Case 2
Don's wife finally convinced him to seek treatment

This 76-year-old male has been unwell for the last few months, but in the early hours of this morning he becomes too breathless to sleep. His wife insists he seek medical help, and he arrives in the Emergency Department shortly after 3 a.m. A careful but timely history and examination identifies the problem and the reason it became particularly bad tonight …

Timeline summary

01:40	Wakes from sleep breathless.
03:05	Arrives at Emergency Department; triaged category 3.
03:15	Led into a cubicle; nursing staff commence assessment.
03:30	Seen by resident medical officer.
03:30	12-lead electrocardiogram (ECG).
03:50	Intravenous access, bloods sent to pathology.
04:15	Chest X-ray (CXR) taken.
04:25	Patient and results reviewed; therapy commenced.
04:45	Referred for medical admission.

Learning objectives

Physiological

- Describe the physiological basis of breathlessness.
- Understand how rheumatic fever as a child may lead to congestive heart failure.
- Explain to what degree the heart is able to compensate for valvular abnormalities.
- Understand how congestive cardiac failure (CCF) can lead to inappropriate reabsorption of salt and water by the kidneys.
- List the factors controlling β-type natriuretic protein (BNP) release.
- Describe the physiological basis of atrial fibrillation.

Clinical

- Describe the possible causes for breathlessness in this context.
- Be able to recognise the symptoms and signs of heart failure.
- Understand the appropriate and timely use of investigations to diagnose CCF.
- Understand the acute and longer-term management of CCF and the evidence behind it.

Context

Don has been fairly healthy all of his life. He had rheumatic fever as a child, but other than that he'd been well for years. He had mildly elevated blood pressure for which he took a tablet daily. He had worked as an engineer for the local council until his retirement 11 years ago. He played bowls regularly and liked nothing better than to have lunch every day with Jan, his wife of over 50 years.

Over the last few weeks he'd been noticing that he had been getting breathless when walking up the gentle hill near his house. He thought this was due to 'old age' and that it would settle down with time. However, it had been getting steadily worse and now he was even getting puffed walking around the house. A couple of times he'd actually been woken up at night gasping for breath.

Tonight was 'one of those nights'. He'd gone to bed feeling well enough but now, just after 1:30 a.m., he felt he couldn't get his breath. His wife was woken by his noisy breathing. He assured her that he would settle down, but she realised that he needed help. Despite feeling better after sitting up in bed for a few minutes, he reluctantly agreed and allowed her to drive him to hospital. They arrived at the Emergency Department just after 3:00 a.m. Don was feeling much better by this time and when seen by the triage nurse he was given a triage 'category 3'. He was led into a cubicle where he changed into a hospital gown and waited for the nurse and doctor to see him.

03:25 hours

The department has been fairly busy for a Tuesday night and Helen, the intern rostered on overnight, has been working hard since her shift began at 10:30 p.m. Things are settling down now, and Helen has finally been able to sit down for a cup of coffee and a bowl of fruit. Feeling refreshed, she returns to the Emergency Department floor and checks to see who is the next patient to be seen.

Helen walks into the cubicle to find the nurse completing her observations.

'Here you are', she says, handing the sheet to the intern. 'Let me know if there's anything else you'd like.'

The observations are as follows:

- Elderly male c/o breathlessness. Now settled.
- PR: 86 bpm, irregular
- BP: 160/110 mmHg
- RR: 20 breaths/min
- temperature: 36.7°C
- SpO_2: 95% room air

Don is quite comfortable sitting on the trolley. Helen introduces herself to him and begins her assessment.

PC

Physiology comment

Dyspnoea (breathlessness)

The American Thoracic Society defines dyspnoea (shortness of breath; breathlessness) as 'a term used to characterise a subjective experience of breathing discomfort that is comprised of qualitatively distinct sensations that vary in intensity'. Others have defined it as an undue awareness of breathing or awareness of breathing difficulty. The subjective nature of dyspnoea makes it difficult to quantify.

