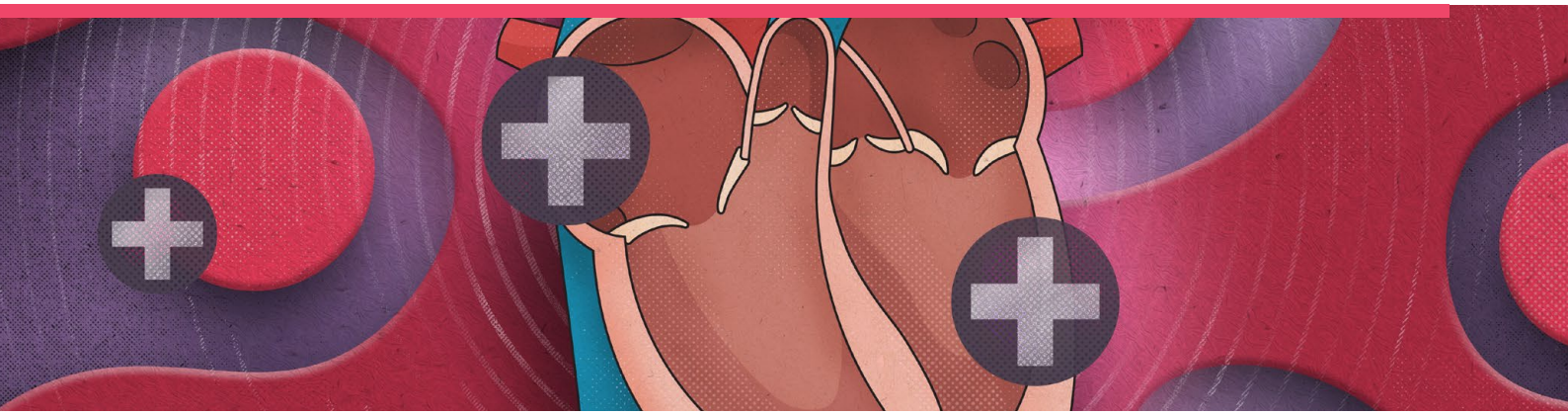


Addressing heart failure in primary care: Part 2 – Initiating and optimising treatment for heart failure

While most people with heart failure will require secondary care input at some stage, primary care still has a significant role in management. There have been substantial advances in pharmacological intervention in recent years that all clinicians should familiarise themselves with, including emphasis on the early introduction and optimisation of guideline-directed medical therapy (GDMT) – also referred to as the "four pillars" of heart failure treatment. GDMT significantly improves patient prognosis, including reducing the risk of hospitalisation and death, as well as improving quality of life.

KEY PRACTICE POINTS

- Following clinical diagnosis, pharmacological treatment for patients with heart failure should immediately proceed under the assumption they have reduced left ventricular ejection fraction (HFrEF)
- Rather than sequential treatment escalation in response to symptoms, patients with heart failure should be promptly established on four guideline-directed medical therapy (GDMT) medicines and up-titrated to the highest tolerated or target dose, unless contraindicated. GDMT maximises prognostic outcomes (e.g. risk of hospitalisation and mortality) and limits disease progression. This includes:
  - An angiotensin receptor-neprilysin inhibitor (ARNI; preferred) or an ACE inhibitor/ARB if this is not possible; and
  - A beta blocker (either bisoprolol, metoprolol succinate or carvedilol); and
  - A mineralocorticoid receptor antagonist (MRA); and
  - A sodium-glucose co-transporter-2 (SGLT-2) inhibitor
- Special Authority restrictions may influence the introduction of GDMT. For example, the ARNI and SGLT-2 inhibitor cannot be initiated (funded) without patients first being established on "concomitant optimal standard chronic heart failure treatments" (among other criteria).
  - When an ARNI is introduced, stop the ACE inhibitor (or ARB) before initiating treatment due to the risk of angioedema
  - SGLT-2 inhibitors have been funded with Special Authority approval for patients with HFrEF (regardless of diabetes status) since 1st December, 2024
- In addition to GDMT, assertive treatment with a loop diuretic is required if the patient has fluid overload. Withhold beta blocker initiation until the patient is euvolemic.
- For patients initially diagnosed in primary care, aim to achieve optimal dosing of all four GDMT medicines within three months where practically possible. If a patient is already taking a GDMT medicine, or is hospitalised for heart failure, aim for optimisation within a shorter timeframe, e.g. within six weeks.
- Regular monitoring is essential in patients with heart failure. Key short-term considerations include medicine adverse effects, clinical/symptomatic status, blood pressure, renal function and serum potassium.
  - Further BNP testing (preferably NT-proBNP) can be considered, but only if it will influence management decisions
  - Repeat echocardiography is helpful to monitor disease progression in the long-term, including consideration for further treatments, e.g. implantable cardioverter defibrillator (ICD), cardiac resynchronisation therapy (CRT) or CRT-defibrillation
- If heart failure with preserved ejection fraction (HFpEF) is confirmed based on echocardiography at any point, a cardiologist should generally be involved to refine treatment
  - HFpEF management focuses mainly on controlling fluid balance using diuretics at the lowest possible dose, in addition to managing associated co-morbidities, especially hypertension and atrial fibrillation
  - SGLT-2 inhibitors are effective in patients with HFpEF, but those with echocardiography-confirmed HFpEF are not currently eligible for Special Authority approval

The general principles of management

Once heart failure has been diagnosed, initiate pharmacological treatment as soon as practically possible:[1]

The short-term goal – improve the patient's symptoms/signs (clinical status) and functional capacity, and reduce the risk of hospital admission

The longer-term objective – improve heart function or slow/prevent progressive deterioration thereby improving patient longevity and quality of life

Patients with acute onset of significant heart failure symptoms may require immediate hospital admission where initial treatment decisions will be made.[2] However, in patients with a more gradual onset of symptoms, the process can begin in primary care. In this scenario, it is unlikely that echocardiography will have been performed yet, and therefore it will not be known whether the patient has heart failure with reduced ejection fraction (HFrEF; i.e. left ventricular ejection fraction [LVEF] of ≤ 40%), mildly reduced ejection fraction (HFmrEF; i.e. LVEF 41 – 49%) or preserved ejection fraction (HFpEF; i.e. LVEF ≥ 50%).

For further information on the terminology associated with heart failure, see: **Part 1 – Identifying and diagnosing heart failure**

Proceed assuming the patient has HFrEF. Given that most evidence regarding effective management relates to patients with HFrEF, it is practical to initiate treatment in primary care assuming they have this subtype (see: "Guideline-directed medical therapy (GDMT): The four pillars of heart failure treatment"), and then modify the approach later on if echocardiography proves otherwise (see: "Treatment of patients with heart failure with preserved ejection fraction (HFpEF)"). The medicines used in GDMT are still likely beneficial if the patient is subsequently found to have HFpEF, and are unlikely to cause harm. For example, sodium-glucose co-transporter-2 (SGLT-2) inhibitors are recommended across international guidelines for all patients with heart failure, regardless of their associated LVEF.

A 2022 Australian study found that patients with heart failure attend general practice over 14 times each year on average (New Zealand data not available, but is reported to be similar); this indicates there is ample opportunity to not only monitor disease progression and medicine tolerance, but to further optimise treatment and improve clinical outcomes.[3]

Guideline-directed medical therapy (GDMT): The four pillars of heart failure treatment

Among cardiovascular conditions commonly encountered in primary care, the approach to heart failure management has perhaps undergone the most substantial changes over recent times (**Figure 1A**). International guidelines increasingly recommend that most patients with heart failure should be established on guideline-directed medical therapy (GDMT) – also referred to as the "four pillars" of heart failure treatment – as early as practically possible.[4, 5] This includes optimised use of:

- An **angiotensin receptor-neprilysin inhibitor** (**ARNI**; preferred) or an **ACE inhibitor/ARB** if this is not possible; and
- A **beta blocker** (either bisoprolol, metoprolol succinate or carvedilol); and
- A **mineralocorticoid receptor antagonist (MRA)**; and
- A **sodium-glucose co-transporter-2 (SGLT-2) inhibitor**

Combined use of these four medicines in patients with HFrEF reduces the relative risk of all-cause mortality by 61%, and cardiovascular mortality or heart failure hospitalisation by 64% (**Figure 1B**). Compared with standard ACE inhibitor/ARB and beta blocker treatment, GDMT is estimated to confer more than six additional years of life (on average) for a patient aged 55 years, or 1.4 additional years for a patient aged 80 years.[6] Despite these benefits, and the otherwise poor prognostic outlook for patients with heart failure, GDMT is significantly underutilised in daily practice (see: "Most patients with heart failure are undertreated").

