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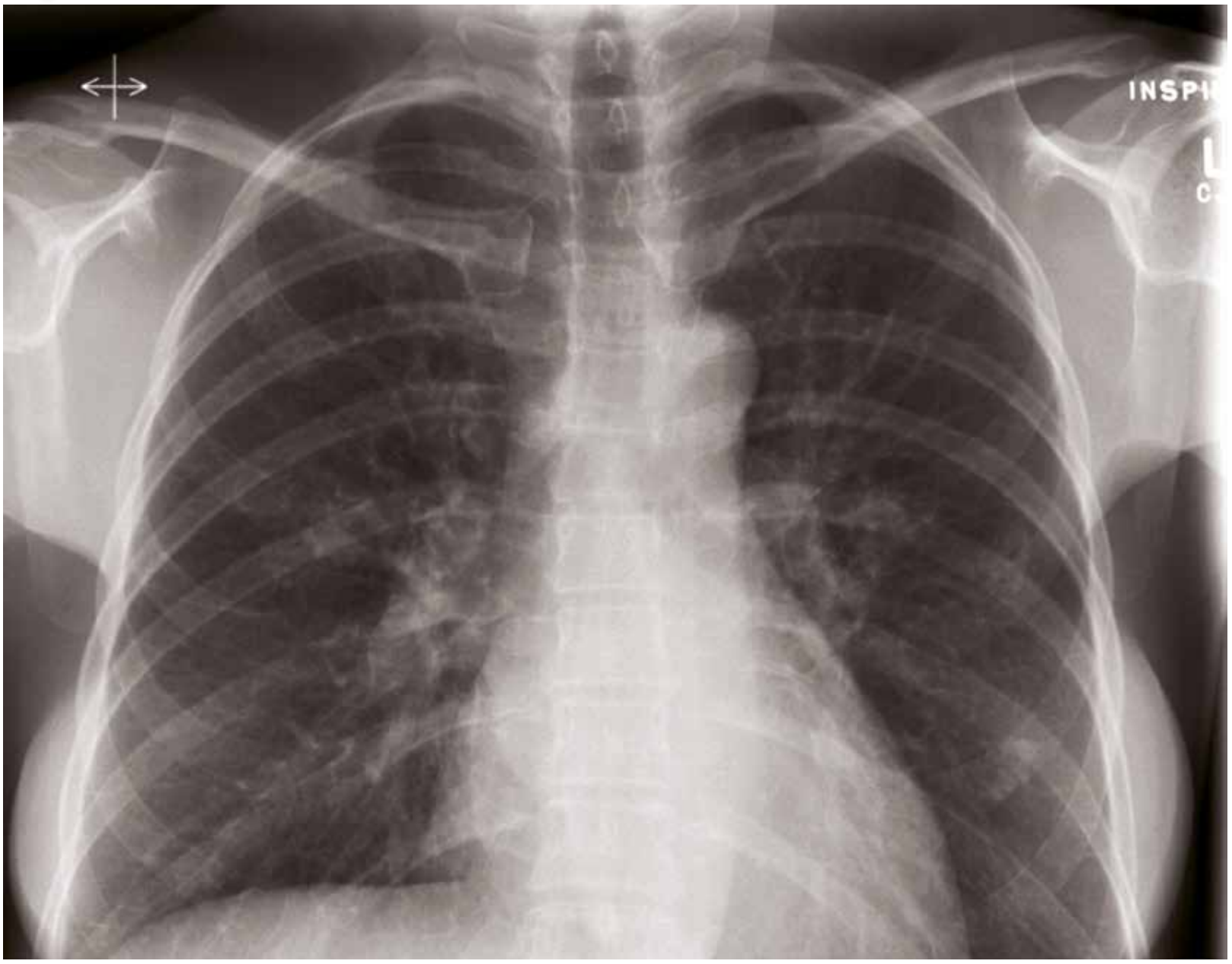

HEART FAILURE

Introduction

HEART failure is the inability of the heart to pump blood adequately around the body, leading to reduced perfusion and congestion. It can be secondary to reduced contraction (systolic heart failure), reduced relaxation (restrictive or diastolic heart failure), high output states or compression (constriction/tamponade).

Heart failure can be due to dysfunction of the left ventricle, right ventricle or both (see figure 1).

More than 20 million people worldwide have heart failure. It affects 280,000 Australians or 1.3% of the population, with 30,000 new cases and 49,000 hospital admissions per year.

One-year heart failure readmission rates are high at 32%. This results in a burden of 200,000 bed days in NSW alone.[1] The annual cost of heart failure readmission in the US is over $35 billion.[2]

This How to Treat summarises the different types of heart failure, aetiology, investigations and current treatment.

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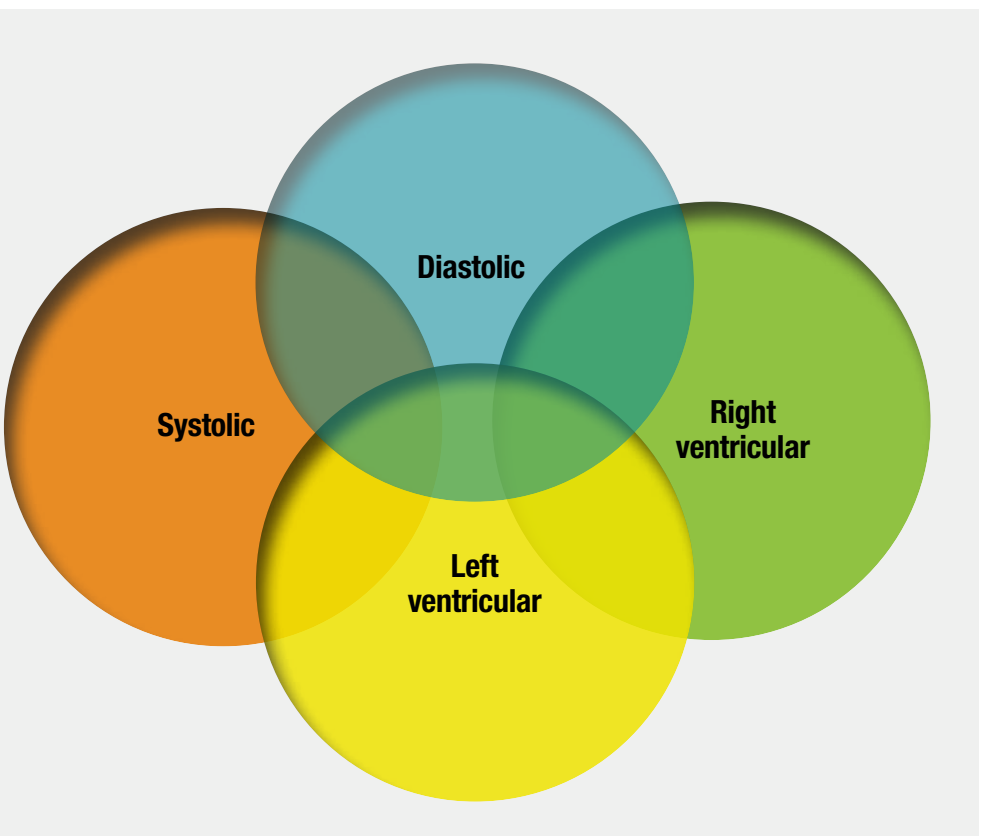

Figure 1: Types of heart failure.

