INSELSPITAL
UNIVERSITÄTSSPITAL BERN
HOPITAL UNIVERSITAIRE DE BERNE

UNIVERSITÄT BERN

Ultrasound assessment of portal hypertension

Annalisa Berzigotti

Department for Visceral Surgery and Medicine
Inselspital, Bern University Hospital,
University of Bern
Switzerland

Outline

- Basic concepts about portal hypertension
- Indications for US in PH
- Signs of PH on US examination
  - B-mode, CDUS and PWUS signs
  - Correlations with clinical aspects in cirrhosis

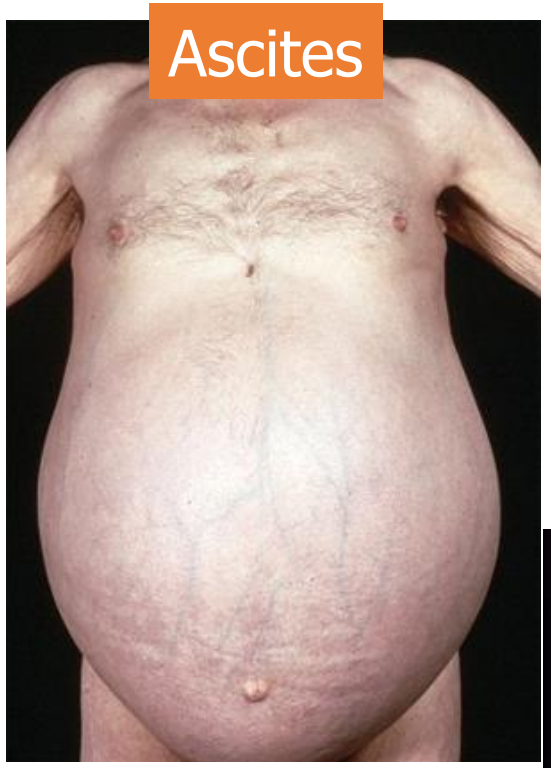
Portal hypertension

Clinical syndrome hemodynamically defined
by the increase in the
portal pressure gradient above normal values
(normal PPG ≤ 5 mmHg)

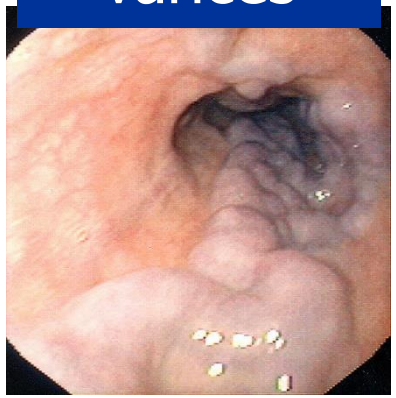
Clinically defined by its complications

Clinical features

Ascites

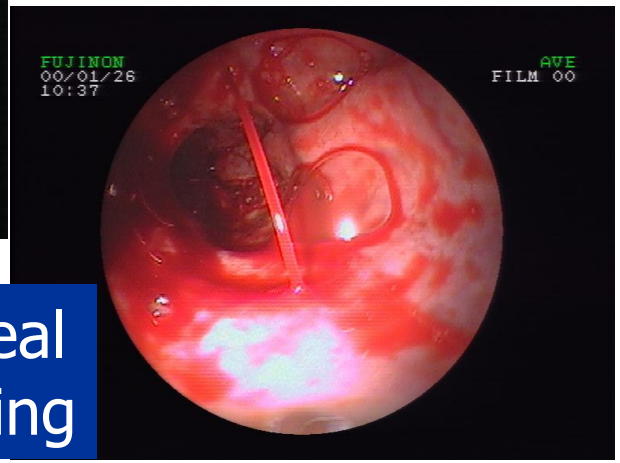


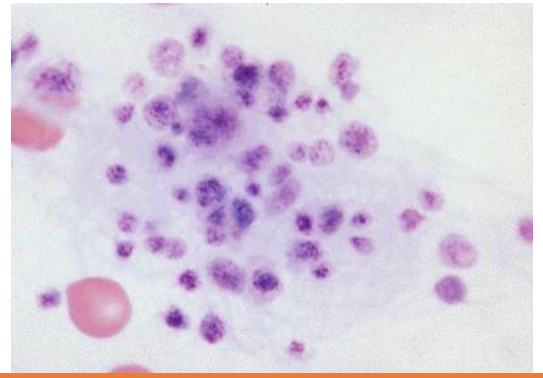
Esophageal varices



Porto-systemic shunting
HE

Variceal bleeding

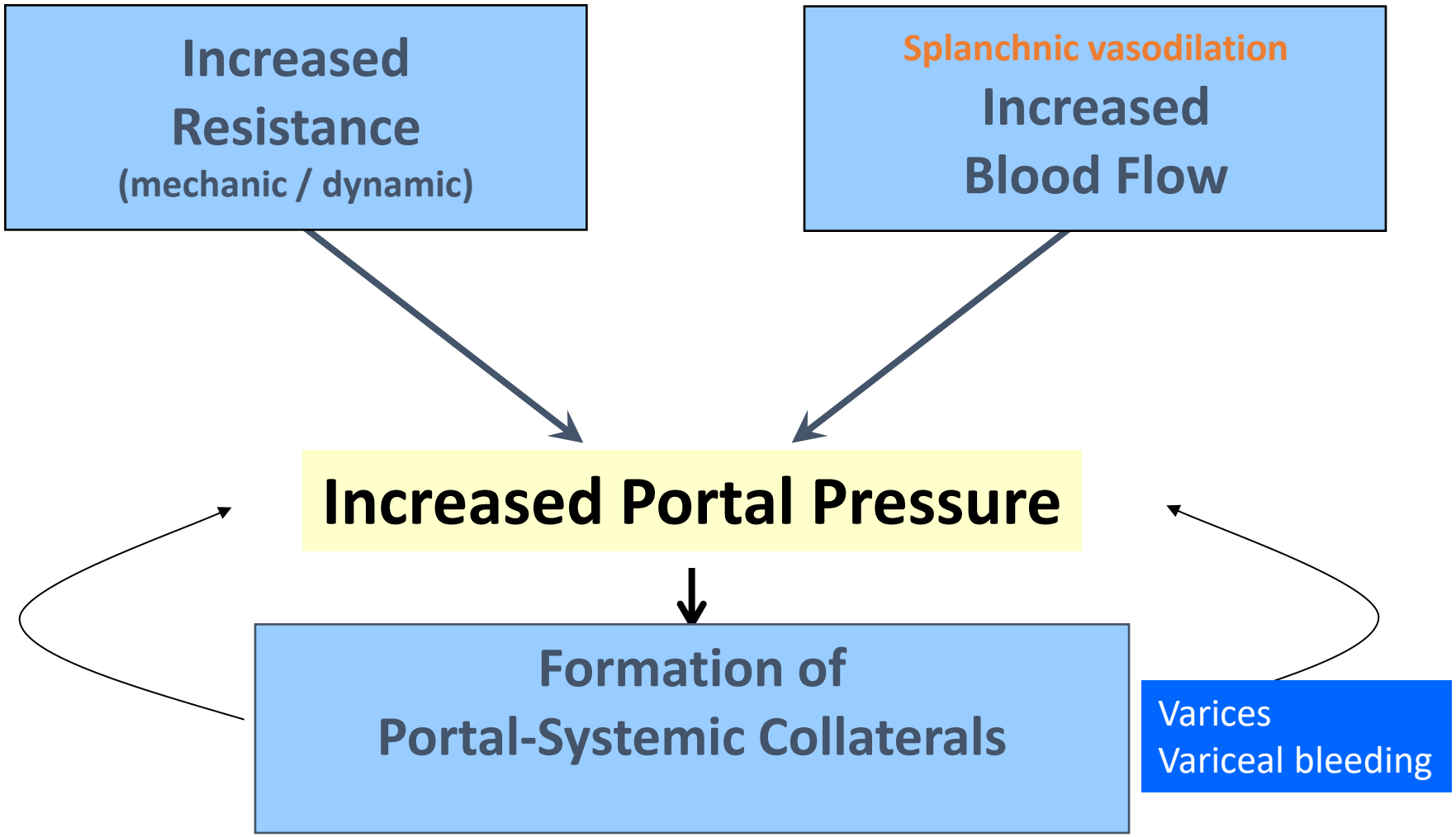




Hypersplenism

Pathophysiology of PH

Δ Portal Pressure = Resistance x Blood Flow



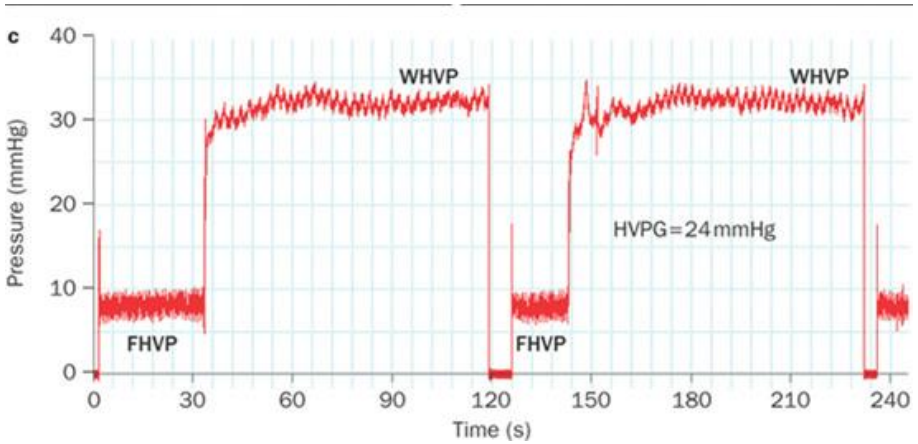
Diagnosis of PH

Clinical features (e.g. hypersplenism, ascites, collaterals)
Low Sensitivity

Anatomic changes

IMAGING **Elastography**

Increase of portal pressure
Gold-standard but Invasive



See Giovanna's and my talk tomorrow

Imaging of PH: US is the first-line approach

Assessment of parenchymas + vessels

Quantitative data

Repeatable

Real-time

Cheap

Safe

"Point-of-care testing"

CLÍNIC BARCELONA Hospital Universitari

Outline

- Basic concepts about portal hypertension
- Indications for US in PH
- Signs of PH on US examination
  - B-mode, CDUS and PWUS signs
  - Correlations with clinical aspects in cirrhosis

Indications for US in PH

- Evaluation of patients with clinical/US/laboratory signs of portal hypertension and no chronic liver diseases

  → CLASSIFICATION OF PORTAL HYPERTENSION

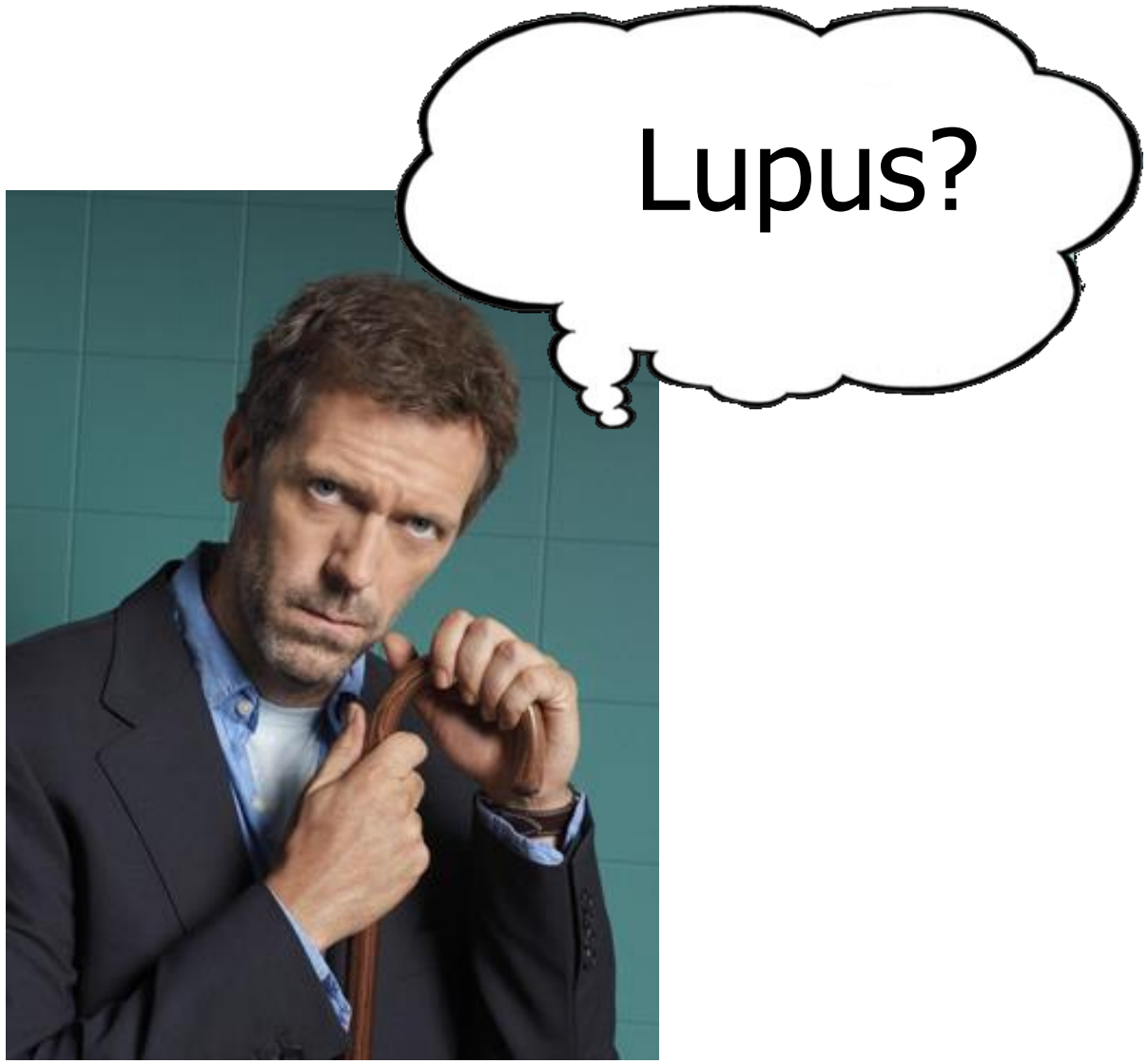
- Screening of signs of PH in patients with known compensated chronic liver disease/cirrhosis

- Prognostic assessment in cirrhosis

Pre-test probability in medicine

My patient has portal hypertension…

… what is causing it?



