

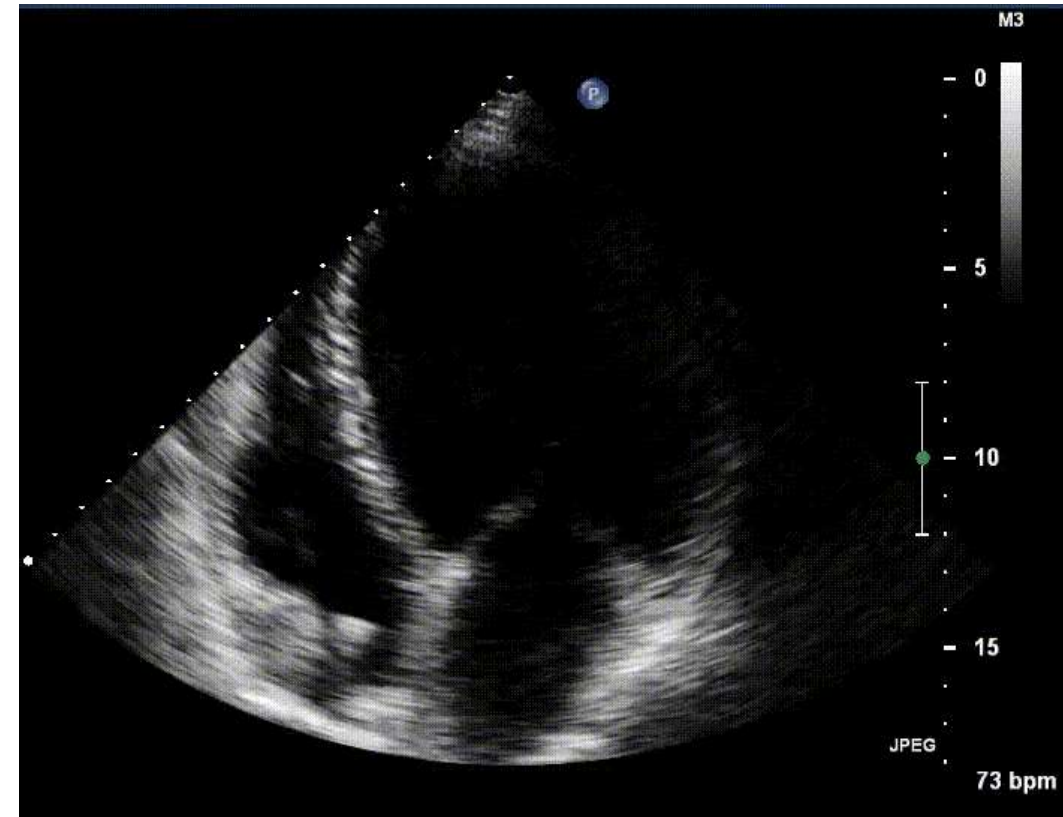
SERVIZIO SANITARIO REGIONALE
EMILIA-ROMAGNA
Azienda Ospedaliero - Universitaria di Parma

LO SCOMPENSO CARDIACO:


Inquadramento eziologico e percorso diagnostico nello scompenso cardiaco

Dott. Federico Barocelli

U.O. CARDIOLOGIA, AZIENDA OSPEDALIERO-UNIVERSITARIA DI PARMA



Heart Failure Is a Major Public Health Problem Worldwide



~64 million
people
have HF¹

US
Data

HF = heart failure; HFpEF = heart failure with preserved ejection fraction; US = United States.

1. GBD 2017 Disease and Injury Incidence and Prevalence Collaborators. *Lancet*. 2018;392:1789-1858; 2. Lippi G et al. *AME Med J*. 2020;5:15; 3. Jones NR et al. *Eur J Heart Fail*. 2019;21:1306-1325; 4. Mamas AM et al. *Eur J Heart Fail*. 2017;19:1095-1104; 5. Omote K et al. Online ahead of print. *Annu Rev Med*. 2021.



Projected **~24% rise in cases** between 2012 and 2030²



5-year **mortality rate ~50%**³



HF **mortality risk is similar** to some of the common cancers in both men and women⁴



Economic burden **~350 billion US dollars**²



Number one cause of hospitalizations in people over 65 years of age^{1,a}



Number one cause of unplanned hospital readmission²

What is heart failure?

Definition by Braunwald:

«A clinical syndrome caused by the inability of the heart to supply blood to the tissues commensurate to the metabolic needs of that tissue or only at the expense of elevated filling pressures»

Proposed Universal Definition of HF¹



Clinical syndrome with symptoms and/or signs caused by a structural and/or functional cardiac abnormality and corroborated by elevated natriuretic peptide levels and/or objective evidence of pulmonary or systemic congestion.

EF = ejection fraction; HF = heart failure; LVEF = left ventricular ejection fraction.

1. Bozkurt B et al. *Eur J Heart Fail.* 2021;23:352-380; 2. McDonagh TA et al. *Eur Heart J.* 2021;42:3599-3726.

HEART FAILURE

What is HEART FAILURE?

A PROBLEM with HOW YOUR HEART PUMPS.

There are two main types of heart failure:



Weak chambers become big over time

REDUCED EJECTION FRACTION
The heart can't pump or squeeze enough blood out to the body.



Stiff chambers fill with less blood than usual

PRESERVED EJECTION FRACTION
The heart can't fill with enough blood.

Ejection fraction
is a measure of how much blood your heart is pumping out with each beat.

SYMPTOMS



Over 6 million Americans have heart failure

It is the leading cause of hospitalization for people 65+

COMMON CAUSES

Coronary artery disease



Heart attack



Diabetes



High blood pressure




Heart rhythm disorders



- Heart inflammation
- Valve problems
- Congenital heart problems
- Obesity
- Some cancer treatments

For more information, visit [CardioSmart.org/HeartFailure](https://www.cardiosmart.org/HeartFailure)

 @ACCinTouch #CardioSmart

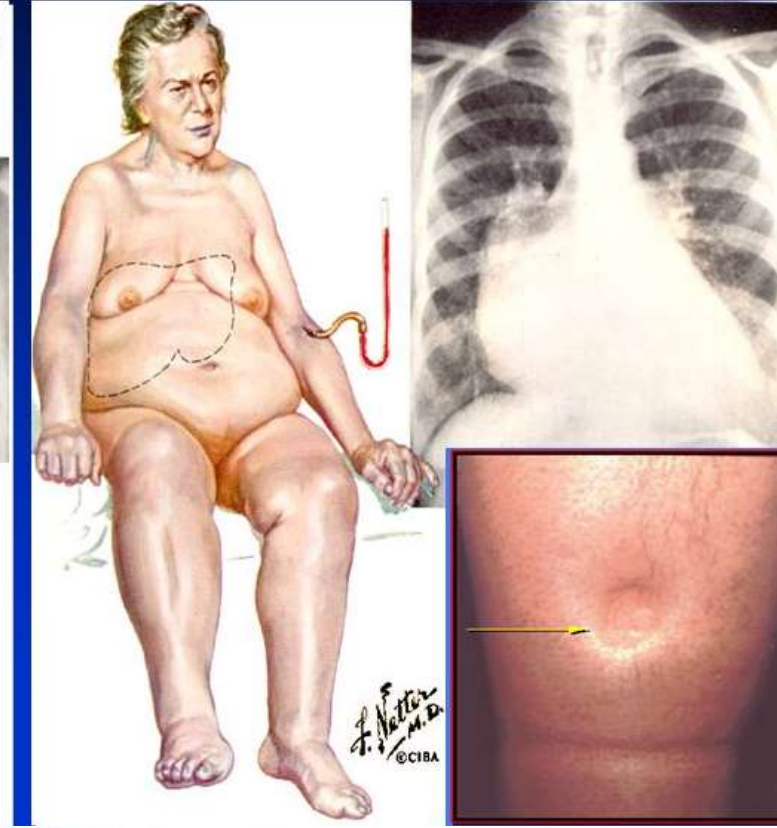


Table 4 New York Heart Association functional classification based on severity of symptoms and physical activity

Class I	No limitation of physical activity. Ordinary physical activity does not cause undue breathlessness, fatigue, or palpitations.
Class II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in undue breathlessness, fatigue, or palpitations.
Class III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results undue breathlessness, fatigue, or palpitations.
Class IV	Unable to carry on any physical activity without discomfort. Symptoms at rest can be present. If any physical activity is undertaken, discomfort is increased.

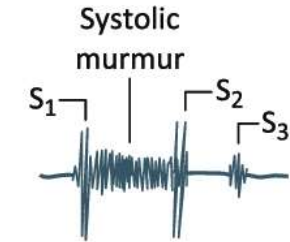
NYHA Functional Classification

McDonagh T.A. et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure, EHJ 2020

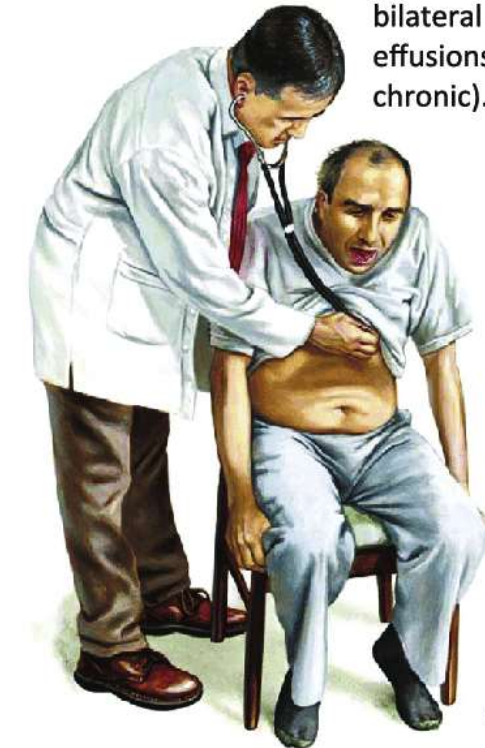
Heart failure signs and symptoms

Left-Sided Cardiac Heart Failure

Cardiac auscultation for third heart sounds (S_3) and murmurs should be performed in standard positions, including that with the patient sitting forward.



Chest auscultation reveals bilateral rales and pleural effusions (when CHF is chronic).



Cyanosis of lips and nail beds may be present if the patient is hypoxic.

Patients with left-sided CHF may be uncomfortable lying down.

Table 6 Symptoms and signs of heart failure

Symptoms of heart failure	
Typical	Breathlessness Orthopnoea ^a Paroxysmal nocturnal dyspnoea ^a Reduced exercise tolerance ^a Fatigue, tiredness ^b Ankle swelling ^a Inability to exercise ^a Swelling of parts of the body other than ankles Bendopnoea
Less typical	Nocturnal cough Wheezing Bloated feeling ^c Postprandial satiety ^c Loss of appetite Decline in cognitive function, confusion (especially in the elderly) ^b Depression Dizziness, syncope ^b
Signs of heart failure	
More specific	Elevated jugular venous pressure ^a Third heart sound ^a Summation gallop with third and fourth heart sounds Cardiomegaly, laterally displaced apical impulse Hepatojugular reflux Cheyne–Stokes respiration in advanced heart failure ^b
Less specific	Peripheral oedema (ankle, sacral, scrotal) Pulmonary rales ^a Unintentional weight gain (>2 kg/week) Weight loss (in advanced heart failure) with muscle wasting and cachexia Cardiac murmur Reduced air entry and dullness to percussion at lung bases suggestive of pleural effusion Tachycardia, irregular pulse Tachypnoea Hepatomegaly/ascites Cold extremities ^b Oliguria Narrow pulse pressure

Table 7 Natriuretic peptide levels supporting definition of heart failure

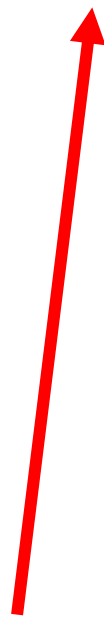
	Ambulatory	Hospitalized/ decompensated
BNP, pg/ml	≥ 35	≥ 100
NT-proBNP, pg/ml	≥ 125	≥ 300

Causes of elevated natriuretic peptide levels other than primary diagnosis of heart failure

Cardiovascular causes	Non-cardiovascular causes
Acute coronary syndrome, myocardial infarction Pulmonary embolism Myocarditis Hypertrophic cardiomyopathy Valvular heart disease Congenital heart disease Atrial or ventricular arrhythmias Heart contusion, cardiac infiltration or malignancy Cardioversion, ICD shock Pericardial disease Invasive or surgical procedures involving the heart Pulmonary hypertension, right ventricular failure	Infiltrative cardiomyopathies Advanced age Kidney disease Critical illnesses including sepsis syndrome, cytokine syndrome Ischaemic or haemorrhagic stroke Pulmonary disease (pneumonia, chronic obstructive pulmonary disease) Liver disease Severe anaemia Severe metabolic and hormone abnormalities (e.g. thyrotoxicosis, diabetes, ketoacidosis, severe burns)

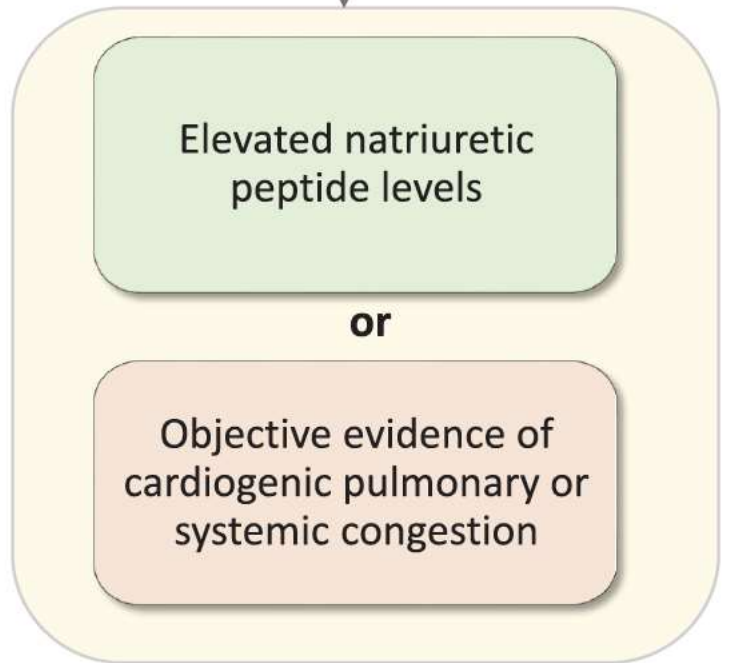
Causes of lower natriuretic peptide levels

- Obesity, or increased BMI
- Pericardial disease^a



Symptoms and/or signs of HF caused by a structural and/or functional cardiac abnormality

corroborated by *at least one* of the following



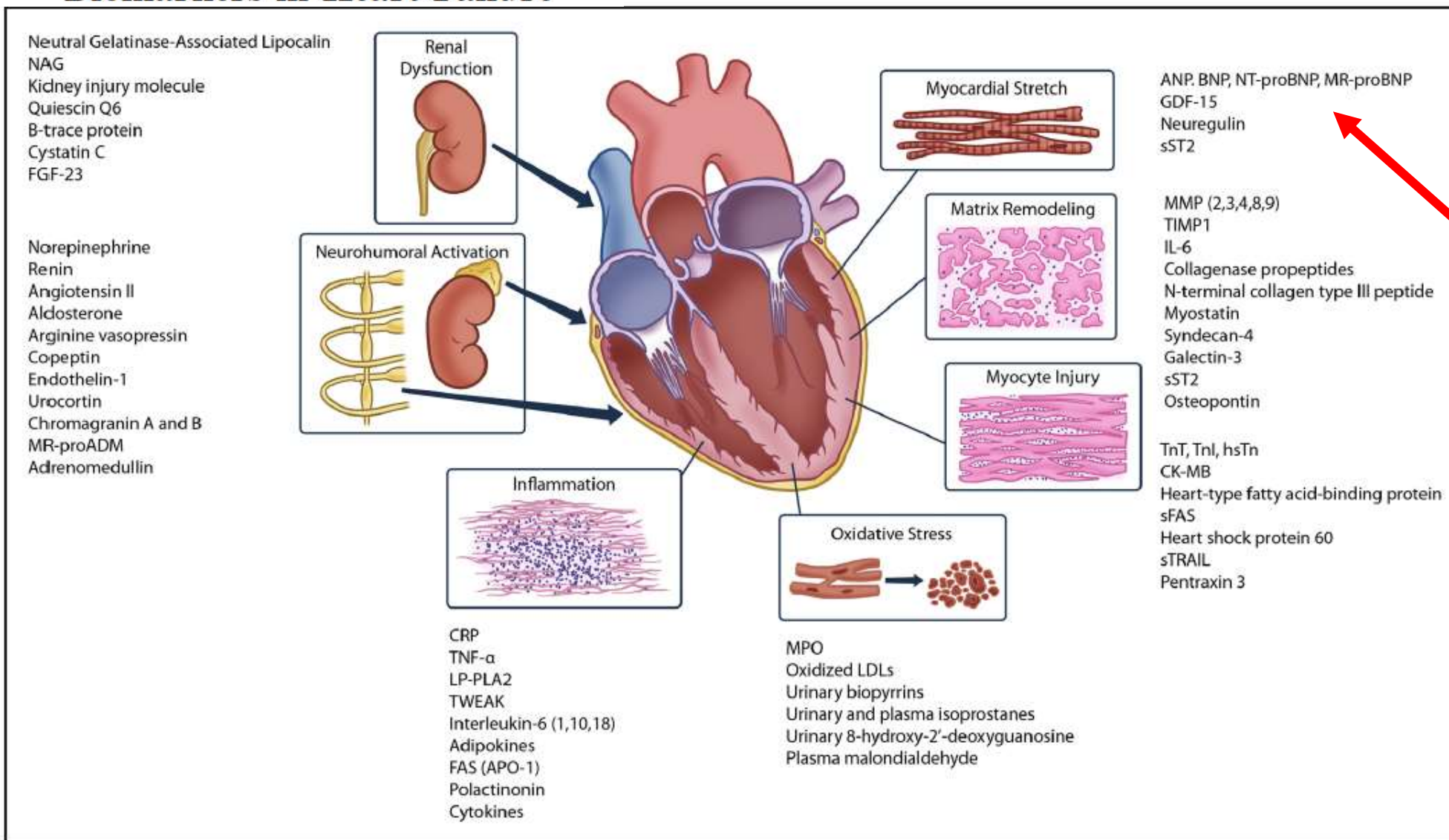
Established and Emerging Roles of Biomarkers in Heart Failure

Nasrien E. Ibrahim, James L. Januzzi Jr

© 2018 American Heart Association, Inc.

Circulation Research is available at <https://www.ahajournals.org/journal/res>

DOI: 10.1161/CIRCRESAHA.118.312706



Myocardial Stretch Biomarkers:
BNP and NT-proBNP.

Left ventricular wall stress is
the most potent trigger for
release of natriuretic peptides

Figure. Various pathophysiological pathways contributing to the development and progression of heart failure and biomarkers representative the various pathways. ANP indicates atrial natriuretic peptide; APO, apolipoprotein; BNP, B-type natriuretic peptide; CK-MB, creatinine kinase-muscle/brain; CRP, C-reactive protein; FAS, Fas cell surface death receptor; GDF, growth differentiation factor; hsTn, high-sensitivity troponin; IL, interleukin; LDL, low-density lipoprotein; LP-PLA2, lipoprotein-associated phospholipase A2; MMP, matrix metalloproteinases; MPO, myeloperoxidase; MR-proADM, midregional proadrenomedullin; MR-proBNP, midregional pro-B-type natriuretic peptide; NT-proBNP, N-terminal pro-B-type natriuretic peptide; sFAS, soluble Fas cell surface death receptor; sST2, soluble ST2; sTRAIL, soluble TNF-related apoptosis-inducing ligand; TIMP, tissue inhibitors of metalloproteinases; TNF, tumor necrosis factor; TnI, troponin I; and TnT, troponin T.

2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

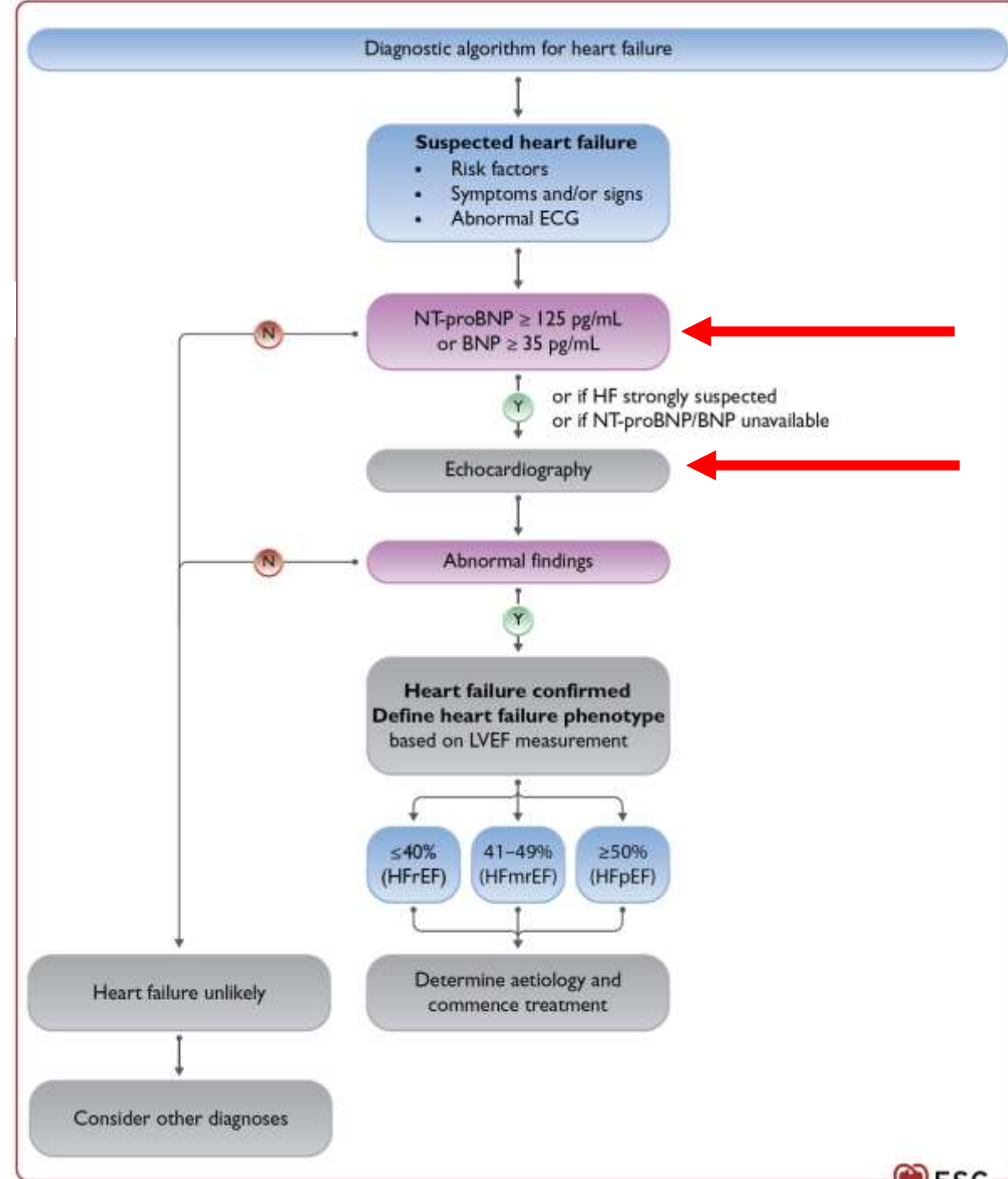
The diagnostic algorithm for heart failure

Recommended diagnostic tests in all patients with suspected chronic heart failure

Recommendations	Class ^a	Level ^b
BNP/NT-proBNP ^c	I	B
12-lead ECG	I	C
Transthoracic echocardiography	I	C
Chest radiography (X-ray)	I	C
Routine blood tests for comorbidities, including full blood count, urea and electrolytes, thyroid function, fasting glucose and HbA1c, lipids, iron status (TSAT and ferritin)	I	C

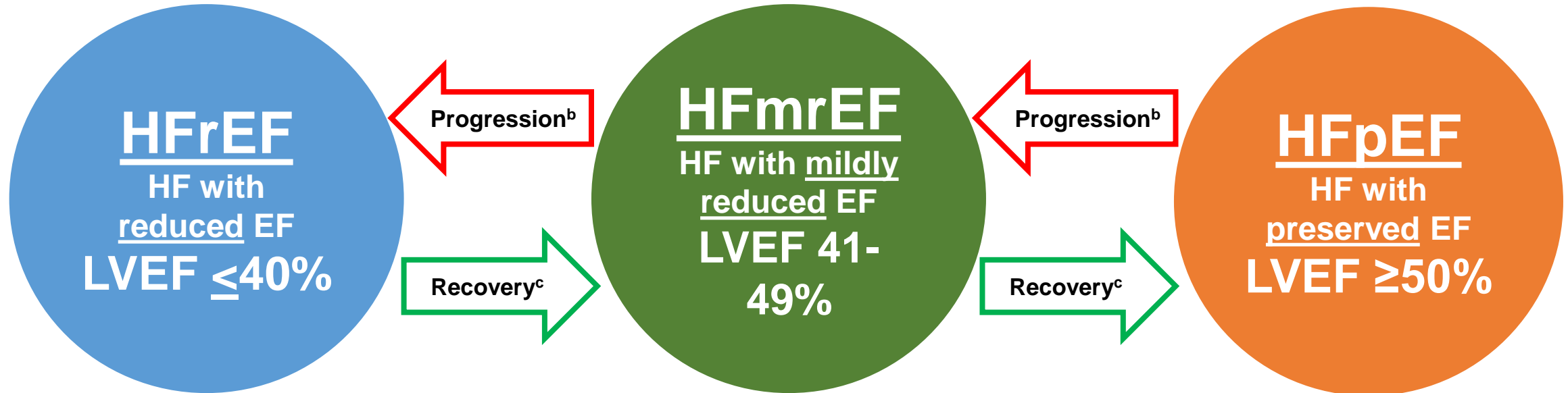
© ESC 2021

BNP = B-type natriuretic peptide; ECG = electrocardiogram; HbA1c = glycated haemoglobin; NT-proBNP = N-terminal pro-B-type natriuretic peptide; TSAT = transferrin saturation. (a) Class of recommendation. (b) Level of evidence. (c) References are listed in section 4.2 for this item.



Changes in LVEF Occur Over Time and Are Associated With Specific Patient Characteristics

HF Categories According to LVEF ^{1,2}



Factors associated with progression^a:

Diabetes, ischemic heart disease, lack of specialized HF follow-up, higher NT-proBNP levels

Factors associated with recovery^b:

Younger age, female, lower HF severity, shorter HF duration, fewer comorbidities

Data from patients with ≥ 2 EF measurements in the SwedeHF study (N=4942) between May 2000 and December 2012.

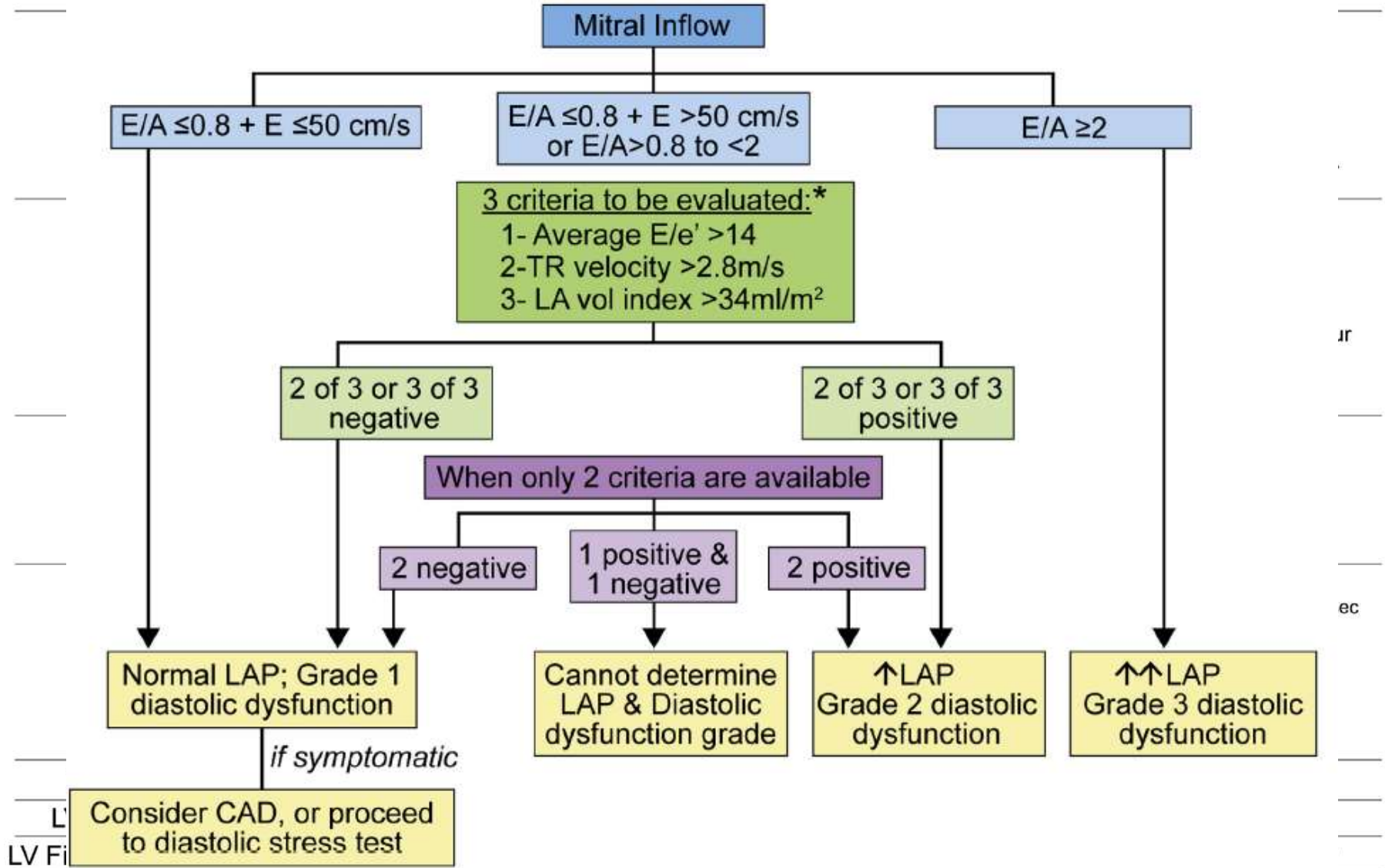
^aEF decrease; ^bEF increase. 1. Bozkurt B et al. *Eur J Heart Fail.* 2021;23:352-380; 2. McDonagh TA et al. *Eur Heart J.* 2021;42:3599-3726.

EF = ejection fraction; HF = heart failure; HFmrEF = heart failure with mildly reduced ejection fraction; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-B-type natriuretic peptide.

Savarese G et al. *JACC Heart Fail.* 2019;7:306-317.

Estimating LAP in Patients with Diastolic Dysfunction

Characteristic hemodynamic pressure and Doppler echocardiographic findings seen with different LV filling patterns



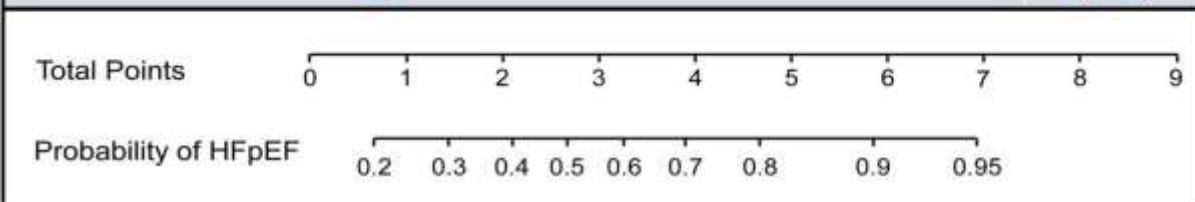
*PV S/D ratio < 1 applicable to conclude increased LAP if LVEF is depressed

A Simple, Evidence-Based Approach to Help Guide Diagnosis of Heart Failure With Preserved Ejection Fraction

Reddy Y.N.V. et al.

