# Cardiology at the interface of primary and secondary care: session 3 – How to improve heart failure outcomes

This webinar will start shortly.

# Cardiology at the interface of primary and secondary care: session 3 – How to improve heart failure outcomes

**Zoom webinar – Wednesday 23 July 2025**

**6.30-8pm**

# Acknowledgement of traditional owners

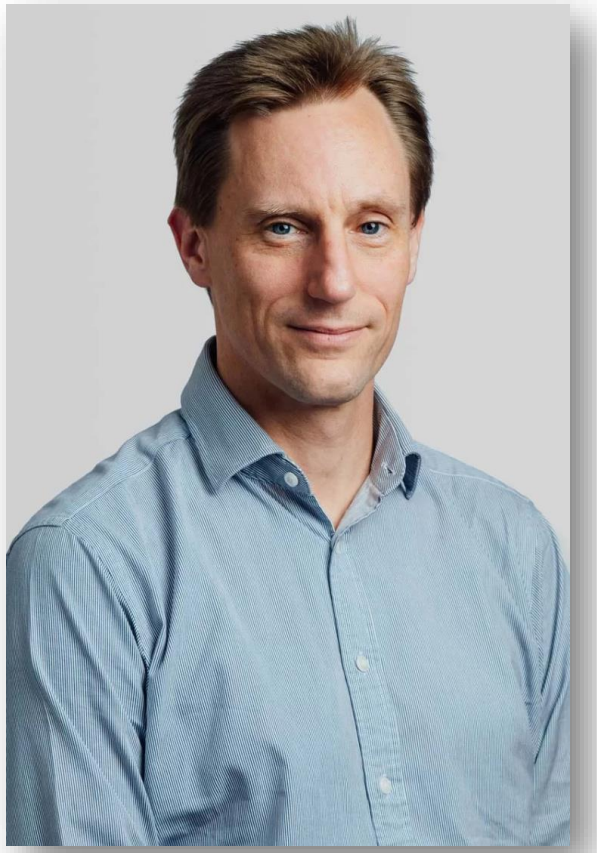
We acknowledge the Tasmanian Aboriginal people as the traditional owners and ongoing custodians of the land on which we are meeting today. We pay our respects to Elders past and present.

We would also like to acknowledge Aboriginal people who are joining us today.

# Some housekeeping

- Tonight's webinar is being recorded
- Please use the Zoom Q&A feature to ask questions
- At the end of the webinar your browser will automatically open an evaluation survey. We appreciate you taking the time to complete this to help us improve our events programme
- Please don't forget to register for your next webinar at: https://www.primaryhealthtas.com.au/for-health-professionals/events/

# Presenters



Dr Nathan Dwyer

Luke Dare

# Panel

Dr Paul MacIntyre

Dr Faline Howes

# How to Improve Heart Failure Outcomes

Dr. Nathan Dwyer

BMedSci (Hons), MBBS (Hons), PhD, FRACP, FCSANZ

# Learning Objectives

1. Understand the mechanisms involved in the development of HFrEF and HFpEF
2. Understand evidence based diagnostic strategies for heart failure
3. Learn a Mnemonic for the evidence-based therapies for heart failure
4. Gain comfort in prescribing and titrating the Four Pillars of heart failure therapy
5. Gain confidence with the management of common adverse events of therapy
6. Learn some approaches for interpreting an echocardiogram report

# Heart failure affects a large number of Australians each year, placing a significant burden on the healthcare system[1]

**61,000 deaths**
were listed as caused by heart failure in Australia in 2016

**511,000 Australians**
(2.1%) had heart failure in 2016

**~67,000 Australians**
are newly diagnosed with chronic heart failure every year

**158,000 hospitalisations**
With primary diagnosis of heart failure in Australia in 2016
1.1 million days of hospital stay

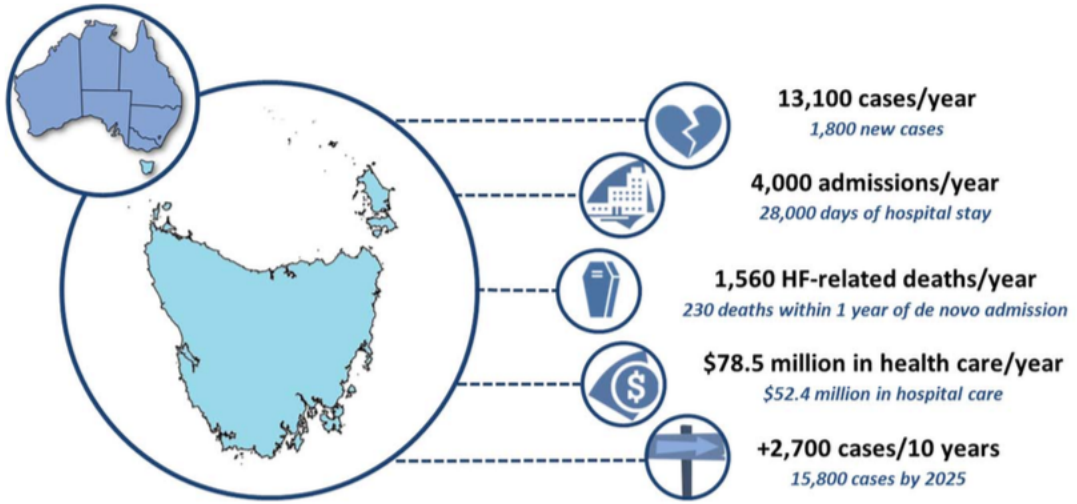
**8.9 days**
average length of stay[2]

**$3.1 billion**
per annum in costs to the Australian economy
$2 billion in hospital care

**+146,000 cases/10 years**
projected growth


1. Chen L, Booley S, Keates AK, Stewart S. Snapshot of heart failure in Australia. May 2017. Mary MacKillop Institute for Health Research, Australian Catholic University, Melbourne, Australia
2. Australian Institute of Health and Welfare 2011. Cardiovascular disease: Australian facts 2011. Cardiovascular disease series. Cat. no. CVD 53. Canberra: AIHW.

# Snapshot of Heart Failure in Tasmania



13,100 cases/year
*1,800 new cases*

4,000 admissions/year
*28,000 days of hospital stay*

1,560 HF-related deaths/year
*230 deaths within 1 year of de novo admission*

$78.5 million in health care/year
*$52.4 million in hospital care*

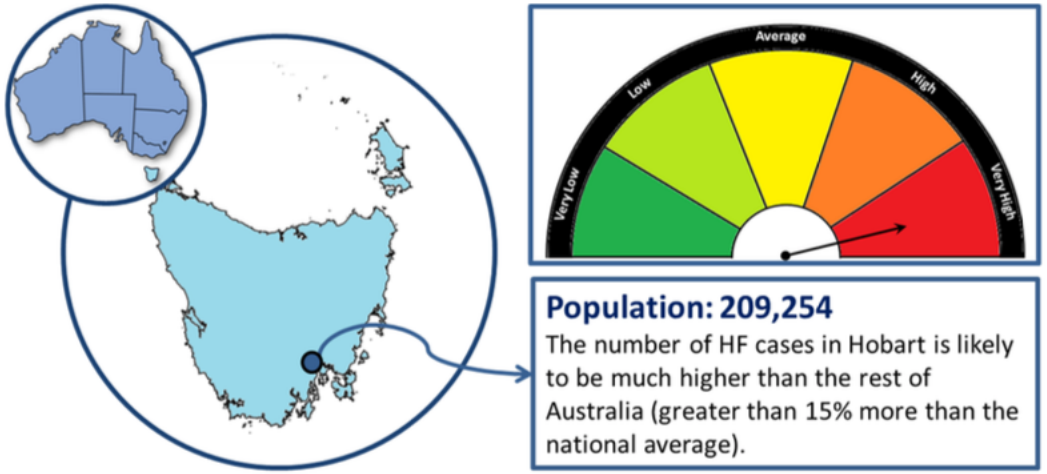
+2,700 cases/10 years
*15,800 cases by 2025*

| | Men | Women |
|---|---|---|
| **Population Profile** (Adults aged ≥45 years) | | |
| All/*New* Cases of HF | 8,700/*800* | 4,400/*1,000* |
| These figures reflect the probable number of Australians with clinical signs and symptoms of HF associated with underlying coronary heart disease and a reduced ejection fraction (HFrEF) with more men than women affected overall. | | |
| **Hospital Burden** (per annum) | | |
| All/*New* Hospital Admissions | 2,000/*420* | 2,000/*350* |
| Days of hospital stay | 14,000 | 14,000 |
| HF rarely occurs in isolation and when present as comorbidity negatively influences health outcomes. As such, these data reflect all hospital admissions where HF is listed as primary or secondary diagnosis. | | |
| **HF-related deaths** (per annum) | | |
| Total deaths | 1,040 | 520 |
| 1 year of de novo admission | 120 | 110 |
| HF is as "malignant" as many forms of cancer; particularly once an individual is hospitalised – within 5 years of a de novo admission ~50% of patients will have died. | | |
| **Health Care Costs** (per annum) | | |
| Total health care costs | $43.6 million | $34.9 million |
| Cost of hospital care | $26.3 million | $26.1 million |
| The costliest and most preventable component of health care attributable to HF is hospital care for those patients who become clinically unstable and have recurrent events. | | |
| **Future burden** (per annum) | | |
| All/*New* Cases of HF in 2025 | 10,200/*1,100* | 5,600/*1,300* |
| Even without any change in the key drivers of HF (e.g. hypertension and coronary heart disease), population dynamics alone will mean substantially more cases in the decade ahead. | | |

## Additional Key Facts about heart failure (HF)

- Beyond those with HF associated with an inability of the heart to contract properly (mostly caused by underlying coronary heart disease and known as HFrEF) an estimated **13,700** adults (with more women affected) have a form of HF that is associated with an inability of the heart to relax - HF with preserved ejection fraction (HFpEF)
- In men and women **1,300** and **700** HF admissions (**65%** and **35%**) per annum respectively, are linked to an coronary heart disease and HFrEF.
- Within 30-days of a *de novo* HF admission one third of surviving patients will be readmitted for any reason (**220** patients/year)
- Within one year of an initial HF-related admission, on average a patient will experience **3 more hospital (re)admissions**
- Around **one third** of hospital admissions for HF (**1,340** are preventable overall)

# Snapshot of Heart Failure in Hobart

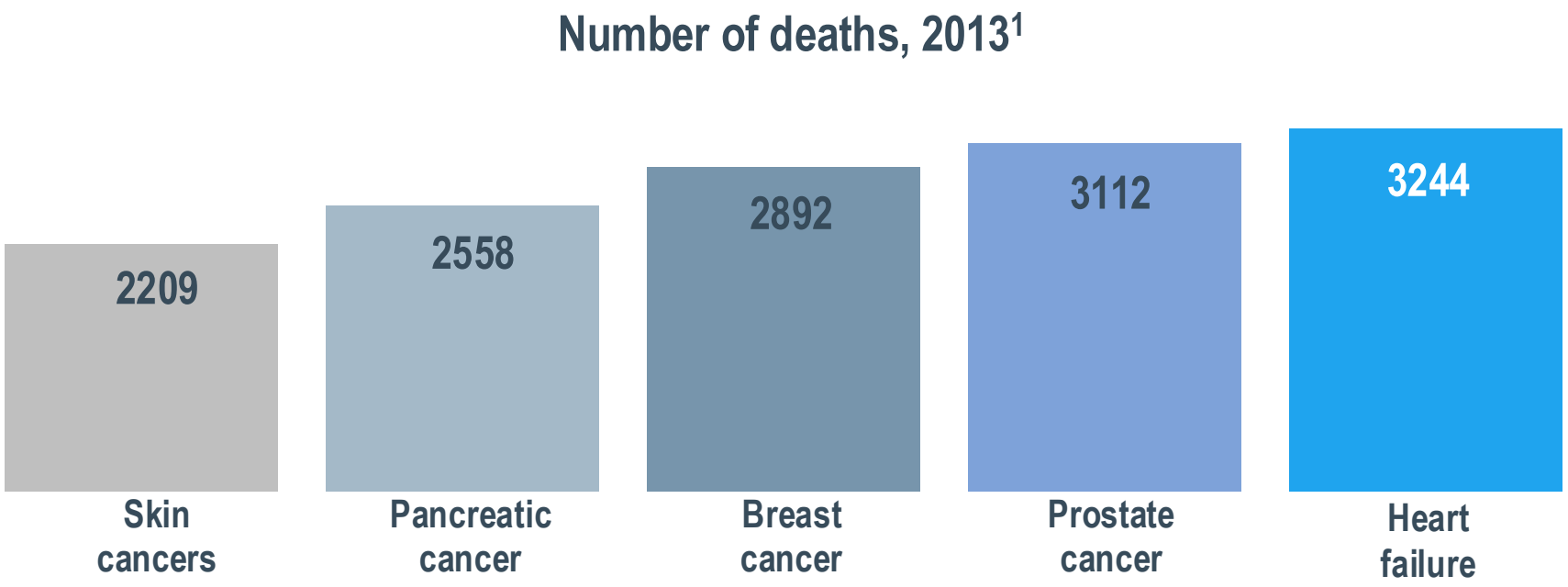



**Population: 209,254**
The number of HF cases in Hobart is likely to be much higher than the rest of Australia (greater than 15% more than the national average).

| | Men | Women |
|---|---|---|
| **Population Profile** (Adults aged ≥45 years) | | |
| All/*New* Cases of HF | 4,100/*370* | 2,200/*490* |
| **Hospital Burden** (per annum) | | |
| All Hospital Admissions | 710 | 750 |
| Days of hospital stay | 4,900 | 5,200 |
| **Health Care Costs** (per annum) | | |
| Total health care costs | $17.6 million | $14.3 million |

Snapshot of heart failure in Australia. May 2017. MMIHR, Australian Catholic University.

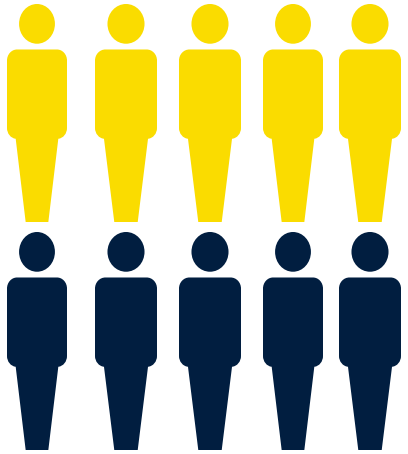
# Annual mortality for HF is higher than that of some common cancers[1]




1. Australian Bureau of Statistics, 3303.0 - Causes of Death, Australia, 2013 Available at http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/by%20Subject/3303.0~2013~Main%20Features~Leading%20Causes%20of%20Death~10001 Accessed July 2016.

# Chronic HF has a significant impact on long-term prognosis[1]

**5**
**years**

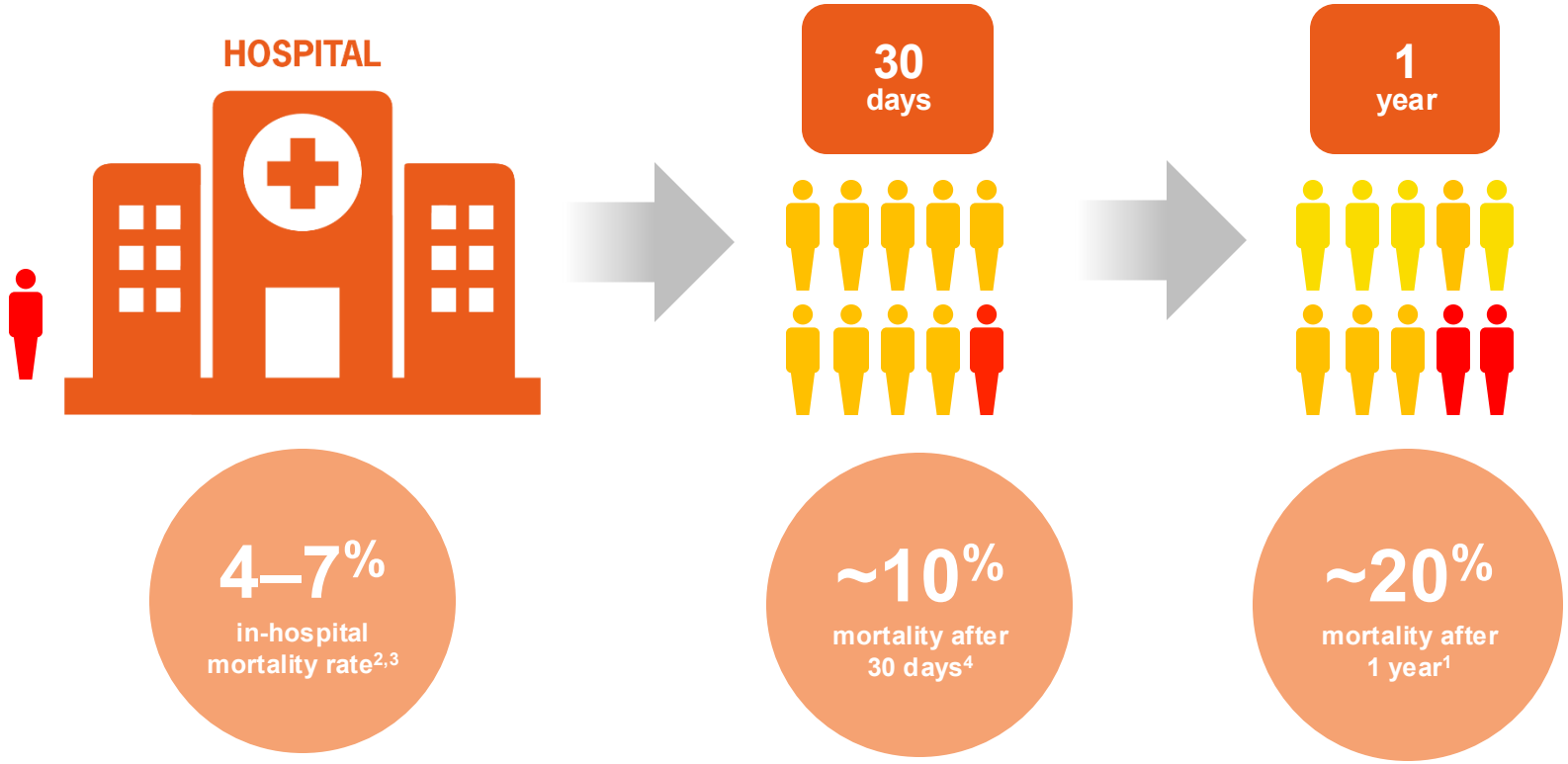


**~50%**
mortality after onset of heart failure[2-4]

- All patients with heart failure, regardless of their symptoms, have a poor prognosis[1]
- Within 3 years, 34% of NYHA class I and II patients, and 42% of NYHA class III and IV patients die[1]


1. Ahmed A. *Am J Cardiol* 2007;99:549–53.
2. Roger VL *et al*. *JAMA* 2004;292:344–50.
3. Levy D *et al*. *N Engl J Med* 2002;347:1397–402.
4. Go AS *et al*. *Circulation* 2014;129:e28–e292.

# HOSPITALISATION FOR ACUTE HEART FAILURE IS ASSOCIATED WITH SIGNIFICANT MORTALITY[1-4]

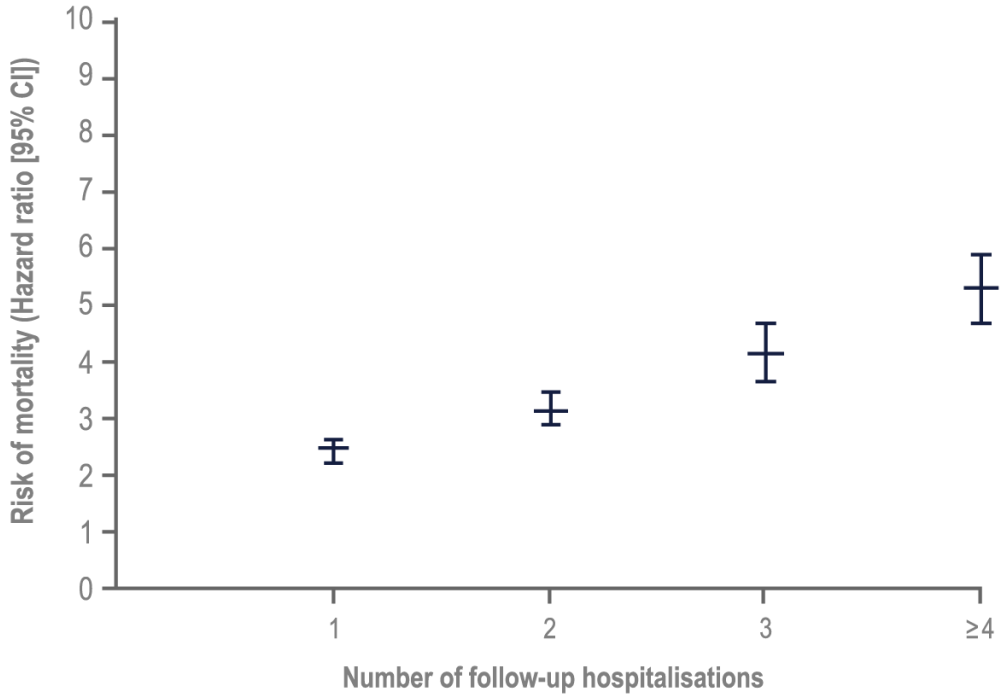




1. Maggioni AP *et al. Eur J Heart Fail* 2013;15:808–17.
2. Maggioni AP *et al. Eur J Heart Fail* 2010;12:1076–84.
3. Nieminen MS *et al. Eur Heart J* 2006;27:2725–36.
4. Loehr LR *et al. Am J Cardiol* 2008;101:1016–22.

# Each time a patient is hospitalised for HF, their mortality risk increases[1]

## Risk of mortality during a median follow-up of 1,024 days, according to the number of re-hospitalisations for heart failure[1]




Adapted from Lee DS et al. (2009).[1] Retrospective clinical audit examining the 'dose-dependent' relationship between heart failure events and death in patients with heart failure (n=9138) in the Enhanced Feedback For Effective Cardiac Treatment Study.

**30% readmission rates[2]**
within 30 days

**3 more hospital admissions[2]**
within 1 year of *de novo* HF admission

**~30% of admission preventable[2]**
(53,000 admissions)


1. Lee DS *et al*. *Am J Med* 2009;122:162–9 e1.
2. Chen L, Booley S, Keates AK, Stewart S. Snapshot of heart failure in Australia. May 2017. Mary MacKillop Institute for Health Research, Australian Catholic University, Melbourne, Australia

# A SIGNIFICANT PROPORTION

of Australians with HF are missing out on guideline-recommended treatment

**SHAPE study[1]**

A retrospective cohort study of HF in the Australian primary care setting using Medical Director data

21,803 people classified as having:

- definite HF (16,930) or
- probable HF (4,873)

**Of those classified as having definite or probable heart failure (HF):**

**<15%**

**had HF recorded as a diagnosis** (although 55% had HF recorded either as a diagnosis or as free text in the notes)

**Only 1 in 5**

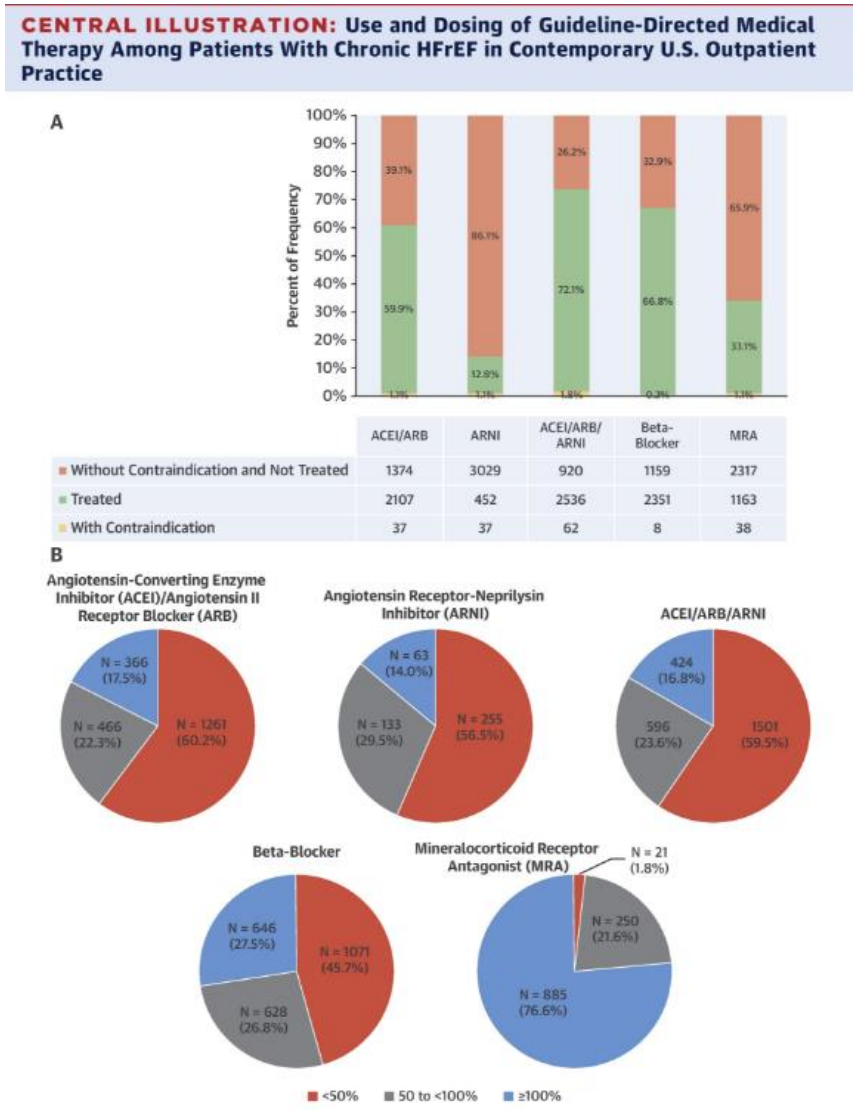
**were on a HF specific medication (21.8%)**

**22%**

**had symptoms/ signs of HF and were on a diuretic**

**Fewer than half**

**(46.8%) had a cardiologist referral within 30 days of a HF diagnosis**

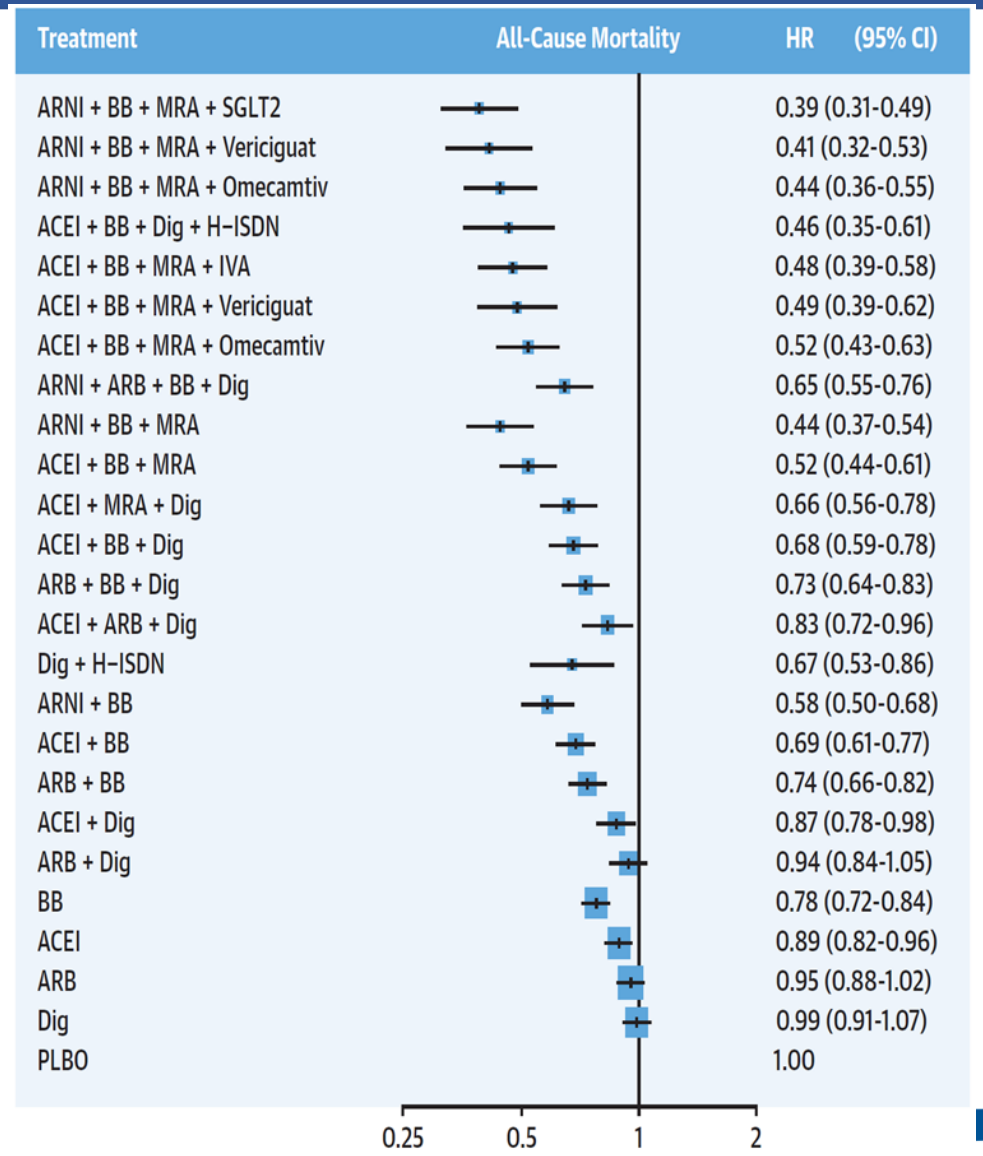



Greene, S.J. et al. J Am Coll Cardiol. 2018;72(4):351-66.

