UCL
Institute for Liver and Digestive Health

Royal Free London NHS
NHS Foundation Trust

Use of Ultrasound in Portal Hypertension

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Portal Hypertension: definition

«**Portal hypertension** (PH) is a frequent **clinical syndrome** haemodynamically defined by an **increase in the portal pressure gradient** (difference between portal vein pressure and inferior vena cava pressure) **over** the normal limit of **5 mmHg**»

Berzigotti A, Piscaglia F. Ultraschall bei Pfortaderhochdruck… Ultraschall in Med 2011; 32: 548–571

HEPATO-RENAL SYNDROME

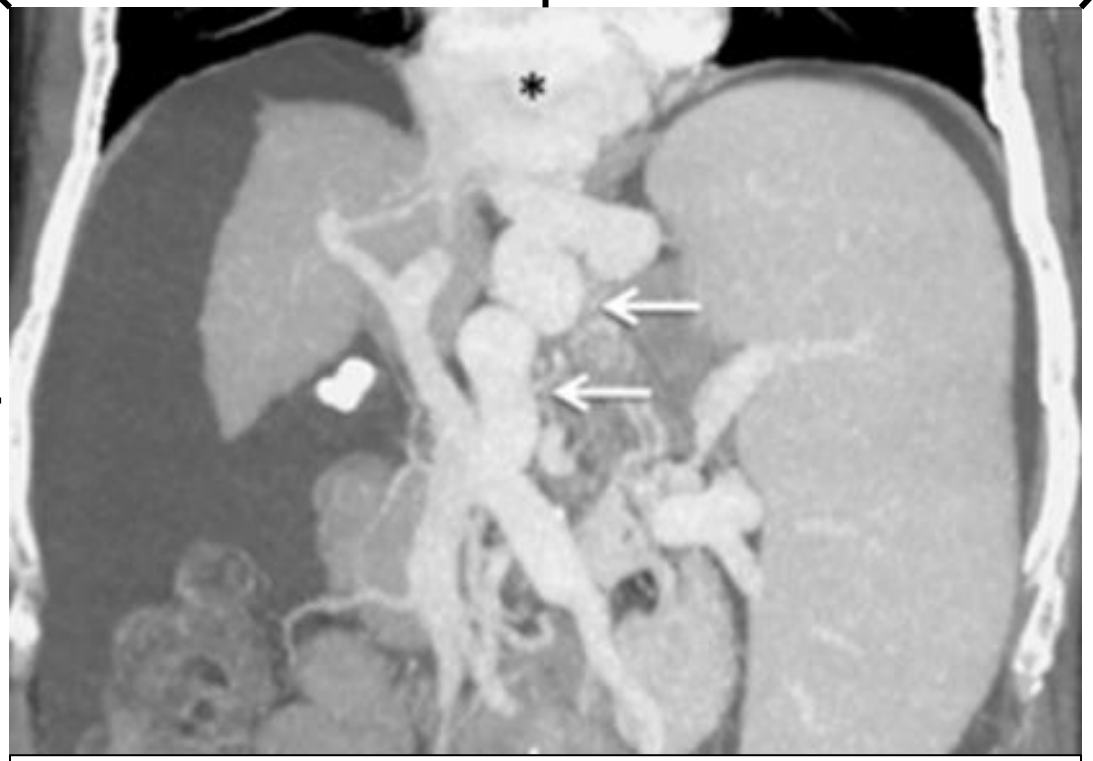
SBP

ESOPHAGEAL VARICES

RAPTURE

BLEEDING

ASCITES



CSPH is an independent predictor of clinical decompensation

OLTx

DEATH

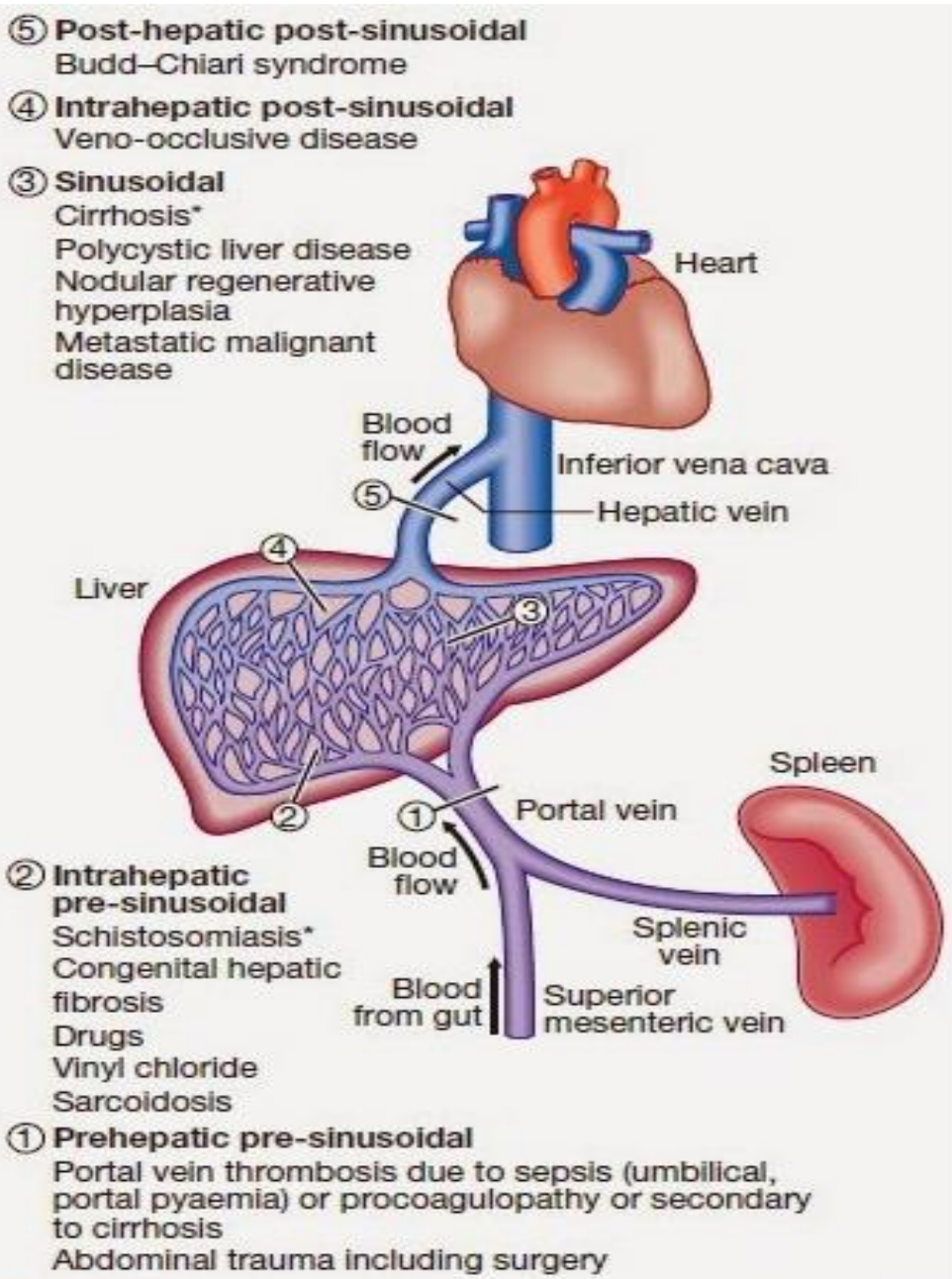


Fig. 23.19 Classification of portal hypertension according to site of vascular obstruction. *Most common cause. Note that splenic vein occlusion can also follow pancreatitis, leading to gastric varices.

1. Pre-hepatic pre-sinusoidal

2. Intra-hepatic pre-sinusoidal

3. **Intra-hepatic sinusoidal**

Cirrhosis

4. Intra-hepatic post-sinusoidal

5. Post-hepatic post-sinusoidal

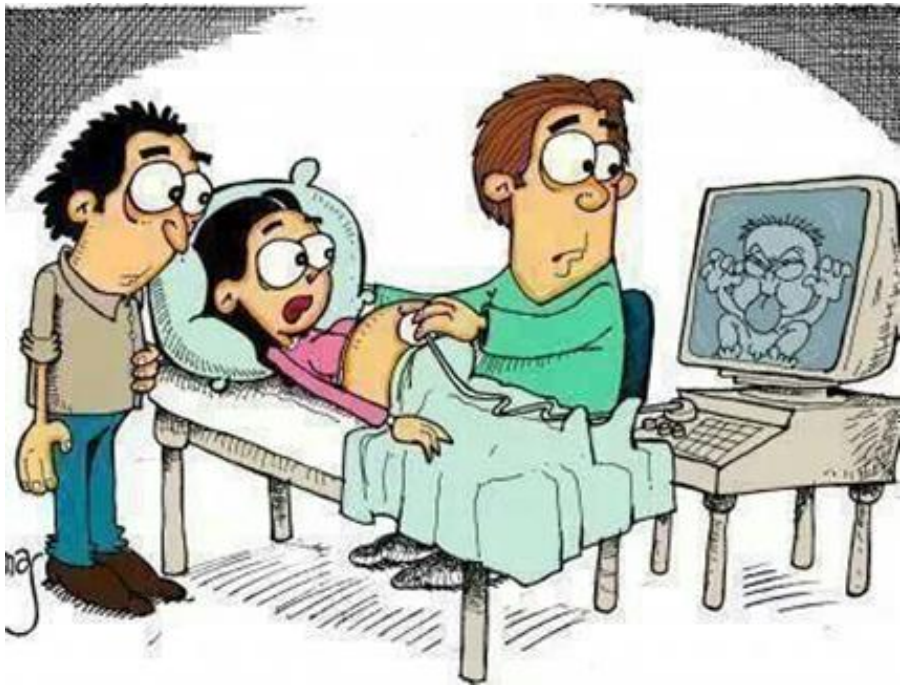
Portal Hypertension: reference standard

Normal

Sub-clinical PH

CSPH
Risk of development of varices, ascites, first clinical decompensation (also after liver resection for HCC), increased risk of HCC

Role of Ultrasound



1. First-line imaging technique used in patients with suspected PH, since it is noninvasive, repeatable and cheap.

2. US examiners should be able to detect and report correctly the most important signs of PH.

3. Most US signs of PH are independent of its underlying cause, and their interpretation should always be integrated with clinical information.

4. When patients already show overt clinical features of PH and no other data is available, US examination facilitates the classification of the mechanism which led to PH.

Instrument-based requirements

- **Convex transducers** between **3.5 and 5MHz**. Higher frequencies might be necessary in children. Moreover, **linear transducers** (**7.5–10MHz**) might be needed to properly assess the liver surface, since they significantly increase the performance of US in the detection of liver cirrhosis.

- The US equipment should be provided with **pulsed and color/power Doppler** modules to assess the patency of the vessels and to characterize the haemodynamic features of portal and splanchnic circulations.

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Examination procedure

- The ***liver, spleen and portal venous system*** should always be examined in any patient with suspected PH.

- Patient needs to be in a **fasting** state for at least 6 hours (food ingestion induces hemodynamic changes)

- Examination should be started after 5-10 minutes of a **supine position** (exercise and posture changes induce hemodynamic changes).

- Quantitative Doppler measurements should be performed in suspended **normal respiration**, avoiding deep inspiration or expiration.

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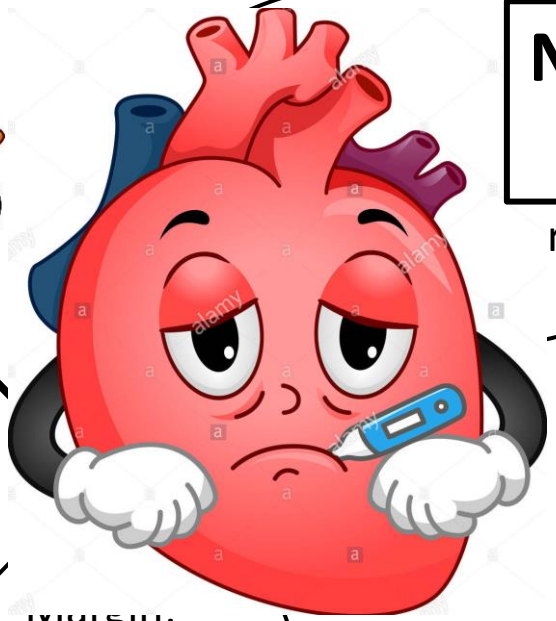
LIVER and SPLEEN assessment: S

Profile/edge/surface:
- normal
- irregular
- nodular

Caudate lobe:
- normal size
- hypertrophy

ASCITES

Echotexture:
- homogeneous
- heterogeneous
- coarsened



Margin:
- sharp
- rounded

Normal or enlarged
(size < 12 cm; area < 45 cm^2)

NEVER FORGET THE HEART!!!

rtrophy

Vascular assessment: B-mode, pulsed-Doppler, color/power Doppler

		where to measure	how to measure and normal values
portal vein (PV)	diameter	oblique-transversal scan in epigastrium/ right subcostal region to visualize PV along its longitudinal axis for at least 3 – 4 cm	– measure PV diameter as distance from inner anterior wall to inner posterior wall, perpendicular to the long portal axis, at the cross with hepatic artery or slightly downstream (but ≥ 2 cm upstream from portal bifurcation) wherever the vessel walls are best visualized. Aim: to maintain a large angle between US waves and portal walls. – the diameter is to be preferably measured with grayscale B-mode ultrasound, since CDUS, despite facilitating identification of the vessel, also implies a risk of overestimation of the diameter, related to the size of the color pixels. – measured during normal suspended respiration in the supine position (forced inspiration or left-side decubitus make measurement unreliable) – **normal** ≤ 12 mm (diameter increases according to body surface and spleen size)
	velocity		– place sample volume (≥ 50% of the diameter of PV) in the middle of the lumen at the cross with hepatic artery [8, 12] – Doppler angle preferably set at 55°, but always ≤ 60° – Doppler flowmetry, recommended PRF = 4 kHz; wall filter = 100 Hz (decrease to 50 Hz if very slow flow) – either use concurrent display of color Doppler image and Doppler flowmetry measurement if feasible (top equipment) or freeze B-mode image while displaying Doppler flowmetry tracings – manual tracing of Doppler signal for at least 2 cardiac cycles or ≥ 2 – 3 seconds; time averaged maximal velocity is calculated by the equipment in cm/s; the mean velocity can be approximated as the time averaged max velocity * 0.57. Direct measurement of the mean portal vein velocity is technically feasible but strongly influenced by Doppler setting [8], resulting in low reproducibility. Measurement of time averaged maximal velocity is recommended – **normal time averaged maximum velocity** > 20 – 24 cm/s[5]
	congestion index		calculated as PV cross-sectional area (diameter/ 2 × diameter/ 2 × π)/mean portal flow velocity **normal** < 0.075

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Vascular assessment: B-mode, pulsed-Doppler, color/power Doppler

splenic vein	diameter	transverse scan in epigastrium, to visualize SV longitudinal axis	measure SV diameter at least 1 – 2 cm upstream from the spleno-portal confluence, during suspended normal respiration in supine position diameter ≥ 10 mm is to be considered enlarged
superior mes. vein	diameter	longitudinal scan in epigastrium, to visualize SMV longitudinal axis	measure SMV diameter about 1 – 2 cm upstream from the mesenteric-portal confluence, during suspended normal respiration in supine position diameter ≥ 10 mm is to be considered enlarged
porto-collateral circulation	presence or absence	at least the following vessels should be actively looked for by US and color Doppler US: paraumbilical vein: falciform ligament left gastric vein: epigastric region posterior to left hepatic lobe. Also check the flow direction. short gastric veins: left hypochondrium posterior to the upper pole of the spleen spleno-renal circulation: left hypochondrium between the lower half of the spleen and the left kidney	
hepatic veins	diameter and patency	right subcostal or right intercostal scan (the latter especially for Doppler flow tracing measurement) allowing main axis visualization sampling at 1–3 cm from IVC	normal diameter ≤ 1 cm
	phasicity of flow		the sample volume should be about the same as the diameter of the vein; quantitative information (flow velocity) is restricted to selected cases (stenosis) **normal** triphasic flow. Flow tracings to be assessed during suspended normal respiration (forced inspiration may flatten the tracing. However, if regularly triphasic during forced inspiration, a normal tracing is still ascertained). Assessment best in supine position, but also acceptable in left decubitus.
inferior vena cava	diameter and patency	transverse/longitudinal scans from the thoraco-abdominal region (intercostal and subcostal)	**normal** tri-quadriphasic flow; tend to collapse in expiration; caliber < 2 cm.

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hepatic artery	intraparenchymal Doppler impedance indexes	main lobar branches in the right and left lobe	color Doppler helps in finding the site of measurement, adjacent to the lobar branches of the portal vein. Right branch usually best visualized through an intercostal scan at its entrance to the liver, left branch through an epigastric scan, either during suspended normal respiration or during forced inspiration (to be maintained no longer than approximately 10 seconds, otherwise hypoxia induces vasodilatation). Increase PRF to improve Doppler tracings, aiming at having a trace occupying approximately ¾ of the screen height. At least 2 identical consecutive complete arterial tracings are required (at best ≥3) to confirm that no change in the pulsed Doppler insonation angle occurred during the recording of tracings in any cardiac cycle. **Normal:** RI < 0.65–0.70; PI < 1.20
splenic artery	intraparenchymal Doppler Impedance indexes	main branches 1 cm after entering the parenchyma	color Doppler helps in finding the site of measurement, usually parallel to the intrasplenic veins. Adjust PRF to improve Doppler tracings, aiming at having a trace occupying approximately between ½ and ¾ of the screen height, after having lowered the zero Doppler line. Angle of insonation preferable between 20° and 60°. Measurements through a left intercostal space in the supine position during either suspended normal respiration or forced inspiration (to be kept no longer than approximately 10 seconds, otherwise hypoxia induces vasodilation). Sample volume usually 2–4 mm, often larger than arterial diameter. At least 2 identical complete arterial tracings are required (at best ≥3) to confirm that no change in pulsed Doppler insonation angle occurred during the tracing of any cardiac cycle. **normal:** RI < 0.63; PI < 1.00
superior mesenteric artery (SMA)	diameter and Doppler impedance indexes	longitudinal scan in epigastrium, to visualize SMA longitudinal axis	site of assessment: 3–5 cm distal to the origin, ideally shortly after the initial curve, where the course is straight. Sample volume set as large as the artery. Adjust PRF to improve Doppler tracings, aiming at having a trace occupying approximately between ½ and ¾ of the screen height. **normal in fasting state:** RI > 0.84; PI > 3.20. Diameter ≤6 mm. At least 2 identical consecutive complete arterial tracings are required (at best ≥3) to confirm that no change in pulsed Doppler insonation angle occurred during the tracing of any cardiac cycle.
renal arteries	intraparenchymal Doppler impedance indexes	interlobar (or interlobular) arteries	visualize the kidney as superficially as possible (usually through a rather posterior approach). Preliminary CDUS is strongly recommended to visualize the arterial tree. Keep CDUS PRF low (700–800 Hz or few cm/sec in equipments reporting PRF as velocity assuming a 0° angle). Measurements taken either during suspended normal respiration or forced inspiration (to be kept no longer than approximately 10 seconds, otherwise hypoxia induces vasodilation). Sample volume usually 2–4 mm, larger than artery diameter. Adjust PRF to improve Doppler tracings, aiming at having a trace occupying approximately ¾ of the screen height, after having lowered the zero Doppler line. At least 2 identical consecutive complete arterial tracings are required (at best ≥3) to confirm that no change in pulsed Doppler insonation angle occurred during the tracing of any cardiac cycle. **normal:** RI < 0.70 (in adult patients); PI < 1.15–1.20

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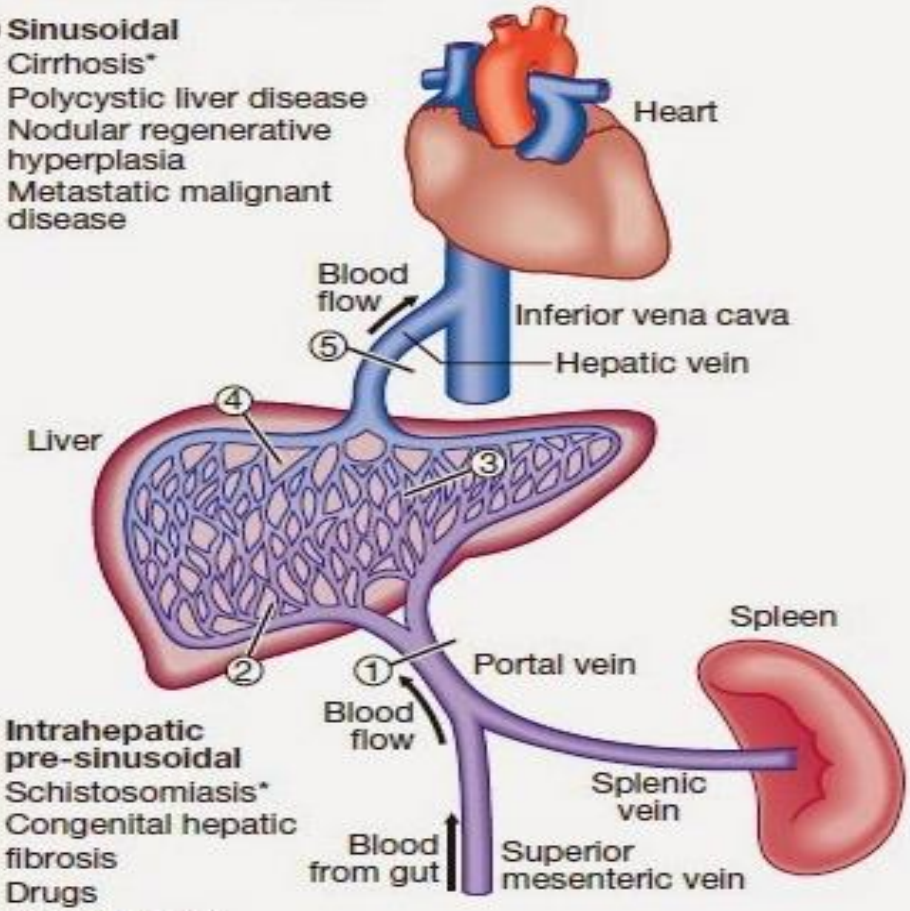
⑤ **Post-hepatic post-sinusoidal**
Budd–Chiari syndrome

④ **Intrahepatic post-sinusoidal**
Veno-occlusive disease

③ **Sinusoidal**
Cirrhosis*
Polycystic liver disease
Nodular regenerative hyperplasia
Metastatic malignant disease



② **Intrahepatic pre-sinusoidal**
Schistosomiasis*
Congenital hepatic fibrosis
Drugs
Vinyl chloride
Sarcoidosis

① **Prehepatic pre-sinusoidal**
Portal vein thrombosis due to sepsis (umbilical, portal pyaemia) or procoagulopathy or secondary to cirrhosis
Abdominal trauma including surgery

Fig. 23.19 Classification of portal hypertension according to site of vascular obstruction. *Most common cause. Note that splenic vein occlusion can also follow pancreatitis, leading to gastric varices.

