20Η ΔΙΗΜΕΡΙΔΑ
«ΕΞΕΛΙΞΕΙΣ ΣΤΗΝ ΚΑΡΔΙΑΓΓΕΙΑΚΗ ΑΠΕΙΚΟΝΙΣΗ 2023»
10-11 ΦΕΒΡΟΥΑΡΙΟΥ 2023
ΑΜΦΙΘΕΑΤΡΟ Γ.Ν.Α. ΙΠΠΟΚΡΑΤΕΙΟ / ΑΘΗΝΑ
ΠΡΟΕΔΡΟΙ ΟΡΓΑΝΩΤΙΚΗΣ ΕΠΙΤΡΟΠΗΣ: Κ. Τσιούφης, Κ. Αγγέλη, Σ. Μπρίλη

DSE IN ESLD

IS IT USEFUL?

Γιάννης Δημητρόγλου
Ειδικευόμενος,
Α' Πανεπιστημιακή Καρδιολογική Κλινική,
ΓΝΑ Ιπποκράτειο

THE CARDIAC OUTPUT AT REST IN LAENNEC'S CIRRHOSIS [1]

By HENRY J. KOWALSKI [2] AND WALTER H. ABELMANN

(*From the Thorndike Memorial Laboratory, Second and Fourth Medical Services* [*Harvard*], *and the Department of Medicine, Harvard Medical School, Boston, Mass.*)

(Submitted for publication April 29, 1953; accepted June 12, 1953)

THE HEMODYNAMIC RESPONSE TO EXERCISE IN PATIENTS WITH LAENNEC'S CIRRHOSIS [1]

By WALTER H. ABELMANN,[2] HENRY J. KOWALSKI,[3] AND WILLIAM F. McNEELY

Review

Preoperative Evaluation of Coronary Artery Disease in Liver Transplant Candidates: Many Unanswered Questions in Clinical Practice

Maria Bonou [1], Sophie Mavrogeni [2], Chris J. Kapelios [1,*], Marina Skouloudi [1], Constantina Aggeli [3], Evangelos Cholongitas [4], George Papatheodoridis [5,†] and John Barbetseas [1,†]

Hyperdynamic circulation

- Increase SV
- Decrease PVR
- Increase HR
- Increase CO

Remodeling

- Increase LVEDV
- Increase LAV
- Increase RVEDV
- Preserved LVEF

Impaired response to physical and pharmacological stress

- Increased mortality during LT

Table 1. Baseline Hemodynamic Changes in Patients With ESLD

Hemodynamic Parameter	Changes
Systemic circulation	Plasma volume ↑, total blood volume ↑, noncentral blood volume ↑, central blood volume ↓ ↔, arterial blood pressure ↔ ↓, systemic vascular resistance ↓, heart rate ↑, cardiac output ↑
Cardiac hemodynamics	LA volume ↑, LV volume ↑ ↔, right atrial volume ↔ ↑ ↓, RV volume ↔ ↑ ↓, RA pressure ↔ ↑, RV end-diastolic pressure ↔, pulmonary capillary wedge pressure ↔, LV end-diastolic pressure ↑ ↔
Pulmonary circulation	Pulmonary blood flow ↑, pulmonary artery pressure ↔ ↑, pulmonary vascular resistance ↔ ↑ ↓

↔ = No change; ↑ = increase; ↓ = decrease; ESLD = end-stage liver disease; LA = left-atrial; LV = left-ventricular; RA = right-atrial; RV = right ventricular.

Garg A, Amstrong W, JACC Cardiovasc Imag 2019

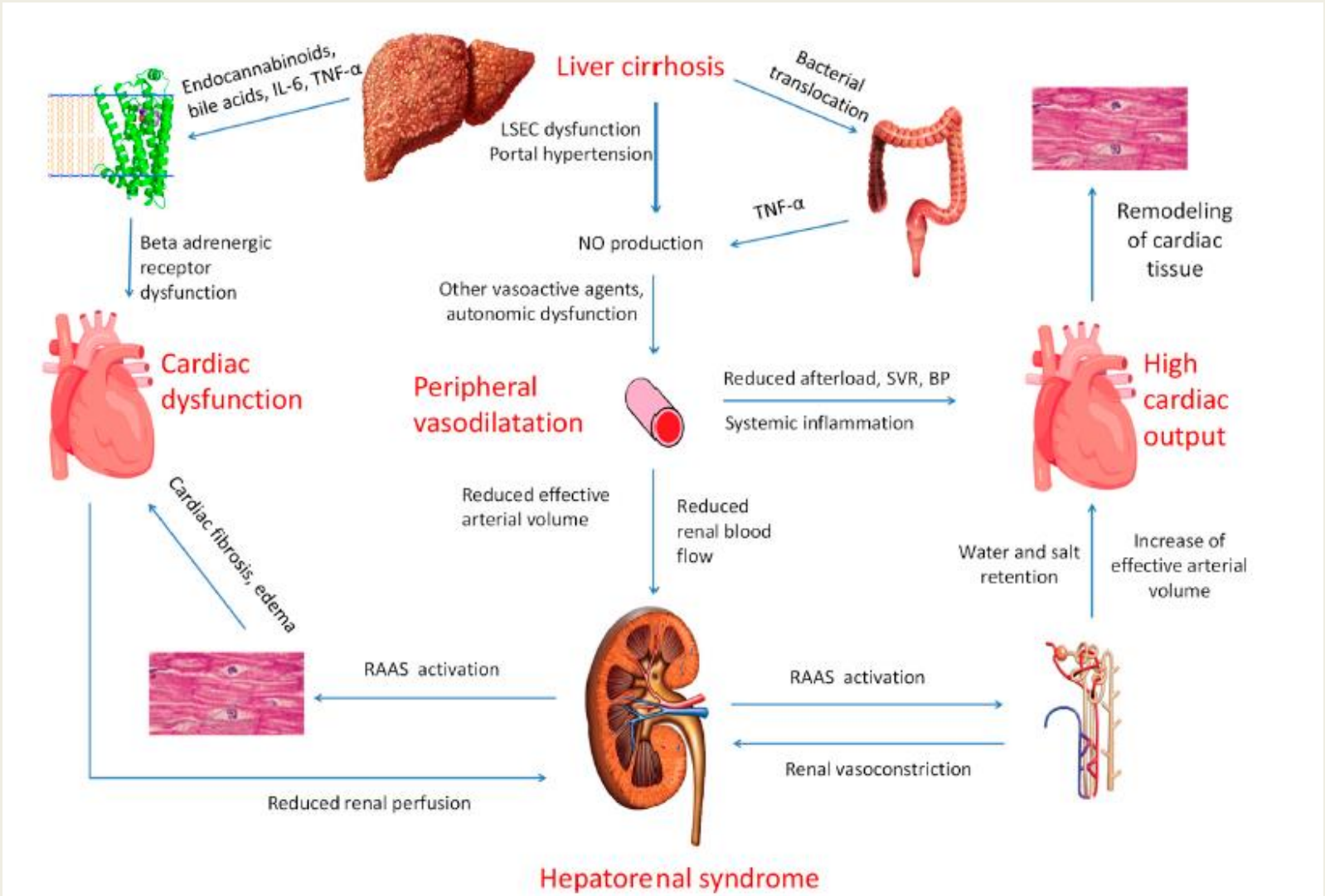
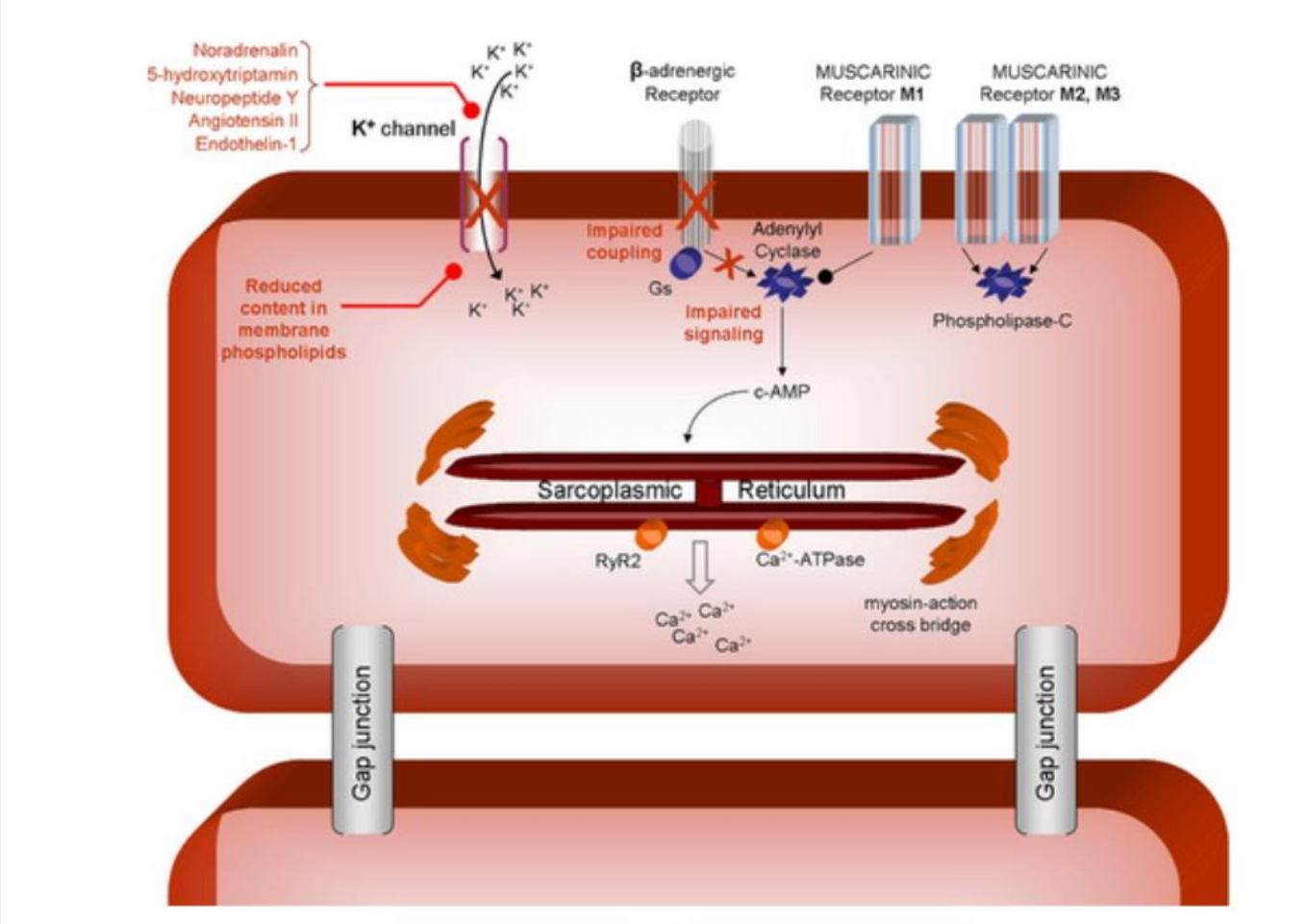


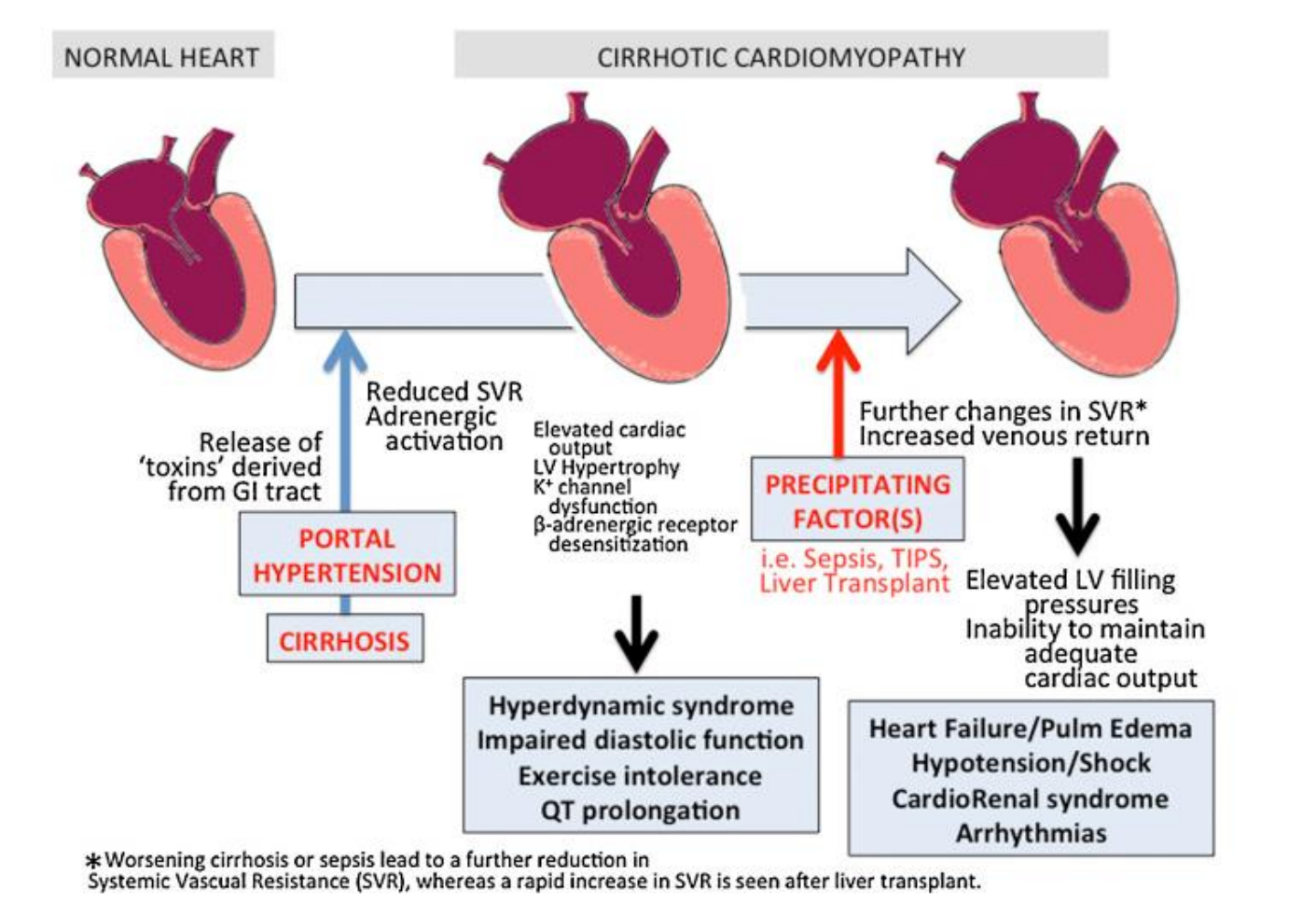
Figure 1. Presentation of pathophysiological interactions between the liver the heart and the kidneys for the pathogenesis of cirrhotic cardiomyopathy. RAAS: renin-angiotensin-aldosterone system, LSEC: liver sinusoidal endothelial cells.

Pathophysiology of LV Dysfunction in LC



JACC, 2010

Pathophysiology of LV Dysfunction in LC



Jardi et al, 2015

Table 1. Cirrhotic cardiomyopathy diagnostic criteria according to the World Gastroenterology Organisation (Montreal 2005).

