Cardiac failure

in public primary care for the family physician

Dr. Liezel Rossouw
Family physician

What will happen in this workshop

- 1. Background to why I am presenting this topic
- 2. Understand basic principles of cardiac failure (short video)
- 3. Newest classification in cardiac failure & new society guidelines
- 4. Clinical case discussion
- 5. 3 questions for the audience
- 6. Diagnosis, investigation and management of cardiac failure patients according to classification

Is heart failure a significant problem?

- In 2008 cardiovascular disease is the most common non-communicable cause of death in SA
- 2022 6177 people per 100 000 population in SA (increased by 5 % from 2017)
- Africa highest case fatality rates 34%, double the world average 16% (INTER-CHF study 2016)
- 5 year mortality 42 to 60 % (Sub Saharan Africa)[1]

1. Adesuyi A. Ajayi, M.D(2020). Explaining Heart failure hyper-morality in Sub Sharan Africa. j.jnm, 112(2) 141 – 154.

Is heart failure a significant problem

- Little documented about our high readmission rates in District Hospitals, as well as mortality rates
- Suspect much higher for those assessed at GSH
- High cost to system and our patients

Why do family physicians need to know this?

Clinical characteristics and causes of heart failure, adherence to treatment guidelines, and mortality of patients with acute heart failure: Experience at Groote Schuur Hospital, Cape Town, South Africa

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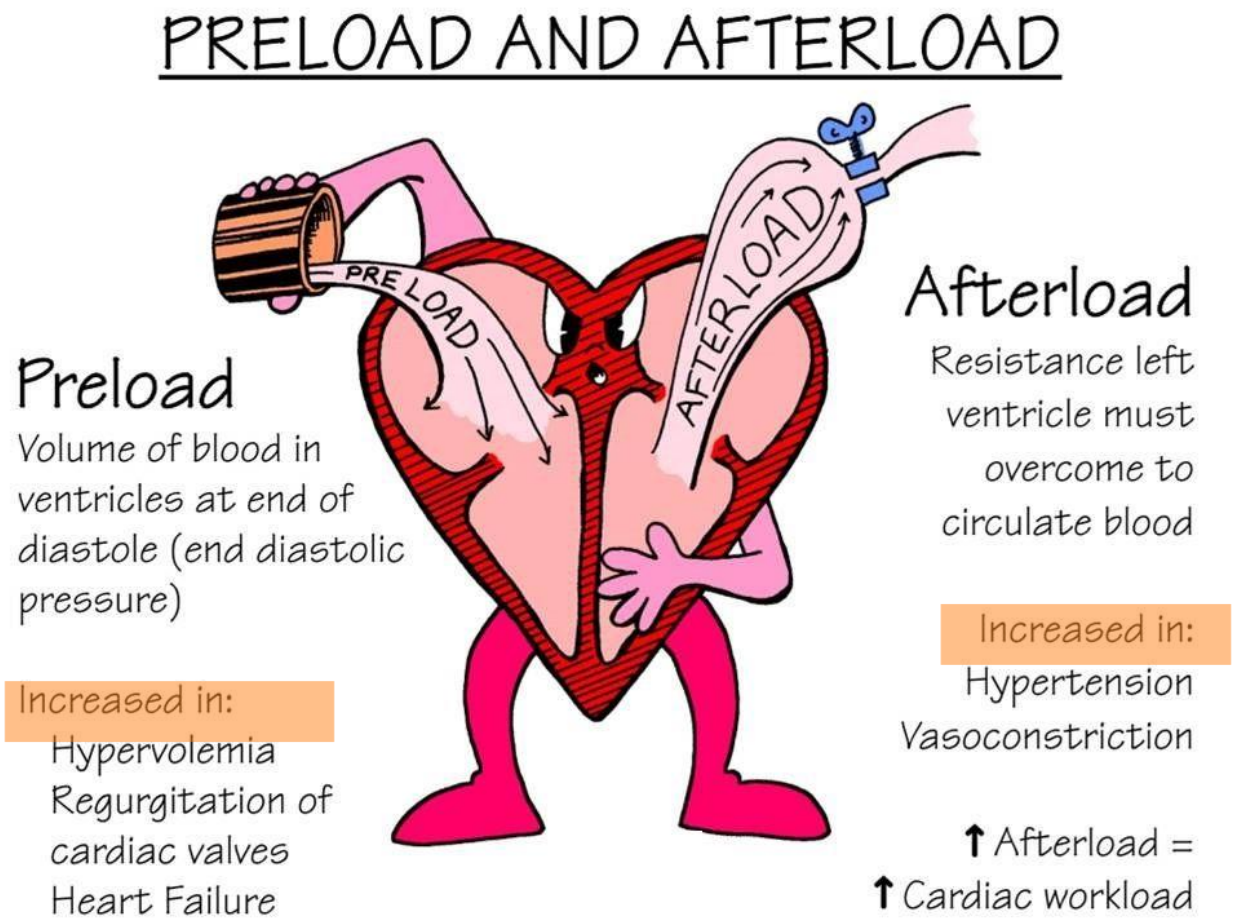
Background. There is limited information on acute heart failure (AHF) and its treatment in sub-Saharan Africa.
Objective. To describe the clinical characteristics and causes of heart failure (HF), adherence to HF treatment guidelines, and mortality of patients with AHF presenting to Groote Schuur Hospital (GSH), Cape Town, South Africa.
Methods. This sub-study of The Sub-Saharan Africa Survey of Heart Failure (THESUS-HF) was a prospective and observational survey that focused on the enrolment and follow-up of additional patients with AHF presenting to GSH and entered into the existing registry after publication of the primary THESUS-HF article in 2012. The patients were classified into prevalent (existing) or incident (new) cases of HF.
Results. Of the 119 patients included, 69 (58.0%) were female and the mean (standard deviation) age was 49.9 (16.3) years. The majority of prevalent cases were patients of mixed ancestry (63.3%), and prevalent cases had more hypertension (70.0%), diabetes mellitus (36.7%), hyperlipidaemia (33.3%) and ischaemic heart disease (IHD) (36.7%) than incident cases. The top five causes of HF were cardiomyopathy (20.2%), IHD (19.3%), rheumatic valvular heart disease (RHD) (18.5%), cor pulmonale (11.8%) and hypertension (10.1%), with the remaining 20.1% consisting of miscellaneous causes including pericarditis, toxins and congenital heart disease. Most patients received renin-angiotensin system blockers and loop diuretics on discharge. There was a low rate of beta-blocker, aldosterone antagonist and digoxin use. Rehospitalisation within 180 days occurred in 25.2% of cases. In-hospital mortality was 8.4% and the case fatality rate at 6 months was 26.1%.
Conclusion. In Cape Town, the main causes of AHF are cardiomyopathy, IHD and RHD. AHF affects a young population and is associated with a high rate of rehospitalisation and mortality. There is serious under-use of beta-blockers, aldosterone antagonists and digoxin. Emphasis on the rigorous application of treatment guidelines is needed to reduce readmission and mortality.