For further information on the individual GDMT options and treatment considerations, see: "A closer look at the treatments for patients with heart failure with reduced ejection fraction (HFrEF)".

The logistics of GDMT use are still being refined

Initiating GDMT medicines. There has been considerable debate in international literature regarding the best approach to achieving optimised GDMT; *should the medicines be initiated simultaneously or introduced sequentially?* If possible, simultaneous initiation of all GDMT medicines at low doses, and subsequent up-titration, is encouraged to maximise patient adherence and outcomes, both in the short and long term.[4] However, this assumes that close monitoring is possible, such as in a hospital setting. In primary care, GDMT initiation and optimisation is based on a shared decision between the clinician

A Guideline-directed medical therapy (GDMT)

Also called the "four pillars" of heart failure treatment – denoted in green boxes

Establish patients on all four medicines as early as possible

- **ARNI** (or ACE inhibitor/ ARB†)
- **Beta blocker** (once euvolemic)
- **MRA****
- **SGLT-2 inhibitor** (all patients unless contraindicated)

Promptly titrate to maximally tolerated or target dose **(see: Table 1)**
- Ideally achieve optimised GDMT within three months of initiation
- Initially favour up-titrating the beta blocker unless congested or heart rate < 50 bpm

Continue at specified dose

N.B. See main text for discussion around the practicalities of implementing GDMT in New Zealand primary care.

Consider need for secondary care referral to guide further medicine optimisation or use of advanced procedures, e.g. in patients with a high symptom burden despite optimised GDMT

Provide assertive loop diuretic* treatment if fluid overload/ congestion is present
- Taper/stop the diuretic once patient is euvolemic
- Avoid continuous long-term use

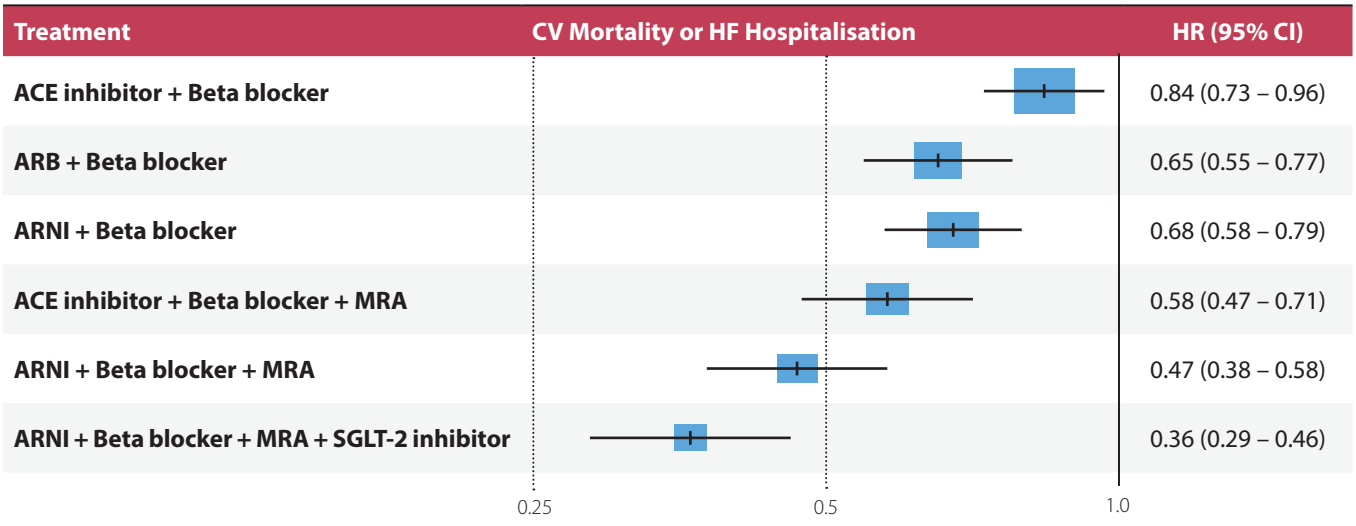
Consider additional treatments depending on co-morbidities. For example:

Medicine	Condition
Digoxin	Atrial fibrillation
Anticoagulants	
Intravenous iron	Anaemia and iron deficiency

Non-pharmacological support:
- Daily exercise, as appropriate if tolerated
- Reduce sodium intake (ideally < 3 g daily; no more than 5 g daily)
- Weight loss
- Adequate fluid intake (1.5 – 2 L daily)
- Reduce alcohol/smoking cessation, if relevant
- Influenza/pneumococcal/ COVID-19 vaccination

* Usually furosemide. Consider use of a thiazide diuretic if loop diuretic is contraindicated or not tolerated.
† If unable to tolerate an ARNI or patient not eligible for funded access (and cannot afford to self-fund treatment)
** Also known as aldosterone antagonist. Examples include spironolactone and eplerenone.

B The benefits of GDMT



A hazard ratio (HR) below 1 means the endpoint (CV mortality or HF hospitalisation) is less likely to occur with the specified treatment regimen versus control. Lower HR values indicate a greater reduction in risk, reflecting a more significant treatment effect.

ACE = angiotensin-converting enzyme inhibitor; ARB = angiotensin II receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; CI = confidence interval; CV = cardiovascular; HF = heart failure; MRA, mineralocorticoid receptor antagonist; SGLT-2, sodium-glucose co-transporter 2

Figure 1. An overview of **(A)** guideline-directed medical therapy (GDMT) for patients with heart failure with reduced ejection fraction (HFrEF) or an undifferentiated clinical diagnosis (i.e. echocardiography results are not yet available) and **(B)** the benefits of GDMT versus other treatment regimens in patients with HFrEF.[4, 5, 7, 8]

N.B. HFrEF refers to patients with symptoms and signs of heart failure and a LVEF ≤ 40% confirmed by echocardiography. Previous definitions for HFrEF have also encompassed the LVEF range 41 – 49%, but this is now distinguished as being heart failure with a mildly reduced ejection fraction (HFmrEF); the management approach for HFrEF and HFmrEF are largely the same.

and patient, balancing clinical status, co-morbidities and capacity for appropriate monitoring.[7, 9] As such, rapid sequential addition is still a reasonable approach in primary care, e.g. starting with one or two medicines initially and then adding/switching additional medicines at subsequent appointments. A 2022 Australian consensus statement on the pharmacological management of heart failure provides a **suggested algorithm for introducing GDMT** based on the patient's degree of congestion, however, this does not account for Special Authority funding restrictions in New Zealand.[10]

Prioritise prompt GDMT optimisation. For patients not taking any GDMT medicines prior to a heart failure diagnosis, aim to introduce and achieve optimal dosing within three months in a community setting.[4] Rapid GDMT optimisation is preferrable, where possible. If any GDMT medicines are already being taken, or patients initially require hospitalisation, an abridged timeline should be targeted (e.g. six weeks or less; see: "The case for more assertive GDMT optimisation: STRONG-HF").[11, 12] In practice, numerous factors influence this process, and particular caution is needed in patients with renal dysfunction.[4] As such, regular review of the patient's clinical status, blood pressure, renal function and electrolytes is required (see: **Table 1** and "Monitoring the effectiveness of treatment").

