B.E.A.T Heart Failure

- The Growing Burden of HF
- The 25in25 initiative
- Anatomy and pathophysiology
- Evidence Based Medical management
- Help Beat HF

Together we can turn the tide on this life limiting condition.

Heart Failure is a major health problem:

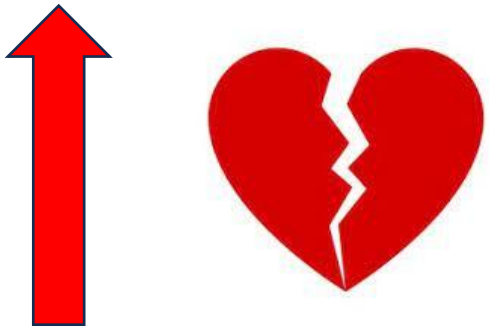
Prevalence:

> 920,000 living with HF[1,2]

Incidence:

>200,000 new diagnosis each year[1,2]

Heart Failure cases projected to double by 2040 :



92%

BY 2040

Total number to increase by approx. 1 million

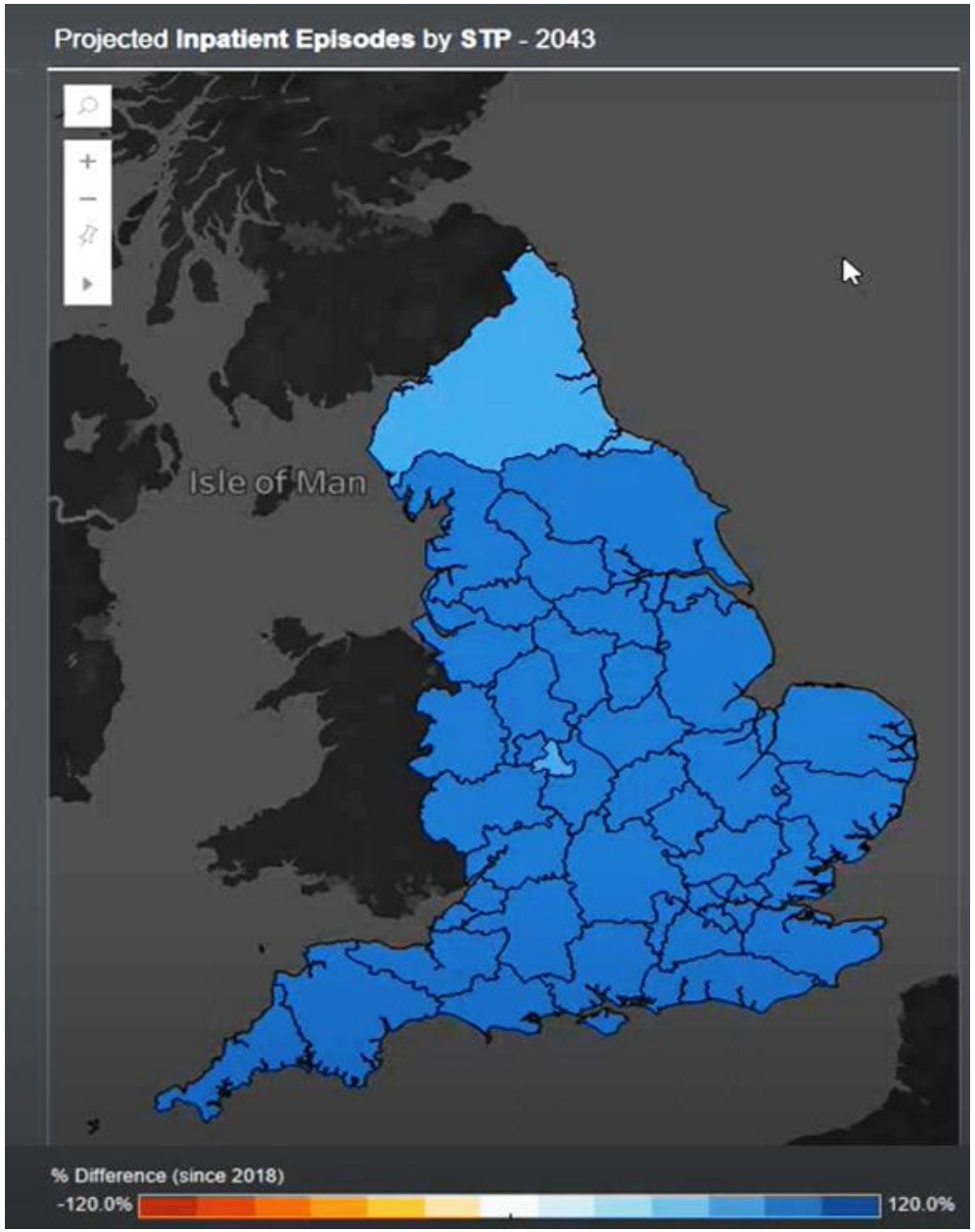
https://reader.health.org.uk/projected-patterns-of-illness-in-england/executive-summary#people-are-living-longer-but-with-major-illness accessed 19/04/24

- 1. Conrad N et al. Lancet 2018;391:572–580; 2. British Heart Foundation. UK Factsheet. 2020. Available at: https://www.bhf.org.uk/what-we-do/our-research/heart-statistics. Accessed February 2020.

Together we can turn the tide on this life limiting condition.

25 IN 25

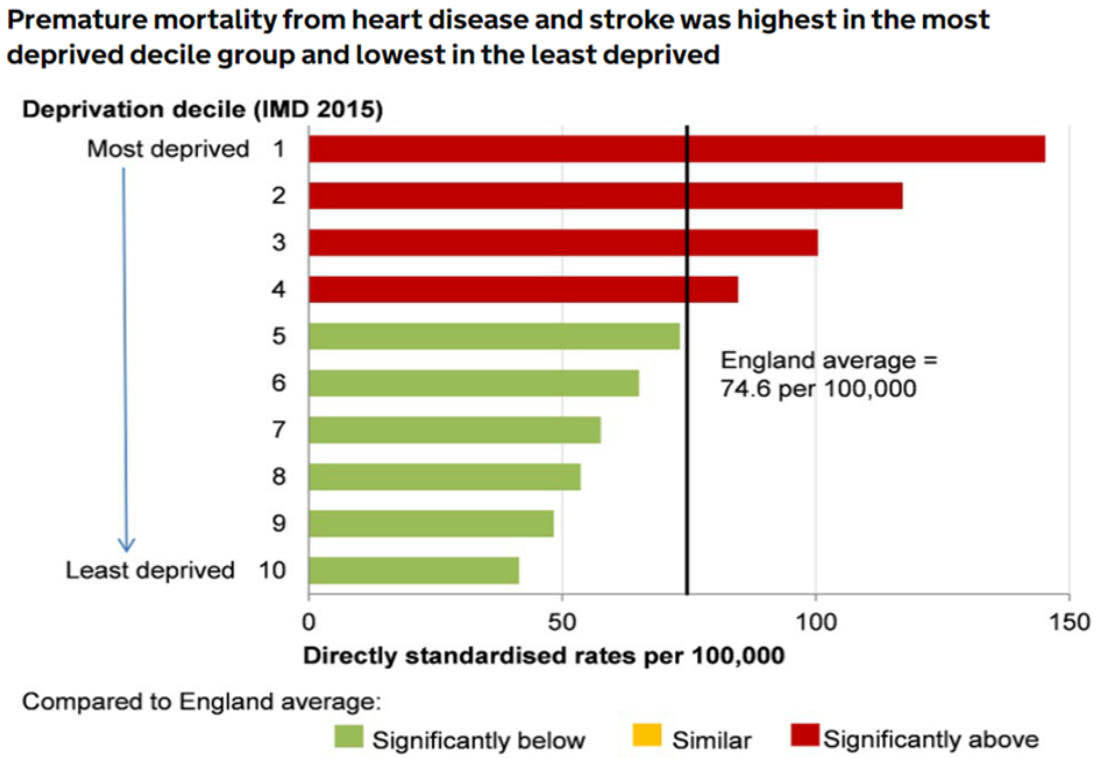
Projected 100% more cardiology inpatients by 2043: HEE



Together we can turn the tide on this life limiting condition.

Why is this happening?

- Baby boom peaked in 1965 in UK
- In 20 years baby boomers approaching 80 years
- Deprivation has a direct impact on premature mortality
- 9 of the 10 most deprived District Wards in Cambridgeshire are in Peterborough



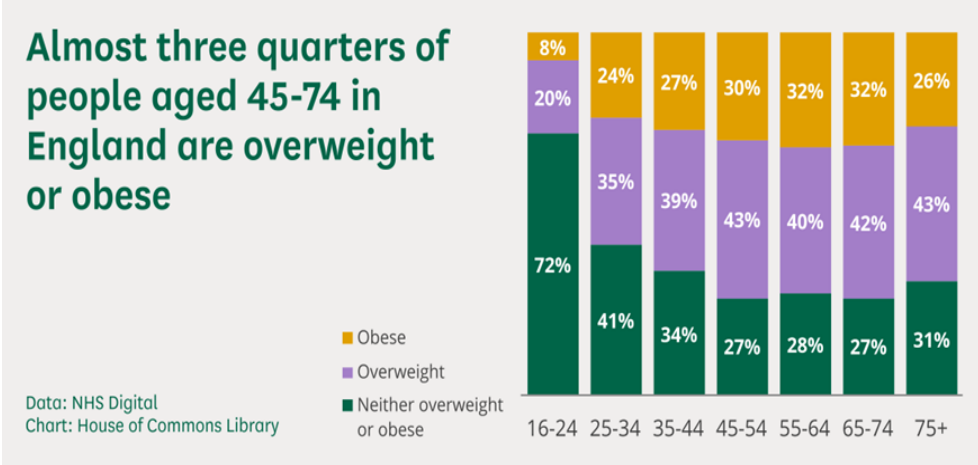
https://cambridgeshireinsight.org.uk/wp-content/uploads/2019/10/Cambridgeshire-Summary-Report-for-IMD-2019-1.0.pdf accessed 20/04/2024

Why is this happening?

Obesity and diabetes have overtaken CV disease as the major risk factors for developing HF

UK obesity rates are

highest in Europe:



Obesity is the biggest risk factor in T2DM for developing HF:

https://modernheartandvascular.com/top-risk-factors-for-congestive-heart-failure/accessed 19/04/24
OECD, organisation fir Economic Co-operation and Development,
Rashani A et al. Risk factors, mortality and cardiovascular outcomes in patients with T2DM. N Engl J Med 2018;379:633-644

Survival in heart failure is worse than in some common cancers[1]

In a primary care-based cohort study in Scotland with 10-year follow-up, the survival of HF patients was compared with that of patients with common cancers

5-year survival

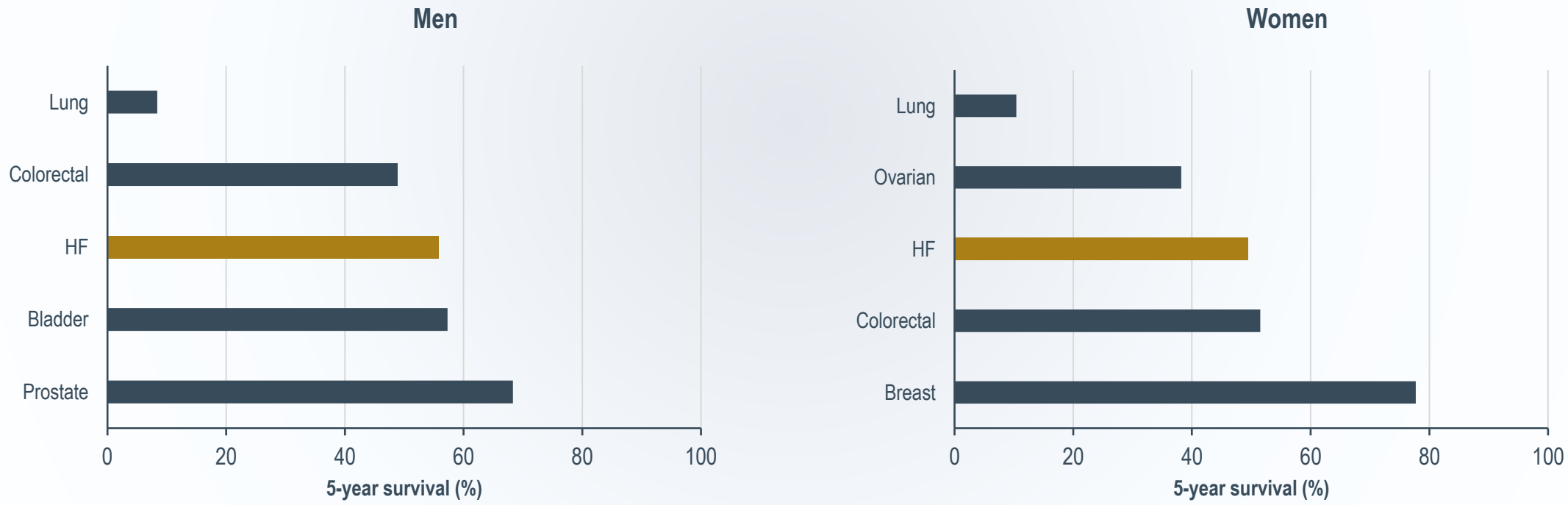


Figure adapted from Mamas MA et al. Eur J Heart Fail 2017;19:1095–1104.
HF, heart failure.