S Afr Med J 2018;108(2):94-98. DOI:10.7196/SAMJ.2018.v108i2.12519

Understanding cardiac failure



What we are looking for with history and clinical exam

Causes Cardiac failure

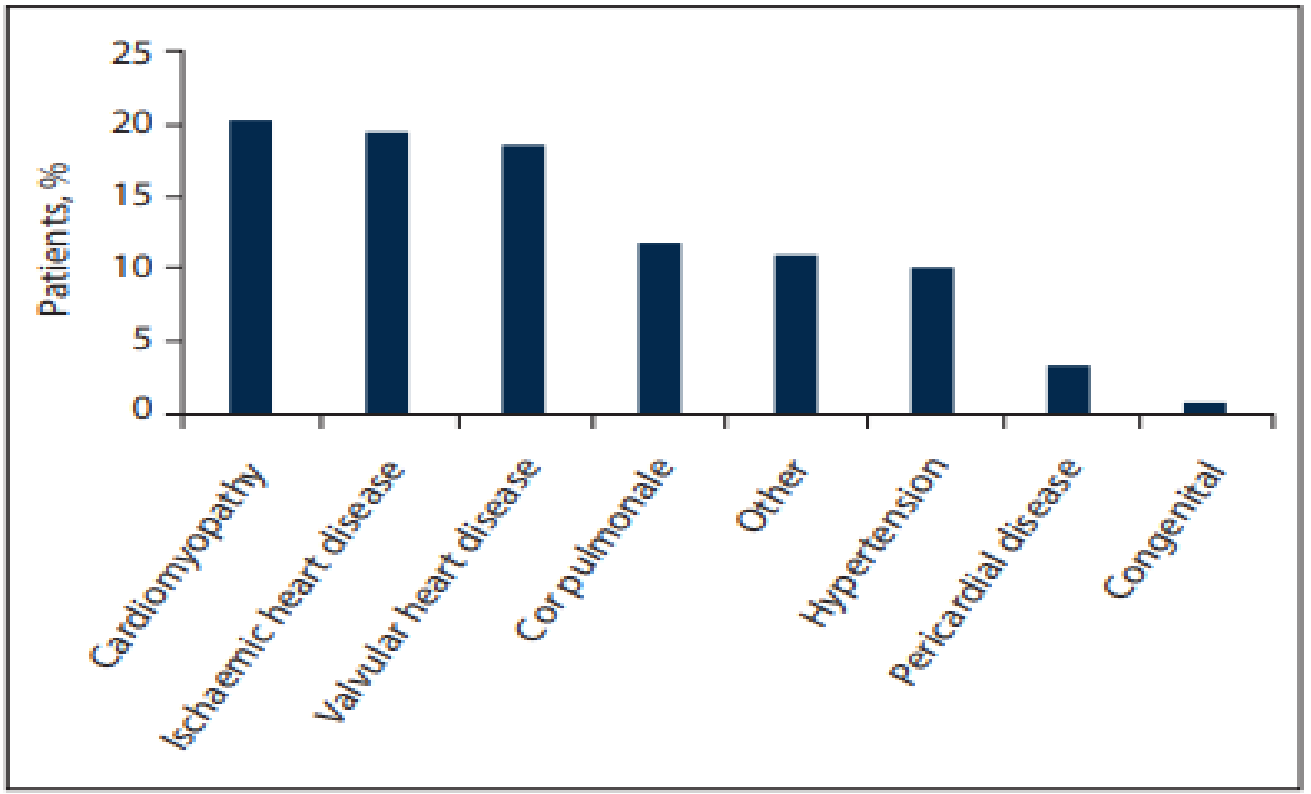


Fig. 1. Causes of heart failure. ('Other' includes toxins, arrhythmias and Graves' disease.)

S Afr Med J 2018;108(2):94-98. DOI:10.7196/SAMJ.2018.v108i2.12519

Causes of heart failure

DESCRIPTION

CCF is a clinical syndrome and has several causes. The cause and immediate precipitating factor(s) of the CCF must be identified and treated to prevent further harm.

Potentially reversible causes include:

- hypertension
- thyroid disease
- valvular heart disease
- constrictive pericarditis
- thiamine deficiency
- ischaemic heart disease
- haemochromatosis
- tachycardia

Adult Hospital Level EDL 2019


CONGESTIVE
HEART FAILURE

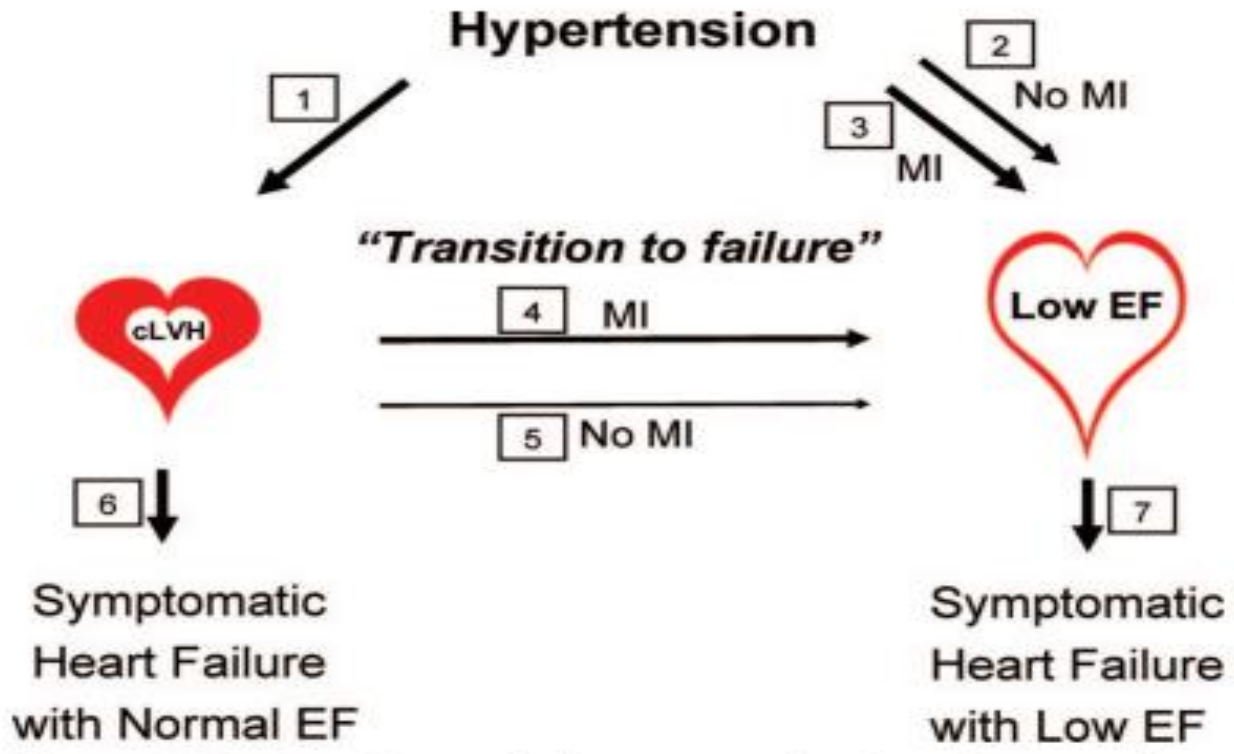


Figure 1. The 7 pathways in the progression from hypertension

From: Mark H. Drazner. The progression of Hypertensive Heart disease. Circulation.2011;123L327-334

Classification Heart failure according to ESC 2022

Table 3 **Definition of heart failure with reduced ejection fraction, mildly reduced ejection fraction and preserved ejection fraction**

Type of HF		HFrEF	HFmrEF	HFpEF
CRITERIA	1	Symptoms ± Signs[a]	Symptoms ± Signs[a]	Symptoms ± Signs[a]
	2	LVEF ≤40%	LVEF 41–49%[b]	LVEF ≥50%
	3	–	–	Objective evidence of cardiac structural and/or functional abnormalities consistent with the presence of LV diastolic dysfunction/raised LV filling pressures, including raised natriuretic peptides[c]

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; LV = left ventricle; LVEF = left ventricular ejection fraction.

[a]Signs may not be present in the early stages of HF (especially in HFpEF) and in optimally treated patients.

[b]For the diagnosis of HFmrEF, the presence of other evidence of structural heart disease (e.g. increased left atrial size, LV hypertrophy or echocardiographic measures of impaired LV filling) makes the diagnosis more likely.

[c]For the diagnosis of HFpEF, the greater the number of abnormalities present, the higher the likelihood of HFpEF.

Symptom based classification

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.

LEFT VS RIGHT symtoms

Clinical assessment of the cardiac failure patient

- You have a 60 year old Hypertensive and Diabetic patient that presents to your clinic with 4 day history of shortness of breath and leg swelling. He had to sit up at night due to shortness of breath. He had no history chest pain.
- BP 150/100 P110 RR 26 hgt 6
- Please discuss in groups of 2/3 (as you sitting)
- What will you look for in your clinical examination?
- What will the side-room and special investigations include?

What do you understand?

1. Which of the following is correct (could be more than one answer):

A patient with uncontrolled hypertension presents to you with shortness of breath. You do not find any clinical signs of cardiac failure. Her sats is 100 % on room air.

a) You can consider the cost-benefit of doing a pro BNP rule out cardiac failure or confirm it

b) You think it is most likely systolic cardiac failure

c) The treatment will consist of an ACE – inhibitor/ARB, a loop diuretic and a b-blocker

d) You can try and do a cardiac ultrasound to determine the ejection fraction in conjunction with telemedical aid

What do you understand?

2. A hypertensive patient presents to you with clear fluid overload. He has peripheral oedema to the knees, a raised JVP. He is not desaturating. Which of the following is correct?

a) You can treat him as an outpatient as the hospital is under bed pressure

b) You can start him on ivi diuresis on the same dose of his chronic diuretic use

c) You will continue the same dose intravenous diuresis until the peripheral oedema resolve

d) You can switch over to oral furosemide in the ward fairly quickly

e) If the patient does not respond to intravenous loop diuretics, hydrochlorotiazide can be added

f) This patient will need ACE-inhibitor, loop diuretics and carvedilol (slowly uptritrated when no creps on chest)

What do you understand

3. Which of the following statements regarding the management of cardiac failure is true:

a) Add spironolactone for all patients with systolic or HFrEF with class III or IV heartfailure already on an ACE-inhibitor/ARB

b) Add digoxin to all patients who is still symptomatic with HFrEF on ACE-Inhibitor/ARB and B blocker, loop diuretic and spironolactone. However not if patient is in renal failure, elderly, low body mass index or hypokalaemic.