Classification of Portal hypertension

Based on the site of increased resistance to portal flow PH can be classified as:

	Main cause
Pre-hepatic	Portal vein thrombosis
Intra-hepatic	Cirrhosis
Post-hepatic	Hepatic veins thrombosis (Budd-Chiari syndrome)

US in suspected portal hypertension

Main role of US:

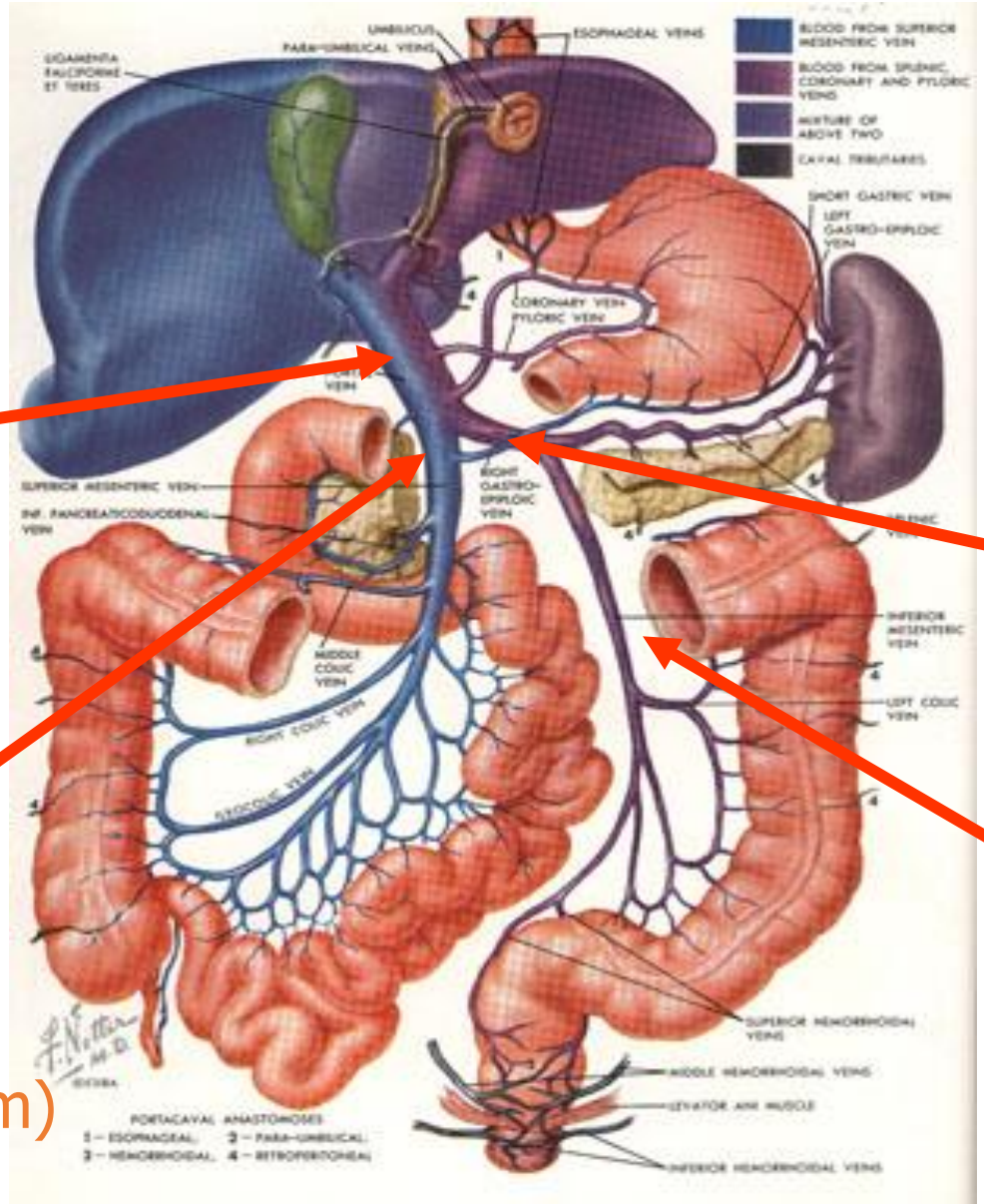
- Rule out thrombosis: check portal venous system patency and hepatic veins patency

- Identify signs of cirrhosis

- Look for rarer causes

Portal venous system

Portal vein
Normal
6-12 mm

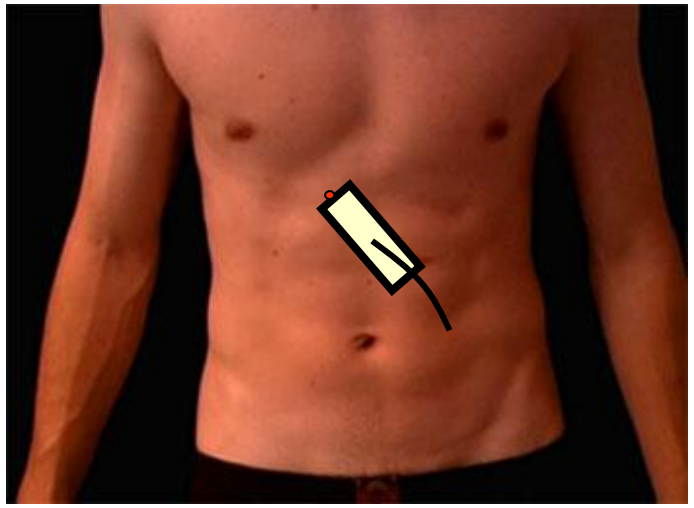


Splenic Vein
(<10 mm)

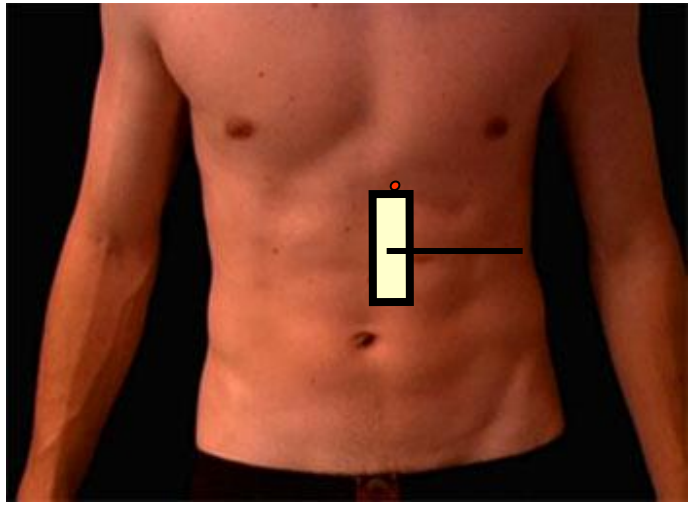
Superior Mesenteric Vein (< 10 mm)

Inferior Mesenteric Vein (< 10 mm)

Portal venous system: scanning technique



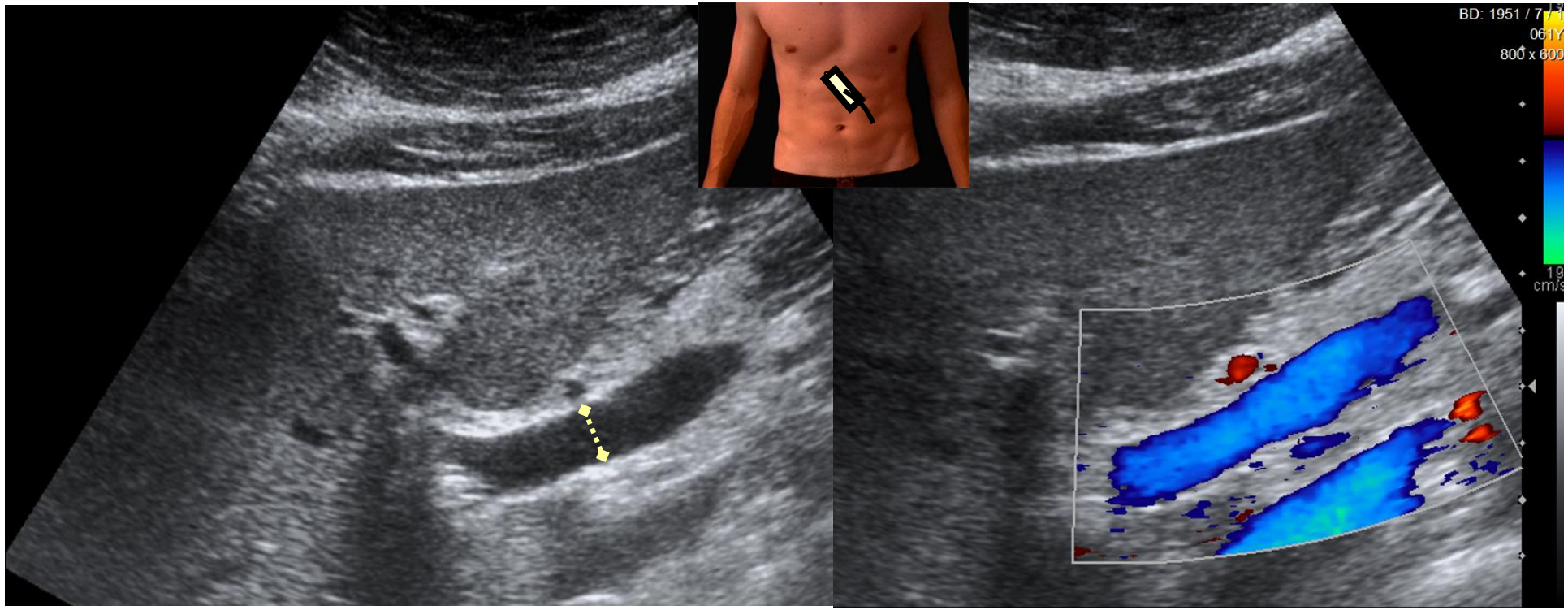
Portal vein: oblique-transversal scan in epigastrium/right subcostal region to visualize PV along its longitudinal axis for at least 3 – 4 cm



Splenic vein and SV-PV confluence: transversal scan in epigastrium to visualize SV along its longitudinal axis

Superior mes. vein and SMV-PV confluence: longitudinal scan in epigastrium to visualize SMV along its longitudinal axis

Normal extrahepatic portal vein



Portal vein: anechoic; measure the diameter in B-mode between the two inner walls; best at the crossing with hepatic artery or slightly downstream

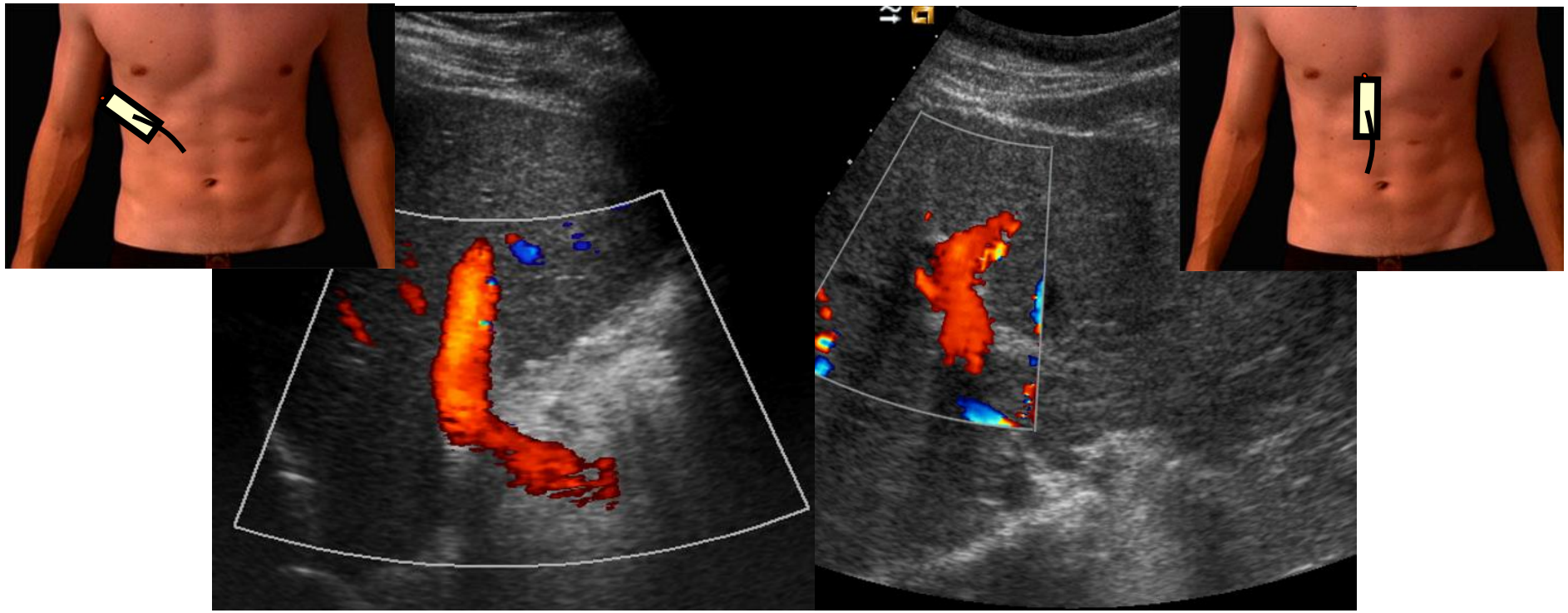
CDUS: hepatopetal flow (away from the probe- blue in this scan)

Normal intrahepatic portal vein

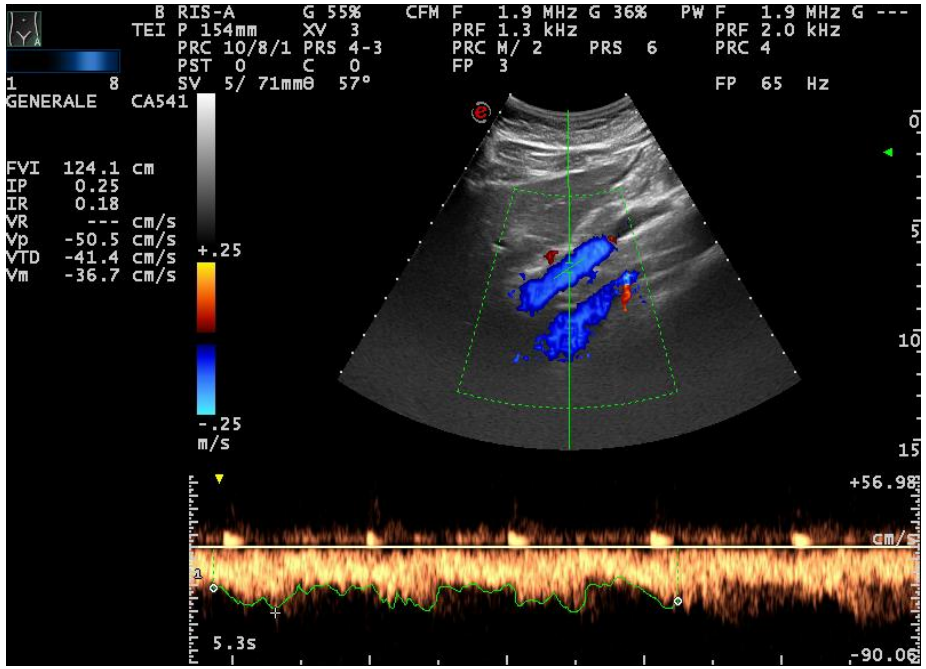
Structures to assess:

- PV at the hepatic hilium and right portal branch
- left portal branch

B-mode: anechoic; CDUS: hepatopetal flow (towards the probe-red in these scans)



Normal portal vein: Doppler flowmetry



Sample: 50% of the diameter of PV, in the middle of the lumen at the cross with hepatic artery

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 flow)

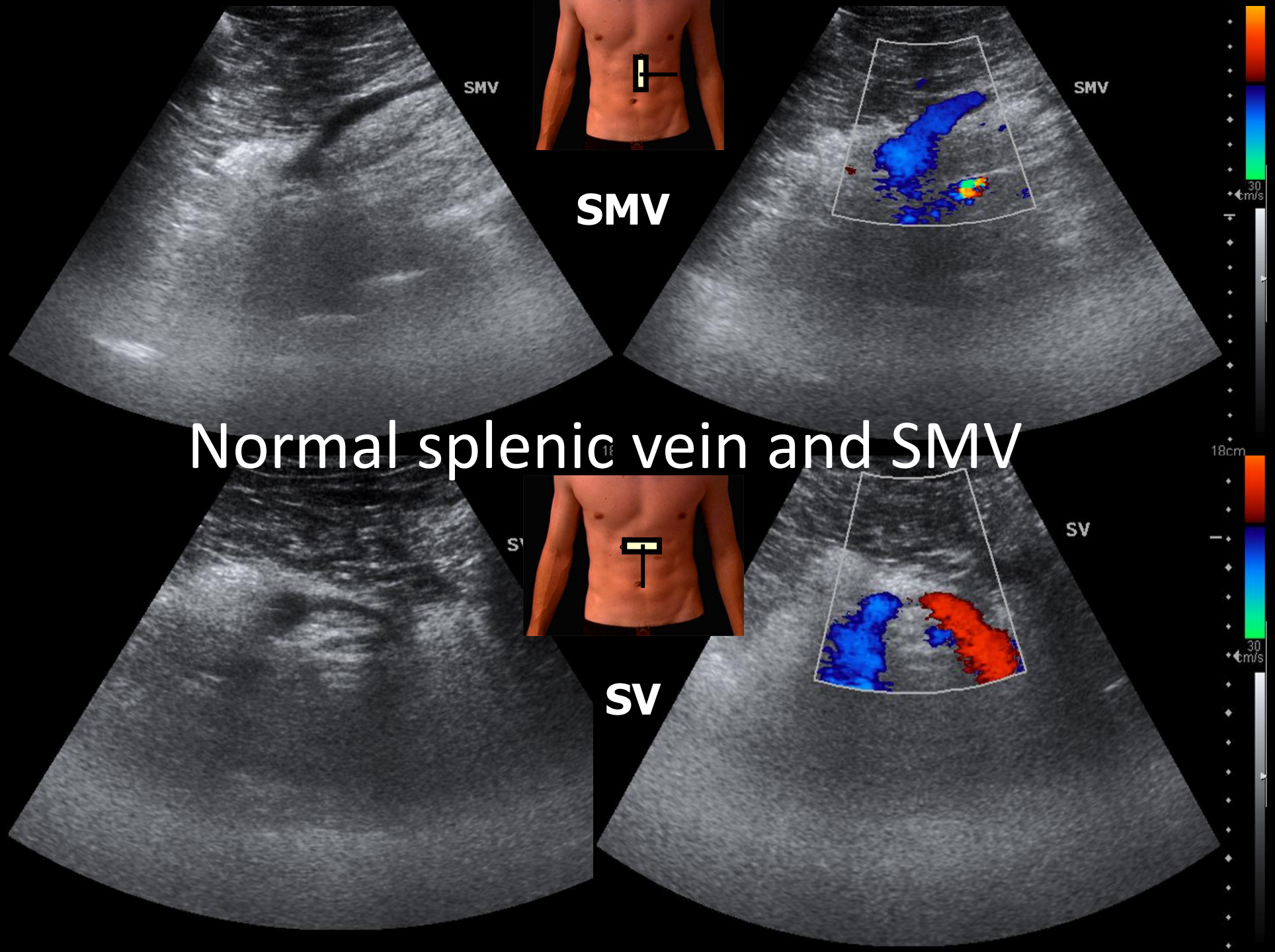
Doppler flowmetry tracings: If feasible use color Doppler image + Doppler flowmetry manual tracing of Doppler signal for at least 2 cardiac cycles or ≥ 2 – 3 seconds;