INSIDE

THE AUTHOR

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Symptoms and signs

SYMPTOMS of heart failure are proportional to the degree of congestion and reduced perfusion caused by the lack of forward blood flow through the heart. Symptoms often vary depending on whether the left or right ventricle is affected (see table 1).

Left ventricular systolic failure results in congestion of the lungs and alveolar oedema, producing dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea (PND). Dyspnoea is categorised by the New York Heart Association (NYHA) classification (see table 2).

Low cardiac output causes reduced tissue perfusion and symptoms of fatigue, dizziness, confusion and cold extremities. When assessing patients with heart failure, it is useful to ask yourself 'Are they warm or cold, wet or dry?' (see figure 2). This helps assess severity of illness and guides treatment.

Right ventricular failure results in venous congestion and may be associated with tricuspid regurgitation (TR). Patients often complain of dyspnoea due to pleural effusions, headaches worse on lying flat (from TR) and symptoms of hepatic congestion such as bloating, early satiety and right upper quadrant pain. Cardiac cachexia is more often associated with right ventricular failure due to hepatic congestion and anorexia. This often catches physicians unaware because of the substitution of muscle and fat by retained fluid and thus there is no change in body weight. Common signs to look for are listed in Table 1.

The physiology and the anatomy of the right ventricle are completely different from the left ventricle. The symptoms of right and left ventricular failure are also different. Right and left ventricular failure should be treated differently. However, all too often their treatment is the same, with the same medication, despite the lack of evidence for this. This incorrect treatment can often make patients feel worse.

Diastolic or restrictive heart failure results in congestion and often presents with dyspnoea or pedal oedema.

Constrictive cardiomyopathy results in signs of right ventricular failure. However, the elevated JVP may increase with inspiration (Kussmaul's sign) and show a rapid Y descent. A 'pericardial knock' may be heard after the second heart sound.

Table 1. Symptoms and signs of heart failure

Symptoms		Signs
Excess fluid	Reduced output	Tachycardia
Dyspnoea	Fatigue	Atrial fibrillation
Paroxysmal nocturnal dyspnoea	Weakness	Pulsus alternans
Orthopnoea	Dizziness	Elevated JVP
Hepatic congestion	Dyspnoea	Hypo- or hypertension
Anorexia	Confusion	Displaced apex
Early satiety	Cold extremities	RV Heave
Right upper quadrant pain		3rd/4th heart sound
Bloating		Murmurs – mitral regurgitation/ tricuspid regurgitation
Ascites		Crepitations/effusions
Leg swelling		Hepatomegaly/hepatojugular reflux
Headaches		Ascites
		Pedal/sacral oedema
		Reduced capillary return

Table 2. New York Heart Association (NYHA) dyspnoea classification

Class	Breathless symptoms
I	Asymptomatic
II	Slight limitation
III A	Ordinary activity
III B	Minimal activity
IV	At rest

Source: American Heart Association, see http://bit.ly/1WE8OaC

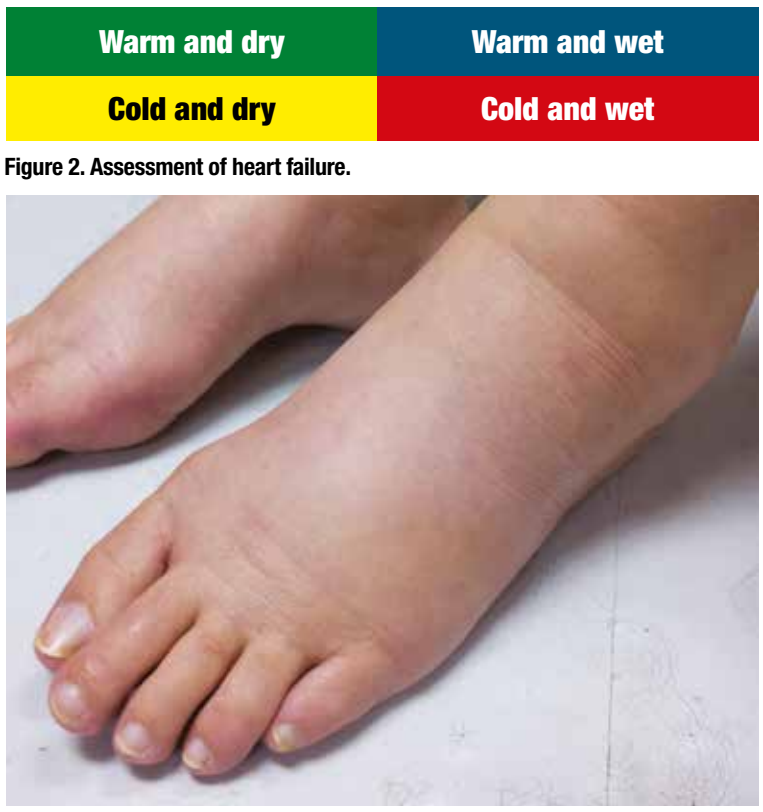


Figure 2. Assessment of heart failure.

Aetiology

THERE are many different aetiologies of systolic heart failure (see figure 3) and it often coexists with multiple comorbidities. When it is due to cardiac muscle dysfunction, it is called cardiomyopathy.

In the Western world, the most common cause is ischaemic heart disease, which is responsible for more than 50% of cases. Other common causes are hypertension, infective (predominantly viral), valvular, tachyarrhythmia, toxic/drug related, endocrine (thyroid and diabetes), hereditary, infiltrative and connective tissue diseases.