# CHAMP-HF

- 3518 patients from 150 primary care and cardiology practices
- Among eligible patients,
  - 27% not prescribed- ACEi/ARNI
  - 33% not prescribed BB
  - 67% not prescribed MRA
- Of those receiving drugs
  - 17% receiving target dose of ACEi
  - 28% receiving target dose of BB
  - 77% receiving target dose of MRA
- In those eligible – only 1% receiving target of all 3

# The Four Pillars



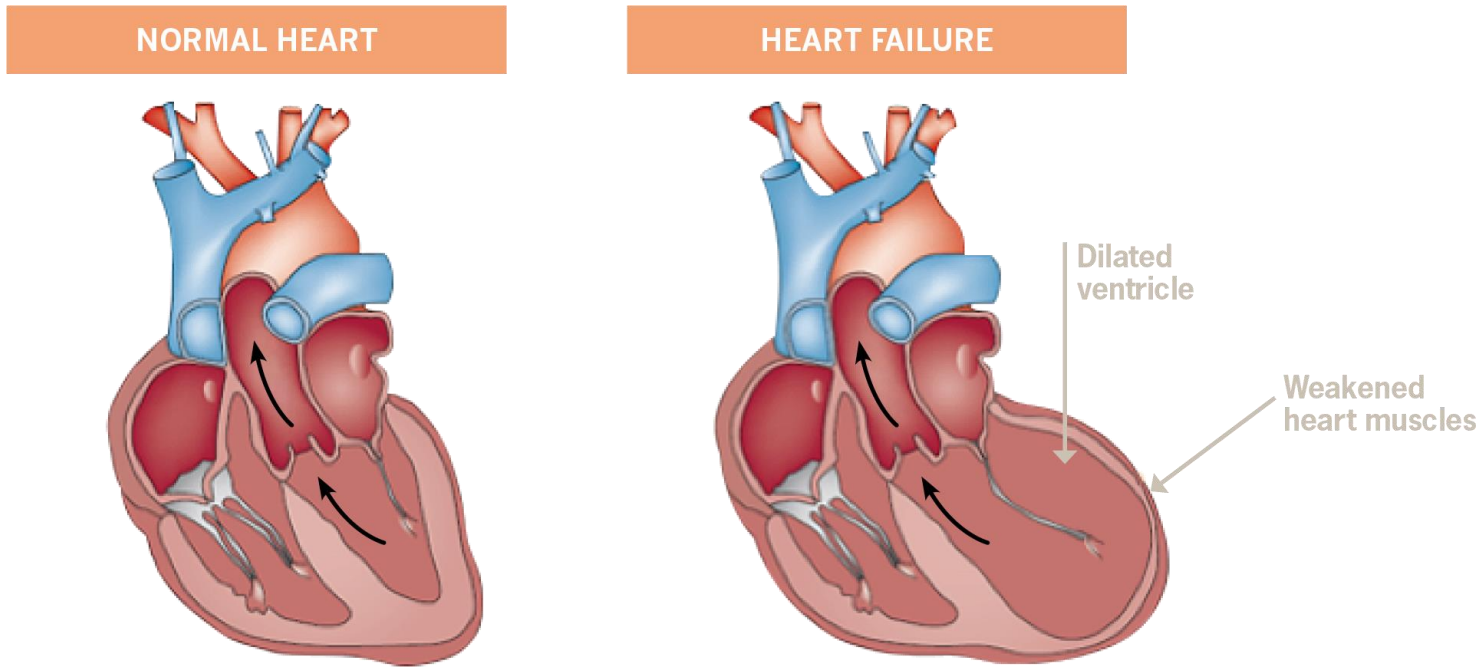

Tromp, J. et al. J Am Coll Cardiol HF. 2022;10(2):73-84.

# What is Heart Failure?

## How Do We Diagnose It?

# Symptoms and Signs of Heart Failure are the Result of Abnormalities of Cardiac Structure/Function

- Abnormality of cardiac structure or function leads to failure of the heart to adequately perfuse organ systems
- Weakening or stiffening of the heart muscle over time leads to pump failure and insufficient delivery of blood around the body



# Heart Failure is a Clinical Syndrome

## Heart failure is characterised by typical symptoms, which include:

- Breathlessness
- Orthopnoea
- Paroxysmal nocturnal dyspnoea
- Ankle swelling
- Fatigue
- Reduced exercise tolerance

## These symptoms may be accompanied by typical signs, such as:

- Elevated jugular venous pressure
- Pulmonary crackles
- Peripheral oedema

# Heart Failure Signs & Symptoms

| Symptoms | Signs |
|---|---|
| **Typical** | **More specific** |
| Breathlessness<br>Orthopnoea<br>Paroxysmal nocturnal dyspnoea<br>Reduced exercise tolerance<br>Fatigue, tiredness, increased time to recover after exercise<br>Ankle swelling | Elevated jugular venous pressure<br>Hepatojugular reflux<br>Third heart sound (gallop rhythm)<br>Laterally displaced apical impulse |
| **Less typical** | **Less specific** |
| Nocturnal cough<br>Wheezing<br>Bloated feeling<br>Loss of appetite<br>Confusion (especially in the elderly)<br>Depression<br>Palpitations<br>Dizziness<br>Syncope<br>Bendopnoea | Weight gain (>2 kg/week)<br>Weight loss (in advanced heart failure)<br>Tissue wasting (cachexia)<br>Cardiac murmur<br>Peripheral oedema (ankle, sacral, scrotal)<br>Pulmonary crepitations<br>Reduced air entry and dullness to percussion at lung bases (pleural effusion)<br>Tachycardia<br>Irregular pulse<br>Tachypnoea<br>Cheyne Stokes respiration<br>Hepatomegaly<br>Ascites<br>Cold extremities<br>Oliguria<br>Narrow pulse pressure |

- Symptoms and signs of heart failure should be assessed at each visit
- Particular attention should be given to evidence of congestion and BP/HR
- Persistence of symptoms despite treatment often indicates the need for additional therapy

# DEFINITION OF HEART FAILURE[1]

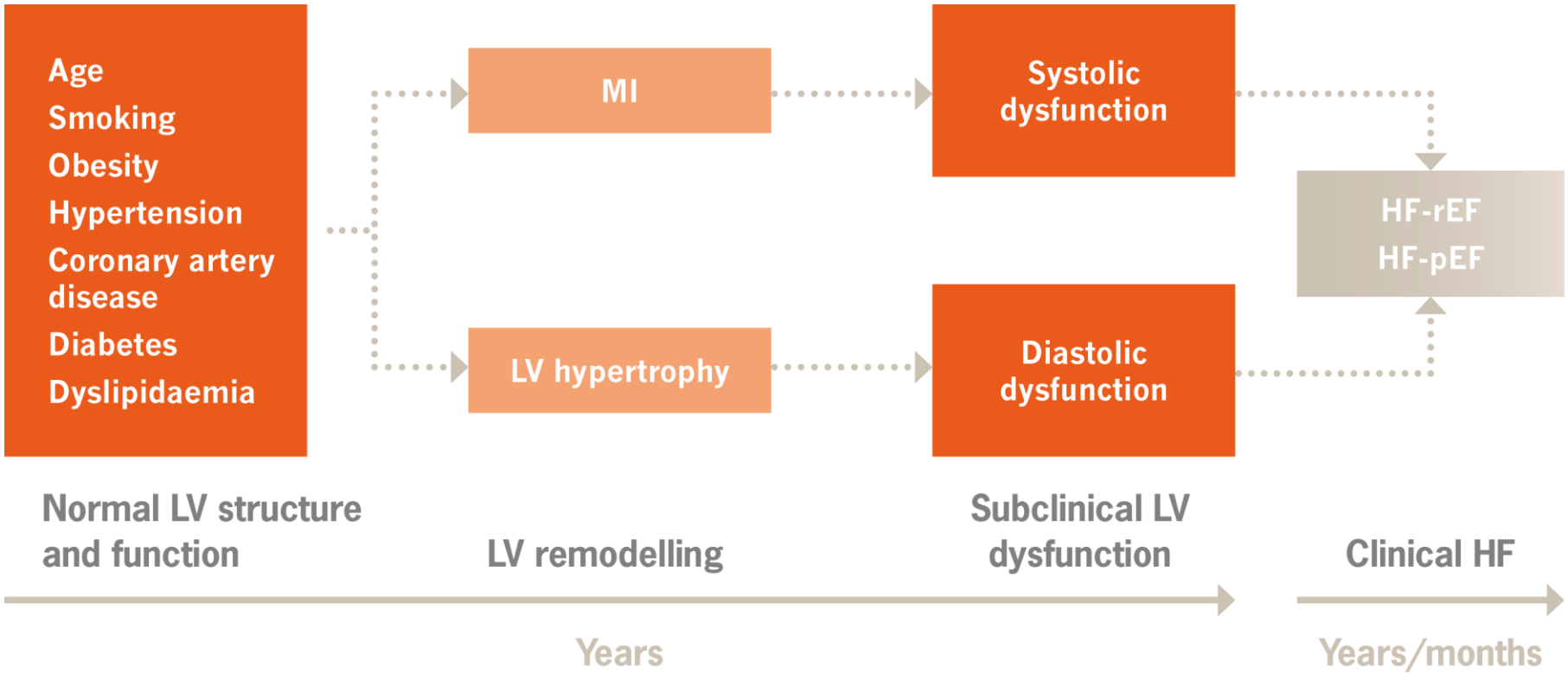
| HF-rEF | HF-pEF |
| --- | --- |
| • Symptoms ± signs of heart failure<br>**and**<br>• LVEF <50%† | • Symptoms ± signs of heart failure<br>**and**<br>• LVEF ≥50%<br>**and**<br>• Objective evidence of:<br>- Relevant structural heart disease (LV hypertrophy, left atrial enlargement)<br>**and/or**<br>- Diastolic dysfunction, with high filling pressure demonstrated by any of the following:<br>• Invasive means (cardiac catheterisation)<br>• Echocardiography<br>• Biomarker (elevated BNP or NT-proBNP)<br>• Exercise (invasive or echocardiography) |

"HF is a complex clinical syndrome with typical symptoms and signs that generally occur on exertion, but can also occur at rest (particularly when recumbent), that is secondary to an abnormality of cardiac structure or function that impairs the ability of the heart to fill with blood at normal pressure or eject blood sufficient to fulfil the needs of the metabolising organs."[1]

CSANZ Heart Failure Guidelines

# Different Co-Morbidities and Pathophysiological Processes Can Lead to Different Types of Heart Failure[1]

## A range of risk factors and co-morbidities contribute to the development of heart failure[9]





1. Krum H *et al. Lancet* 2003;362:147–58
2. Ponikowski P *et al. Eur Heart J* 2016; 37:2129-2200

# HFrEF is a cardiac syndrome; HFpEF is a systemic syndrome

**HF-REF: pathophysiology**

Cardiac syndrome driven by myocardial injury leading to loss of myocardial cells[1]

Triggers adaptive neurohormonal signals that are eventually deleterious[2]

Neurohormonal signals cause LV remodelling that impairs LV function and causes HF[2]

**HF-PEF: pathophysiology hypothesis**

Cumulative expression of risk factors and comorbidities (eg female gender, diabetes, chronic kidney disease, ageing, obesity, COPD)[1,3]

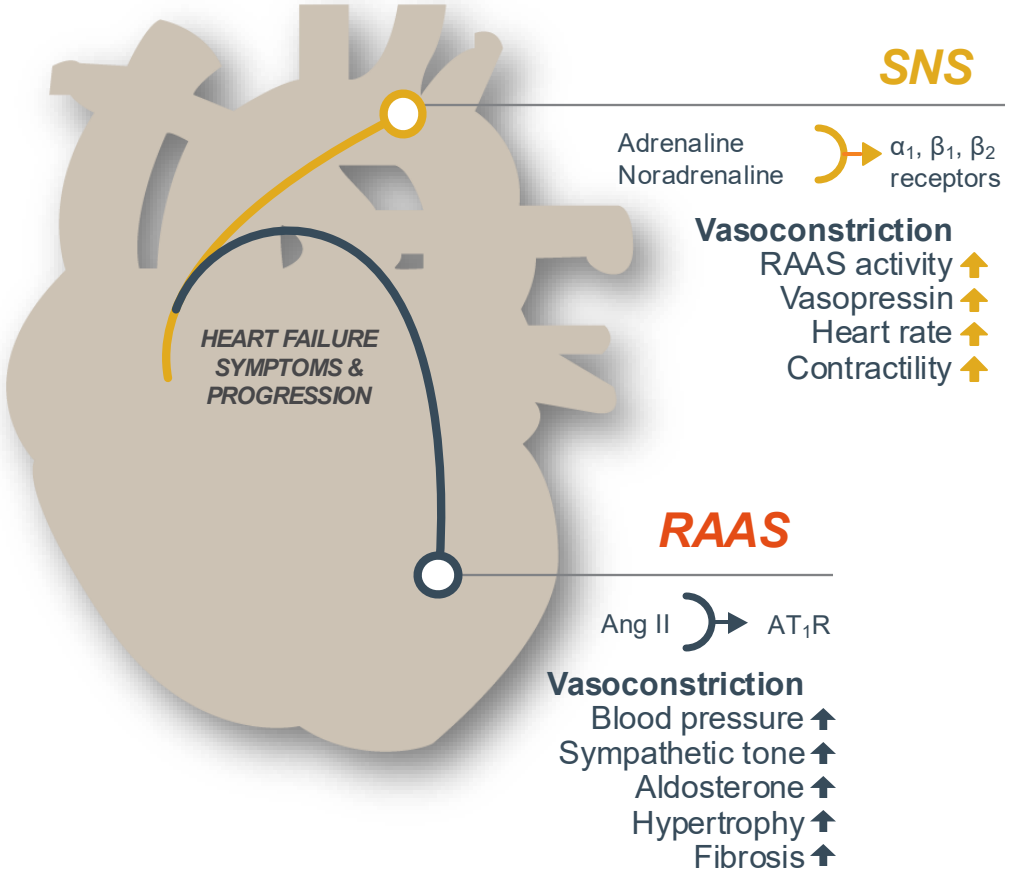
Systemic disease state places increased stress on LV wall promoting LV remodelling (hypertrophy)[3]

LV hypertrophy associated with myocardial fibrosis and stiffness[3]

Eventual diastolic dysfunction

1. Ferrari R, et al. Eur J Heart Fail. 2015; 17:665–671.
2. McMurray JJV, et al. Eur Heart J. 2012; 33:1787–1847.
3. Heinzel F, et al. J Appl Physiol. 2015; 119:1233–1242.

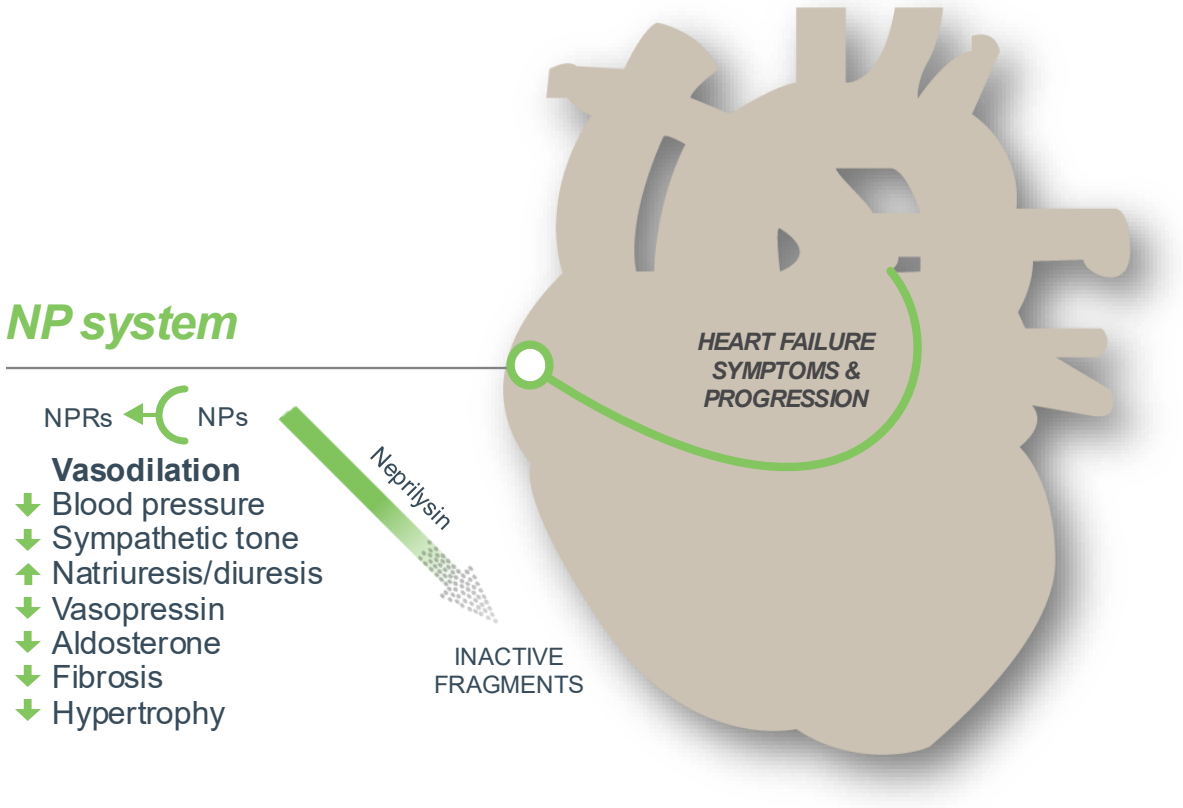
# The SNS and RAAS are over-activated in HF and are responsible for many of the pathophysiological responses that contribute to disease progression[1-3]




Ang: angiotensin; $AT_1R$: angiotensin type 1 receptor; HF: heart failure; RAAS: renin-angiotensin-aldosterone system; SNS: sympathetic nervous system.

1. Kemp CD *et al. Cardiovasc Pathol* 2012;21:365–71. 2. Schrier RW *et al. N Engl J Med* 1999;341:577–85. 3. Langenickel *et al. Drug Discov Today: Ther Strateg* 2012;9:e131–9.

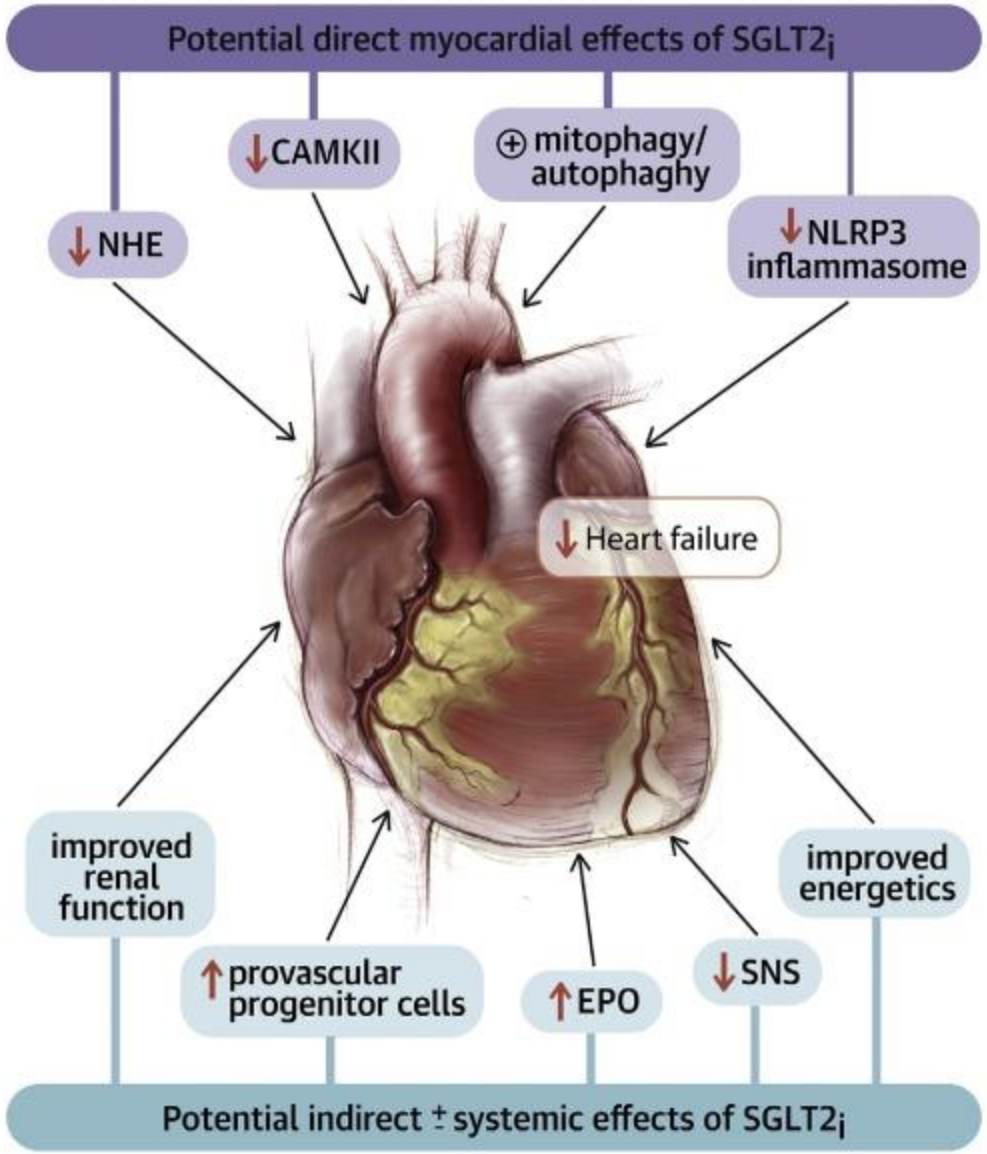
# Secretion of natriuretic peptides results in a number of responses that act to reduce the symptoms and progression of HF[1,2]




NP: natriuretic peptide; NPR: natriuretic peptide receptor.

