The adult patient with cardiac failure: a clinical nursing perspective

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The global disease burden posed by heart failure (HF) is significant. HF constitutes an often-encountered diagnosis in clinical nursing practice, especially amongst elderly patients. The prognosis of HF remains poor. This article aims to provide the nursing practitioner with a basic overview and refresher of this debilitating condition, its classification and treatment, as well as the associated nursing care implications.

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Introduction

The global disease burden posed by heart failure (HF) is significant. HF constitutes an often-encountered diagnosis in clinical nursing practice, especially amongst ageing patient populations, as well as those with neglected congenital and valvular heart disease. Elderly HF patients often present with the added complexities of multiple comorbid conditions and polypharmacy.

At its simplest level, heart failure (*syn.* cardiac failure, or CF) refers to a complex clinical syndrome, which is caused by impaired ventricular filling or contractility, and which typically manifests itself through varying degrees of dyspnoea, fatigue and oedema. The underlying impairment could either be structural, or functional in nature, and it is always preferable to establish the exact cause in an attempt to optimise the patient's management.[1-3]

Thus, heart failure refers to the inability of the heart to meet the metabolic demands and blood flow requirements of the body. Refer to Figure 1, which also contains the typical signs and symptoms that are associated with HF. Lately, the term 'heart failure' is preferred over the term 'congestive heart failure' (CHF), because not all patients display signs of being oedematous or overloaded with fluid.[1-3]

Based on the left ventricular ejection fraction (EF)—a parameter that has been widely used as a determining factor for patient selection during clinical trials (as well as being a

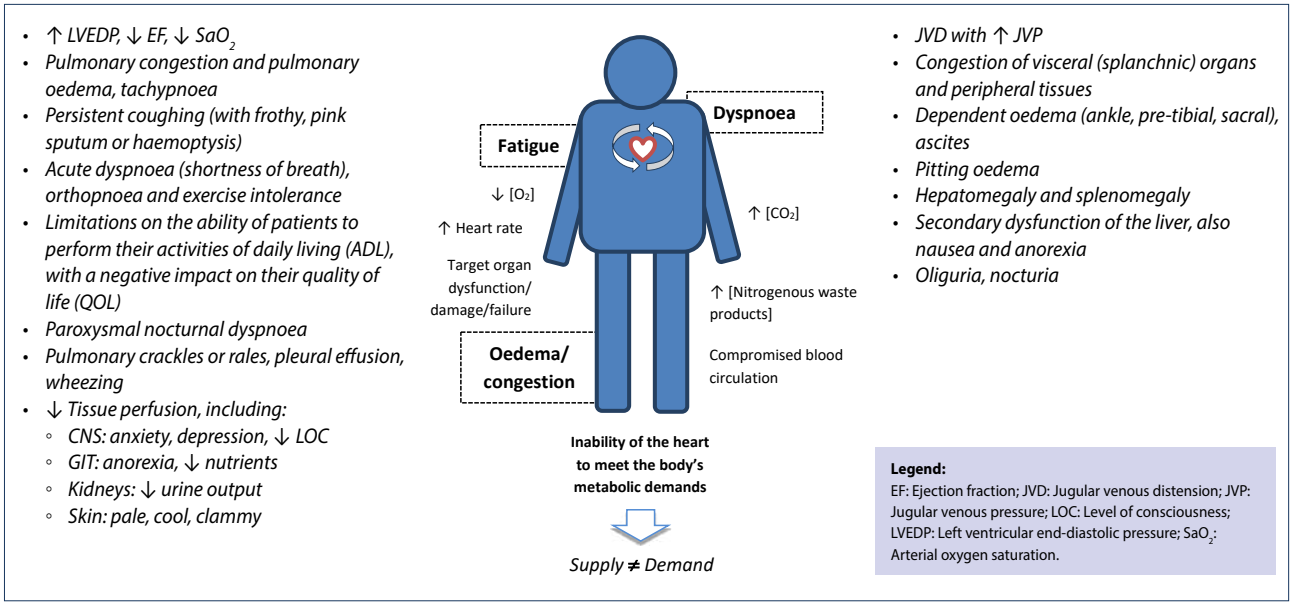

Figure 1: The heart failure 'triad' of dyspnoea, fatigue and oedema, with the associated signs and symptoms (patients do not always present with all three cardinal features in equal measure)[1-3]

significant prognostic aid)—there are two main categories of heart failure, namely:[1]

- HF with preserved ejection fraction (HF*p*EF), and
- HF with reduced ejection fraction (HF*r*EF).