1. Mamas MA et al. Eur J Heart Fail 2017;19:1095–1104.

British Society for Heart Failure
25in25 Initiative

In UK:

- 80% of HF is diagnosed in hospital, (BMJ 2019) of those
- 40% had symptoms that should have triggered an earlier assessment in primary care in the months prior to admission
- Approx 400,000 people are living with HF currently undetected, undiagnosed and missing out on life-preserving treatment[1]
- Deferring heart failure treatment for *one month*, even in low-risk patients, increases the risk of death by 1% (Zaman, 2017)

1. National Institute Clinical Effectiveness Impact CVD Management Report (2021)

Together we can turn the tide on this life limiting condition.

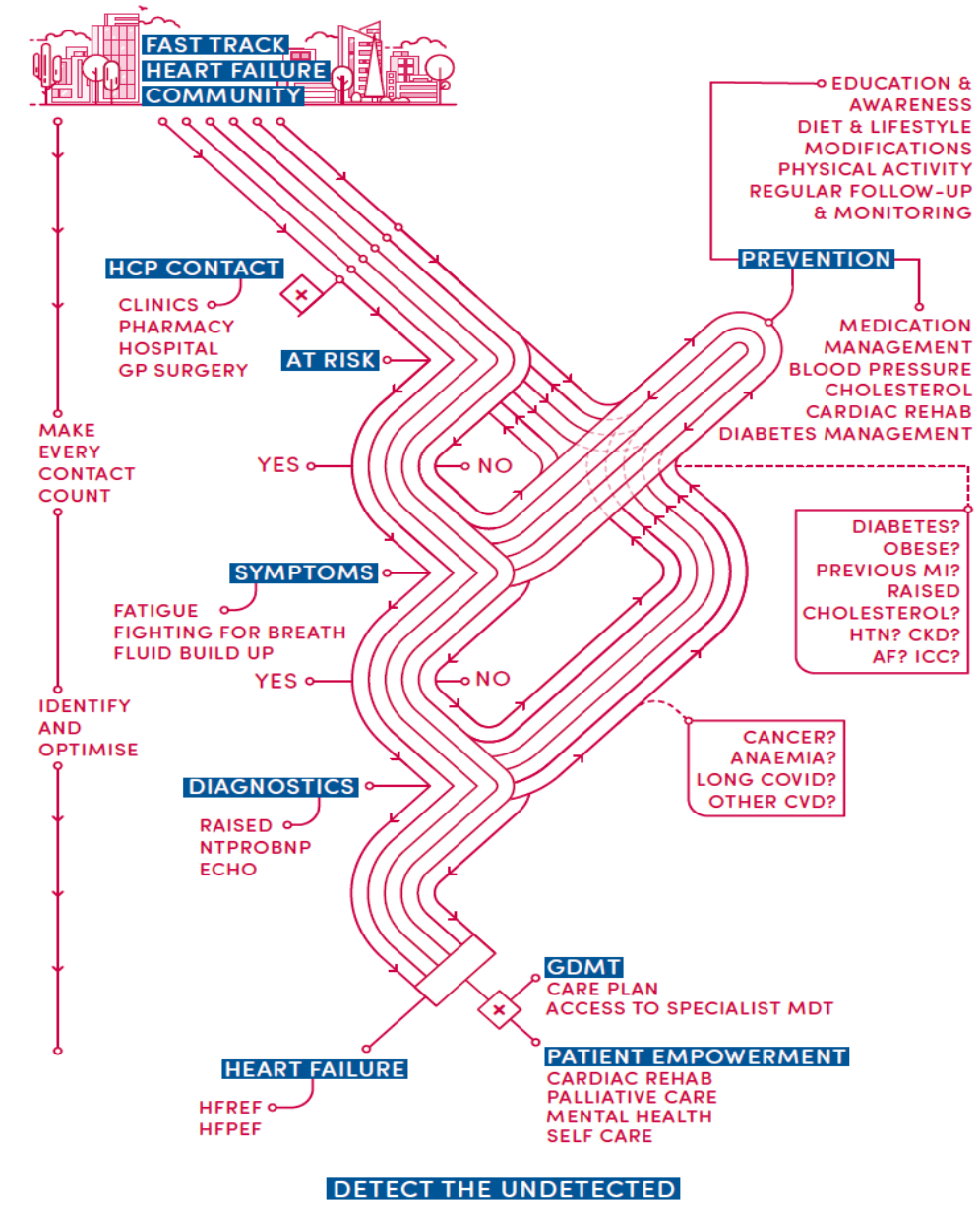
British Society for Heart Failure

25in25 Initiative:

Changing the trajectory of heart failure: reduce mortality from HF in the first year of diagnosis by 25%in 25 years

This would mean 5 fewer deaths for every 100 patients newly diagnosed with heart failure every year, translating to over 10,000 lives saved per year

Together we can turn the tide on this life limiting condition.



Prevention strategies

Identifying those at risk

Early and accurate diagnosis

Appropriate treatment

DETECTING THE UNDETECTED:

Heart Failure is Treatable

Together we can turn the tide on this life limiting condition.

BSH 25in25 initiative:
A population health approach to reducing HF mortality

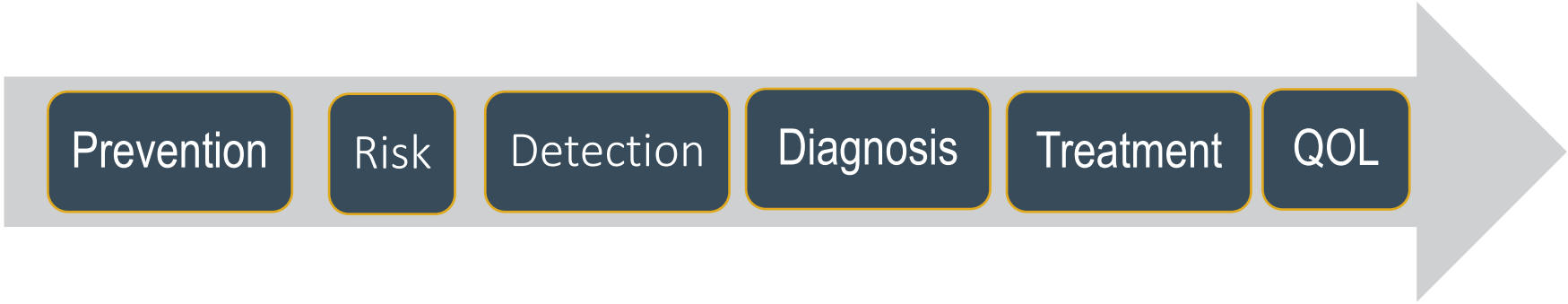
Bringing together organisations with a vested interest in improving CV outcomes and creating a pact to reduce HF mortality

BSH has 3 workstreams:

1)Quality Improvement: Identifying adaptable projects that can rolled out across the UK, that can demonstrate positive change in outcomes/ performance

2) Collaboration: Working with likeminded organisations to drive the 25in25 message/ brand and encourage further adoption

3) Lobbying: Campaigning for policy makers to address the challenges and achieve a greater investment in HF.



Together we can turn the tide on this life limiting condition.

What is Heart failure?
Pathophysiology of heart failure is complex:

- Heart failure is caused by a structural and / or functional cardiac abnormality that results in **reduced cardiac output** and / or **elevated intracardiac pressures** at rest or during stress[1]
- In patients with HFrEF, myocardial injury results in maladaptive changes in myocytes and extracellular matrix, which leads to **pathological ventricle remodelling** with **ventricular dilatation** and **impaired ventricular contractility**[2]

Heart failure is defined by the European Society of Cardiology as:

*"a **clinical syndrome** characterised by typical **symptoms** (e.g. breathlessness, ankle swelling and fatigue) that may be accompanied by **signs** (e.g. elevated jugular venous pressure, pulmonary crackles and peripheral oedema) caused by a **structural and / or functional cardiac abnormality**, resulting in a **reduced cardiac output** and / or elevated intracardiac pressures at rest or during stress"*[3]

1. National Health Service. Overview: heart failure. Available at: https://www.nhs.uk/conditions/heart-failure/. Accessed February 2020; 2. Ponikowski P et al. Eur Heart J 2016;37:2129–2200.

Symptoms and signs of heart failure:

Common symptoms of HF include[1]*:

- Shortness of breath / dyspnoea
- Orthopnoea
- Paroxysmal nocturnal dyspnoea
- Reduced exercise tolerance
- Fatigue
- Ankle swelling / oedema

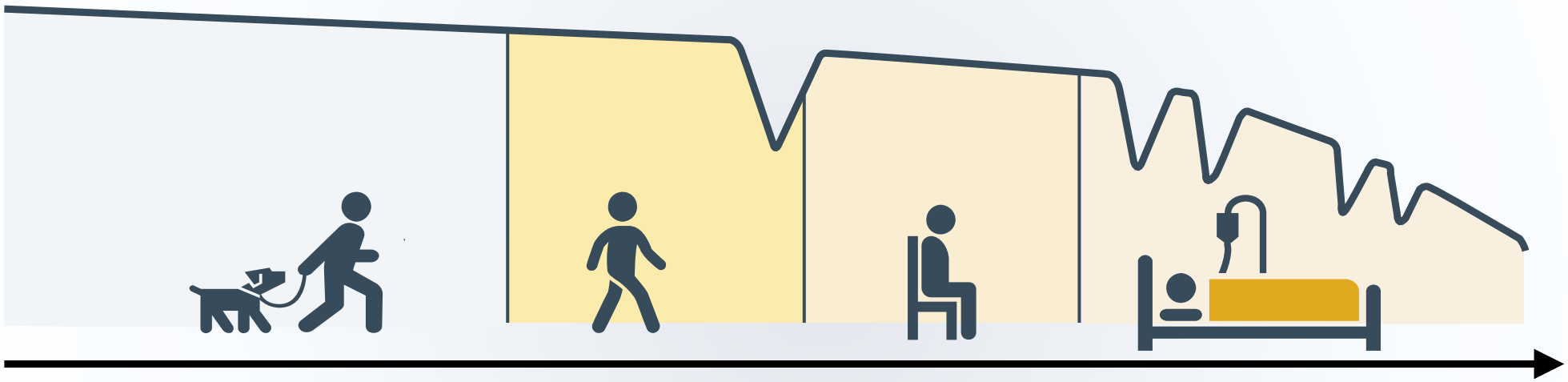
Common signs of HF include[1]*:

- Elevated jugular venous pressure
- Third heart sound (gallop rhythm)
- Laterally displaced apical impulse
- Pulmonary crepitations
- Peripheral oedema

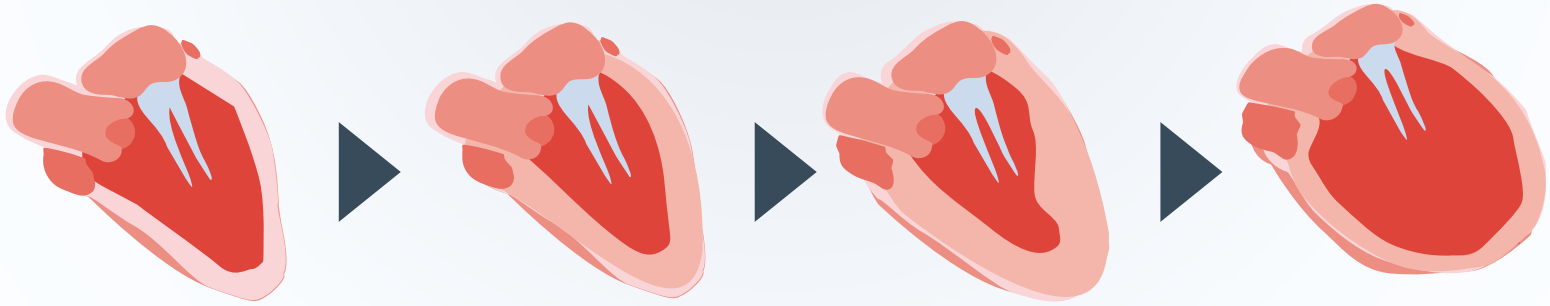
*This list is not exhaustive.
HF, heart failure.

1. Ponikowski P et al. Eur Heart J 2016;37:2129–2200.

Cardiac remodelling is associated with abnormal neurohormonal regulation, culminating in heart failure with high morbidity and mortality[1]



- **Myocardial remodelling is central in the pathophysiology of advancing of HF.**[2]



1. Wang Y et al. Am Heart Assoc 2019;8(13):e012272. 2. Konstam et al. JACC Cardiovasc Imaging. 2011; 4: 98-108

How is heart failure defined?

