Children's Hospital
Cleveland Clinic

Hemodynamics: Beyond the Book

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What is Hemodynamics?

- Physical principles governing the distribution of blood flow and blood pressure in the vascular system

Why Hemodynamics?

- Echo started with anatomy and function.
- Doppler opened the window to blood flow.
- Echo excels at blood flow compared to the competition.
- Neha knows I love understanding blood flow.

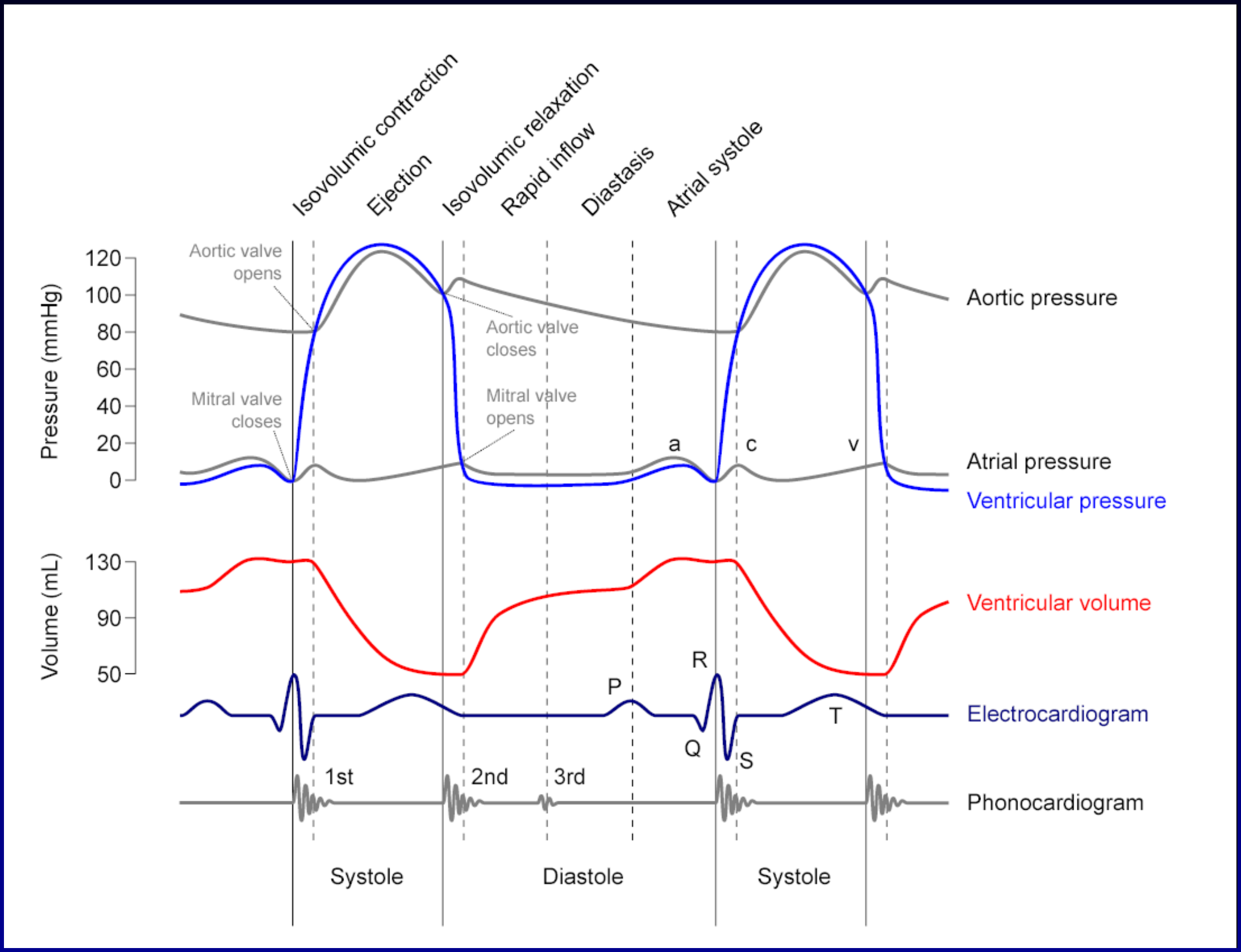
Figure 5. *William R. Milnor, lecturing in 1990.*

Is Hemodynamics Just For CHD?

- It is important in all normal and abnormal circulations.
- Sometimes you just need to look for it to see the impact.

It is All About Physiology: Wiggers Diagram

RV and LV talk to each other but do their own thing



After retiring as professor emeritus in 1953, Wiggers joined the Frank Bunts Institute of the Cleveland Clinic Foundation, taking part in postgraduate training for student doctors and in medical and scientific seminars.

http://en.wikipedia.org/wiki/File:Wiggers_Diagram.svg

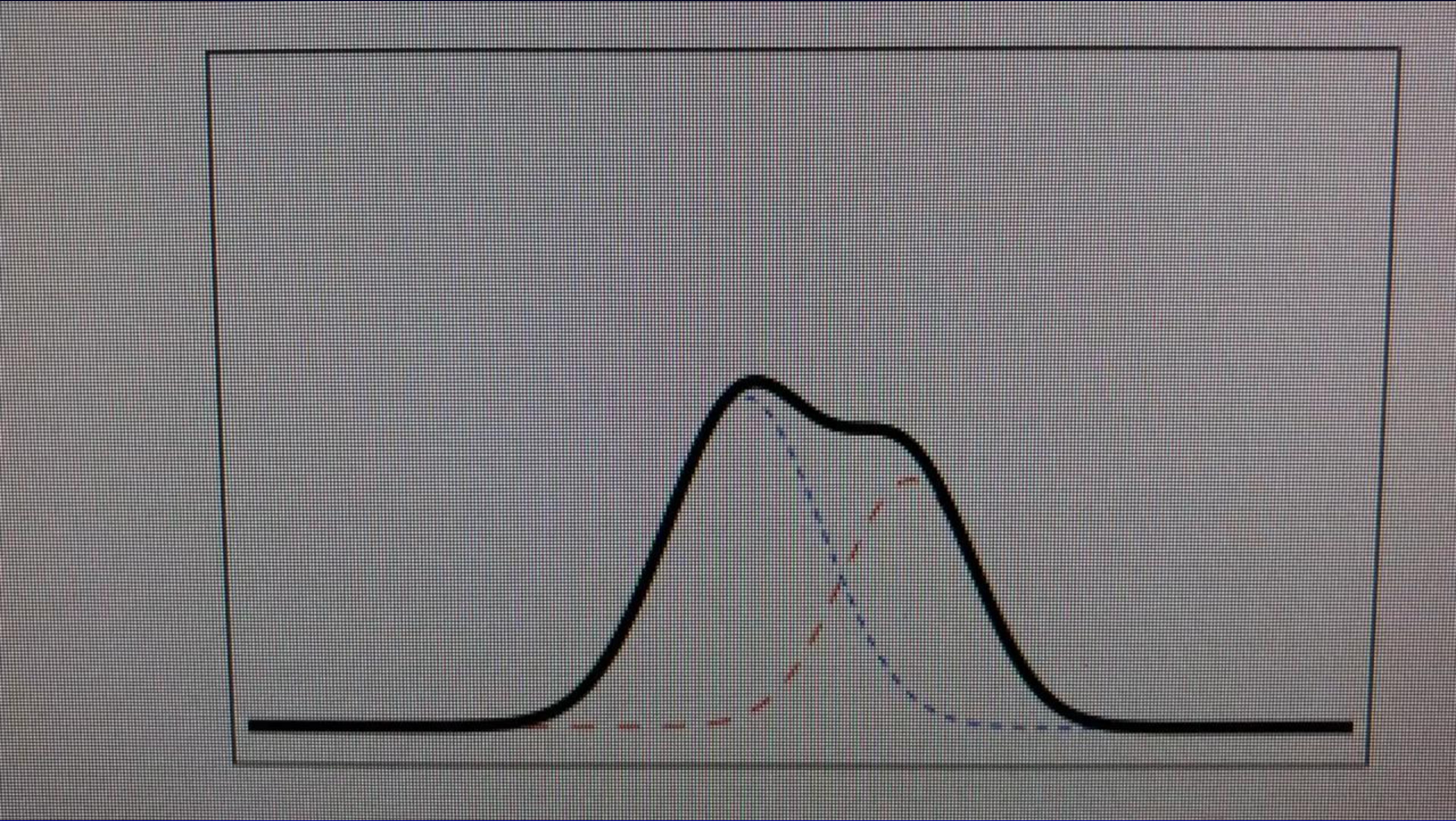
Pressure, Resistance and Blood Flow

- Pressure/Flow = Resistance
- Increase the flow without changing the resistance and the pressure goes up (exercise)
- Increase the resistance without changing flow and the pressure goes up (high blood pressure)
- Increase the resistance without changing the pressure and the flow goes down (severe AS)

Resistance v Impedance

- Resistance (R) is the obstruction to steady state flow
- Impedance (Z) includes resistance and accounts for the extra energy required to move blood in a pulsatile manner including arterial size, arterial length, distensibility and wave reflections.

Wave Reflections



https://www.acs.psu.edu/drussell/Demos/superpositi
on/superposition.html#standing

Pressure and Flow Depends on Where You Look

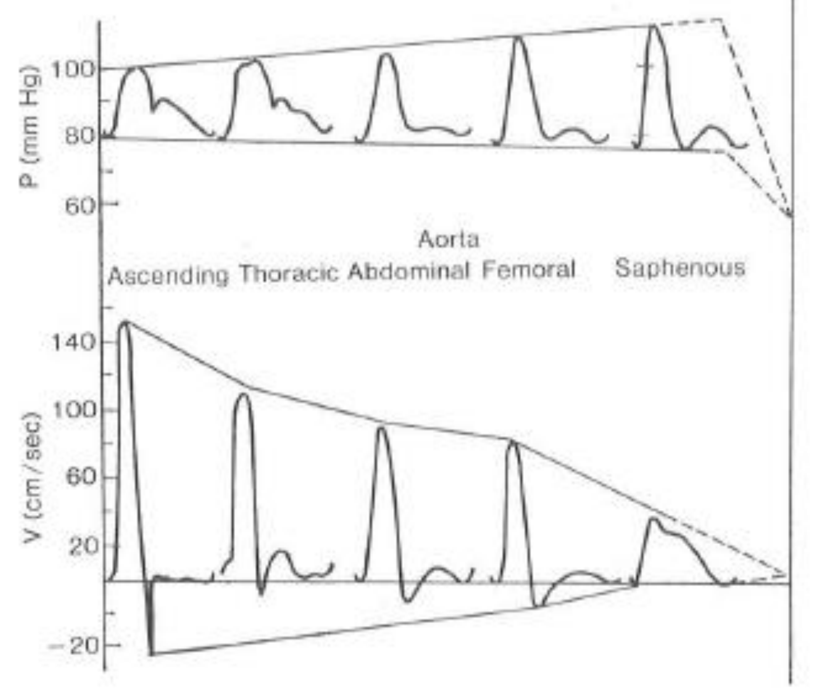


Fig. 6.5. Reprinted with permission from D. A. McDonald. *Blood Flow in Arteries*. Edward Arnold, London, 1974. Pressure (*above*) and average velocity (*below*) in the aorta and arterial branches in the dog, showing amplification of the pressure pulses by reflected waves.

Milnor WR Hemodynamics 1989 p 149, p 226

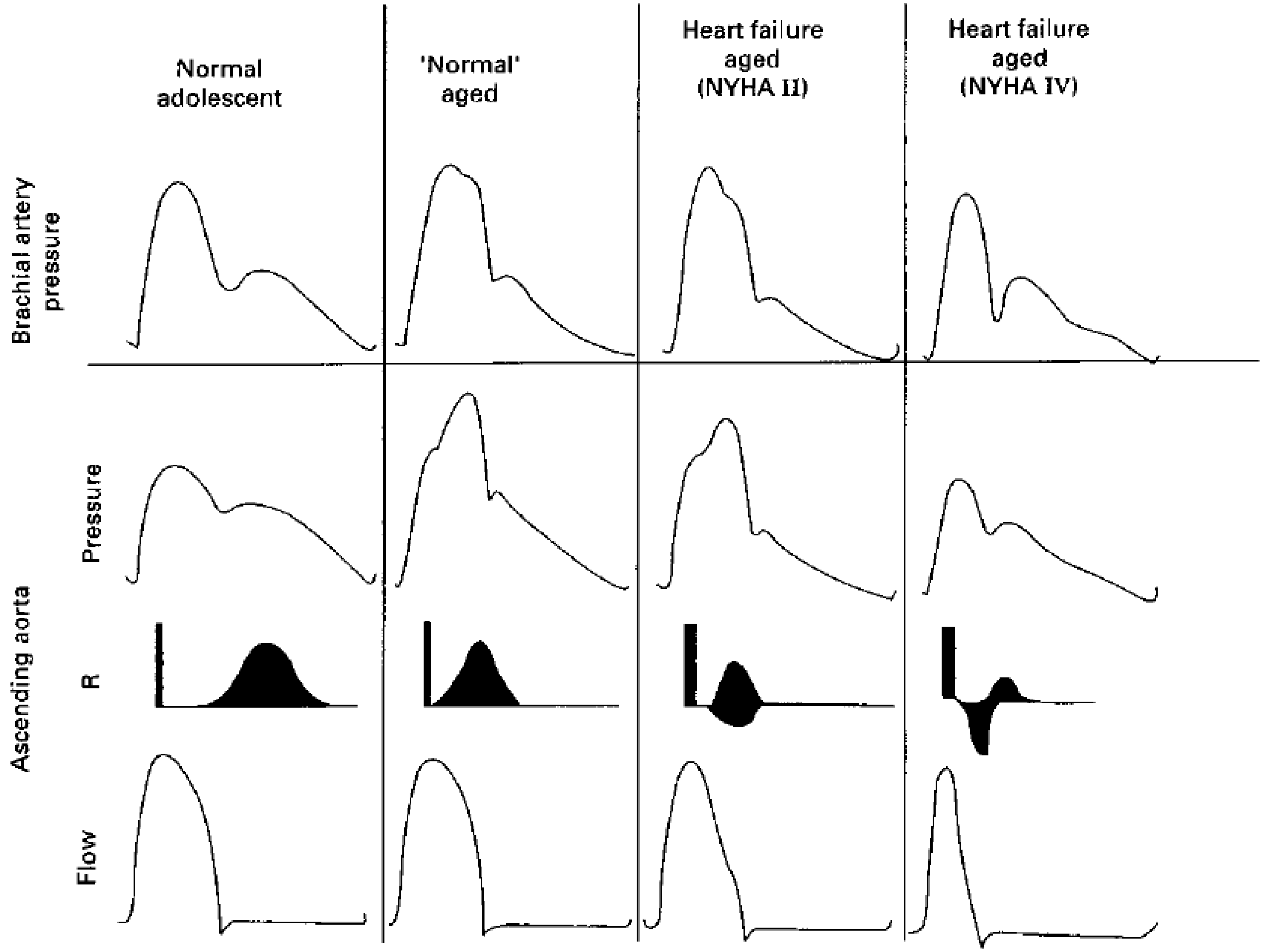


FIG 32. Effects on wave reflection on aortic and brachial pressure and left ventricular output with the development of heart failure in a patient with isolated hypertension, all shown schematically. From top: brachial artery pressure, aortic pressure, wave reflection *(R)* with effect on pressure shown as upward deflection, and effect on flow *(bottom tracing)* as downward reflection. The pressure assumes a dicrotic configuration with the development of heart failure. From Nichols and O'Rourke.[7]

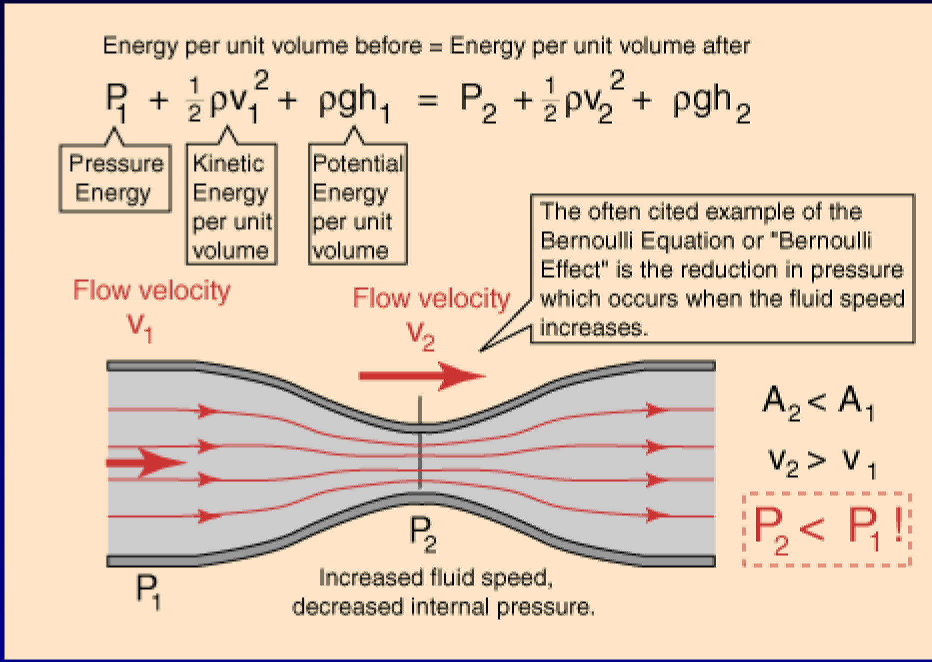
O'Rouke- Curr-Prob-Cardiol-2000

Conservation of Energy

- Pressure is a form of potential energy
- If there is a pressure difference that change in pressure must be accounted for by another form of energy, usually kinetic energy.
- K.E. = ½ mV^2
- Other forms of energy play a minor role: mechanical, heat, light, sound, nuclear
- The 3 cousins Venturi, Bernoulli and Coanda effects are governed by these principles.

Bernoulli Effect

- **The Bernoulli Equation can be considered to be a statement of the conservation of energy principle appropriate for flowing fluids. The qualitative behavior that is usually labeled with the term "Bernoulli effect" is the lowering of fluid pressure in regions where the flow velocity is increased. This lowering of pressure in a constriction of a flow path may seem counterintuitive, but seems less so when you consider pressure to be energy density. In the high velocity flow through the constriction, kinetic energy must increase at the expense of pressure energy.**

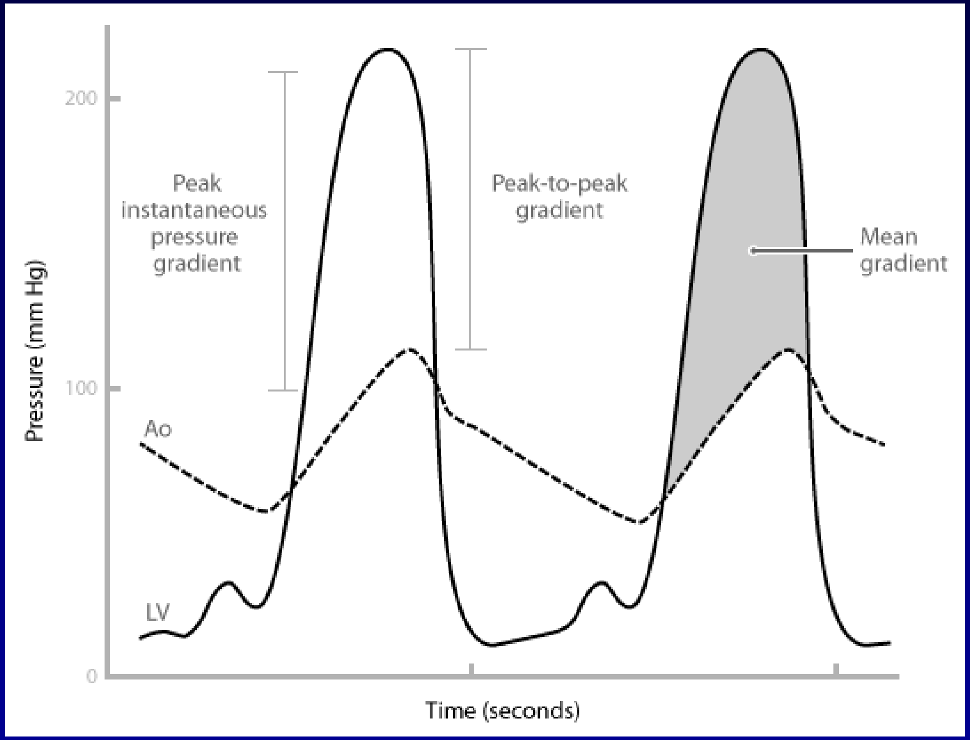


http://hyperphysics.phy-astr.gsu.edu/hbase/hframe.html

What Would I like you to Remember So Far?