Up-titration is important but should not come at the expense of initiating additional GDMT medicines. The ideal scenario is that any patient with newly diagnosed heart failure should receive all four GDMT medicines up-titrated to the target* dose.[4] However, if this is not possible, it is suggested that "*receiving some GDMT is still far more important than receiving none*" and that "*below-target doses of multiple classes of GDMT are likely more effective in reducing risk than large doses of 1 or 2 agents*".[4] Given the diverse maladaptive pathways and multi-system dysfunction associated with heart failure (**Figure 2**), using multiple medicines with distinct mechanisms of actions is important as they have independent, yet additive, benefits.[13]

* Target dose refers to the dose of GDMT medicines used in clinical trials to demonstrate efficacy (**Table 1**)

The case for more assertive GDMT optimisation: STRONG-HF

The STRONG-HF trial (N = 1,078) assessed the benefits of high-intensity management among patients admitted to hospital with acute heart failure, who were previously not treated with target doses of GDMT medicines, aiming to achieve optimal dosing within two weeks of discharge.[12] Outcomes were compared against patients randomised to "usual care", i.e. treatment according to a clinician's standard practice.

STRONG-HF was stopped early because of the significant benefit demonstrated for high-intensity treatment.[12] After 90 days of follow up, blood pressure, heart rate, functional status, congestion, weight and NT-proBNP concentrations had decreased more significantly among patients randomised to high-intensity treatment.[12] The primary endpoint of heart failure re-admission or all-cause mortality up to Day 180 occurred in 15.2% of the high-intensity group versus 23.3% in the usual care group (a 35% relative reduction).[12] Overall rates of adverse effects were elevated in the high-intensity treatment group, but there was no significant difference in discontinuation rates or the incidence of serious or fatal adverse effects.[12] Secondary analyses also demonstrated improved quality of life among patients treated with assertive GDMT,[17] and more effective/sustained decongestion outcomes at Day 90 (despite a lower mean loop diuretic dose being required).[18]

Taken together, these findings demonstrate substantial benefits can be achieved with assertive GDMT optimisation, without significantly compromising patient safety. The two-week optimisation timeframe used in STRONG-HF is more ambitious than the six weeks recommended for patients post-hospital discharge in the 2023 focused update of European guidelines, which may be a more practical target for primary care if assertive management is being considered.[11, 12]

A closer look at the treatments for patients with heart failure with reduced ejection fraction (HFrEF)

Immediately prescribe a loop diuretic if the patient has fluid overload

Patients with symptomatic heart failure often have presenting features such as shortness of breath, elevated jugular venous pressure, bibasilar crackles or peripheral oedema – all of which are caused by hypervolaemia (fluid overload).[19] Persisting hypervolaemia is a marker for poor prognosis in patients with heart failure.[19] Many patients hospitalised for acute heart failure are discharged while still congested.

Immediate and assertive treatment with a loop diuretic such as furosemide is a first priority for managing patients with heart failure and fluid overload:[19, 20]

- Initially 20 – 40 mg daily
- For resistant fluid overload, up-titrate in 20 – 40 mg increments to the minimum dose that improves symptoms and achieves weight loss of approximately 1 kg/day with a return to dry body weight
- The frequency of up-titration will depend on patient response and the severity of fluid overload (weekly is common)
- The usual dose range is 40 – 240 mg daily (patients with renal impairment may require higher doses within this range, but prolonged use may further compromise renal function)

Diuretics have not been shown to improve survival in patients with heart failure and are therefore not considered one of the "pillars of heart failure treatment". However, they significantly improve quality of life by alleviating fluid overload, which can be particularly important in severely congested patients.

Patients should initially be weighed and a "dry weight" target* established to progressively evaluate diuresis. Measure blood pressure, serum potassium and renal function throughout treatment, and ask patients about urine output.[2] Taper diuretic dosing over time as control is achieved to the lowest effective dose that maintains euvolemia; avoid long-term continuous treatment where possible unless patients are symptomatic.[1] Diuretics can be re-started in response to re-emerging symptoms/signs of fluid overload. Some patients may be able to self-manage diuretic dosing.

* Dry weight is the patient's recorded/reported average weight before they began experiencing symptoms/signs of fluid overload

Practice point: Following acute treatment, establish a heart failure action plan with the patient, which includes daily self-monitoring of changes in weight, swelling and shortness of breath. This can facilitate better self-management and treatment adherence, as well as prompt identification of symptom recurrence. Patient resources are available from the Heart Foundation, see: **www.heartfoundation.org.nz/resources/heart-failure-action-plan-tear-off-pad**.

Heart failure apps are also available which may assist some patients with aspects of self-management. **Healthify has reviewed two apps**; they are free to download, but note that they have both been developed in the United States so may contain information not relevant to the New Zealand context, but still include useful features, e.g. tracking symptoms/weight, fluid status, reminders to take medicines, educational material.

Other medicines for promoting diuresis

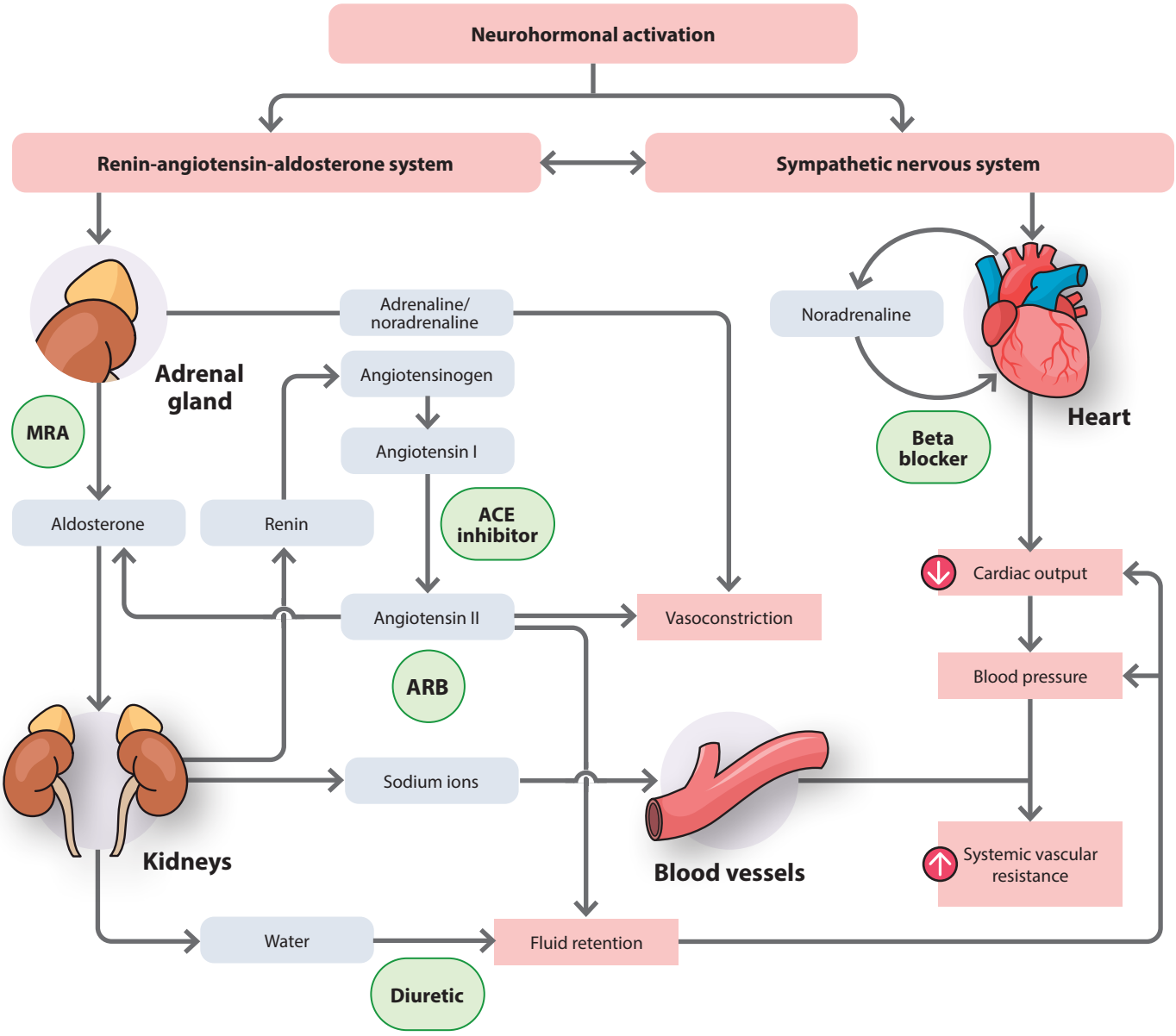
Bumetanide is an alternative loop diuretic to furosemide, which some congested patients respond more favourably to due to increased oral absorption (gastrointestinal absorption may be compromised due to gut congestion).[21] If loop diuretics are contraindicated or ineffective, consider a thiazide diuretic as an alternative or an add-on, but these are contraindicated in patients with poor renal function (e.g. Stage IV chronic kidney disease or eGFR < 30 mL/min/1.73 m^2) and patients taking a thiazide diuretic require more frequent monitoring (e.g. electrolytes, fluid balance).[1, 21] MRAs, ARNIs and SGLT-2 inhibitors also have a mild diuretic effect, so may influence the dose of other diuretics being used concurrently.[19]