1. Levin ER *et al. N Engl J Med* 1998;339:321–8. 2. Mangiafico S *et al. Eur Heart J* 2013;34:886–93c.

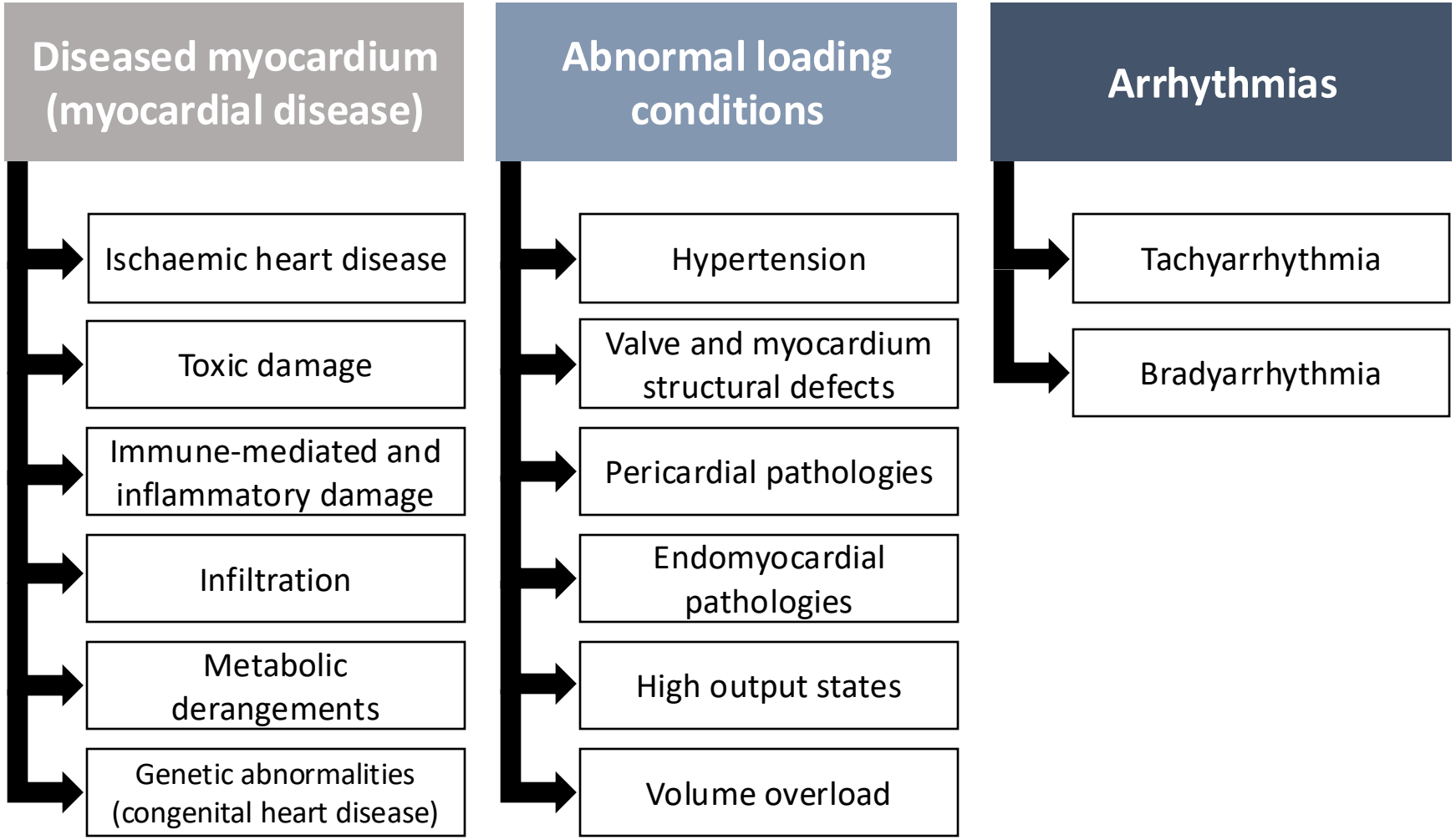
**CENTRAL ILLUSTRATION: Potential Direct Myocardial and Indirect ± Systemic Effects of SGLT2$_i$**




Lopaschuk, G.D. et al. J Am Coll Cardiol Basic Trans Science. 2020;5(6):632-44.

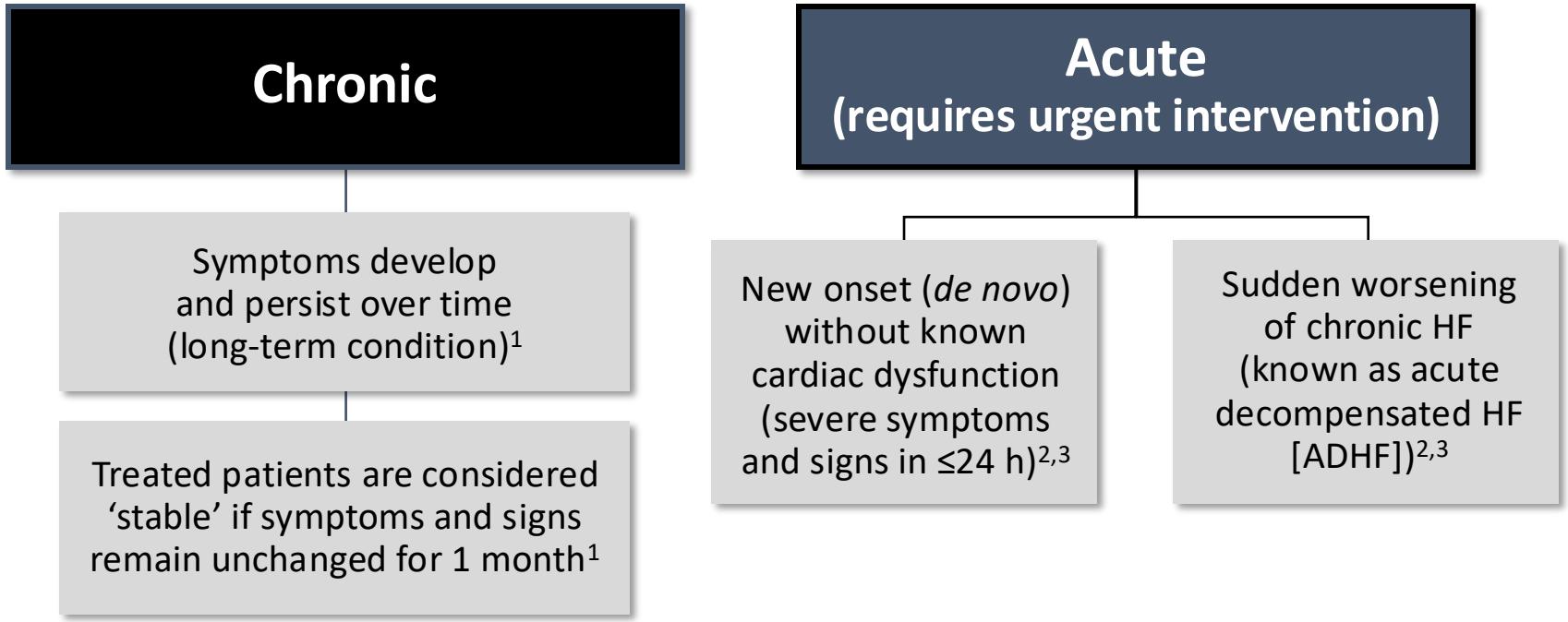
- Increasing diuresis/natriuresis
- Blood pressure lowering
- Improved cardiac energy metabolism
- Preventing inflammation
- Weight Loss
- Improving glucose control
- Inhibiting the SNS
- Preventing adverse cardiac remodelling
- Preventing ischaemia/reperfusion injury
- Inhibiting cardiac Na/H exchanger
- Reducing hyperuricemia
- Increasing autophagy and lysosomal degradation
- Decreasing epicardial fat mass
- Increasing EPO levels
- Increasing circulating provascular progenitor cells
- Decreasing oxidative stress
- Improving vascular function

# Aetiology of HF



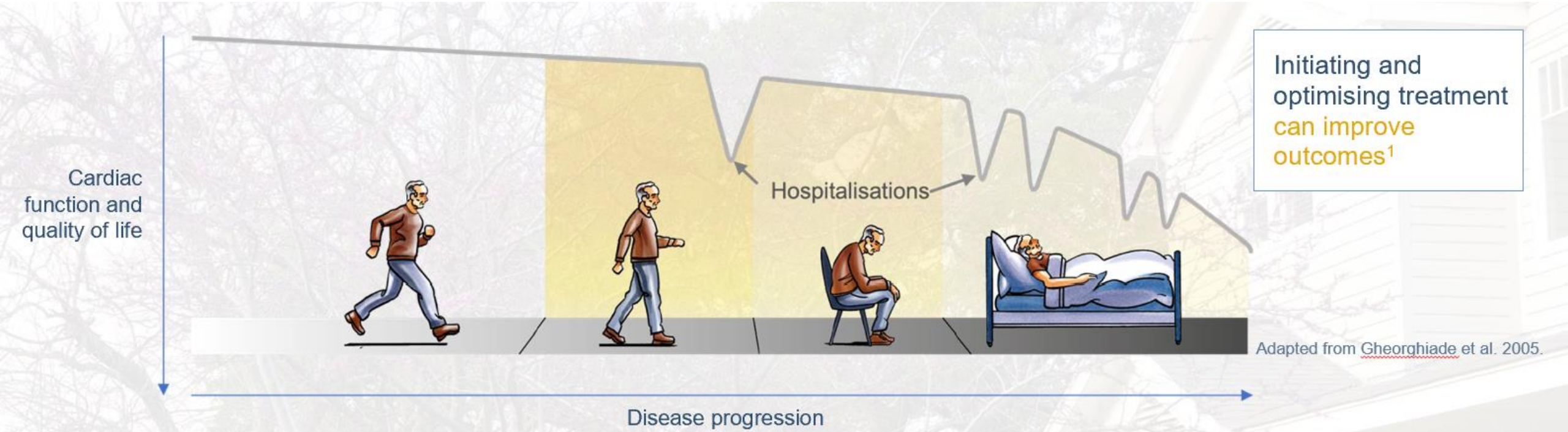

1. Reprinted from Ponikowski P, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J, 2016. doi:10.1093/eurheartj/ehw128 by permission of Oxford University Press on behalf of the European Society of Cardiology, www.escardio.org/.

# HF may develop chronically or acutely




1. Ponikowski P, et al. Eur Heart J. 2016; doi: 10.1093/eurheartj/ehw128. 2. NICE Guideline CG187. Acute heart failure: diagnosis & management. Clinical guideline. 2014. Available at: www.nice.org.uk/guidance/cg187. 3. Mosterd A, Hoes AW. Heart. 2007; 93:1137–1146.

# Chronic Progressive Disease




Increasing frequency of acute events with disease progression leads to high rates of hospitalisation, increased morbidity and increased risk of mortality.[1-3]

# Causes of Acute Heart Failure Decompensations

- Non-adherence to diet or medications
- Arrhythmias
- Ischaemia
- Infection
- Anaemia
- Thyroid disease
- Hypertension
- Renal Failure
- Acute or worsening valvular disease
- Addition of exacerbating medications
  - NSAIDs, prednisolone, non-dihydropyridine calcium channel antagonist

# NYHA classification is important for evaluating the symptoms of patients with HF

- HF can be graded according to NYHA functional classification.
- NYHA functional classification is widely used and accepted and is based on exercise capacity and symptoms of the disease.[1]

| NYHA Classes | | | |
|---|---|---|---|
| **NYHA class I** | **NYHA class II** | **NYHA class III** | **NYHA class IV** |
| No limitation of physical activity | Slight limitation of physical activity | Marked limitation of physical activity | Unable to carry on any physical activity without discomfort |
| No overt symptoms | Comfortable at rest, but ordinary physical activity causes symptoms of heart failure | Comfortable at rest, but less than ordinary activity causes symptoms of heart failure | May have symptoms even at rest which increases with any activity |

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

# A NUMBER OF DIAGNOSTIC ASSESSMENTS CAN BE USED TO SUPPORT THE PRESENCE OF HEART FAILURE

| | |
|---|---|
| **Assessment of symptoms** | Compatible symptoms include breathlessness, fatigue, angina, palpitations or syncope |
| **Assessment of signs** | Compatible signs should include appearance, pulse, BP, fluid overload, respiratory and heart rate |
| **ECG** | ECG changes are common (e.g. presence of new Q waves reflecting a MI; wave abnormalities reflecting ischaemia, or an arrhythmia). If the ECG is completely normal, heart failure, especially with systolic dysfunction, is unlikely (<10%) |
| **Laboratory analyses** | Elevated BNP/NT-proBNP, hyponatraemia, renal dysfunction, mild elevations of troponin |
| **Chest X-ray** | Permits assessment of pulmonary congestion and may demonstrate important pulmonary or thoracic causes of dyspnoea |
| **Echocardiography** | Provides extensive information on cardiac anatomy, wall motion and valvular and ventricular function; used to confirm heart failure diagnosis |

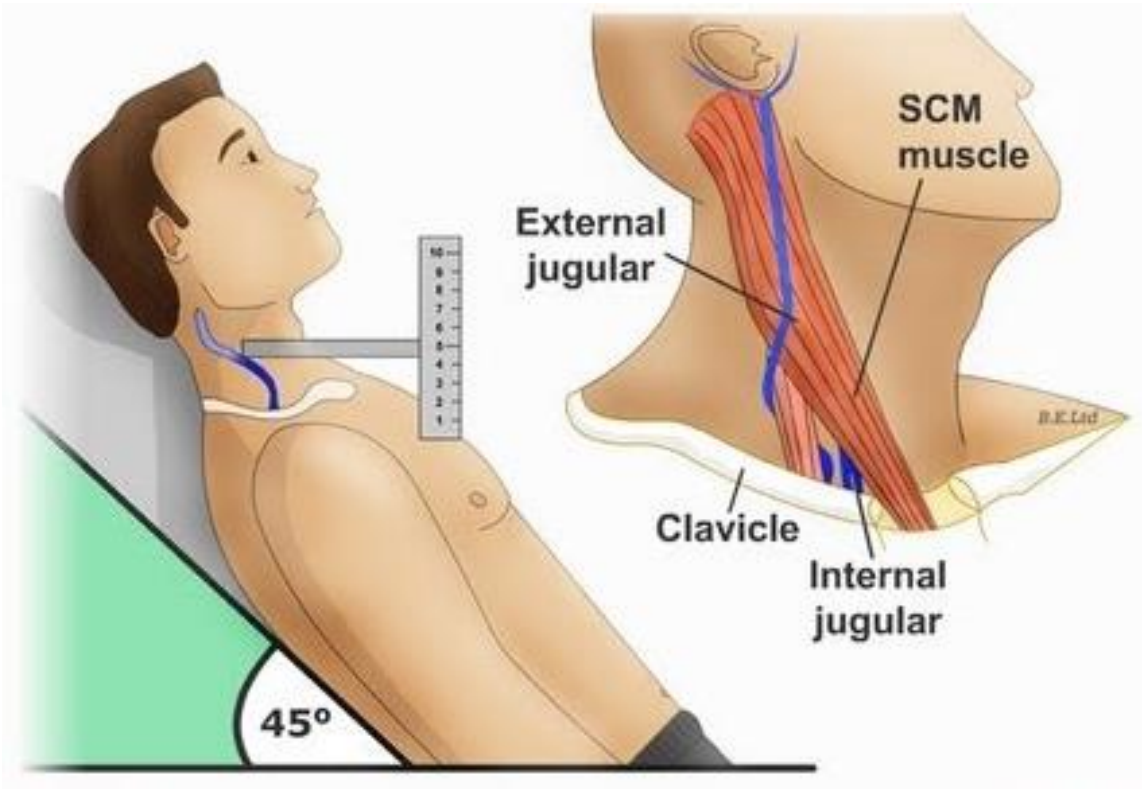
# Elevated jugular venous pressure

**Relevance**[1]

- Jugular venous pressure (JVP) provides an indirect measure of central venous pressure
- The JVP consists of certain waveforms, and abnormalities of these can help to diagnose certain conditions

**Method**[1]

- Patient is positioned at 45° and filling pressure of external jugular vein determined
- Visualise internal jugular vein
- In healthy people, the filling level should be less than 3 centimetres vertical height above sternal angle




1. Reproduced with permission from: Moffatt, J., St George's, University of London, http://www.nataliescasebook.com/tag/jugular-venous-pressure

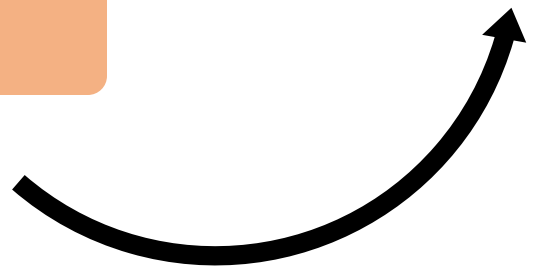
# Electrocardiogram (ECG)

- For all patients with suspected HF[1]
- HF is unlikely in patients with a completely normal ECG (sensitivity 89%)[2]

**Does not exclude diagnosis of HF[1]**

**ECG can identify certain cardiac abnormalities[1]**

- Can provide information on aetiology (eg myocardial infarction)[2]
- May provide indications for therapy[2]
  - Anticoagulation for AF
  - Pacing for bradycardia
  - CRT if broadened QRS complex



1. National Heart Foundation Australia. Guidelines for the prevention, detection and management of chronic heart failure in Australia. 2011. 2. Ponikowski P, et al. Eur Heart J. 2016; doi: 10.1093/eurheartj/ehw128.

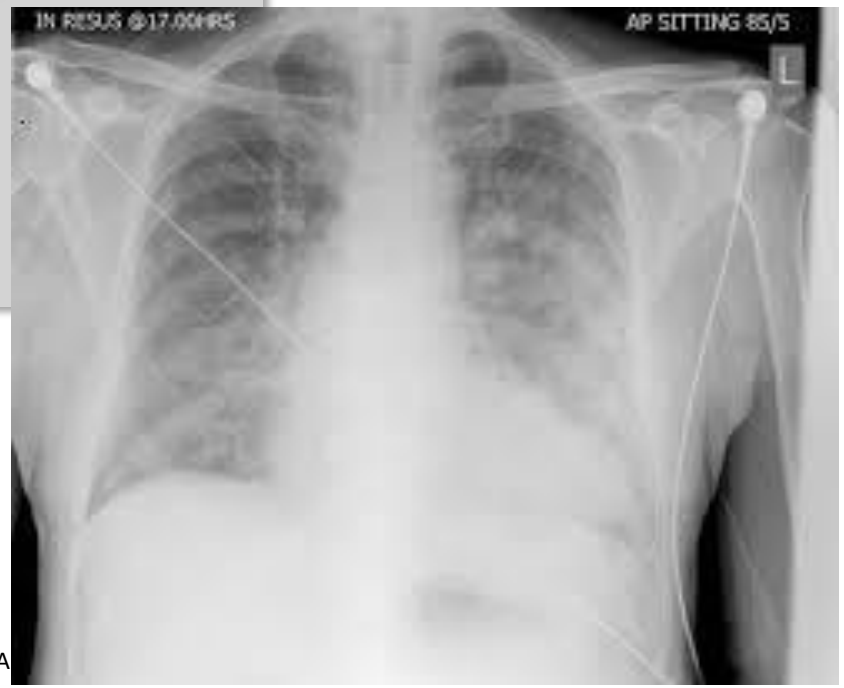
# Chest x-ray

- For all patients with suspected HF
- Important in making diagnosis of HF
- Normal x-ray does not exclude diagnosis

**May reveal alternative explanation for patient symptoms**

**X-ray may reveal signs of worsening HF**

- Interstitial oedema
- Lymphatic oedema due to raised left atrial pressure




1. National Heart Foundation Australia. Guidelines for the prevention, detection and management of chronic heart failure in A

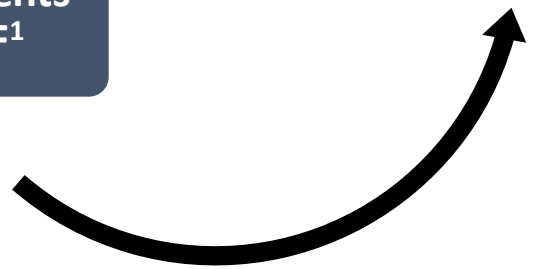
# Echocardiography

- For all patients with suspected HF[1]
- Crucial in establishing diagnosis and determining appropriate treatment[2]

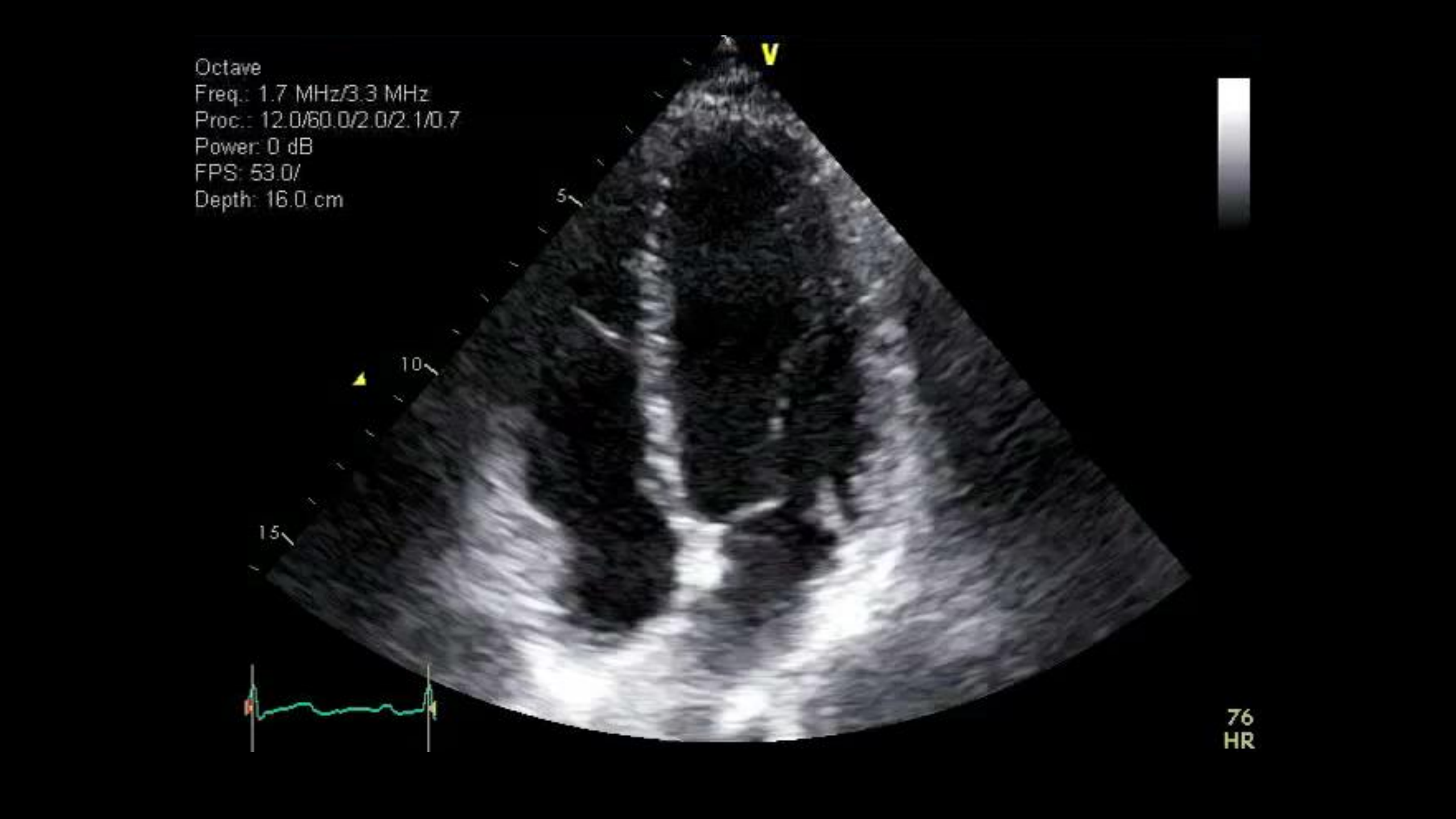
**Single most useful investigation in patients with suspected HF[1]**

**Can identify certain cardiac abnormalities[1,2]**

- Shows chamber volumes, ventricular systolic and diastolic function, wall thickness and motion, valve function, pericardial disease and pulmonary hypertension[1,2]
- May confirm cause of HF[1]



1. National Heart Foundation Australia. Guidelines for the prevention, detection and management of chronic heart failure in A ustralia. 2011.
2. Ponikowski P, et al. Eur Heart J. 2016; doi: 10.1093/eurheartj/ehw128.