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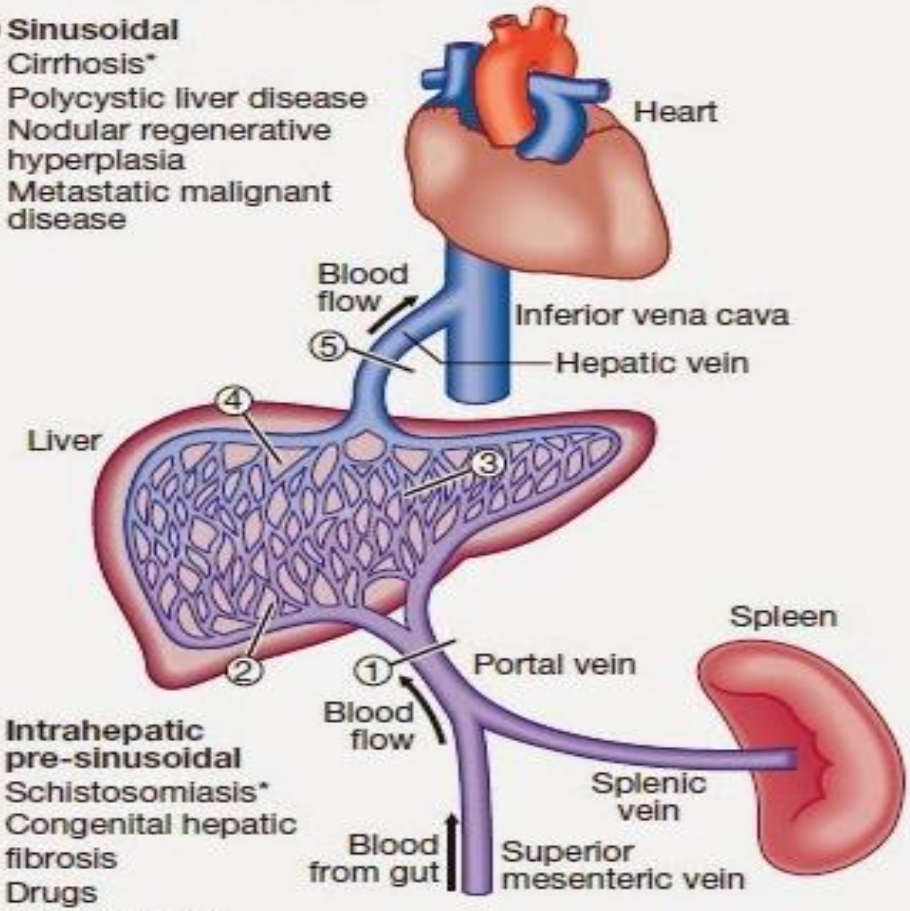

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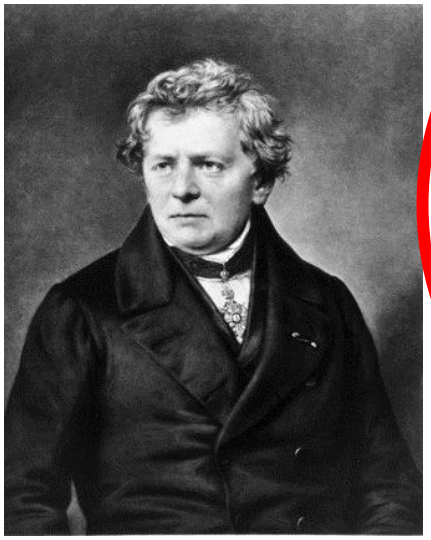
Cirrhotic Portal Hypertension: pathogenesis

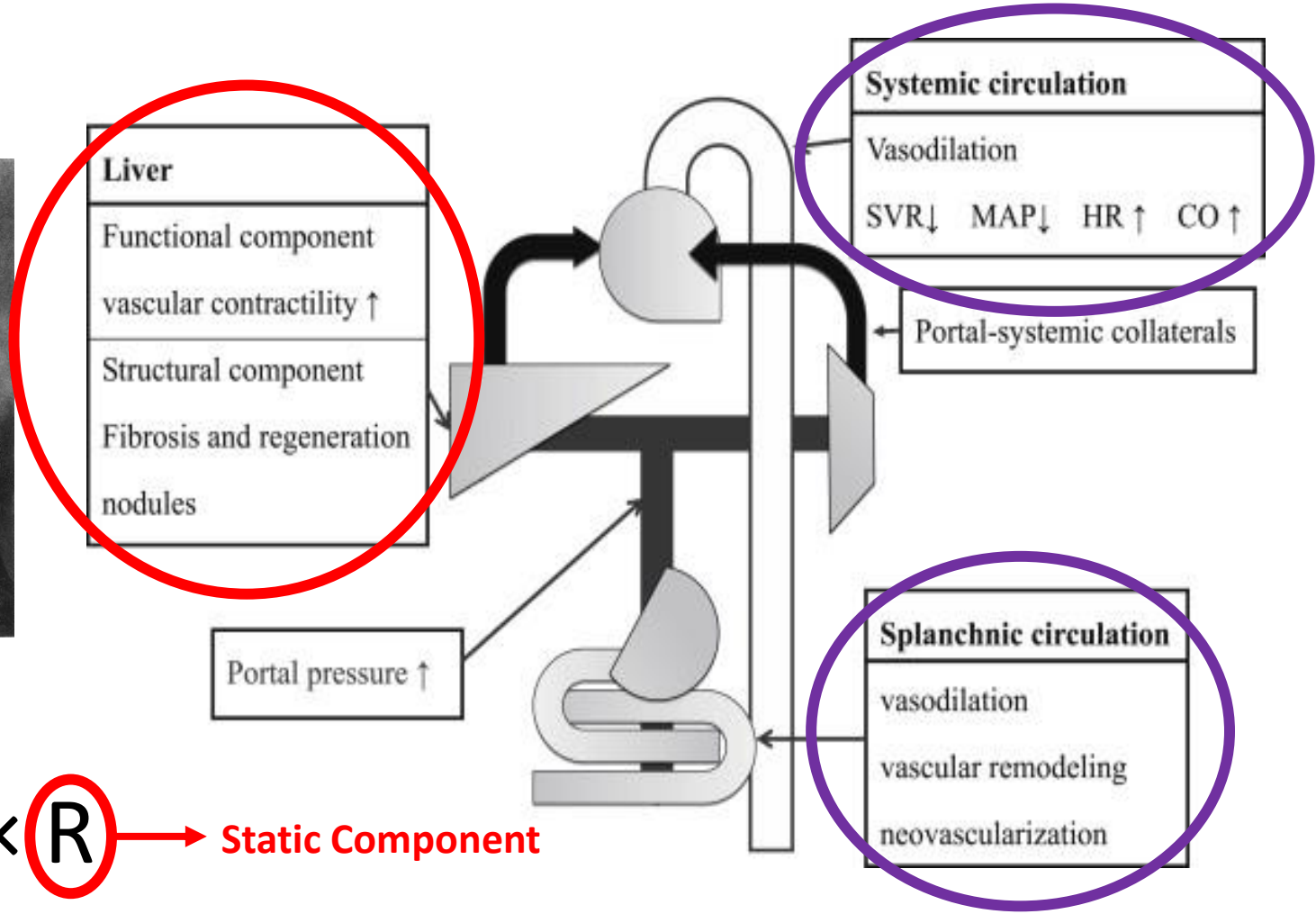


OHM'S LAW

$$\Delta P = Q \times R$$

Dynamic Component

Static Component



LIVER CIRRHOSIS

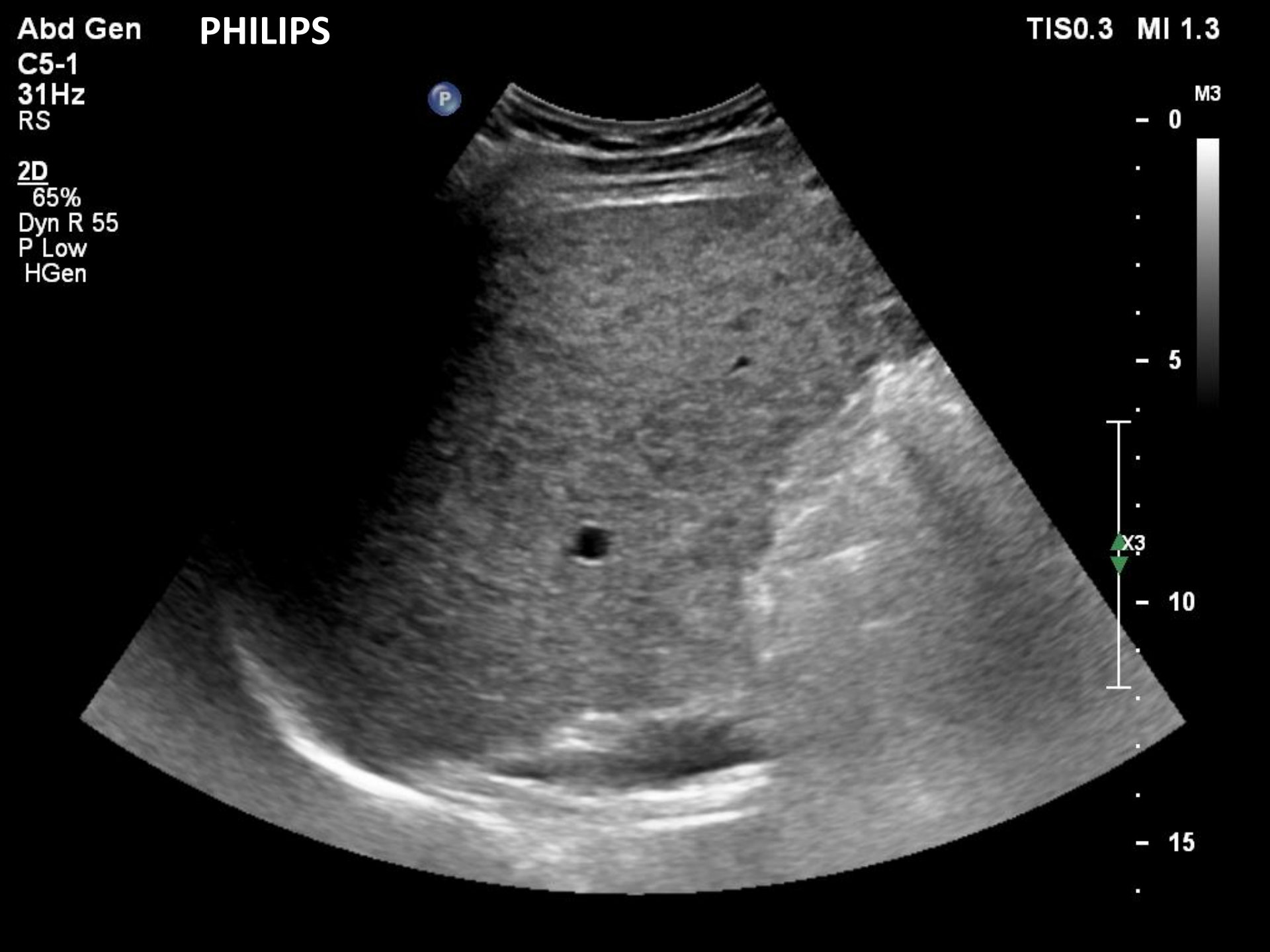
Eco-color Doppler:

- **Portal Vein**:
  - ✓Flow velocity (TAPV) < 20-24 cm/sec
  - ✓Hepatofugal flow
- **Hepatic Artery:**
  - ✓RI > 0,70
- **Hepatic Veins:**
  - ✓Monophasic/biphasic Doppler waveform
- **Other vessels:**
  - ✓Umbilical vein recanalization
  - ✓Collateral vessels/varices

LIVER CIRRHOSIS

B-mode US:

- Irregular/nodular liver profile
- Caudate hypertrophy/right hypotrophy/relative left hypertrophy
- Heterogeneous/coarse echo-texture
- Rounded margin
- PV calibre > 13 mm
- No changes of PV calibre with respiration
- Absence of respiratory phasicity
- Enlarged spleen (size > 13mm, area > 45 cm^2)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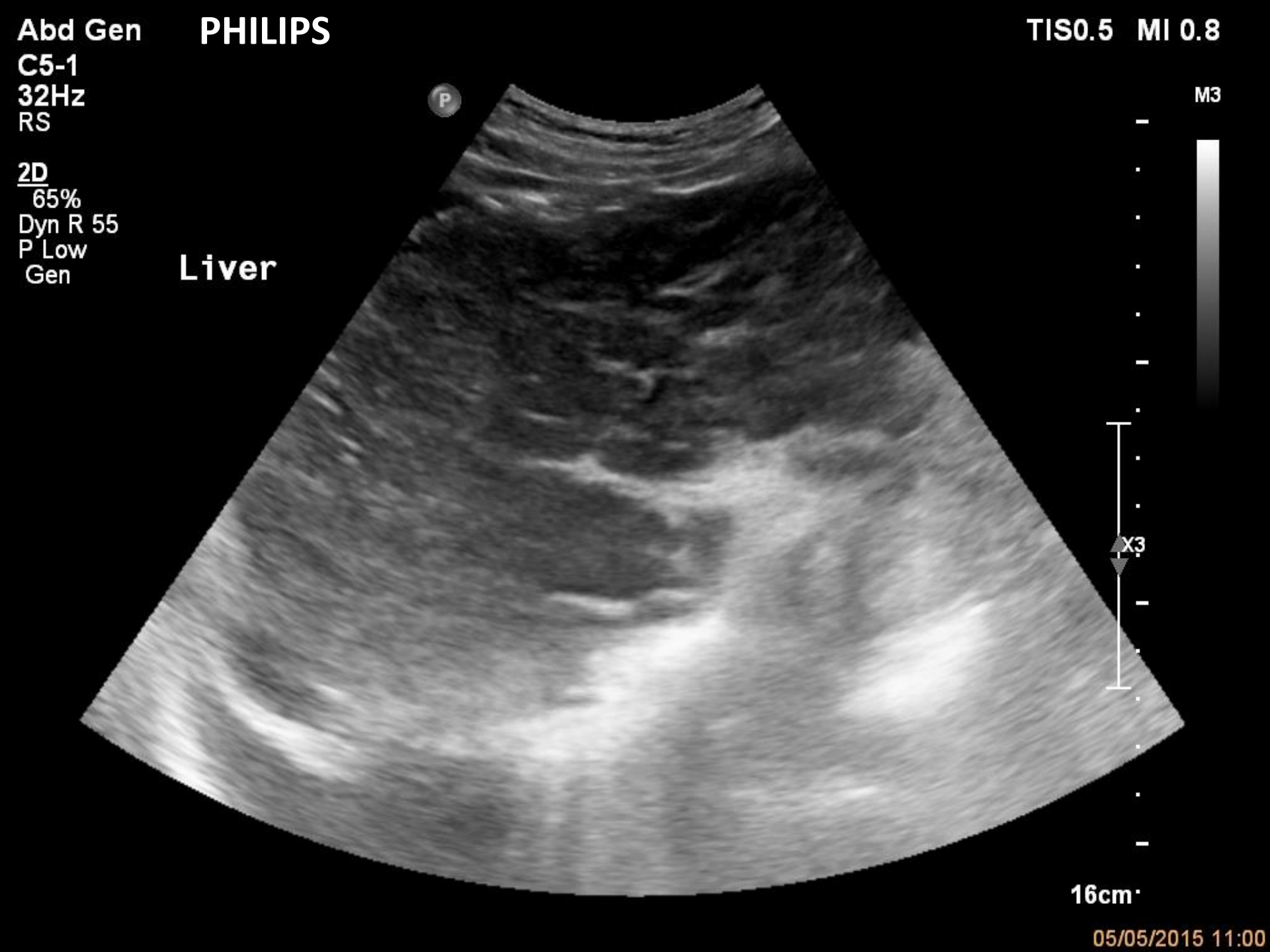
C5-1
31Hz
RS
2D
65%
Dyn R 55
P Low
HGen
PHILIPS
TIS0.3 MI 1.3
M3
0
5
10
15
X3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C5-1
32Hz
RS
2D
65%
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P Low
Gen
PHILIPS
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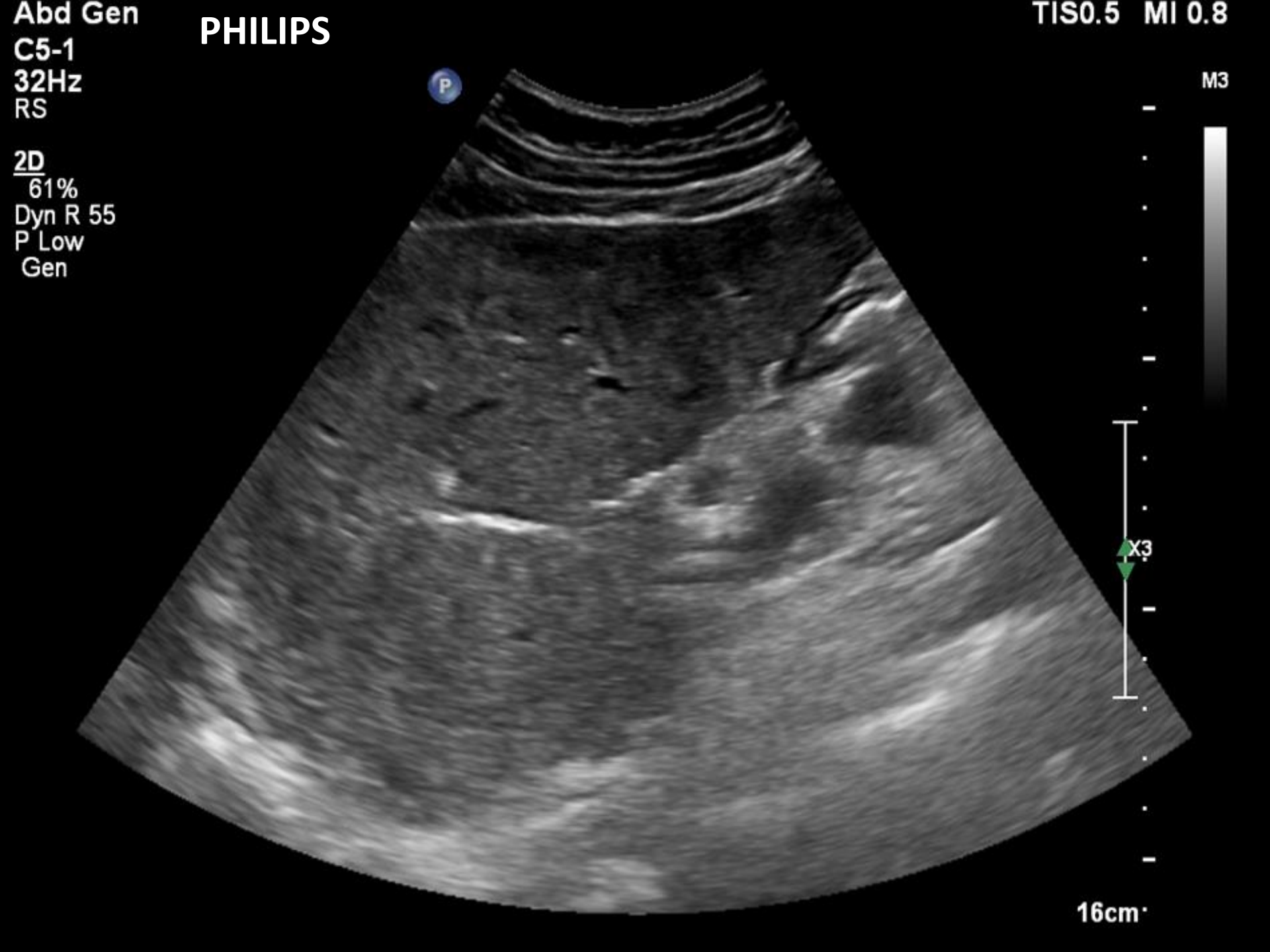
M3
Liver
X3
16cm
05/05/2015 11:00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32Hz
RS
2D
61%
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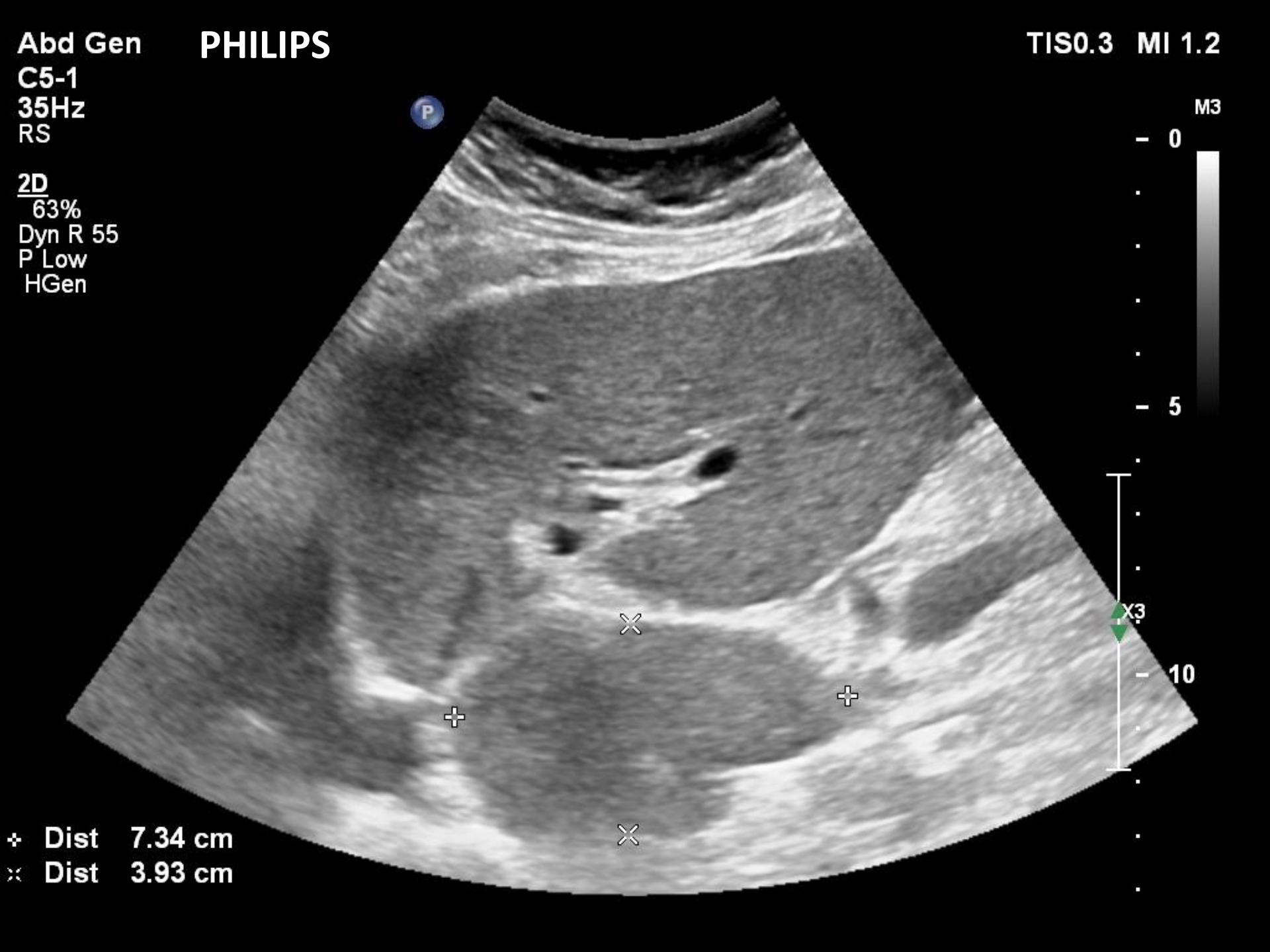
P Low
Gen
PHILIPS
TIS0.5 MI 0.8
M3
X3
16cm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RS
2D
63%
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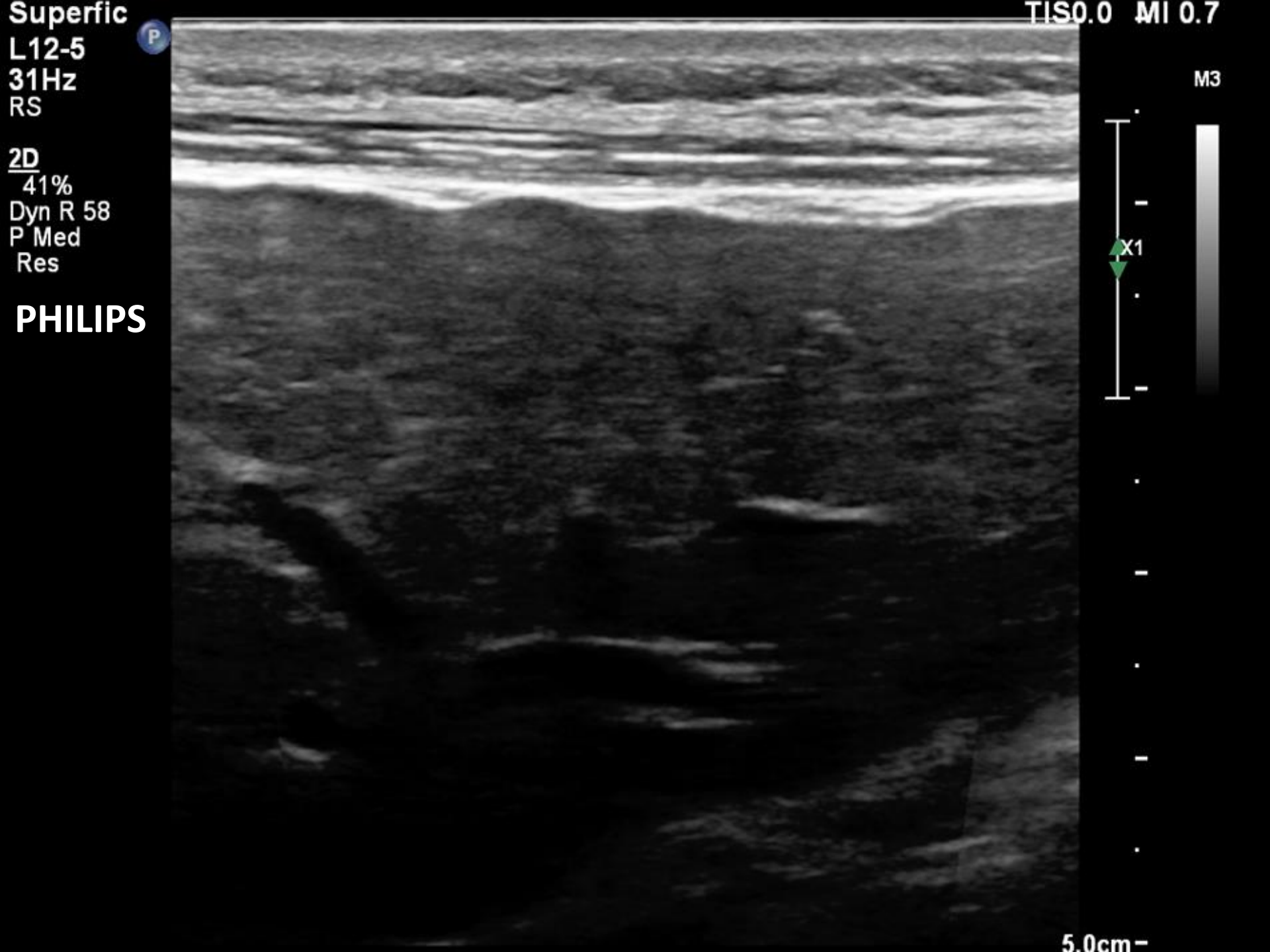
0
5
10
X3
Dist 7.34 cm
Dist 3.93 cm