Supress the renin-angiotensin-aldosterone system (RAAS)

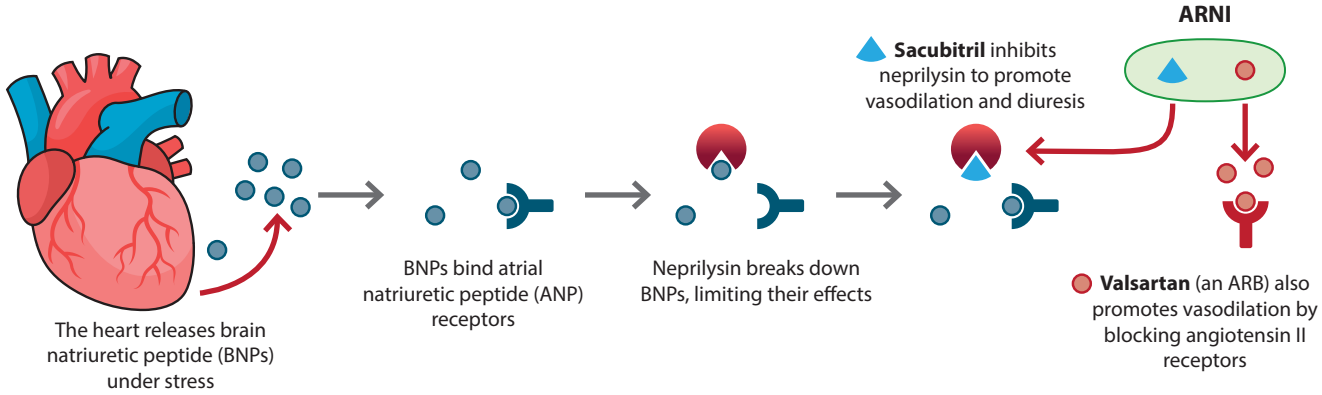
The RAAS regulates blood volume, electrolyte balance and systemic vascular resistance, and comprises three main components: renin, angiotensin II (derived from angiotensin I) and aldosterone.[22] In a normal physiological context, these factors help maintain blood pressure and fluid balance.[22] However, in heart failure, the RAAS is often overactivated as a compensatory mechanism that drives excessive vasoconstriction, sodium retention and fluid overload, contributing to increased blood pressure, worsening oedema and adverse cardiac effects, e.g. hypertrophy, fibrosis (**Figure 2A**).

RAAS inhibition is therefore an important early objective in patients with heart failure.[23] For decades this was achieved using ACE inhibitors or ARBs, but the development of ARNIs

A Heart failure pathophysiology and targets of "traditional" treatments



B (Dual) mechanism of action – ARNI (sacubitril + valsartan)



C Possible mechanisms of action – SGLT-2 inhibitor

Mechanism for cardioprotective benefits of SGLT-2 inhibitors not fully understood – numerous pathways have been implicated

Direct cardiac effects
- Improves cardiac remodelling
- Improves cardiac energy metabolism
- Decreases ischaemia/reperfusion injury
- Inhibits SGLT-1
- Increases cardiac autophagy and lysosomal degradation
- Reduces epicardial fat mass
- Reduces oxidative stress

Vasculature effects
- Decreases blood pressure
- Reduces inflammation
- Increases pro-vascular progenitor cells
- Improves overall vascular function

Systemic effects
- Increases erythropoietin levels
- Inhibits sympathetic nervous system activity

Renal effects
- Decreases hyperuricaemia
- Inhibits Na^+/H^+ exchange
- Increases natriuresis/diuresis

Figure 2. Heart failure pathophysiology and mechanism of action for **(A)** "traditional" heart failure medicines,**(B)** ARNIs and **(C)** SGLT-2 inhibitors.[14–16] Part A adapted from Schwinger *et al,* 2021.[14]

has since provided an alternative and more effective option (see: "Angiotensin receptor-neprilysin inhibitor (ARNI)").[23] Special Authority funding restrictions in New Zealand for ARNIs mean that ACE inhibitors/ARBs are still often the first step for RAAS inhibition due to the requirement that patients are receiving "*concomitant optimal standard chronic heart failure treatments*".[13]

ACE inhibitor and ARB treatment

ACE inhibitors reduce symptom severity and mortality in patients with HFrEF, and any option is suitable as beneficial effects are class-wide (**Table 1**).[4] ARBs, such as losartan or candesartan, are an alternative if an ACE inhibitor is not tolerated (e.g. due to cough or angioedema).[2] Preference for an ACE inhibitor first is largely based on their wider use in clinical trials, but some guidelines do not prioritise ACE inhibitors over ARBs.[4] If a patient is already taking an ARB (e.g. for pre-existing hypertension) there is no need for them to switch to an ACE inhibitor following a diagnosis of heart failure. Furthermore, changing from an ARB to an ARNI is more straightforward than from an ACE inhibitor to ARNI, due to the requirement for a wash out period (see next section).

Following diagnosis, ACE inhibitor/ARB treatment should be initiated at a low dose as soon as practically possible unless contraindicated (**Table 1**), including when symptoms of fluid overload are still present.[1, 4, 24] A conservative approach to up-titration was previously recommended (doubling doses every two to four weeks), however, recent guidelines advocate for more assertive treatment escalation (increasing doses every one to two weeks under close supervision).[4] In primary care, this schedule will be dictated by the capacity for regular patient review (i.e. whether appropriate monitoring can still occur with each dose change) and in many cases, fortnightly up-titration remains a reasonable strategy. Minor increases in serum creatinine and asymptomatic decreases in blood pressure are to be expected following initiation of the ACE inhibitor/ARB; if these exceed acceptable limits (**Table 1**), delaying up-titration (until resolution) or dose decreases may be needed.[2]

Angiotensin receptor-neprilysin inhibitor (ARNI)

Sacubitril + valsartan (Entresto) is the only available example in a medicine class combining an ARB with a neprilysin inhibitor (ARNI), providing a dual mechanism of action (**Figure 2B**). ARNI treatment is superior to ACE inhibitor/ARB use across a range of key outcomes in patients with HFrEF, including all-cause and cardiovascular death, major adverse cardiac events and hospitalisation.[25] ARNIs also improve markers of cardiac function such as left ventricular function (systolic and diastolic), BNP concentrations, burden of ventricular arrhythmias, as well as other clinical endpoints, e.g. quality of life, duration of hospitalisation required.[4] As such, there is a growing consensus that immediate or early ARNI use should be prioritised for RAAS inhibition where possible, including in patients already stabilised on an ACE inhibitor/ARB.[4]

Read the evidence

The original trial investigating sacubitril + valsartan (**PARADIGM-HF**) was stopped early after a median follow-up of 27 months due to the significant benefit associated with ARNI use versus ACE inhibitor treatment.[26] When taken concomitantly with a beta blocker, ARNIs reduce the absolute risk of cardiovascular death or hospitalisation by almost 5% compared with an ACE inhibitor in patients with symptomatic heart failure.[26] The number need to treat (NNT) during the study to prevent one primary event (involving a composite of CV death or hospitalisation relating to heart failure) was 21.[26]

Funded with Special Authority approval. Special Authority applications for sacubitril + valsartan can be made by any relevant practitioner for patients who have NYHA class II – IV symptoms and who are receiving concomitant optimal standard chronic heart failure treatments.[20] The criteria state that patients should either have a confirmed LVEF ≤ 35%, or if echocardiography is not practically possible, the medicine can be initiated if the clinician believes they are "*likely to benefit from treatment*".[20]

A 2023 Position Statement from the Cardiac Society of Australia and New Zealand and the New Zealand Heart Foundation recommended that changes should be made to facilitate ARNI treatment being fully funded without Special Authority requirements.[16] This would align with international guidelines that prefer an ARNI as the first line RAAS inhibitor. For current Special Authority criteria see: **https://schedule.pharmac.govt.nz/latest/SA2302.pdf**.