Normal time averaged maximum velocity (TAMv, automatic) > 20 – 24 cm/s

Mean velocity =TAMv *0.57

More details in:

Kruskal J B et al. Radiographics 2004
Berzigotti & Piscaglia Ultraschall Med 2012



Normal splenic vein and SMV

US in suspected portal hypertension

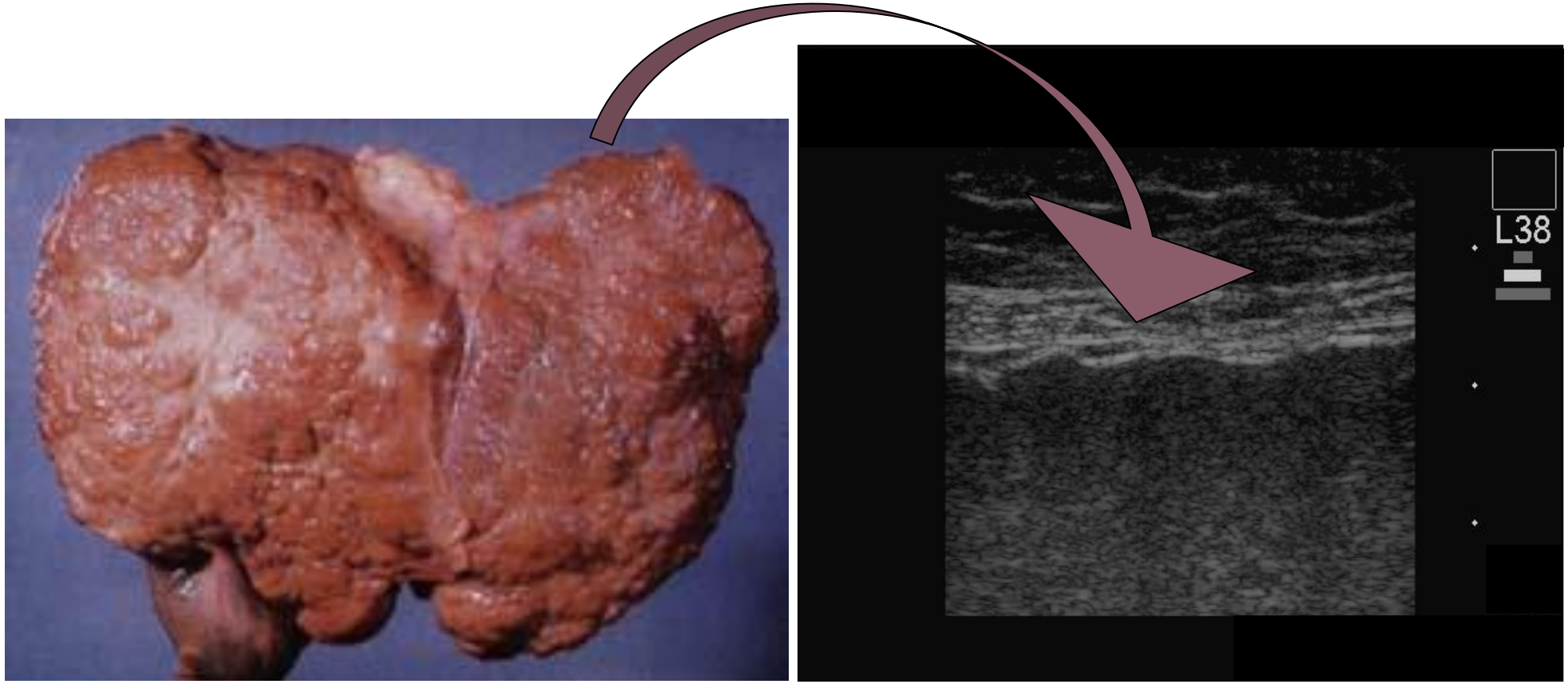
Main role of US is:

- Rule out thrombosis: check portal venous system and hepatic vein system patency

- Identify signs of cirrhosis (Dr. Rosselli)

- Look for rarer causes

High frequency US assessment of liver surface to diagnose cirrhosis in "difficult" patients



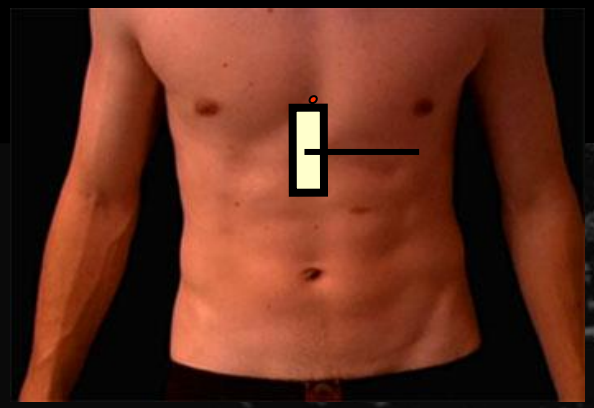
Nodular liver surface : +LR 11.15

Smooth liver surface: -LR 0.10

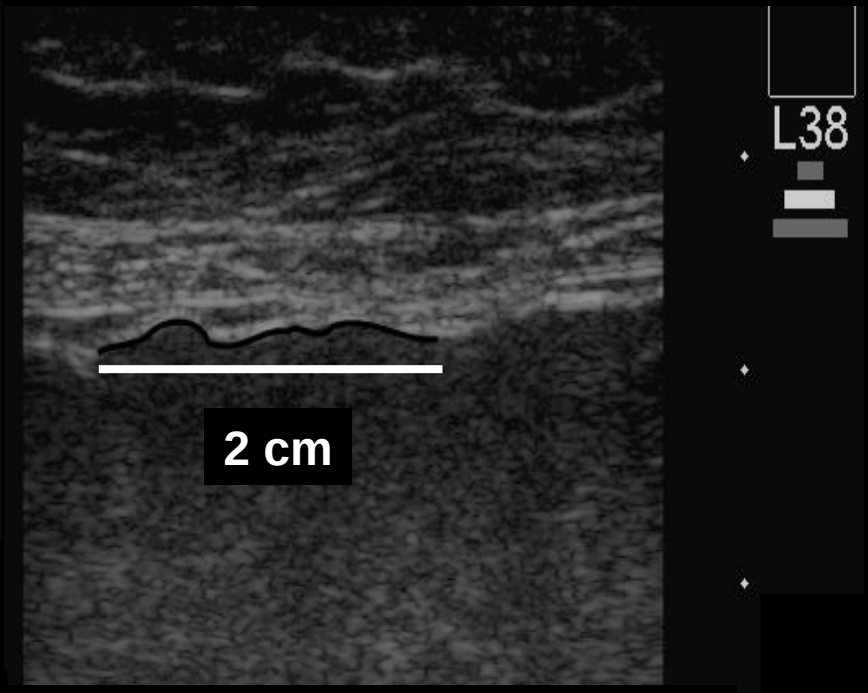
Berzigotti et al. J Hepatol 2010

Irregular (indeterminate) 35%

High frequency US assessment of liver surface to diagnose cirrhosis in "difficult" patients



Digital measurement 2.01 cm



Digital measurement: 2.11 cm

Berzigotti et al. J Hepatol 2010

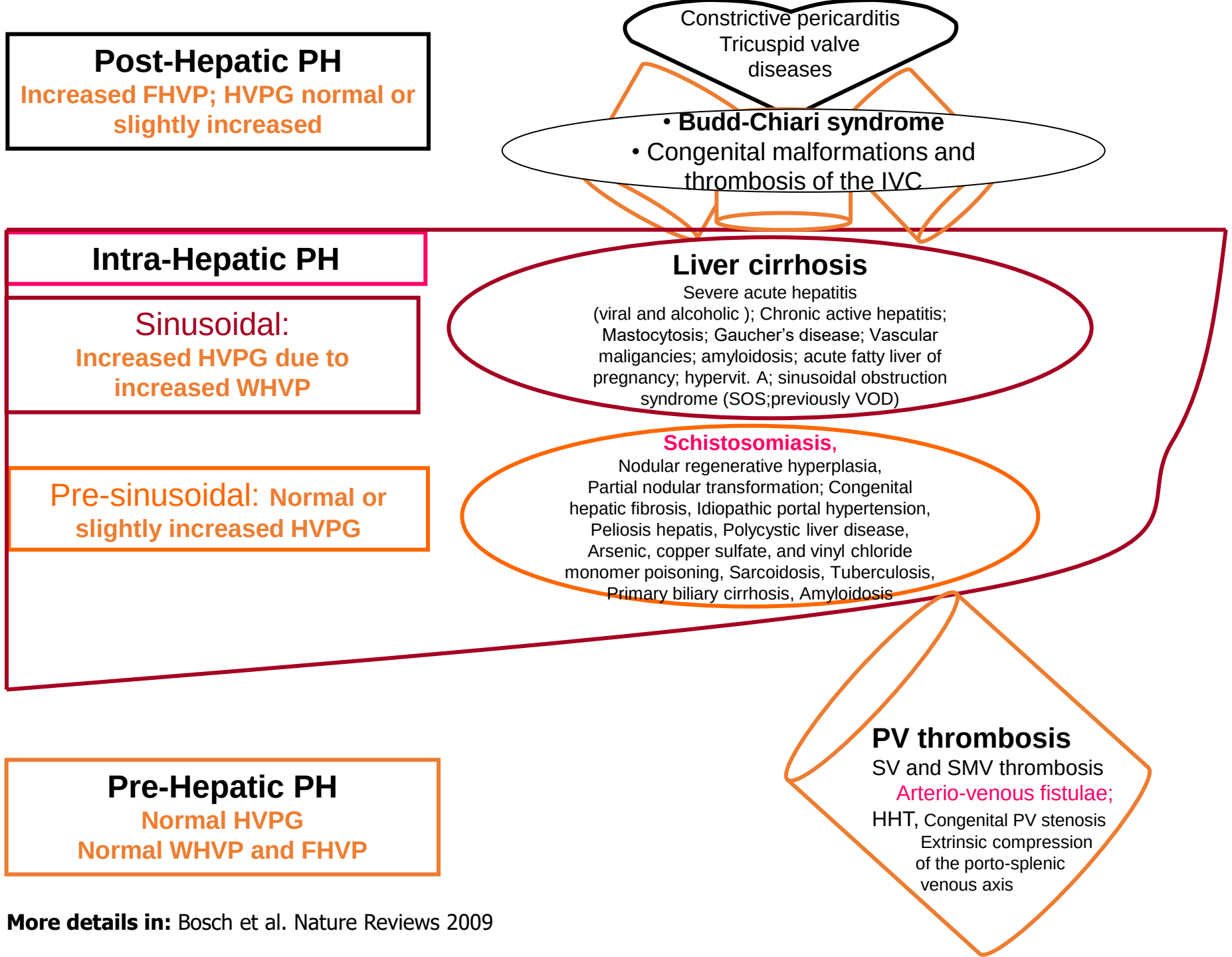
US in suspected portal hypertension

Main role of US is:

- Rule out thrombosis: check portal venous system and hepatic vein system patency

- Identify signs of cirrhosis

- Look for rarer causes



More details in: Bosch et al. Nature Reviews 2009

Artero-portal Fistula

Indications for US in PH

- Evaluation of patients with clinical/laboratory signs of portal hypertension, without known chronic liver diseases (or with basic sonographic signs of possible portal hypertension)
  → CLASSIFICATION OF PORTAL HYPERTENSION

- Screening of signs of PH in patients with known compensated chronic liver disease/cirrhosis

- Prognostic assessment in cirrhosis

Most important US signs of PH

		Sens	Spec
Portal venous system	Dilatation of portal vein (≥13 mm)	<50%	90-100%
	Reduction of portal vein blood flow velocity (TAMvel < 20 cm/s; mean vel < 14 cm/ s)	80-88%	80-96%
	Reversal	rted; sign e: 8.3% of cted pts	**100%**
	Increased	95%*	100%
	Dilatation of splenic vein (SV) and erior mesenteric vein (SMV) (≥11 mm)	72%	100%
	Reduction of respiratory variation ameter in SV or SMV (<40%)	79.7%	100%
Presence of porto-systemic collateral circulation		83%	**100%**
artery	hepatis (>0.78)		
Renal artery	Increased resistive index of the right interlobar renal artery (≥ 0.65)	79.5%*	59.3%
SMA	Decrased pulsatility index (≤ 2.70)	85.7%*	65.2%

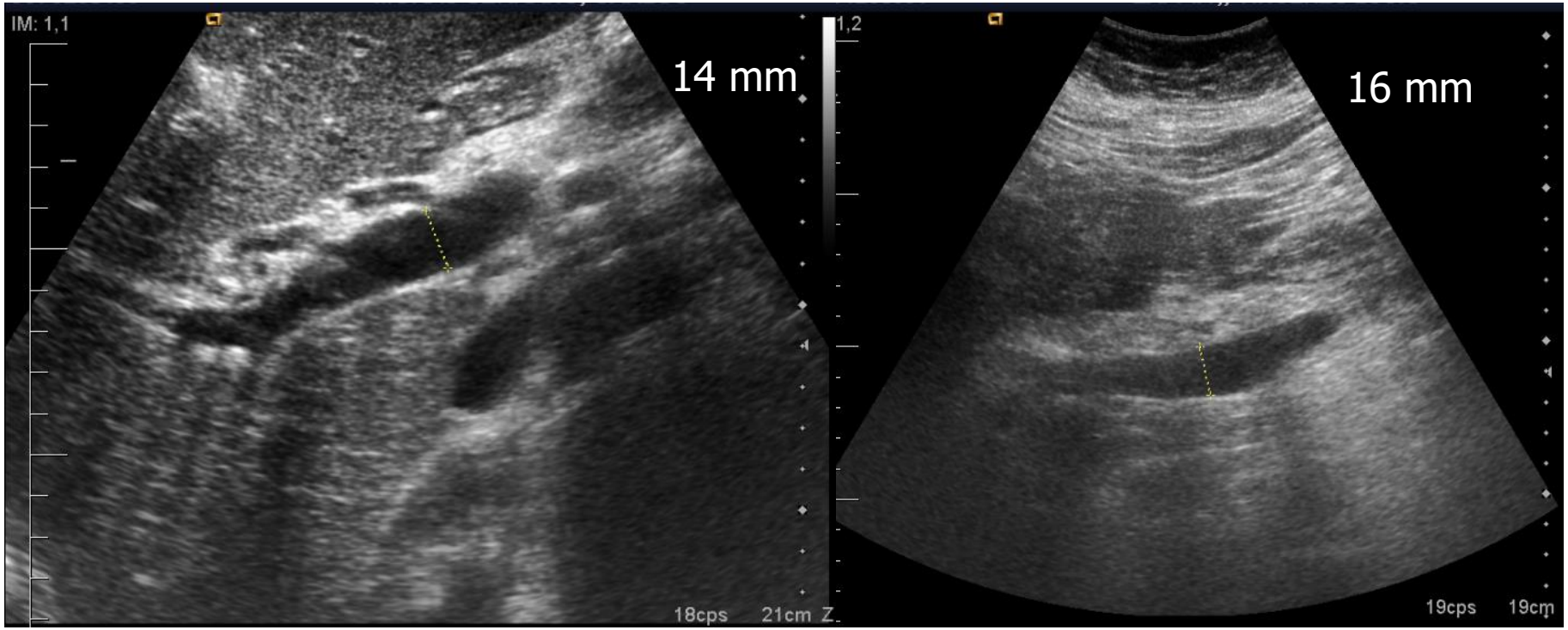
Moderate sensitivity
High specificity

Presence of a sign (or a combination of signs) allows a confident diagnosis, while in the absence of signs the diagnosis cannot be excluded