The prognosis of HF remains poor.[3]

The pathophysiology of HF

The most prominent and defining feature of heart failure is the inability of the heart to meet the metabolic demands and blood flow requirements of the body. There are multiple causes and a variety of underlying conditions that may be implicated in the pathophysiological mismatch between metabolic supply and demand, and this often complicates the approach to treatment.[2,3]

The two underlying causes that are most often encountered, are longstanding and poorly controlled hypertension, and coronary artery disease (CAD) with its associated complications. Other primary causes may include valvular heart disease, pericardial disease, uncontrolled cardiac arrhythmias, high ventricular output syndromes (such as chronic anaemia and hyperthyroidism), arteriovenous shunting (as seen in several of the congenital cardiac abnormalities), and the cardiomyopathies.[1-4]

Once the heart starts to fail, and a resultant inability to adequately supply in the blood flow and metabolic demands of the body ensues, a number of physiological, compensatory mechanisms are invariably activated. These so-called **neurohormonal compensatory mechanisms** initially limit the harmful effects of the failing heart on the systemic blood circulation, but eventually these mechanisms will result in a downward spiral of worsening cardiac output (CO) and upregulated neurohormonal responses.[3] Normal cardiac output, and the interplay between stroke volume (SV) and the EF, is explained in Figure 2.

There are three major compensatory mechanisms that become activated when the CO decreases, as well as a number of additional mechanisms that augment these responses. The three major mechanisms are:[1-6]

- **The sympathetic nervous system (SNS):** Noradrenaline, together with dopamine and adrenaline, are called the catecholamines. In the context of cardiac failure, catecholamine stimulation increases the blood pressure via a potent vasopressor effect on peripheral arterioles, increases the heart rate, as well as the force of myocardial contraction, via the cardiac β_1-adrenoceptors, and acts as one of the trigger mechanisms for the activation of the renin-angiotensin-aldosterone system (RAAS).[2]
- **The renin-angiotensin-aldosterone system (RAAS):** In patients with heart failure, it is the reduced renal perfusion that results in the activation of this intricate and potent compensatory mechanism. This results in a significant vasopressor effect, combined with sodium and water retention, and cardiac remodelling.[2,3]
- **The natriuretic peptide system (NPS):** The activation of the NPS actually serves to counteract the pressor effects of the SNS and the RAAS, as well as the accompanying sodium and water retention (via aldosterone release) and cardiac remodelling. The natriuretic peptides are released in response to an increase in ventricular (i.e. myocardial) wall tension.[3,6]

In the case of acute heart failure (AHF), which is the term used to refer to a new-onset or sudden worsening of existing signs and symptoms of heart failure, the characteristic clinical presentation is most often the result of significant congestion and fluid overload. The latter, in turn, is most likely the result of fluid redistribution within the body. AHF frequently requires urgent intervention. Acute congestion may have dire consequences on the normal functioning of

