Elastography in cirrhotic and non-cirrhotic portal hypertension – from guidelines to clinical practice

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The compensated patient: my vision

Standard US

- Morphology of liver, spleen and vessels

Doppler US

- Hemodynamics
  - Presence of flow
  - Flow direction
  - Velocity

Complementary

Elastography

- Liver stiffness
  - Inflammation
  - Fibrosis
  - Congestion
  - other
- Spleen stiffness
  - Portal hypertension
  - Hematological diseases

Framework for the use of elastography in PH

Similar to what discussed yesterday as for the use of ultrasound

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

  → CLASSIFICATION OF PORTAL HYPERTENSION

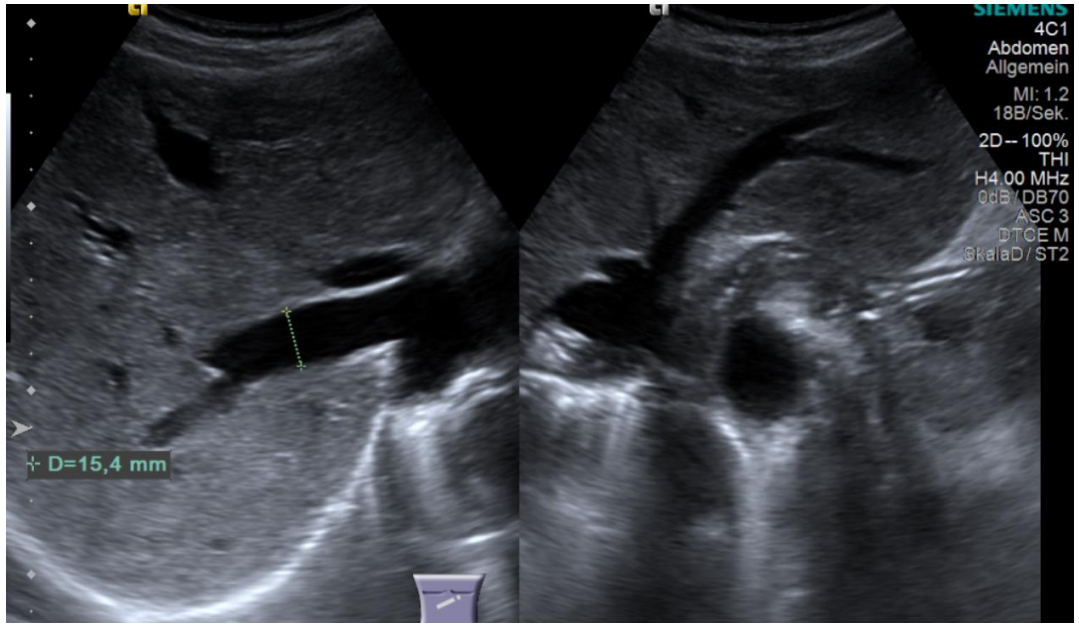
- Screening of CSPH in patients with known compensated advanced chronic liver disease

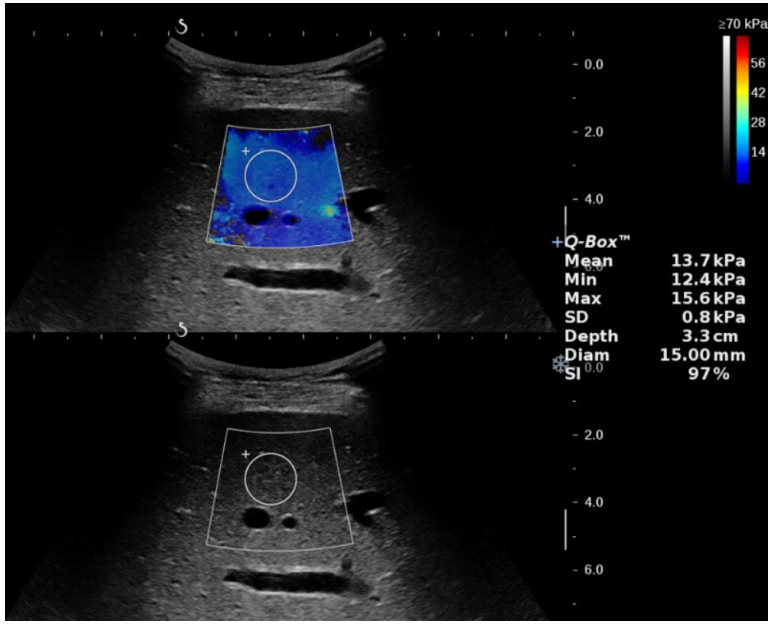
- Prognostic assessment in cACLD/cirrhosis off and on treatment

Obviously combining US and elastography is of great benefit

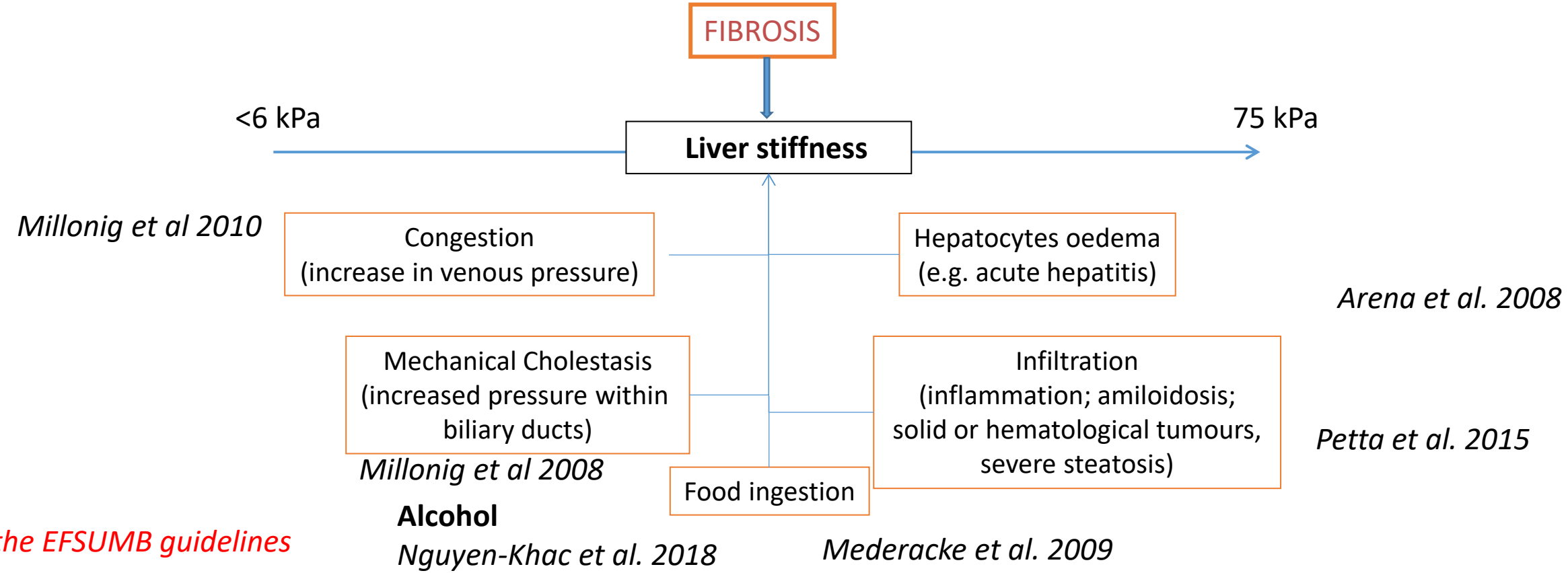
In the era of elastography pay attention to US!

- Man, 65 y/o, metabolic syndrome; steatosis diagnosed 8 years ago; normal LSM (4.6 kPa): MASLD
- Yearly follow-up until now: always stable
- Last control: 14 kPa and edemas; sent to us with suspicion of MASH cirrhosis with PH
- Ultrasound:



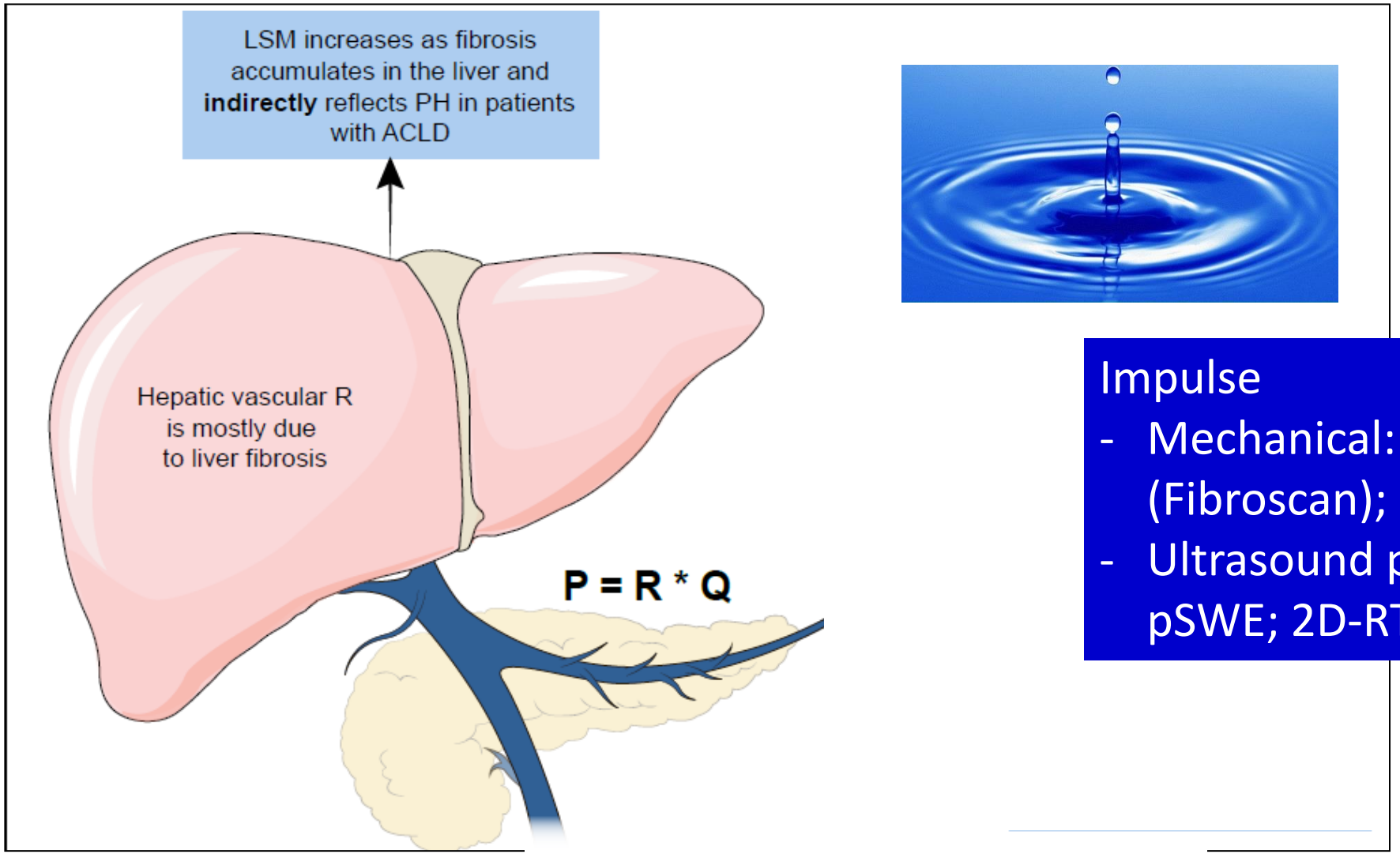


Mind the confounders and check with POCUS



Doppler-US can rule-out and rule-in many of the confounders

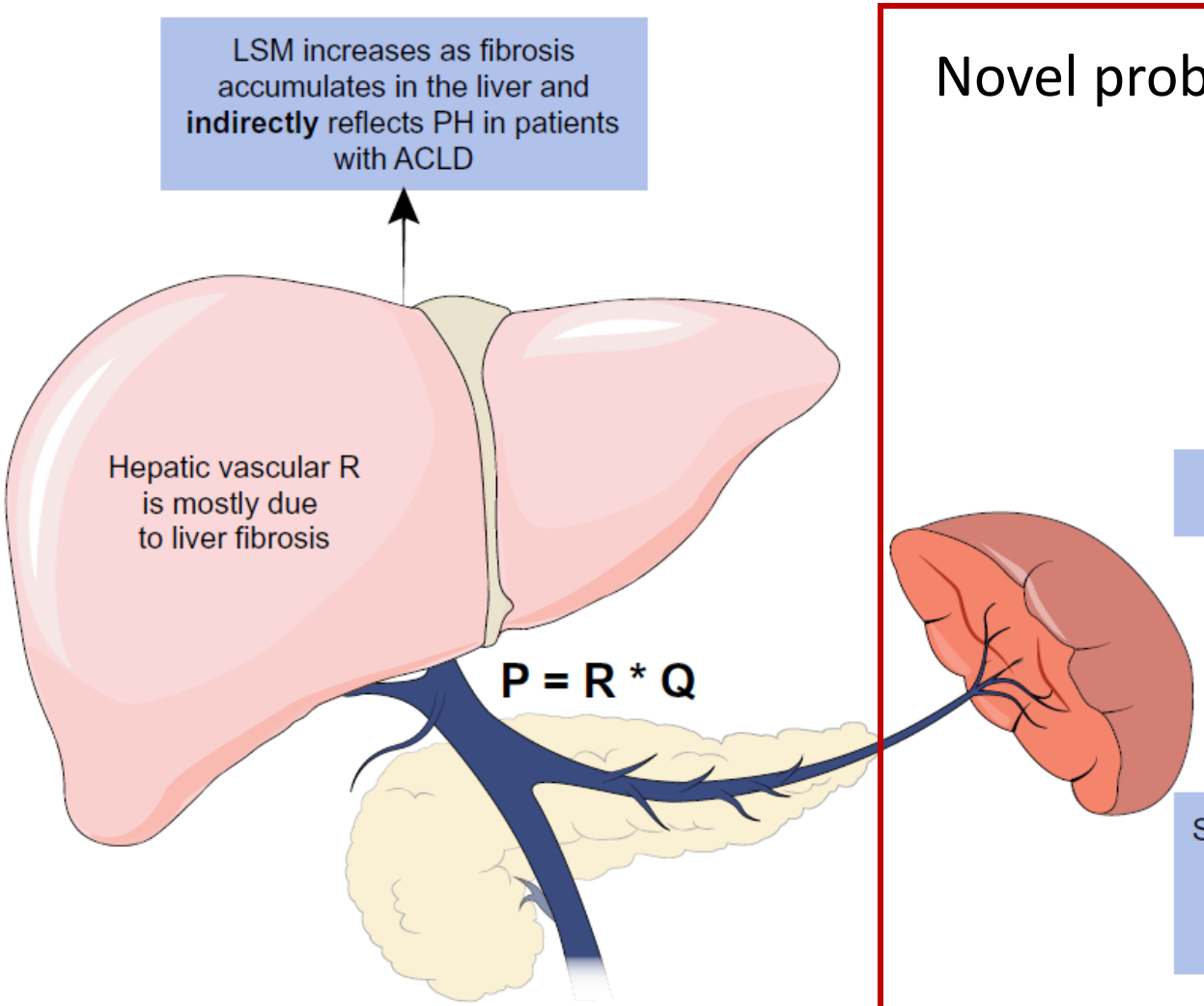
Rationale of liver elastography for portal hypertension



Impulse
- Mechanical: TE (Fibroscan); MRE
- Ultrasound pulse(s): pSWE; 2D-RT-SWE

Berzigotti A., J Hepatol 2017

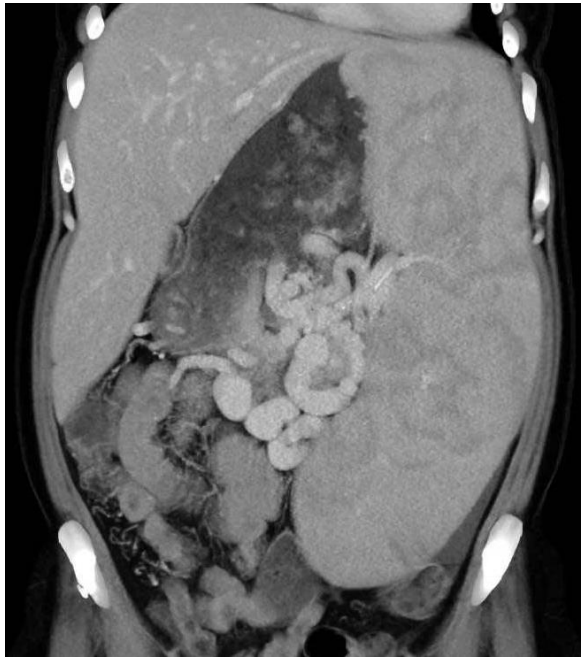
Rationale of spleen stiffness measurement for PH



Novel probe 100 Hz

SSM **directly** reflects PH

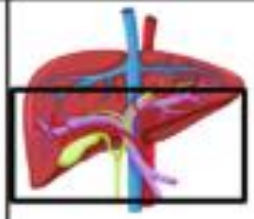
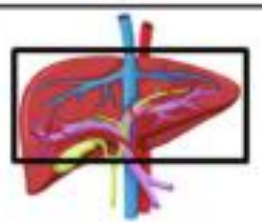
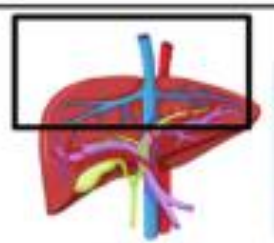
Stiffness is due to congestion and other changes related directly to PH independent of its cause



Berzigotti, J Hepatol 2017

Classification of PH is greatly facilitated by elastography

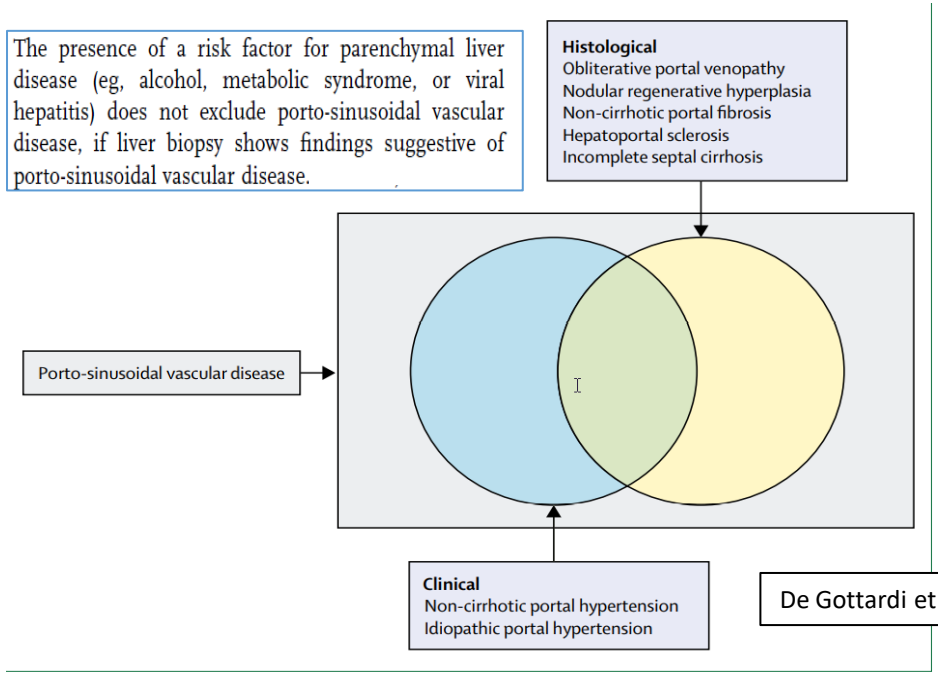
Intrahepatic pre-sinusoidal causes are increasing!

Type of PH	Pre-hepatic PH		Hepatic (sinusoidal) PH (cACLD)		Post-hepatic PH	
Stiffness measurement patterns						
	Normal LSM	High SSM	High SSM	High SSM	High LSM	High SSM
Examples	Extrahepatic portal vein obstruction		Liver cirrhosis		Budd-Chiari syndrome Congestive liver (heart)	

PH, portal hypertension; LSM, liver stiffness measurement; SSM, spleen stiffness measurement; cACLD, compensated advanced chronic liver disease

Mendoza YP et al. 2020

Porto-sinusoidal vascular disease (PSVD)



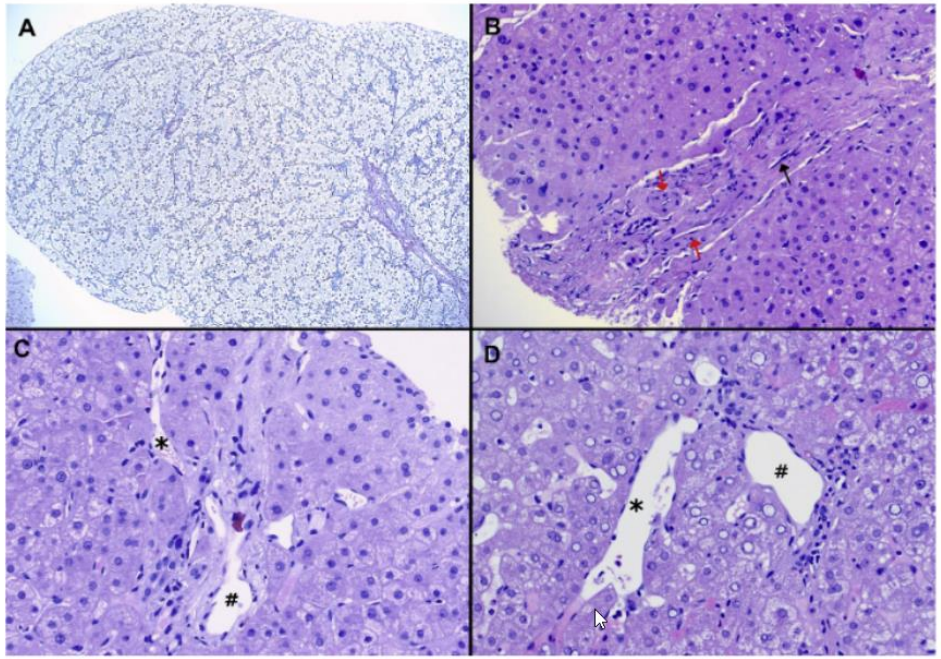
Vascular liver disease characterized by portal hypertension in the absence of cirrhosis

Requires histological confirmation

Commonly complicated by portal vein thrombosis

De Gottardi et al. Lancet Gastroenterol Hepatol 2019

Clinical suspicion of PSVD:
signs of PH (splenomegaly, varices, etc) with normal or almost normal liver function

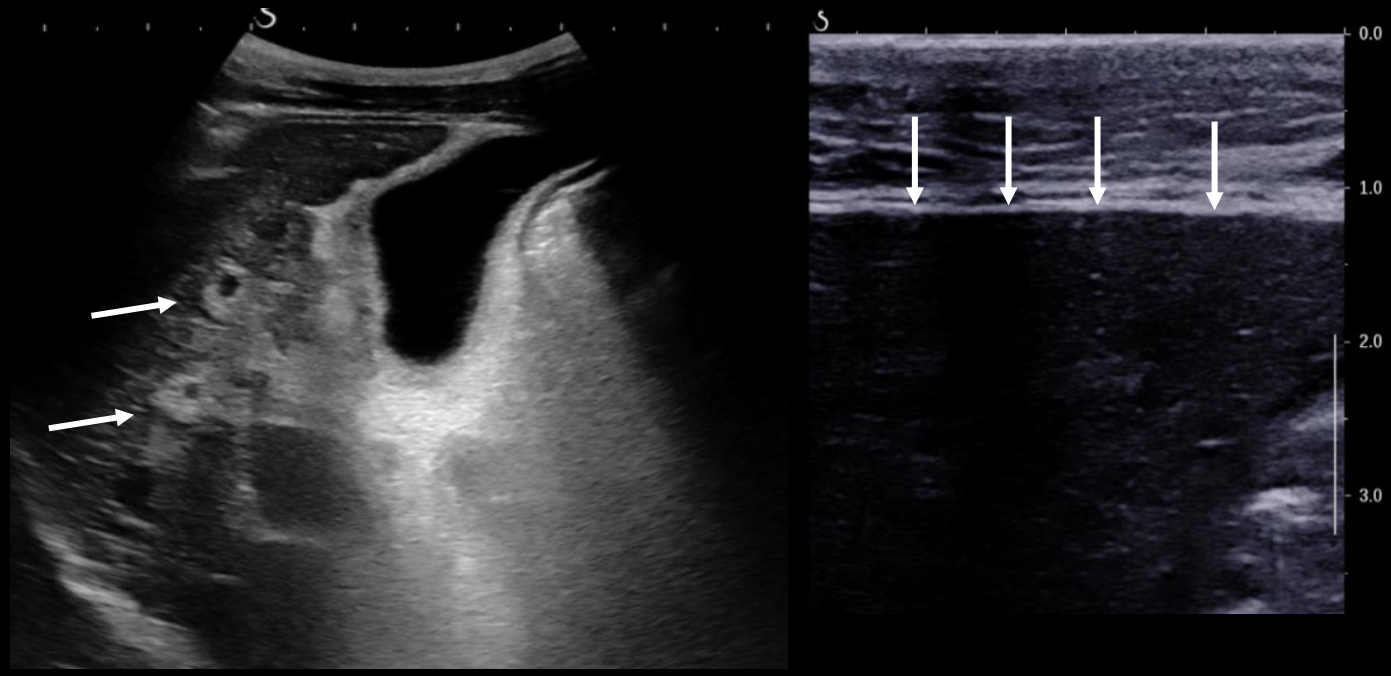


Supplementary Figure 1. Sections showing histological signs of PSVD: (*A*) nodular regenerative hyperplasia (stained with argentic reticulin stain), (*B*) obliterative portal venopathy (black arrows) and multiplication of arteries (red arrows, stained with hematoxylin and eosin), (*C*) hypervascularized portal tracts with periportal vessels (#, stained with hematoxylin and eosin), and (*D*) herniating portal veins (*, stained with hematoxylin and eosin).