Octave
Freq.: 1.7 MHz/3.3 MHz
Proc.: 12.0/60.0/2.0/2.1/0.7
Power: 0 dB
FPS: 53.0/
Depth: 16.0 cm
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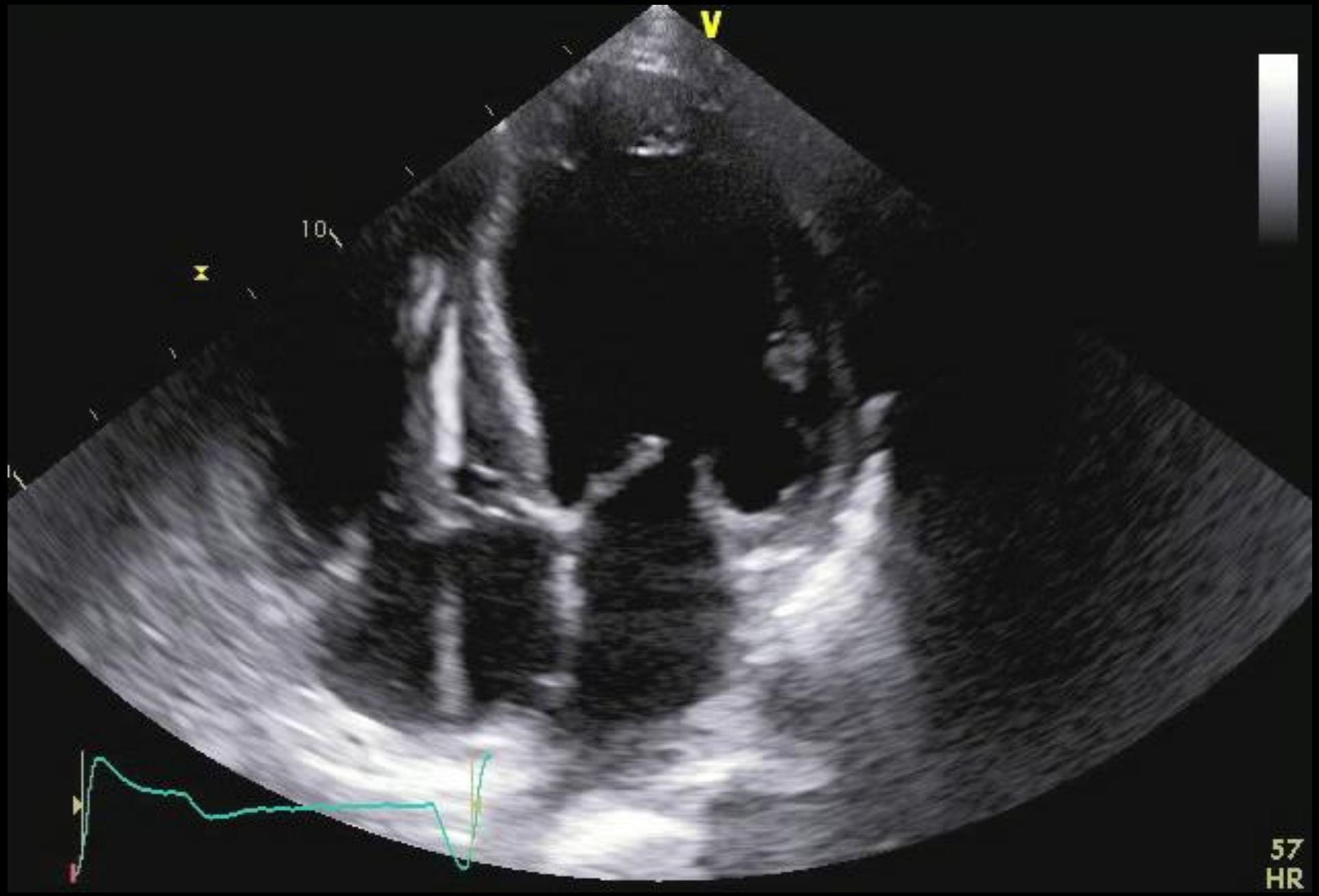
HR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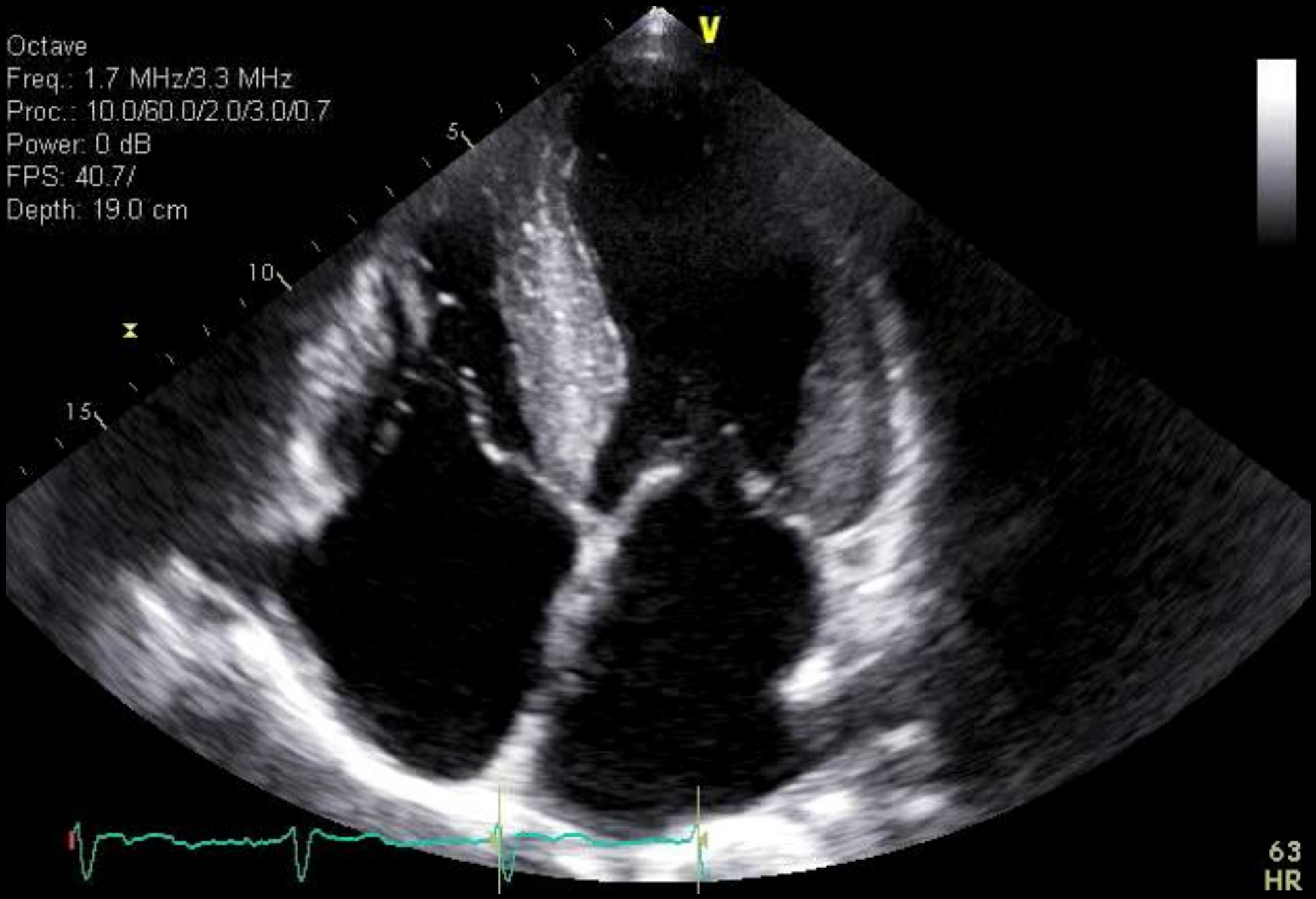
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FPS: 40.7/
Depth: 19.0 cm
5
10
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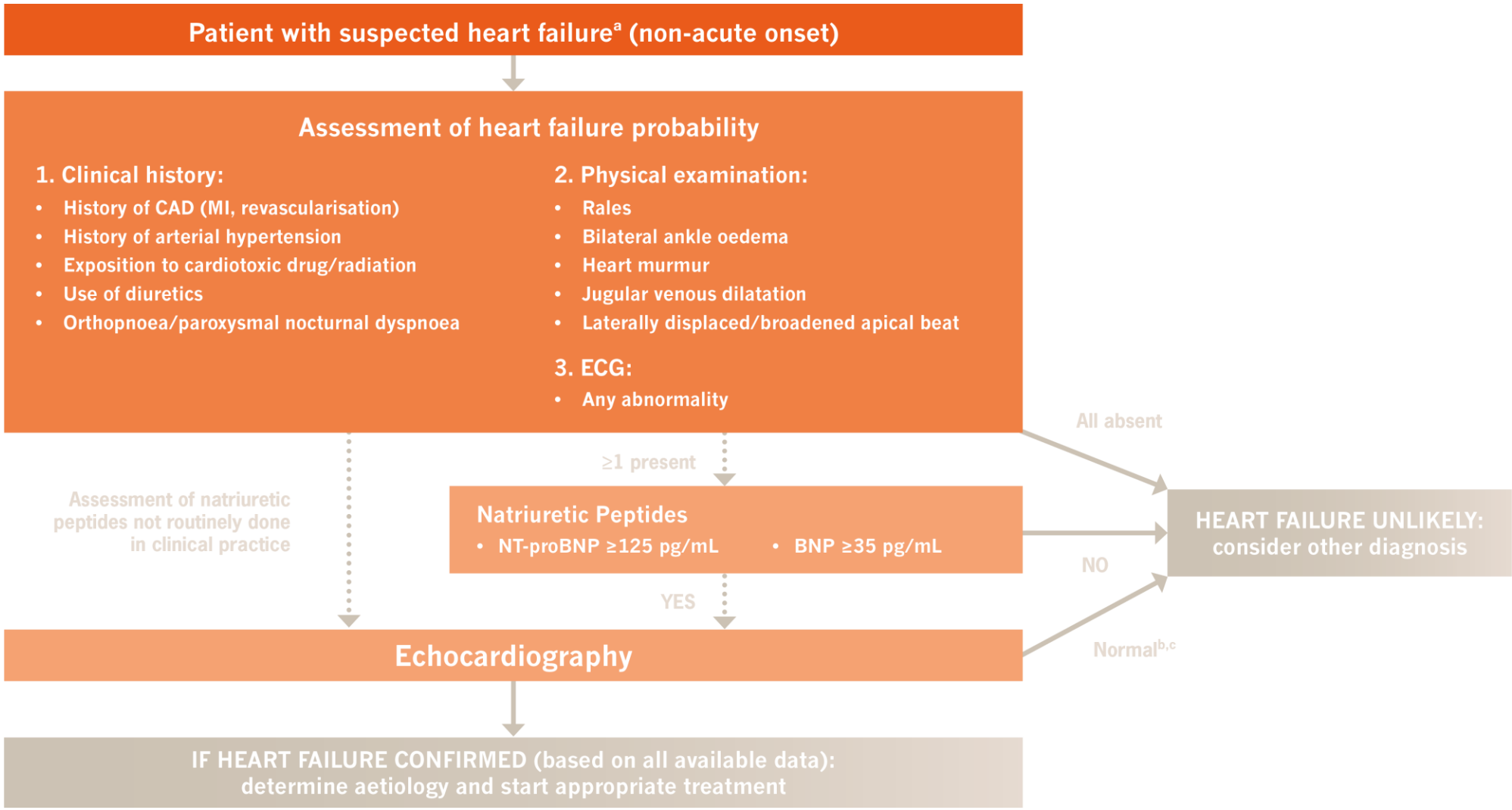
63
HR

# Diagnostic Algorithm for Non-Acute Heart Failure



# What additional tests?

- Coronary angiography
- Right heart catheter
- Endomyocardial biopsy
- Cardiac magnetic resonance imaging (cMRI)

# Spirometry

- For confirmation of alternative causes of HF symptoms and common comorbidities
  - COPD
  - Asthma

- HF impact on spirometry results
  - HF may restrict lung volume[1]
    - Total lung capacity <80%
    - May also indicate other diseases: pleural effusion, pneumonia, pulmonary fibrosis

1. Barreiro T, Perillo I. Am Fam Physician. 2004; 69:1107–1115.

# BNP/NT-pro-BNP Cut-Offs

## BNP

- <100 pg/mL – HF unlikely
- 100–500 pg/mL – HF is possible, but consider other diagnoses
- >500 pg/mL – HF is very likely

**Increase**

Age
Renal Dysfunction
Atrial Fibrillation
ARNIs (BNP not NTproBNP)

**Reduce**

Obesity

## NT-pro-BNP

| Age | HF is unlikely | HF is possible, but consider other diagnoses | HF is very likely |
|---|---|---|---|
| **<50** | <300 pg/mL | 300–450 pg/mL | >450 pg/mL |
| **50–75** | <300 pg/mL | 450–900 pg/mL | >900 pg/mL |
| **>75** | <300 pg/mL | 900–1,800 pg/mL | >1,800 pg/mL |

Moe GW *et al*. Canadian Journal of Cardiology. 2015; 31(1): 3-16

# MBS Item 66829: NTproBNP

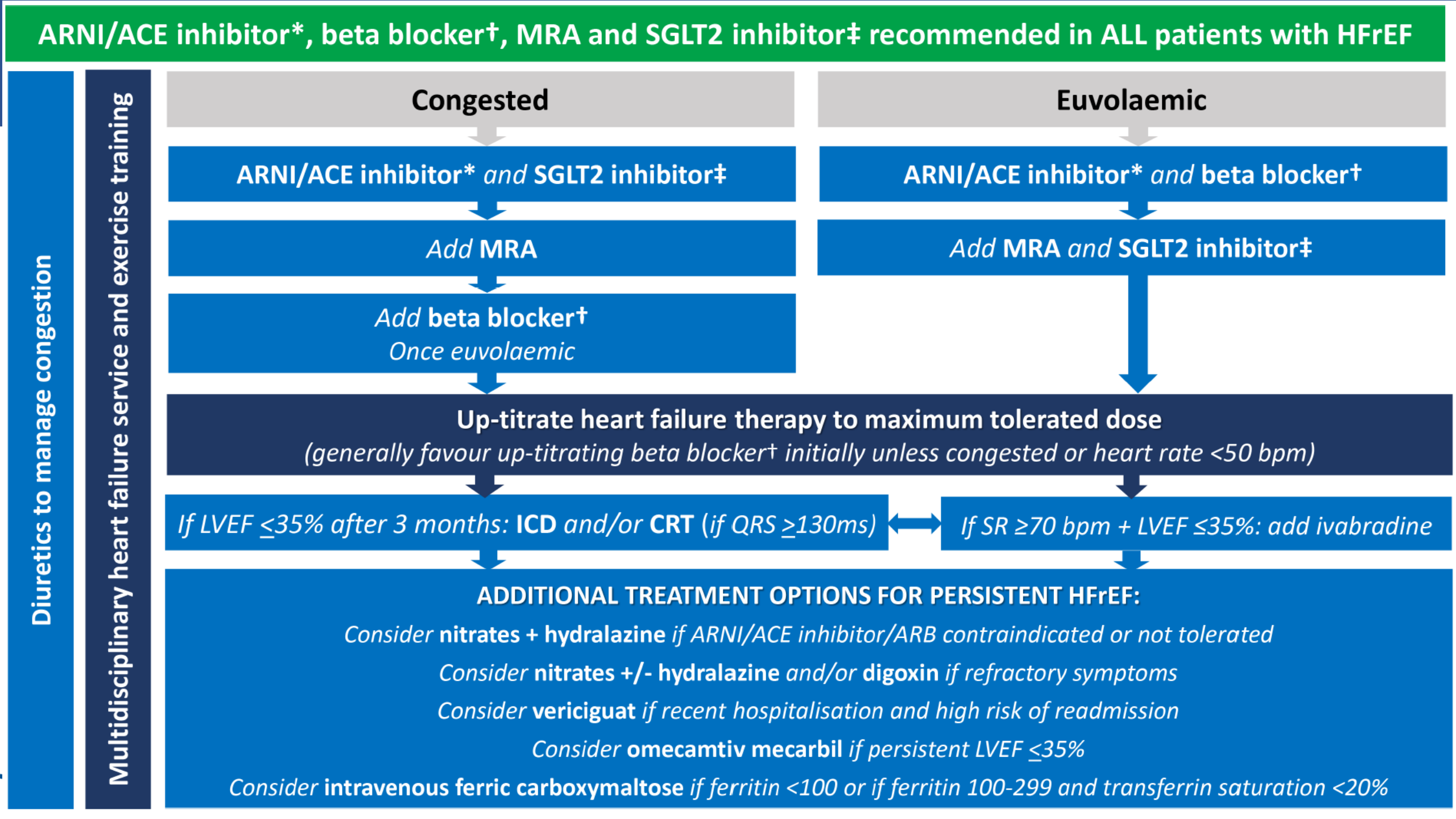
- 1st November 2024
- Quantification of BNP or NT-pro-BNP for the exclusion of a diagnosis of heart failure in patients presenting to a non-hospital setting to assist in decision-making regarding the clinical necessity of an echocardiogram, where heart failure is suspected based on signs and symptoms, but diagnosis is uncertain. Applicable not more than once in a 12-month period.

# Management of Heart Failure Reduced Ejection Fraction

# What are the treatment objectives for chronic HF?

## Objectives of treatment for chronic HF[1]


Adapted from Dickstein *et al.* (2008).[1]
HF: heart failure.
1. Dickstein K *et al. Eur Heart J* 2008;29:2388–442.


ARNI/ACE inhibitor*, beta blocker†, MRA and SGLT2 inhibitor‡ recommended in ALL patients with HFrEF
Diuretics to manage congestion
Multidisciplinary heart failure service and exercise training
Congested
Euvolaemic
ARNI/ACE inhibitor* and SGLT2 inhibitor‡
ARNI/ACE inhibitor* and beta blocker†
Add MRA
Add MRA and SGLT2 inhibitor‡
Add beta blocker†
Once euvolaemic
Up-titrate heart failure therapy to maximum tolerated dose
(generally favour up-titrating beta blocker† initially unless congested or heart rate <50 bpm)
If LVEF ≤35% after 3 months: ICD and/or CRT (if QRS ≥130ms)
If SR ≥70 bpm + LVEF ≤35%: add ivabradine
ADDITIONAL TREATMENT OPTIONS FOR PERSISTENT HFrEF:
Consider nitrates + hydralazine if ARNI/ACE inhibitor/ARB contraindicated or not tolerated
Consider nitrates +/- hydralazine and/or digoxin if refractory symptoms
Consider vericiguat if recent hospitalisation and high risk of readmission
Consider omecamtiv mecarbil if persistent LVEF ≤35%
Consider intravenous ferric carboxymaltose if ferritin <100 or if ferritin 100-299 and transferrin saturation <20%

# Standard of Care from Landmark Clinical Trials

**1987**

**ACE inhibitors[1]**
**CONSENSUS**
enalapril vs placebo
(n=253)

**1991/92**

**ACE inhibitors[2,3]**
**SOLVD**
enalapril vs placebo
(n=6797)

**1999**

**Beta-blockers[4]**
**CIBIS-II**
bisoprolol vs placebo
(n=2647)

**Beta-blockers[5]**
**MERIT-HF**
metoprolol vs placebo
(n=3991)

**Aldosterone antagonists[6]**
**RALES**
spironolactone vs placebo
(n=1663)

**2001**

**ARBs[7]**
**Val-HeFT**
valsartan vs placebo
(n=5010)

**Beta-blockers[8]**
**CAPRICORN**
carvedilol vs placebo
(n=1959)

**Beta-blockers[9]**
**COPERNICUS**
carvedilol vs placebo
(n=2289)

**2003**

**ARBs[10]**
**CHARM-Alternative**
candesartan vs placebo
(n=2028)

**2004**

**Nitrates/vasodilators[11]**
**A-HeFT**
isosorbide dinitrate/
hydralazine vs placebo
(n=1050)

**SHIFT (2010)**
6,558 patients
***($I_f$ inhibitor) vs. placebo:***
18% ↓ CV death or heart failure hospitalisation

**2011**

**Aldosterone antagonists[12]**
**EMPHASIS-HF**
eplerenone vs placebo
(n=2737)

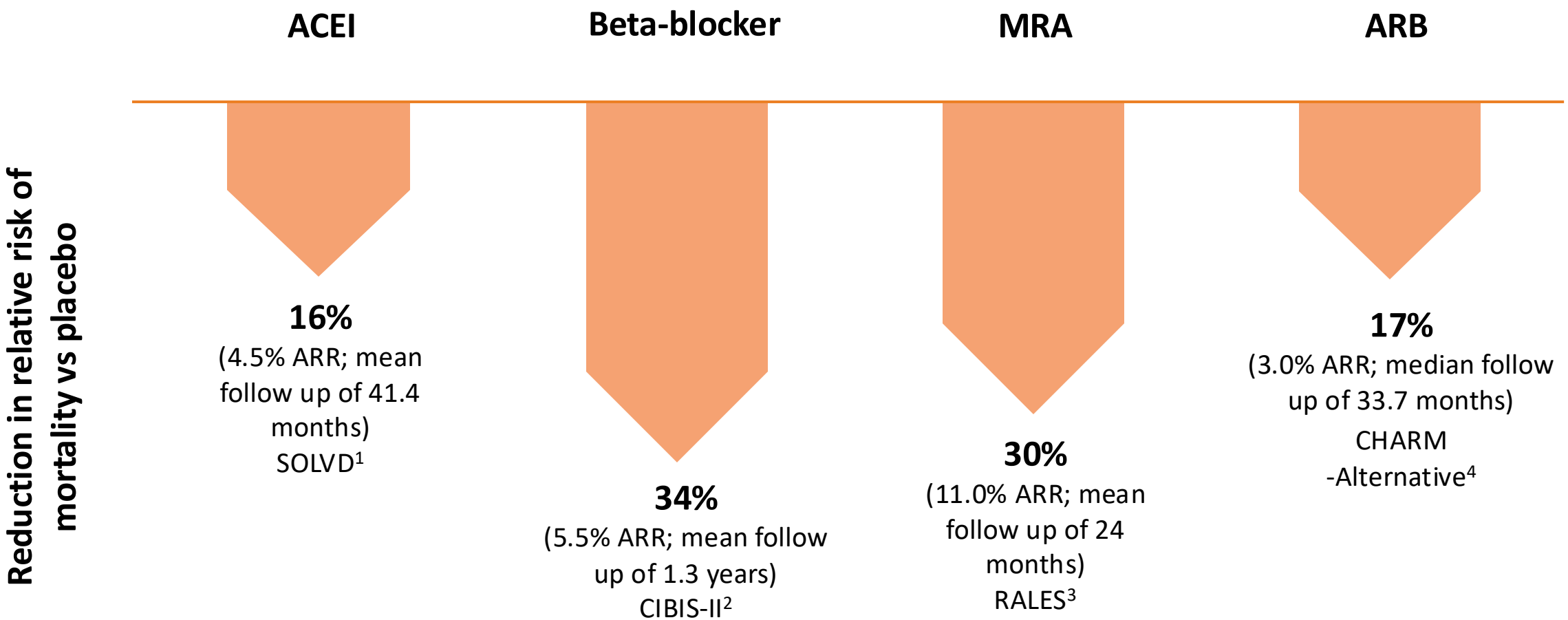
**PARADIGM-HF (2014)**
8,442 patients
***(ARNI ) vs. ACEi:***
20% ↓ CV mortality or heart failure hospitalisation

1. The CONSENSUS Trial Study Group. *N Engl J Med* 1987;316(23):1429–35. 2. The SOLVD Investigators. *N Engl J Med* 1991;325(5):293–302. 3. The SOLVD Investigators. *N Engl J Med* 1992;327(10):685–91. 4. The CIBIS-II Investigators. *Lancet* 1999;353(9146):9–13. 5. MERIT-HF Working Group. *Lancet* 1999;353(9169):2001–7. 6. Pitt B *et al. N Engl J Med.* 1999;341(10):709–17. 7. Cohn J *et al. N Engl J Med* 2001;345(23):1667–75. 8. Dargie HJ. *Lancet* 2001;357(9266):1385–90. 9. Packer M *et al. N Engl J Med* 2001;344(22):1651–8. 10. Granger CB *et al. Lancet.* 2003;362(9386):772–6. 11. Taylor AL *et al. N Engl J Med* 2004;351(20):2049–57. 12. Zannad F *et al. N Engl J Med* 2011;364(1):11–21.

**DAPA HF (2019)**
4,700 patients
**Dapagliflozin *vs. placebo:***
17% ↓ all-cause mortality 30% reduction HF hospitalisation

**EMPEROR-Reduced (2020)**
3,700 patients
**Empagliflozin *vs. placebo:***
30% reduction HF hospitalisation

# Standard Therapy Improves Survival





1. The SOLVD Investigators. *N Engl J Med* 1991;325(5):293–302
2. The CIBIS-II Investigators. *Lancet* 1999;353(9146):9–13.
3. Pitt B *et al. N Engl J Med.* 1999;341(10):709–17.
4. Granger CB *et al. Lancet.* 2003;362(9386):772–6.

# SGLT2i Meta-analysis

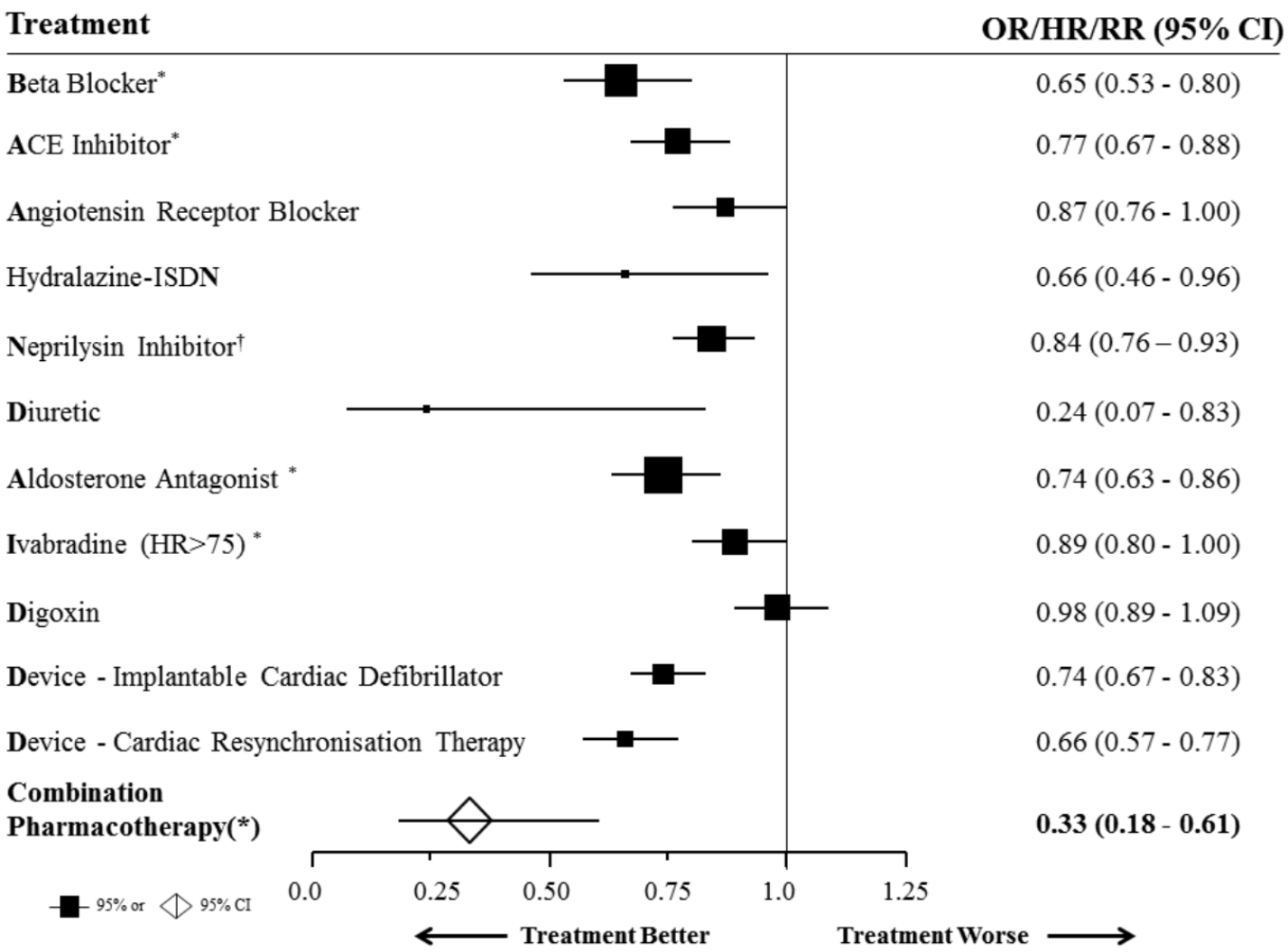
- Significant relative risk reductions in
  - all-cause mortality (13%)
  - cardiovascular mortality (14%)
  - first hospitalisation for heart failure (31%)
  - first kidney composite event (38%)
- HF benefits in absence of background therapy

Lancet 2020; 396: 819-829.

