

Heart Failure: End-stage management

Queensland Health Guideline

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Disclaimer

This guideline is for information purposes only. The guideline is not a substitute for clinical judgement, knowledge and expertise, or medical advice. Variation from the guideline, considering individual circumstances, may be appropriate.



Overview

Purpose and Scope

This guideline provides recommendations regarding best practice to support quality of life for people with heart failure during the different phases of advanced disease. The specific aims are to assist the health care team to:

1. Recognise when a patient is approaching end-stage heart failure (using identifiers) and how to shift palliative intent towards a focus on quality of life.
2. Appropriately manage implantable cardiac defibrillator (ICD) deactivation
3. Manage common symptoms of end-stage heart failure.

Recommendations are for all clinical staff within their professional scope of practice and independent of the location of care.

Identifying end-stage heart failure

A palliative care approach is suitable alongside heart failure therapy, when the focus is on preventing disease progression, and can be provided by a range of health professionals, including cardiologists and GPs. A palliative care approach can be provided by addressing needs, providing symptom relief, support, and services.

Due to the unpredictable trajectory of heart failure, it may be difficult to determine when to shift the intent of care from a focus on preventing disease progression to a focus on quality of life. The identifiers of end-stage heart failure, outlined in the table below, indicate the need to start discussions with specialist palliative care and shift the intent of care to focus on quality of life.

Identifiers of patients with end-stage heart Failure

1. Symptoms consistently present on any physical activity or at rest (i.e. New York Heart Association (NYHA) Class IV).

And

2. Not suitable for any further procedural interventions (such as revascularisation with coronary bypass surgery, coronary angioplasty, valve surgery/procedure, biventricular pacing, or cardiac transplantation).

Plus, meets at least one of the criteria below

3. Increasing heart failure symptoms despite maximum tolerated heart failure therapy (as indicated).
 4. Worsening or irreversible end organ damage (including cardiac cachexia).
 5. Repeated hospital readmissions with deteriorating heart failure, ventricular arrhythmias, or cardiac arrest.
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The trajectory of heart failure and the palliative approach to care

The illness trajectory for heart failure is complex. The end-stage is characterised by intermittent acute exacerbations with periods of remission (which can last for months or even years) with sudden death possible at any time. It is difficult to predict the severity or timing of the next exacerbation or when sudden death might occur. Following an exacerbation, patients often do not return to their previous level of function (see figure 1).

Due to the unpredictability of heart failure, patients should be encouraged to undertake advance care planning soon after diagnosis regardless of their clinical status.[1] In Queensland, patient choices can be recorded using an [Advance Health Directive, Enduring Power of Attorney \(health and financial\), and Statement of Choices document](#). The Office of Advance Care Planning accepts these documents from all hospitals, health services, Residential Aged Care Facilities and individuals (email to acp@health.qld.gov.au). Documents are uploaded to The Viewer (this is an application used within Queensland Health that gives access summary records).

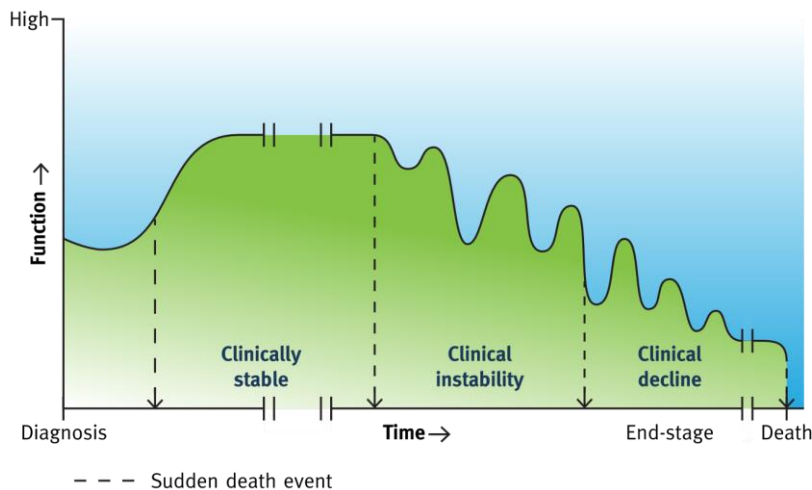


Figure 1: Trajectory of Heart Failure, adapted from Goodlin[2]