Circulation. 2018;138:861–870. DOI: 10.1161/CIRCULATIONAHA.118.034646

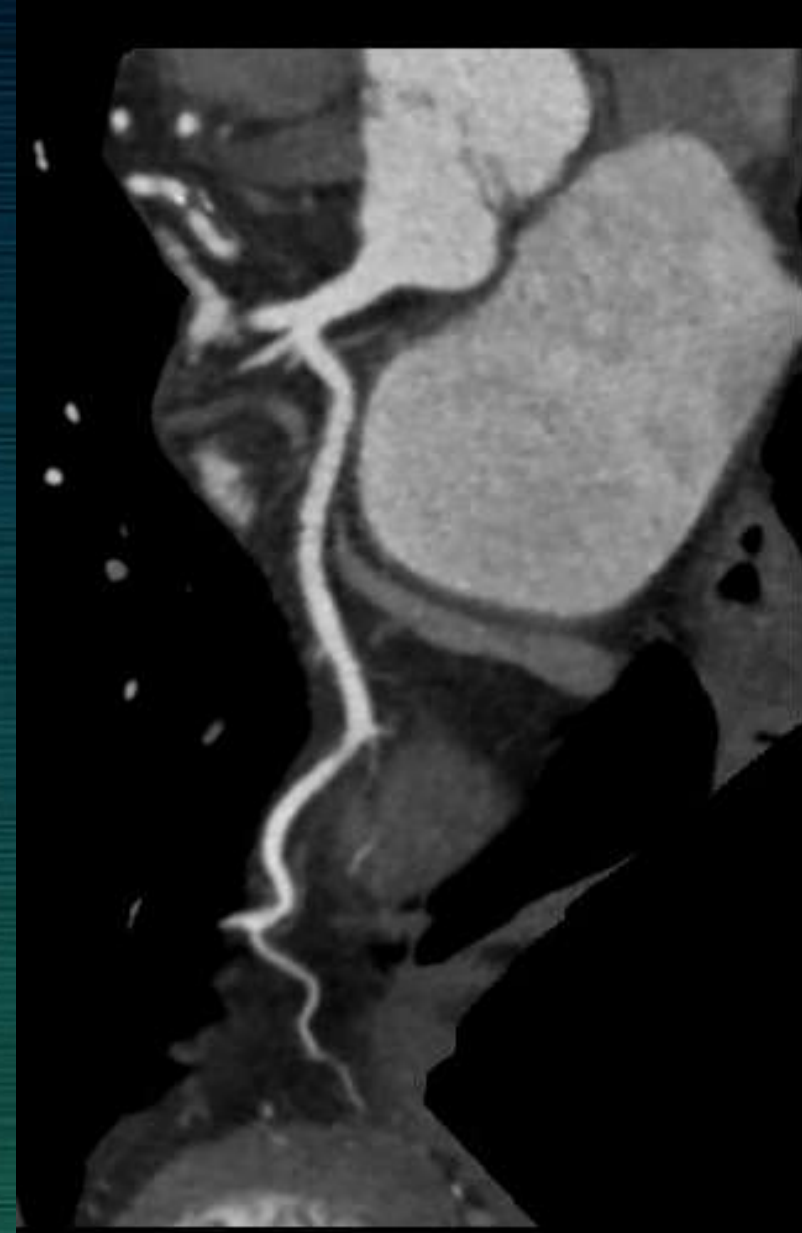
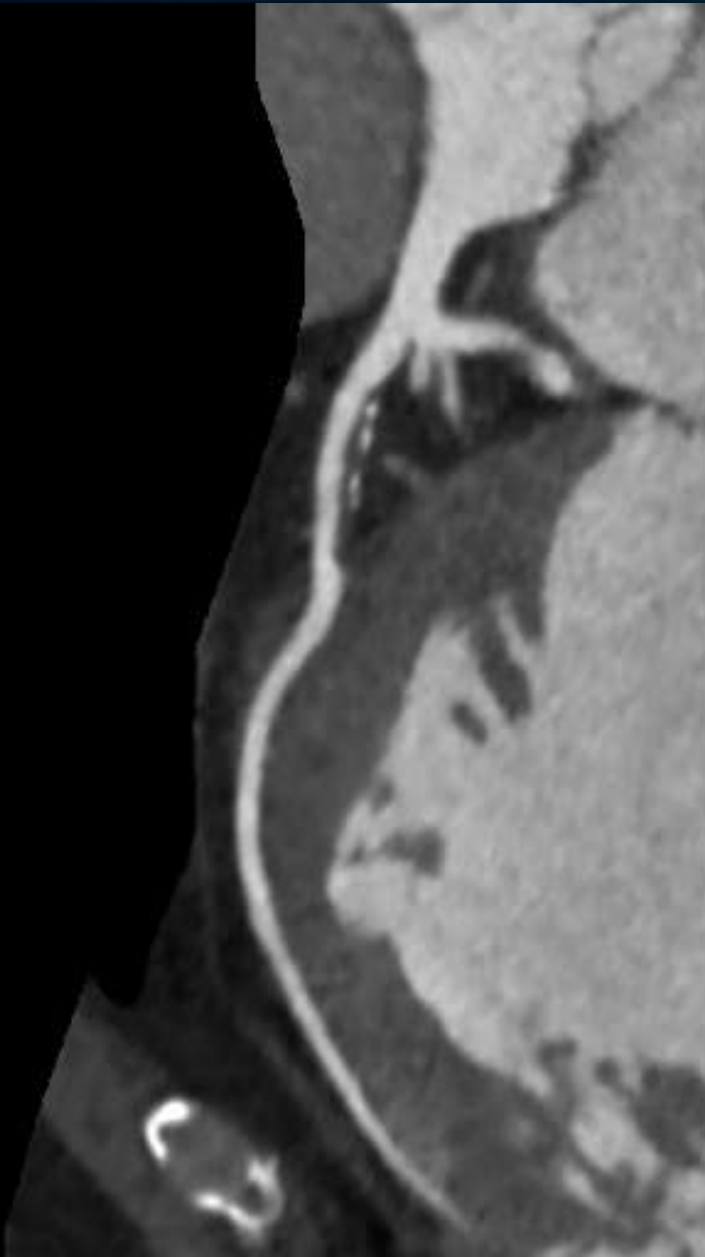
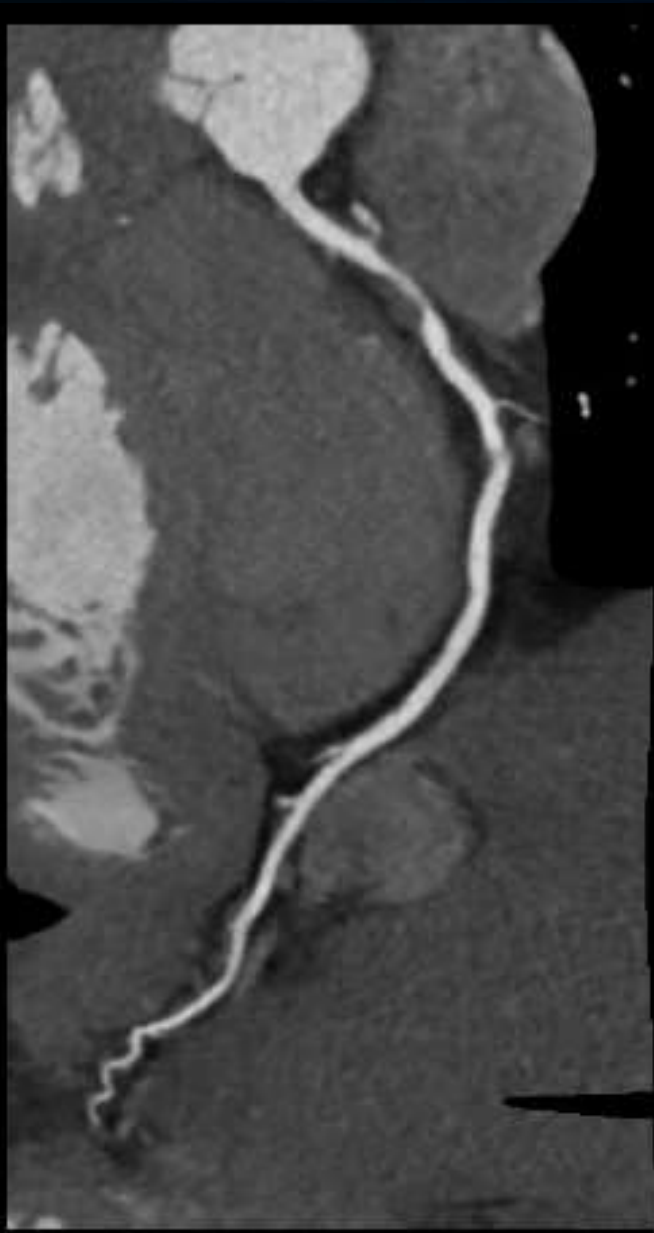
	Clinical Variable	Values	Points
H₂	H heavy	Body mass index > 30 kg/m ²	2
	H ypertensive	2 or more antihypertensive medicines	1
F	Atrial F ibrillation	Paroxysmal or Persistent	3
P	P ulmonary Hypertension	Doppler Echocardiographic estimated Pulmonary Artery Systolic Pressure > 35 mmHg	1
E	E lder	Age > 60 years	1
F	F illing Pressure	Doppler Echocardiographic E/e' > 9	1
H₂FPEF score			Sum (0-9)



How to diagnose heart failure with preserved ejection fraction: the HFA-PEFF diagnostic algorithm: a consensus recommendation from the Heart Failure Association (HFA) of the European Society of Cardiology (ESC)

	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/m ² or LVMI ≥ 149/122 g/m ² (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		

HFpEF diagnosis



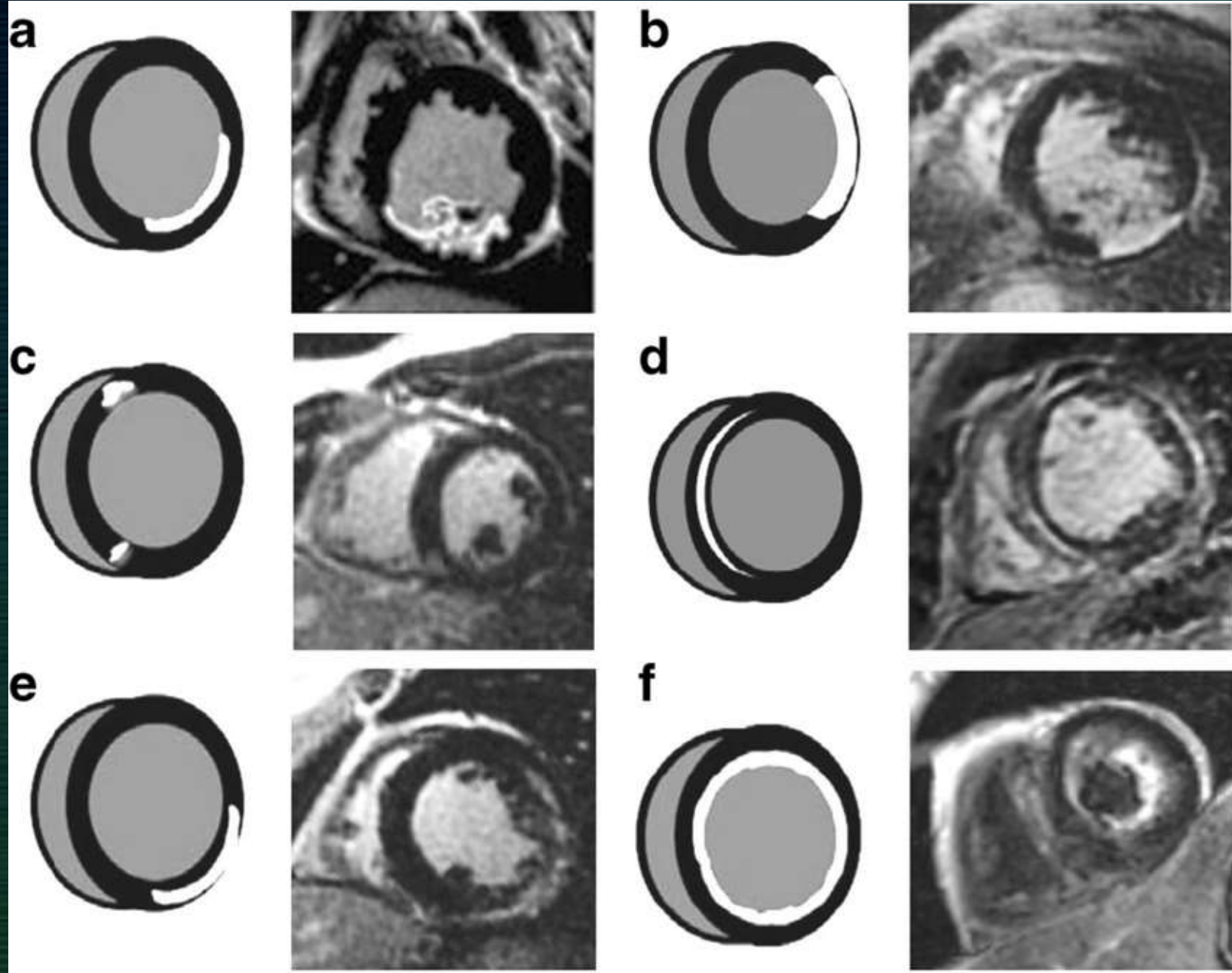
Advanced diagnostic tests

Cardiac Magnetic Resonance Imaging (CMR)

CMR represents the "gold standard" for the evaluation of cardiac morphology, function, and mass and is extremely useful for obtaining additional information for diagnostic, therapeutic, and prognostic purposes. It is the only non-invasive method at our disposal for tissue characterization, particularly through the assessment of the presence of "late gadolinium enhancement" (LGE), an increase in signal caused by the contrast medium lingering in the interstitial tissue, mostly attributable to fibrotic replacement.

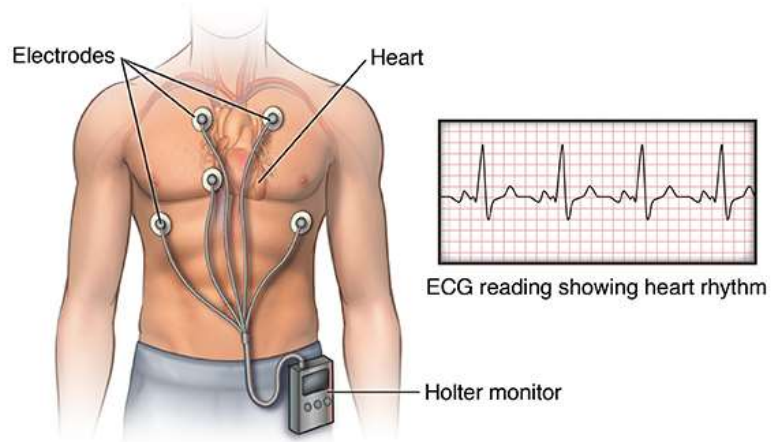
The distribution of LGE in the myocardium provides diagnostic indications (non-ischemic patterns: "mid-wall" and/or subepicardial) useful in differentiation from ischemic forms, and its detection has significant prognostic implications, in terms of distribution site and percentage compared to healthy myocardium.

New promising MRI techniques include the evaluation of "T1-mapping" and **extracellular volume**, allowing for the quantification of unorganized fibrosis not visible through standard post-contrast methods.



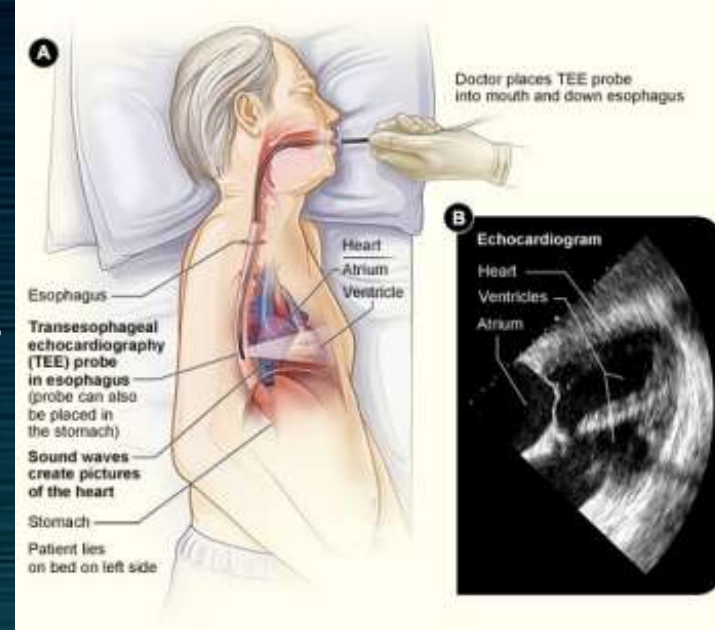
24-Hour Holter ECG/ 48-Hour Holter ECG

Holter monitor with ECG reading



Advanced diagnostic tests

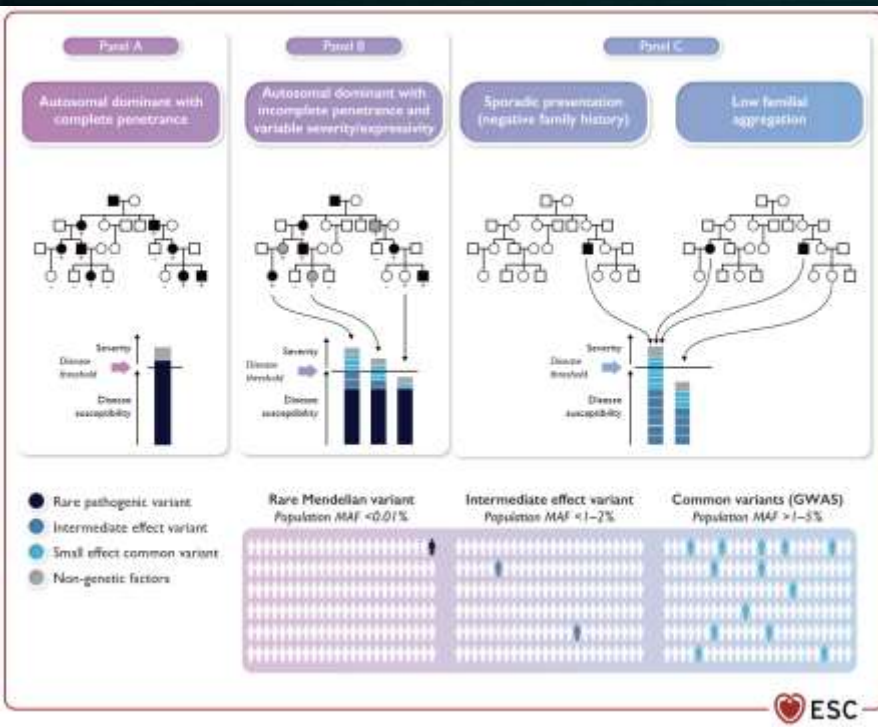
Transesophageal echocardiogram



Cardiopulmonary Exercise Test (CPET)



Genetic analysis

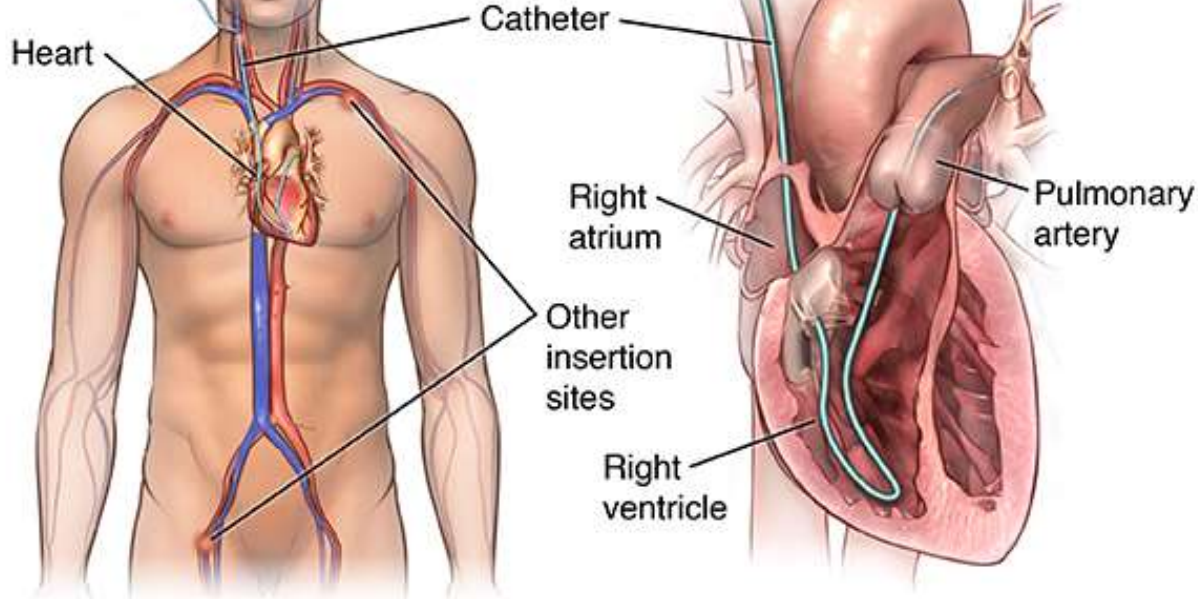


2023 ESC Guidelines for the management of cardiomyopathies

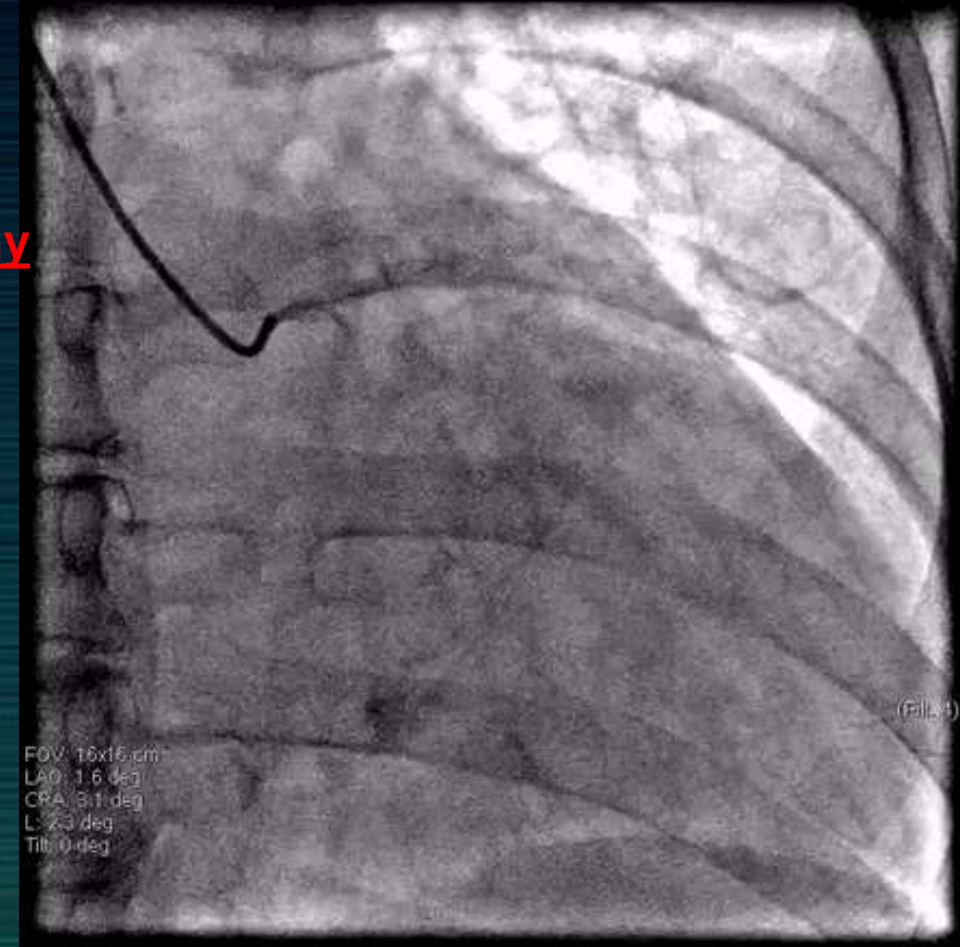
2023 ESC Guidelines for the management of cardiomyopathies (European Heart Journal; 2023 – doi:10.1093/eurheartj/ehad 194)

Advanced diagnostic tests

Right heart catheterization

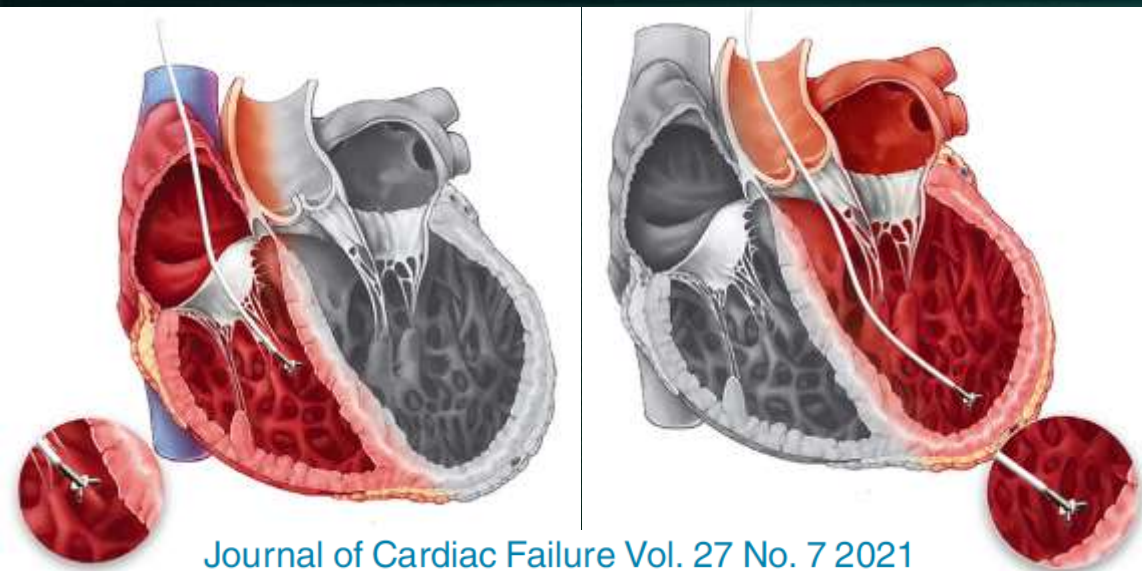


Coronary angiography



Endomyocardial Biopsy (EMB)

It is reasonable to consider endomyocardial biopsy (EMB) indicated in cases of severe, newly onset, unexplained, and refractory to conventional therapy heart failure, especially if associated with absent or mild left ventricular remodeling, and in cases presenting with life-threatening unexplained tachyarrhythmias or advanced atrioventricular blocks associated with left ventricular dysfunction. Moreover, EMB can be useful in cases of unclear etiology or in suspicion of storage or infiltrative diseases.



La diagnosi
nello scompenso cardiaco:
non fermiamoci al livello «letterale»



Dante col libro della Commedia, tre regni e la città di Firenze,
Domenico di Michelino e Alesso Baldovinetti, 1465, Santa Maria del Fiore, Firenze

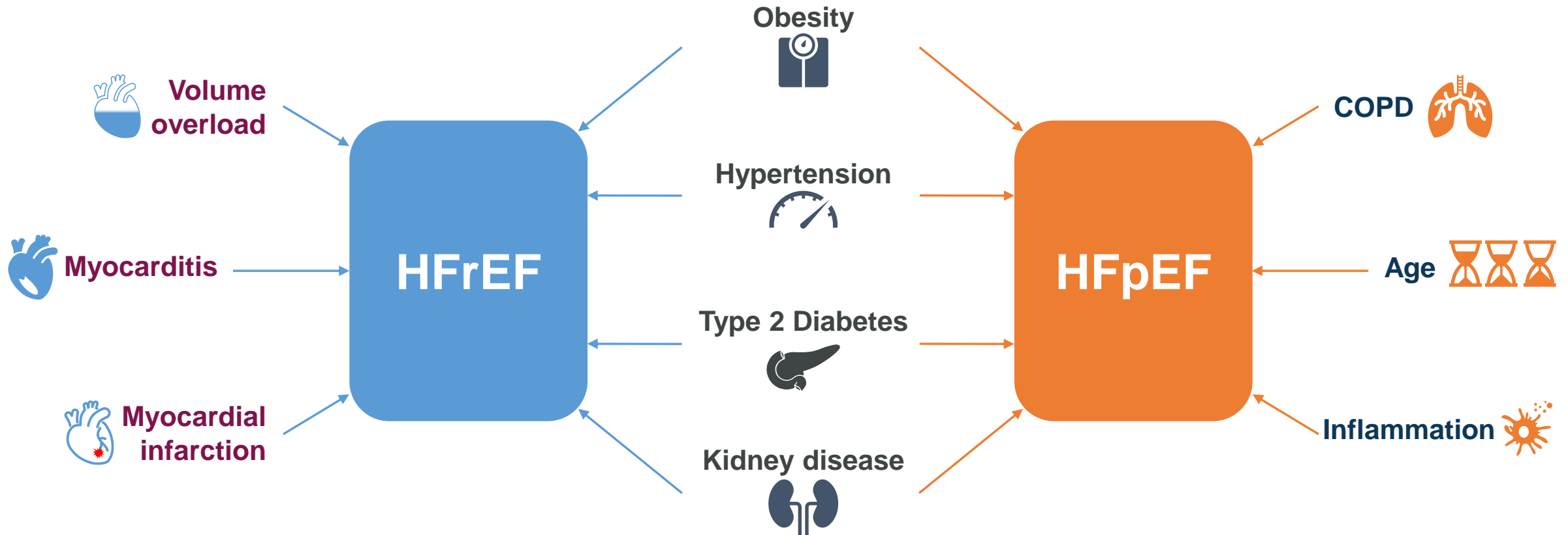
2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

Causes of heart failure, common modes of presentation and specific investigations

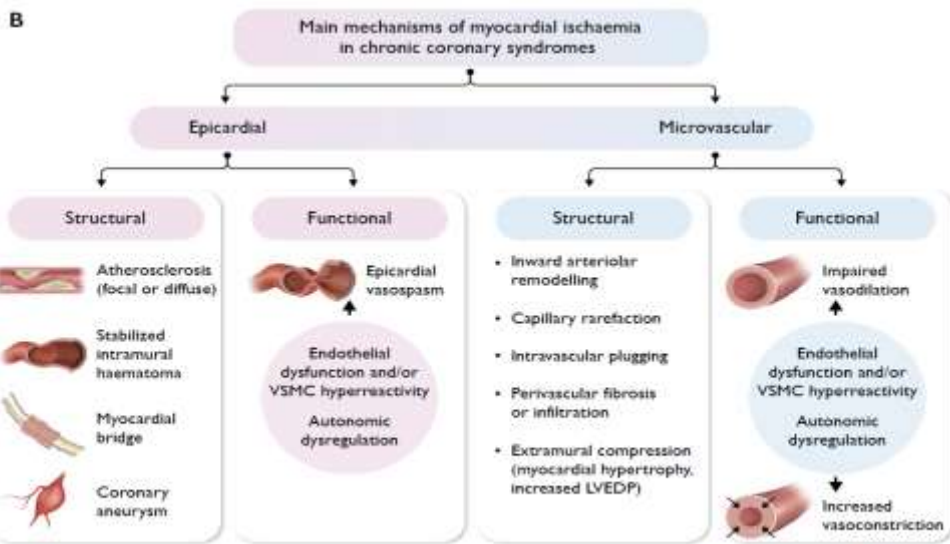
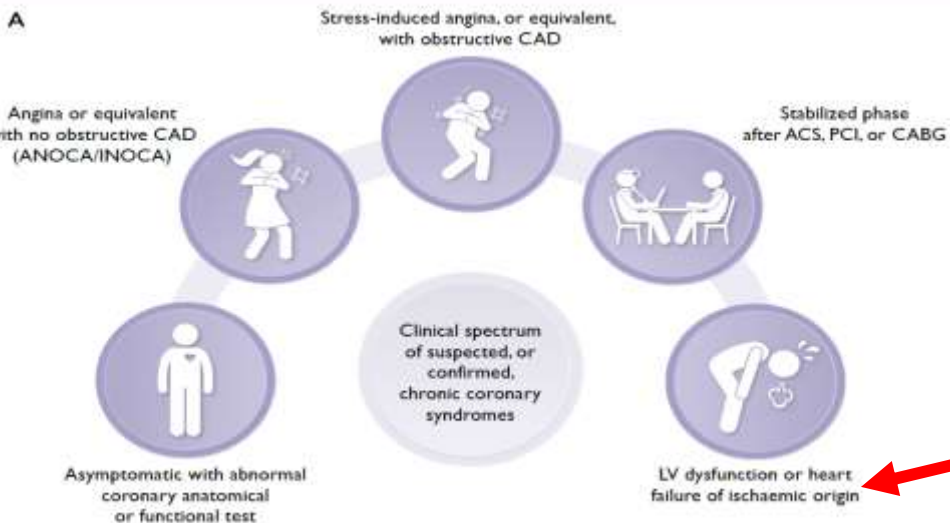
Cause	Examples of presentations	Specific investigations
CAD	Myocardial infarction Angina or "angina-equivalent" Arrhythmias	Invasive coronary angiography CT coronary angiography Imaging stress tests (echo, nuclear, CMR)
Hypertension	Heart failure with preserved systolic function Malignant hypertension/acute pulmonary oedema	24 h ambulatory BP Plasma metanephrines, renal artery imaging Renin and aldosterone
Valve disease	Primary valve disease e.g. aortic stenosis Secondary valve disease, e.g. functional regurgitation Congenital valve disease	Echo – transoesophageal/stress
Arrhythmias	Atrial tachyarrhythmias Ventricular arrhythmias	Ambulatory ECG recording Electrophysiology study, if indicated
CMPs	All Dilated Hypertrophic Restrictive ARVC Peripartum Takotsubo syndrome Toxins: alcohol, cocaine, iron, copper	CMR, genetic testing Right and left heart catheterization CMR, angiography Trace elements, toxicology, LFTs, GGT
Congenital heart disease	Congenitally corrected/repai red transposition of great arteries Shunt lesions Repaired tetralogy of Fallot Ebstein's anomaly	CMR
Infective	Viral myocarditis Chagas disease HIV Lyme disease	CMR, EMB Serology
Drug-induced	Anthracyclines Trastuzumab VEGF inhibitors Immune checkpoint inhibitors Proteasome inhibitors RAF+MEK inhibitors	
Infiltrative	Amyloid Sarcoidosis Neoplastic	Serum electrophoresis and serum free light chains, Bence Jones protein, bone scintigraphy, CMR, CT-PET, EMB Serum ACE, CMR, FDG-PET, chest CT, EMB CMR, EMB
Storage disorders	Haemochromatosis Fabry disease Glycogen storage diseases	Iron studies, genetics, CMR (T2* imaging), EMB α -galactosidase A, genetics, CMR (T1 mapping)
Endomyocardial disease	Radiotherapy Endomyocardial fibrosis/eosinophilia Carcinoid	CMR EMB 24 h urine 5-HIAA
Pericardial disease	Calcification Infiltrative	Chest CT, CMR, right and left heart catheterization
Metabolic	Endocrine disease Nutritional disease (thiamine, vitamin B1 and selenium deficiencies) Autoimmune disease	TFTs, plasma metanephrines, renin and aldosterone, cortisol Specific plasma nutrients ANA, ANCA, rheumatology review
Neuromuscular disease	Friedreich's ataxia Muscular dystrophy	Nerve conduction studies, electromyogram, genetics CK, electromyogram, genetics