Superfic
L12-5
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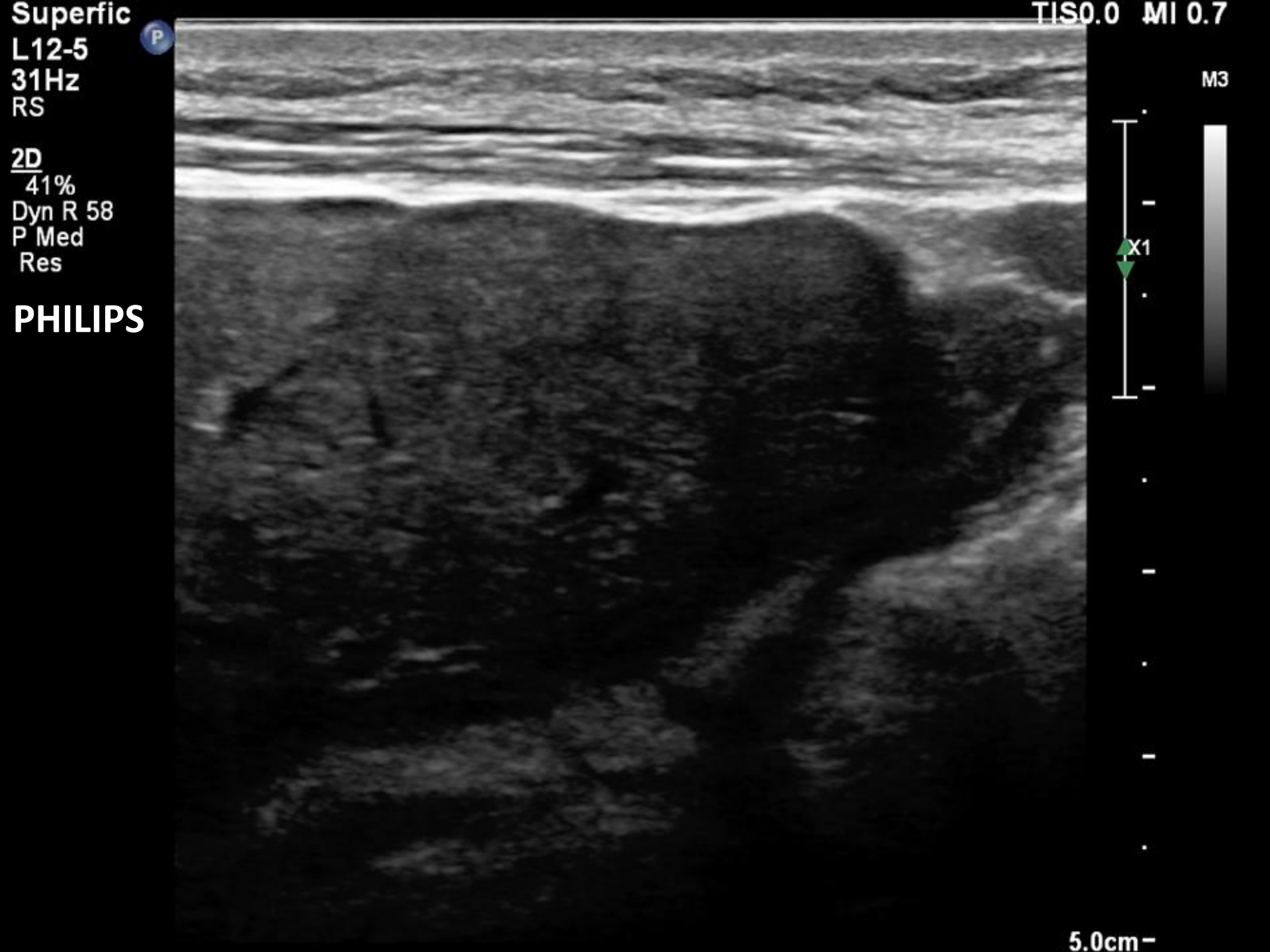
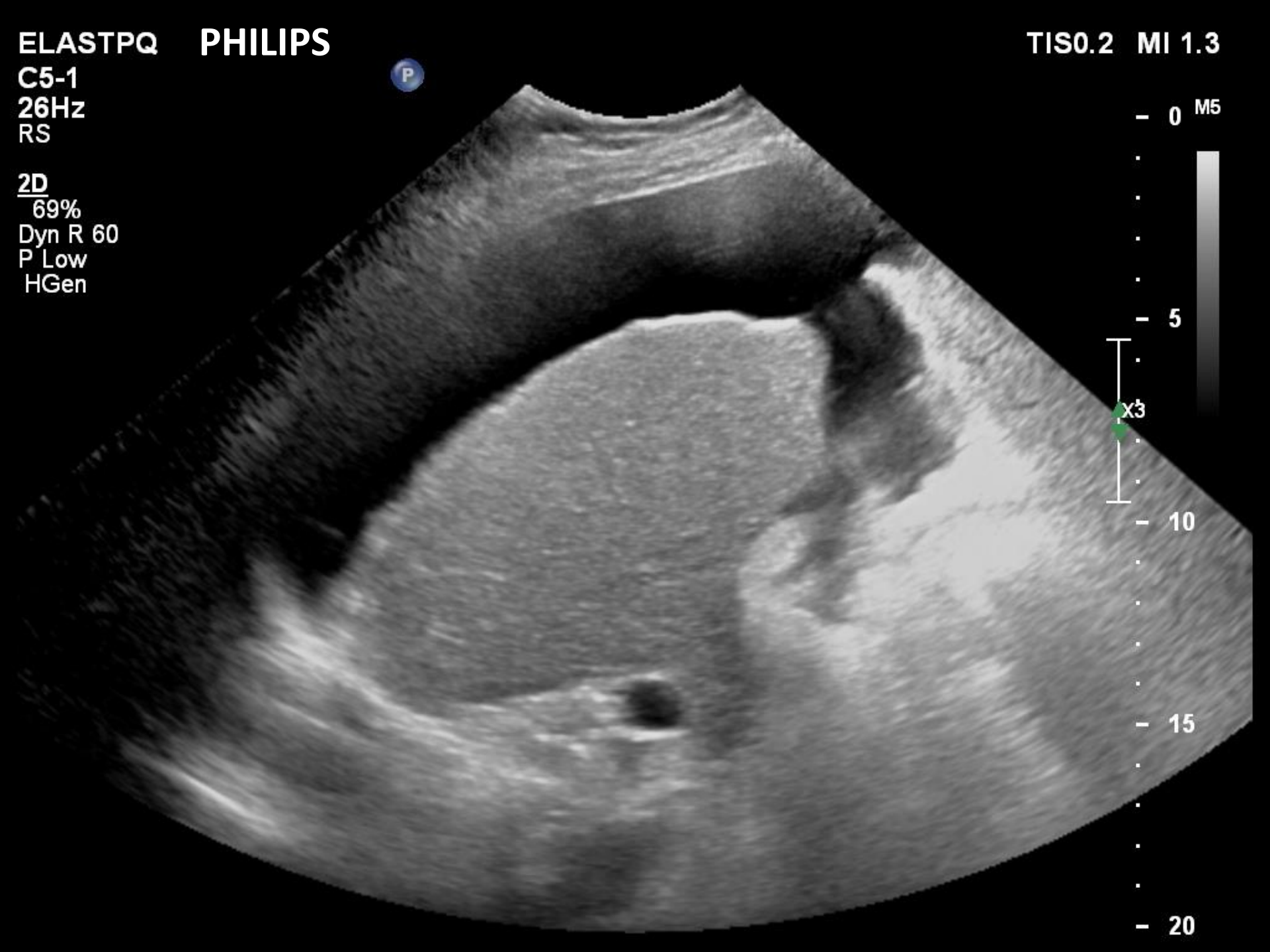
RS
2D
41%
Dyn R 58
P Med
Res
PHILIPS
TIS0.0 MI 0.7
M3
X1
5.0cm


Superfic
L12-5
31Hz
RS
2D
41%
Dyn R 58
P Med
Res
PHILIPS
TIS0.0 MI 0.7
M3
X1
5.0cm


ELASTPQ
PHILIPS
C5-1
26Hz
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HGen
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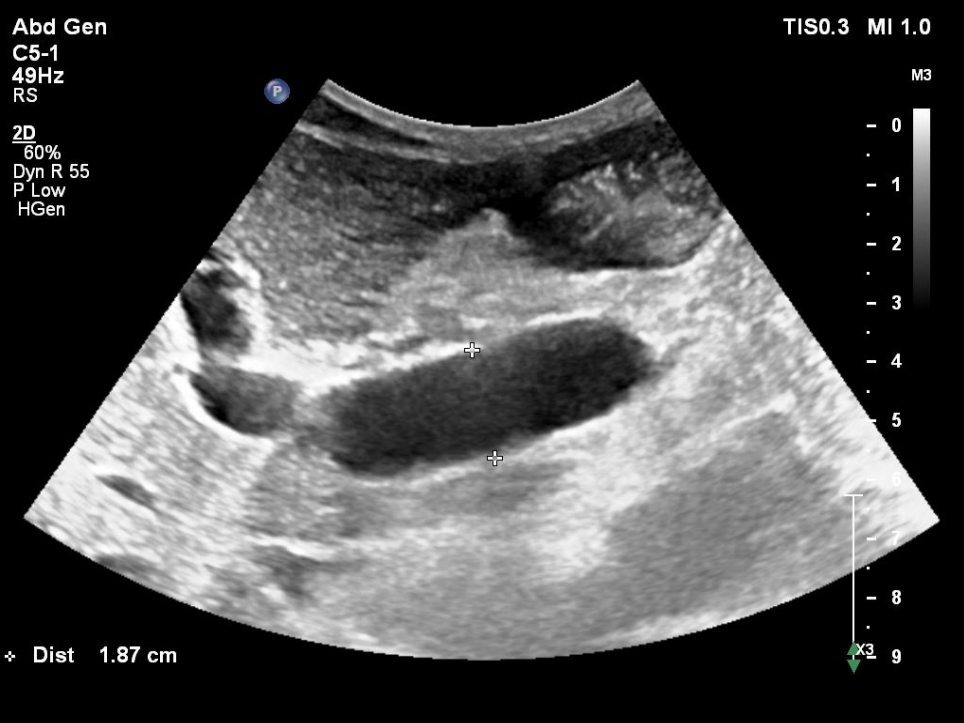
M5
0
5
10
15
20
x3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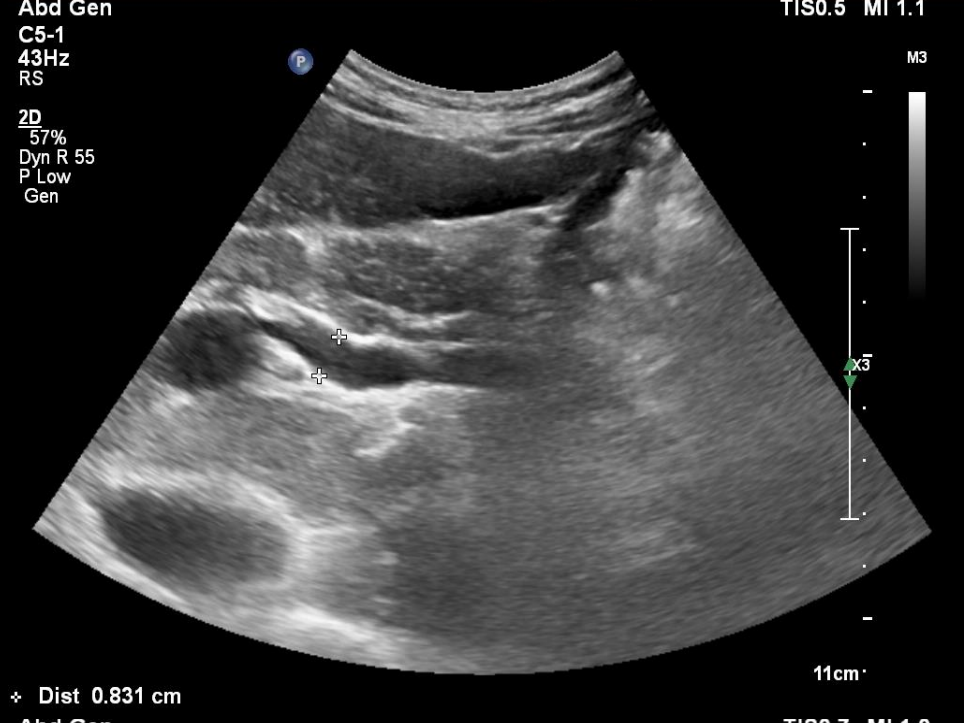
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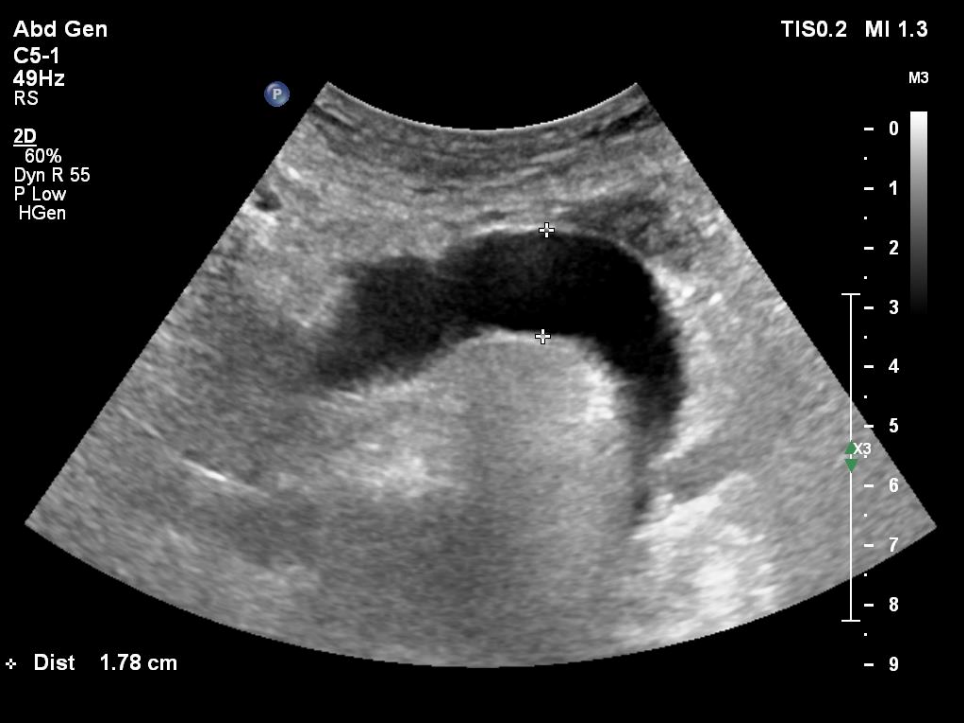
11cm
Dist 0.831 cm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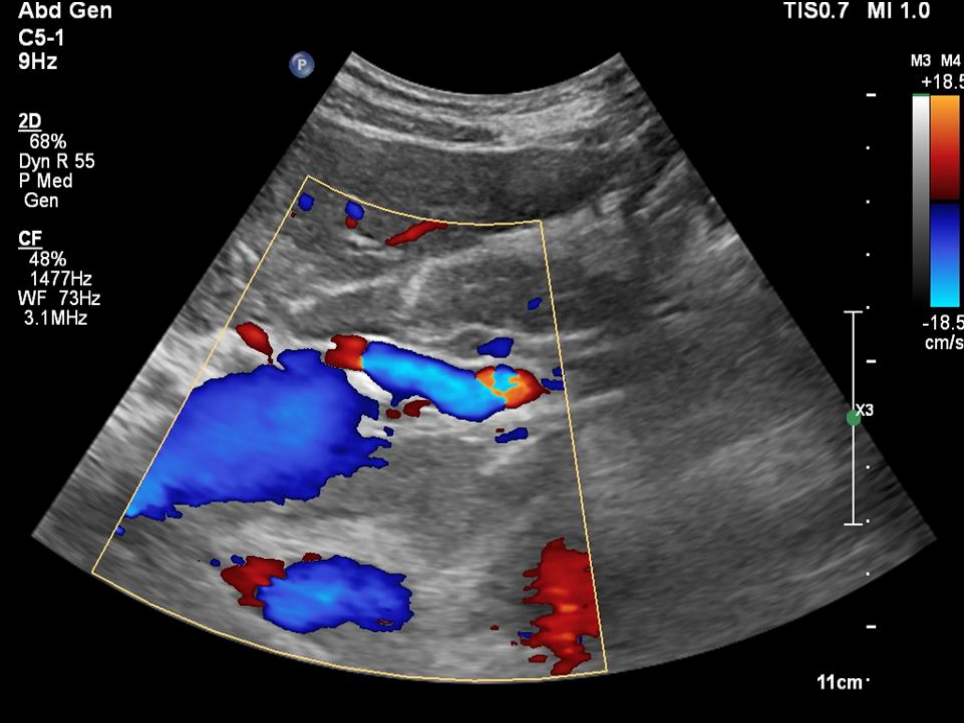
49Hz
RS
2D
60%
Dyn R 55
P Low
HGen
TIS0.2 MI 1.3
M3
Dist 1.78 cm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C5-1
9Hz
2D
68%
Dyn R 55
P Med
Gen
CF
48%
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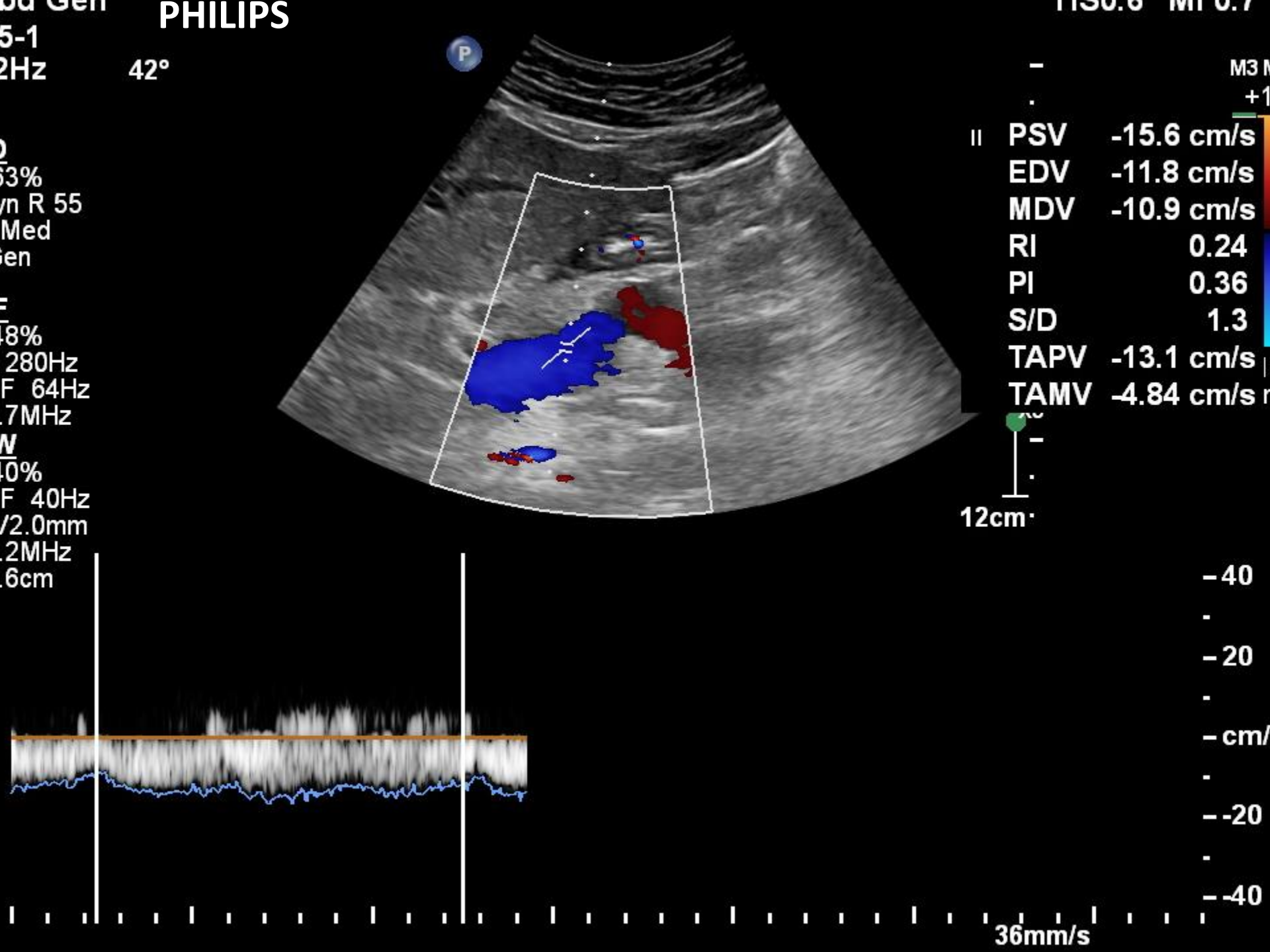
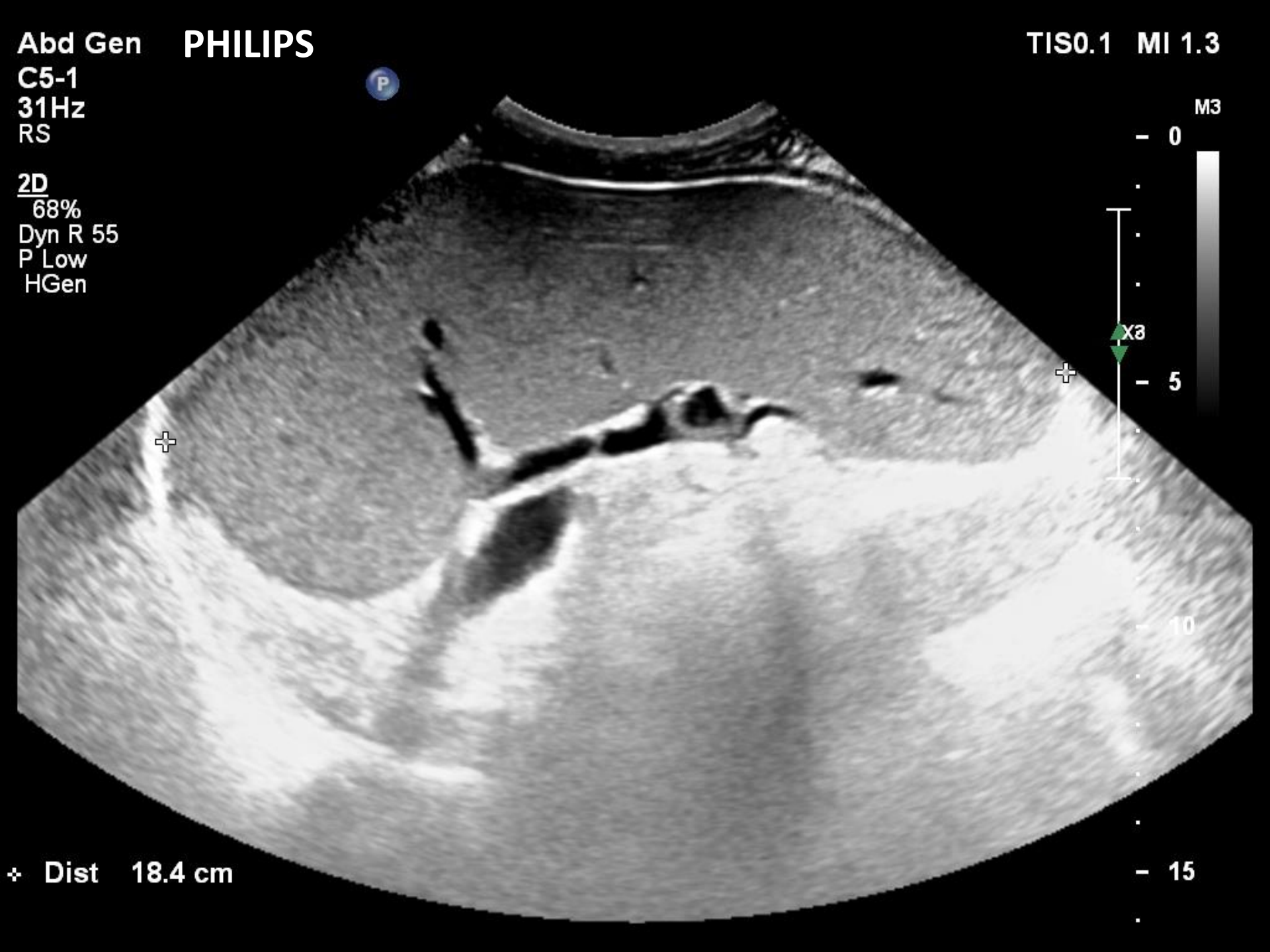
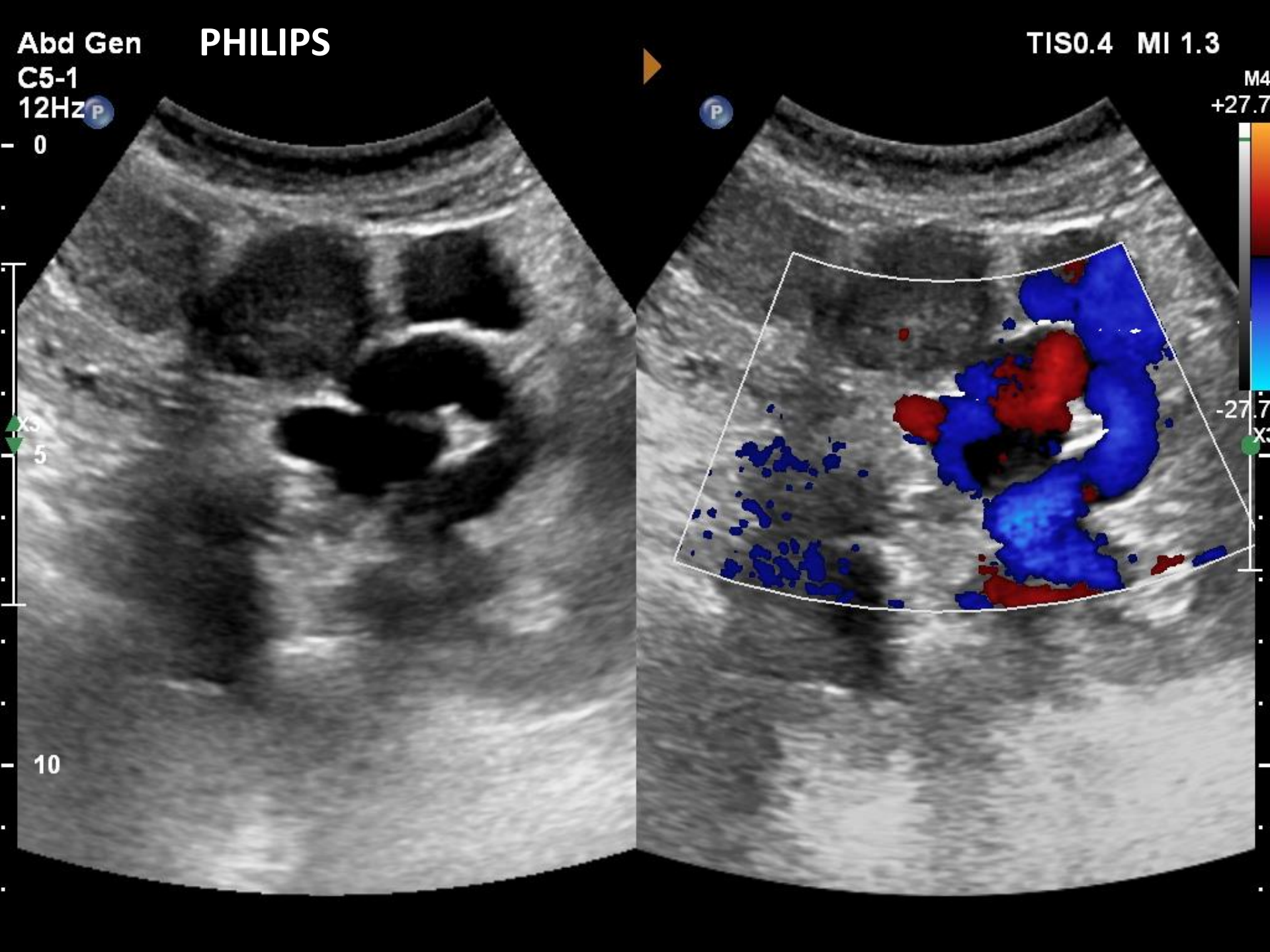
WF 73Hz
3.1MHz
TIS0.7 MI 1.0
M3 M4
-18.5
cm/s
11cm