A palliative approach is relevant across the heart failure trajectory. Care can be provided by a range of health care professionals, including specialist palliative care clinicians, based on the patient and family's needs. Treatment goals should be discussed between the patient, family, and the healthcare team periodically throughout the trajectory of their illness and the intent to care adjusted accordingly.

The palliative intent is particularly important for individuals with end-stage heart failure (as per the identifiers above) and involves shifting towards treatment goals that prioritise quality of life over preventing disease progression. This change in intent includes managing increasingly emerging symptoms and acute exacerbations, as well as psychosocial support for the patient and the family.

Medication management

Medications for heart failure frequently need to be continued as they also provide symptom relief in addition to improving life expectancy. As end-stage heart failure progresses, the approach to medications may shift away from achieving target doses and move to focussing on controlling refractory symptoms. Symptom control may require down titrating medicines to minimise hypotension or prevent unnecessary kidney damage.[3] Such changes in the goal of medications require an ongoing conversation with the patient and their family.

Medication review

A full medication review should be undertaken with a view to rationalising medications with questionable benefit in the face of limited prognosis. Target groups of medication to consider deprescribing would usually include statins, vitamins, bisphosphonates (for osteoporosis) and proton pump inhibitors.

Caution when deprescribing

- Cessation of medications will need sensitive explanation as patients may have been informed that these are lifelong therapies.
- Beta-blockers should be weaned slowly due to risk of reflex tachyarrhythmias.
- Withdrawal of anti-coagulation needs to be discussed and individualised for each patient due the risk of significant morbidity such as stroke.

Managing drug interactions

Drug-disease interactions are common for patients with end-stage heart failure due to changes in pharmacokinetics as a result of impaired renal function, reduced hepatic metabolism, and gut oedema. Some interactions are acceptable as they provide a substantial benefit. The acceptability of risk will depend upon the potential severity of the interactions, patient and family wishes, the phase of palliation, disease trajectory, location of care, and availability of alternative options.

Opioids

Health providers and patients often have fears relating to opioids. Patients may think that death is imminent or that there is danger of addiction. It is very important to communicate with patients that opioids are used for both pain and dyspnoea with no demonstrated increase in risk of respiratory depression. [4] Specialist palliative care nurses and physicians are skilled in administering opioids and this may be an opportunity to seek/consult them for their expertise.

Renal dysfunction

Renal failure is a common feature of end-stage heart failure. The balance between the control of fluid overload and renal function may need to be relaxed towards the final phases of end-stage heart failure to maximise symptom control. For example, to avoid recurrent pulmonary oedema, a deterioration in renal function may be deemed acceptable.

Implantable cardioverter defibrillators (ICDs)

Implantable cardioverter defibrillators (ICDs) treat potentially lethal arrhythmias with either anti-tachycardia pacing or electric shock. Ventricular arrhythmias may trigger repeated shocks at the end of life that are extremely traumatic, painful and ineffective.

Discussion regarding deactivation of the defibrillator should occur at implantation and periodically thereafter as the condition progresses.[5] NB. The pacing or resynchronization therapy function (if present in the ICD) can continue to function to provide symptomatic relief until the end of life.

Indications for ICD deactivation:

- Patient preference
- Irreversible terminal condition
- A decision is made not to provide cardiopulmonary resuscitation
- Withdrawal of anti-arrhythmic medications related to a downward illness trajectory
- Imminent death

Planned ICD deactivation

Check whether there is a local deactivation policy and procedure.