When no other cause can be found it is termed 'idiopathic'. However, over time, it is subsequently found to be hereditary in 30% of idiopathic cases.[4]

The causes of left and right ventricular failure are similar, but there are some conditions predominantly affecting the right ventricle. These include chronic thromboembolic disease, arrhythmogenic right ventricular dysplasia, pulmonary hypertension, left to right cardiac shunts and congenital heart disease.

Restrictive cardiomyopathies are often genetic (hypertrophic cardiomyopathy) or due to infiltration disorders, such as cardiac amyloid, haemochromatosis or Fabry's disease.

Diastolic heart failure can be secondary to stiff scar tissue as a result of myocardial infarction, but is more commonly a disease of the elderly, particularly hypertensive, diabetic, overweight females.

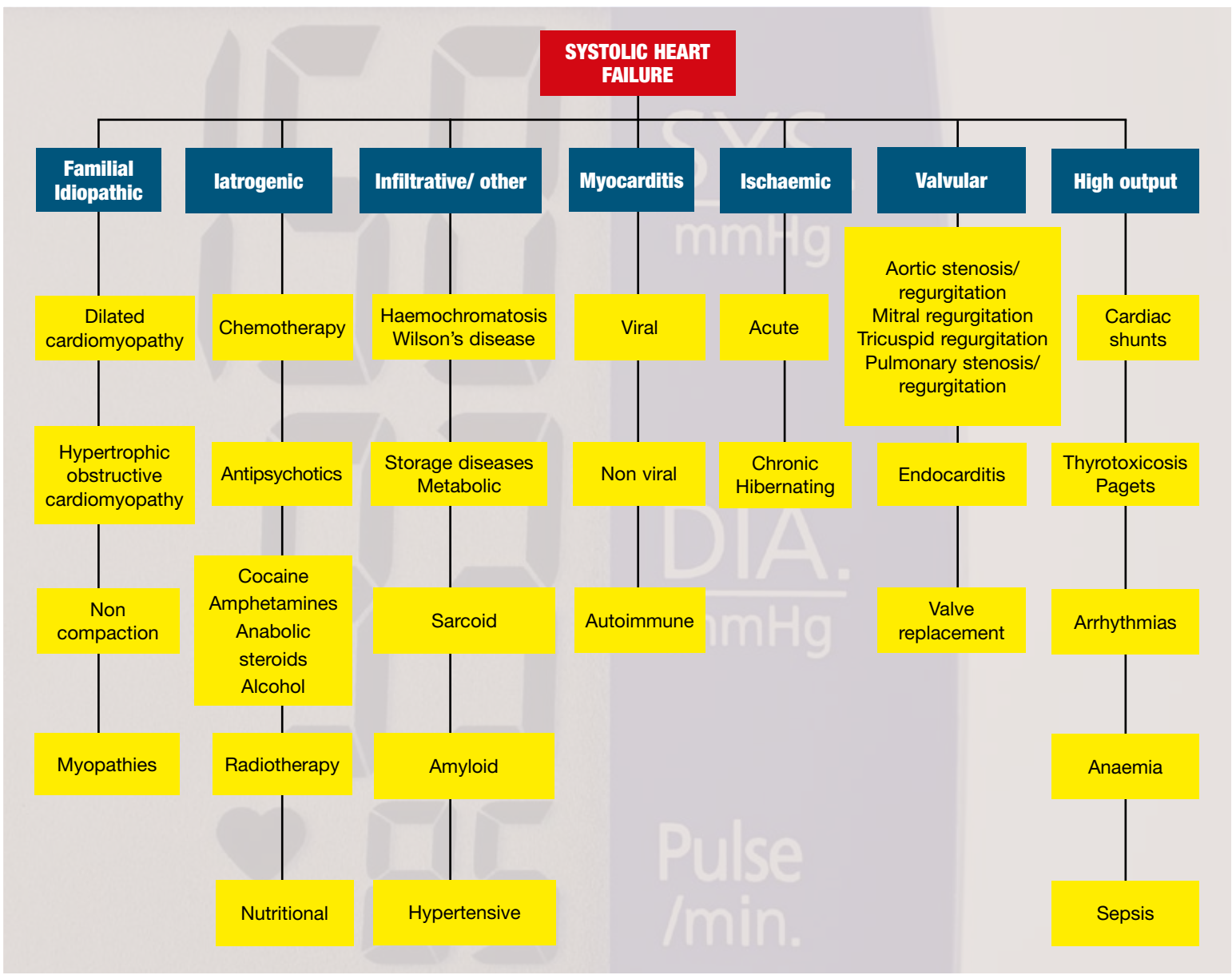


Figure 3. Aetiology of systolic heart failure.

Diagnosis

DIAGNOSIS starts with a thorough history, particularly assessing risk of coronary artery disease, arrhythmias, genetics, autoimmune disease, infections, endocrine or toxic drug exposure. Examination helps in establishing whether it is predominantly left sided, right sided or biventricular, as well as other possible aetiologies, such as valvular, hypertensive or pulmonary hypertension.