PHILIPS
42°
PSV -15.6 cm/s
EDV -11.8 cm/s
MDV -10.9 cm/s
RI 0.24
PI 0.36
S/D 1.3
TAPV -13.1 cm/s
TAMV -4.84 cm/s
12cm
40
20
-20
-40
36mm/s


Abd Gen
PHILIPS
C5-1
31Hz
RS
2D
68%
Dyn R 55
P Low
HGen
TIS0.1 MI 1.3
M3
0
5
10
15
X3
Dist 18.4 cm


Abd Gen
C5-1
12Hz
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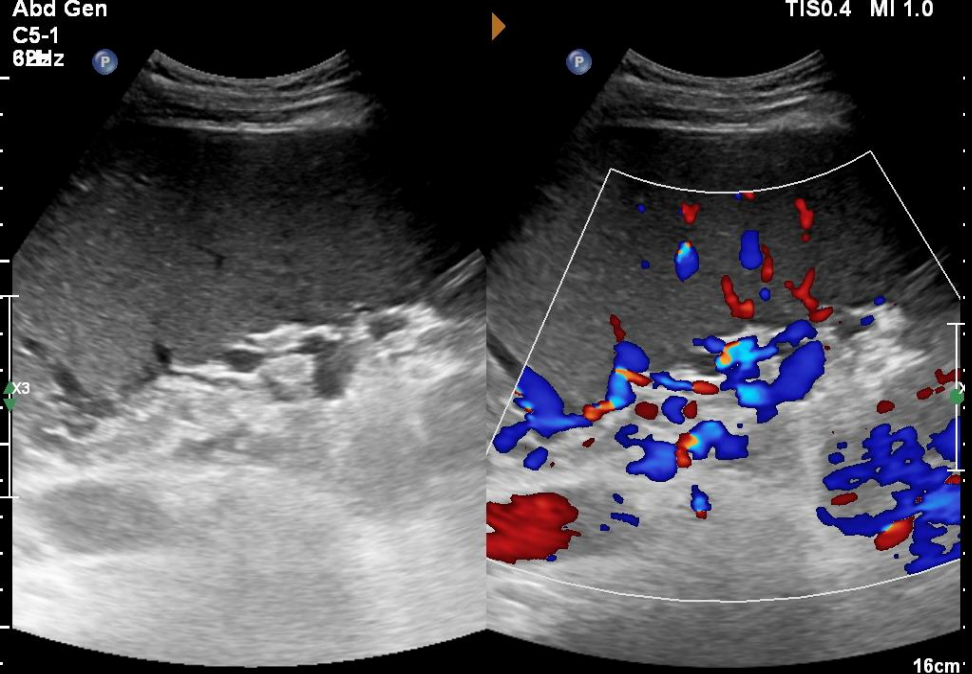
TIS0.4 MI 1.3
M4
+27.7
-27.7
0
5
10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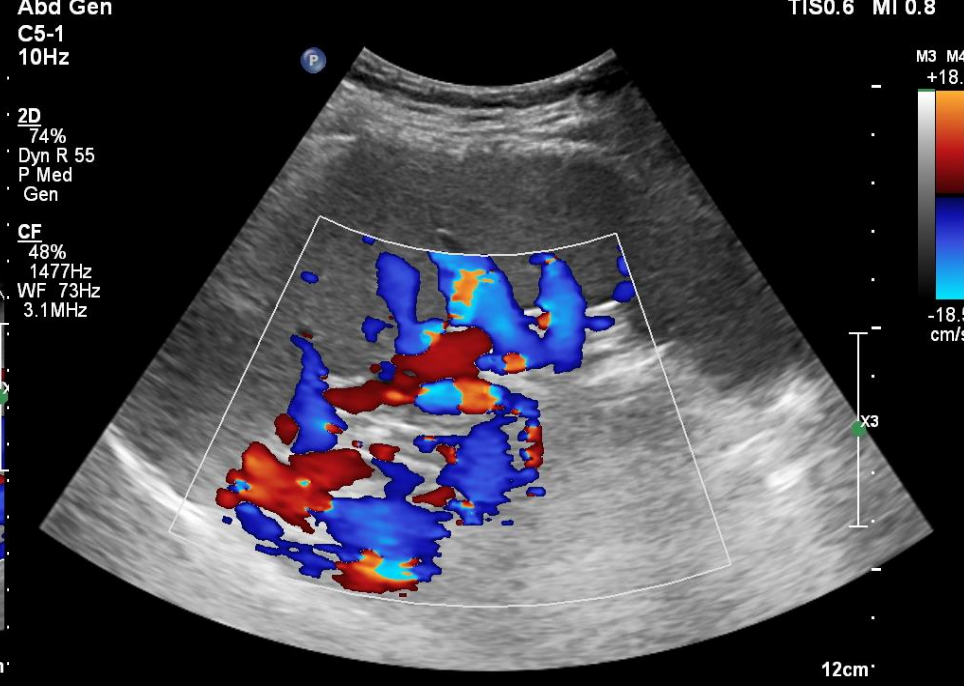
16cm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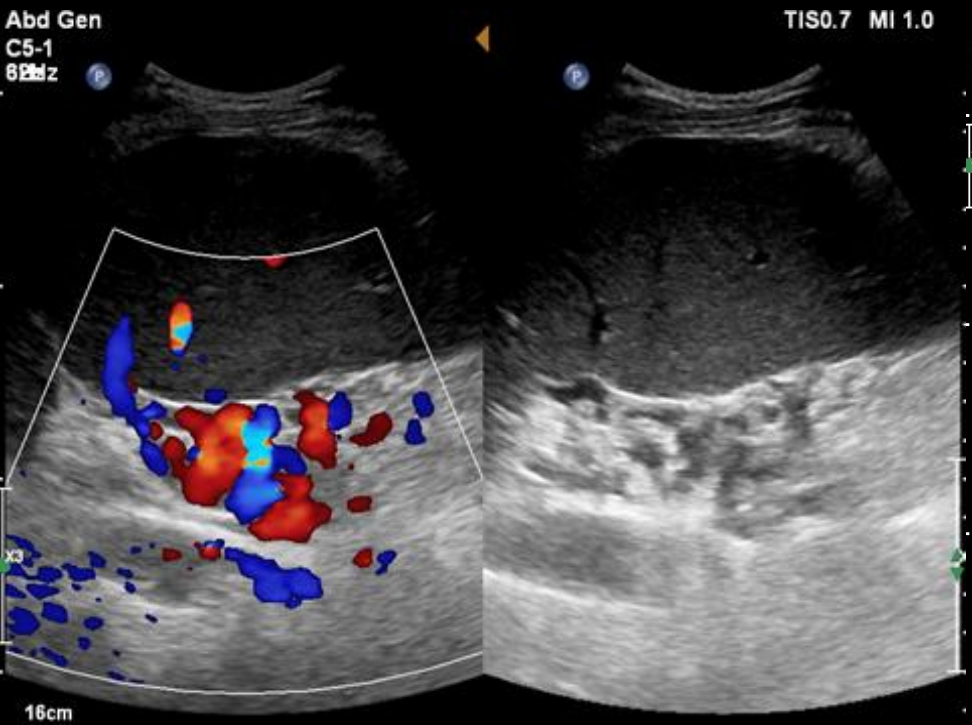
C5-1
10Hz
2D
74%
Dyn R 55
P Med
Gen
CF
48%
1477Hz
WF 73Hz
3.1MHz
TIS0.6 MI 0.8
12cm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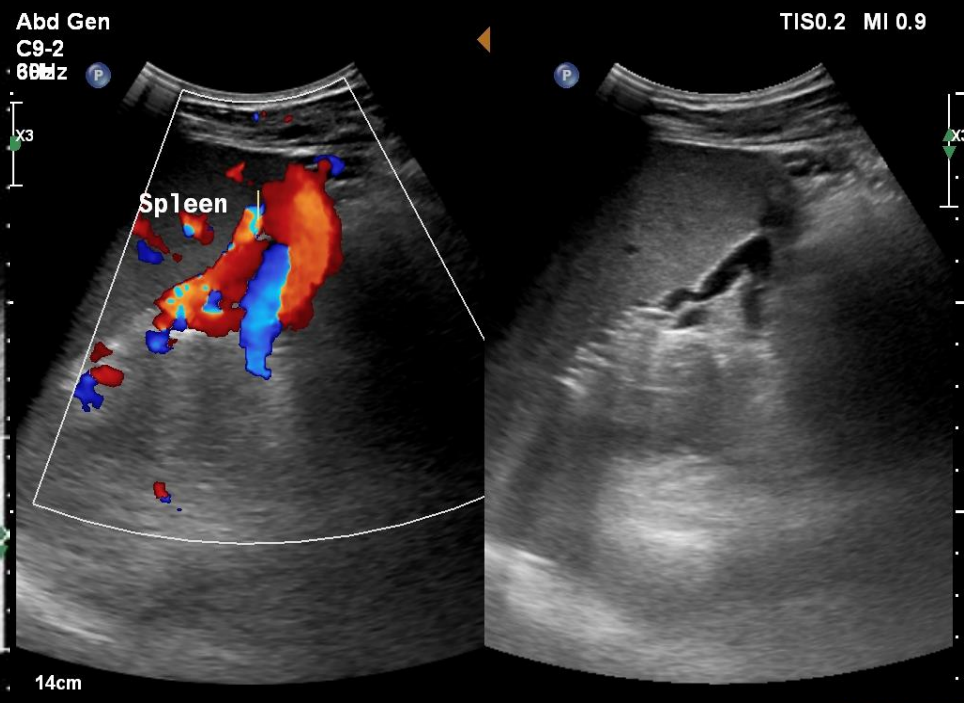
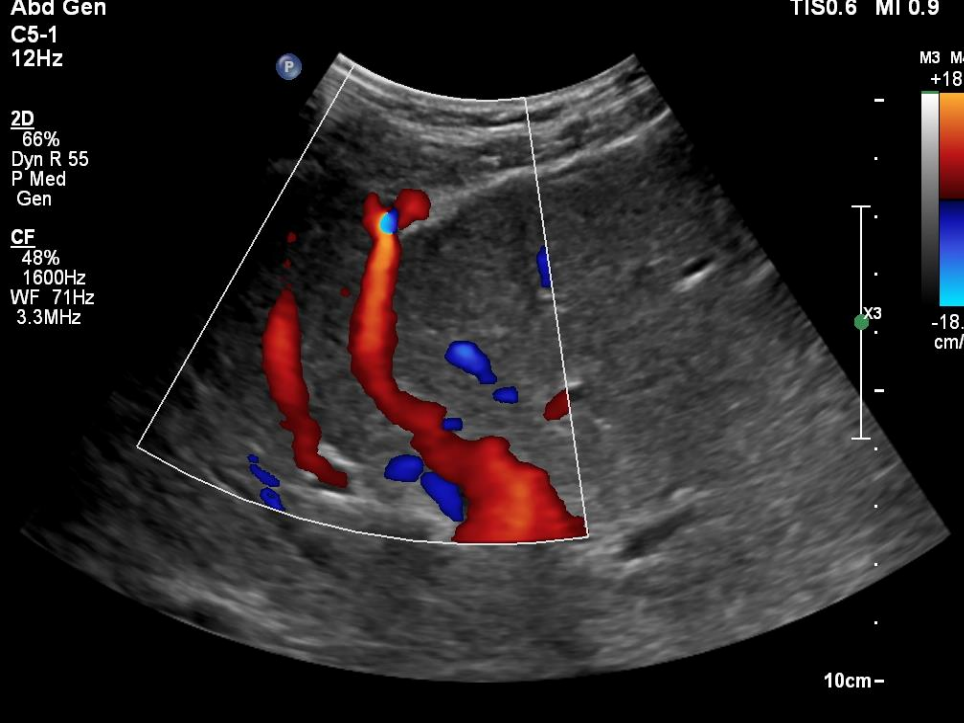
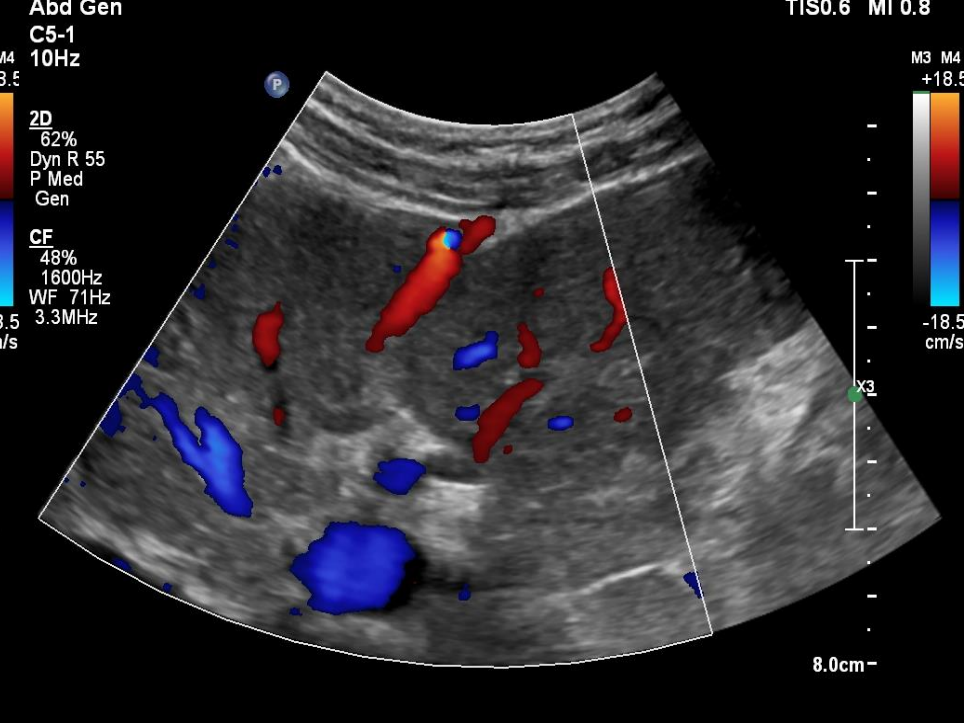
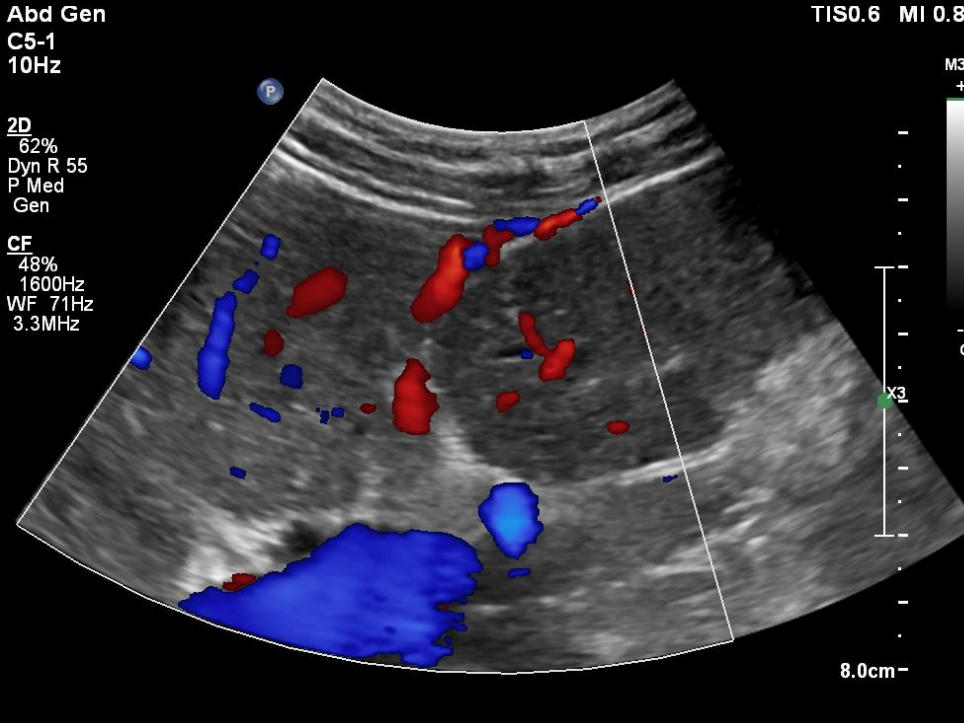
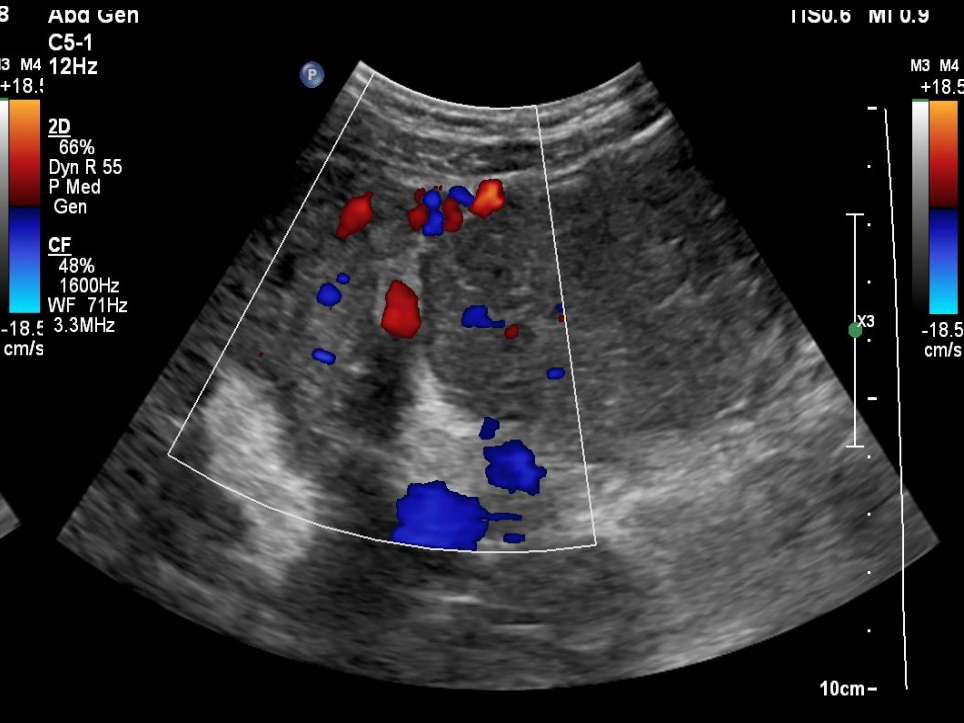
C5-1
TIS0.7 MI 1.0
16cm