Cirrhotic patient with	1. Abnormal contractile response to stress 2. Diastolic dysfunction 3. Absence of another clinically significant cardiopulmonary Disease
Systolic function (at least 1)	➢ Blunted increase in cardiac output with exercise, volume challenge or pharmacologic stimuli. ➢ Resting left ventricular ejection fraction (LVEF) < 55%
Diastolic function (at least 1)	➢ E/A ratio < 1 (age corrected) ➢ Prolonged mitral deceleration time (DT > 200 ms) ➢ Prolonged isovolumetric relaxation time (>80 ms)
Supportive criteria	➢ Abnormal chronotropic response to stress ➢ Electromechanical uncoupling ➢ Dysychrony ➢ Prolonged QTc interval ➢ Enlarged left atrium ➢ Increased left ventricular mass ➢ Increased BNP or proBNP

and then…

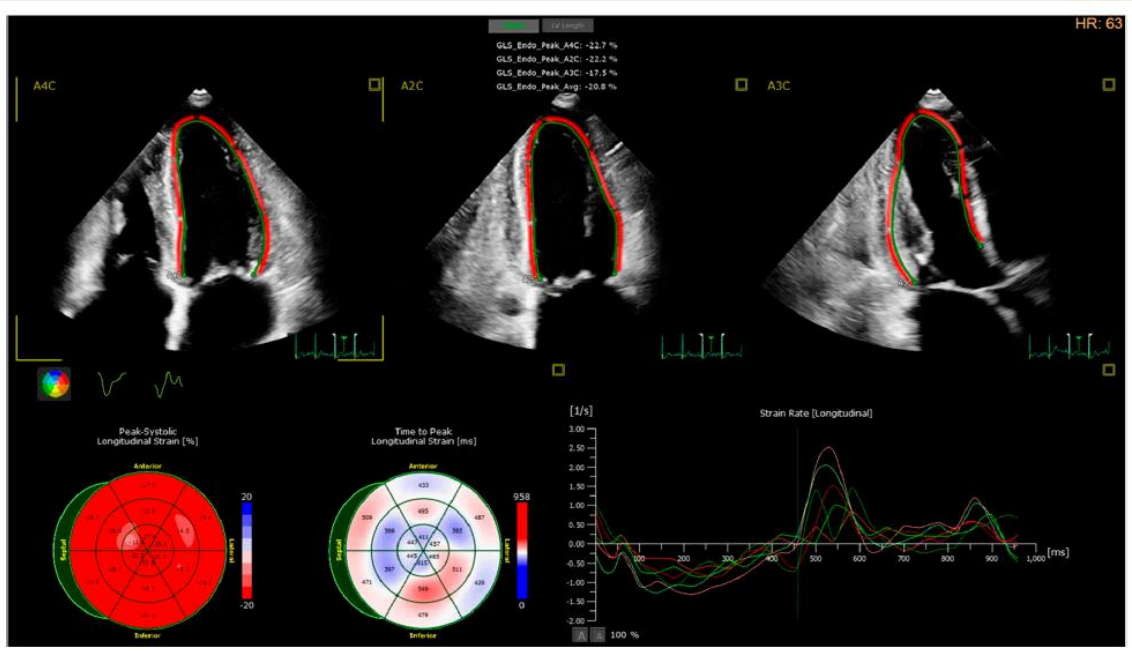
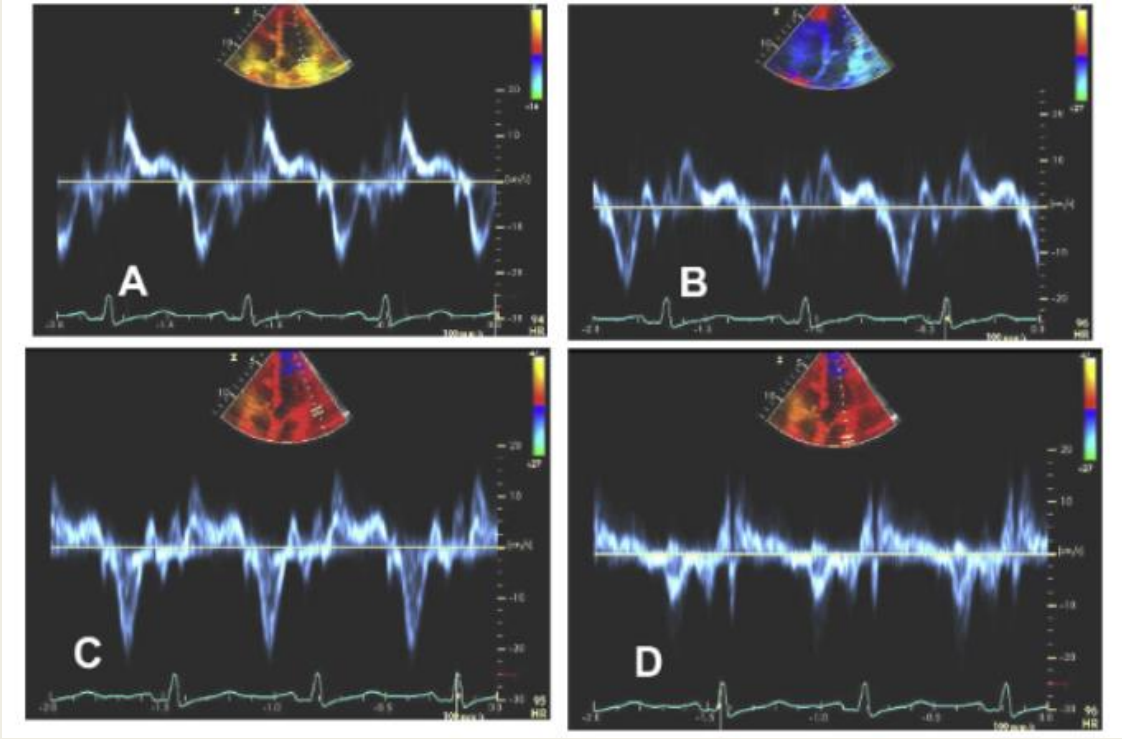


Figure 2. Speckle tracking echocardiography. Semiautomatic calculation of Global longitudinal strain (GLS), with possible simultaneous identification of segmental wall motion abnormalities (left low), dysychrony (middle low) and diastolic function abnormalities-strain rate (right low).

Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging



HEPATOLOGY

AASLD

SPECIAL ARTICLE | HEPATOLOGY, VOL. 71, NO. 1, 2020

Redefining Cirrhotic Cardiomyopathy for the Modern Era

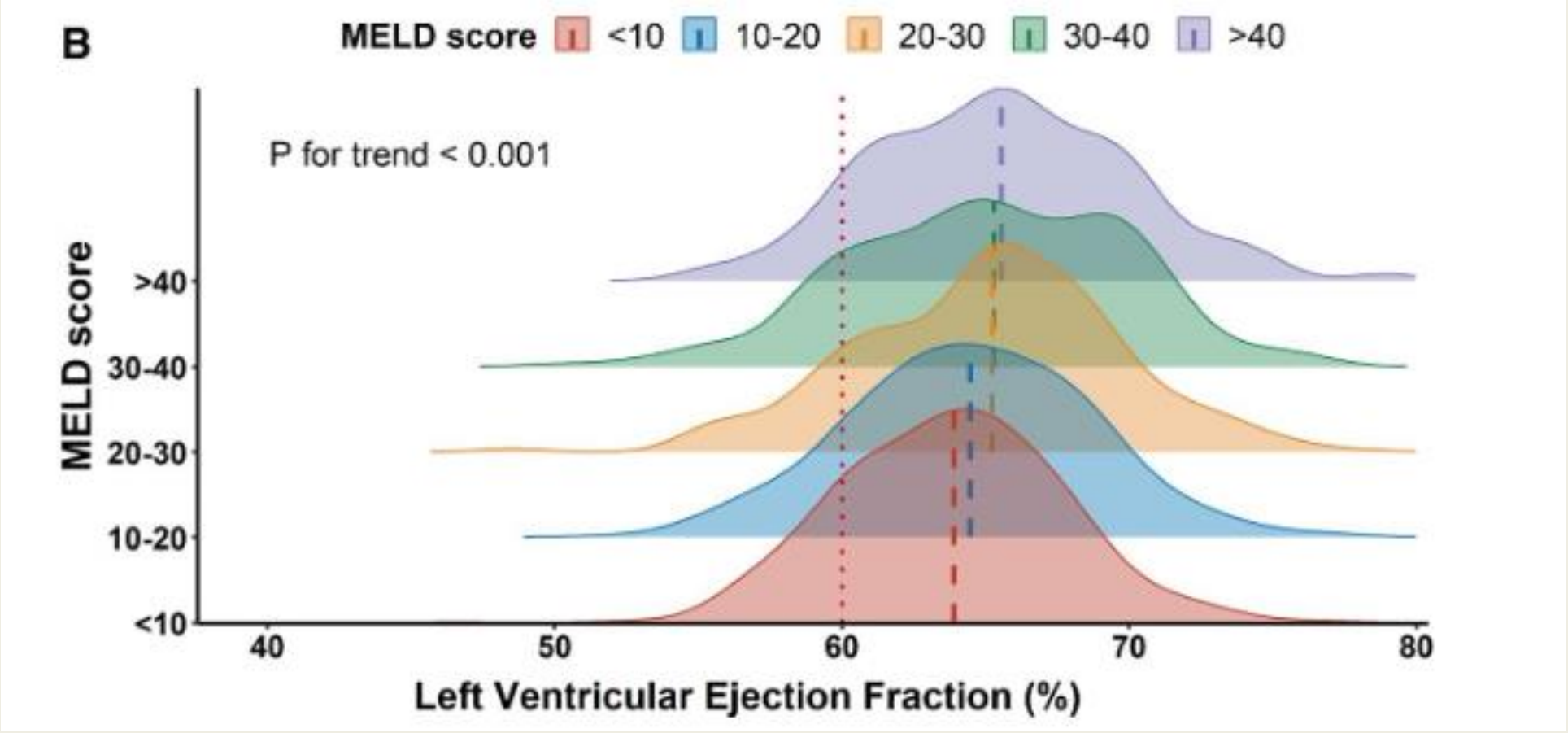
Manhal Izzy [1*] Lisa B. VanWagner [2,3*] Grace Lin,[4] Mario Altieri,[5] James Y. Findlay,[6] Jae K. Oh,[4] Kymberly D. Watt,[7] and Samuel S. Lee[8]; on behalf of The Cirrhotic Cardiomyopathy Consortium

Systolic Dysfunction	Advanced Diastolic Dysfunction	Areas for Future Research
Any of the following • LV ejection fraction ≤50% • Absolute* GLS <18%	≥3 of the following • Septal e′ velocity <7 cm/s • E/e′ ratio ≥15 • LAVI >34 ml/m2 • TR velocity >2.8 m/s	• Abnormal chronotropic or inotropic response§ • Electrocardiographic changes • Electromechanical uncoupling • Myocardial mass change • Serum biomarkers • Chamber enlargement • CMRI

GLS, global longitudinal strain; e′, early diastolic mitral annular velocity; E/e′, ratio of mitral peak velocity of early filling to early diastolic mitral annular velocity; CMRI, cardiac magnetic resonance imaging.

Izzy et al, Hepatology 2020

LVEF in LC



Kwon et al,Hepatology, 2020

Evaluation of subclinical ventricular systolic dysfunction assessed using global longitudinal strain in liver cirrhosis: A systematic review, meta-analysis, and meta-regression

Denio A. Ridjab[1]*, Ignatius Ivan[2], Fanny Budiman[2], Riki Tenggara[3]

Study or Subgroup	Cirrhotic Patients Mean [%]	SD [%]	Total	Non-Cirrhotic Patients Mean [%]	SD [%]	Total	Weight	Mean Difference IV, Random, 95% CI [%]
Kim HM (2020)	24.2	2.7	33	18.6	2.2	17	5.4%	5.60 [4.21, 6.99]
Zamirian M (2019)	22.6	2.4	20	19.2	1.9	10	5.3%	3.40 [1.82, 4.98]
von Köckritz F (2021)	21.39	4.06	80	18.73	2.95	30	5.4%	2.66 [1.28, 4.04]
Huang CH (2019)	21.43	1.51	80	20.77	3.04	29	5.5%	0.66 [-0.49, 1.81]
Rimbas RC (2017)	20.9	3	46	20.7	2.8	46	5.5%	0.20 [-0.99, 1.39]
Koç DÖ (2020)	19.42	2.83	50	19.49	2.33	33	5.5%	-0.07 [-1.19, 1.05]
Sampaio F (2015)	18.47	3.47	36	18.57	4.02	8	4.5%	-0.10 [-3.11, 2.91]
Ibrahim MG (2020)	20.83	7.25	50	21.67	8.4	50	4.4%	-0.84 [-3.92, 2.24]
Hassan AAA (2019)	19.5	2.7	45	20.7	4.3	30	5.2%	-1.20 [-2.93, 0.53]
Chen Y (2016)	18.6	2.6	103	20.1	2.8	48	5.6%	-1.50 [-2.44, -0.56]
İnci SD (2019)	16	3.2	40	17.6	2.2	26	5.4%	-1.60 [-2.90, -0.30]
Sampaio F (2013)	20.19	2.38	100	22.1	1.54	18	5.6%	-1.91 [-2.76, -1.06]
Hammami R (2017)	19.8	2.8	80	22.01	2.6	80	5.6%	-2.21 [-3.05, -1.37]
Özdemir E (2019)	19.9	3.4	40	22.8	1.9	40	5.5%	-2.90 [-4.11, -1.69]
Novo G (2018)	18.3	3.23	39	21.3	1.46	39	5.5%	-3.00 [-4.11, -1.89]
Isaak A (2020)	18.5	4	42	22.5	3.6	18	5.1%	-4.00 [-6.06, -1.94]
Anish PG (2019)	19.52	2.41	55	23.66	2.31	30	5.5%	-4.14 [-5.18, -3.10]
Altekin RE (2014)	20.57	2.1	38	28.74	3.11	37	5.5%	-8.17 [-9.37, -6.97]
Al-Hwary S (2015)	19.98	7.65	40	29.5	5.92	20	4.1%	-9.52 [-13.03, -6.01]
Total (95% CI)			**1017**			**609**	**100.0%**	**-1.43 [-2.79, -0.07]**

Heterogeneity: Tau² = 8.41; Chi² = 384.29, df = 18 (P < 0.00001); I² = 95%
Test for overall effect: Z = 2.06 (P = 0.04)