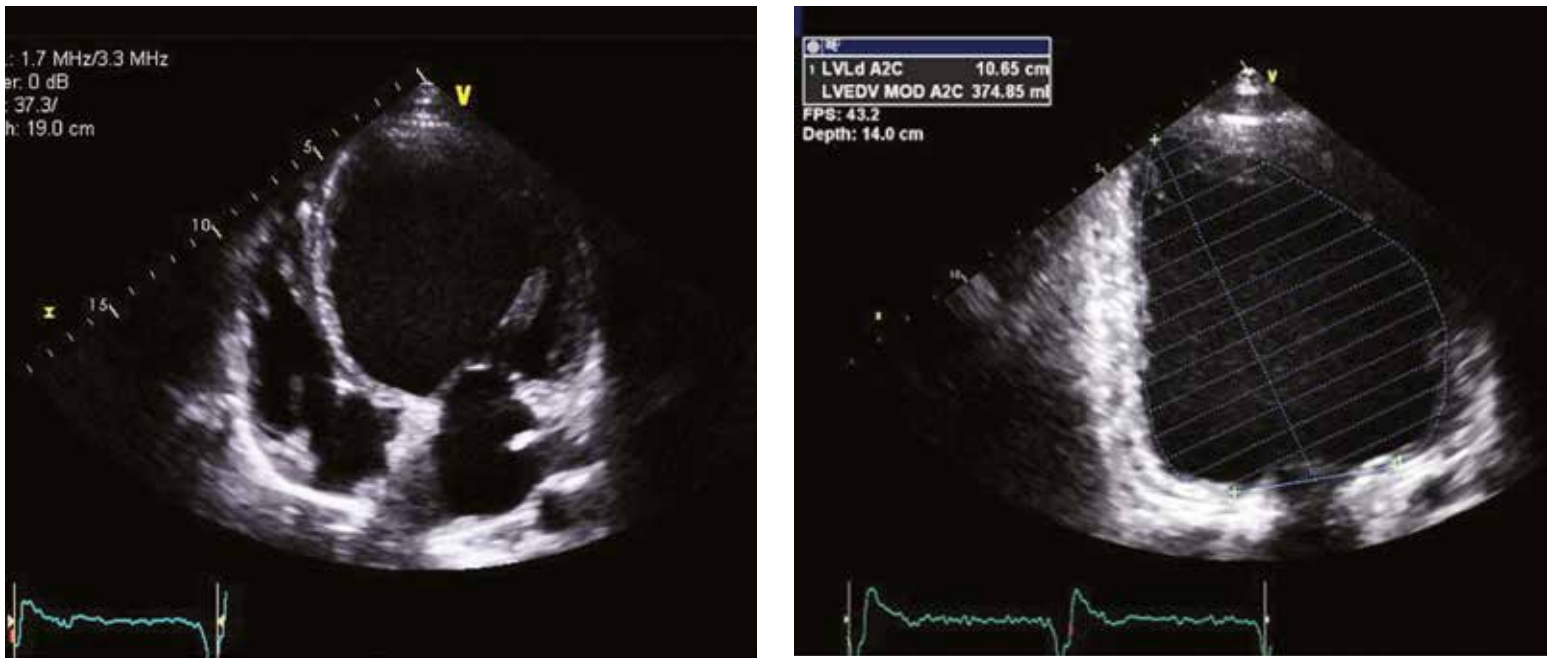
Investigations will depend on the clinical scenario. Table 3 lists the possible investigations and their main indications. A good starting point is renal and hepatic biochemistry, full blood count, glucose, thyroid function, B12, folate and iron studies.

Brain natriuretic peptide (BNP or N-Terminal Pro BNP) is a good screening test, but is only covered by Medicare for investigation of heart failure in the ED (it costs around $70). It is useful as a rule-out test for heart failure as it has a high negative predictive value. However, caution must be used interpreting a positive test or a negative test in obese patients (see table 4).

An ECG, chest X-ray and transthoracic echocardiogram are easily accessible tests that form the first line of investigation. Up to 20% of patients have poor echocardiographic windows and may require the use of trans-oesophageal echocardiography, contrast agents or other imaging techniques to elucidate the severity and cause of heart failure.

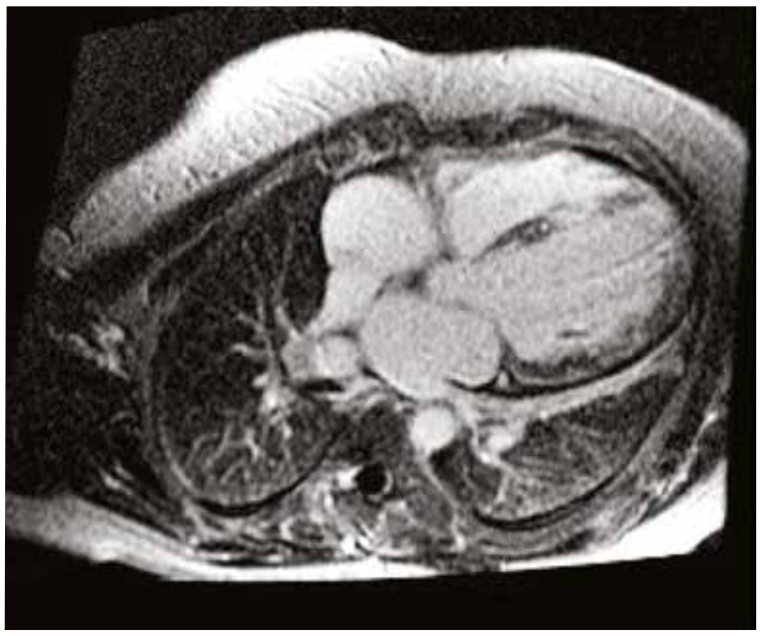
Coronary angiography — either by CT or gold standard invasive technique — helps to assess for aberrant coronary anatomy or cardiac ischaemia. A left heart study assesses left ventricular function, valvular abnormalities and diastolic dysfunction. In patients with heart failure, a right heart catheter is often performed to assess fluid status, cardiac function, pulmonary pressures and presence of significant left to right shunts.

A fluid challenge may be given to test for diastolic heart failure. If constrictive heart failure is suspected, then a simultaneous left and right heart catheter is indicated.



Transthoracic two-dimensional echocardiogram in apical four chamber view, showing dilated left ventricle.

CT coronary angiogram showing normal coronary arteries.



Cardiac MRI showing myocarditis of ventricular septum and left ventricular lateral wall.

A cardiac biopsy may be performed in cases of suspected myocardial infiltration (sarcoid, amyloid, and haemochromatosis), storage disorders (Fabry's disease), giant cell myocarditis or eosinophilic myocarditis.[4-8]

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Table 3. Diagnosis of heart failure

Investigation/assessment	Details and conditions to consider and exclude
History and examination	
Bloods	BNP, ANA, ENA, dsDNA, ANCA, B12, folate, iron, Hep B/C, HIV, EBV, CMV, Toxo, TSH, ACE, 1,25 - Vit D, carnitine, alpha galactosidase, EPG, serum light chains
ECG	Rhythm, LVH, ischaemia, BBB, RVH, low voltage (amyloid)
Chest X-ray	Cardiomegaly, pulmonary oedema, LA size, pericardial calcification
BNP	Systolic/ diastolic/ restrictive heart failure
Echo +/- contrast	LV/RV function, valves, congenital defects, thrombus, pulmonary pressures
Cardiac MRI	Ischaemia, infiltration, congenital abnormalities
Stress test/ Imaging Exercise stress test Myocardial perfusion scan Dobutamine stress echocardiogram	Ischaemia, valvular
Holter/event monitor	Arrhythmias / heart rate
Cardiac CT/CT coronary angiogram	Structural, coronary anatomy, coronary atherosclerosis
Invasive coronary angiogram	Ischaemia, aberrant coronary origins, left ventricular-end diastolic pressure
Right heart catheter	Fluid status, pulmonary hypertension, cardiac output, shunts
Cardiac biopsy	Autoimmune, eosinophillic, sarcoid, amyloid, storage disorders
Genetic screening	Hypertrophic, dilated
PET	Myocardial viability, lymphoma, sarcoid

Table 4. BNP levels in conditions other than left ventricular heart failure

Increased	Normal/decreased
Pulmonary disease	Acute pulmonary oedema
Right ventricular strain	Obesity
Oedema/low protein	Drugs
Septicaemia	Heart failure proximal to left ventricle (eg, mitral stenosis) Pulmonary veno-occlusive disease
Tachyarrhythmia	
Myocardial ischaemia	
Renal failure	
Age	
Diastolic heart failure	

Treatment

TREATMENT depends on the cause, and whether the heart failure is acute or chronic, systolic or diastolic and which ventricle is affected; often all conditions co-exist.[4-8]

Systolic left ventricular failure

Figure 4 shows an algorithm for the treatment of systolic left ventricular failure. As mentioned, when assessing patients with heart failure, it is useful to ask yourself 'are they warm or cold, wet or dry?'.