Most common histological features

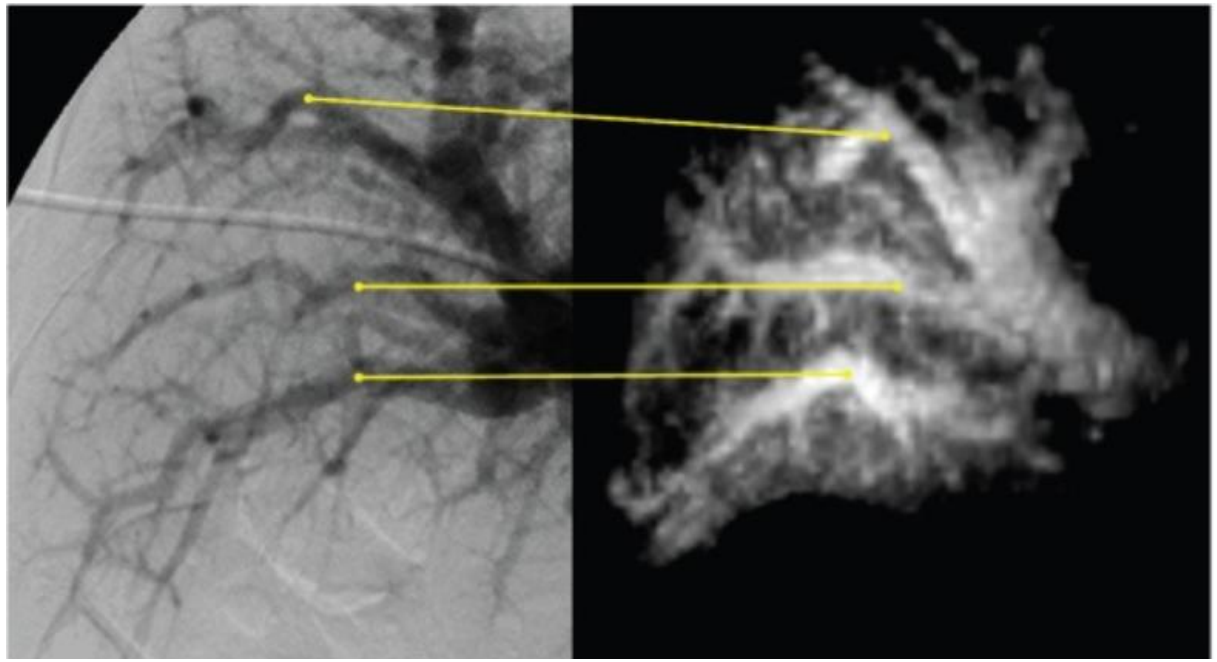
1. NRH
2. Obliterative portal venopathy
3. Abnormal portal tracts with periportal vessels and herniating PVs

Often confounded with cirrhosis: US signs of PSVD/IPH



Thickening of portal walls (Schistosomiasis-like)
+ dysmorphic liver + splenomegaly +/- P-S collaterals and other signs of PH

CEUS using Sonazoid for non-invasive portography to diagnose PSVD/IPH



5 with biopsy-proven IPH

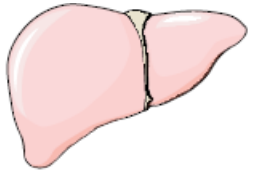
3D CEUS compared to invasive portography

Maruyama et al.
Br J Radiol 2012

1. Periportal delayed enhancement in the liver strongly suggests PSVD
2. In the post-vascular phase greater accumulation of intrahepatic microbubbles

Maruyama et al. Radiology 2009
Maruyama et al. Hep Intern 2012

Elastography in PSVD: very useful



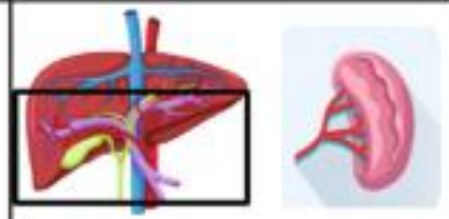
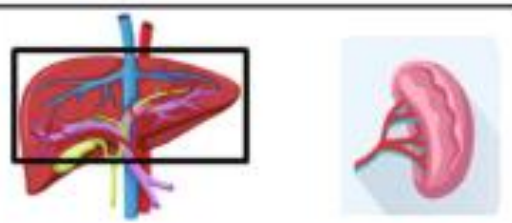
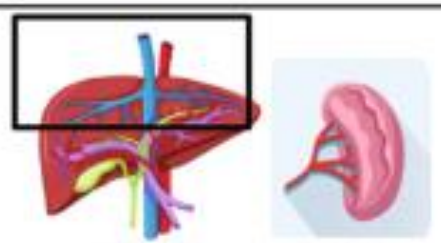
Site of increased resistance to portal flow	Liver stiffness	Spleen stiffness
PSVD: intrahepatic pre-sinusoidal	< 10 kPa Is 97% specific	Elevated If < 40 kPa + Bilirubin < 1 mg/dl no HRV

No cirrhosis or severe liver fibrosis!

Elkrief L et al Hepatology 2021

Moga L et al Hepatology 2024

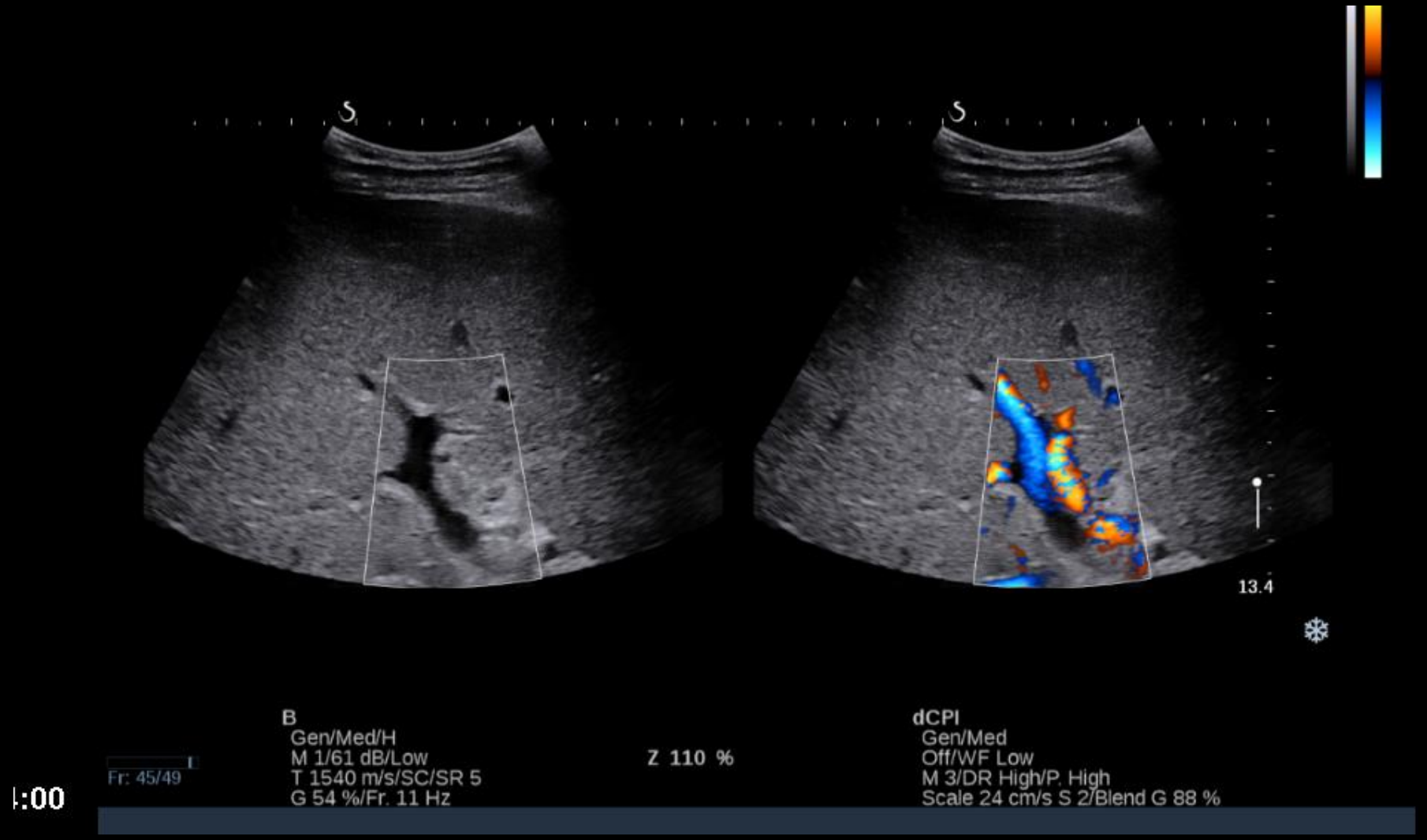
Back to the patient with advanced chronic liver disease

Type of PH	Pre-hepatic PH	Hepatic (sinusoidal) PH (cACLD)	Post-hepatic PH
Stiffness measurement patterns	 Nor mal LSM High SSM	 High SSM High SSM	 High LSM High SSM
Examples	Extrahepatic portal vein obstruction	Liver cirrhosis	Budd-Chiari syndrome Congestive liver (heart)

PH, portal hypertension; LSM, liver stiffness measurement; SSM, spleen stiffness measurement; cACLD, compensated advanced chronic liver disease