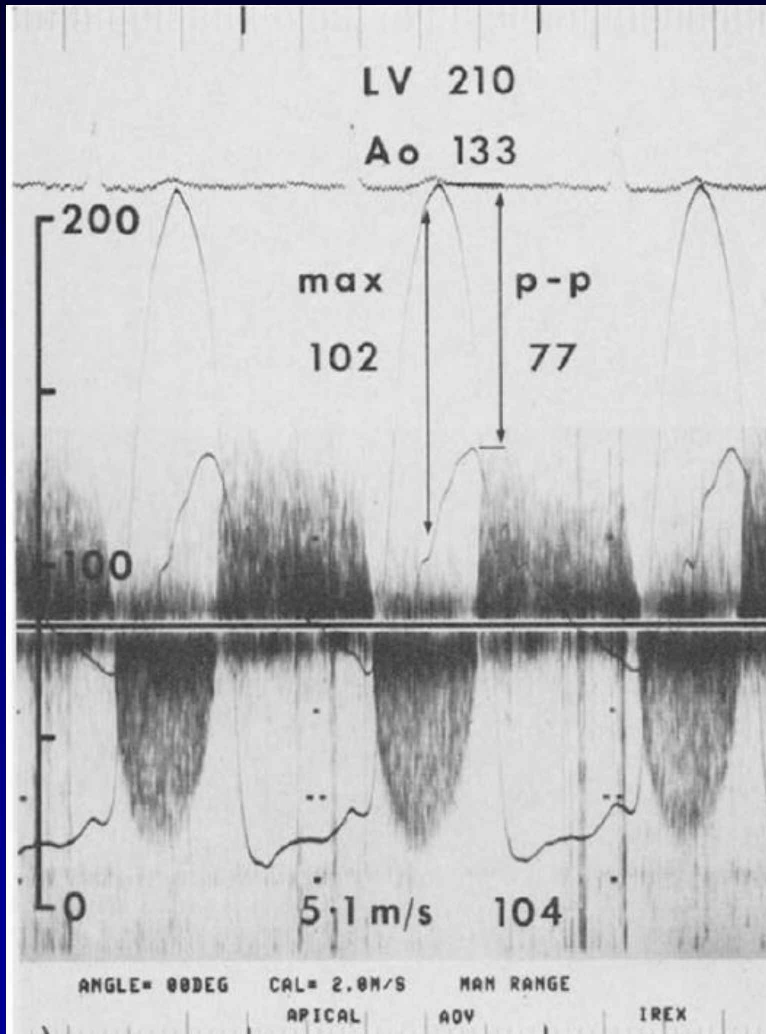
- High Pressure to Low Pressure increases velocity by v^2
- Blood moves toward low resistance or impedance
- Blood flow is a combination of forward flow and reflected waves
- It is important to break down the cardiac cycle into more than just diastole and systole

Why Do We Look at Mean Gradients?



- Peak aortic pressure is not at the same time as the peak LV pressure
- Cath measures P-P pressure
- Echo measures peak instantaneous pressure

What Is the Difference Between Cath and Echo Valve Gradients?



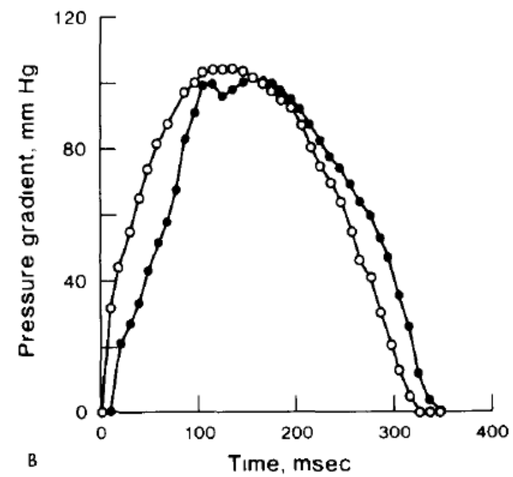


Figure 1. A, Simultaneous Doppler and dual catheter pressure recordings in a patient with severe valvular aortic stenosis. The maximal catheter gradient (max) of 102 mm Hg corresponded closely to the maximal instantaneous Doppler gradient of 104 mm Hg. The peak to peak catheter pressure gradient (p-p) was 77 mm Hg. Note that the peak left ventricular (LV) pressure and the peak aortic (Ao) pressures are nonsynchronous; therefore, the peak to peak catheter pressure gradient does not represent an instantaneous pressure gradient. **B**, Same patient. Instantaneous Doppler and catheter pressure gradients from the digitized Doppler spectral velocity envelope and simultaneous left ventricular and ascending aortic pressure waveforms of the second beat from **A**. The instantaneous catheter gradient (**closed circles**) is comparable with the Doppler-derived gradient (**open circles**). Note the slight phase delay related to the fluid-filled catheter system. The mean catheter gradient (area under the curve divided by gradient time) was 69 mm Hg and the mean Doppler-derived gradient was 74 mm Hg.

Currie et al J Am Coll Cardiol. 1986 Apr;7(4):800-6

What Is the Difference Between Cath and Echo Valve Gradients?

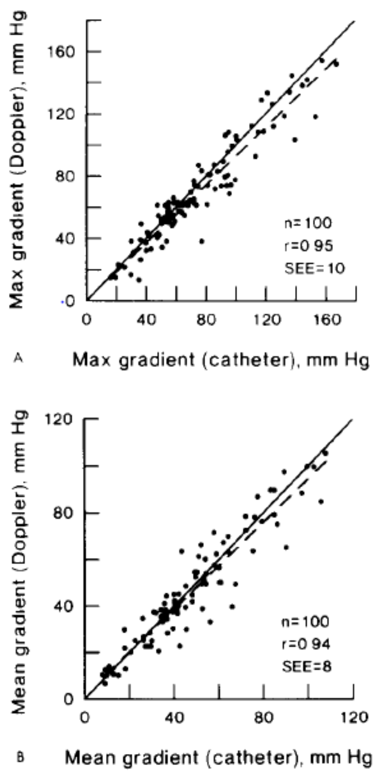


Figure 3. A, Correlation of simultaneous maximal (Max) Doppler and catheter pressure gradients in the 100 stenotic lesions. The regression equation is: Doppler gradient = 0.5 + 0.93 × catheter gradient. **B**, Correlation of simultaneous mean Doppler and catheter pressure gradients in the 100 lesions. The regression equation is: Doppler gradient = 1.8 + 0.93 × catheter gradient. The **dotted lines** represent the regression lines and the **solid lines** represent the lines of identity.

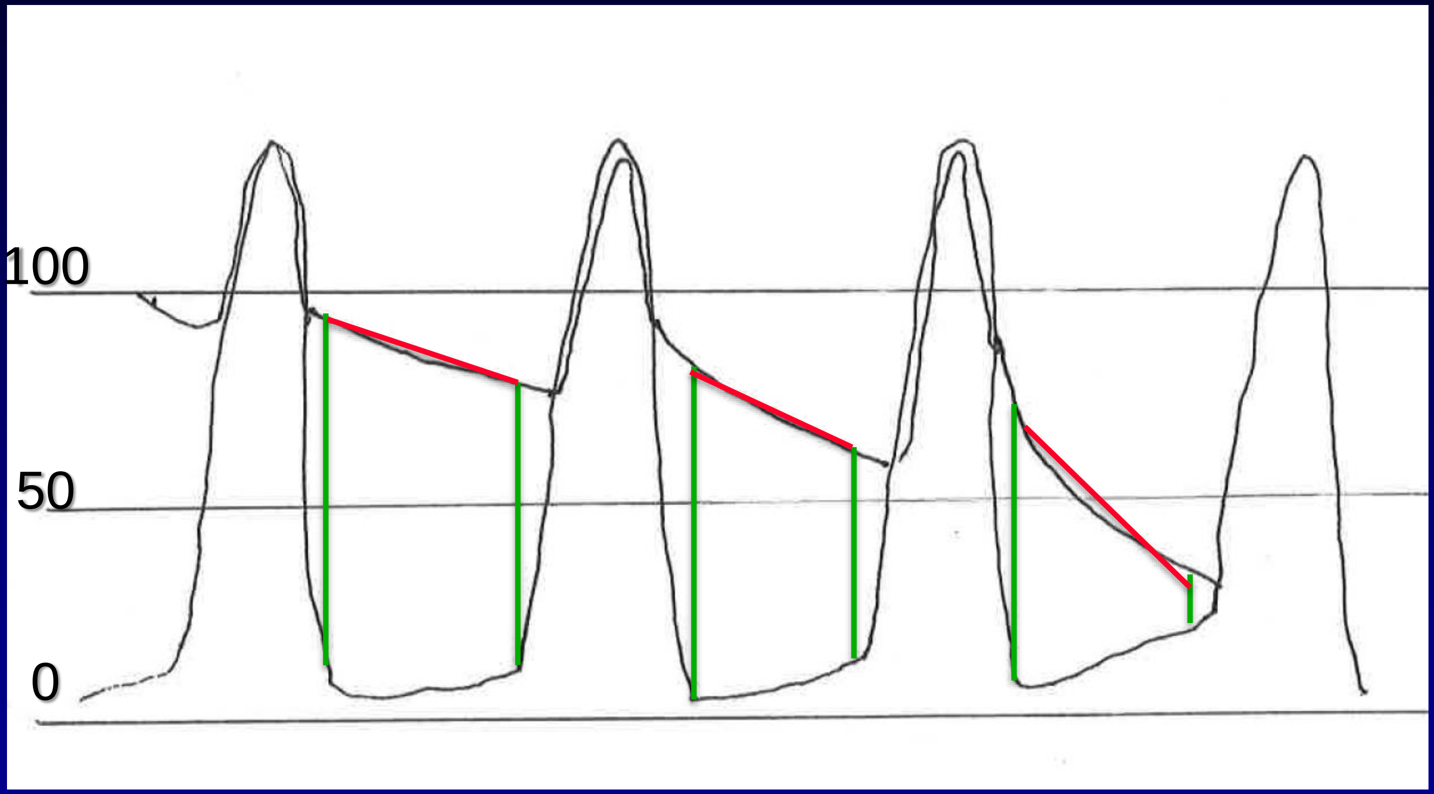
Table 1. Comparisons of Right Ventricular (RVOT) and Left Ventricular (LVOT) Outflow Tract Obstructive Lesions

	RVOT (n = 38) (mm Hg)	LVOT (n = 62) (mm Hg)	p Value
Maximal Doppler gradient	76 ± 32	69 ± 34	NS
Maximal catheter gradient	80 ± 32	74 ± 35	NS
Peak to peak (catheter) gradient	71 ± 33	51 ± 33	<0.01
Mean Doppler gradient	50 ± 21	45 ± 24	NS
Mean catheter gradient	49 ± 20	47 ± 25	NS
Maximal catheter − maximal Doppler gradient	4 ± 9	5 ± 11	NS
Mean catheter − mean Doppler gradient	−1 ± 8	3 ± 8	NS
Peak to peak − maximal Doppler gradient	−5 ± 10	−19 ± 13	<0.001
Peak to peak − maximal catheter gradient	−9 ± 6	−23 ± 11	<0.001

NS = not significant.

Currie et al J Am Coll Cardiol. 1986 Apr;7(4):800-6

Why is Aortic Regurgitation High Velocity? Why Does it have a pressure half time?



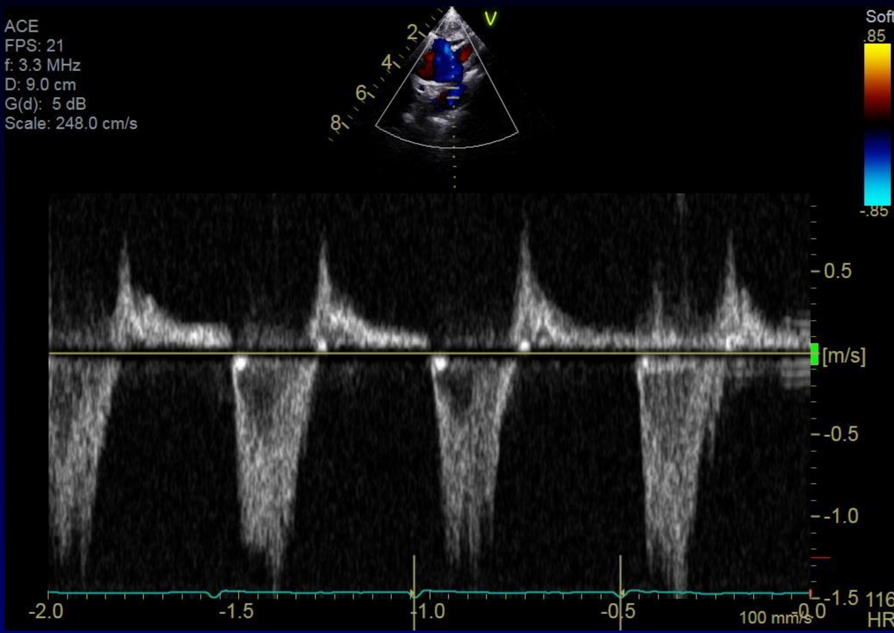
Mild

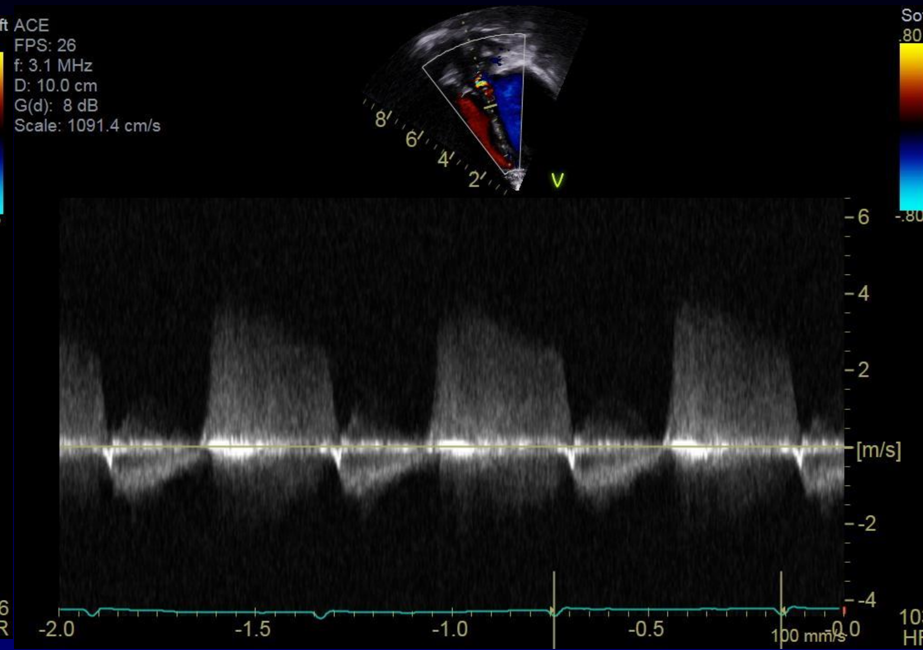
High Velocity and P1/2

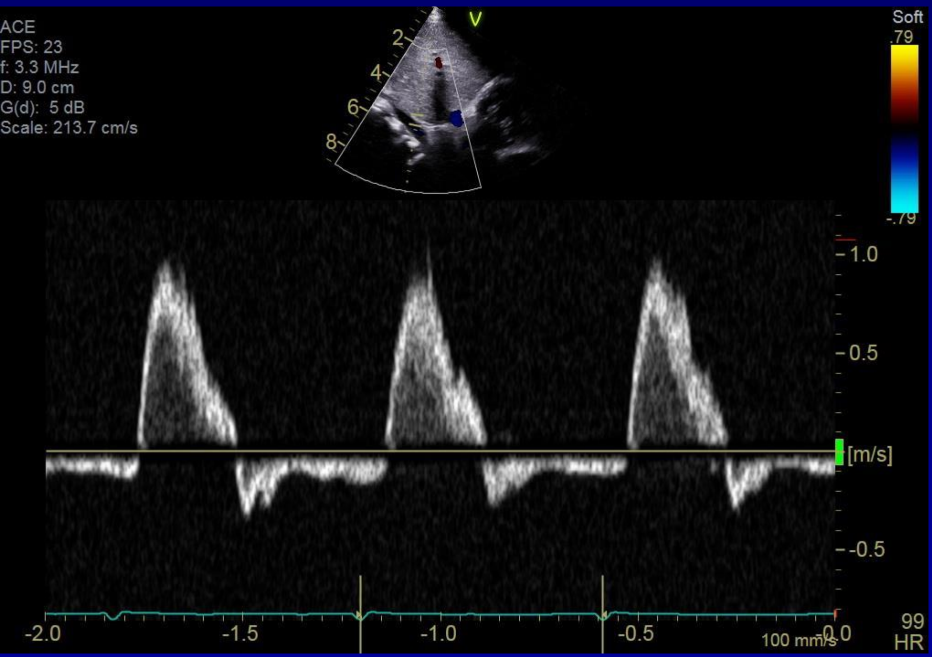
Mod

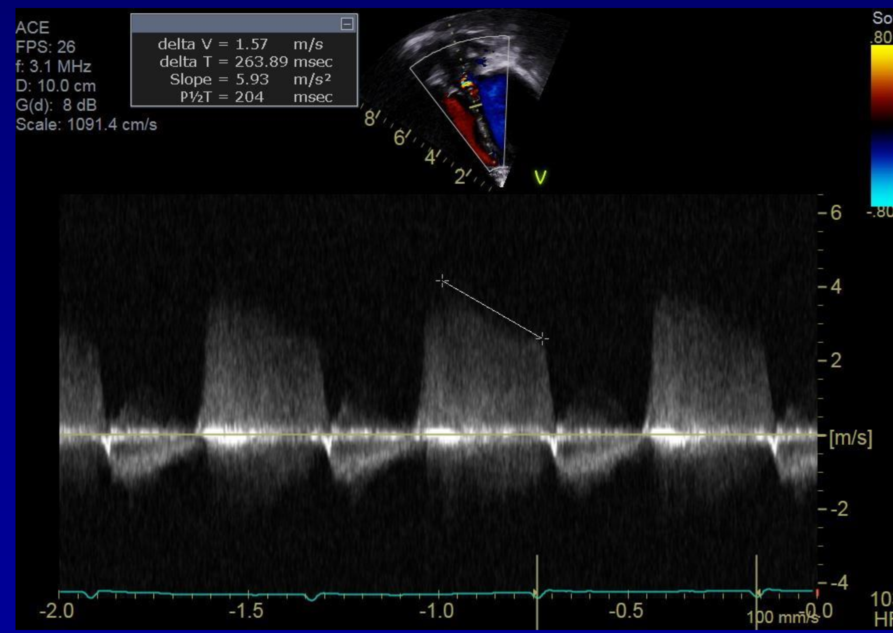
Severe

Low Velocity and P1/2

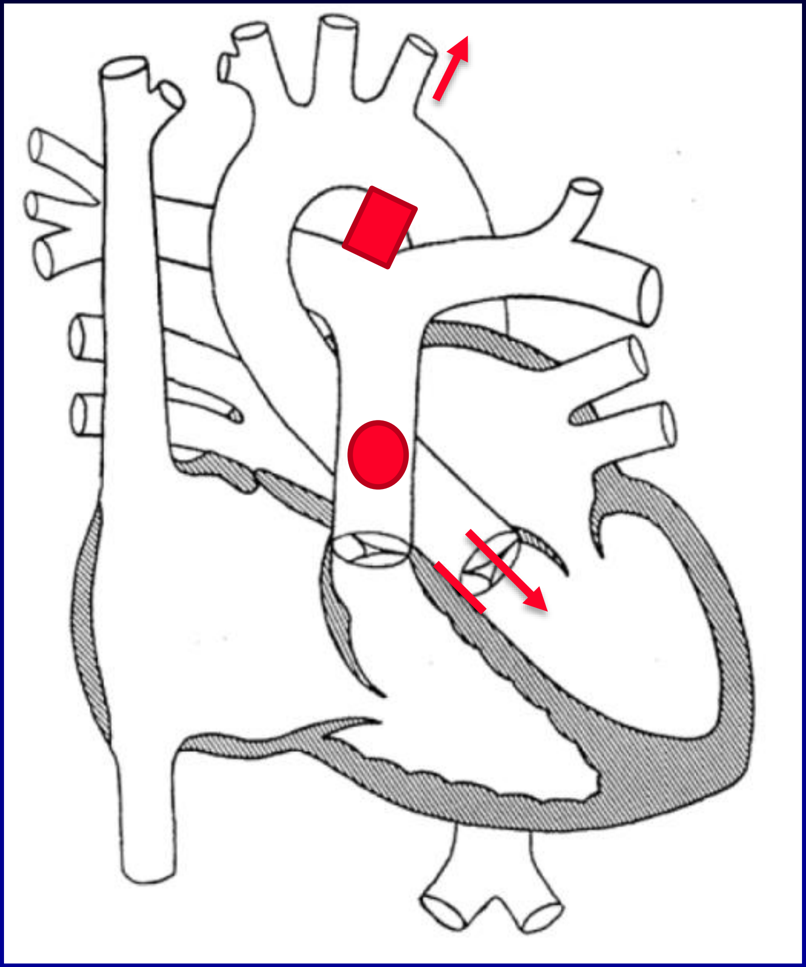

ACE
FPS: 21
f: 3.3 MHz
D: 9.0 cm
G(d): 5 dB
Scale: 248.0 cm/s
Soft
.85
-.85
0.5
[m/s]
-0.5
-1.0
-1.5
116
HR
-2.0
-1.5
-1.0
-0.5
0.0
100 mm/s