Patients who are warm and dry are the most desirable, and may allow for up-titration of heart failure therapy or reduction of diuretics. Patient who are wet require diuresis, and patients who are cold either require reduction of vasodilator therapy or inotropes. Cold and wet patients are often the sickest, frequently requiring hospital admission.

Lifestyle modification and withdrawal of exacerbating factors or treatment of the underlying causes are essential. Patients should be advised to weigh themselves daily, in the morning after micturition, have a low salt diet (2g/day), and monitor their fluid input (2L/day).

Revascularisation with coronary stents or surgery and valve replacements often have a marked beneficial effect.

Diuretics

Diuretics (see table 5) are used for symptom relief in patients who are congested or wet, but they have not (except aldosterone antagonists) been shown to increase survival.

Options are loop diuretics (frusemide, bumetanide, ethacrynic acid), thiazide diuretics (hydrochlorothiazide, bendrofluazide, chlorthalidone, metolazone), potassium sparing (amiloride, triamterene), aldosterone antagonists (spironolactone, eplerenone), carbonic anhydrase inhibitors (acetazolamide) and osmotic diuretics (urea, mannitol).

Rarely, demeclocycline, lithium or tolavptan are used to induce nephrogenic diabetes insipidus with resultant free water loss.

Angiotensin converting enzyme inhibitors

Angiotensin converting enzyme inhibitors (ACEI) have been shown to improve survival by 30%. These should be started at a low dose and up-titrated frequently (2-4 weekly) to maximum tolerated dose as per dosing guidelines.

Angiotensin 2 receptor blockers (A2RB) should only be used instead of ACEI if patients are allergic or intolerant. Currently only candesartan, valsartan and losartan have evidence in systolic left ventricular failure.

The evidence for adding an A2RB to ACEI is currently conflicting and adding candesartan should only be used when other options are exhausted. Hydralazine (300mg) and oral nitrates (180mg) can be substituted for patients intolerant of ACEI/A2RB, but they are not as beneficial in reducing mortality (see table 6).

Table 5. Diuretics

Loop	Thiazides (distal convoluted tubule)	K sparing DCT/collecting tubules	
Frusemide 40mg od — 500mg bd	Hydrochlorothiazide 12.5mg-50mg od	Amiloride 5-15mg daily	
Bumetanide 1mg od — 5mg bd	Indapamide 2.5mg od	Triamterene 50-200mg daily	
Ethacrynic acid 50mg od —200mg bd	Chlorthalidone 12.5mg-50mg od		
Aldosterone antagonists	**Proximal tubule**	**Osmotic**	**Vasopressin antagonist/ nephrogenic diabetes insipidus**
Spironolactone 12.5mg -50mg/ day	Acetazolamide 125-250mg bd	Mannitol	Tolvaptan
Eplerenone 12.5-50mg/ day		Urea	Demeclocycline
			Lithium

