

Chapter 1

Clerking Patients

Clerking patients may seem a daunting task initially, but it is an important skill to master. A well performed history and examination allows you to not only reach an appropriate differential diagnosis, and thus request relevant investigations, but also to develop an effective doctor–patient relationship. This chapter will ensure that you structure your history taking, and will point out the important questions to ask. It will help you to focus on understanding symptomatology, common abnormal examination findings and your presentation technique.

1.1 History Taking

It is vital to have a structure, at least in your head, on which to base your history as the patient rarely gives you the information needed in an orderly fashion.

The key cardiac symptoms are:

- Chest pain
- Dyspnoea
- Palpitations
- Oedema
- Syncope

1.1.1 *Five simple steps to taking a cardiac history*

Step 1: Ask about the presenting complaint:

Also make sure you cover the following, which are the main cardiac symptoms:

- chest pain
- dyspnoea/shortness of breath

- palpitations
- syncope
- oedema.

Bear in mind that none of the above symptoms are cardiac *specific*; they can be caused by non-cardiac pathology, and therefore thoughtful *directed* questioning of each symptom can help determine its cause. If a patient presents with any one of these symptoms, do not forget to enquire about the others. Try to keep in mind the causes of each symptom, as this will help direct your questioning.

Step 2: Always ask about the five major risk factors, namely:

- | | | |
|-------------------------------------------------------------------------------------------------------------------------------------------------|---|-----------------------|
| <ul style="list-style-type: none"> • high blood pressure • hypercholesterolaemia • smoking • diabetes | } | <i>all modifiable</i> |
| <ul style="list-style-type: none"> • family history of cardiac disease — <i>non-modifiable</i>. | | |

Step 3: Don't neglect the past medical history, including:

- heart disease and previous cardiac investigations or procedures including angioplasty/stent/bypass grafting
- history of stroke/transient ischaemic attack
- history of peripheral vascular disease
- asthma — as B-blockers and adenosine can cause bronchospasm
- rheumatic fever — predisposition to valve disease
- thyroid disease — can cause palpitations and exacerbate heart failure.

Step 4: A full drug history:

- remember that patients don't often list aspirin or inhalers spontaneously
- any drug allergies? *Be specific* — patients do not always understand this question. Did the patient have an anaphylactic reaction/rash/nausea/vomiting, or did they simply not benefit from taking the drug?

Step 5: Patient's social circumstances:

- occupation (for example, taxi drivers and airline pilots have strict occupational health regulations)
- implications post-myocardial infarction
- who lives at home with the patient?
- pre-morbid health — before this admission what was the patient's level of independence and activity? Gives an idea on what to aim for prior to discharge
- alcohol history — predisposition to hypertension, cardiomyopathy and atrial fibrillation.

1.2 Chest Pain

Chest pain is a simple shorthand that we use to describe a wide variety of experiences. You'll be surprised at how often you and the patient may not be referring to the same thing! For example, does the patient mean an ache, heartburn, or heaviness in the chest? Finding out the nature or characteristics of the pain can provide important clues as to whether or not the pain is cardiac in nature.

There are two critical features of *cardiac* chest pain:

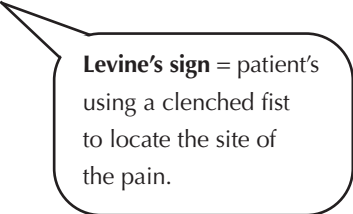
1. **Location** of the pain may be typically symmetrical across the centre of the chest, potentially including both shoulders and arms, and may radiate up the neck to the jaw. It may be more dominant on the left side.
2. **Exertional relationship** — coronary pain will always get worse when the patient is physically active. Pain that occurs randomly at rest and during exercise with no exertional link is usually non-cardiac in origin. Pain that has a habitual pattern of only occurring at rest is very unlikely to be cardiac.

Ask about associated shortness of breath and autonomic symptoms such as nausea and sweatiness. Glyceryl trinitrate (GTN) will usually offer some symptom relief, whether the patient is experiencing angina or a myocardial infarction. However, if pain lasts for more than

20 minutes with or without relief by GTN, consideration of a myocardial infarction (MI) is warranted.

1.2.1 *The key features of cardiac chest pain*

- crushing pain/tightness over central chest
- +/- radiation to jaw/down the left arm
- +/- autonomic symptoms, for example, nausea, sweatiness



Levine's sign = patient's using a clenched fist to locate the site of the pain.

Other cardiac causes of chest pain include:

- *Aortic dissection* — this is a life-threatening condition where blood is tracking between the layers of the wall of the aorta. Left untreated, there is a high risk of rupture and death. This is characterised by a 'tearing' central chest pain radiating to the back between the scapulae. May be accompanied by haemodynamic compromise.
- *Pericarditis* — this is inflammation of the pericardium which causes chest pain worse on inspiration or lying flat.
- *Arrhythmias* — these can sometimes cause chest pain if the rate becomes really fast.

The main non-cardiac causes of chest pain that are often mistaken for angina can be divided into **respiratory**, **gastrointestinal** and **musculoskeletal** causes. For example, *sharp* chest pain is often referred to as *pleuritic* chest pain and is suggestive of a pleuritic/respiratory cause, such as an infection (for example, pneumonia), inflammation (for example, pleurisy), or infarction (for example, a Pulmonary Embolism [PE]). It is caused by inflamed contact between the lung and pleura.

Some important differentiating questions for pleuritic pain include:

- Is it worse on deep inspiration? On coughing? With movement?
- Is the pain sharp? Like a sharp knife? Note that some patients will use the word ‘sharp’ to mean ‘severe’ rather than to describe the type of pain.

Consider a **gastrointestinal cause** such as oesophagitis, oesophageal reflux or spasm if the chest pain is *burning* and is worse on lying flat or is related to meals. Pain that is relieved on sitting forward may suggest pancreatitis.

- Is the pain worse in any one position? Such as lying flat?
- Is the pain relieved by sitting forward?
- Is the pain worse before/after eating?

Don’t forget musculoskeletal/cutaneous chest pain which is confined to the chest wall and may be tender on palpation, for example, Tietze’s syndrome (costochondritis = inflammation of the costal cartilages), or herpes zoster (shingles), which can also give rise to a vesicular rash on the chest wall in a dermatomal distribution.

You can use the questions based on the well-known mnemonic SOCRATES below to guide your history taking.