HF can be defined, based on LVEF, as[1]:

- **HF with reduced ejection fraction (HFrEF):** previously known as systolic HF, with **LVEF <40%**
- **HF with mildly reduced ejection fraction (HFmrEF**): LVEF 40–49%*
- **HF with preserved ejection fraction (HFpEF):** previously known as diastolic HF, with **LVEF ≥50%**

In the UK, the most common type of HF is HFrEF due to impaired contraction of the left ventricle[2]

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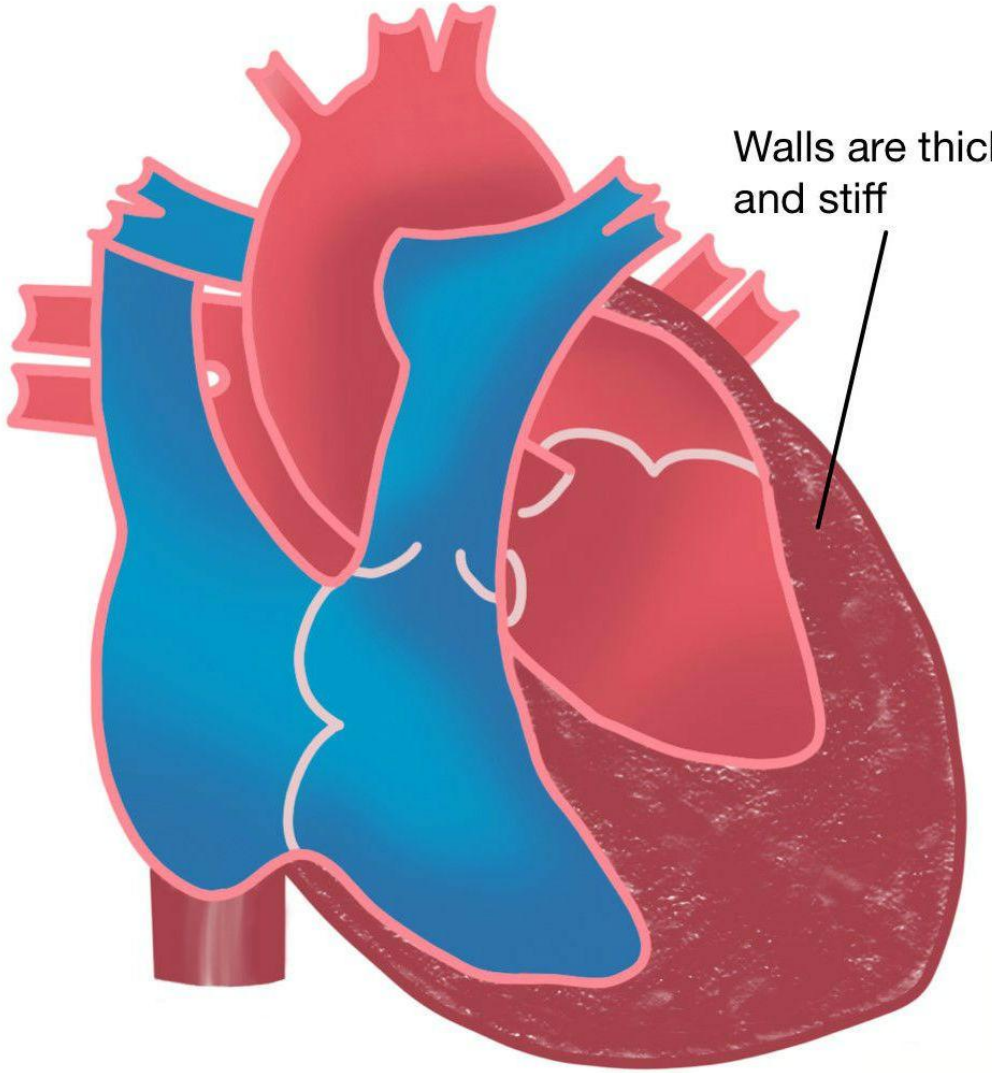
*HFmrEF is not recognised in the current National Institute for Health and Care Excellence guidance as it has not been fully clinically validated and remains the topic of further research.[4]

HF, heart failure; LVEF, left ventricular ejection fraction.

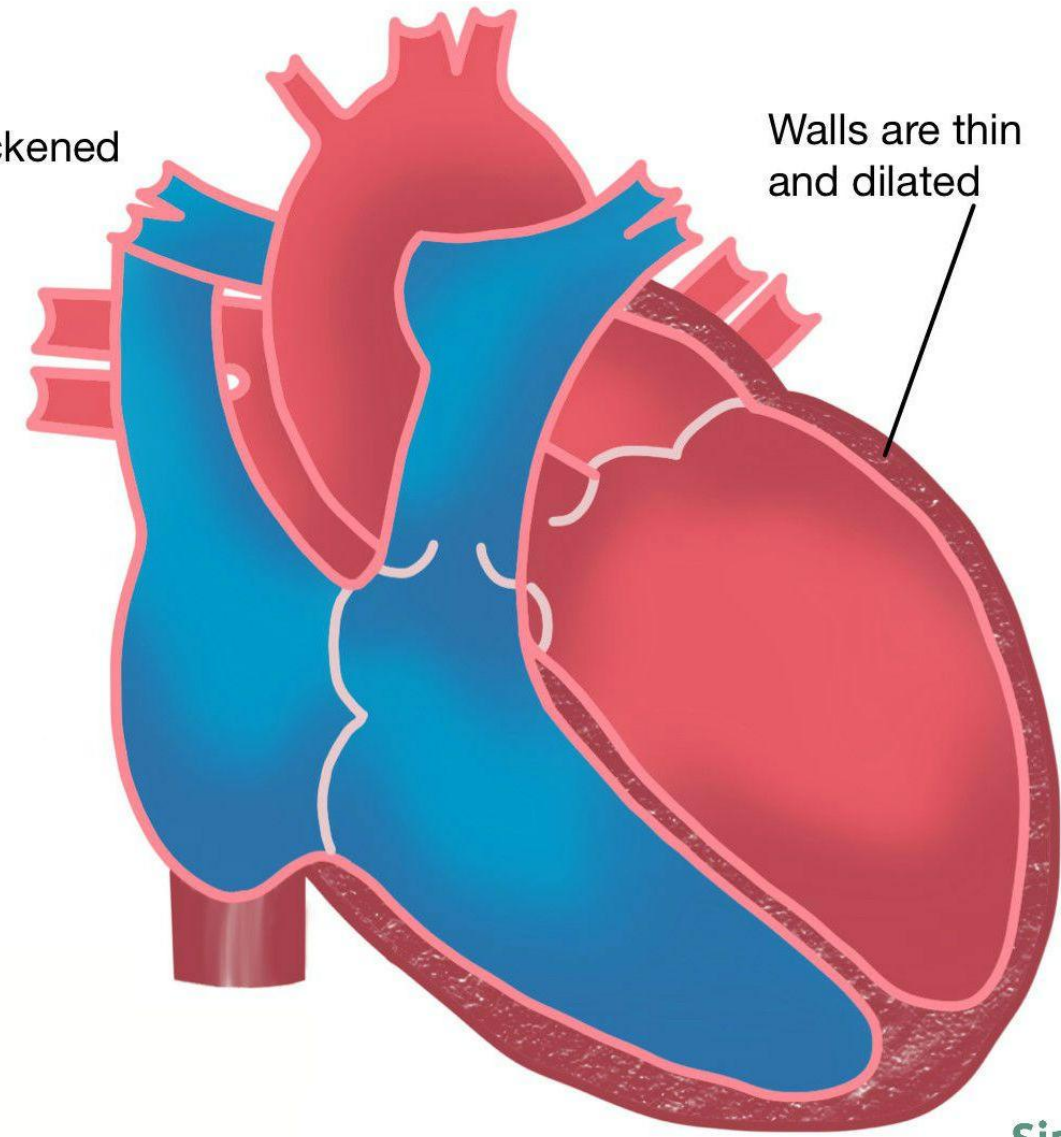
1. Ponikowski P et al. Eur Heart J 2016;37:2129–2200; 2. National Institute for Cardiovascular Outcomes Research. National Heart Failure Audit 2019 Summary Report (2017/2018 data). Available at: https://www.nicor.org.uk/wp-content/uploads/2019/09/Heart-Failure-2019-Report-final.pdf. Accessed February 2020; 3. Entresto Summary of Product Characteristics. Available at: https://www.medicines.org.uk/emc/product/7751/smpc. Accessed February 2020; 4. National Institute for Health and Care Excellence. Chronic heart failure in adults: diagnosis and management. NG106. 2018. Available at: https://www.nice.org.uk/guidance/ng106. Accessed February 2020.

Heart failure with preserved ejection fraction
(Diastolic heart failure)