Abd Gen
C9-2
TIS0.2 MI 0.9
Spleen
14cm


Abd Gen
C5-1
12Hz
2D
66%
Dyn R 55
P Med
Gen
CF
48%
1600Hz
WF 71Hz
3.3MHz
TIS0.6 MI 0.9
M3 M4
+18.5
-18.5
cm/s
10cm


Abd Gen
C5-1
10Hz
2D
62%
Dyn R 55
P Med
Gen
CF
48%
1600Hz
WF 71Hz
3.3MHz
TIS0.6 MI 0.8
M3 M4
+18.5
-18.5
cm/s
8.0cm

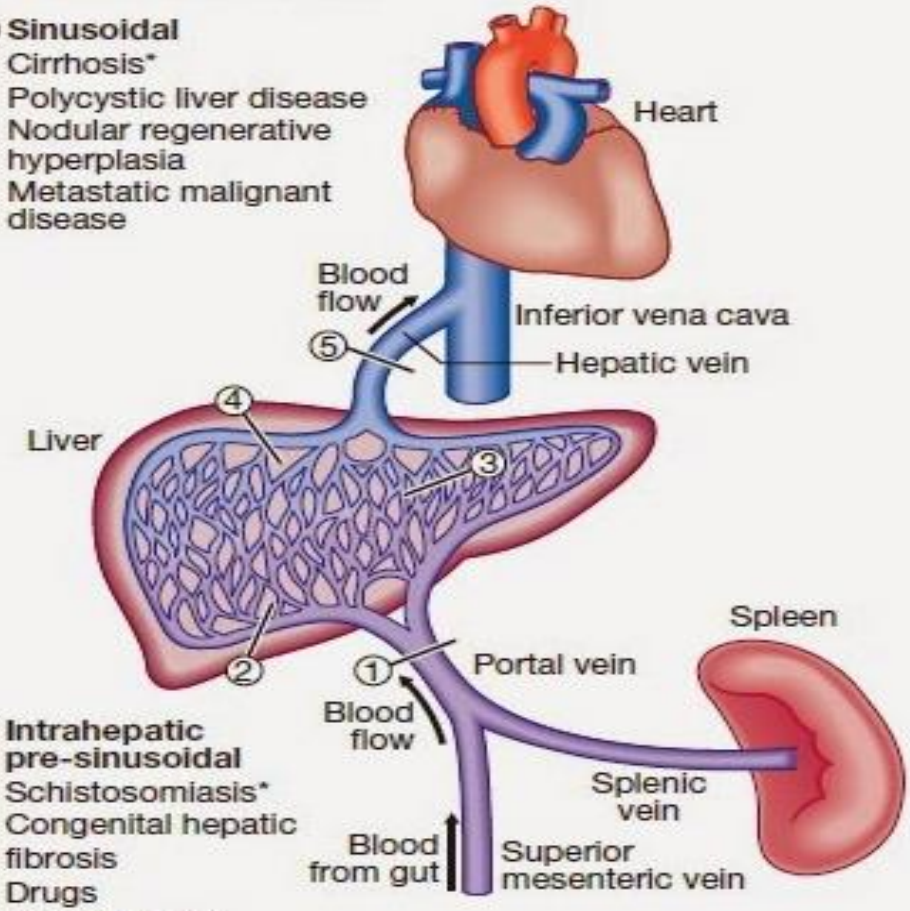

Abd Gen
C5-1
10Hz
2D
62%
Dyn R 55
P Med
Gen
CF
48%
1600Hz
WF 71Hz
3.3MHz
TIS0.6 MI 0.8
M3 M4
+18.5
-18.5
cm/s
8.0cm


Abd Gen
C5-1
12Hz
2D
66%
Dyn R 55
P Med
Gen
CF
48%
1600Hz
WF 71Hz
3.3MHz
TIS0.6 MI 0.9
M3 M4
+18.5
-18.5
cm/s
10cm

⑤ **Post-hepatic post-sinusoidal**
Budd–Chiari syndrome

④ **Intrahepatic post-sinusoidal**
Veno-occlusive disease

③ **Sinusoidal**
Cirrhosis*
Polycystic liver disease
Nodular regenerative hyperplasia
Metastatic malignant disease



② **Intrahepatic pre-sinusoidal**
Schistosomiasis*
Congenital hepatic fibrosis
Drugs
Vinyl chloride
Sarcoidosis

① **Prehepatic pre-sinusoidal**
Portal vein thrombosis due to sepsis (umbilical, portal pyaemia) or procoagulopathy or secondary to cirrhosis
Abdominal trauma including surgery

Fig. 23.19 Classification of portal hypertension according to site of vascular obstruction. *Most common cause. Note that splenic vein occlusion can also follow pancreatitis, leading to gastric varices.

1. Pre-hepatic pre-sinusoidal

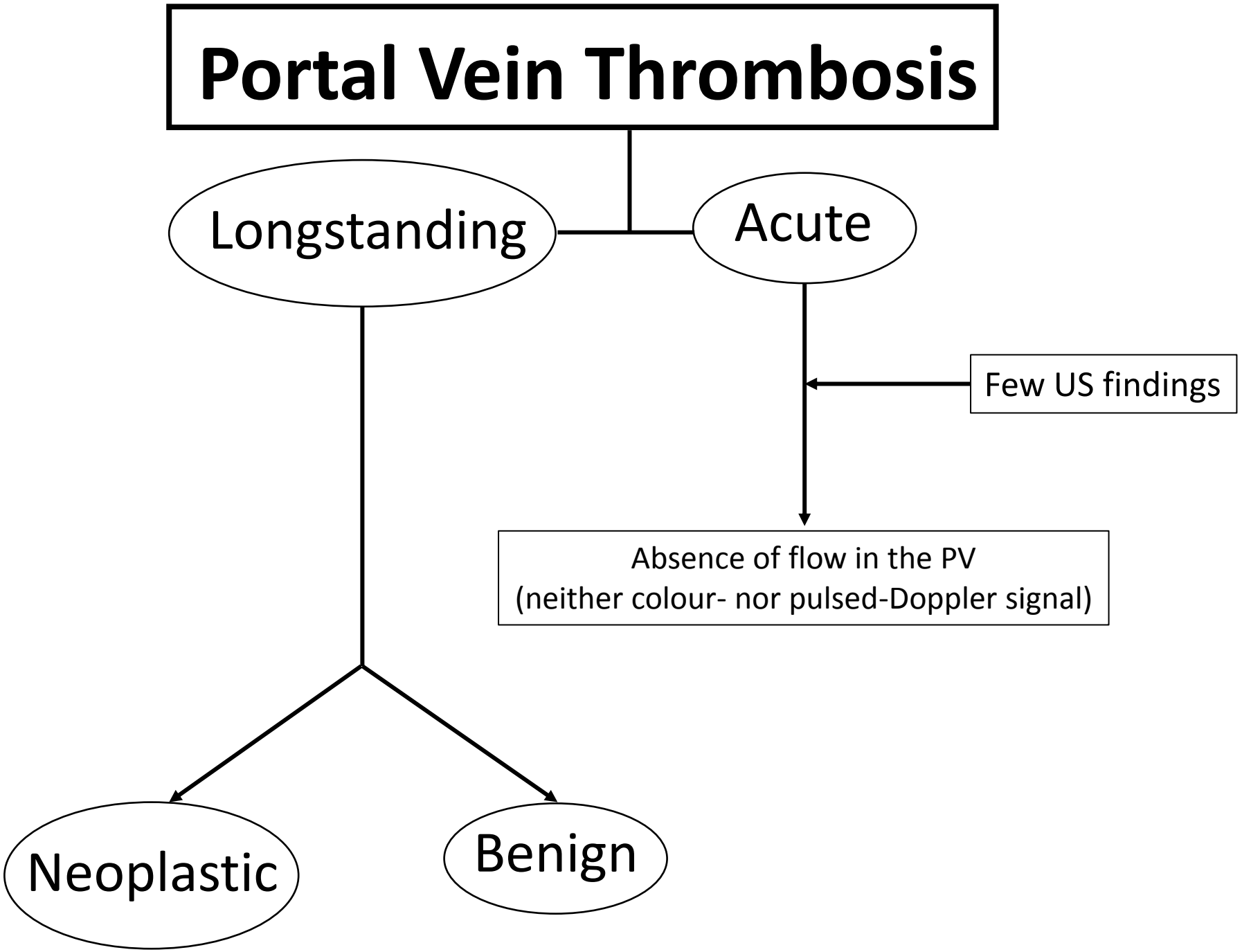
2. Intra-hepatic pre-sinusoidal

Portal Vein Thrombosis

3. Intra-hepatic post-sinusoidal
Cirrhosis

4. Intra-hepatic post-sinusoidal

5. Post-hepatic post-sinusoidal


Portal Vein Thrombosis
Longstanding
Acute
Few US findings
Absence of flow in the PV
(neither colour- nor pulsed-Doppler signal)
Neoplastic
Benign

Portal Vein Thrombosis

Longstanding

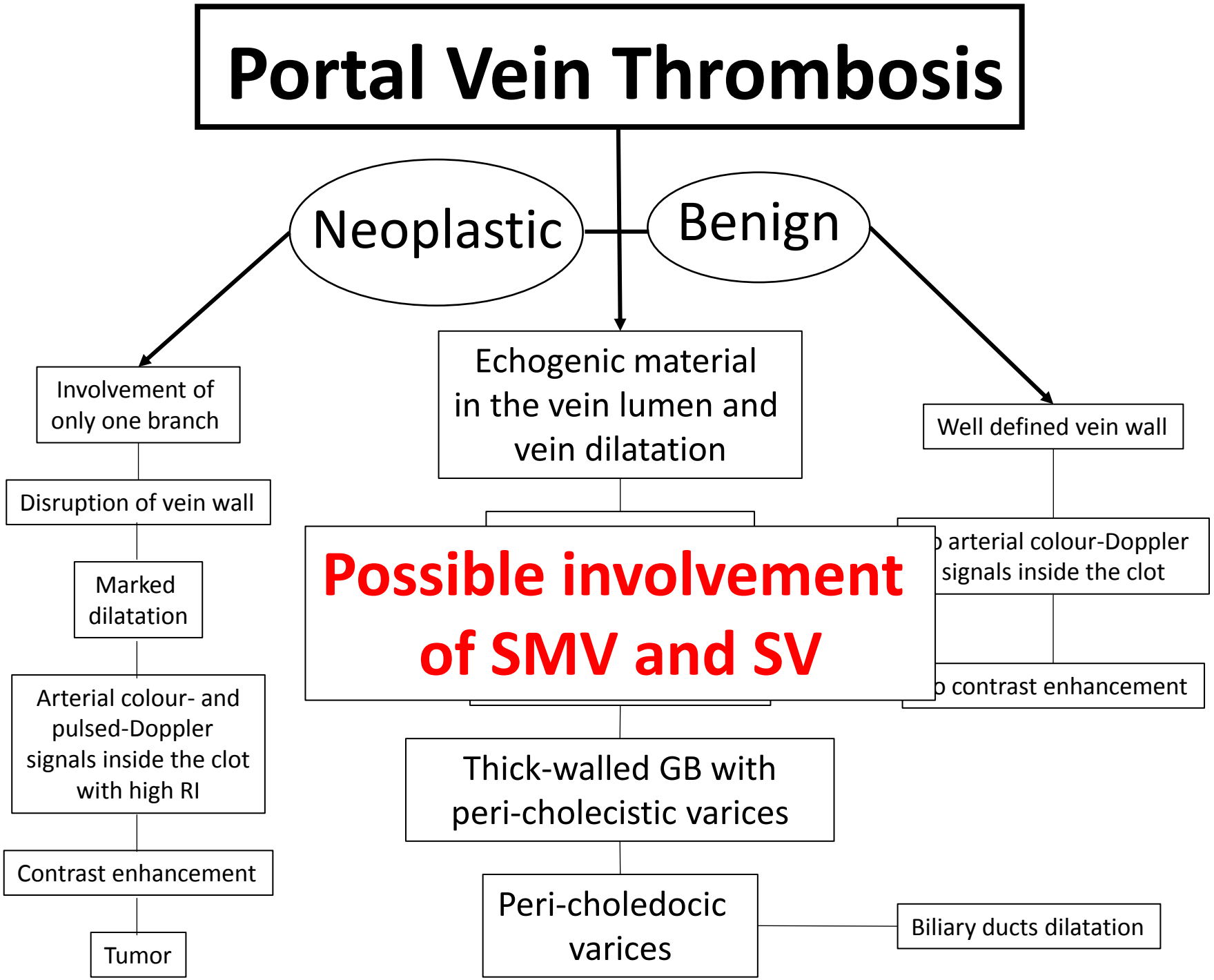
Acute

Few US findings

Absence of flow in the PV
(neither colour- nor pulsed-Doppler signal)

Neoplastic

Benign


Portal Vein Thrombosis
Neoplastic
Benign
Involvement of only one branch
Disruption of vein wall
Marked dilatation
Arterial colour- and pulsed-Doppler signals inside the clot with high RI
Contrast enhancement
Tumor
Echogenic material in the vein lumen and vein dilatation
Possible involvement of SMV and SV
Thick-walled GB with peri-cholecistic varices
Peri-choledocic varices
Biliary ducts dilatation
Well defined vein wall
arterial colour-Doppler signals inside the clot
contrast enhancement

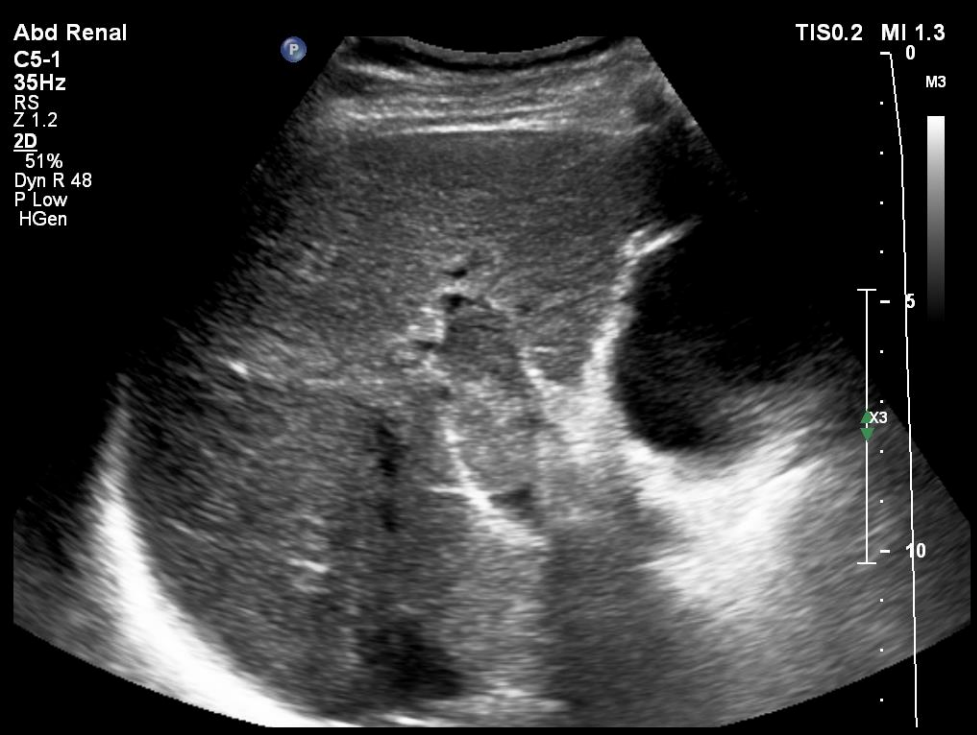
Portal Vein Thrombosis

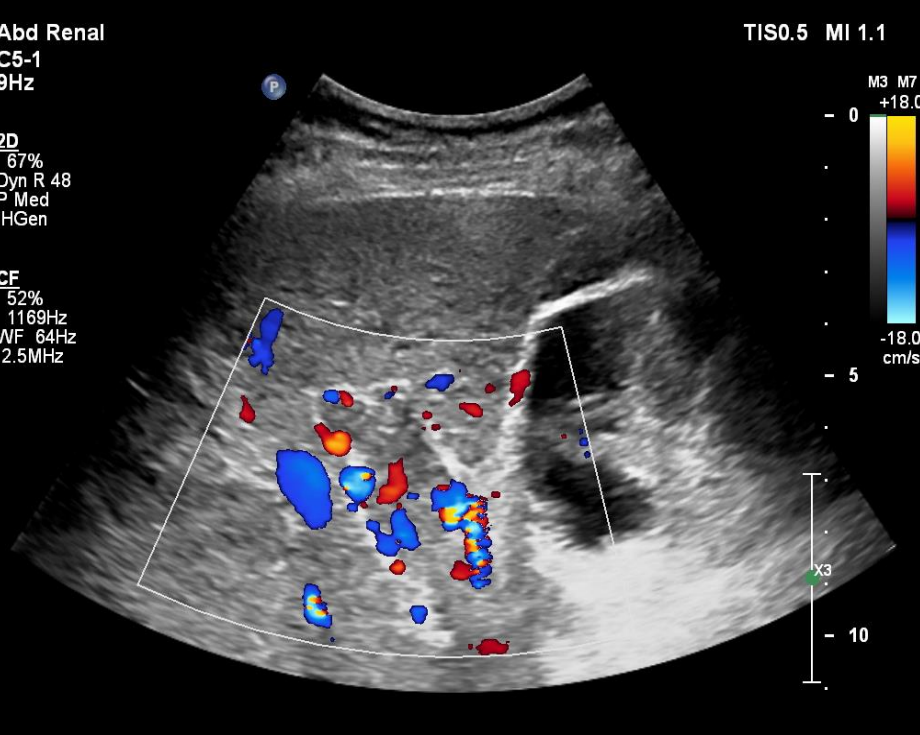
B-mode US:

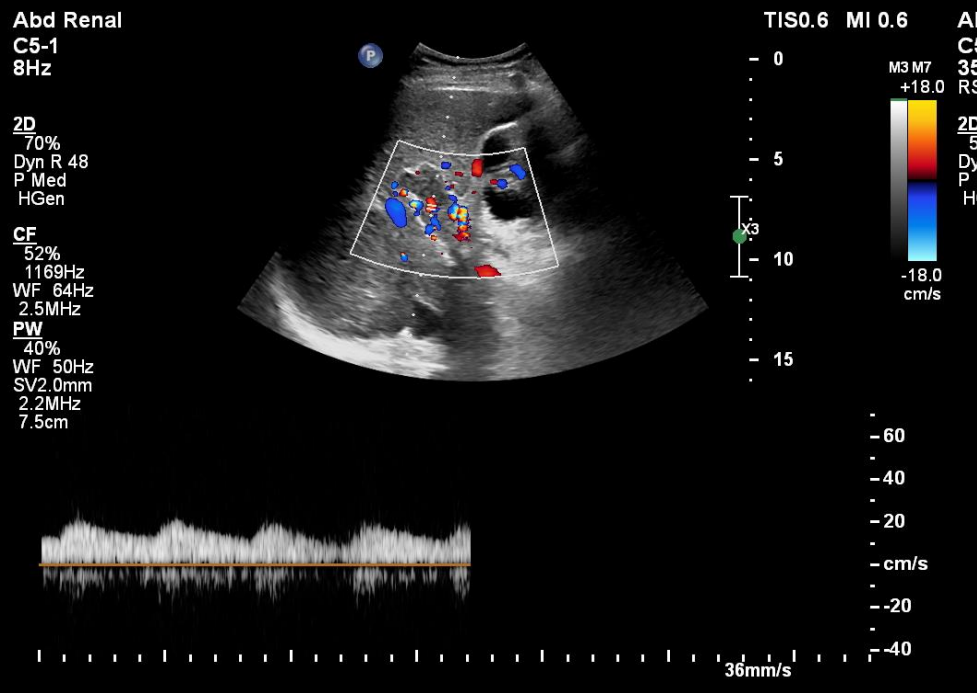
- Normal or heterogeneous liver parenchyma
- Focal liver lesion
- A marked heterogenous hepatic area: infiltrating carcinoma
- Hepatic artery hypertrophy
- Enlarged spleen (size > 13mm, area > 45 cm^2)

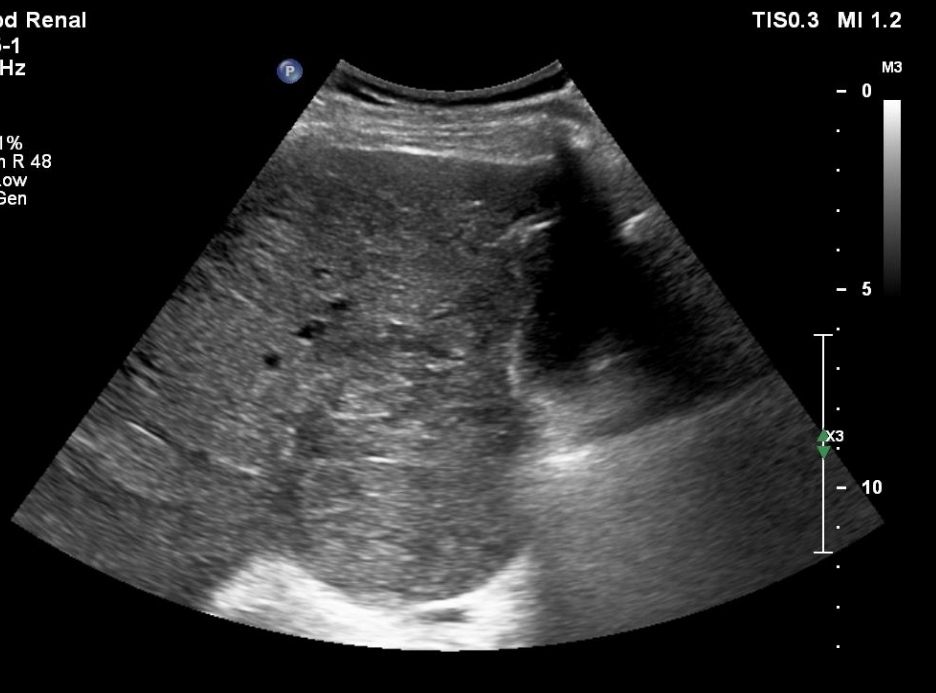
Eco-color Doppler:

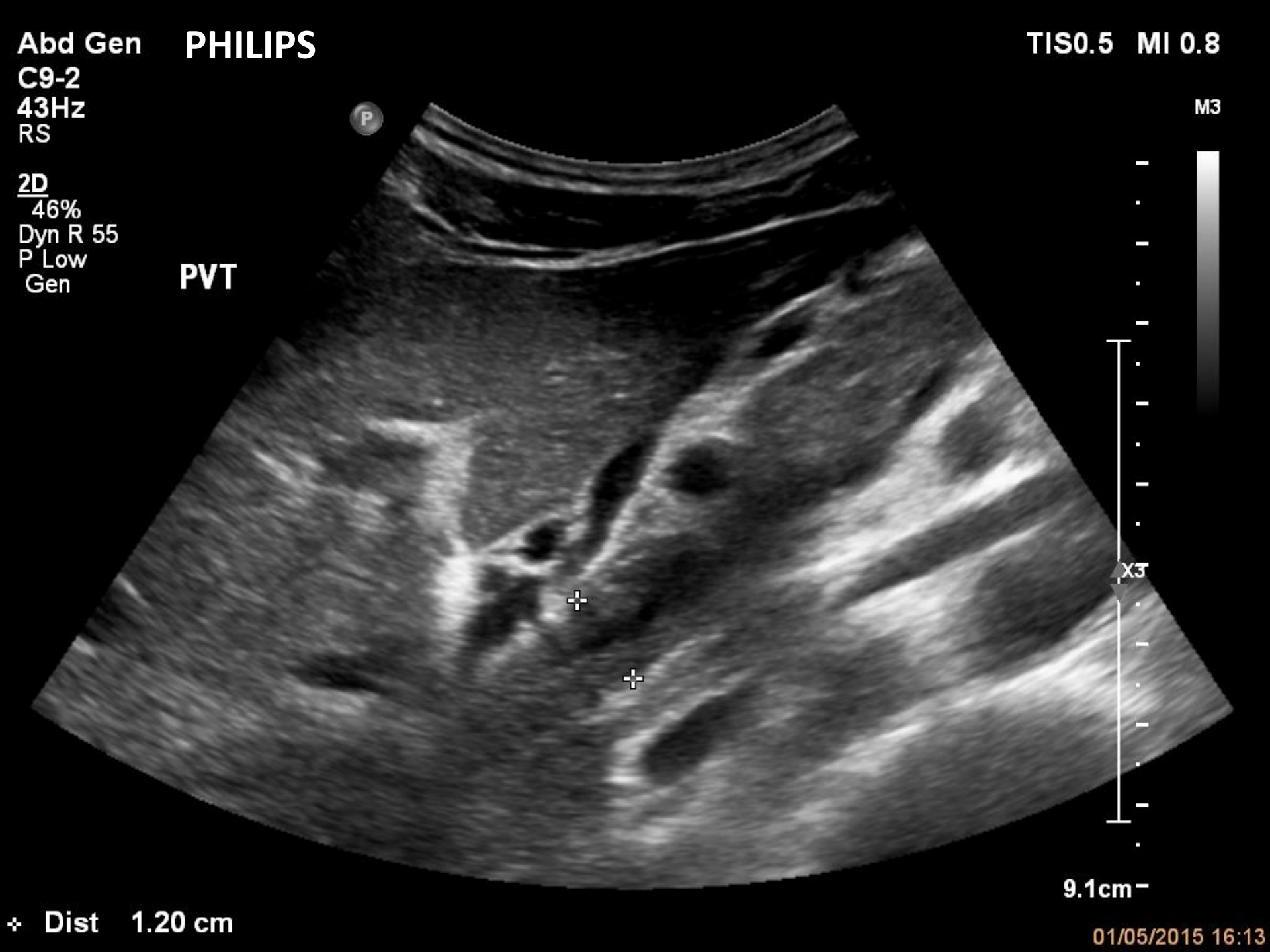
- **Portal cavernoma transformation**
- **Hepatic Artery:**
  ✓ RI > 0,70
- **Collateral vessels/varices**

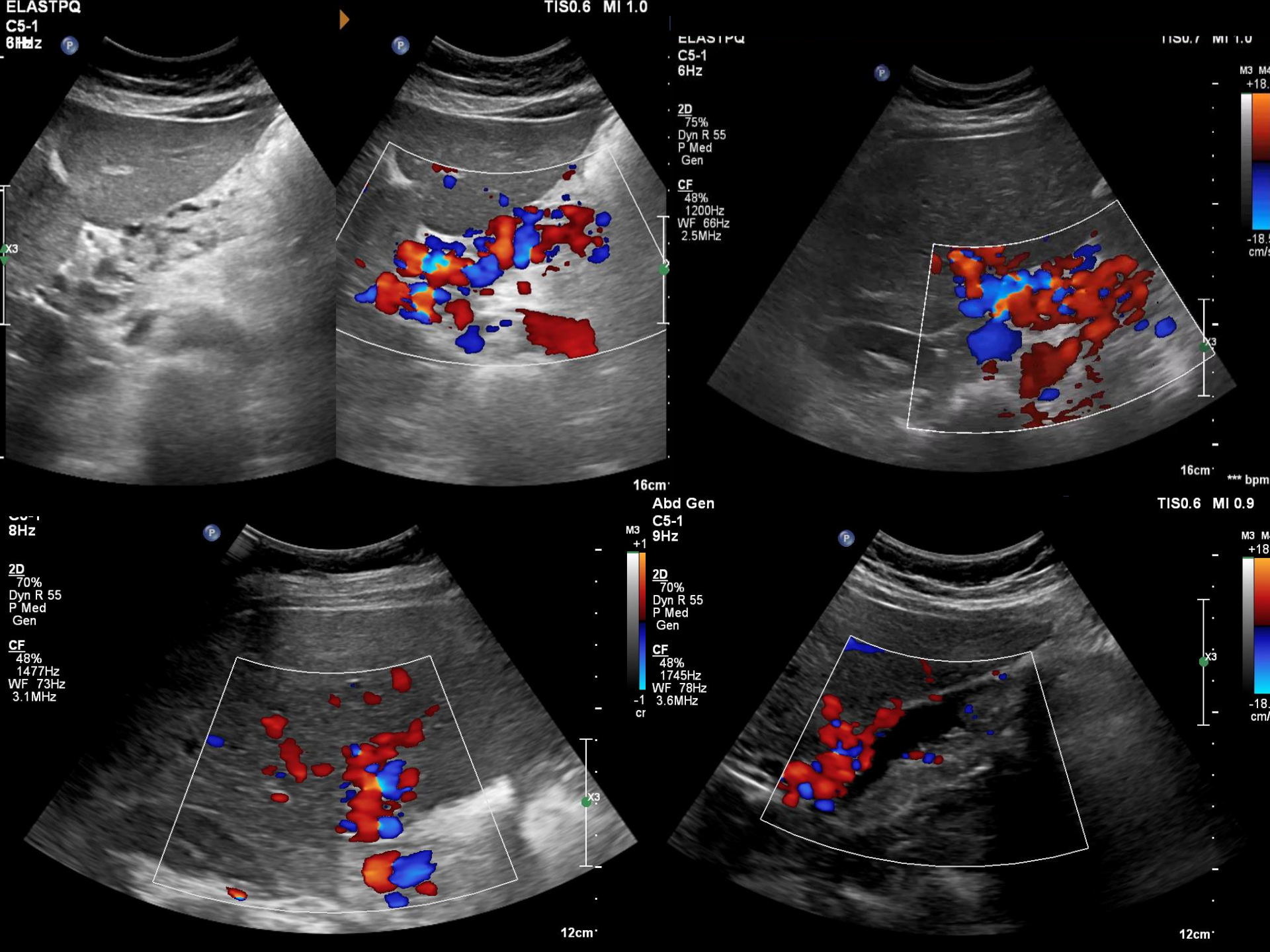

Abd Renal
C5-1
35Hz
RS
Z 1.2
2D
51%
Dyn R 48
P Low
HGen
TIS0.2 MI 1.3
M3
X3

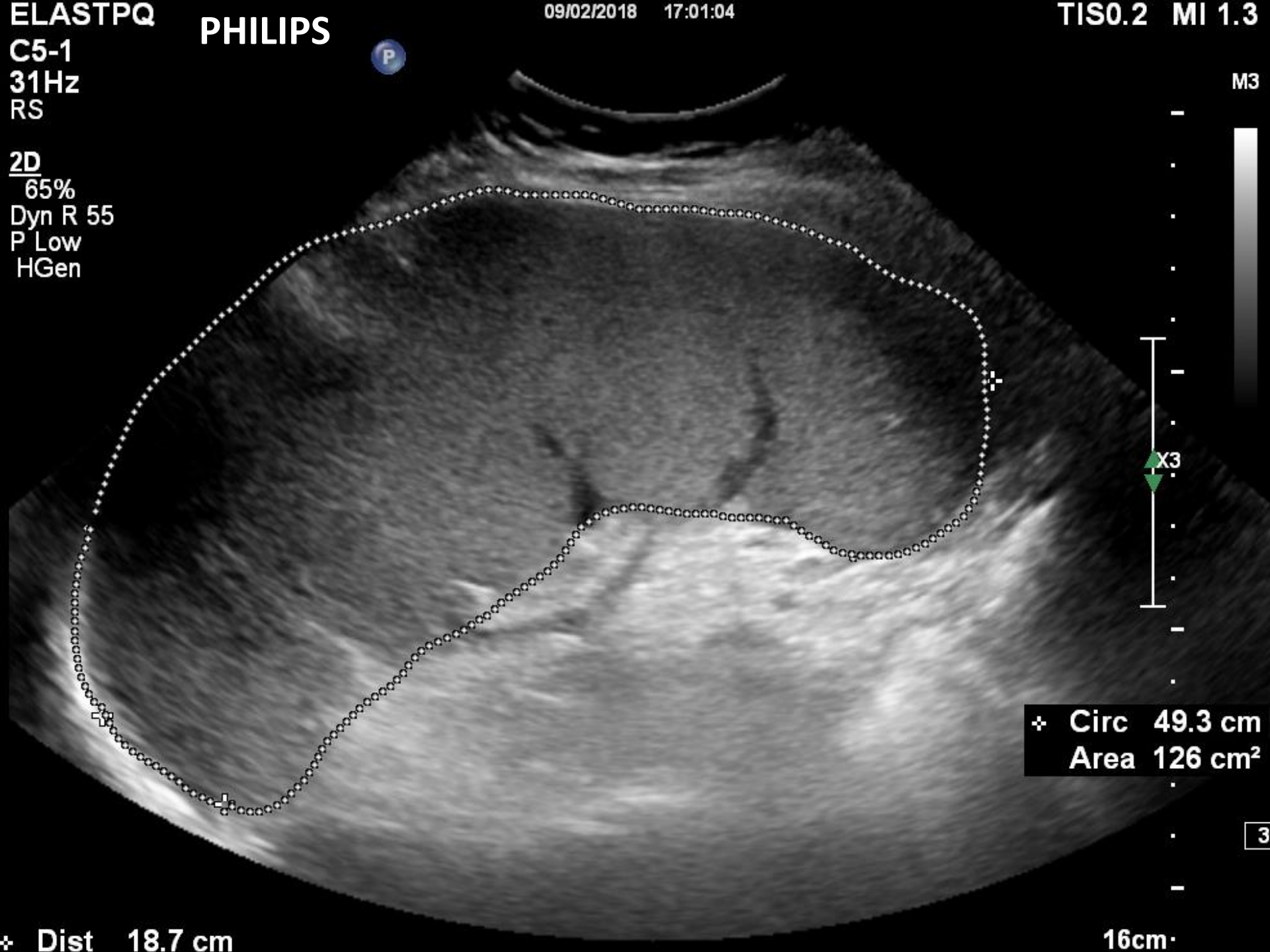

Abd Renal
C5-1
9Hz
2D
67%
Dyn R 48
P Med
HGen
CF
52%
1169Hz
WF 64Hz
2.5MHz
TIS0.5 MI 1.1
M3 M7
X3


Abd Renal
C5-1
8Hz
2D
70%
Dyn R 48
P Med
HGen
CF
52%
1169Hz
WF 64Hz
2.5MHz
PW
40%
WF 50Hz
SV2.0mm
2.2MHz
7.5cm
TIS0.6 MI 0.6
M3 M7
+18.0
-18.0
cm/s
X3
cm/s
36mm/s


Abd Renal
C5-1
35Hz
RS
2D
51%
Dyn R 48
P Low
HGen
TIS0.3 MI 1.2
M3
X3


Abd Gen
C9-2
43Hz
RS
2D
46%
Dyn R 55
P Low
Gen
PHILIPS
PVT
TIS0.5 MI 0.8
M3
X3
9.1cm
Dist 1.20 cm
01/05/2015 16:13

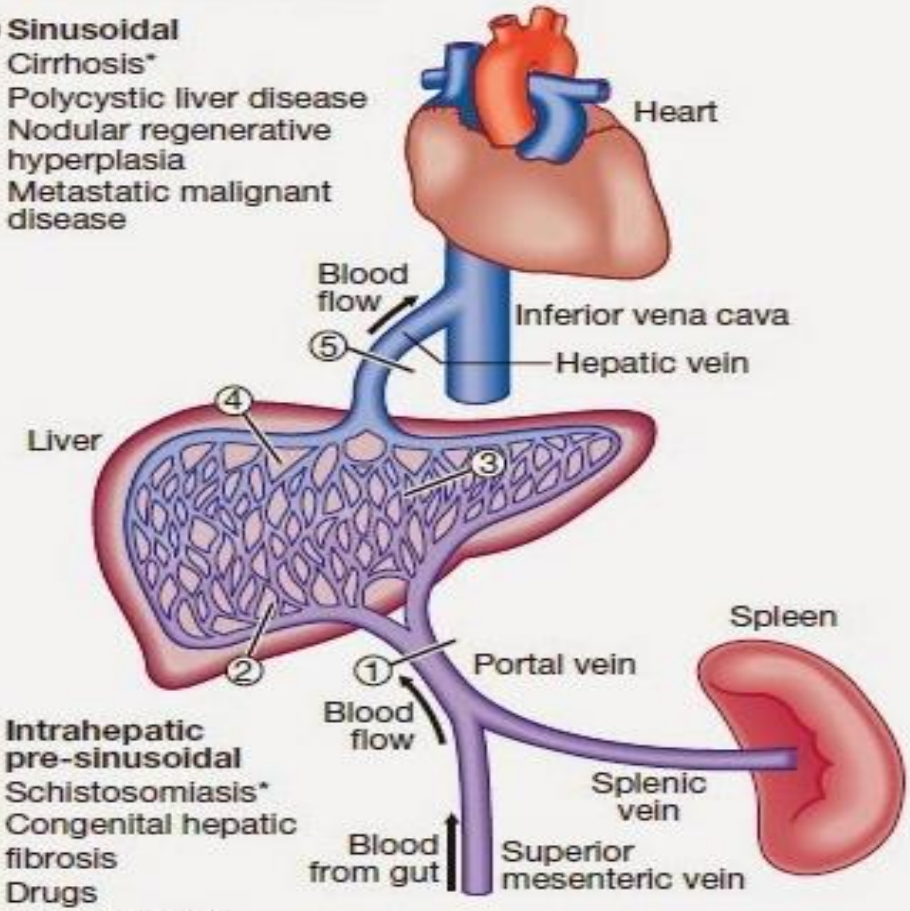

ELASTPQ
C5-1
6Hz
TIS0.6 MI 1.0
2D
75%
Dyn R 55
P Med
Gen
CF
48%
1200Hz
WF 66Hz
2.5MHz
16cm
ELASTPQ
C5-1
6Hz
16cm
*** bpm
Abd Gen
C5-1
9Hz
2D
70%
Dyn R 55
P Med
Gen
CF
48%
1745Hz
WF 78Hz
3.6MHz
TIS0.6 MI 0.9
8Hz
2D
70%
Dyn R 55
P Med
Gen
CF
48%
1477Hz
WF 73Hz
3.1MHz
12cm
12cm


ELASTPQ
C5-1
31Hz
RS
2D
65%
Dyn R 55
P Low
HGen
PHILIPS
09/02/2018 17:01:04
TIS0.2 MI 1.3
M3
X3
Circ 49.3 cm
Area 126 cm²
3
Dist 18.7 cm
16cm

⑤ **Post-hepatic post-sinusoidal**
Budd–Chiari syndrome

④ **Intrahepatic post-sinusoidal**
Veno-occlusive disease

③ **Sinusoidal**
Cirrhosis*
Polycystic liver disease
Nodular regenerative hyperplasia
Metastatic malignant disease



② **Intrahepatic pre-sinusoidal**
Schistosomiasis*
Congenital hepatic fibrosis
Drugs
Vinyl chloride
Sarcoidosis

① **Prehepatic pre-sinusoidal**
Portal vein thrombosis due to sepsis (umbilical, portal pyaemia) or procoagulopathy or secondary to cirrhosis
Abdominal trauma including surgery

Fig. 23.19 Classification of portal hypertension according to site of vascular obstruction. *Most common cause. Note that splenic vein occlusion can also follow pancreatitis, leading to gastric varices.

1. **Chronic right heart failure with cardiac liver cirrhosis**
2. **Tricuspid valve disease**
3. **Constrictive pericarditis**

5. Post-hepatic post-sinusoidal

PH related to Heart disease

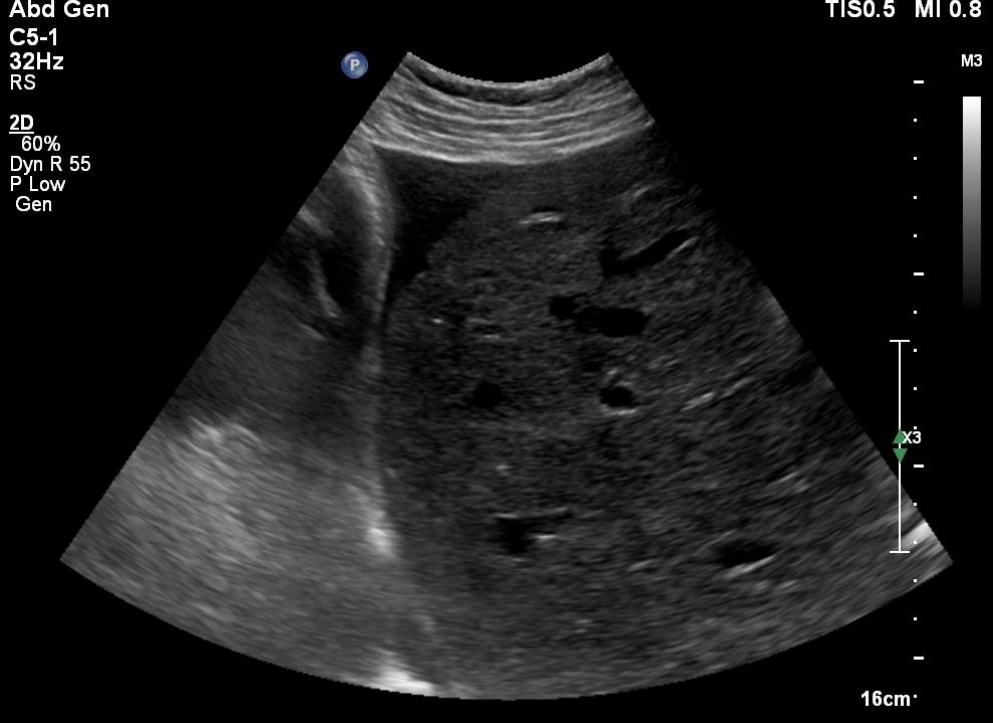
B-mode US:

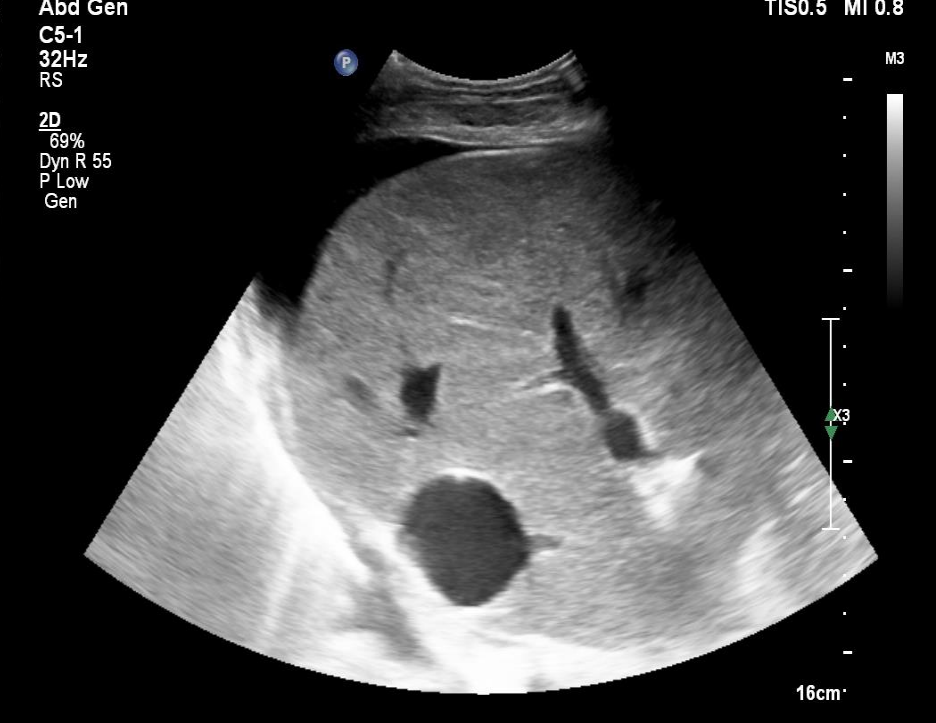
- Enlarged/congested liver
- US features of liver cirrhosis (cardiac cirrhosis)
- Enlarged spleen (size > 13mm, area > 45 cm^2)
- US features of tricuspid regurgitation and/or heart failure
- PV size > 13 mm
- No PV calibre changes with respiration
- IVC calibre > 2 cm
- HVs calibre > 1 cm
- No IVC inspiratory collapse or < 40%

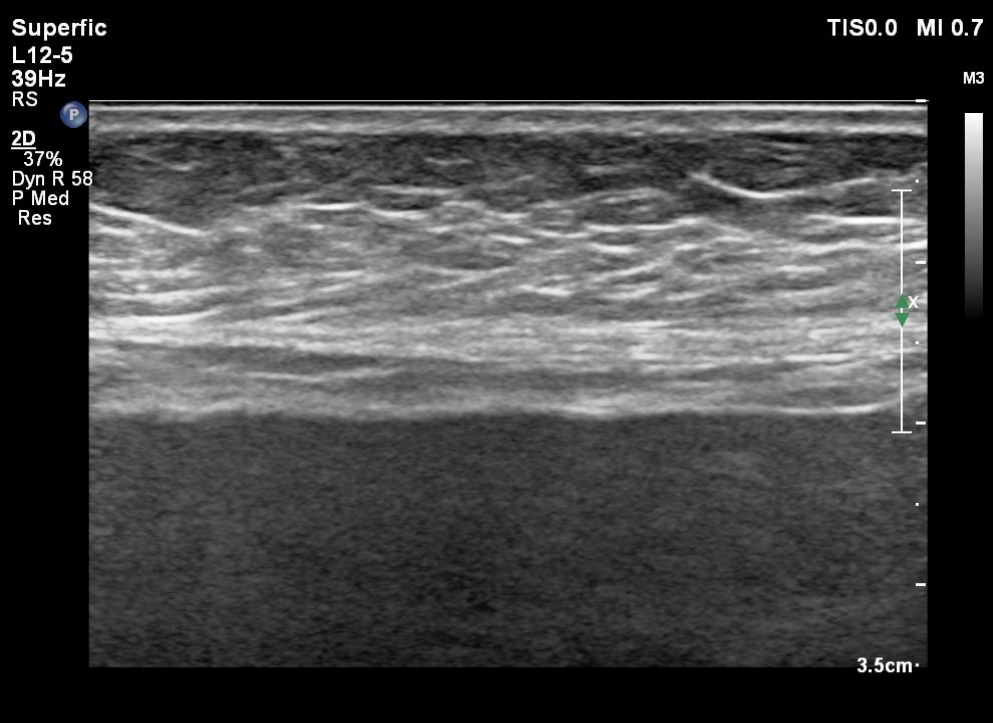
PH related to Heart disease

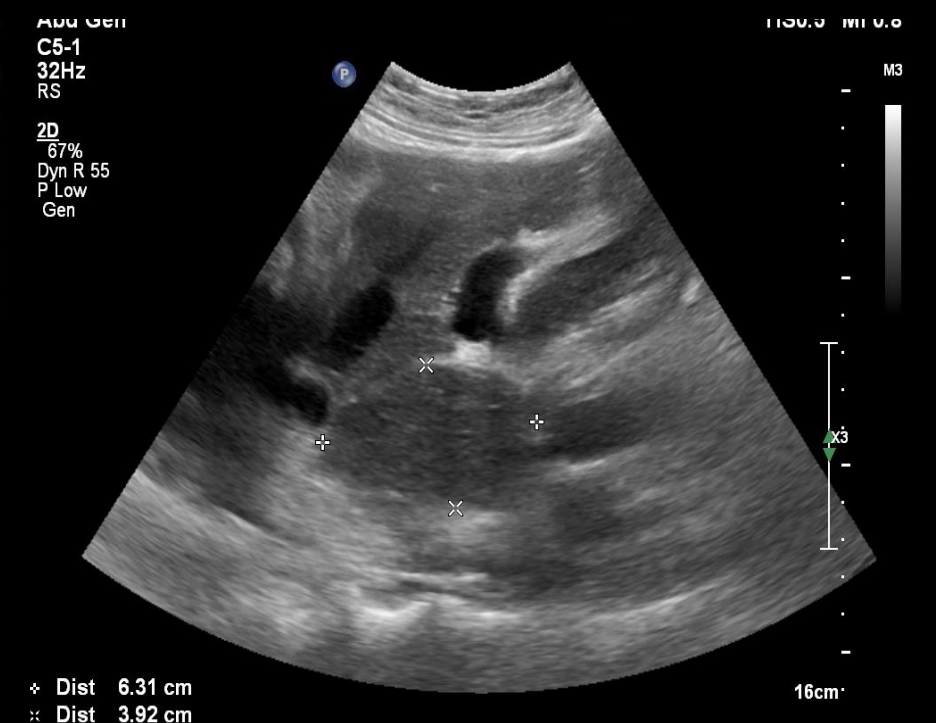
Eco-color Doppler:

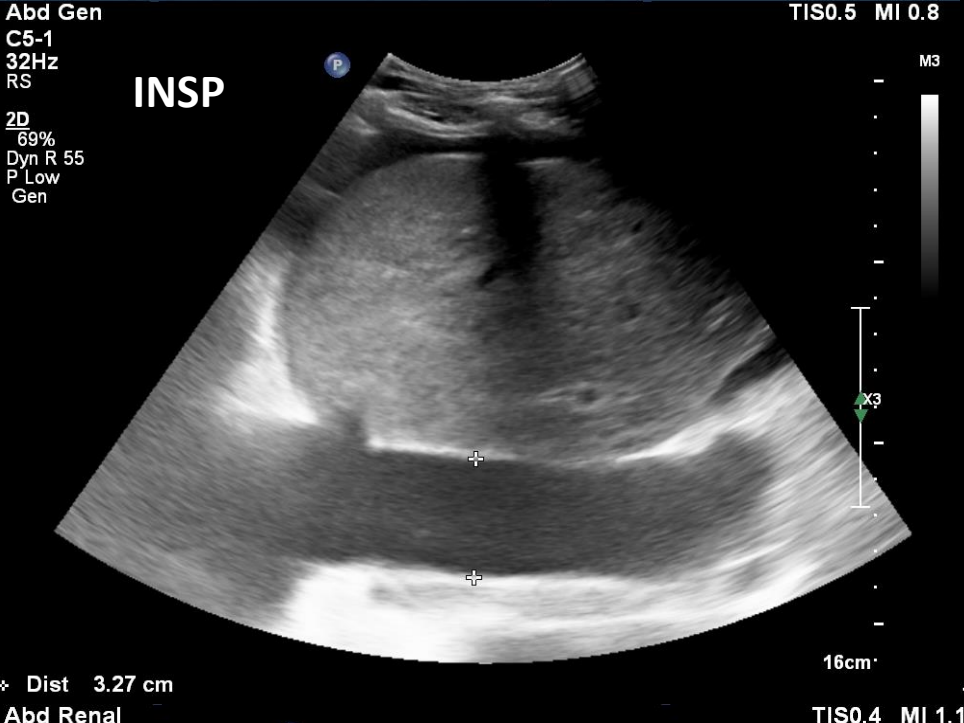
- **Portal Vein**:
  - ✓Phasic flow
  - ✓Hepatofugal flow
  - ✓Flow velocity (TAPV) < 20-24 cm/sec
- **Hepatic artery:** RI > 0,70
- **Hepatic Veins:**
  - ✓Quadriphasic Doppler waveform
- **Other vessels:**
  - ✓Umbilical vein recanalization
  - ✓Collaterals vessels/varices

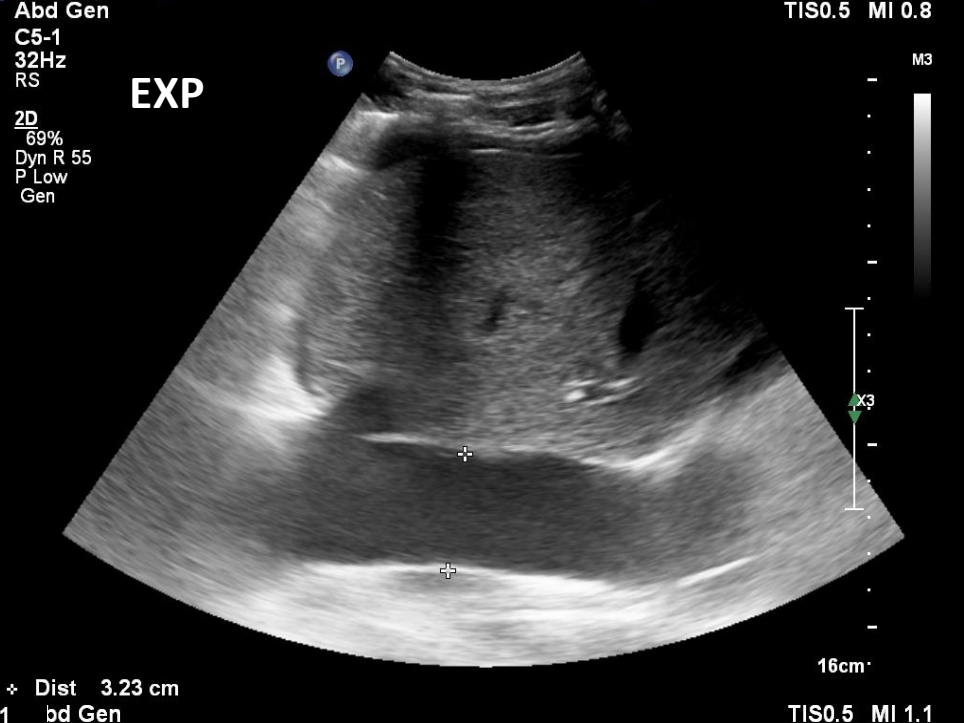

Abd Gen
C5-1
32Hz
RS
2D
60%
Dyn R 55
P Low
Gen
TIS0.5 MI 0.8
M3
16cm

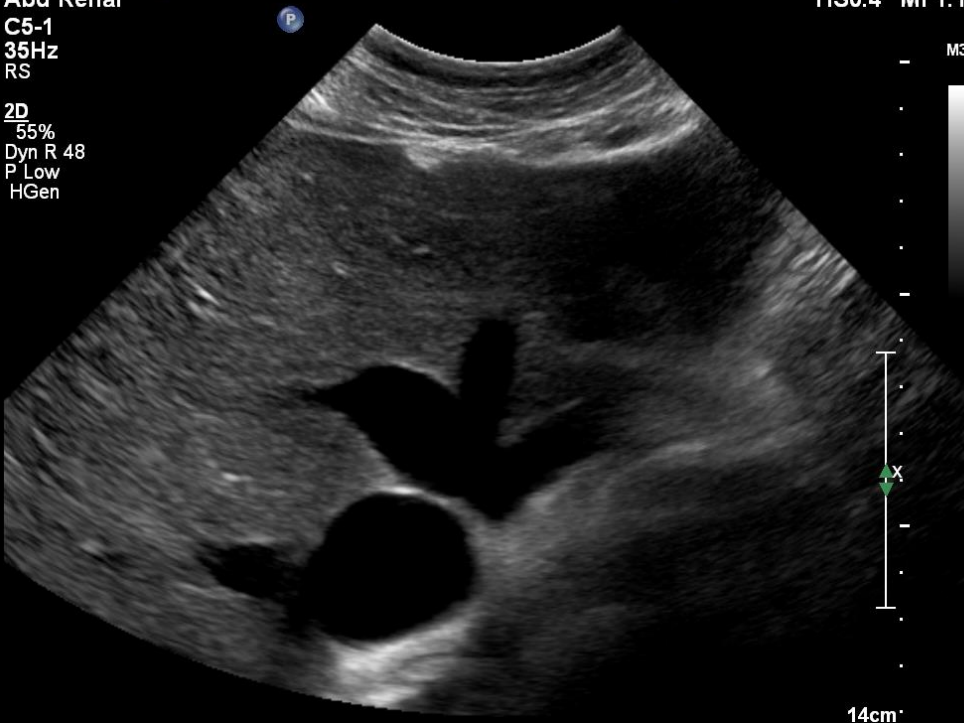

Abd Gen
C5-1
32Hz
RS
2D
69%
Dyn R 55
P Low
Gen
TIS0.5 MI 0.8
M3
16cm

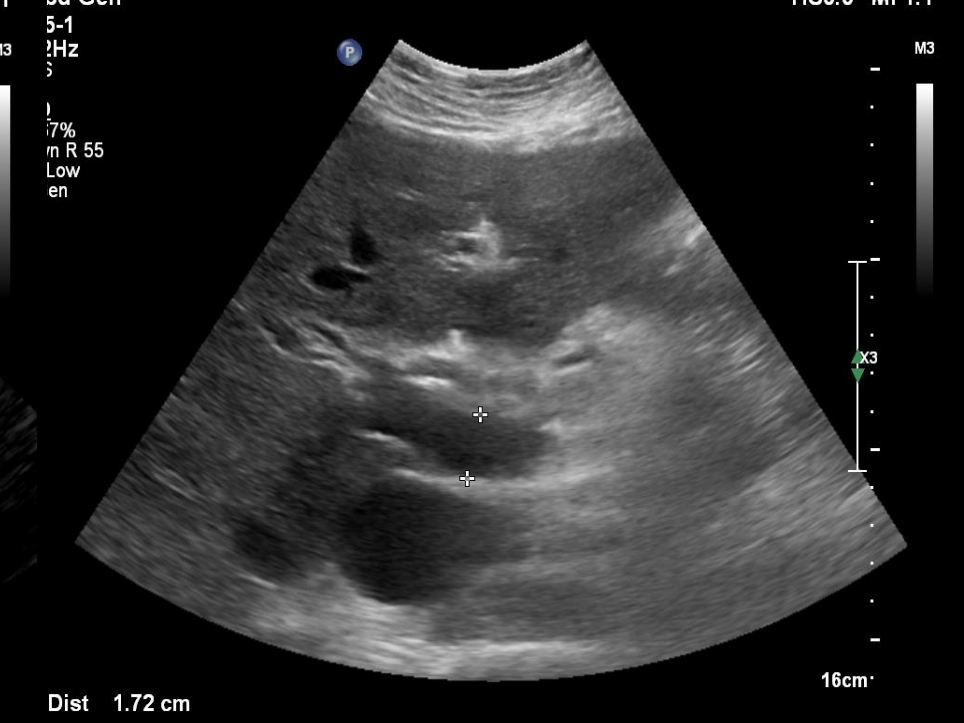

Superfic
L12-5
39Hz
RS
2D
37%
Dyn R 58
P Med
Res
TIS0.0 MI 0.7
M3
3.5cm

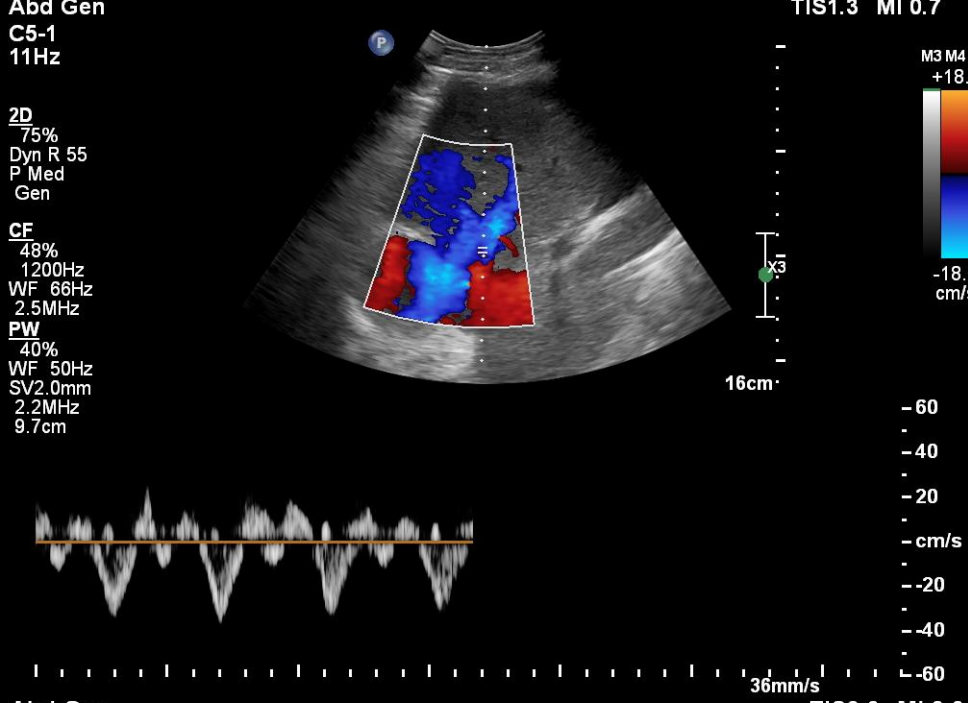

C5-1
32Hz
RS
2D
67%
Dyn R 55
P Low
Gen
M3
16cm
Dist 6.31 cm
Dist 3.92 cm

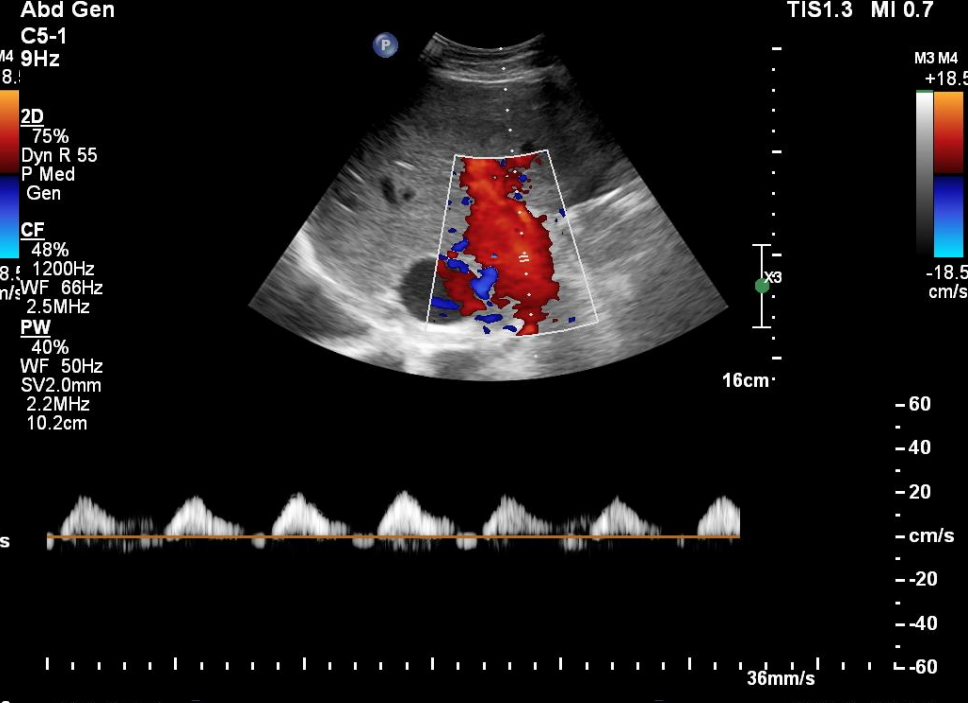

Abd Gen
C5-1
32Hz
RS
2D
69%
Dyn R 55
P Low
Gen
TIS0.5 MI 0.8
INSP
M3
X3
16cm
Dist 3.27 cm

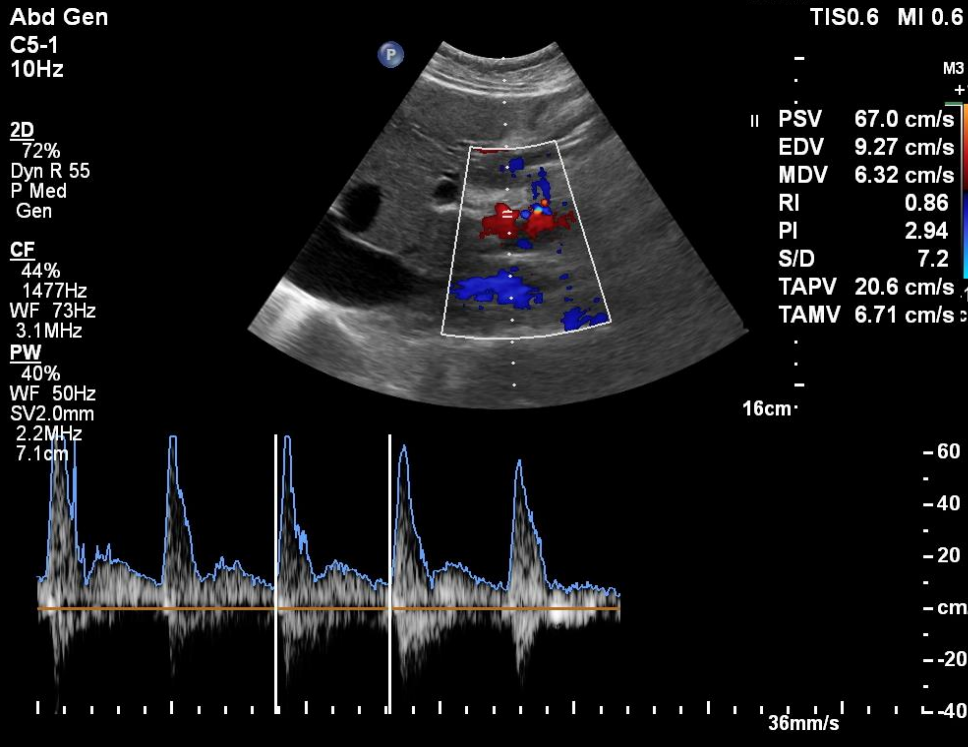

Abd Gen
C5-1
32Hz
RS
2D
69%
Dyn R 55
P Low
Gen
TIS0.5 MI 0.8
EXP
M3
X3
16cm
Dist 3.23 cm