- S** — Site: Where is the pain?
- O** — Onset: When did the pain first start?
- C** — Character: Describe the pain.
- R** — Radiation: Does the pain spread anywhere?
- A** — Associated symptoms: Any nausea, pallor, sweating or dizziness?
- T** — Timing and duration: How long does the pain last?
- E** — Exacerbating and relieving factors: This includes drug treatment, that is, GTN.
- S** — Severity: Score out of ten. (Ten being the worse pain the patient has ever experienced.)

Despite learning all of these well recognised patterns of chest pain, some patients may have *atypical* chest pain; that is, chest pain that doesn't follow the typical pattern of presentation for the disease. For example, a patient with a myocardial infarction may present with only left arm pain, or shortness of breath. They may even present with *pleuritic* sounding, *sharp* chest pain or heartburn. Pain that is very well localised or point-like, that is, a pain that the patient can point to with a fingertip, rarely turns out to be coronary in origin. Multifocal pain is also very unlikely to be cardiac in origin as coronary chest pain is characteristically stereotyped within any one single individual in the core location in which it affects, although as it becomes more severe it can radiate to more areas. Furthermore, some patients may not experience any chest pain at all, particularly those who are elderly, with diabetes, and/or following cardiac transplantation. It is thought that the weakening of the peripheral nerve function (through age, diabetes, or heart transplant surgery) prevents the information travelling to the brain in the way that typically causes pain.

Multifocal pain describes pain that occurs randomly at various points across the chest which may all present at the same time.

Radiation on the other hand, implies a more temporal relationship from the onset of pain in one place followed by another.

Even asymptomatic patients can have coronary disease or a silent myocardial infarction. In any admitted patient with multiple background risk factors for atherosclerosis, it is worth doing simple tests such as an electrocardiogram (ECG).

1.3 Dyspnoea

Dyspnoea or shortness of breath is another common cardiac symptom. It can be a symptom of heart failure, myocardial infarction or

valvular disease. Symptoms to illicit with patients who present with dyspnoea include:

1. **Orthopnoea** (difficulty in breathing when lying flat) —
 - a) ‘Do you need to sleep propped up to avoid breathlessness?’
Sleeping on more than two pillows may be significant. **Beware** though that orthopnoea can also occur in obese persons and people suffering from lung disorders, and that patients with back pain may also choose to sleep on several pillows.
 - b) ‘What happens to your breathing if you lie flat?’
2. **Paroxysmal nocturnal dyspnoea (PND)** —
 - ‘Do you wake up in the night, gasping for breath, which can be relieved by sitting up or getting out of bed?’

Orthopnoea and PND are two key symptoms of heart failure. Difficulty in breathing is often worse when patients are lying flat because fluid otherwise pooled in the lower limbs returns to the heart, which backs up into the lungs.

The **onset** of dyspnoea often gives a clue as to whether the cause is cardiac or non-cardiac in origin:

- Did it come on suddenly? (Consider pulmonary embolism/myocardial infarction.) Or did it come on gradually? (As in angina, heart failure, chronic obstructive pulmonary disease [COPD], pneumonia, or asthma.)
- Has it been getting worse?

It is important to ascertain the **severity** of dyspnoea which gives you a clinical indication of the severity of the disease. Although it is traditional to ask patients how far they can walk before they become short of breath, when this has been studied formally, it has been found to be unrelated to patients’ true exercise capacity, even after correcting for their perception of distance.

To quantify the severity of dyspnoea, the **New York Heart Association (NYHA)** classification can be used:

- Class 1: able to perform ordinary activities
- Class 2: dyspnoea on ordinary activity, but not at rest
- Class 3: dyspnoea on minimal exertion
- Class 4: dyspnoea at rest

- How many flights of stairs can you climb before becoming breathless?
- Do you get short of breath at rest?

To Impress!

Save time by asking key questions:

To distinguish between a patient in Class 1 and 2:

- If you were walking along with other people of the same age and sex, do they generally have to slow down for you? Or do you keep up with them? If they keep up, they are in **Class 1**.

To distinguish between Class 2 and 3:

- When you move around from room to room at home on the same level, do you get breathless or fatigued? If yes, they are in **Class 3**. If not, they are in **Class 2**.

Class 4 should be easy to identify!

Beware: Some patients may have limited exercise tolerance due to arthritis or other conditions. See if they can tell you what symptom is actually limiting them.

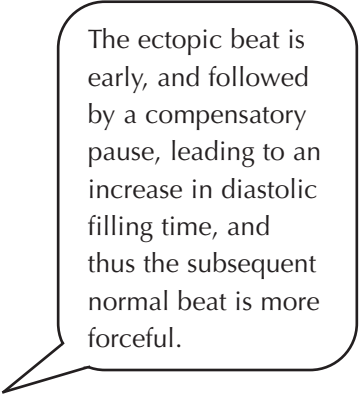
1.4 Palpitations

Palpitations can be a very difficult symptom to investigate. The word ‘palpitation’ means the *abnormal* awareness of the heart beating in the chest, and again may mean different things to different

people. Some patients feel that their heart is beating abnormally fast or slow. Other patients are aware that their heart is beating irregularly. A third group feel the heart is beating at a normal rate and regularly, but simply more intensely without good reason. It is important to identify what the patient means by palpitations. It can be helpful to ask the patient to tap out the rate and rhythm on the table. Patients with genuine significant arrhythmias are keen to do so. Patients with very brief and non-specific palpitations often decline to even try.

Some of the key questions that will help you differentiate the cause of the palpitation include:

- Is it brought on by worry?
- Does it occur on exercise or when you're excited?
- Do you notice it mainly when you're lying down? These features suggest a benign cause.
- Does it feel as though your heart drops or misses a beat? This suggests atrial or ventricular ectopics.
- Do the palpitations have an abrupt start or end? Many minutes to hours of palpitations with an abrupt start or end suggests significant pathology.



The ectopic beat is early, and followed by a compensatory pause, leading to an increase in diastolic filling time, and thus the subsequent normal beat is more forceful.

Has the patient had a 24-hour monitor/ECG? Don't forget the non-cardiac causes which are important to exclude in your history. These include:

- Drugs — that is, caffeine, nicotine, cocaine and any sympathomimetic, although remember they can both trigger pathological arrhythmias and cause simple sinus tachycardia.
- Metabolic disorders — anaemia, hyperthyroidism, and phaeochromocytoma (rare).

1.5 Syncope

Syncope can be defined as the temporary impairment of consciousness due to cerebral ischaemia. Taking an account of the attack can be divided into **three** key parts:

1) What happened *before* the faint?

- Did you have any warning before the faint?
- Were there any specific triggers? For example, standing up? Passing urine? Cold? The presence of a precipitant suggests a non-cardiac cause.