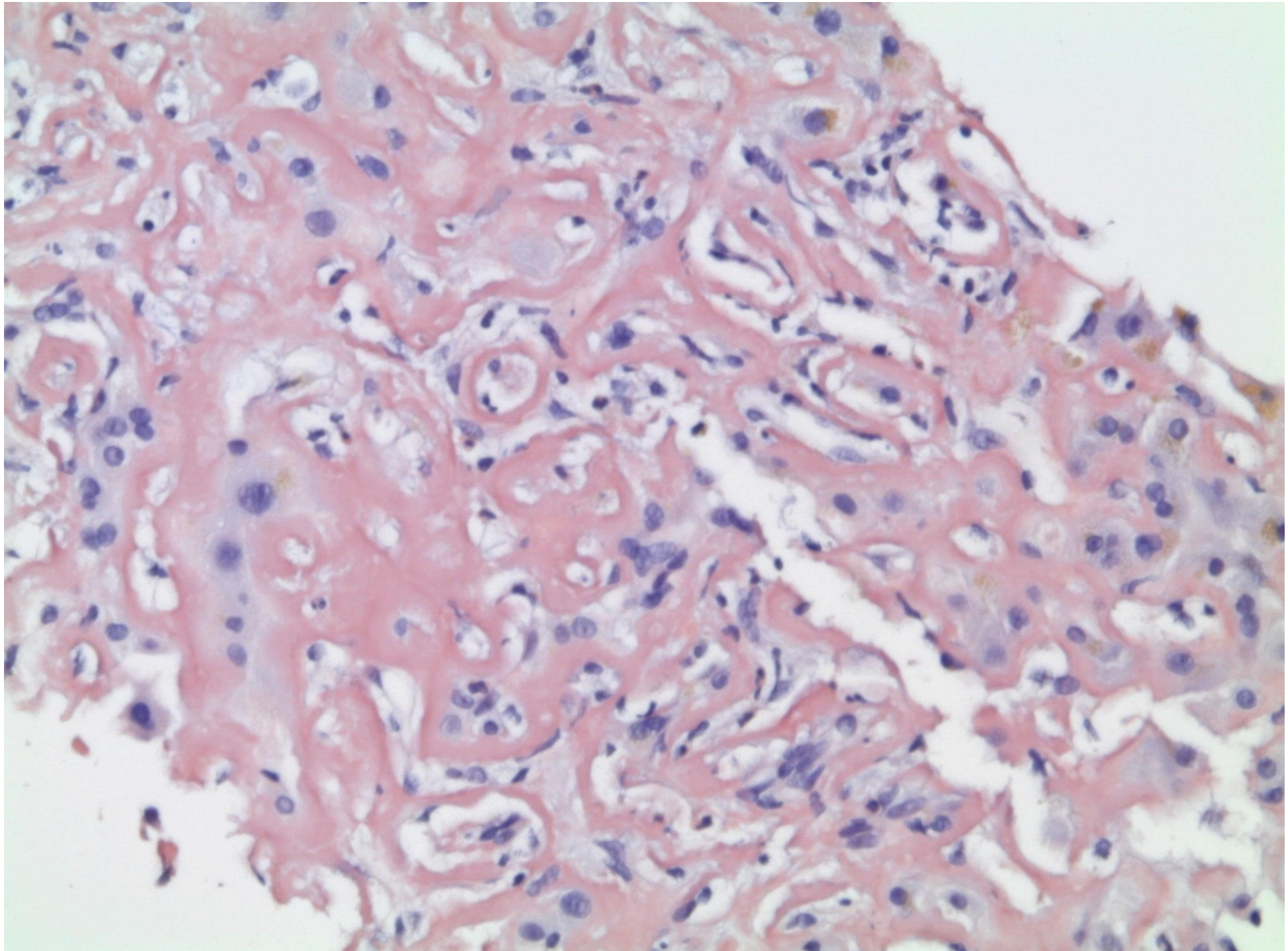
Mendoza YP et al. 2020

Male 69 y, sent to our attention for ascites and suspected alcoholic cirrhosis

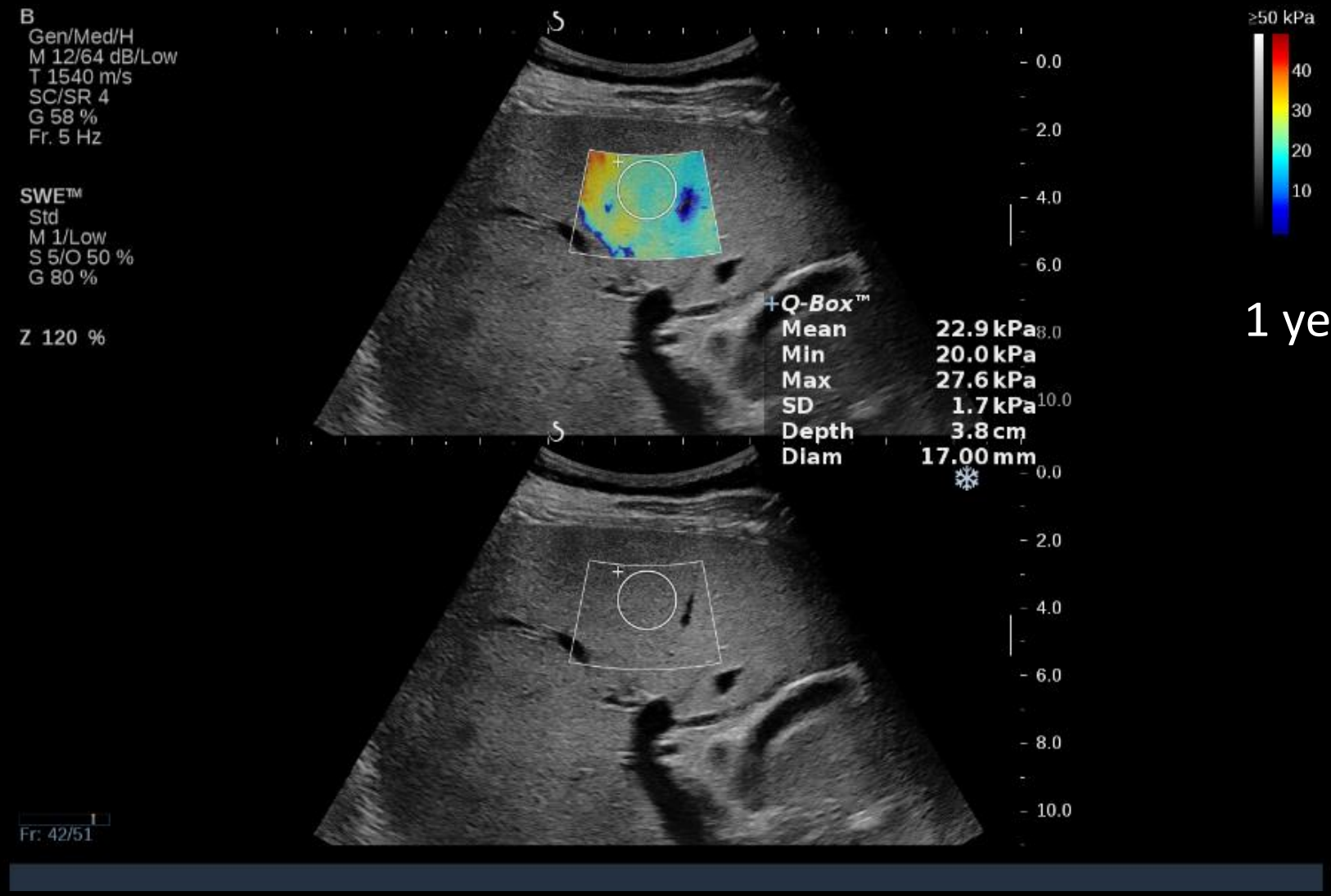



B
Gen/Med/H
M 1/61 dB/Low
T 1540 m/s
SC/SR 5
G 61 %
Fr. 11 Hz
CFI
Gen/Med
Off/WF Med
M 7/P. Low
Scale 18 cm/s
S 2
G 78 %
Z 145 %
0.0
5.0
10.0
18.0
-18.0
cm/s
Fr: 90/90

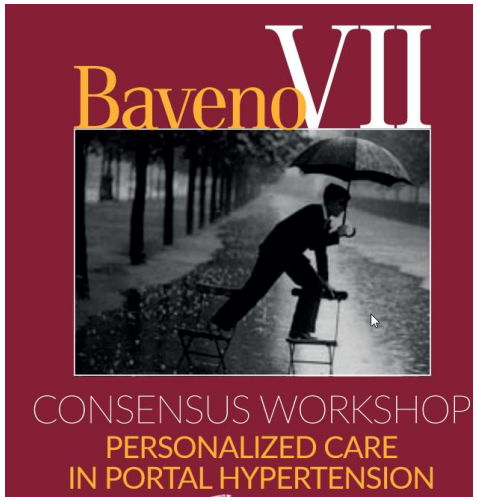
Really cirrhosis?



Congo red staining


B
Gen/Med/H
M 12/64 dB/Low
T 1540 m/s
SC/SR 4
G 58 %
Fr. 5 Hz
SWE™
Std
M 1/Low
S 5/O 50 %
G 80 %
Z 120 %
Q-Box™
Mean 22.9 kPa
Min 20.0 kPa
Max 27.6 kPa
SD 1.7 kPa
Depth 3.8 cm
Diam 17.00 mm
≥50 kPa
40
30
20
10
0.0
2.0
4.0
6.0
8.0
10.0
Fr: 42/51

1 year later



In cACLD to rule-in and rule-out CSPH using elastography is possible but there is a ”grey zone” problem

Relative risk of decompensation & liver-related death

+Plat >150, Baveno VI - avoid endoscopy

+Plat ≥150, Exclude CSPH

“Grey Zone” for CSPH

5 kPa → 10 kPa → 15 kPa → 20 kPa → 25 kPa

Normal

CSPH > 90%

40-60% of patients…

LSM < 15 kPa + Platelet count >150 G/L: very low risk of CSPH (<5%)

LSM > 20 kPa + Plt < 150 G/L
CSPH > 90%

Using ARFI-SWE techniques take advantage of the ”rule of four”

Interpretation of liver stiffness measurement obtained using ARFI-SWE techniques (rule of four)

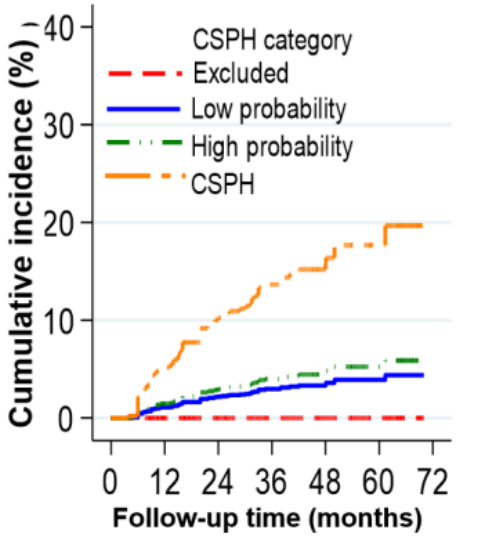
ARFI-SWE LSM	Interpretation
≤5 kPa (1.3 m/s)	High probability of being normal
<9 kPa (1.7 m/s)	In the absence of other known clinical signs, rules out cACLD. If there are known clinical signs, further testing may be needed for confirmation.
9–13 kPa (1.7–2.1 m/s)	Suggestive of cACLD but further testing is required for confirmation.
>13 kPa (2.1 m/s)	Rules in cACLD
>17 kPa (2.4 m/s)	Suggestive of CSPH
>21 kPa (2.6 m/s)	High probability of CSPH

Barr et al. Radiology 2020
Endorsed by Ferraioli et al, WFUMB guidelines 2024

Validation of the ”grey zone”: risk of first clinical decompensation

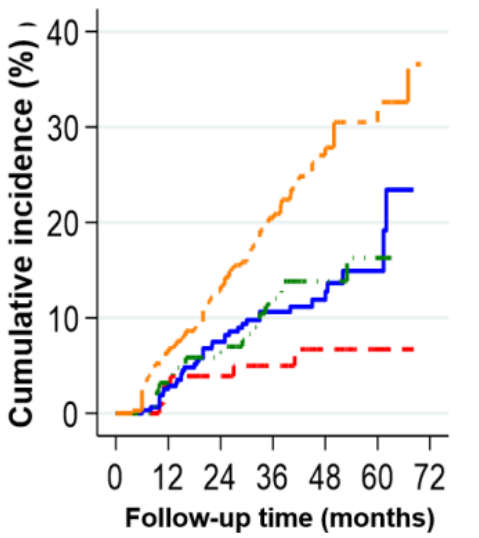
N=1159 cACLD; median FUP 40 months; decompensation in the FUP: 7.2% (n=83)