Dosing recommendations. Sacubitril + valsartan should not be initiated until at least 36 hours after the last dose of ACE inhibitor (a "wash out" period) due to the risk of angioedema.[1, 4] A wash out period is not required for patients switching from an ARB; the ARNI can be initiated 24 hours after the last ARB dose, i.e. when the next ARB dose would have been due. There are three strengths of sacubitril + valsartan available, each prescribed twice daily (**Table 1**):[20]

- 24.3 mg sacubitril/25.7 mg valsartan (rounded to 24 mg/26 mg)
- 48.6 mg sacubitril /51.4 mg valsartan (rounded to 49 mg/51 mg)
- 97.2 mg sacubitril /102.8 mg valsartan (rounded to 97 mg/103 mg)

In general, patients can be initiated on the "medium" 49 mg/51 mg dose, and up-titrated to the "high" 97 mg/103 mg dose if tolerated. The "low" 24 mg/26 mg starting dose is reserved for high risk patient groups, e.g. those with hypotension, older or frail patients, severe renal or hepatic impairment.[20]

Monitoring considerations. Blood pressure, renal function and serum potassium should be reviewed at initiation, with each dose increase, and every three to six months once the patient is stable (in the absence of relevant risk factors; **Table 1**).[1] If a patient's systolic blood pressure is < 100 mmHg at initiation, delay use until this is resolved.[20] Using BNP as a biomarker for treatment efficacy is not routinely recommended, but NT-proBNP (N-terminal proBNP) monitoring can be considered if necessary if this specific test is available at the local community laboratory (see: "Long-term monitoring").

Practice point: If symptomatic hypotension and/or systolic blood pressure < 95 mmHg occurs at any stage during sacubitril + valsartan treatment, ideally first address this by lowering the dose of any diuretic or other antihypertensive medicines.[4, 20] If hypotension persists, a dose decrease or discontinuation of sacubitril + valsartan can be considered.[20] However, restarting and/or up-titration should always be considered at later reviews.

Select a beta blocker with evidence of effectiveness in heart failure

Like RAAS inhibitors, beta blockers also reduce symptom severity and mortality in patients with HFrEF.[1] These medicines help to counteract the effects of prolonged sympathetic stimulation (**Figure 2A**), reversing adverse left ventricular remodelling, improving the ejection fraction, in addition to their rate controlling and anti-arrhythmic properties.[9, 27]

Bisoprolol, metoprolol succinate or carvedilol are recommended for patients with HFrEF.[1, 4] In general, bisoprolol and carvedilol are better tolerated, and generally safe in patients with asthma/COPD. Only these beta blockers with evidence of effectiveness in heart failure should be used in patients with HFrEF,[4] so switching is appropriate if a patient is already taking a different beta blocker for a co-morbidity.

Beta blockers should be initiated at a low dose as soon as practically possible (**Table 1**), but only once symptoms of acute fluid overload have resolved, if present (to improve tolerance).[1, 4, 24] Up-titration every two weeks until the target or maximum tolerated dose is reached is usually recommended in a community setting;[24] general resting heart rate targets are 50 – 60 bpm for patients in sinus rhythm or < 80 bpm for those with atrial fibrillation (although international guidelines differ in their recommendations for this target).[28] Blood pressure, serum potassium and creatinine should also be monitored at

each dose increase (**Table 1**). For some patients, up-titration needs to be more gradual to reduce the risk of adverse effects, and it can take longer to observe a change in symptoms.[24] Once beta blocker treatment is established, dose reduction or discontinuation is not needed during episodes of acute deterioration (see: "Decompensation in previously stable patients") or fluid overload, unless there are obvious signs of hypoperfusion.[1, 4]

Mineralocorticoid receptor antagonists (MRAs) recommended for most patients regardless of symptom severity

Aldosterone levels are frequently elevated in patients with heart failure, contributing to sodium retention (and potassium loss), sympathetic activation and myocardial fibrosis.[9] MRAs block these effects in patients with heart failure (**Figure 2A**), and when added to a background of RAAS inhibition and beta blocker treatment, significantly bolster reductions in hospitalisation/cardiovascular mortality risk (**Figure 1B**).[8]

Given these benefits, there has been a shift from the conventional sequence of starting treatment with an ACE inhibitor/ARB and a beta blocker, and only adding a MRA if the patient remains symptomatic, to early MRA introduction regardless of heart failure severity.[4, 5] **Spironolactone** is the first MRA option used (**Table 1**).[1, 2] This medicine should be prescribed with caution in patients with impaired renal function and may cause hyperkalaemia; creatinine and electrolytes should therefore be monitored regularly, and patients should be advised to avoid over the counter NSAIDs.[2]

If spironolactone is not tolerated or the patient experiences significant anti-androgenic adverse effects, an alternative dose-equivalent MRA, **eplerenone**, can be prescribed with Special Authority approval, provided that the patient has a LVEF < 40%.[20, 29] Both MRAs act on the same biological receptor, but eplerenone is more selective, and is associated with lower rates of gynaecomastia or breast tenderness which are common reasons for treatment discontinuation in males taking spironolactone.[29] A 2024 meta-analysis demonstrated that eplerenone is superior to spironolactone in reducing all-cause (HR = 0.78) and cardiovascular (HR = 0.54) mortality events.[29]

Sodium-glucose co-transporter-2 (SGLT-2) inhibitors address the cardiovascular-kidney connection

There is substantial interconnectedness between the cardiovascular system and the kidneys. SGLT-2 inhibitors (e.g. empagliflozin) were initially developed for the treatment of diabetes, but are also highly effective for improving prognostic outcomes in patients with heart failure (regardless of LVEF) and protect against the progression of proteinuric renal dysfunction in patients with chronic kidney disease (which is common in this setting; **Figure 2C**).

Most patients with heart failure are undertreated

Contemporary data on adherence to GDMT recommendations in New Zealand is not available. However, international evidence indicates that sub-optimal treatment is common.[33] Among 1,062 patients with HFrEF enrolled in the European multicentre SMYRNA Study:[33]

- RAAS inhibitors (ACE inhibitors/ARBs/ARNIs) were prescribed to 76% of patients, of which 24% were receiving the target dose
- Beta blockers were prescribed to 89% of patients, of which 11% were receiving the target dose
- MRAs were prescribed to 55% of patients, of which 11% were receiving the target dose

An observational study following 2,588 outpatients with chronic HFrEF in the United States found that after 12 months of follow up, < 25% were simultaneously receiving a RAAS inhibitor, beta blocker and MRA, and < 1% were treated with all three medicines at target doses.[34] Subsequent analysis demonstrated that low blood pressure, medicine intolerance or contraindications did not explain the lack of treatment escalation in most cases (< 2% of patients had absolute contraindications to treatment with all three medicines).[9, 35] These studies pre-date the widespread inclusion of SGLT-2 inhibitors as a component of GDMT; a more recent analysis (EVOLUTION HF) suggests SGLT-2 initiation is often delayed compared with other components of GDMT.[36]

Multiple factors might explain why so few patients are prescribed optimised GDMT, including the perception that they are "too sick" to tolerate the combination of medicines, or that they are "not sick enough" to require them.[7] However, as a progressive condition, optimising heart failure treatment is always an important goal in the absence of contraindications, even if the patient currently has a good functional status and a low symptom burden. A lack of familiarity with GDMT and a rapidly evolving evidence base may also contribute to underuse.

