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CLINICAL GUIDELINES ID TAG	
Title:	Guidelines for Management of Patients Hospitalized with Heart Failure
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Speciality / Division:	MEDICINE
Directorate:	CARDIOLOGY
Date Uploaded:	5TH AUGUST 2020
Review Date	3RD AUGUST 2023
Clinical Guideline ID	

Guidelines for Management of Patients Hospitalized with Heart Failure

Table of Contents:

These guidelines are based predominantly on:

- ✓ ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC): ESC clinical practice guidelines 2016: EHJ (2016) 37 (27):2129-2200 - https://doi.org/10.1093/eurheartj/ehw128

- ✓ Chronic heart failure in adults: diagnosis and management NICE guideline (NG106). Published date: 12 September 2018

These guidelines are a summary of the current guidelines and consensus documents of the ESC and are intended for internal use within the SHSCT only.

1. Abbreviations:

Table 2	
ACEi	Angiotensin Converting Enzyme Inhibitor
ADHF	Acute decompensated Heart Failure
AI	Aortic incompetence
ARB	Angiotensin Receptor Blocker
ARNI	Angiotensin Receptor-Neprilysin Inhibitors
AS	Aortic stenosis
AVR	Aortic Valve Replacement
BP	Blood pressure
CXR	Chest X-ray
ECG	Electrocardiogram
FBC	Full blood count
GDMT	Guideline-Directed Medical Therapy
HDU	High Dependency Unit
HF	Heart Failure
HFrEF	Heart Failure reduced ejection fraction
HFpEF	Heart Failure preserved Ejection Fraction
I+Os	Ins and Outs
JVP	Jugular venous pressure
LVSD	Left ventricular systolic dysfunction
MR	Mitral regurgitation
MRA	Mineralocorticoid Receptor Antagonist
MV	Mitral Valve
PND	Paroxysmal Nocturnal Dyspnoea
P2	Pulmonary component of second heart sound
RAAS	Renin-Angiotensin-Aldosterone System
RAS	Renin-Angiotensin System
RUQ	Right upper quadrant
TAVI	Transcatheter Aortic Valve Implantation
S3	Third heart sound
TTE	Transthoracic echo
TR	Tricuspid regurgitation

2. Introduction:

Heart failure is a condition in which the heart does not pump enough blood to meet all the needs of the body. It is caused by dysfunction of the heart due to muscle damage (HFrEF or HFpEF), valvular dysfunction, arrhythmias or other rare causes. Acute heart failure can present as new-onset heart failure in people without known cardiac dysfunction, or as an acute decompensation of chronic heart failure.

Acute HF is the leading cause of hospital admission in people 65 years or older in the UK. Heart failure accounts for a total of 1 million inpatient bed-days – 2% of all NHS inpatient bed-days – and 5% of all emergency medical admissions to hospital. Hospital admissions because of heart failure are projected to rise by 50% over the next 25 years largely as a result of the ageing population.

Heart failure has a poor prognosis: 30–40% of patients diagnosed with heart failure die within a year, but thereafter the mortality is < 10% per year. There is evidence of a trend of improved prognosis in the past 10 years. Effective multidisciplinary specialist services for people with CHF can have a positive effect on patients' life expectancy and quality of life and evidence suggests they can help to reduce recurrent hospital stays by 30–50%.

Early therapy for acute HF is crucial. There is evidence that both pharmacological and non-pharmacological treatments can improve patient quality of life, both in terms of physical functioning and well-being. There is also a strong evidence base for treatments to improve the prognosis of heart failure. Nevertheless, many patients remain sub-optimally treated, and the period during hospitalisation should be recognised as a time when therapies can be optimised.

3. Purpose of this policy:

The pathway to improve outcomes during and after HF hospitalisation begins with admission, continues through decongestion and transition to oral therapies before the day of discharge, and connects to a planned post discharge follow up.

This guideline covers the care of adults (aged 18 years or older) who have a diagnosis of acute heart failure, have possible acute heart failure, or are being investigated for acute heart failure. It includes the following key clinical areas:

- *the role of early natriuretic peptide testing and echocardiography*
- *the role of specialist management units*
- *the use of ventilatory support, pharmacological therapy after stabilisation, selected surgical interventions and initiation of pharmacological therapies that are used in the management of chronic heart failure.*

Drug recommendations

The guideline will assume that prescribers will use a drug's summary of product characteristics to inform decisions made with individual patients.

4. Scope:

This document provides guidance for any professional involved in the clinical management of patients with HF in hospital, and includes:

- Consultants
- SAS doctors
- SpRs
- Junior Doctors
- Specialist Nurses
- Nursing Staff
- General Practitioners

5. Key Points:

- Heart failure is a descriptive term for signs and symptoms of cardiac dysfunction that can arise due to many causes
- The heart failure team should be involved in the management of all patients with heart failure at some stage in their journey
- Accurate and timely diagnosis and management are key to improving the outcome for these patients
- Care should be taken with discontinuation, initiation and up-titration of HF medications to achieve a balance between optimal guideline directed therapy and adverse effects. This often takes several weeks – months to achieve
- Patients are frequently discharged to soon from hospital having residual oedema/ on-going symptoms. This has a negative impact on their trajectory. Euvolaemia should be achieved prior to discharge in most circumstances
- Patients should have a robust follow up plan established pre-discharge.

6. Diagnosis and Assessment:

Take a history, perform a clinical examination and undertake standard investigations.