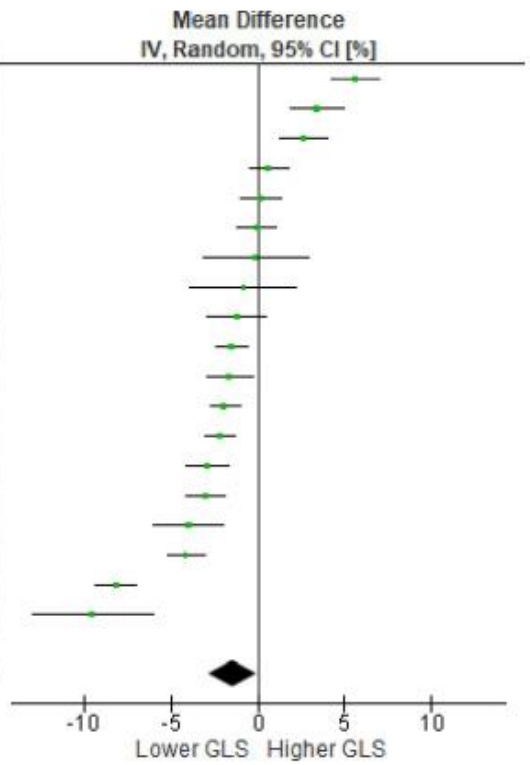
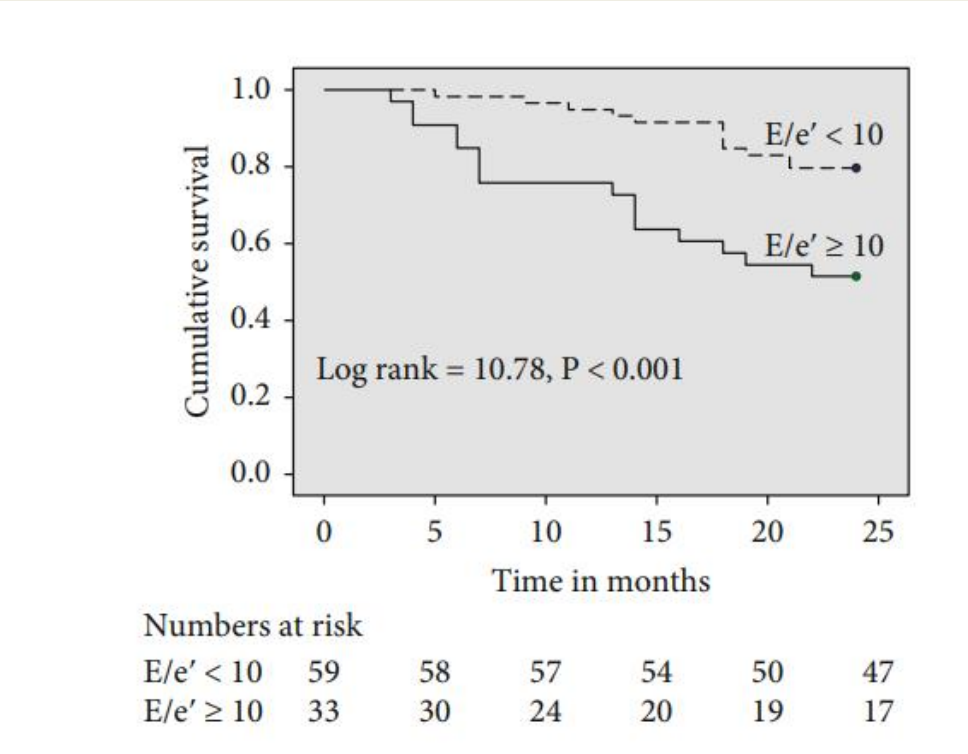
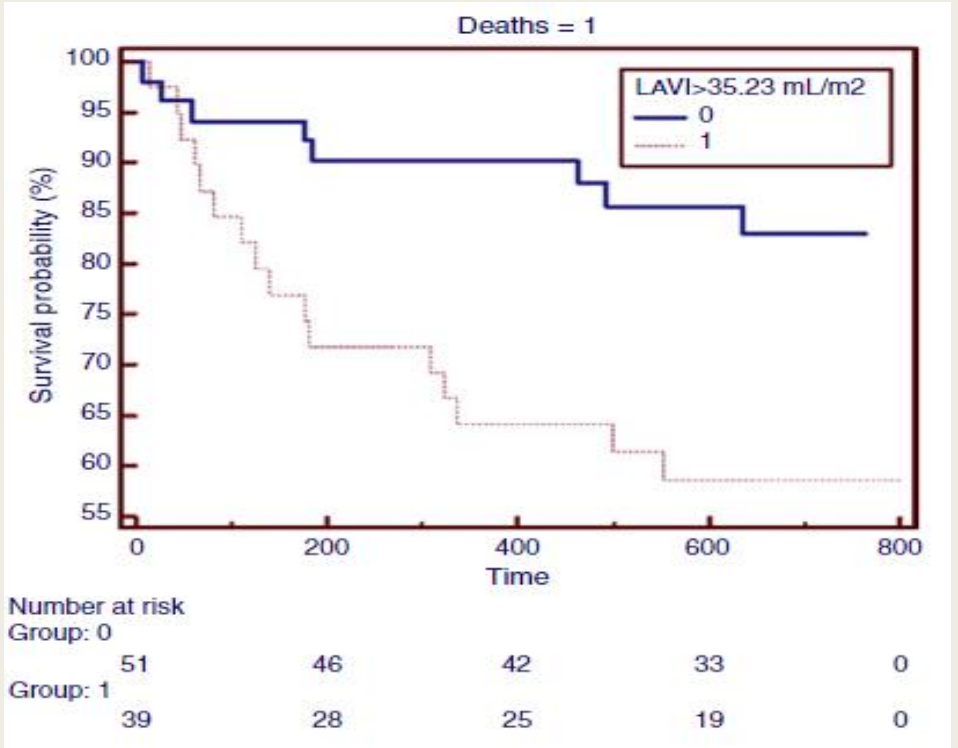


Fig 2. Mean difference of left ventricular global longitudinal strain in patiens with cirrhosis versus patients without cirrhosis evaluated using the random effect model. SD, standard deviation; IV, inverse variance; CI, confidence interval; df, degrees of freedom; Chi^2, chi-squared statistic; p, p-value; I^2, I-squared heterogeneity statistic; Z, Z statistic.

DD is associated with worse prognosis in LC



Behera et al, 2021



Merdli et al 2016

HFA-PEFF score as an independent predictor of 2-year mortality in liver cirrhosis patients

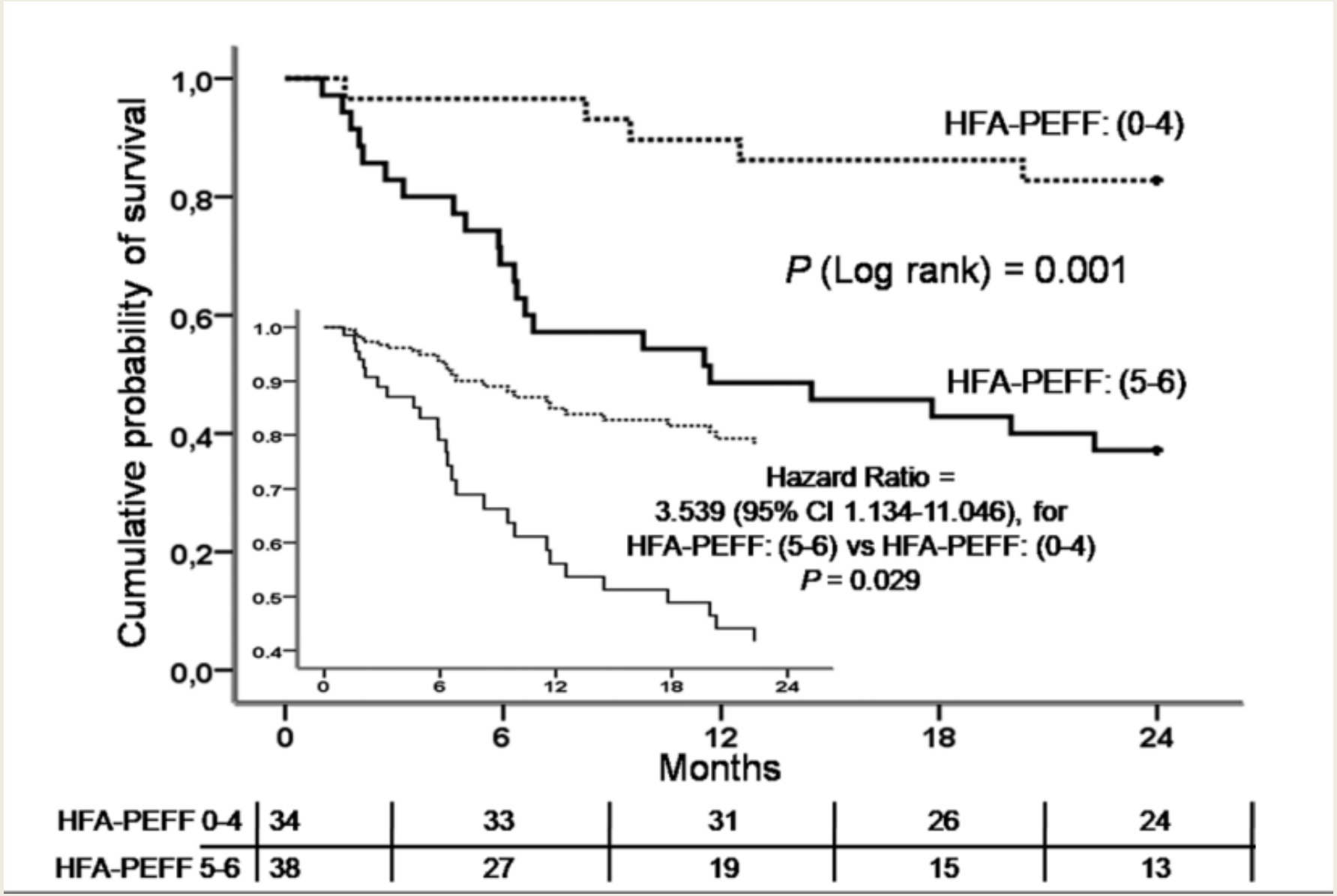
Yannis Dimitroglou[a], Dimitrios Tsartsalis[a], Larisa Vasilieva[b], Dimitrios Patsourakos[a], Argyro Kalompatsou[a], Alexandra Alexopoulou[b], Dimitris Tousoulis[a], Konstantinos Tsioufis[a] and Constantina Aggeli[a]

	Functional	Morphological	Biomarker (SR)	Biomarker (AF)
Major	septal e' < 7 cm/s or lateral e' < 10 cm/s or Average E/e' ≥ 15 or TR velocity > 2.8 m/s (PASP > 35 mmHg)	LAVI > 34 ml/m² or LVMI ≥ 149/122 g/m² (m/w) and RWT > 0,42 #	NT-proBNP > 220 pg/ml or BNP > 80 pg/ml	NT-proBNP > 660 pg/ml or BNP > 240 pg/ml
Minor	Average E/e' 9 -14 or GLS < 16 %	LAVI 29-34 ml/m^2 or LVMI > 115/95 g/m^2 (m/w) or RWT > 0,42 or LV wall thickness ≥ 12 mm	NT-proBNP 125-220 pg/ml or BNP 35-80 pg/ml	NT-proBNP 365-660 pg/ml or BNP 105-240 pg/ml

Major Criteria: 2 points	**≥ 5 points: HFpEF**
Minor Criteria: 1 point	**2-4 points: Diastolic Stress Test or Invasive Haemodynamic Measurements**

Table 3. Correlation of the variables included in the HFA-PEFF score with the severity of the liver disease

	Variable	Child-Pugh A	Child-Pugh B	Child-Pugh C	*P* value
HFA-PEFF functional	Septal e′ (cm/sc)	7.1 (5.8–8.4)	6.2 (5.1–8.4)	7.6 (5.8–9.6)	0.505
	Lateral e′ (cm/s)	9.4 (7.1–11.5)	9.4 (8.0–11.9)	8.9 (7.5–11.8)	0.812
	Average E/e′	7.0 (6.5–9.4)	8.6 (5.5–9.8)	9.7 (7.8–11.6)	0.071
	TR velocity (m/s)	2.36 (2.27–2.55)	2.34 (2.21–2.55)	2.48 (2.24–2.63)	0.733
	Absolute GLS (%)	20.3 (18.7–21.8)	21.1 (20.3–24.1)	22.5 (20.2–24.4)	0.022
HFA-PEFF morphological	LAVi (mL/m^2)	29.3 (23.6–34.0)	35.3 (29.6–41.9)	48.0 (32.8–58.0)	<0.001
	LVMi (g/m^2)	72.9 (60.5–90.3)	69.5 (59.2–77.8)	89.2 (77.6–101.1)	0.001
	RWT	0.38 (0.31–0.41)	0.36 (0.30–0.41)	0.40 (0.34–0.45)	0.431
HFA-PEFF biomarker	NT-pro-BNP (pg/mL)	57 (11–209)	140 (54–324)	294 (133–716)	0.002
	BNP (pg/mL)	35 (10–75)	80 (45–109)	135 (73–344)	0.002



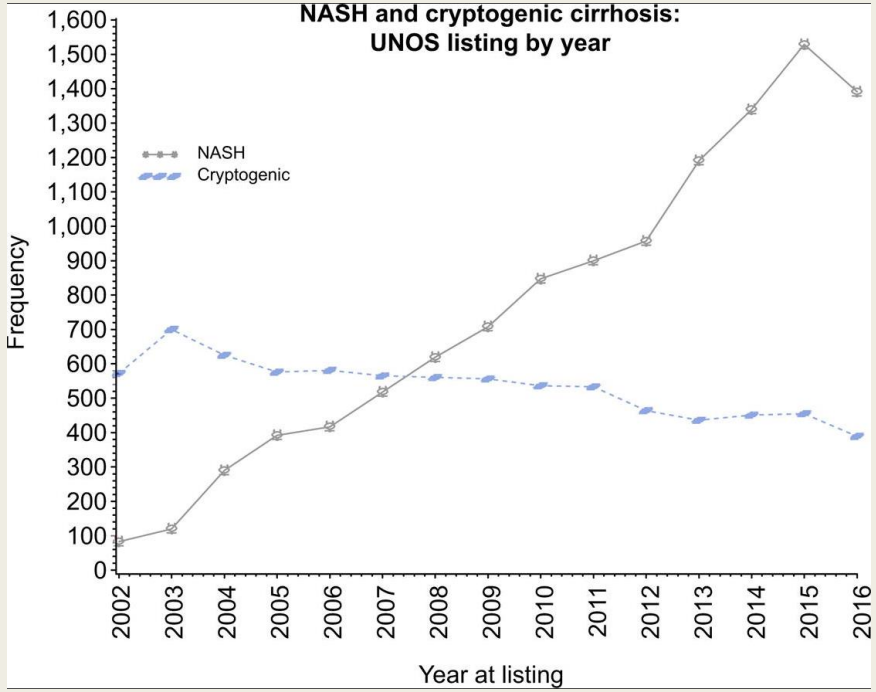
HFA-PEFF 0-4	34	33	31	26	24
HFA-PEFF 5-6	38	27	19	15	13

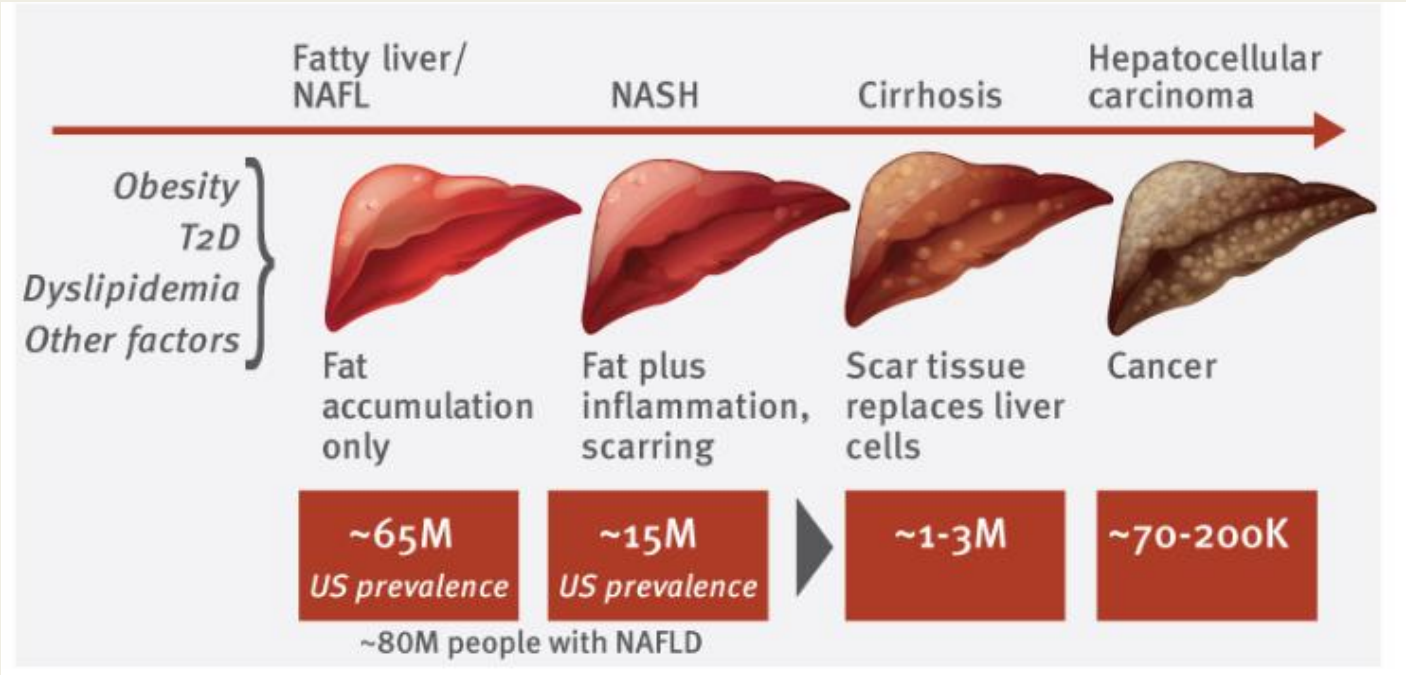
The Low Incidence of Myocardial Infarction in Patients With Portal Cirrhosis of the Liver: A Review of 639 Cases of Cirrhosis of the Liver From 17,731 Autopsies

William L. Howell, M.D., and William C. Manion, M.D.,** Washington, D.C.*

Of the 17,731 patients autopsied, 639 were found to have anatomic diagnoses of portal cirrhosis, Laennec's cirrhosis, or postnecrotic cirrhosis. Of the 639 cirrhotic patients, 32 (4.9 per cent) were found to have myocardial infarcts. The incidence of myocardial infarction in the remaining noncirrhotic group, on the basis of examination of a random 5 per cent sample from the 17,731 autopsies in 1957, was 20.2 per cent. Hence, in the group studied, myocardial infarction was less than one fourth as common in cirrhotic patients as in noncirrhotic patients.