* Study using as a gold-standard HVPG measurement

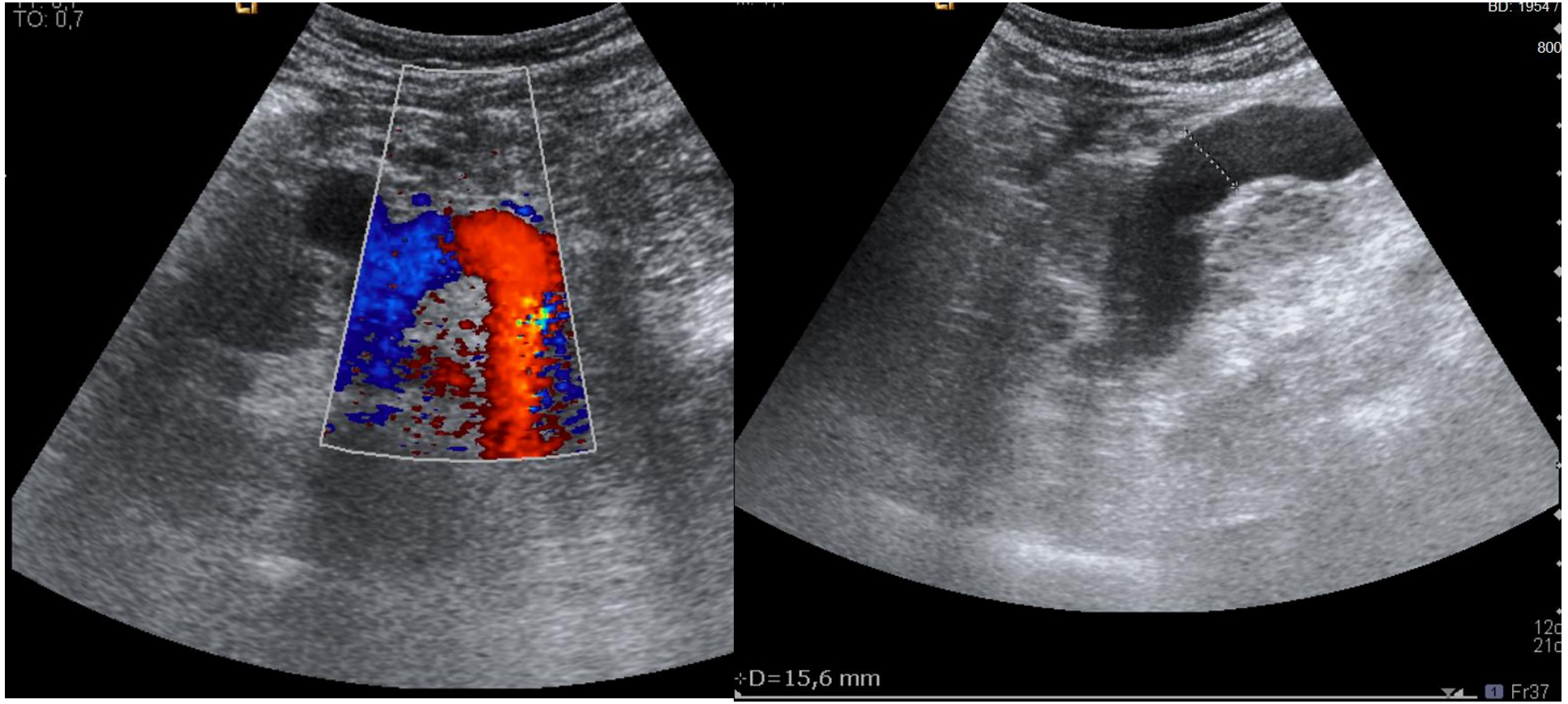
Enlargement of PV in PH

PV (and SV/SMV) diameter increases as a consequence of PH



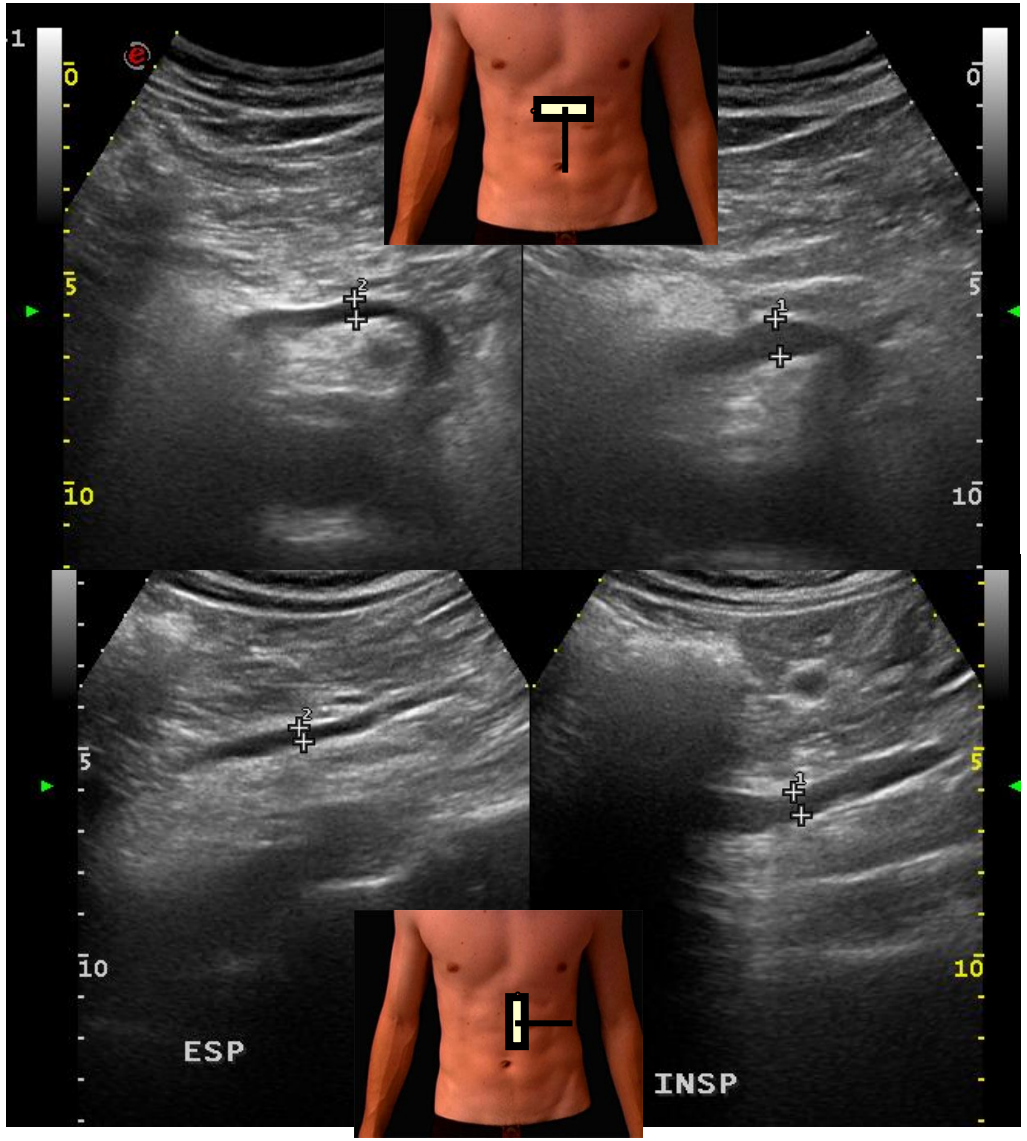
Normal PV < 13 mm

SV and SMV diameter in PH

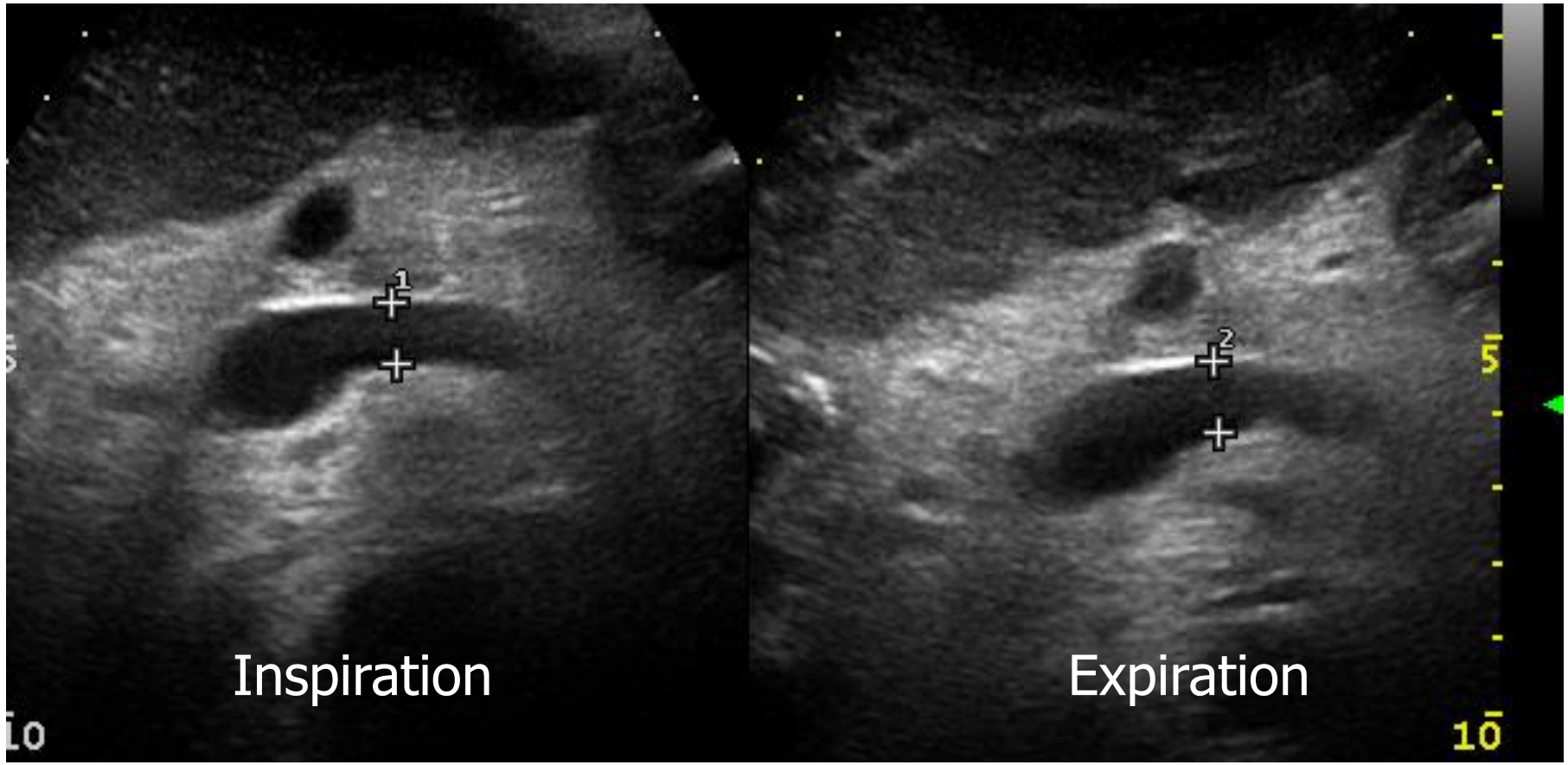


Normal respiratory variation of the diameter of splenic vein and SMV

Normal ≥ 40%



Lack of respiratory variation of SV and SMV

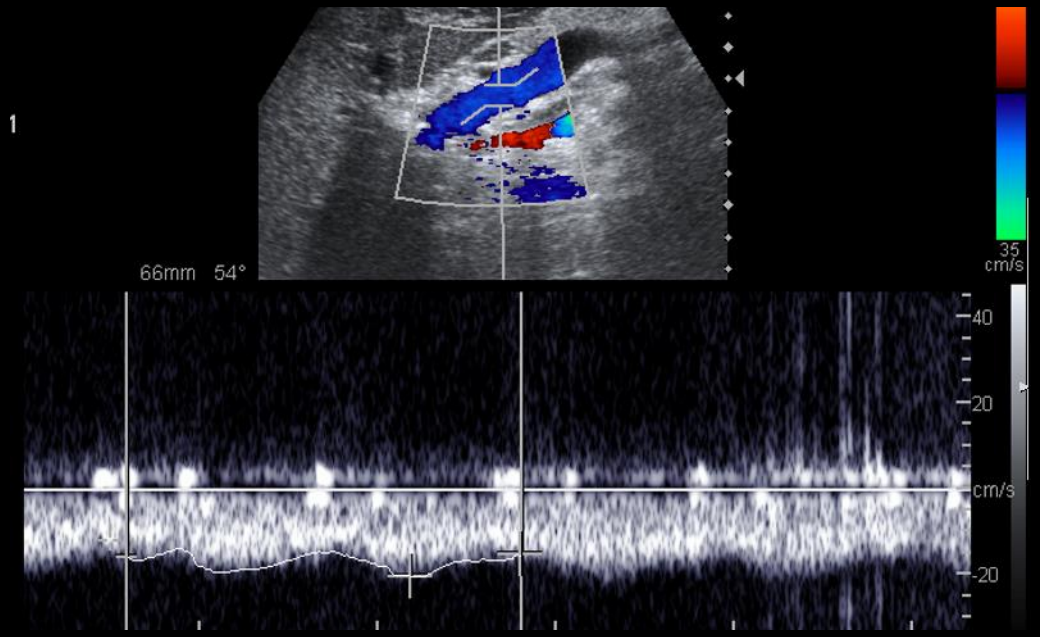


Bolondi et al. Radiology 1982

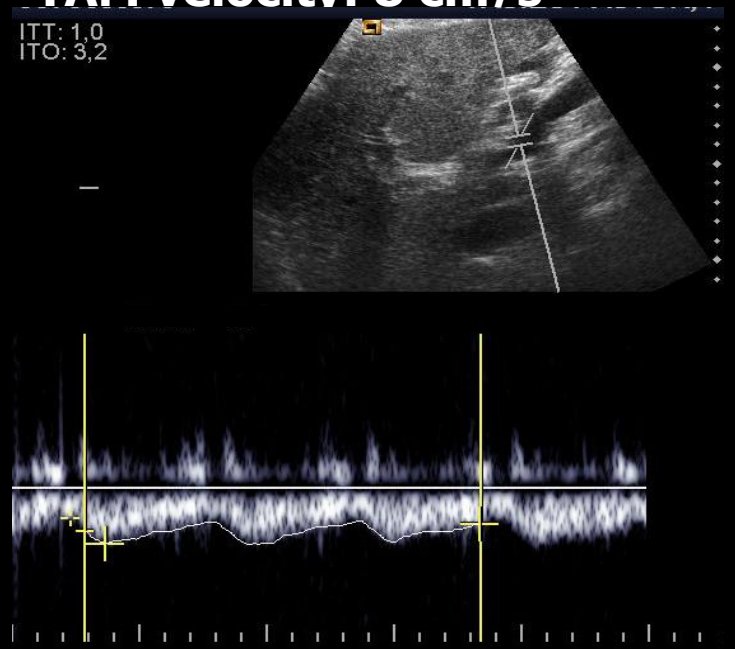
PV blood flow velocity and PH

The velocity of blood flow in the PV decreases due to increased resistance opposed by the liver and by porto-systemic shunting

Slightly reduced portal vein velocity
TAM velocity: 18 cm/s



Markedly reduced portal vein velocity
TAM velocity: 8 cm/s



PV diameter and blood flow velocity: clinical correlations

Both parameters are increasingly observed in decompensated cirrhosis and numerically (but slightly) correlate with PH and the severity of liver disease