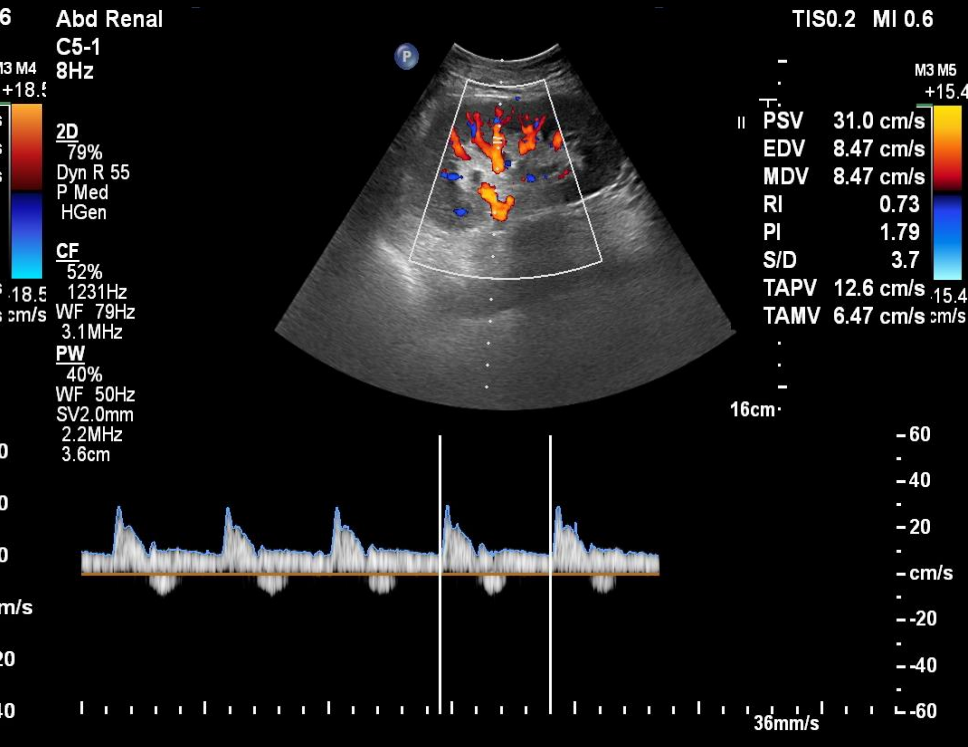

Abd Renal
C5-1
35Hz
RS
2D
55%
Dyn R 48
P Low
HGen
TIS0.4 MI 1.1
M3
X
14cm

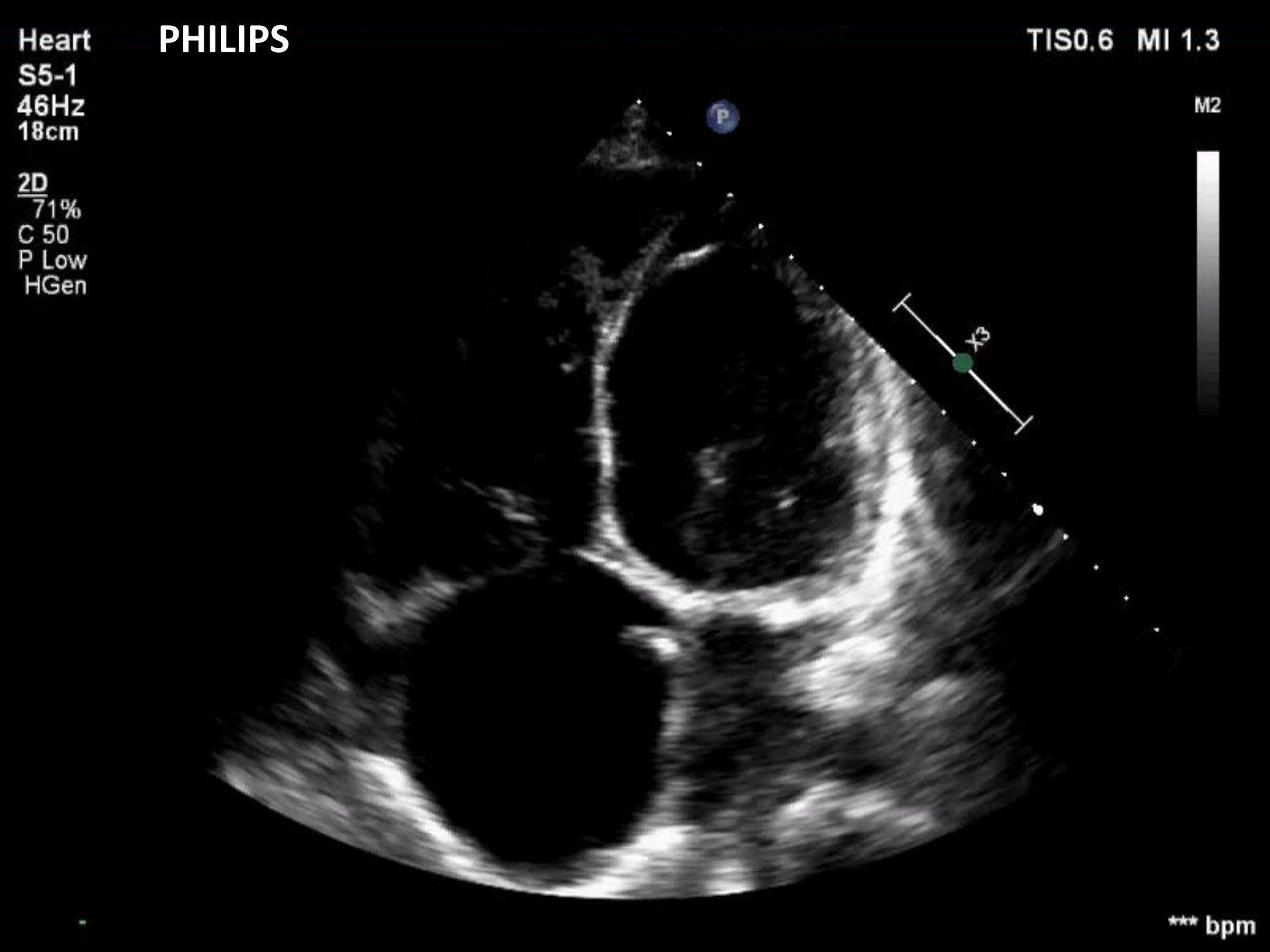

TIS0.5 MI 1.1
M3
X3
16cm
Dist 1.72 cm

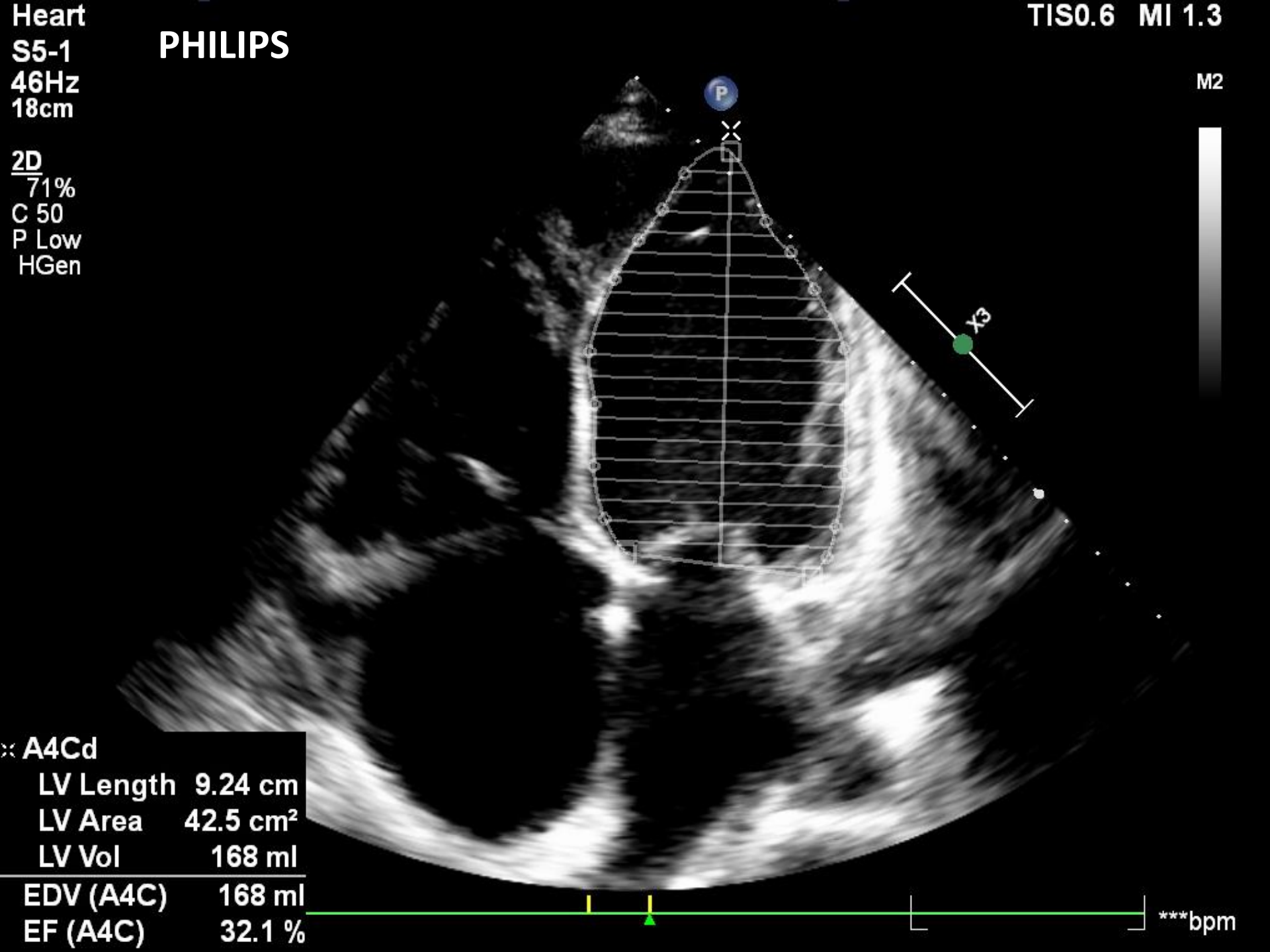

Abd Gen
C5-1
11Hz
TIS1.3 MI 0.7
2D
75%
Dyn R 55
P Med
Gen
CF
48%
1200Hz
WF 66Hz
2.5MHz
PW
40%
WF 50Hz
SV2.0mm
2.2MHz
9.7cm
M3 M4
16cm
60
40
20
cm/s
-20
-40
-60
36mm/s


Abd Gen
C5-1
9Hz
TIS1.3 MI 0.7
2D
75%
Dyn R 55
P Med
Gen
CF
48%
1200Hz
WF 66Hz
2.5MHz
PW
40%
WF 50Hz
SV2.0mm
2.2MHz
10.2cm
M3 M4
16cm
60
40
20
cm/s
-20
-40
-60
36mm/s


Abd Gen
C5-1
10Hz
TIS0.6 MI 0.6
2D
72%
Dyn R 55
P Med
Gen
CF
44%
1477Hz
WF 73Hz
3.1MHz
PW
40%
WF 50Hz
SV2.0mm
2.2MHz
7.1cm
PSV 67.0 cm/s
EDV 9.27 cm/s
MDV 6.32 cm/s
RI 0.86
PI 2.94
S/D 7.2
TAPV 20.6 cm/s
TAMV 6.71 cm/s
M3 M4
16cm
60
40
20
cm/s
-20
-40
36mm/s


Abd Renal
C5-1
8Hz
TIS0.2 MI 0.6
2D
79%
Dyn R 55
P Med
HGen
CF
52%
1231Hz
WF 79Hz
3.1MHz
PW
40%
WF 50Hz
SV2.0mm
2.2MHz
3.6cm
PSV 31.0 cm/s
EDV 8.47 cm/s
MDV 8.47 cm/s
RI 0.73
PI 1.79
S/D 3.7
TAPV 12.6 cm/s
TAMV 6.47 cm/s
M3 M5
16cm
60
40
20
cm/s
-20
-40
-60
36mm/s

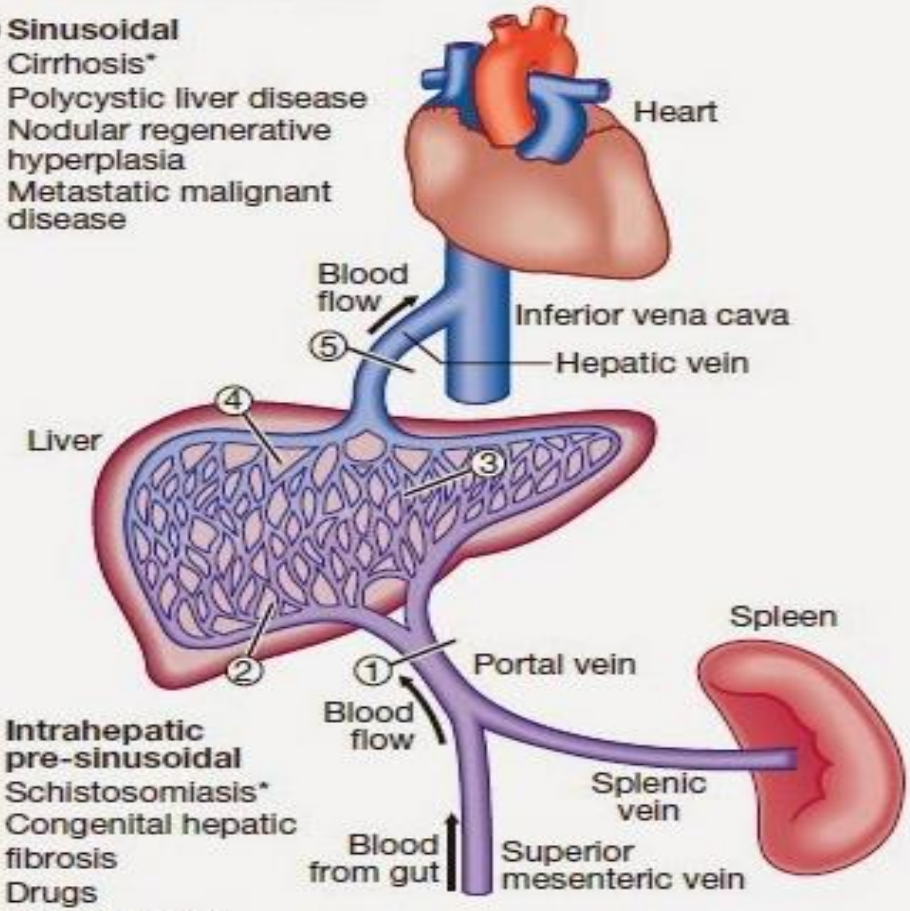

Heart
S5-1
46Hz
18cm
2D
71%
C 50
P Low
HGen
PHILIPS
TIS0.6 MI 1.3
M2
P
x3
*** bpm


Heart
S5-1
46Hz
18cm
2D
71%
C 50
P Low
HGen
PHILIPS
TIS0.6 MI 1.3
M2
P
X3
A4Cd
LV Length 9.24 cm
LV Area 42.5 cm²
LV Vol 168 ml
EDV (A4C) 168 ml
EF (A4C) 32.1 %
***bpm

⑤ **Post-hepatic post-sinusoidal**
Budd–Chiari syndrome

④ **Intrahepatic post-sinusoidal**
Veno-occlusive disease

③ **Sinusoidal**
Cirrhosis*
Polycystic liver disease
Nodular regenerative hyperplasia
Metastatic malignant disease



② **Intrahepatic pre-sinusoidal**
Schistosomiasis*
Congenital hepatic fibrosis
Drugs
Vinyl chloride
Sarcoidosis

① **Prehepatic pre-sinusoidal**
Portal vein thrombosis due to sepsis (umbilical, portal pyaemia) or procoagulopathy or secondary to cirrhosis
Abdominal trauma including surgery

Fig. 23.19 Classification of portal hypertension according to site of vascular obstruction. *Most common cause. Note that splenic vein occlusion can also follow pancreatitis, leading to gastric varices.

1. Pre-hepatic pre-sinusoidal

2. Intra-hepatic pre-sinusoidal

3. Intra-hepatic post-sinusoidal

Cirrhosis

Budd-Chiari syndrome (HVs thrombosis)

4. Intra-hepatic post-sinusoidal

5. Post-hepatic post-sinusoidal

Budd-Chiari Syndrome

In the acute form of BCS

Eco-color Doppler:

- Lack of visualization of one or more hepatic veins at the color-Doppler

B-mode US:

- A thrombus filling the vein, vein stenosis, or a tumor invading or compressing the veins.

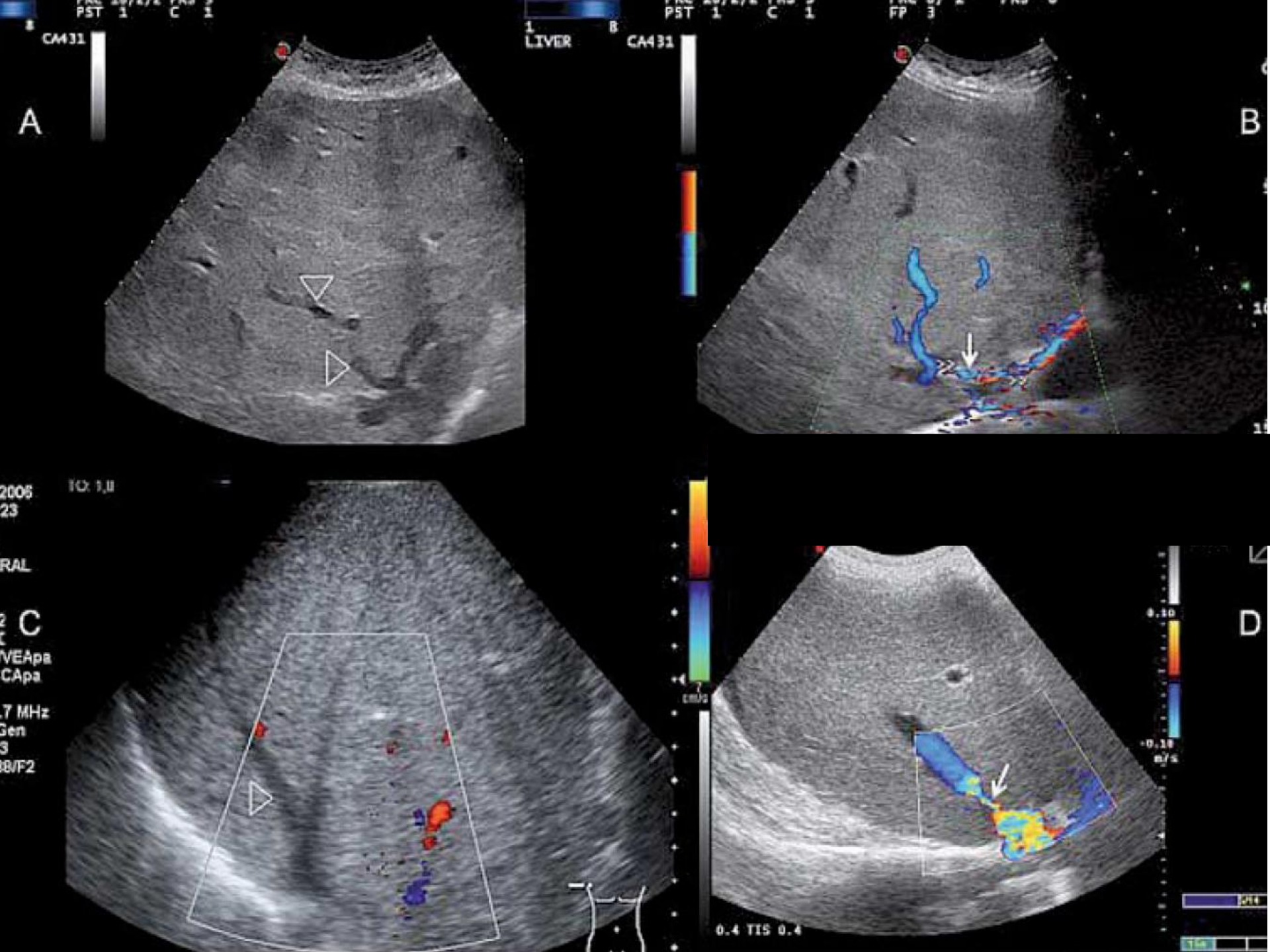
In subacute and chronic forms of BCS

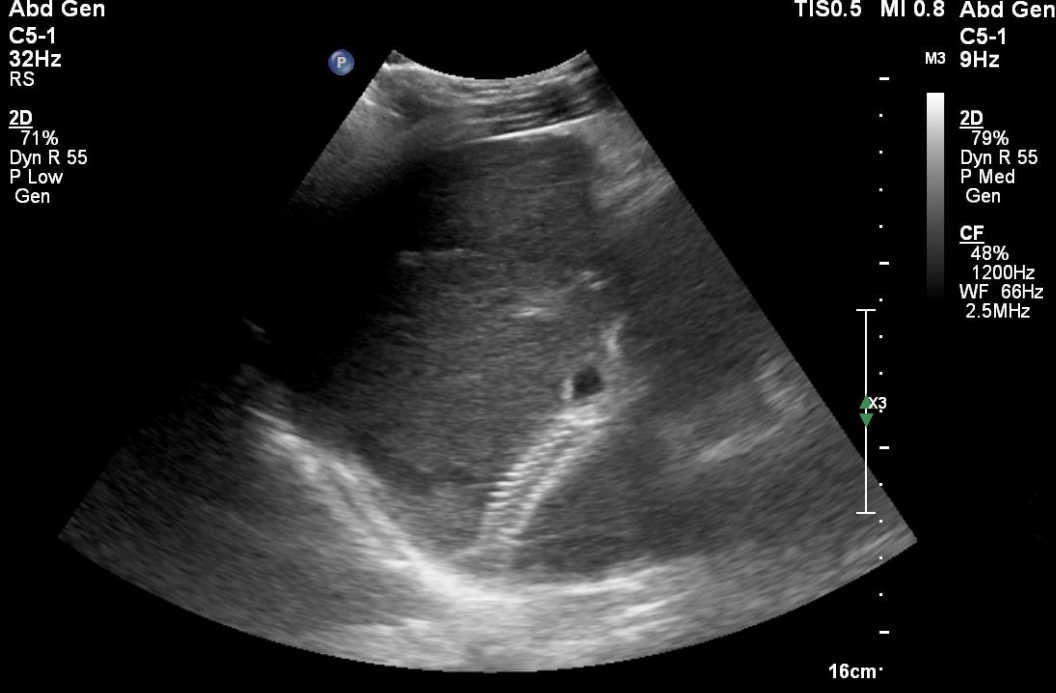
Eco-color Doppler:

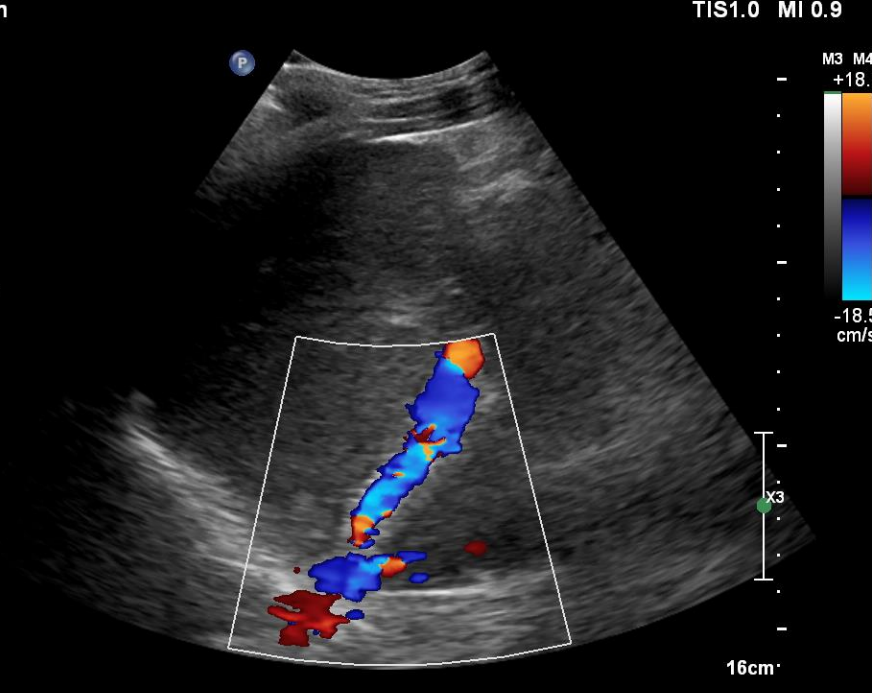
- A fragmented vein with flow reversal, or new venous vessels that drain sub-capsular circulation to another hepatic vein or directly to the inferior vena cava

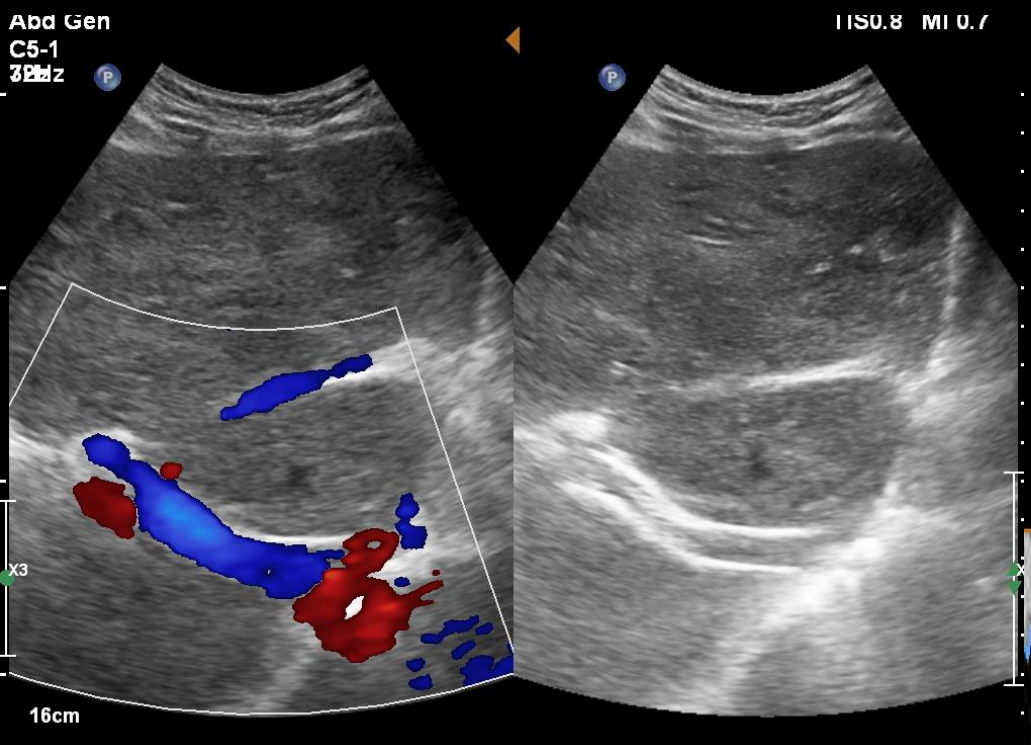
B-mode US:

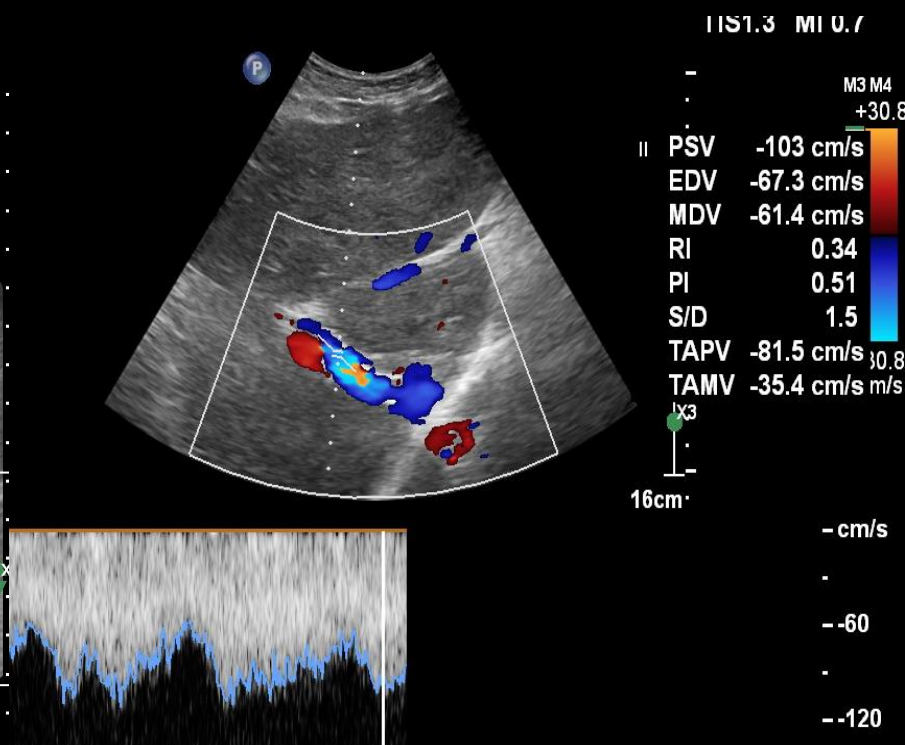
- A fibrous tract replacing the obstructed hepatic veins
- Caudate lobe and caudate vein hypertrophy
- Enlarged liver with grossly heterogeneous echo-texture


A
B
C
D
LIVER
0.4 TIS 0.4


Abd Gen
C5-1
32Hz
RS
2D
71%
Dyn R 55
P Low
Gen
TIS0.5 MI 0.8
Abd Gen
C5-1
9Hz
M3
2D
79%
Dyn R 55
P Med
Gen
CF
48%
1200Hz
WF 66Hz
2.5MHz
X3
16cm


TIS1.0 MI 0.9
M3 M4
+18.
-18.
cm/s
X3
16cm


Abd Gen
C5-1
TIS0.8 MI 0.7
X3
16cm


TIS1.3 MI 0.7
M3 M4
+30.8
PSV -103 cm/s
EDV -67.3 cm/s
MDV -61.4 cm/s
RI 0.34
PI 0.51
S/D 1.5
TAPV -81.5 cm/s
TAMV -35.4 cm/s
X3
16cm
cm/s
-60
-120

Limitations of US examination in this field

- **Abdominal air interposition** which may prevent correct and complete visualization of the abdominal organs and vessels.
- **Massive ascites** also impairs the imaging of the liver and abdominal vessels.
- A well recognized limitation of quantitative Doppler measurements is the **inter-equipment and inter-observer variability** which reduces the comparability of this data among different centers.
- Patients in follow-up should preferably be examined by the same operator and with the same equipment whenever possible.
- Cooperative studies have shown that it is possible to reduce inter-observer variability by using a standardized protocol of examination

THANK YOU