Chapter 10
Heart Failure

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Key Points
- NIV should be considered early in the management of heart failure.
- NTG is an excellent single agent for hypertensive heart failure.
- For normotensive pulmonary oedema, treatment should be focused on diuresis, supplemental oxygen and finding the precipitating cause.
- Vasopressors and inotropes should be started for cardiogenic shock, in the setting of hypotension and signs of poor peripheral perfusion. The options include norepinephrine, dobutamine, dopamine and milrinone.

Introduction

Heart failure is one of the most common reasons for hospitalisation among patients older than 65 years of age [1]. It is the third most common cardiovascular disease in the United States affecting almost five million people [2]. Also, these numbers are increasing with time for various reasons. This makes heart failure a major public health problem associated with a huge financial cost [1].

This chapter is focused on pathophysiology, diagnosis and management of heart failure from an ED perspective.

Emergency physicians play an extremely important role in the management of heart failure, as nearly 80 % of all the heart failure admissions come from the emergency department. Care given in the emergency department certainly affects the final outcome of these critically ill patients.

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S.S. David (ed.), *Clinical Pathways in Emergency Medicine: Volume I*,
DOI 10.1007/978-81-322-2710-6_10

Based on expert consensus, heart failure is defined as 'traditional signs and symptoms of heart failure requiring urgent or emergent therapy' [3].

Pathophysiology

- In pulmonary oedema, there is leakage of fluid from the pulmonary capillaries and venules into the alveolar space as a result of increased capillary hydrostatic pressure. There can be multiple causes for fluid leakage like LV systolic/diastolic dysfunction, excessive preload or venous return, excessive afterload or peripheral resistance (see Textbox 10.1).
- Initially, the body compensates though the patient may have orthopnoea and paroxysmal nocturnal dyspnoea (PND) as the initial symptoms. Once the system is overwhelmed, it leads to florid LVF.
- This involves a self-perpetuating cycle where acute LV systolic dysfunction leads to decreased myocardial contractility and cardiac output (CO), leading to catecholamine surge that causes increased SVR (afterload) and blood pressure.
- This rise in blood pressure increases the myocardial wall tension and myocardial oxygen demand. This cycle continues and eventually diminishes the myocardial contractility and CO.

Note: Almost half of the patients with acute pulmonary oedema have preserved ejection fraction and have a problem with fluid distribution, not fluid overload [4].

Causes of Heart Failure

The common causes of heart failure that are encountered in the ED are enumerated in Textbox 10.1.

Textbox 10.1 Common Causes of Heart Failure

- Ischaemic heart disease
- Arrhythmias
- Hypertensive heart failure
- Anaemia
- Cardiomyopathies
- Valvular dysfunction
- Cardiac tamponade
- Metabolic: hypothyroidism and hyperthyroidism
- Toxin induced: alcohol, cocaine and doxorubicin
- Iatrogenic: beta blockers and CCB

Diagnosis

History

- Heart failure is primarily a clinical diagnosis.
- Most patients with heart failure present with dyspnoea. Questioning about changes in their peak effort tolerance is important to grade and assess the severity of the disease.
- Orthopnoea and PND suggest acute exacerbation of the disease.
- Symptoms like weight gain, apparent lower extremity or sacral oedema or dyspnoea at rest suggest gradual worsening of the disease.
- It is also important to identify the underlying precipitant of the decompensating factor (see Textbox 10.2).

Textbox 10.2 Precipitants of HF
- Medications non-compliance
- Dietary non-compliance
- Arrythmias
- Infections
- Acute coronary syndrome
- Missed dialysis
- Exacerbation of chronic lung diseases
- Pulmonary embolus

Clinical Examination

- Look at the general appearance, posture, respiratory effort and diaphoresis.
- Assess for pallor, oedema, JVD or hepatojugular reflux, cyanosis, capillary refill time and mental status of the patient to assess cerebral perfusion.
- Check vital signs assessment and classify them as hypertensive, hypotensive or hypertensive heart failure.
- Lung examination often reveals crepitations. Auscultate on the front, back and at the bases of the lungs to diagnose occult disease. A wheeze might also be heard due to smaller airway narrowing and spasm (cardiac wheeze or cardiac asthma). If the air entry is less, then suspect a pleural effusion.
- Auscultation might reveal murmurs that might have precipitated the illness or an S3, which is a specific sign for heart failure.