Steps	Process and documentation for permanent ICD deactivation
1. Clinical discussion	<input type="checkbox"/> Indications for deactivation discussed with the patient's main cardiologist/general physician (or on-call consultants in urgent situations)
2. Patient and family discussion	Careful discussion with the patient and/or their family about the benefits, risks, and consequences of deactivation ICD. Key points: <ul style="list-style-type: none"> <input type="checkbox"/> Deactivation will avoid repeated shocks that are ineffective and painful and will not cause pain or sudden death <input type="checkbox"/> The pacing function will continue (if it is present) <input type="checkbox"/> The ICD can be reactivated if circumstances change
3. ICD deactivation	<ul style="list-style-type: none"> <input type="checkbox"/> An acute resuscitation plan (ARP) is required prior to deactivation <input type="checkbox"/> Modification of ICD settings may be done by the hospital implant team or device company representative (location dependent).
4. Document	<ul style="list-style-type: none"> <input type="checkbox"/> Patient consent <input type="checkbox"/> Reason for defibrillator deactivation <input type="checkbox"/> Persons present at the discussion about deactivation <input type="checkbox"/> Discussion outcomes with patient's usual cardiologist/ general physician <input type="checkbox"/> Acute resuscitation plan completed and reviewed <input type="checkbox"/> Confirmation that the defibrillator has been turned off <input type="checkbox"/> Sign off by the senior clinician

Urgent or temporary deactivation

Urgent deactivation can be considered if planned deactivation has not been arranged and the patient has reached the stage of imminent death. An ICD should ideally be deactivated by the implanting team or clinical representative from the ICD device company. If this is not possible (or cannot be performed in the required time) temporary deactivation using a magnet can be undertaken by any healthcare practitioner while awaiting manufacturer's representative to attend.

Magnet

Temporary deactivation is achieved by placing a strong magnet directly over the implant site of the ICD. Tape the magnet to the chest so it stays in place (see figure).

The magnet resets the device to the default manufacturer's programming which stops the device delivering a shock or anti-tachycardia pacing (ATP) but does not disable bradyarrhythmia pacing. This is a temporary solution as the device will revert to the planned programming if the magnet is removed.



Figure: taping magnet

Managing an ICD in situ after death?

Interrogation of the device should be considered following sudden death to obtain information about the cardiac rhythm and device therapy immediately preceding death to aid in establishing cause of death.

ICDs should be deactivated prior to autopsy or removal from the body to avoid risk of shock to the person performing the procedure.

All implanted electronic devices should be removed prior to cremation to avoid explosions at intense temperatures.[6]

Symptom control in end-stage heart failure

End-stage heart failure symptom management		
Symptom	Non pharmacological	Pharmacological*
General	Causes of symptoms are often multifactorial. Assess and address reversible causes	Avoid nonsteroidal anti-inflammatory drugs (NSAIDs) as they may exacerbate fluid overload; gastrointestinal bleeding; or cardiac and renal dysfunction
Dyspnoea	Fan to move air, repositioning, and oxygen (see also oedema, pain, and anxiety)	Diuretics for fluid related dyspnoea, oral opioid for pain and benzodiazepine for anxiety (see oedema, pain, and anxiety)
Oedema	< 2 gm salt daily and fluid restrictions. Restrictions may be relaxed if nearing end of life. Ice cubes etc for thirst. Rest with feet up	Oral frusemide up to 120 mg 2-3 times a day. Add in thiazides or spironolactone if needed or substitute with bumetanide. Beta-blocker dose reduction or cessation with caution not to unmask arrhythmias IV diuretic intermittent bolus or infusion
Pain	Massage, heat packs, physiotherapy, meditation etc.	Mild pain: paracetamol (avoid NSAIDs) Moderate to severe pain: Where eGFR is <30 extend morphine dosing interval or use hydromorphone (pre-empt opioid constipation with laxatives) Neuropathic pain: pregabalin or amitriptyline
Anxiety and depression	Mindfulness, cognitive behavioural therapy, coping skills Spiritual support, clear and open communication, support groups	Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants Short acting benzodiazepines for anxiety (caution can exacerbate delirium)
Constipation	High-fibre foods, exercise, and enema for severe impaction	Stool softeners like Coloxyl® and osmotic laxatives such as Movicol®
Nausea and vomiting	Eat small amounts, often and slowly. Cold foods to minimise smell and taste	1st line: metoclopramide and domperidone 2nd line: haloperidol, or if anxiety is contributing, lorazepam
Anorexia & cachexia	Small, frequent servings and nutritional supplements. Maintain family mealtime routines/rituals	Megestrol acetate may stimulate appetite Fish oil may assist in treatment of cachexia
Fatigue	Energy conservation, relaxation, mindfulness, and meditation Aerobic and resistance exercise	Optimise cardiac output by maintaining cardiac medications where possible Consider reducing dose of beta blockers*
* Palliative care may consider providing injectable drugs for symptom management at home at the end of life in the event a patient cannot swallow		