- Diameter >13 mm: predictive of EV — Schepis 2001
- Diameter > 15 mm: predictive of large EV — Cottone 1985
- Mean PV velocity < 10 cm/s: reduced survival — Zoli, J Hepatol 1996
- Low PV velocity might predict onset of PV thrombosis — Zocco, J Hepatol 2009

PV diameter and velocity should be always reported

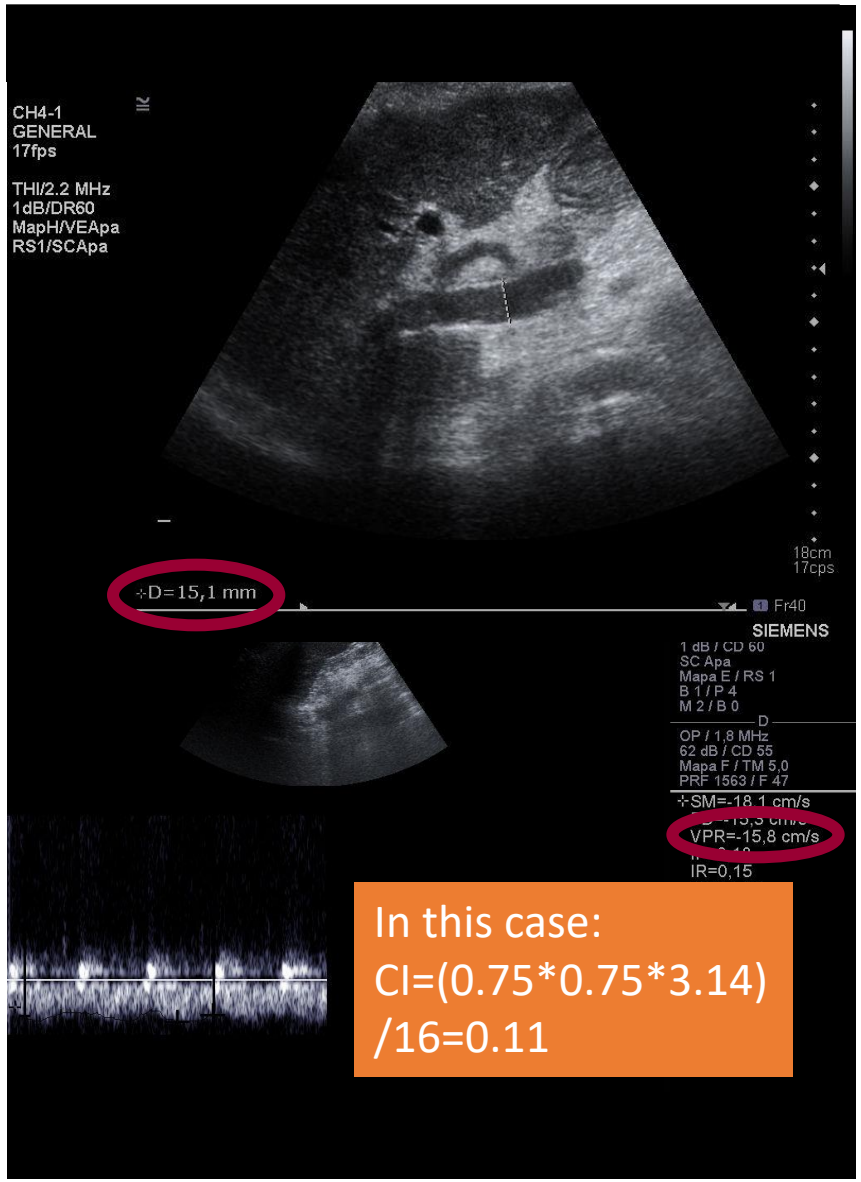
EFSUMB recommendations on performance and reporting of US in PH

Limitations of quantitative parameters on Doppler-Us

Limitation	Solution
Intra-observer and inter-observer variability	Standardized protocols Sabba et al 1990 and 1995; Zoli et al. 1996; Sacerdoti et al. 1997
Inter-equipment variability	Preferably use the same equipment on follow-up; assess normal values on your machine
Modified by several physiological or external factors (drugs, ageing…)	Always check fasting state; report other potential modifiers on US report
Abdominal air	No solution yet

Combination diameter and velocity: congestion index of the PV

Moriyasu 1986



CI =area (cm^2)/mean vel. (cm/s)

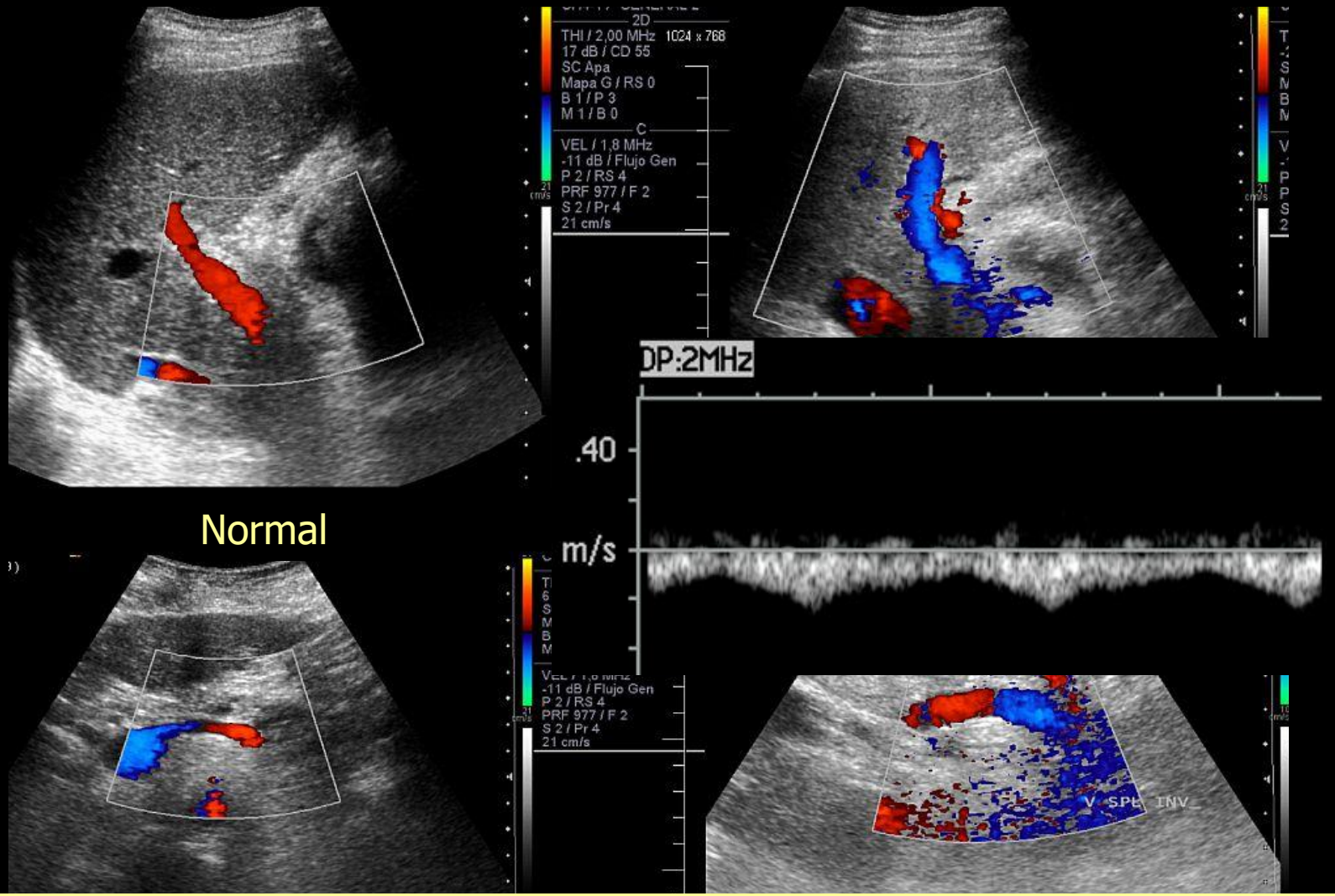
87 pts (prospective) without previous bleeding → follow-up (mean 24 months); Clinical variables, endoscopy, CDUS

↓

CI independently associated with the risk of first variceal bleeding (with variceal size, RWM and bilirubin)
Improves prediction

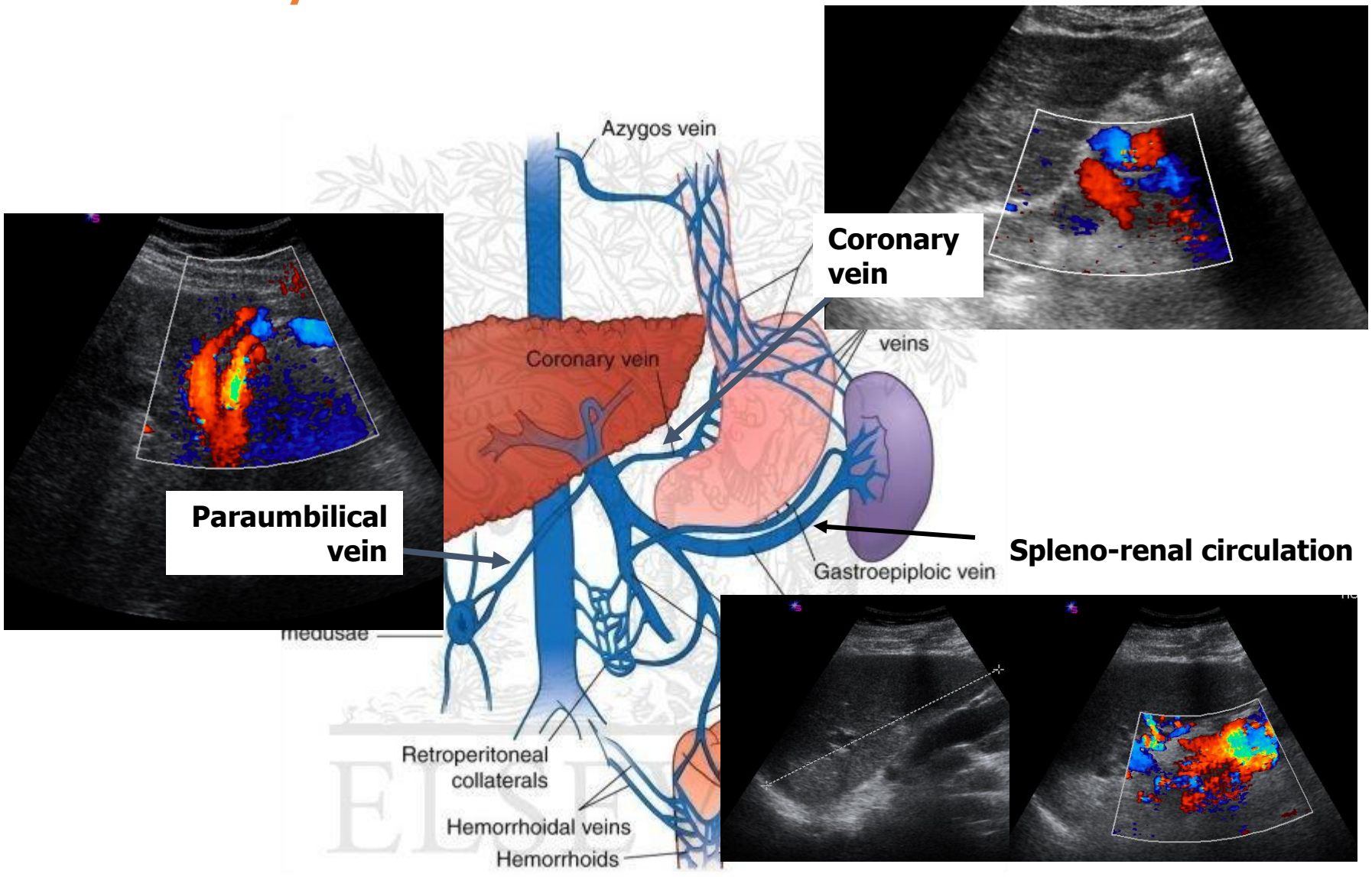
Siringo et al. Hepatology 1994

Portal flow reversal: a pathognomonic sign of PH



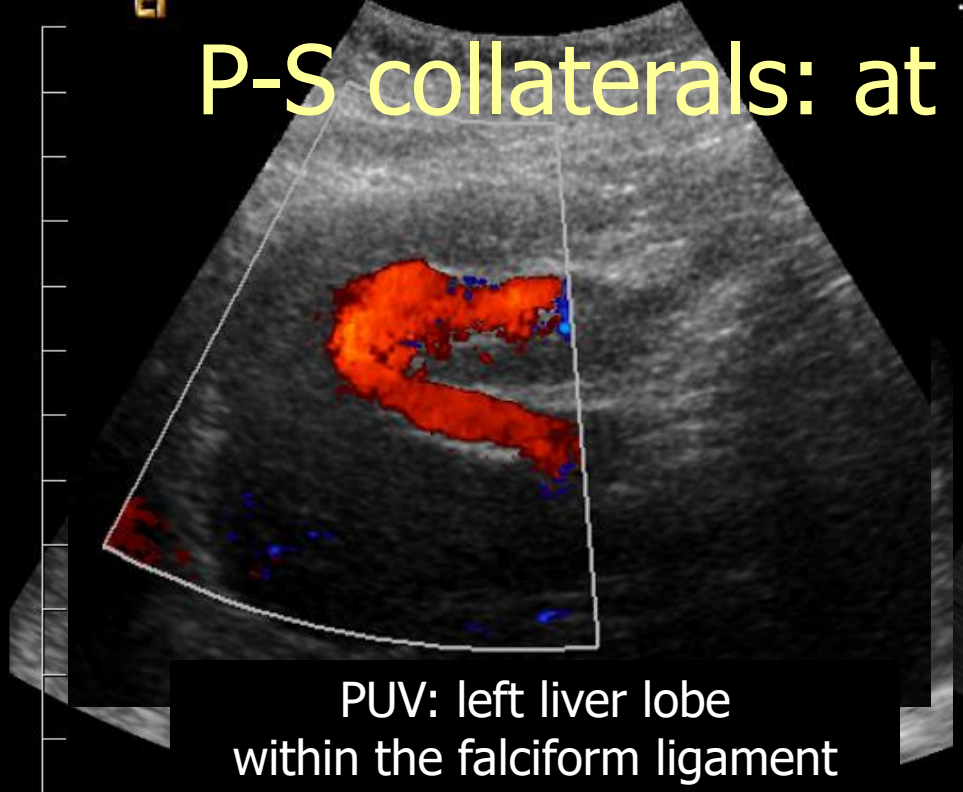
8.3% of unselected cirrhotics; very rare in compensated pts (look for large collateral vessels)

Porto-systemic collaterals on US



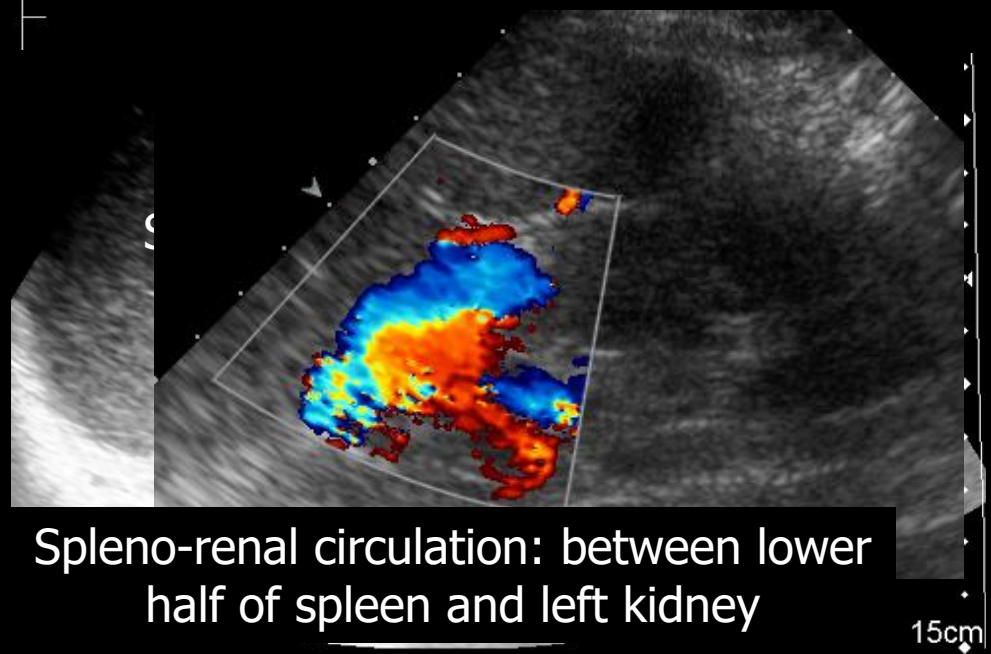
From Rikkers LF. Portal hypertension. In Miller TA: Physiologic Basis of Modern Surgical Care. St. Louis, CV Mosby, 1988