5-HIAA = 5-hydroxyindoleacetic acid; ACE = angiotensin-converting enzyme; ANA= anti-nuclear antibody; ANCA= anti-nuclear cytoplasmic antibody; ARVC = arrhythmogenic right ventricular cardiomyopathy; BP = blood pressure; CAD= coronary artery disease; CMP = cardiomyopathy; CMR= cardiac magnetic resonance; CK = creatinine kinase; CT = computed tomography; ECG = electrocardiogram; Echo = echocardiography; EMB = endomyocardial biopsy; FDG = fluorodeoxyglucose; GGT= gamma-glutamyl transferase; HIV = human immunodeficiency virus; h = hour; LFT = liver function test; LGE = late gadolinium enhancement; MEK =mitogen-activated protein kinase; PET = positron emission tomography; TFT = thyroid function test; VEGF = vascular endothelial growth factor

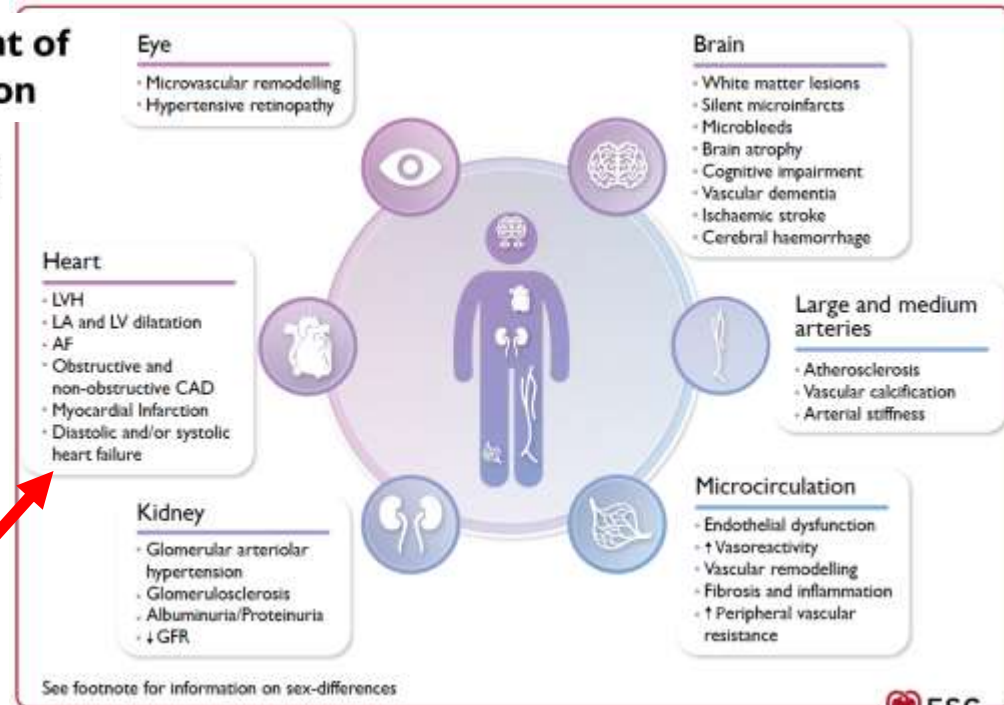
HFrEF and HFpEF Share Many Comorbidities and Risk Factors, While Others Differ



2024 ESC Guidelines for the management of chronic coronary syndromes

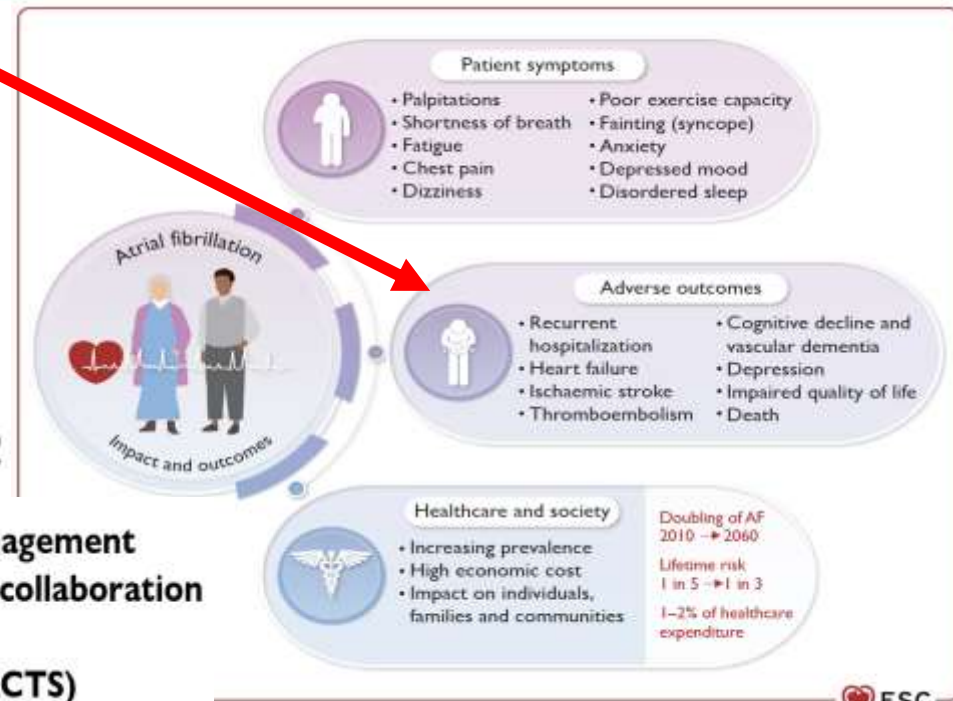


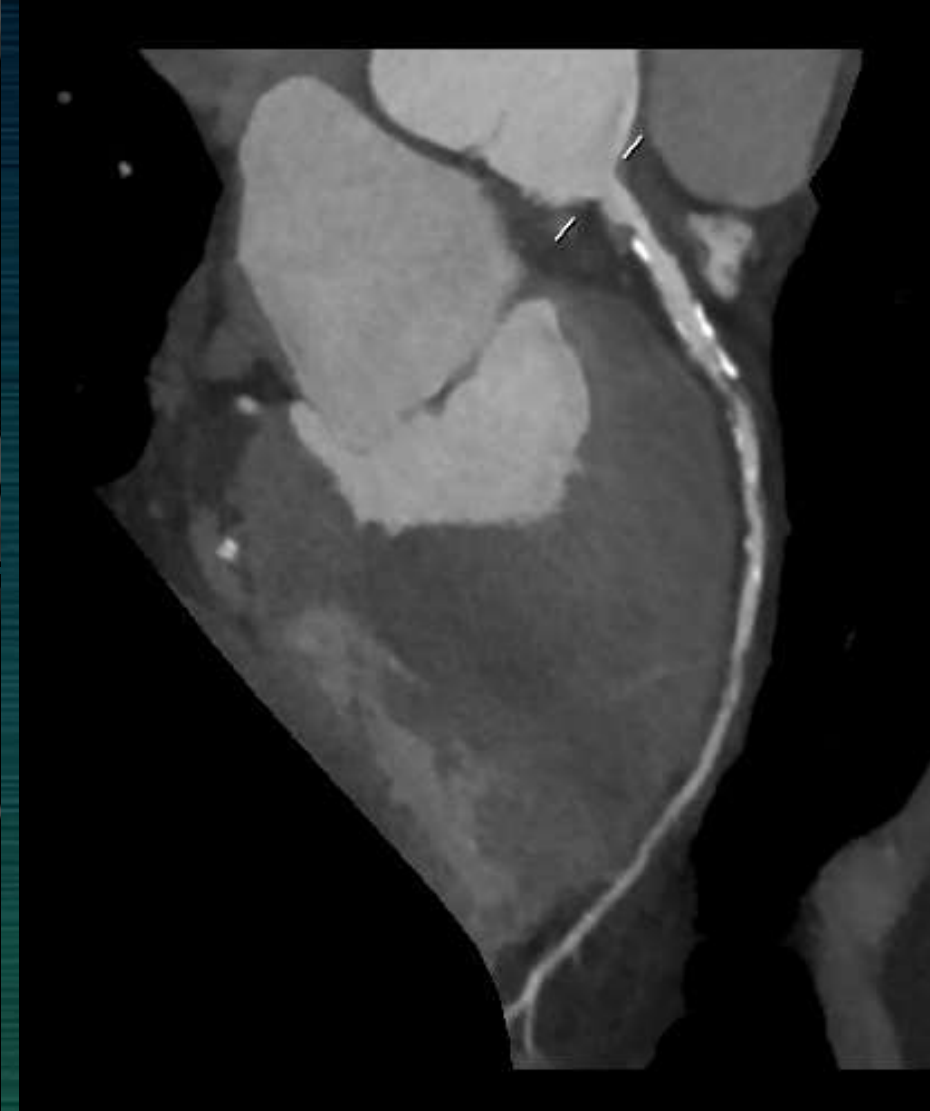
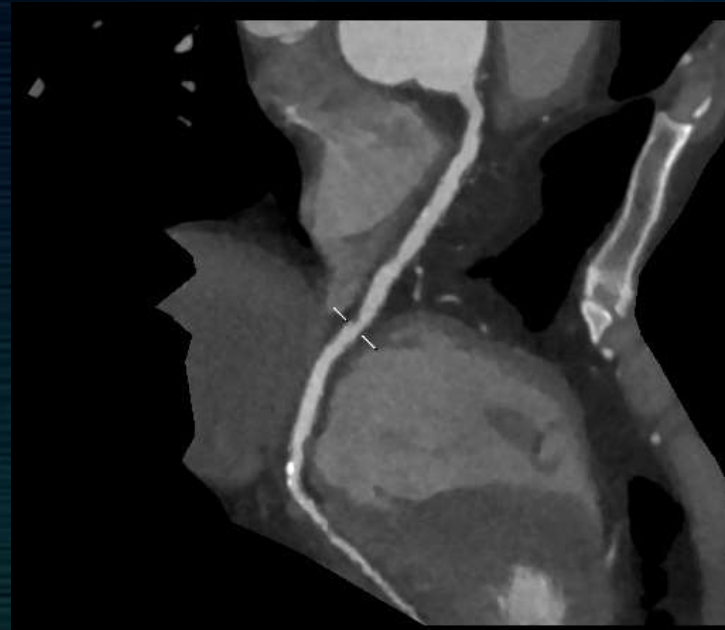
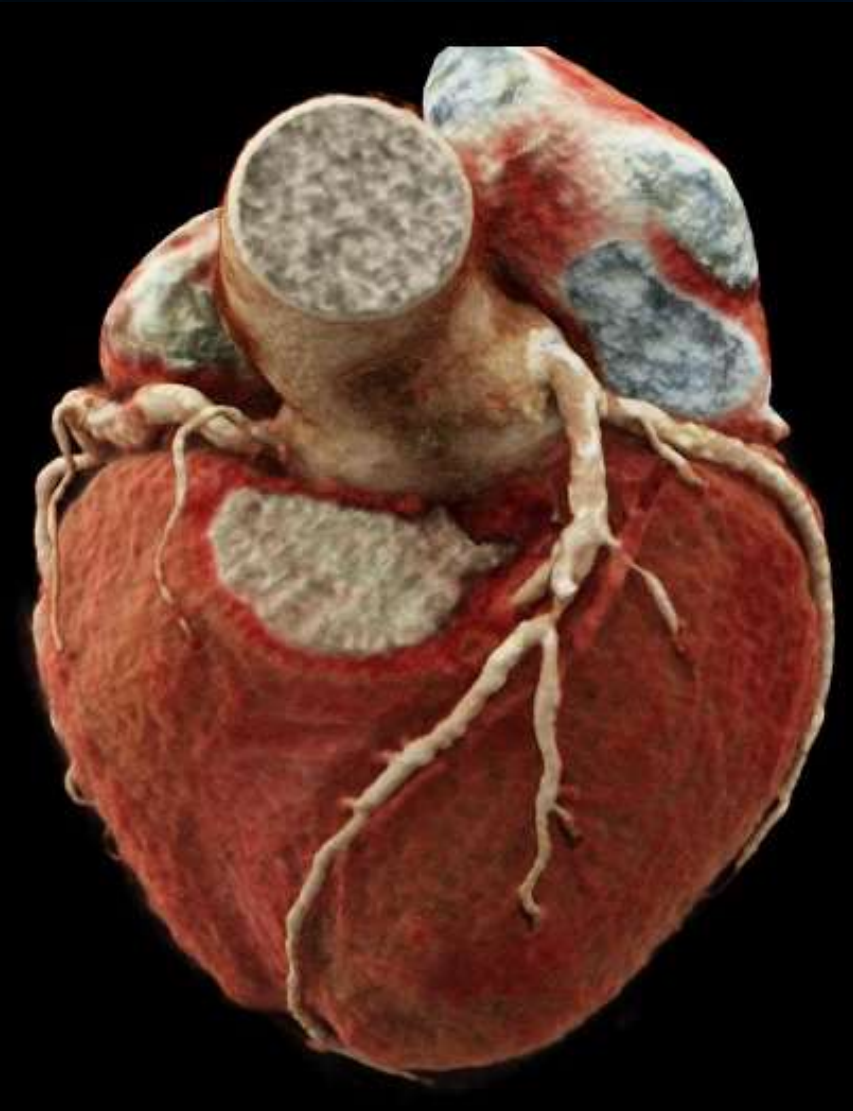
2024 ESC Guidelines for the management of elevated blood pressure and hypertension



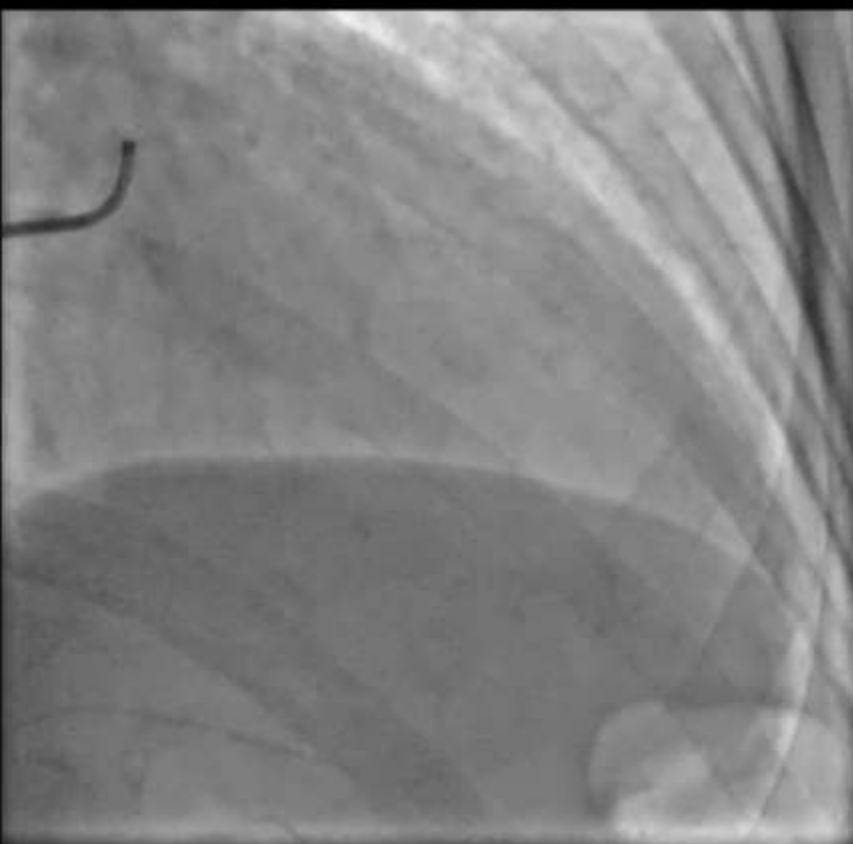
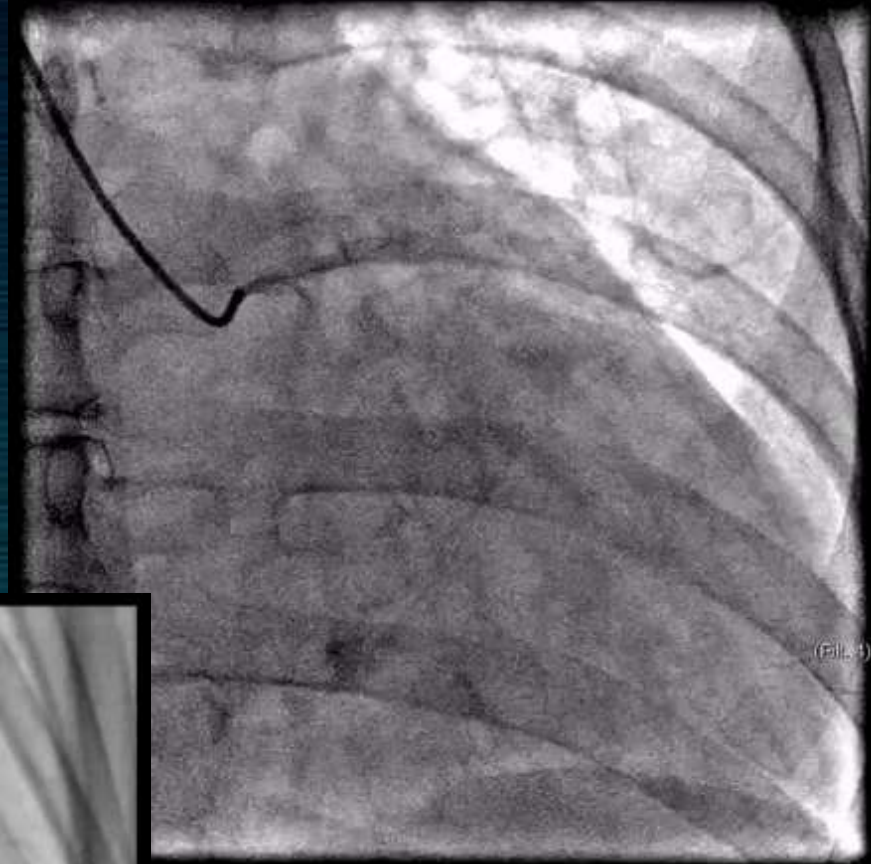
Heart failure, a central and current issue

2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS)



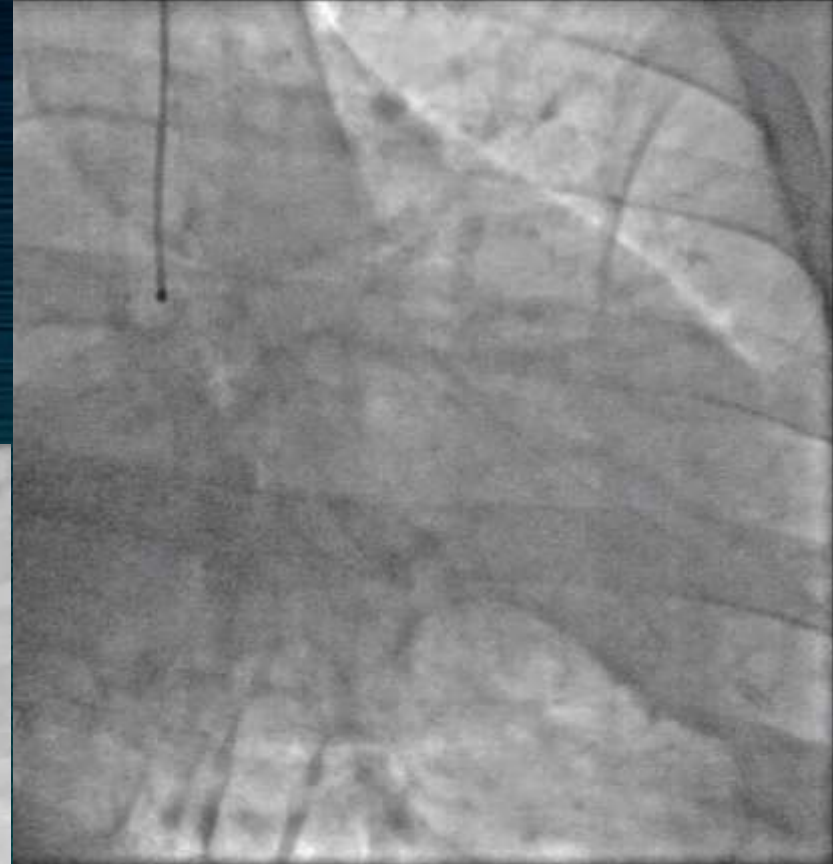
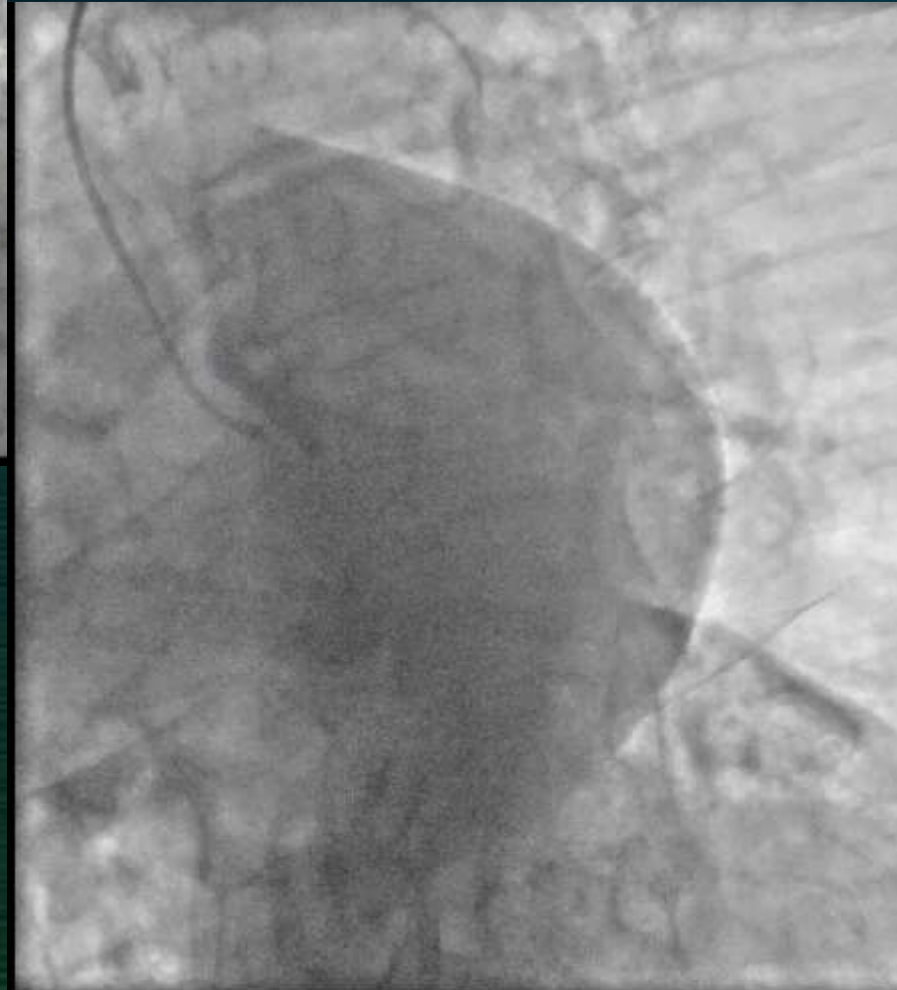


Coronary angiography



Normal

Coronary angiography:

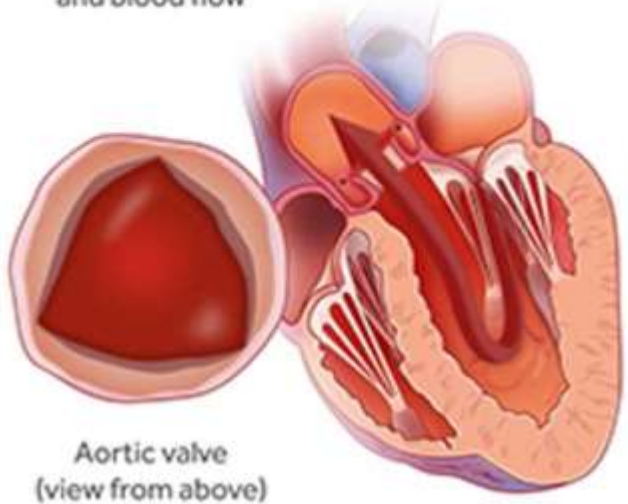


Three-vessel coronary artery disease with left main disease and chronically collateralized occlusion of the dominant right coronary artery

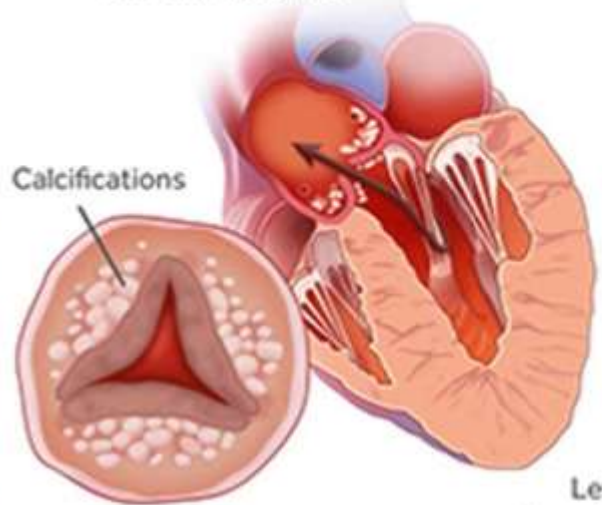
Valvular diseases

Aortic valve stenosis

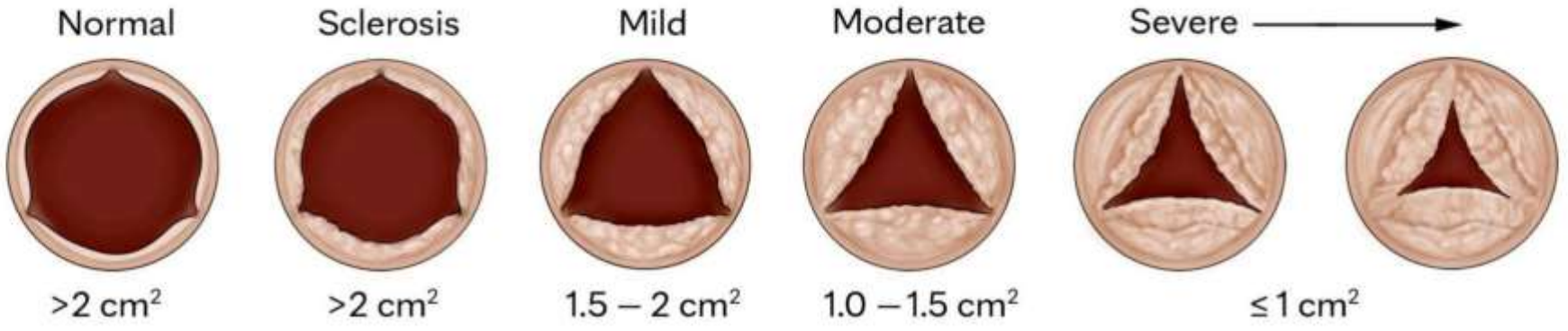
Normal aortic valve and blood flow



Calcifications narrow the aortic valve (stenosis) causing reduced blood flow

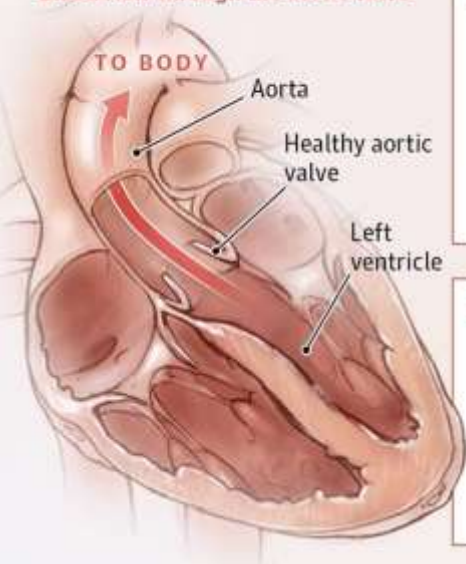


Left ventricular hypertrophy caused by severe aortic stenosis

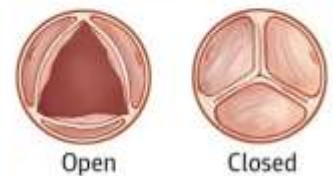


Aortic Valve Stenosis

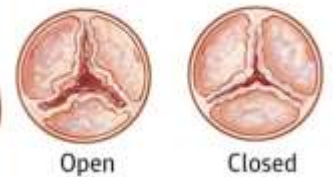
Blood flow through the aortic valve



Healthy aortic valve (top view)



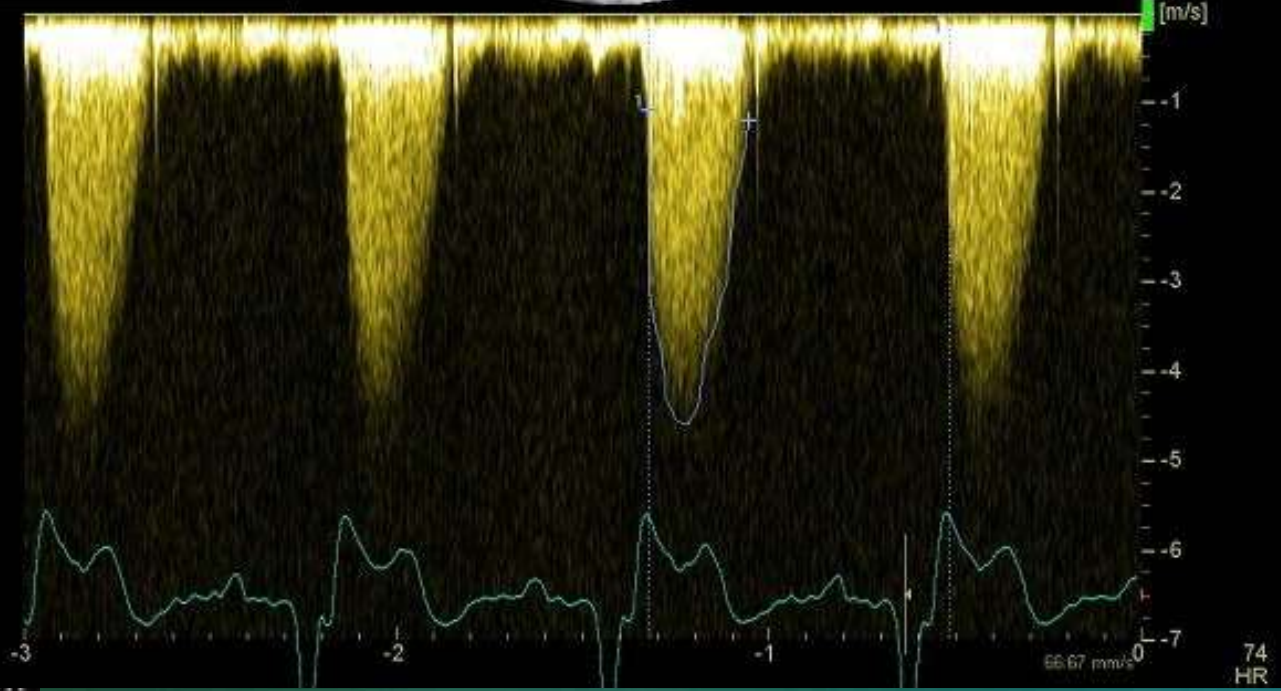
Aortic valve stenosis (top view)