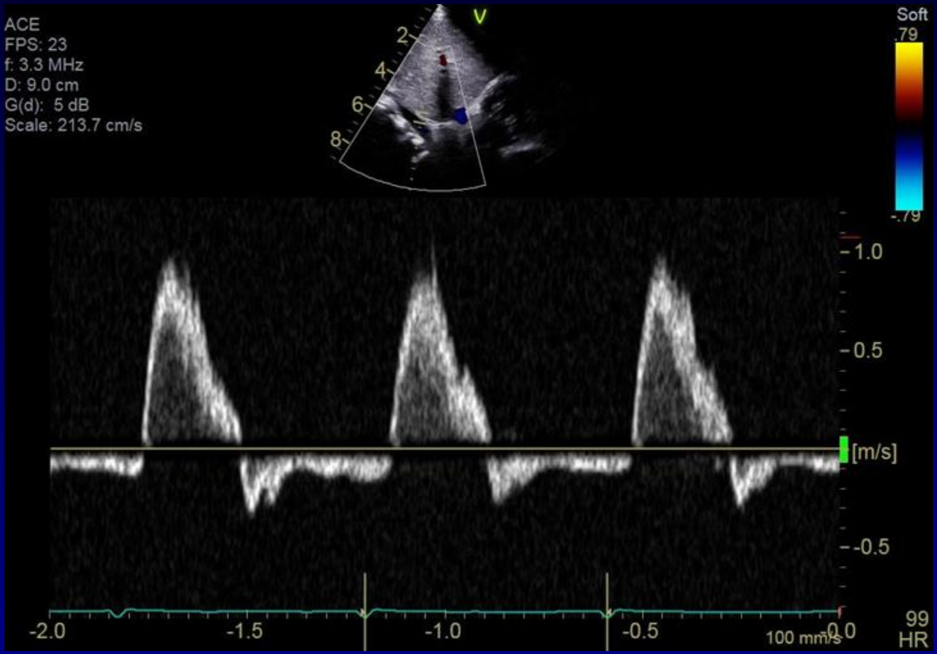

ACE
FPS: 26
f: 3.1 MHz
D: 10.0 cm
G(d): 8 dB
Scale: 1091.4 cm/s
Soft
.80
-.80
6
4
2
[m/s]
-2
-4
103
HR
-2.0
-1.5
-1.0
-0.5
0.0
100 mm/s


ACE
FPS: 23
f: 3.3 MHz
D: 9.0 cm
G(d): 5 dB
Scale: 213.7 cm/s
Soft
.79
-.79
1.0
0.5
[m/s]
-0.5
99
HR
-2.0
-1.5
-1.0
-0.5
0.0
100 mm/s


ACE
FPS: 26
f: 3.1 MHz
D: 10.0 cm
G(d): 8 dB
Scale: 1091.4 cm/s
delta V = 1.57 m/s
delta T = 263.89 msec
Slope = 5.93 m/s²
P½T = 204 msec
Soft
.80
-.80
6
4
2
[m/s]
-2
-4
103
HR
-2.0
-1.5
-1.0
-0.5
0.0
100 mm/s

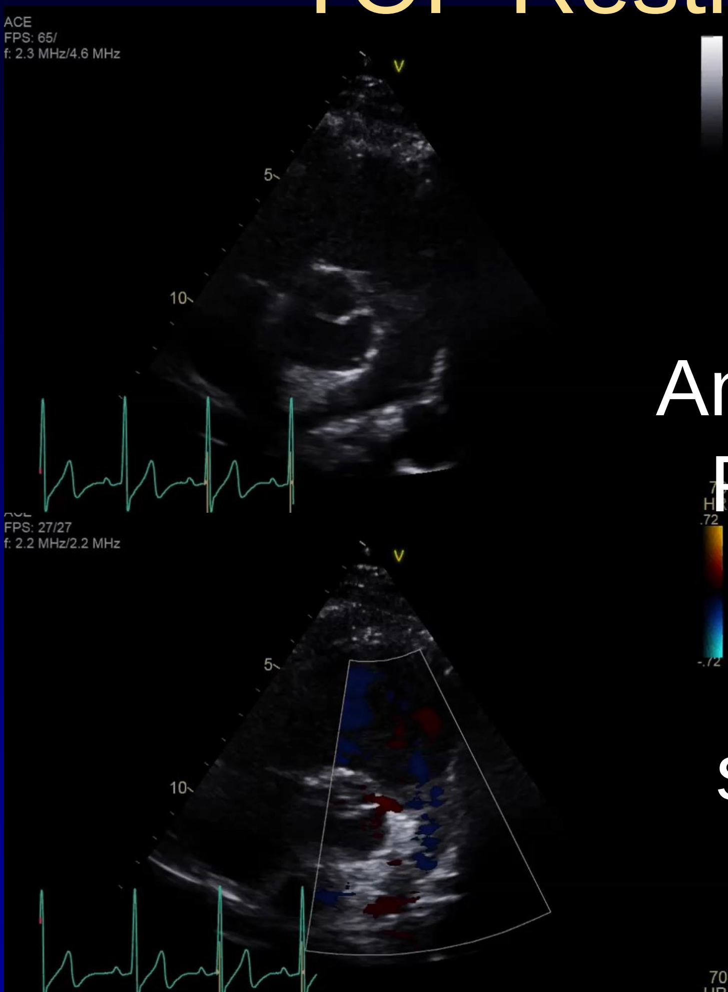
Does Retrograde Descending Aortic Flow Always Mean Aortic Regurgitation?

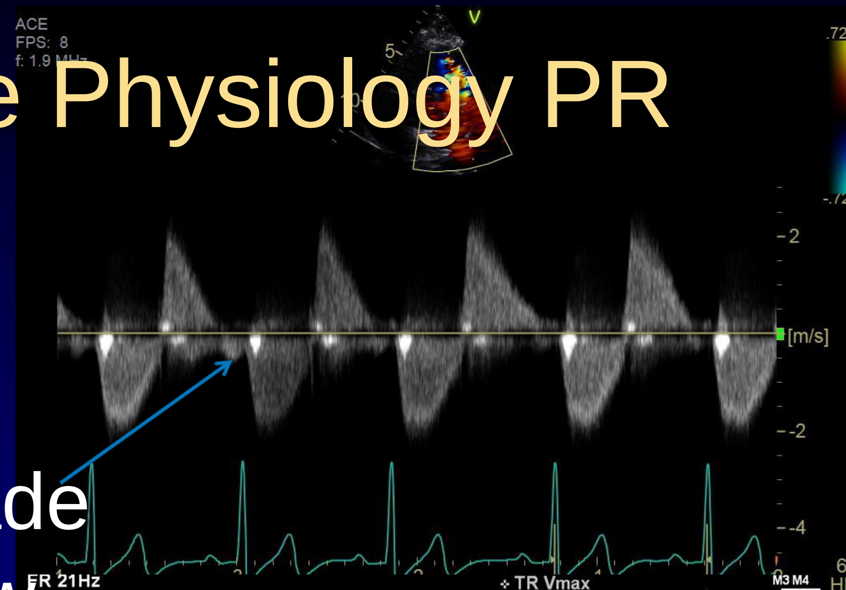




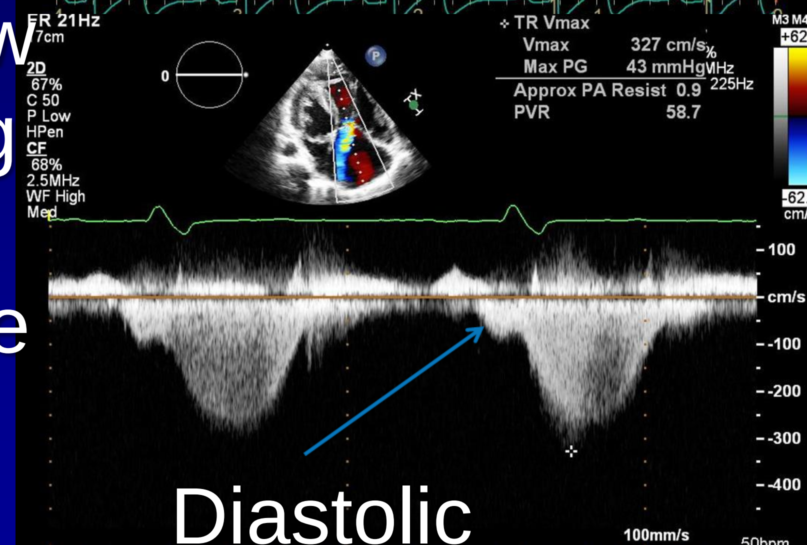
Aortic Regurgitation
PDA
Cerebral AVM
AP Window
LV-Aortic Tunnel
Coronary Fistula

TOF Restrictive Physiology PR



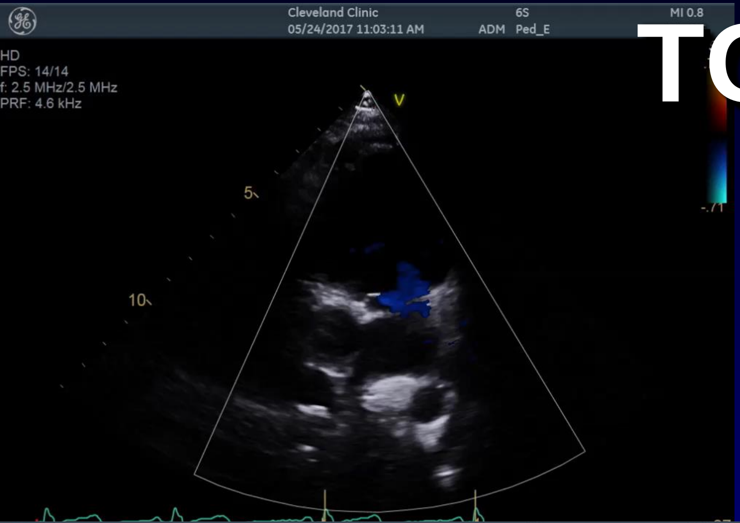


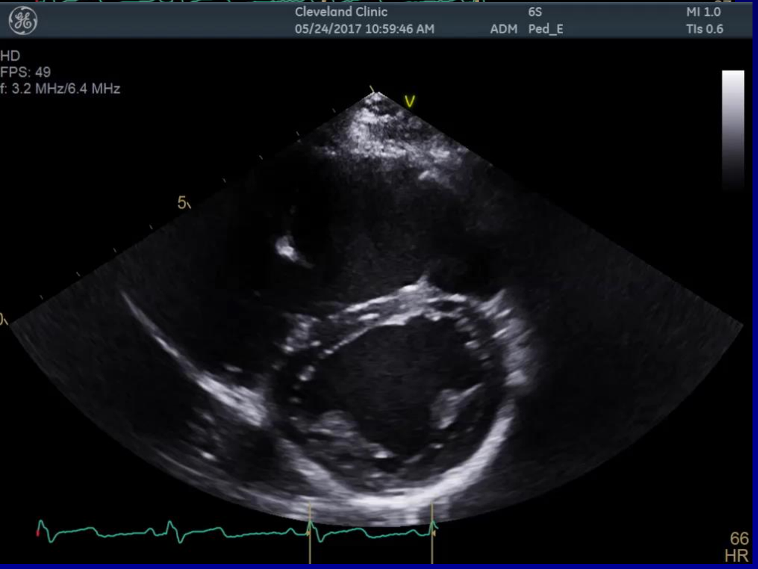
Antegrade PA flow during atrial systole

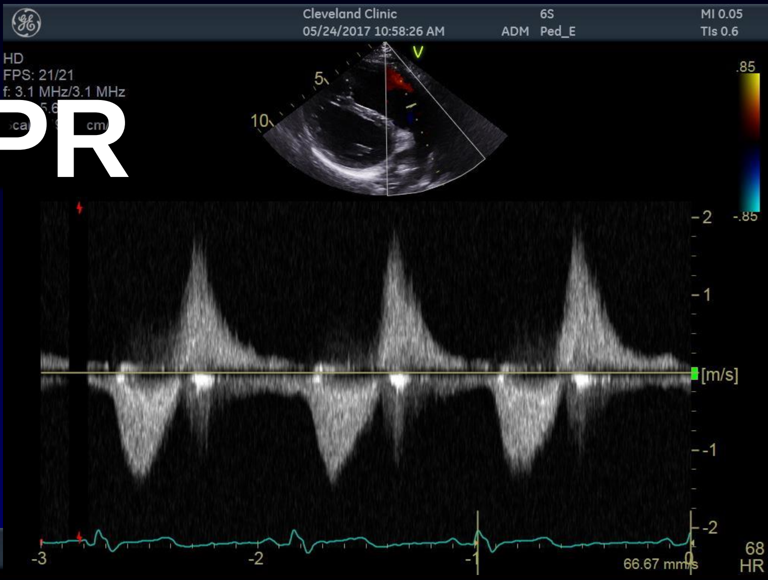


Diastolic TR

TOF free PR

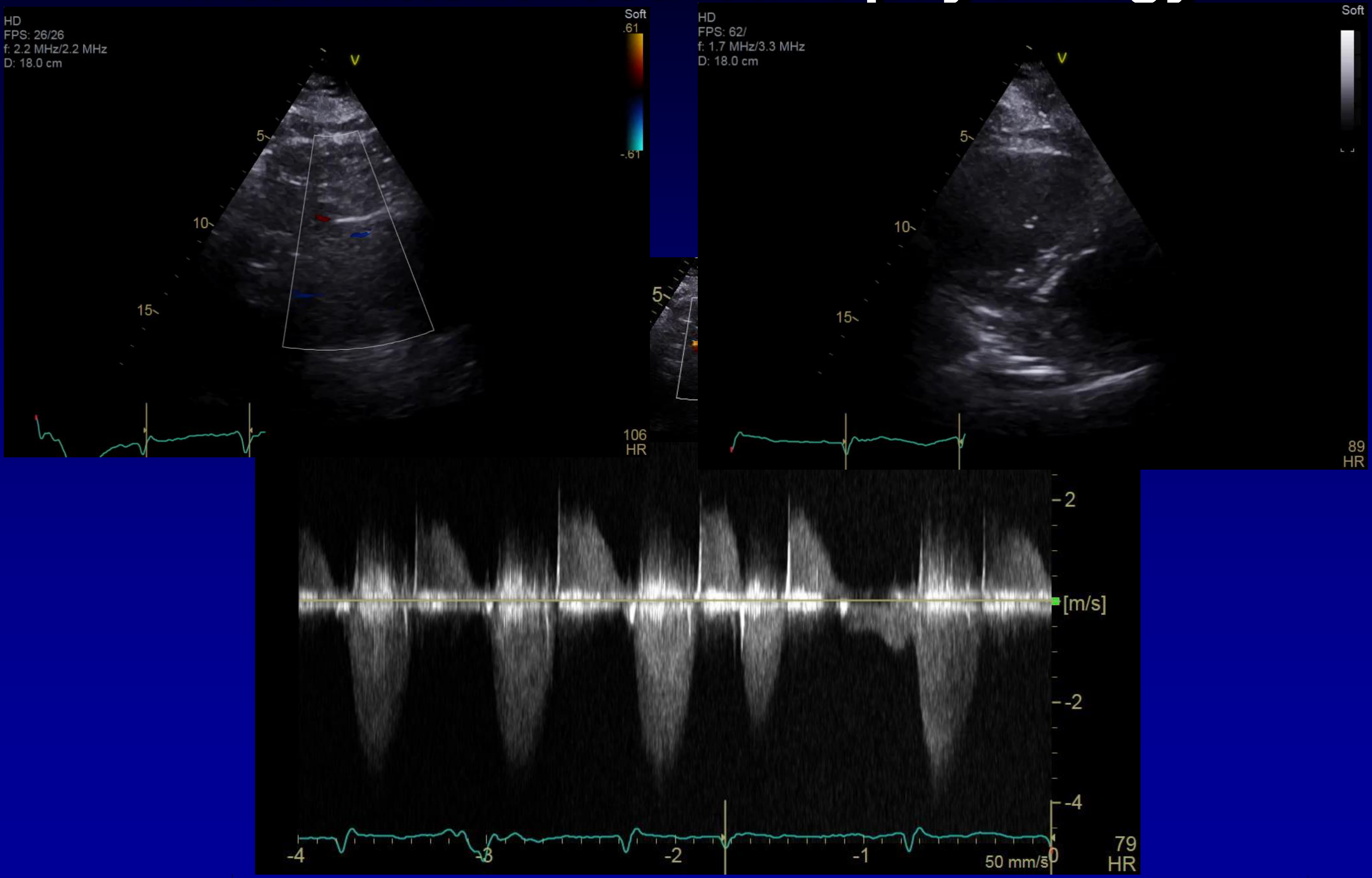

Cleveland Clinic
05/24/2017 11:03:11 AM
ADM Ped_E
6S
MI 0.8
HD
FPS: 14/14
f: 2.5 MHz/2.5 MHz
PRF: 4.6 kHz


Cleveland Clinic
05/24/2017 10:59:46 AM
ADM Ped_E
6S
MI 1.0
TIs 0.6
HD
FPS: 49
f: 3.2 MHz/6.4 MHz
66
HR


Cleveland Clinic
05/24/2017 10:58:26 AM
ADM Ped_E
6S
MI 0.05
TIs 0.6
HD
FPS: 21/21
f: 3.1 MHz/3.1 MHz
[m/s]
66.67 mm/s
68
HR


ACE
FPS: 44
f: 2.3 MHz/4.6 MHz
64
HR

60 year old congenital PS/PR intermittent restrictive physiology



Can TR and PR occur at the same time?

- It depends on the rate of relaxation of the RV.
- Similarly the rate of rise of RV pressure may delay the onset of right to left shunting across a VSD until late systole.

When is systole not systole and diastole not diastole?