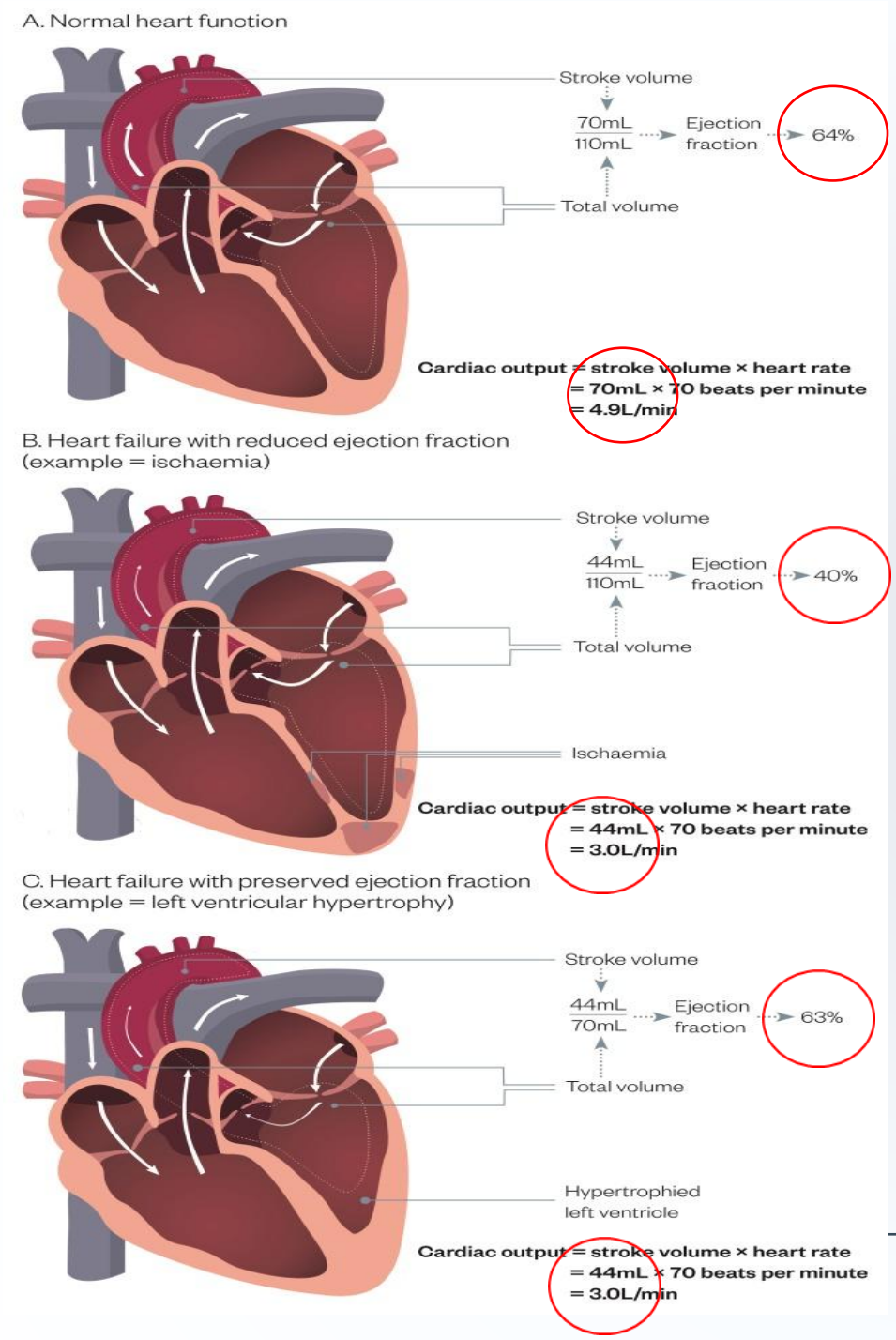


Heart struggles to fill

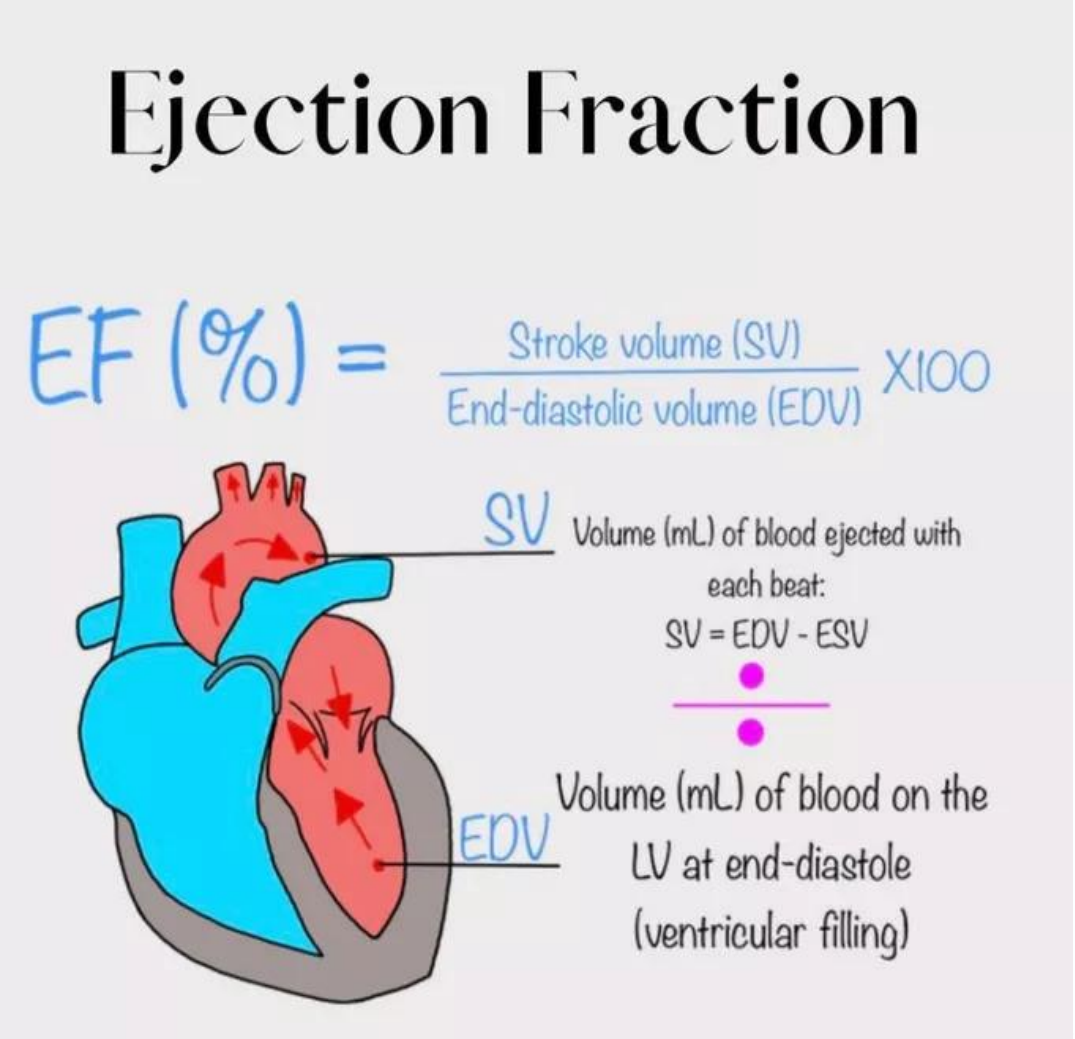
Heart failure with reduced ejection fraction
(Systolic heart failure)



Heart struggles to pump

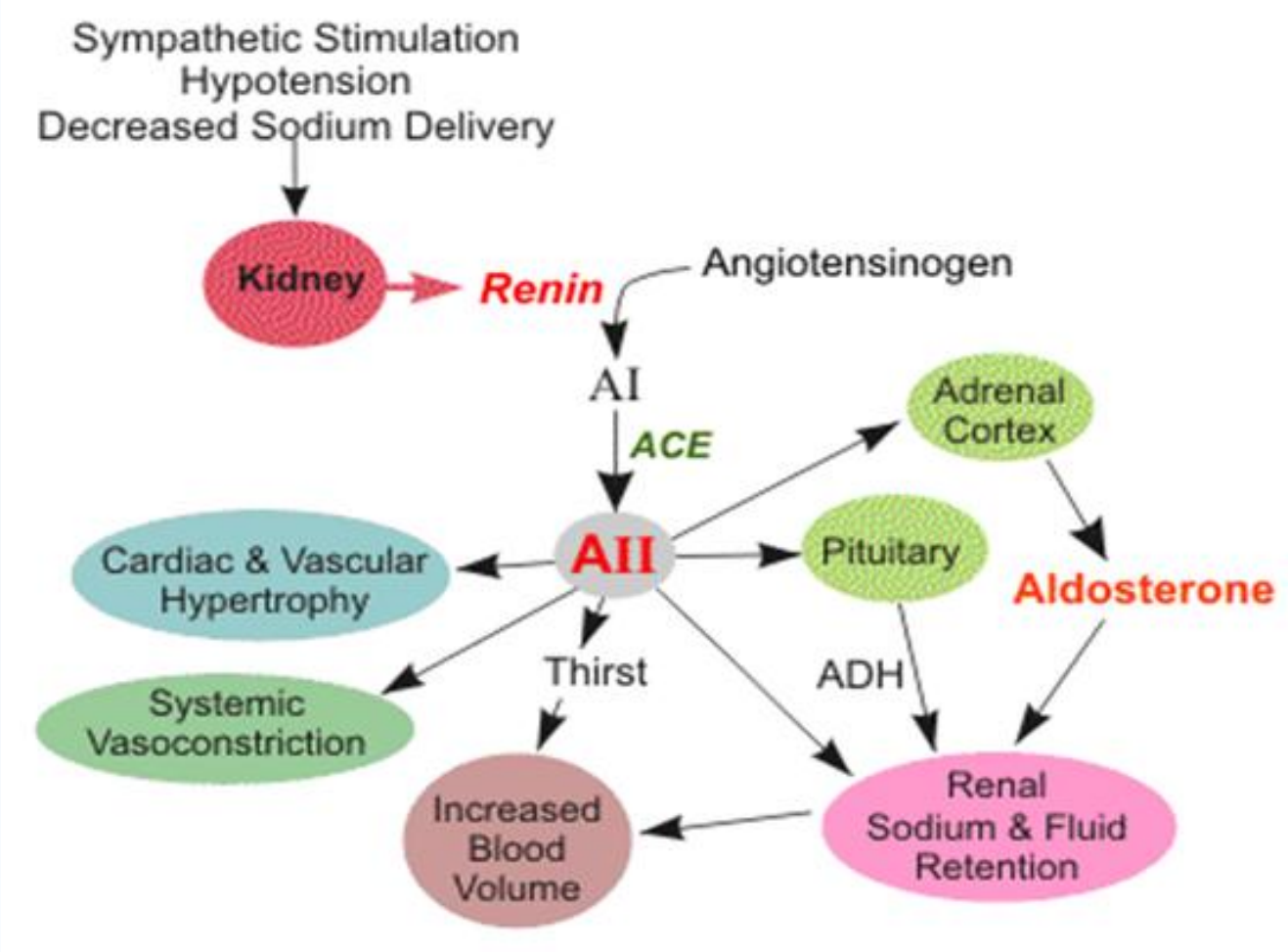


What do we mean by Ejection Fraction?

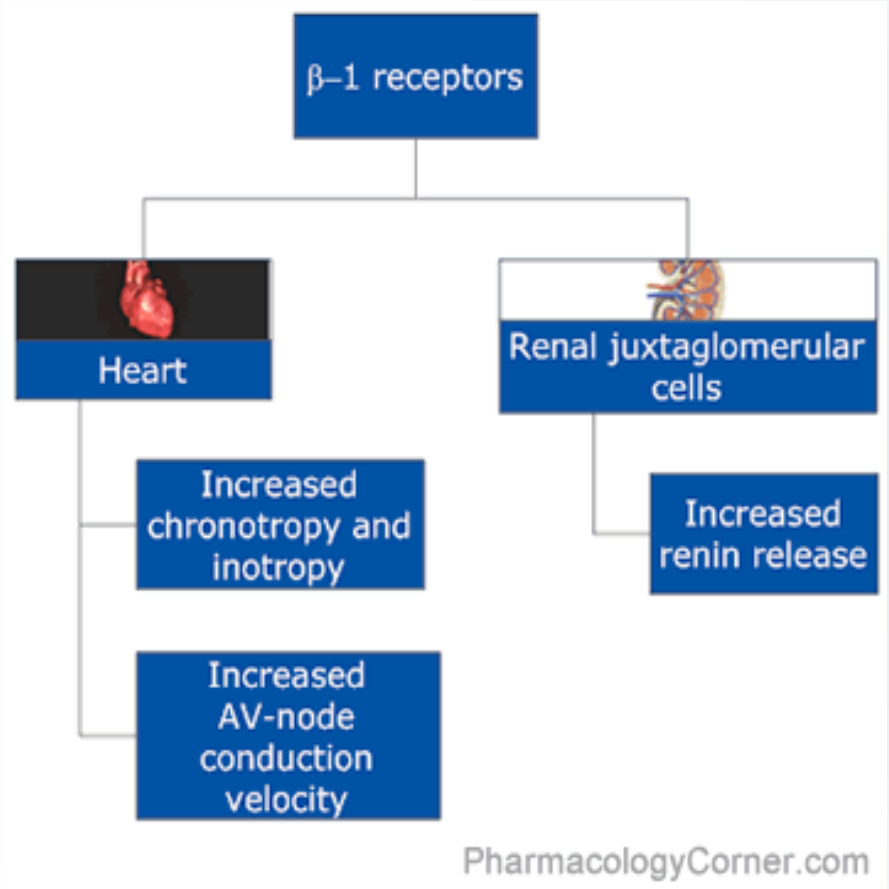


https://pharmaceutical-journal.com/article/ld/pathophysiology-and-management-of-heart-failure accessed 23/09/22

Renin Angiotensin Aldosterone System



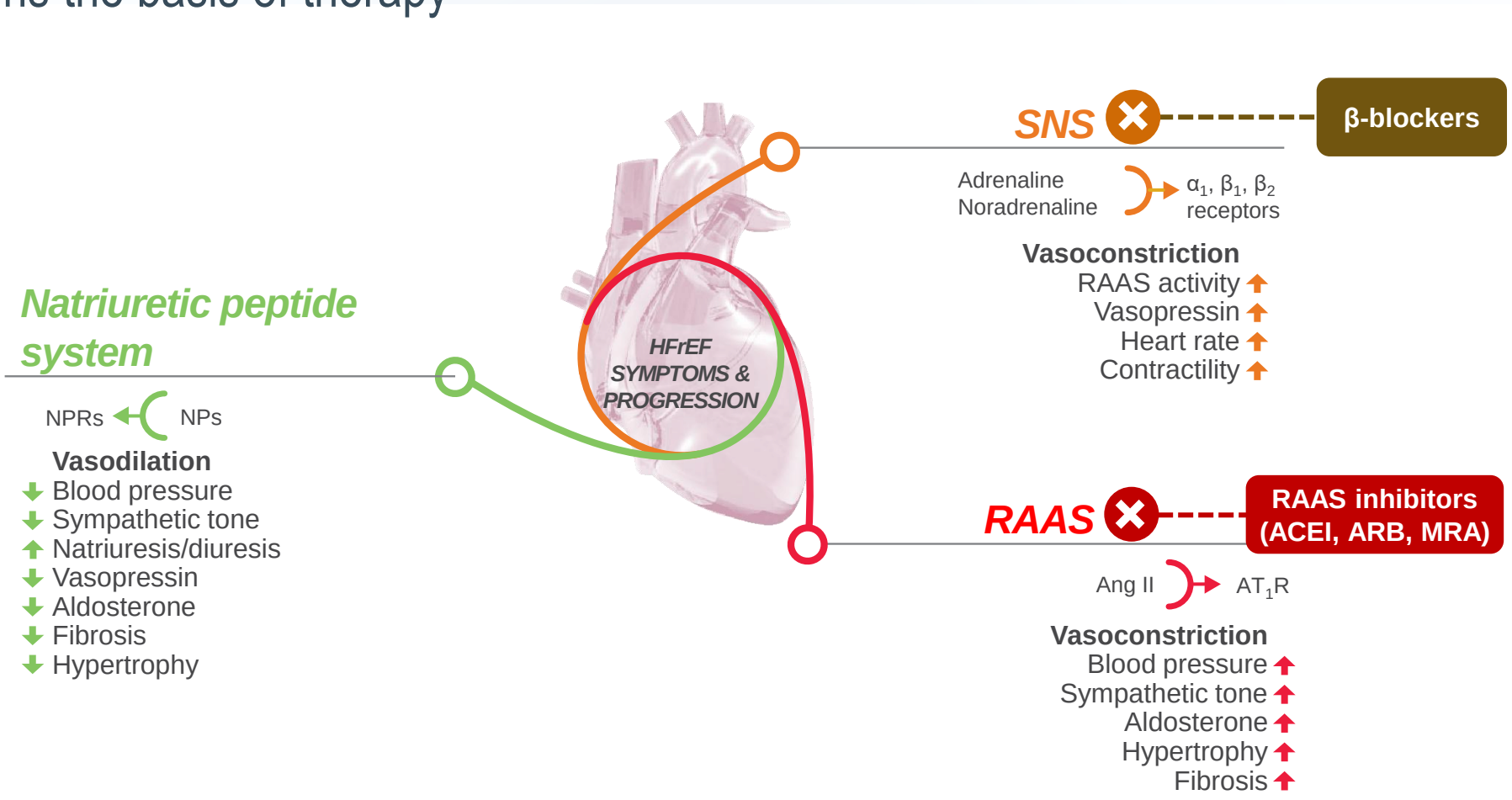
Sympathetic Nervous System: Beta-adrenergic receptor sites of action (stimulated by epinephrine or norepinephrine)