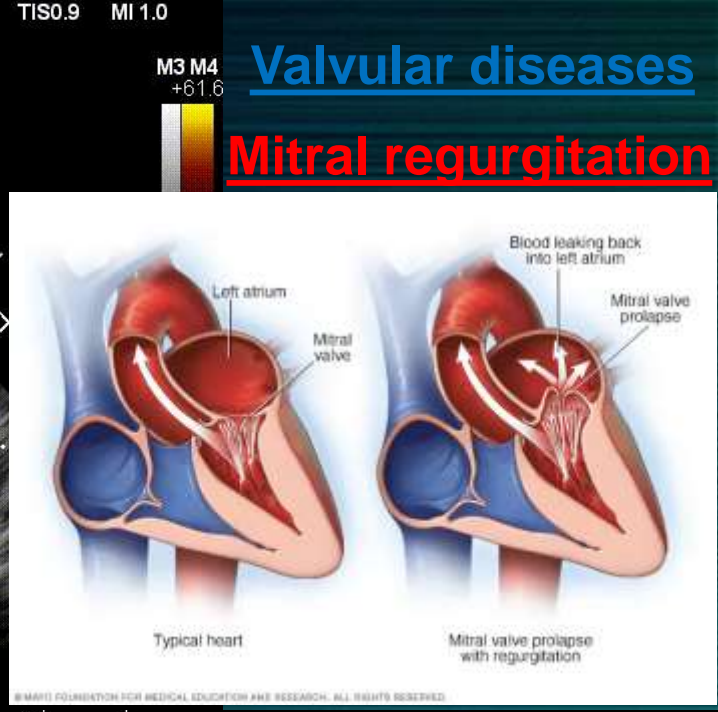
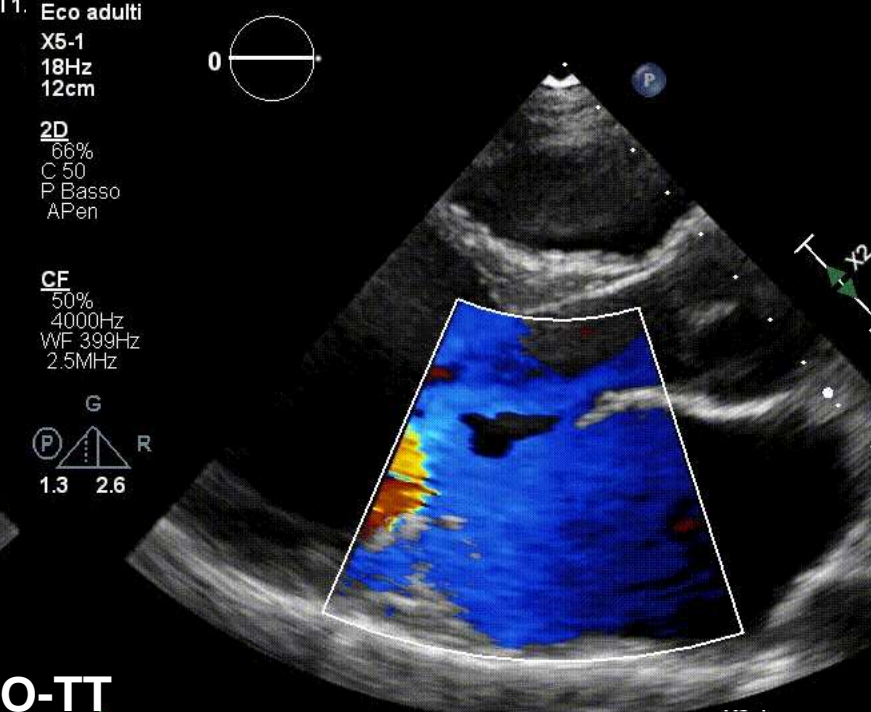
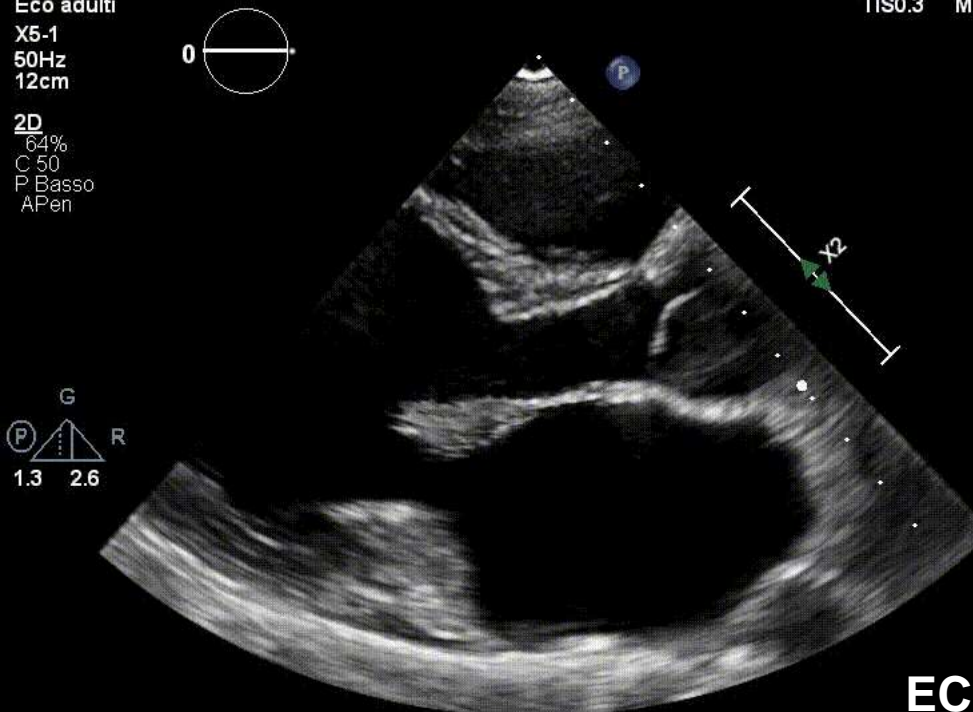


1 AV Vmax	4.58 m/s
AV Vmean	3.50 m/s
AV maxPG	83.93 mmHg
AV meanPG	52.84 mmHg
AV VTI	94.4 cm
AV Env.Ti	270 ms
HR	74 BPM

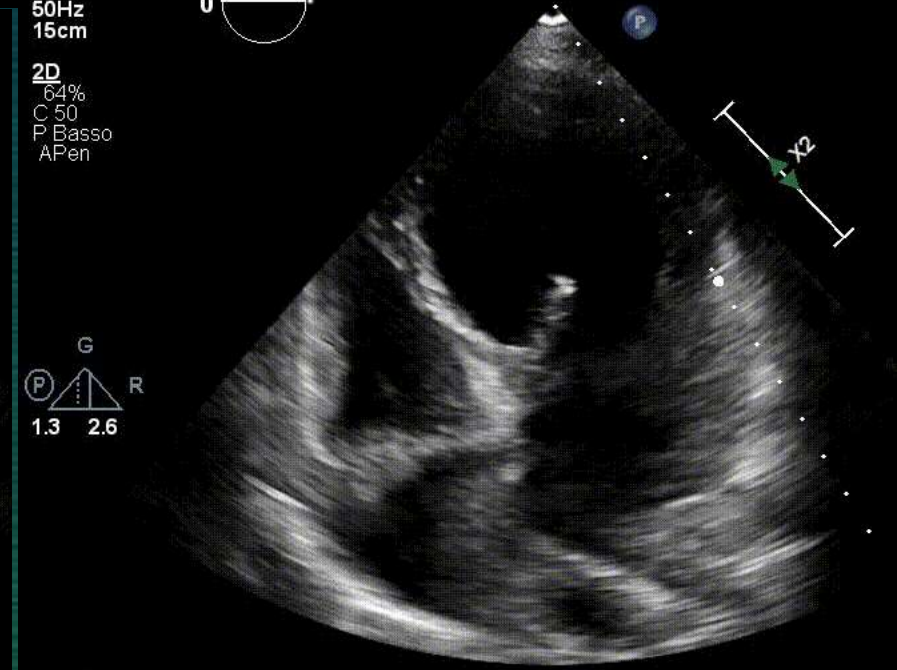
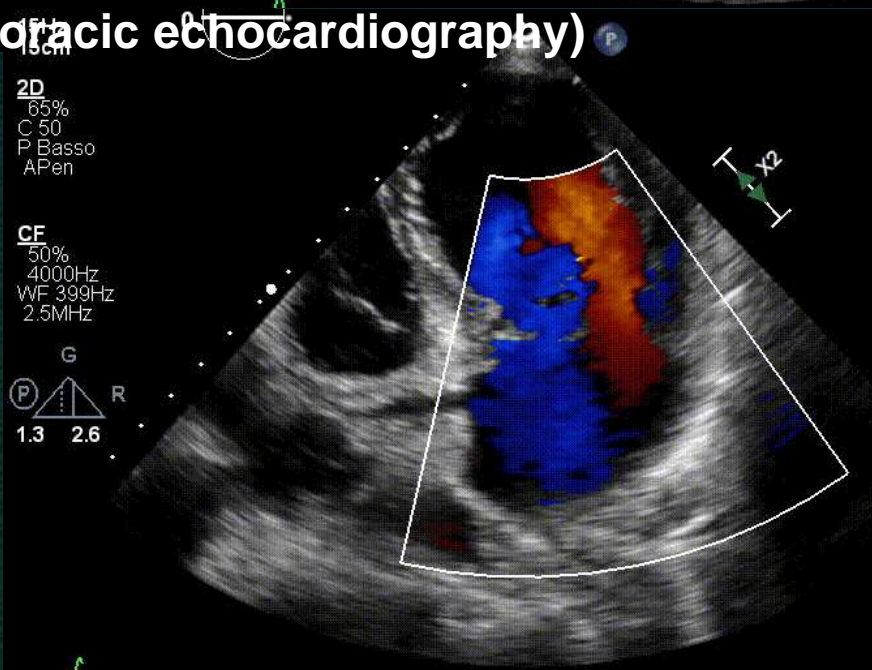
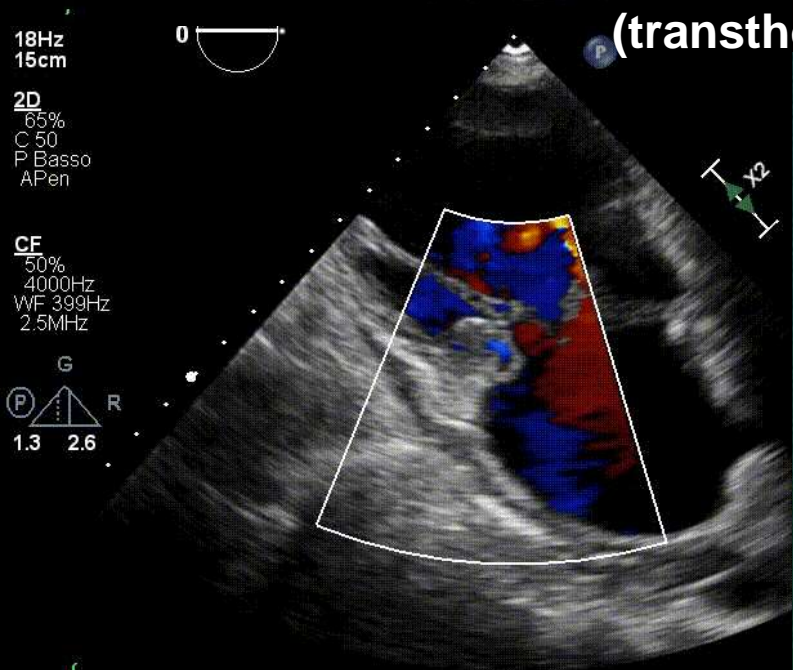
Valvular diseases

Aortic valve stenosis





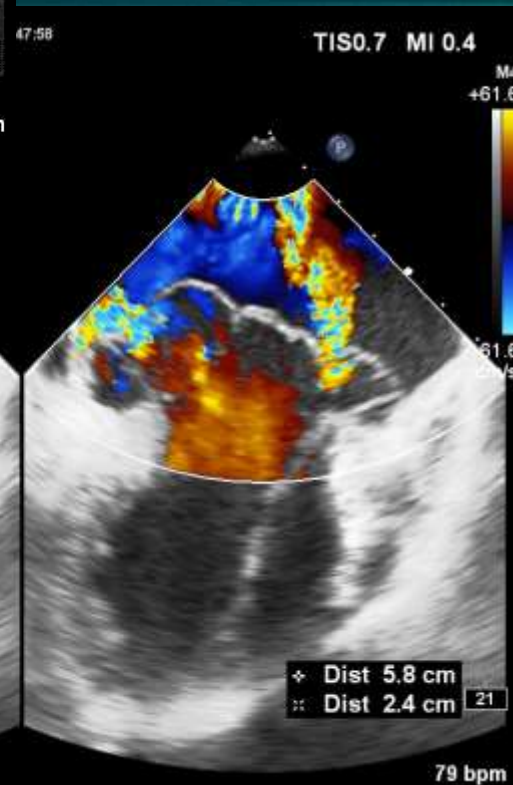
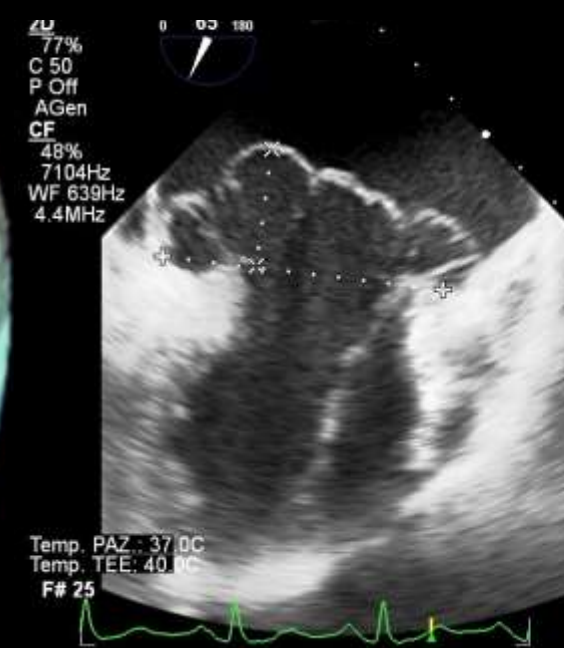
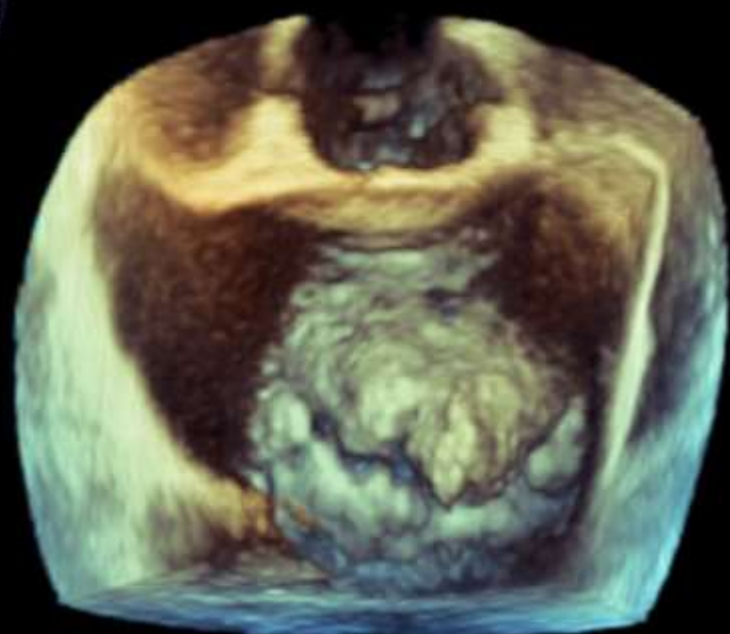
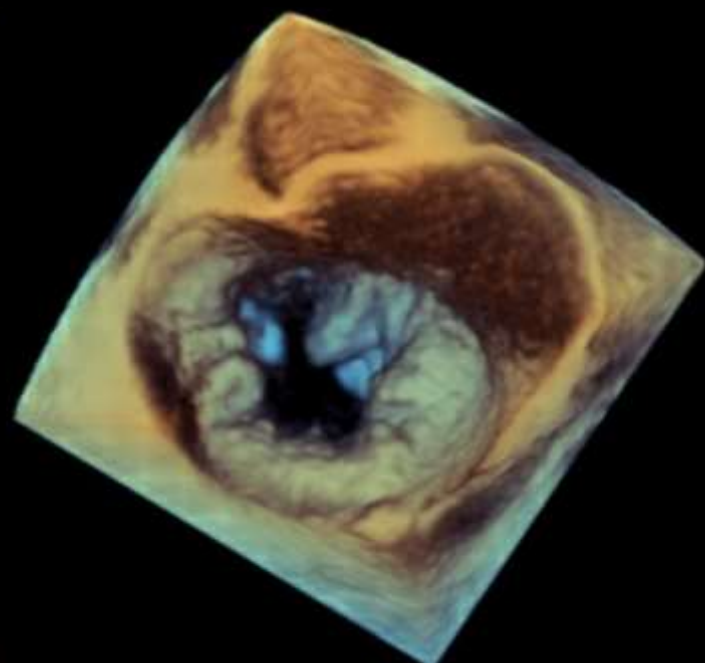
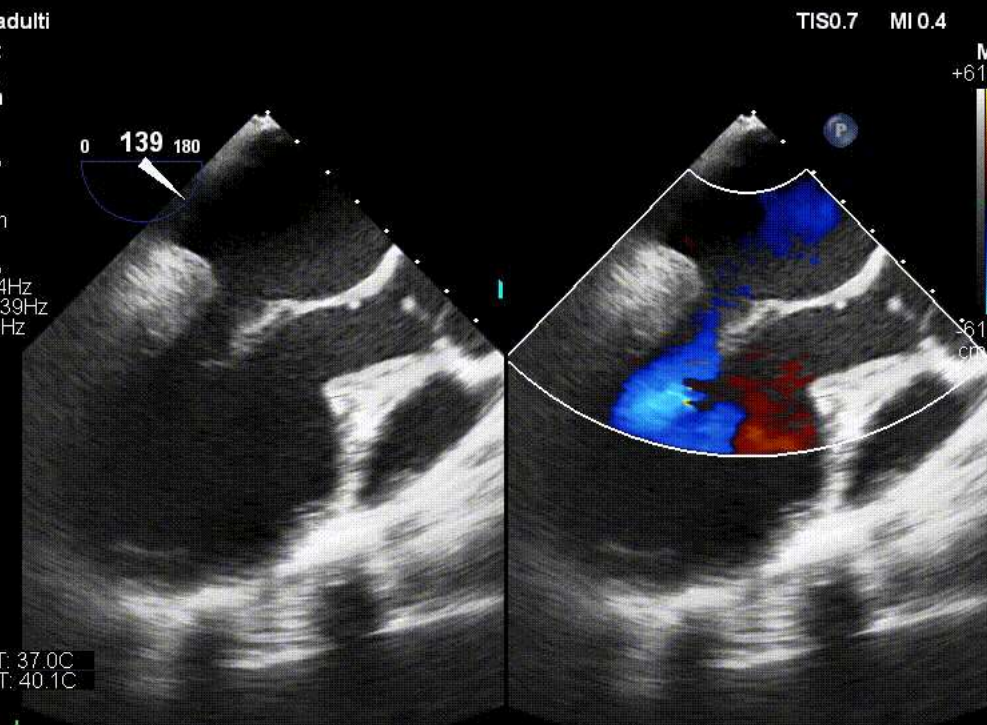
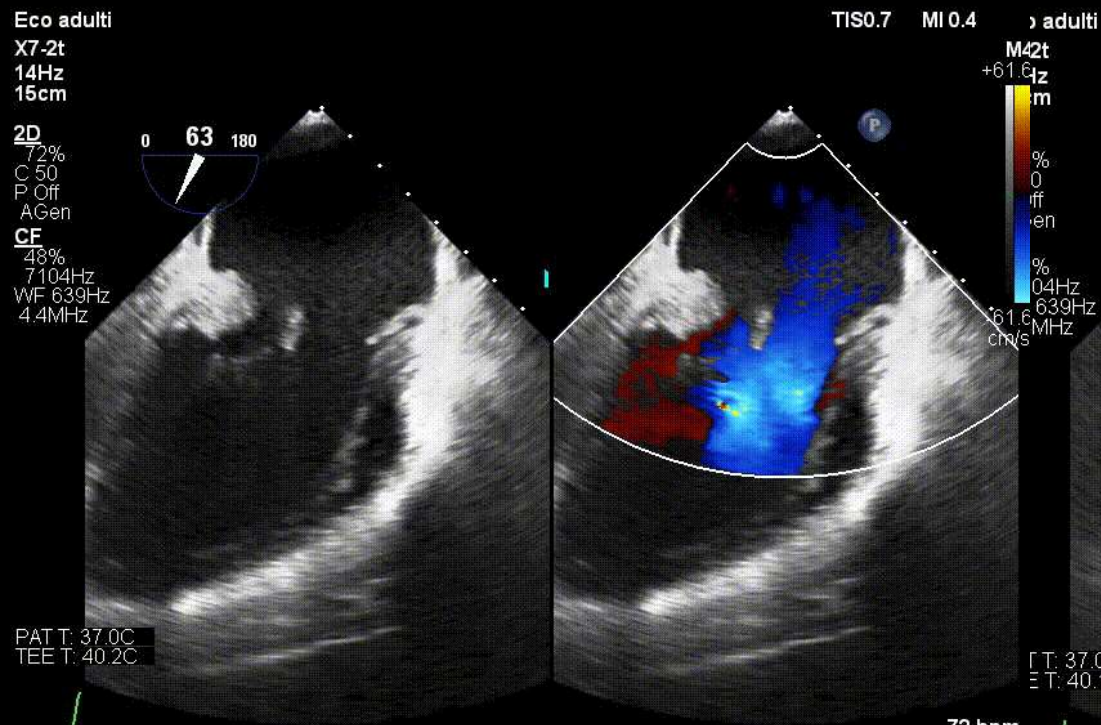
ECO-TT (transthoracic echocardiography)



Valvular diseases

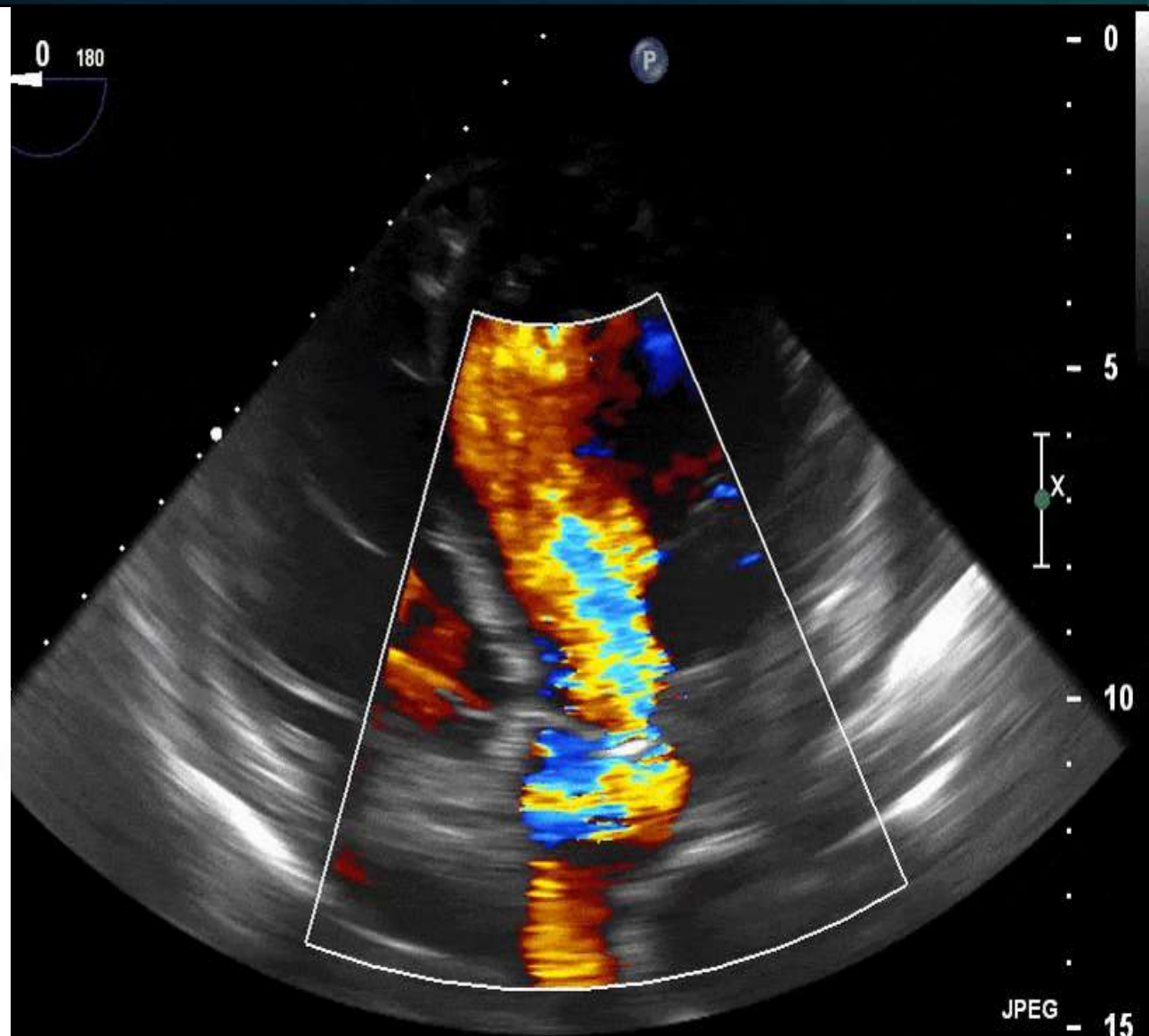
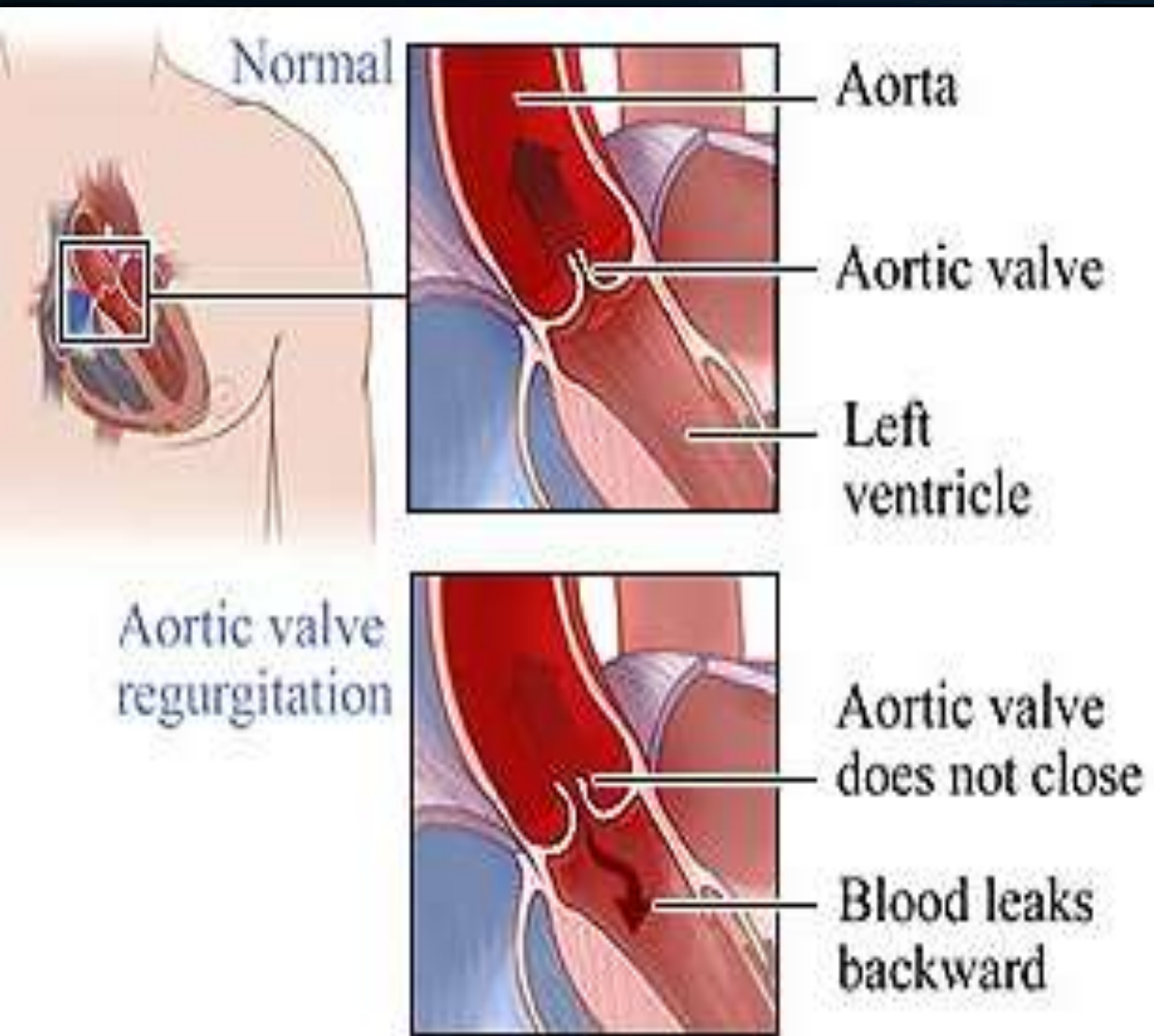
Mitral regurgitation

ECO-TE (transesophageal echocardiography)



Valvular diseases

Aortic valve regurgitation



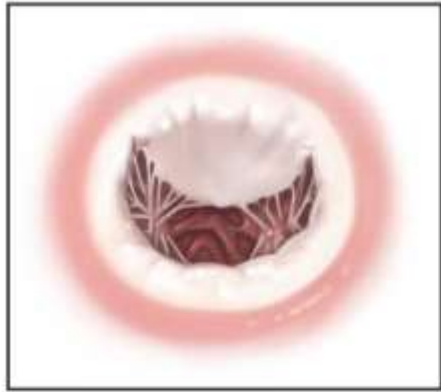
TT: 37.0C
ET: 38.9C

JPEG 15
74

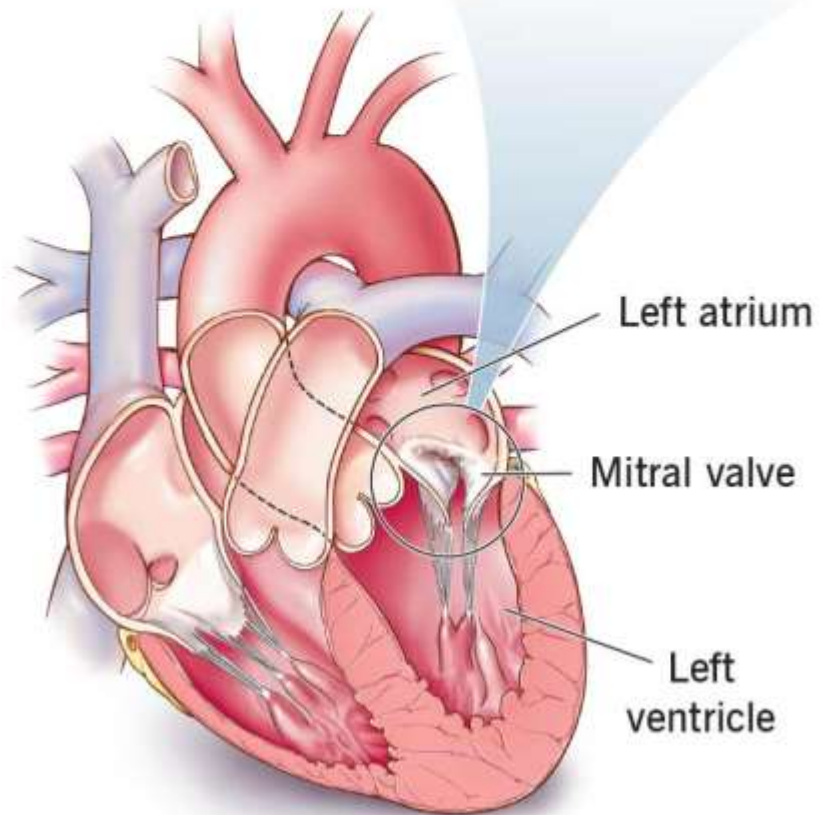
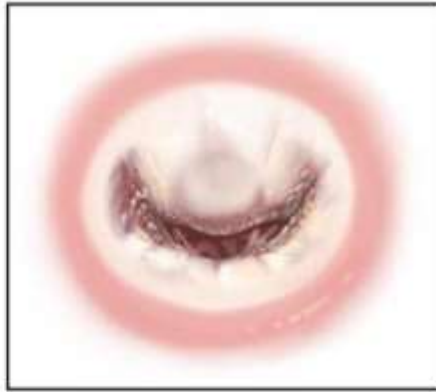
Valvular diseases