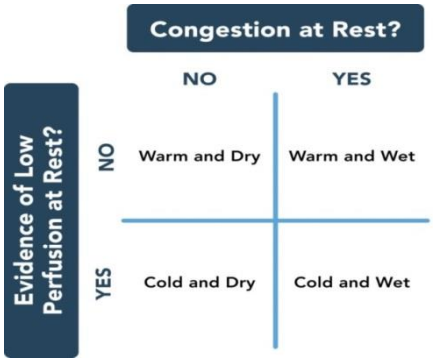
History and Physical Examination:

Table 3	
Clinical Evidence of Congestion	
SYMPTOMS	Dyspnoea on exertion
	Orthopnoea
	PND
	Nocturnal cough when supine
	Abdominal swelling
	Early satiety, anorexia, nausea/vomiting
	Peripheral oedema
	Rapid weight gain
	RUQ pain
SIGNS (**also see figure 1 and its explanatory text below**)	ASSESS VOLUME + PERFUSION STATUS (see figure 1 below)
	Elevated JVP
	Rales*
	Pleural effusion*
	S3
	Increased intensity of P2
	Murmurs of MR/TR, also AS/AI
	Ascites
	Pulsatile hepatomegaly
	Peripheral oedema – making sure to check for pre-sacral oedema/scrotal oedema

* = important to note that clear lungs does NOT rule out ADHF

Figure 1 outlines assessment of hemodynamic profiles for patients with HF.

Figure 1: Clinical Hemodynamic Assessment of Patients presenting with ADHF



The jugular venous pressure (JVP) reflects elevated right-sided filling pressures and is also a sensitive indicator of elevated left-sided filling pressures in patients with HF.

The 'warm and dry' profile represents a compensated patient and therefore HF is not the cause of their presentation (consider transient ischemia, arrhythmias, pulmonary disease).

The 'wet and warm' clinical profile (volume up without evidence of hypoperfusion) characterizes over 80% of patients admitted with ADHF. Rales, when present, usually indicate higher filling pressures than baseline, but are often absent in chronic HF due to pulmonary lymphatic compensation. Extensive pitting edema, ascites, or large pleural effusions reflect large extravascular reservoirs that may take many days to mobilize.

The 'cold and wet' profile describes congestion accompanied by clinical evidence of hypoperfusion, as suspected from narrow pulse pressure, cool extremities, oliguria, reduced alertness, and often recent intolerance to neurohormonal inhibition. Sleepiness, impaired concentration, and very low urine output may also be present. These patients may require adjunctive therapy with vasodilator or inotropic agents or decrease of medications with negative inotropic effects to improve cardiac output and facilitate diuresis. 15% of ADHF admissions.

True hypoperfusion without elevated filling pressures ('cold and dry') accounts for fewer than 5% of admitted patients and usually reflects aggressive prior therapy.

Investigations:

Table 4	
Investigations	
ECG	evaluate possible aggravating and precipitating factors (active ischemia, dysrhythmias)
CXR	Look for radiographic evidence of congestion/ cardiomegaly
BNP	In people presenting with new suspected acute heart failure, measurement of serum natriuretic peptides on admission (B-type natriuretic peptide [BNP] or N-terminal pro-B-type natriuretic peptide [NT-proBNP]): the following thresholds rule out the diagnosis of heart failure: BNP less than 100 ng/litre, NT-proBNP less than 300 ng/litre In patients with known chronic heart failure, admission BNP is required (compare with baseline BNP where available)
Other blood tests	• electrolytes, urea and creatinine • eGFR (estimated glomerular filtration rate) • thyroid function tests • liver function tests • fasting lipids • fasting glucose • full blood count • If new HFrEF is diagnosed, a cardiomyopathy screen should be sent (see Trust Intranet)
Urinalysis	evaluate possible precipitating factors (eg UTI)
TTE	If NEW suspected HF - Perform transthoracic Doppler 2D echocardiography to exclude important valve disease, assess cardiac structure and ventricular function, and detect intracardiac shunts. Ideally within 48 hours of admission. If ADHF in known chronic HF, no need to repeat TTE if performed within last 6-12 months (discuss with cardiology if unsure)

Assessing Risk during Hospitalisation:

A key message of this document is the importance of serial assessment from admission through discharge; therefore the risk factors listed in Table 5 are categorized according to the time when they may be known during the hospitalization. In setting goals to decrease risk and improve outcomes after hospitalization and later, it may be helpful to focus on those risk factors most likely to be modifiable.

Table 5	
Assessment of Risk: Markers of Higher Risk	
Chronic History Prior to Admission	Older Age
	Number of previous HF hospitalisations
	Comorbidities; renal dysfuntion, anaemia, diabetes, COPD, liver disease, cancer, dementia
	Frailty
	Known low LVEF in HFrEF
	Known Significant RV dysfunction
Assessment at Admission	Class IV symptoms
	Nonadherence to medications or salt/fluid restriction
	Progressively higher risk with rising natriuretic peptide levels
	Renal dysfunction markers: • Elevated serum creatinine or low GFR • Additional risk of high BUN • Diuretic resistance with high outpatient doses
	Degree of congestion at admission not predictive of outcome except longer length of stay with greater excess volume
	Low systolic blood pressure
	Hemodynamic profile of “cold and wet” at admission (see figure 1 below)
	Troponin elevation
	Hyponatremia
	Increased risk at admission if: • No RAAS therapy • No beta blocker therapy
	Intravenous inotropic therapy even if brief
	Resuscitation or Intubation
Assessment at Discharge	Assessment of volume and perfusion status
	BNP level measurement

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Natriuretic peptide levels are the most robust predictors of readmissions and death, and also highly modifiable with decongestion, and levels continue to decrease for days after discharge. The magnitude of decrease in natriuretic peptide levels during therapy is closely associated with decreased risk, and increase or failure to decrease levels is associated with higher risk. Absolute levels at discharge are also highly predictive of rehospitalization, need for advanced therapies such as transplant or mechanical circulatory support, and mortality.