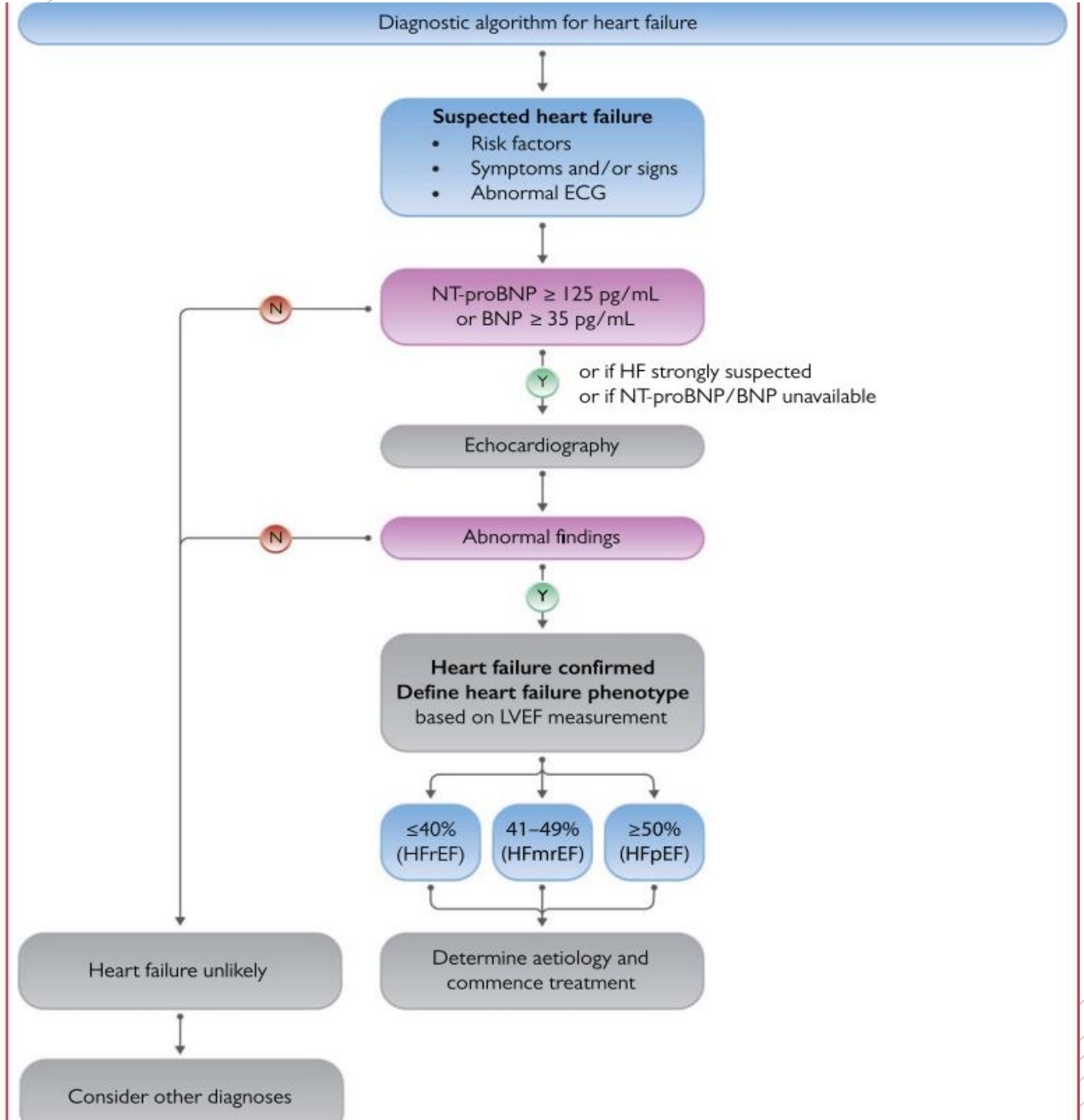
c) Carvedilol is contraindicated in a COAD patients with systolic HFrEF

d) Carvedilol can be safely used in a patient with Asthma and systolic HFrEF

e) The optimum treatment of a patient with Right sided isolated heart failure involves a diuretic, beta blocker and ACE-inhibitor

Diagnosis

Price nhls proBNP R466
Price in District hospital bed – R1543. day
(Day et al., 2011)
ESC guidelines 2022



Pro BNP

- Sensitivity 91% and specificity 91%
- More than 125pg/ml is positive for heart failure
- Other causes for elevated pro BNP include:
- AF, increasing age, renal failure and proBNP may be lower than expected in obese patients

Pro-BNP
In those with symptoms, but you are sure

4.2.1 Use in the non-acute setting

The diagnostic value of NPs, in addition to signs and symptoms and other diagnostic tests, such as an ECG, has been assessed in several studies in the primary care setting.[68,76–80] The aim of these studies was to either exclude or establish a diagnosis of HF. The Task Force considered studies of adequate quality that included NP cut-off points in their diagnostic algorithms, below which the probability of having HF was extremely low. The upper limits of normal in the non-acute setting are 35 pg/mL for BNP, and 125 pg/mL for NT-proBNP. In these studies, the negative predictive values of NP concentrations below these thresholds range from 0.94 to 0.98.[76–78] Fewer data are available for MR-proANP in CHF than in AHF. A concentration of <40 pmol/L can be used to rule out HF.[68]

Investigating cardiac 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

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.

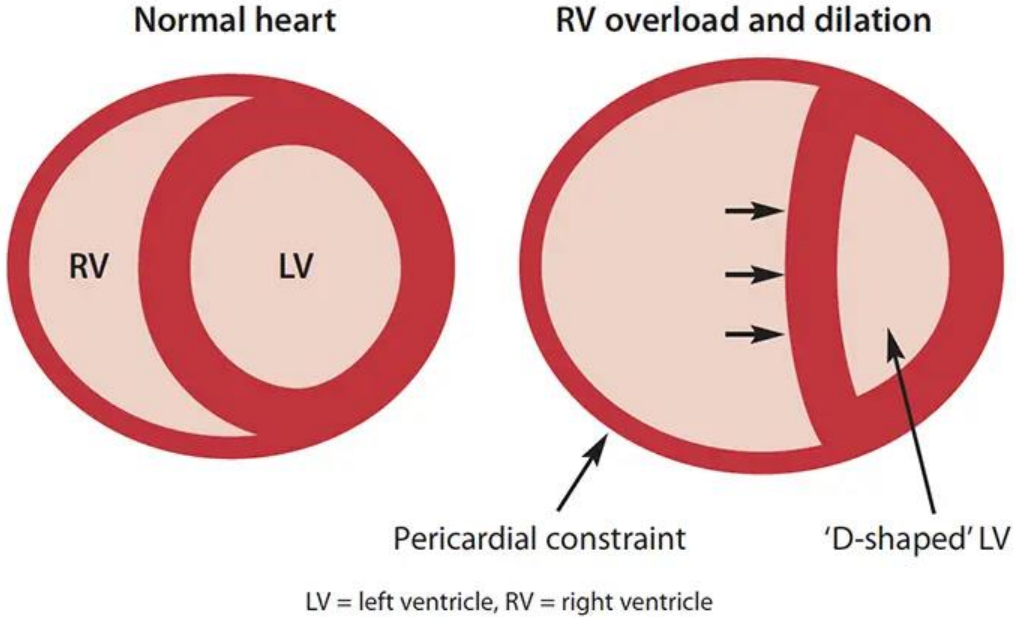
[b]Level of evidence.

[c]References are listed in section 4.2 for this item.

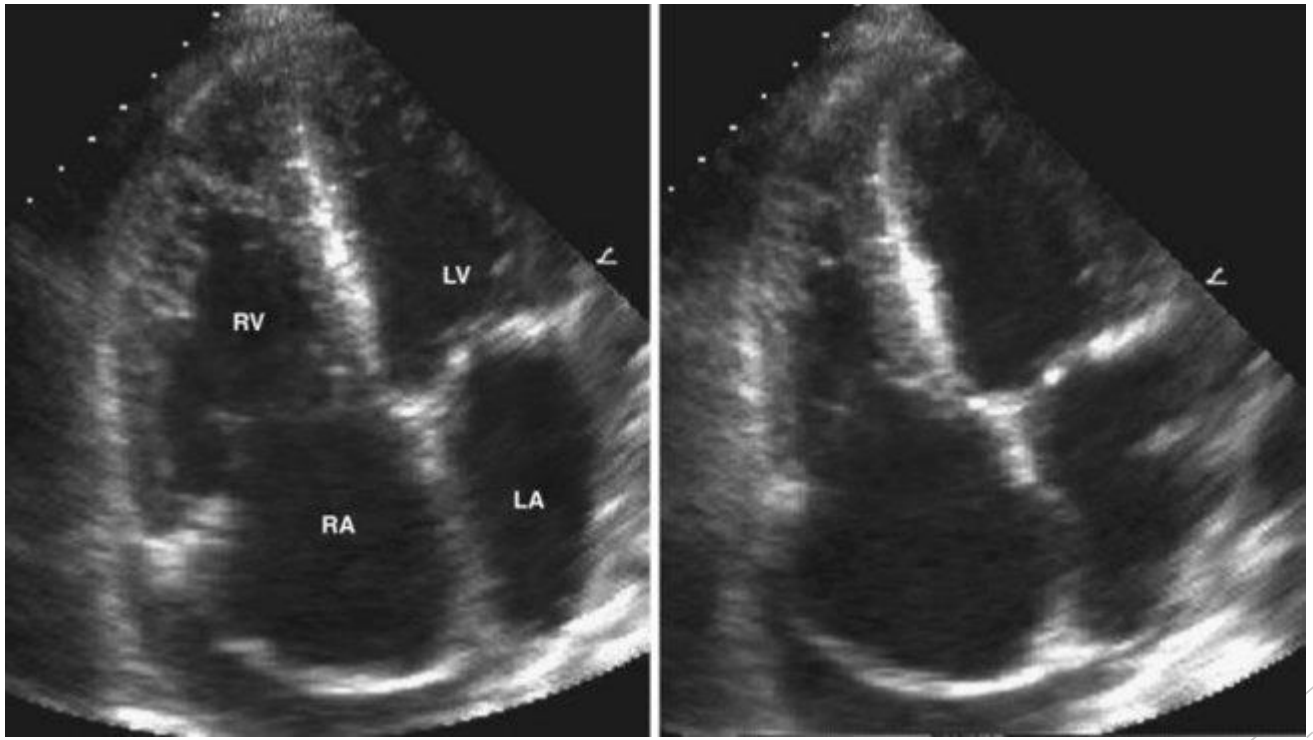
A deducted classification according to treatment principles