Mitral valve stenosis

Healthy mitral valve



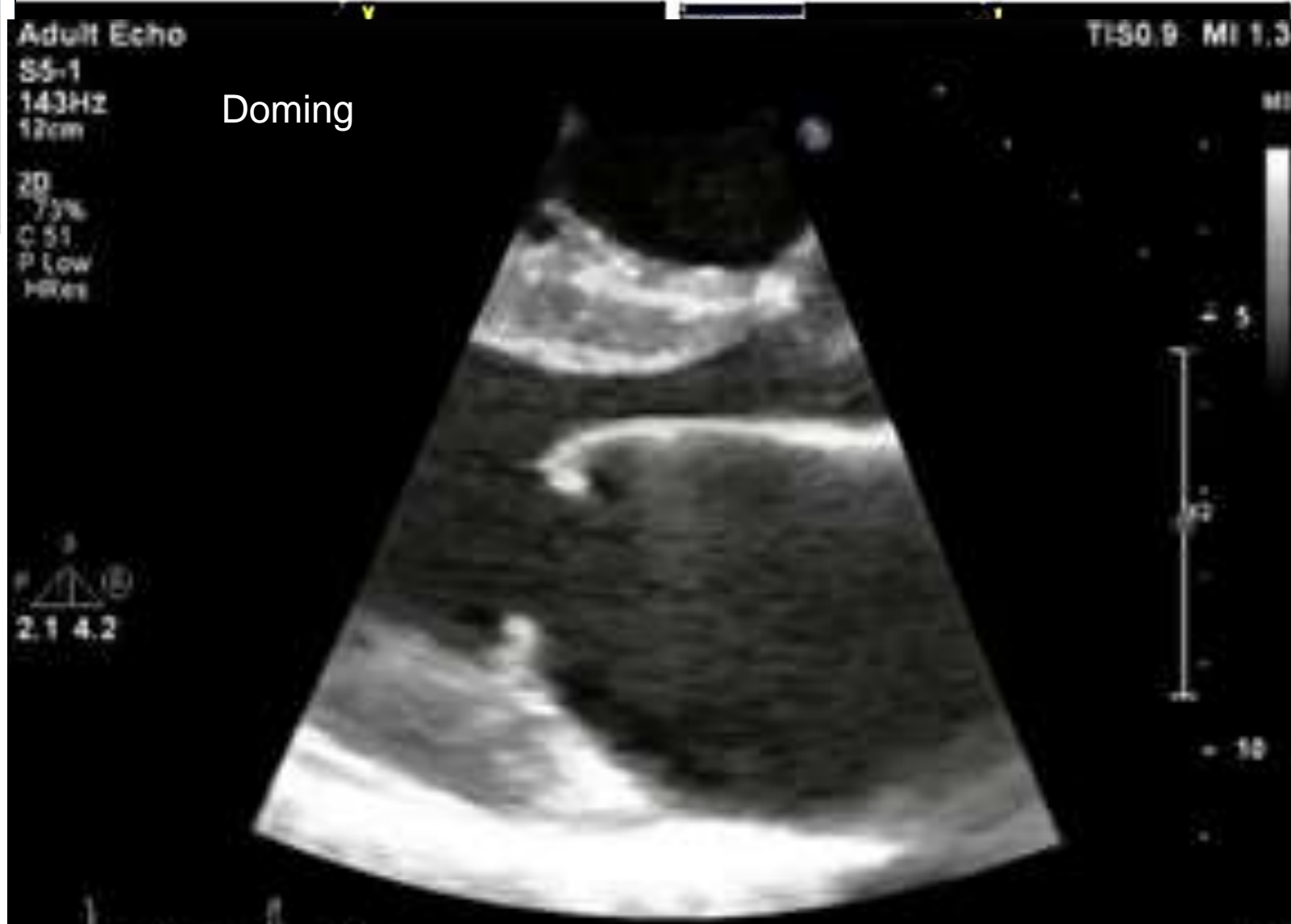
Mitral valve stenosis



Adult Echo
S5-1
143Hz
12cm
2D
73%
C 51
P Low
HR 81

Doming

2.1 4.2

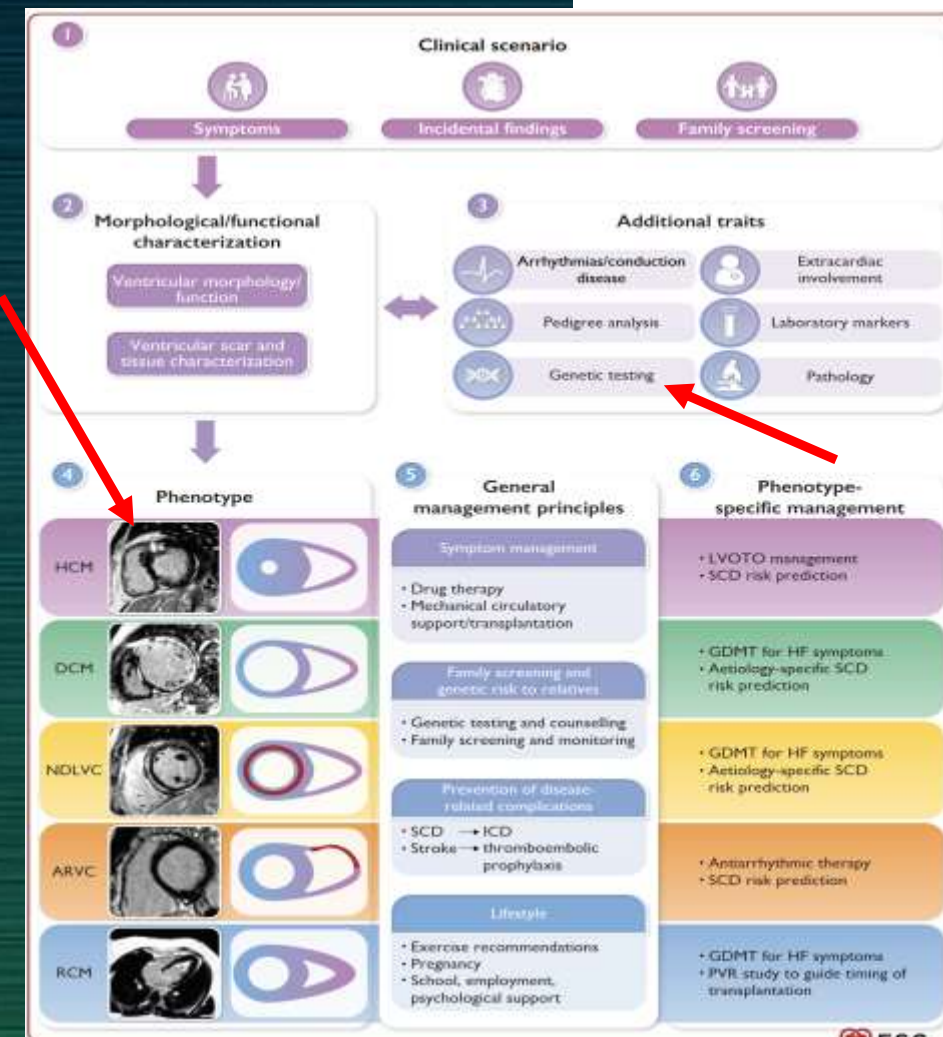
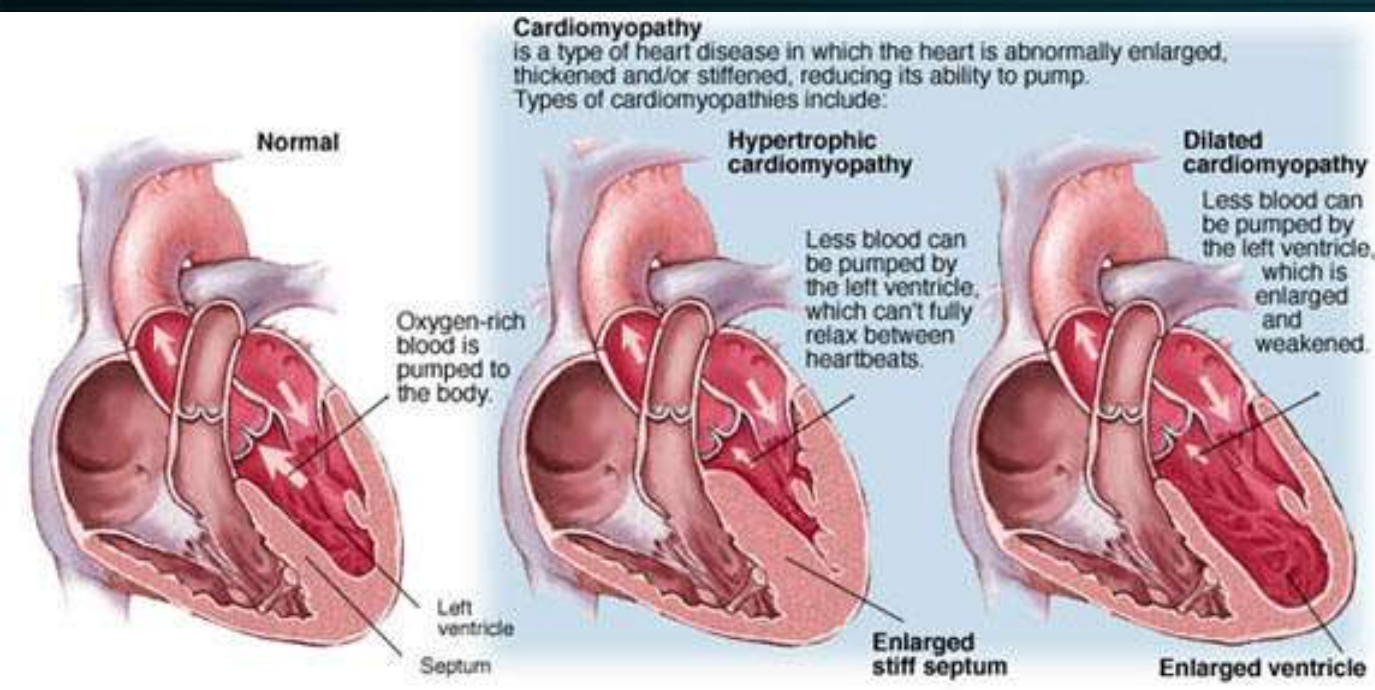


Handwritten signature

Cardiomyopathy is disease of the heart muscle.

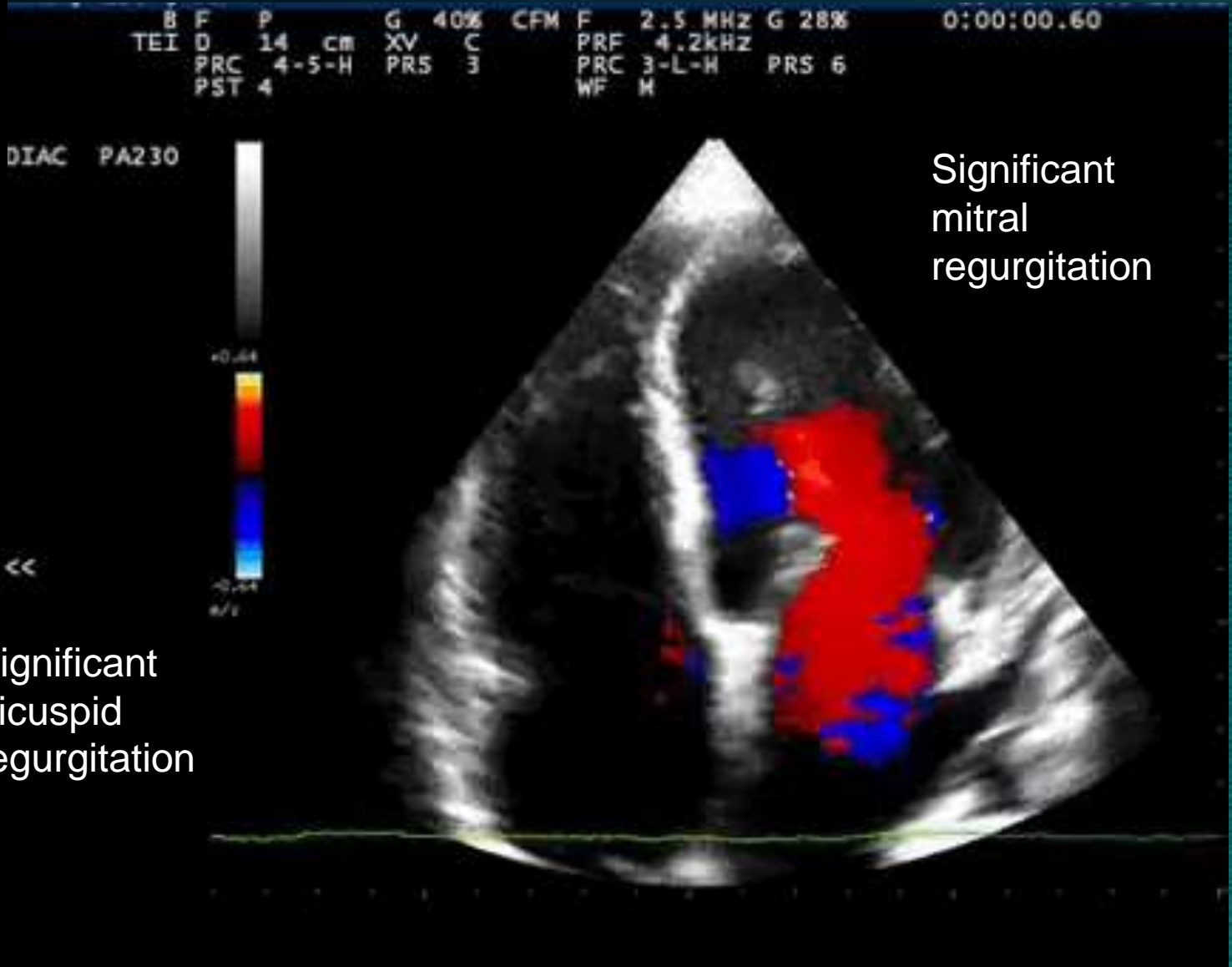
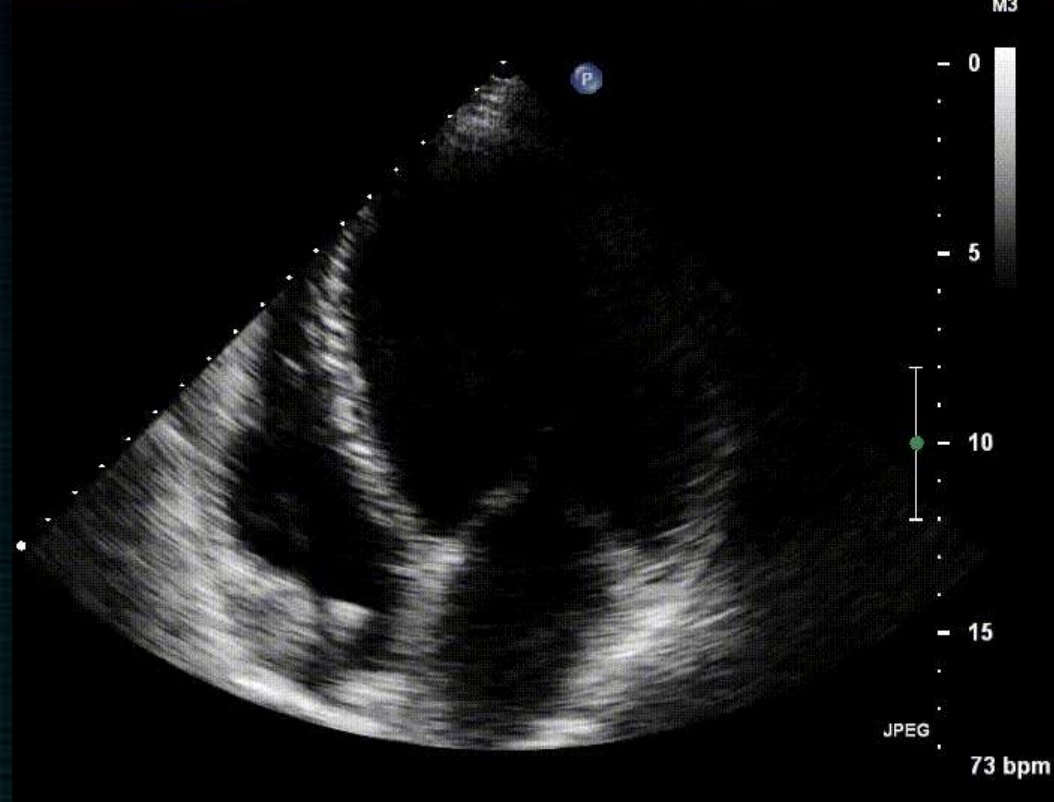
A **cardiomyopathy** is defined as ‘a myocardial disorder in which the **heart muscle is structurally and functionally abnormal, in the **absence** of coronary artery disease (CAD), hypertension, valvular disease, and congenital heart disease (CHD) sufficient to cause the observed myocardial abnormality**

Key aspects in the evaluation and management of cardiomyopathies



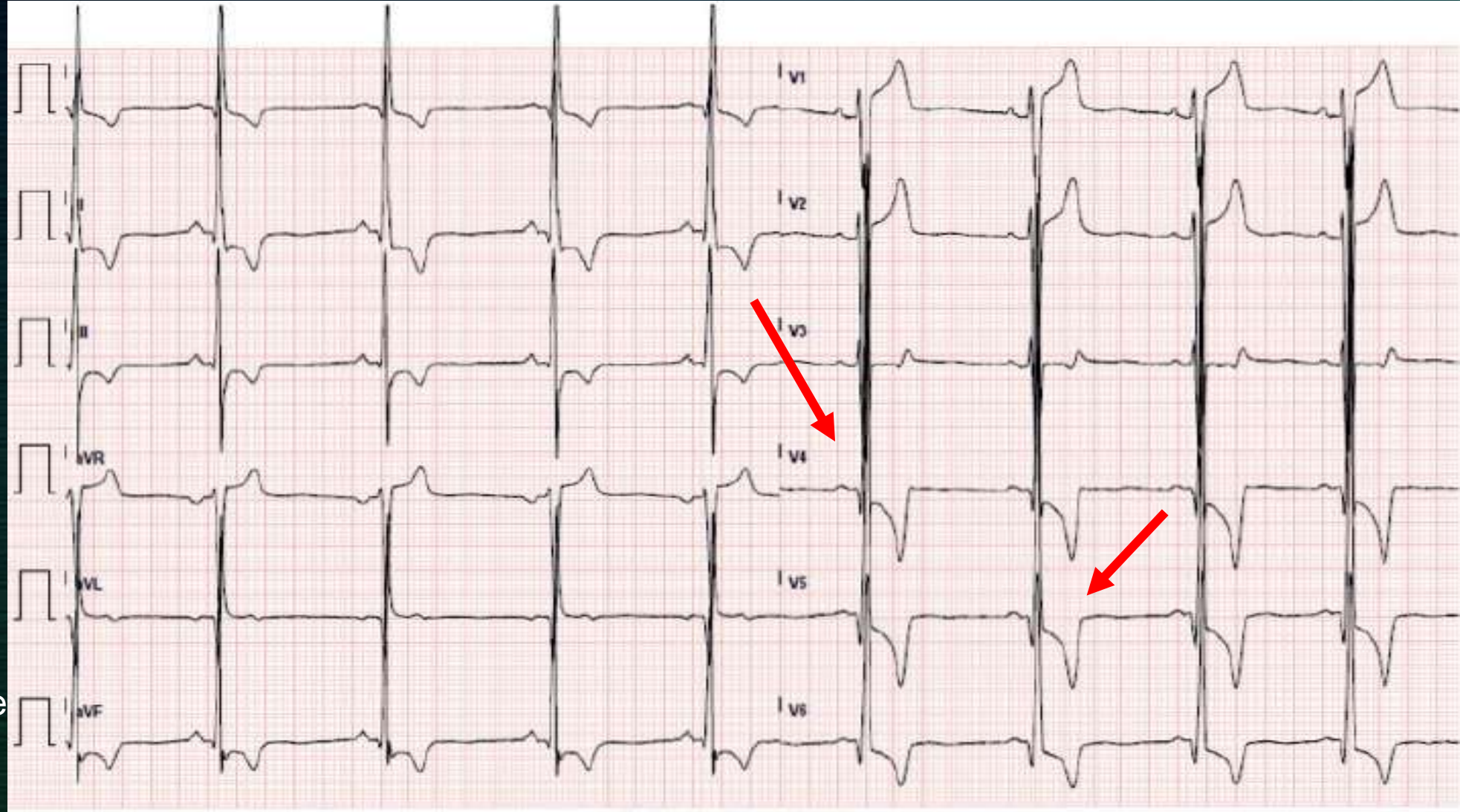
DILATED CARDIOMYOPATHY

Transthoracic Echocardiogram



HYPERTROPHIC CARDIOMYOPATHY

ECG of a young patient with incidental findings of hypertrophic cardiomyopathy, presenting with a syncopal episode.



ECG is characterized by:

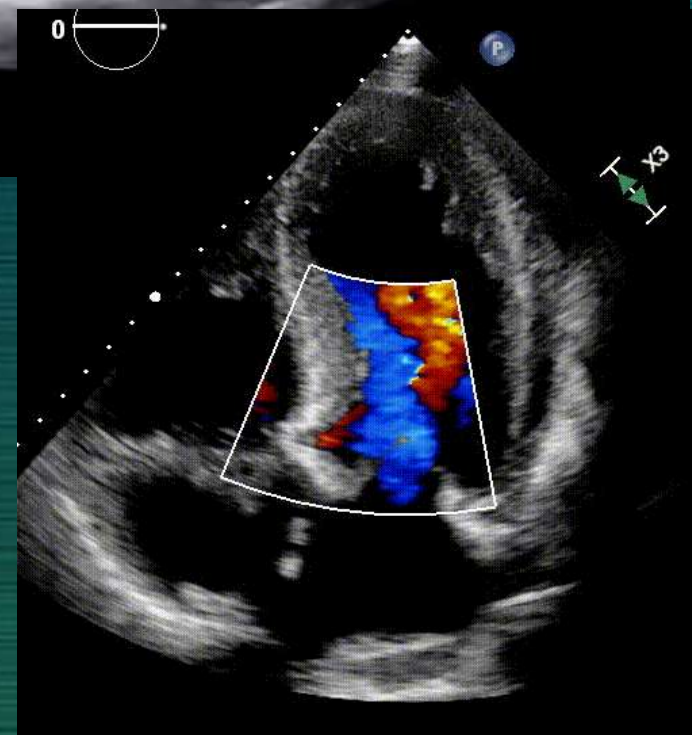
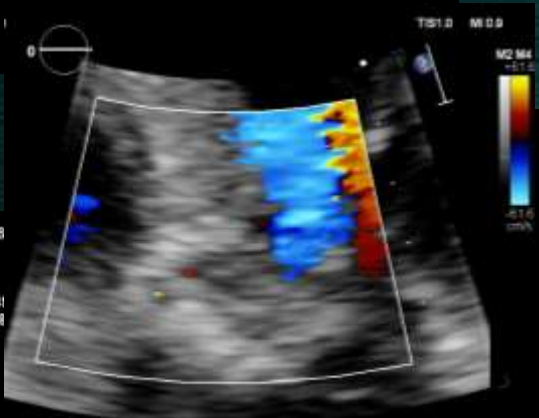
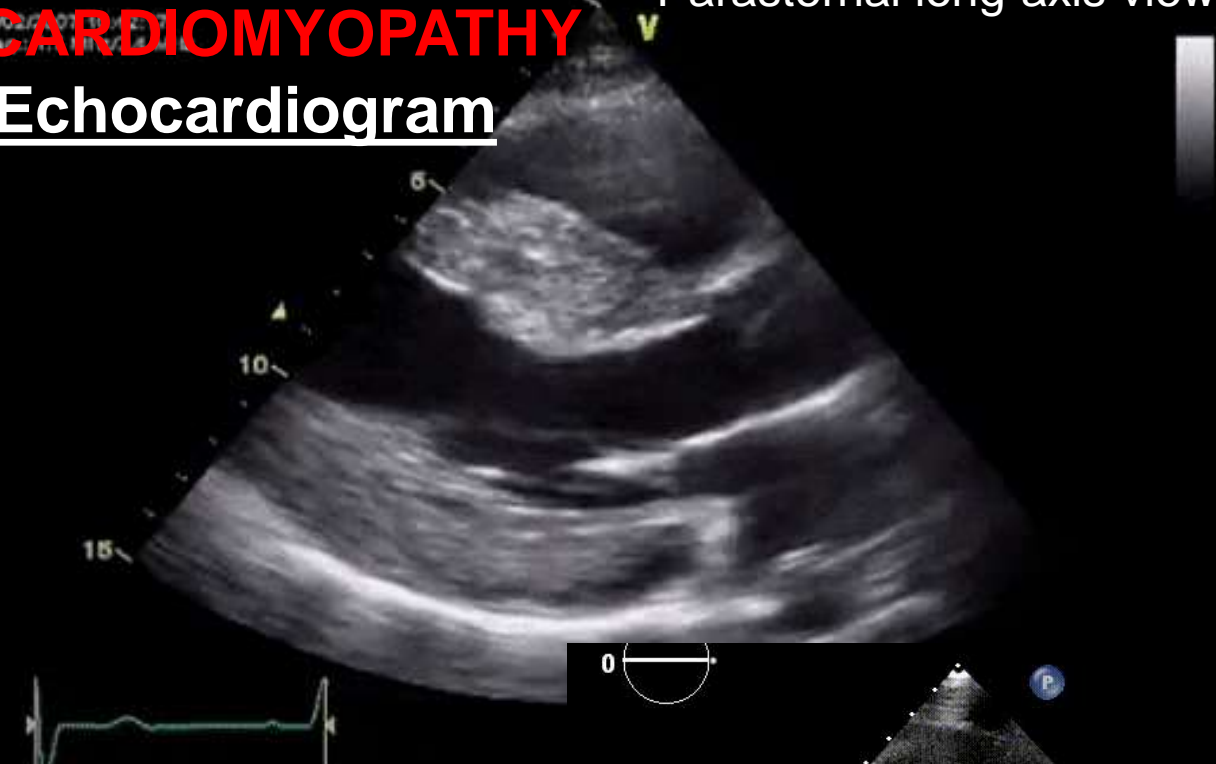
- high QRS voltages, indicating increased ventricular mass,
- pseudonecrosis Q waves,
- and deep negative T waves, particularly in the inferior-lateral leads.

Apical 4-chamber view

Parasternal long-axis view

HYPERTROPHIC CARDIOMYOPATHY

Transthoracic Echocardiogram



ECOCARDIO
X5-1
17Hz
20cm

TD
70%
C 45
P Basic
APen
CF
50%
4000Hz
WF 189Hz
2.5MHz

CW
50%
WF 225Hz
1.8MHz

100mm/s

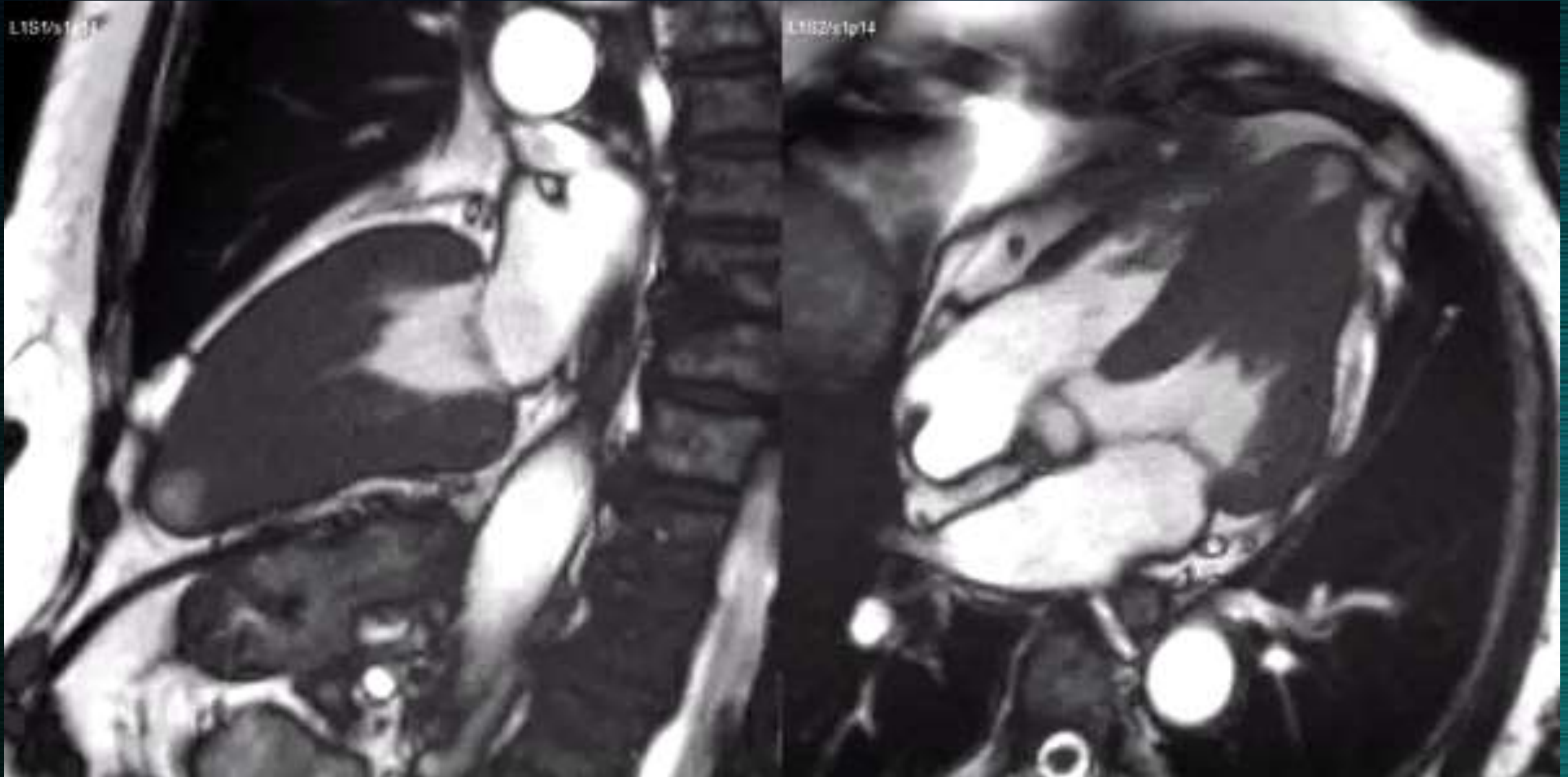
82 bpm

12:45 95 HR

SAM (Systolic anterior motion of the mitral valve)

HYPERTROPHIC CARDIOMYOPATHY

CMR



Restrictive heart diseases

Screening for ATTR amyloidosis in the clinic: overlapping disorders, misdiagnosis, and multiorgan awareness

Restrictive heart diseases

Intrinsic myocyte dysfunction

Genetic

Primary RCM

Variants in sarcomeric, cytoskeletal, nuclear envelope, filamin, titin genes

Storage

Desmin

AFD

Danon

Glycogenoses

PRKAG2 variants

Iron overload/storage disorders

Non-genetic

Drugs (e.g. chloroquine)

Endomyocardial disorders

Endomyocardial fibrosis

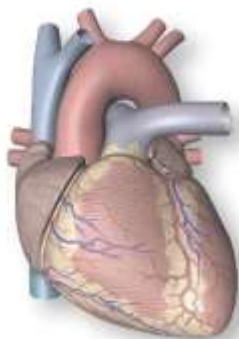
Hypereosinophilia

Carcinoid

Endocardial fibroelastosis

Endocardial neoplasms

Iatrogenic/drug toxicity



Myocardial extracellular matrix disorders

Infiltrative

Hyperoxaluria

Amyloidosis

Sarcoidosis

Fibrosis

Radiation

Chemotherapy

Systemic sclerosis

Inflammatory/granulomatous

Diabetic heart disease

Cardiac

Heart failure



Atrial fibrillation



Bradyarrhythmias/
conduction abnormalities/
pacemakers



Musculoskeletal

Carpal tunnel syndrome



Back pain/lumbar spinal stenosis



Ruptured distal biceps tendon/
Popeye sign



Shoulder, knee and hip pain or surgery

Trigger finger



Polyneuropathy

Painful neuropathy in hands and feet



Muscle weakness, difficulty walking, and falls



Autonomic Dysfunction

Orthostatic hypotension/
intolerance to blood pressure meds



Chronic diarrhea/
constipation/weight loss



Erectile dysfunction



RCM



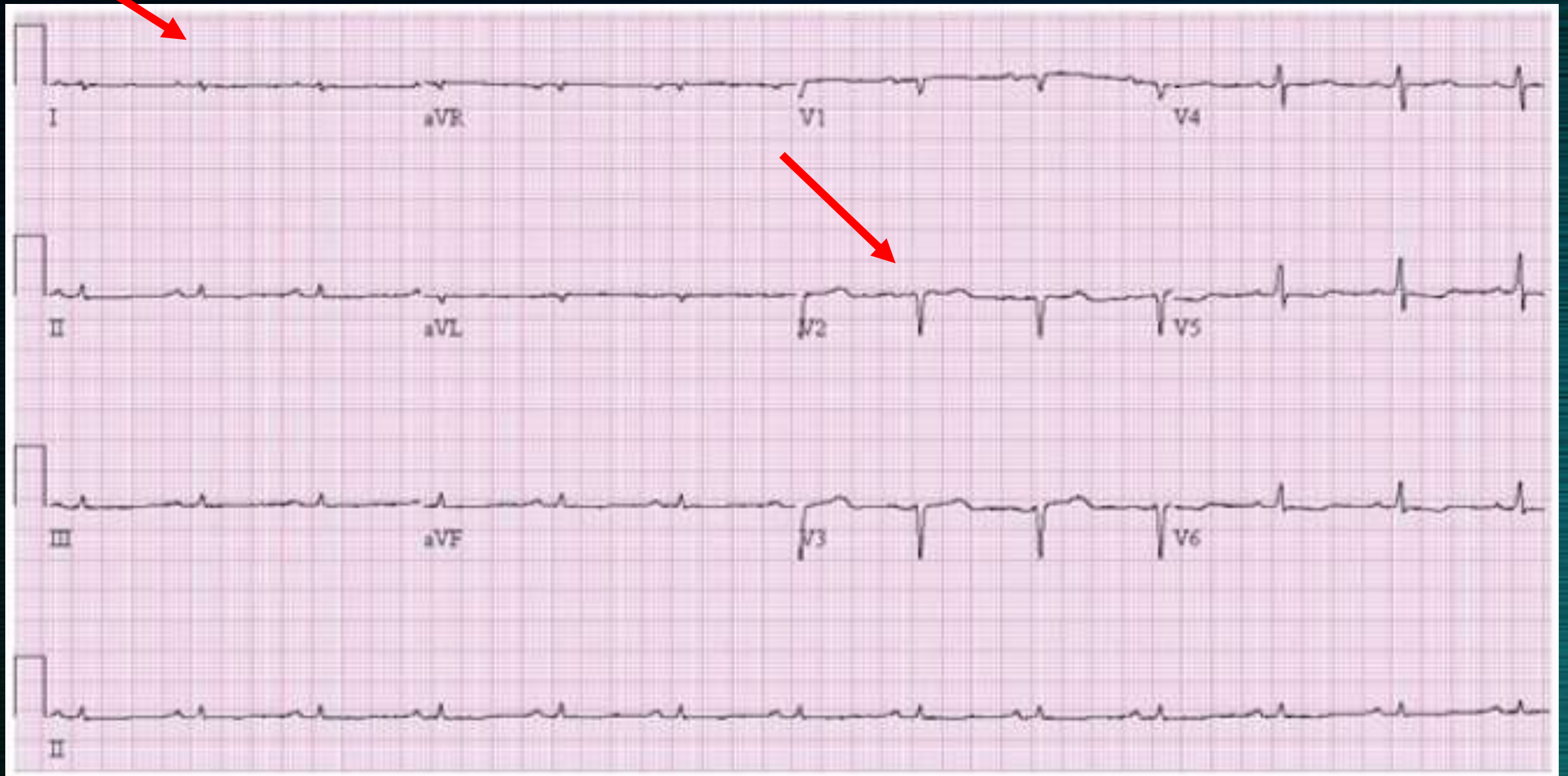
Myocardial diseases with occasional restrictive physiology, often in the context of LVH

2023 ESC Guidelines for the management of cardiomyopathies (European Heart Journal; 2023 – doi:10.1093/eurheartj/ehad194)



Heart Failure Reviews (2022) 27:785–793
<https://doi.org/10.1007/s10741-021-10080-2>