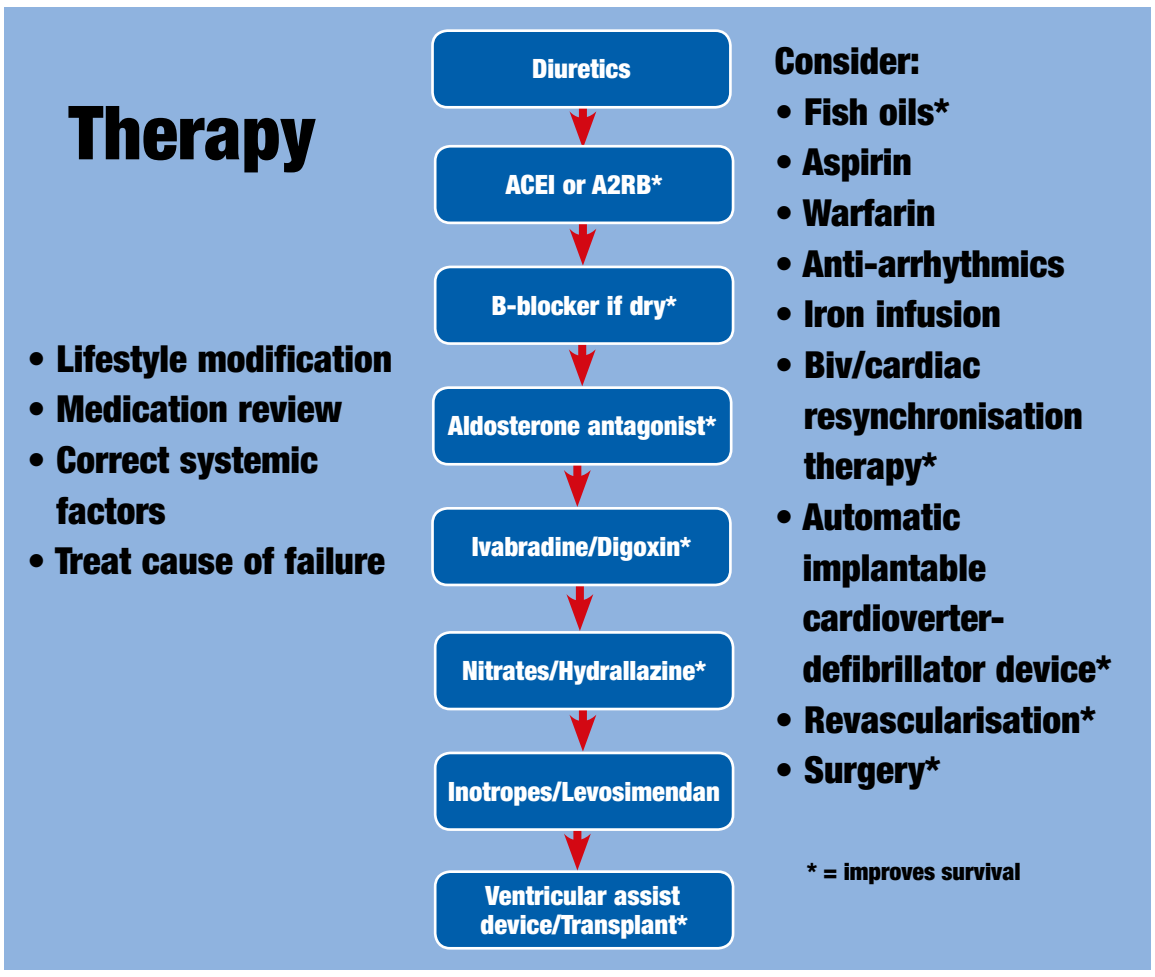

Figure 4. Treatment of systolic left ventricular failure

Table 6. ACEIs

ACEI	Starting dose	Target dose
Captopril	6.25mg tds	50mg tds
Enalapril	2.5mg bd	20mg bd
Fosinopril	10mg od	40mg od
Lisinopril	2.5mg od	20-35mg daily
Perindopril	2.5mg od	10mg od
Ramipril	2.5mg od	10mg od

Table 7. Beta-blockers

Beta-blocker	Start dose	Target dose
Carvedilol	3.125mg bd	25-50mg bd
Metoprolol succinate	11.875mg od	190mg od
Bisoprolol	1.25mg od	10mg od
Nebivolol	1.25mg od	10mg od

Beta-blockers

There are currently four beta-blockers available in Australia for the treatment of heart failure. They have been shown to improve survival by a further 30% when added to ACEI.[9, 10] Beta-blockers should only be started or up-titrated when patients are euvolaemic. Patients can often feel unwell for 2-4 weeks after up-titration. More cardioselective beta-blockers (bisoprolol, nebivolol) should be used in patients with hypotension, asthma, fatigue and impotence.

Beta-blockers can be started before an ACEI or A2RB for control of arrhythmias, such as atrial fibrillation or ventricular tachycardia (see table 7).

The only treatments currently of potential benefit in diastolic heart failure are diuretics and spironolactone.

Aldosterone antagonists

Aldosterone antagonists (spironolactone, eplerenone) also reduce mortality when added to ACEI and beta-blocker therapy. Spironolactone is currently indicated for New York Heart Association (NYHA) class IV, but a recent trial of eplerenone in NYHA class II-III was stopped prematurely due to survival benefit.

Other treatments

Ivabradine (see New treatments section) is a sinus node slowing agent. It is indicated as an add-on therapy for patients with systolic left ventricle failure in sinus rhythm and with a resting heart rate higher than 77/min, who have a contraindication to, or are on maximum tolerated dose of, beta-blockers.

Fish oils — 3000mg daily (850mg/day DHA/EPA) — have been shown to reduce mortality by 2% over four years. There is no evidence for krill or calamari oil.

Routine use of aspirin or warfarin has not been shown to improve outcomes, but these may be used if there are other indications for their use. Warfarin can be considered in severely dilated and impaired systolic left ventricle failure or the presence of either spontaneous echo contrast or an LV thrombus within the left ventricle.

Both cardiac resynchronisation therapy (CRT) and prophylactic implantable cardioverter-defibrillator (ICD) devices have been shown to reduce mortality in systolic left ventricle failure. Current indications for an ICD are LVEF <35%, NYHA II-III and life expectancy greater than two years. CRT is indicated for LVEF <35%, NYHA II-IV with QRS > 120ms, or patients with NYHA class II, LVEF <30% and LBBB (QRS >150ms).[6]

Iron carboxymaltose infusions have been shown to improve morbidity but not mortality in systolic left ventricle failure patients with transferrin saturations < 20% and ferritin <300µg/L, irrespective of haemoglobin concentration.

For end-stage systolic left ventricle failure, mechanical circulatory support with a left ventricular assist device (LVAD) or heart transplantation may be required. The average survival post-heart transplantation in Australasia is 14 years.[11]

Diastolic heart failure/ Restrictive cardiomyopathy/ Constrictive cardiomyopathy

The only treatments currently of potential benefit in diastolic heart failure are diuretics and spironolactone. Good fluid balance, blood pressure control and maintenance of sinus rhythm are paramount. There is no evidence for the use of ACEI, A2RB, beta-blockers, nitrates, hydralazine or sildenafil.

In constrictive cardiomyopathy, diuretics may improve symptoms but the only definitive treatment is pericardiectomy.

Systolic right ventricular failure

There is no evidence for the use of ACEI, A2RB or beta blockers in isolated systolic right ventricular failure. The only potential benefits are with diuretics, including aldosterone antagonists, digoxin, pulmonary vasodilators and inotropes.

cont'd page 24

Prognosis

PRIOR to the discovery of ACEI, A2RB, beta blockers and devices, the mortality at one year from heart failure was around 50% — the same as for lung cancer. That figure has now reduced to around 10% per annum, but is higher in patients presenting with Class IV NYHA.

Patients can be roughly classified into three even groups: full recovery; partial recovery; or no recovery or deterioration (see figure 5). Recovery can often take up to two years, but most occurs within the first 3-6 months. Patients with ischaemic cardiomyopathy usually have less recovery because of scar tissue.

As there are currently no data on medication cessation, patients should be advised to continue therapy with ACEI/A2RB and beta-blockers lifelong once recovery occurs.