2) What happened *during* the faint?

- Discover whether there were any eye witnesses. If so, obtain an eye witness account.
- Was there any jerking, tongue biting, or incontinence? These are features of epilepsy but can occur with prolonged cardiac syncope.
- How long did it last?

3) What happened *after* the faint?

In general, cardiac causes of syncope involve a loss of consciousness for 1–2 minutes with complete recovery in seconds to minutes with no subsequent confusion.

The common causes of cardiac syncope are:

- structural heart disease leading to obstruction to outflow, i.e. seen in any of the valvular stenoses, for example, aortic stenosis and hypertrophic obstructive cardiomyopathy
- arrhythmias such as atrial fibrillation, supraventricular tachycardias or ventricular tachycardia/fibrillation
- pulmonary artery hypertension (rare).

Stokes Adams attack describes a transient bradycardia, a decrease in cardiac output and loss of consciousness in which there is no warning.

The patient becomes pale and drops to the floor. This can occur in any position and was originally used to describe the consequences of intermittent heart block.

Other commonly described syncopal events include vasovagal attacks, situational and postural hypotension. Vasovagal attacks are provoked by pain, fear, emotion, prolonged standing and warm environments. The response is due to **vasodilatation** and/or **bradycardia**. It doesn't occur lying down. The patient may experience preceding nausea; sweatiness and dizziness then fall to the floor and lose consciousness for 1–2 minutes.

Situational syncope is associated with specific triggers such as coughing and micturition. Postural hypotension is common in the elderly and causes dizziness or collapse on standing from lying or sitting position. This is due to inadequate reflex vasoconstriction. This response can be exacerbated if the patient is on anti-hypertensives or anti-anginals.

Don't forget to consider neurological causes such as epilepsy — that is why an eye-witness account is important (to report tongue biting, urinary incontinence, confusion, and so on). Lastly, don't forget metabolic causes such as hypoglycaemia, and drug-induced syncope (for example, blood pressure medications!).

1.6 Oedema

Peripheral oedema is the accumulation of fluid in the body's tissues. It can be divided into two types: non-pitting and pitting oedema. In non-pitting oedema the skin cannot be indented by external pressure and is due to reduced lymphatic drainage or thyroid disease. Heart failure causes pitting oedema, which is due to an increase in venous pressure secondary to ineffective pumping of the right side of the heart, together with salt and water retention. It characteristically affects both legs, often worsens as the day progresses, and is more severe the higher up the body it is located. The table below

summarises a simple way to remember the causes of peripheral oedema:

Table 1.1 Causes of non-pitting and pitting oedema

Non-pitting oedema	Pitting oedema
Hypothyroidism (mucopolysaccharide deposition)	<i>Usually bilateral</i>
Impaired lymph drainage	High venous pressures
<ul style="list-style-type: none"> surgical, radiation, malignant infiltration, infectious (filariasis), congenital (Milroy's disease) 	<ul style="list-style-type: none"> heart failure, renal failure, excessive IV fluids, steroids
Increased capillary permeability	Low albumin states
<ul style="list-style-type: none"> angio-oedema 	<ul style="list-style-type: none"> liver failure, nephrotic syndrome, protein losing enteropathy
	Vasodilatation
	<ul style="list-style-type: none"> drugs: dihydropyridines (amlodipine) and alpha-blockers (doxazosin)
	<i>Unilateral</i>
	Deep vein thrombosis
	Local infection, including burns

1.7 Assessment of Cardiac Risk Factors

Once you have obtained a thorough history of each of the symptoms above, a thorough assessment of the patient's cardiac risk factors can help to support or counter your diagnosis. You will be expected to be able to rattle off the list of risk factors without hesitation and to have asked the patient about each one. The greater the number of cardiac risk factors present, the greater their risk of a cardiac event.

1.7.1 Previous cardiac history

Start with asking about any previous cardiac history.

Remember to avoid using jargon and stick to simple terminology the patient will understand.

- Have you had any cardiac events before?
- If so, were the symptoms similar to the current ones? What happened on that occasion, that is, what tests were performed and what were the results?
- Have you had any previous interventional procedures such as a balloon angioplasty, stent insertion or coronary artery bypass surgery (CABG)? When did you have these procedures (dates)?

1.7.2 Hypertension

In patients with high blood pressure, ask about duration as an indication of severity. The longer the history of hypertension, the greater the likelihood of cardiovascular disease. (See the *Hypertension* section in Chapter 4 *Commonly Encountered Patients* for more information.)

- How long have you had high blood pressure for?
- What blood pressure medications have been tried before? (Why were they stopped?)
- What blood pressure medications are you currently taking?
- Do you measure your blood pressure at home?
- What are your recent blood pressure readings?

1.7.3 Diabetes

Don't just say ask if they have diabetes, find out roughly what age they were diagnosed, whether they went straight to insulin therapy, the duration of their disease and severity. There are two types of diabetes. Type 1 typically presents in childhood/youth and is treated immediately with insulin. Type 2 typically is of adult onset, commonly associated with obesity and often treated with diet and

HbA1c is a molecule formed when glucose is attached to haemoglobin in the blood. As haemoglobin circulates in the blood for 8–12 weeks, measurement of HbA1c gives an indication of the average blood glucose in a patient over the last 8–12 weeks.

tablets first. Look for the presence of end organ damage to eyes, nerves and kidneys.

- Have you got any problems with your eyes or kidneys?
- Do you get pins and needles in your hands or feet?

Finding out whether or not the patient is insulin dependent can give you an idea as to the stage. Patients with diabetes have a two to four-fold increase in relative risk of developing coronary heart disease. Aim to keep the blood glucose values between 4 and 6 mmol/l and HbA1c < 6%.

- Do you measure your blood sugar at home?
- If so, what is your usual range?

1.7.4 *Hypercholesterolaemia*

Often patients won't know if they have high cholesterol, therefore it is quite useful to ask if the patient is on any cholesterol-lowering medication instead. Occasionally, there may be evidence of familial hypercholesterolaemia (a fairly rare group of severe genetic disease).

Familial hypercholesterolaemia is defined by two criteria in the patient — NOT the family!

- 1) Total cholesterol concentration >7.5 mmol/l, or Low Density Lipoprotein (LDL) cholesterol >4.9 mmol/l.
- 2) Presence of tendon xanthoma or genetic mutation of **LDL receptor/apoB-100** in 1st/2nd degree relative.

