

Part 2: Heart failure

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James Edwards does a two-part chat with Dr. Sean Lal. Sean's clinical and research interests are in heart failure.

In this second part of the podcast, we look at:

- how best to investigate heart failure,
- the difference between systolic and diastolic heart failure,
- what are the common precipitants,
- what type of drugs are used, and
- when to escalate care.

In [Part 1](#) we go through what heart failure is and its common causes.

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Systolic and diastolic heart failure

Heart failure (HF) is a clinical condition. It's a result of an impaired ability of the heart to provide output to perfuse organs sufficiently. This is due to abnormalities of heart structure and function. There are two main mechanisms by which heart failure can occur.

Systolic heart failure results in impaired contractile function. Thereby, it reduces the output to the body.

Diastolic dysfunction results from abnormal cardiac relaxation. And this happens where a stiff ventricle is not able to fill adequately. These two pathological processes often coexist together.

About Dr. Sean Lal

Sean Lal is a clinical academic cardiologist at Royal Prince Alfred Hospital and the [University of Sydney](#), NSW, Australia. He undertook his training at:

- the University of Sydney,
- Royal Prince Alfred Hospital, and
- Harvard Medical School.

Part 2: Heart failure

With Dr Sean Lal, Clinical Academic Cardiologist at the University of Sydney and Royal Prince Alfred Hospital, New South Wales, Australia

Introduction

Heart failure (HF) is a clinical condition resulting from an impaired ability of the heart to provide output to perfuse organs sufficiently due to abnormalities of heart structure and function. There are two main mechanisms by which heart failure can occur. Systolic heart failure results in impaired contractile function thereby reducing output to the body. Diastolic dysfunction, on the other hand, results from abnormal cardiac relaxation where a stiff ventricle is not able to fill adequately. These two pathological processes often coexist together. In this second part of the podcast, we will look at how best to investigate heart failure, what are the common precipitants, what type of drugs are used and when to escalate care.



1. Is it possible to distinguish between HFpEF and HFrEF clinically?

- Heart failure is diagnosed clinically and then further stratified into two broad categories based on the left ventricular ejection fraction (LVEF)
 - $EF = (EDV - ESV) / EDV$
 - EF - Ejection fraction
 - EDV - End diastolic volume
 - ESV - End systolic volume
 - Heart failure with reduced ejection fraction (HFrEF)
 - Symptoms ± signs of heart failure and
 - LVEF < 50%
 - Pathophysiology
 - Numerous underlying pathologies cause reduced systolic function which causes reduced cardiac output (CO)
 - Reduced CO causes reduced end-organ perfusion, activates RAAS (Renin-angiotensin-aldosterone system) and inflammatory systems and promotes cardiac remodelling all resulting in poorer cardiac function
 - Heart failure with preserved ejection fraction (HFpEF)
 - Symptoms ± signs of HF and
 - LVEF > 50% and
 - Objective evidence of:
 - Structural heart disease - LV hypertrophy, LA enlargement
 - Diastolic dysfunction with high filling pressure
 - Pathophysiology
 - Less well defined
 - Typically seen in co-morbid older patients
 - Prevalent in older female patients with a history of hypertension, obesity, diabetes and atrial fibrillation
- Clinically these two syndromes are difficult to distinguish between
- Dyspnoea and fatigue are two key features that are common between both types
 - Breathlessness in diastolic heart failure occurs when unable to fill efficiently - can't do as LV is stiff
 - This often occurs in tachycardia as there is not enough time for the ventricle to fill

2. What investigations can be done on the ward?

- Bedside Investigations
 - ABG/ VBG - If hypoxia is present + needing oxygenation support
 - ECG - To determine if underlying ischaemia and/ or cardiac rhythm abnormalities and/or LV hypertrophy is present
- Bloods
 - FBE -? decrease in Hb contributing to shortness of breath
 - UEC -? Renal impairment causing low output HF
 - LFT -? Liver congestion in the context of RV HF
 - TFT - especially in the context of tachycardia
 - Troponin - If any suggestion of ischaemic event
 - High sensitivity is a very useful tool in the emergency department to rule out an ischaemic event
 - It is noted that the high-sensitivity troponin may also be elevated in heart failure
 - Look at the trend of the troponin rise - how quickly is it increasing, and does it continue to rise?
 - What is the peak value?
 - E.g. If the troponin is initially 20 then jumps to 40 in an elderly individual w/ stiff ventricle and a tachycardia this may be consistent with a troponin leak
 - If the troponin is high initially and continues to rise or is initially normal and then rises significantly may be consistent w/ an additional acute coronary syndrome

