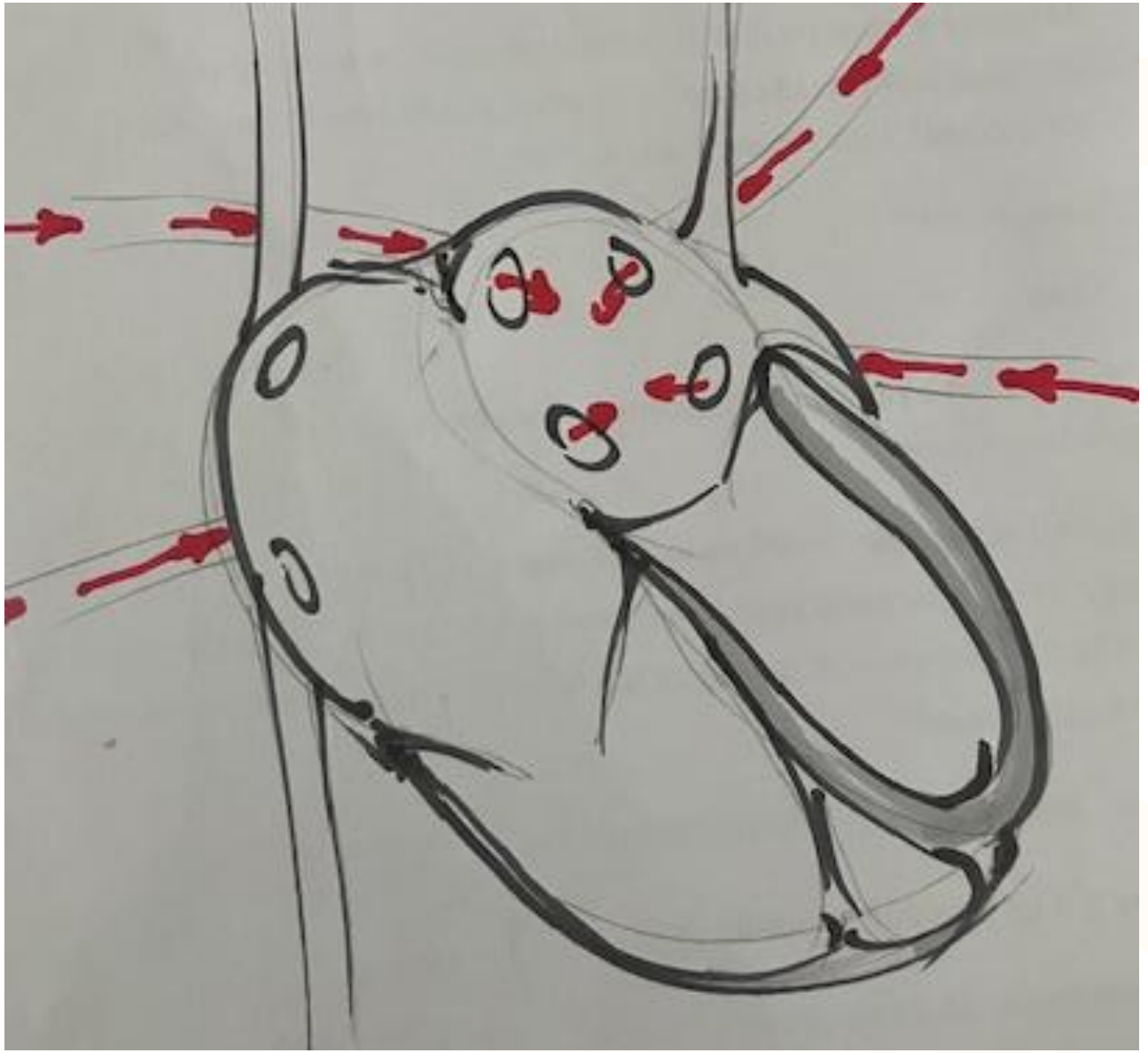
Mean PCWP: 26mmHg

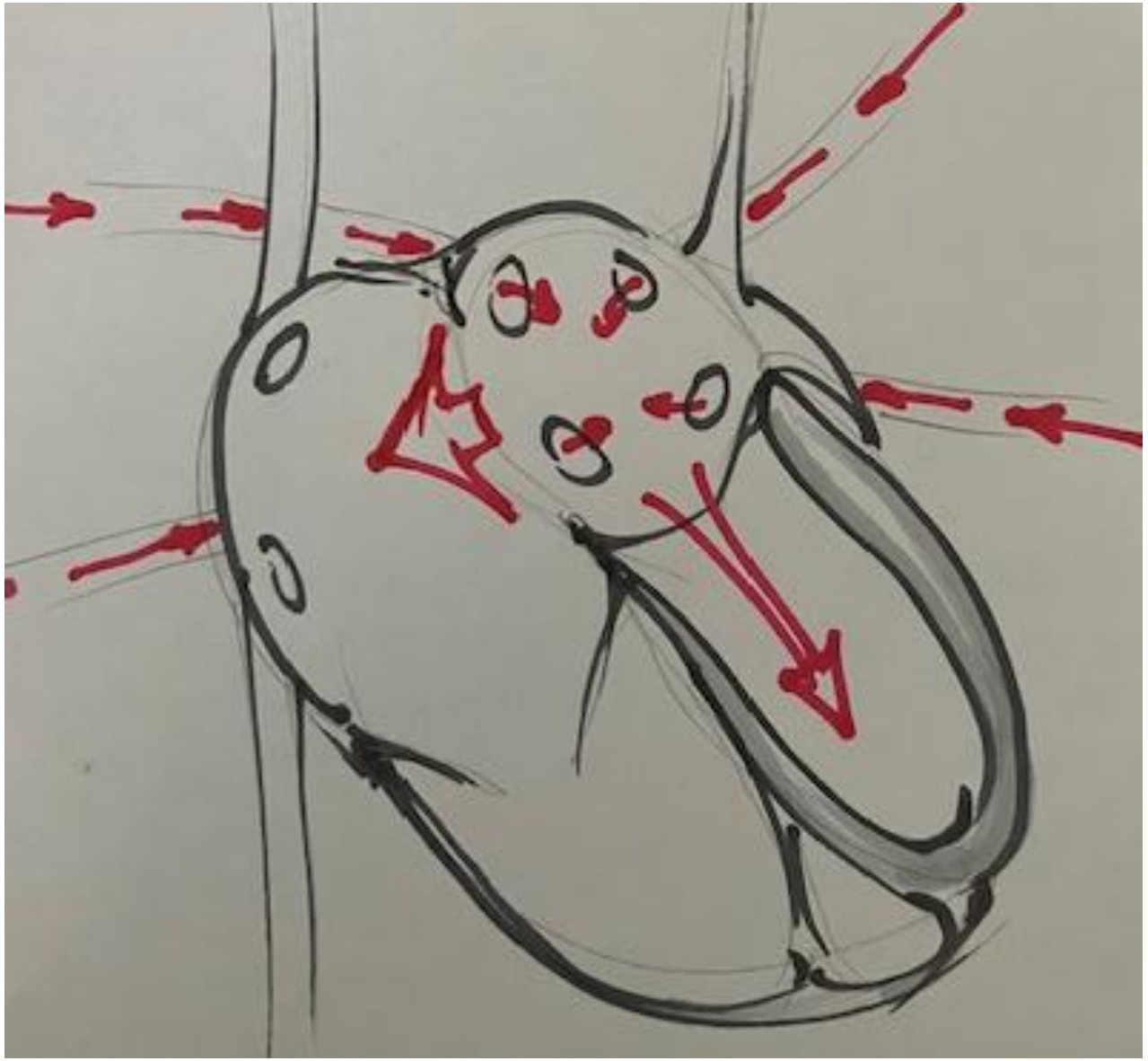


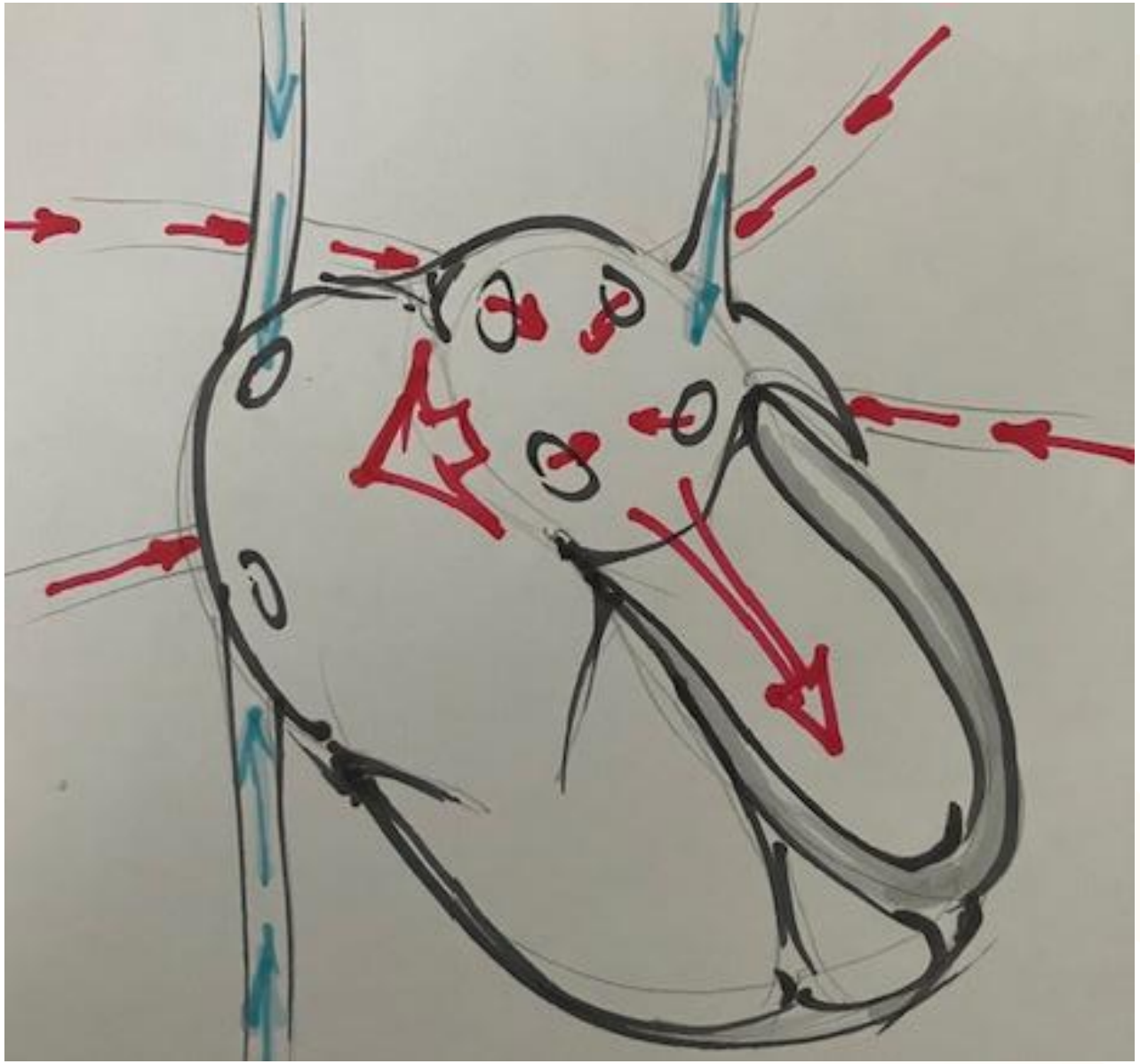
With a large ASD
LAP = RAP = PCWP

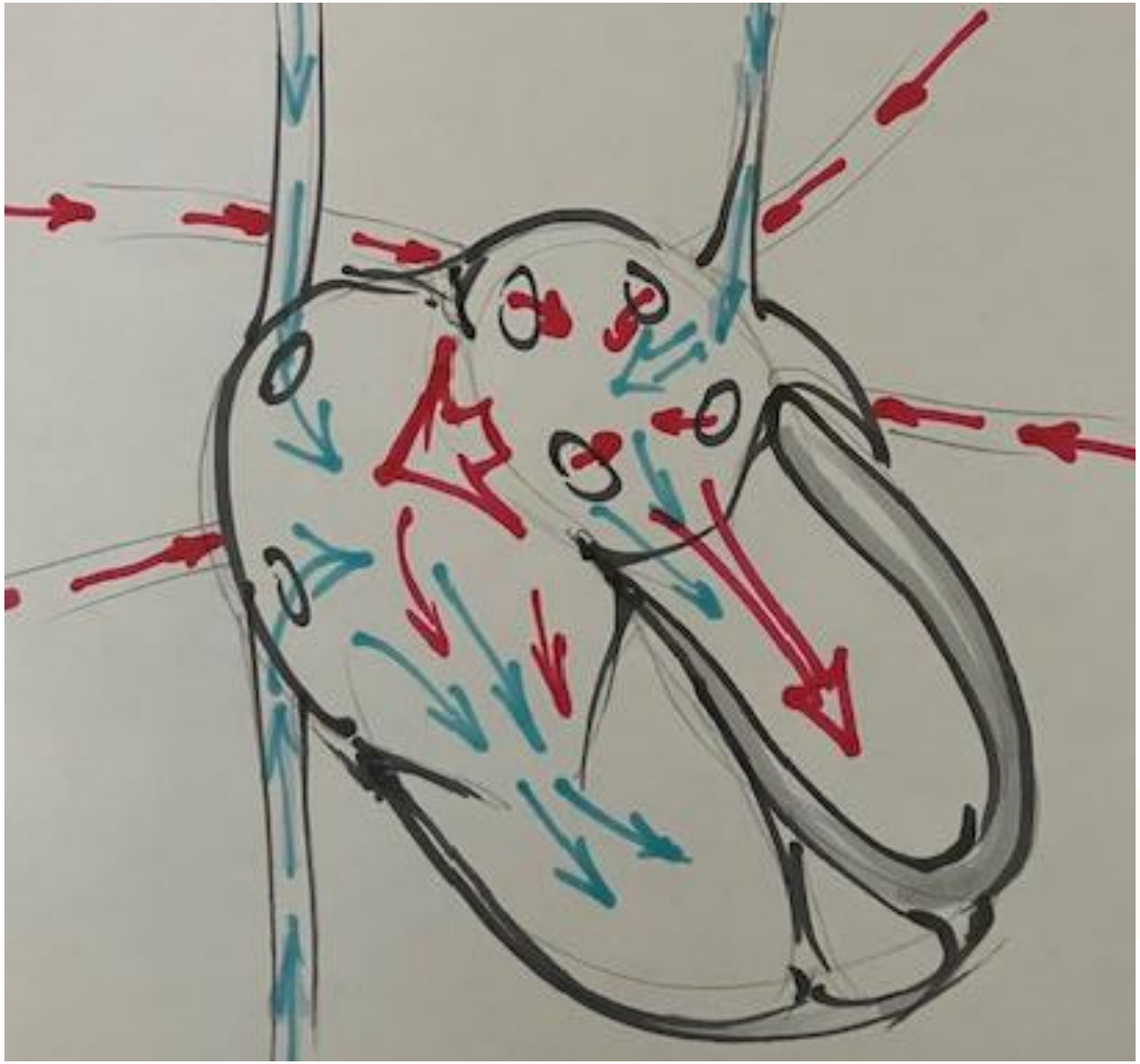

SVC
L SVC
LA
ASD
RA
LV
RV











- ASD – with large left to right shunt