1.7.5 *Smoking*

- Do you smoke? If so, how many cigarettes do you smoke a day?
- Have you tried to stop smoking? Have you sought any specialist advice on how to stop smoking?
- If you are an ex-smoker, how long ago did you give up?

Pack years

Textbooks and tutorials often teach you to calculate the number of pack years that a patient has smoked. You will notice experienced consultants rarely actually elicit this data in practice and certainly never present it. There are two reasons

for this. Firstly, the critical distinction to make is the division of patients into three groups — never smoked, ex-smoker and current smoker. This is because the ex-smokers and current smokers have a substantially elevated cardiovascular risk compared to the non-smokers. The extent of this elevation is relatively easy to judge from the age of the patient if you assume most patients start smoking in their teens. It saves time, when time is limited, not to get into the ups and downs of the patient's cigarette consumption over the years.

Secondly, the 'current smoker' group is vital to identify because these patients are targets for aggressive intervention. There is good evidence to show that patients who are offered advice and counselling on smoking cessation have higher odds of quitting than those without any help.

The *pack year* assumes a standard pack of cigarettes has 20 cigarettes, therefore 1 pack year is equal to a patient smoking 20 cigarettes a day for a year. You can easily calculate this by this formula:

$$\text{Pack years} = \frac{\text{number of cigarettes per day} \times \text{no. of years}}{20}$$

1.7.6 Alcohol

Small amounts of alcohol, around 1 unit per day, are consistently associated with reduced cardiac event rates for reasons that are less clear. Higher amounts of alcohol, however, can cause hypertension,

One unit of alcohol equals

- Small glass of wine
- Half a pint of normal strength beer
- Single 25 mls shot of spirit

cardiomyopathy and atrial fibrillation. Ask about weekly alcohol consumption:

- How much alcohol do you drink a week? In units?
- Is this in the form of beer? Spirits? Wine? Other?

Try to quantify consumption as much as possible (see above). Keeping their alcohol consumption below the recommended limits, of up to 21 units per day for men and up to 14 units per day for women, is advisable.

1.7.7 *Family history*

Family history of cardiovascular disease (CVD) and or stroke, especially present in first degree relatives under the age of 65 in males and 55 in females, increases a patient's risk of heart disease by 1.5.

In addition to the five major risk factors discussed, physical inactivity, obesity and a high fat and high salt diet are also risk factors, although less easily quantifiable. The drug history may also highlight any medical problems the patient may have forgotten to tell you

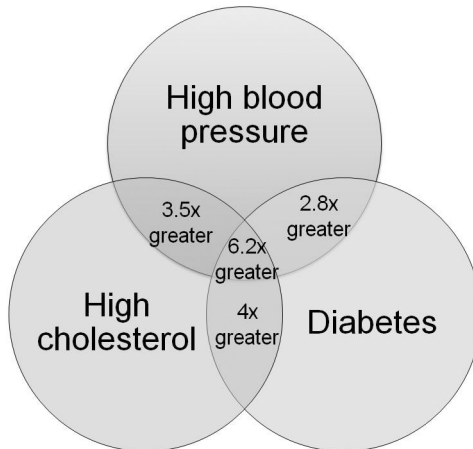


Figure 1.1 Combinational effect of each additional risk factor on cardiovascular risk

about, for example, the patient may have neglected to tell you about their hypothyroidism as they have become so accustomed to it but this can be easily picked up when the patient tells you that they take thyroxine regularly.

1.8 Social Circumstances

Don't forget the implications of the symptoms and the disease to the patients.

- What physical activity do you need to do for your work?

A myocardial infarction may cost a construction worker his job but not an office worker. What family support or responsibility does the patient have? What physical activity could the patient manage at home prior to the disease? This will help work out a realistic goal for post-discharge rehabilitation.

1.9 Implications in Primary Care

Primary prevention of cardiovascular disease in the community, even in apparently healthy individuals, is increasingly practiced. In the UK, all adults over the age of 40 undergo a comprehensive cardiovascular risk assessment in primary care once every five years. Many risk assessment models exist including the Framingham equation, Sheffield risk tables, the European coronary risk chart and the New Zealand risk assessment tool. The coronary risk prediction chart is the one recommended by the joint British societies and is based on assessment of all the risk factors including ethnicity, smoking, family history, weight, blood pressure and lipid levels. It stratifies patients according to their percentage risk over a ten-year period. These charts are useful in deciding the degree of intervention necessary, that is, simple lifestyle measures or initiating drug therapy with aspirin, anti-hypertensives and lipid lowering medications. However, the disadvantages of these charts are that they underestimate the risk in some ethnic groups and also in diabetics, patients with renal failure and inherited dyslipidaemias.

(See the joint British societies' risk prediction chart: http://www.bhsoc.org/Cardiovascular_Risk_Prediction_Chart.stm.)

- Low risk patients (green area) = calculated cardiovascular risk <10% over the next ten years.
- Intermediate risk patients (orange area) = cardiovascular risk between 10–20% in the next ten years.
- High risk patients (red area) = cardiovascular risk over 20% in the next ten years. These patients should be targeted and treated to prevent disease progression.

1.10 Examination of a Patient

Introduce yourself to the patient, explain what you would like to do and gain their permission. By shaking the patient's hand, you are not only being friendly, but also have the opportunity to assess his/her peripheral circulation.

Remember to be systematic in your approach to the patient:

general → hands → BP → neck → face → chest → oedema → finish

Adequately expose the patient's chest and position the patient at 45 degrees.

1.10.1 *General examination*

Stand at the end of the bed and briefly observe the patient's general condition. Does the patient look well or do they look unwell? Are there any devices by the bedside that would give you clues such as inhalers, cardiac monitor, external pacing, or walking aids? Does the patient look in pain or in respiratory distress? Does the patient have any dysmorphic features that may allude to an underlying congenital heart disease, for example:

- **Down's syndrome** — look for upslanting palpebral fissures, bilateral epicanthal folds (almond shaped eyes), protruding tongue and low set ears.

- **Marfan's syndrome** — tall stature, abnormally long and slender limbs (arachnodactyly).
- **Turner's syndrome** — short stature, webbing of the neck, low set ears.

Mechanical prosthetic valves can sometimes be heard from the end of the bed!

As part of your general examination, 'without touching the patient, examine the chest in a swift and focused manner for:

- Scars and external devices
 - A midline sternotomy scar could indicate a previous coronary artery bypass graft (CABG) or valve replacement.
 - A lateral thoracotomy scar could indicate mitral valve surgery.
 - A subclavicular scar and bump under the skin could indicate pacemaker/automated implantable cardiac defibrillator (AICD).
 - Vein harvesting scars on legs and radial artery harvesting scars could be a sign of bypass surgery.