- Right sided heart failure (preload, CLD)
- Biventricular cardiac failure (DCMO) and HFrEF (Left sided)
- HFpEF (diastolic disfunction in context HPT)

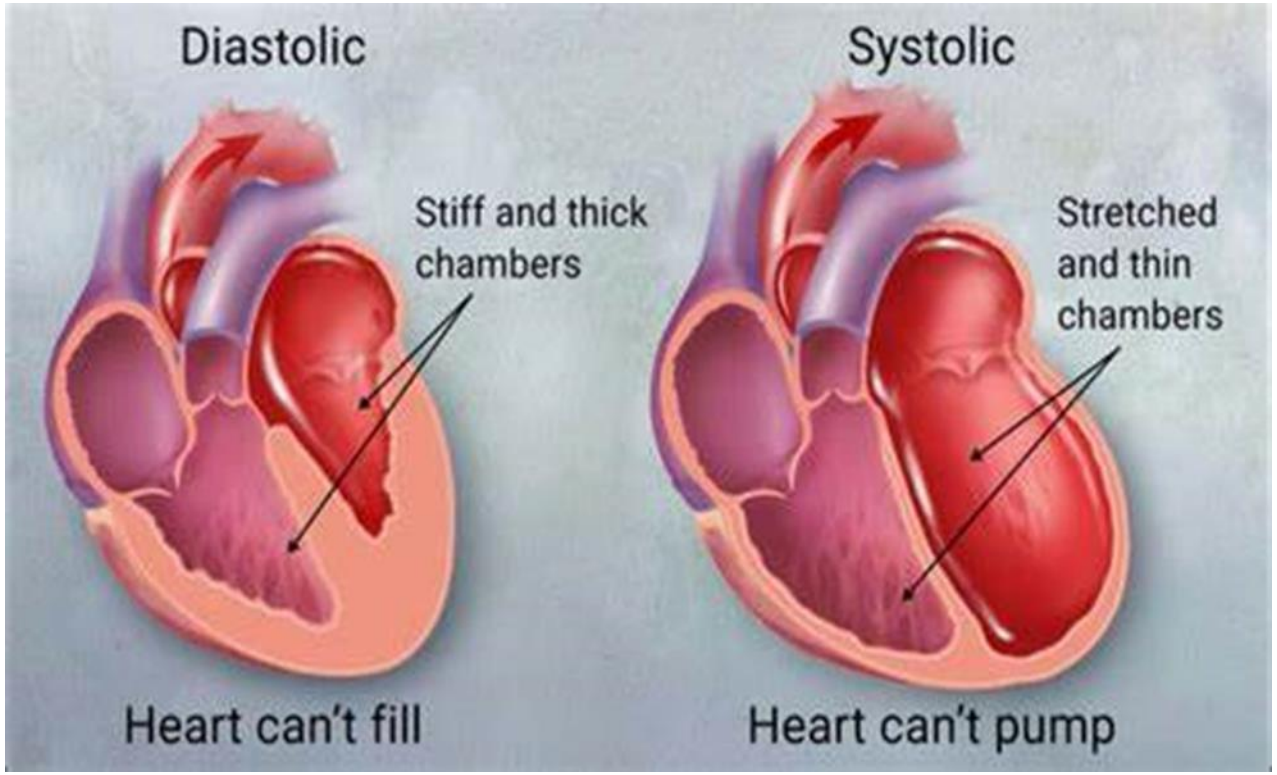
1. Right heart failure dilatation RV due to CLD



LV = left ventricle, RV = right ventricle



2. Biventricular heart failure or HFrEF (systolic dysfunction)



Pharmacological treatments indicated in patients with (NYHA class II–IV) heart failure with reduced ejection fraction (LVEF ≤40%)

Recommendations	Class[a]	Level[b]
An ACE-I is recommended for patients with HFrEF to reduce the risk of HF hospitalization and death.[110–113]	I	A
A beta-blocker is recommended for patients with stable HFrEF to reduce the risk of HF hospitalization and death.[114–120]	I	A
An MRA is recommended for patients with HFrEF to reduce the risk of HF hospitalization and death.[121,122]	I	A
Dapagliflozin or empagliflozin are recommended for patients with HFrEF to reduce the risk of HF hospitalization and death.[108,109]	I	A
Sacubitril/valsartan is recommended as a replacement for an ACE-I in patients with HFrEF to reduce the risk of HF hospitalization and death.[105]	I	B

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ACE-I = angiotensin-converting enzyme inhibitor; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; LVEF = left ventricular ejection fraction; MRA = mineralocorticoid receptor antagonist; NYHA = New York Heart Association.

[a]Class of recommendation.

[b]Level of evidence.

Treatment systolic dysfunction EDL 2019

Significant volume overload or abnormal renal or hepatic function, loop diuretic:

- Furosemide, oral, daily.
  - Initial dose: 40 mg/day.
  - Higher dosages may be needed, especially if comorbid renal failure.
  - Advise patients to weigh themselves daily and adjust the dose if necessary.

LoE:III

Note:

» Unless patient is clinically fluid overloaded, reduce the dose of diuretics before adding an ACE-inhibitor. After introduction of an ACE-inhibitor, try to reduce diuretic dose and consider a change to hydrochlorothiazide.

» Routine use of potassium supplements with diuretics is not recommended. They should be used short-term only, to correct documented low serum potassium level.

LoE:I

Renin-angiotensin-aldosterone system (RAAS) blockers

- ACE-inhibitor, e.g.:
- Enalapril, oral, 2.5 mg 12 hourly, titrated to 10 mg 12 hourly.
  - In the absence of significant side-effects always try to increase the

Treatment systolic disfunction EDL 2019

If ACE-inhibitor intolerant, i.e. intractable cough:

- Angiotensin receptor blocker (ARB), e.g.:
- Losartan, oral, 50–100 mg daily. Specialist initiated.

Spironolactone

Use with an ACE-inhibitor and furosemide in patients presenting with Class III or IV heart failure.

Do not use if eGFR <30 mL/minute.

Monitoring of potassium levels is essential if spironolactone is used with an ACE-inhibitor or other potassium sparing agent or in the elderly.

- Spironolactone, oral, 25–50 mg once daily.

LoE:III[iii]

ß-blockers

For all stable patients with heart failure who tolerate it:

Note: Patients should not be fluid overloaded or have a low BP before initiation of therapy.

- Carvedilol, oral.
  - Initial dose: 3.125 mg 12 hourly.
  - Increase at 2-weekly intervals by doubling the daily dose until a maximum of 25 mg 12 hourly, if tolerated.
  - If not tolerated, i.e. worsening of cardiac failure symptoms, reduce the dose to the previously tolerated dose.
  - Up-titration should take several weeks or months.

LoE:I[iii]

Digoxin

Treatment systolic dysfunction EDL 2019

Digoxin

Patients in sinus rhythm remaining symptomatic despite the above-mentioned agents (Specialist consultation):

- Digoxin, oral, 0.125 mg daily, adjust according to response and trough plasma level.
  - Digoxin trough plasma levels (before the morning dose) should be maintained between 0.6-1 nmol/L.
  - Patients at high risk of digoxin toxicity: the elderly, patients with renal dysfunction, hypokalaemia and patients with lean body mass.