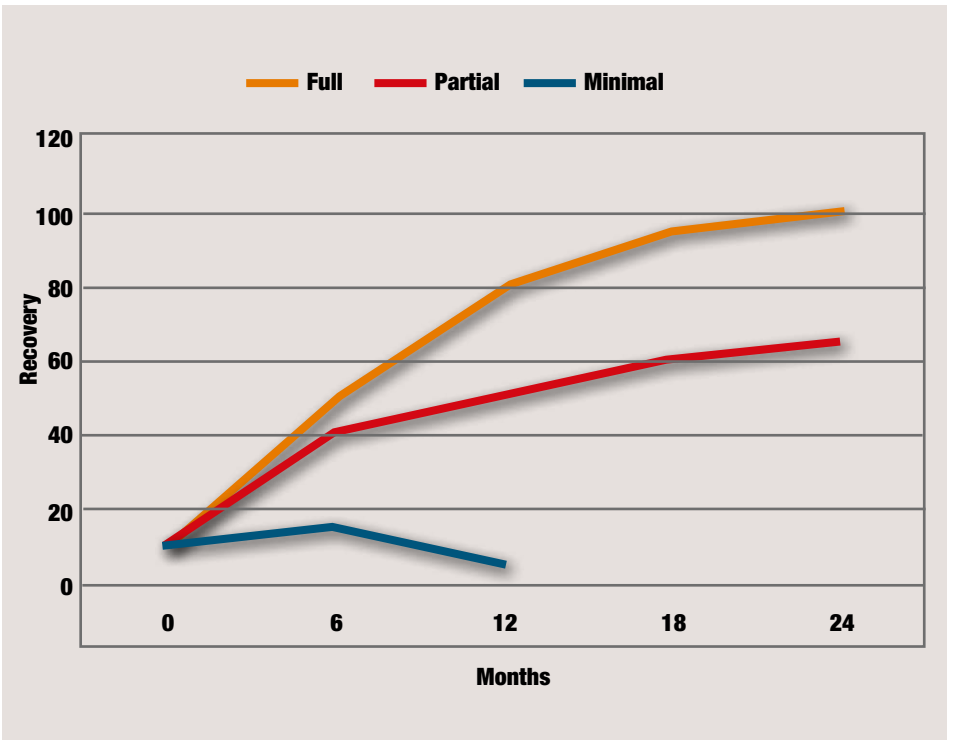

Figure 5. Prognosis of systolic left ventricular failure.

References
Available on request from howtotreat@cirrusmedia.com.au

Follow-up

HEART failure is a chronic disease with high mortality and hospital readmission rates, for both cardiovascular and non-cardiovascular causes. Strategies to reduce the risk of mortality or readmission include: early review post-discharge; frequent review for unstable patients; multidisciplinary team approach (physicians, GPs, dieticians, physiotherapists, pharmacists and nurses); and nurse practitioner or heart failure nurse clinics focusing on education, weight surveillance and medication titration.

Studies show that patients who receive no follow-up within 30 days post-hospital discharge have 30% higher mortality than those seen by their local practitioner or specialist.

Interestingly, the mortality difference is no greater if they are seen by a GP rather than a specialist, but there is a 30% greater reduction if conjoint care occurs with both GP and specialist. This mortality reduction is statistically as beneficial as an ACEI or beta-blocker.

If the patient is seen within one week post-hospital discharge, then there is a 30% reduction in one month readmission rates.[12-14]

Patients with rapidly deteriorating symptoms or shock should be referred to hospital urgently.

New treatments

Ivabradine

TRIALS have shown the selective sinus node inhibitor ivabradine can be added to the treatment options for patients with heart failure. Ivabradine works on the I-f channel and decreases resting heart rate. It was shown in the SHIFT study to reduce the combined endpoint of heart failure admissions and death by 18%.[15] This was driven by a reduction in heart failure admissions by 26%.

Although there was no overall benefit for mortality; subgroup analysis has shown that if the heart rate is greater than 77bpm, there is a 19% reduction in all-cause mortality and a 39% reduction in heart failure mortality. It is now PBS-listed for the treatment of chronic stable heart failure in patients with normal sinus rhythm with a heart rate greater than 77bpm, left ventricular ejection fraction < 35% and class II-III NYHA dyspnoea who have a contraindication for beta-blockers or are taking maximum tolerated dose.

Riociguat

Riociguat is the first-in-class of soluble guanylate cyclase agonists and it has recently been approved by the US Food and Drug Administration for the treatment of primary pulmonary arterial hypertension and chronic thromboembolic disease. Two recent trials — PATENT-1 and CHEST-1 — have shown this add-on therapy significantly improves exercise capacity.[16,17] Its primary area of action is on the pulmonary vascular bed resulting in pulmonary vasodilatation.

Spironolactone

Spironolactone has recently been shown to be of potential benefit in patients with diastolic heart failure. The Aldo-DHF study showed that a dose of 25mg daily for one year reduced echocardiographic parameters of diastolic heart failure, but did not improve clinical symptoms.[18]

The recently published TOPCAT study showed no improvement in mortality, but a 15% reduction in heart failure admissions.[19] Apart from diuretics for symptomatic relief, there is no other treatment in the armamentarium against diastolic heart failure. Based on these trials, I currently use 25mg-50mg spironolactone daily with careful renal and potassium monitoring (creatinine < 200mmol/L and K^+ < 5mmol/L) in symptomatic patients with diastolic heart failure.

Omecamtiv mecarbil

This shows promise in reducing symptoms of dyspnoea in acute heart failure according to a recent finding of ATOMICAHF, a phase II trial in systolic heart failure.[20] Omecamtiv mecarbil is a cardiac myosin activator, which increases stroke volume and cardiac output without significantly increasing myocardial oxygen consumption or heart rate. Phase III trials are currently underway.

Ultrafiltration

Aquapheresis is a portable ultrafiltration unit that can remove up to 500mL of free water per hour in patients with end-stage, diuretic-resistant heart failure and renal impairment. Current studies have shown it is equally as effective as intravenous diuretics and safe. No studies have shown a mortality benefit, but it does reduce hospital readmission rates and length of stay by approximately 50%, two important markers of morbidity.[21]

Entresto

Entresto (Valsartan/Sacubitril) is a new combination of an angiotensin II receptor blocker and a novel neprilysin inhibitor, sometimes called an ARNI. When compared with enalopril in the PARADIGM-HF study, there was a 16% relative risk reduction in all-cause death.[22] The TGA approved this drug for use in Australia in February 2016, but it is currently unavailable on the PBS.

cont'd page 26

Case study

MARK, aged 40, presents with dyspnoea and pedal oedema secondary to a viral cardiomyopathy three months ago. His LV ejection fraction at the time was 30%.

He is a non-smoker and non-alcohol drinker with no past medical history. His blood work-up was all normal. Mark was started on frusemide 40mg, perindopril 2.5mg and bisoprolol 2.5mg daily. His diuretic is ceased after one week because of reduction in oedema and symptoms.

He now presents to you for his three-month review. He is well, Class I NYHA (namely, not breathless) with BP 120/80mmHg, heart rate 76 bpm, sinus rhythm. He is euvolaemic, warm and perfused, with a clear chest and normal heart sounds. His echo shows an improvement in LV ejection fraction to 45%.

What will you do next?

A. Nothing, he is stable
B. Add diuretics
C. Increase ACEI
D. Increase beta-blocker
E. Add digoxin
F. Add Ivabradine
G. Stop medications as he no longer needs them as his ejection fraction is now 45%

Answer

With these patients we should use the warm or cold, wet or dry assessment table to determine our approach.