Imaging and Labs

1. *ECG*
   - A completely normal ECG is extremely unlikely in the setting of heart failure, and it puts the diagnosis into question.
   - ECG may suggest underlying precipitants like pulmonary embolism and chronic hypertension and states leading to acute decompensation of heart failure like myocardial ischaemia and arrhythmias. ST-T changes may also be seen due to LV strain or NSTE-ACS.
   - The ECG image (Fig. 10.1) shows LBBB with LV strain and changes suggestive of LVH.
2. *Chest X-ray*
   - A chest radiograph (CXR) can help in reinforcing the diagnosis of heart failure with characteristic findings like Kerley B lines, prominent pulmonary vasculature in upper lung fields, cardiomegaly and pleural effusions.
   - It may also suggest other differential diagnosis like pneumonia, aortic dissection or a pulmonary embolus.
   - CXR is specific to pick up heart failure but not very sensitive as about 18 % of patients with acute decompensated heart failure have normal CXR [5].
   - A normal CXR cannot rule out heart failure. Always try to compare the current CXR with the former ones.
3. *Lung USG*
   - With the advent of lung ultrasound, it is now possible to do a bedside assessment for these critically ill patients. Lung USG can give valuable information about interstitial oedema (B lines), pleural effusions and consolidation.

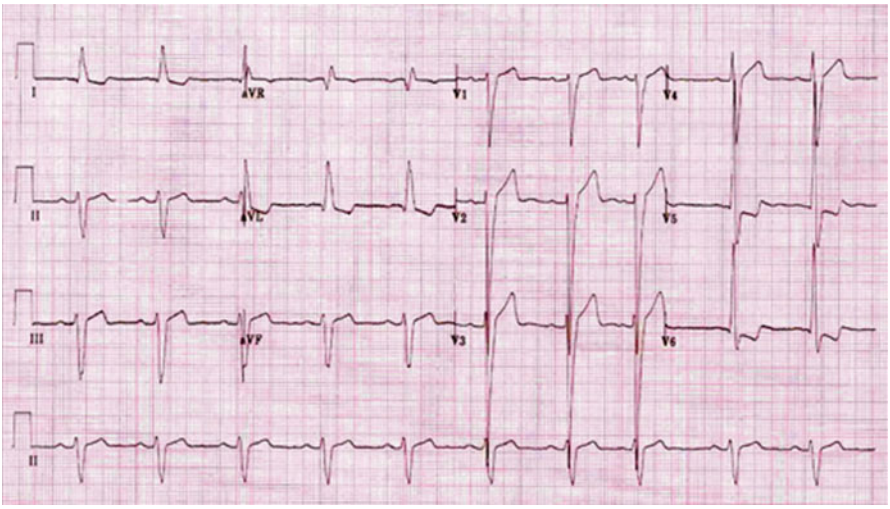


Fig. 10.1 ECG of a 56-year-old male with HF, showing LV strain in the lateral chest leads

4. *Echogram*

   Bedside echogram can assess LV function, presence of pericardial effusion, dilated right heart and valvular heart disease. This helps in diagnosing as well as prognosticating the disease. Emergency department bedside ECHO is often used for undifferentiated hypotension and dyspnoea that aids the diagnosis of heart failure.
5. *Laboratory investigations*

   - Laboratory testing is not required to diagnose heart failure, but to find out the precipitating causes, looking for the complications and prognosticating the disease.
   - The tests which are generally done include a complete blood profile, BNP, troponin and renal function tests (creatinine, urea and electrolytes).
   - Electrolyte abnormalities are commonly found due to the use of diuretics and volume overload. Potassium derangements may precipitate arrhythmias. Hyponatraemia, if present, suggests a poor prognosis and two- to threefold post-discharge mortality.
   - A majority of patients with heart failure suffer from CAD, and also ischaemia might be a potential precipitant of heart failure; therefore, troponin is frequently requested for these patients. But heart failure itself can raise the troponin levels, so often serial troponins are required to diagnose ACS unless straightforward ECG changes are present [6].

BNP

- Natriuretic peptide is synthesised as by the ventricular myocardium in response to raised ventricular pressures.
- Current guidelines recommend BNP assays to aid in the diagnosis or exclusion of acute heart failure [7].
- It is elevated in heart failure and a low level strongly suggests other potential diagnosis. *As always, biomarkers are not stand-alone tests; always use your clinical acumen while interpreting any biomarker and consider additional testing if required.*
- BNP also aids in determining the prognosis, morbidity and mortality. Other illnesses that may affect BNP levels are sepsis or renal dysfunction.