With the introduction of minimally invasive interventional techniques, patients who have had angiography or other interventions may only have a small scar in the groin crease (usually the right) which you are unlikely to see. Antecubital fossa brachial artery scars are more likely to be observed.

1.10.2 *Hands*

Take your patient's hands. Warm hands suggest adequate perfusion (unless the patient is pyrexial). Start by looking at the fingertips.

Look for *clubbing* which can be described in four stages:

1. Increased fluctuancy of nail bed
2. Loss of nail bed angle
3. Increased longitudinal curvature of nail
4. Drum stick appearance of the nail caused by expansion of the terminal phalanx.

To detect clubbing, ask the patient to hold the nails of both index fingers, facing each other. In the absence of clubbing, a diamond-shaped space can be seen, caused by the angulation of both nail beds. In patients with clubbing, the diamond shape is obliterated.

The cardiac causes of clubbing can be remembered by **ABC**:

- **A**trial myxoma (rare)
- **B**acterial endocarditis (subacute)
- **C**yanotic congenital heart disease.

However, you would be expected to know a few non-cardiac causes of clubbing. Use the following mnemonic to help you remember both cardiac and non-cardiac causes:

- congenital *Cyanotic* heart disease
- **L**ung abscess, fibrosis
- **U**lcerative colitis/Crohn's disease
- **B**iliary cirrhosis
- **B**ronchiectasis
- **I**nfective endocarditis
- **N**eoplastic disease, for example, lung cancer and Hodgkin's disease
- **G**astrointestinal malabsorption.

Look closely at the nails also for signs of bacterial endocarditis characterised by splinter haemorrhages (which can also occur due to trauma, for example, in manual labourers), Janeway lesions and Osler nodes (see section on *Endocarditis in Chapter 2: Bedside teaching*). A simple thing to comment on (which shows you are observant) is tar staining on the fingers, as smoking is a risk factor for cardiovascular disease.

Look for hypercholesterolaemic deposits in the skin as yellow nodules known as tendon and palmar xanthomas.

1.10.3 *Radial pulse*

Feel for the pulse for at least 15 seconds and comment on the rate and rhythm. Is it slow (bradycardic) or fast (tachycardic), regular or

irregular? A congenital condition known as coarctation of the aorta is where there is a narrowing somewhere along the descending aorta. This manifests as radial-radial delay or radial-femoral delay which can be detected by feeling pulses in two places simultaneously. Most aortic coarctations are distal to both subclavian arteries so only the radio-femoral delay is abnormal.

1.10.4 *Blood pressure*

Moving up the arm in your examination ensures you don't miss the blood pressure. Comment on it, if it's available, otherwise say you would like to measure it. (See the *Appendix* on how to measure blood pressure for tips.) The pulse pressure is the difference between the systolic and diastolic pressures. The pulse pressure is typically wide in aortic regurgitation and although many textbooks say it is narrow in aortic stenosis, this is simply not the case and patients with aortic stenosis often have normal pulse pressures. There is also a phenomenon known as **pulsus paradoxus** where there is a drop in systolic blood pressure during inspiration of 10 mmHg. It is something you might get asked about on ward rounds as it is associated with pericardial constriction, tamponade and severe asthma. Look also for **pulsus alternans**, the alternating of strong and weak beats sometimes seen in severe left ventricular systolic impairment. Comment on any postural blood pressure changes. A postural drop in blood pressure is defined as a drop in systolic BP of >15 mmHg or a diastolic drop of >10 mmHg after a patient stands from lying down.

1.10.5 *Neck*

There are two important structures in the neck in the cardiovascular examination, the jugular venous pulse (JVP) and the carotid pulse. The internal jugular vein (IJV) gives an indirect measure of the pressure in the right atrium (RA) and provides some information about cardiac function — this is because there are no valves between the RA and IJV. The IJV enters the neck just behind the mastoid process, passes deep to the sternocleidomastoid muscle (SCM) and then runs

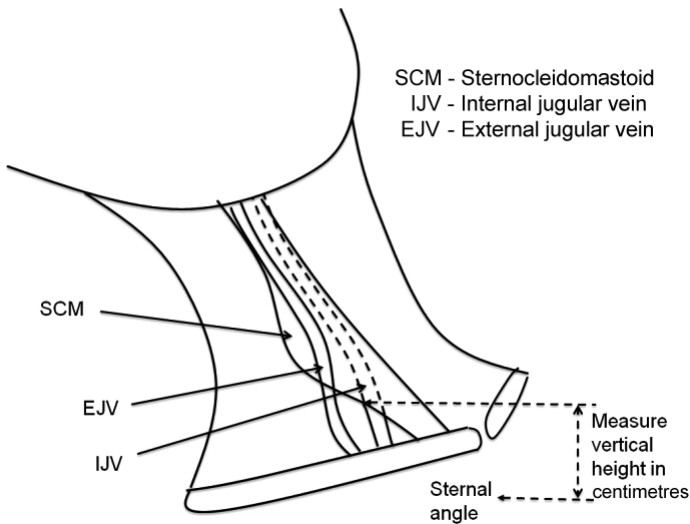


Figure 1.2 Measuring the JVP

between the sternal and clavicular heads of the SCM before entering the thorax. The IJV itself is not visible.

To measure the JVP, sit the patient at a 45 degree angle, with the neck muscles relaxed and head turned slightly to the left. Look for diffuse pulsations. The JVP is the vertical height of the pulse in the IJV above the sternal angle. The normal JVP is <4 cm.

A common question you are likely to encounter is how you would differentiate between the JVP and the carotid pulse.

The six key features to distinguish JVP from carotid artery:

The JVP:

- 1) pulsation is easily obliterated by finger pressure
- 2) has two pulsations for each single arterial pulse
- 3) is much weaker in force (impalpable)
- 4) varies with respiration
- 5) varies with position
- 6) rises transiently with pressure on the liver (hepatojugular reflux) or on the abdomen (abdominojugular reflux).

Two important things to note about the JVP are the **height** and **waveform**.

A *raised JVP* can be a sign of:

- fluid overload
- right-sided heart failure
- SVC obstruction
- constrictive pericarditis.

In constrictive pericarditis, an elevated JVP is characteristically associated with a paradoxical rise in inspiration — known as **Kussmaul's sign**.

To understand waveform abnormalities, it is important to understand the actual waveform. The waveform corresponds to the changes in right atrial pressure. There are two peaks and two descents.