Dyspnoea

Dyspnoea (breathlessness) is a very common symptom in patients with heart failure. The experience of dyspnoea is subjective and may not reflect oxygen saturation, particularly if anxiety is significant. Clinicians should address reversible causes where possible such as:

- Respiratory related causes (e.g. pulmonary oedema, pleural effusions, pulmonary embolism, pneumonia, chronic obstructive pulmonary disease (COPD), respiratory muscle fatigue)
- Poor cardiac function (low output state), cardiac ischaemia
- Anaemia and iron deficiency
- Anxiety

Non pharmacological measures for dyspnoea

- A fan providing moving air may reduce perception of dyspnoea
- Supportive care, a reassuring presence, and a calm environment
- Repositioning
- Oxygen therapy and positive pressure ventilation may help breathing or sleep apnoea in some cases.

Pharmacological measures for dyspnoea

See principles of medication management in end-stage heart failure.

Pharmacological measures will vary depending on the primary cause of the dyspnoea.

- Start with standard treatments including diuretics
- **Opioids.** Low dose opioids are shown to be effective for management of dyspnoea and have a low risk of side-effects.[7] NB. Regular laxatives are highly recommended when using opioids.
 - If opioid naïve, use a low dose oral morphine 1-2 mg one to two hourly PRN. If there is concurrent renal impairment, the dosing interval may need to be extended or use an alternative opioid such as hydromorphone. NB. Oral hydromorphone is 5-7 times more potent than morphine and only requires very small doses i.e. 0.25-0.5 mg PRN. If 3 sequential doses of oral hydromorphone are ineffective, then medical review is recommended to consider dose titration.
 - A higher starting dose may be suitable for patients currently prescribed opioids.
 - If a short acting opioid is effective, consider long acting sustained release (SR) preparations in an equivalent dose. NB. Contact your local palliative care service for advice before switching to SR preparations.
- **Benzodiazepines** can alleviate anxiety. Initial doses should be small. NB. Exclude delirium as this may be exacerbated using benzodiazepines.
- **Beta blockers.** Review dose and continuation of beta blockers especially in the presence of ongoing fluid overload in end-stage heart failure.

Oedema

Oedema in end-stage disease can be challenging due to altered drug absorption, pharmacokinetics, and worsening renal failure.

Non pharmacological measures for oedema

- Salt and fluid restrictions can help control oedema but may need to be relaxed in the terminal phase of the illness.
- Thirst can be quenched by ice cubes, frozen fruit and sugar free lollies or gum.
- Low salt diet (< 2 gm daily) and avoid high sodium content medicines.
- Resting with feet up (compression stockings or bandages are unlikely to help.[8])

Pharmacological measures for oedema

Cautions

- Non-steroidal anti-inflammatory drugs (NSAID) may exacerbate fluid overload.
- Beta-blockers can exacerbate congestion; however, reduction or cessation may unmask arrhythmias or worsen heart failure. A compromise in dose reduction may be needed.
- Frusemide up titration requires attention to the potassium and renal function (although electrolyte monitoring in the very end-stage disease is not usually clinically relevant).