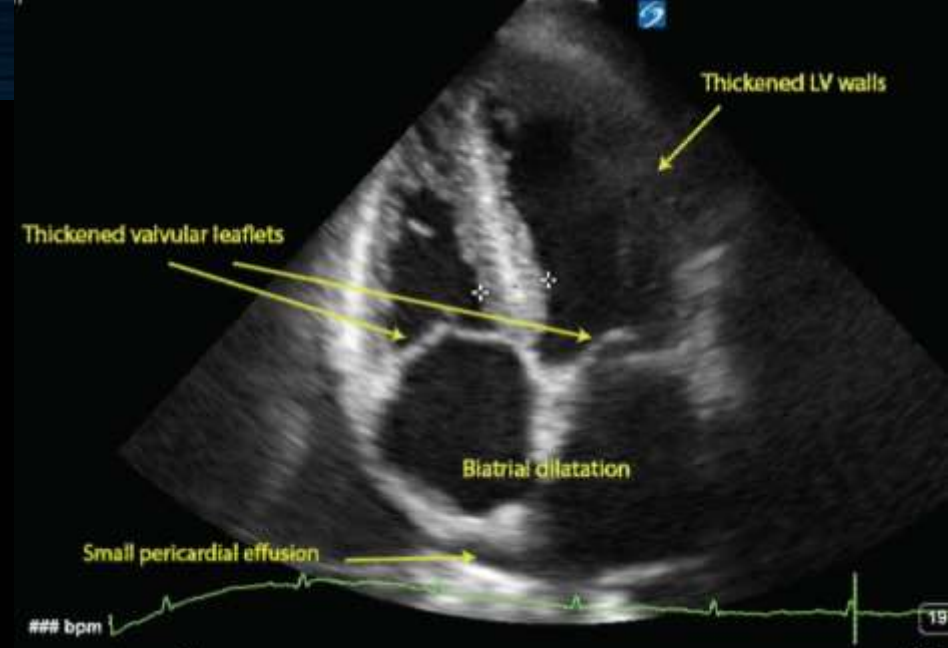
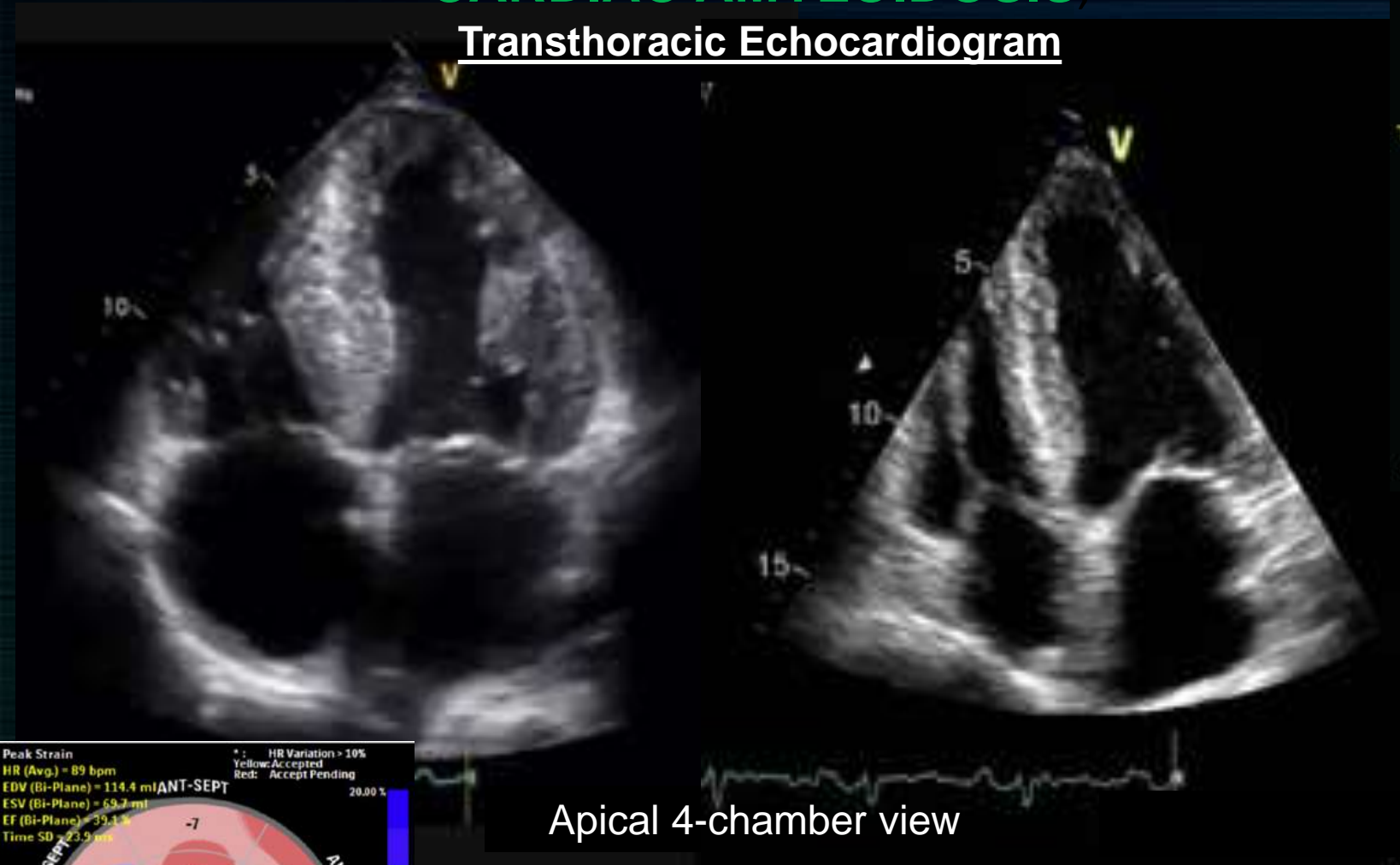
CARDIAC AMYLOIDOSIS, ECG



Typical electrocardiogram (ECG) in a patient with cardiac amyloidosis. Low QRS voltages in peripheral leads, Pathologic Q waves.

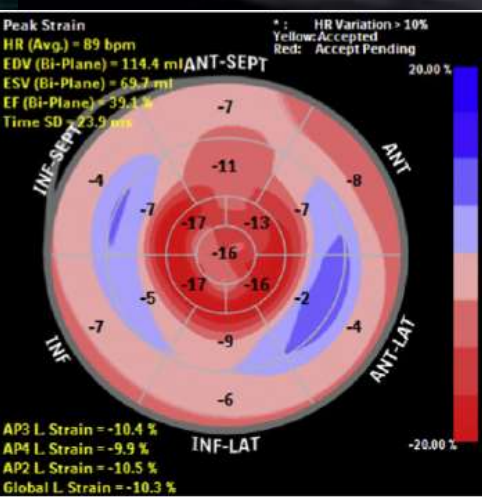
CARDIAC AMYLOIDOSIS

Transthoracic Echocardiogram



Apical 4-chamber view

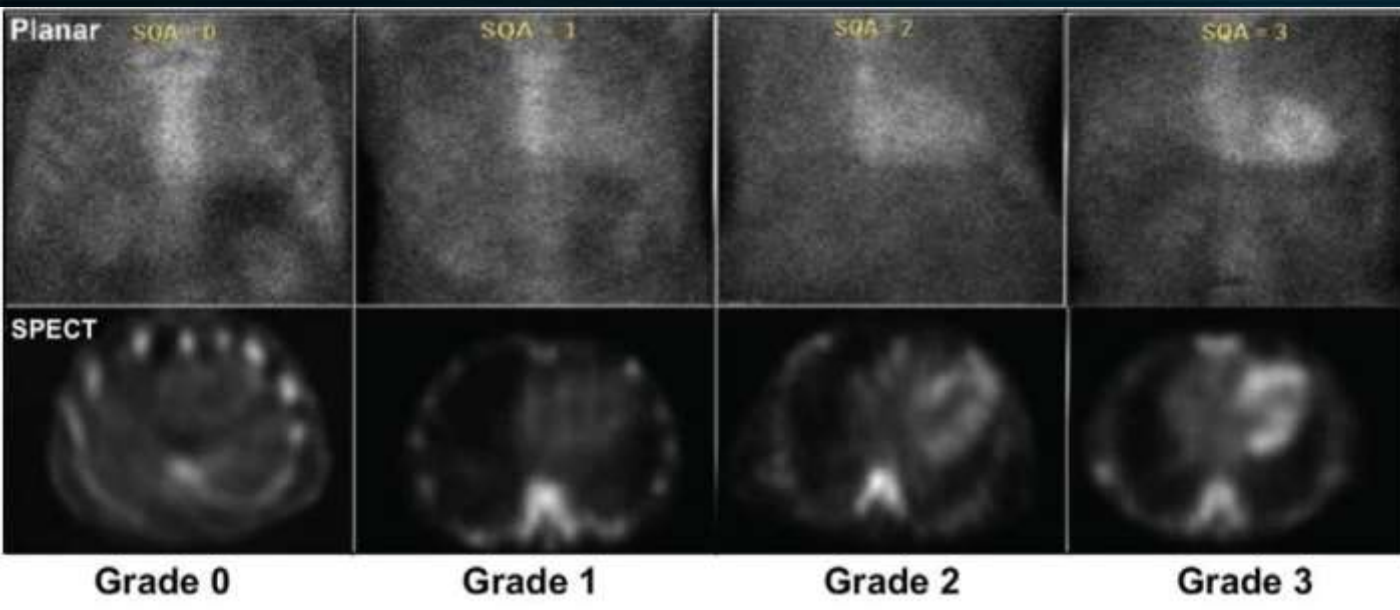
Subcostal view



Longitudinal **strain** presents with a typical pattern that distinguishes it from other cardiomyopathies: a severe reduction in deformation values at the base of the left ventricle with apical sparing (>2:1 apical/basal ratio or “cherry on top”) pattern.

CARDIAC AMYLOIDOSIS, bone scintigraphy

Bone tracers, in particular, ^{99m}Tc -3,3-diphosphono-1,2-propanodicarboxylic acid (^{99m}Tc -DPD) has been documented to have the ability to bind to TTR deposits but not to AL deposits. Therefore, the method allows, within certain limits, the differential diagnosis between the two etiological forms



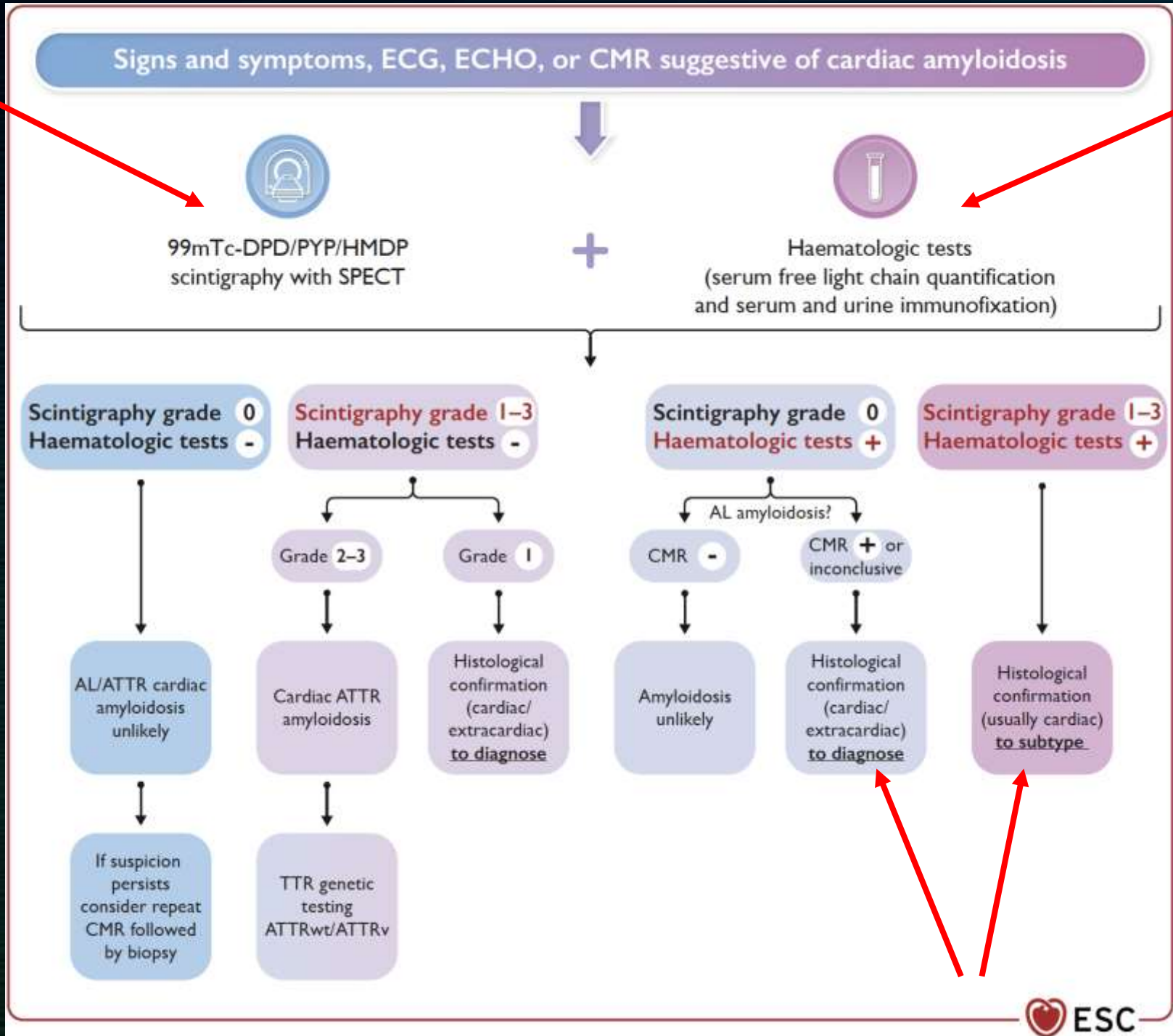
^{99m}Tc imaging procedures for cardiac amyloidosis. SPECT imaging to identify myocardial retention of technetium-based isotopes

Perugini grading scale:

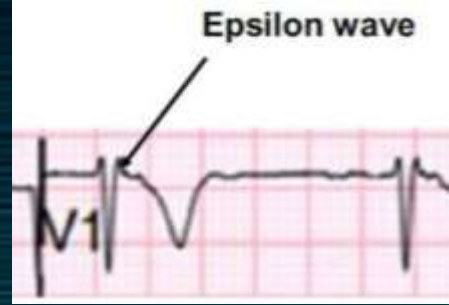
- Strong uptake (2-3),
- weak uptake (1), a
- bsent uptake (0))



Diagnosis of cardiac amyloidosis



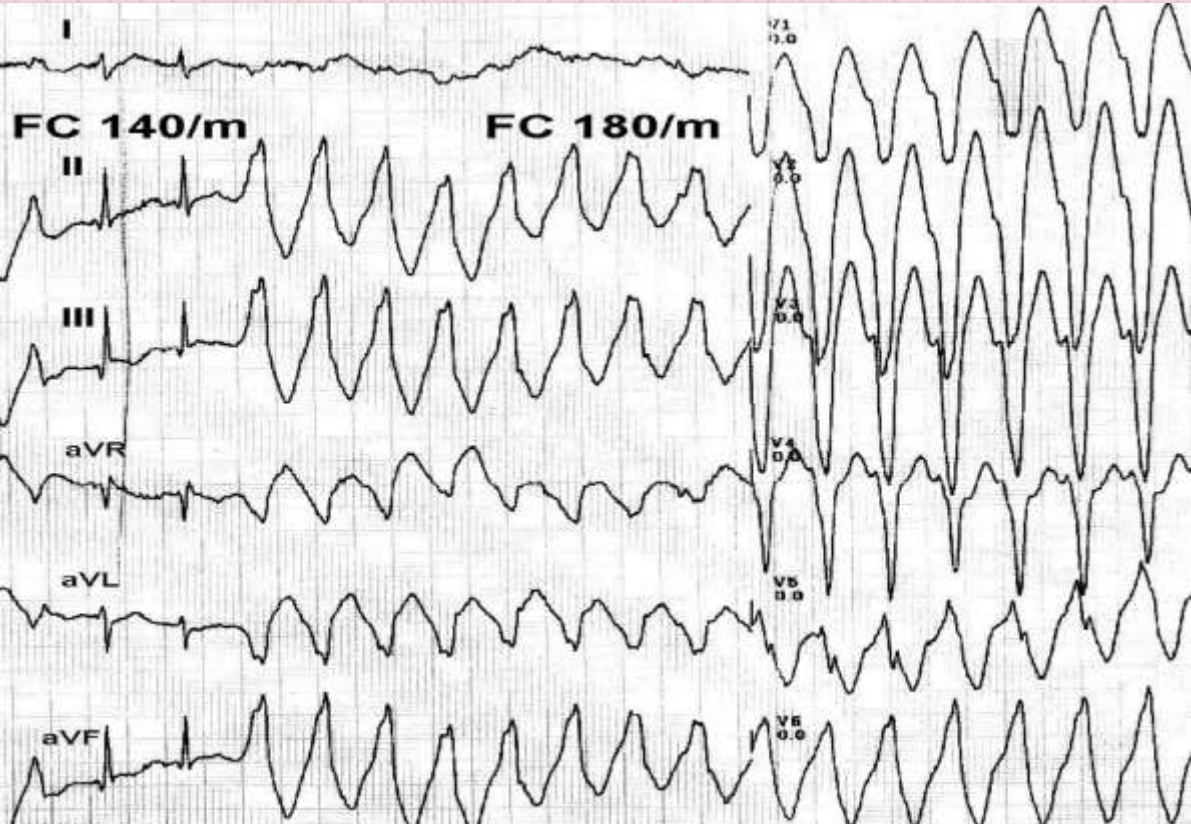
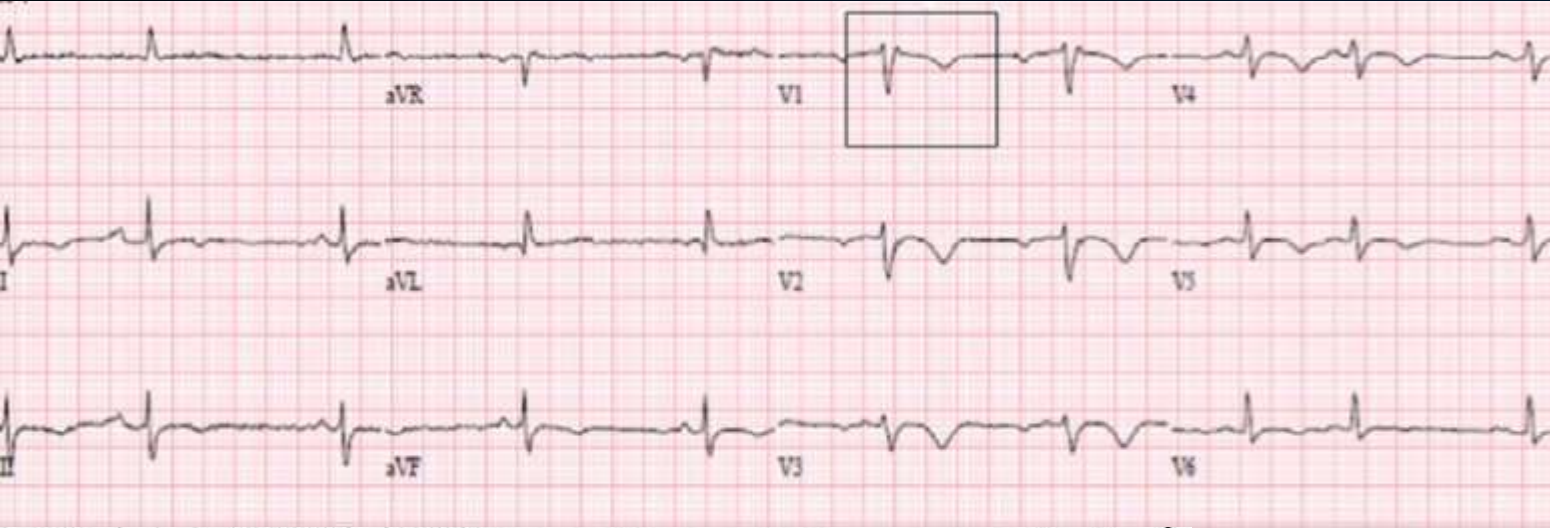
ARRHYTHMOGENIC CARDIOMYOPATHY, ECG



ECG in ARVC patient (PKP2 mutation).
Inversion of T-waves in V1-V6 leads and epsilon wave in V1.

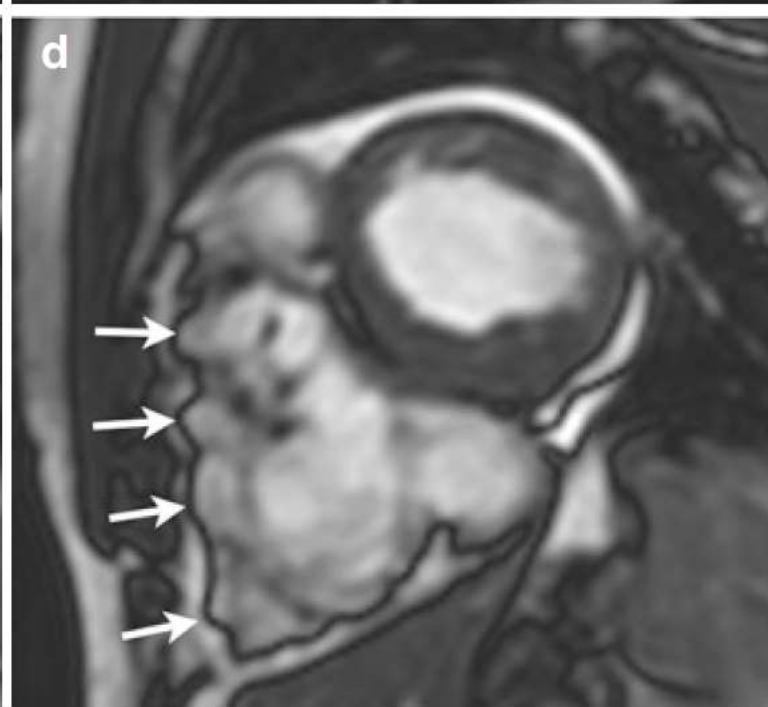
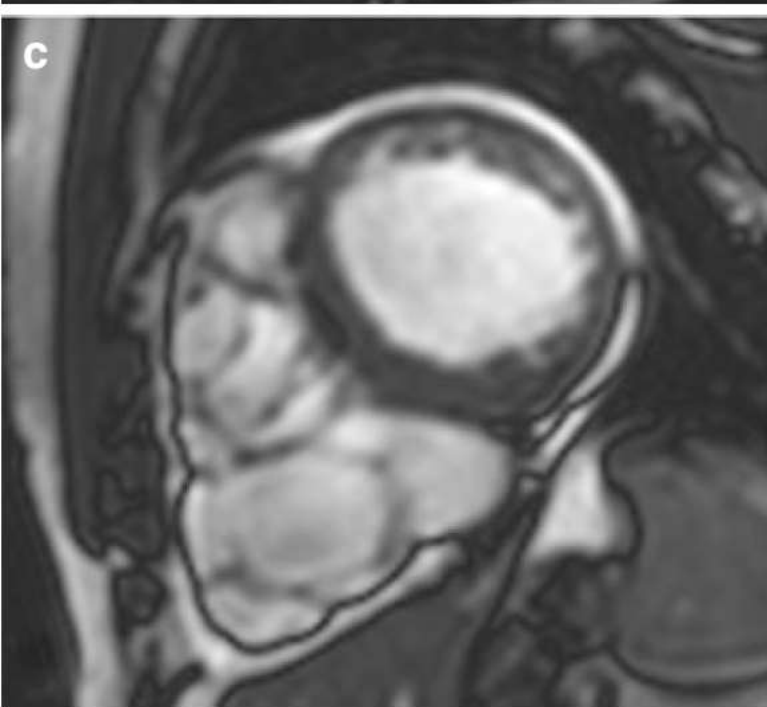
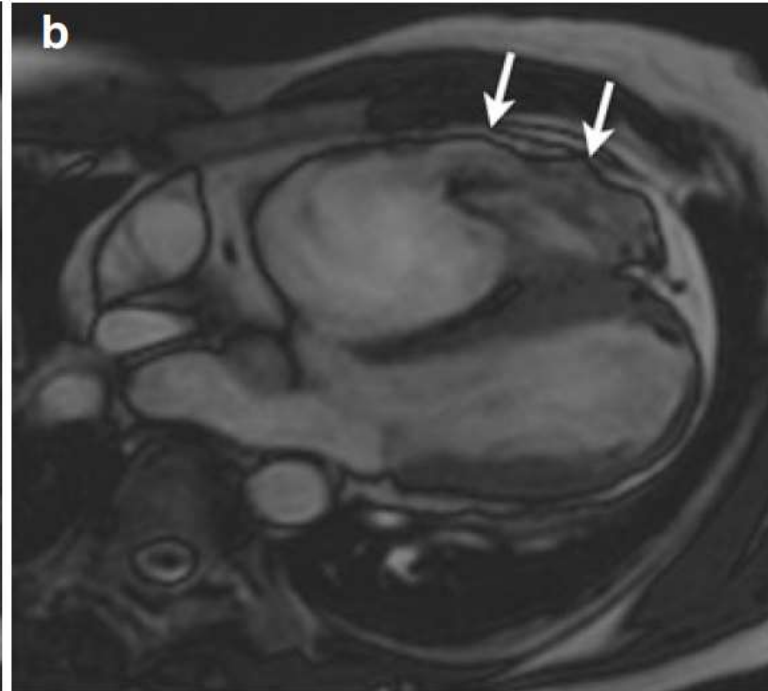
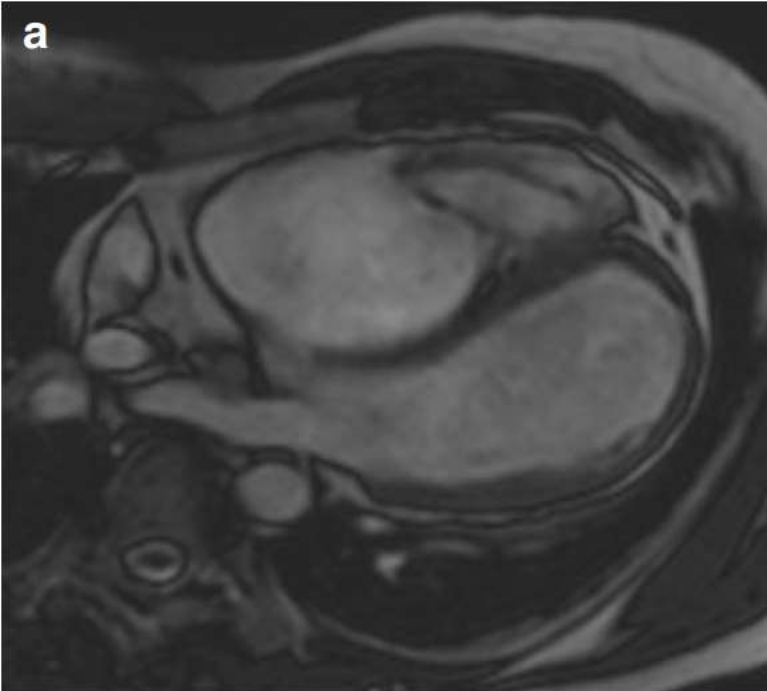
nadir-to-end of S-wave interval >55 msec

The echocardiogram in arrhythmogenic right ventricular cardiomyopathy (ARVC).



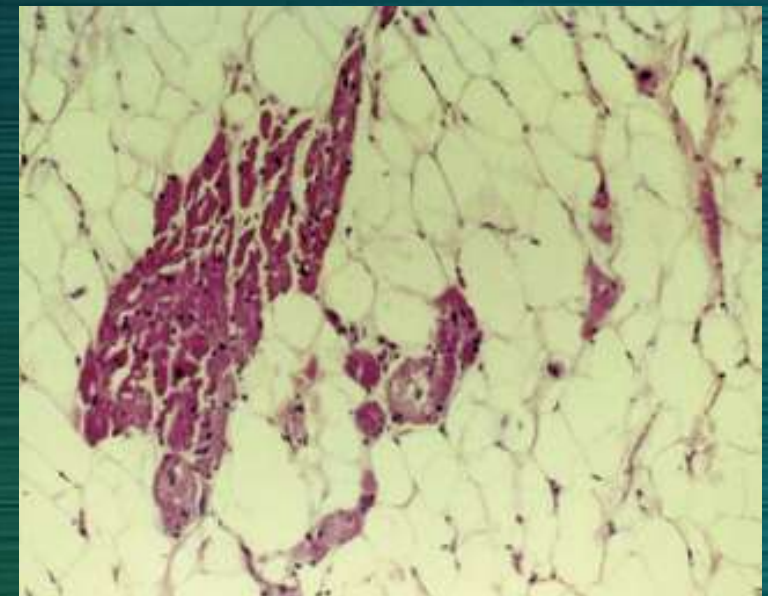
ECG during exercise-induced ventricular tachycardia (VT) in a patient with arrhythmogenic right ventricular cardiomyopathy (ARVC). Morphology of VT resembling a left bundle branch block (LBBB) pattern with a lower axis, delayed transition, wide QRS, and the presence of notches in the QRS complexes.