Betablockers primarily block the ß-1receptor sites reducing heart rate and renin release

Some betablockers eg Carvedilol have some ß-2 receptor properties and can cause smooth muscle constriction

Overactivation of the RAAS and SNS is detrimental in HFrEF and underpins the basis of therapy



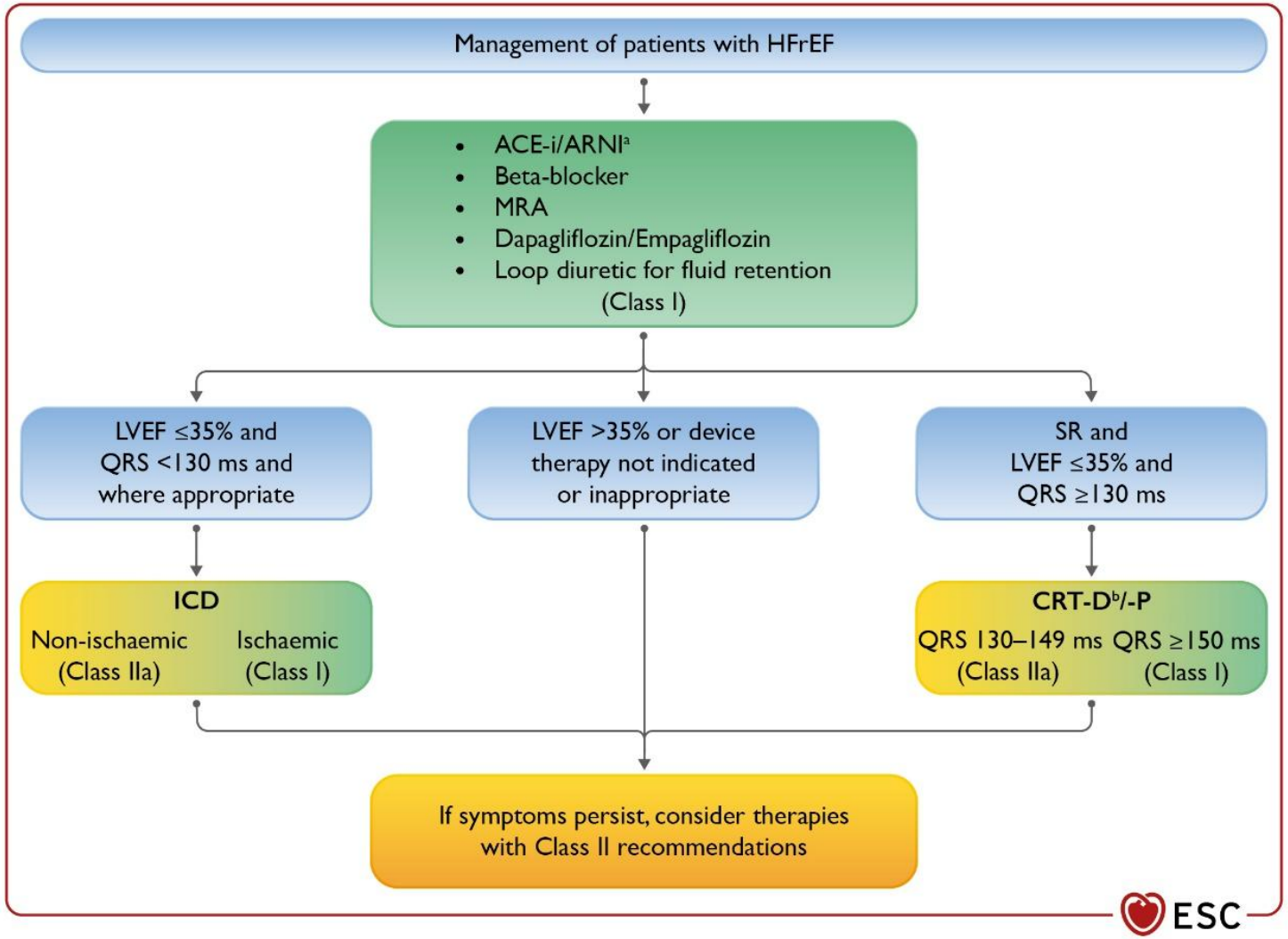
- The crucial importance of the RAAS is supported by the beneficial effects of ACEIs, ARBs and MRAs[1]
- Benefits of β-blockers indicate that the SNS also plays a key role[1]
- The benefits of Natriuretic peptides have lead to the development of Neprilysin inhibitor

RAAS = renin aldosterone angiotensin system
SNS = sympathetic nervous system
ACEI = angiotensin-converting-enzyme inhibitor
ARB = angiotensin receptor blocker
MRA = mineralocorticoid receptor antagonist

1. McMurray et al. Eur Heart J 2012;33:1787–847

Figure references: Levin et al. N Engl J Med 1998;339:321–8; Nathisuwan & Talbert. Pharmacotherapy 2002;22:27–42; Kemp & Conte. Cardiovascular Pathology 2012;365–371; Schrier & Abraham. N Engl J Med 1999;341:577–85

Therapeutic algorithm of Class I Therapy Indications for a patient with heart failure with reduced ejection fraction



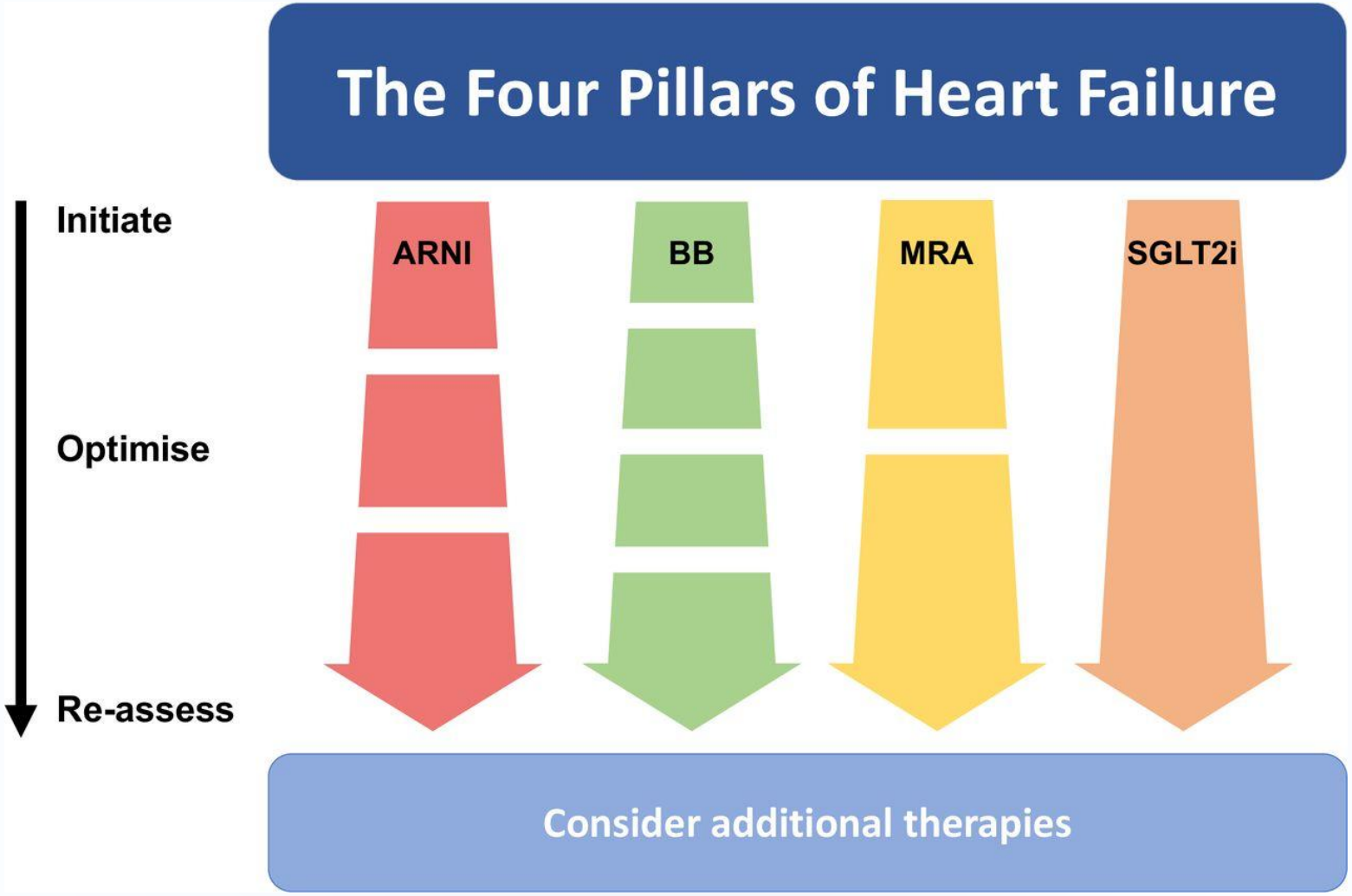
ACE-I = angiotensin-converting enzyme inhibitor; ARNI = angiotensin receptor-neprilysin inhibitor; CRT-D = cardiac resynchronization therapy with defibrillator; CRT-P = cardiac resynchronization therapy pacemaker; ICD = implantable cardioverter-defibrillator; HFrEF = heart failure with reduced ejection fraction; MRA = mineralocorticoid receptor antagonist; QRS = Q, R, and S waves of an ECG; SR = sinus rhythm.

[a]As a replacement for ACE-I.

[b]Where appropriate. Class I=green. Class IIa=Yellow.

2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure (European Heart Journal 2021 – doi:10.1093/eurheartj/ehab368)

Initiation and optimisation of the Four Pillars of Heart Failure.



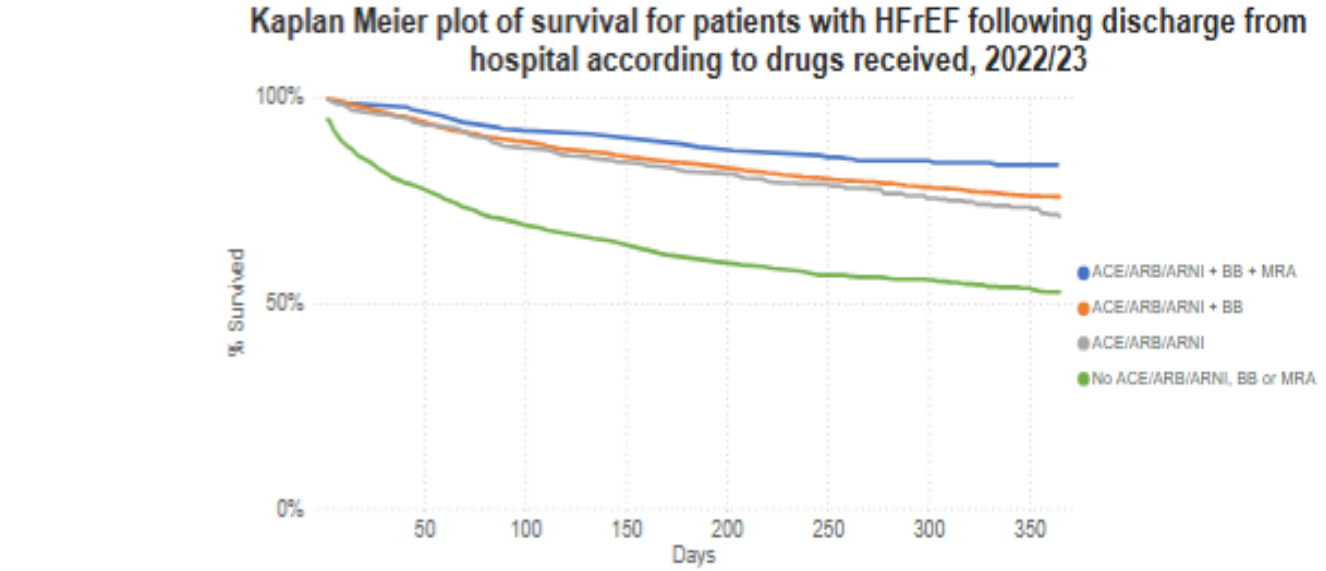
Sam Straw et al. Open Heart 2021;8:e001585