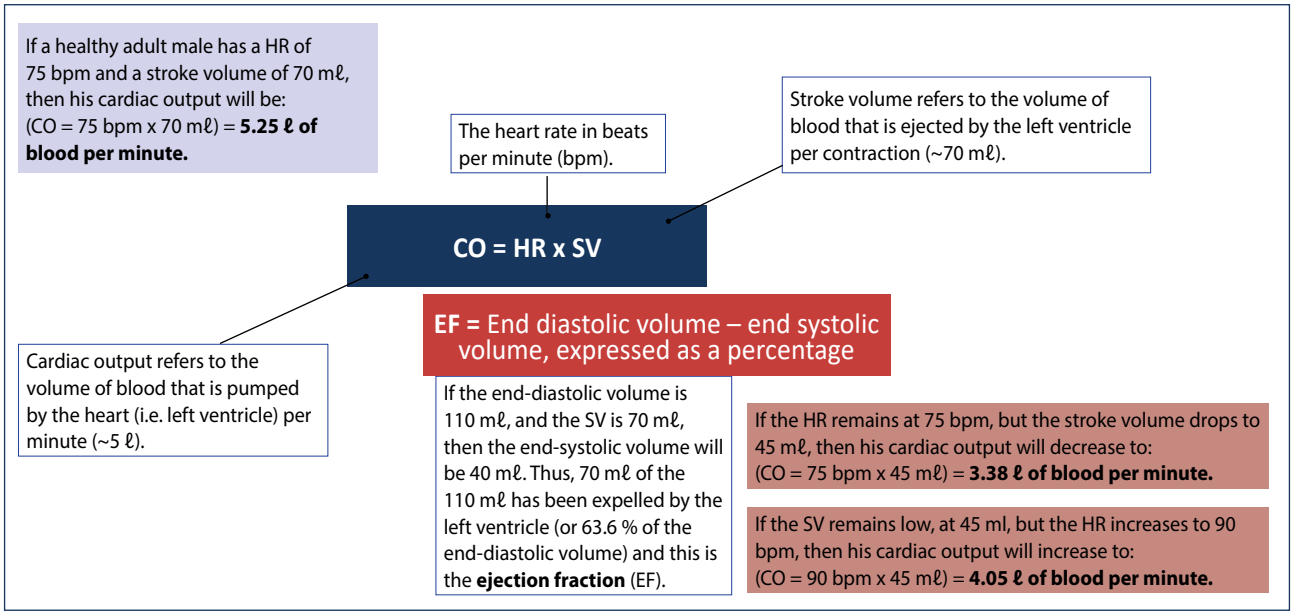

Figure 2: Cardiac output (CO) and the interplay between stroke volume (SV), heart rate (HR) and ejection fraction (EF)

multiple organs and organ systems in the body, including the lungs, intestines, liver, kidneys and heart.[7]

Disease classification and severity

The classification of symptoms is often utilised in an attempt to grade the severity of a patient's condition. The most widely recognised functional classification system is that of the New York Heart Association (NYHA) and is further illustrated in Figure 3.

In addition to the abovementioned NYHA functional classes, which are independent predictors of mortality,[1] there are a number of other classification systems for HF as well. Examples include the following:

- **The American College of Cardiology Foundation/ American Heart Association (ACCF/AHA) stages of HF:** According to this classification system, there are four stages, labelled A to D (see Figure 3).[1]

NYHA:	AACF / AHA:
Class I: • *No limitation of physical activity.* • *Ordinary physical activity does not cause undue dyspnoea, fatigue or palpitations.* The dysfunction is asymptomatic.	**Stage A:** The patient is at high risk, but without any structural heart disease or symptoms of HF.
Class II: • *Slight limitation of physical activity.* • *Comfortable at rest, but ordinary physical activity results in undue dyspnoea, fatigue or palpitations.* The dysfunction is mild.	**Stage B:** Structural heart disease, but without signs or symptoms of HF.
Class III: • **Marked limitation** *of physical activity.* • *Comfortable at rest, but less than ordinary physical activity results in undue dyspnoea, fatigue or palpitations.* The dysfunction is moderate.	**Stage C:** Structural heart disease with prior or current symptoms of HF.
Class IV: • *Unable to carry out any physical activity without discomfort.* • *Symptoms at rest can be present.* • *If any physical activity is undertaken, discomfort is increased.* The dysfunction is severe.	**Stage D:** Refractory HF requiring specialised interventions.

Figure 3: The stratified, functional classification of heart failure (Class I to IV) according to the New York Heart Association (NYHA) and compared to the ACCF/AHA stages of HF (note that these two classification systems do not necessarily align)[1,3]

- **Left and right-sided heart failure:** Irrespective of the ventricle, left or right, which was affected by the primary insult or underlying pathology, it is often inevitable that both ventricles will ultimately fail. This is referred to as biventricular failure.[3]
- **Systolic versus diastolic dysfunction:** The former refers to the existence of a primary dysfunction in the contractility or pumping mechanism of the ventricle, as opposed to a problem that exists with the relaxation or subsequent filling of the cardiac chamber during diastole.[3] It is also common to find some elements of diastolic dysfunction in patients that have been diagnosed by primary systolic dysfunction.[1]
- **HF with preserved ejection (HF*p*EF) fraction *versus* reduced EF (HF*r*EF):** The ejection fraction may be measured non-invasively through the use of transthoracic echocardiography (ultrasound) and represents the percentage of blood being ejected by the left ventricle during a single cardiac contraction. The normal range is 55–70%. Patients with HF*p*EF usually have an ejection fraction of ≥ 50%; conversely, in HF*r*EF, the EF typically falls below the 40% mark (≤ 40%). Those patients that fall within the 41–49% range, constitute an intermediate or borderline group (HF*p*EF, *borderline*) and would be managed more like HF*r*EF patients than like those with a preserved EF. Lastly, there is a subset of HF*p*EF patients that would have previously had HF*r*EF and that now show an improvement or recovery in their ejection fraction to above the 40% level. These latter patients are classified as being HF*p*EF, *improved*.[1,3]