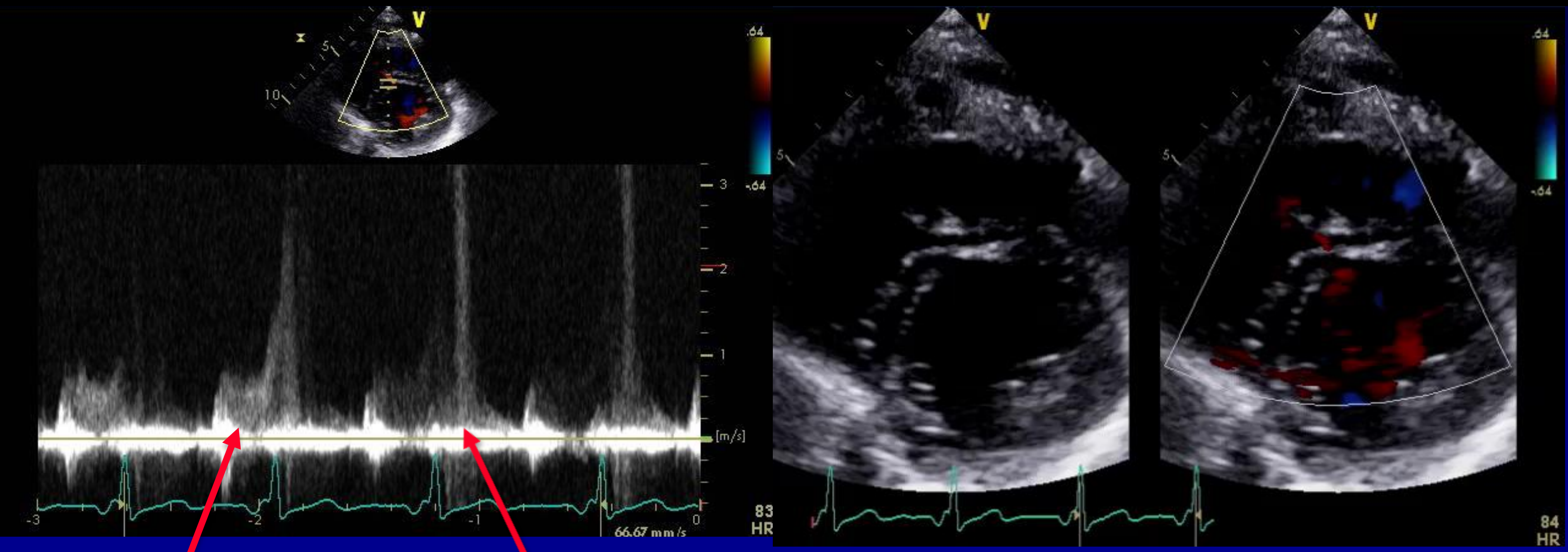
PR

TR

VSD

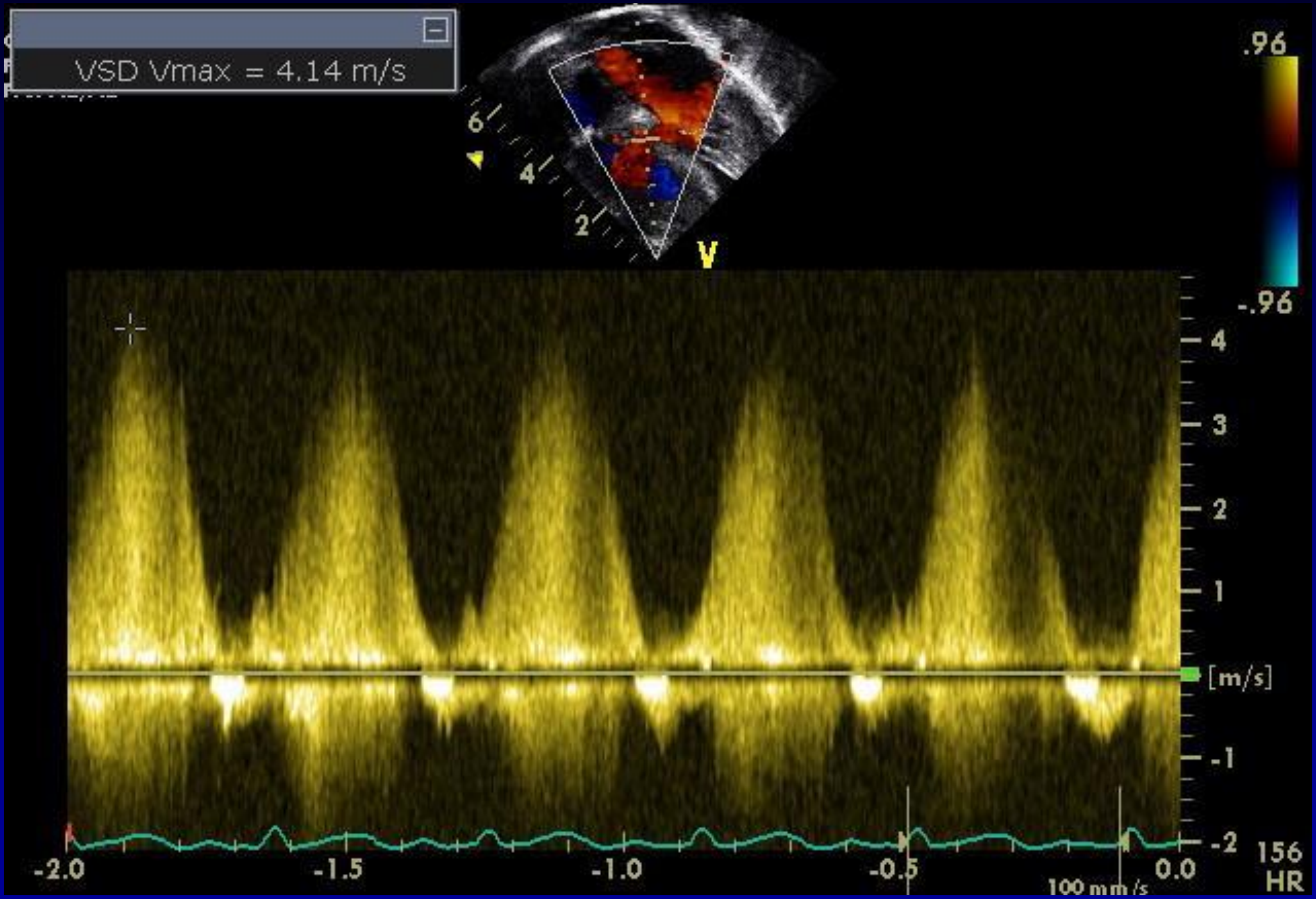
- Size, location matters
- Peak velocity is important but it is not the only thing you should measure

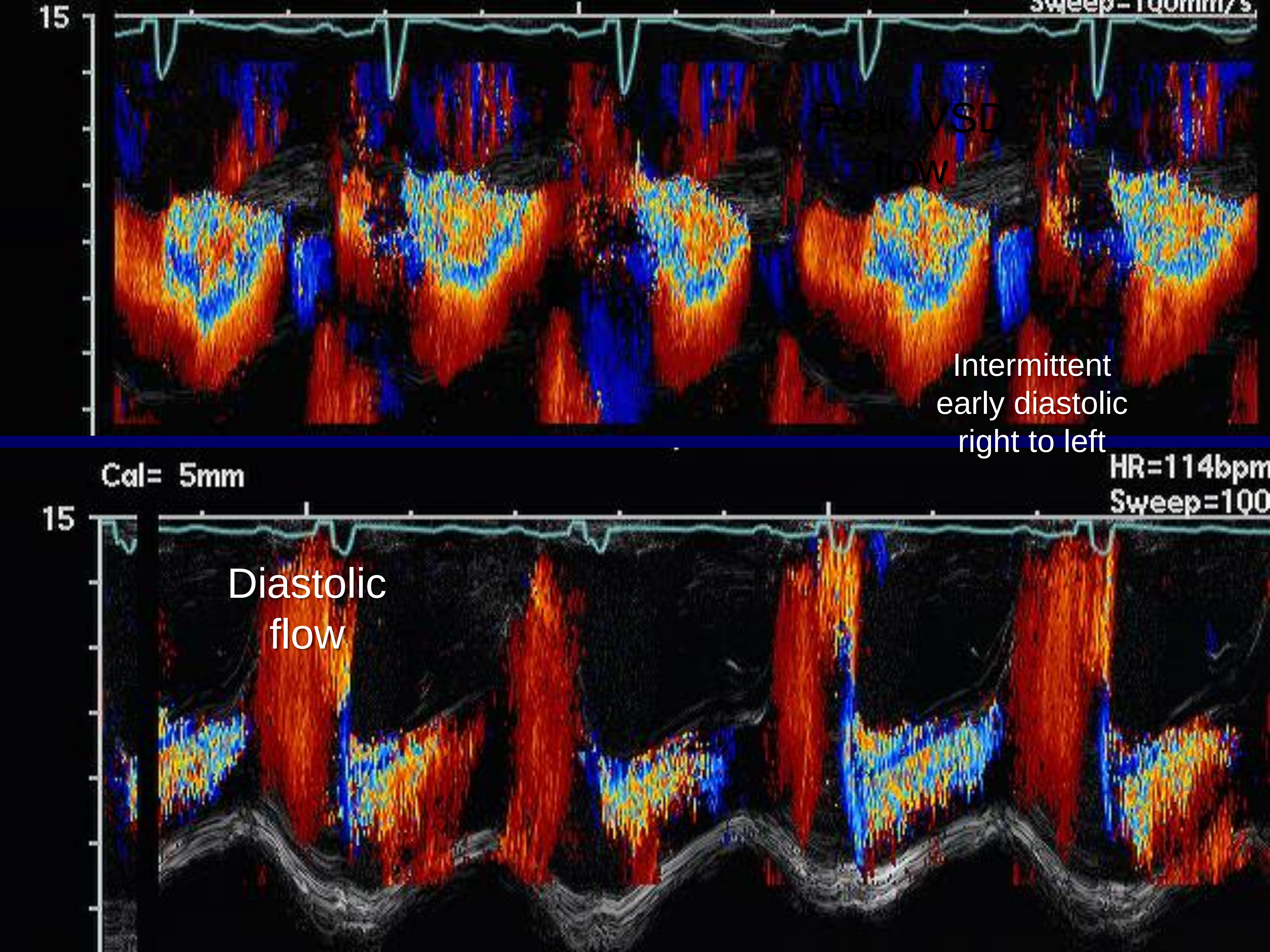
16 year old with high-frequency systolic murmur that ends 1/3 into systole



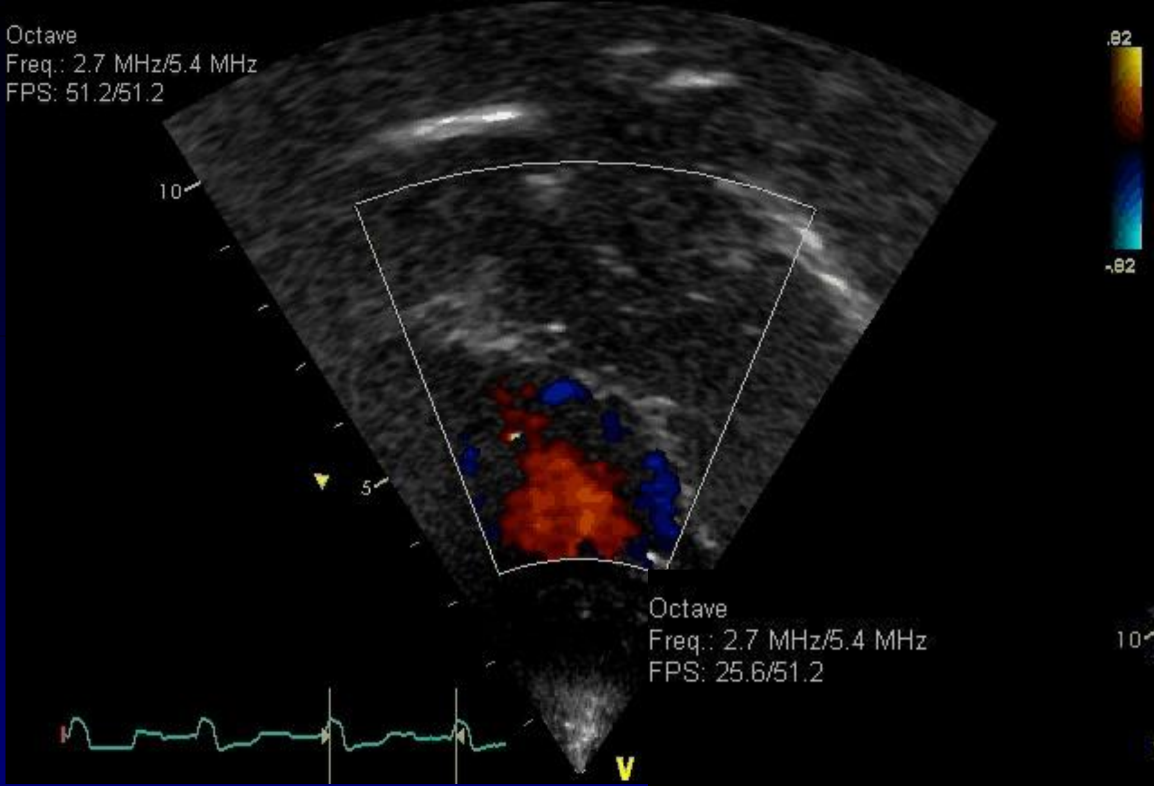
Diastolic flow Early Systolic flow

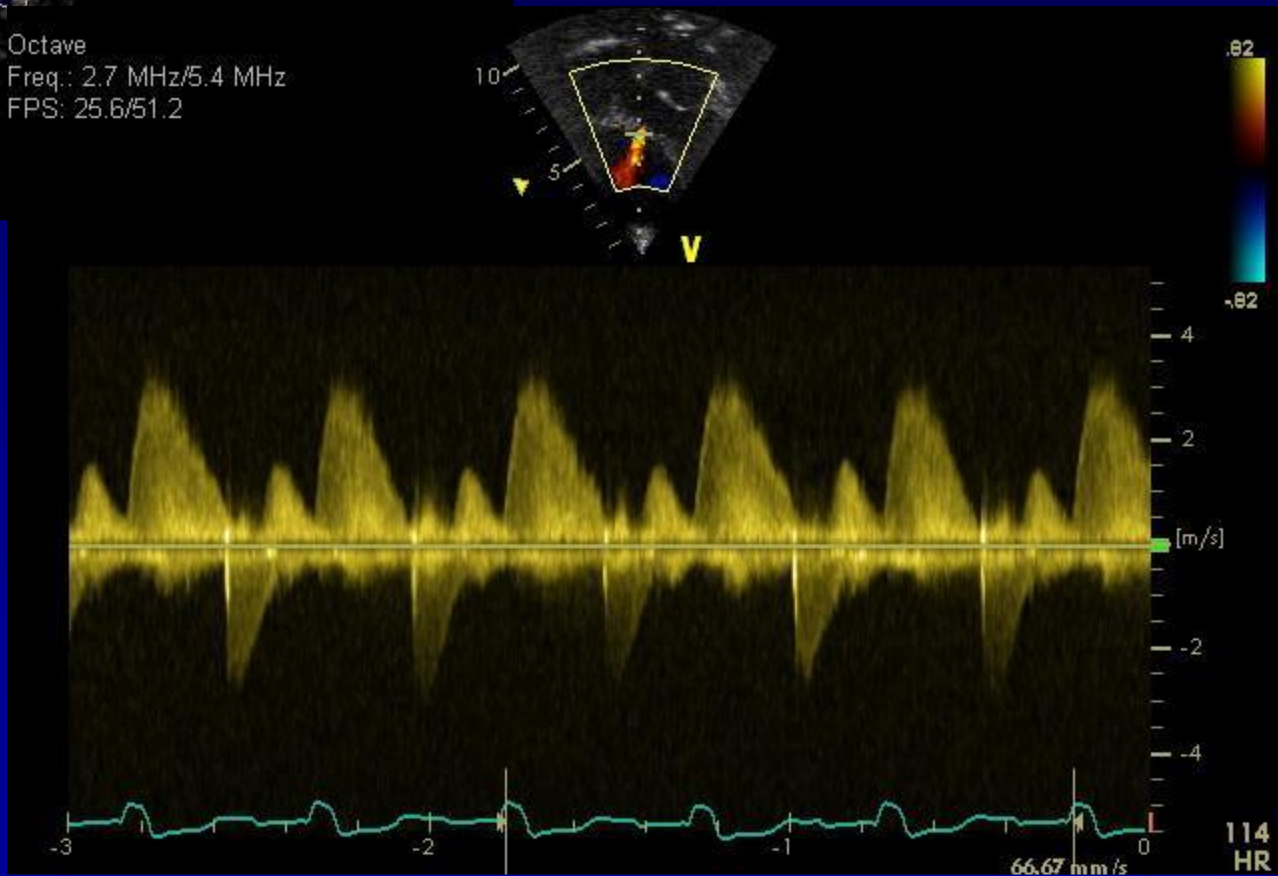
VSD- LV-RV pressure gradient

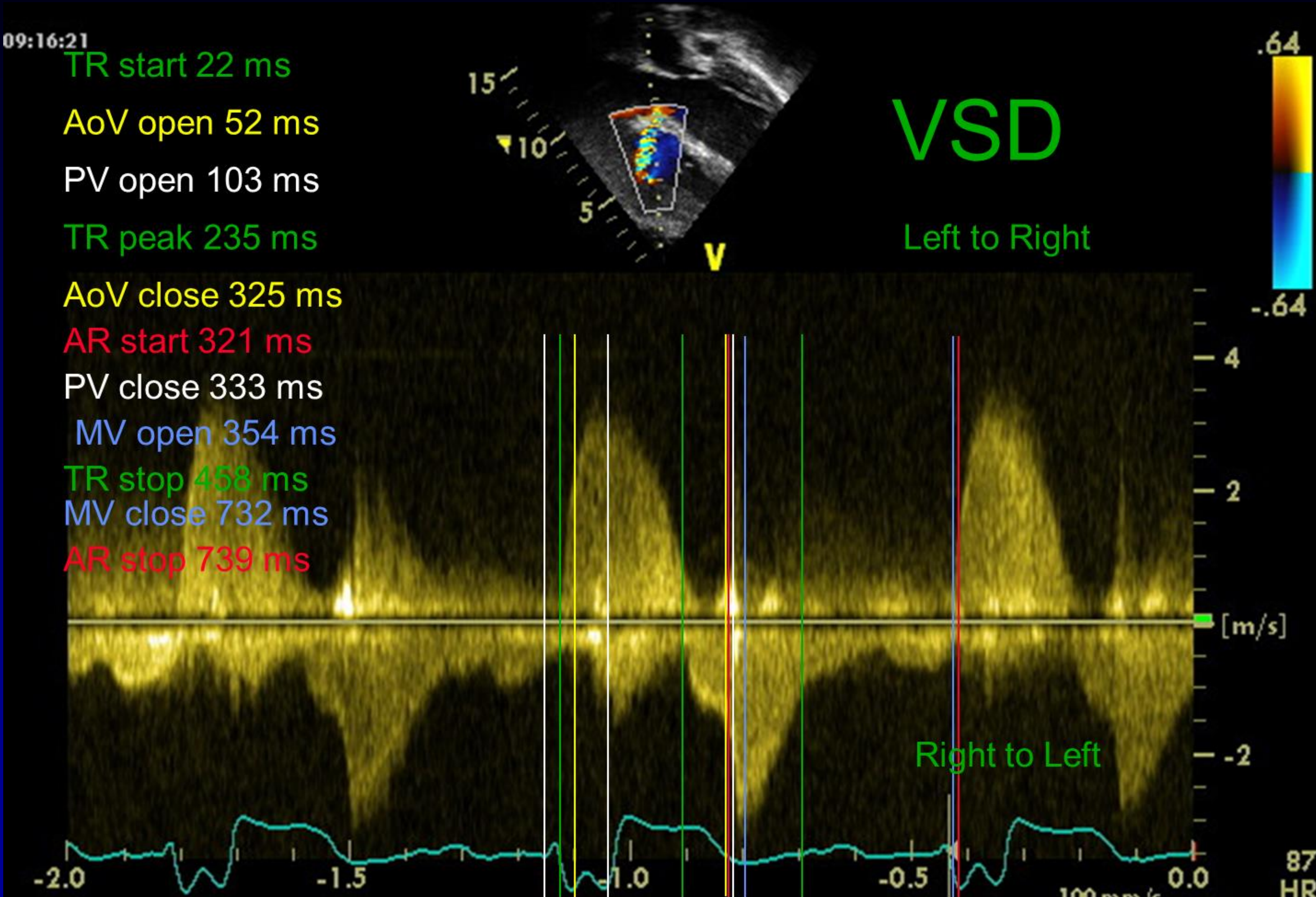



15
Peak VSD flow
Intermittent early diastolic right to left
Cal= 5mm
HR=114bpm
Sweep=100
15
Diastolic flow

TOF Repair with equal systolic RV and LV pressure






09:16:21
TR start 22 ms
AoV open 52 ms
PV open 103 ms
TR peak 235 ms
AoV close 325 ms
AR start 321 ms
PV close 333 ms
MV open 354 ms
TR stop 458 ms
MV close 732 ms
AR stop 739 ms
VSD
Left to Right
Right to Left
.64
-.64
4
2
[m/s]
-2
15
10
5
V
-2.0
-1.5
-1.0
-0.5
0.0
100 mm/s
87
HR