P-S collaterals: at least look for these



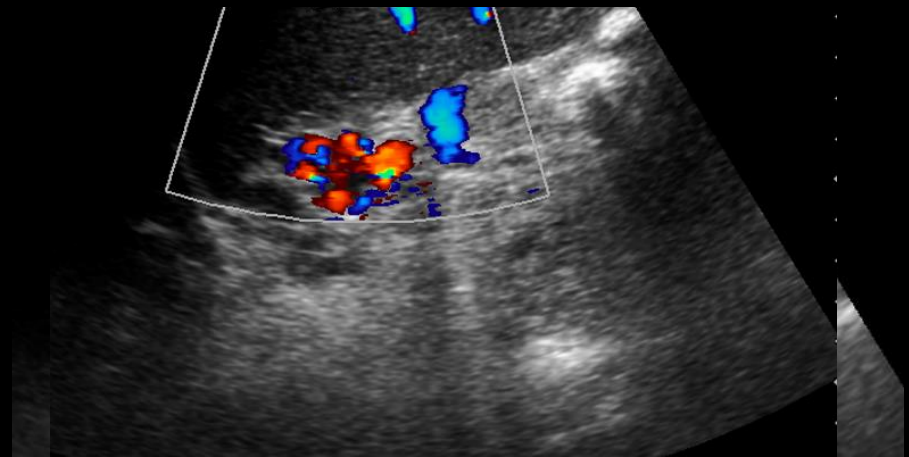
PUV: left liver lobe
within the falciform ligament

Left gastric vein: epigastrium
posterior to the left hep. lobe



Spleno-renal circulation: between lower
half of spleen and left kidney

Short gastric veins:
left hypochondrium posterior to the
upper pole of the spleen



P-S collaterals and PH

Pathognomonic sign of PH (100% specific); "compensatory" but not protective

Prevalence increases with severity of liver failure and PH: <20% in Child A, 40-50% in Child B-C >60% in Child C and/or large varices

Are a predictor of very severe PH (HVPG > 16 mmHg) in compensated pts

Berzigotti J Gastroenterol 2011

Their de novo appearance on follow-up (or their increase in number) is associated with the appearance and growth of EV

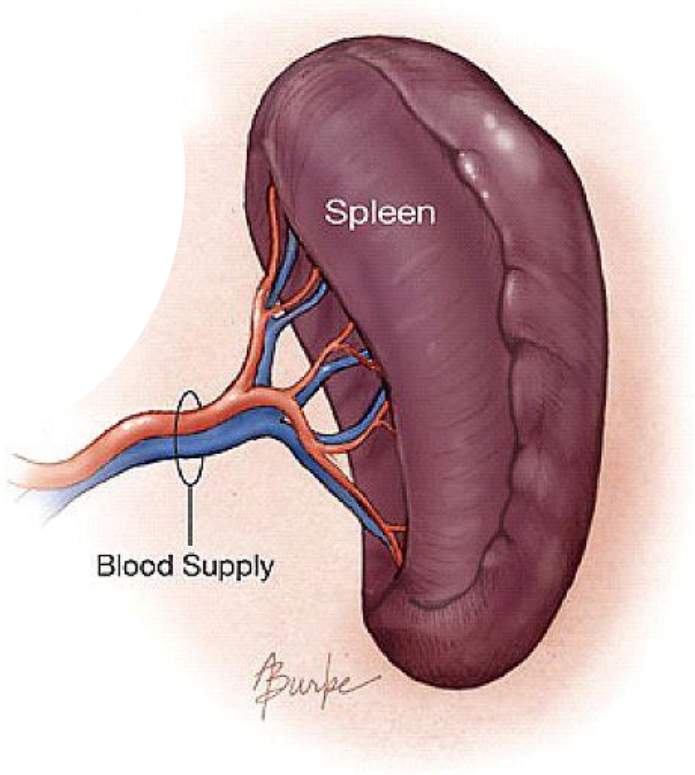
Berzigotti DLD 2008

SHOULD BE ACTIVELY RESEARCHED by CDUS

EFSUMB recommendations on performance and reporting of US in PH

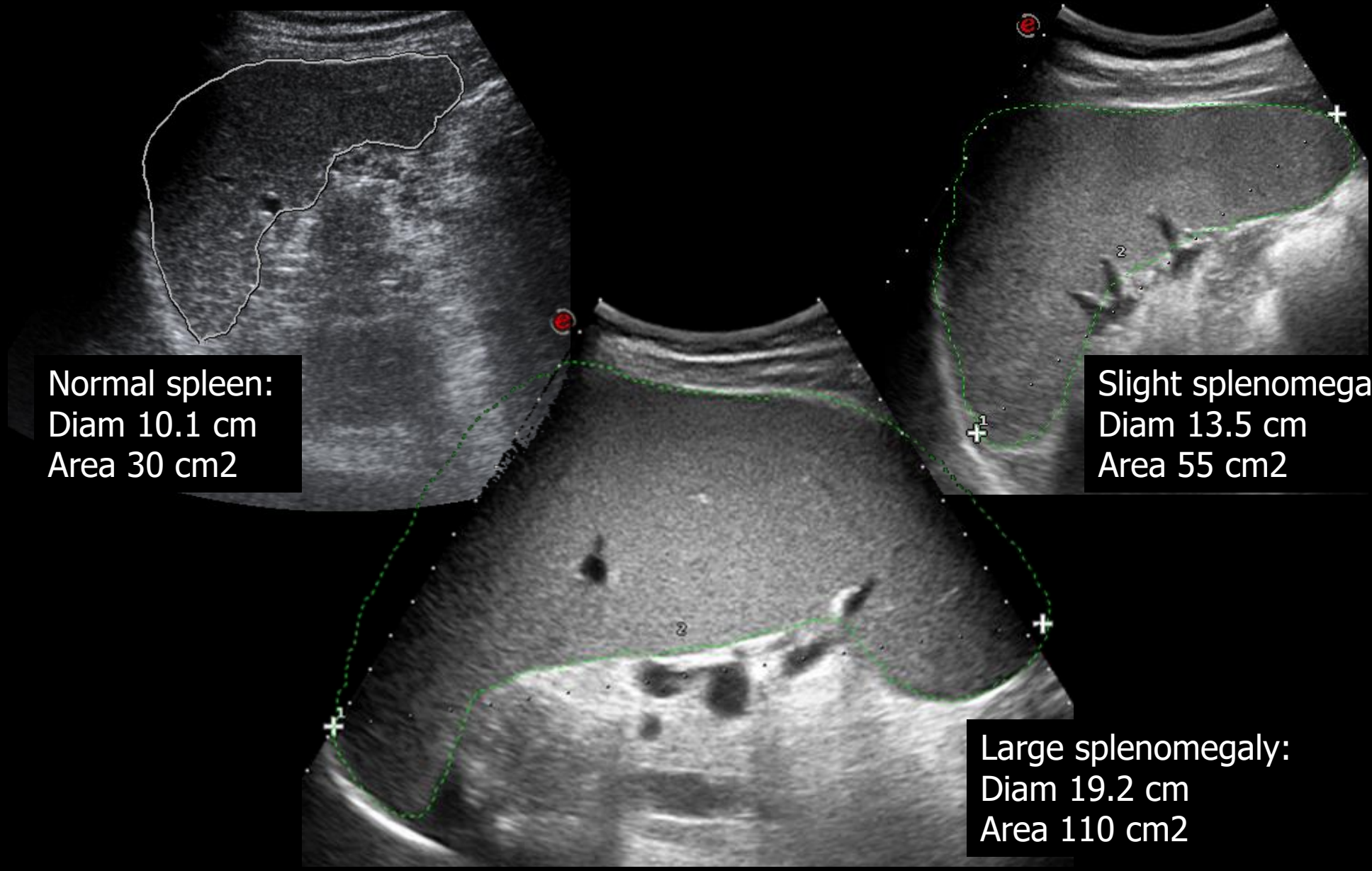
Splenomegaly: most sensitive sign of CSPH

Spleen size increases as a consequence of PH (any cause): congestion, lymphoid tissue hyperplasia, angiogenesis, fibrogenesis



Mejias, J Hepatol 2010

Spleen size assessment by US



Normal spleen:
Diam 10.1 cm
Area 30 cm2

Slight splenomegaly:
Diam 13.5 cm
Area 55 cm2

Large splenomegaly:
Diam 19.2 cm
Area 110 cm2

Splenomegaly in PH

Compensated cirrhosis: splenomegaly in 65-70%; prevalence increases in decompensated cirrhosis

Piscaglia et al, Scand J Gastro 2002

Associated with CSPH in very well-compensated pts

Berzigotti et al, AJG 2008

Spleen size correlates with presence and size of EV and their bleeding risk

Many authors

Increase of spleen size on follow-up is associated with increased formation and growth of EV, and with first clinical decompensation in compensated cirrhosis

Berzigotti, Zappoli et al, Clin Gastroenterol Hepatol 2007

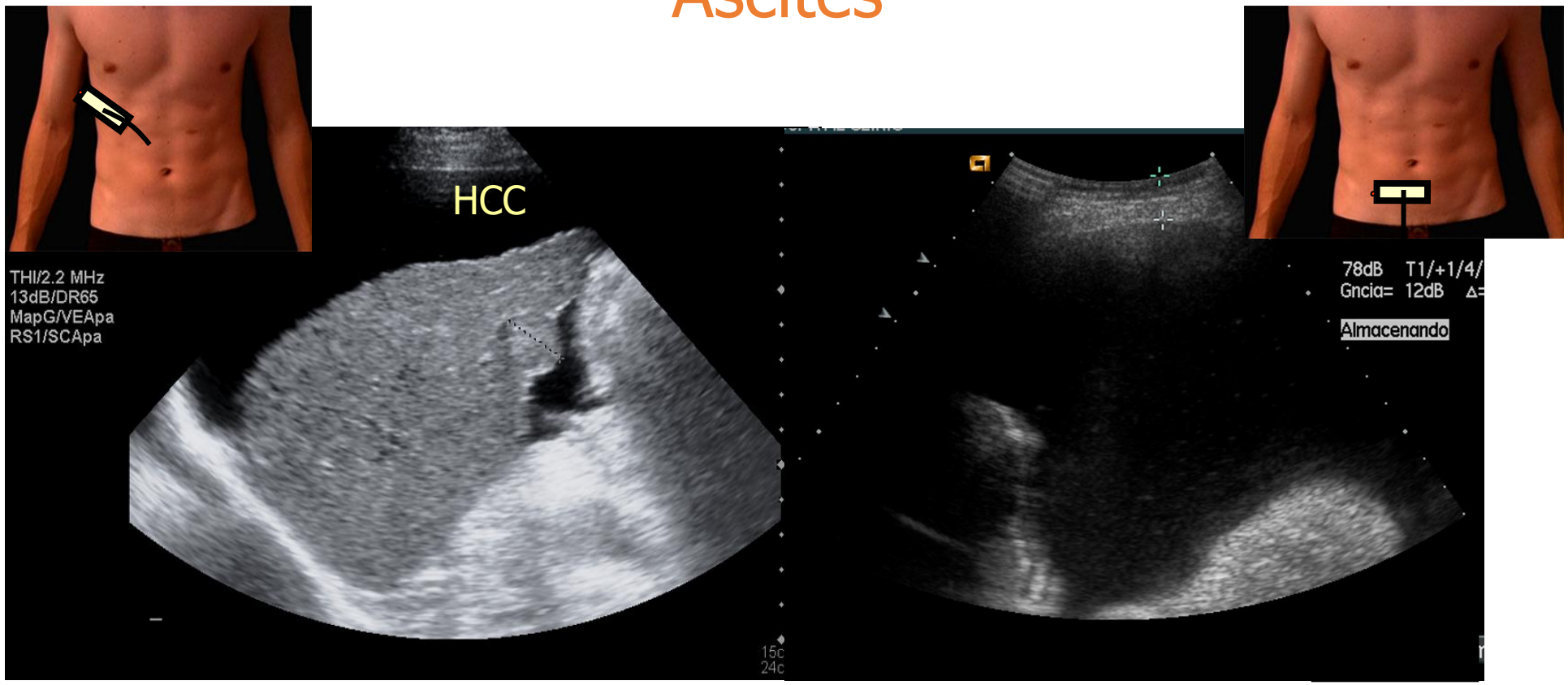
Its combination with other non-invasive parameters (platelet and liver stiffness) is the best predictor of EV up to date

Kim et al. AJG 2011

ALWAYS REPORT SPLEEN SIZE

Drawback: low specificity

Ascites



Defines a decompensated stage of cirrhosis with poor prognosis; a semiquantitative assessment can be used

Even minimal ascites, not clinically evident, should be reported

Zipprich et al AASLD 2011

Sometimes: hepatic hydrothorax → remember to look at pleural spaces

Diagnosis of PH

- Doppler-US allows diagnosing the main causes of PH
- 100% specific signs: reversal of flow in the portal system; porto-systemic collaterals
- Most sensitive sign: splenomegaly (scarcely specific)
- Other useful signs: reduction of PV velocity; dilatation of PV axis; lack of respiratory variation of the diameter of SV and SMV

EFSUMB recommendations on performance and reporting of US in PH

Portal venous system:
- Portal vein and axis patency
- Portal vein diameter
- Flow direction and TAMv
- Presence of collateral vessels

Spleen size

Hepatic veins patency

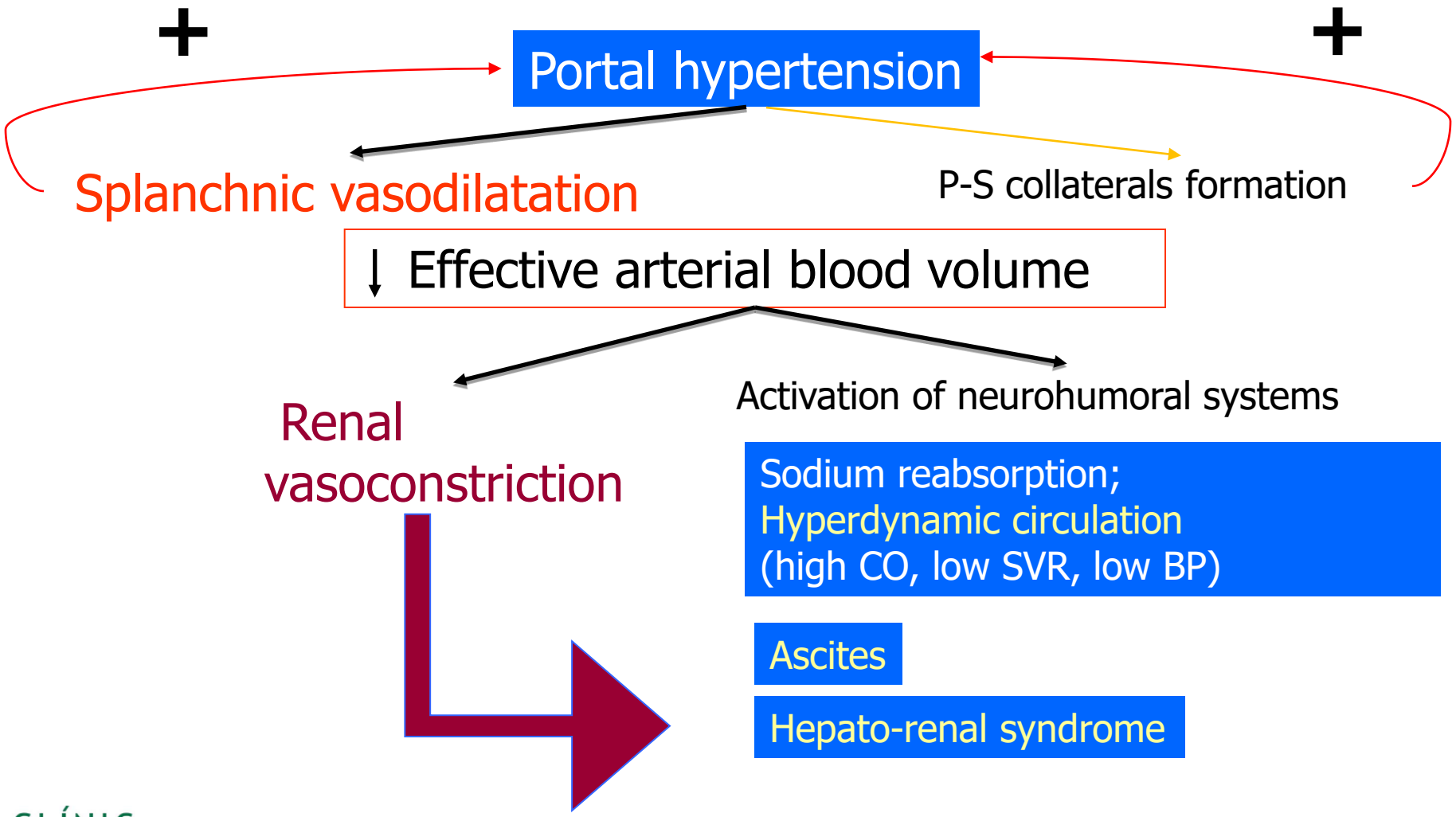
Peritoneal cavity (ascites)