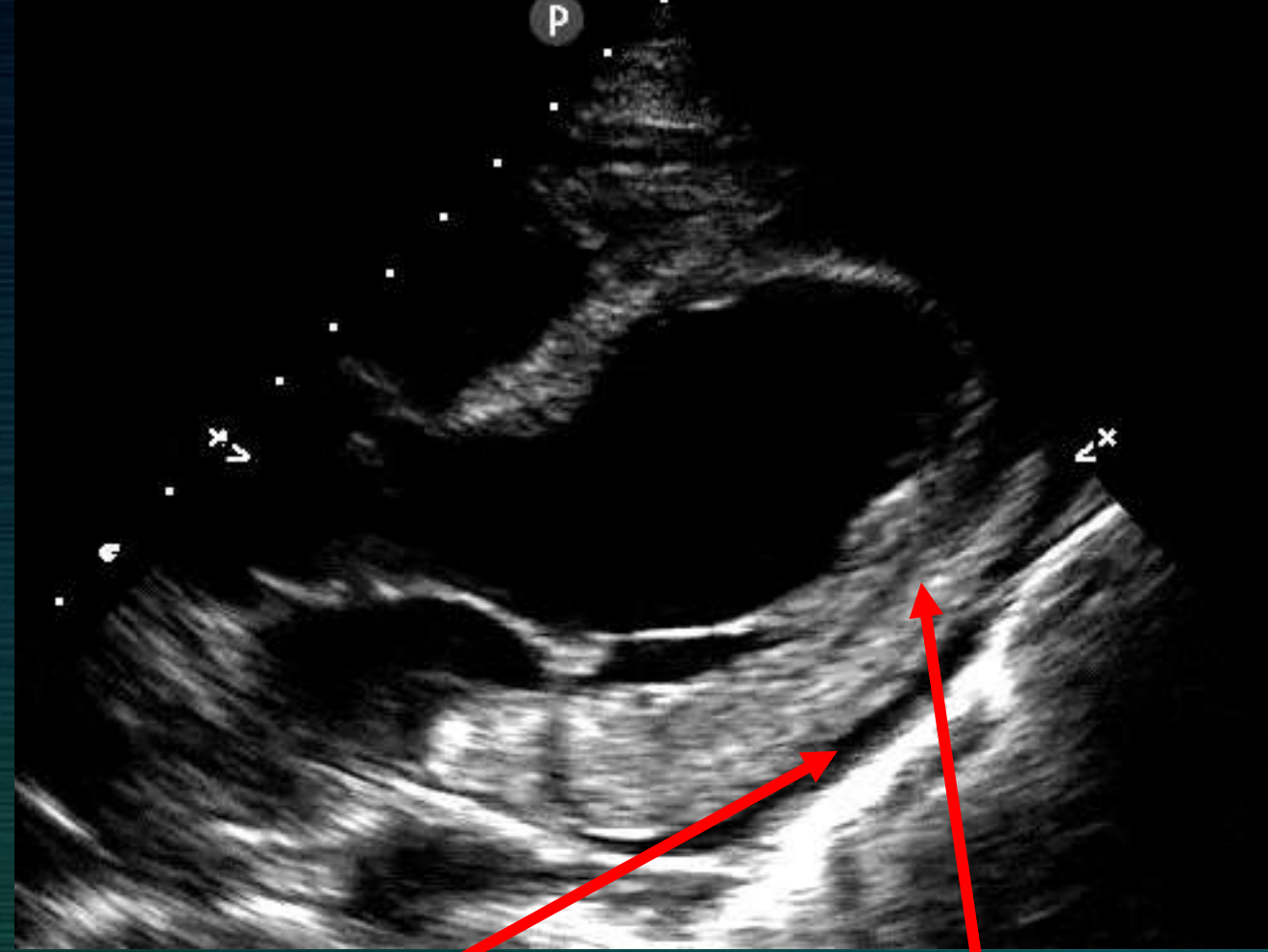
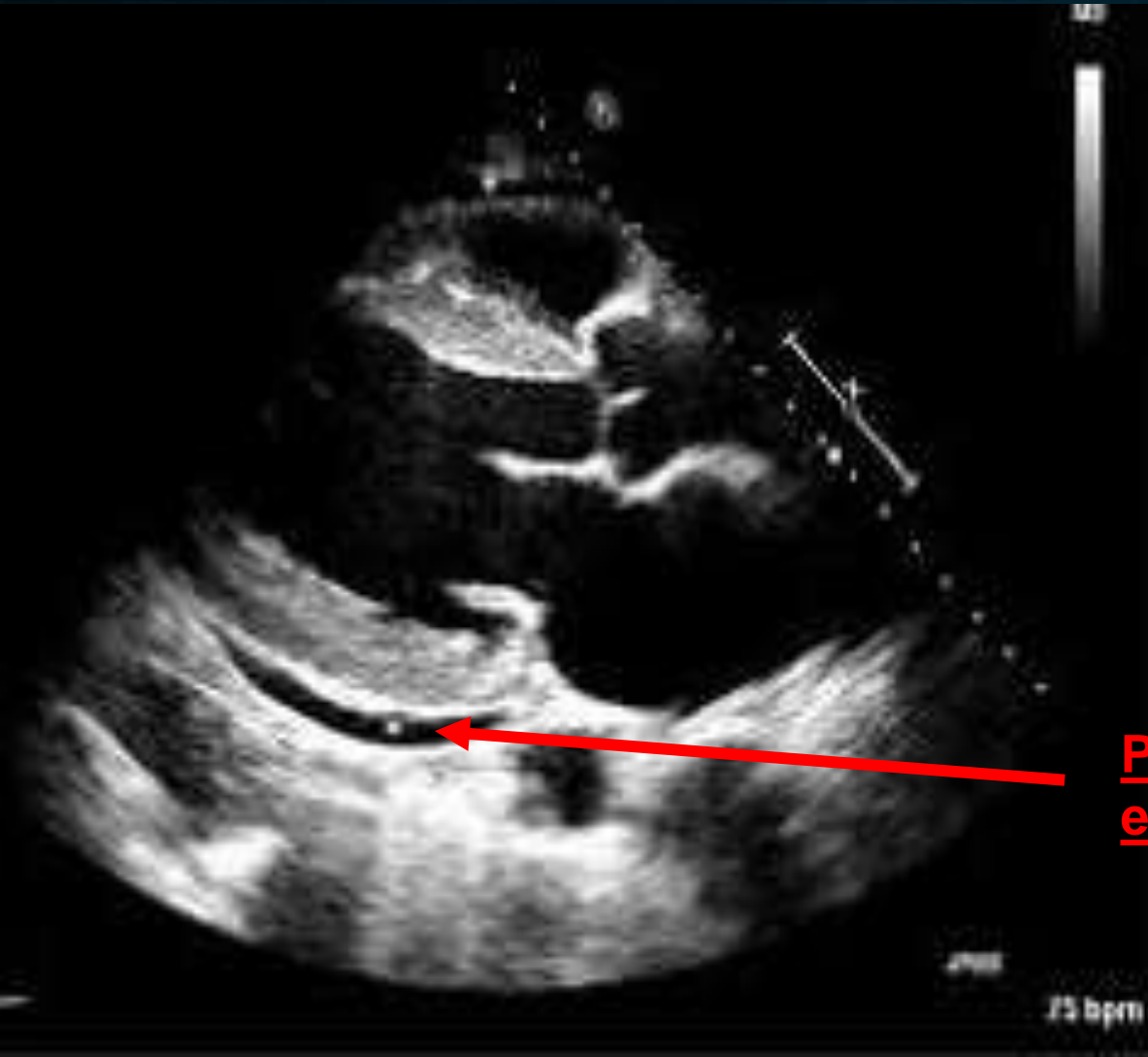


ARRHYTHMOGENIC CARDIOMYOPATHY, CMR

Biventricular wall motion abnormalities (WMA) in a patient with arrhythmogenic right ventricular cardiomyopathy (ARVC) and left ventricular (LV) involvement seen at steady-state free precession (SSFP) imaging. End-diastole (a , c), end-systole (b , d). Four-chamber off-axis (a , b), and short-axis (c , d) slices. Note RV end-systolic bulging areas in b and d (arrows). Apical LV hypokinesia is also present



MYOCARDITIS and
MYOPERICARDITIS DIAGNOSIS:
Troponin and Echocardiogram

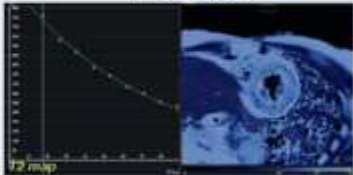

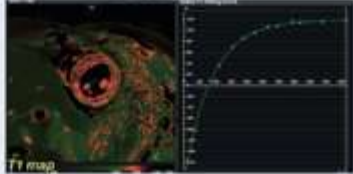
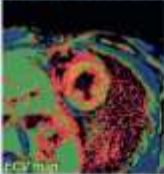

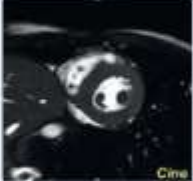



Pericardial effusion

Depression of the kinetics of the left ventricle

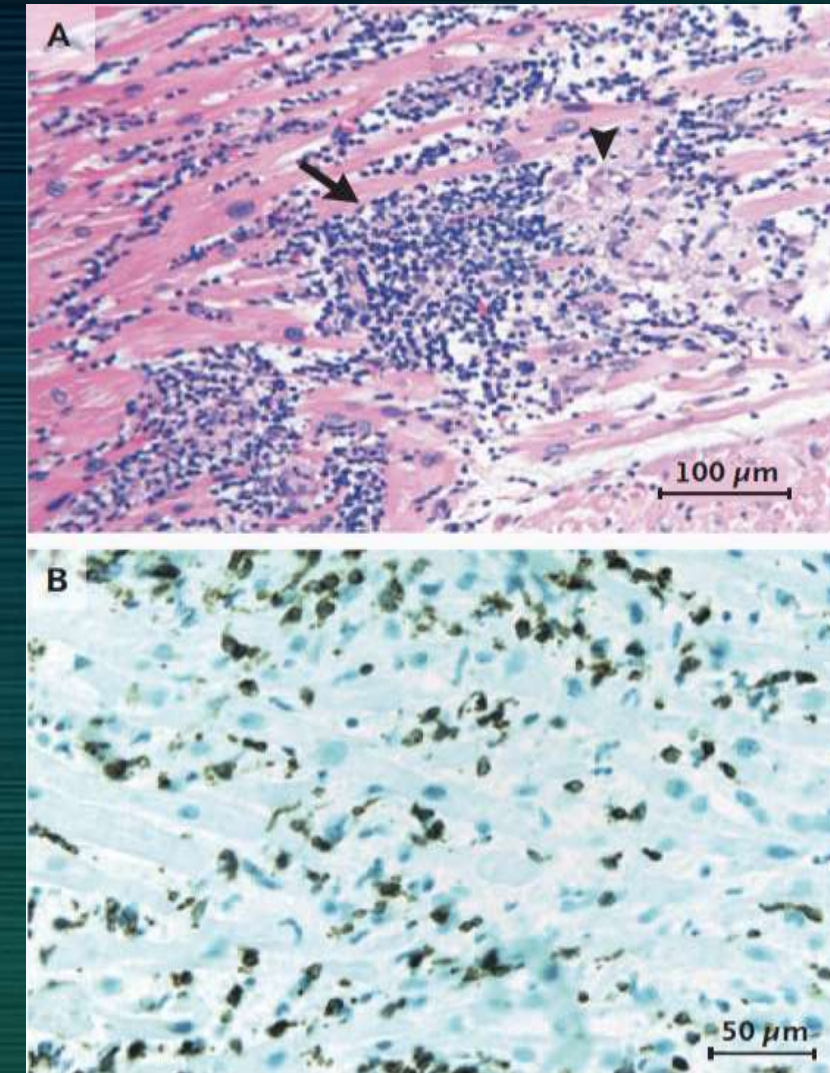
Cardiac Magnetic Resonance Imaging (CMR)

Histology:

	2018 Lake Louise Criteria	CMR Image Examples
Main Criteria	Myocardial Edema (T2-mapping or T2W images)	Regional or global increase of native T2  or Regional or global increase of T2 signal intensity 
	Non-ischemic Myocardial Injury (Abnormal T1, ECV, or LGE)	Regional or global increase of native T1  or Regional or global increase of ECV  or Regional LGE signal increase 
Supportive Criteria	Pericarditis (Effusion in cine images or abnormal LGE, T2, or T1)	Pericardial effusion 
	Systolic LV Dysfunction (Regional or global wall motion abnormality)	Regional or global hypokinesis 

Ferreira, V.M. et al. J Am Coll Cardiol. 2018;72(24):3158-76.

ECV . extracellular volume; LGE . late gadolinium enhancement; T2W . T2-weighted.



Lymphocytic and Histiocytic Infiltrate and T Lymphocytes in Heart-Tissue.
 Sections from Patients with Acute Myocarditis.
 Panel A shows acute myocarditis with widespread lymphocytic and histiocytic infiltrate (arrow) and associated myocyte damage (arrowhead) (hematoxylin and eosin).
 Panel B shows CD3 immunostaining of T lymphocytes in a patient with acute myocarditis

The definitions for PH are based on haemodynamic assessment by [right heart catheterization \(RHC\)](#).

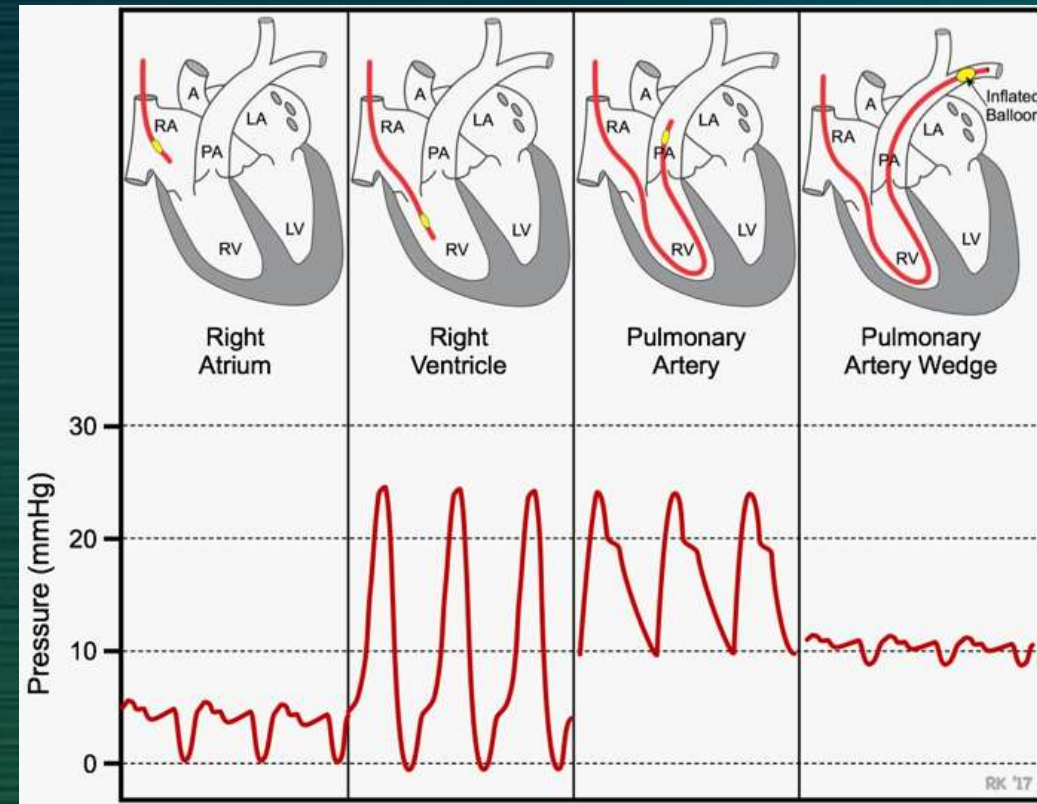
Although haemodynamics represent the central element of characterizing PH, the final diagnosis and classification should reflect the whole clinical context and consider the results of all investigations.

[Pulmonary hypertension](#) :
a **mean pulmonary arterial pressure (mPAP) > 20 mmHg** at rest

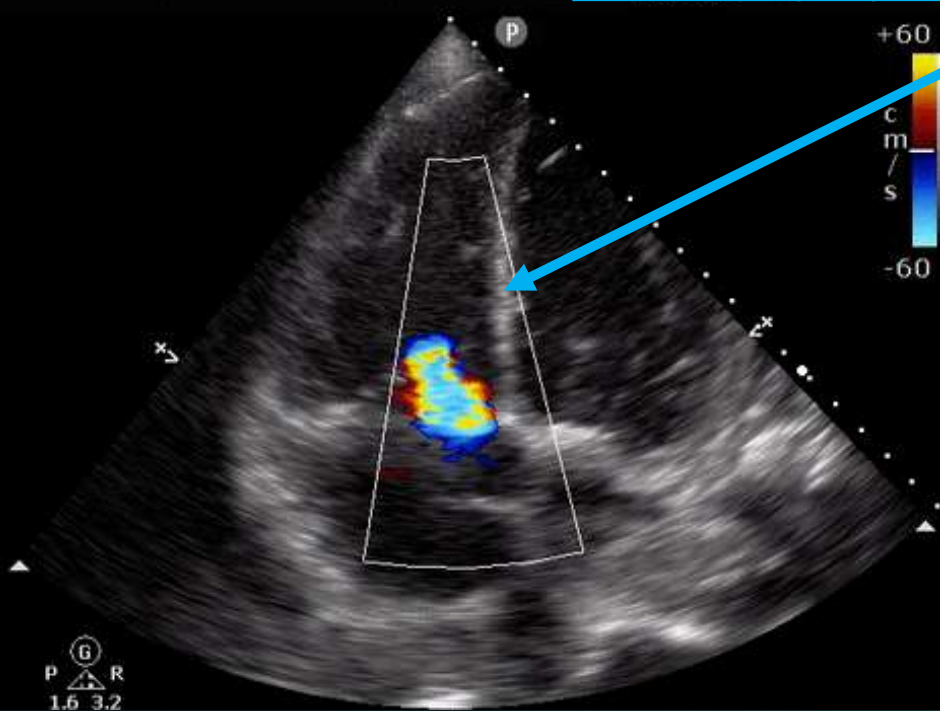
It is essential to include

- [Pulmonary Vascular Resistance \(PVR\)](#)
- and [Pulmonary Arterial Wedge Pressure \(PAWP\)](#)

in the definition of pre-capillary PH

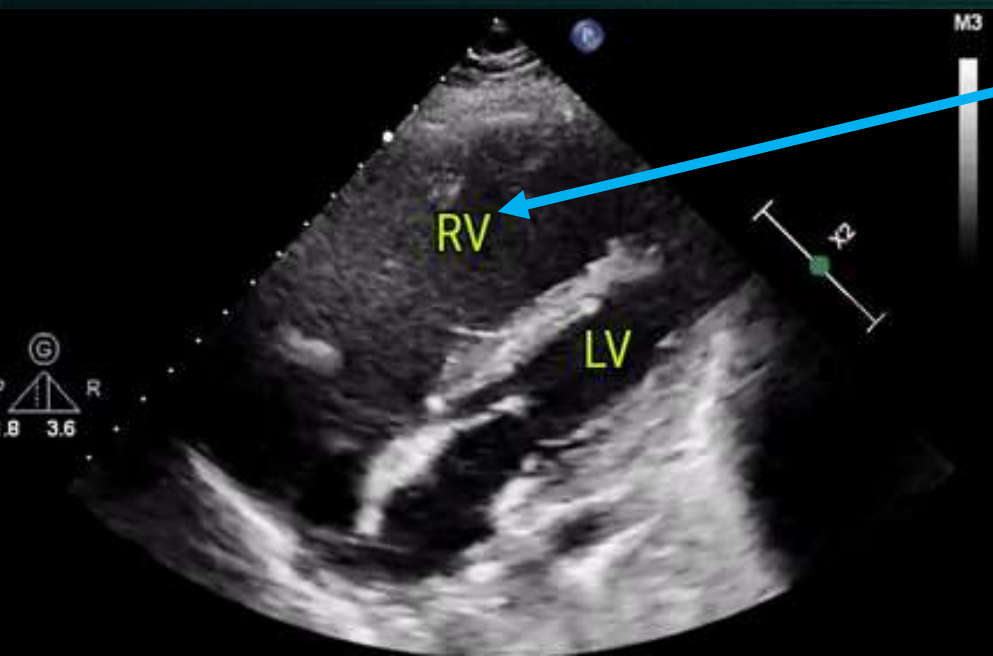
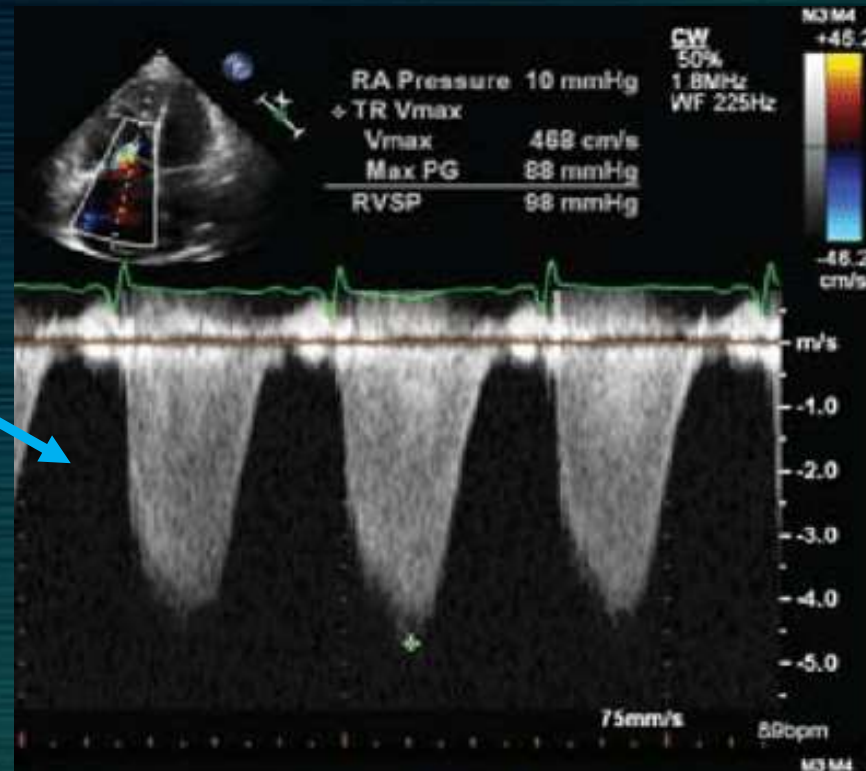


Pulmonary Hypertension: TransThoracic Echocardiography



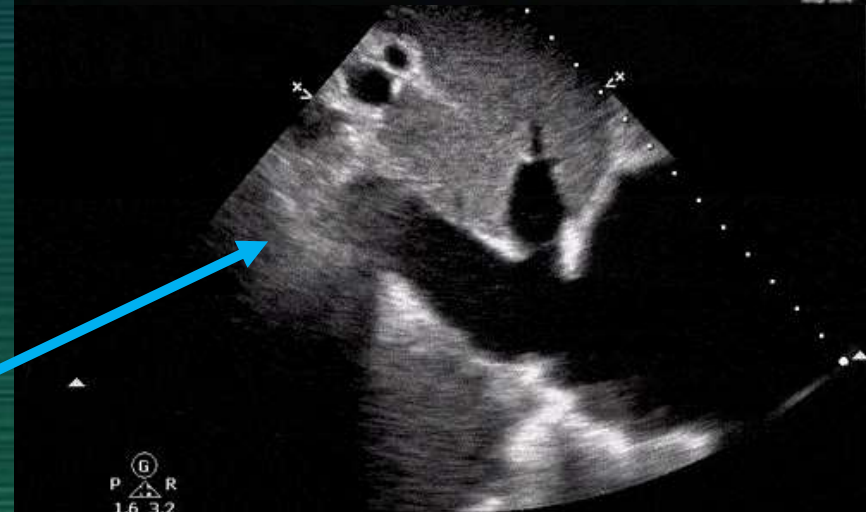
Tricuspid regurgitation seen with color doppler flow

Increase systolic peak tricuspid regurgitation velocity (peak TRV), measured with continuous wave Doppler

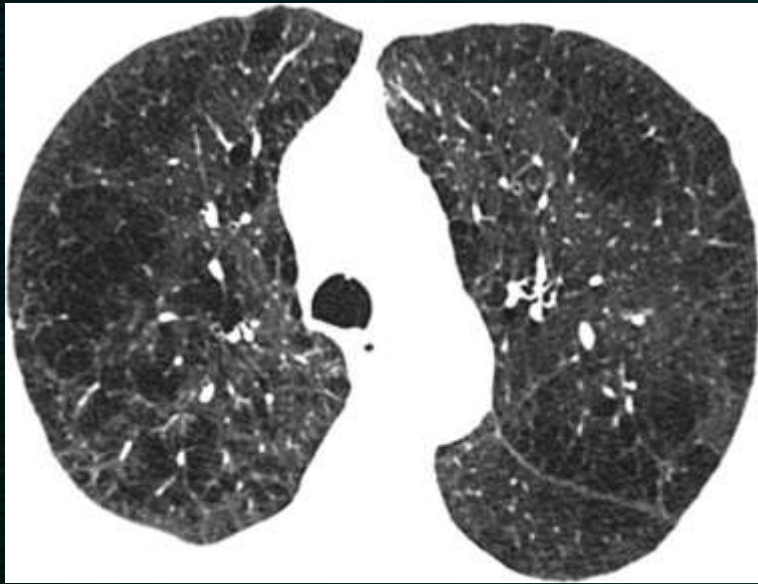
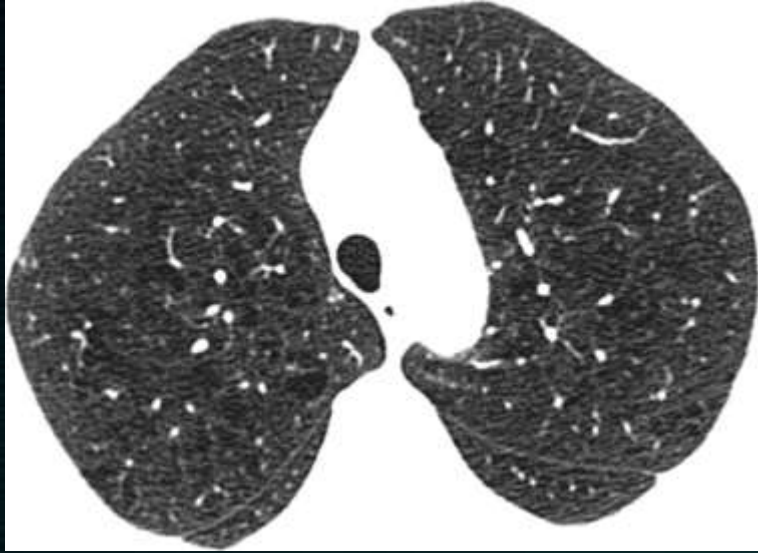


A case of severely dilated right ventricle (RV size more than that of LV) in a patient with severe pulmonary hypertension.

Subcostal view: distended inferior vena cava with diminishing inspiratory collapsibility

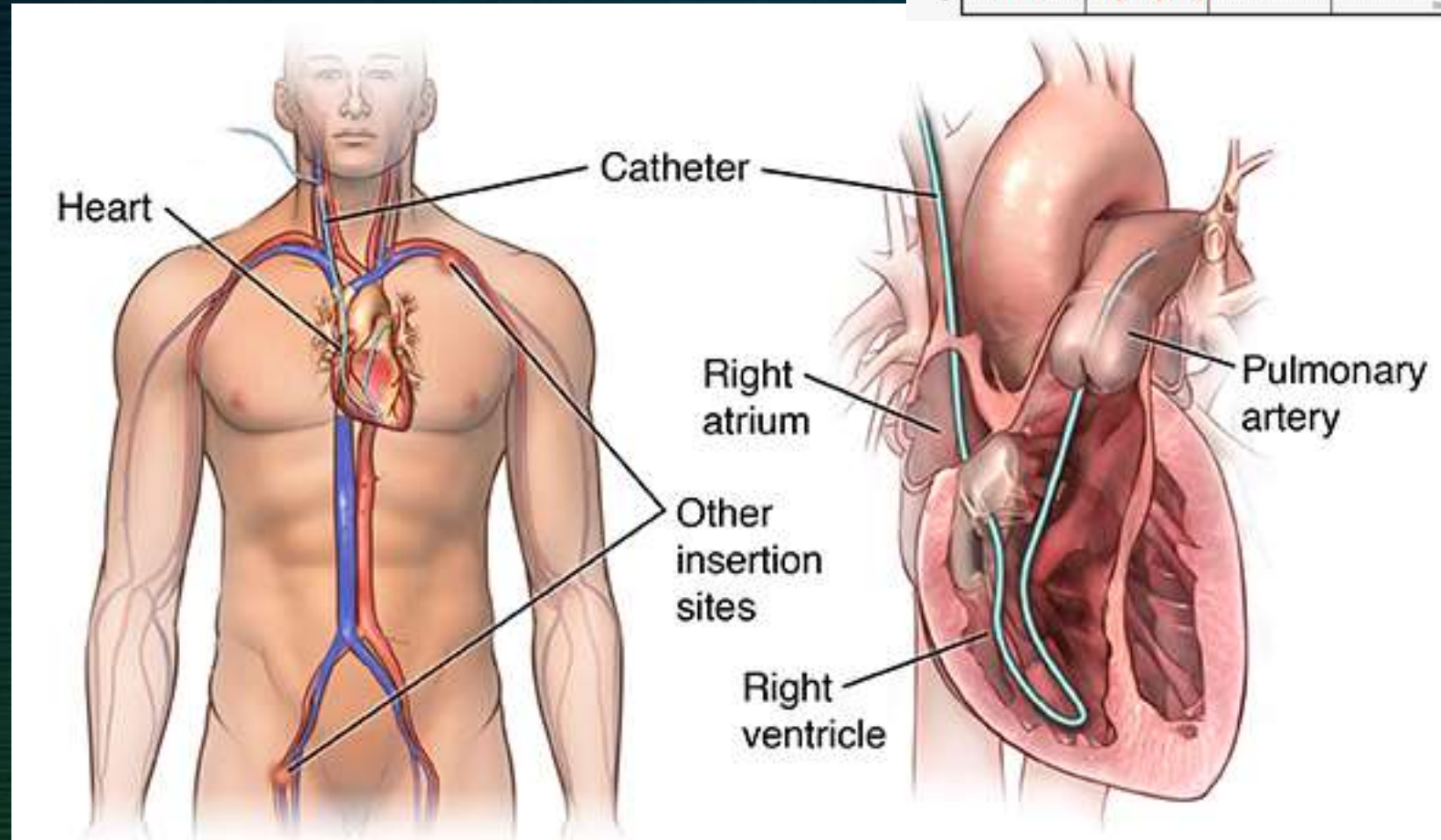
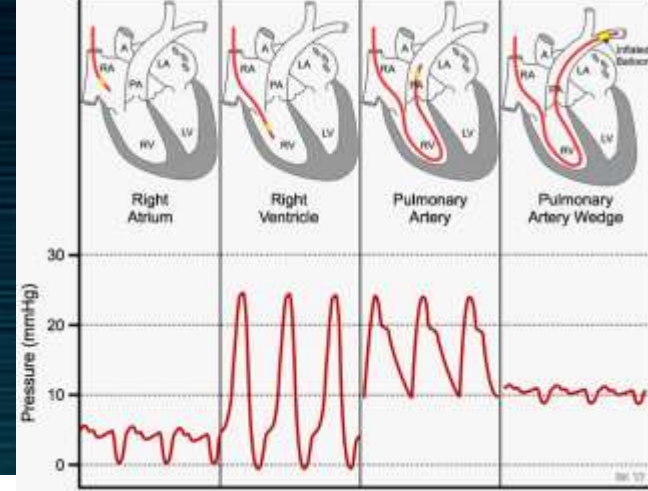


DIAGNOSIS of PH.
**PULMONARY FUNCTION AND
LUNG IMAGING**



Figures: Axial CT images show severity grades of parenchymal emphysema

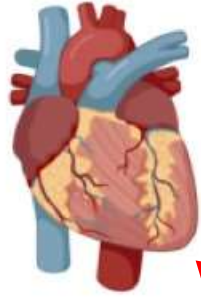
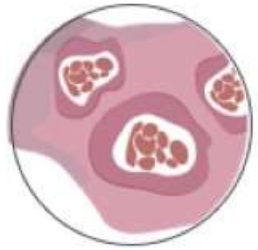
DIAGNOSIS : **INVASIVE
CARDIOPULMONARY HEMODYNAMICS:
Right Heart Catheterization (RHC).**



2022 ESC/ERS Guidelines for the Diagnosis and Treatment of PH

Central Illustration

Clinical Classification

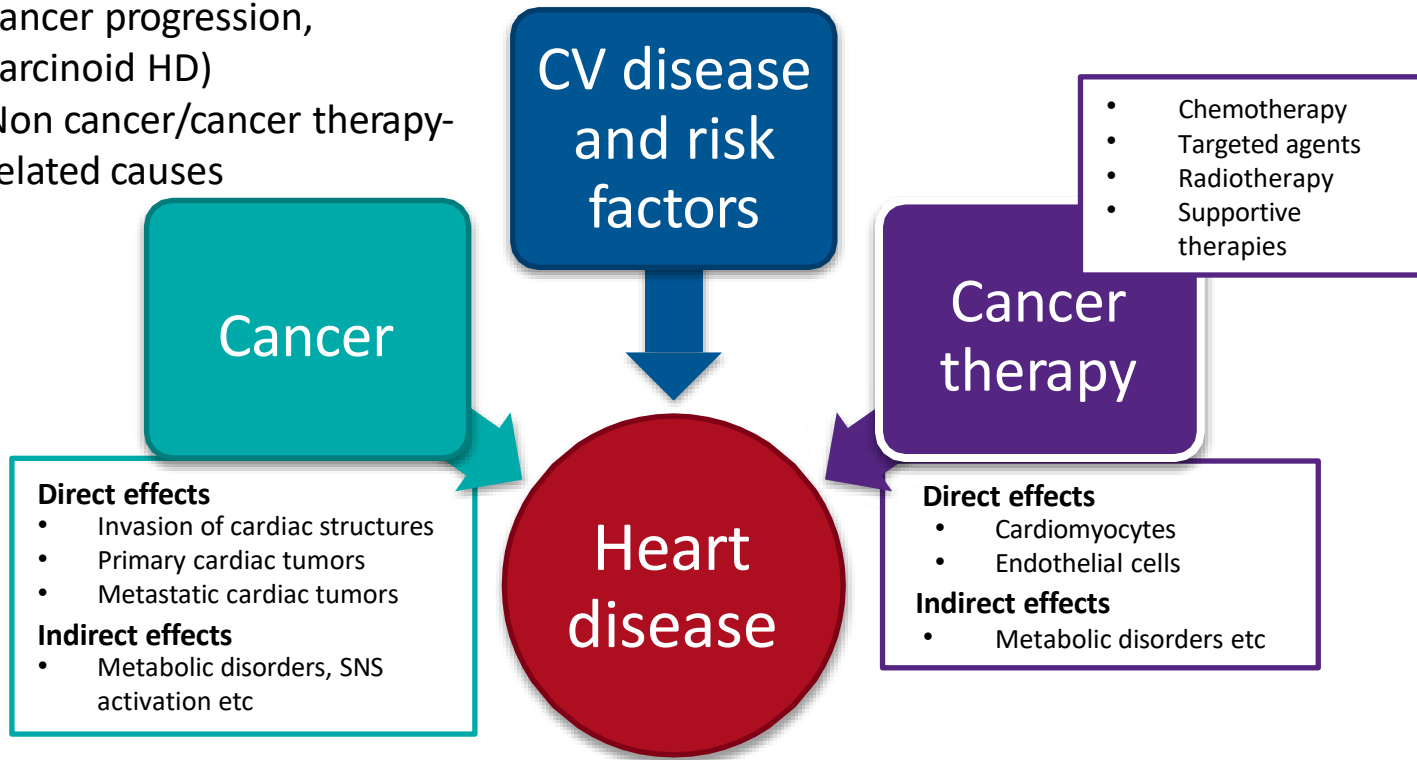


Pulmonary arterial hypertension (PAH) 1	PH associated with left heart disease 2	PH associated with lung disease 3	PH associated with pulmonary artery obstructions 4	PH with unclear and/or multifactorial mechanisms 5
<ul style="list-style-type: none"> Idiopathic/ heritable Associated conditions 	<ul style="list-style-type: none"> IpcPH CpcPH 	<ul style="list-style-type: none"> Non-severe PH Severe PH 	<ul style="list-style-type: none"> CTEPH Other pulmonary obstructions 	<ul style="list-style-type: none"> Hematologic disorders Systemic disorders
<p>Rare</p>	<p>Very common</p>	<p>Common</p>	<p>Rare</p>	<p>Rare</p>