Liver decompensation



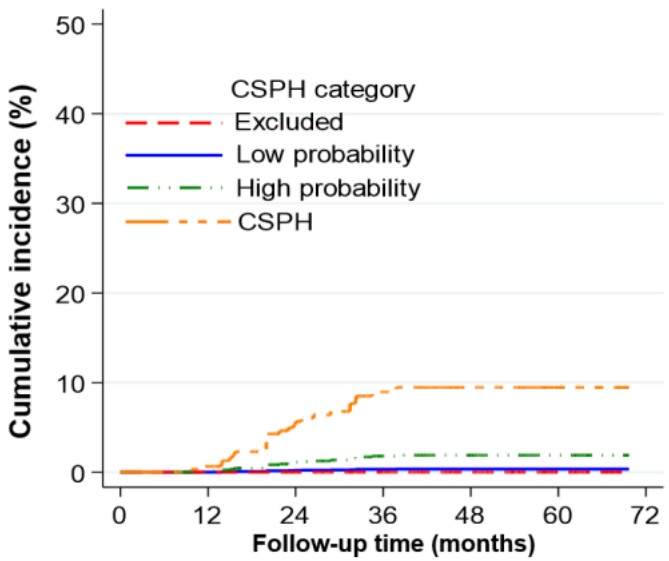
CSPH category	sHR (95% CI)	p-value
Excluded	0	NA
Low probability	Reference	-
High probability	1.3 (0.9-1.8)	0.06
CSPH	5.5 (4.0-7.4)	< 0.01

Liver-related events



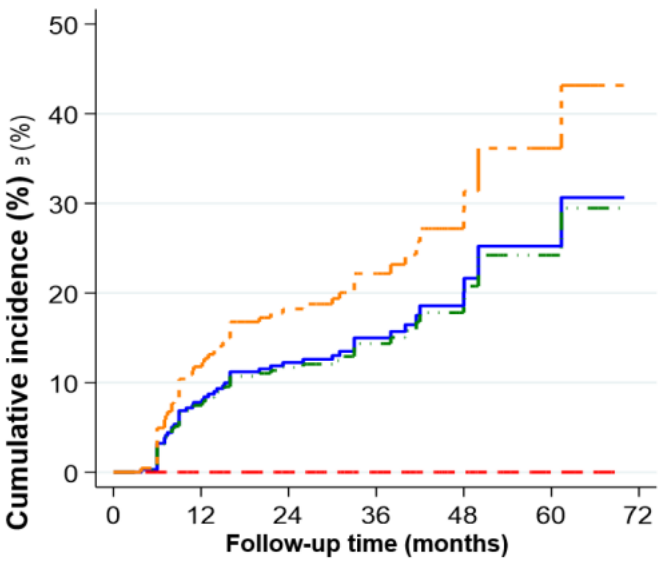
CSPH category	HR (95% CI)	p-value
Excluded	Reference	-
Low probability	2.2 (0.9-4.8)	0.06
High probability	1.8 (0.8-4.4)	0.16
CSPH	3.9 (1.8-8.4)	< 0.01

Viral-related



CSPH category	sHR (95% CI)	p-value
Excluded	Reference	-
Low probability	0	NA
High probability	5.4 (1.0-27.8)	0.04
CSPH	27.9 (4.2-183.3)	< 0.01

Non Viral-related



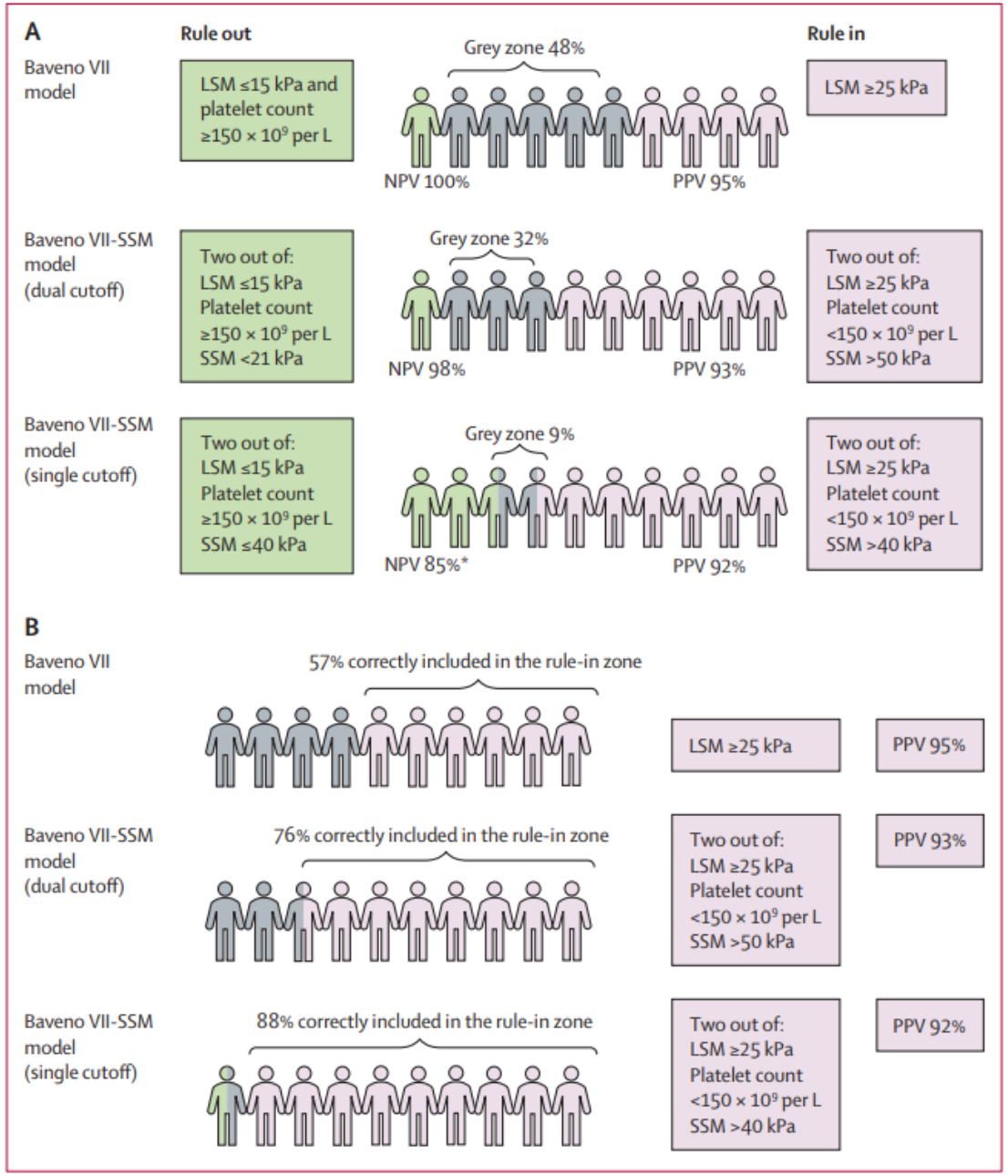
CSPH category	sHR (95% CI)	p-value
Excluded	Reference	-
Low probability	0	NA
High probability	1.0 (0.6-1.5)	0.8
CSPH	1.5 (1.3-1.9)	< 0.01

LSM≥25: CSPH

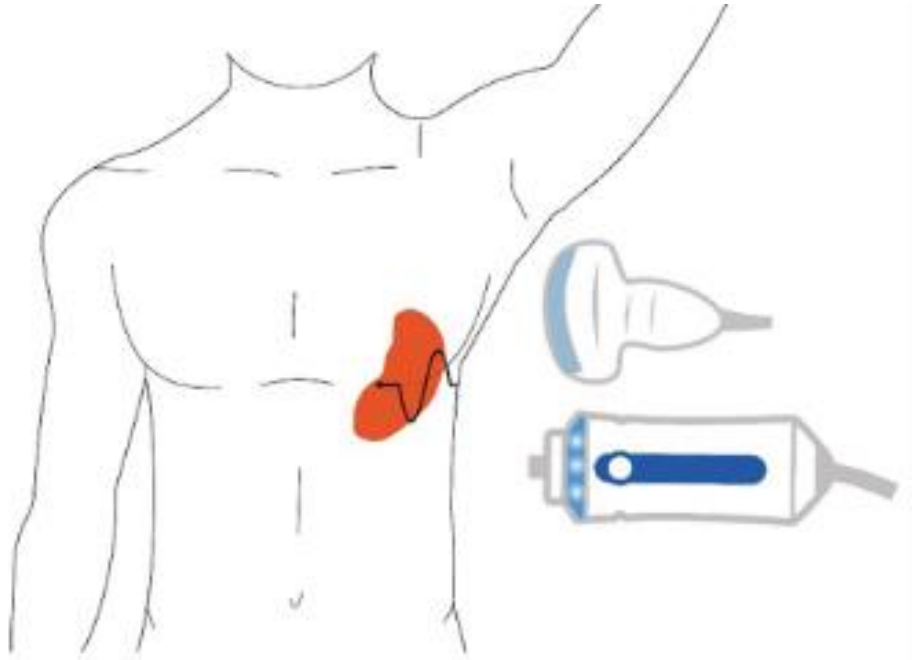
LSM<15 and Plt >150 G/L: exclude CSPH

Grey Zone: all the remaining; high prob CSPH if LSM 20-25 kPa + Plt < 150 G/L or LSM 15-20 + Plt < 110 G/L

Wong YJ et al. Clin Mol Hepatol 2023



Spleen stiffness improves risk stratification for CSPH



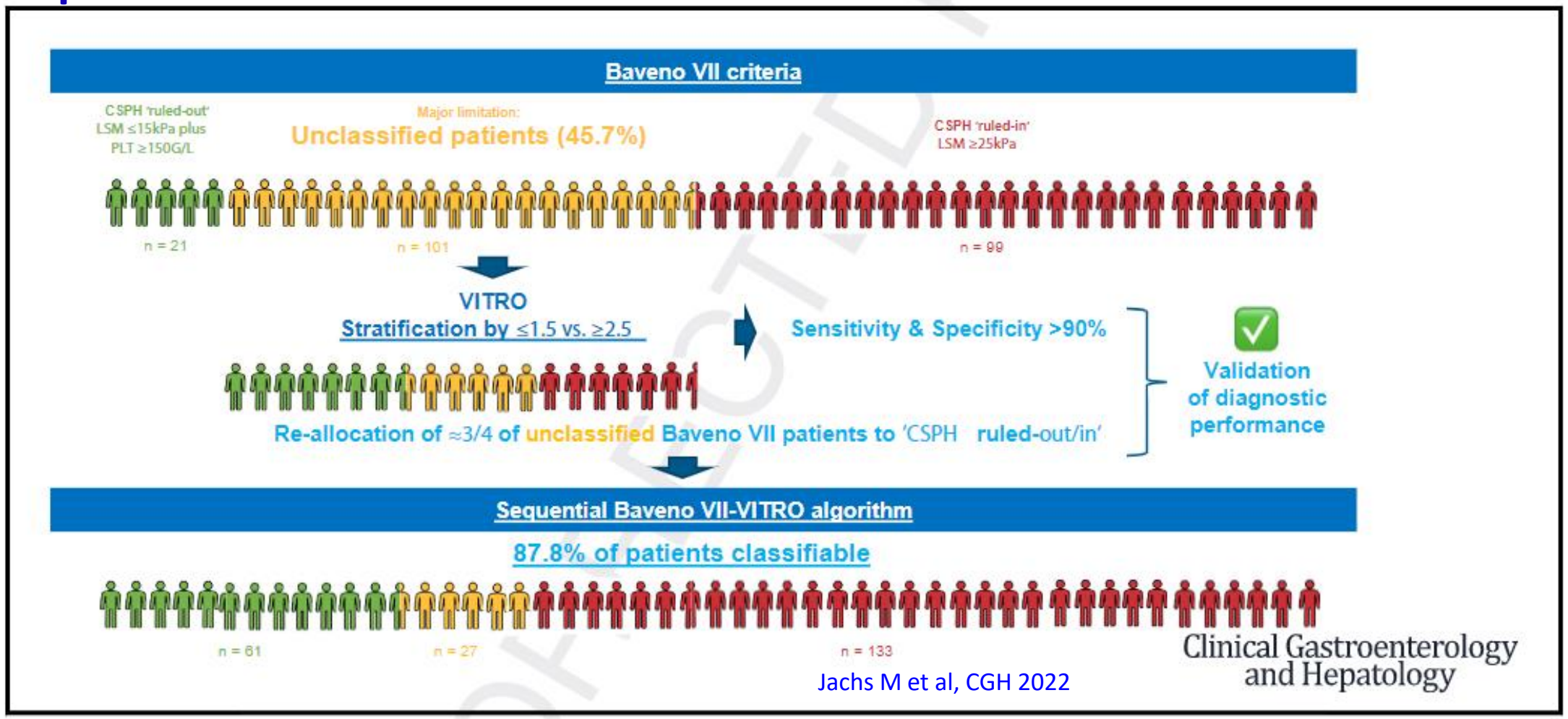
Dajti et al. Lancet Gastroenterol Hepatol 2023