Pulmonary Hypertension -PVR

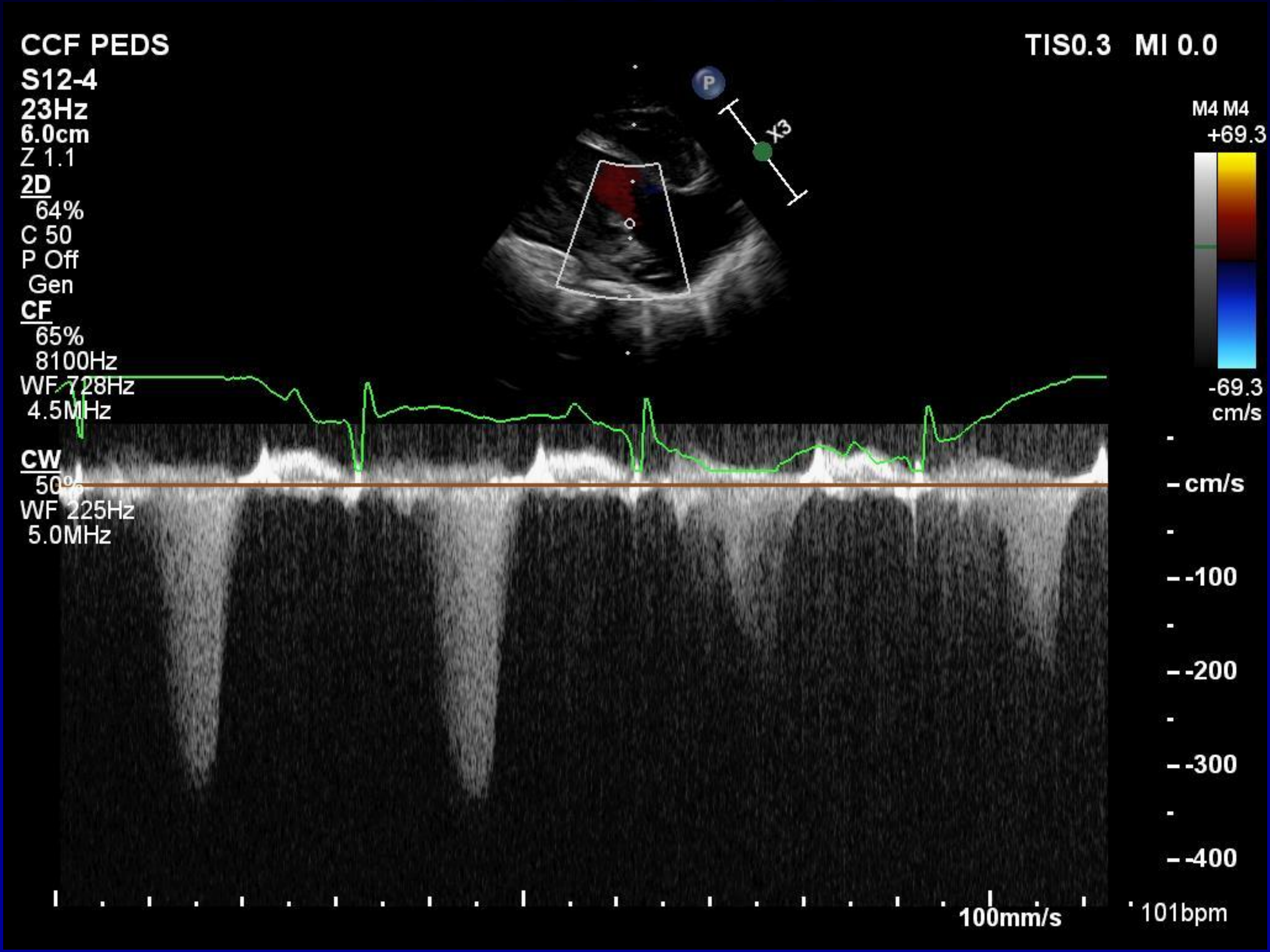
- High TR velocity does not necessarily mean PHT, just RV hypertension
- Pulmonary regurgitation velocity can help estimate diastolic PA pressure
- Branch PA waveforms can be a window into PVR

When Are Wave Reflections Useful?

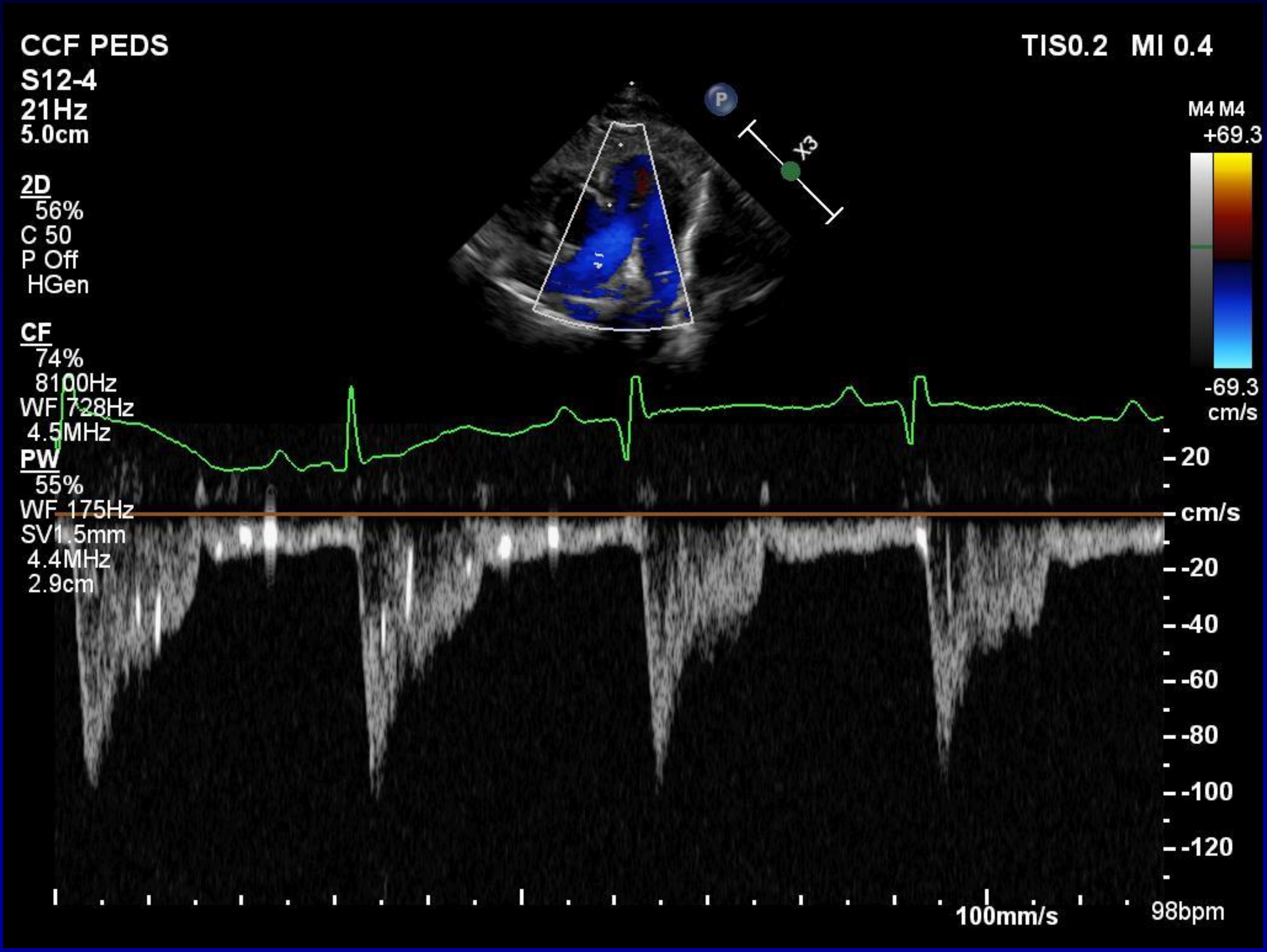
- Understanding the shape of the arterial wave form
- Understanding the shape of the Doppler flow pattern in arteries.

Newborn with VSD, PDA, high PVR and arch hypoplasia

TR 3.8 m/s

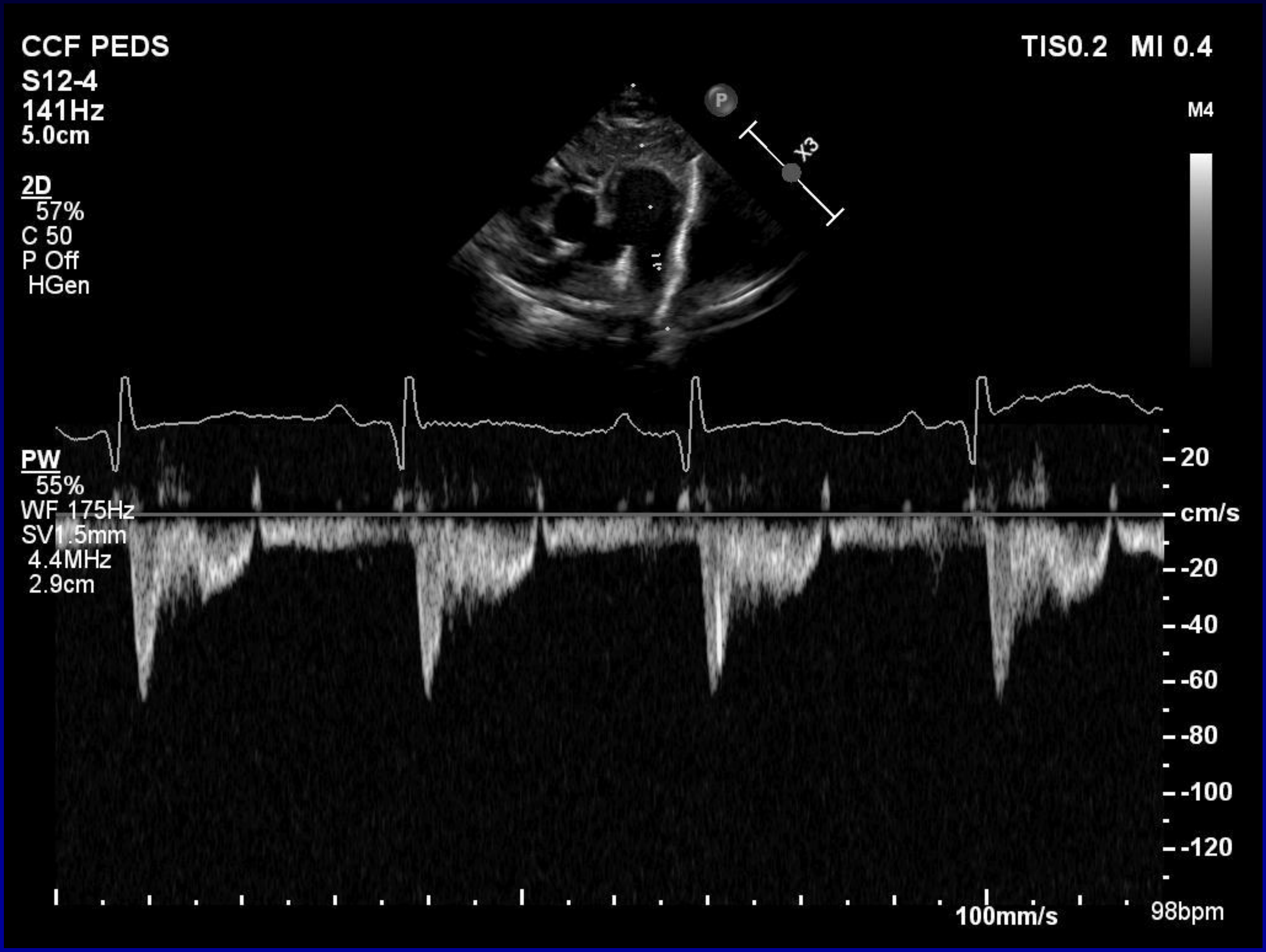

CCF PEDS
S12-4
23Hz
6.0cm
Z 1.1
2D
64%
C 50
P Off
Gen
CF
65%
8100Hz
WF 728Hz
4.5MHz
CW
50%
WF 225Hz
5.0MHz
TIS0.3 MI 0.0
X3
M4 M4
+69.3
-69.3
cm/s
cm/s
-100
-200
-300
-400
100mm/s
101bpm

RPA Doppler



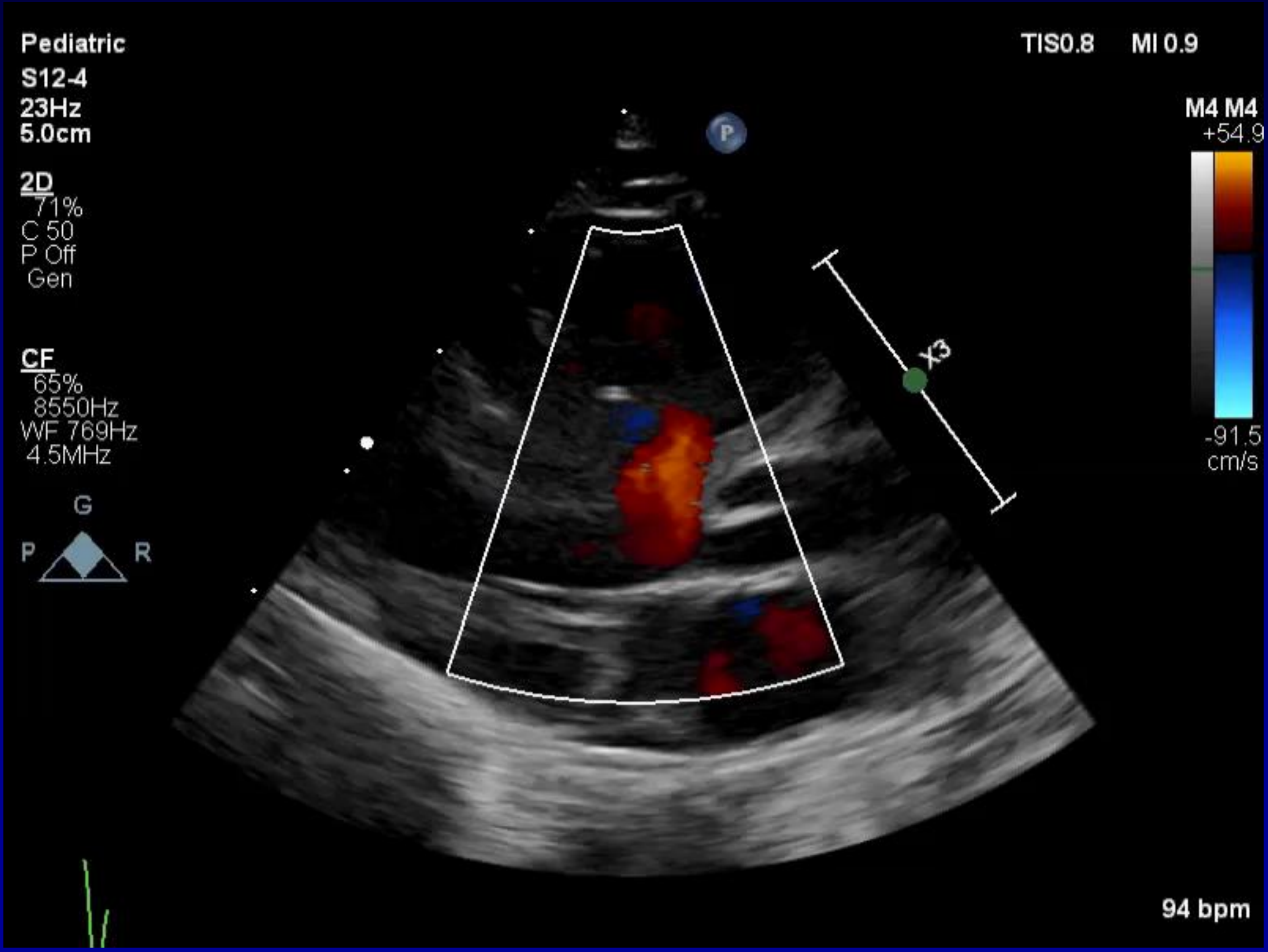
CCF PEDS
S12-4
21Hz
5.0cm
2D
56%
C 50
P Off
HGen
CF
74%
8100Hz
WF 728Hz
4.5MHz
PW
55%
WF 175Hz
SV1.5mm
4.4MHz
2.9cm
TIS0.2 MI 0.4
M4 M4
+69.3
-69.3
cm/s
X3
P
20
cm/s
-20
-40
-60
-80
-100
-120
100mm/s
98bpm

LPA Doppler



CCF PEDS
S12-4
141Hz
5.0cm
2D
57%
C 50
P Off
HGen
TIS0.2 MI 0.4
M4
X3
P
PW
55%
WF 175Hz
SV1.5mm
4.4MHz
2.9cm
20
cm/s
-20
-40
-60
-80
-100
-120
100mm/s
98bpm

VSD Color

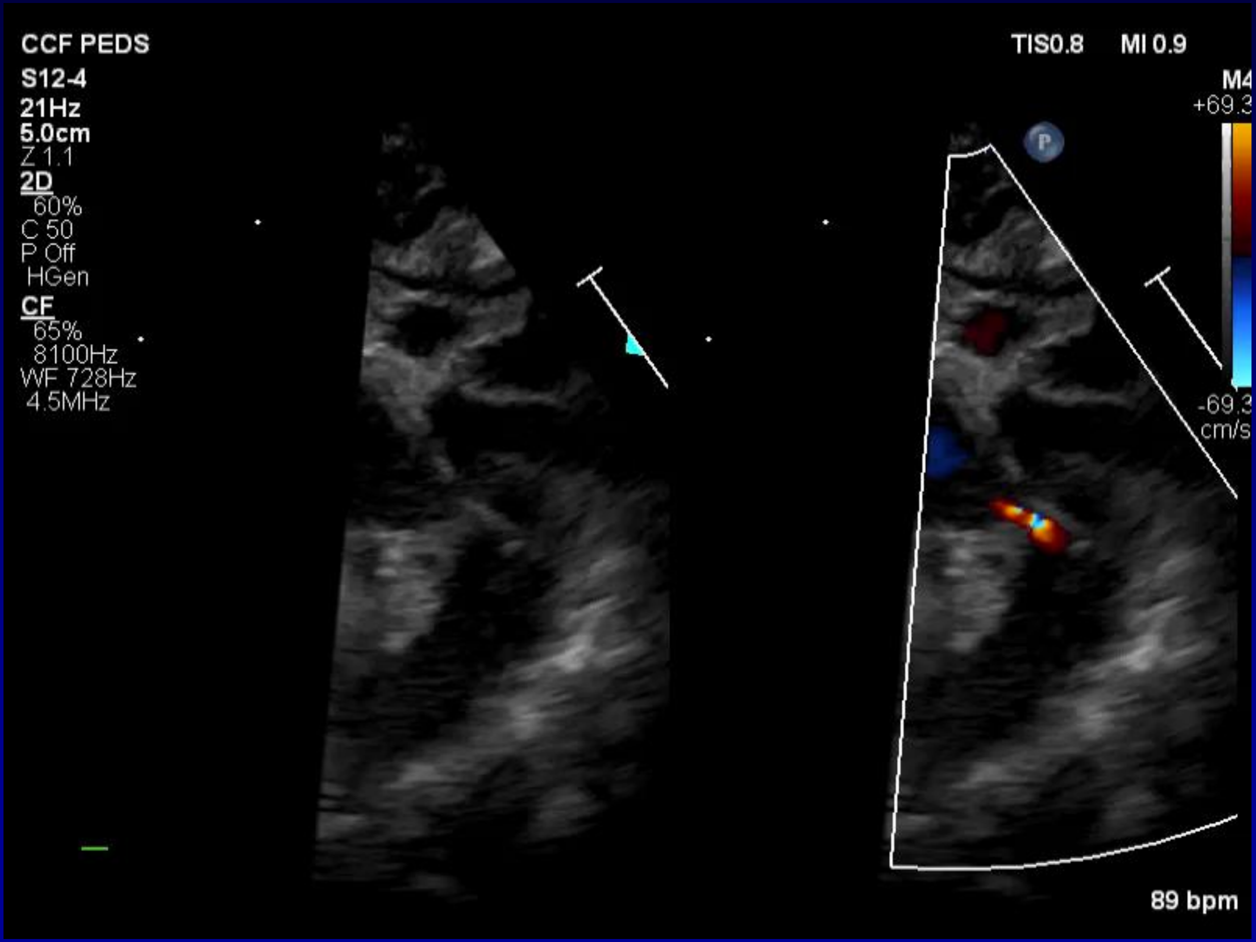


Pediatric
S12-4
23Hz
5.0cm
2D
71%
C 50
P Off
Gen
CF
65%
8550Hz
WF 769Hz
4.5MHz
G
P
R
TIS0.8
MI 0.9
M4 M4
+54.9
-91.5
cm/s
X3
94 bpm