Causes of acute/sub-acute CVD in active cancer

Differential diagnosis

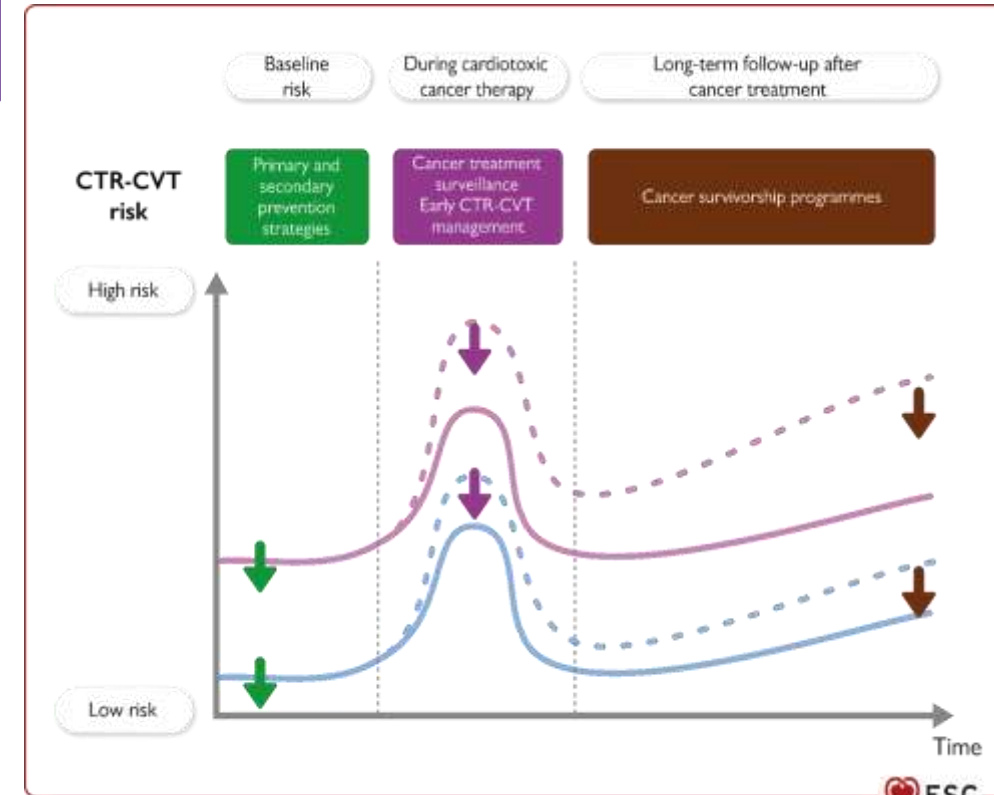
- Cardiotoxicity
- Cancer (eg. cardiac tumors, cancer progression, carcinoid HD)
- Non cancer/cancer therapy-related causes



Farmakis et al. Eur J Heart Fail 2018

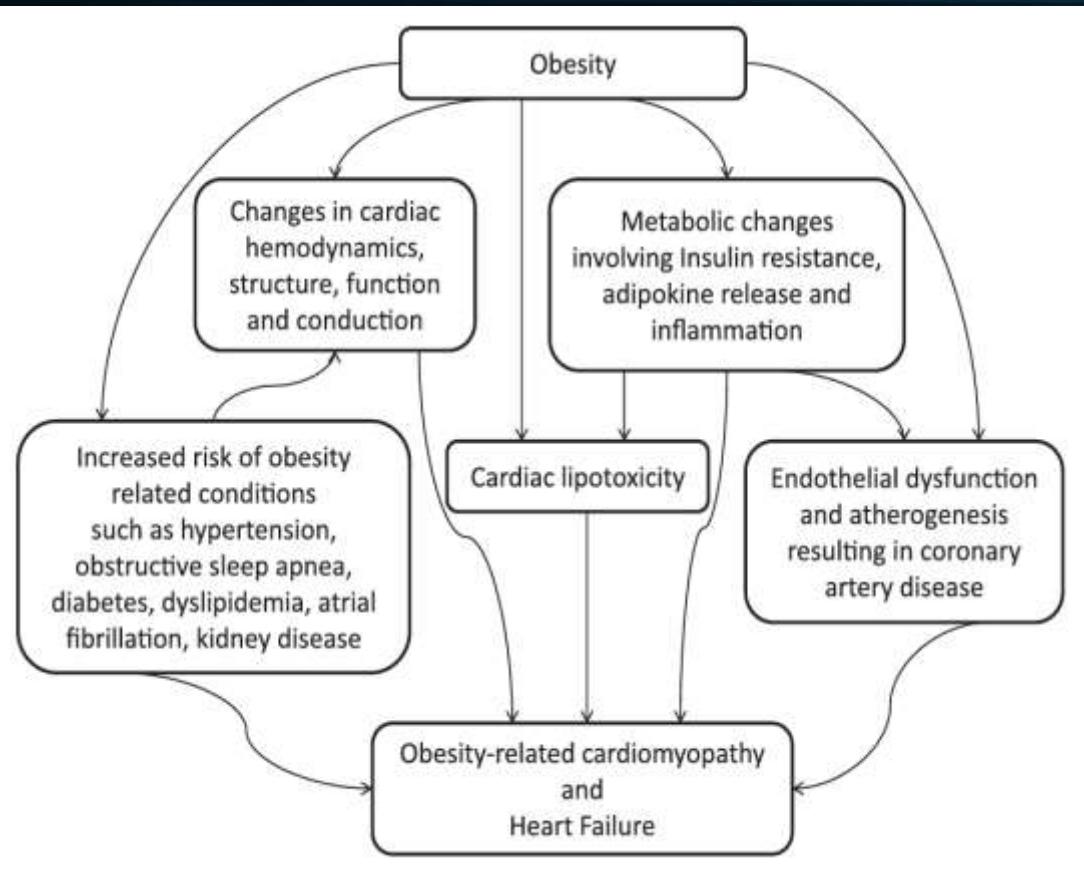
Dynamics of CV toxicity risk

CTR-CVT:
cancer therapy-related cardiovascular toxicity



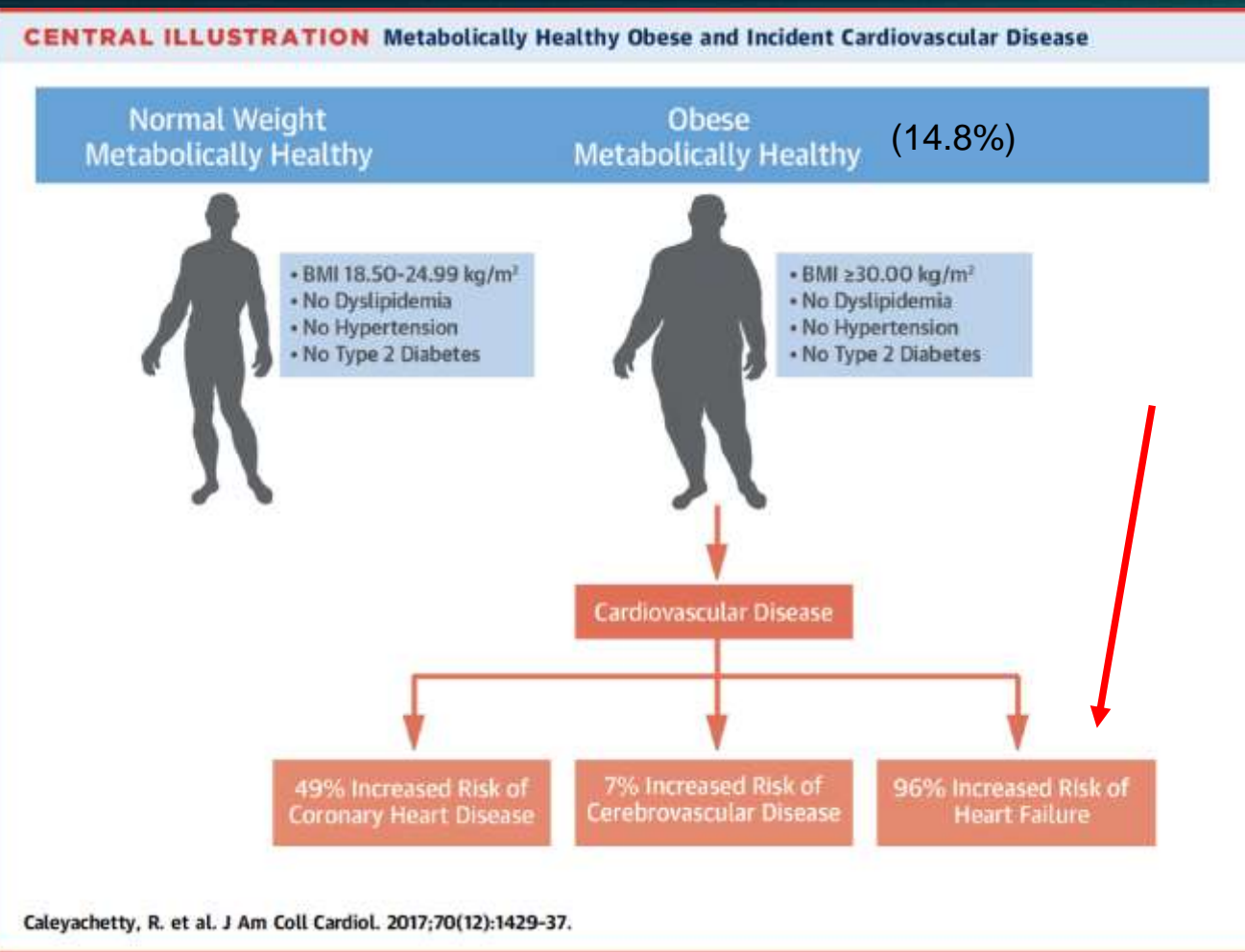
Mechanisms of heart failure in obesity

Imo A. Ebong^{a,*}, David C. Goff Jr.^b, Carlos J. Rodriguez^{c,d}, Haiying Chen^e, Alain G. Bertoni^{c,d}



Metabolically Healthy Obese and Incident Cardiovascular Disease Events Among 3.5 Million Men and Women

Rishi Caleyachetty, MBBS, PhD,^a G. Neil Thomas, PhD,^a Konstantinos A. Toulis, MD, PhD,^{a,b} Nureddin Mohammed, PhD,^a Krishna M. Gokhale, MSc,^a Kumarendran Balachandran, MBBS, MD,^{a,c} Krishnarajah Nirantharakumar, MPH, MD^a



An illustration of the mechanisms of heart failure in obesity

Fernando Domínguez^{1,2}, Eric Adler³, and Pablo García-Pavía^{1,2*}

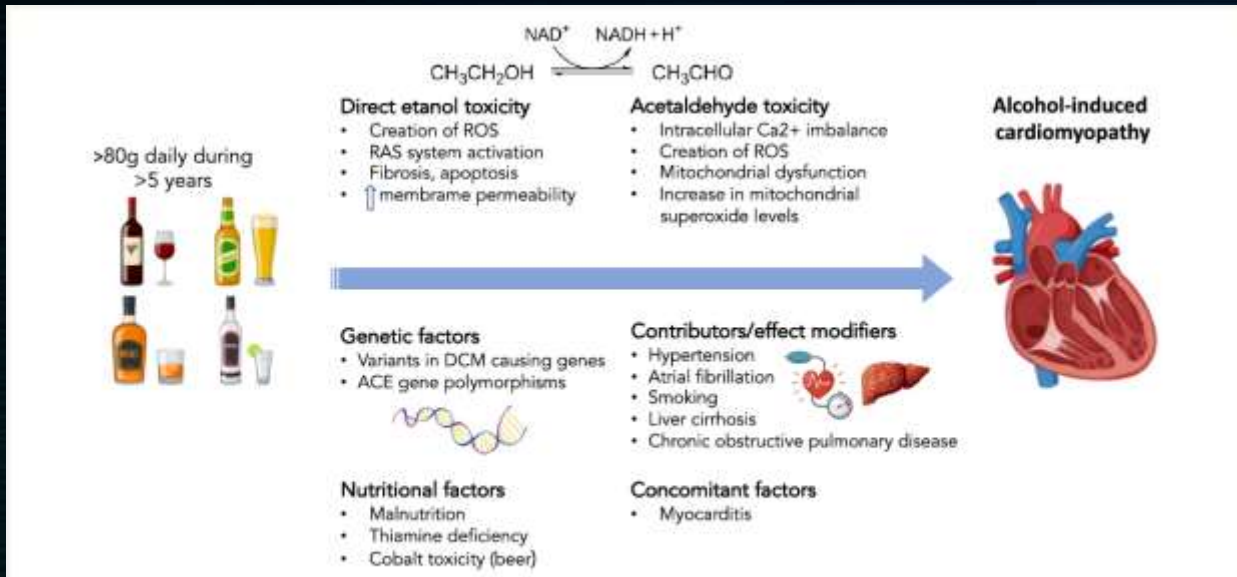


Figure 1 Pathogenic mechanisms of alcohol-induced cardiomyopathy. ACE, angiotensin-converting enzyme; DCM, dilated cardiomyopathy; RAS, renin–angiotensin system; ROS, reactive oxygen species

Echocardiographic changes caused by alcohol consumption

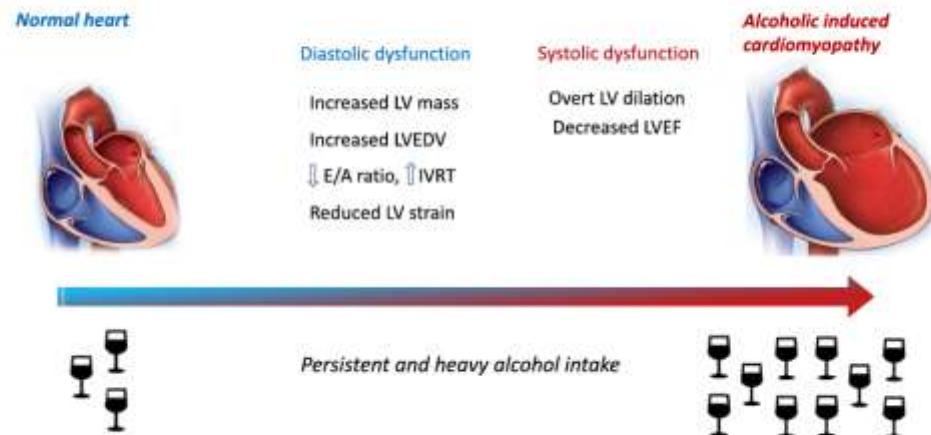


Figure 2 Echocardiographic consequences of heavy and persistent alcohol consumption. IVRT, isovolumic relaxation time; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction

Alcoholic cardiomyopathy

Definition

- ✓ >80g of alcohol, daily, > 5 years
- ✓ Dilated LVEDD > 2SD + LVEF < 50%
- ✓ Exclusion of other causes of DCM

Epidemiology

- ✓ 4–47% of unexplained DCM
- ✓ Male >>> female (9:1)

Pathogenesis

- ✓ Ethanol and acetaldehyde toxicity
- ✓ Genetic factors
- ✓ Comorbidities/effect modifiers
- ✓ Nutritional factors

↓

Treatment

- Alcohol abstinence
- ✓ Naltrexone
- ✓ Acamprostate
- ✓ Disulfiram (caution in HF)
- HF rEF drugs

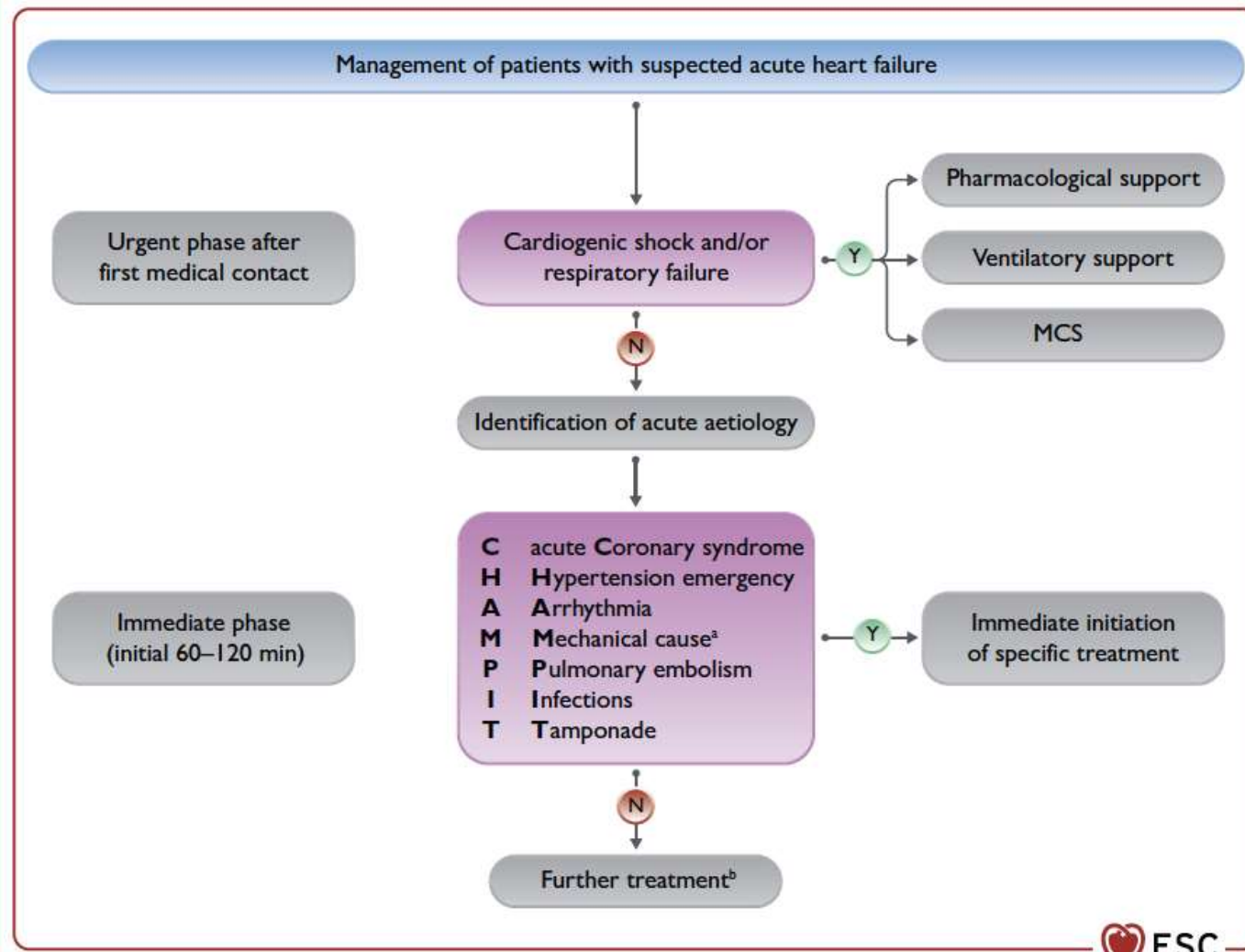
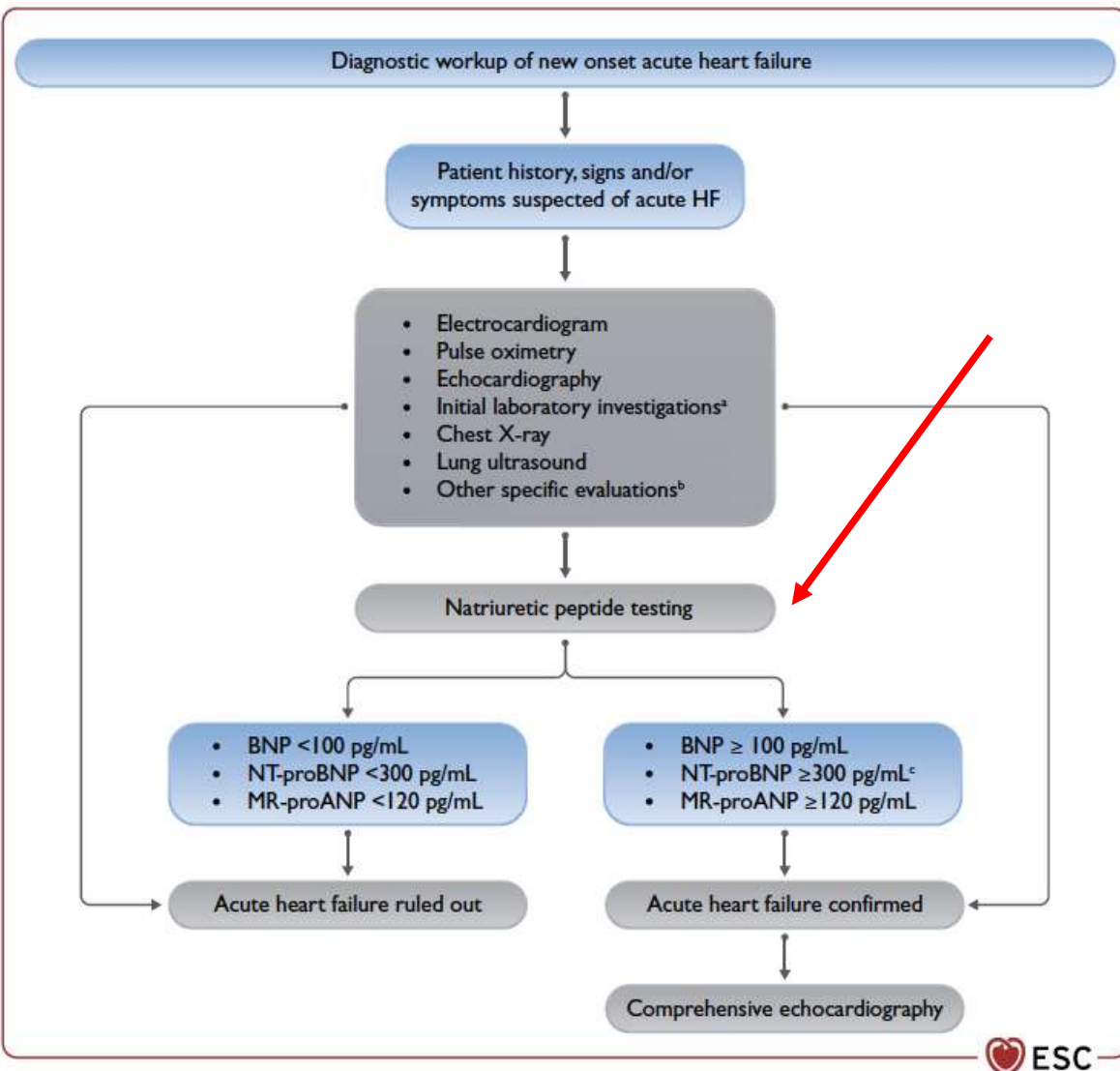
Future therapeutic targets?
 Aim: Limit the extent of cardiac damage

Prognosis

- Relies on abstinence
- ↑ LVEF
- ↑ HTx free survival

Clinical overview, pathogenesis, treatment and prognosis of alcoholic cardiomyopathy. DCM, dilated cardiomyopathy; HF, heart failure; HF rEF, heart failure with reduced ejection fraction; HTx, heart transplant; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; SD, standard deviation

Alcohol-induced cardiomyopathy (AC) is an **acquired** form of dilated cardiomyopathy (DCM) caused by prolonged and heavy alcohol intake in the absence of other causes.



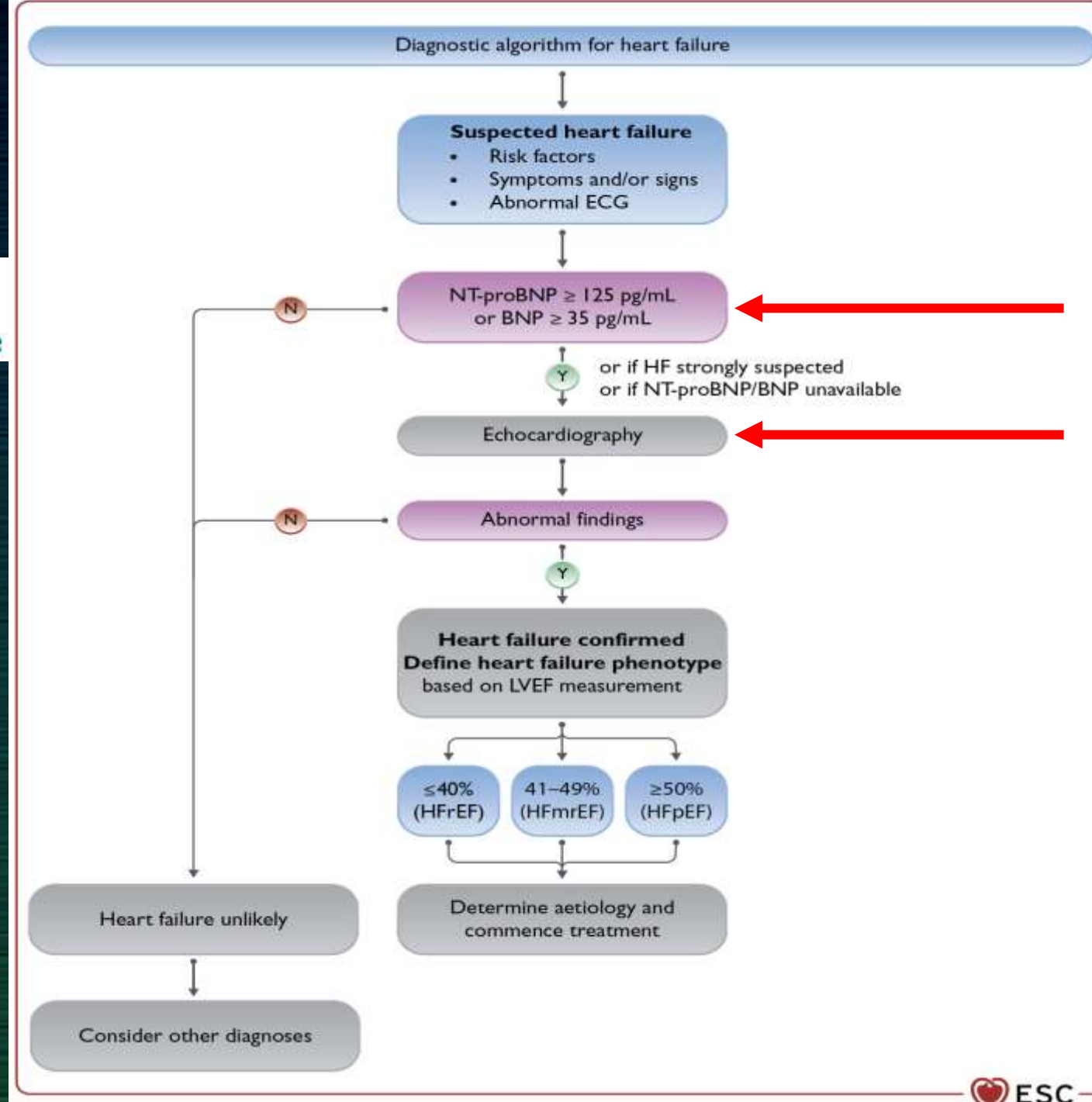
Diagnostic workup of new onset acute heart failure. ACS = acute coronary syndrome; BNP=B-type natriuretic peptide; CT=computed tomography; HF=heart failure; MR-proANP=mid-regional pro-atrial natriuretic peptide; NT-proBNP=N-terminal pro-B-type natriuretic peptide; TSH =thyroid-stimulating hormone. ^aInitial laboratory exams include troponin, serum creatinine, electrolytes, blood urea nitrogen or urea, TSH, liver function tests as well as D-dimer and procalcitonin when pulmonary embolism or infection are suspected, arterial blood gas analysis in case of respiratory distress, and lactate in case of hypoperfusion. ^bSpecific evaluation includes coronary angiography, in case of suspected ACS, and CT in case of suspected pulmonary embolism. ^cRule-in values for the diagnosis of acute HF: >450 pg/mL if aged <55 years, >900 pg/mL if aged between 55 and 75 years and >1800 pg/mL if aged >75 years. 433,434

Initial management of acute heart failure. MCS=mechanical circulatory support. ^aAcute mechanical cause: myocardial rupture complicating acute coronary syndrome (free wall rupture, ventricular septal defect, acute mitral regurgitation), chest trauma or cardiac intervention, acute native or prosthetic valve incompetence secondary to endocarditis, aortic dissection or thrombosis. ^bSee Figures 7–10 for specific treatments according to different clinical presentations

TAKE-HOME MESSAGE

2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure

McDonagh T.A. et al.



TAKE-HOME MESSAGE

Prime valutazioni in sospetto scompenso cardiaco

1. Esami di laboratorio:

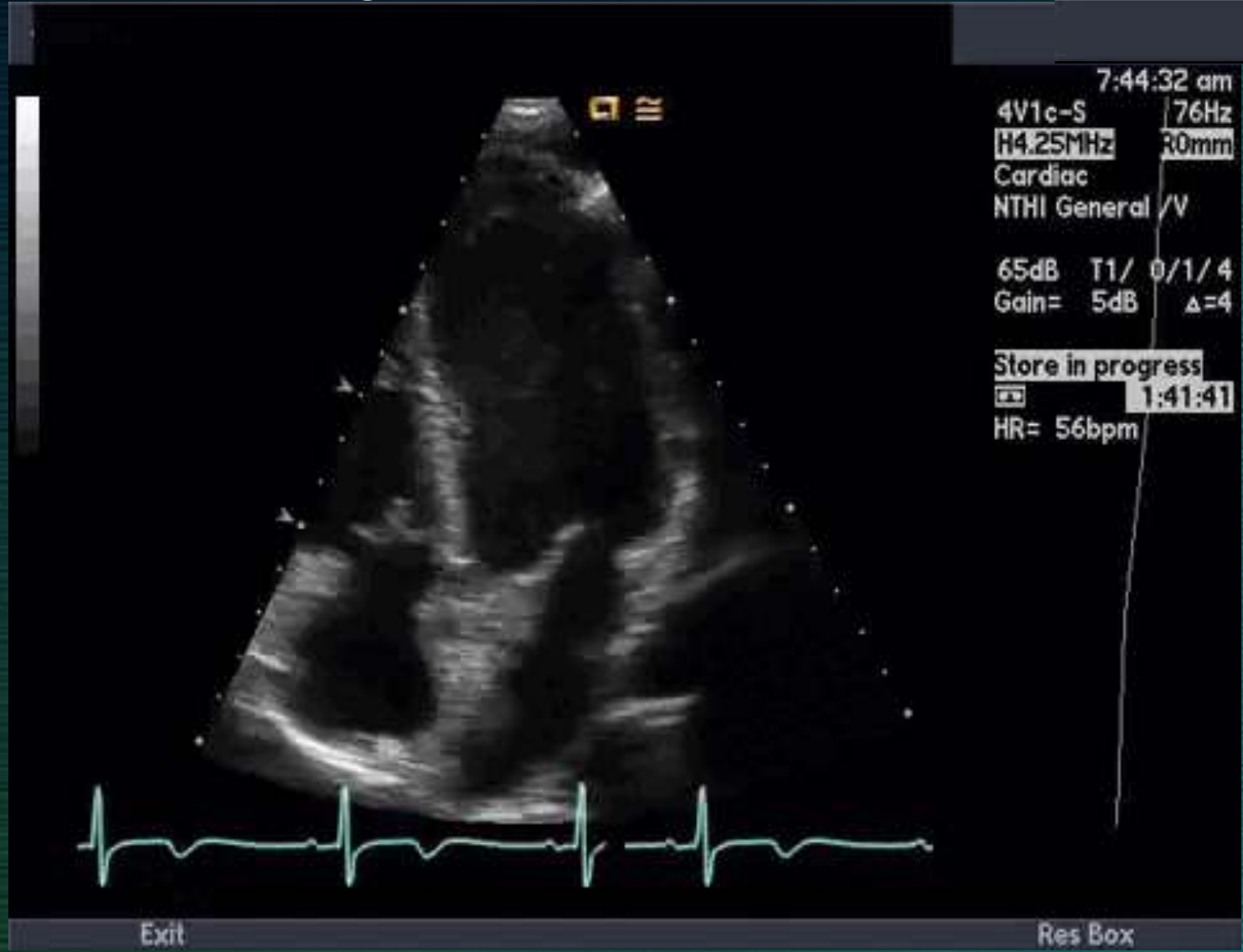
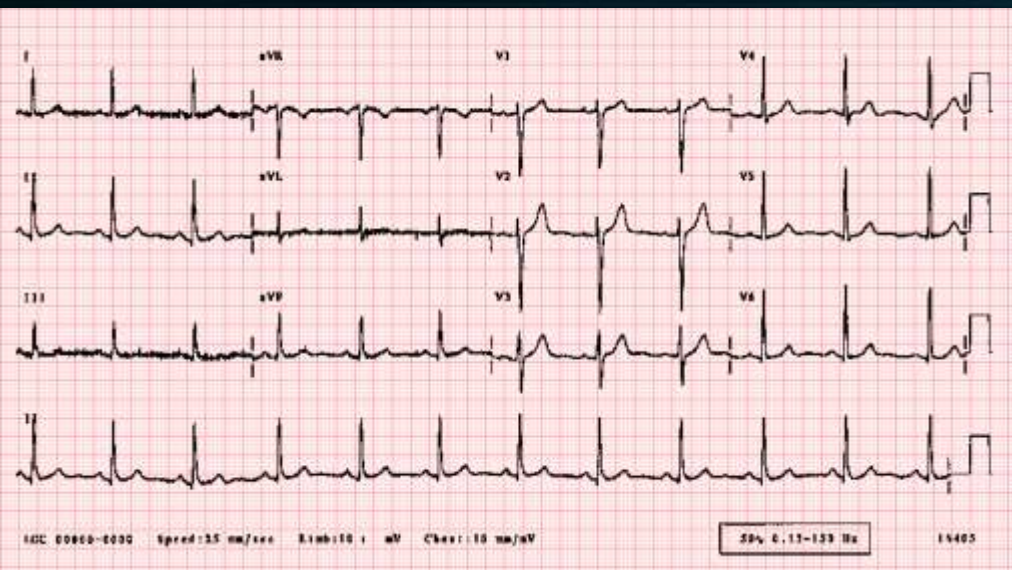
- EMOCROMO: WBC, Hb, PLT.
- Funzionalità renale ed elettroliti: Creatinina (GFR), Urea, K+, Na +, Acido urico
- BNP o NT-proBNP
- Proteine totali, albumina, AST, ALT, fosfatasi alcalina, gamma-GT, LDH, CPK, Elettroforesi delle sieroproteine. Bilirubina totale.
- Profilo lipidico: Colesterolo totale, LDL, HDL, Trigliceridi.
- Emoglobina glicata (HbA1c) e glicemia.
- Funzionalità tiroidea (TSH reflex).
- Assetto marziale:
 - **Ferro**,
 - **Ferritina**,
 - Indice di saturazione,
 - Transferrina.



TAKE-HOME MESSAGE

Prime valutazioni in sospetto scompenso cardiaco

2. Visita cardiologica con ECG ed ecocardio:



HEART FAILURE, A GIANT TO DEFEAT



Francisco Goya (1746-1828), *The Giant, The Colossus*





*Grazie per
l'attenzione!!!*