Combined BVII-SSM criteria to rule-out and rule-in CSPH

	Baveno VII–SSM single-cutoff model	Baveno VII–SSM dual-cutoff model
Rule out CSPH if	≥2 of the following criteria: LSM <15 kPa Platelet count ≥150 × 10^9/L SSM ≤40 kPa	≥2 of the following criteria: LSM <15 kPa Platelet count ≥150 ×10^9/L SSM <21 kPa
Rule in CSPH if	≥2 of the following criteria: LSM ≥25 kPa Platelet count <150 × 10^9/L SSM >40 kPa	≥2 of the following criteria: LSM ≥25 kPa Platelet count <150 × 10^9/L SSM >50 kPa

Dajti et al. Lancet Gastroenterol Hepatol 2023; endorsed by WFUMB – Ferraioli et al. UMB 2024

Sequential use of the Baveno VII criteria and VITRO



VITRO score = von Willebrand factor antigen to PLT ratio

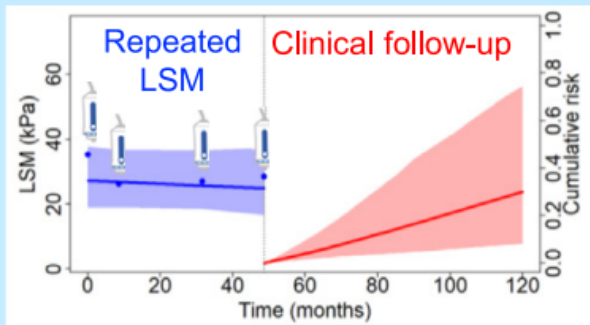
“Dynamic” assessment of liver disease: validation

Dynamics in liver stiffness measurements predict outcomes in advanced chronic liver disease

Cohort

- **2508** patients (757 cACLD)
- **8561** single reliable liver stiffness measurements (LSM)
- **71 months** of clinical follow-up

Joint modeling
updated risk assessment



LSM-dynamics in cACLD

20% increase → 50% increased risk

20% decrease → 50% decreased risk

✓ **Superior** to **single LSM**

✓ **Superior** to **FIB-4** & **MELD dynamics**

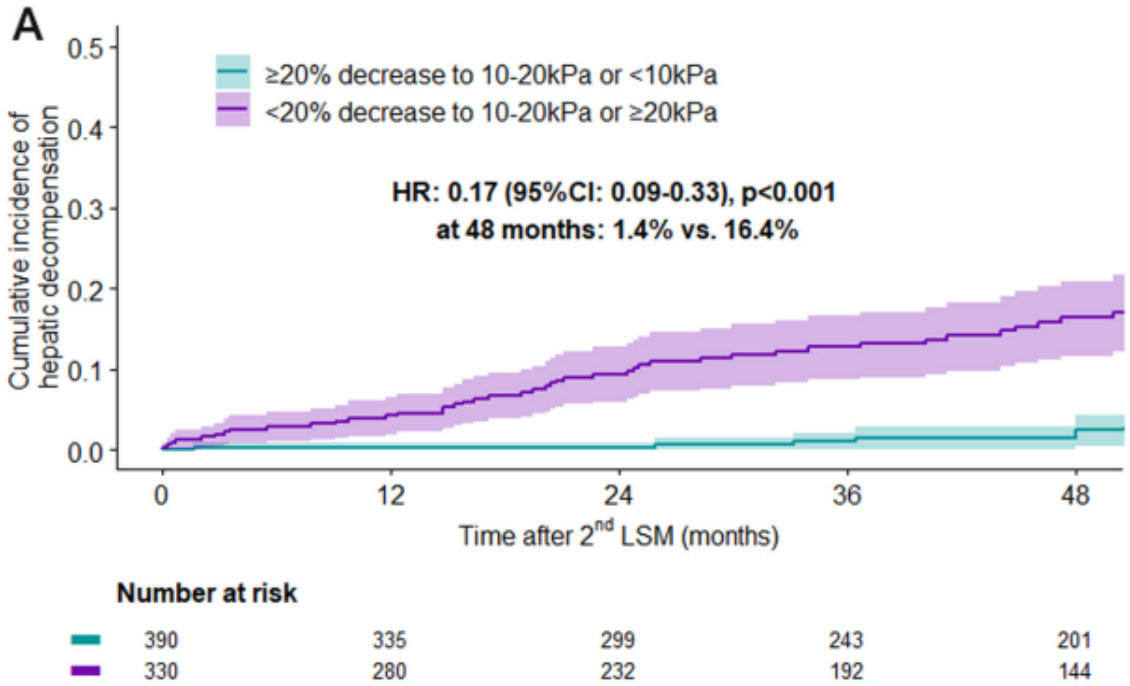
Decrease to <20kPa → favorable outcome

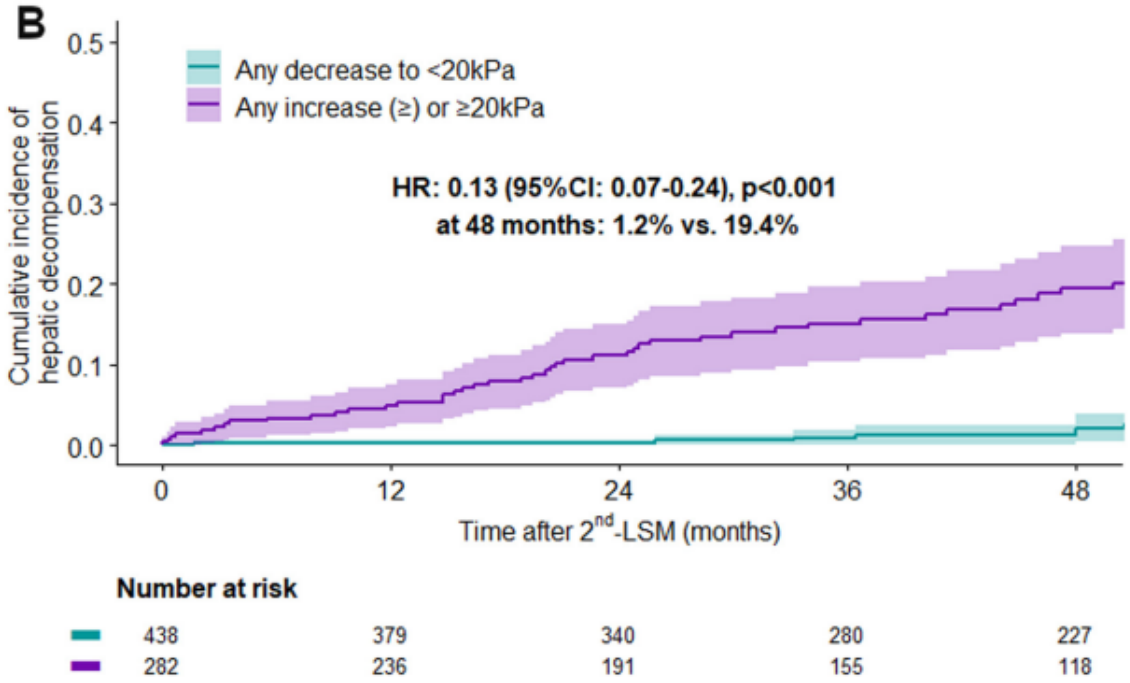
Repeat LSM

- **to monitor progression** (nonACLD)
- **to predict outcome** (cACLD + dACLD)

Gastroenterology

Semmler G et al. Gastroenterology 2023



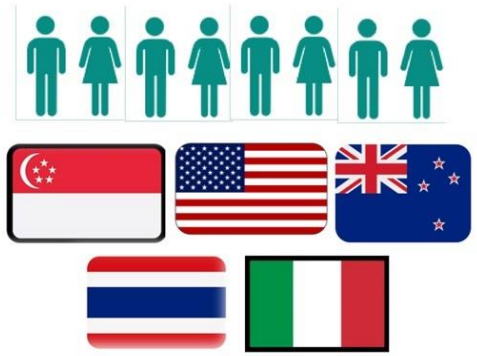


Persistent decrease of LSM below 20 kPa or over 20% is a strong predictor of better outcomes in cACLD of any etiology

Exact timing: still to be determined

Semmler G et al. Gastroenterology 2023

Serial Liver Stiffness Measurements to predict Liver-Related Events in Compensated Advanced Chronic Liver Disease Patients



Intercontinental, multinational cohort of cACLD patients

Repeat LSM

68% → cACLD

32% → "Resolved" cACLD

Using the current LSM only:

Liver-related events: 5.8%

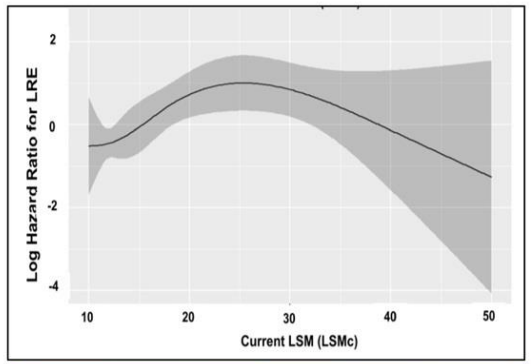
C-statistic: 0.77

Follow-up: 34 months

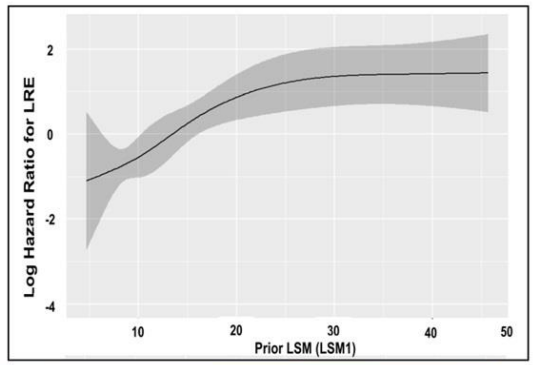
Using both prior & current LSM:

C-statistic: 0.79

Prediction of LRE with prior LSM:

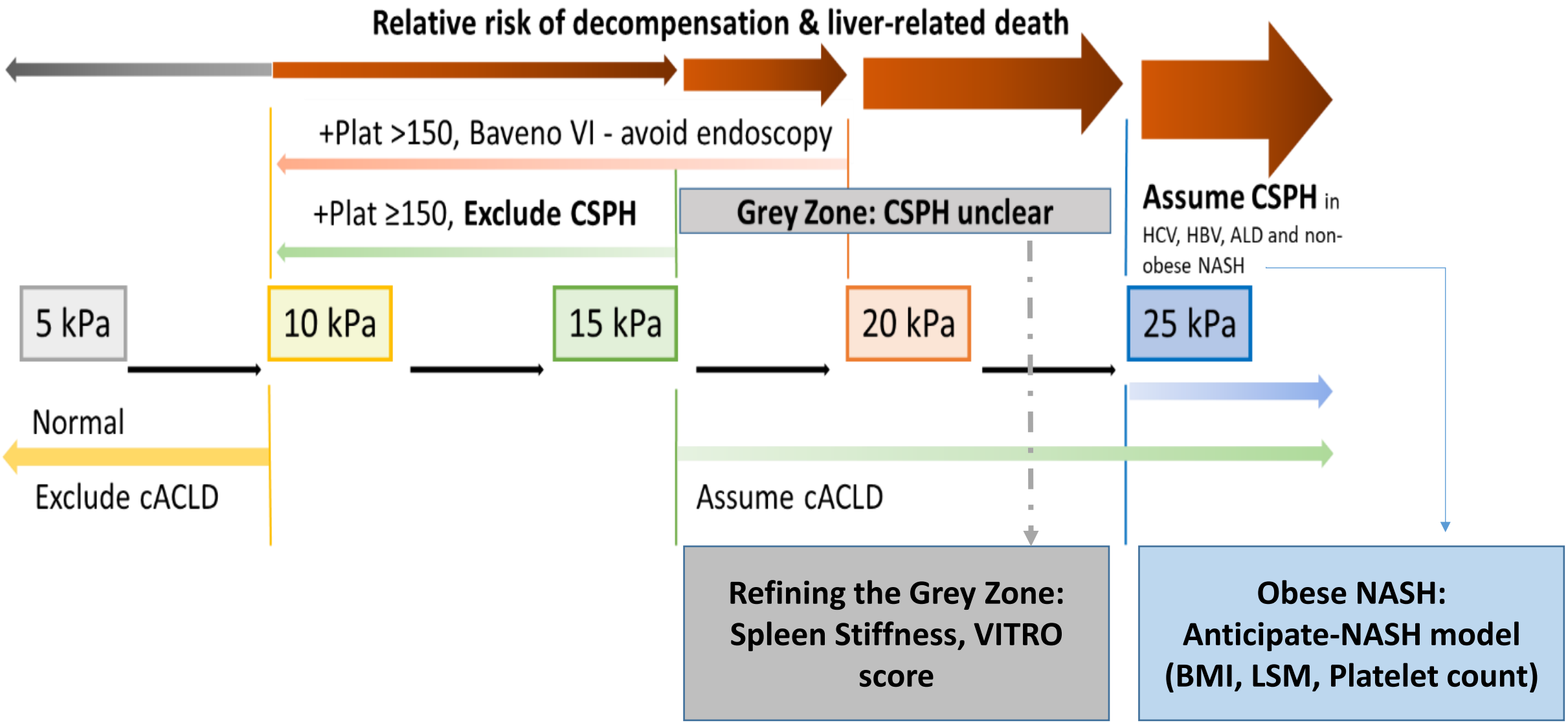


Prediction of LRE with current LSM:

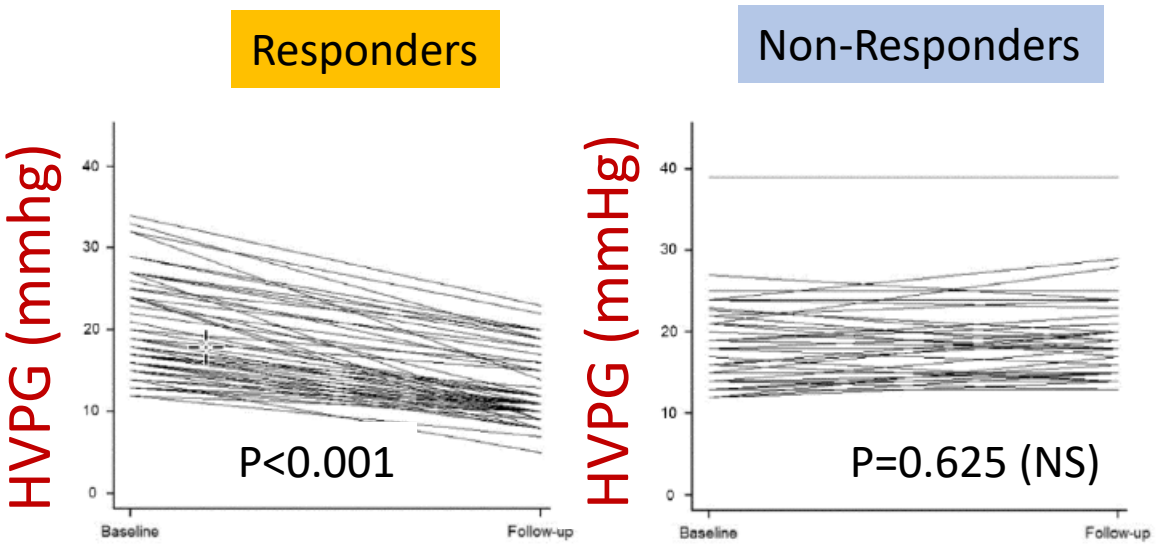


Once the current LSM is known, previous LSM values do not add to the prediction of Liver-related events in cACLD patients

In Summary, as for the Baveno VII criteria, my take is:



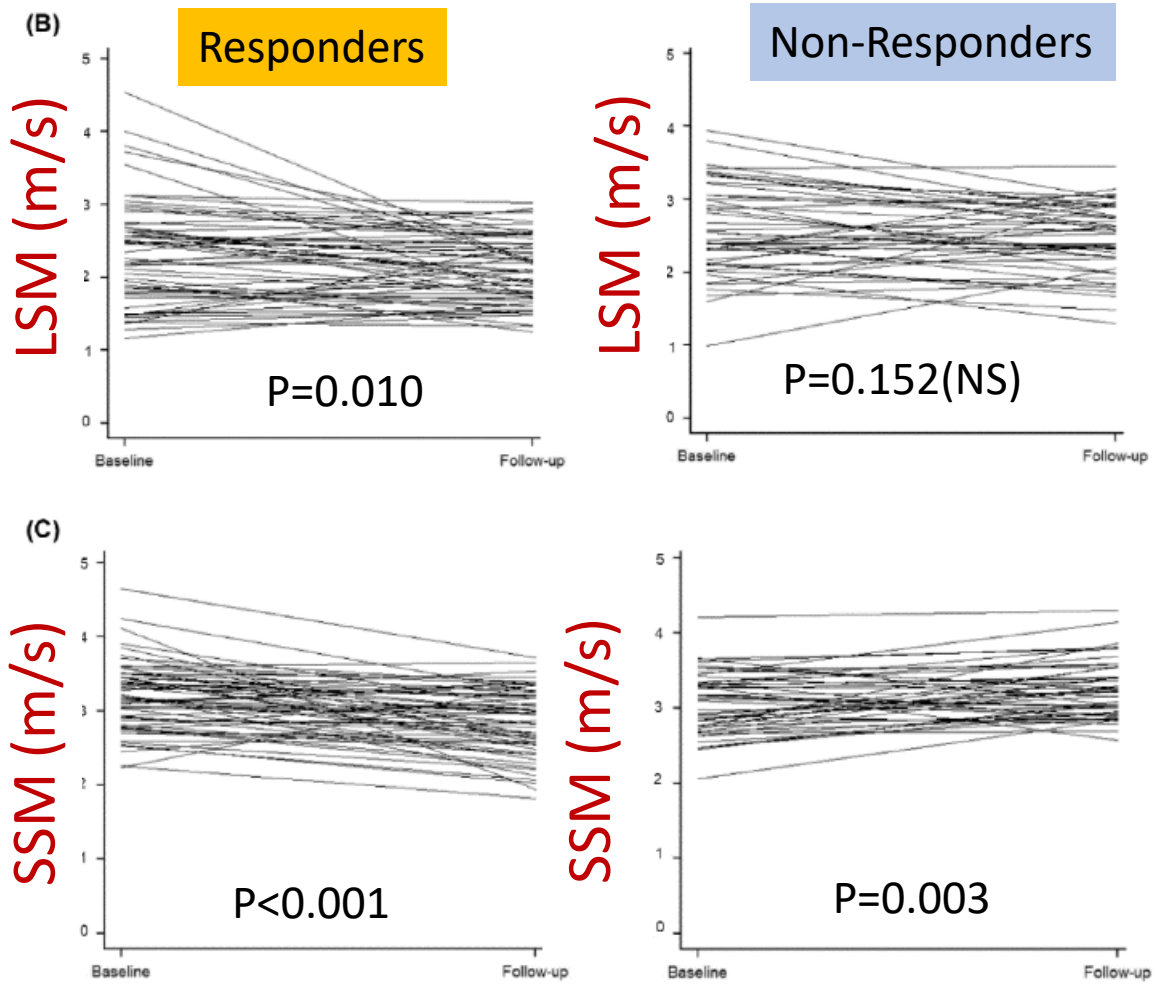
In the setting of compensated patients with CSPH treated with NSBB, the only valuable NIT to assess response is SSM (pSWE)



$$\text{Model}_{\Delta ss}\ (=0.0490-2.8345\times\Delta SS)$$

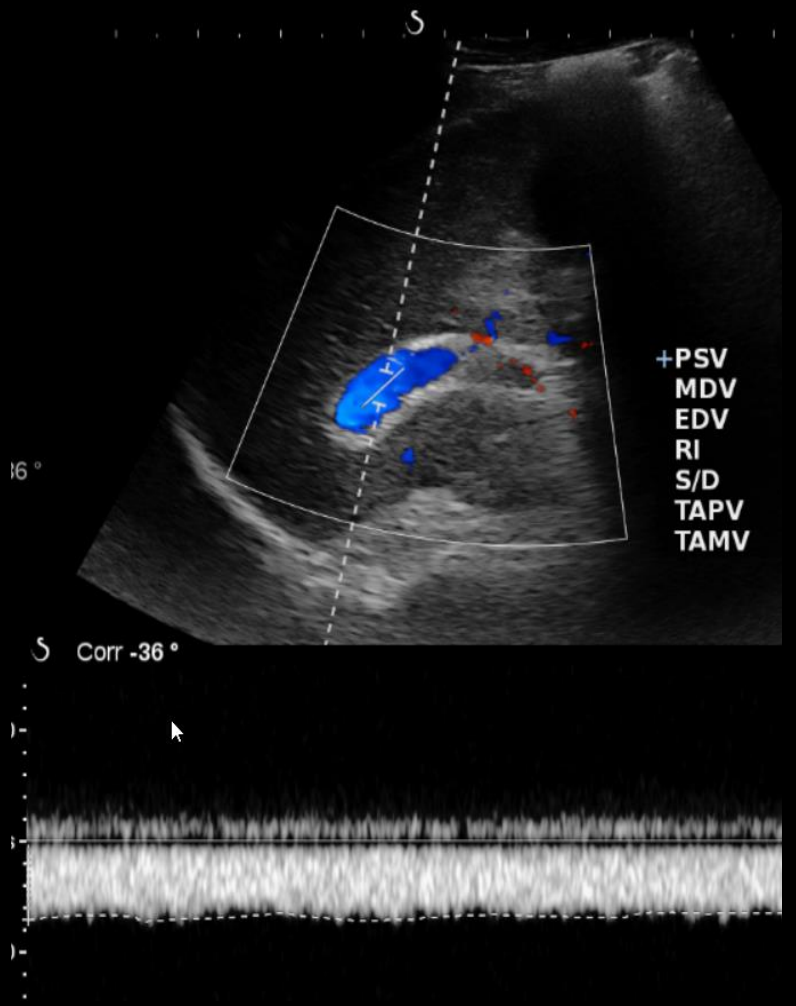
AUROC: training set: 0.801; validation set: 0.848