B blocker, cardiac failure

Common classes

B1 receptors inhibitors (cardioselective) – bisoprolol, metoprolol, atenolol

Inhibit B1 and B2 inhibitors (smooth muscle)receptors (non cardioselective) carvedilol, propranolol, labetalol, timolol

Gulea *et al. Respir Res* (2021) 22:64
https://doi.org/10.1186/s12931-021-01661-8

Beta-blocker therapy in patients with COPD: a systematic literature review and meta-analysis with multiple treatment comparison

Claudia Gulea[1,2*], Rosita Zakeri[3], Vanessa Alderman[4], Alexander Morgan[5], Jack Ross[6] and Jennifer K. Quint[1,2,7]

B blockers, cardiac failure and COAD

Abstract

Background: Beta-blockers are associated with reduced mortality in patients with cardiovascular disease but are often under prescribed in those with concomitant COPD, due to concerns regarding respiratory side-effects. We investigated the effects of beta-blockers on outcomes in patients with COPD and explored within-class differences between different agents.

Methods: We searched the Cochrane Central Register of Controlled Trials, Embase, Cumulative Index to Nursing and Allied Health Literature (CINAHL) and Medline for observational studies and randomized controlled trials (RCTs) investigating the effects of beta-blocker exposure versus no exposure or placebo, in patients with COPD, with and without cardiovascular indications. A meta-analysis was performed to assess the association of beta-blocker therapy with acute exacerbations of COPD (AECOPD), and a network meta-analysis was conducted to investigate the effects of individual beta-blockers on FEV1. Mortality, all-cause hospitalization, and quality of life outcomes were narratively synthesized.

Results: We included 23 observational studies and 14 RCTs. In pooled observational data, beta-blocker therapy was associated with an overall reduced risk of AECOPD versus no therapy (HR 0.77, 95%CI 0.70 to 0.85). Among individual beta-blockers, only propranolol was associated with a relative reduction in FEV1 versus placebo, among 199 patients evaluated in RCTs. Narrative syntheses on mortality, all-cause hospitalization and quality of life outcomes indicated a high degree of heterogeneity in study design and patient characteristics but suggested no detrimental effects of beta-blocker therapy on these outcomes.

Conclusion: The class effect of beta-blockers remains generally positive in patients with COPD. Reduced rates of AECOPD, mortality, and improved quality of life were identified in observational studies, while propranolol was the only agent associated with a deterioration of lung function in RCTs.

Asthma, cardiac failure and b blockers

The safety of cardioselective β_1-blockers in asthma: literature review and search of global pharmacovigilance safety reports

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ABSTRACT

Introduction: Beta-blockers are key in the management of cardiovascular diseases but blocking airway β_2-receptors can cause severe and sometimes fatal bronchoconstriction in people with asthma. Although cardioselective β_1-blockers may be safer than non-selective β-blockers, they remain relatively contraindicated and under-prescribed. We review the evidence of the risk associated with cardioselective β_1-blocker use in asthma.

Methods: We searched "asthma" AND "beta-blocker" in PubMed and EmbaseOvid from start to May 2020. The World Health Organization (WHO) global database of individual case safety reports (VigiBase) was searched for reports of fatal asthma or bronchospasm and listed cardioselective β_1-blocker use (accessed February 2020). Reports were examined for evidence of pre-existing asthma.

Results: PubMed and EmbaseOvid searches identified 304 and 327 publications, respectively. No published reports of severe or fatal asthma associated with cardioselective β_1-blockers were found. Three large observational studies reported no increase in asthma exacerbations with cardioselective β_1-blocker treatment. The VigiBase search identified five reports of fatalities in patients with pre-existing asthma and reporting asthma or bronchospasm during cardioselective β_1-blocker use. Four of these deaths were unrelated to cardioselective β_1-blocker use. The circumstances of the fifth death were unclear.

Conclusions: There were no published reports of cardioselective β_1-blockers causing asthma death. Observational data suggest that cardioselective β_1-blocker use is not associated with increased asthma exacerbations. We found only one report of an asthma death potentially caused by cardioselective β_1-blockers in a patient with asthma in a search of VigiBase. The reluctance to use cardioselective β_1-blockers in people with asthma is not supported by this evidence.

Loop diuretics and AHF

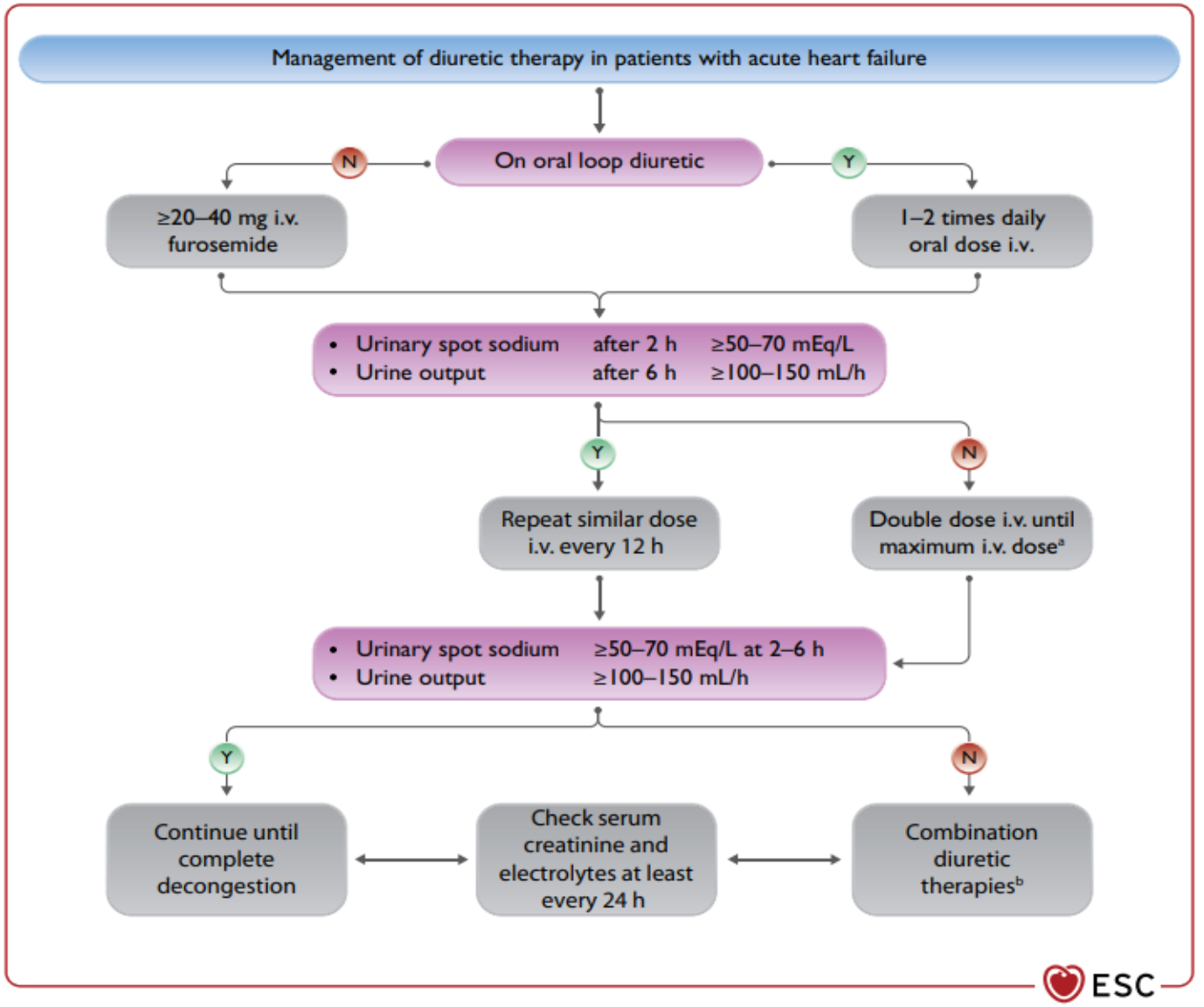


Figure 13 Diuretic therapy (furosemide) in acute heart failure. i.v. = intravenous. [a]The maximal daily dose for i.v. loop diuretics is generally considered furosemide 400–600 mg though up to 1000 mg may be considered in patients with severely impaired kidney function. [b]Combination therapy is the addition to the loop diuretic of a diuretic with a different site of action, e.g. thiazides or metolazone or acetazolamide. Modified from [145].

Diuretics ESC 2022 guidelines

- Poor response to loop diuretic – addition of diuretics acting on different sites namely thiazides
- BUT monitor serum e and renal function
- Decrease diuretic dose when negative fluid balance obtained
- NB Care must also be taken to avoid patients being discharged from hospital with persistent congestion as this is Evidence based of increased deaths and rehospitalisation

vasodilators

- Indicated (nitroprusside) where pulmonary oedema is caused by increased afterload and mainly fluid retention in lungs – dilate venous and arterial vessels
- *however recent rct did not find evidence for NP to be better than loop diuretics

MEDICINE TREATMENT

Where heart failure is due to left ventricular systolic dysfunction, mortality is significantly reduced by the use of ACE-inhibitors, ß-blockers and spironolactone and every effort should be made to ensure eligible patients receive all these agents in appropriate doses.