However epidemiology of LC has changed





J Hepatol 2017

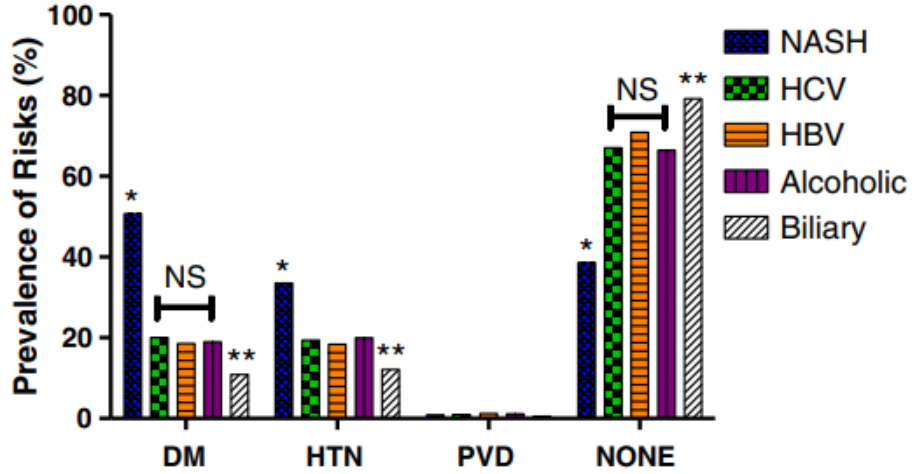


Fig. 2 Prevalence of coronary artery disease (CAD) risk factors. DM = diabetes mellitus, HTN=hypertension, PVD=peripheral vascular disease in various etiological groups, HBV=hepatitis B, HCV=hepatitis C, NASH=nonalcoholic steato-hepatitis, NS=non-significant. *$P < 0.001$ vs all other groups; **biliary cirrhosis: $P < 0.001$ vs all other groups.

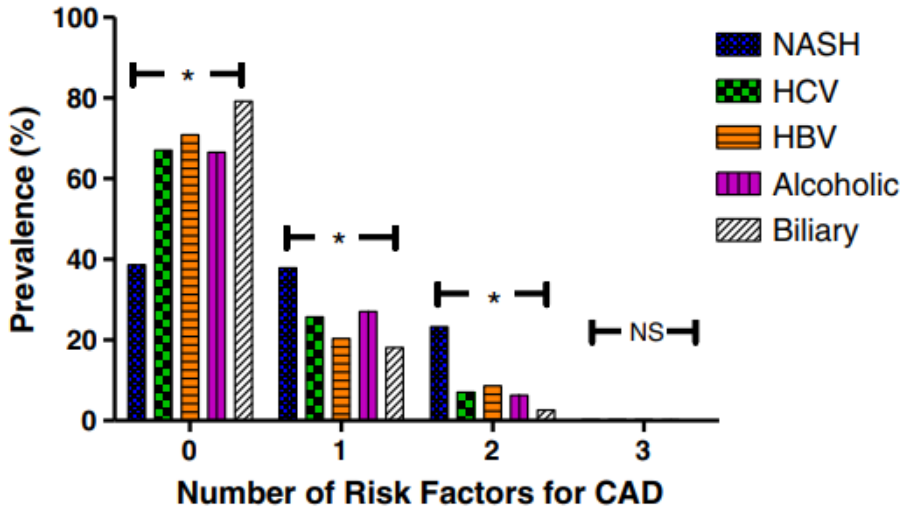


Fig. 3 Distribution of the number of coronary artery disease (CAD) risk factors within various etiological groups. DM=diabetes Mellitus, HBV=hepatitis B, HCV=hepatitis C, HTN=hypertension, NASH=nonalcoholic steato-hepatitis, PVD=peripheral vascular disease, NS=nonsignificant. *$P < 0.001$ among each etiology of end-stage liver disease.

Comparison of the Frequency of Coronary Artery Disease in Alcohol-Related Versus Non-Alcohol-Related Endstage Liver Disease

Sanjay Patel, MBBS, PhD[a,c,*], Todd L. Kiefer, MD[a], Aijaz Ahmed, MD[b], Ziad A. Ali, MBChB, DPhil[a], Jennifer A. Tremmel, MD[a], David P. Lee, MD[a], Alan C. Yeung, MD[a], and William F. Fearon, MD[a]

The American Journal of Cardiology (www.ajconline.org)

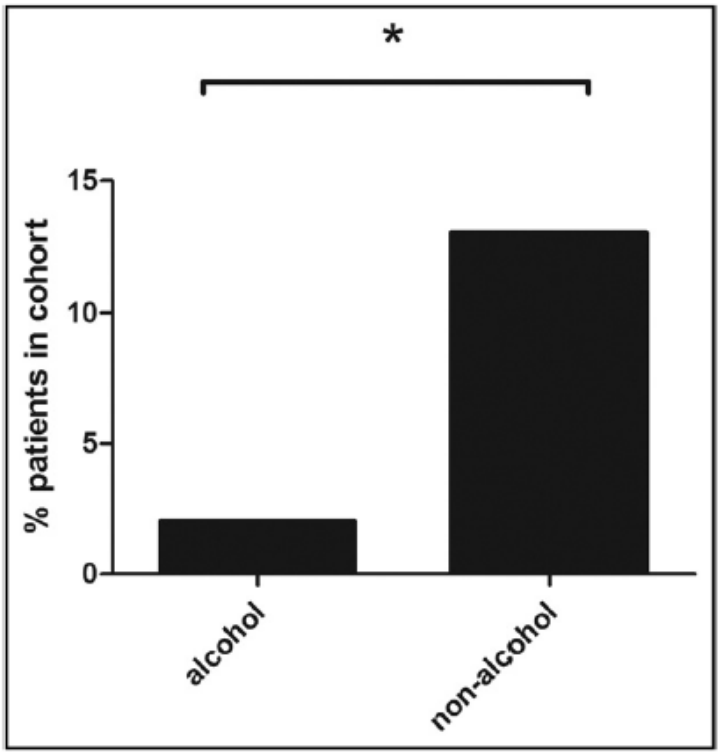


Figure 1. Comparison of the incidence of severe CAD (>70% stenosis in any major epicardial coronary artery) on the basis of the cause of ESLD.

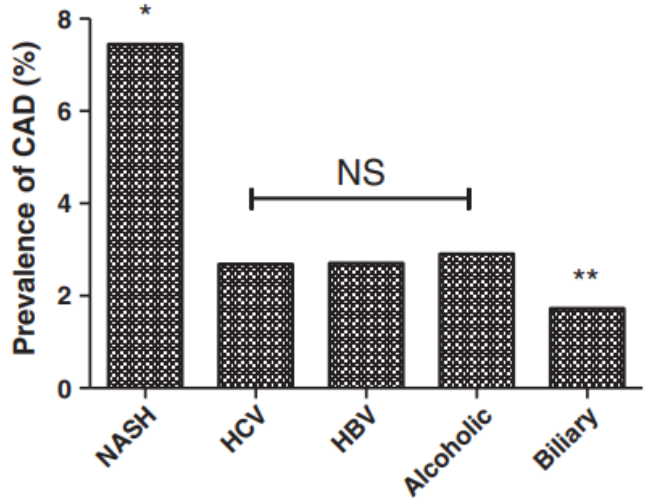
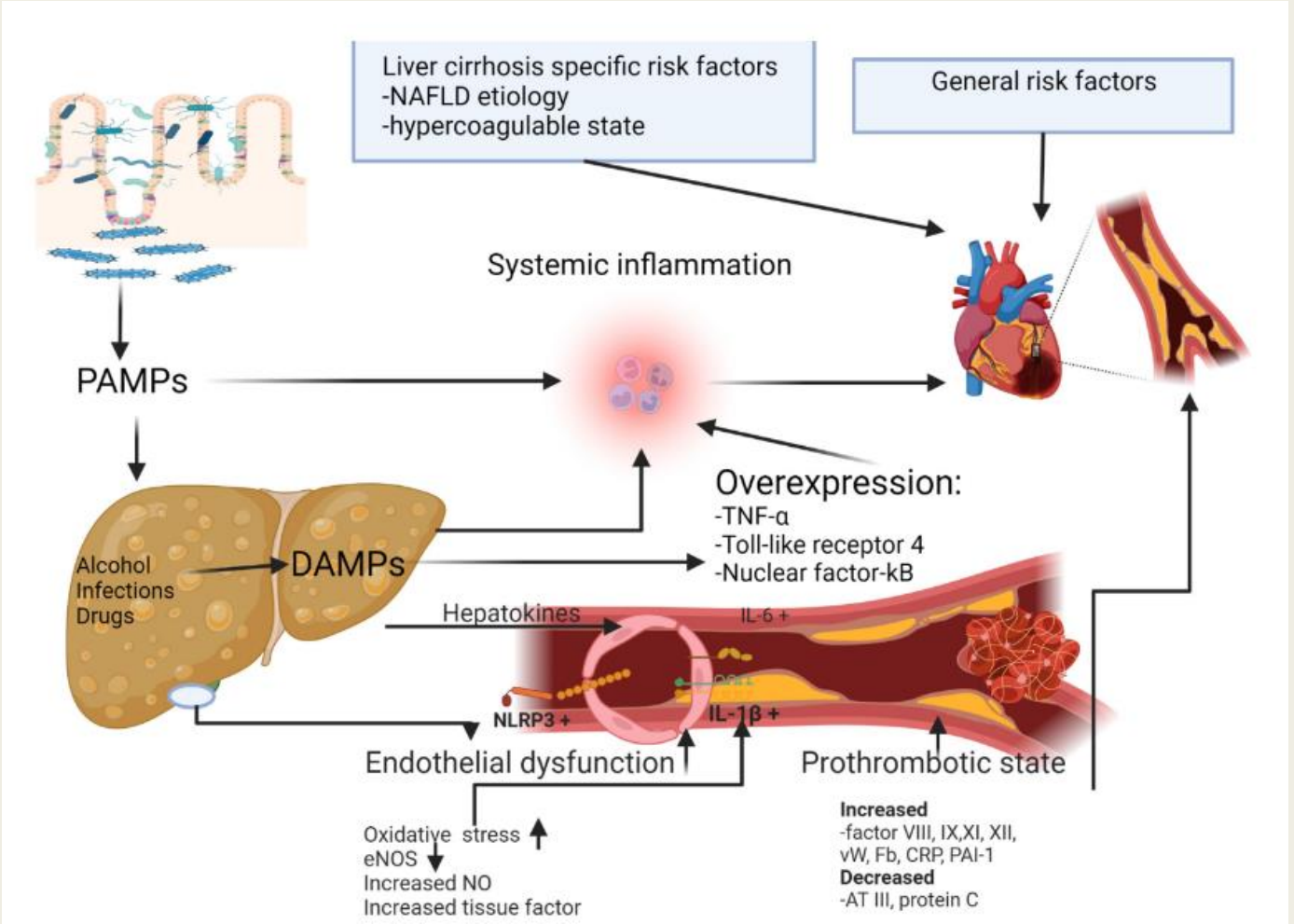


Fig. 1 Prevalence of coronary artery disease (CAD) in various etiological groups. HBV=hepatitis B, HCV=hepatitis C, NASH= nonalcoholic steato-hepatitis. *$P < 0.0001$, vs all other groups; **biliary cirrhosis vs NASH: $P < 0.0001$; vs HCV: $P = 0.037$; vs HBV: not significant; vs alcoholic cirrhosis: $P = 0.018$.


Liver cirrhosis specific risk factors
-NAFLD etiology
-hypercoagulable state
General risk factors
Systemic inflammation
PAMPs
Alcohol
Infections
Drugs
DAMPs
Overexpression:
-TNF-α
-Toll-like receptor 4
-Nuclear factor-kB
Hepatokines
IL-6 +
NLRP3 +
IL-1β +
Endothelial dysfunction
Prothrombotic state
Oxidative stress
eNOS
Increased NO
Increased tissue factor
Increased
-factor VIII, IX,XI, XII,
vW, Fb, CRP, PAI-1
Decreased
-AT III, protein C

Prevalence and Prediction of Coronary Artery Disease in Patients With Liver Cirrhosis

A Registry-Based Matched Case–Control Study

Jihyun An, MD; Ju Hyun Shim, MD, PhD; Seon-Ok Kim, MS; Danbi Lee, MD, PhD; Kang Mo Kim, MD, PhD; Young-Suk Lim, MD, PhD; Han Chu Lee, MD, PhD; Young-Hwa Chung, MD, PhD; Yung Sang Lee, MD, PhD

Circulation October 14, 2014

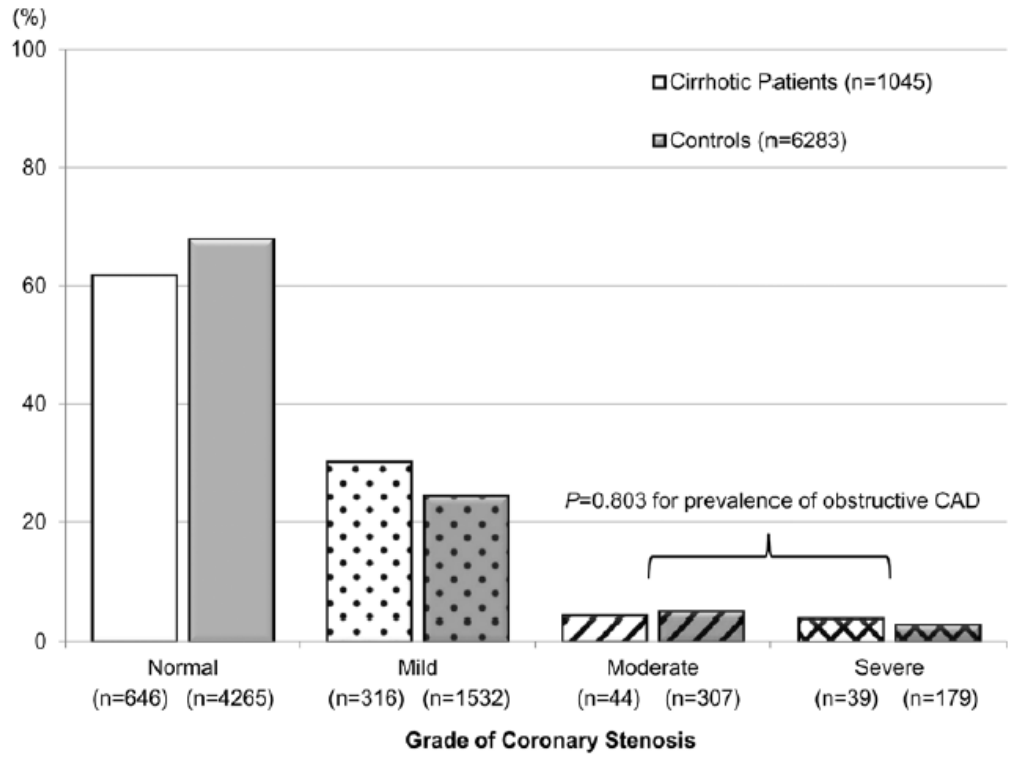


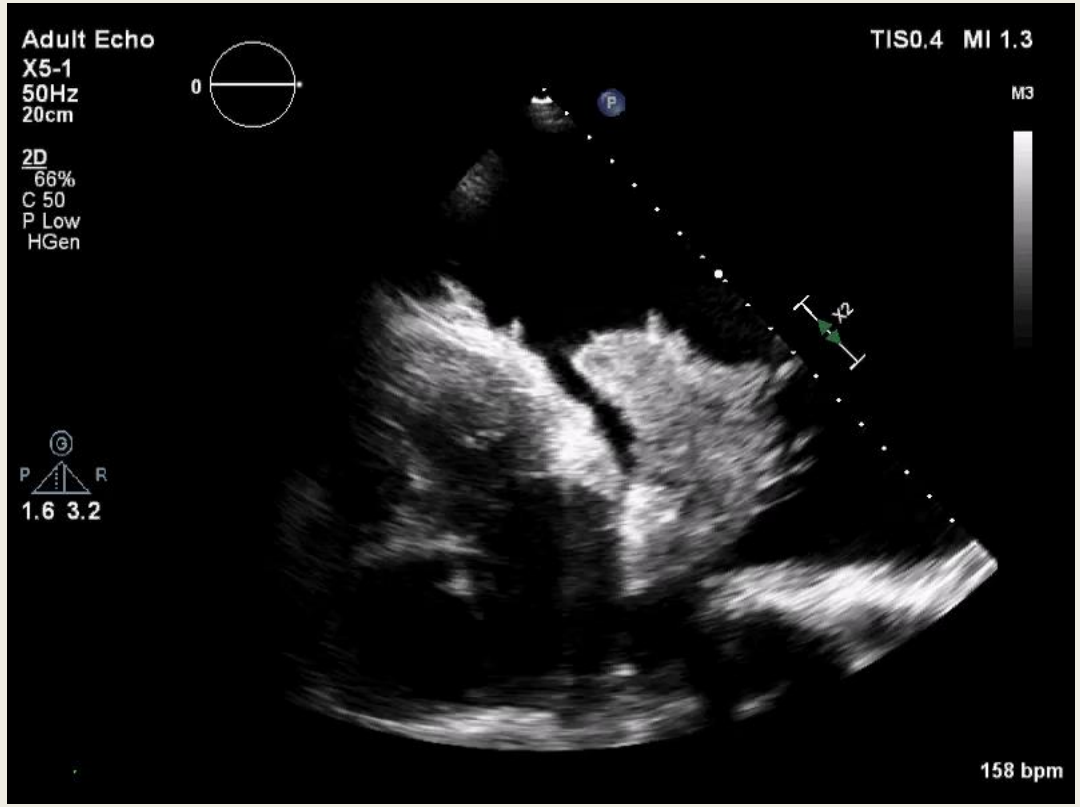
Figure 1. Distribution of coronary artery stenosis on coronary CT angiography among all cirrhotics (n=1045) and controls (n=6283). The prevalence of significant obstructive CAD was similar in the 2 groups (7.9% [83/1045] vs 7.7% [486/6283]; *P*=0.803), in contrast to that of mild nonobstructive CAD (30.2% [316/1045] vs 24.4% [1532/6283]; *P*<0.001). CAD indicates coronary artery disease; and CT, computed tomography.