Diagnosing heart failure

Given the fact that HF is a clinical syndrome, certain characteristic signs and symptoms (refer back to Figure 1) allow for a comprehensive medical history, combined with a thorough physical examination of the patient, to be used to arrive at a diagnosis of heart failure based on the clinical judgement of the clinician. This may be augmented by diagnostic tests that are mostly aimed at identifying the underlying cause and quantifying the severity of the condition. B-type natriuretic peptide (BNP), with the 'B' referring to the brain, and N-terminal pro-B-type natriuretic peptide (NT-proBNP) may be used as biomarkers. Non-invasive echocardiography should form part of the clinical assessment of the patient.[3,6]

Pharmacological management of heart failure

The two basic methodological approaches, from a pharmacological standpoint to the management of HF, are to strengthen the force of myocardial contraction, and to decrease the cardiac workload. The latter, in turn, will decrease the myocardial oxygen demand.[2]

Drugs that exert positive inotropic effects on the heart will strengthen the force of myocardial contraction to improve

signs and symptoms of hypoperfusion, but this mechanism forces the myocardium to work harder and therefore increases its oxygen demand. Conversely, inhibition of the abovementioned neurohormonal pathways has the advantage of counteracting the detrimental effects of the compensatory vasoconstriction, increased peripheral resistance, and sodium and water retention seen in patients with chronic heart failure, without increasing myocardial oxygen demand. The positive inotropic agents are used in **acute settings** to maintain adequate vital organ perfusion.[2]

Treatment according to the ACCF/AHA stages A to D

The following is a brief synopsis of the main treatment recommendations per stage (a detailed discussion does not fall within the scope of this article).

Stage A

Patients need to be optimally treated for their hypertension, dyslipidaemia, obesity and diabetes mellitus. Relevant and contemporary treatment guidelines need to be followed in this regard. In terms of hypertension, the following agents are known to be effective in helping to prevent HF in this setting: diuretic-based antihypertensive therapy, ACE-inhibitors, ARBs and suitable β-blockers. Patients need to be counselled in terms of other relevant risk factors, such as alcohol consumption, weight loss and the need to quit smoking. In the case of high blood pressure, both systolic and diastolic hypertension need to be controlled in the long term. The aldosterone antagonists are indicated in patients with refractory hypertension.[1]

Stage B

Generally speaking, all of the abovementioned recommendations for stage A heart failure should be applied to stage B as well. In patients with stage B HF with a reduction in LVEF, the ACE-inhibitors or, if needed, suitable β-blockers, are the agents of choice to improve morbidity and mortality in this setting. Should the ACE-inhibitors not be tolerated, the ARBs may be used as effective substitutes.[1]

Stage C

Patients with symptomatic HF require a sodium-restricted diet. In addition, those suffering from sleep apnoea may benefit from a suitable treatment intervention. Other measures that may warrant more serious consideration and that may have been deemed unnecessary in asymptomatic HF patients, include effective weight management, exercise training or regular physical activity, and the use of a cardiac rehabilitation programme, if indicated.[1]

Wherever appropriate, the pharmacological measures listed for stage A and B patients will also apply to stage C. To reduce morbidity and mortality in these patients, the following options may be utilised to achieve the goal of inhibiting the RAAS:[1,8]

- ACE-inhibitors, or ARBs, or an ARNI (i.e. an angiotensin receptor neprilysin inhibitor), in conjunction with
- an evidence-based β-blocker, and
- in selected patients, the addition of an aldosterone antagonist.

Selected and treatment-resistant patients should be evaluated for the need to add additional drugs to their treatment regimen, such as loop diuretics, digoxin, hydralazine, isosorbide dinitrate, anticoagulants, statins, a suitable second-generation calcium-channel blocker (i.e. amlodipine), etc. It is also recommended that symptomatic HF patients receive an omega-3 polyunsaturated fatty acid (PUFA) supplement, unless otherwise contraindicated.[1]

Another potential treatment option is the I_f channel inhibitor, ivabradine—see further down in the text for more details.

Stage D

These are patients with advanced, refractory or end-stage cardiac failure, with a persistently increasing severity of their condition. The management of these severely ill patients does not fall within the scope of this article. Nevertheless, a few important measures or considerations that these patients will require are as follows:[1]

- Fluid restriction in the range of 1.5 to 2 litres per day.
- Inotropic support to preserve the functioning of their vital organs.
- The use of mechanical circulatory support, such as a ventricular assist device (VAD).
- In the case of very carefully selected and eligible patients, a heart transplantation is the ultimate treatment option for end-stage cardiac failure.