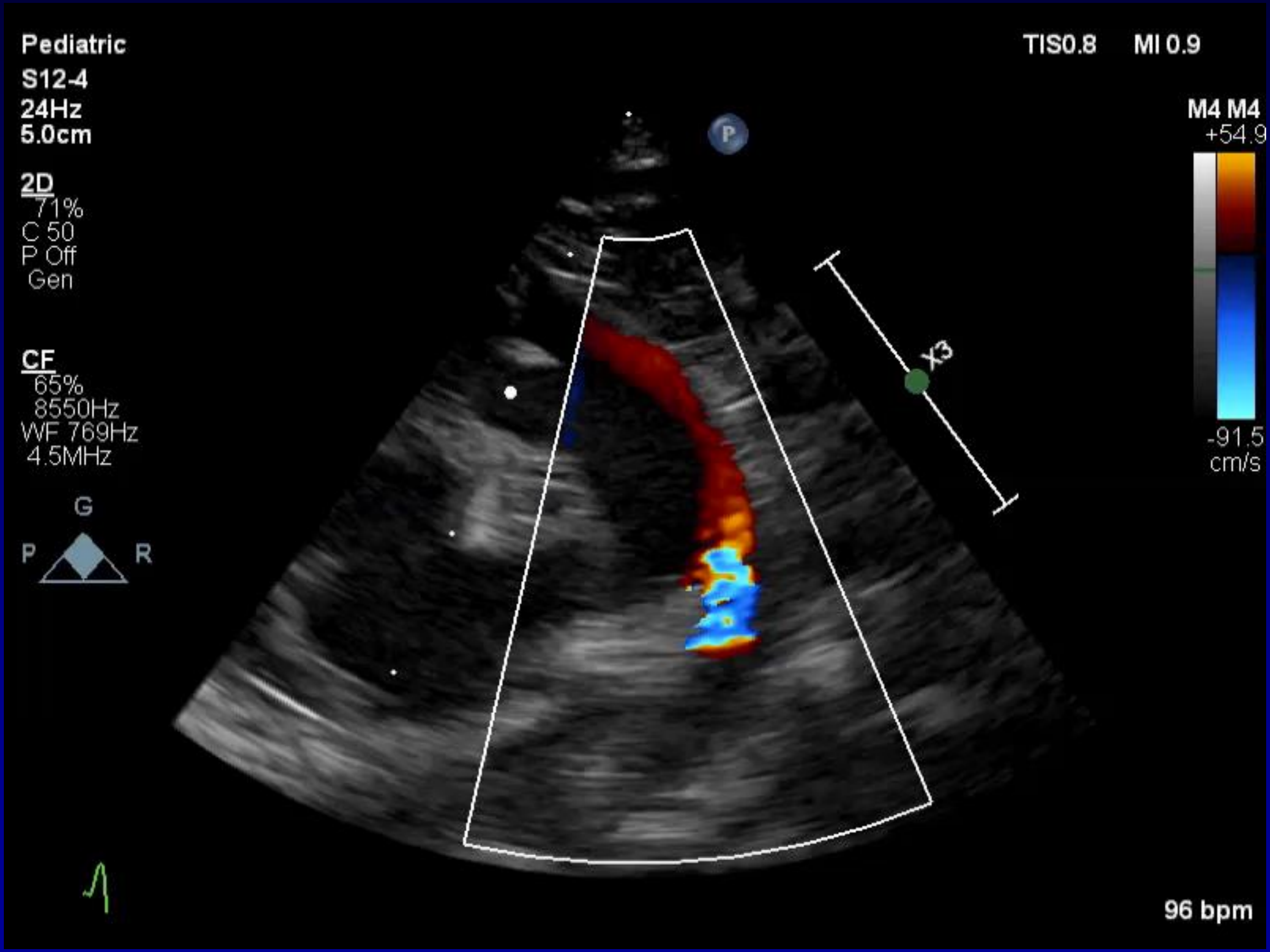
LVOT/VSD


CCF PEDS
S12-4
21Hz
6.0cm
Z 1.1
2D
58%
C 50
P Off
HGen
CF
72%
6930Hz
WF 623Hz
4.5MHz
CW
50%
WF 225Hz
5.0MHz
TIS0.2 MI 0.0
M4 M4
+59.3
-59.3
cm/s
80
40
cm/s
-40
-80
-120
100mm/s
95bpm
4 M4
+54.9
-91.5
cm/s
WF 769Hz
4.5MHz
PW
63%
WF 200Hz
SV1.5mm
4.4MHz
2.5cm
120
80
40
cm/s
-40
-80
100mm/s
94bpm

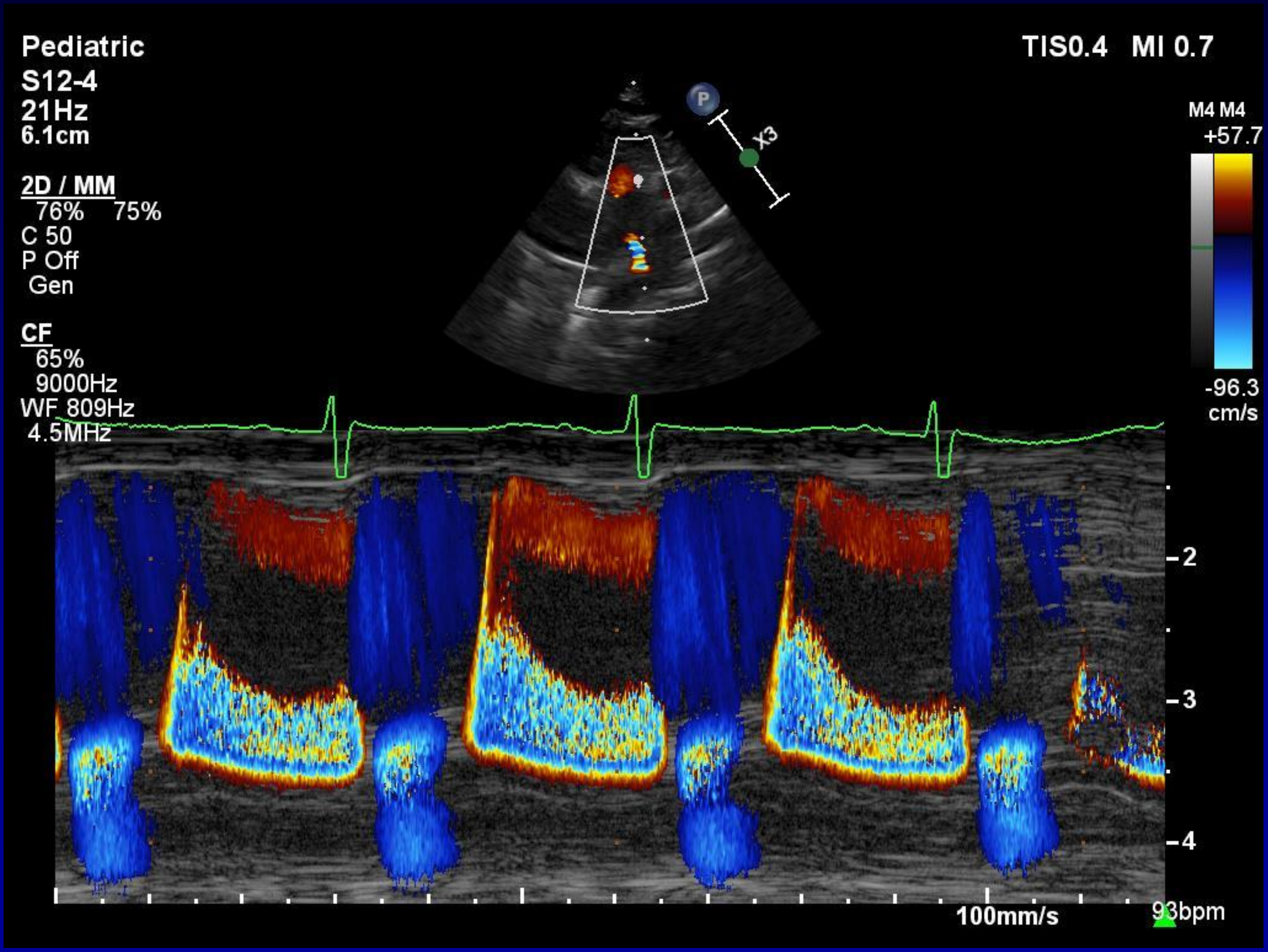
PDA


CCF PEDS
S12-4
21Hz
5.0cm
Z 1.1
2D
60%
C 50
P Off
HGen
CF
65%
8100Hz
WF 728Hz
4.5MHz
TIS0.8
MI 0.9
M4
+69.3
-69.3
cm/s
89 bpm

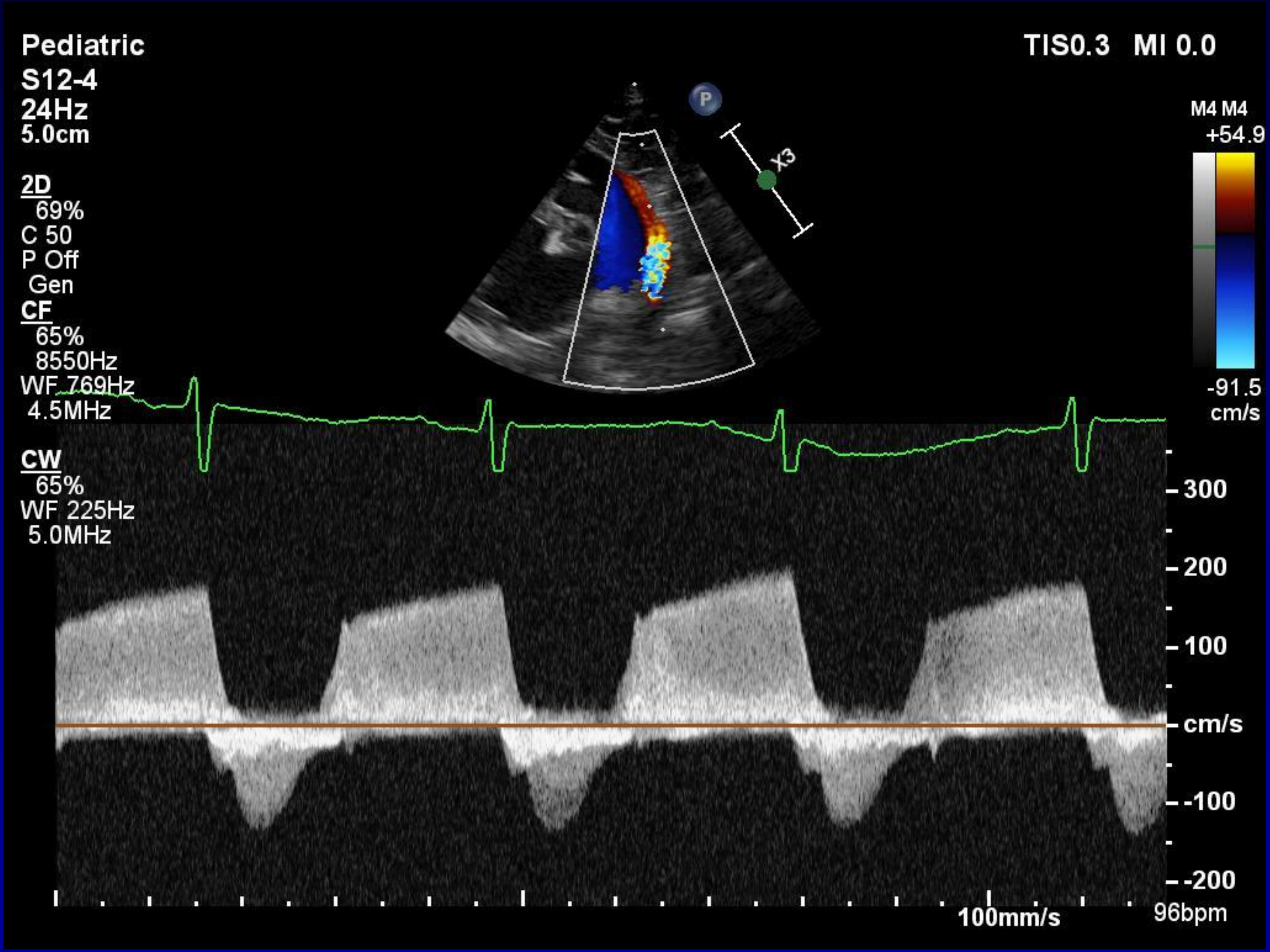
PDA Bidirectional color



PDA Color M-mode



PDA CW


Pediatric
S12-4
24Hz
5.0cm
2D
69%
C 50
P Off
Gen
CF
65%
8550Hz
WF 769Hz
4.5MHz
CW
65%
WF 225Hz
5.0MHz
TIS0.3 MI 0.0
M4 M4
+54.9
-91.5
cm/s
X3
300
200
100
cm/s
-100
-200
100mm/s
96bpm