Mark is obviously not fluid overloaded but dry, and therefore does not require diuretics. His ejection fraction has improved, but not yet back to normal, so we must try to increase his medications. He is warm with a good blood pressure and so he is in the 'green box '. This means we are safe to carefully up-titrate his heart failure medications (ACEI and/or beta-blocker). He does not require digoxin as his cardiac failure is not severe enough and he is not on maximal ACEI/beta-blocker.

Warm and dry	Warm and wet
Cold and dry	Cold and wet

He still has an impaired left ventricle, so we cannot stop his heart failure medications. The answers are therefore C and D.

Conclusion

HEART failure is the inability of the heart to pump blood adequately and results in reduced perfusion and congestion and is predominantly, but not limited to, left ventricular systolic failure.

There are many aetiologies occurring at any age that need to be considered, but the majority of diagnoses can be made with simple blood tests and readily available investigations, such as a chest X-ray, ECG and echo.

When assessing patients, think about their fluid status and perfusion as this will help guide their management. The treatment will depend on whether the heart failure is left- or right-sided, and systolic or diastolic, as successful treatments for one cannot be extrapolated to work in the other.

Patients with systolic left ventricular failure should be constantly up-titrated to maximum tolerated doses of ACEI and beta-blocker, with consideration of add-on therapies or devices.

Overall, patients who are compliant should be reassured they have a good prognosis, but may take up to two years to recover.

Whether the diagnosis is right- or left-ventricular failure, it is a chronic disease that is best managed by a multidisciplinary team that includes input from a dietitian, physiotherapist and pharmacist, with collaboration between the GP and the cardiologist.

Early review post-hospital discharge facilitates the best patient outcomes.

How to Treat Quiz

Heart failure — 1 July 2016

INSTRUCTIONS

Complete this quiz online and fill in the GP evaluation form to earn 2 CPD or PDP points.
We no longer accept quizzes by post or fax.

The mark required to obtain points is 80%. Please note that some questions have more than one correct answer.

GO ONLINE TO COMPLETE THE QUIZ

www.australiandoctor.com.au/education/how-to-treat

1. Which TWO statements regarding heart failure are correct?
a) Heart failure can be secondary to reduced contraction (systolic heart failure).
b) Heart failure can be secondary to increased contraction (restrictive or diastolic heart failure).
c) Heart failure can be due to dysfunction of the left atrium, right atrium or both.
d) Heart failure can be secondary to high output states.

2. Which THREE statements regarding the symptoms and signs of heart failure are correct?
a) Symptoms of heart failure are proportional to the degree of congestion and reduced perfusion caused by the lack of forward blood flow through the heart.
b) Left ventricular systolic failure results in congestion of the lungs and alveolar oedema, producing dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea.
c) Right ventricular failure results in venous congestion and may be associated with tricuspid regurgitation.
d) Cardiac cachexia is more often associated with left ventricular failure due to hepatic congestion and anorexia.

3. Which TWO conditions predominantly affect the right ventricle?
a) Chronic thromboembolic disease.
b) Hypertrophic obstructive cardiomyopathy.
c) Pulmonary hypertension.
d) Ischaemic heart disease.

4. Which THREE conditions are more commonly associated with diastolic heart failure?
a) Hypertension.
b) Thyrotoxicosis.
c) Diabetes.
d) Obesity.

5. Which TWO tests are easily accessible and are first line in assessing heart failure?
a) Stress ECG.
b) Chest X-ray.
c) CT scan with contrast.
d) Transthoracic echocardiogram.

6. Which THREE statements regarding brain natriuretic peptide (BNP) are correct?
a) BNP is a good screening test, but is only covered by Medicare for investigation of heart failure in the ED.
b) It is useful as a rule-out test for heart failure as it has a high negative predictive value.
c) BNP levels decrease with increasing age.
d) Caution must be used interpreting a positive test or a negative test in obese patients.

7. Which TWO statements regarding the treatment of systolic left ventricular failure are correct?
a) Patients who are warm and dry may allow for up-titration of heart failure therapy or reduction of diuretics.
b) Patient who are wet require diuresis.
c) Patients who are cold either require an increase of vasodilator therapy or inotropes.
d) Warm and dry patients are often the sickest, frequently requiring hospital admission.

8. Which THREE statements regarding the use of beta blockers to treat systolic left ventricular failure are correct?
a) Beta blockers have been shown to improve survival by a further 10% when added to ACEI.
b) Beta blockers should only be started or up-titrated when patients are euvolaemic.
c) More cardioselective beta blockers should be used in patients with hypotension, asthma, fatigue and male impotence.
d) Beta blockers can be commenced before an ACEI or A2RB for control of arrhythmias, such as atrial fibrillation or ventricular tachycardia.

9. Which THREE statements regarding treatment are correct?
a) The only treatments currently of potential benefit in diastolic heart failure are diuretics and ACEIs.
b) There is no evidence for the use of ACEI, A2RB or beta blockers in isolated systolic right ventricular failure.
c) In constrictive cardiomyopathy, diuretics may improve symptoms, but the only definitive treatment is pericardiectomy.
d) In systolic right ventricular failure, the only potential benefits are with diuretics including aldosterone antagonists, digoxin, pulmonary vasodilators and inotropes.

10. Which THREE statements regarding prognosis and follow-up are correct?
a) Patients can roughly be classified into three even groups: i) full recovery, ii) partial recovery or iii) no recovery or deterioration.
b) Patients can be weaned off their medication after around two years, as at this stage, maximal recovery will have been attained.
c) Studies show that patients who receive no follow-up within 30 days post-hospital discharge have 30% higher mortality than those seen by their local practitioner or specialist.
d) If the patient is seen within one week post-hospital discharge, there is a 30% reduction in one-month readmission rates.

CPD QUIZ UPDATE

The RACGP requires that a brief GP evaluation form be completed with every quiz to obtain category 2 CPD or PDP points for the 2014-16 triennium. You can complete this online along with the quiz at **www.australiandoctor.com.au.** Because this is a requirement, we are no longer able to accept the quiz by post or fax. However, we have included the quiz questions here for those who like to prepare the answers before completing the quiz online.

Australian Doctor Education

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NEXT WEEK

Next week's How to Treat describes the management of strabismus in children. A high index of suspicion, low threshold for referral, plus prompt treatment make a difference to treatment outcomes. The author is **Dr Caroline Catt,** adult & paediatric ophthalmologist, Sydney Ophthalmic Specialists, the Children's Hospital at Westmead and Sydney Eye Hospital.