**TABLE 15** Benefits of Evidence-Based Therapies for Patients With HFrEF (3-6,8,10-14,23,31-42)

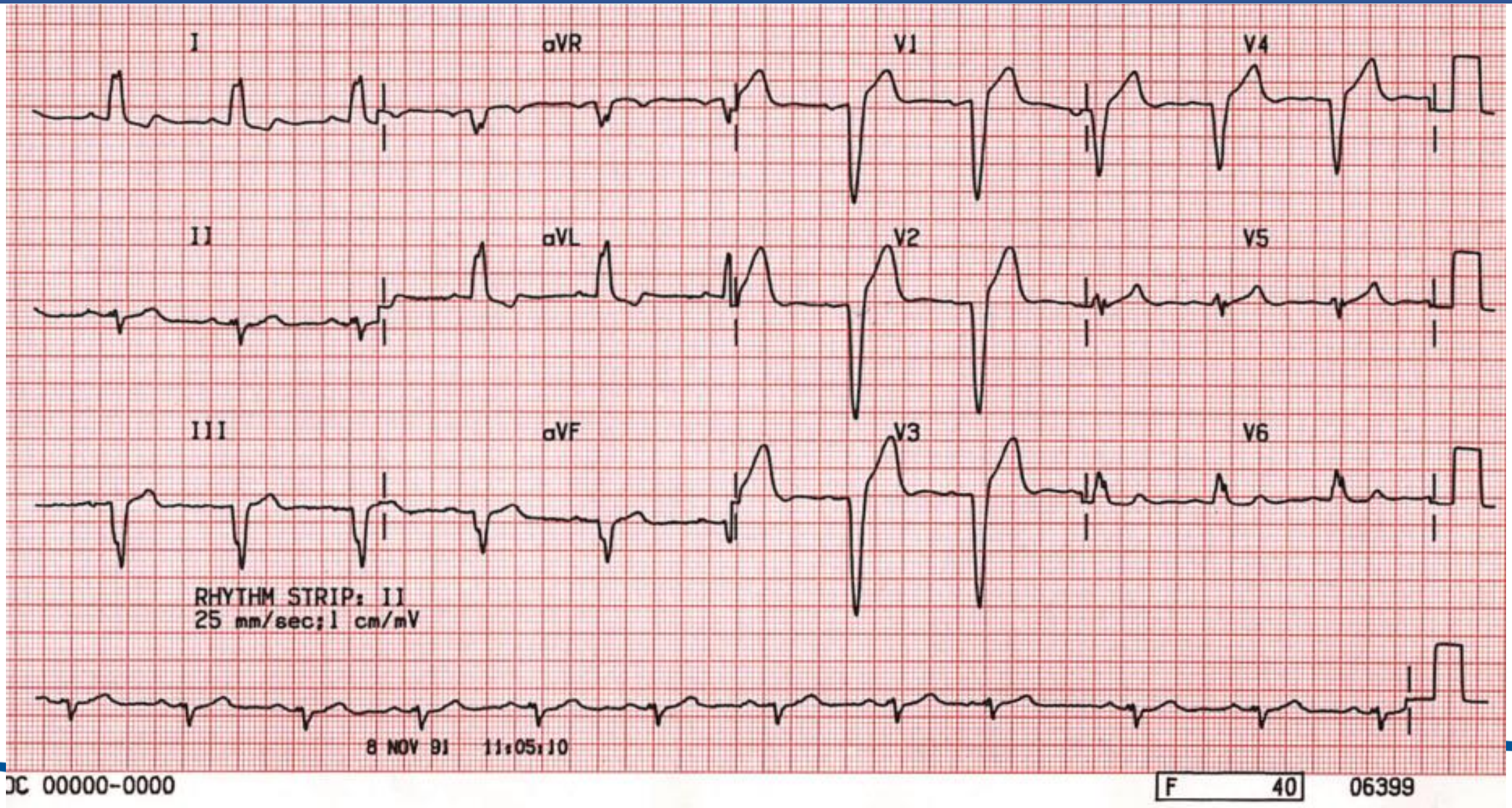
| Evidence-Based Therapy | Relative Risk Reduction in All-Cause Mortality in Pivotal RCTs, % | NNT to Prevent All-Cause Mortality Over Time* | NNT for All-Cause Mortality (Standardized to 12 mo) | NNT for All- Cause Mortality (Standardized to 36 mo) |
|---|---|---|---|---|
| ACEi or ARB | 17 | 22 over 42 mo | 77 | 26 |
| ARNi† | 16 | 36 over 27 mo | 80 | 27 |
| Beta blocker | 34 | 28 over 12 mo | 28 | 9 |
| Mineralocorticoid receptor antagonist | 30 | 9 over 24 mo | 18 | 6 |
| SGLT2i | 17 | 43 over 18 mo | 63 | 22 |
| Hydralazine or nitrate‡ | 43 | 25 over 10 mo | 21 | 7 |
| CRT | 36 | 12 over 24 mo | 24 | 8 |
| ICD | 23 | 14 over 60 mo | 70 | 23 |

# BANDAID$^2$S

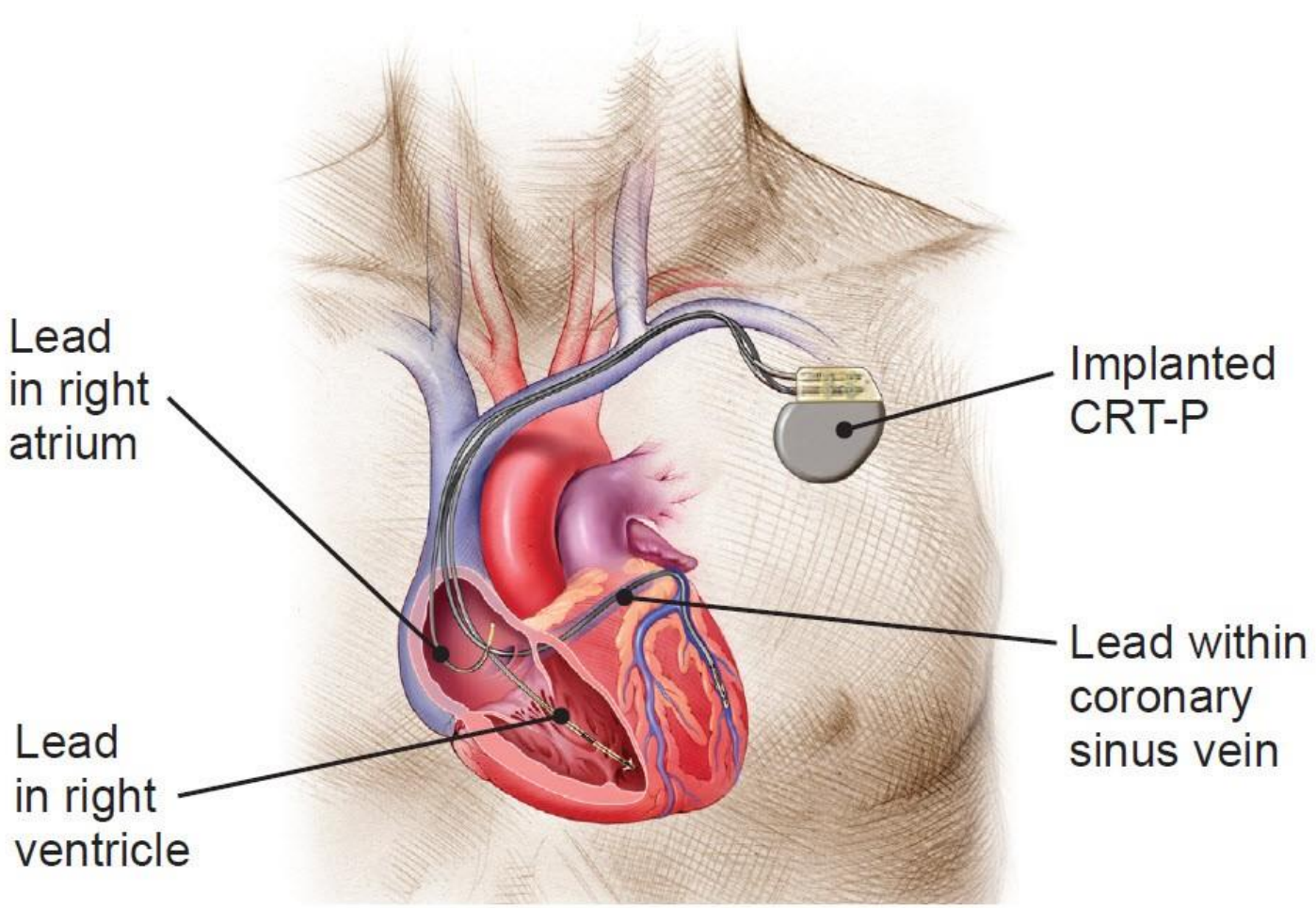


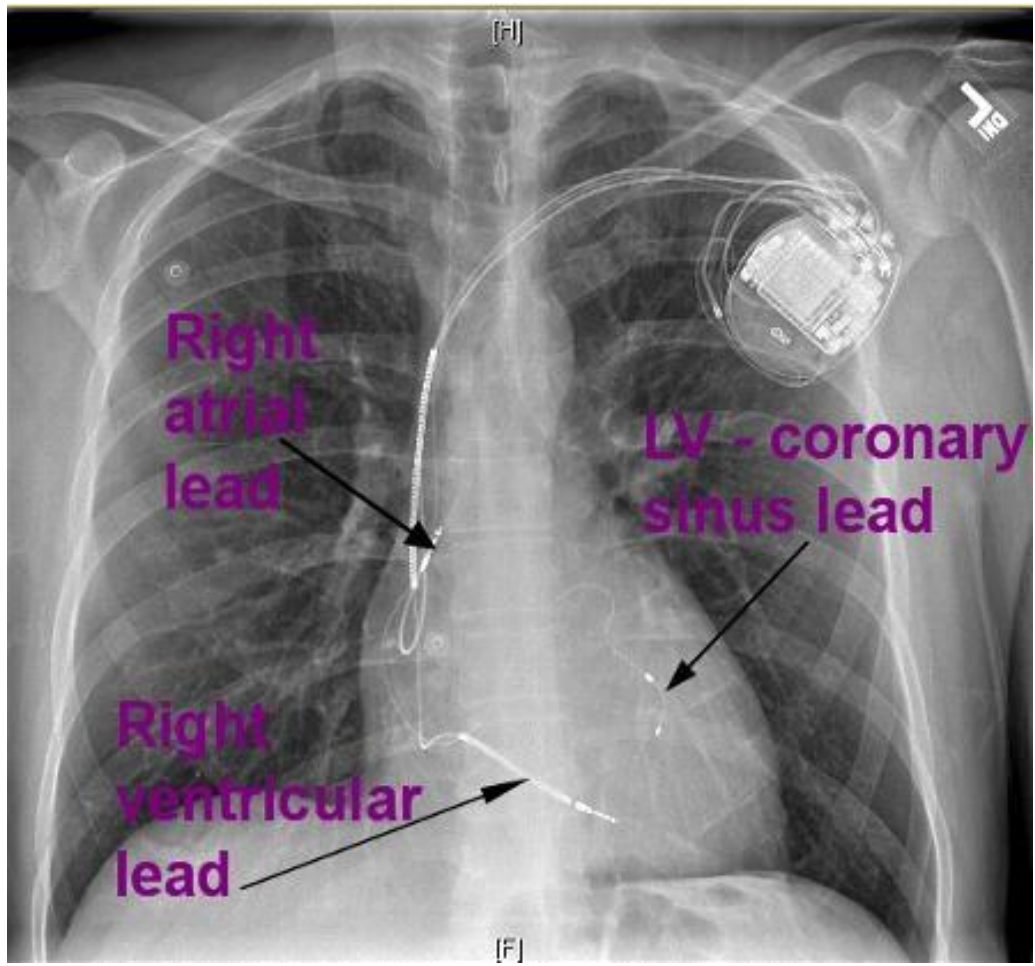

Intern Med J. 2016 Jun;46(6):653-62. doi: 10.1111/imj.12839

# LBBB


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec;1 cm/mV
8 NOV 91 11:05:10
OC 00000-0000
F 40 06399

# Cardiac Resynchronisation Therapy (CRT)

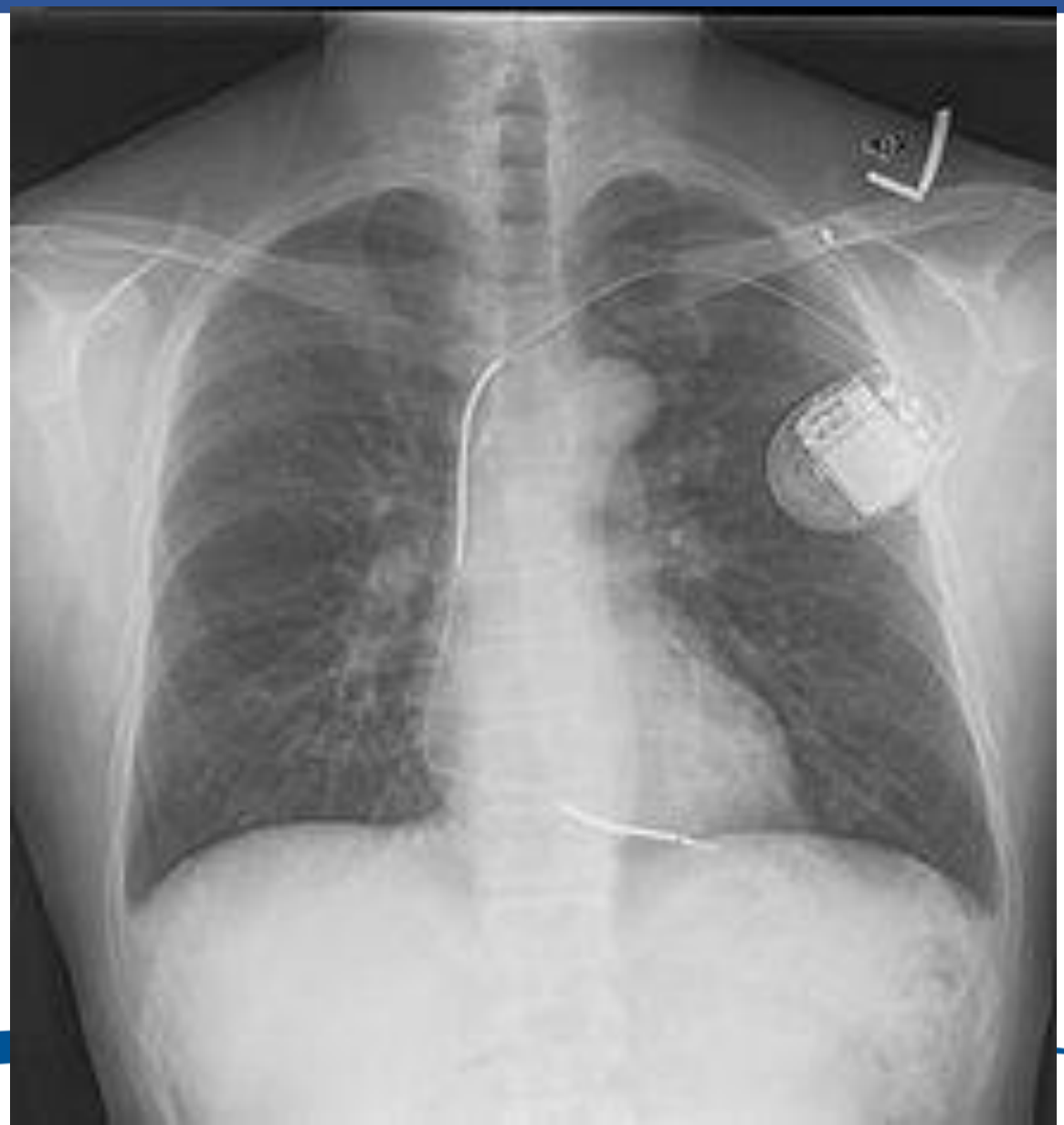


# Implantable Defibrillator

- Primary Prevention
- Secondary Prevention

- Antitachycardic Pacing (ATP)
- Defibrillation



# Australian Guidelines for Heart Failure (2018) Recommend a Number of Non- and Pharmacological Interventions[1]

## Non-pharmacological management recommendations

- Multi-disciplinary care team for the patient
- Physical activity program
- Restrict dietary sodium to <2 g/day
- Fluid limitation (1–1.5 L/day) appropriate to symptom severity
- Cease smoking
- Limit alcohol intake to 10–20 g/day – ideally nil
- Daily weighing – advised to seek medical attention if >2 kg gained in a two-day period
- Vaccinations against pneumococcal disease and influenza
- For obese patients – weight loss
- Address sleep apnoea
- Diet with reduced saturated fat intake and high fibre
- Limit caffeine

## Pharmacological management recommendations

- ACEI or ARB
- Beta-blocker
- Diuretic
- Mineralocorticoid receptor antagonist
- Direct sinus node inhibition
- Digoxin

**1.** Atherton JJ et al. Heart Lung Circ 2018; 27: 1123–1208

# Keep heart failure in mind

## when managing your comorbid patients

### Who is the typical Australian HF patient?

- 75% of HF patients are NYHA class II–IV[a1]
- 1 in 4 have been admitted to hospital in past year[a1]
- HF prevalence increases with age (13.9% of those aged ≥75 years)[a1]

Persistent symptoms despite treatment of comorbidities may be a sign that current chronic HF therapy should be re-evaluated[5]

### 94.5% of HF patients have two or more other chronic conditions[b2]

**Prevalence of comorbidity in patients with HF[3]**

**HYPERTENSION:** ~66%

**ATRIAL FIBRILLATION (AF):** up to 50%[c]
(AF is a common precipitant of HF, and conversely, HF is the strongest predictor for AF)

**CORONARY ARTERY DISEASE:** up to 50%
(most common cause of incident HF-rEF: 16.5% incidence of HF at 1 year post AMI[d4])

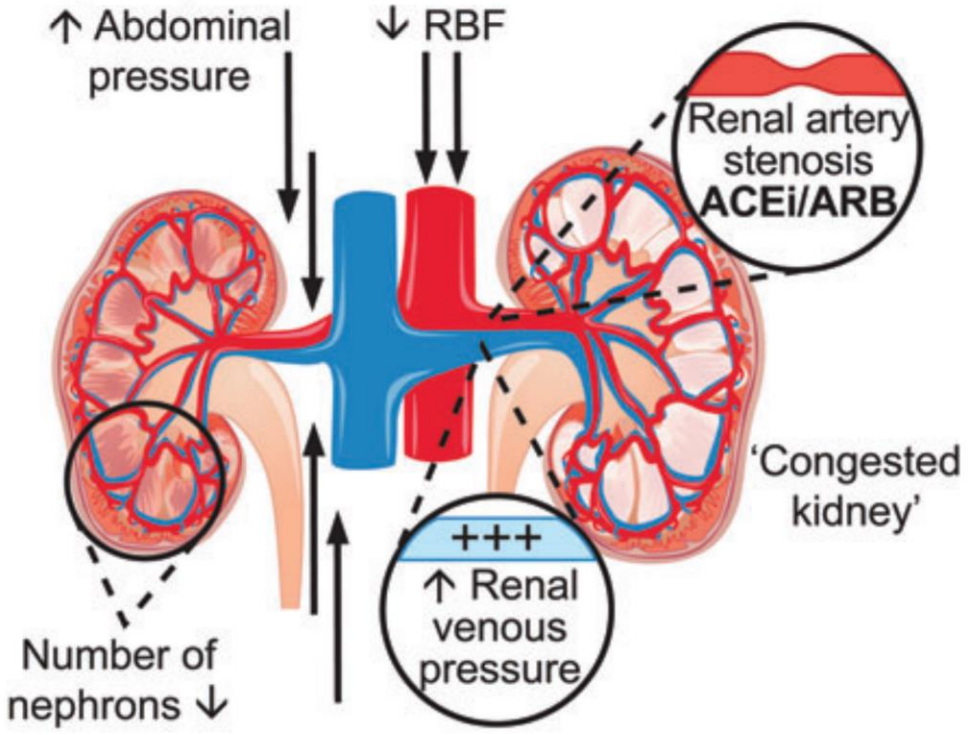
**CHRONIC KIDNEY DISEASE (CKD):** >60%
(~30% with moderate to severe[e] CKD)

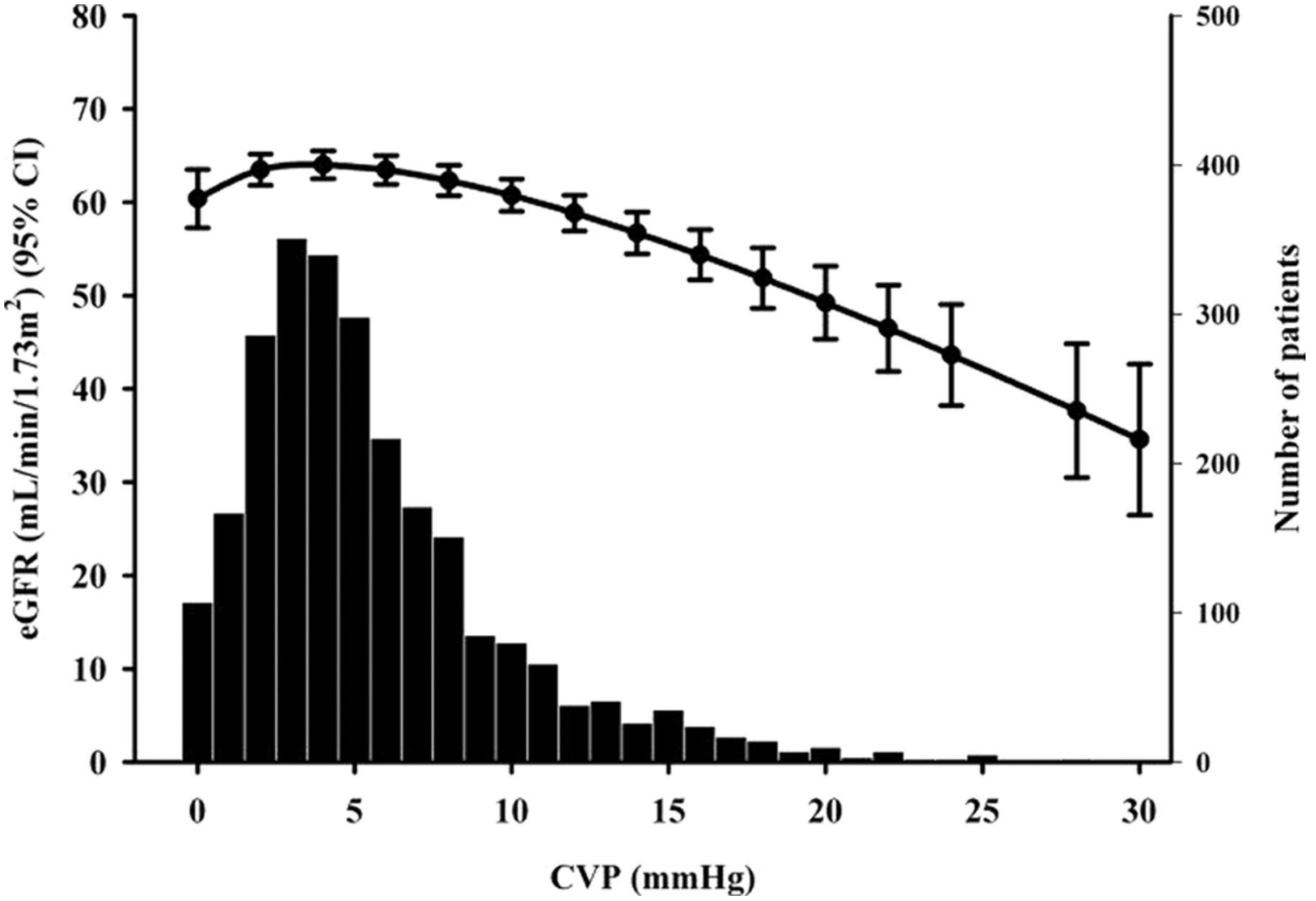
**COPD:** ~20%

**DIABETES:** 30–40%

**SLEEP-DISORDERED BREATHING:** 50–75%

# Relationship Between CVP and eGFR




Bock & Gottlieb. Circulation. 2010;121:2592-2600

# Iron Deficiency

- **Fe essential**
  - Oxygen uptake, transport and storage
  - Oxidative metabolism in skeletal and cardiac muscle
  - Erythropoiesis
- **Clinical consequences in absence of anaemia**
  - Repletion of Fe in those without anaemia improves cognitive, symptomatic, and exercise performance.

# Anaemia

| COR | LOE | Recommendations | Comment/ Rationale |
|---|---|---|---|
| **IIb** | **B-R** | In patients with NYHA class II and III HF and iron deficiency (ferritin <100 ng/mL or 100 to 300 ng/mL if transferrin saturation is <20%), intravenous iron replacement might be reasonable to improve functional status and QoL. | NEW: New evidence consistent with therapeutic benefit. |
| **III: No Benefit** | **B-R** | In patients with HF and anemia, erythropoietin-stimulating agents should not be used to improve morbidity and mortality. | NEW: Current recommendation reflects new evidence demonstrating absence of therapeutic benefit. |

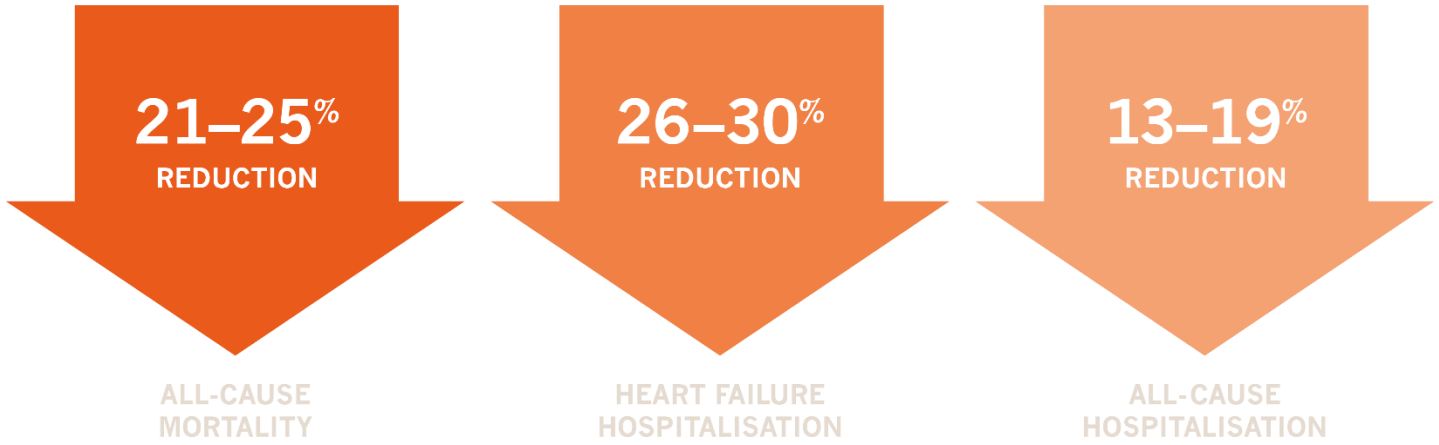
Yancy, *et. al*. *Circulation*. 2017. DOI:10.1161/CIR.509/-/DC1

# Withdrawal of Treatment?

| COR | LOE | RECOMMENDATION |
|---|---|---|
| 1 | B-R | 1. In patients with HFimpEF after treatment, GDMT should be continued to prevent relapse of HF and LV dysfunction, even in patients who may become asymptomatic (1). |

# Multidisciplinary Strategies Optimise the Management of Patients with Heart Failure and Improve Patient Outcomes

- **Coordination of care** along the continuum of heart failure is crucial to achieving the goal of heart failure management – providing a 'seamless' system of care, **optimising the management** of patients[1]
- Multidisciplinary management programs have been reported to reduce rates of **heart failure hospitalisation, all-cause hospitalisation and mortality** in patients with heart failure when compared with usual care[2-4]




1. McMurray JJ *et al. Eur Heart J* 2012;33:1787–847.
2. Yancy CW *et al. J Am Coll Cardiol* 2013;62:e147–239.
3. Holland R *et al. Heart* 2005;91:899–906.
4. McAlister FA *et al. J Am Coll Cardiol* 2004;44:810–9.