Note: All the guideline evidence presented here relates to treatment of patients in whom the heart failure syndrome is due to left ventricular systolic dysfunction and cannot necessarily be extrapolated to patients in whom heart failure is due to other causes of the syndrome.

Additional therapy

Thiamine

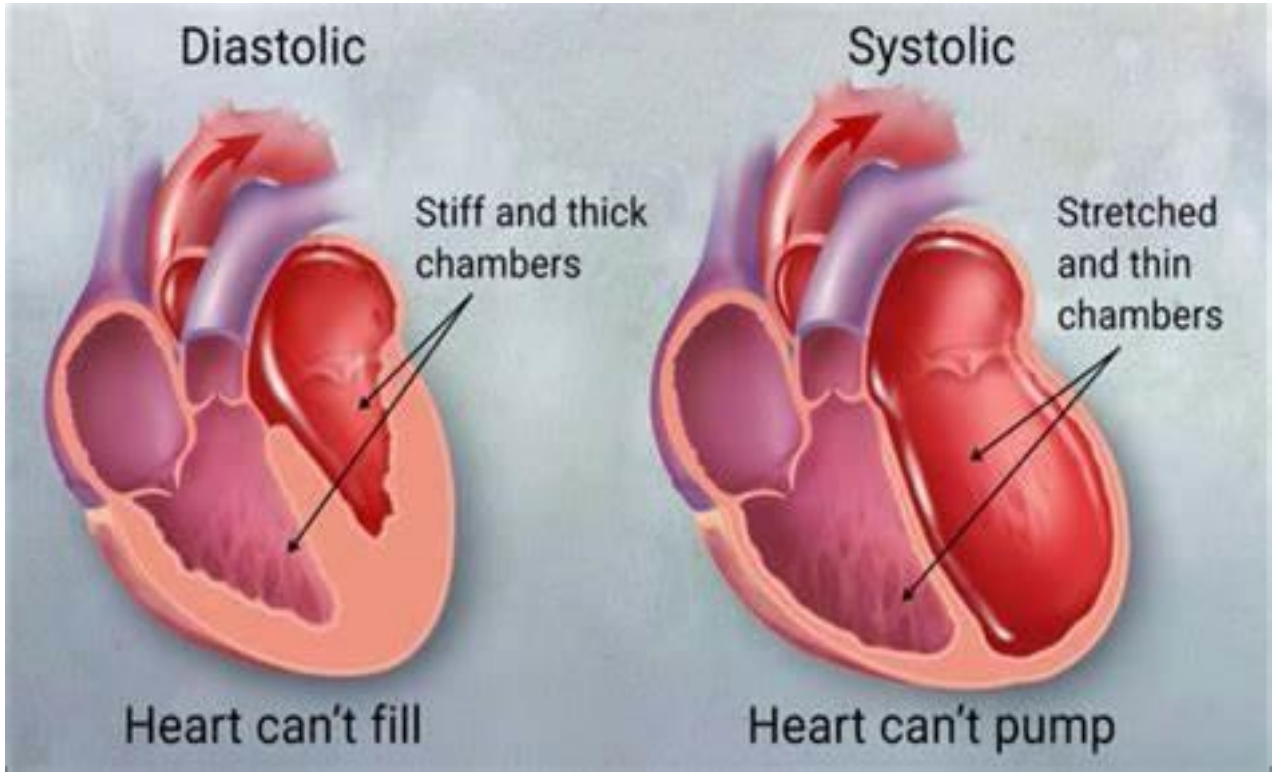
Consider as a trial of therapy in all unexplained heart failure:

- Thiamine, oral/IM, 100 mg daily for 4 weeks.

Prophylaxis (Z29.2)

- Annual influenza vaccine. See section 9.2: Adult vaccination.

3. HFrEF (diastolic disfunction in hypertensive context)



Recommendations for the treatment of patients with heart failure with preserved ejection fraction

Recommendations	Class[a]	Level[b]
Screening for, and treatment of, aetiologies, and cardiovascular and non-cardiovascular comorbidities is recommended in patients with HFpEF (see relevant sections of this document).	I	C
Diuretics are recommended in congested patients with HFpEF in order to alleviate symptoms and signs.[137]	I	C

HFpEF = heart failure with preserved ejection fraction.
[a]Class of recommendation.
[b]Level of evidence.

Mx risk factors in HFpEF

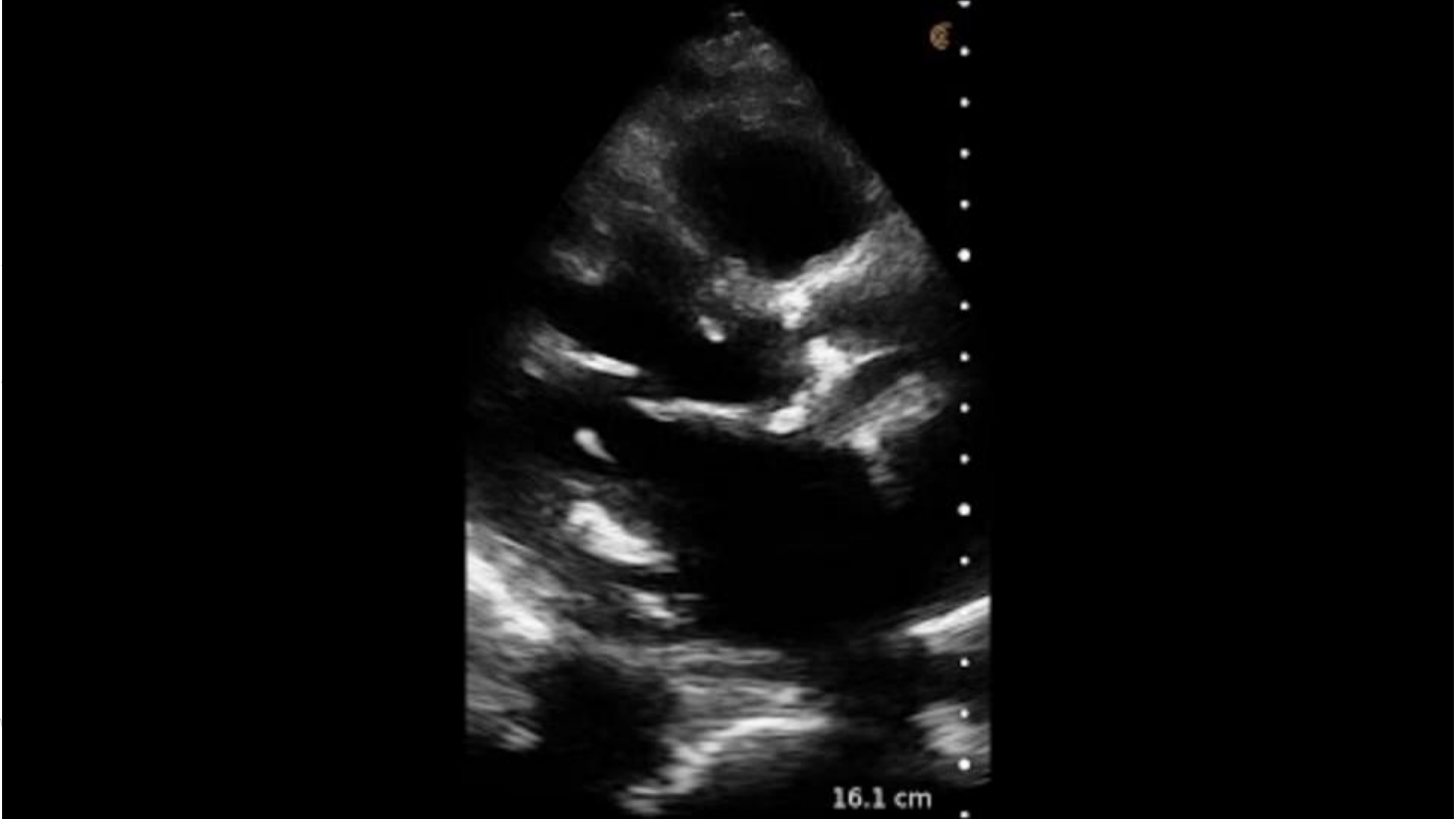
Table 10 **Risk factors for the development of heart failure and potential corrective actions**

Risk factors for heart failure	Preventive strategies
Sedentary habit	Regular physical activity
Cigarette smoking	Cigarette smoking cessation
Obesity	Physical activity and healthy diet
Excessive alcohol intake[287]	General population: no/light alcohol intake is beneficial Patients with alcohol-induced CMP should abstain from alcohol
Influenza	Influenza vaccination
Microbes (e.g. *Trypanosoma cruzi*, Streptococci)	Early diagnosis, specific antimicrobial therapy for either prevention and/or treatment
Cardiotoxic drugs (e.g., anthracyclines)	Cardiac function and side effect monitoring, dose adaptation, change of chemotherapy
Chest radiation	Cardiac function and side effect monitoring, dose adaptation
Hypertension	Lifestyle changes, antihypertensive therapy
Dyslipidaemia	Healthy diet, statins
Diabetes mellitus	Physical activity and healthy diet, SGLT2 inhibitors
CAD	Lifestyle changes, statin therapy

CAD = coronary artery disease; CMP = cardiomyopathy; SGLT2 = sodium-glucose co-transporter 2.