29 year old AVC/TOF-Diastolic MR

Onset atrial systole

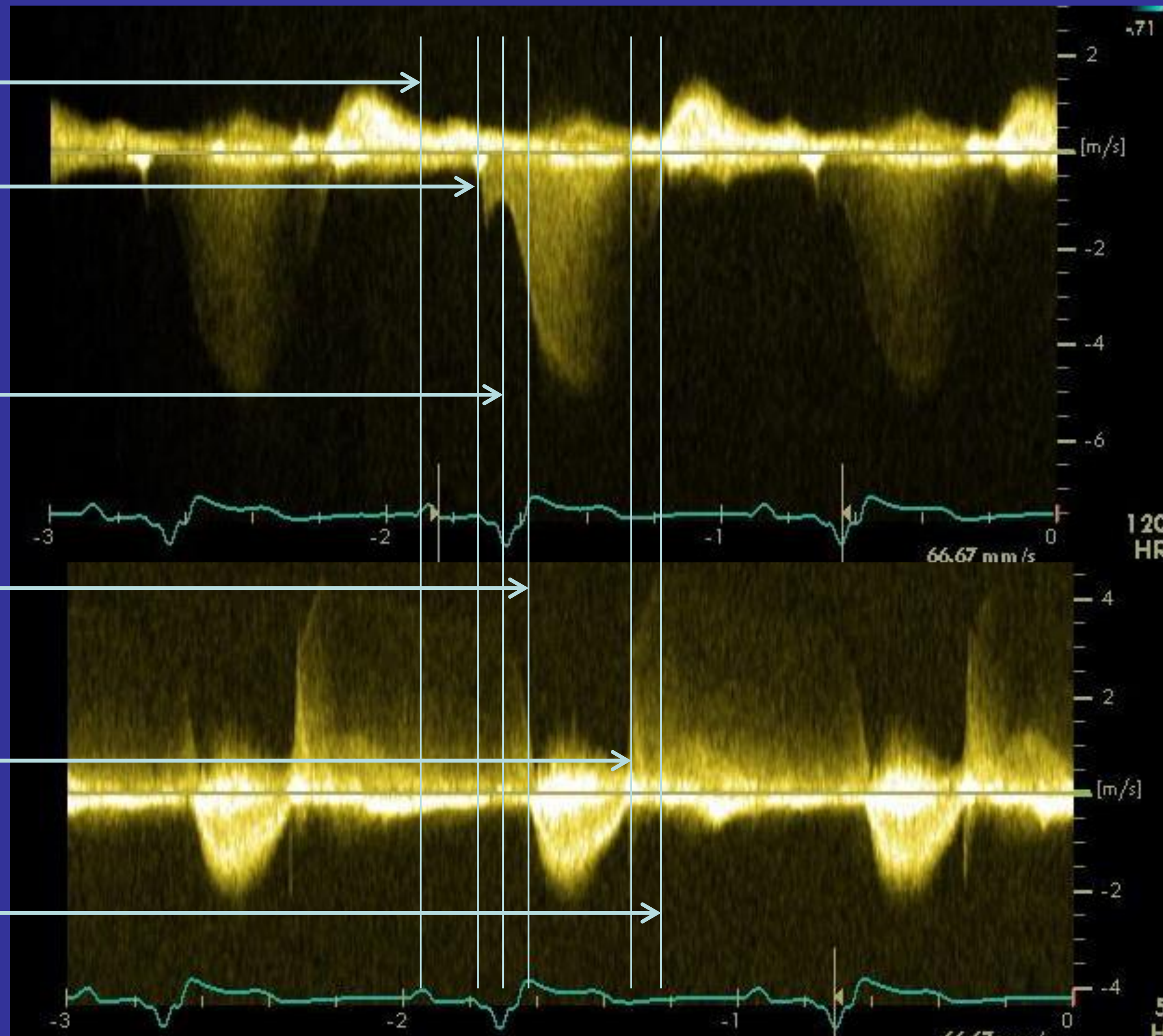
Onset diastolic mitral regurgitation

Onset systolic mitral regurgitation

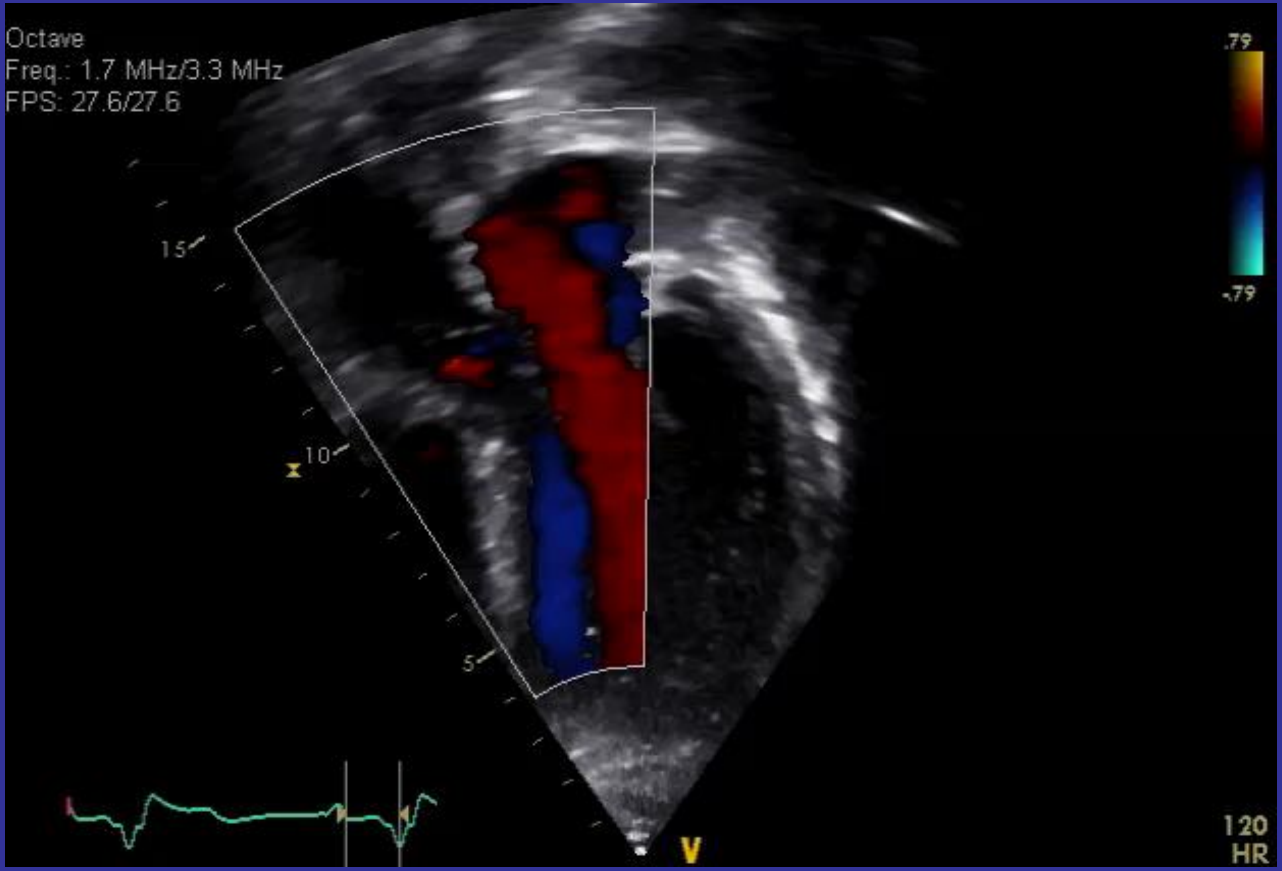
Onset systolic aortic flow- end AR

End systolic aortic flow- onset AR

End MR- onset MV inflow



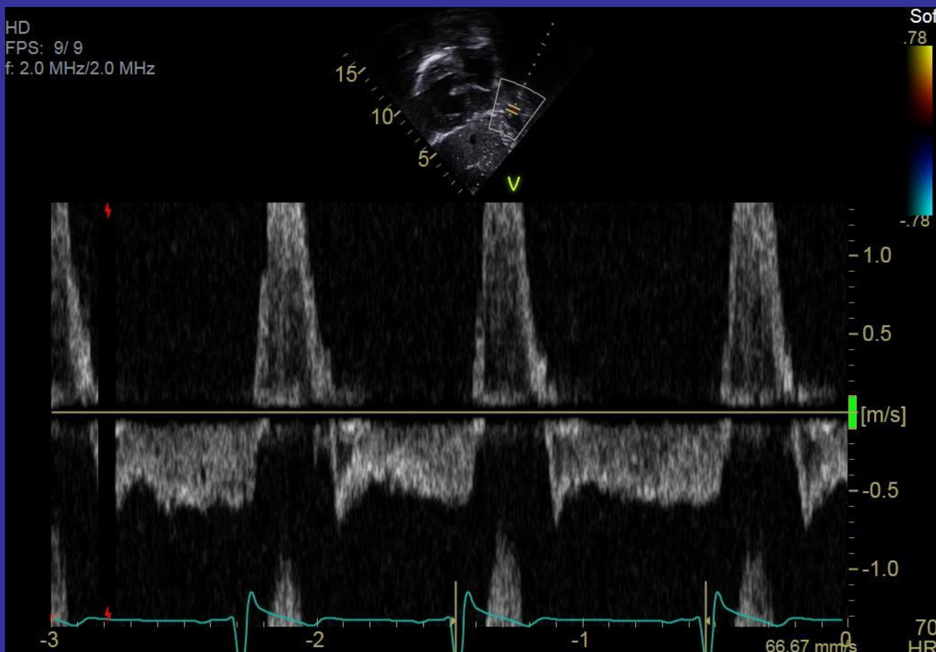
Diastolic MR occurs in the setting of a relatively long PR, relatively stiff LV with associated aortic regurgitation

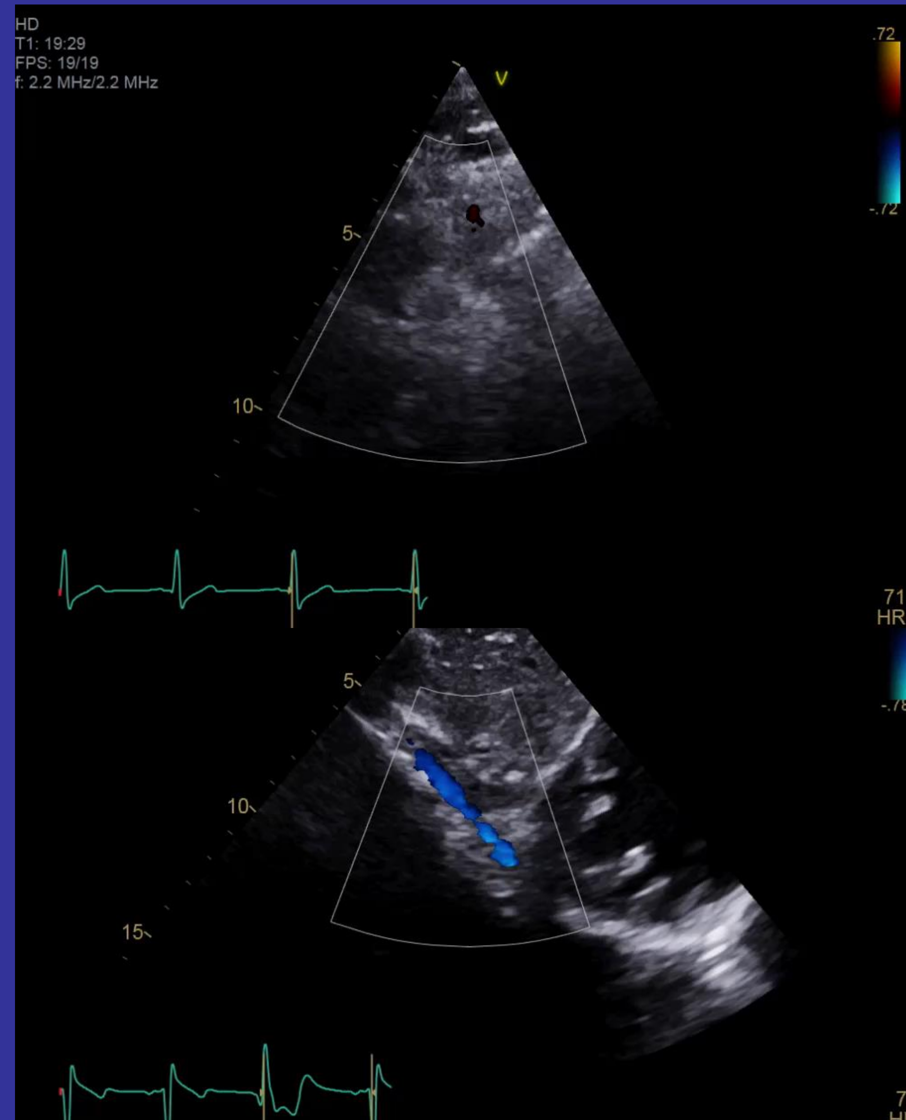


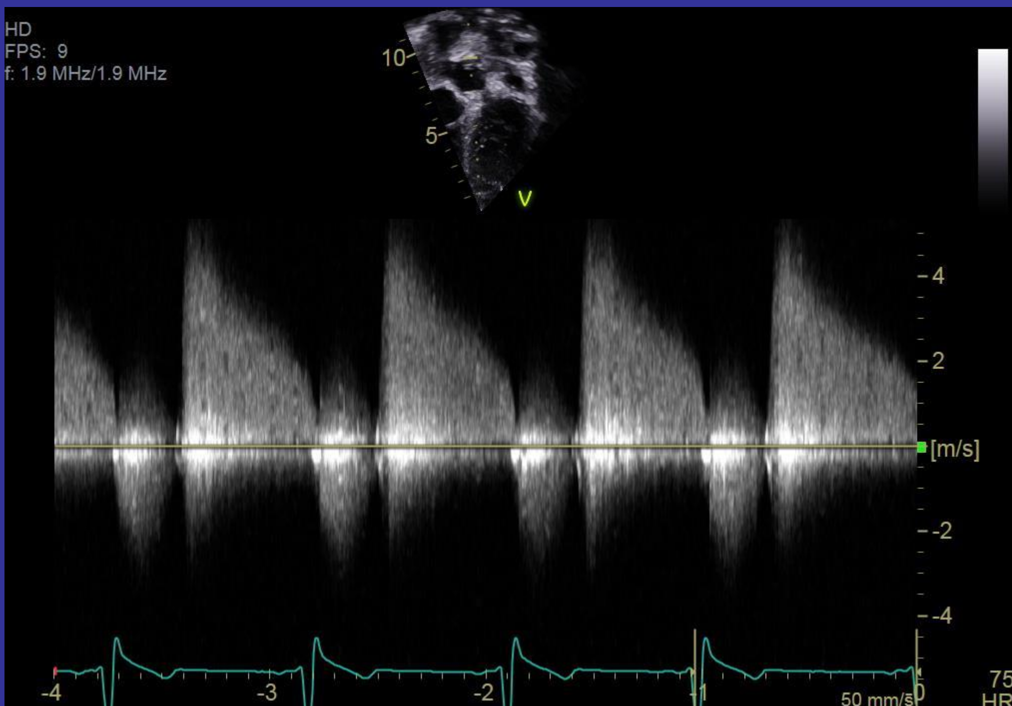
Octave
Freq.: 1.7 MHz/3.3 MHz
FPS: 27.6/27.6
.79
-.79
15
10
5
V
120
HR

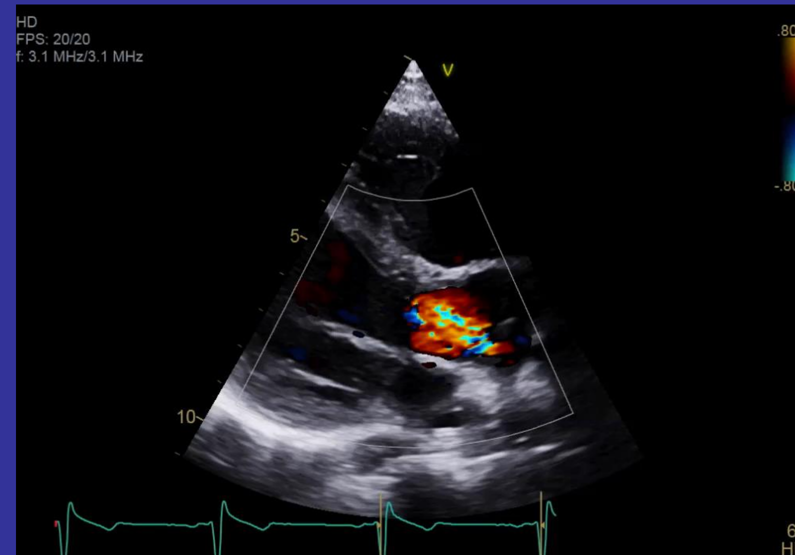
Diastolic MR

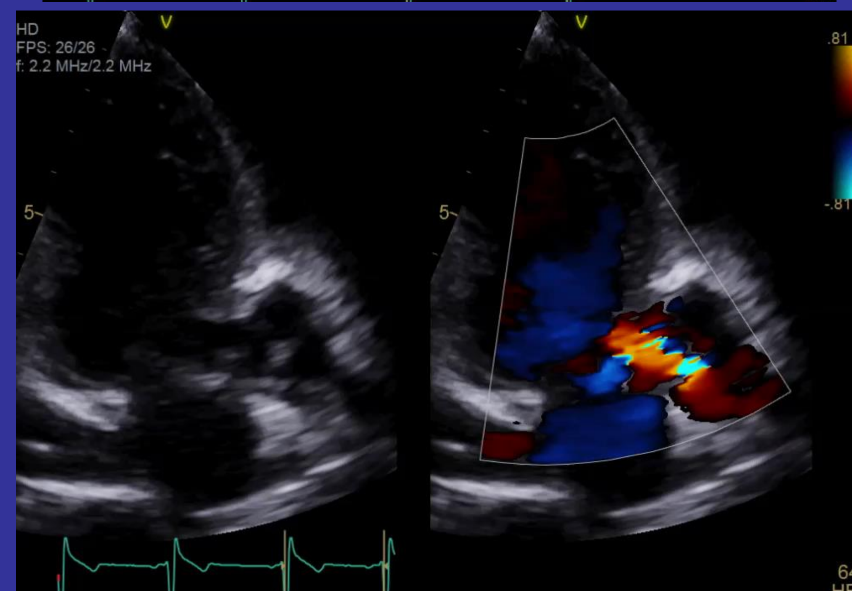
- 6-year-old with aortic atresia, aortiopulmonary window, outlet ventricular septal defect, hypoplastic ascending aorta, interrupted aortic arch status post 2 ventricle repair now with severe aortic regurgitation

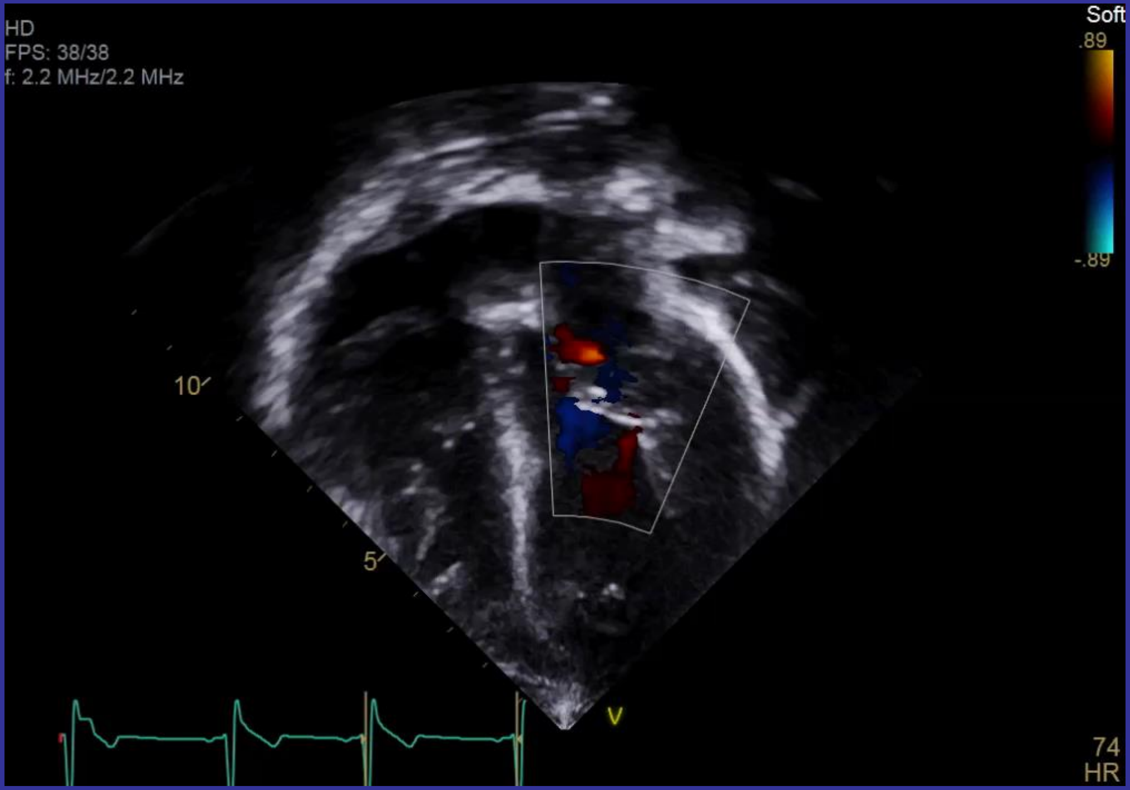

HD
FPS: 9/ 9
f: 2.0 MHz/2.0 MHz
Soft
.78
-.78
1.0
0.5
[m/s]
-0.5
-1.0
-3
-2
-1
0
66.67 mm/s
70
HR