# GPs ARE WELL PLACED TO IDENTIFY

## patients with HF-rEF who are symptomatic on their current treatment[1]

The CSANZ encourages a collaborative 'shared care' model between GPs and specialists:

"**GPs have a vital role in the management of patients with heart failure in the community**"[2]

CSANZ Heart Failure Guidelines

A patient with congestive heart failure will see their GP 12 times per year on average[1]

**Symptoms of fatigue and/or breathlessness in a patient with HF-rEF should be a red flag**

- The signs and symptoms of heart failure can be subtle and non-specific, and may be mistaken for other health conditions or old age – patients themselves may not even recognise them
- Regularly questioning your heart failure patients about their symptoms can help you to identify when their heart failure treatment may need to be reviewed

If you are not asking about their HF symptoms, then who will?

# TIPS FOR IDENTIFYING SYMPTOMATIC HF-rEF[1]

**Ask your patients about their symptoms:**

- Do you need to sleep propped up on pillows to breathe easier?
- Do you struggle to catch your breath walking up stairs?
- Do you have swollen feet or ankles at the end of the day?
- Do you cough, even when you don't have a cold?
- Do you no longer do the things you used to enjoy due to exhaustion?

**ASK ABOUT**

CHECK FOR

EVALUATE

REVIEW

# TIPS FOR IDENTIFYING SYMPTOMATIC HF-rEF[1]

**Check for:**

- Peripheral oedema – press the skin of the ankles to detect pitting
- Increased use of diuretics to control symptoms

ASK ABOUT

**CHECK FOR**

EVALUATE

REVIEW

# TIPS FOR IDENTIFYING SYMPTOMATIC HF-rEF[1]

**Are the symptoms/signs evidence of worsening HF-rEF?**

- Consider additional investigations for underlying causes (such as worsening comorbidities)*

**If you suspect symptomatic HF-rEF despite treatment:**

- Is a repeat ECHO and/or cardiologist referral needed?
- Re-evaluate current HF-rEF treatment – does it need to be adjusted/intensified?

ASK ABOUT

CHECK

**EVALUATE**

REVIEW

# TIPS FOR IDENTIFYING SYMPTOMATIC HF-rEF[1]

**Review every 6–12 months once stabilised, or following a change in clinical status:**

- Symptom assessment
- Serum biochemistry (electrolytes, urea, creatinine, and glucose)
- Full blood count

ASK ABOUT

CHECK

EVALUATE

**REVIEW**

# Management of Heart Failure Preserved Ejection Fraction

# Principles of Management in HFpEF

- A: Avoid tachycardia
  - Digoxin or beta blockers with atrial fibrillation (restore and maintain SR)
- B: Blood pressure control
  - ACEi, ARBs and MRA may be of greatest benefit
- C: Comorbid condition treatment
  - Manage obesity, sleep apnoea, pulmonary disease, anaemia, ischaemia
- D: Diuretics to relieve congestion
  - Judicious use of loop diuretic with careful monitoring of renal function
- E: Exercise training encouraged
  - Improves exercise capacity, physical function and QoL[1]

1. Circ Heart Fail 2014; 8: 33-40

# Guideline Recommendations

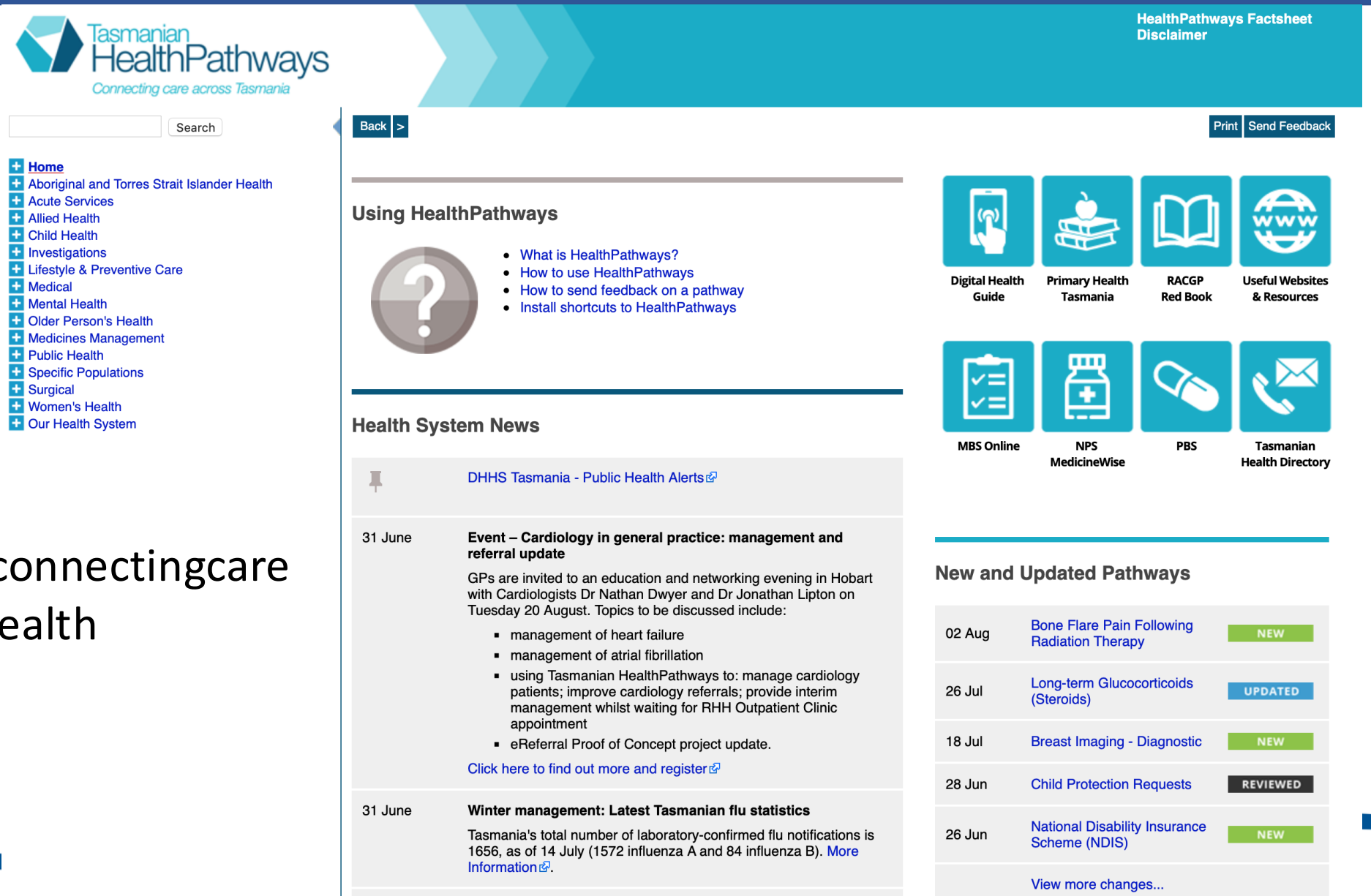
- Current guidelines (2022 AHA/ACC/HFSA Heart Failure guidelines) recommend **SGLT2 inhibitors** as a class for patients with HFpEF to reduce the risk of hospitalization and improve symptoms, without specifically favouring one over the other.

# Guidelines for HFpEF (LVEF ≥ 50%): Focus on Management of Symptoms and Co-Morbidities

| ACCF/AHA recommendations for the treatment of HF-pEF | Class of recommendation | Level of evidence |
|---|---|---|
| Systolic and diastolic blood pressure should be controlled according to published clinical practice guidelines | **I** | **B** |
| Diuretics should be used for relief of symptoms due to volume overload (irrespective of LVEF) | **I** | **C** |
| Coronary revascularisation for patients with CAD in whom angina or demonstrable myocardial ischemia is present despite GDMT | **IIa** | **C** |
| Management of AF according to published clinical practice guidelines for HF-pEF to improve symptomatic HF | **IIa** | **C** |
| Use of beta-blockers, ACEIs and ARBs for hypertension in HF-pEF | **IIa** | **C** |
| ARBs might be considered to decrease hospitalisations in HF-pEF | **IIb** | **B** |
| Nutritional supplementation is not recommended in HF-pEF | **III: no benefit** | **C** |

Yancy CW *et al. J Am Coll Cardiol* 2016. doi:10.1016/j.jacc.2016.05.011

# https://tasmania.healthpathways.org.au




Username: connectingcare
Password: health

TASMANIAN HEALTH SERVICE | Tasmanian Government

# HEART FAILURE MEDICATION TITRATION PLAN

STATEWIDE

FACILITY: _____

| PT ID | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|

SURNAME..... D.O.B.....
OTHER NAMES.....
ADDRESS.....
.....

Attach Patient Sticker Label

*(Tick ☑ as appropriate, format time as 00:00 (24 hour) and date as DD/MM/YYYY)*

**RECENT RESULTS**

| Date | EF % | Weight kg | eGFR mL/min | Potassium (K+) mmol/L | BP mmHg | HR bpm |
|---|---|---|---|---|---|---|
| DD / MM / YYYY | | | | | | |

**MONITORING RECOMMENDATIONS** *(see overleaf for guidance)*

- Check blood pressure (BP) including postural drop and heart rate (HR) each visit
- ACEI/ARB/ARNI/MRA*: check serum potassium (K+), renal function 1 to 2 week(s) after commencing or titrating (if K+ is high recheck in 48 hours). For MRAs check every 4 weeks for 12 weeks, at 6 months, then 6-monthly
- SGLT2i*: Before commencing check volume status and for type 1 diabetics seek endocrinologist approval
- Diuretic dose changes beyond 3 days require medical review and checking of blood chemistry and volume status
- Iron: Order Hb*, CRP*, ferritin and transferrin saturation at first assessment and every 3 to 6 months if iron deficient

| The 4 drug classes that reduce heart failure mortality and morbidity | | Combination therapy is more effective than a single medication at a higher dose BUT avoid simultaneous up titration | | |
|---|---|---|---|---|
| **Class*** | **Medication Name** | **Current Dose/ Frequency** | **Target Dose/ Frequency** | **Schedule/Instructions** |
| **ACEI**<br>**ARB**<br>**ARNI** | | mg | mg | Washout for 36 hours or more if switching from ACEI to ARNI or vice versa<br>Increase dose by: mg every weeks(s) |
| **Beta-Blocker** | ☐ Bisoprolol<br>☐ Carvedilol<br>☐ Metoprolol XL<br>☐ Nebivolol | mg | mg | Increase dose by: mg every weeks(s) |
| **MRA** | ☐ Eplerenone<br>☐ Spironolactone | mg | mg | Increase dose once stable on other heart failure medications. |
| **SGLT2i** | ☐ Dapagliflozin<br>☐ Empagliflozin | mg | N/A | A transient fall in eGFR (up to 30%) is common and not usually clinically significant. Withhold if perioperative or unwell/fasting. |

**MEDICATIONS THAT PROVIDE SYMPTOM RELIEF**

| | | |
|---|---|---|
| **Diuretic** | ☐ Furosemide ☐ Bumetanide<br>☐ Patient has a diuretic action plan | Adjust diuretic dose according to clinical assessment (for example increase dose 50 –100% if fluid overloaded) |
| **Iron Infusion** | Date of infusion (if given): DD / MM / YYYY (oral iron is ineffective with heart failure)<br>☐ Check iron studies (see monitoring above). Give an iron infusion if ferritin is less than100 mcg/L **OR** 100 to 299 mcg/L with a transferrin saturation below 20%. Contact hospital if unable to provide infusion | |

**NOTES**

| | |
|---|---|
| Consultant *(print name)*: | **Heart Failure Service** |
| Authorised by *(print name)*: | Name: |
| Authoriser signature: | |
| Designation: Date: DD / MM / YYYY | Phone Number: |

HEART FAILURE MEDICATION TITRATION PLAN



**Patient** *(print name)*:_____ DOB: DD / MM / YYYY PT ID

**Medications that may cause or worsen HF**

Non-steroidal anti-inflammatories, cyclooxygenase-2 inhibitors; centrally acting calcium channel blockers (verapamil, diltiazem), corticosteroids, tricyclic antidepressants, saxagliptin, moxonidine, thiazolidinediones (glitazones)

**Hypotension**

Asymptomatic hypotension usually requires no change in therapy (unless systolic BP is consistently less than 90mmHg).

**Symptomatic hypotension**

I. Stop or reduce calcium-channel blockers and/or other vasodilators unless essential for example for angina.

II. Consider reducing diuretic dose if there are no signs or symptoms of congestion.

III. Temporarily reduce ACEI, ARB, ARNI or beta-blocker dose if above measures do not work, Avoid abrupt cessation of beta blockers unless patient is in shock*.

IV, Review patient within a week and seek specialist advice if the above measures do not work.

* For severe hypotension or shock, refer to hospital emergency department (ED).

**Worsening renal function**

**Cautions for renal function**

- Caution with ARNI if eGFR is less than 30mL/min.
- eGFR does not accurately reflect renal function where body weight is very low (tending to overestimate) or when volume change is rapid.
- Where there is severe dehydration, sepsis, or medication induced nephrotoxicity refer to ED. Consider withholding MRA first, then SGLT2i, followed by ACEI, ARB or ARNI until patient is reviewed,

**After commencing or titrating therapy:**

- Expect a rise in creatinine, urea, and potassium (K+) for ACEI, ARB, ARNI, or MRA. A decline in eGFR up to 30% is acceptable if it stabilises within 2 weeks (or 4 to 12 weeks for SGLT2i).
- If eGFR declines by more than 30%, review fluid status and nephrotoxic medications and seek specialist advice about safety of continuing therapy.

**Congestion or peripheral oedema**

- Increase the diuretic dose, then gradually reduce beta-blocker dose (avoiding abrupt cessation).
- Liaise with the heart failure service and review the patient daily or weekly (as appropriate).
- Seek specialist advice if symptoms do not improve. If deterioration is severe, refer patient to ED.

**Bradycardia**

- Where HR is less than 50 beats per minute, and the patient is on a beta-blocker, review the need for other drugs that slow heart rate (for example digoxin, amiodarone) in consultation with specialist; and arrange ECG to exclude heart block.
- Consider reducing beta-blocker (avoiding abrupt cessation) if bradycardia is symptomatic,
- If pacemaker is present, seek specialist review.

**Hyperkalaemia**

Monitor K+ for ACEI, ARB, ARNI and MRA, Urgently check K+, creatinine and urea for dehydration or sepsis.

If serum K+ is:

- 5.0 to 5.5 mmol/L reduce or withhold K+ supplements and check diet
- 5.6 to 5.9 mmol/L perform ECG and withhold K+ supplements and reduce K+ retaining agents especially MRAs (less so for ARNI, ACEI and ARB)
- 6 mmol/L or more, urgently seek specialist advice
- Recurrently high, seek specialist advice

**Volume depletion**

SGLT2i, MRA and ARNI have a mild diuretic effect,

Assess volume status before commencing or adjusting doses and reduce the dose of loop diuretic in euvolaemic patients if required.

**Cough**

- Exclude pulmonary oedema or reflux as a cause if cough is new or worsening.
- Only stop implicated drugs if cough is not tolerable and consider substituting ACEI with ARB or ARNI.

**Angioedema (rare)**

- Stop ACEI, ARB, or ARNI immediately, and consider referral to an immunologist.
- If there is a history of ACEI related angioedema, seek specialist advice before trialling ARB due to possible cross-sensitivity,
- Avoid ARNI if angioedema is due to ACEI or ARB.

**Euglycemic ketoacidosis (rare)**

SGLT2i increase the risk of ketoacidosis in diabetic patients. Endocrinologist review is advised before commencing in patients with type 1 diabetes. The risk increases when the patient has missed or reduced insulin doses, is fasting, perioperative, on a ketogenic diet, dehydrated, or has vomiting or diarrhoea,

**This guide is not intended to replace clinical judgment**



**Abbreviation key: ACEI** angiotensin-converting-enzyme inhibitor | **ARB** angiotensin II receptor blockers | **ARNI** angiotensin receptor neprilysin inhibitor | **bpm** beats per minute | **CRP** C-reactive protein | **EF** ejection fraction | **eGFR** estimated glomerular filtration rate | **Hb** haemoglobin | **kg** kilograms | **mg** milligrams | **mL/min** millilitres per minute | **mmHg** millimetres of mercury | **mmol/L** millimoles per litre | **MRA** mineralocorticoid receptor antagonist | **SGLT2i** sodium-glucose cotransporter-2 inhibitor |

HEART FAILURE MEDICATION TITRATION PLAN

# Titration Strategies:

- Aim to achieve maximally tolerated dose of all agents (RASi/Beta Blocker/MRA/SGLT2i)
- Aim to Commence all 4 agents at time of diagnosis (either in hospitalised patients or as outpatient) followed by rapid titration with close follow up.
- Recent evidence from STRONG-HF trial demonstrates that rapid drug implementation and up-titration is superior to traditional and more gradual step-by-step approach.

# Caution

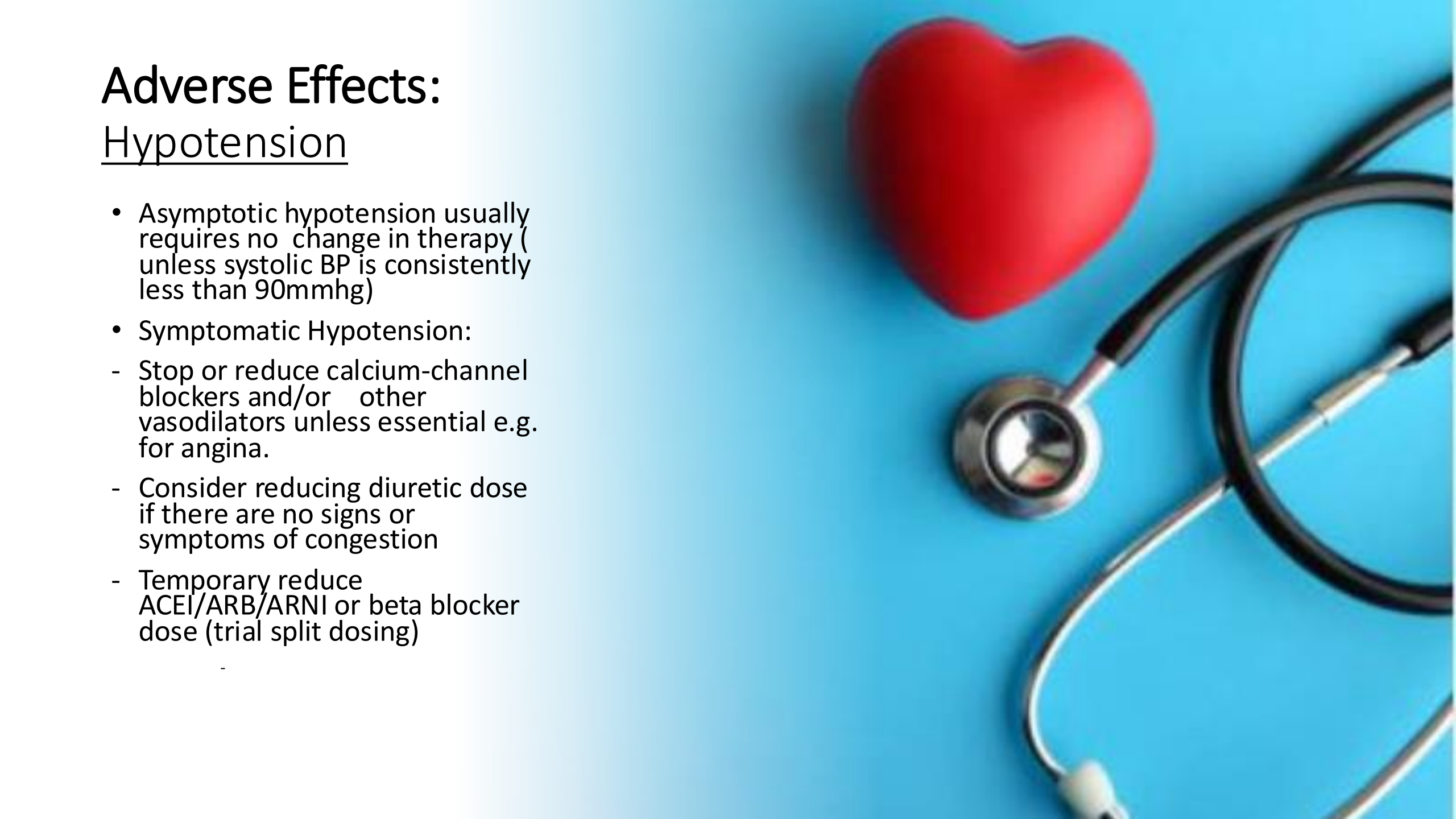
- ARNI – BP<100mmHg, Hx of dialysis, K>5.5 on discharge, allergy
- BB – HR<60bpm, known contraindication ie asthma or recent inotrope
- MRA – eGFR<30, Hx of dialysis, K>5.0 on discharge, allergy
- SGLT2i – eGFR<20, Hx of dialysis, T1DM, ketoacidosis or allergy

# Adverse Effects:

## Hypotension

- Asymptotic hypotension usually requires no change in therapy ( unless systolic BP is consistently less than 90mmhg)
- Symptomatic Hypotension:

- Stop or reduce calcium-channel blockers and/or other vasodilators unless essential e.g. for angina.
- Consider reducing diuretic dose if there are no signs or symptoms of congestion
- Temporary reduce ACEI/ARB/ARNI or beta blocker dose (trial split dosing)



# Adverse effects:
## Worsening renal function

Caution with ARNI if eGFR< 30 ml/minute.

eGFR < 20 ml/minute, starting treatment with SGLT2i is not recommended, however may be continued to if eGFR falls below this level (unless dialysis commenced)

Where there is severe dehydration, sepsis, or medication induced nephrotoxicity refer to ED. Consider withholding MRA first, then SGLT2i, followed by ACEI, ARB or ARNI until patient is reviewed.

# Adverse effects:
## After commencing or titrating therapy

- Expect a rise in creatinine, urea, and potassium (K+) for ACEI, ARB, ARNI, or MRA. A decline in eGFR up to 30% is acceptable if it stabilises within 2 weeks (or 4 to 12 weeks for SGLT2i).

- If eGFR declines by more than 30%, review fluid status and nephrotoxic medications and seek specialist advice about safety of continuing therapy.

# Adverse Effects:
# Bradycardia

If HR < 50 beats per minute and on a beta-blocker, review the need for other drugs that slow heart rate (e.g. digoxin, amiodarone) and arrange ECG to exclude heart block.

Consider reducing beta-blocker (avoiding abrupt cessation) if bradycardia is symptomatic.

If pacemaker is present seek specialist review.

# Adverse effects
## Hyperkalaemia

Monitor K+ for ACEI, ARB, ARNI and MRA. Urgently check K+, creatinine and urea for dehydration or sepsis. If serum K+ is:

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- 6 mmol/L or more, urgently seek specialist advice
- Recurrently high, seek specialist advice

# Adverse Effects: Volume Depletion

SGLT2i, MRA and ARNI have mild diuretic effects.

Assess volume status before commencing or adjusting doses and reduce the dose of loop diuretics in euvolemic patients if required.

Ease fluid restriction if patient euvolemic.

# Monitoring Recommendations

- Check blood pressure (BP) including postural drop and heart rate (HR) each visit
- ACEI/ARB/ARNI/MRA*: check serum potassium (K+), renal function 1-2 week/s after commencing or titrating (if K+ is high recheck in 48 hours). For MRAs check every 4 weeks for 12 weeks, at 6 months, then 6-monthly
- SGLT2i*: before commencing check volume status and for type 1 diabetics seek endocrinologist approval
- Diuretic dose changes beyond 3 days require medical review and checking of blood chemistry and volume status
- Iron: Order Hb*, CRP*, ferritin & transferrin saturation at first assessment and every 3-6 months if iron deficient

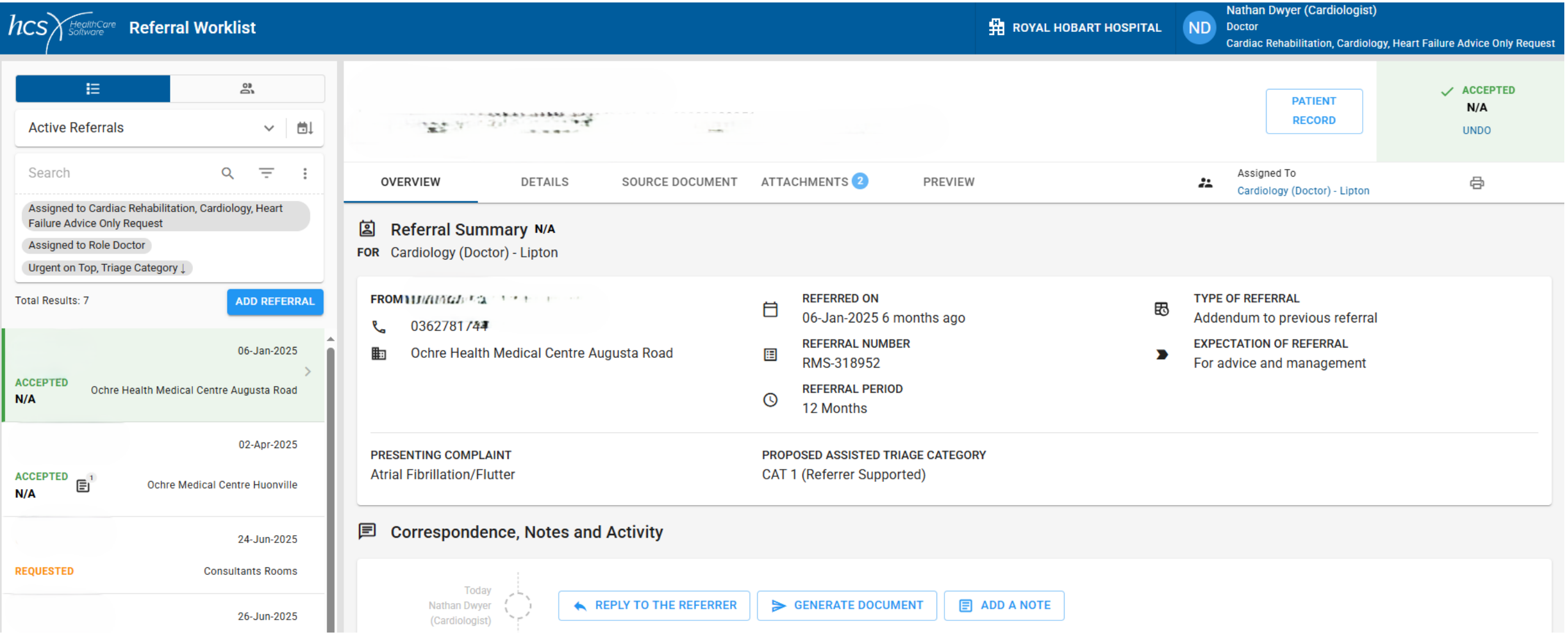
# Take Home Message:

- Four pillars are universally recommended in the management of HFrEF.
- Titration has historically been slow and cautious, recent trials have shown improved outcomes with rapid titration.
- Barriers can be overcome with utilisation of a multidisciplinary and collaborative approach. Telephone/Tele-Health titration is safe and effective.
- HF medication titration is not a one size fits all approach.