2024 Annual Summary Report

GDMT

- 55 year old gains 6.3 years
- 80 year old gains 2.3 years

1-year survival much better for those with HFrEF discharged on all three classes of disease-modifying drugs



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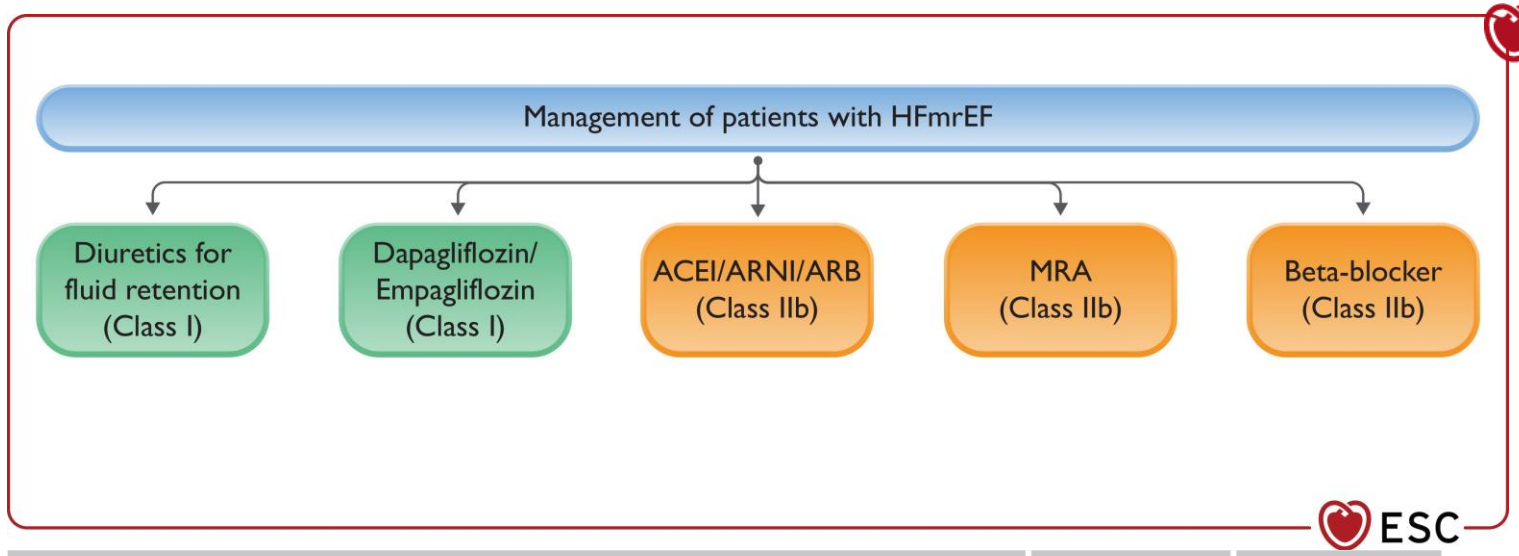
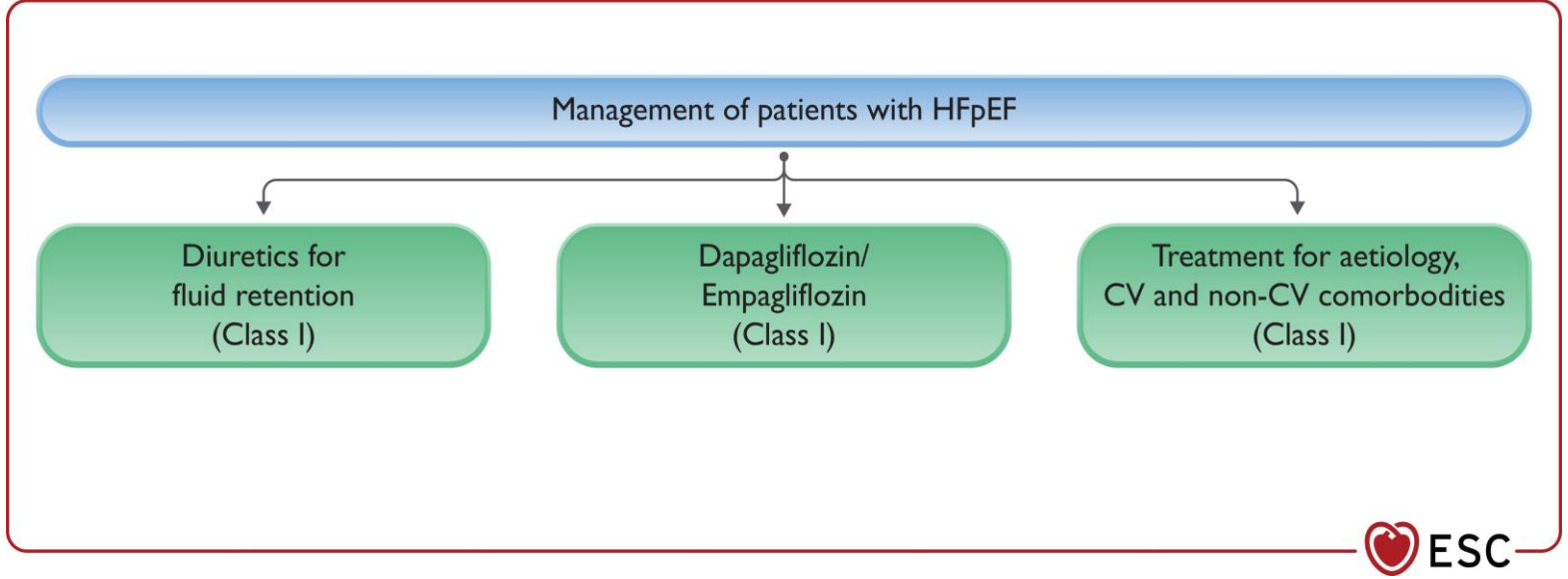
NICOR NHFA report 2024: accessed 11/01/2025



Recommendation	Class[a]	Level[b]
An SGLT2 inhibitor (dapagliflozin or empagliflozin) is recommended in patients with HFmrEF to reduce the risk of HF hospitalization or CV death.[c] [6,8]	I	A

CV, cardiovascular; HF, heart failure; HFmrEF, heart failure with mildly reduced ejection fraction; SGLT2, sodium–glucose co-transporter 2.
[a]Class of recommendation.
[b]Level of evidence.
[c]This recommendation is based on the reduction of the primary composite endpoint used in the EMPEROR-Preserved and DELIVER trials and in a meta-analysis. However, it should be noted that there was a significant reduction only in HF hospitalizations and no reduction in CV death.

Eur Heart J, Volume 44, Issue 37, 1 October 2023, Pages 3627–3639, https://doi.org/10.1093/eurheartj/ehad195



ESC European Society of Cardiology

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Eur Heart J, Volume 44, Issue 37, 1 October 2023, Pages 3627–3639, https://doi.org/10.1093/eurheartj/ehad195

Pathway for the Optimisation of Medicines in Heart Failure with Reduced Ejection Fraction.

East of England Cardiac Network

Cardiac rehabilitation should be offered to all HF patients unless unstable

Vaccinations for HF patients – offer annual influenza vaccine, pneumococcal vaccine (required once), Covid-19 Vaccine as per current recommendations

Patients with AF and HFrEF

ALL PEOPLE WITH LVSD AND AF SHOULD BE ANTICOAGULATED

If HR > 75bpm and unless contraindicated a b-blocker should be commenced and up titrated to the maximum tolerated dose. If HR remains over 60bpm and/or a b-blocker is contraindicated consider low dose digoxin.

If **iron deficiency** is established— defined as either
1] Serum Ferritin <100mg/L or
2] Serum Ferritin 100–299mg/L AND Transferrin Saturation <20%
— consider specialist referral for intravenous iron therapy in accordance with locally developed guidance/pathways. (ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Eur Heart J (2016) Jul 14;37(27):2096, https://www.gov.uk/drug-safety-update/ferric-carboxymaltose-ferinject-risk-of-symptomatic-hypophosphataemia-leading-to-osteomalacia-and-fractures)

Patient with symptomatic HFrEF

The East of England Cardiac Network recommend initiation of all the agents in the four classes below over four weeks from HFrEF diagnosis or at the next medicines optimisation review for patients with established HFrEF in primary care. This guidance is on the basis that each component is disease modifying and the effects are additive in terms of both morbidity and mortality with evidence illustrating benefits very early on. Optimise treatment by sequential up-titrations to evidence-based doses and follow up as appropriate.

Start at the first opportunity Initiate **ACE-I or ARB**, up titrate to maximum tolerated dose or dose used in clinical trial (table 1) (NICE NG106)

Start at the first opportunity Initiate **B-blocker**, up titrate to maximum tolerated dose or dose used in clinical trial (table 3) (NICE NG106)

Start at the first opportunity Initiate **SGLT2 inhibitor** licensed for HF treatment Dapagliflozin 10mg or Empagliflozin 10mg (table 4). (NICE TA 679, NICE TA 773)

Start at the first opportunity Initiate **MRA** (table 5) Spironolactone or Eplerenone (NICE NG106)

ASAP Refer to HF Specialist to Replace ACEI (or ARB) with **sacubitril – valsartan (Entresto)** (table 2) if ejection fraction <35% and up titrate to maximum tolerated dose (NICE TA388)

If contraindicated to ACE-I/ARB/ARNI consider hydralazine with nitrate (NICE NG106)

Use **loop diuretic** (Furosemide or Bumetanide) (table 6) to reduce symptoms of fluid overload, Educate patient on fluid restriction, morning weight and adjusting loop diuretic dose accordingly. (NG106)

Is the patient still symptomatic?
Specialist re-assessment. If symptoms persist despite first-line treatment, seek specialist advice and consider one or more of the following. The treatments may be combined if indicated and not contraindicated

Add **Ivabradine** for patients in SR (can not be used in AF) with heart rate >75 and ejection fraction <35% (NICE TA267)

Add **Hydralazine** and **Nitrate** (especially if of African-Caribbean descent) (NICE NG106)

Add **Digoxin** for elderly patients with sinus rhythm who are relatively inactive (NICE NG106)

Evaluate need for CRT-P or CRT-D, if QRS duration ≥ 120 msec (NICE TA 314)

Is patient still symptomatic ?

Patient deteriorating despite optimal treatment and effective management of underlying medical condition(s) – discuss palliative care needs with the specialist HF MDT, patient and family should have access to professionals with palliative care skills.

Specialist referral to consider advanced therapies (LVAD or Heart Transplant)

Consider use of Thiazide diuretic in resistant oedema in conjunction with loop (table 6)

The manufacturer's summary of product characteristics (SPC) and the most current online edition of the British National Formulary should be consulted for full information on contraindications, warnings, side effects and drug interactions. Consult the SPS Drug monitoring site for monitoring advice.

TABLE 1. ACE-I /ARB

- Check **baseline U&Es and BP, then increase the dose in 2 weeks' intervals (up to max tolerated dose) checking U&Es and BP after each increase** (double doses increases).
- Continue to the next step if systolic BP >100mmHg, no symptoms of hypotension, serum potassium < 5.5 mmol/L, serum creatinine < 200ummol/L or if increase <30% of the baseline and eGFR> 30 ml/min. Otherwise seek specialist advice. If serum creatinine increases by > 30% above baseline (equivalent to fall in eGFR > 25%), reduce or stop ACE-I / ARB unless alternative cause is found.
- Don't start ACE-I in people with Chronic kidney disease (CKD) with a pre-treatment serum potassium concentration > 5.0 mmol/L.
- A rise of serum potassium to ≥5.5 mmol/L should prompt discontinuation of the ACE-inhibitor or reducing to previous tolerated dose and seeking specialist advice. ACE –I/ARB should be withdrawn in all patients with serum K+ ≥ 6 mmol/l who do not meet the criteria for treatment with novel potassium binders.

- **Ramipril, starting dose 1.25mg OD, target dose 10mg OD (or 5mg BD).**
- **Lisinopril, starting dose 2.5mg OD – 5mg OD, target dose up to 35mg OD.**
- **Enalapril, starting dose 2.5mg BD, target dose up to 20mg BD.**
- **Perindopril, starting dose 2mg OD, target dose 4mg OD.**

- **Losartan , starting dose 12.5mg OD, target Losartan 100mg OD, up to maximum 150mg OD.**
- **Candesartan, starting dose 4mg OD, target dose Candesartan 32mg OD.**
- **Valsartan, starting dose 40mg BD, target dose Valsartan 160mg BD.**

- If symptomatic hypotension or Creat≥200 µmol/L or increase >50% from baseline, or eGFR <30ml/min/1.73m2 - consider stopping concomitant nephrotoxic drugs or reducing loop diuretics if no congestion. Recheck U&Es in 1 week.
- If U&Es return to acceptable levels continue up titration.
- If problems persist half the dose of ACE-I and recheck U&Es 1 week. If still no improvement stop ACE-I /ARB.
- STOP ACE-I / ARB if K >6.0 or serum creatinine >300 µmol/L or increase >100% from baseline. Consider seeking specialist advice/cardiorenal MDT.
- SICK DAY RULES - advise patient to stop for 1 -2 days until the patient recovers.