Oral diuretics

If the patient is prescribed a diuretic and congestion is still problematic, consider:

- a) Titrating frusemide up to 120 mg two or three times a day. If this dose is of limited benefit, consider combining with the following oral agents:
 - Thiazide diuretic such as hydrochlorothiazide, 12.5 -25 mg once or twice daily
 - Spironolactone (use caution due to potassium accumulation)
- b) Bumetanide has better oral bioavailability than frusemide. Dosing: 1 mg bumetanide is equivalent to 40 mg of frusemide. (Bumetanide is subsidised when dispensed from a Queensland public hospital pharmacy.)
- c) Metolazone needs cardiologist permission to access under Therapeutic Goods Administration (TGA) Special Access Scheme (<https://www.tga.gov.au/form/special-access-scheme>). Dose: 2.5-5 mg once a day PRN (use caution due to electrolyte depletion).

Intravenous (IV) diuretics

- IV frusemide dose is based on current oral dose and degree of diuresis required. Dosing: IV frusemide 20mg is equivalent to oral daily dose of 40mg.
- Intermittent frusemide bolus injections can be provided up to 2 to 3 times a day for inpatients or once daily for outpatients.
- A frusemide continuous infusion of 5 mg an hour can achieve a gentle diuresis of 1-2 litre over 24 hours. Hospital initiation helps to clarify the dose prior to home IV diuresis.

Pain

Pain is common in all phases of heart failure and is often under undertreated.[9] Assess for the cause of the pain, duration, intensity and whether there is currently a pain management regime. Treat the source of pain where possible, e.g. where the pain is of cardiac origin ensure that cardiac treatments (such as anti-anginal agents) are optimised.

Non pharmacological measures for pain

Consider treatment options such as massage, heat packs, physiotherapy, and meditation.

Pharmacological measures

Avoid nonsteroidal anti-inflammatory drugs (NSAID) such as aspirin and ibuprofen as they may exacerbate fluid overload; gastrointestinal bleeding; or cardiac and renal dysfunction. Mild pain can be managed with regular paracetamol.

Neuropathic pain

Neuropathic pain is poorly responsive to opioids and will likely require alternatives. Consider pregabalin 25 mg and titrate as tolerated (limited by renal function). Pregabalin can cause peripheral oedema in 5% of patients. Other treatments may include low dose amitriptyline 10mg daily.

Opioids for moderate to severe pain

Morphine is the drug of choice for preserved renal function and hydromorphone for patients with impaired renal function (i.e. eGFR <30). Pre-emptively manage constipation with regular laxatives.

- If opioid naïve, use a low dose oral morphine 1-2 mg one to two hourly PRN. If there is concurrent renal impairment, the dosing frequency may need to be extended or use an alternative opioid such as hydromorphone. NB. oral hydromorphone is 5-7 times more potent than morphine and only requires very small doses i.e. 0.25-0.5 mg PRN. If 3 sequential doses of oral hydromorphone are ineffective, then medical review is recommended to consider dose titration.
- If a short acting opioid is effective for 2 to 3 days, consider long acting sustained release (SR) preparations in an equivalent dose. NB. Contact your local palliative care service for advice before switching to SR preparations.
- For patients on regular opioids, consider additional short acting opioids for managing “breakthrough” pain. The breakthrough pain top-up dose is usually between 1/10th and 1/6th of the daily opioid dose. (If the total daily oral morphine dose is 30 mg, then the top-up dose would range from 3 mg to 5 mg PRN.)

Anxiety and depression

Comorbid depression and anxiety are very common in end-stage heart failure.

Non pharmacological measures for anxiety and depression

Emotional and psychosocial support is extremely important.