- L SVC draining into the LA – resulting in slightly low arterial 02 saturations
- Severe RV dilatation with significant functional TR
- Raised LA pressures - ?due to diastolic dysfunction

- Unexplained neurological event –
  - Could it represent TIA?
  - Is there any link to the anatomical abnormalities identified

- Referred to Auckland for surgical management

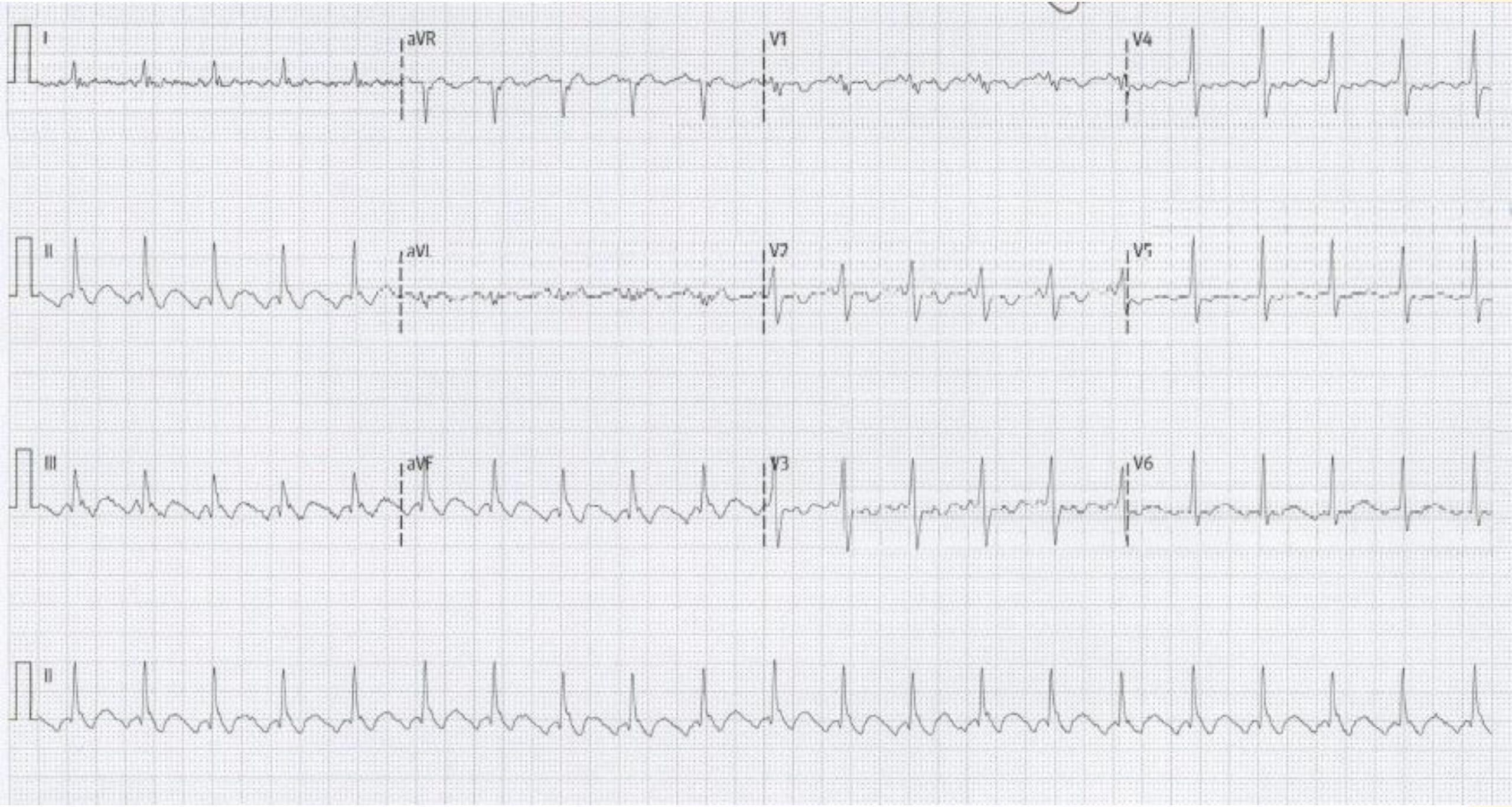
- Diagnostic work-up relying on multimodality imaging.
  - CT/CMR helpful for anatomy
  - Echo/CMR/Cardiac catheterisation helpful for clarifying useful for clarifying haemodynamic effect of anatomical lesions.