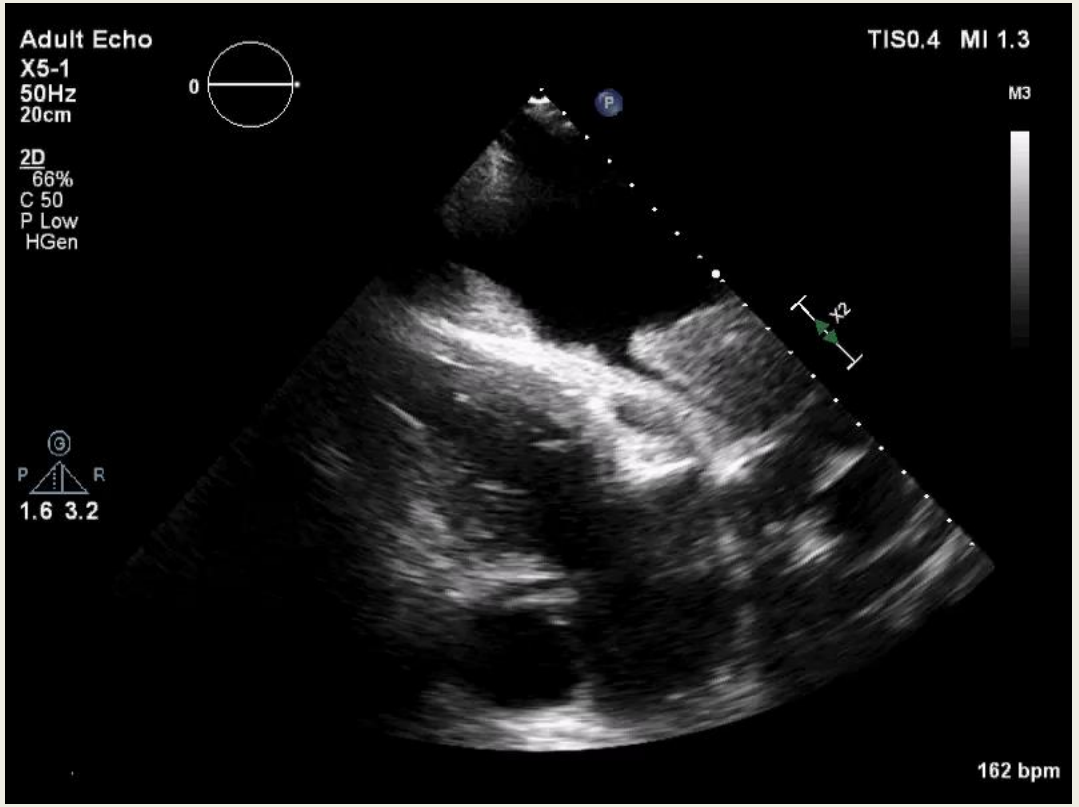
Stress echocardiography

- Dobutamine stress echo???
  - *Role of BB and autonomic dysfunction*
- Vasolidatory stress tests??
  - *ESLD is characterized by vasodilation*
- Exercise stress echo…..Not so feasible for all pts

Blunted response to adrenergic stimulation is translated into lower peak heart rate, lower increase in LVEF and cardiac index in cirrhotics compared to controls during peak exercise [69]. These findings result from the autonomic dysfunction characterizing end-stage cirrhosis [70]. Besides that, blunted response during dobutamine stress echo has been accompanied by higher baseline ejection fraction [71], a finding which highlights that contractile reserve is lower in patients with decompensated cirrhosis and high output cardiovascular state. Some data with the use of STE reinforce these findings. In a recently published study, while GLS at rest was better in cirrhotics than controls, groups did not differ at stress, because lower absolute GLS increase was observed in cirrhotic patients [72]. However, due to temporal resolution limitations, use of speckle tracking echocardiography in current clinical practice of stress echocardiography is questionable and more data from future studies are needed.

A 60 years old man with advanced cirrhosis





EDITORIAL COMMENT

Noninvasive Testing in Patients With End-Stage Liver Disease

When and How?*

Luc A. Pierard, MD, PhD

JACC: CARDIOVASCULAR IMAGING

Liver transplantation is considered a high-risk surgical procedure (4). It is urgent in acute liver failure and elective in most cases. The guidelines by the American Association for the Study of Liver Diseases and the American Society of Transplantation recommend cardiac evaluation with stress echocardiography as an initial screening test with cardiac catheterization as clinically indicated (Class Ib) (5).

Patients with end-stage liver disease (ESLD) who are candidates for liver transplantation have a small prevalence of severe coronary artery disease (CAD). Their hemodynamic characteristics include vasodilation, anemia, and high cardiac output. They are usually treated with beta-blockers to reduce the incidence of esophageal variceal bleeding. The sensitivity of DSE is highly variable, but low (32%) according to a meta-analysis (7).

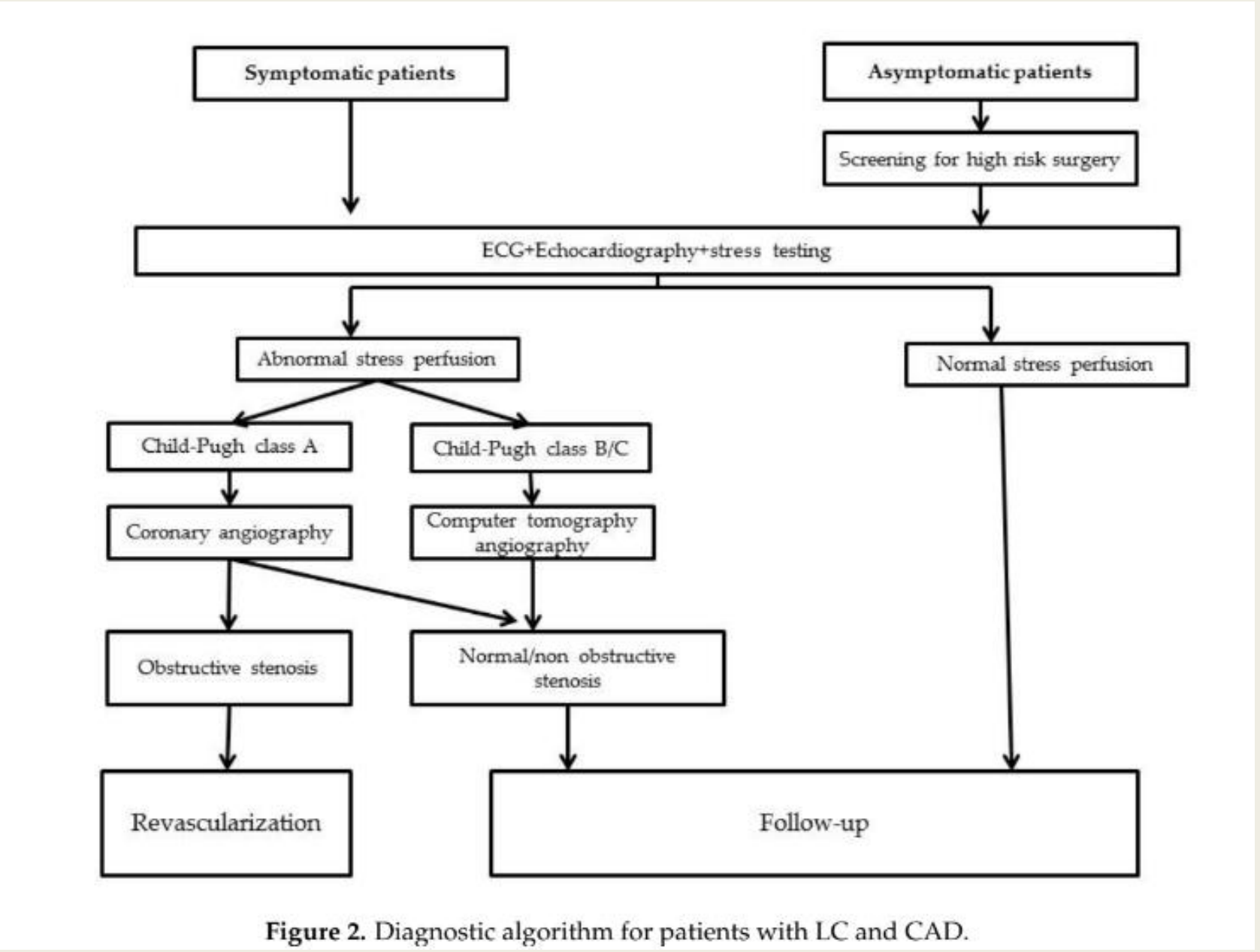


Figure 2. Diagnostic algorithm for patients with LC and CAD.

JACC: CARDIOVASCULAR IMAGING VOL. 12, NO. 11, 2019

PUBLISHED BY ELSEVIER

ORIGINAL RESEARCH

Diagnostic Performance of Dobutamine Stress Echocardiography in End-Stage Liver Disease

Anisiia T. Doytchinova, MD,[a,b] Thomas D. Feigenbaum, MS,[a] Roja C. Pondicherry-Harish, MD,[a] Peter Sepanski, PhD,[a] Deborah Green-Hess, BS,[a] Harvey Feigenbaum, MD,[a] Stephen G. Sawada, MD[a]

TABLE 3 Diagnostic Performance of Dobutamine Stress Echocardiography

	n	Sensitivity	Specificity	PPV	NVP
≥70% stenosis, any vessel	631	24 (17/72)	90 (503/559)	23 (17/73)	90 (503/558)
1-vessel CAD	43	23 (10/43)	89 (525/588)	14 (10/73)	94 (525/558)
2-vessel CAD	18	22 (4/18)	89 (544/613)	5 (4/73)	97 (544/558)
3-vessel or left main CAD	11	27 (3/11)	89 (550/620)	4 (3/73)	99 (550/558)
≥70% stenosis, no branch or distal disease	631	23 (12/53)	89 (517/578)	16 (12/73)	93 (517/558)
≥50% stenosis, any vessel	631	19 (21/108)	90 (471/523)	29 (21/73)	84 (471/558)
≥70% stenosis, any vessel, duplicate studies	686	30 (28/94)	89 (525/592)	29 (28/95)	89 (525/591)

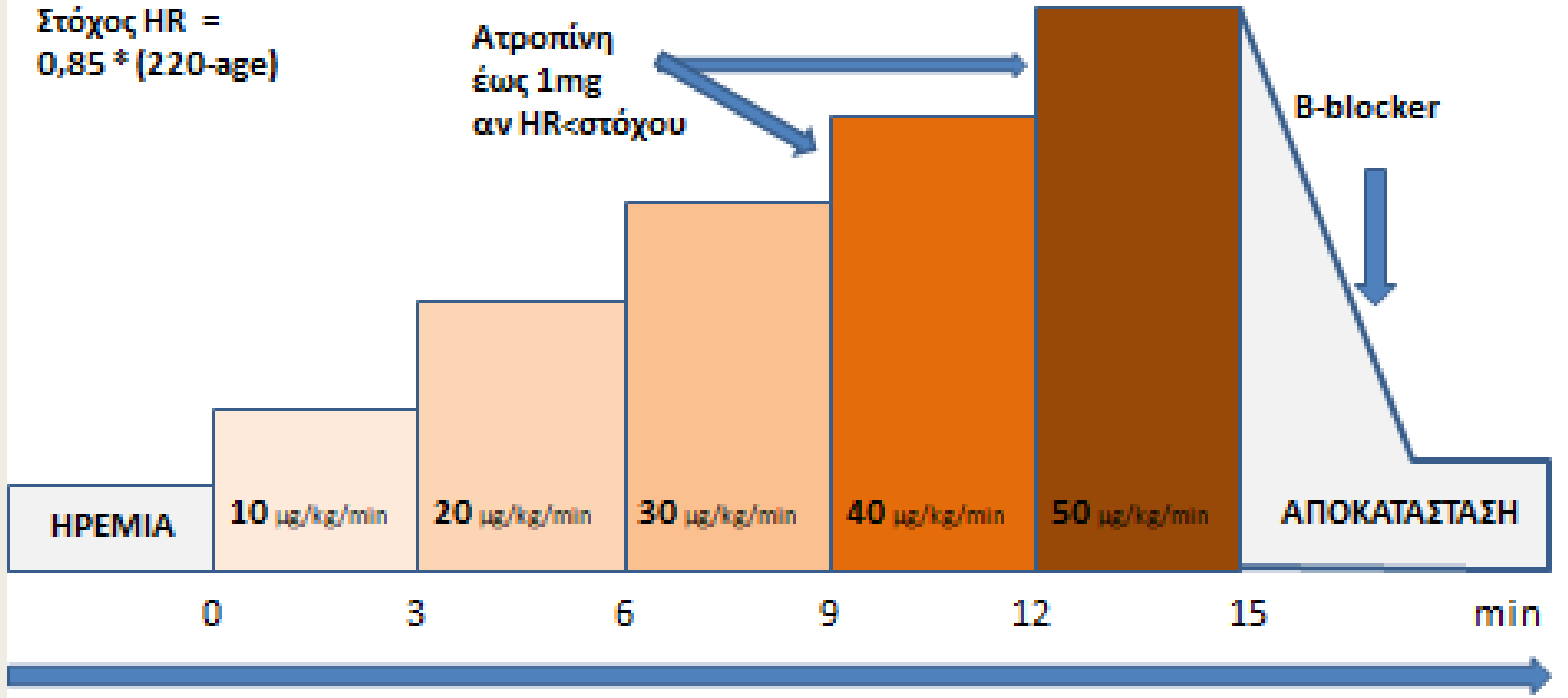
Values are n or % (n/N).
NPV = negative predictive value; PPV = positive predictive value; other abbreviation as in Table 1.

In 633 ESLD patients with a low prevalence of CAD, DSE sensitivity was low (24%) and specificity was high (90%). The sensitivity varied according to the probability of disease and ranged up to 32% in the high-risk group. DSE sensitivity was significantly higher in subjects with LVIDd >4.8 cm, when tardokinesis or lack of hyperkinesis from low-to-peak dose were considered markers of ischemia, and in studies performed after January 1, 2015. Sensitivity was not improved by achievement of the target heart rate. Patients with CAD and an abnormal DSE had significantly worse outcome than those with CAD and a normal DSE.