Should be always assessed AND reported

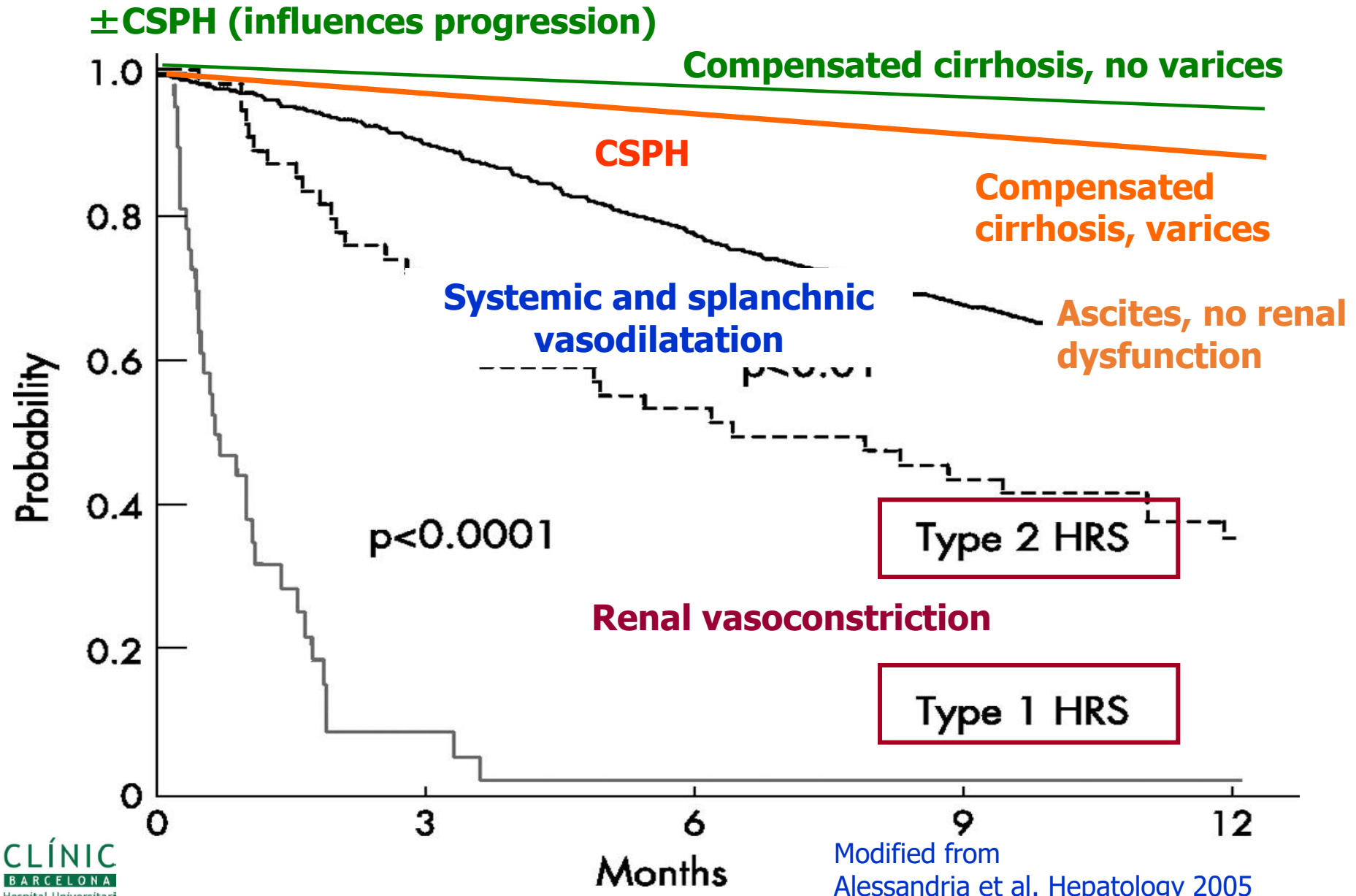
Doppler-US assessment of abdominal arteries in PH

"AREN'T THERE ENOUGH PROBLEMS IN THE WORLD ALREADY?"

Pathophysiology of PH-late stages

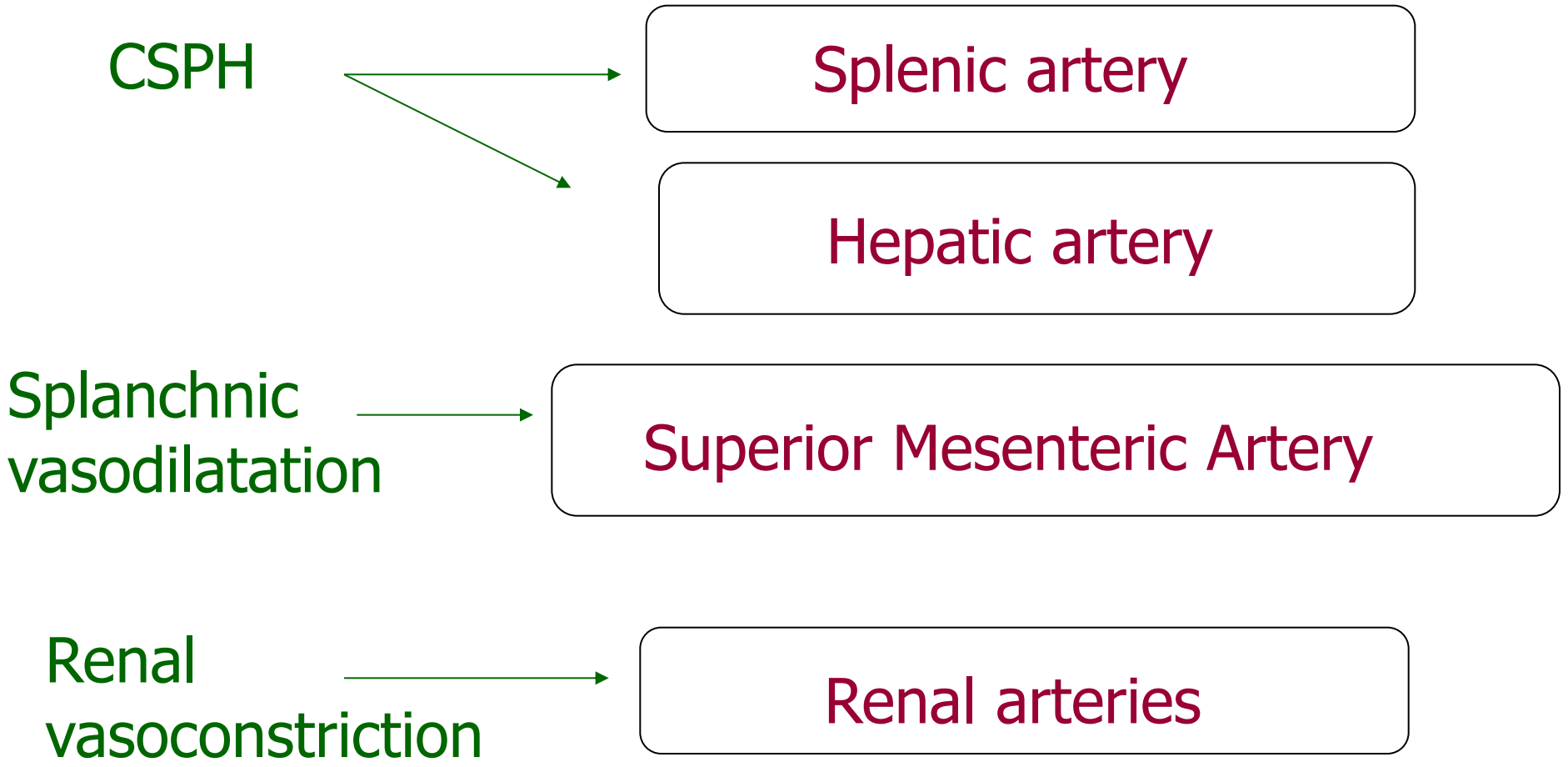


PH and prognosis in cirrhosis



Modified from
Alessandria et al. Hepatology 2005

Rationale for arterial Doppler in PH



Parameters to be measured

Quantitative parameters

Systolic, diastolic and <u>mean velocity</u>

Problem: measurement-angle dependent (need < 60º)

Semi-quantitative parameters

Impedance indexes→ estimate vascular resistance

Not angle-dependent→ parenchymas

Automatically calculated from the Doppler tracing as:

$$RI = \frac{peak\ systolic\ velocity - end\ diastolic\ velocity}{peak\ systolic\ velocity}$$

$$PI = \frac{peak\ systolic\ velocity - end\ diastolic\ velocity}{mean\ velocity}$$

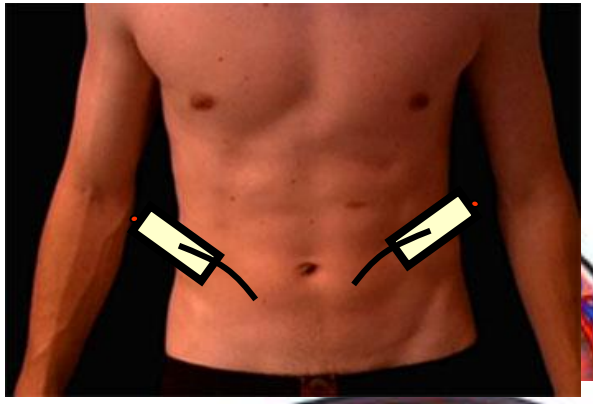
Less dependent on heart rate

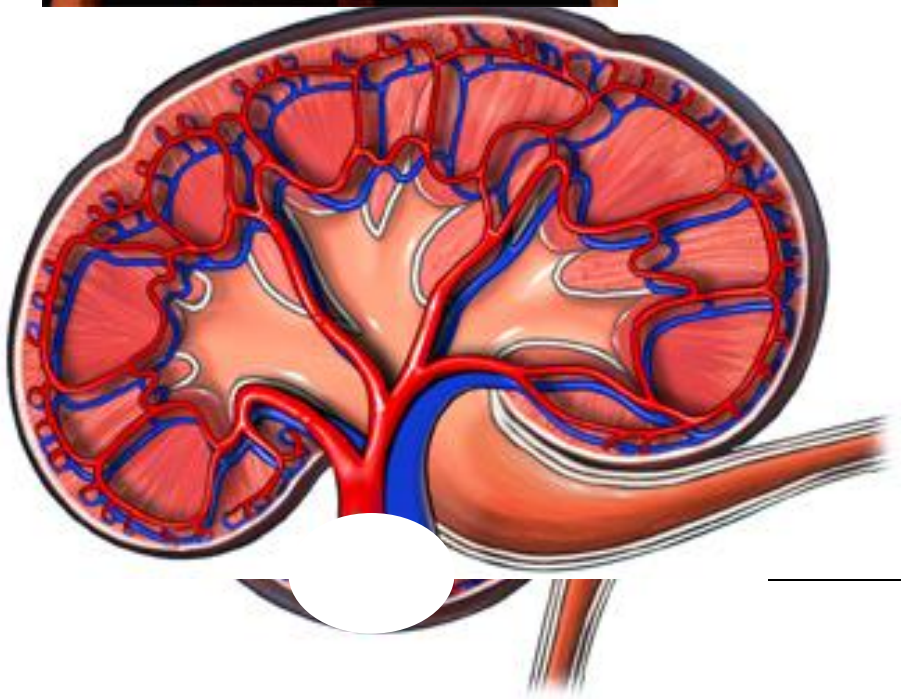
Which conditions should I control?

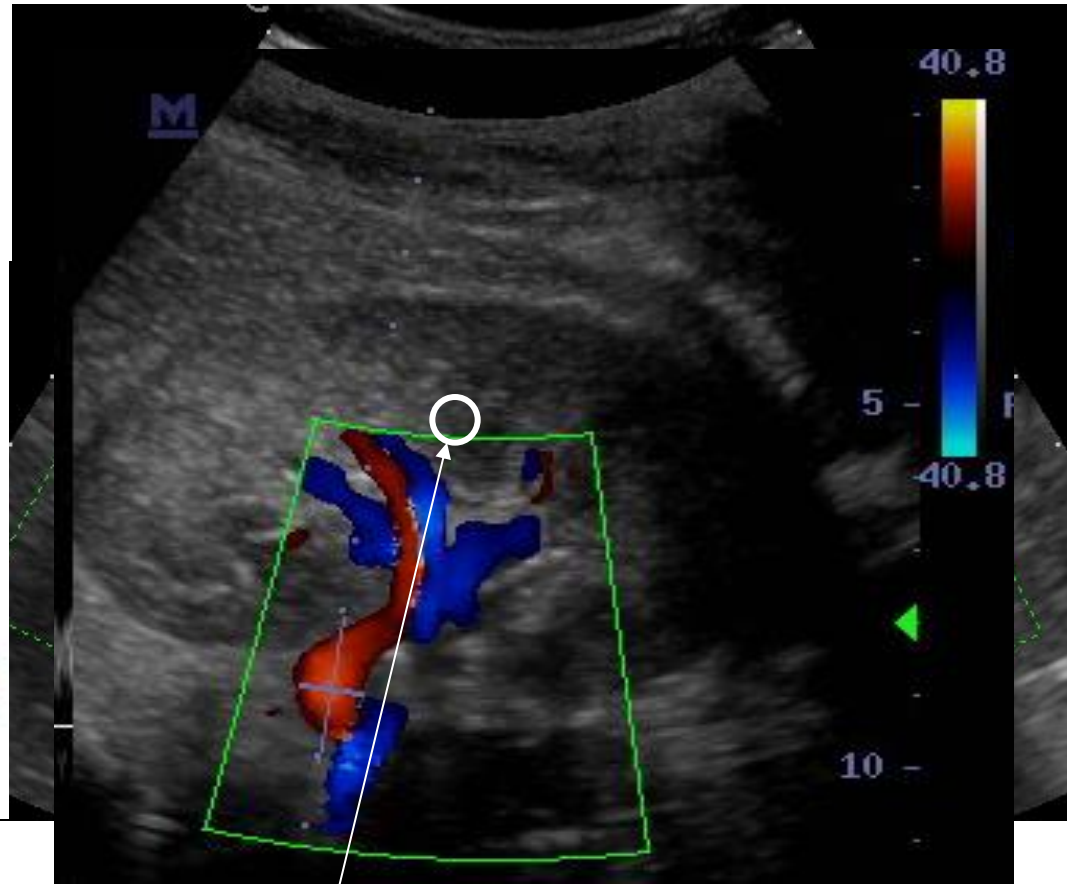
Several physiologic conditions induce changes in systemic haemodynamics and consequently in Doppler measurements

- Meal ingestion → control fasting state
- Exercise → provide a resting period (>10 minutes)
- Take into account: heart rate, age and vasoactive drugs
- Consider that causes of vascular/parenchymal damage other than cirrhosis may exist