Ivabradine and heart failure

Ivabradine offers a different mechanism of action to many of the more traditional treatment approaches to heart failure. Ivabradine is a selective inhibitor of the I_f ion channel found in the cardiac pacemaker cells of the sinoatrial (SA) node. Ivabradine selectively blocks cardiac pacemaker cell I_f or *'funny'* current and exerts significant inhibition thereof at concentrations that do not affect other cardiac ionic currents.[9] Myocardial contractility and atrioventricular conduction are maintained while heart rate, both at rest and during exercise, is reduced.[10]

Sacubitril and valsartan combination therapy

Sacubitril is the first in a new class of therapeutic agents to be made available on the local market. It is a neprilysin inhibitor and is currently available in combination with the angiotensin receptor blocker (ARB), valsartan. Neprilysin is responsible for the degradation of the natriuretic and other vasoactive peptides. It is presently indicated as second-line treatment option for symptomatic HF, Class II, III or IV on the NYHA classification system, with systolic dysfunction.[1,8,11]

Non-pharmacological interventions

As mentioned in the previous section, a number of non-pharmacological measures form part of the treatment guidelines for heart failure. These include, but are not necessarily limited to:[1]

- Avoiding excessive alcohol consumption and the use of recreational drugs.
- Quitting the smoking habit (and/or other forms of tobacco use).
- Eating a healthy diet (with a firm sodium restriction where needed) and losing excess body weight.
- Sleep disorders are quite frequently observed in patients with heart failure, with more than 60% of adults with chronic HF having been found to suffer from either central or obstructive sleep apnoea. These patients may benefit significantly from the use of a device to provide continuous positive airway pressure, or CPAP, during the night.
- Lastly, a number of invasive cardiac procedures, including coronary revascularisation, as well as mechanical devices, such as the implantable cardioverter-defibrillator (ICD) or the use of cardiac resynchronisation therapy (CRT), may be employed in the management of selected patients with cardiac failure.

Nursing care considerations

A number of aspects need to be considered in terms of the general nursing care of patients with cardiac failure. The following aspects should be included as part of the assessment, planning and execution of carefully tailored care plans:[2,12]

- Monitoring of the patient's vital signs, with special reference to heart rate (for tachycardia and abnormalities of rhythm), blood pressure (with emphasis on hypertension as an underlying cause of HF, but also hypotension as a sign of decompensation and inadequate tissue perfusion).
- In addition to the above, the patient's ability to breathe readily, and without undue discomfort or exertion, should be assessed. This assessment should include the patient's breathing rate and pattern, oxygen saturation, obvious signs of inadequate oxygenation, rapid or laboured breathing, orthopnoea and haemoptysis.
- Monitoring the patient's heart rhythm and electrocardiogram (ECG), serum electrolyte levels and body weight (where acute weight gain is indicative of fluid retention).
- Monitoring the patient's renal function and fluid balance (intake and output), noting a decrease in urine output or concentrated urine, and carefully managing the patient's total fluid intake (including intravenous fluids, nutritional supplements and oral fluid intake). Patients with congestion and fluid overload will be placed on fluid restriction, which will require careful and judicious monitoring on an ongoing basis.
- Monitoring of the patient's mood and level of consciousness, nothing signs of irritability, anxiousness, lethargy, confusion, disorientation, depression and loss of consciousness. These may be signs of both decreased cerebral perfusion, and the effects of prolonged and debilitating illness on a person's mental wellbeing.

Suitable interventions that need to be employed in the nursing unit may include the following:[2,12]

- Correct positioning of the patient in bed, trying to find a good compromise between optimum comfort and ease of breathing. Typically, a high Fowler's position would provide optimal expansion of the lungs in bedridden patients.
- Provisioning of supplemental oxygen therapy.
- Limiting physical exertion.
- Administering suitably prescribed pharmacological agents.
- Providing appropriate health education to patients and their caregivers. Typically, three pillars form the basis of the medical treatment modality, namely adequate rest, necessary lifestyle modifications, and suitable pharmacotherapy.

Conclusion

This article attempted to provide a basic refresher of heart failure, including its classification, diagnosis, treatment, and nursing care considerations. According to the latest international guidelines, the most important differentiation in the setting of HF, is to determine which patients have HF*p*EF as opposed to HF*r*EF. The basis of HF treatment starts with the correct classification or grading of the severity of the patient's condition, the identification and management of modifiable risk factors, and then, the initiation of appropriate measures that are aimed at counteracting the detrimental effects of the neurohormonal response towards the mismatch in metabolic supply and demand, which is brought about by a failing heart.

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