Στα πλαίσια του προμεταμοσχευτικού ελέγχου ο ασθενής υποβλήθηκε αρχικά σε πρωτόκολλο δυναμικής υπερηχογραφίας με δοβουταμίνη...

STRESS ECHO ΜΕ ΔΟΒΟΥΤΑΜΙΝΗ

ECHO 4C,2C,3C στο τέλος κάθε σταδίου. Χορήγηση παραγόντων αντίθεσης και αποθήκευση εικόνων στην ηρεμία, σε χαμηλές δόσεις, στην κορύφωση και στην αποκατάσταση

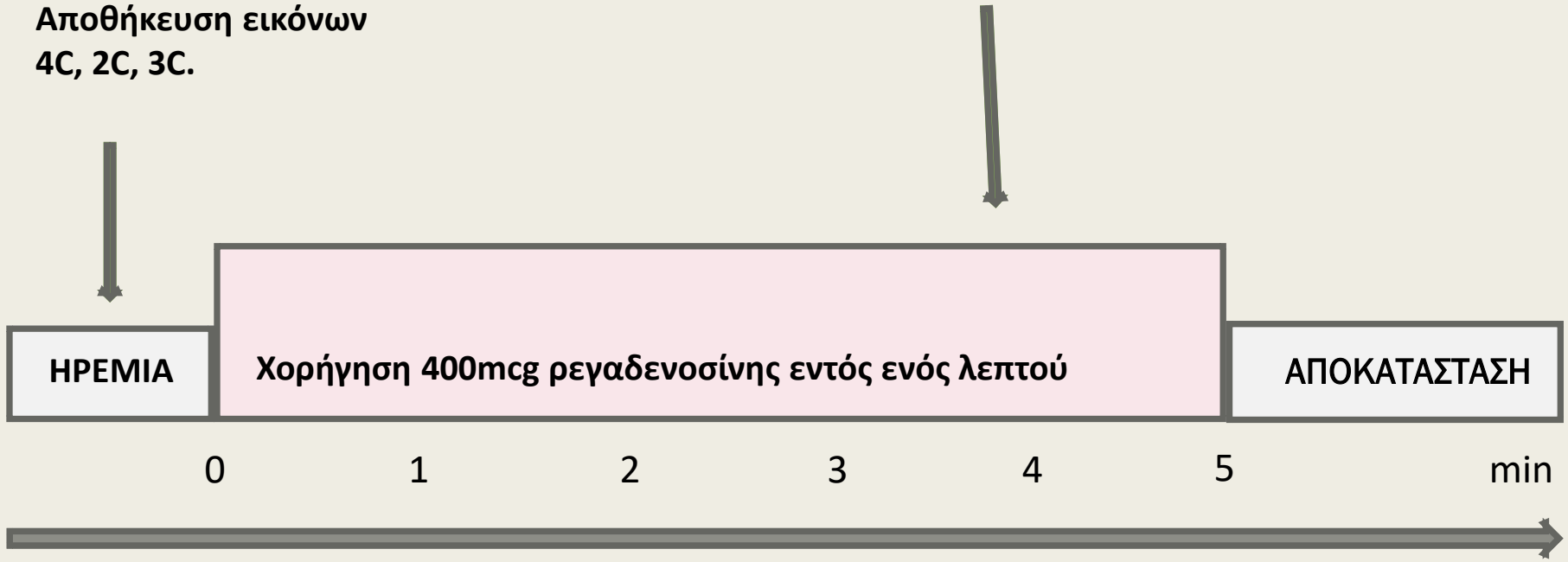


Συνεχής κλινική εκτίμηση και καταγραφή αρτηριακής πίεσης, ΗΚΓ.

STRESS ECHO ΜΕ ΡΕΓΑΔΕΝΟΣΙΝΗ

ECHO ηρεμίας με ενδοφλέβια χορήγηση παραγόντων αντίθεσης. Αποθήκευση εικόνων 4C, 2C, 3C.

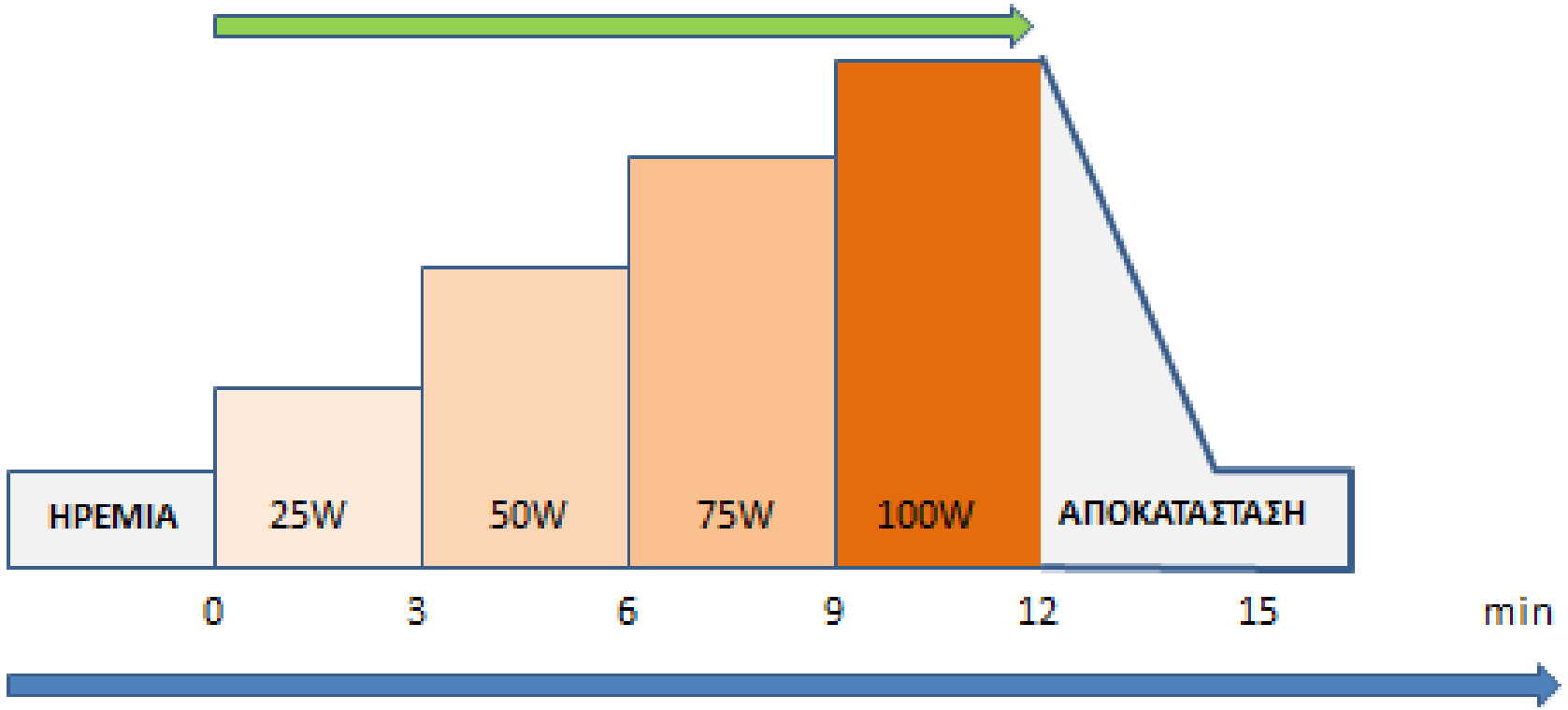
ECHO κατά τη διάρκεια της χορήγησης αδενοσίνης με ενδοφλέβια χορήγηση παραγόντων αντίθεσης. Αποθήκευση εικόνων 4C, 2C, 3C



Συνεχής κλινική εκτίμηση και καταγραφή αρτηριακής πίεσης, ΗΚΓ.

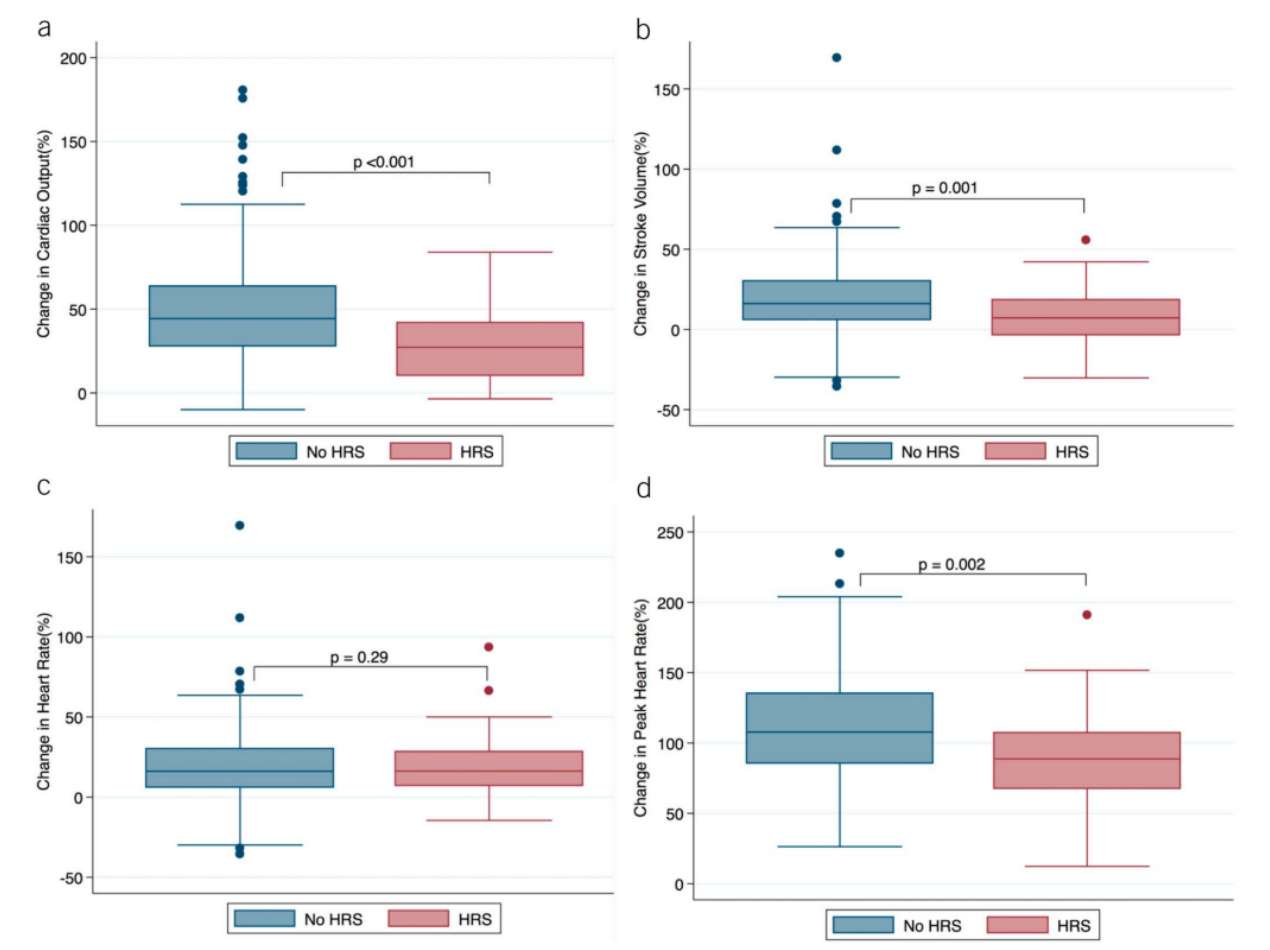
STRESS ECHO ΜΕ ΑΣΚΗΣΗ (ΠΟΔΗΛΑΤΟ)

ECHO με ενδοφλέβια χορήγηση παραγόντων αντίθεσης στην ηρεμία, στο τέλος κάθε σταδίου και στην αποκατάσταση. Αποθήκευση εικόνων 4C, 2C, 3C



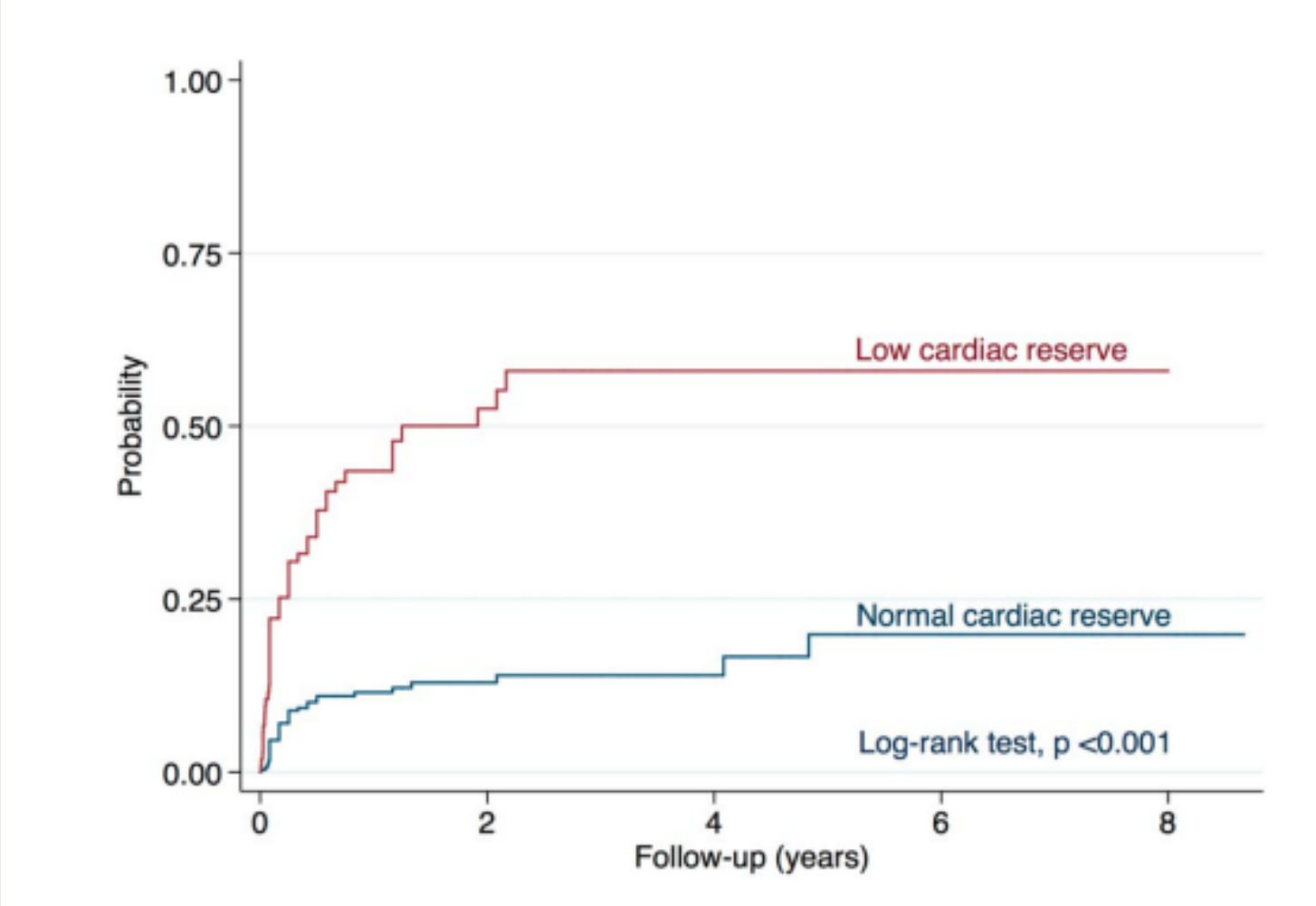
Συνεχής κλινική εκτίμηση και καταγραφή αρτηριακής πίεσης, ΗΚΓ.