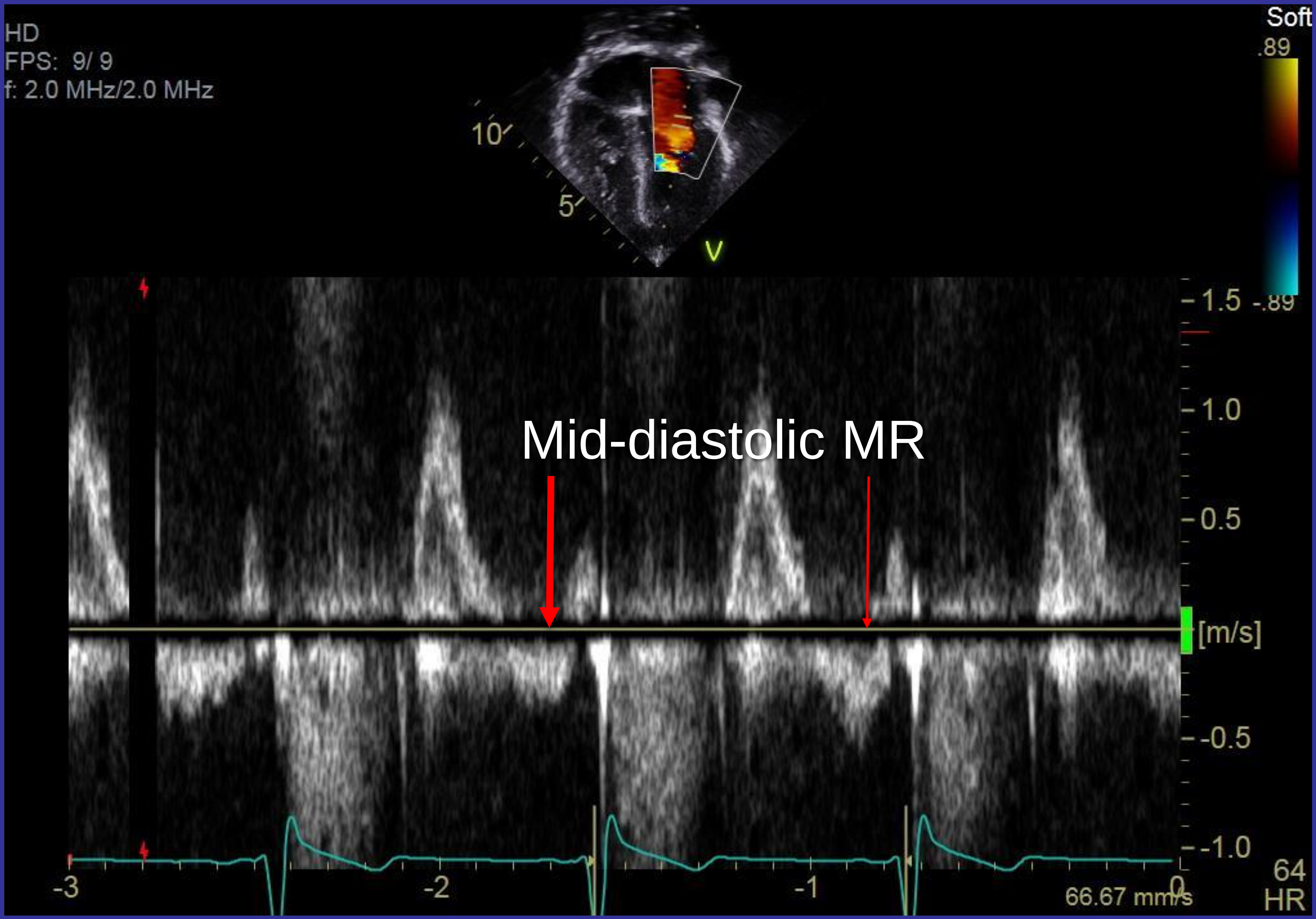

HD
T1: 19:29
FPS: 19/19
f: 2.2 MHz/2.2 MHz
.72
-.72
71
HR
-.78
70
HR


HD
FPS: 9
f: 1.9 MHz/1.9 MHz
[m/s]
50 mm/s
75
HR


HD
FPS: 20/20
f: 3.1 MHz/3.1 MHz
65
HR

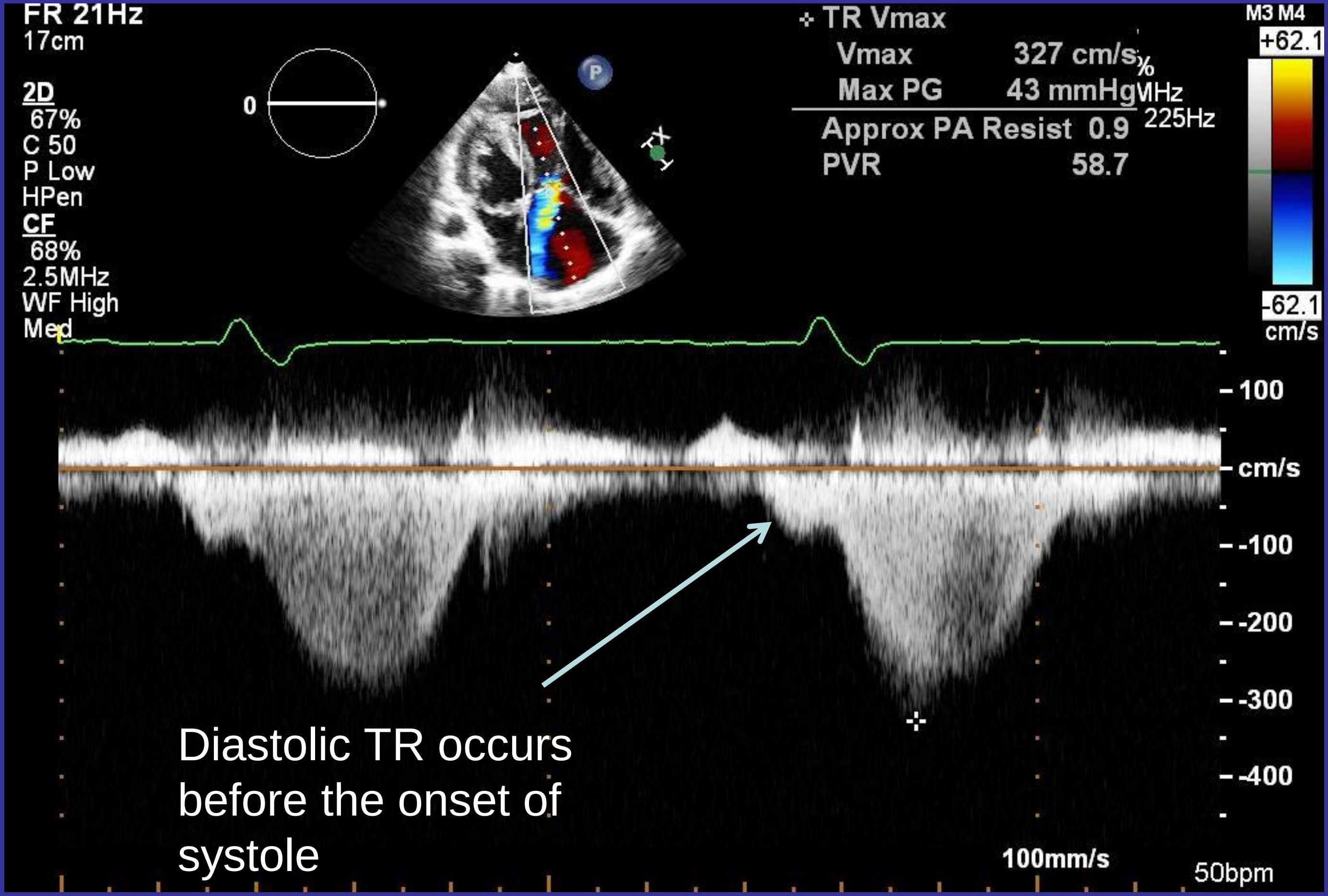

HD
FPS: 26/26
f: 2.2 MHz/2.2 MHz


HD
FPS: 38/38
f: 2.2 MHz/2.2 MHz
Soft
.89
-.89
10
5
V
74
HR

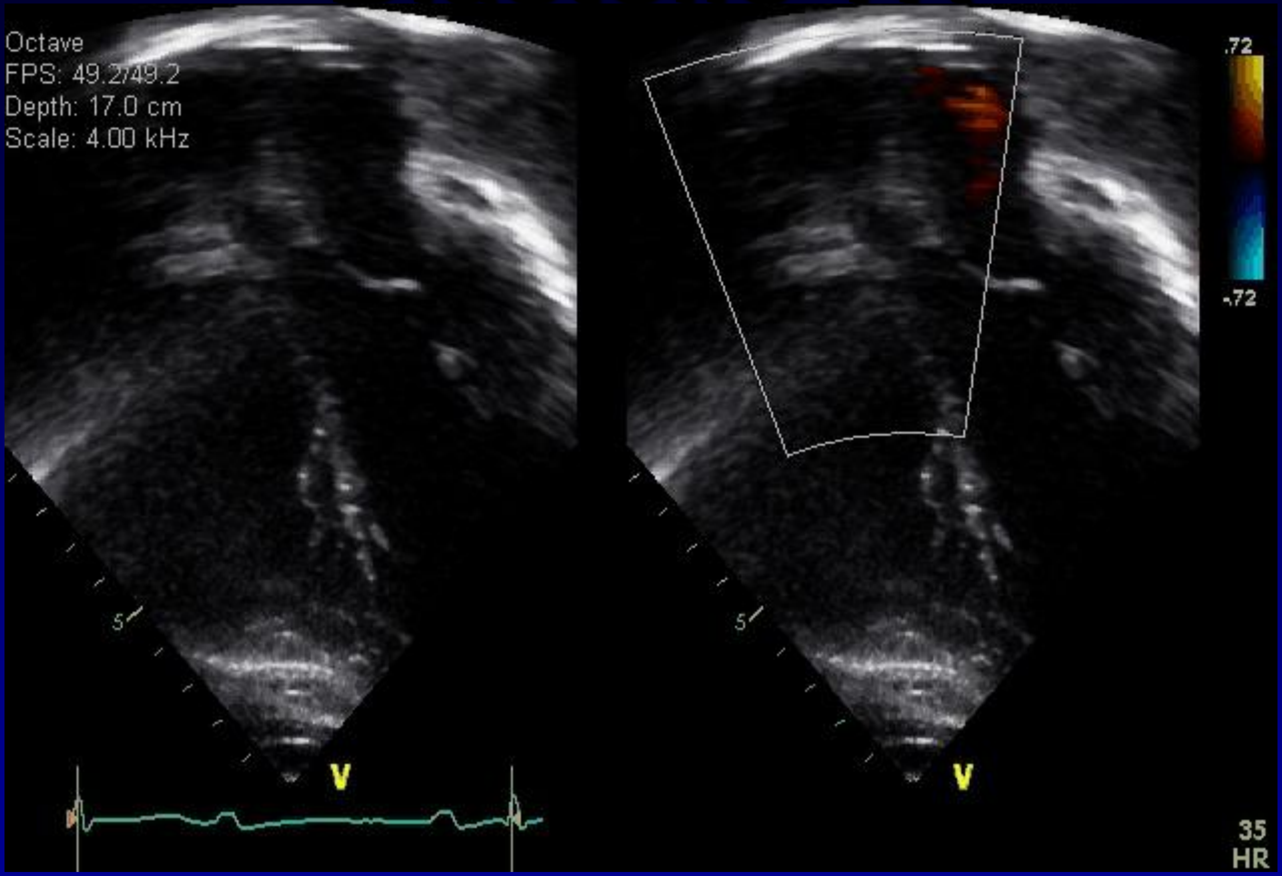


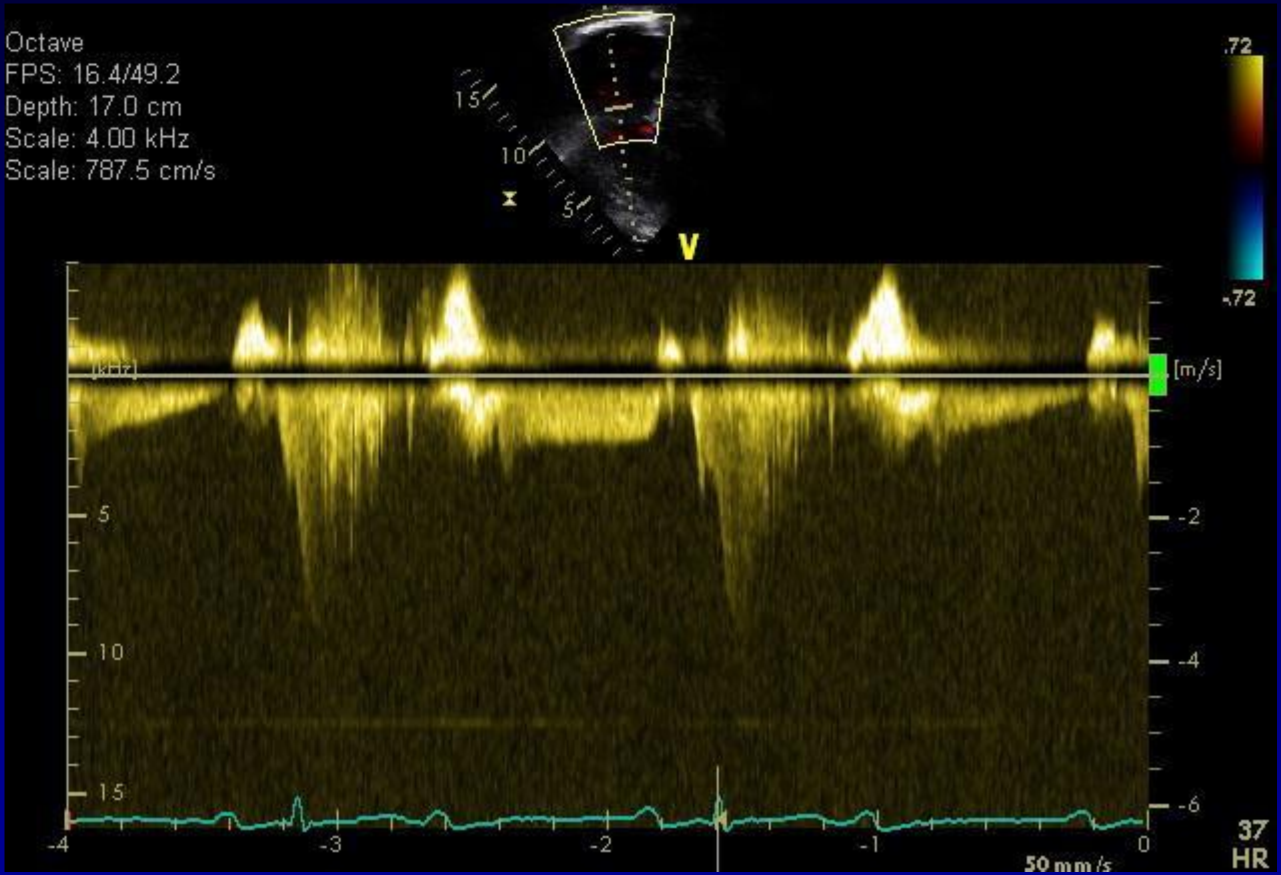
HD
FPS: 9/ 9
f: 2.0 MHz/2.0 MHz
Soft
.89
-.89
10
5
V
Mid-diastolic MR
1.5
1.0
0.5
[m/s]
-0.5
-1.0
-3
-2
-1
0
66.67 mm/s
64
HR

Diastolic TR occurs with severe PR and typically requires a slower heart rate and longer PR to be easily recognized

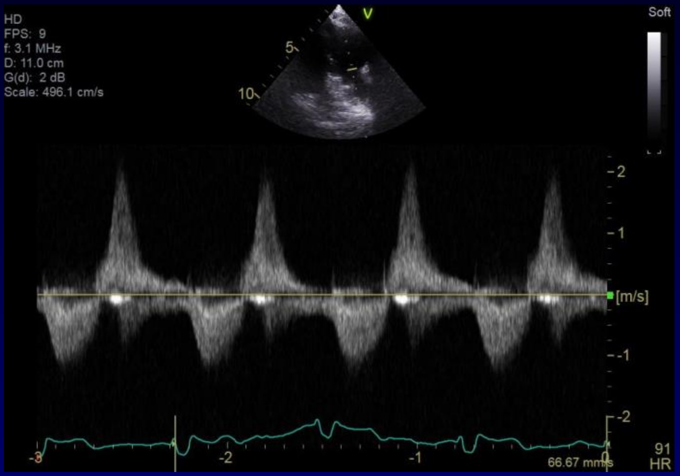


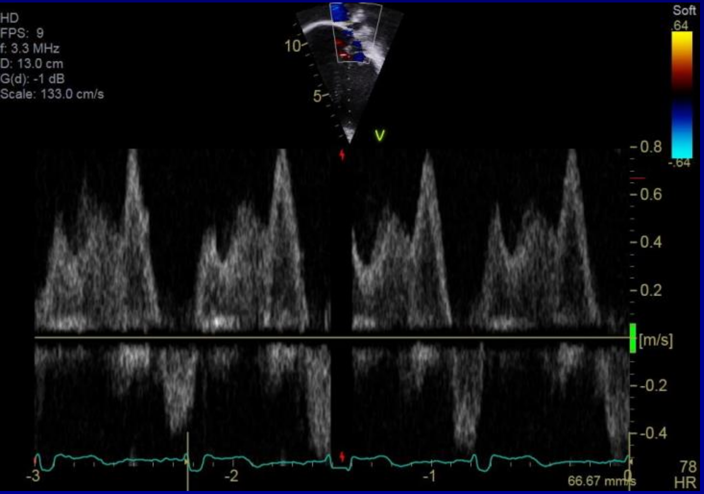
Eisenmenger VSD with CHB
Diastolic TR

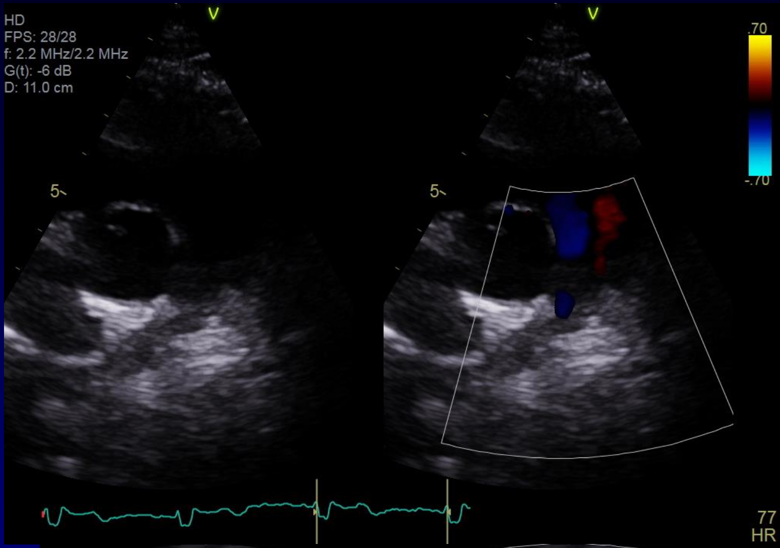


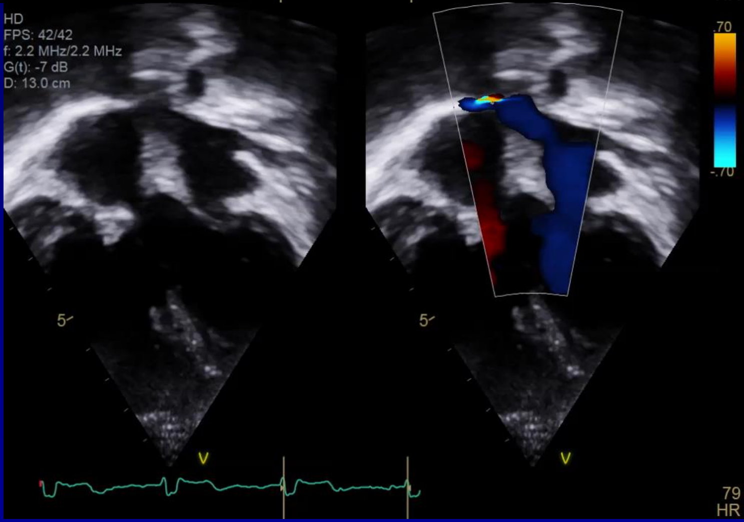

Octave
FPS: 16.4/49.2
Depth: 17.0 cm
Scale: 4.00 kHz
Scale: 787.5 cm/s
[kHz]
[m/s]
50 mm/s
37
HR

RV-LV interaction: TOF Free PR



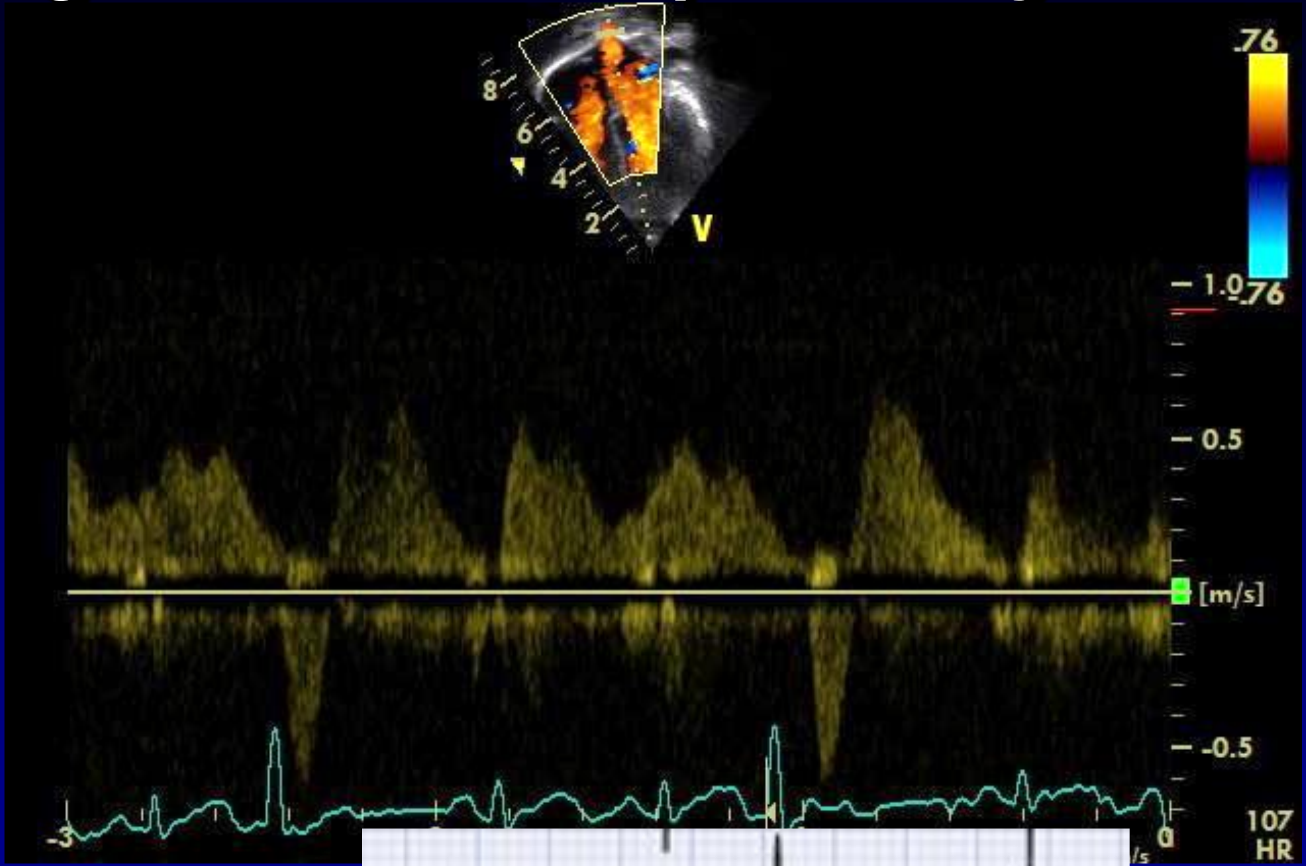






Pulmonary Vein Doppler

Structurally normal heart with Prominent Retrograde A waves in pulmonary vein Doppler



What Would I Like You to Remember?

- Blood flow is complicated but it usually plays by the rules.
- There is a wealth of information in the Doppler tracings to help understand hemodynamics if you look for it.
- The RV and LV can work in different ways
- Systole and Diastole sometimes overlap
- I love wave reflections and diastolic AV valve regurgitation
- Sonographers are indispensable team members