- Group sessions for training in mindfulness, coping skills and support can reduce anxiety and depression in heart failure. [10, 11]
- If clinical depression is suspected, a psychiatric evaluation may be required before completing an Advance Health Directive.[3]
- Cognitive behavioural therapy and spiritual support can help.
- Anxiety may be relieved through actions that create a greater sense of control such as support groups and clear information and open communication.

Pharmacological measures for anxiety and depression

Depression

- Selective serotonin reuptake inhibitors (SSRI's) can be used with caution but can cause fluid retention or hyponatremia if there is renal insufficiency. The effect of onset is 1-2 weeks. Sertraline is the SSRI of choice in renal impairment due to the lack of accumulation of active metabolites. Start orally with a dose of 25 mg mane.

Anxiety

Benzodiazepines can alleviate anxiety if non pharmacological interventions are ineffective.[2]

When prescribing benzodiazepines, it is best to:

- First exclude delirium in the differential diagnosis of anxiety as this may exacerbated the delirium
- Preference short-acting benzodiazepines such as lorazepam and oxazepam
- Initiate with small doses

Gastrointestinal symptoms

Gastrointestinal symptoms causes are often multifactorial. Assess and address reversible causes.

Constipation

Laxative therapy requires clinical judgement as there is no evidence on which laxatives are better than others.[11] Constipation in end-stage heart failure may be due to medications, dehydration, and lack of physical activity.

Constipation in end-stage heart failure may be prevented or relieved by :

- Consumption of high-fibre foods and exercise such as walking
- Stool softeners with low fluid contents such as like Coloxyl® and Dulcolax®
- Osmotic laxatives (fluid is not absorbed by the gastrointestinal tract).
- Avoiding high fibre preparations that require a lot of fluid such Metamucil® or Fybogel®
- An enema if impaction is severe

Nausea and vomiting

- Avoid skipping meals as this can make nausea worse
- Aim to eat small amounts, often and slowly.
- Cold or room temperature foods may be better tolerated (as taste and smell is milder)
- First line agents include metoclopramide 10 mg up to three times a day, or domperidone 10 mg as prokinetic. Both are to be taken 30 minutes before meals.
- Second line agents include haloperidol 0.5 to 2 mg (orally or subcutaneously) two times a day up to 4 mg daily or lorazepam (if anxiety is contributing to nausea) 0.5- 1.0 mg PRN.

Cautions

- If more than one antiemetic is required long term that prolongs QT (metoclopramide, domperidone, and haloperidol etc.) consider obtaining specialist advice and weighing the risks and benefits. Lorazepam does not prolong QT and may be a preferable in some HF patients.
- Avoid ondansetron as it causes significant constipation and the cost is prohibitive.

Anorexia and cachexia

Poor appetite can compound weight loss and fatigue leading to physiological worsening of disease (including cachexia) and distress for the patient and family. Assess nutritional status and/or refer to a dietician.

Malnutrition in end-stage heart failure may be prevented or relieved by :

- Small, frequent servings that prioritise food with high calorie and protein content, use of nutritional supplements and maintaining family mealtime routines and rituals.[12]
- Megestrol acetate may stimulate appetite and promote weight gain.[12]
- N-3 polyunsaturated fatty acids (fish oil) may assist in treatment of cachexia.[13]

Fatigue

Assess and address reversible cause such as: anaemia, iron deficiency, hypothyroidism, depression, sleep disorders, breathlessness, pain, cachexia, infections, low muscle mass etc.

Non pharmacological measures for fatigue

- Energy conservation techniques (refer to occupational therapy)
- Regular aerobic and light resistance exercise (refer to physiotherapy)
- Relaxation techniques such as mindfulness and meditation

Pharmacological measures for fatigue

- Optimise cardiac output by maintaining cardiac medications where possible
- Consider reducing dose of beta blockers as they commonly cause fatigue

Document history/Version control

Version	Date	Comments
1.0	July 2021	<i>New</i>
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