Association of HR and CO increase with HRS

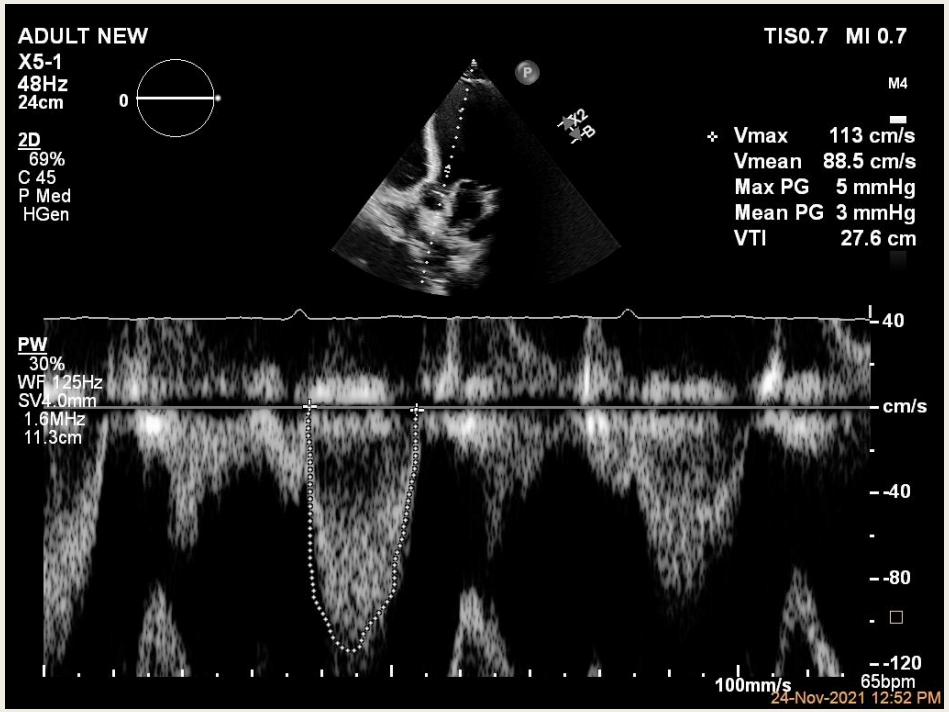


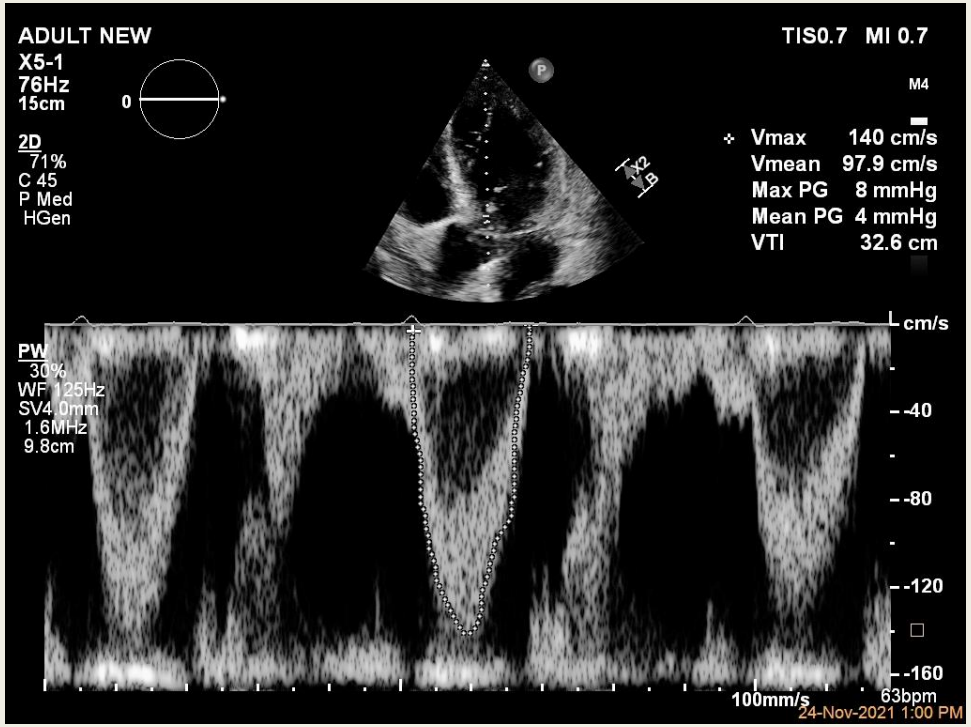
Am J Gastroenterol 2019;00:1–10.

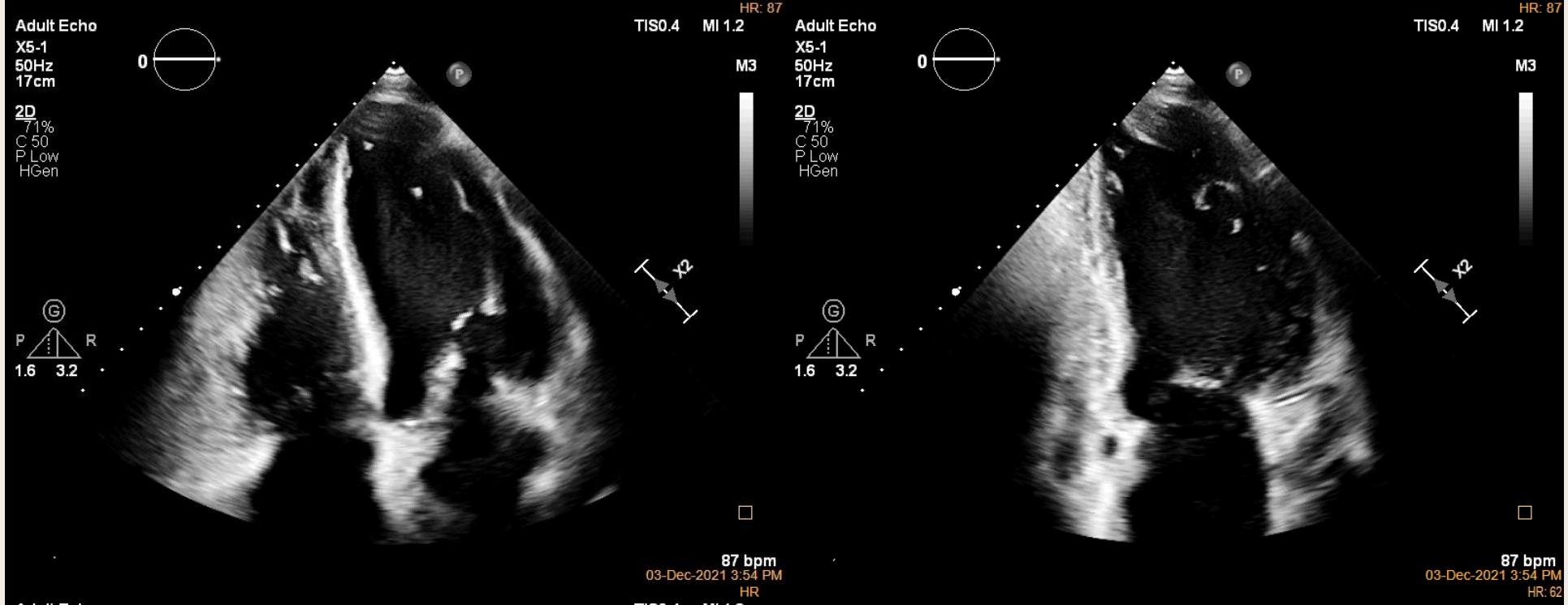
Association of HR and CO increase with prognosis



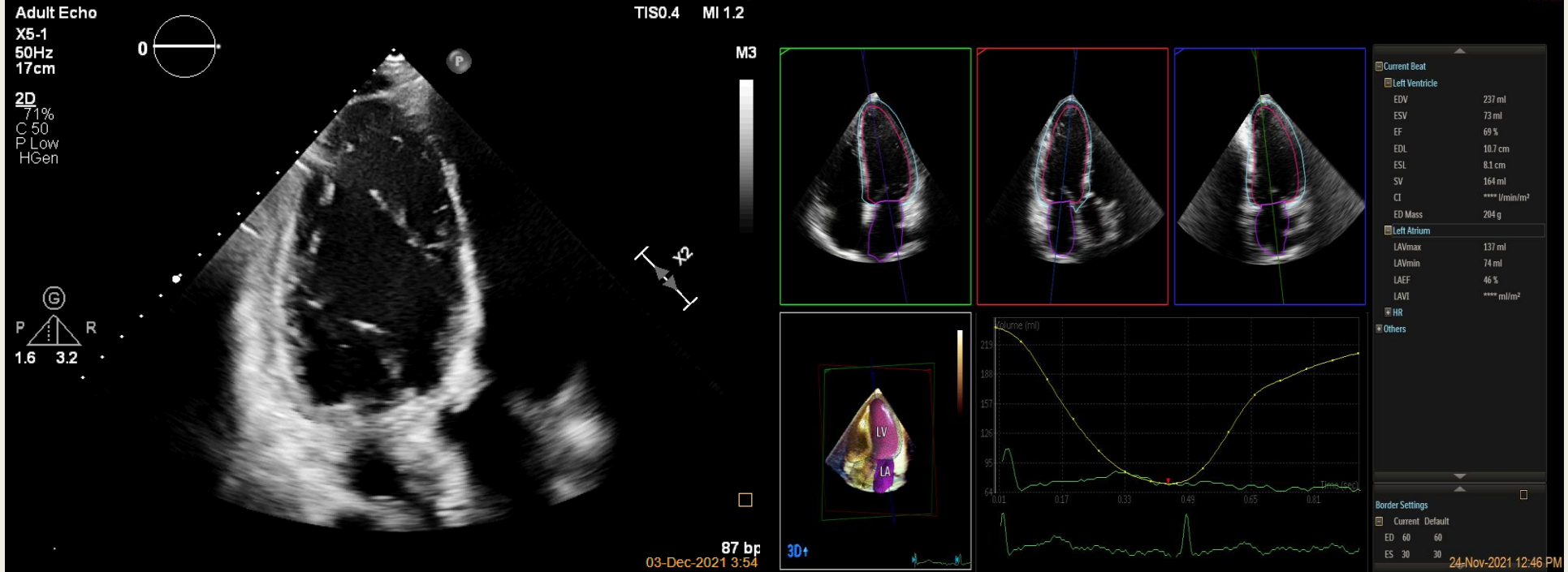
Am J Gastroenterol 2019;00:1–10.

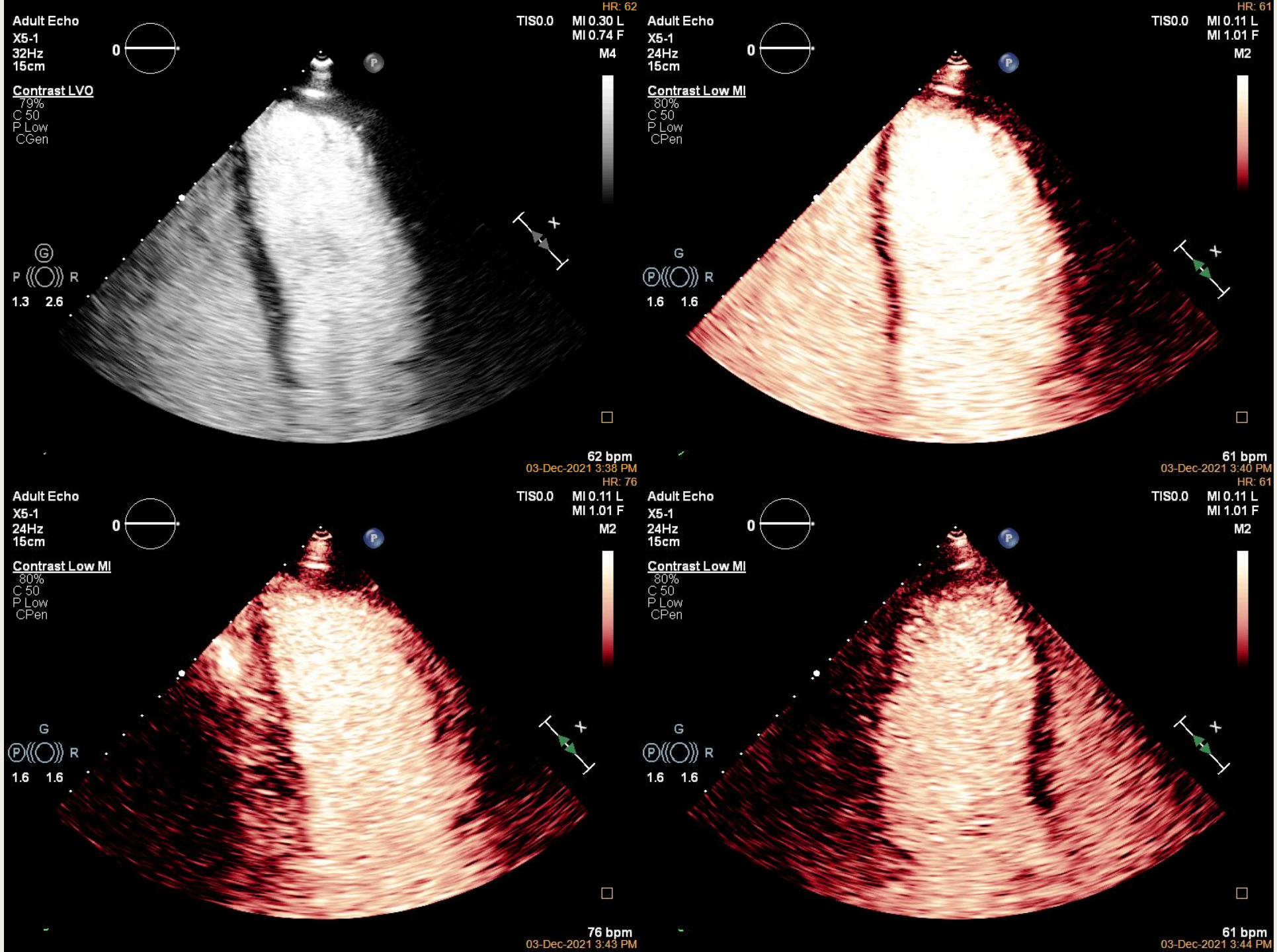

ADULT NEW
X5-1
48Hz
24cm
2D
69%
C 45
P Med
HGen
TIS0.7 MI 0.7
M4
Vmax 113 cm/s
Vmean 88.5 cm/s
Max PG 5 mmHg
Mean PG 3 mmHg
VTI 27.6 cm
PW
30%
WF 125Hz
SV4.0mm
1.6MHz
11.3cm
40
cm/s
-40
-80
-120
100mm/s
65bpm
24-Nov-2021 12:52 PM