# When to Refer to Heart Failure Physician

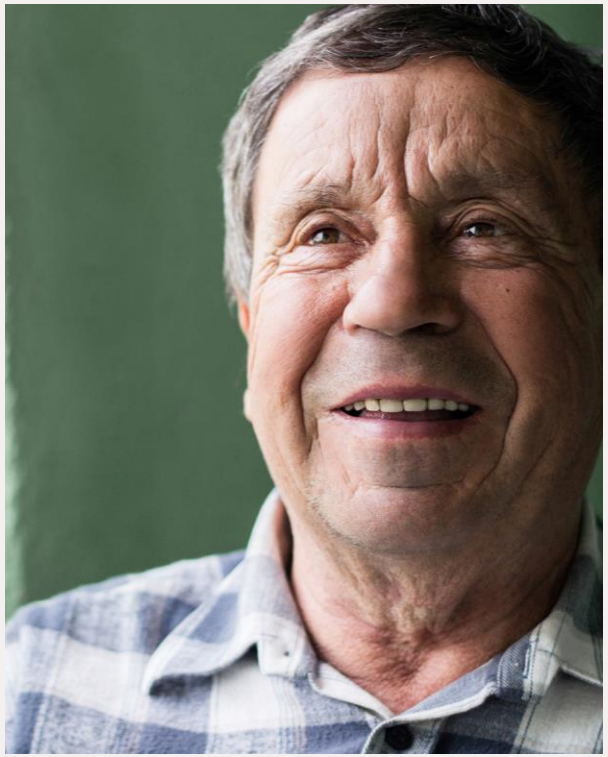
- New onset HF (especially in young)
- LVEF ≤ 35%
- Oedema despite escalating doses of diuretics
- Low blood pressure
- High heart rate
- End-organ dysfunction
- NYHA FC III/IV
- Hospitalisation
- Intolerance or down-titration of GDMT

# Heart Failure Advice Service - eReferrals



# MICK'S STORY

## Mick, aged 66, presents to his GP for a routine check-up and prescriptions



He seems generally well, although he remarks that he has been experiencing some recent fatigue and shortness of breath with moderate exertion.

> *"I haven't been out to mow the lawn for a few weeks; must be old age catching up with me"*

**Medical History**

- MI (2 years; non-obstructive CAD)
- HF-rEF (diagnosed 4 months post-MI)
- LVEF 36% (mild mitral regurgitation) at last cardiologist visit 18 months ago
- History of hypertension
- Mild COPD (ex-smoker)

CAD is the most common cause of HF-rEF, with 16.5% incidence at 1 year post acute MI

# MICK'S STORY

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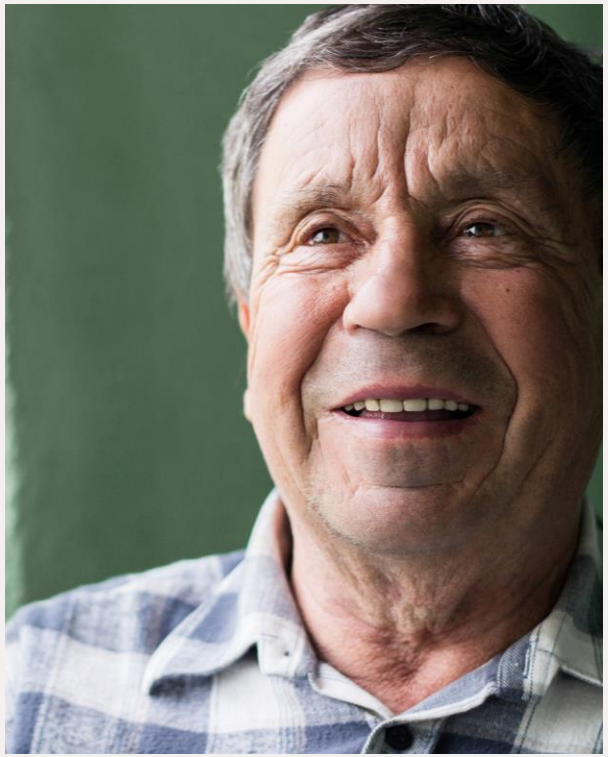


He seems generally well, although he remarks that he has been experiencing some recent fatigue and shortness of breath with moderate exertion.

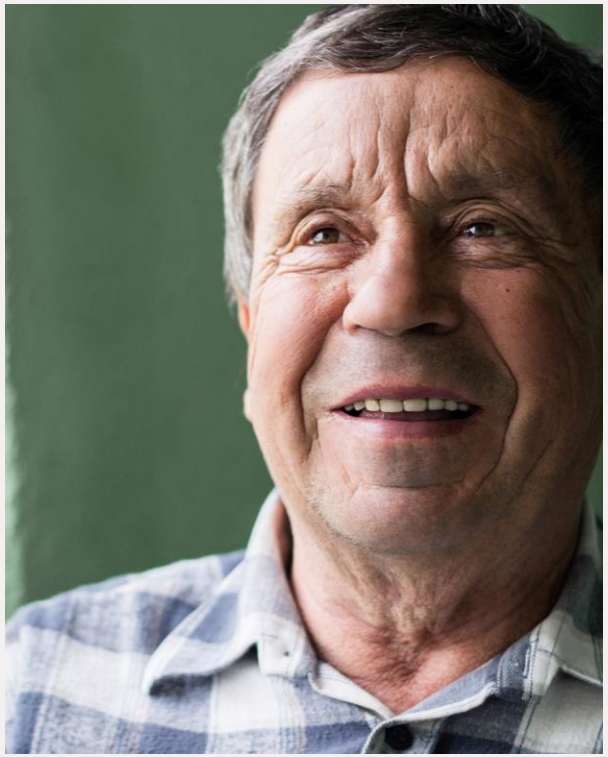
> *"I haven't been out to mow the lawn for a few weeks; must be old age catching up with me"*

**Current Medication**

- Perindopril arginine 5 mg once daily
- Carvedilol 25 mg twice daily
- Atorvastatin 40 mg once daily
- Aspirin 100 mg once daily
- Fluticasone/salmeterol 500/50 µg twice daily

# MICK'S STORY

## Mick, aged 66, presents to his GP for a routine check-up and prescriptions



He seems generally well, although he remarks that he has been experiencing some recent fatigue and shortness of breath with moderate exertion.

> *"I haven't been out to mow the lawn for a few weeks; must be old age catching up with me"*

**Examination**

- BP 130/65
- HR 75
- Occasional crackles at lung base; systolic murmur
- No oedema
- JVP not assessed (overweight)

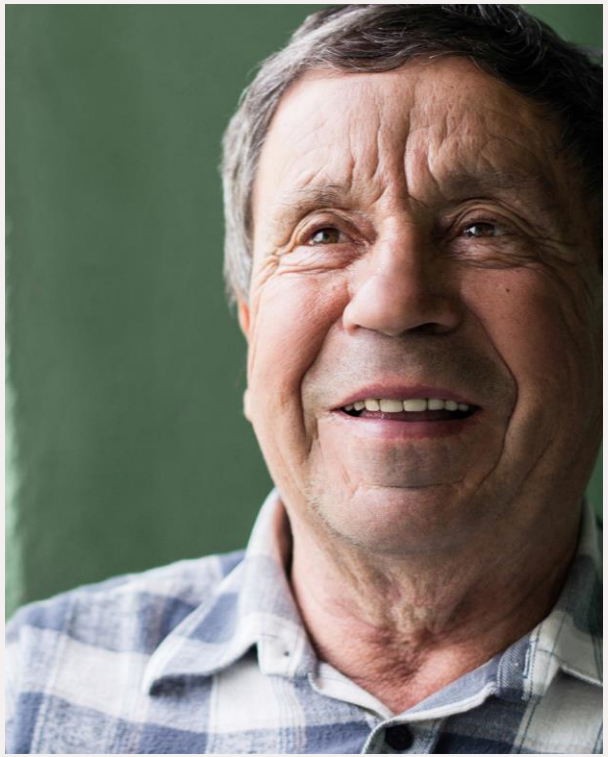
# WHAT WOULD YOU HAVE DONE FOR MICK?

Mick's GP concluded Mick was having an exacerbation of his known COPD which was resolving. He reiterated the importance of taking his medication regularly and did not change any of his medications.
He asked him to come back in 4 weeks for a review.

Several weeks later, he received a call from Mick's wife to let him know Mick had died suddenly.

# MICK – AN OPPORTUNITY MISSED

## Mick, aged 66, presents to his GP for a routine check-up and prescriptions



**What clues were overlooked in Mick's presentation?**

He seems generally well, although he remarks that he has been experiencing some recent fatigue and shortness of breath with moderate exertion

**Medical History**

- MI (2 years; non-obstructive CAD)
- HF-rEF (diagnosed 4 months post-MI)
- LVEF 36% (mild mitral regurgitation) at last cardiologist visit 18 months ago
- History of hypertension
- Mild COPD (ex smoker)

**Examination**

- BP 130/65
- HR 75
- Occasional crackles at lung base; systolic murmur
- No oedema
- JVP not assessed (overweight)

> *"I haven't been out to mow the lawn for a few weeks; must be old age catching up with me"*

Mick was not a 'stable' HF-rEF patient – his presentation should have triggered urgent further investigation

# IF YOU COULD TURN BACK THE CLOCK, WHAT COULD YOU HAVE DONE FOR MICK?

The treatment Mick was receiving was appropriate for an **uncomplicated MI (SAAB)**:

**S**tatin

**A**spirin

**A**CE inhibitor

**B**eta-blocker (note, carvedilol is only indicated for patients with HF)

**Crackles at the lung bases**.

What else could have been done for Mick?

# IF YOU COULD TURN BACK THE CLOCK, WHAT WOULD YOU HAVE DONE FOR MICK?

Would you:

a) Increase Perindopril

b) Add frusemide

c) Add spironolactone

d) Refer to a cardiologist

e) B and C

f) Other

# YOU GET MICK BACK THE FOLLOWING WEEK AND HIS CRACKLES HAVE RESOLVED.

What would you do now?

a) Titrate spironolactone

b) Switch Perindopril to Sacubitril/Valsartan

c) Add empagliflozin or dapagliflozin

d) Refer to a cardiologist

# GPs are crucial in the heart failure journey

- GPs see patients more frequently than their physicians
- There is no such thing as a ‘stable’ heart failure patient – even mildly symptomatic patients with HF are at risk for sudden death
- Regularly ask patients with heart failure about their symptoms and check for ‘red flags’:
  - Persistent symptoms of heart failure despite treatment
  - Peripheral (pitting) oedema
  - Increased use of diuretics to control symptoms
- Use of the 4 Pillars at maximal tolerated doses would be expected to reduce hospitalisation, improve QoL and reduce mortality
- GPs are a key component to the MDT

# Final Key Points

- Heart failure is a common clinical syndrome with high morbidity and mortality
- Echocardiography is an important clinical tool to distinguish between HFrEF and HFpEF as management strategies differ
- NT-pro-BNP is now available annually to assess for HF and can be used to triage echo.
- The management of heart failure is complex and requires a multidisciplinary approach
- The use of Tasmanian Health Pathways and eReferral advice can assist in optimising your patient's health


# How to Interpret a Formal Echocardiogram Report

An echocardiogram is a comprehensive ultrasound assessment of the heart. To make sense of it, focus on these key sections:

1. Heart Size and Structure
2. Valve Function
3. Pumping Strength (Ejection Fraction)
4. Additional findings

# Left Ventricle (LV)

## Ejection Fraction (EF)

Main measure of global heart pump function.

- Normal: 55–70%
- Mildly reduced: 45–54%
- Moderately reduced: 30–44%
- Severely reduced: <30%

## Global Longitudinal Peak Systolic Strain (GLPSS)

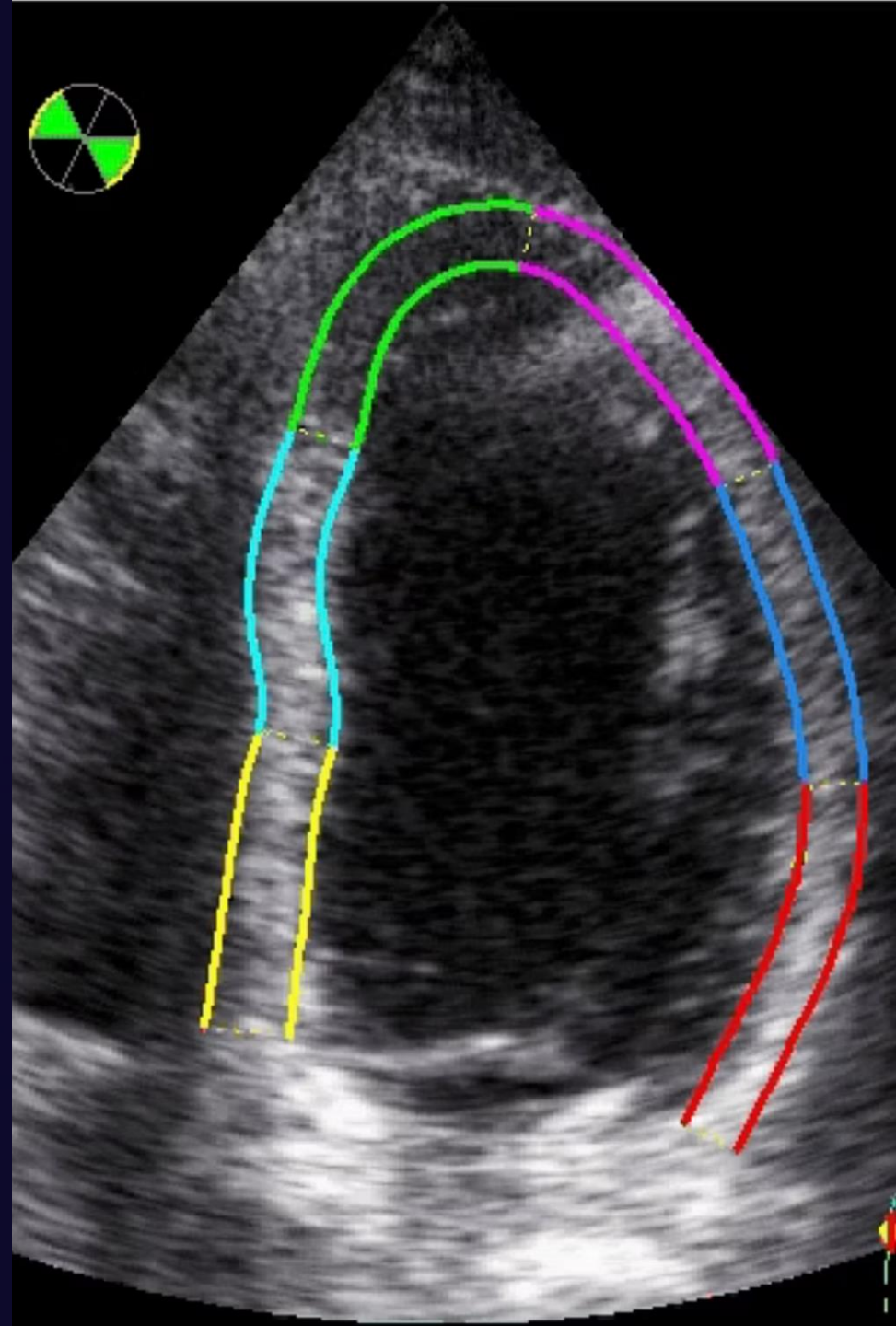
Normal > -18%

## Wall Motion Abnormalities

Suggest coronary artery disease (regional), cardiomyopathy (global), or prior infarction.

## Wall thickness

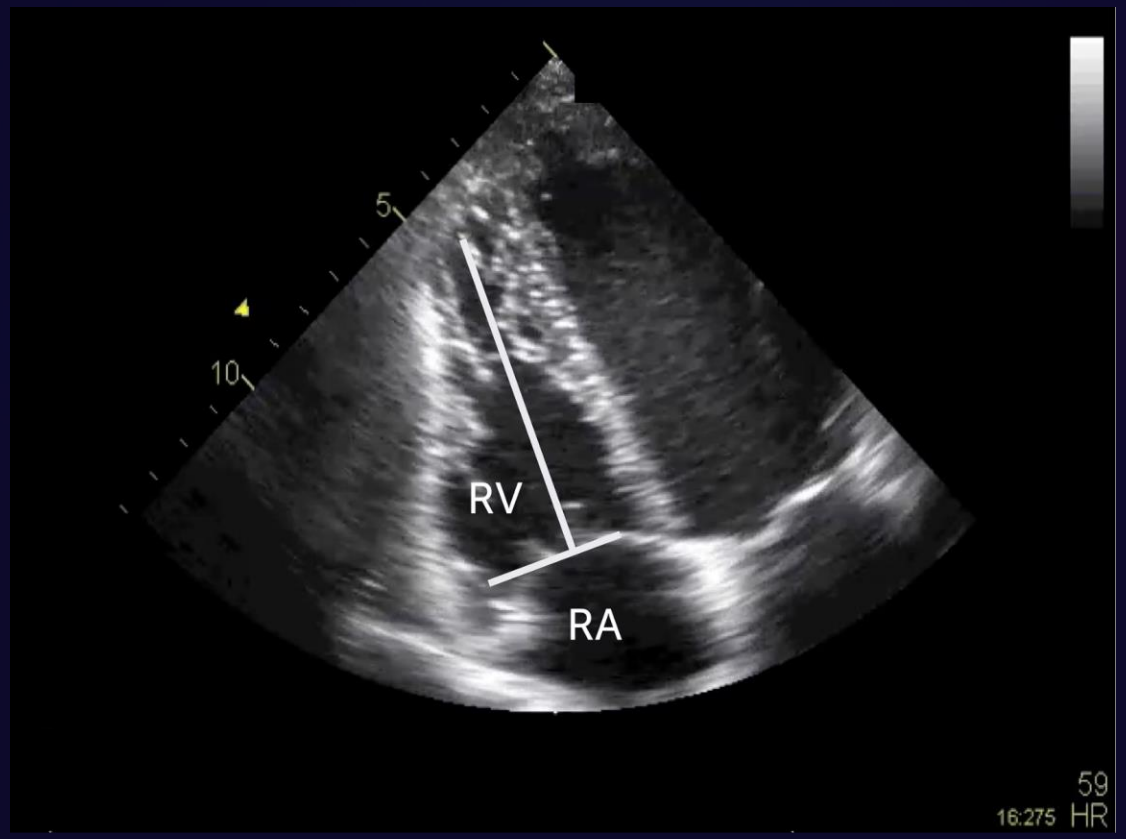
Suggests long-standing hypertension, valvular disease, hypertrophic cardiomyopathy, oedema (myocarditis) or infiltrative process.



# Right Ventricle (RV)

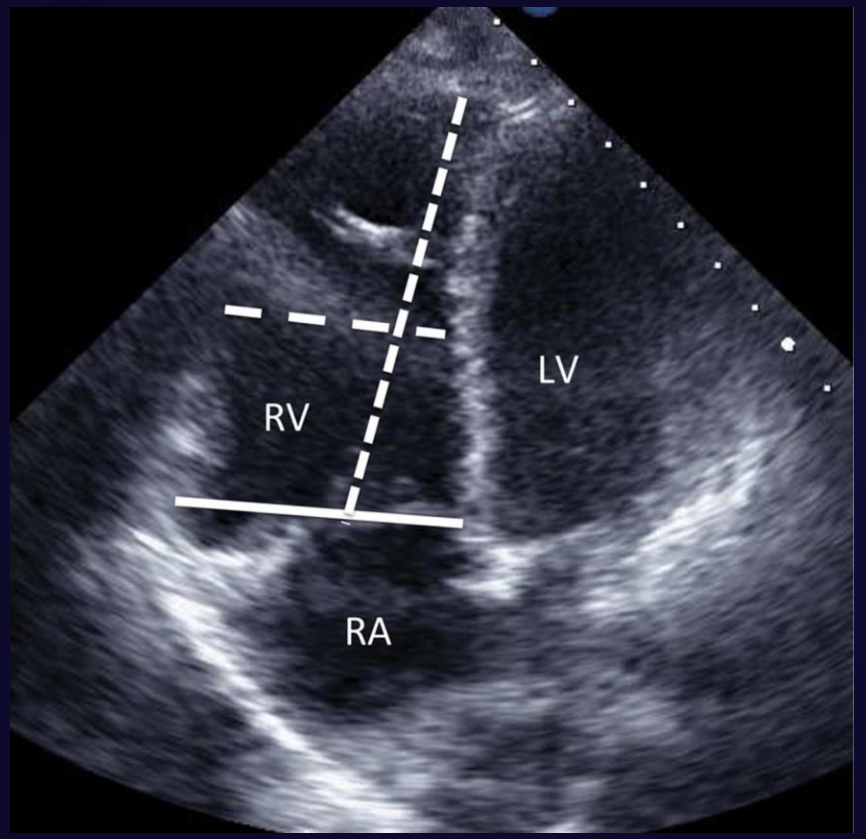
## Normal Function

Normal function suggests low likelihood of significant pulmonary hypertension or right heart strain.




## Abnormal Findings

Dilatation or reduced function may imply pulmonary disease or right-sided heart disease.



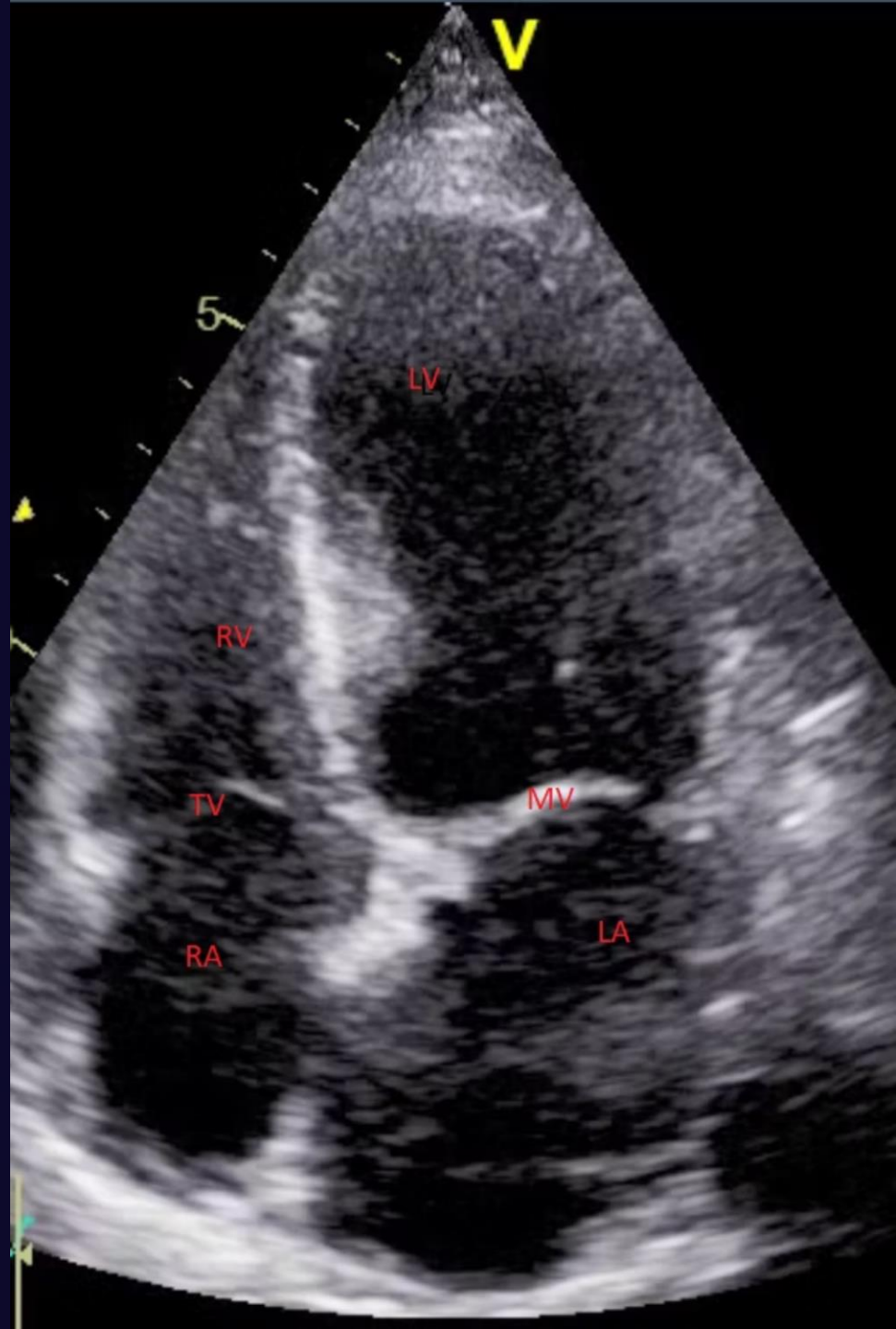
# Left Atrium (LA)

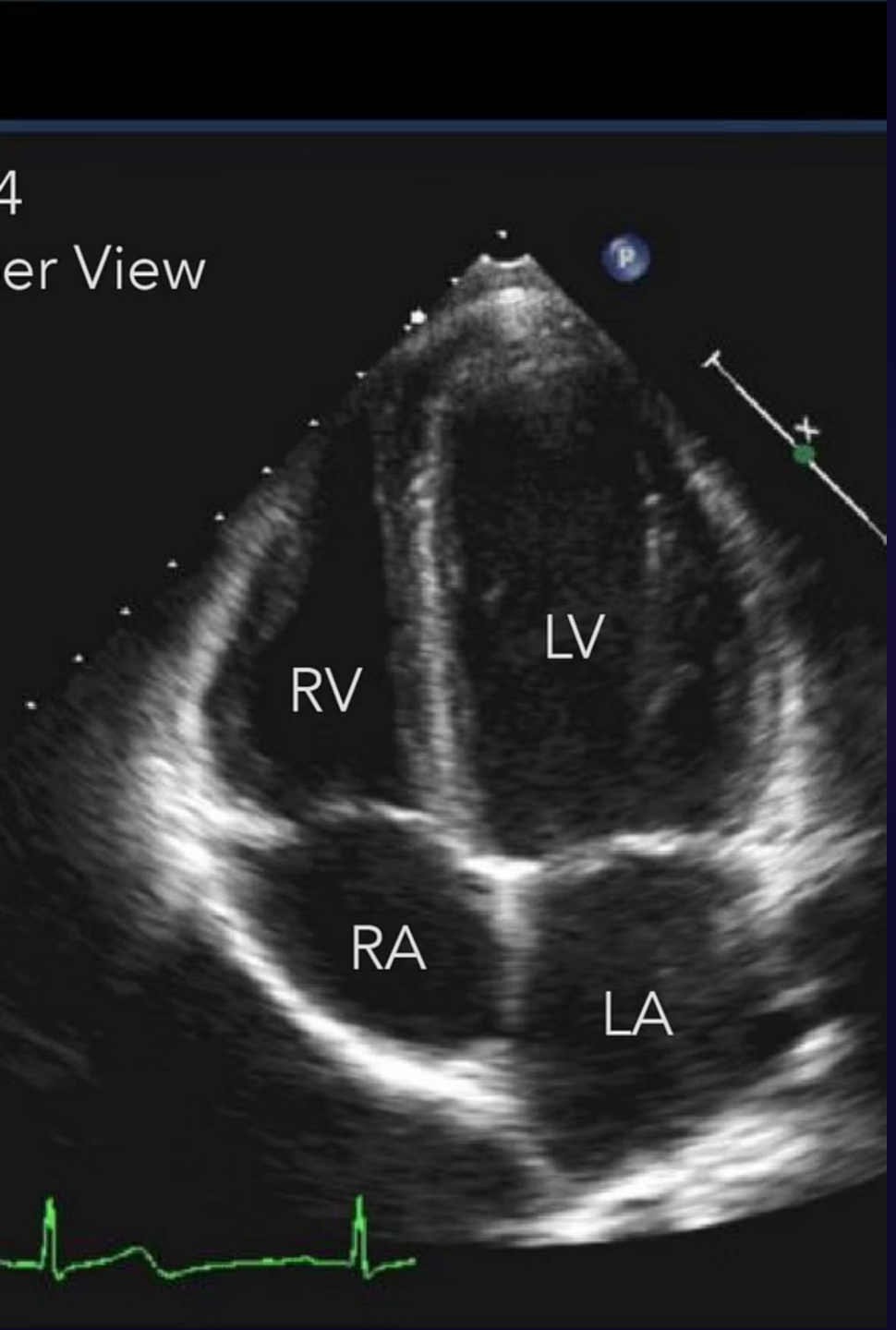
## <34 mL/cm²

### Normal LA Size

Measurements below this threshold indicate normal left atrial dimensions

Enlargement is a marker of chronic diastolic dysfunction, atrial fibrillation risk, and long-standing hypertension.