The normal JVP goes down in systole (x descent). Systole starts with the brief c wave and then proceeds with the x descent as the right atrial floor moves down as a result of right ventricular contraction. Late in systole, the veins start to fill the atrium faster than its capacity is being increased by ventricular contraction and so there is a passive accommodation of blood and therefore increase in pressure which corresponds to the v wave.

The abnormalities can be divided broadly into a wave and v wave abnormalities.

'A wave' abnormalities

An *absent a wave* (and so the JVP rate is similar to the pulse rate) indicates no atrial contraction and occurs in atrial fibrillation. A *large a wave* is hard to diagnose clinically but in an exam you need to understand

To Impress!

Positive hepatojugular reflux

sign is not simply the elevation of the JVP on hepatic compression as this occurs in everyone but rather that the JVP **remains** elevated for a 15 second compression. This is because the RV is unable to pump out the increased venous return — a sign of RV heart failure.

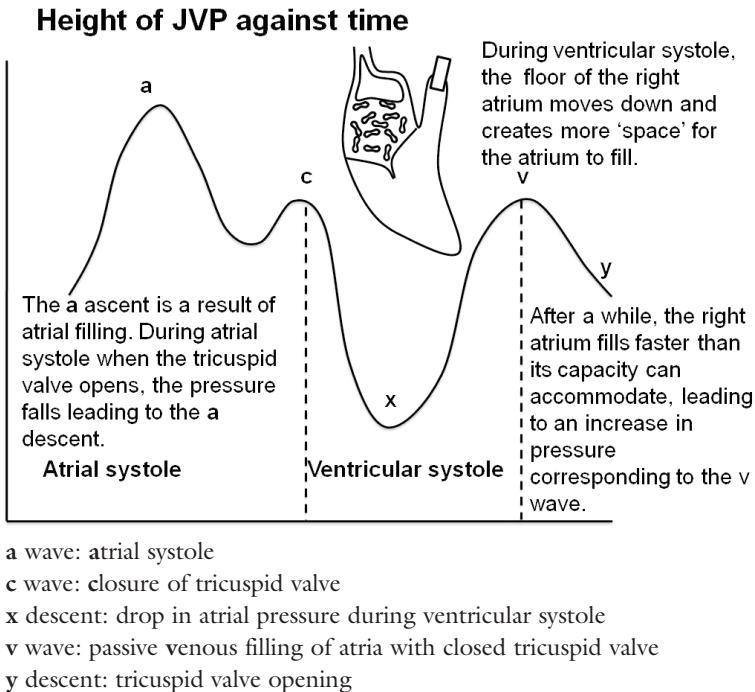


Figure 1.3 Height of JVP against time

that this occurs when the right atrium is contracting against resistance, that is, in pulmonary hypertension and pulmonary stenosis. *Cannon 'a' waves* are caused by a right atrium contracting intermittently against a closed tricuspid valve, which occurs in complete heart block.

V wave abnormalities

In tricuspid regurgitation, right ventricular contraction does not only pull down the floor of the right atrium, but also ejects a lot of blood into the right atrium, hence the JVP goes up. This is **not** passive venous filling, so it is not a large **v** wave. In fact, it starts at the **c** wave and continues to the end of the **v** wave. Its proper name is a giant *CV* wave, and can be thought of as an upside down **x** descent. The JVP

may be so high in the neck that it can only be seen by looking behind the ear — a large v wave can cause the ears to waggle!

In practice, it is difficult to pick up a JVP waveform abnormality, and valvular heart disease is rarely diagnosed on the basis of an abnormal JVP waveform. However, it is important to recognise whether the JVP is raised or not, and to know what conditions are associated with the different waveform abnormalities both for written and clinical examinations. Tricuspid regurgitation and atrial fibrillation are two common examination cases. In addition, there is some evidence that an elevated JVP in patients with heart failure is associated with an increased risk of hospital admission, death and subsequent hospitalisation for heart failure.

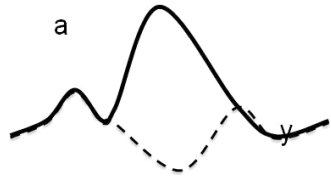


Figure 1.4 Giant CV wave

The carotid pulse

The carotid artery can be found by placing the thumb gently on the trachea and sliding the thumb laterally until it hits the sternocleidomastoid where the carotid pulse should be palpable. Four things should be noted about the pulse — the *rate*, *rhythm*, *volume* and *character*. The rate and rhythm should already have been assessed when examining the radial pulse. The volume and character should be assessed at the carotid rather than the radial as it is closer to the heart.

The volume may be:

- **low** — in shock and heart failure, or
- **large** — in aortic regurgitation, vasodilatation (exercise, fever).

There are several distinct character pulses:

- **slow rising** occurs in aortic stenosis
- **collapsing pulse** occurs in regurgitation
- **bisferens pulse**, where two distinct systolic peaks are present, characteristic of aortic regurgitation and stenosis.

Don't forget to listen for carotid bruits — ask patients to hold their breath whilst simultaneously listening with the bell of the stethoscope (it's a good idea to hold your breath with the patient as it reminds you how long you're asking them to do this). The presence of carotid bruits can suggest local disease or radiation from elsewhere, that is, in aortic stenosis. Ask yourself whether the patient has any other clinical features of peripheral arterial disease and remember in an exam situation to at least offer to palpate peripheral pulses at the end of the examination.

1.10.6 *Face*

Start from top down — look at the eyes for evidence of cataracts, which can be a result of diabetes or hypertensive retinopathy. A pale conjunctiva is indicative of anaemia. Pallor with jaundice suggests haemolytic anaemia. Lid lag and exophthalmos indicates thyroid disease. Look for the presence of corneal arcus, a grey opaque line surrounding the cornea and xanthelasmata which are yellow fatty deposits commonly found around the eyes. Both of these signs are associated with raised cholesterol levels.

Jaundice can signify haemolysis which can result as a complication of prosthetic valves. Central cyanosis on the tongue can indicate congenital heart disease.

1.10.7 *Praecordium*

Examine for scars particularly at the apex where they can be easily missed. Feel the position of the apex by placing the palm of your right hand over the left chest wall: is it displaced? If so, where — for example, laterally? Downwards? Be specific. The normal position of the apex is in the fifth intercostals space, mid-clavicular line.

Top tip: When you know the apex beat is abnormal but do not know if it is heaving or thrusting say '*forceful*'.