Differential Diagnoses

The differential diagnoses of heart failure include the conditions enumerated in Textbox 10.3.

Textbox 10.3 Differential Diagnoses of Heart Failure

- Exacerbation of chronic lung disease
- Pneumonia
- Pulmonary embolus

- Hepatic failure
- Sepsis
- Acute valvular insufficiency
- ARDS (non-cardiogenic pulmonary oedema)

Emergency Department Management

- From a treatment perspective, acute heart failure can be divided into three subtypes: hypertensive heart failure, normotensive heart failure and hypotensive heart failure (cardiogenic shock).
- Although the majority of patients with acute heart failure syndrome respond well to medical therapy, some patients will require ventilatory assistance.
- Timely interventions and appropriate use of nitrates, diuretics, ACE inhibitors and non-invasive ventilation (NIV) can reduce the need for mechanical ventilation and ICU admissions.

1. *Hypertensive HF*

   - This is the most toxic looking subtype of heart failure patients. Some physicians also refer to this as acute hypertensive decompensated heart failure.
   - The treatment should begin immediately here with non-invasive ventilation (NIV), followed by high-dose NTG drip, diuretics and ACE inhibitors.
   - *Setting up NIV early in the illness is crucial* [8]. Either CPAP or BiPAP may provide non-invasive ventilatory assistance. These devices reduce the need for endotracheal intubation, hospital length of stay and mortality.
   - *NTG is a single excellent first-line agent to relieve symptoms of hypertensive LVF* [8]. Although a large group of experts recommend loop diuretics as the first-line agents for ADHF, NTG is more beneficial in the emergency department set-up. It works almost instantaneously and it has a very short half-life.
   - It lessens both the RV and the LV pressures, decreases the systemic blood pressure and increases the cardiac.
   - Output. In contrast, furosemide, during its initial phases, actually worsens the haemodynamics [8].
   - The oral dose of NTG 0.4 mg is approximately equal to 80 mcg/min infusion. Therefore, starting an infusion at 10 mcg/min after giving an oral dose of 0.4 mg leads to dramatic dose reduction. *Therefore, NTG should be commenced at 80–100 mcg/min at least to start with and then escalated rapidly 10–20 mcg every minute up to 400 mcg/min*, guided by the blood pressure [8].
   - High-dose NTG results in dramatic improvement in symptoms over minutes and averts mechanical ventilation. At such high doses, it acts as both arteriolar and a veno-dilator that decreases both afterload and preload, respectively.
   - Now there is robust literature that proves the efficacy of high-dose NTG in the setting of hypertensive LVF [9–16].
   - *Aggressive diuretic monotherapy is unlikely to prevent the need for endotracheal intubation compared with aggressive nitrate monotherapy* [7].

- Loop diuretics, if given alone, can potentially cause hypotension and worsen the renal function by causing diuretic-induced azotaemia and increasing the mortality [10]. Patients in hypertensive LVF have compromised renal blood flow, so diuretics take up to 1–2 h to show the desired effect. So, add diuretics only after nitroglycerine drip is on flow for at least 30 min.
- When studied it was found that almost half of heart failure are not actually in fluid overload [4]; they just have fluid in the wrong compartment of their body. So, *the treatment should be focused on fluid redistribution with optimisation of haemodynamics using nitroglycerine, not eliminating the fluid and dehydrating them with diuretics.* Therefore, administration of diuretics is recommended only in the smallest possible dose in cases of apparent fluid overload.
- Traditionally, morphine has been recommended to ease anxiety in this subgroup of patients, but the current literature questions the use of morphine. It has been associated with worse outcomes with increased rates of ICU admissions and intubation [17].
- Angiotensin-converting enzyme (ACE) inhibitors may also be used in the initial management of acute heart failure syndromes. Overall, they have been less studied in APE and the evidence is not robust. ACE inhibitors decrease the preload and afterload by shutting off the renin-angiotensin-aldosterone system [8].
- *In summary, the treatment of hypertensive LVF should start with NIV and then high-dose NTG and then followed by diuretics only of there is apparent fluid overload.*

2. *Normotensive heart failure*

- This subtype generally presents with an SBP of 90–140 mmHg.
- They may present with a gradual onset of breathlessness and decreased peak effort tolerance. Treatment for them includes diuresis as the primary modality.
- An initial bolus of furosemide 20–40 mg is recommended [18]. If using high doses of diuretics, beware of their potential to cause renal dysfunction.
- In addition to diuresis, also consider using supplemental oxygen and non-invasive ventilation, guided by the general appearance, respiratory rate/effort and SpO_2.