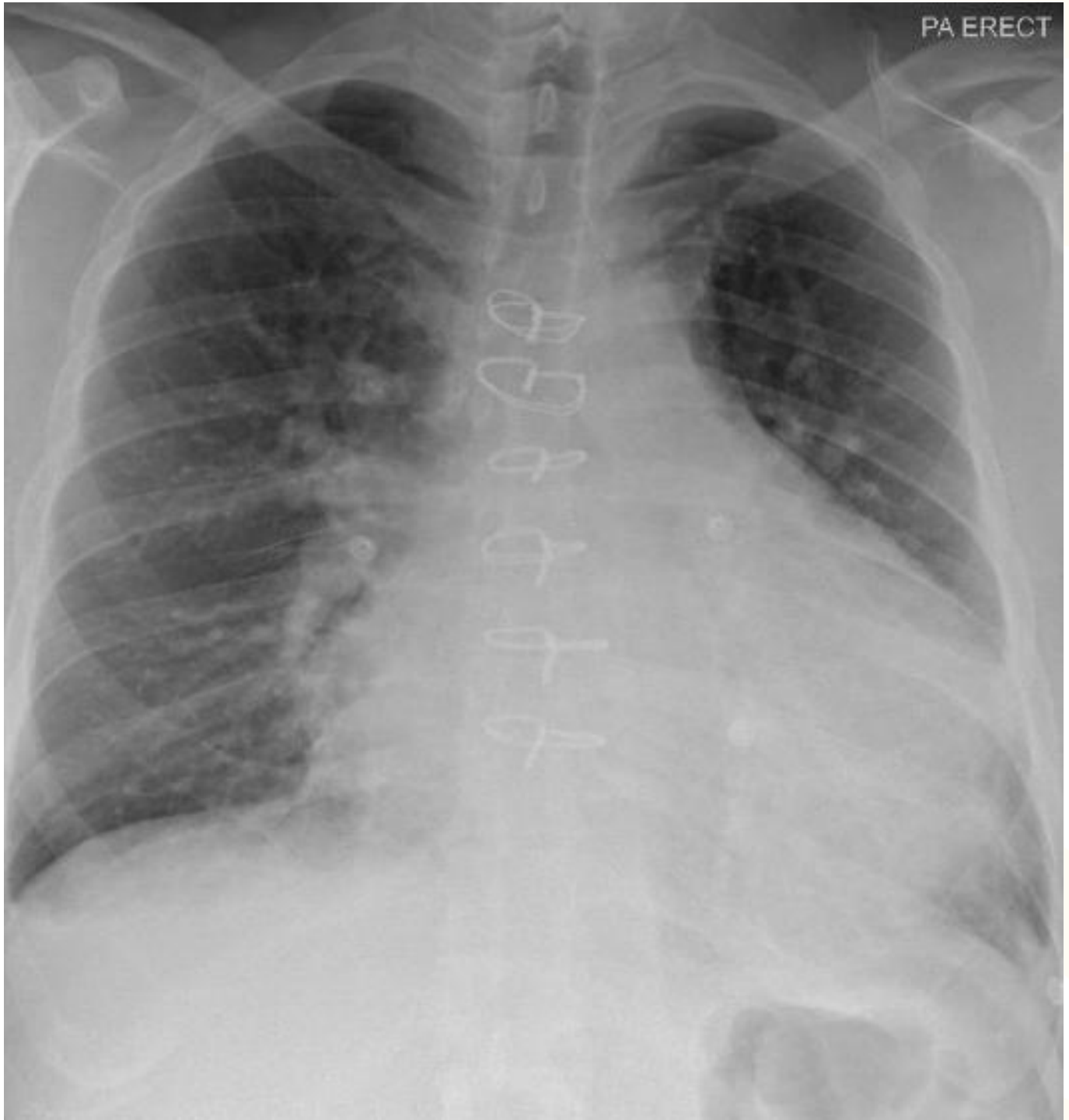
Operation

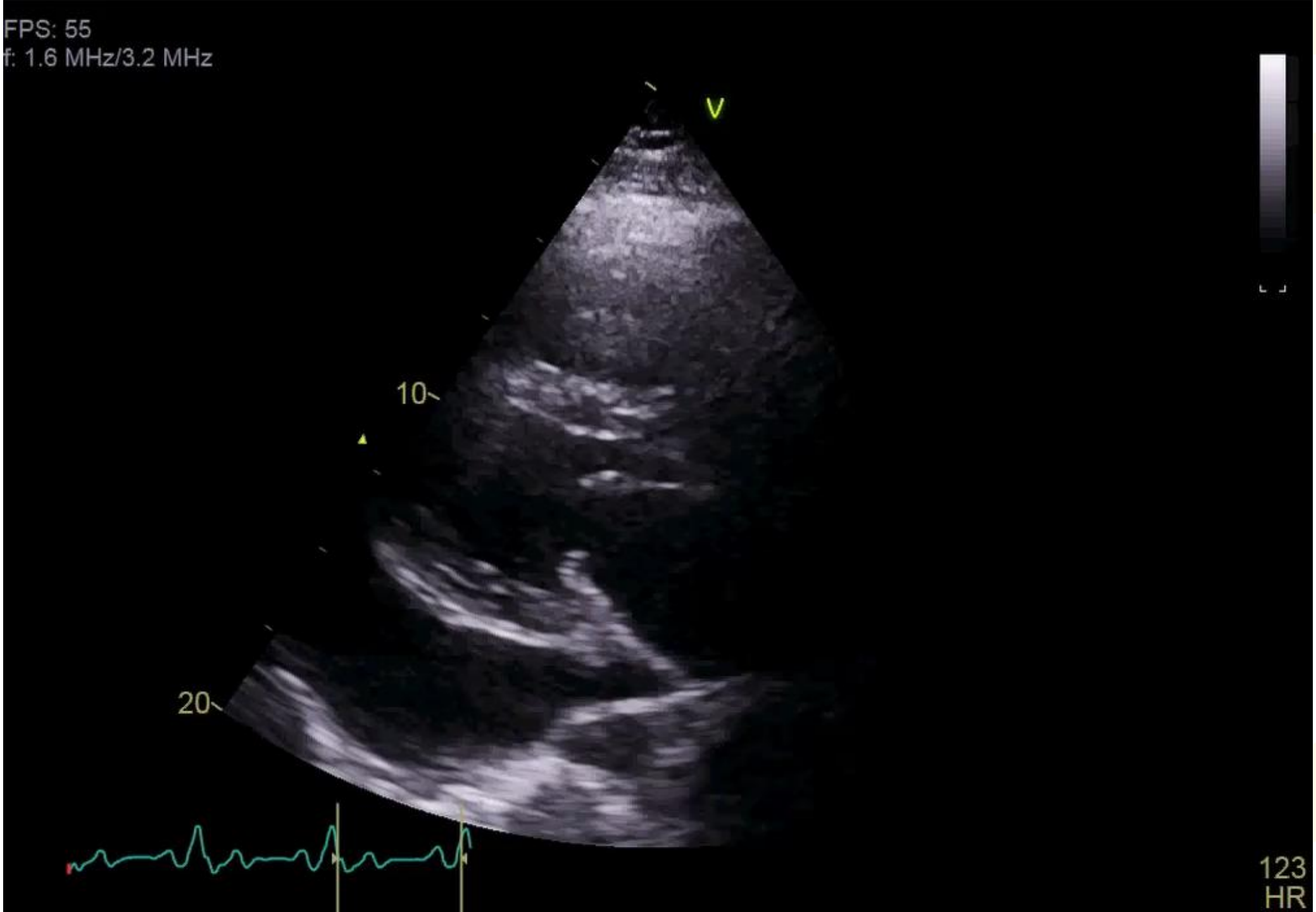
- ASD closure
- TV repair with 36mm Cosgrove annuloplasty ring
- Ligation of persistent left SVC (which communicates to LA)
- Right atrial MAZE
- In sinus rhythm at discharge