Abnormal characteristics of the apex beat can be described as 1) heaving, which occurs in volume

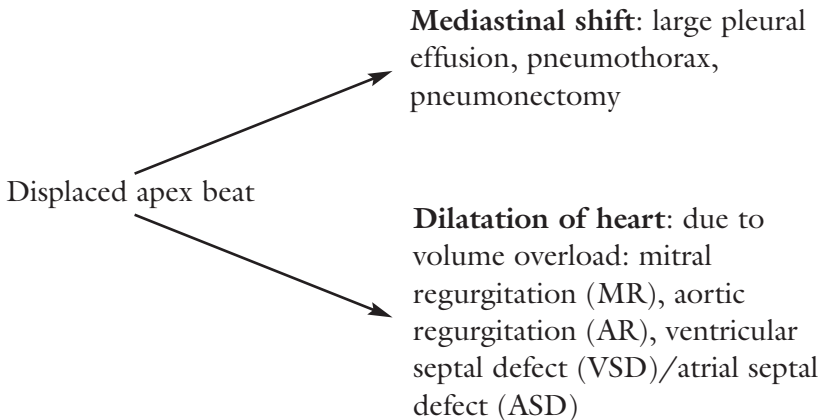
overloaded conditions like mitral and aortic regurgitation; 2) thrusting, which occurs in pressure overloaded conditions like aortic stenosis.

Volume overload — conditions: mitral or aortic regurgitation
 — leads to dilatation of the LV → displacement of the apex
 — **detectable on Chest X-ray (CXR) if severe**

Pressure overload — conditions: aortic stenosis, hypertension
 — leads to concentric hypertrophy of the LV → thicker LV wall (hypertrophy)
 — **detectable on ECG** (sometimes).

Only echocardiography is truly reliable for detecting these.

If the apex is displaced, this is due to either mediastinal shift or dilatation of the heart:



To feel for thrills, keep your hand over the apex and then palpate over both sides of the sternum. *A thrill is a palpable murmur — it feels like a purring cat.* Now feel for a right ventricular (RV) heave by placing your hand in the left parasternal position. A RV heave is a palpable

beat and suggests RV enlargement, which can occur in cor pulmonale or pulmonary stenosis.

Cor pulmonale is right ventricular failure secondary to chronic pulmonary hypertension which can be a result of lung disease, pulmonary vascular disorders, neuromuscular and skeletal diseases.

Stenosis of the **pulmonary artery** causes increased pressure in the right ventricle and leads to hypertrophy.

1.10.8 *Auscultation of the heart*

Most stethoscopes have a bell and diaphragm. When auscultating, you are listening for:

- normal heart sounds
- added heart sounds, that is, murmurs (due to turbulent blood flow) or a third or fourth heart sound.

The **bell** is best used for low pitched sounds:

- diastolic murmurs, for example, mitral stenosis
- third and fourth heart sounds.

The **diaphragm** is better for detecting higher pitched sounds:

- normal heart sounds
- systolic murmurs
- aortic regurgitation.

With the patient sitting at 45 degrees first listen at the apex, tricuspid, pulmonary and aortic areas with the bell and then again with the diaphragm. Place your thumb on the patient's carotid pulse whilst auscultating the heart sounds — this helps to differentiate the first (S1) and second (S2) heart sounds and any murmurs.

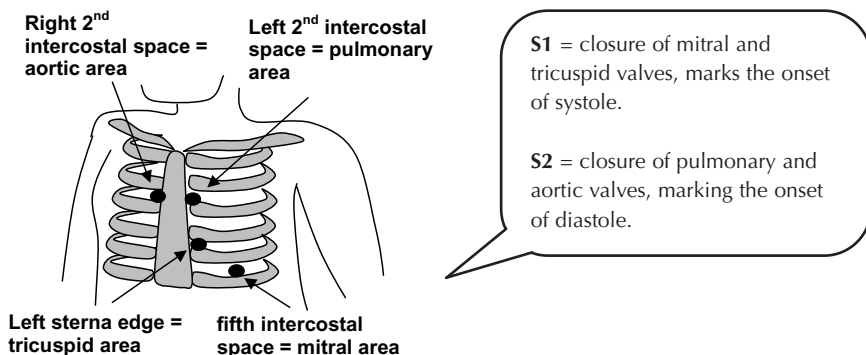


Figure 1.5 Key areas of auscultation

Splitting of the second heart sound:

S2 has two components: A2 (closure of aortic valve) and P2 (closure of pulmonary valve). In most people *S2* is heard as one sound. However, it is normal to hear a split *S2* on inspiration in the young.

Why is this? — On inspiration, intrathoracic pressures drop which leads to an increase in venous return to the right heart. This in turn leads to a delay in right heart emptying and so the pulmonary valve remains open for longer, closing later (after the aortic valve).

Abnormal splitting:

Wide 'fixed' splitting in atrial septal defect (ASD): *S2* remains split in inspiration and expiration.

Wide 'physiological' splitting in right bundle branch block (RBBB): *S2* is split in expiration, but more so in inspiration.

Reverse splitting in left bundle branch block (LBBB): *S2* is split in expiration and not inspiration.

Roll the patient onto the left side and listen at the apex for mitral murmurs and then in the axilla for any radiation of the murmur. Sit the patient up and leaning forwards. This time listen in the aortic and tricuspid areas with the diaphragm. If a murmur is heard, determine the timing (that is, systolic or diastolic). Ask the patient to take a deep

breath in and hold their breath. Then get the patient to take a deep breath in, out and hold their breath at the end of the expiration. Right-sided murmurs are louder on inspiration and left-sided murmurs louder on expiration — **RILE**. For a more detailed description on murmurs see Chapter 2 *Bedside Teaching*. Listen to the bases of the lungs for bilateral crepitations (as in pulmonary oedema) or reduced air entry and dullness (pleural effusions). Feel for sacral oedema.

To complete examination:

- **Check the abdomen for:** an enlarged liver (right-heart failure), a pulsatile liver edge occurs in tricuspid regurgitation or midline pulsatile mass (aortic aneurysm).
- **Check the legs and sacrum** for pitting oedema: use a finger to gently but firmly push against a bony surface and look to see if the indentation remains.
- **Dip the urine for haematuria** (in infective endocarditis), glucose (diabetes — a risk factor for cardiovascular disease).

1.11 Worked Example

Below is a worked example to highlight the key points in clerking and presenting a patient complaining of chest pain:

Student: Good morning. Can I check that I have come to the right person? What is your name?

Patient: My name is Mr. R.