Renal arteries

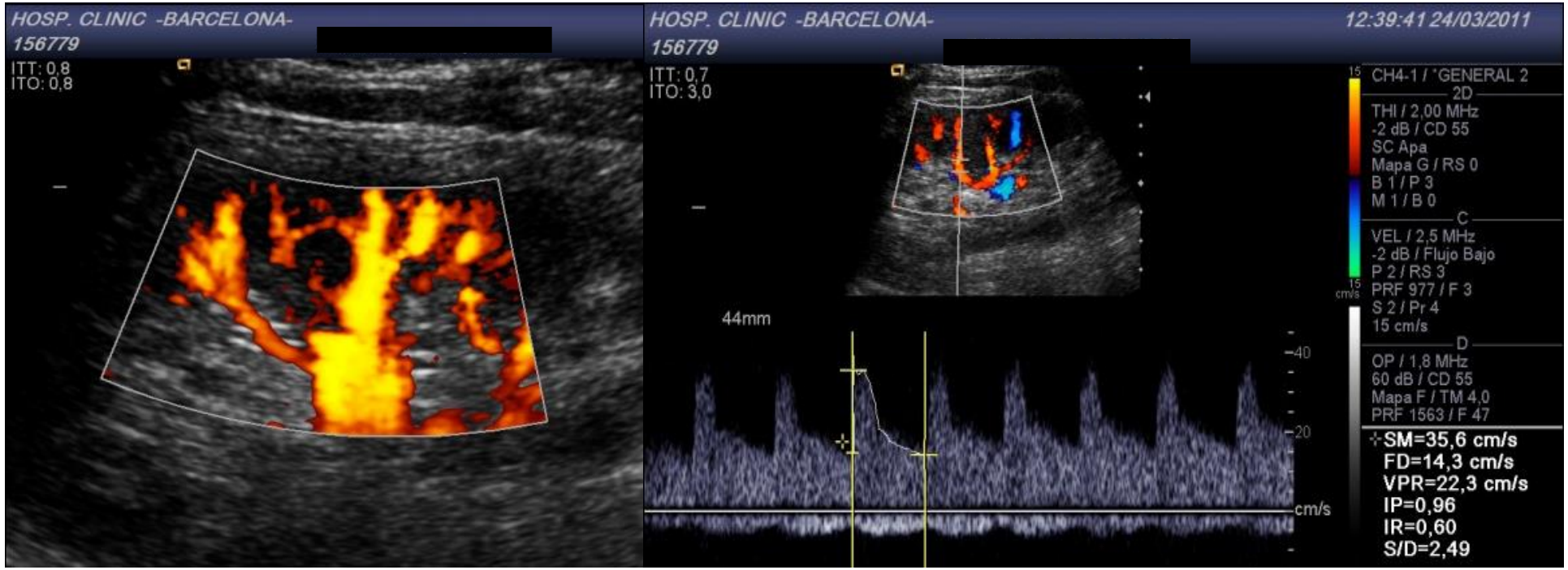






Interlobar arteries

Renal arteries RI and PI: normal subjects

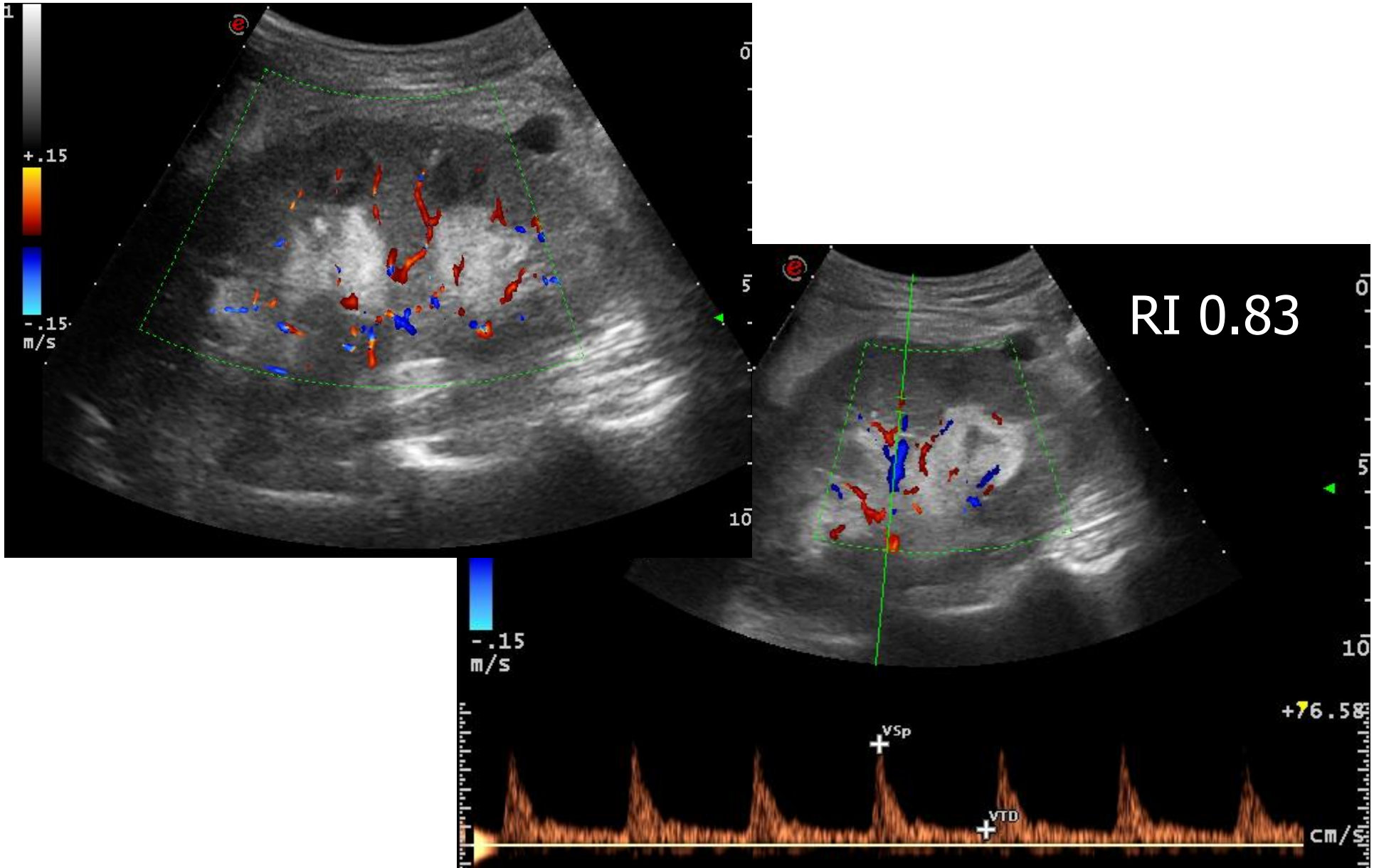


The interlobar arteries can be easily visualized and can be sampled with spectral Doppler to measure PI and RI

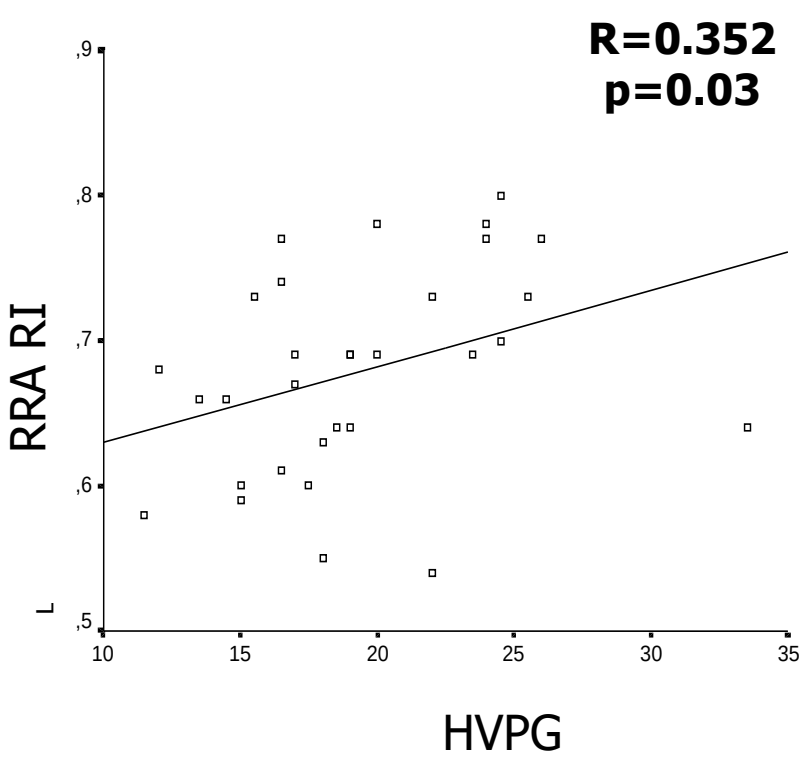
UNL: 0.70

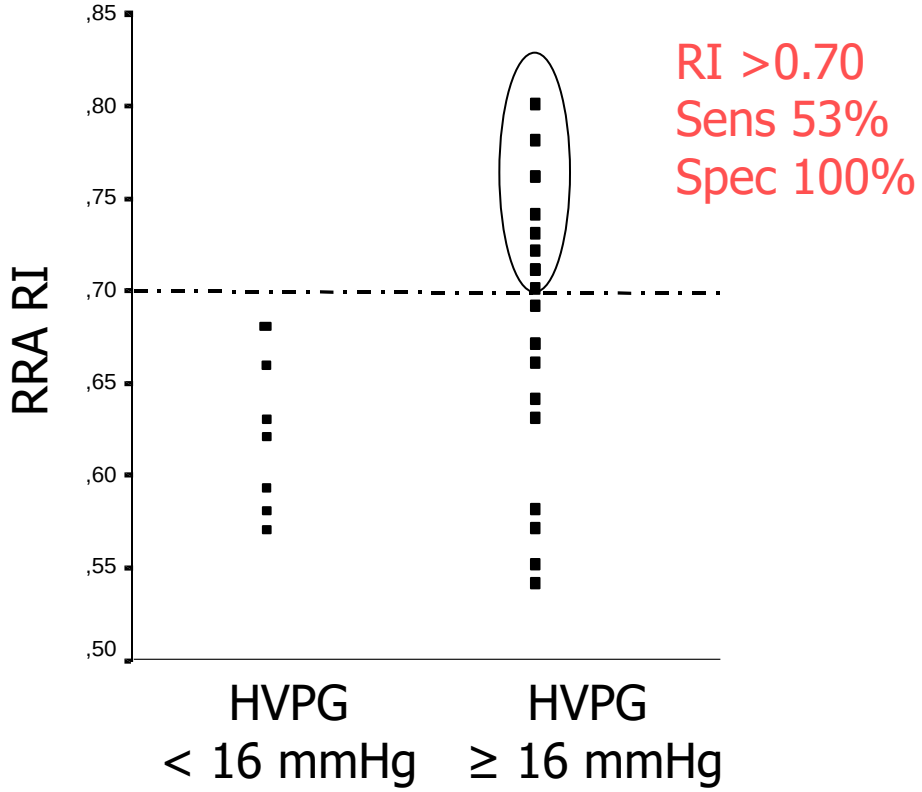
Platt J et al. Radiology 1989
Tublin E et al. AJR 2003

Renal vasoconstriction by Doppler in cirrhosis



Renal arteries RI and PP



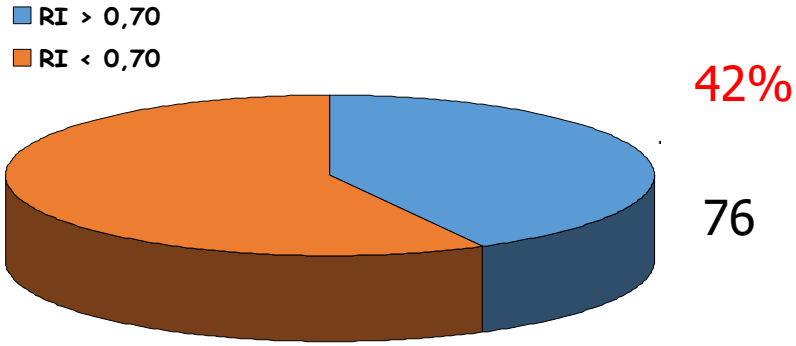


RI >0.70
Sens 53%
Spec 100%

Berzigotti et al, Radiology 2006

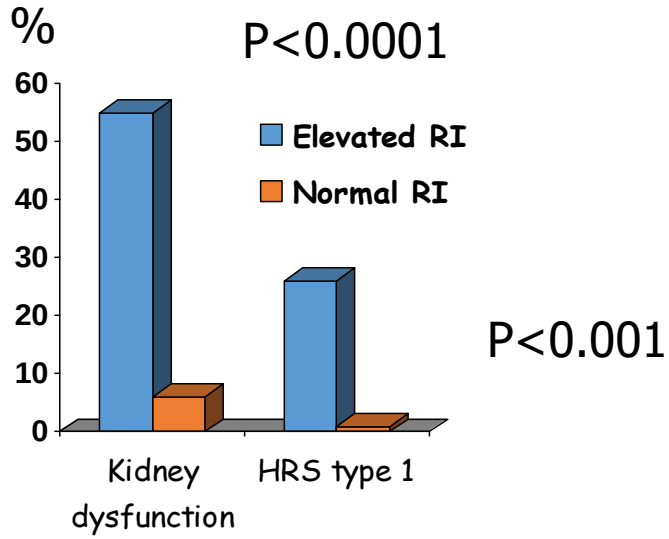
Renal arteries RI and HRS

N=180 non-azotemic cirrhosis



42%

76



P<0.001

RI > 0.70: increased risk of hepato-renal syndrome

Platt, Hepatology 1994

Predictor of kidney failure and mortality in pts with ascites

Maroto et al, Hepatology 1994

Doppler assessment of abdominal arteries in cirrhosis

- Worth doing: adds prognostic information to the standard Doppler examination of the portal system
- Vessels to be studied: renal arteries (prognostic implications); HA, SA and SMA complementary information
- Technically demanding: needs skilled operators

EFSUMB recommendations on performance and reporting of US in PH

Portal venous system:
- Portal vein and axis patency
- PV diameter
- Flow direction and TAMv
- Presence of collateral vessels

Spleen size

Hepatic veins patency

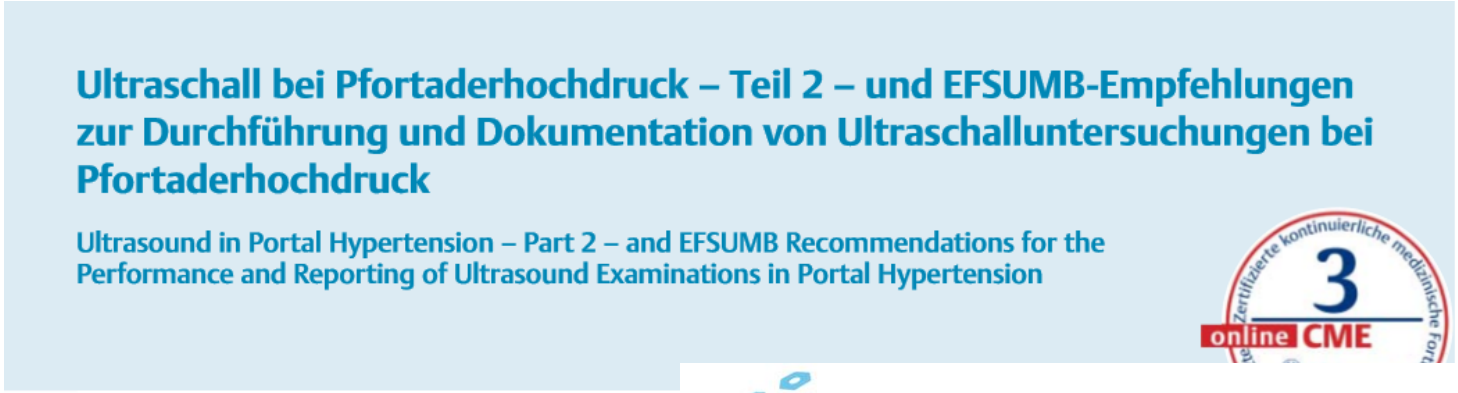
Peritoneal cavity (ascites)

Arterial vessels
- Splenic artery, hepatic artery
- SMA
- Renal arteries

Should be always assessed AND reported

Assessment is restricted to tertiary (referral) US Centers

https://www.thieme-connect.de/products/ejournals/pdf/10.1055/s-0031-1299145.pdf?update=true



Ultraschall Med 2012

https://learning.easl.eu/course/index.php?categoryid=2