Dyspnoea can be caused by a range of factors, including:

- reduced oxygenation;
- acidosis;
- stimulation of a range of mechanoreceptors in the lung;
- inadequate delivery to, or utilisation of, oxygen by peripheral tissues;
- inability to respond to ventilatory demands, for example due to increased resistance to breathing, inspiratory muscle fatigue;
- psychogenic issues.

Clinical question 1

(a) Describe your approach to a patient such as Don.
(b) List the differential diagnoses you are considering at this early stage.
(c) What questions will you ask him?

Helen proceeds to take a detailed history from Don. He tells her about his breathlessness over the preceding few weeks, and how he sometimes felt as if he couldn't breathe when he was lying in bed at night and how he would feel better when he sat up. In fact, he would feel better if he slept propped up on a couple of pillows. This prompts Helen to ask some more specific questions.

Clinical question 2

(a) What are the medical terms for Don's symptoms?
(b) What condition are they suggestive of?
(c) Describe how you will proceed now.

Helen thinks that this points towards heart failure as a possible cause of Don's symptoms. She glances at his ankles, and notes that they looked swollen. She takes his pulse, and notes that it felt irregular. She looks at Don and continues to take her history.

Dr Helen: 'Have you ever had any chest pain?'

Don: 'No, never.'

Dr Helen: 'Have you ever had any problems with your heart?'

Don: 'I had rheumatic fever when I was a child and I was told it could affect my heart later, but I've never noticed anything. A doctor once said I had a murmur …'

Dr Helen: 'Do you take any medication?'

Don: 'I'm on Diltiazem for my blood pressure. I've been on it for years.'

Dr Helen: 'Your legs seem quite swollen—has this been a problem for long?'

Don: 'Come to think of it, I've only noticed them in the last few weeks … my shoes have become a bit tight …'

Clinical question 3

(a) Do these answers help you make a diagnosis?
(b) What do they reveal?
(c) Are there any significant findings in this history that lead you towards a particular diagnosis?

Clinical comment

Don has fairly typical symptoms of CCF, as evidenced by his paroxysmal nocturnal dyspnoea (waking up breathless from sleep), orthopnoea (inability to breathe except in an upright position) and evidence of peripheral oedema. These form part of various diagnostic criteria in existence, such as the Framingham criteria and the WHO criteria for the diagnosis of heart failure. Of particular significance are the findings of a history of rheumatic heart disease (which predisposes to the development of valvular heart disease), the presence of an irregular pulse (suggesting atrial fibrillation) and his use of calcium channel blocker medication (which has a negative inotropic effect and may exacerbate heart failure). The role of the medical practitioner in this instance is to make and confirm the diagnosis, consider precipitants and commence appropriate therapy.

Physiology comment

Aetiology of congestive cardiac failure

It is believed to be the immunological component of rheumatic disease that causes scarring of the endothelial layer of the heart valves, which can later become symptomatic. In the disease, the mitral and tricuspid valves are often affected, along with the aortic valve. If a history of rheumatic disease is elicited, it is important to consider that often multiple valves are affected and therefore cardiac symptoms can be more complex.

Helen thinks to herself that the diagnosis of CCF is likely, but she needs more information. She performs a physical examination. She requests that a 12-lead ECG and a CXR be performed, and she proceeds to insert an intravenous cannula and obtain blood to be sent to pathology.

Clinical question 4

(a) What blood tests would you request at this stage?
(b) Discuss the role of BNP in the diagnosis of heart failure.
(c) What physical findings would you expect to find?
(d) Would you commence any treatment at this time?
(e) List the possible precipitants of heart failure.

Helen records her physical examination findings as follows (see Fig. 2.1).