# Right Atrium (RA)

## <18 mL/cm²

### Normal RA Size

Measurements below this threshold indicate normal right atrial dimensions

Enlargement can be associated with tricuspid disease, pulmonary hypertension, or right heart strain.

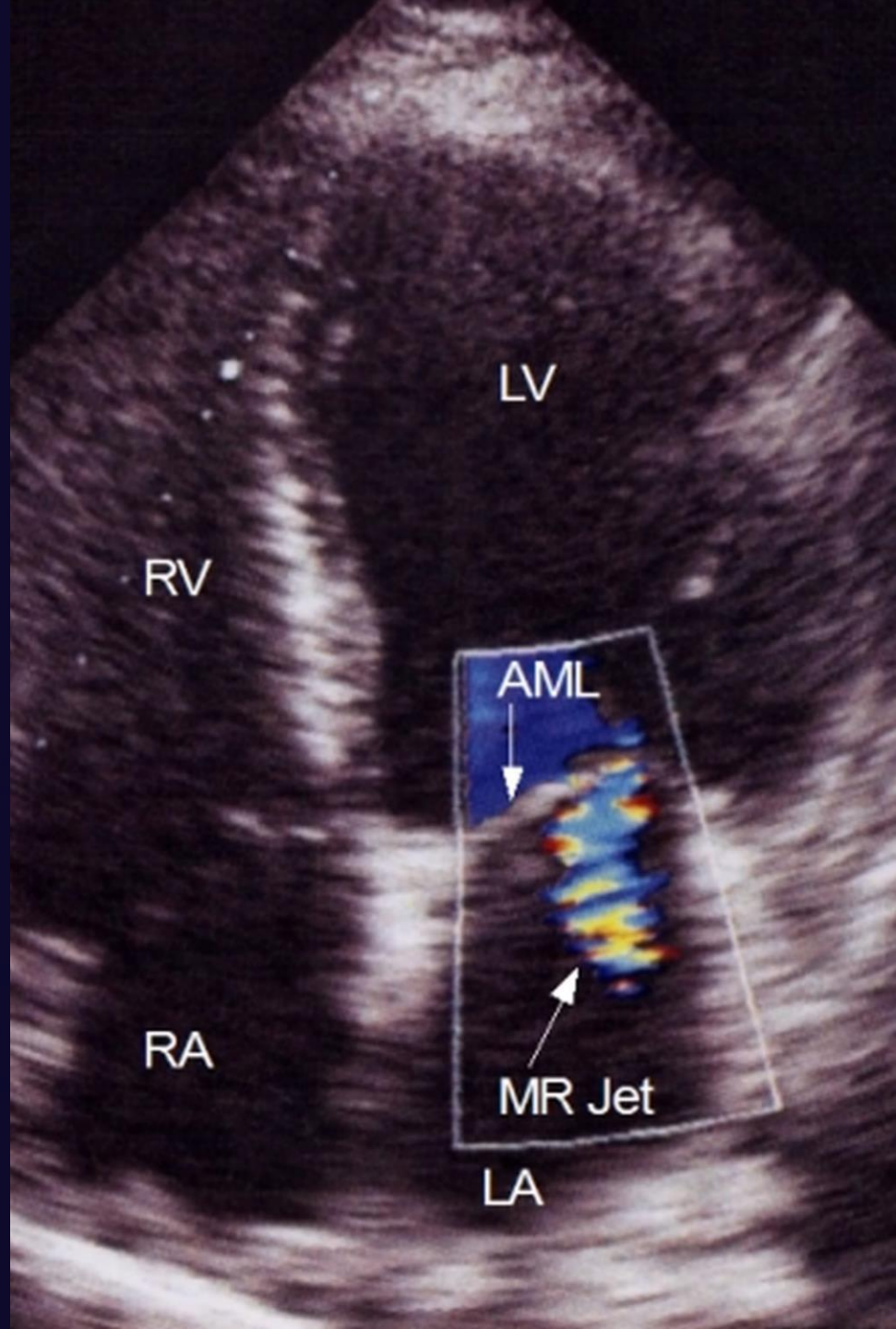
# Valves (Stenosis or Regurgitation)

## Severity Levels

- Mild: Usually monitored periodically.
- Moderate: May require closer clinical follow-up.
- Severe: Needs consideration for intervention.

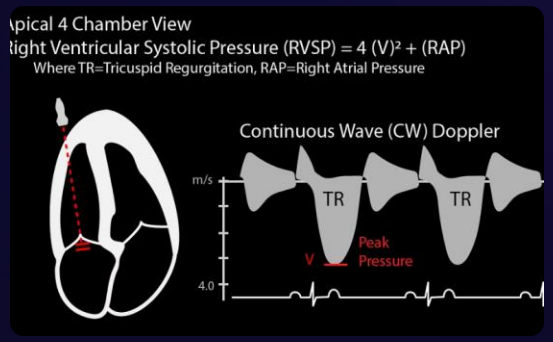
## Valve Types

- Aortic: Stenosis (AS) or Regurgitation (AR).
  - Bicuspid or Tricuspid.
- Mitral: Stenosis (MS) or Regurgitation (MR).
- Tricuspid: Regurgitation (TR).
- Pulmonic: Rare, often congenital.



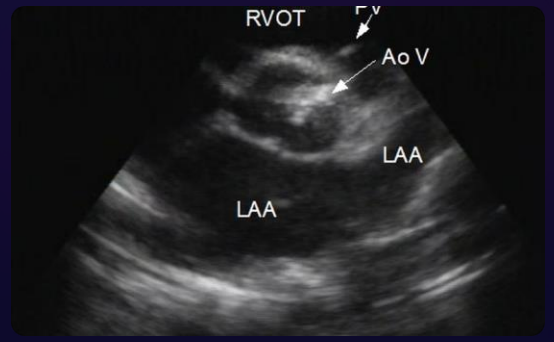
# Pulmonary Artery Systolic Pressure (PASP = RVSP + RAP)

Estimated from tricuspid regurgitation velocity:



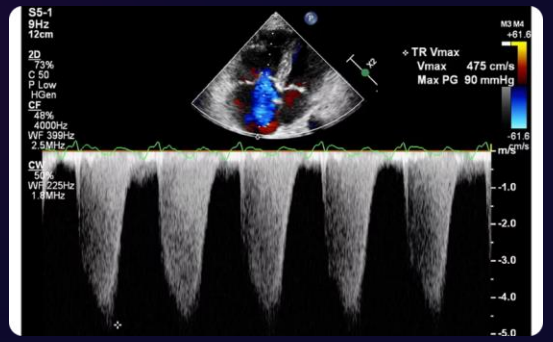

## Normal (<25 mmHg)

Indicates healthy pulmonary circulation



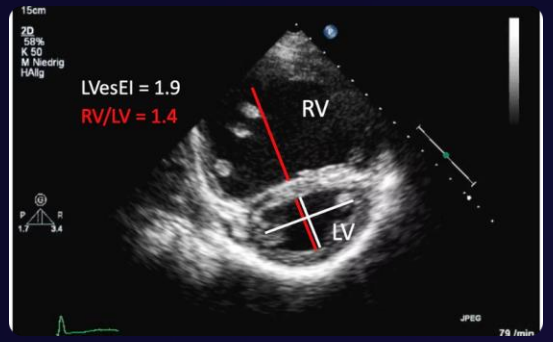

## Mildly elevated (25-40 mmHg)

May require monitoring




## Moderately elevated (40-55 mmHg)

Suggests significant pulmonary hypertension




## Severely elevated (>55 mmHg)

Indicates severe pulmonary hypertension

# Diastolic Function

**Why it matters:** Evaluates how well the left ventricle relaxes and fills. Abnormalities can indicate heart failure with preserved ejection fraction (HFpEF).

1

## E/A Ratio

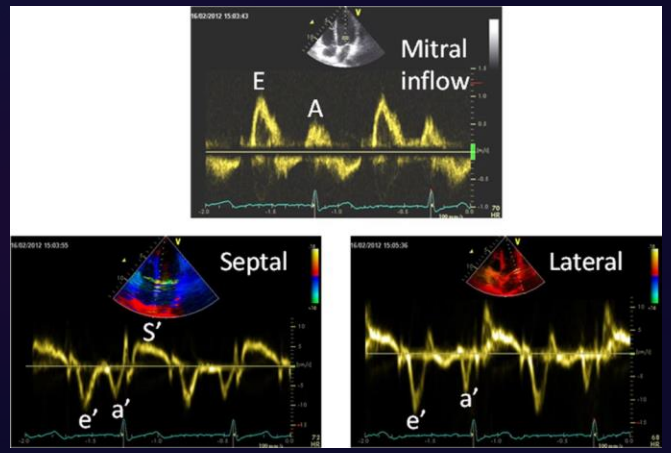
Ratio of early (E) to late (A) diastolic mitral inflow.

- Normal: ~1–2.
- Low E/A (<0.8): Suggests impaired relaxation (Grade I diastolic dysfunction).
- High E/A (>2): Suggests restrictive filling (Grade III).

2

## E/e' Ratio

Estimate of left atrial pressure.




- <8: Normal filling pressure.
- 9–14: Indeterminate.
- 14: Suggests elevated filling pressure (diastolic dysfunction).

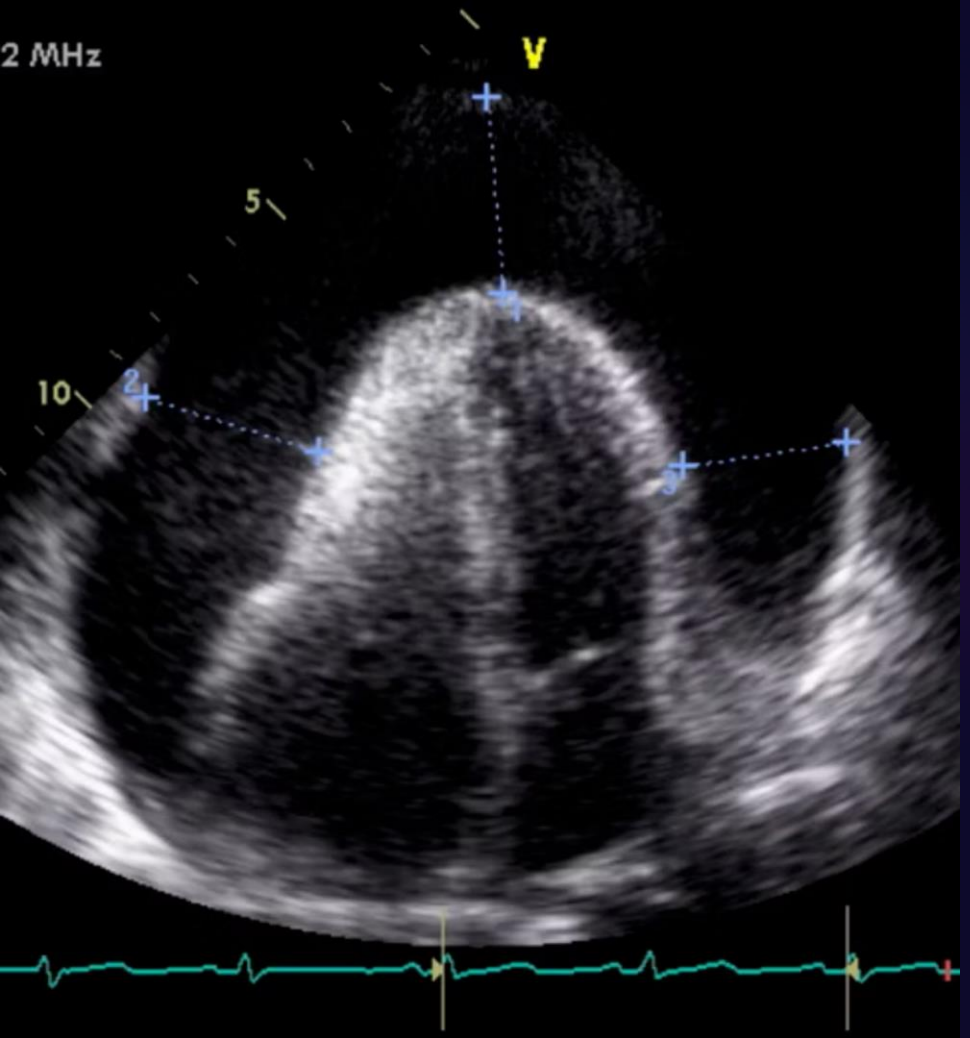
3

## Left Atrial Volume Index (LAVI)

34 mL/m²: Suggests chronic diastolic pressure overload.

## Implication for Practice:

- Abnormal diastology can cause dyspnoea despite a normal EF.
- Supports clinical suspicion of HFpEF and guides therapy (diuretics, blood pressure control, consideration for further investigation).




# Additional Findings

## Pericardial Effusion

Small, moderate, or large. Significance depends on clinical context.

## Aorta

Dilatation root and ascending aorta.

## Masses or Thrombus

Rare but critical to identify.

# Overall Impression

Look for the final Summary/Conclusions section. This typically gives a succinct clinical takeaway:

- Left and right heart function status.
- Significant valvular disease.
- Specific recommendations (e.g., clinical follow-up, referral for valve intervention, further studies).

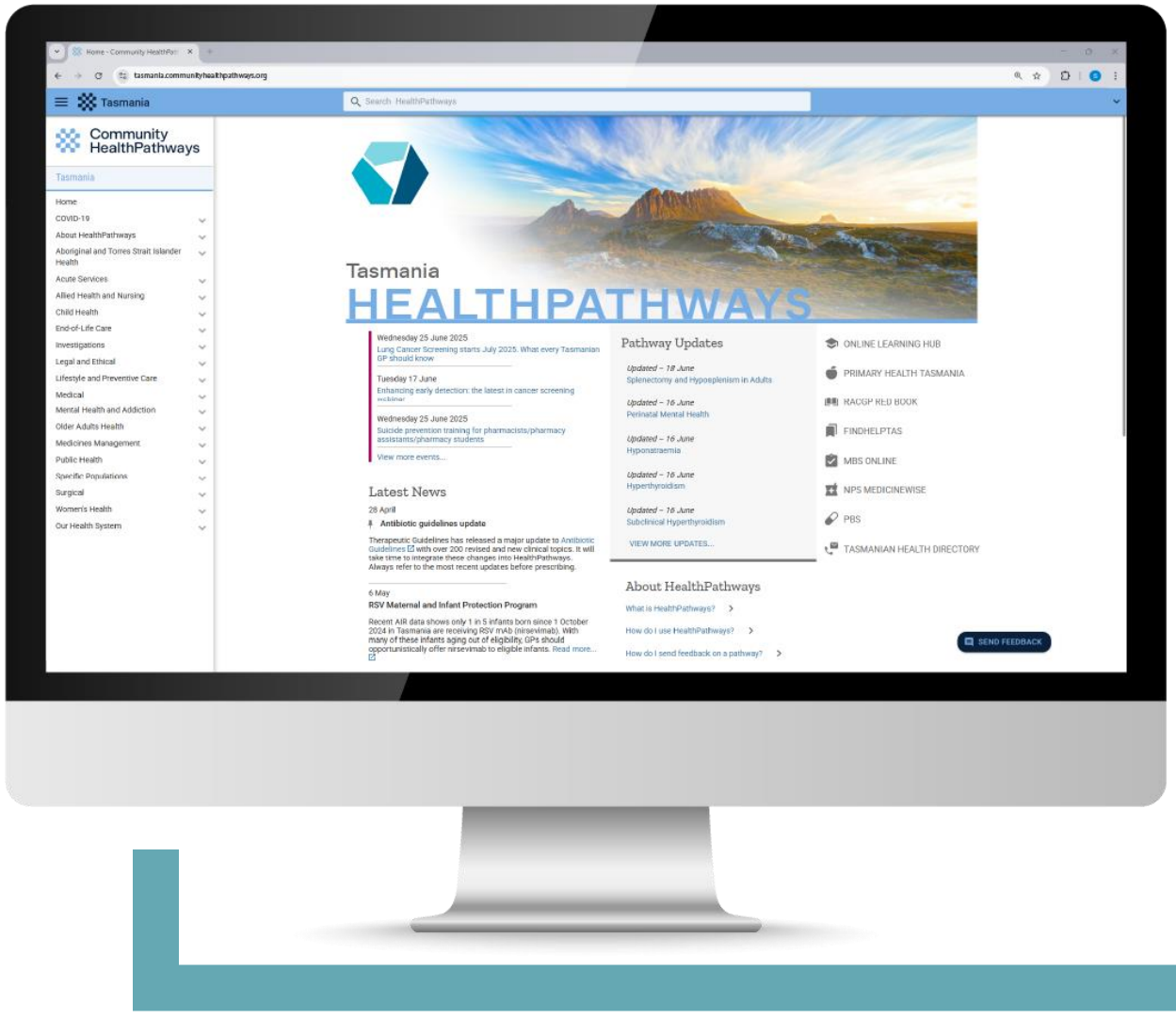
# QUESTIONS AND DISCUSSION

Tasmanian HealthPathways

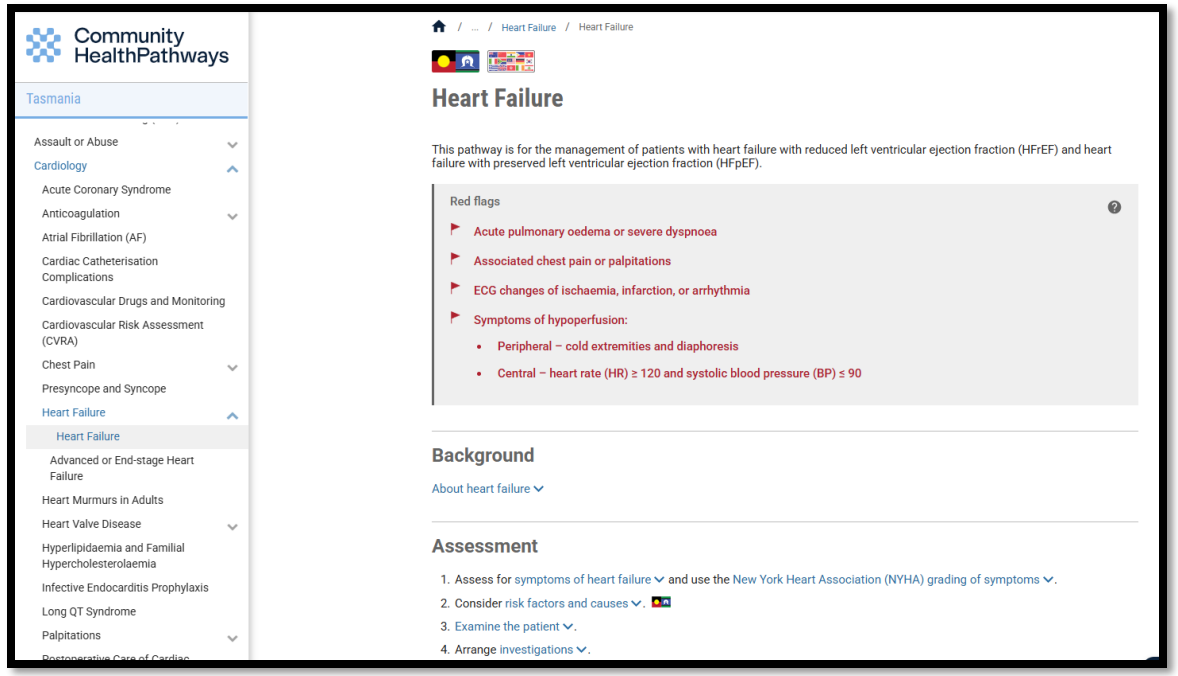

# Tasmanian HealthPathways

is a web-based information portal developed by Primary Health Tasmania. It is designed to help primary care clinicians plan local patient care through primary, community and secondary healthcare systems.

**tasmania.communityhealthpathways.org**



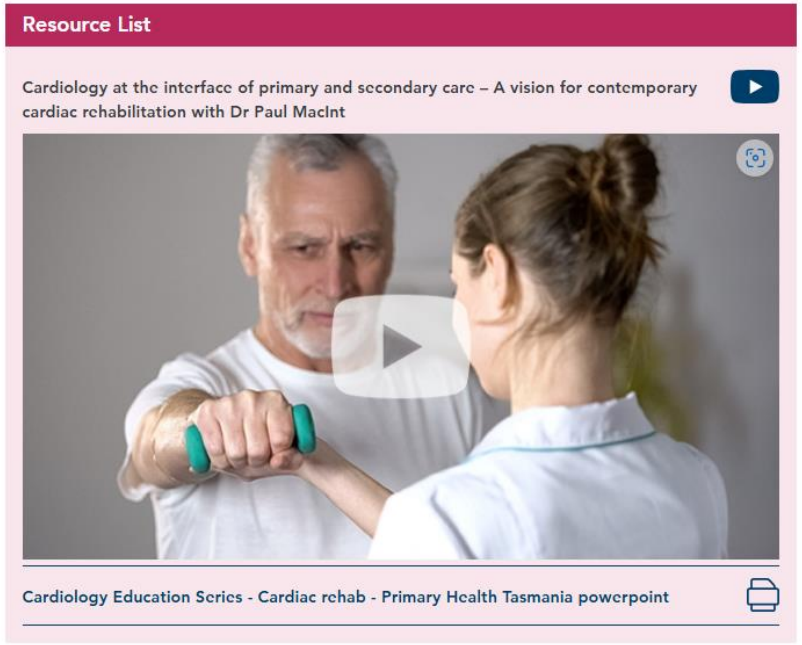

Tasmanian HealthPathways



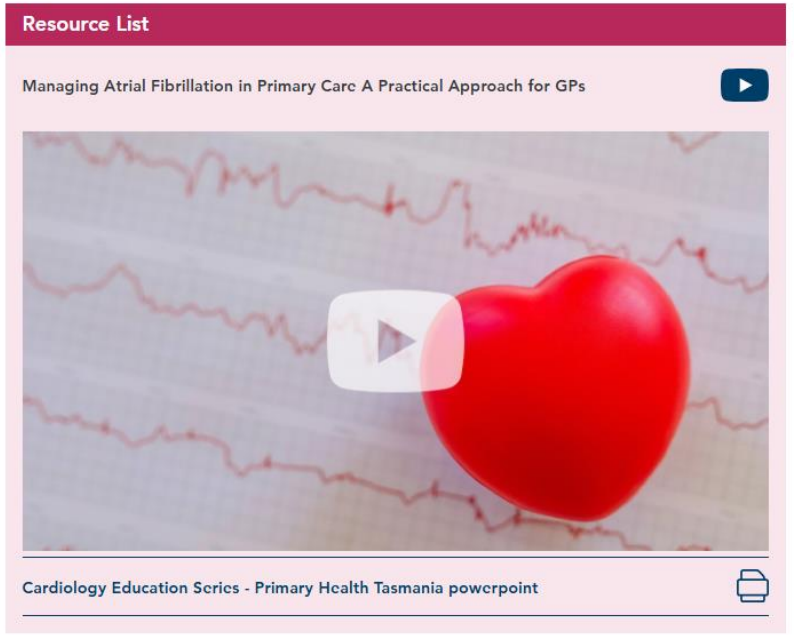

To gain access to HealthPathways, please email
**healthpathways@primaryhealthtas.com.au**

# Cardiology Series

## Cardiology at the interface of primary and secondary care – A vision for contemporary cardiac rehabilitation with Dr Paul MacIntyre




## Cardiology at the interface of primary and secondary care – Managing Atrial Fibrillation in Primary Care: A Practical Approach for GPs

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# Some final words

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