Table 1. Recommendations for medicine use in patients with HFrEF or an undifferentiated clinical diagnosis of heart failure. See **Figure 1** for further information. N.B. If the target dose cannot be achieved, aim for the highest tolerated dose.[1, 4, 20]

	Usual starting dose*	Target dose	Up-titration strategy	Initiate only if	Monitoring
ACE inhibitor			**Conventional approach:** Gradual increases to maintenance dose; in general, doubling dose no sooner than every two to four weeks **Assertive approach:** Guidelines now indicate that up-titration every one to two weeks is reasonable, but rapid titration should only occur with close supervision Some treatment protocols recommend switching to ARNI after achieving mid-range ACE inhibitor or ARB dose (see below)	▪ Systolic blood pressure is ≥ 100 mmHg ▪ Serum potassium is < 5.5 mmol/L; significant caution is still required between 5.0 – 5.5 mmol/L ▪ Creatinine is < 250 micromol/L or eGFR is ≥ 30 mL/min/1.73 m² (seek cardiology advice if not) ▪ In general, discontinue potassium supplements and potassium-sparing diuretics before introducing an ACE inhibitor ▪ ACE inhibitor contraindicated in patients with history of idiopathic or hereditary angioedema	▪ Check serum potassium and creatinine† one week after first dose ▪ Check blood pressure, serum potassium and creatinine prior to each dose increase; delay dose increase or seek cardiologist advice if systolic blood pressure is < 95 mmHg, serum potassium is > 5.5 mmol/L or creatinine is > 25% above baseline ▪ Regular physical examination: weight, pulse, jugular venous pressure, chest auscultation ▪ Once stable dosing is achieved, continue long-term and monitor every three months (or more frequently if required depending on the patient)
Enalapril	2.5 mg, once or twice daily	10 – 20 mg, twice daily (higher doses indicated in some patients, e.g. those with hypertension). Once stabilised, total daily dose can be given once daily, if tolerated.			
Quinapril	2.5 – 5 mg, twice daily	20 – 40 mg, daily, in 1 – 2 divided doses (higher doses indicated in some patients, e.g. those with hypertension)			
Lisinopril	2.5 mg, once daily	20 – 40 mg, once daily			
Perindopril	2 mg, once daily	4 mg, once daily			
Ramipril**	1.25 mg, once daily	10 mg daily, preferably taken in two divided doses			
ARB					
Candesartan	4 mg, once daily	32 mg, once daily			
Losartan	12.5 mg, once daily	150 mg, once daily			
ARNI‡					
Sacubitril/ valsartan	▪ 49 mg/51 mg, twice daily, for most patients ▪ 24 mg/26 mg, twice daily, may be suitable for higher risk patients (see main text)	97 mg/103 mg, twice daily	Increase dose every two weeks	▪ As for ACE inhibitor/ ARB (above) ▪ Patient has stopped taking an ACE inhibitor/ARB ▪ It has been at least 36 hours since last ACE inhibitor dose or at least 24 hours since last ARB dose	
Beta blocker					
Carvedilol	3.125 mg, twice daily	25 mg, twice daily, for patients weighing < 85 kg or 50 mg, twice daily, for patients weighing ≥ 85 kg	**Conventional approach:** Gradual increases to maintenance dose; in general, doubling dose no sooner than every two to four weeks **Assertive guideline approach:** Increase dose every two weeks until maximum tolerated or target dose is reached (ensure appropriate monitoring occurs at each dose increase)	▪ Symptoms of fluid overload have resolved and there are no symptoms of worsening heart failure ▪ No symptomatic bradycardia, hypotension or second- or third-degree heart block	▪ As for ACE inhibitor/ ARB/ARNI above ▪ If the patient has first degree heart block (i.e. PR interval > 0.2 seconds), an ECG is recommended before each dose increase. If an ECG is not available, seek cardiology advice.
Bisoprolol	1.25 mg, once daily	10 mg, once daily			
Metoprolol succinate (modified-release)	23.75 mg, once daily	190 mg, once daily			

Table 1 Cont.

	Usual starting dose*	Target dose	Up-titration strategy	Initiate only if	Monitoring
MRAs					
Spironolactone **Eplerenone‡**	25 mg, once daily	50 mg, once daily	▪ Increase dose after two weeks	▪ eGFR is > 30 mL/min/1.73 m^2 ▪ Serum potassium is < 5.0 mmol/L	▪ Check creatinine and electrolytes regularly, i.e. at one week, one month and then at least six monthly
SGLT-2 inhibitor‡					
Empagliflozin	10 mg, once daily		Not applicable; continue treatment at 10 mg, once daily	▪ eGFR is > 20 mL/min/1.73 m^2 ▪ Patient does not have type 1 diabetes (due to risk of diabetic ketoacidosis)	▪ Assess renal function before initiation of concomitant medicines that may reduce renal function, then at least annually thereafter ▪ Warn patients about increased risk of Fournier's gangrene (rare). Recommend patients self-check their genitals and surrounding skin regularly for changes in integrity, inflammation or signs of infection. Consider temporarily stopping treatment in patients with active genital or urinary tract infections until resolved.

* For more specific dosing information refer to the NZ Formulary (NZF) at **nzf.org.nz**. In some cases, cardiologists may recommend slightly different dosing regimens, or general practitioners may decide on a different regimen depending on patient-specific factors.

† An increase in serum creatinine of up to 30% above baseline is acceptable following initiation assuming it does not exceed 250 micromol/L; subsequent up-titrations should only occur if the creatinine increase is ≤ 25% above baseline (otherwise seek cardiologist advice)

** Ramipril doses listed are for patients with heart failure without previous myocardial infarction (unapproved indication). Dosing recommendations differ for patients with heart failure post-myocardial infarction (approved indication), however, treatment will likely be initiated and supervised in hospital – refer to the NZF at **nzf.org.nz/nzf_1286** for further information.

‡ Special Authority approval required for funded access

Read the evidence

Pivotal trials (**DAPA-HF** and **EMPERPOR -Reduced**) found that SGLT-2 inhibitor use in patients with HFrEF reduced the:[30]

- Combined relative risk of first hospitalisation for heart failure and cardiovascular mortality by 26%
- Composite of recurrent hospitalisations for heart failure or cardiovascular mortality by 25%
- Risk of a composite renal endpoint (≥ 50% decline in eGFR, end-stage renal disease or renal death) by 38%

Benefits were observed in patients regardless of diabetes status, age/sex, ARNI use or baseline health status. Further analysis indicates that cardioprotective effects occur for both patients with HFrEF and those with HFpEF (see: "Treatment of patients with heart failure with preserved ejection fraction (HFpEF)").[31]

SGLT-2 inhibitors are taken at one specified dose in patients with heart failure, without the need for up-titration (**Table 1**).[20] Empagliflozin is the only SGLT-2 inhibitor available in New Zealand, funded with Special Authority approval. As of 1st December, 2024, **the empagliflozin Special Authority criteria has been widened** to include patients with symptomatic HFrEF regardless of their diabetes status.[32] These changes now facilitate funded delivery of the final GDMT component for patients with HFrEF in New Zealand.

Practice point: Given the changes in Special Authority access, consider reviewing and adding a SGLT-2 inhibitor to the treatment regimen of any patient with HFrEF or an undifferentiated clinical diagnosis of heart failure under your care, even if they do not have diabetes and are already stabilised on otherwise optimised medicine use.

A clinical audit on optimising heart failure treatment for primary care is available at: **https://bpac.org.nz/audits/heart-failure.aspx**

Monitoring the effectiveness of treatment

Monitoring patients with newly diagnosed heart failure is influenced by multiple factors, including their baseline clinical status and co-morbidities, the presentation setting (i.e. community versus hospital) and profile of medicines introduced. In some cases, patients may already be taking some GDMT medicines, whereas others may require the introduction of several within a short timeframe. A unifying principle is that assessment should be more intensive initially, and then gradually relaxed as clinical improvements and treatment optimisation occurs.