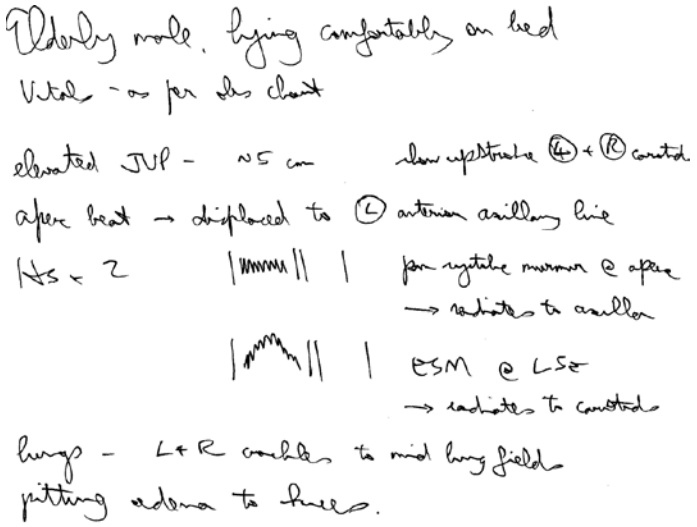

Figure 2.1 Doctor's handwritten notes

03:30 hours

The ECG is performed by the nurse and shown to the intern (see Fig. 2.2).

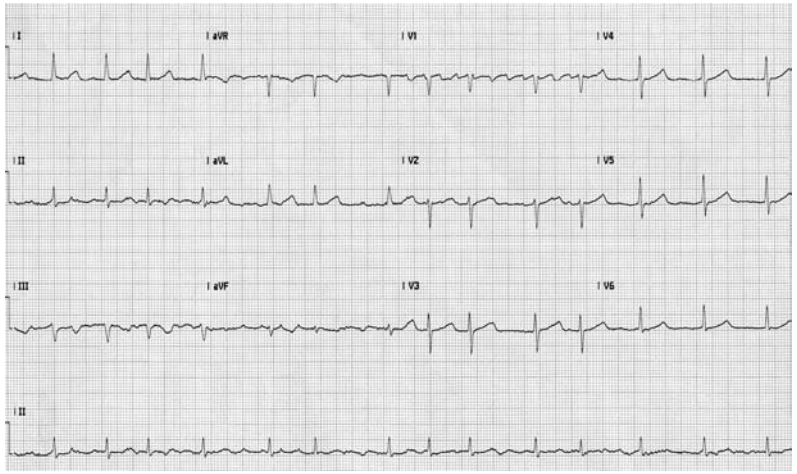

Figure 2.2 ECG

The CXR is performed (see Fig. 2.3).

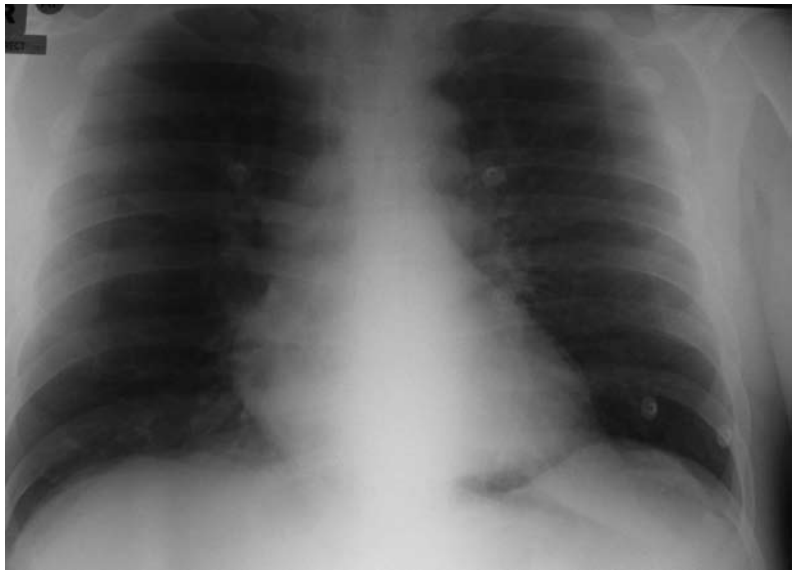

Figure 2.3 Chest X-ray

Clinical question 5

(a) Describe the ECG and the CXR as if you are presenting your findings to a colleague.
(b) Summarise the findings into a diagnosis and write a problem list. Discuss what may have precipitated the condition.
(c) Does Don require admission to hospital?
(d) If so, describe how you will refer him to the inpatient team. Include your diagnosis and initial management plan.