7. Therapies:

7A.Initial pharmacological treatment

Offer intravenous diuretic therapy to people with acute heart failure. Start treatment using either a bolus or infusion strategy.

For people already taking a diuretic, consider a higher dose of diuretic than that on which the person was admitted (unless there are serious concerns with patient adherence to diuretic therapy before admission). Change total daily oral dose to equivalent frusemide total dose administered IV at 1 to 2.5 times the total daily dose (e.g., an outpatient bumetanide dose of 1 mg twice daily would convert to 80 mg daily furosemide equivalent, and the IV dose would be furosemide 80 to 200 mg IV daily).

For those patients who have not been on diuretics as an outpatient, initial furosemide dose can vary according to patients' fluid overload, kidney function, and age, and usually is around 40 to 80 mg IV daily dose.

IV diuretics are usually continued throughout the early hospital stay either by IV bolus every 8 to 12 hours or by continuous IV infusion (particularly in those patients with marked peripheral oedema)

Closely monitor the person's renal function, weight and urine output during diuretic therapy (daily U+E, daily early morning weights, daily I+Os). Discuss the best strategies of coping with an increased urine output and thirst with the patient (e.g. ice chips rather than boluses of water).

Do not routinely offer opiates to people with acute heart failure.

Do not routinely offer nitrates to people with acute heart failure. If intravenous nitrates are used in specific circumstances, such as for people with concomitant myocardial ischaemia, severe hypertension or regurgitant aortic or mitral valve disease, monitor blood pressure closely in a setting where at least level 2 care can be provided (Coronary Care or HDU).

Do not offer sodium nitroprusside to people with acute heart failure.

Do not routinely offer inotropes or vasopressors to people with acute heart failure. Consider inotropes or vasopressors in people with acute heart failure with potentially reversible cardiogenic shock. Administer these treatments in a cardiac care unit or high dependency unit or an alternative setting where at least level 2 care can be provided.

7B. Initial non-pharmacological treatment

We do not offer non-invasive ventilation routinely for patients with ADHF and pulmonary oedema. However, if a person has cardiogenic pulmonary oedema with severe dyspnoea and acidaemia consider starting non-invasive ventilation without delay: either

- at acute presentation or
- as an adjunct to medical therapy if the person's condition has failed to respond.

Consider invasive ventilation in people with acute heart failure that, despite treatment, is leading to or is complicated by:

- respiratory failure or
- reduced consciousness or physical exhaustion.

7C. Treatment after stabilisation

See Section 9 for optimization of GDMT, however the following provide guidance on continuing therapy and initiation of therapies.

7Ci. Beta-Blockers

In a person presenting with acute heart failure who is already taking beta-blockers, continue the beta-blocker treatment unless they have a heart rate less than 50 beats per minute, second or third degree atrioventricular block, or shock.

Start or restart beta-blocker treatment during hospital admission in people with acute heart failure due to LVSD, once their condition has been stabilised – for example, when intravenous diuretics are no longer needed.

Ensure that the person's condition is stable for typically 48 hours after starting or restarting beta-blockers and before discharging from hospital.

Non-dihydropyridine calcium channel blockers should be avoided in HFrEF. Ivabradine can be used in sinus rhythm but is usually reserved for chronic therapy.

7Cii. ACEi or ARB or ARNI, and MRA

Offer an ACEi (or ARB if there are intolerable side effects) or ARNI, and an MRA during hospital admission to people with ADHF due to LVSD. If ACEi / ARB / ARNI is not tolerated an MRA should still be offered.

Closely monitor the person's renal function, electrolytes, heart rate, blood pressure and overall clinical status during treatment with beta-blockers, ACEi / ARB or MRAs.

7D. Valvular surgery and percutaneous intervention

Where necessary, additional invasive procedures may be appropriate and will be guided by the consultant cardiologist.

Offer coronary revascularisation where HF is caused by active ischemia with viable myocardium.

Consider referral for surgical AVR to people with heart failure due to severe aortic stenosis assessed as suitable for surgery, and consider TAVI in those with heart failure caused by severe aortic stenosis who are assessed as unsuitable for surgical AVR.

Consider surgical MV repair or replacement for people with heart failure due to severe MR assessed as suitable for surgery.

Where there is refractory cardiogenic shock, at an early stage, the cardiologist should have a discussion with a centre providing mechanical circulatory support about:

- people with potentially reversible severe acute heart failure or
- people who are potential candidates for transplantation.