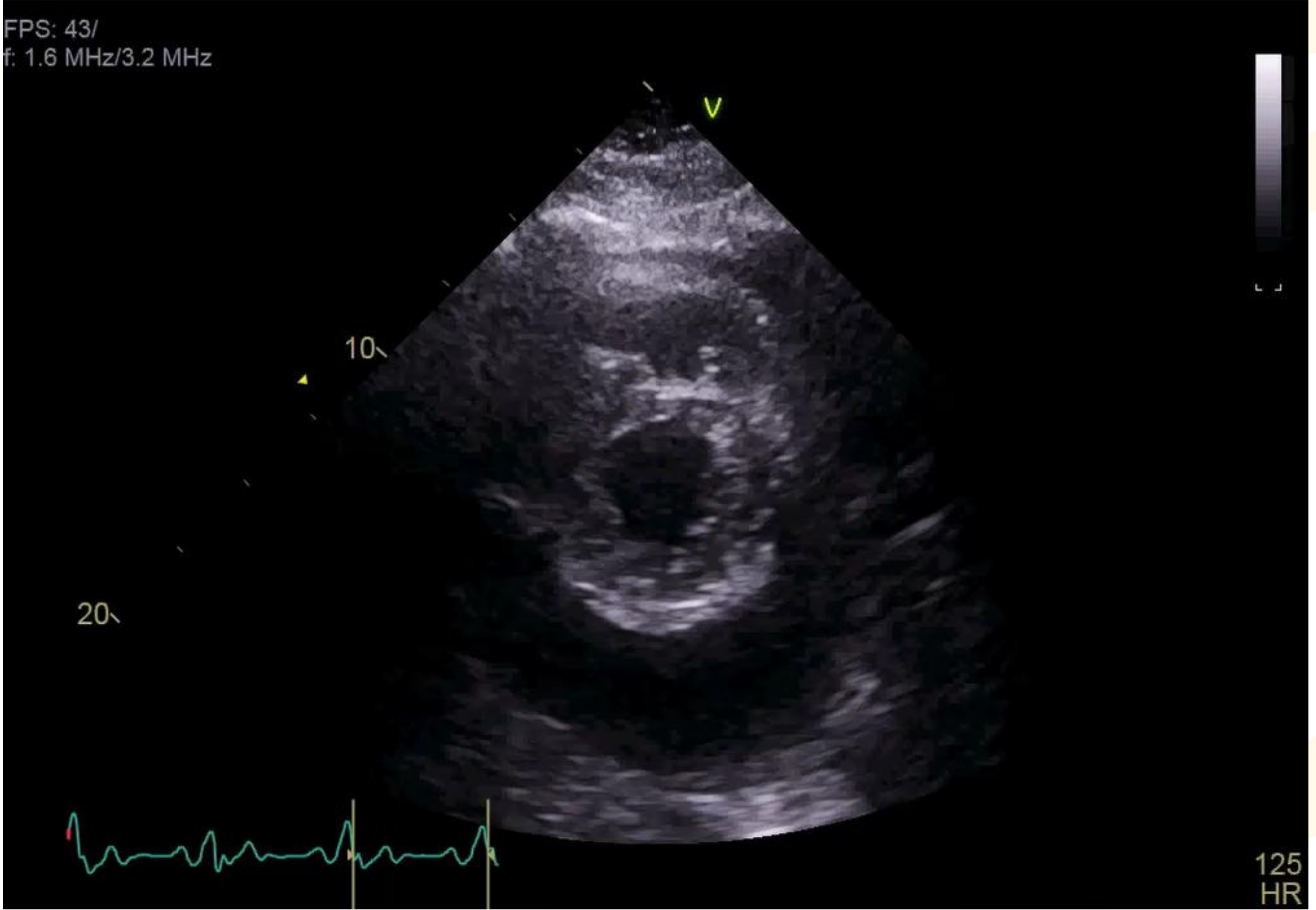
Presentation to GP 10/7 post discharge

- Since discharge aware of exercise intolerance and palpitations
- PND over past week
- Light-headed in GP surgery.
- Noted to be in A flutter with rapid ventricular response