3. *Normotensive heart failure*

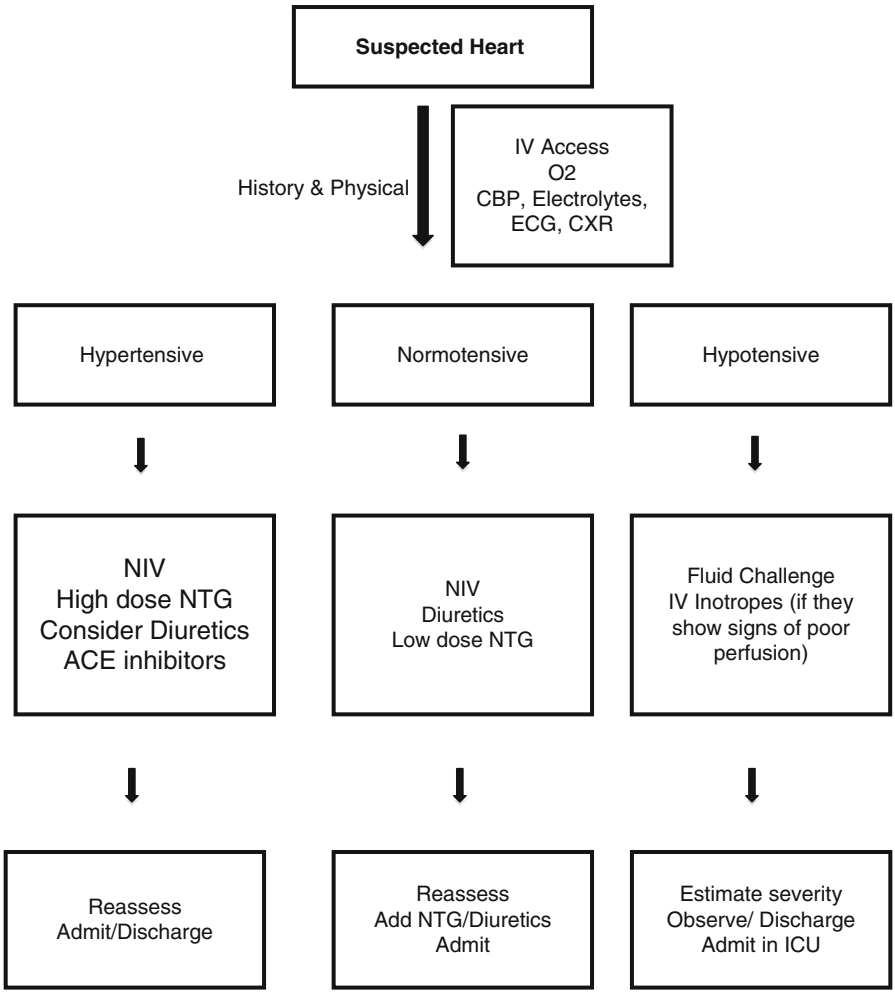
- Only a minority of patients present with hypotension and heart failure; undoubtedly this is the most difficult subtype of patients to manage.
- Look for signs of poor perfusion, do a good history and try to get an estimate about their baseline blood pressure before starting inotropes. If there is an acute decompensation, look for the precipitating event.
- If there are signs of poor perfusion along with hypotension, only then add inotropes temporarily to improve the haemodynamics. Inotropes are necessary evils, associated with an increased mortality, and thus, they are not routinely recommended.
- The choice of inotropes is largely based on the clinician's preference, but in general, dobutamine is the drug of choice for cardiogenic shock [18].
- In cases of florid shock, norepinephrine should be commenced first and supplemented by dobutamine due to initial hypotensive effects of dobutamine via beta 2 receptors. Other potential options are dopamine and milrinone [19].

Other therapies that can be used to buy time or treat heart failure include the use of IABP, LVAD and ECMO. This should be discussed with the patient's cardiologist.

Disposition

Currently, most of the patients who come to the ED with heart failure get admitted. The situation may change in the future with the development of ED-based observation units. There are no well-accepted and validated clinical rules to risk stratify these patients and decide about the disposition. In addition, there is data that suggests high mortality if they are discharged from the ED.

Treatment Algorithm



Patients with the following features are high risk and have a low threshold to admit them.

High-Risk Features
- Poor response
- Inadequate urine output
- Elevated troponin
- Renal dysfunction
- Persistent hypoxia despite using NIV

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