8. Daily Clinical Trajectory Check

Checking the clinical trajectory daily during hospitalization enables one to record responsiveness to therapy in terms of

- clinical HF symptoms and signs and
- laboratory and diagnostic tests

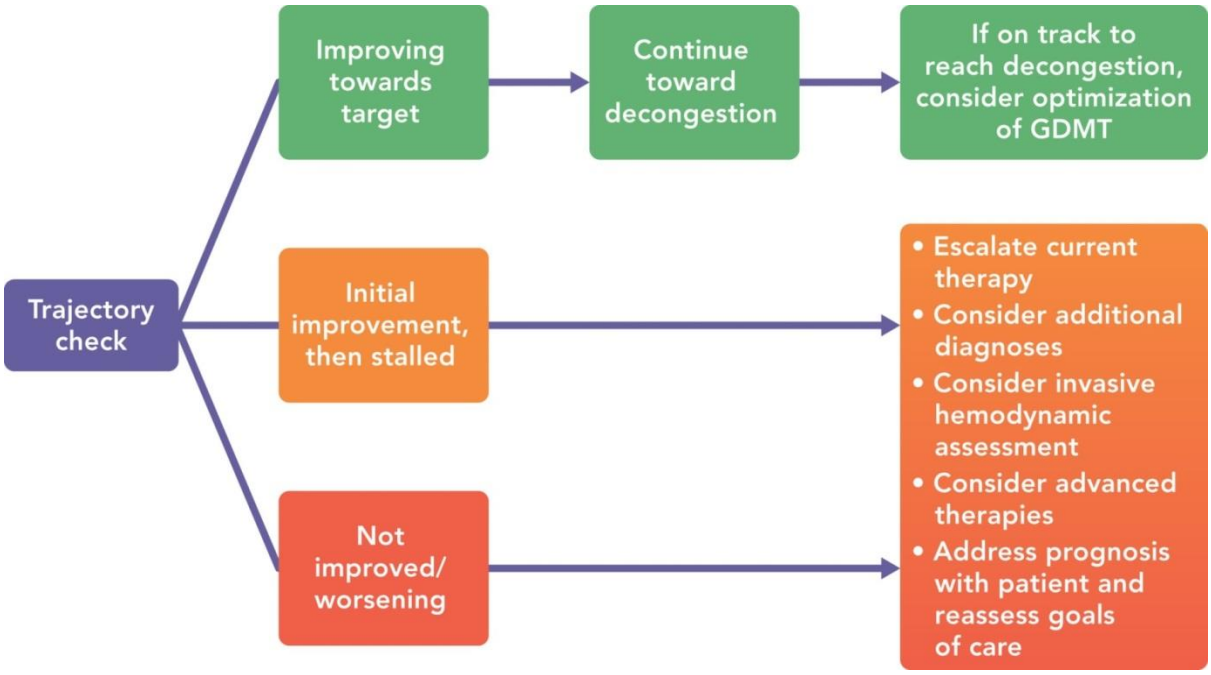
This trajectory helps define the next steps for management, risk and prognosis, and planning discharge and future care.

Determination should be made daily of one of 3 main in-hospital trajectories:

- improving towards target
- stalled after initial response
- not improved/worsening.

And follow the advice in figure 2 below to determine next steps.

Figure 2. Clinical Trajectories and Their Implications for Therapy



The usual goal is for complete decongestion, with absence of signs and clinical symptoms of elevated resting filling pressures. Rates of rehospitalization and death are consistently lower in patients rendered free of clinical congestion by the time of discharge.

Specific targets:

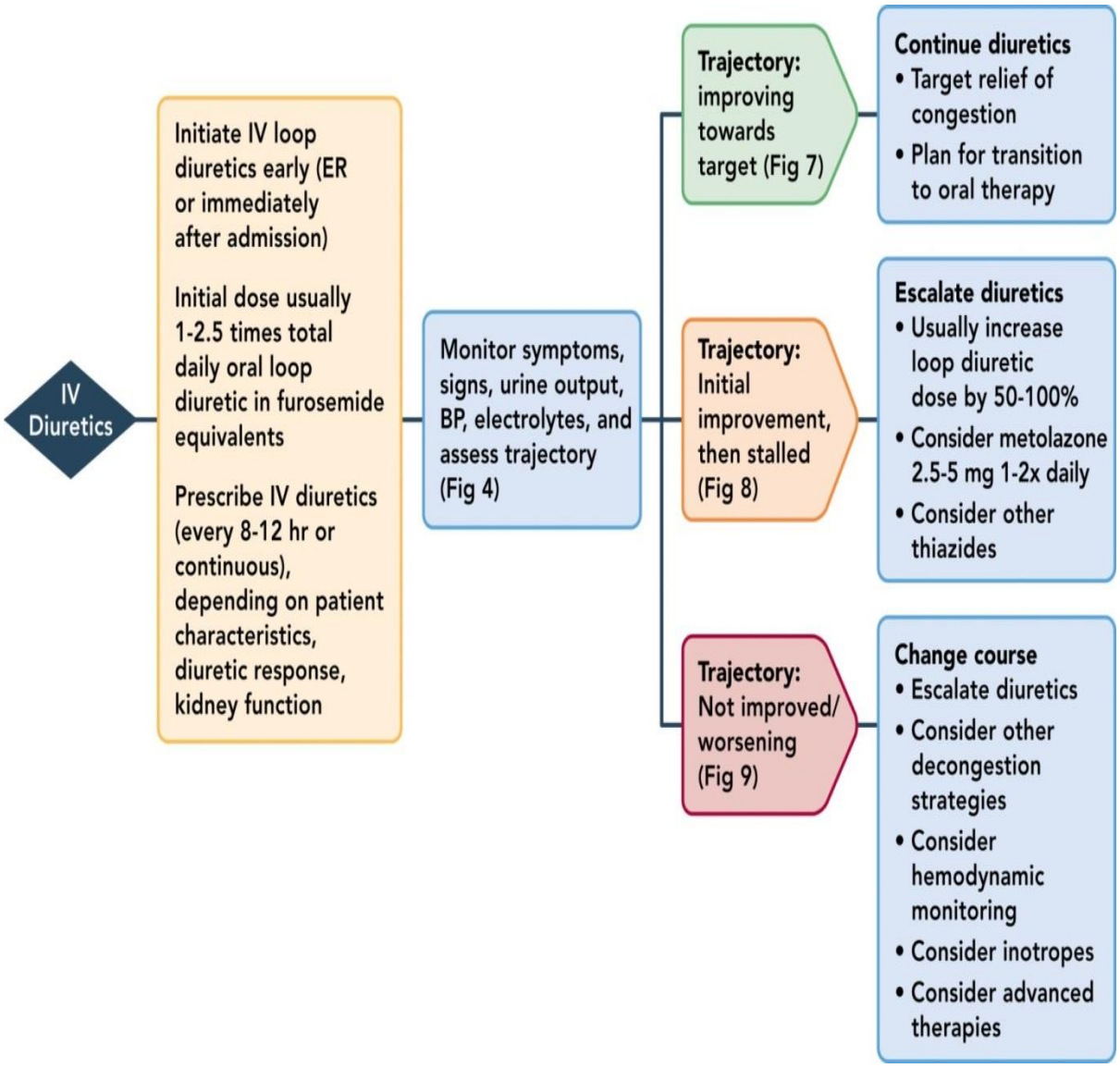
- JVP should generally be reduced to <8 cm total (ie not 8cm above the sternal angle)
- dyspnoea at rest should be relieved
- no residual orthopnoea or oedema