04:25 hours

Helen returns to the cubicle and sits down with Don and his wife.

'The results have come back and there's a good reason you've been feeling so breathless recently. You do have two murmurs, and they're contributing to your heart condition. Also, your heart is in an irregular rhythm called "atrial fibrillation". This is all causing your heart to not pump as effectively as it should, and fluid is accumulating in your lungs and the rest of your body. Your condition is called "congestive cardiac failure", and we need to admit you to hospital.'

Helen proceeds to discuss the nature of Don's condition with him and his wife, and to initiate therapy to treat his condition (see Table 2.1).

Table 2.1 Medication administered to patient

Treatment	Route	Rationale
40 mg frusemide	IV	To reduce the amount of fluid overload
500 µg digoxin—first of his loading dose	Oral	To control rate of atrial fibrillation
80 mg enoxaparin	Subcutaneous	To provide therapeutic anti-coagulation (as prophylaxis for thromboembolism secondary to his atrial fibrillation) as well as treatment for a possible ischaemic cause
150 mg aspirin	Oral	Presumptive treatment for a possible ischaemic cause for his condition
Cessation of calcium channel blocker		Removal of a negative inotropic agent which may worsen his cardiac failure
Commenced ACE inhibitor: 10 mg enalapril daily		ACE inhibitors have been shown to improve morbidity and mortality in patients with CCF
Patient weighed and placed onto fluid balance observations		To track efficacy of diuretic therapy
Request form for transthoracic echocardiogram sent to cardiology department		To assess heart structure and function

04:45 hours

Don is referred to the medical registrar for admission.

PC

Physiology comment

Atrial fibrillation

Atrial fibrillation is an irregular rhythm generated by disorganised atrial depolarisation, with random conduction of some impulses through the atrio-ventricular (AV) junction to the ventricles. Chronic CCF is associated with atrial fibrillation due to enhanced automaticity and multiple re-entry circuits created by atrial deformation. The increased resistance at the AV node normally blocks most atrial impulses, but the ventricular rate is often in excess of 200 bpm. The faster the ventricular rate, the greater the decrease in cardiac output owing to reduced stroke volume and cardiac output. An irregular rhythm with p waves replaced with a characteristic wavy baseline (f-waves) can be observed on the ECG. The QRS complex will be normal unless additional conduction abnormalities occur downstream of the AV node.

Cardiac compensation in aortic stenosis

In aortic stenosis, a pressure gradient develops across the damaged valve as the left ventricle is forced to generate an increased pressure to overcome the stenosed opening. The patient often remains asymptomatic during the early stages of the disease, as the left ventricle is able to compensate adequately (via the mechanism of increased preload). However, as the left ventricular wall begins to hypertrophy in response to the increased pressure the reduced wall compliance decreases filling volume, leading to a drop in cardiac output. In order to maintain blood pressure, total peripheral resistance (TPR) increases. As diastolic pressure is determined by the ease of blood run-off to the venous side of the circulation, this increase in TPR often leads to an increase in diastolic pressure. Over time, usually several symptoms gradually appear:

- angina;
- syncope;
- heart failure.

Angina occurs when the coronary blood flow is unable to meet the requirements of the hypertrophied myocardium, especially during exercise. Syncope may result from an inappropriate haemodynamic reflex in which the TPR falls, thereby causing decreased cerebral perfusion. Arrhythmias and/or heart block secondary to the hypertrophied myocardium may also lead to reduced blood pressure and syncope. Dyspnoea can result from pulmonary capillary congestion (secondary to left ventricular

PC

(LV) failure), which reduces haemoglobin oxygenation and diminishes lung compliance.