- At this stage it is unclear as to why some events result only in a small release of troponin
 - Troponin lies in the cytoplasm of cardiac myocytes
 - Mild irritation of cardiac myocytes may only cause a small leak whereas myocyte death causes a larger release of troponin
- BNP – Reflective of stretch of the atria and is affected by age, obesity and pregnancy
 - In the acute setting:
 - Ideally not utilised where clinical signs + radiographic evidence are sufficient to make a clinical diagnosis of heart failure
 - Is useful to determine if undifferentiated dyspnoea is due to heart failure
 - In the chronic setting – to monitor/ track progress of heart failure
- Imaging
 - Chest X-Ray – to detect signs of pulmonary congestion and to identify if there are other causes for a patient’s symptoms
 - Look for any new changes on the chest XRAY – compare with previous films
 - Upper lobe diversion / perihilar congestion
 - Frank new pleural effusions
 - Alveolar infiltrates
 - Cardiac silhouette enlarged
 - NB: Diastolic failure patient may have chronic signs of the above

3. When should an ECHO be performed? What are the indications for an urgent ECHO vs a non-urgent outpatient ECHO?

- Urgent inpatient bedside ECHO is required when:
 - Patient is haemodynamically unstable – tachycardia and hypotension
 - Low output state
 - Reduced urine output w/ increasing creatinine
 - Signs of cardiac tamponade
- Non-urgent inpatient vs outpatient ECHO when
 - New diagnosis but haemodynamically stable
 - Exacerbation of underlying CCF
 - Call cardiology to gain advice re urgent management however bedside echocardiogram is not required

4. Management of heart failure

- ACE inhibitor/ ARB
 - Recommended in HFrEF with LVEF < 40% unless not tolerated
- Beta Blockers (bisoprolol, carvedilol, metoprolol XR, nebivolol are approved for use in heart failure)
 - Recommended in HFrEF with LVEF < 40% unless not tolerated. Should only be commenced when there is minimal/absent congest to prevent further decompensation
 - In HFpEF where there is diastolic dysfunction and an associated tachycardia/ hypertension may actually need beta-blockers such as metoprolol early to slow HR and allow filling time
- Loop Diuretic
 - Recommended for use in patients with heart failure and symptoms or signs of congestion
 - Frusemide also enables pulmonary venodilation
 - Measure urine output to gauge response to diuretics
- Mineralocorticoid receptor antagonist (MRA)
 - Recommended in HFrEF with LVEF < 40% unless not tolerated

- Angiotensin receptor neprilysin inhibitor (ARNI)
- Ivabradine
 - Recommended in HFrEF with LVEF < 35% + sinus rate > 70 beats per minute despite max use of ACEI and beta blocker
- GTN infusion may be used as venodilation to reduce preload in HF exacerbation

5. What are some of the common precipitants of heart failure?

- Heart failure can be caused by myocyte damage/ loss, conditions which cause abnormal loading and arrhythmias. Below are some examples
- Myocyte damage/loss
 - Ischaemia - infarction, ischaemia
 - Inflammation - infection, immune
 - Toxic damage - alcohol, drugs (anthracyclines, amphetamines, anabolic steroids, clozapine)
 - Infiltration - malignancy, amyloids, sarcoid, haemochromatosis, iron overload, glycogen storage disease
 - Metabolic abnormalities - thyroid, growth hormone, cortisol, diabetes mellitus, pheochromocytoma
 - Nutritional abnormalities
 - Genetic abnormalities
 - Pregnancy
- Abnormal loading conditions
 - Hypertension - uncontrolled hypertension leads to increased afterload
 - Valvular dysfunction
 - Pericardial pathology
 - High output states - anaemia, sepsis thyrotoxicosis
 - Volume overload - renal failure, iatrogenic fluid overload
- Arrhythmias
 - Tachyarrhythmias - AF, ventricular arrhythmias
 - Bradyarrhythmia - Sinus node or AV node dysfunction

6. When should a junior doctor escalate care in patients with heart failure?

- Abnormalities in ECG in someone with heart failure
- Significant increase in O2 requirements
- Hypertensive requiring GTN infusion - needs cardiology input and monitored bed
- Tachycardia either as a cause or result of heart failure
- When CPAP required for management of pulmonary congestion, escalate to cardiology/ respiratory/ ICU
- When inotropic support is required to maintain perfusion in low output states and cardiogenic shock

Take home messages

- Heart failure is an extremely common presentations in all areas of the hospital
- Heart failure is not always systolic - think of risk factors for diastolic heart failure - diabetes, HTN (hypertension), sleep apnoea and consider this as a potential cause of undifferentiated dyspnoea in these individuals
- Always rule out ischaemic/ coronary artery events

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