NOTE: Most patients report early improvement in symptoms, particularly dyspnoea. Symptoms of congestion usually improve before the signs of congestion have fully resolved. If guided only by symptom relief, diuresis will often be stopped too soon. Average weight loss in recent inpatient HF trials ranges from 4 to 8 kg.

When high furosemide doses are not effective, metolazone can be added at 2.5 to 5 mg doses once or twice daily. Other thiazide diuretics can be considered. See figure 3.

Intravenous vasodilators (e.g., nitroglycerin) represent another strategy in patients with refractory congestive symptoms. When added to diuretic therapy, IV vasodilators improve symptoms and hemodynamic evidence of congestion, but have not been associated with reductions in length of stay or mortality. Vasodilators may be particularly helpful in patients with symptomatic crashing acute pulmonary edema.

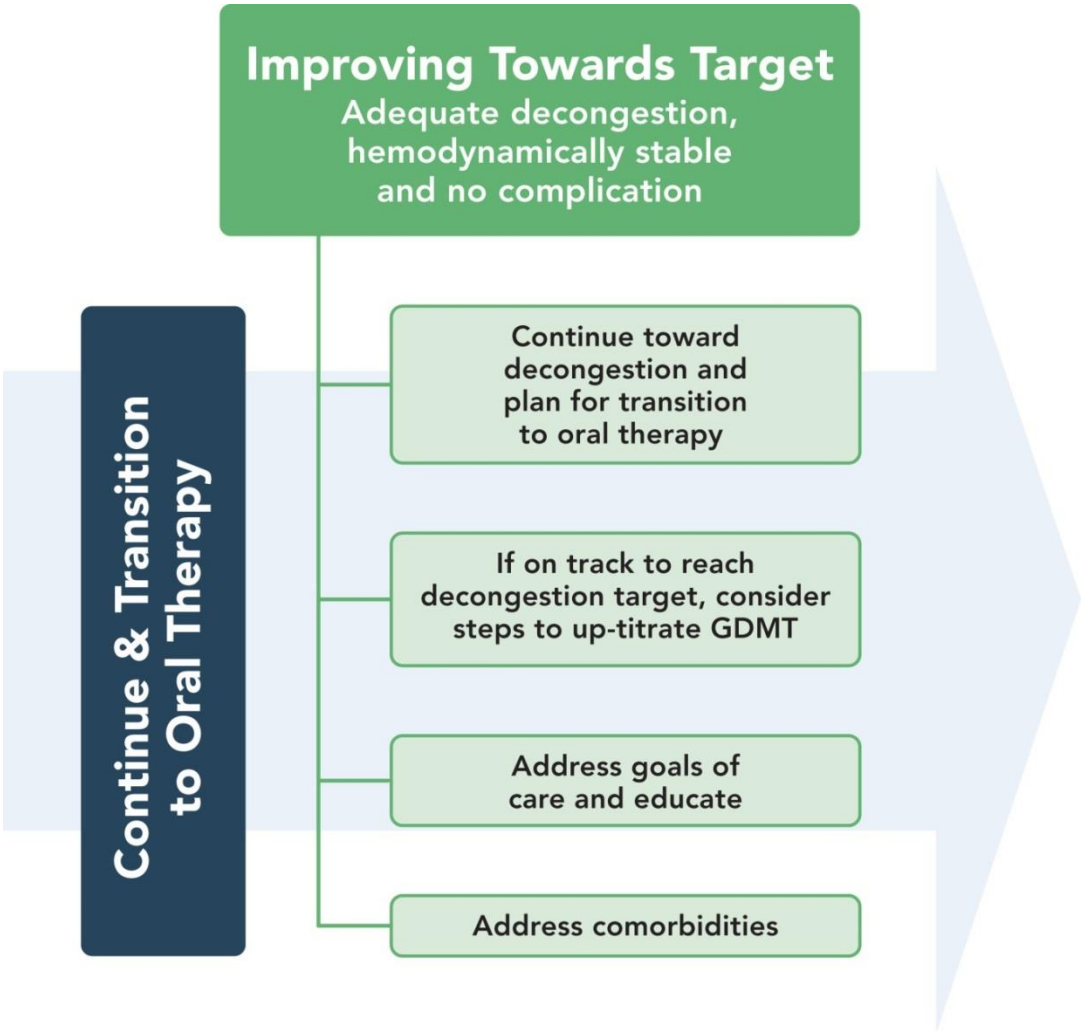
Figure 3. Diuretic Therapy in Different Clinical Trajectories



9. TRAJECTORY: Improving Towards Target - Optimization of GDMT

Continuation and optimisation of GDMT through hospitalization or initiation before discharge is associated with substantially better outcomes. This should be considered for all patients whose daily trajectory check shows they are improving towards target (see figure 4).