Short-term monitoring

Patients with newly diagnosed heart failure who do not require hospitalisation should initially be reviewed at least weekly.[4, 11] Key monitoring requirements include:[1, 4]

- Medicine-specific adverse effects (see **Table 1** and **NZF**)
- Changes in symptoms and signs (particularly congestion), exercise tolerance and the associated impact on daily activities
- Blood pressure
- Routine laboratory tests, e.g. electrolytes, renal function. There is some evidence from the STRONG-HF trial that NT-proBNP testing may assist decision-making when undertaking assertive GDMT up-titration (i.e. at one week post-initiation), but this is not routinely recommended in primary care.[37]

Review can progress to every two weeks once the clinical situation improves and initial progress with GDMT has been made. In patients who required hospitalisation, close follow up is particularly important during the first six weeks post-discharge to reduce the risk of re-hospitalisation and death.[4, 11]

Adverse effects should be expected with GDMT, and occur in 75 – 80% of patients.[38] However, a meta-analysis suggests that overall reported rates across RCTs are not substantially different between intervention and placebo arms;[38] this may reflect the significant impact heart failure has on general health, rather than effects solely attributable to specific medicines.[38] Renal impairment and/or hyperkalaemia are common barriers to the initiation and up-titration of GDMT.[4] In patients with hyperkalaemia, educate about the importance of a low potassium diet and prioritise introducing a SGLT-2 inhibitor (alleviates high potassium).[4]

Practice point: Serum creatinine increases up to 30% above baseline are common and acceptable following initiation of ACE inhibitor/ARB, ARNI or SGLT-2 inhibitor treatment.[24, 39] This is partly due to increased diuresis, therefore furosemide dosing should be reviewed and decreased if the patient is euvolemic. Subsequent up-titration should only occur if serum creatinine is < 250 micromol/L and increases are ≤ 25% above baseline.

The cumulative blood pressure lowering effects of GDMT medicines may be a limiting factor when trying to achieve target doses, particularly in older frail patients.[4] However, unless blood pressure is < 90 – 100/60 mmHg (or there is evidence of orthostatic hypotension, hypoperfusion or low output) and all other potential causes have been excluded, low blood pressure alone should not be a reason to withhold

GDMT.[7] In addition, a potentially paradoxical effect for ARNIs, beta blockers and MRAs on blood pressure has been noted in clinical trials depending on the patients initial blood pressure, where:[40–42]

- Blood pressure reduces in patients who at the start of treatment have systolic measurements > 135 – 140 mmHg
- Blood pressure gradually increases in patients who at the start of treatment have systolic measurements < 105 – 110 mmHg

Long-term monitoring

Most patients with stable heart failure on optimised GDMT dosing can be reviewed every three to six months in primary care.[4] However, certain patients may require more frequent follow up, e.g. those who are frail or at increased risk of decompensation.

Repeat echocardiography is recommended to assess structural and functional cardiac changes in the long-term for patients receiving treatment, e.g. three to six months after medicine titration has been completed.[4] Many patients diagnosed with HFrEF who are treated using GDMT can achieve a LVEF > 40%.[4] This approach allows for further decisions regarding the use of advanced treatments (see: "Beyond primary care"), and further imaging may be considered based on the patient-specific symptoms and characteristics.

N.B. While the frequency of echocardiography should depend on clinical indication, the timing of access may be influenced by region-specific resource availability.

If BNP levels were substantially elevated at diagnosis, monitoring for decreases can be considered as a further marker of treatment efficacy. However, serial measurements are not routinely recommended in the setting of chronic heart failure and decreases may not occur in all patients.[4] BNP monitoring is likely only suitable if it will influence management decisions, particularly if there is uncertainty about the cause of a change in symptoms, e.g. whether improvement in shortness of breath is due to the changing status of COPD or heart failure. If serial BNP testing is done for any reason, do not repeat tests within two weeks, and ideally request no more than four tests per year.[43]

Practice point: If serial BNP monitoring is being considered in patients taking an ARNI (sacubitril + valsartan), NT-proBNP testing is preferable, if available (this can differ depending on the region).[4] Valsartan inhibits neprilysin which normally breaks down BNP-32 (**Figure 2B**); this leads to modestly elevated BNP-32 levels during ARNI use, potentially confounding interpretation of treatment efficacy (levels would otherwise be expected to drop if treatment was successful).[4] In contrast, the biologically inactive NT-proBNP is not a substrate for neprilysin and therefore not affected by valsartan neprilysin inhibition. As such, NT-proBNP levels generally decrease consistently with effective treatment, meaning it remains a more accurate measure of heart failure severity.[4]

Additional medicines to consider based on patient co-morbidities

Digoxin. Consider digoxin if patients have heart failure associated with atrial fibrillation and symptoms cannot be adequately controlled with a beta blocker.[1, 2] There is some evidence digoxin may improve symptoms and reduce the rate of hospitalisation, however, it does not improve survival.[1] If digoxin is used, lower doses (e.g. 62.5 – 125 micrograms once daily) are generally recommended;[2, 21] there is evidence that mortality is significantly higher in patients with serum levels ≥ 1.2 ng/ml.[21] Monitoring of digoxin serum levels is not routinely required and treatment effect is assessed based on the patient's heart rate. If serum digoxin needs to be investigated to rule out high or toxic levels (e.g. significant adverse effects), consider assessment after four weeks, aiming for levels of 0.5 – 0.9 ng/mL.[2]

Anticoagulants. An anticoagulant should be considered in patients with heart failure associated with atrial fibrillation who are at risk of stroke.[1] The **CHA_2DS_2-VASc** is a recommended tool to assess stroke risk.

Intravenous iron. Anaemia is common in patients with heart failure and often occurs as a result of iron deficiency; other potential causes include vitamin B12 and folate deficiency or chronic kidney disease.[1] Iron deficiency can also occur without anaemia.[1] After addressing any reversible causes, e.g. blood loss, consider iron replacement for patients with heart failure who have serum ferritin levels < 100 micrograms/L, or serum ferritin levels 100 – 300 micrograms/L and transferrin saturation (TSAT) < 20%.[1, 2] Conventional thresholds for diagnosing iron deficiency (usually serum ferritin ≤ 20 micrograms/L) are not reliable in patients with heart failure as this condition involves a systemic inflammatory state and ferritin levels become elevated in response to inflammation.[44] Oral iron supplementation has minimal benefit in such patients.[1] Instead, administering intravenous iron is often preferred in patients with heart failure who are iron deficient and correction can improve symptoms, exercise tolerance and reduce the risk of hospitalisation, as well as improve quality of life outcomes.[1, 2]

N.B. Ferric carboxymaltose (Ferinject) is suitable for administration in primary care and is funded with Special Authority approval for patients

Decompensation in previously stable patients

Episodes of acute deterioration (decompensation) can occur for various reasons in patients with heart failure who have previously been stable.[1] Some patients are prone to recurrent episodes of decompensation despite ongoing treatment adherence.

Multiple factors can result in decompensation, including:[2]

- Poor medicine adherence or changes to the patient's regimen, e.g. reducing diuretic dose, adding a new medicine (including over-the-counter, particularly NSAIDs)
- Uncontrolled hypertension
- Cardiac arrhythmia (most often atrial fibrillation)
- Cardiac ischaemia
- Cardiac infection or inflammation
- Systemic infection (secondary to increased haemodynamic demand on the heart)
- Changes in diet (primarily affecting sodium) and fluid intake
- Changes in exercise levels
- Physical or mental exhaustion
- Chronic right ventricular pacing
- Substance misuse

If patients experience acute deterioration requiring hospitalisation, a key focus post-discharge is to ensure GDMT is optimised, and to introduce or up-titrate medicines if not, as appropriate (see: "The case for more assertive GDMT optimisation: STRONG-HF").[11,12] This is also an important time to reinforce lifestyle advice and refer for additional support, if required, e.g. dietitian.

with anaemia or iron deficiency anaemia who meet specific criteria. As of January, 2025, patients with iron deficiency alone are not eligible for funded treatment in primary care but some PHOs may offer funding for select patients. See the **Special Authority form** for the most recent criteria as this may change over time.