Kim HY et al J Hepatol 2018



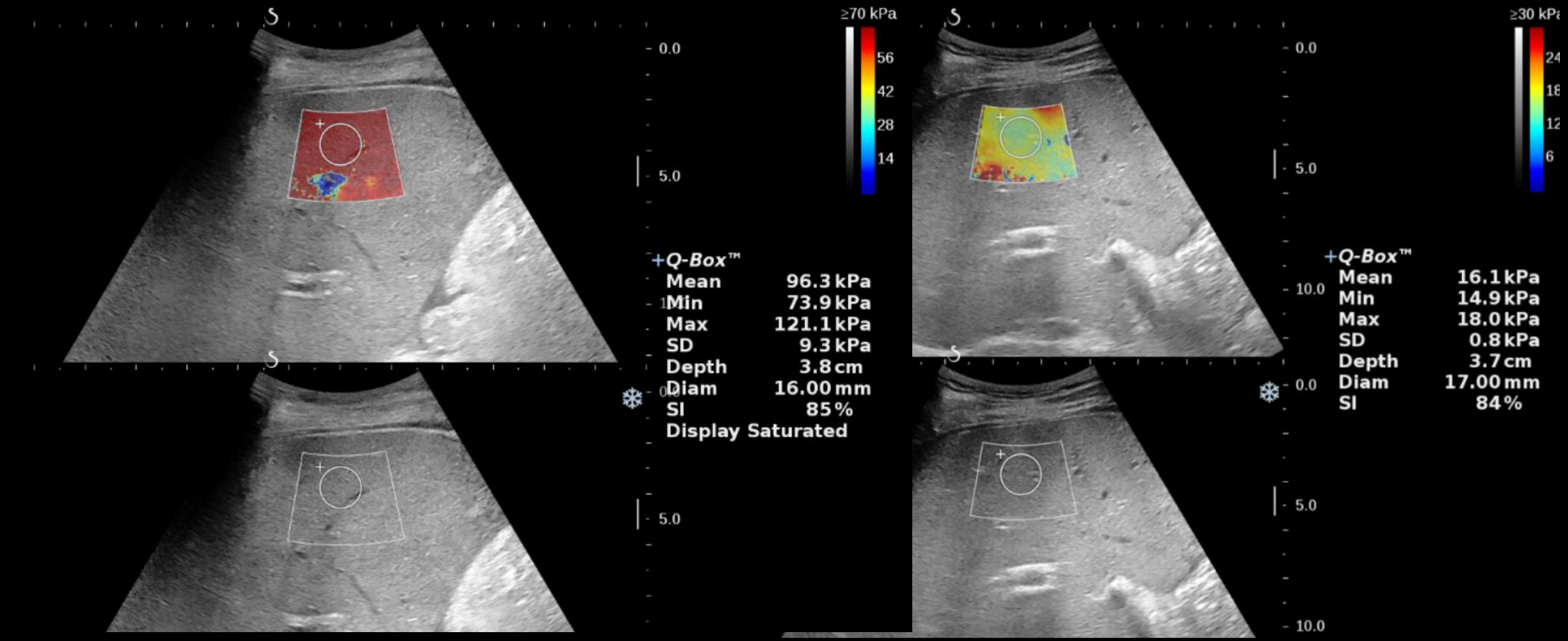
TIPS dysfunction on Doppler US

Rarely visible in B mode and Color-Doppler

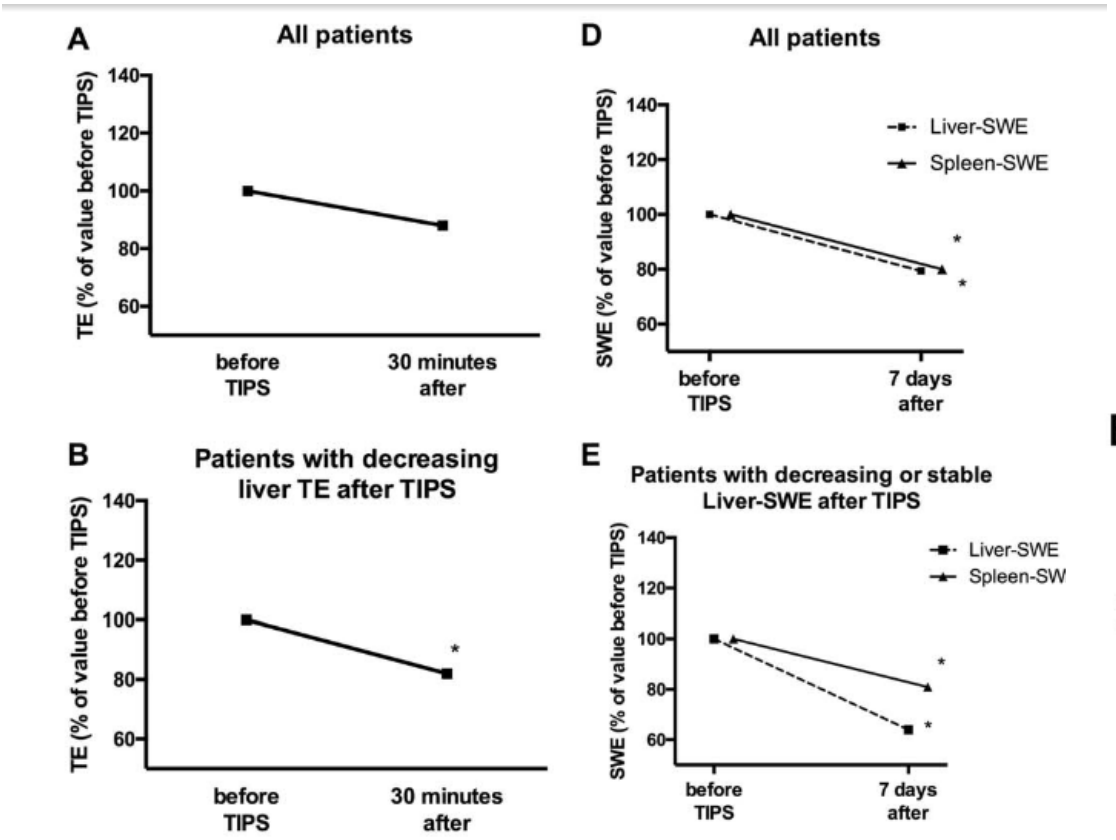


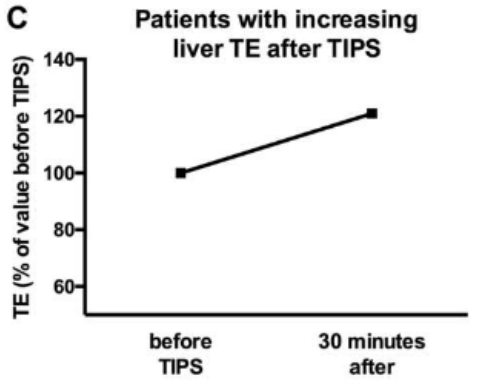
Pulsed Doppler: low portal vein velocity and low intrastent velocity (sometimes aliasing at the stenosis)

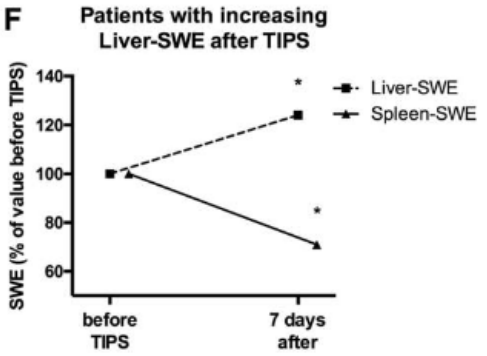
But we can also use elastography as an additional tool

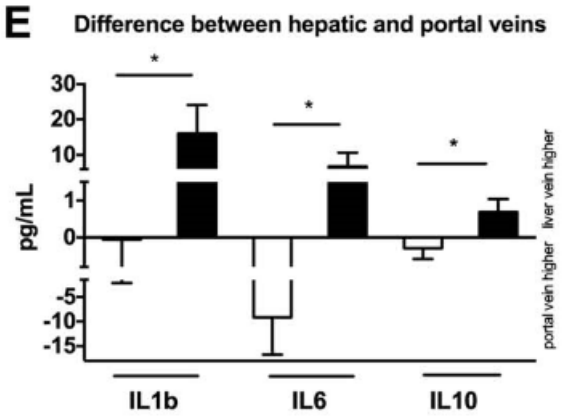


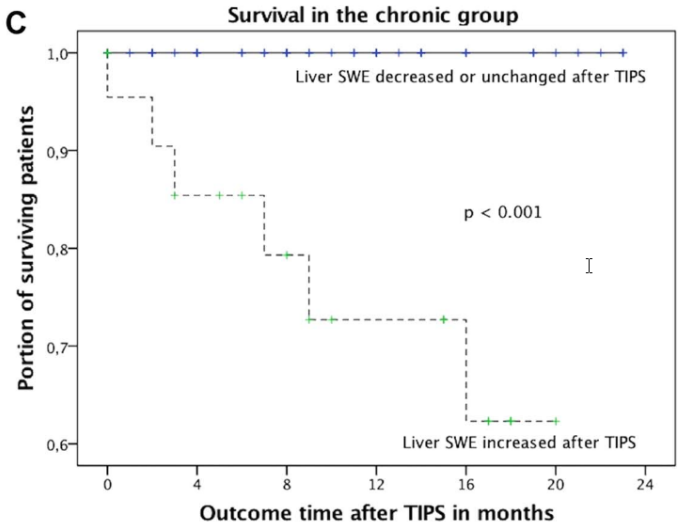
LSM changes correlate with post TIPS prognosis







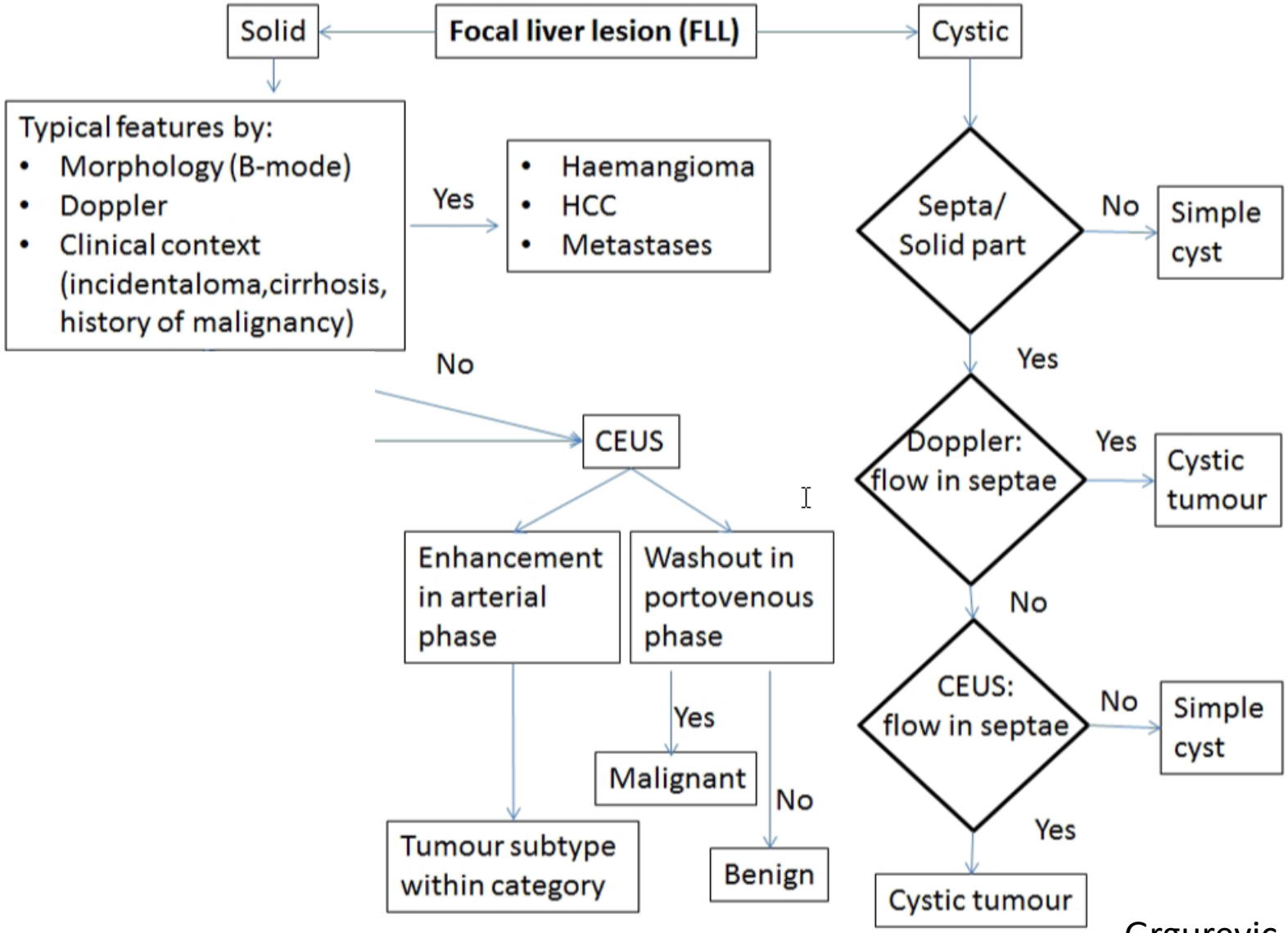




Jansen, Moeller et al Hepatology 2018

An increase in LSM after TIPS is associated with intrahepatic inflammation and poor prognosis

Multiparametric US in the setting of focal liver lesions



Grgurevic et al. Postgrad Med J 2019

Conclusions: Doppler ultrasound continues to play a very important role in the diagnosis and monitoring of chronic liver disease.

Compensated liver disease, unclear origin	cACLD	Decompensated cirrhosis	Focal liver lesions
First approach: Ultrasound and Doppler Ultrasound • If there are signs of portal hypertension (PH): combination of both for the diagnosis of the etiology.	US: HCC screening Doppler US: PVV, P-S collaterals, follow-up Elastography: • Liver: CSPH, prognosis • Spleen: CSPH; possibly hemodynamic response to NSBB; prognosis	Doppler US: Screening of PVT, follow-up of TIPS Elastography: added value in patients with TIPS	Doppler US and CEUS: characterizatio; PVT Elastography possibly

Complementary tools, increasing indications, bright future for both techniques

JOIN US!

Meet the Baveno Cooperation at the Track Hub on Saturday at 10 am

@bavenocoop www.bavenocoop.com

Baveno

25-28 March 2026 VIII