PATIENTS WITH IMPAIRED RENAL FUNCTION
If eGFR is 30 to 45 ml/min/1.73 m2, **consider lower doses or slower titration of ACEI/ARBs/ARNI or MRAs**

TABLE 2. ARNI – Sacubitril/Valsartan.

- Requires specialist initiation.
- A washout period of at least 36 h after ACE-I therapy is required in order to minimize the risk of angioedema.
- Increase the dose in 2-4 weeks' intervals, checking U&Es and BP after each increase. For patients naïve to previous ACE inhibitor or ARB therapy or on low-dose therapy (equivalent to <10 mg enalapril/day) slow titration – doubling every 3 - 4 weeks is recommended.

- **Start Sacubitril/valsartan 24/26mg BD , if patient on low dose ACE-I/ARB, BP 100 – 110mmHg, eGFR 30 – 60 ml/min.**
- **Starting dose Sacubitril/valsartan 49/51mg BD for patients on target ACE-I/ ARB, BP >110mmHg, eGFR >60 ml/min.**
- **Target dose Sacubitril/valsartan 97/103mg BD.**
- Take advice about initiation or dose increase if:
    - Issues with ACE-I or ARB such as hypotension, allergy, renal decline or angioedema: seek advice do not start Sacubitril/valsartan.
    - Systolic BP <100mmHg or symptoms of hypotension.
    - Cr > 200umol or if increase > 50% from baseline, eGFR ≤ 30 ml/min/1.73m2. A reduction in eGFR up to ≤ 30 mL/min/1.73 m2 is acceptable .
- If K+ rises to >5.5 mmol/L or eGFR lowers to <30 mL/min/1.73 m2, seek specialist advice.
- SICK DAY RULES - advise patient to stop for 1 -2 days until the patient recovers.

TABLE 3. BETA-BLOCKERS

- Check baseline BP and HR, increase the dose in 1 to 4 weeks' intervals (up to max tolerated dose) depending on side effects, BP and HR.
- Continue to the next up titration if systolic BP> 100mmHg and HR> 50bpm (optimal HR 60 bpm in SR, 80-90 bpm in AF).
- Beta-blockers should be initiated in clinically stable (without acute HF), euvolaemic, patients at a low dose and gradually uptitrated to the maximum tolerated dose.

- **Bisoprolol, starting dose 1.25mg OD, target dose Bisoprolol 10mg (increase every 1 to 4 weeks).**
- **Carvedilol, starting dose 3.125mg BD, target dose Carvedilol 25mg BD (50mg BD in patients > 85kg) (increase intervals every 2 weeks).**
- **Nebivolol, starting dose 1.25mg OD, target dose Nebivolol 10mg OD (increase every 1 to 2 weeks).**

- If decrease in heart rate <50bpm or symptomatic hypotension or evidence of respiratory problem but no weight gain. Reduce to previous dose or if extreme discontinue.

TABLE 4. SGLT2

- Initiate on the advice of specialist in heart failure either in hospital, community or by a healthcare professional with specialist interest.

- **Option a, Dapagliflozin 10mg OD** If eGFR≥15 ml/min1.73 m2, If eGFR falls to <15 ml/min do not stop Dapagliflozin. All cause mortality benefit is demonstrated in patients with HFrEF.
- **Option b, Empagliflozin 10mg OD,** not recommended if eGFR <20 ml/min1.73 m2.

- Not for T1DM patients and be cautious if the patient has type 2 diabetes and a history of DKA.
- There may be a need to adjust other diuretics, diabetic agents or insulin. Discussion with the diabetic team may be necessary.
- Discuss with patient risk of potential side effects: risk of UTIs, thrush, genital infections etc. Advise regarding importance of personal hygiene.
- SICK DAY RULES - advise patient to stop for 1 -2 days until the patient recovers then restart the SGLT2.

TABLE 5. MRA.

- Check **baseline U&Es and BP, then increase the dose in 2 weeks' intervals, checking U&Es and BP after each increase**. MRA therapy should be avoided if baseline K+ >5.0mmol/l or eGFR<30ml/min.
- If Creatinine ≥ 200 µmol/L or Urea ≥ 11.2 mmol/L , or CKD stage ≥4–5 seek specialist advice.
- Caution/seek specialist advice if K+> 5.0 mmol/L .

If K+ between 5.0 – 5.4 mmol/L maintain current dose and repeat UEs.
If K+ rises ≥5.5 mmol/L -or creatinine rises to 200 µmol/L or eGFR <30 mL/min/1.73 m2, **halve a dose** and monitor UEs closely and seek specialist advice/cardiorenal MDT.
If K+ rises to ≥ 6.0 mmol/L or creatinine to >300 µmol/L or eGFR <20 mL/min/1.73 m2, **stop MRA immediately or refer to specialist to start potassium binder (Patiromer or SZC) and monitor U&Es .**

- Continue the next step uptitration if K+ ≤ 5.0 mmol/L and serum creatinine < 200ummol/L or if Cr increase <50% from the baseline, no diarrhoea or vomiting.
- Continue treatment and monitor U&Es at: 1w after initiation/up-titration, then 4w> 8w> 12w then every 3m – 6m.
- **Spironolactone starting dose 25mg OD (12.5mg if frail) , max dose 50mg OD (25mg if frail)**
- **Eplerenone starting dose 25mg OD, max dose 50mg OD.**

- If issues with breast tenderness or lactation on Spironolactone - switch to eplerenone and up titrate in the same manner.
- SICK DAY RULES: diarrhoea or vomiting or any other cause of sodium and water loss – stop MRA until symptoms settle.

TABLE 6. LOOP DIURETICS +/- THIAZIDE.

- Advise on regular weight monitoring and what to do if rapid weight gain observed (2kg in 3 days).
- Check baseline U&Es and BP, re-check renal function, serum electrolytes and BP in 1 to 2 weeks' after starting treatment.
- Titrate the dose of a loop diuretic up and down according to symptoms and signs of fluid overload.
- Advise on 'sick days' rules, stop for 1 to 2 days until the patient recovers.

- **Furosemide, starting dose 20 to 40mg OM**, can be increased up to 120mg BD (max 240 mg daily) if necessary, check U&Es and BP in 1 to 2 weeks after each dose increase.
- **Bumetanide, starting dose 0.5mg to 1mg OM**, can be increased up to 5mg OD, if necessary, check U&Es and BP in 1 to 2 weeks after each dose increase.
- **Torasemide**, **Starting dose 5mg OD**, can be increased to 20mg OD, check U&Es and BP in 1 to 2 weeks after each dose increase.

- Sudden increase in weight (>1Kg above dry weight sustained over ≥2 days - patient's stable weight with no signs of fluid overload) and/or increasing by oedema and breathlessness.
    - Furosemide is normally increased by 40mg daily at any one time or 1mg daily if Bumetanide, maintain increased dose for 3 days.
    - If dry weight not achieved/symptoms not improved continue and reassess in a further 3--4 days. If dry weight still not achieved, then consider further increase or seek specialist advice.
    - If weight/symptoms increased consider use of thiazide-like diuretic, initially **stat dose bendroflumethiazide 2.5mg or metolazone (Xaqua) 2.5mg** alongside loop diuretics, repeat U&Es the following day and assess weight/fluid status response ensuring potassium levels do not fall or renal function becomes compromised.
    - If dry weight achieved -return to original dose. Reverse the titration process if no peripheral oedema, JVP not raised. (seek specialist advice if present).
    - If repeated episodes (>2) in 2--3 weeks of weight gain/worsening symptoms discuss permanent increase in dose.

Aetiologies of HF:

Understanding the aetiology will inform the management plan

Table 3.4 Aetiologies of heart failure

DISEASED MYOCARDIUM		
Ischaemic heart disease	Myocardial scar	
	Myocardial stunning/hibernation	
	Epicardial coronary artery disease	
	Abnormal coronary microcirculation	
	Endothelial dysfunction	
Toxic damage	Recreational substance abuse	Alcohol, cocaine, amphetamine, anabolic steroids.
	Heavy metals	Copper, iron, lead, cobalt.
	Medications	Cytostatic drugs (e.g. anthracyclines), immunomodulating drugs (e.g. interferons monoclonal antibodies such as trastuzumab, cetuximab), antidepressant drugs, antiarrhythmics, non-steroidal anti-inflammatory drugs, anaesthetics.
	Radiation	
Immune-mediated and inflammatory damage	Related to infection	Bacteria, spirochaetes, fungi, protozoa, parasites (Chagas disease), rickettsiae, viruses (HIV/AIDS).
	Not related to infection	Lymphocytic/giant cell myocarditis, autoimmune diseases (e.g. Graves' disease, rheumatoid arthritis, connective tissue disorders, mainly systemic lupus erythematosus), hypersensitivity and eosinophilic myocarditis (Churg-Strauss).
Infiltration	Related to malignancy	Direct infiltrations and metastases.
	Not related to malignancy	Amyloidosis, sarcoidosis, haemochromatosis (iron), glycogen storage diseases (e.g. Pompe disease), lysosomal storage diseases (e.g. Fabry disease).
Metabolic derangements	Hormonal	Thyroid diseases, parathyroid diseases, acromegaly, GH deficiency, hypercortisolaemia, Conn's disease, Addison disease, diabetes, metabolic syndrome, phaeochromocytoma, pathologies related to pregnancy and peripartum.
	Nutritional	Deficiencies in thiamine, L-carnitine, selenium, iron, phosphates, calcium, complex malnutrition (e.g. malignancy, AIDS, anorexia nervosa), obesity.
Genetic abnormalities	Diverse forms	HCM, DCM, LV non-compaction, ARVC, restrictive cardiomyopathy (for details see respective expert documents), muscular dystrophies and laminopathies.
ABNORMAL LOADING CONDITIONS		
Hypertension		
Valve and myocardium structural defects	Acquired	Mitral, aortic, tricuspid and pulmonary valve diseases.
	Congenital	Atrial and ventricular septum defects and others (for details see a respective expert document).
Pericardial and endomyocardial pathologies	Pericardial	Constrictive pericarditis Pericardial effusion
	Endomyocardial	HES, EMF, endocardial fibroelastosis.
High output states		Severe anaemia, sepsis, thyrotoxicosis, Paget's disease, arteriovenous fistula, pregnancy.
Volume overload		Renal failure, iatrogenic fluid overload.
ARRHYTHMIAS		
Tachyarrhythmias		Atrial, ventricular arrhythmias.
Bradyarrhythmias		Sinus node dysfunctions, conduction disorders.

ARVC = arrhythmogenic right ventricular cardiomyopathy; DCM = dilated cardiomyopathy; EMF = endomyocardial fibrosis; GH = growth hormone; HCM = hypertrophic cardiomyopathy; HES = hypereosinophilic syndrome; HIV/AIDS = human immunodeficiency virus/acquired immune deficiency syndrome; LV = left ventricular.

Comorbidities associated with heart failure

Patients with heart failure have a wide range of comorbidities, in part due to their advanced age[1]
This can lead to the concurrent use of multiple medications[2]
Most comorbidities are associated with worse clinical status and are predictors of poor prognosis in heart failure[2]

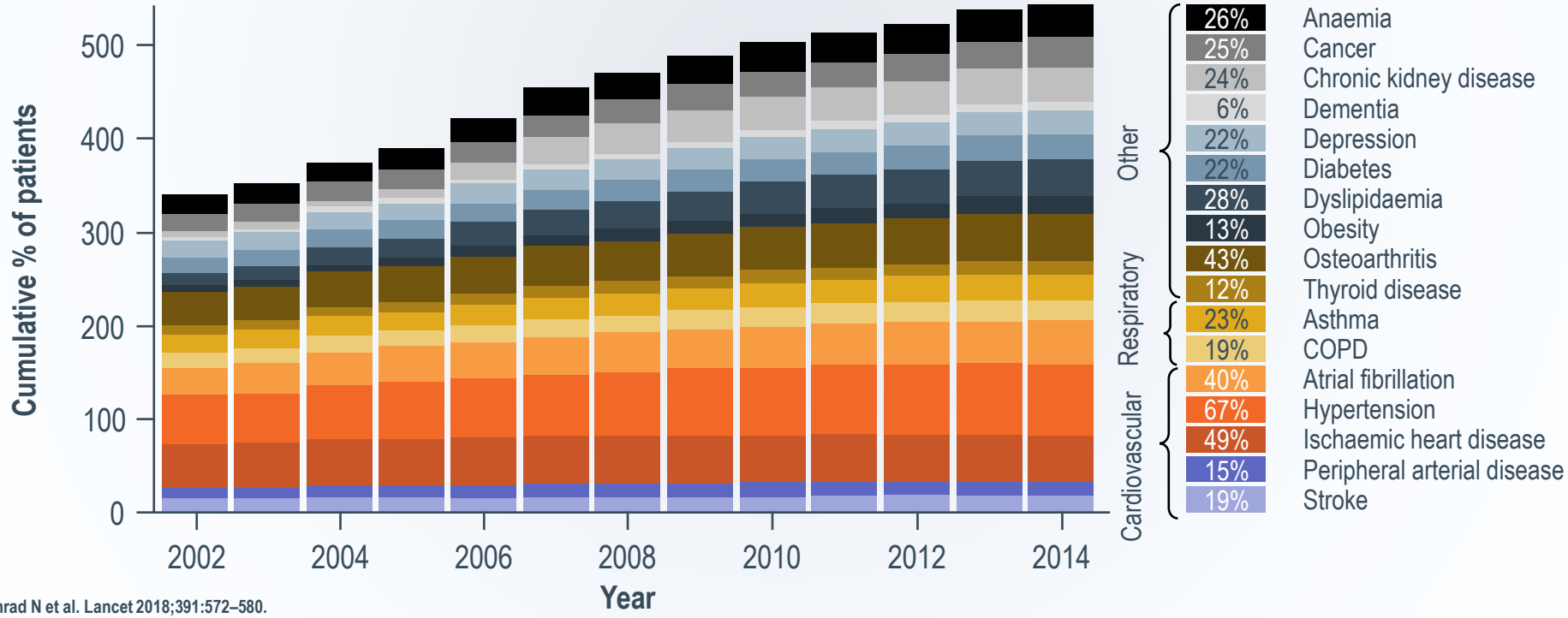


Figure adapted from Conrad N et al. Lancet 2018;391:572–580.
COPD, chronic obstructive pulmonary disease.