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II


PA ERECT


FPS: 55
f: 1.6 MHz/3.2 MHz
V
10
20
123
HR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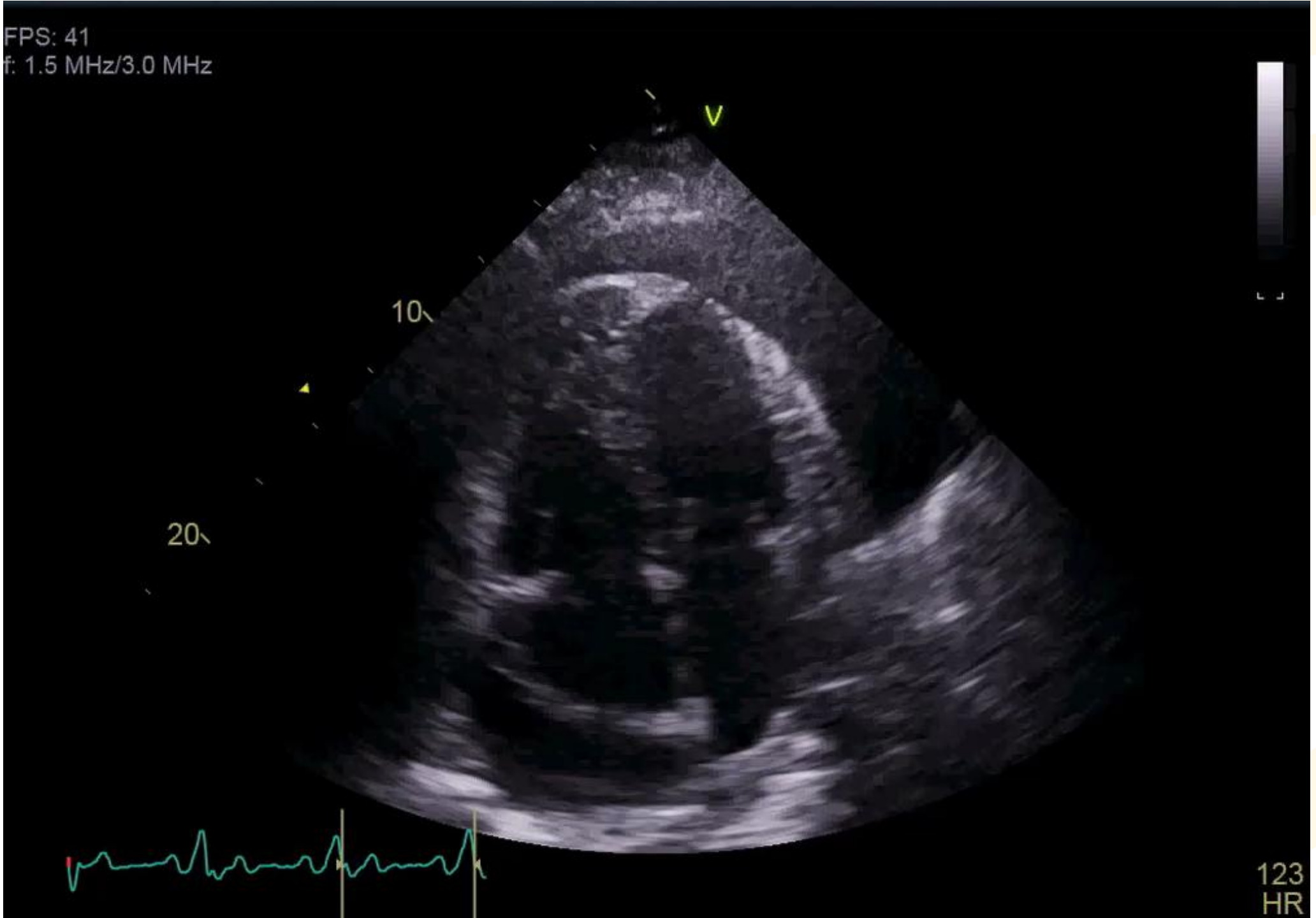
f: 1.6 MHz/3.2 MHz
V