Orthopnoea refers to dyspnoea that occurs while lying and results from pooling of blood in the central vasculature due to gravity, which leads to an increase in cardiac volume. This increased LV preload leads to an increased gradient across the aortic valve, and therefore backup of blood in the pulmonary capillaries. Orthopnoea is relieved by propping the upper body up in order to reduce the venous return. Paroxysmal nocturnal dyspnoea is the occurrence of sudden dyspnoea that wakes the patient from sleep. It may occur when the patient inadvertently slips off the pillows used to elevate the upper body.

CC

Clinical comment

Don has been diagnosed with congestive cardiac failure. He is classified as Class 3 heart failure using the ACC/AHA classification system, evidenced by dyspnoea, fatigue and reduced exercise tolerance. His immediate problem list looks like this:

biventricular CCF, precipitated by:
- valvular heart disease (likely aortic stenosis and mitral regurgitation);
- atrial fibrillation;
- possible underlying ischaemic heart disease;

and exacerbated by use of a calcium channel blocker.

Don has clinical features of left heart failure (orthopnoea and paroxysmal nocturnal dyspnoea) and right heart failure (elevated jugular venous pressure and peripheral oedema). The distinction is usually artificial, and most patients seem to have some element of both. In this circumstance he was not in acute respiratory distress, so it was reasonable to commence therapy once more information became available.

His ECG showed that he was in atrial fibrillation. Although there were no specific features of ischaemia on this particular ECG, cardiac ischaemia is an important cause to consider for patients in this age group. His CXR revealed no overt pulmonary oedema, but he had a mild cardiomegaly consistent with the clinical diagnosis of CCF.

The use of the BNP as a diagnostic aid is growing around the world. It may be of benefit in distinguishing cardiac causes from respiratory causes of breathlessness. While Don's diagnosis may seem quite straightforward, in some circumstances it is difficult to distinguish between different causes of breathlessness. In fact, many patients have concurrent illnesses, and

CC

it is not uncommon for patients with emphysema to have an element of right heart failure as well.

Congestive cardiac failure is a growing problem in Australia, and the National Institute of Clinical Studies has focused on four key areas:

- recognition of heart failure symptoms;
- diagnosis of heart failure;
- prescription of appropriate drugs, particularly angiotensin-converting enzyme (ACE) inhibitors and β-blockers;
- adherence to treatment, particularly medication.

In this case, emphasis is on the first two points. Don should notice a significant improvement in his symptoms once appropriate therapy has been commenced. He will require further investigation of his heart function and of the precipitants of his heart failure. Medical therapy may be sufficient, but surgical therapy of his valvular disease may be required. He may require long-term anticoagulation for his atrial fibrillation. These issues will need to be discussed with his treating cardiologist.

PC

Physiology comment

Congestive cardiac failure and renal function

In the later stages of CCF the renal system often inappropriately reabsorbs both salt and water. This leads to an expansion of the extracellular fluid volume, further exacerbating the central and peripheral oedema already present. The renal system is stimulated to reabsorbed salt and water due to the reduction in blood pressure brought about by the cardiac failure. The lowered blood pressure is sensed by both intrarenal (afferent arteriolar and macula densa) pressure/flow and extrarenal (baroreceptors) receptors that bring about activation of the renin/angiotensin/aldosterone system.

The renal system has no way of directly responding to extracellular fluid (ECF) volume changes, and instead relies upon an indirect measure of blood pressure to alter salt and water balance. Under normal conditions ECF volume and total body sodium decrease and increase in step with blood pressure. In CCF, however, the link between increasing ECF volume (preload) and increasing blood pressure is lost due to the failing heart. Therefore even though the ECF volume is progressively increasing the blood pressure remains low (and may even fall if the myocardium is overstretched or damaged) and the kidney responds by increasing the ECF volume even further.

PC

Factors controlling B-type natriuretic factor release

Cells in the cardiac atria secrete a peptide hormone called B-type natriuretic protein (BNP) when the atria are overstretched (as seen during ECF volume expansion) in CCF. B-type natriuretic factor acts to inhibit sodium reabsorption directly or indirectly by the nephron, therefore offsetting to some degree the almost total reabsorption of sodium that occurs in CCF.