Figure 4: Clinical Trajectory in Patients Improving Toward Target



Hospitalization provides a pivotal opportunity to decrease risk and improve clinical trajectory in patients who respond well to diuresis and who have not previously received adequate trials of GDMT. GDMT modifies and frequently reverses disease progression. The introduction of GDMT during hospitalization for HFrEF is thus a key target to reduce risk. This has been shown for ACEI, beta blockers, and most recently supported for angiotensin receptor– neprilysin inhibitor (ARNI).

Patients with good early response to diuresis should be considered for addition of recommended therapies or up-titration toward trial targets for neurohormonal antagonist

therapy as decongestion is approached. Caution is advised in those with marked volume overload, recognizing that the diuretic response may diminish acutely with increasing neurohormonal antagonism, particularly if BP is lowered by these therapies.

Caution should be used when initiating beta blockers in patients who have required inotropes during their hospital course or when initiating ACEIs, ARBs or aldosterone antagonists in those patients who have experienced marked azotaemia or hyperkalaemia. In these circumstances, specialist advice should be sought.

For patients with HFpEF, beyond diuretics, clinical trial evidence that medical therapy improves outcomes is limited, but it seems reasonable to titrate RAAS inhibitors to desired blood pressures in hospital.

An important common principle is to start at a low dose and titrate slowly upward as tolerated. High starting doses and/or overly aggressive titration can result in hypotension and worsening renal function, setbacks that limit both decongestion and initiation of different components of GDMT.

9.1 RAS Inhibition Therapies

RAS inhibition is part of GDMT for patients with HFrEF, and should be continued or initiated in the absence of hypotension or unstable kidney function.

RAS inhibition can decrease BP in patients with pre-existing intense neurohormonal activation, so particular care should be taken in patients recently weaned from intravenous inotropic therapy. Caution should be exerted also in patients with acute kidney injury or hyperkalaemia.

If prior therapy was held during hospitalization, lower doses may be required when therapy is resumed. Transition through a short-acting agent such as captopril is rarely necessary, although it may be better tolerated in some patients with advanced HF.

If therapy was held in hospital and not resumed, discharge information to the outpatient clinician should include a reminder to consider re-initiation of neurohormonal therapies stopped in the hospital.

9.2 Angiotensin Receptor–Neprilysin Inhibitors

This class of agent is licensed for patients with chronic HFrEF, instead of ACEi therapy.

Initially, data from the pivotal PARADIGM-HF (Prospective Comparison of ARNI with ACEI to Determine Impact on Global Mortality and Morbidity in Heart Failure) trial for ARNI focused exclusively on stable chronic HF, and excluded patients recovering from acute decompensated HF.

However, the PIONEER-HF (Comparison of Sacubitril-Valsartan versus Enalapril on Effect on NT-proBNP in Patients Stabilized from an Acute Heart Failure Episode) trial now provides evidence to support safety of careful initiation of sacubitril-valsartan for hospitalized patients with and without prior exposure to ACEI or ARB, selected for hemodynamic stability, with systolic blood pressure >100 mm Hg and without escalation of intravenous diuretics or vasodilators for 6 hours, and without intravenous inotropic therapy within the previous 24 hours.

Data from this trial suggests that consideration of initiation of ARNI during the hospitalization is warranted, either in patients who have stabilized after initial diuresis, or in the period prior transitioning to oral therapy, but still excludes patients with recent hypotension or marked kidney dysfunction.

Patients need to be off ACEI therapy for 36 hours before starting ARNI therapy to decrease the risk of angioedema. Diuretic dosing may need to be adjusted after ARNI, as diuretic requirements sometimes decrease, but anticipatory reductions are not recommended, unless guided by HF specialist.

9.3 Beta blockers

In patients with HF with the 'wet and warm' profile who are taking beta blockers on admission, they should generally be continued, unless blood pressure is low or there is concern for significant bradycardia/AV block.

If HF remains refractory to diuretics, the dose should be halved.

Discontinuation should be considered if congestion remains unresponsive and if the addition of intravenous inotropic therapy is contemplated.

If decreased or held, beta blockers may be initiated or resumed in the absence of symptomatic hypotension or bradycardia, but a margin of stability is required in view of the known acute effects to lower cardiac output and increase filling pressures.
In hospitalized patients in whom GDMT medications have been held or not initiated, the optimal sequence of reinitiation of ACEI and beta blockers has not been established, although outpatient studies suggest that that either an ACEI or beta blocker may be initiated first.

Ensure that the person's condition is stable for typically 48 hours after starting or restarting beta-blockers and before discharging from hospital. Patients who have required temporary IV inotropic therapy during hospitalization represent a higher-risk cohort and require longer periods of observation prior to and after beta blocker initiation. When it has been difficult to wean inotropic therapy, use of beta blockers is often deferred until stability has been confirmed after discharge.

9D. Aldosterone Antagonists

Patients in whom an MRA was initiated or continued while receiving IV loop diuretics should be monitored closely for rebound hyperkalemia as the diuretic dose is decreased or transitioned to oral therapy.