10
20
125
HR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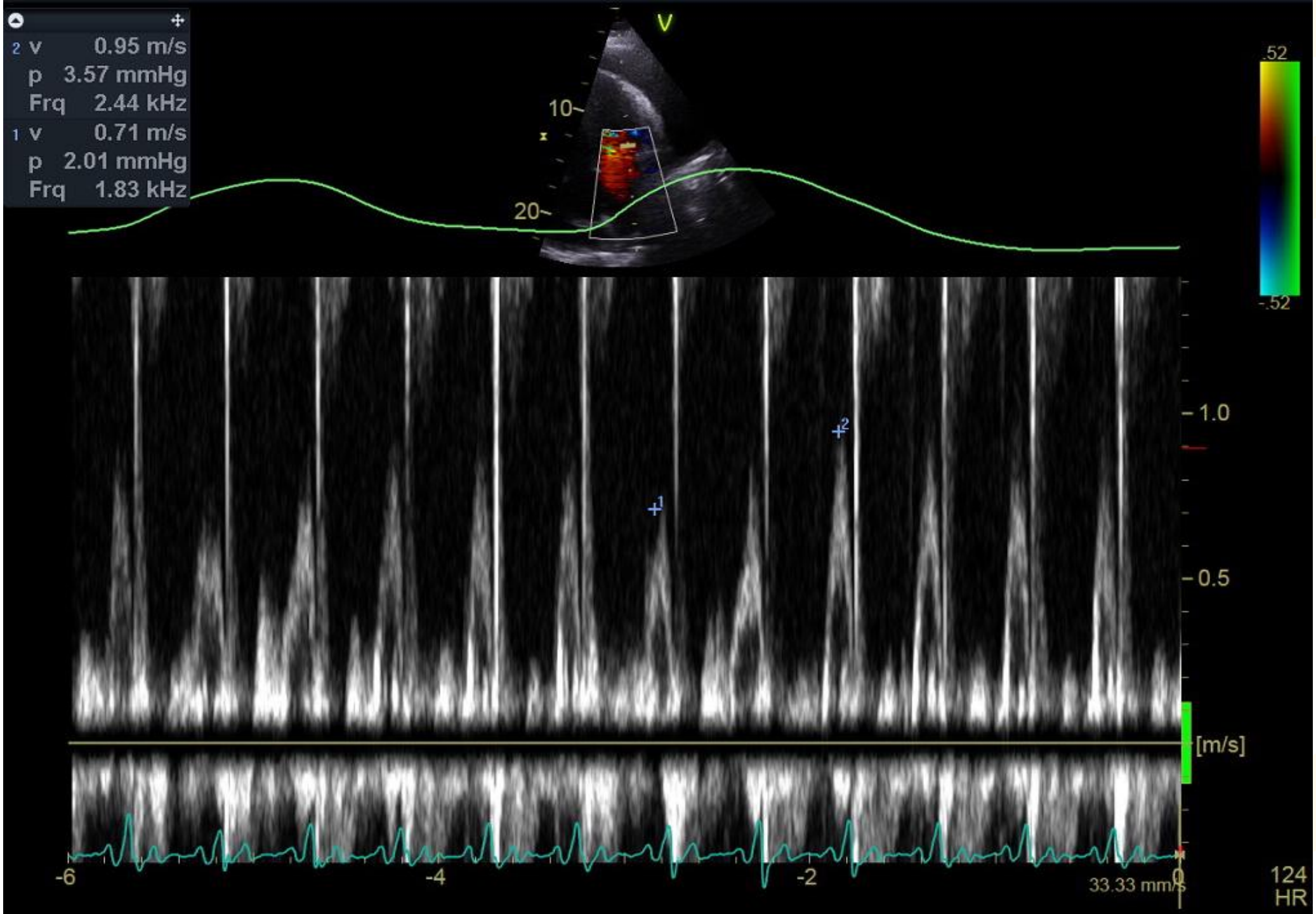
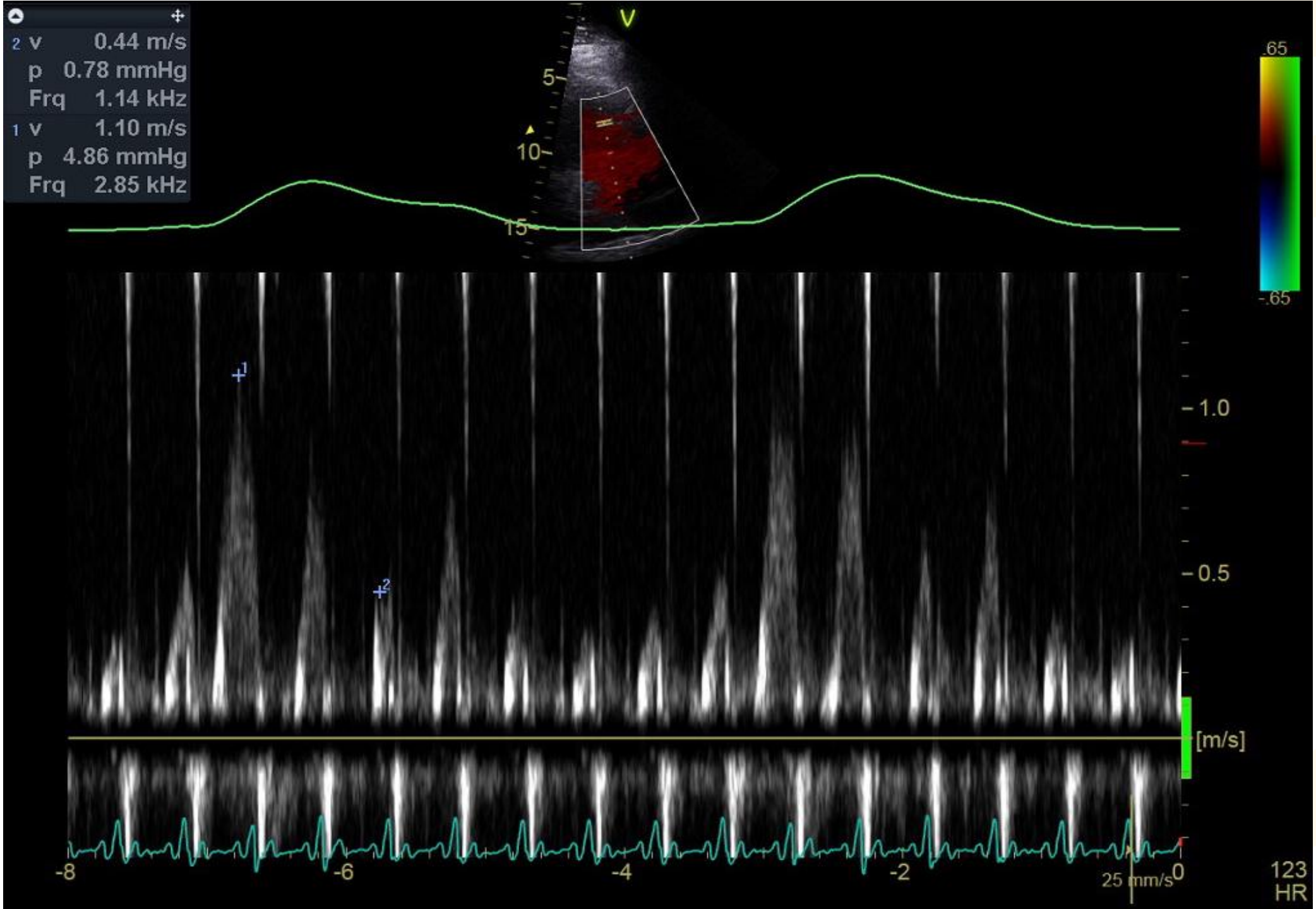
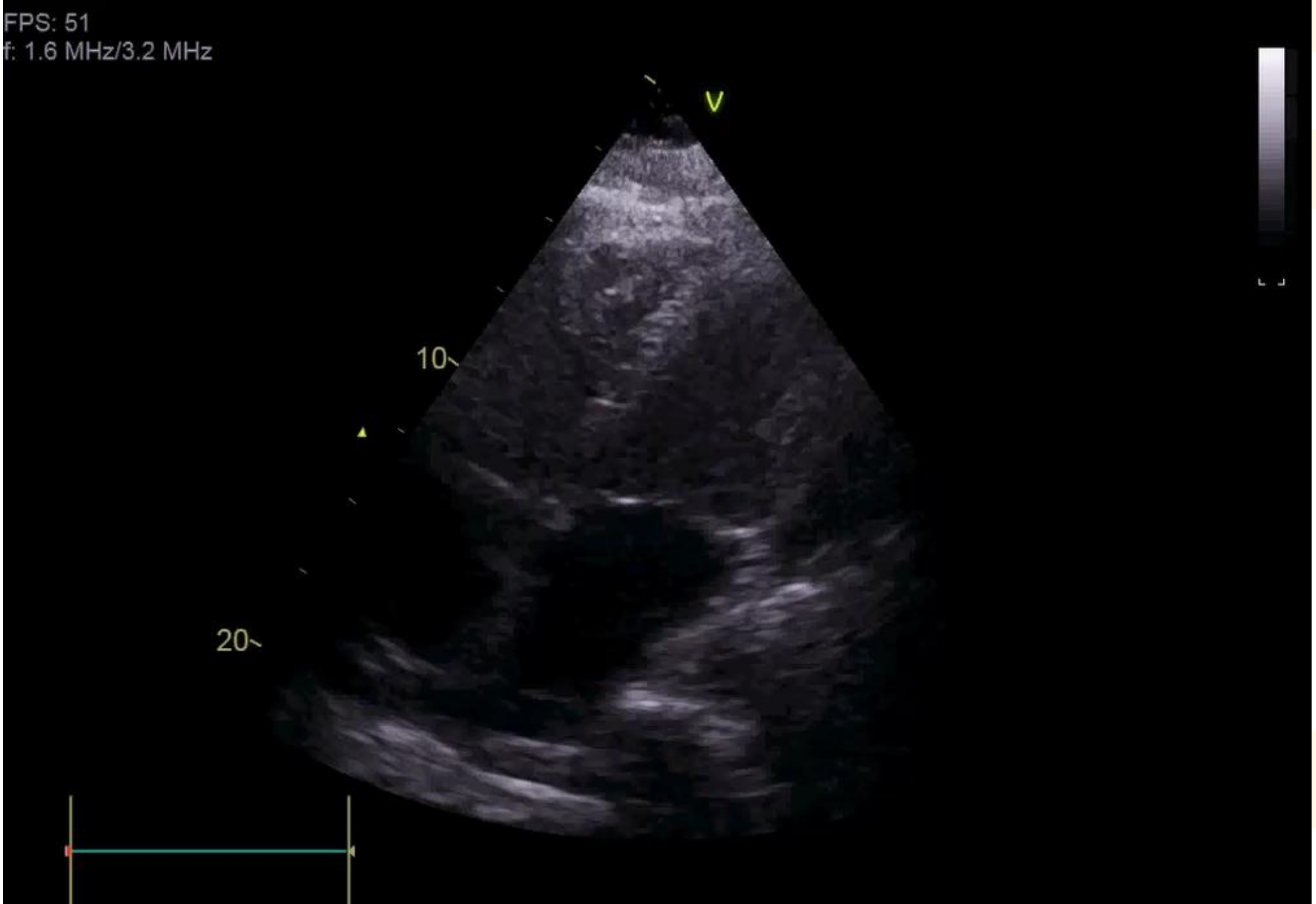
f: 1.5 MHz/3.0 MHz
V
10
20
123
HR