ECHO or CARDIAC ultrasound with telemedicine

- TO CONFIRM CARDIAC FAILURE WHERE DOUBT
- TO look for evidence of ischaemic heart disease (HYPOKINETIC LV/SEPTUM)
- TO determine EF if less 40 % as an indication for CARVEDILOL
- To exclude isolated Cor pulmonale
- To exclude pericardial effusion causing acute Right heart failure
- IN IHD – WE WANT TO USE ATENOLOL IN EF 40 OR MORE (HR control)
- To show if Valvular pathology causing heart failure
- TO look at acute Aorta stenosis as cause for acute cardiac failure


16.1 cm

Aorta stenosis as cause cardiac failure

- TAVI (Transcatheter aorta valve replacement)

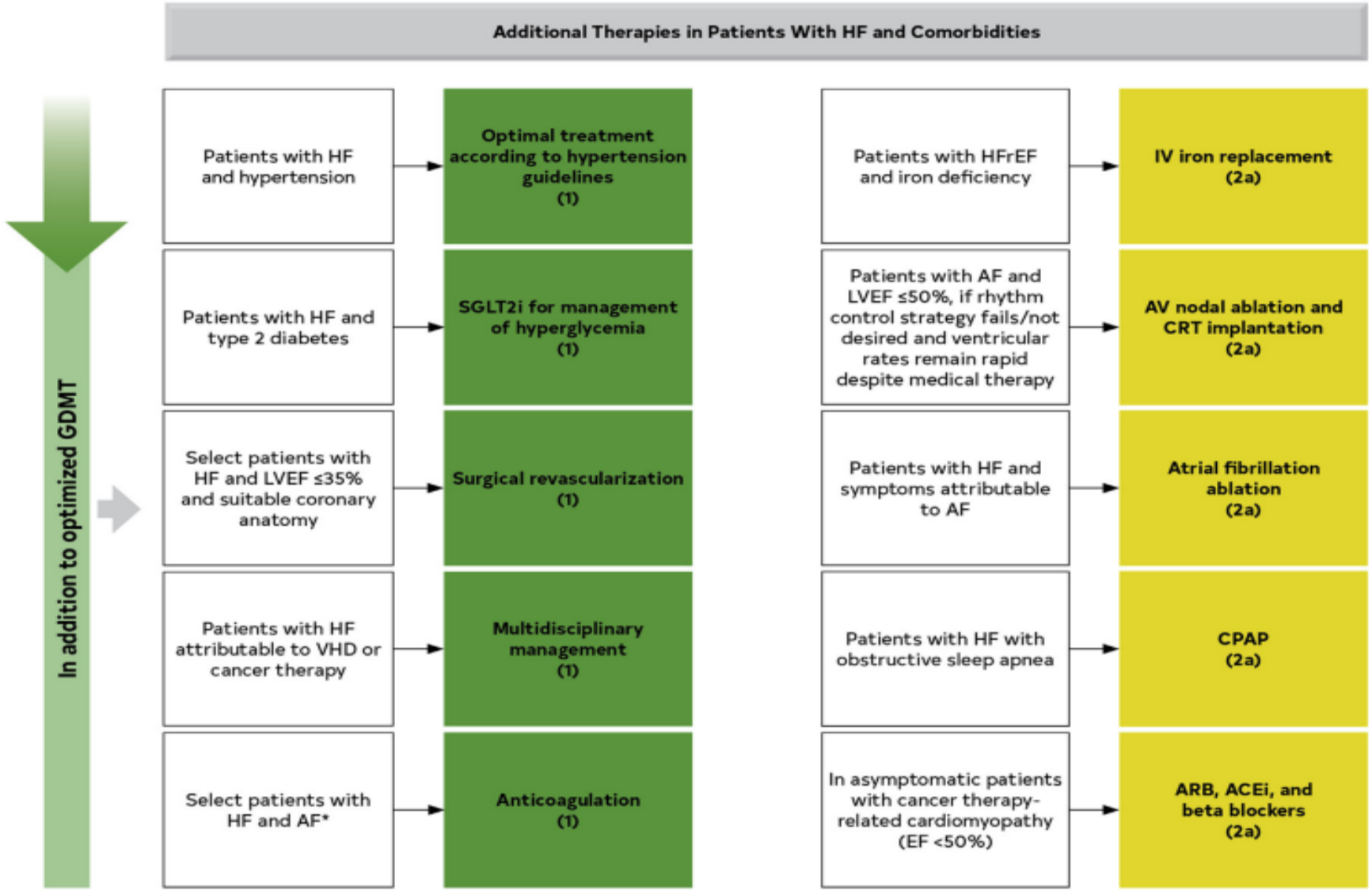
When to refer? EDL 2019

REFERRAL

- Where specialised treatment and diagnostic work-up is needed and to identify treatable and reversible causes.
- All patients with audible cardiac murmurs should undergo specialist evaluation, as should all patients with potentially reversible causes of the heart failure syndrome and those with persistent and severe symptoms and signs of fluid overload despite adequate doses of diuretic.
- Patients who have LBBB on the ECG are potential candidates for cardiac resynchronization therapy. An ECG should be recorded at baseline and repeated at 6-monthly intervals.
- Patients with LBBB should be referred for consideration for resynchronsation therapy, discussed with a specialist.

Defibrillators

An ICD is recommended to reduce the risk of sudden death and all-cause mortality in patients who have recovered from a ventricular arrhythmia causing haemodynamic instability, and who are expected to survive for >1 year with good functional status, in the absence of reversible causes or unless the ventricular arrhythmia has occurred <48 h after a MI.	I	A
An ICD is recommended to reduce the risk of sudden death and all-cause mortality in patients with symptomatic HF (NYHA class II–III) of an ischaemic aetiology (unless they have had an MI in the prior 40 days—see below), and an LVEF ≤35% despite ≥3 months of OMT, provided they are expected to survive substantially longer than 1 year with good functional status.	I	A



2022 ACC/AHA/HFSA Guideline for the Management of Heart Failure - DOI: 10.1016/j.cardfail.2022.02.010

Serum ferritin less 100 or 100 to 299 with a TF sats less 20%
&
Heart failure
Consider IVI iron