Epilogue

Don spent 5 days in hospital. He lost 6.5 kg in weight, indicating that he was significantly overloaded with fluid upon admission. His echocardiogram showed significant disease of his aortic and mitral valves, and a subsequent angiogram confirmed this. The same angiogram revealed no significant coronary artery disease. He was still in atrial fibrillation upon discharge, and he had been commenced on oral anticoagulation. His discharge medications were:

- warfarin: 3 mg nocte
- digoxin: 125 μg mane
- enalapril: 20 mg mane
- carvedilol: 3.125 mg b.d.
- frusemide: 40 mg mane

He was referred to a cardiothoracic surgeon for valve replacement surgery. A nurse practitioner specialising in heart failure will visit him regularly to ensure optimal use of his medication and to identify any early signs of deterioration.

References and further reading

1 National Heart Foundation of Australia and Cardiac Society of Australia and New Zealand Chronic Heart Failure Clinical Practice Guidelines Writing Panel. Guidelines for management of patients with chronic heart failure in Australia. MJA 2001; 174: 459–66. www.mja.com.au
2 National Institute of Clinical Studies. The diagnosis and recognition of congestive cardiac failure. Prepared by the Centre for Evidence Based Practice at the University of Queensland. 2002. NICS, Melbourne. http://www.nicsl.com.au
3 Ewald, B. B-type natriuretic peptide: a new diagnostic tool for congestive heart failure. Aust Prescr 2003; 26: 64–5.
4 Talley, N.J. and O'Connor, S. Examination medicine. 5th edn, 2006. Sydney, Churchill & Livingstone.

5 Hunt, S.A., Baker, D.W., Chin, M.H., Cinquegrani, M.P., Feldman, A.M., Francis, G.S., Ganiats, T.G., Goldstein, S., Gregoratos, G., Jessup, M.L., Noble, R.J., Packer, M., Silver, M.A. and Stevenson, L.W. ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1995 Guidelines for the Evaluation and Management of Heart Failure). 2001. American College of Cardiology website: http://www.acc.org/clinical/guidelines/failure/hf_index.htm

6 Mueller, C., Scholer, A., Laule-Kilian, K. et al. Use of B-type natriuretic peptide in the evaluation and management of acute dyspnea. N Engl J Med 2004 Feb 12; 350(7): 647–54.

Review

Level 1: Content knowledge

1 The inappropriate retention of salt and water by the kidneys often seen in cases of chronic cardiac failure is caused by:
- **A** Increased circulating volume stimulating the baroreceptor response
- **B** Increased circulating volume not resulting in an increase in cardiac output and blood pressure
- **C** A hypovolaemic state stimulating the baroreceptor response
- **D** A lack of the baroreceptor response

2 Which of the following is not a cause of dyspnoea?
- **A** Acidosis
- **B** Stimulation of mechanoreceptors in the lung
- **C** Inadequate delivery to, or utilisation of, oxygen by peripheral tissues
- **D** Increased haematocrit

3 In aortic stenosis:
- **A** A pressure gradient develops across the stenosed valve.
- **B** Pressure increases after the stenosed valve.
- **C** The ventricle will progressively become more compliant.
- **D** Total peripheral resistance will decrease.

Level 2: Clinical applications

1 Which of the following therapies have proven benefit in the management of congestive heart failure?
- **A** Digoxin
- **B** Angiotensin converting enzyme (ACE) inhibitors
- **C** Verapamil (a calcium channel blocker)
- **D** Amiodarone
- **E** All of the above have a role in the management of congestive heart failure.

2 Which of the following symptoms are not suggestive of congestive cardiac failure as a cause of breathlessness?
- **A** Orthopnoea
- **B** Fatigue
- **C** Cough
- **D** Paroxysmal nocturnal dyspnoea
- **E** Haemoptysis