1. Conrad N et al. Lancet 2018;391:572–580; 2. Ponikowski P et al. Eur Heart J 2016;37:2129–2200.

Outcomes associated with early identification, diagnosis and treatment of heart failure[1]

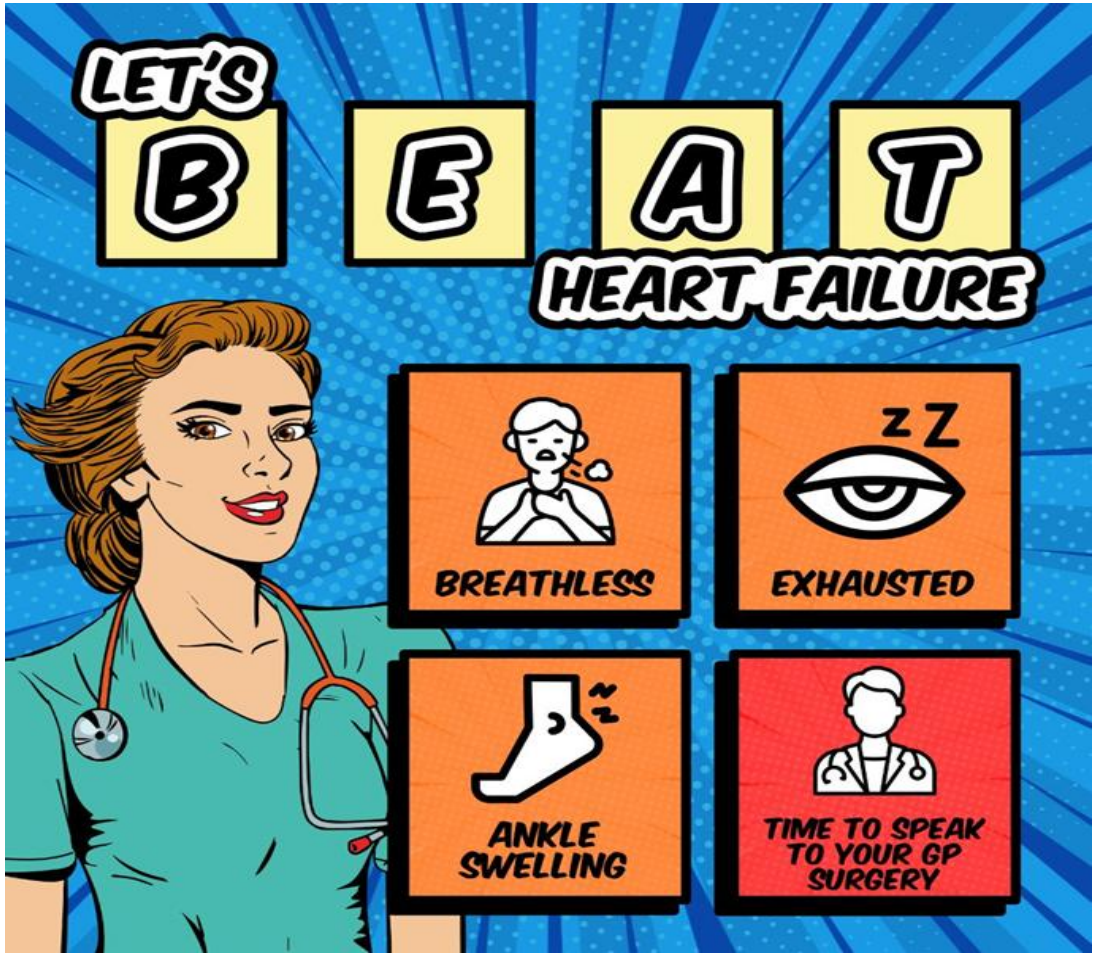
Implementation of recommended guidelines should lead to improved outcomes in heart failure:

- Early and accurate diagnosis to enable patients to start appropriate drug treatment
- Reduced re-admission rates due to patients being stabilised before discharge
- Improved quality of life for patients with heart failure

1. National Institute for Health and Care Excellence. Costing statement: acute heart failure. Implementing the NICE guideline on acute heart failure (CG187). 2014. Available at: https://www.nice.org.uk/guidance/cg187/resources/cg187-acute-heart-failure-costing-statement2. Accessed February 2020.

Help Detect the Undetected

Raise Awareness: Think BEAT



Breathless

Exhausted

Ankle Swelling

Time to speak to GP /HCP

Remember:

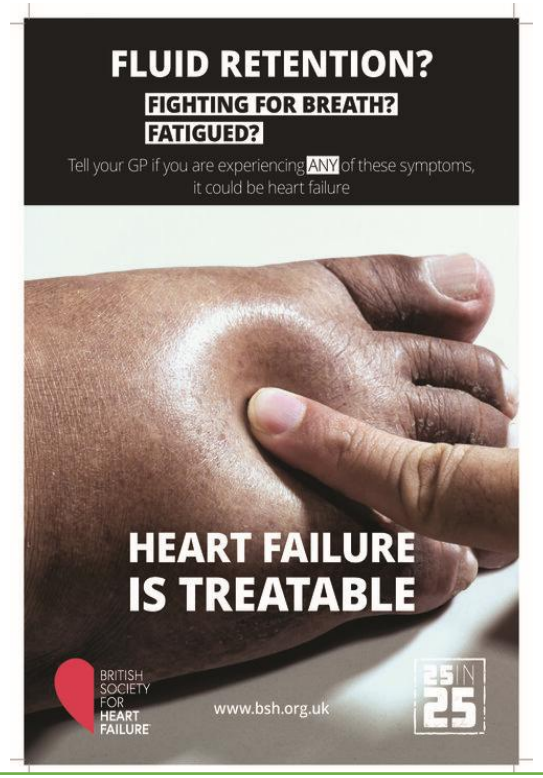
-Patients are either unaware of HF symptoms, or think symptoms relate to existing comorbidities

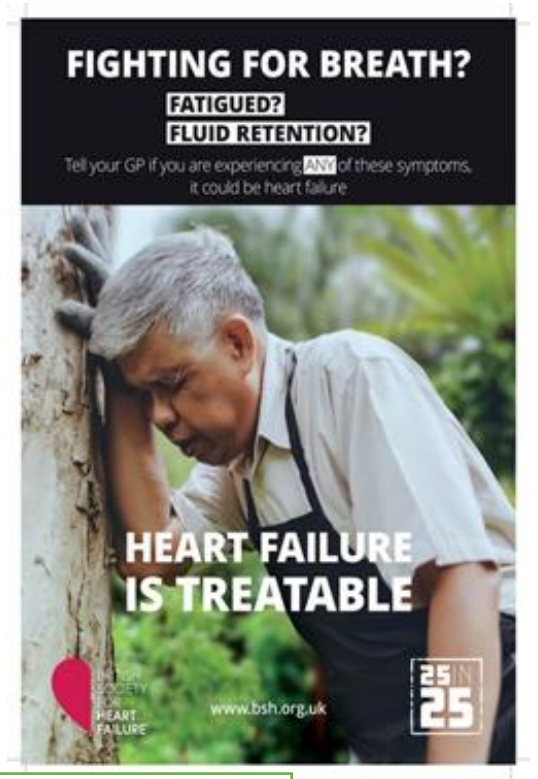
-Patients don't present early enough to prevent further decline

https://pumpingmarvellous.org

Together we can turn the tide on this life limiting condition.

Help Raise Awareness: The ‘F’ word





Heart Failure is treatable

Together we can turn the tide on this life limiting condition.

.https://www.bsh.org.uk

Be Heart Failure Aware

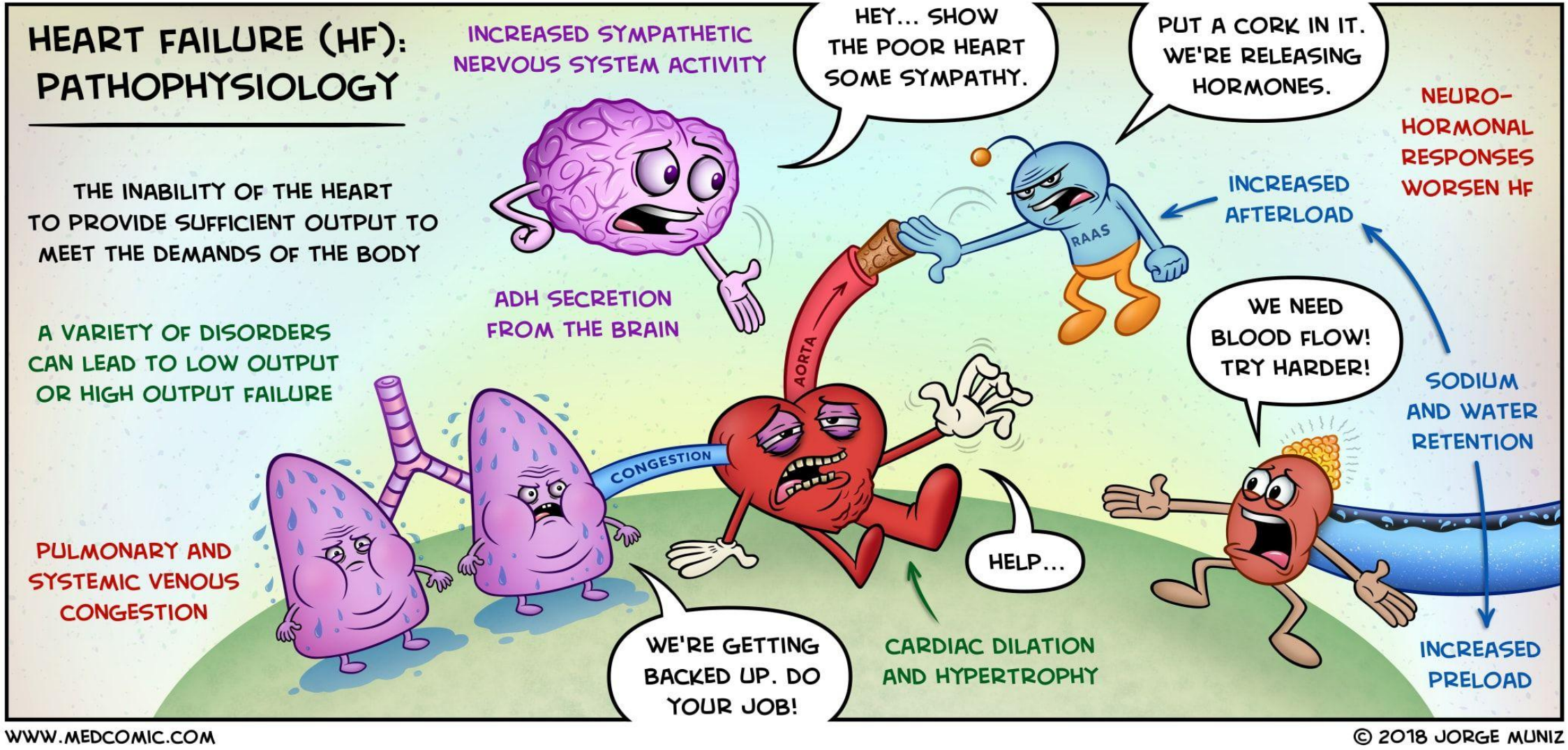
- Be aware of those at risk of developing HF
- Be aware of the signs and symptoms
- Be aware of the importance of early and accurate diagnosis
- Be aware of appropriate, timely evidence-based treatment

Make Every Contact Count

Together we can make a difference

25 IN 25

Together we can turn the tide on this life limiting condition.



Thank you for listening

Any questions?