Discontinuation of potassium supplementation may also be required.

Lower than standard doses (i.e., less than eplerenone 50 mg or spironolactone 25 mg daily) may also be considered in those with at least moderate kidney impairment or other risks for hyperkalaemia.

For patients in whom 2 inhibitors of the RAAS system are being initiated, reinitiated or uptitrated, a sufficient period of time should be allowed to observe the combined effects of the 2 therapies on kidney function and serum potassium concentrations. It should be emphasized that the peak effect on potassium retention is generally not observed for several days; kidney function and potassium should be checked within 72 hours of discharge, if changes in these medicines have occurred within 2 days prior to discharge. Nonetheless, initiation in the hospital is safe with careful monitoring, and inpatient initiation will most likely lead to greater long-term use.

10. TRAJECTORY: Initial Improvement, Then Stalled

This represents a patient who has had some improvement in symptoms and signs of congestion but does not reach the targeted goals of decongestion.

Specific targets:

- JVP should generally be reduced to <8 cm total (ie not 8cm above the sternal angle)
- dyspnoea at rest should be relieved
- no residual orthopnoea or oedema

Such patients tend to have more advanced disease, a history of frequent hospitalizations, and worse baseline kidney function. They commonly have high outpatient diuretic doses, and kidney function may worsen progressively with diuresis, a pattern associated with residual congestion and worse outcomes. Consideration must be given to the aetiology of their heart failure and efforts must be made where possible to address potential reversible causes. Efforts should be made to maintain biochemistry within normal limits to promote exchange of residual fluid from the extravascular space to the intravascular space. For example hyponatraemia will effect osmotic gradients and prevent removal of fluid from the extravascular space to the intravascular compartment. Escalation of diuretic, addition of thiazide diuretic or induction of inotrope support may be considered following review by the heart failure team.

In appropriate circumstances consideration may also be given to the need for advanced supportive therapies such as implantable devices, bridging therapies and cardiac transplantation.

11. Update and review

- This document will be updated every 3 years.
- Revisions will be made ahead of the review date if new, relevant national guidelines are published. Where the revisions are significant and the overall policy is changed, the authors will ensure the revised document is taken through the standard consultation, approval and dissemination processes.