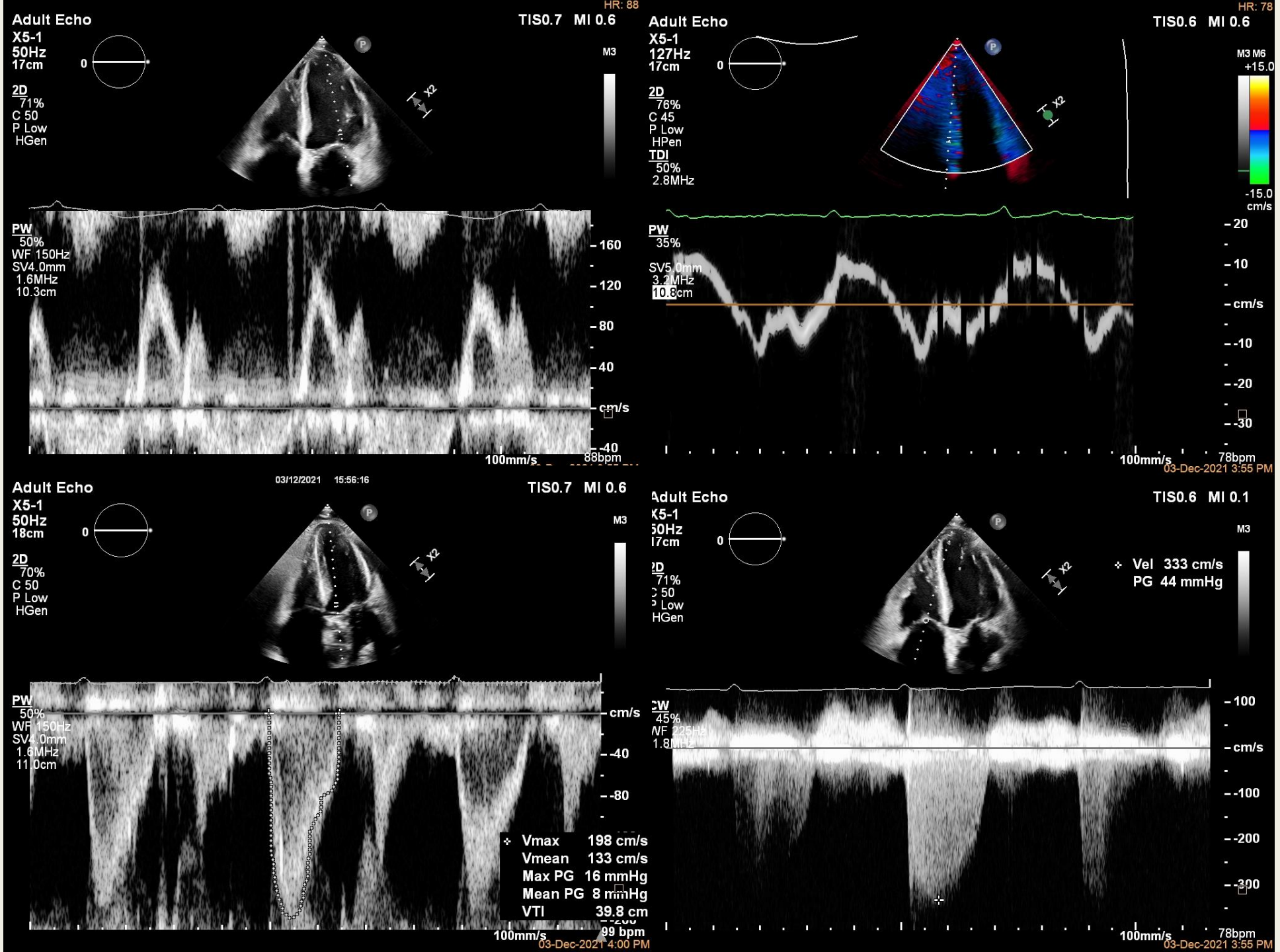

ADULT NEW
X5-1
76Hz
15cm
2D
71%
C 45
P Med
HGen
TIS0.7 MI 0.7
M4
Vmax 140 cm/s
Vmean 97.9 cm/s
Max PG 8 mmHg
Mean PG 4 mmHg
VTI 32.6 cm
PW
30%
WF 125Hz
SV4.0mm
1.6MHz
9.8cm
cm/s
-40
-80
-120
-160
100mm/s
63bpm
24-Nov-2021 1:00 PM


Adult Echo
X5-1
50Hz
17cm
2D
71%
C 50
P Low
HGen
TIS0.4 MI 1.2
M3
HR: 87
87 bpm
03-Dec-2021 3:54 PM
HR
1.6 3.2

Adult Echo
X5-1
50Hz
17cm
2D
71%
C 50
P Low
HGen
TIS0.4 MI 1.2
M3
HR: 87
87 bpm
03-Dec-2021 3:54 PM
HR: 62
1.6 3.2


Adult Echo
X5-1
50Hz
17cm
2D
71%
C 50
P Low
HGen
TIS0.4 MI 1.2
M3
87 bp
03-Dec-2021 3:54
1.6 3.2
LV
LA
3D
Current Beat
Left Ventricle
EDV 237 ml
ESV 73 ml
EF 69 %
EDL 10.7 cm
ESL 8.1 cm
SV 164 ml
CI **** l/min/m²
ED Mass 204 g
Left Atrium
LAVmax 137 ml
LAVmin 74 ml
LAEF 46 %
LAVI **** ml/m²
HR
Others
Border Settings
Current Default
ED 60 60
ES 30 30
24-Nov-2021 12:46 PM


HR: 62
Adult Echo
X5-1
32Hz
15cm
Contrast LVO
79%
C 50
P Low
CGen
TIS0.0
MI 0.30 L
MI 0.74 F
M4
1.3 2.6
62 bpm
03-Dec-2021 3:38 PM
HR: 61
Adult Echo
X5-1
24Hz
15cm
Contrast Low MI
80%
C 50
P Low
CPen
TIS0.0
MI 0.11 L
MI 1.01 F
M2
1.6 1.6
61 bpm
03-Dec-2021 3:40 PM
HR: 76
Adult Echo
X5-1
24Hz
15cm
Contrast Low MI
80%
C 50
P Low
CPen
TIS0.0
MI 0.11 L
MI 1.01 F
M2
1.6 1.6
76 bpm
03-Dec-2021 3:43 PM
HR: 61
Adult Echo
X5-1
24Hz
15cm
Contrast Low MI
80%
C 50
P Low
CPen
TIS0.0
MI 0.11 L
MI 1.01 F
M2
1.6 1.6
61 bpm
03-Dec-2021 3:44 PM


Adult Echo
X5-1
50Hz
17cm
2D
71%
C 50
P Low
HGen
TIS0.7 MI 0.6
M3
HR: 88
PW
50%
WF 150Hz
SV4.0mm
1.6MHz
10.3cm
160
120
80
40
cm/s
-40
100mm/s
88bpm
Adult Echo
X5-1
127Hz
17cm
2D
76%
C 45
P Low
HPen
TDI
50%
2.8MHz
TIS0.6 MI 0.6
M3 M6
+15.0
-15.0
cm/s
HR: 78
PW
35%
SV5.0mm
3.2MHz
10.8cm
20
10
cm/s
-10
-20
-30
100mm/s
78bpm
03-Dec-2021 3:55 PM
03/12/2021 15:56:16
Adult Echo
X5-1
50Hz
18cm
2D
70%
C 50
P Low
HGen
TIS0.7 MI 0.6
M3
PW
50%
WF 150Hz
SV4.0mm
1.6MHz
11.0cm
cm/s
-40
-80
Vmax 198 cm/s
Vmean 133 cm/s
Max PG 16 mmHg
Mean PG 8 mmHg
VTI 39.8 cm
99 bpm
100mm/s
03-Dec-2021 4:00 PM
Adult Echo
X5-1
50Hz
17cm
2D
71%
C 50
P Low
HGen
TIS0.6 MI 0.1
M3
Vel 333 cm/s
PG 44 mmHg
CW
45%
WF 225Hz
1.8MHz
100
cm/s
-100
-200
-300
100mm/s
78bpm
03-Dec-2021 3:55 PM

SPECT

2.4. SPECT. SPECT is the most widely known nuclear test for evaluating myocardial perfusion using diffusible radiotracers. The sensitivity of SPECT is approximately 90%, and the specificity is 75–80% in pharmacological stress studies that use thallium [38]. In liver transplant candidates, however, SPECT imaging is known to be an inaccurate screening test. The sensitivity of SPECT is 37% and its positive predictive value is 22% in comparison with coronary angiography in liver transplant candidates [39]. Adenosine-SPECT has a sensitivity of 62% and a positive predictive value of 30% for diagnosing severe CAD in patients with end-stage liver disease [40]. In patients undergoing orthotopic liver transplantation, SPECT has a sensitivity of 57%, a positive predictive value of 28%, and a negative predictive value of 91% for predicting early cardiac events [13]. A primary deficiency of SPECT is associated with the vasodilating agents used (adenosine and dipyridamole). Chronically decreased arterial vascular resistance in patients with advanced liver failure may limit the typical vasodilating response of the coronary arteries to adenosine or regadenoson [39, 40].

Original Article

JOURNAL OF **CLINICAL** AND **EXPERIMENTAL HEPATOLOGY**

Use of Computed Tomography Coronary Calcium Score for Coronary Artery Disease Risk Stratification During Liver Transplant Evaluation☆

Sabha Bhatti [*,a], Blanca Lizaola-Mayo [†,a], Mohammad Al-Shoha [‡], Mauricio Garcia-Saenz-de-Sicilia [§], Fuad Habash [‖], Karam Ayoub [‖], Michael Karr [‖], Zubair Ahmed [*], Daniel Borja-Cacho [¶], Andres Duarte-Rojo [*,#]

*Division of Cardiology, University of Arkansas for Medical Sciences, 4301 W. Markham Slot #567, Little Rock, AR, 70205, United States, [†] Division of Gastroenterology and Hepatology, Mayo Clinic, 13400 East Shea Blvd, Scottsdale, AZ, 85259, United States, [‡] Division of Gastroenterology and Hepatology, University of Arkansas for Medical Sciences, 4301 W. Markham Slot #567, Little Rock, AR, 70205, United States, [§] Division of Gastroenterology and Hepatology, Washington University, 660 S. Euclid Ave, St. Louis, MO, 63110, United States, [‖] Department of Internal Medicine, University of Arkansas for Medical Sciences, 4301 W. Markham Slot #567, Little Rock, AR, 70205, United States, [¶] Division of Transplant Surgery, Northwestern University, 676 N Saint Clair, Chicago, IL, 60611, United States and [#] Thomas E. Starzl Transplantation Institute and Division of Gastroenterology, Hepatology and Nutrition; University of Pittsburgh Medical Center, 3471 Fifth Avenue, Suite 916, Pittsburgh, PA, 15213, United States

Table 1: Clinical applications of CCTA in combination with the CACS in LT candidates.

Study	Patients (n)	Positive criteria; positive patients, n (%)	Clinical outcomes
Jodocy et al. [24]	54	CACS > 300 or > 50% stenosis on CCTA; 24 (44%)	CCTA and CACS are useful tools for perioperative cardiovascular risk assessments.
Cassagneau et al. [10]	52	> 50% stenosis on CCTA; 6 (12%)	The prognostic value of CCTA is comparable to dobutamine stress echocardiography.
Chae et al. [11]	247	Mild to moderate involvement on CCTA; 27 (11%)	CCTA should be included in routine pretransplant cardiac workups.
Kemmer et al. [25]	85	CACS > 100; 30 (35%)	CACS is a valid alternative tool for risk stratification of LT candidates.
Kong et al. [20]	443	CACS > 400; 11 (3%)	CACS > 400 is a predictor of cardiovascular complications following LT.
Poulin et al. [26]	100	≥ 70% stenosis on CCTA and/or CAG; 20 (20%)	Using CCTA in the evaluation of LT candidates is challenging but is feasible and safe.

CACS, coronary artery calcium score; CAD, coronary artery disease; CAG, coronary angiography; CCTA, coronary computed tomography angiography; LT, liver transplantation.

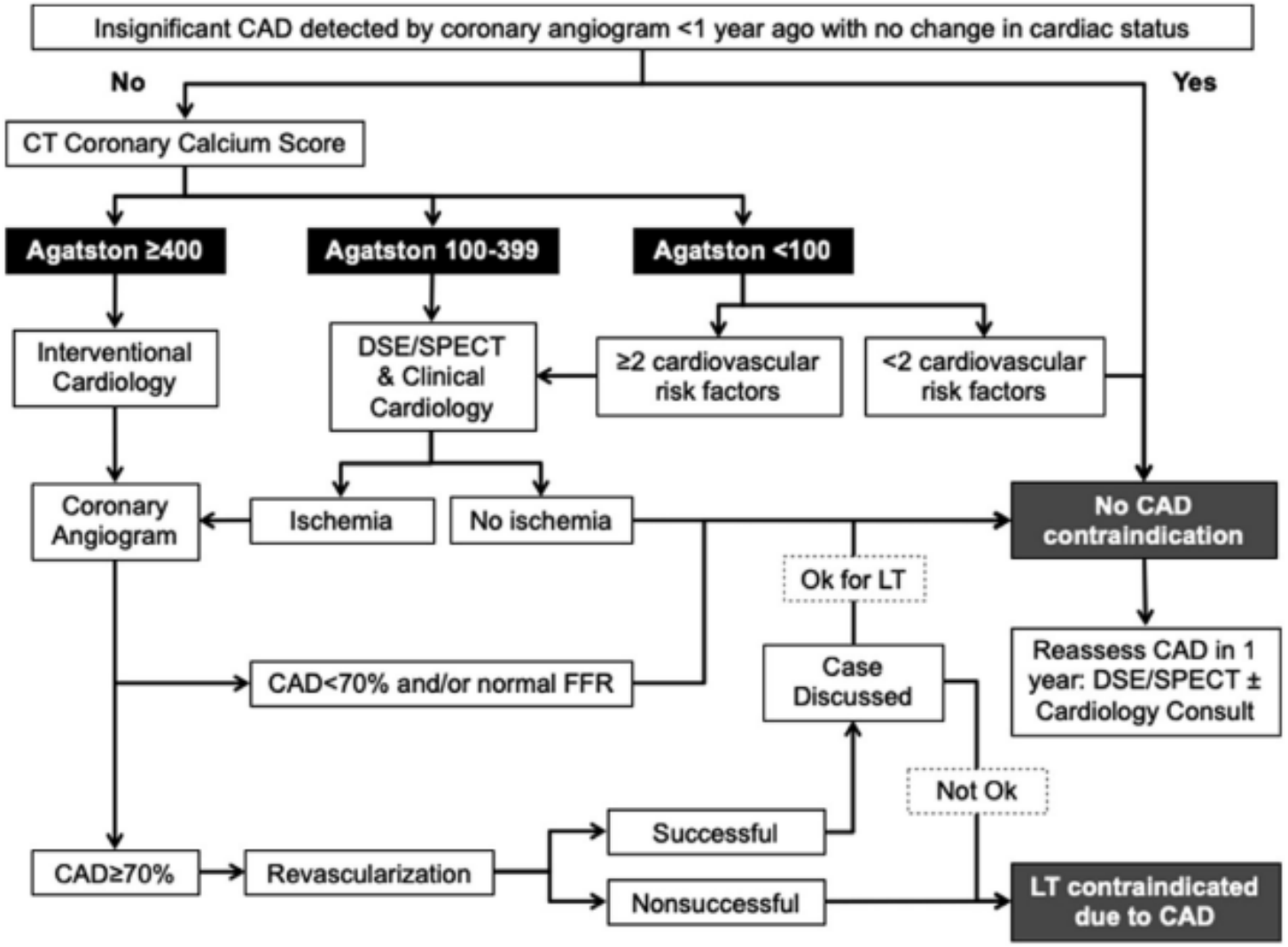


Figure 1 Coronary artery disease evaluation protocol in liver transplant candidates. CAD, coronary artery disease; CT, computed tomography; DSE, dobutamine stress echocardiogram; FFR, fractional flow reserve; LT, liver transplantation; SPECT, single-photon emission computed tomography.

Table 1. Preoperative assessment of CAD in ESLD patients.

Screening Tests		PPV *	NPV *	Disadvantages in ESLD Patients
Noninvasive tests	DSE [1]	0–40%	48–100%	Limited accuracy of DSE to detect CAD due to: - ESLD patients typically have hypercontractile LV - the use of b-blockers results in lower heart rates during the test - the presence of ascites may result in pseudodyskinesis of the posterior wall - microcirculatory disorders
	MPI's [2]	15–28%	77–100%	Limited accuracy of MPI to detect CAD due to: - the impaired vasodilatory reserve in ESLD patients may reduce the effectiveness of a vasodilator stress test - the presence of image artifacts secondary to splenomegaly and ascites
	CCTA [3]	86% in general population	97% in general population	False-positive results are possible in case of elevated diffuse calcification Major limitations: - nephrotoxicity - the need for relative bradycardia
	CACs	no data comparing CCTA to CA in ESDL patients		Contraindications: - severe ascites - orthopnea - hepatic encephalopathy
	CMR stress [4]	77% in general population	91% in general population	Limitations: - lack of availability/expertise - high cost - concern about contrast use in patients with reduced GFR - impossible to scan non MRI conditional devices (metallic clips, pacemakers and defibrillators)
		no data comparing CMR stress to CA in ESDL patients		Contraindications:- severe ascites- orthopnea- hepatic encephalopathy- claustrophobia

Bonou et al 2021

Take home messages

- ESLD is characterized by vasodilation, increased CO and DD
- DSE in ESLD has lower utility than in the general population
- Increase in CO and HR has prognostic significance
- Every patient with ESLD is different
- For excluding CAD CCTA is superior

A multidisciplinary team including both hepatologists and cardiologists can take advantage of the multimodality imaging