Student: My name is X. I'm one of the student doctors and I would like to ask you some questions. Is that alright?

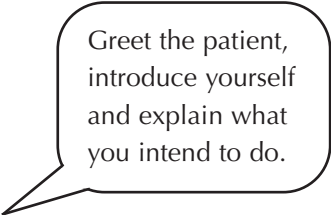
Patient: Yes, that's fine.

Student: How old are you?

Patient: 72.

Student: Are you retired now?

Patient: Yes. Six years now. I used to be an electrician.



Greet the patient, introduce yourself and explain what you intend to do.

Step 1 Presenting Complaint

Student: What brought you into hospital?

Patient: Well, I just wasn't feeling right yesterday.

Student: In what way?

Patient: I had this discomfort across my chest.

Student: Where was this discomfort on your chest?

Patient: Here on the left side.

Student: Did it spread anywhere else?

Patient: Not really, but I did start to get a heaviness in my left hand.

Student: Can you describe the chest discomfort?

Patient: It was like a pressure — like someone sitting on my chest.

Student: When did it start?

Patient: Probably about 1pm. I was walking the dog and had just reached the hill.

Student: What did you do then?

Patient: I carried on walking for a few minutes but the pain became unbearable, so I stopped and sat on one of the park benches.

Student: Did this help?

Patient: A little. It definitely helped me get my breath back.

Student: Were you feeling short of breath at the time?

Patient: Yes. That's strange for me — I never get short of breath when I'm walking the dog.

Student: Going back to the chest discomfort, at its worst, how severe was the pain on a scale of one to ten, where ten is the worst discomfort that you've ever felt?

Patient: I'd say about nine.

Student: What did you do after sitting on the bench?

Use open-ended questions and avoid too many leading questions.

Use of **SOCRATES** questions.

Patient: Well after ten minutes, the pain was still there so I got up and went back home. My wife said I looked really pale and called the ambulance.

Student: Did you feel clammy at all?

Patient: Yes and nauseous.

Student: Did you actually vomit?

Patient: No.

Student: Any dizziness?

Patient: No.

Student: Did you blackout at any point?

Patient: No.

Student: Did you feel your heart fluttering in your chest?

Patient: No.

Student: Do you ever get short of breath on lying flat?

Patient: No.

Student: How many pillows do you sleep with?

Patient: Only the one.

Student: Do you ever find that your ankles become puffy or swollen?

Patient: No, but I do often get these pains in my ankles and knees. My GP said it was probably arthritis. I hope I don't need an operation. My wife had to have her knees replaced last year.

Student: Ok, we will talk about the arthritis a bit later. Can I just clarify a few things... Going back to the chest discomfort — did anything make it better?

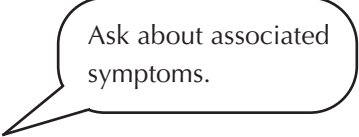
Patient: Not really. It only went away after I reached the hospital.

Student: Were you given anything at the hospital or by the paramedics that helped ease the pain?

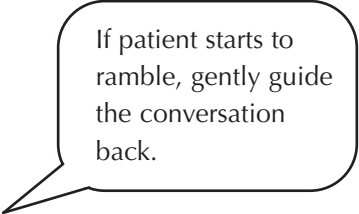
Patient: Oh yes — the oxygen really helped, but I also had a tablet and a spray under my tongue.

Student: How long did you have the pain for in total?

Patient: About an hour or so.



Ask about associated symptoms.



If patient starts to ramble, gently guide the conversation back.

Step 2 Cardiac Risk Factors

Student: Have you ever had this kind of discomfort before or any chest pain in the past?

Patient: No, not at all.

Student: Have you ever had a heart attack?

Patient: No, but my father has.

Student: How old was he when he had the heart attack?

Patient: He was 59 I think.

Student: Do you have **diabetes**?

Patient: No.

Student: Do you have **high blood pressure**?

Patient: Not anymore. I'm on tablets.

Student: Do you **smoke**?

Patient: No, never have done.

Student: What about your **cholesterol**?

Patient: I think my cholesterol is fine doctor.

Risk factors for cardiovascular disease.

Here's where you discover your patient who previously told you he didn't have high cholesterol does not have high cholesterol because he is being controlled with a statin!

Step 3 Drug History

Student: Are you on any medication?

Patient: Yes, I take a cholesterol tablet once a night and a water tablet.

Step 4 Past Medical History

Student: Have you ever been admitted to hospital before?

Patient: No, I've never been sick in my life.

Student: Have you ever had any operations?

Patient: No.

Student: Do you have any medical problems?

Patient: No.

Step 5 Social History

Student: Who do you live with?

Patient: I live with my wife.

Student: Do you live in a house? Flat?

Patient: In a house.

Student: Do you manage the stairs ok?

Patient: Yes.

Student: Do you drink any alcohol?

Patient: Yes, 2–3 pints only on Sundays.

Student: Have you ever tried recreational drugs? Such as cocaine.

Patient: Oh no, I would never touch those things.

1.12 Presenting Patients

When presenting patients, for example on a ward round, it is important to be logical and coherent. Some consultants may have specific ways in which they wish to have the information presented, but on the whole the following can be used.

1.13 Summarising Your History

In the first opening statement you should include:

- name
- age
- occupation
- sex
- *brief* presenting complaint (in patient's own words)
- any previous cardiac disease
- any cardiac risk factors.

When presenting the history, it is important to mention the relevant *positive and negative findings*. For example, in a patient in whom you suspect congestive cardiac failure you must mention whether he/she reports any orthopnoea, paroxysmal nocturnal dyspnoea (PND), or ankle swelling, and what their exercise tolerance is.

1.13.1 *An example of a presentation based on the previous history*

Student. Mr R is a 72-year old retired electrician who presented to the Emergency Department yesterday complaining of ‘chest discomfort’. He has no previous history of ischaemic heart disease but his cardiac risk factors are hypertension, hypercholesterolaemia and a positive family history.

The chest pain occurred on exertion, whilst the patient was walking his dog, was not relieved by rest and lasted a total of one hour. It was located on the left of the chest and was radiating to the left arm. Associated symptoms were nausea, dyspnoea and feeling clammy. There were no palpitations, loss of consciousness or dizziness, orthopnoea or ankle swelling. With regards to cardiac risk factors he has hypertension and high cholesterol, for which he is being treated and his father suffered a myocardial infarction aged 59 years. He has not had a previous myocardial infarction, denies ischaemic heart disease and is not diabetic. He is a non-smoker, lives with his wife and is independent of his activities of daily living.