12. References

- ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC): ESC clinical practice guidelines 2016: EHJ (2016) 37 (27):2129-2200 - https://doi.org/10.1093/eurheartj/ehw128
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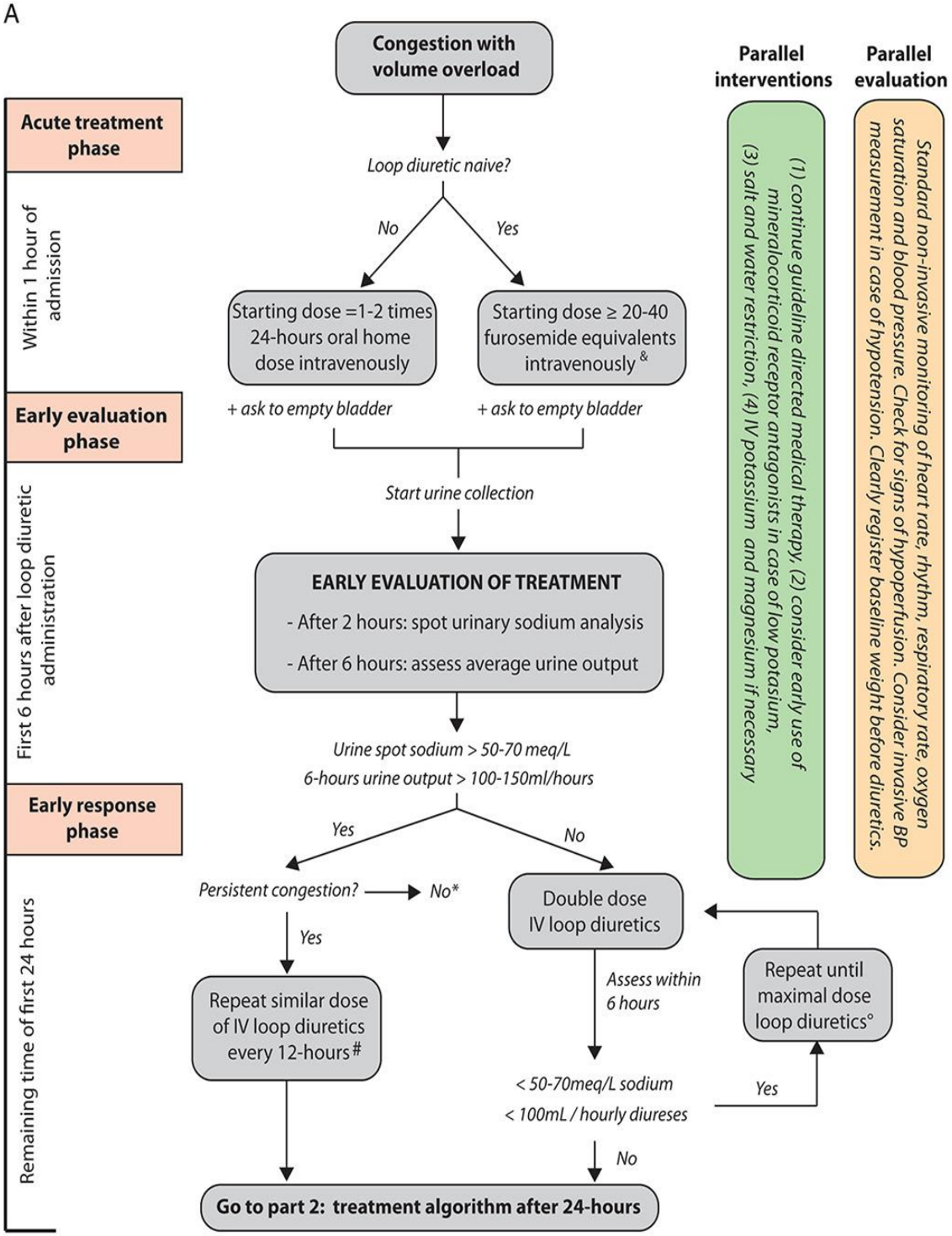
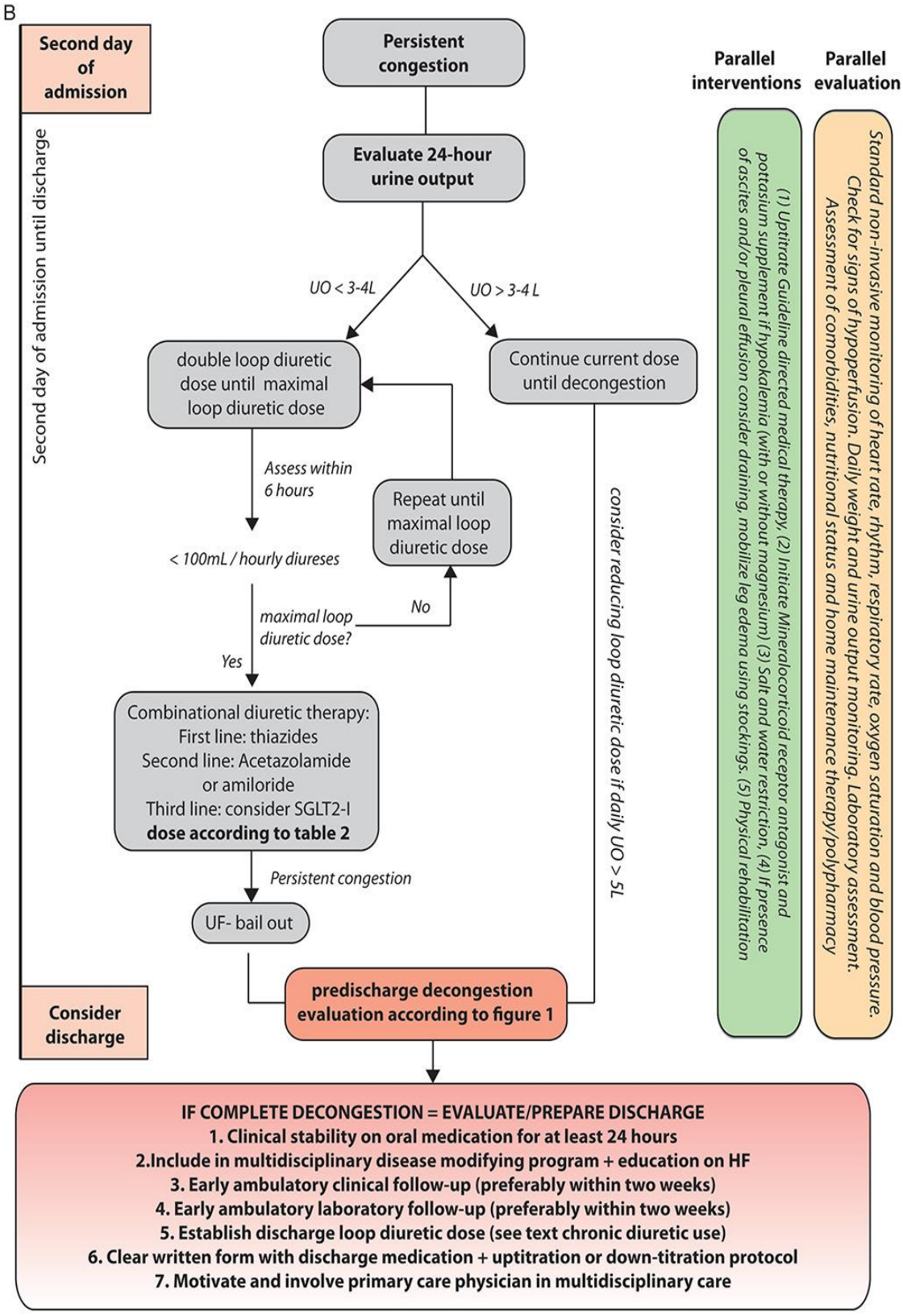
Appendix 1A: Acute phase treatment and early evaluation / early response

A



Appendix 1B: Second day of admission and discharge considerations



Appendix 2 – Governance information

Document title	Guidelines for Management of Patients Hospitalized with Heart Failure
Date issued / approved:	**5th August 2020**
Date valid from:	**5th August 2020**
Date valid to:	**5th August 2023**
Brief summary of contents	This document provides guidance for any professional involved in the clinical management of patients presenting with heart failure.
Policy objectives	To provide clear speciality agreed guidelines and pathways for the diagnosis and clinical management of patients with heart failure.
Keywords	Cardiology Heart failure
Authorship	Dr David Mc Eneaney (cardiology clinical lead, consultant cardiologist) Kay Carroll (cardiology head of service) Dr Patricia Campbell (consultant cardiologist) Dr Alastair Gray (consultant cardiologist) Dr Mick Connolly (consultant cardiologist) Dr Ian Menown (consultant cardiologist)