Treatment of patients with heart failure with a preserved ejection fraction (HFpEF)

The prognosis for patients with HFpEF is less well defined compared with HFrEF. Evidence suggests morbidity outcomes (e.g. hospitalisation rates, symptom severity) are similar between the two groups.[4,5] Patients with HFpEF have an increased risk of all-cause mortality (potentially reflecting the higher burden of co-morbidities among this group), whereas those with HFrEF have higher rates of cardiovascular mortality.[4,6]

Unlike HFrEF, there is minimal evidence for the effective treatment of patients with HFpEF. With the exception of SGLT-2 inhibitor treatment, trials investigating GDMT in these patients have not demonstrated the same level of efficacy.[13] If at any point echocardiography results demonstrate that the patient has HFpEF, seek cardiology advice which will guide further treatment decisions (if not already detailed on the echocardiography report).

General principles of management for patients with HFpEF includes:[2,5]

- Loop diuretic at the lowest possible dose if the patient has fluid overload
- Management of co-morbidities, e.g. atrial fibrillation, ischaemic heart disease, hypertension, diabetes
- Non-pharmacological management strategies (see: "Non-pharmacological changes to support treatment success")
- ARB (rather than ACE inhibitor) and/or beta blocker as required without the need to maximise the dose. Diltiazem and verapamil can be considered as an alternative to beta blockers for rate control (these are contraindicated in patients with HFrEF). ARNIs are a possible alternative to ARBs, however, patients may not be eligible for funded treatment (see: "Angiotensin receptor-neprilysin inhibitor (ARNI)")
- MRA can be considered to reduce the risk of hospitalisation
- SGLT-2 inhibitor, but Special Authority restrictions may limit access (see below, and: "Sodium-glucose co-transporter-2 (SGLT-2) inhibitors address the cardiovascular-kidney connection")

Medicine selection depends on underlying co-morbidities (and optimisation should be directed by a cardiologist), but ideally should include a SGLT-2 inhibitor; international guidelines strongly advocate for their use in all patients with HFpEF without contraindications.[5, 11, 13] However, despite demonstrated benefits, patients with HFpEF (i.e. LVEF ≥ 50%) are not eligible for initial SGLT-2 inhibitor (empagliflozin) Special Authority approval unless the application is submitted prior to imaging confirmation (i.e. an echocardiogram is "*not reasonably practical, and in the opinion of the treating practitioner the patient would benefit from treatment*"). Patients with echocardiography-confirmed HFpEF not already established on a SGLT-2 inhibitor may wish to consider self-funding treatment, but this will be a barrier to access for some patients.

Non-pharmacological changes to support treatment success

Lifestyle advice and education are important to improve the outcomes of pharmacological treatment in all patients with heart failure, regardless of the subtype.[2]

This includes:[1, 2, 4]

- Understanding appropriate action(s) to take if symptoms worsen
- Regular exercise as appropriate/tolerated
- Reducing daily sodium intake (preferably < 3 g daily; no more than 5 g daily)

  Read the evidence

  The SODIUM-HF trial demonstrated that a strict low sodium diet of < 1.5 g daily did not reduce the risk of a composite endpoint including cardiovascular-related admission to hospital, cardiovascular-related emergency department visit, or all-cause death in patients with heart failure compared with those receiving "general advice to restrict dietary sodium" (N.B. This comparator group is therefore still sodium restricted).[47] However, modest improvements in patient-reported quality of life and clinician-assessed NYHA functional class were identified in the low sodium group.[47]
- Weight loss if the patient is overweight
- Consuming an adequate but not excessive amount of fluid, e.g. 1.5 – 2 L daily
- Reducing alcohol intake and smoking cessation, if relevant
- Encouraging influenza, pneumococcal and COVID-19 vaccination (these infections can be a significant cause of decompensation); people with congestive heart failure are eligible for funded annual influenza vaccination

Beyond primary care

The journey for patients with heart failure can vary substantially. For patients who remain symptomatic despite optimal medicine use, or who require frequent secondary care involvement, additional options to improve survival include surgery or device management, e.g. with an implantable cardioverter defibrillator, cardiac resynchronisation therapy (CRT) or CRT-defibrillation.[2]

Consider referral to a cardiologist to discuss these advanced procedures for patients with:[2]

- LVEF persistently < 40% or high symptom burden even with GDMT
- Valvular heart disease, or other forms of confirmed underlying cardiac pathology
- Heart failure and syncope – insertion of a pacemaker may be required
- Heart failure and left bundle branch block, with a wide QRS complex on ECG associated with ventricular dyssynchrony – CRT may be indicated
- A history of cardiac arrest or ventricular tachycardia – defibrillator therapy may be indicated

Despite advances in pharmacological, surgical and device interventions over time, fewer than one in five patients hospitalised for heart failure are alive ten years later (mean survival following first hospitalisation for those aged 75 – 84 years = 2.87 years).[48] If patients with advanced heart failure continue to experience deteriorating and distressing symptoms when treated, the focus may need to shift from prevention of disease progression to improving quality of life outcomes in a palliative care setting. Given that this can be a confronting topic, it is important to introduce the concept as early as possible during a shared discussion between the patient, family/whānau or carer, cardiologist and primary care team.[2] Through this approach, patients can progressively evaluate and convey when they feel the time is right to make this transition.

For further information on advance care planning, see: **www.hqsc.govt.nz/our-programmes/advance-care-planning/**

Acknowledgement: Thank you to **Dr Raewyn Fisher**, Cardiologist and Clinical Lead for Cardiology, Te Whatu Ora Te Tai Tokerau (Northland) for expert review of this article.

N.B. Expert reviewers do not write the articles and are not responsible for the final content. bpac^nz^ retains editorial oversight of all content.

This publication was supported by an unrestricted educational grant by Novartis NZ Ltd. The publication was independently written and Novartis had no control over the content. The views expressed in this publication are those of the author and not necessarily those of Novartis.

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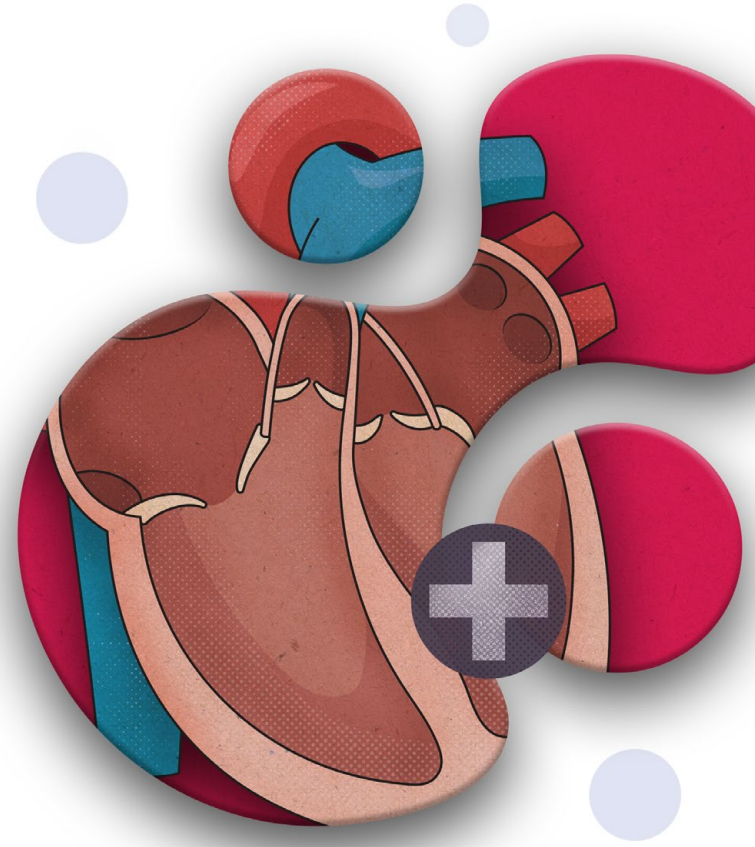

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