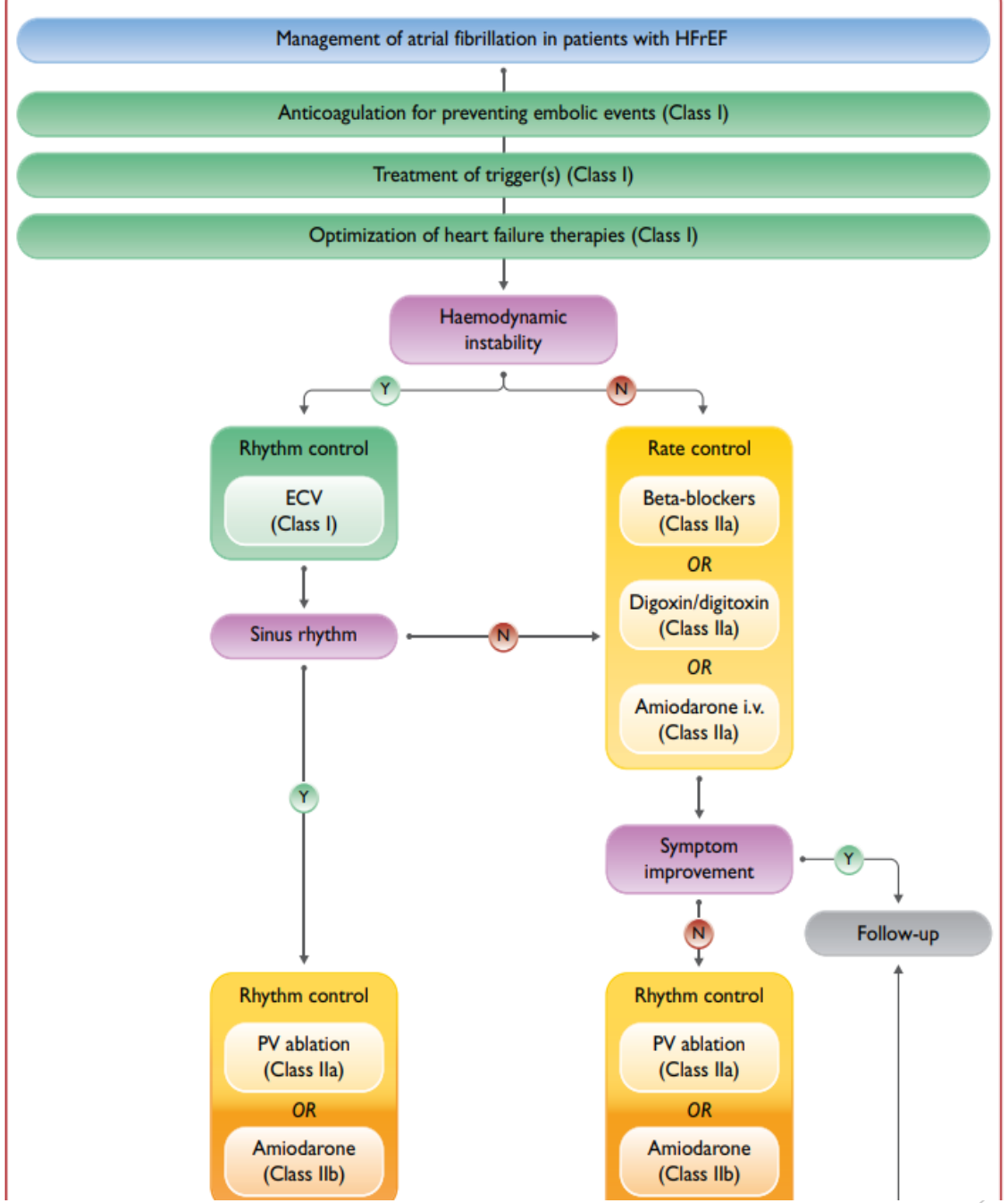
Iron deficiency and cardiac failure

Recommendations for the management of anaemia and iron deficiency in patients with heart failure

Recommendations	Class[a]	Level[b]
It is recommended that all patients with HF be periodically screened for anaemia and iron deficiency with a full blood count, serum ferritin concentration, and TSAT.	I	C
Intravenous iron supplementation with ferric carboxymaltose should be considered in symptomatic patients with LVEF <45% and iron deficiency, defined as serum ferritin <100 ng/mL or serum ferritin 100–299 ng/mL with TSAT <20%, to alleviate HF symptoms, improve exercise capacity and QOL.[721,723,725]	IIa	A
Intravenous iron supplementation with ferric carboxymaltose should be considered in symptomatic HF patients recently hospitalized for HF and with LVEF <50% and iron deficiency, defined as serum ferritin <100 ng/mL or serum ferritin 100–299 ng/mL with TSAT <20%, to reduce the risk of HF hospitalization.[513]	IIa	B

HF = heart failure; LVEF = left ventricular ejection fraction; QOL = quality of life; TSAT = transferrin saturation.
[a]Class of recommendation.
[b]Level of evidence.

Arrythmias and cardiac failure



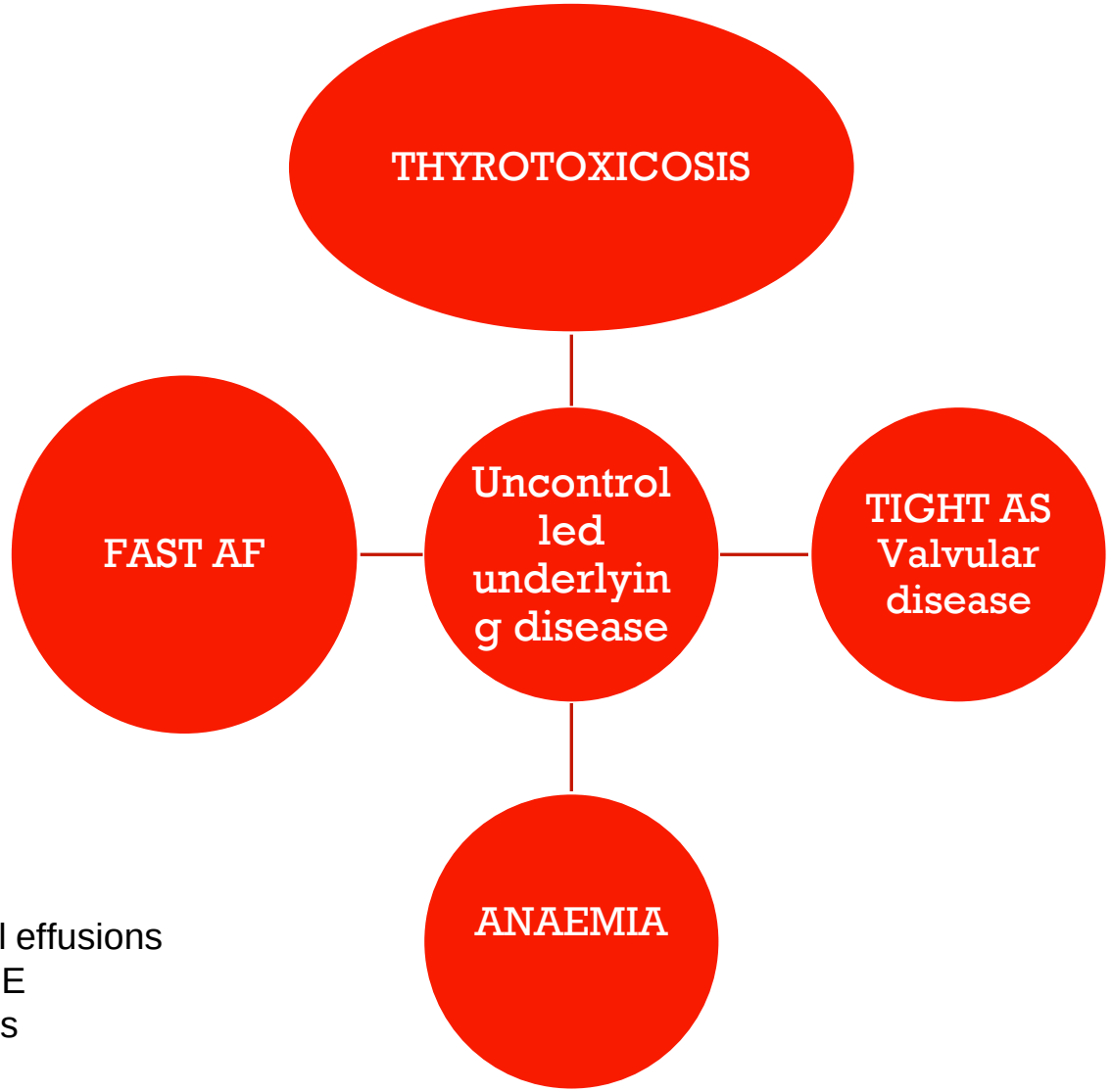
Post partum

- Bromocriptine and specialist consultation for echo

Non pharmacological treatment

1. Educate about the disease and prognosis
2. Medication: joint decision always bring meds with and to understand meds, especially how loop diuretics could be altered
3. To encourage regular exercise and to adapt to circumstances
4. Sleep and rest is important (recognise obstructive elements)
5. Fluids: To avoid large volumes restrict 1.5 to 2 L per day but increase if high heat/N and V
6. Dieet – avoid more than 5g/day salt
7. Alcohol – abstain or avoid excessive use
8. Immunise to pneumococcal and influenza
9. Stop smoking and taking recreational drugs
10. Recognise changes in signs and symptoms

THINK CAUSE
treat cause



Other:
IHD
Pericardial effusions
Massive PE
Myocarditis
ETOHx

What do you understand?

1. Which of the following is correct (could be more than one answer):

A patient with uncontrolled hypertension presents to you with shortness of breath. You do not find any clinical signs of cardiac failure. Her sats is 100 % on room air.

a) You can consider the cost-benefit of doing a pro BNP rule out cardiac failure or confirm it

b) You think it is most likely systolic cardiac failure

c) The treatment will consist of an ACE – inhibitor/ARB, a loop diuretic and a b-blocker

d) You can try and do a cardiac ultrasound to determine the ejection fraction in conjunction with telemedical aid

What do you understand?

2. A hypertensive patient presents to you with clear fluid overload. He has peripheral oedema to the knees, a raised JVP. He is not desaturating. Which of the following is correct?

a) You can treat him as an outpatient as the hospital is under bed pressure

b) You can start him on ivi diuresis on the same dose of his chronic diuretic use

c) You will continue the same dose intravenous diuresis until the peripheral oedema resolve

d) You can switch over to oral furosemide in the ward fairly quickly

e) If the patient does not respond to intravenous loop diuretics, hydrochlorotiazide can be added

f) This patient will need ACE-inhibitor, loop diuretics and carvedilol (slowly uptritrated when no creps on chest)

What do you understand

3. Which of the following statements regarding the management of cardiac failure is true:

a) Add spironolactone for all patients with systolic or HFrEF with class III or IV heartfailure already on an ACE-inhibitor/ARB

b) Add digoxin to all patients who is still symptomatic with HFrEF on ACE-Inhibitor/ARB and B blocker, loop diuretic and spironolactone. However not if patient is in renal failure, elderly, low body mass index or hypokalaemic.

c) Carvedilol is contraindicated in a COAD patients with systolic HFrEF

d) Carvedilol can be safely used in a patient with Asthma and systolic HFrEF

e) The optimum treatment of a patient with Right sided isolated heart failure involves a diuretic, beta blocker and ACE-inhibitor

OUR GOAL