2 v 0.95 m/s
p 3.57 mmHg
Frq 2.44 kHz
1 v 0.71 m/s
p 2.01 mmHg
Frq 1.83 kHz
V
10
20
.52
-.52
1.0
0.5
[m/s]
-6
-4
-2
0
33.33 mm/s
124
HR


2 v 0.44 m/s
p 0.78 mmHg
Frq 1.14 kHz
1 v 1.10 m/s
p 4.86 mmHg
Frq 2.85 kHz
V
5
10
15
65
-.65
1
2
1.0
0.5
[m/s]
-8
-6
-4
-2
0
25 mm/s
123
HR

Large Haemodynamically significant pericardial effusion


FPS: 51
f: 1.6 MHz/3.2 MHz
V
10
20

Progress

- Anticoagulation 2 days post drainage of pericardial collection
- No reaccumulation of pericardial collection
- Successful DCCV after 4 week anticoagulation

Summary

- Likely incidental finding of congenital cardiac anomaly
- Understanding of normal cardiac and thoracic vascular anatomy helpful in interpreting imaging (particularly chest CT scanning)
- Multimodality imaging helpful for:
  - Diagnosis (delineating cardiac anatomy and vascular connections)
  - Planning appropriate management
  - Diagnosis of post-operative complications

Cardiac Output and Shunt determination

- Fick's principle: "the total uptake or release of a substance by an organ is the product of the blood flow to the organ and the AV concentration difference of that substance"

- Cardiac output = O2 consumption x BSA/(Art sat - Venous sat) x Hb x 13.6

- NB: O2 consumption assumed to be:
  - 125ml/min/m2 in normal pts and
  - 110ml/min/m2 in older pts.

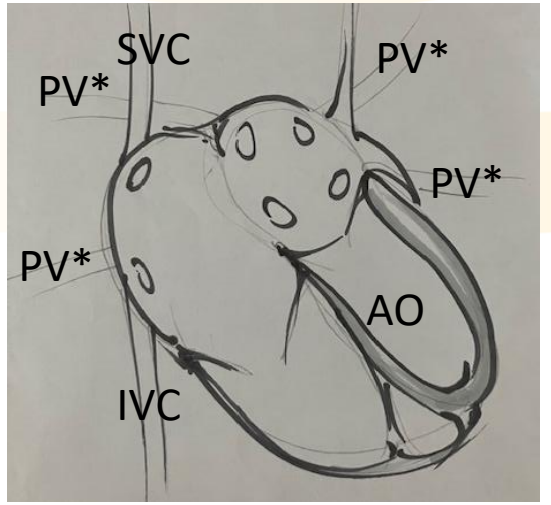
In presence of Shunt slightly more challenging

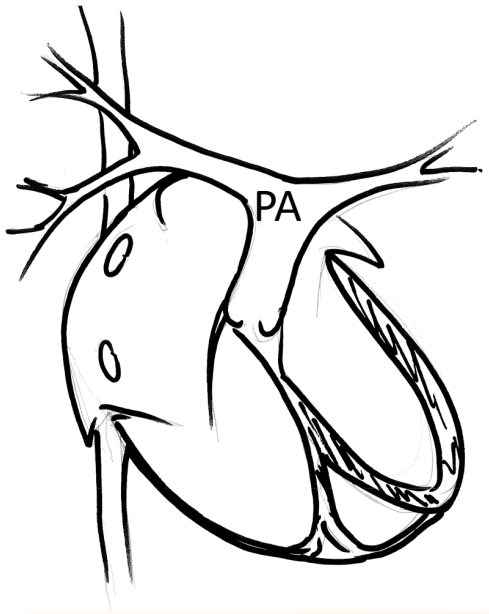
Can use sats run to assess shunt size (QP/QS)

Can use QP to estimate PVR

QP/QS

- QP= O2 consumption x BSA/(PV sat*-PA sat)
- QS= O2 consumption x BSA/(art sat-Mixed Venous sat^)
- QP/QS = (art sat – MV sat) / (PV sat – PA sat)
- QP/QS= (89%-61%)/(98%-86%) = 2.3 to 1





- ^NB: In presence of left to right shunt use ‘Flamm formula:
- Mixed venous sat = (3x SVC) + 1xIVC/4
- MV sat = (3x59%) + 68% / 4 = 61%

*PV sat- Assume normal: 0.97=1.0

QP

- QP = O2 consumption x BSA / (PV sat – PA sat) x Hb x 13.6
- QP = 125ml/min/m2 x 2.37 / (0.98 – 0.86) x 18 x 13.6
- QP = 10 l/min

QS

- QS = O2 consumption x BSA / (AO sat – MV sat) x Hb x 13.6
- QS = 125ml/min/m2 x 2.37 / (0.89 – 0.61) x 18 x 13.6
- QS = 4.3 l/min

Pulmonary vascular resistance

- PVR = Mean PA pressure – Mean PCWP / QP

- PVR = (44mmHg – 26mmHg) / 10 l/min
- PVR = 1.8 WU (NR < 3WU)

Post- script –Clinically in HF even after DCCV to SR.

- Likely to be ongoing problems with RV compliance – measurement of RV function pre-op difficult due to severe TR but clearly increased RVSP (58mmHg) and very raised RVEDP (24mmHg) hinting at RV systolic and RV diastolic dysfunction pre-op. Maybe patient relying on high HR to maintain CO.
- Now has a TV ring. Also L SVC flow has been diverted to RV volume loading.
- LV now has marked increase in flow, which may affect function. Pre-existing increased LV compliance likely (LA pressures very high pre-op – with increased venous return they may be even higher)
- The period of Aflutter may have affected both LV and RV function contributing to decompensation
- On the positive side – PVR pre-op not very high so there may be some reversibility of PHT