

CHAPTER 1

The Cardiovascular History and Physical Examination

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Summary

A cardiovascular history and examination are fundamental to accurate diagnosis and the subsequent delivery of appropriate care for an individual patient. Time spent on a thorough history and examination is rarely wasted and goes beyond the gathering of basic clinical information as it is also an opportunity to put the patient at ease and build confidence in the physician's ability to provide a holistic and confidential approach to their care. This chapter covers the basics of history taking and physical examination of the cardiology patient but then takes it to a higher level by trying to analyse the strengths and weaknesses of individual signs in clinical examination and to put them into the context of common clinical scenarios. In an ideal world there would always be time for a full clinical history and examination, but clinical urgency may dictate that this is impossible or indeed, when time critical treatment needs to be delivered, it may be inappropriate. This chapter provides an insight into delivering a tailored approach in certain, common clinical situations. Skills in clinical history and examination evolve with time and experience and this chapter provides a structured approach to clinical cardiovascular history and examination which should be seen as a framework on which to build clinical experience. It also provides a hierarchical approach to the importance of certain symptoms and signs in a variety of cardiovascular conditions. Clinical history and examination has changed with modern advances in cardiology and the development of sophisticated imaging techniques. Eponymous signs beloved of the 'Old Masters of Examination' are now of historical interest but are listed in → Table 1.20 for information.

Introduction

An accurate history and a careful examination are probably the least expensive and the fastest and most powerful of the tools available to a physician [1]. Their effective use requires skill and experience. It is important to recognize that it is unusual to obtain the classic history of a condition combined with all the classic physical signs. Variations in history and difficulty in eliciting physical signs need to be understood. It is always a serious mistake to massage the history and the physical signs to fit a particular diagnosis.

History

Introduction

There needs to be an awareness of potential difficulties in obtaining a clear history [2]. Patients usually, but not always, try to tell the truth, as they perceive it, to their physician who will develop an instinct for this, and must always consider whether they are misinterpreting the information given to them by the patient.

The interaction between the patient and doctor is complex and determined by many factors (↻ Table 1.1). Patients with a moderate rather than good command of the language may have difficulty in expressing subtle aspects of their story and may agree to suggestions made to them by the doctor, rather than admitting they do not completely understand what is being said to them. Many patients have a poor perception of parameters such as time and distance, which are frequently important in the cardiovascular

Table 1.1 History taking: factors that may lead to problems

Patient-based factors
Command of language (usually patient)/history via an interpreter
Patient's cultural background
Education and employment
Inaccurate perception of variables, e.g. time and distance
Underplaying/exaggeration
Responses altered by:
Alcohol
Recreational drugs
Physician-based factors
Badly structured questions
Assumptions about patient's medical knowledge
Lack of patience
Asking 'leading questions', i.e. suggesting the required answer

history, and rather than admit they cannot answer the question put to them will sometimes make up an answer simply to please the doctor. The way the patient presents a history will also depend on the level of their education, and the type of work that they do [3]. The doctor taking a history must not make assumptions about the patient's underlying medical knowledge. There is a tendency among doctors to slip into medical jargon which is poorly understood or completely incomprehensible to the patient. A good example is that some patients do not understand the difference between a myocardial infarction and a cerebrovascular accident and the term 'stroke' may confuse them as they are not quite sure about what it is. Similarly the patient may describe either a cardiac arrest or a myocardial infarction as 'heart attack'. This problem can sometimes be avoided if it is clear that the patient is becoming confused by asking the patient how the particular illness affected them rather than by giving it a name. The same applies to the description of symptoms. For example, a patient may use the term 'palpitation' without really understanding what it means. This aspect of history taking is discussed in more detail in the sections dealing with specific symptoms.

Particular difficulties can arise when the history is taken via an interpreter and under these circumstances much more time must be put aside and strenuous efforts made to try to establish that the doctor is being given a clear account by the patient rather than by the interpreter. There is a strong tendency for the interpreter to make up their own version of the history and deliver it to the doctor rather than asking the patient exactly what the symptoms are and describing them in detail. This may be prominent when there are major cultural differences between the doctor and the patient (and interpreter). In some situations, particularly where women do not speak the language and their husband acts as an interpreter, there may be very considerable interpretation of the history rather than the language. It may be impossible to overcome these problems, but it is important that the doctor is aware of the difficulties and how they may obscure the transmission of information (see ↻ Key point 1, p. 26).

The reaction of a patient to their symptoms is often complex [4]. Florid descriptions of symptoms (e.g. 'I had seven heart attacks' when in fact there had been one myocardial infarct and some episodes of angina) may become important to the patient, particularly if the patient is validated by their illness. In addition, it becomes real to the patient with repeated re-telling of the history. Conversely, in emergency situations, particularly with young patients, there may be a major downplaying of symptoms particularly

Box 1.1 Scenario 1: downplaying symptoms—the patient who wants to go home

A 45-year-old Asian male who smokes 20 cigarettes a day arrives in the emergency department at 02.00 hours. There is a history of a heavy feeling in the central and lower chest present for 45min, but easing off before the patient arrived. The patient admits to two or three alcoholic drinks and a spicy meal at 21.00 hours the previous evening. He now maintains that his problem is ‘just indigestion’, but the electrocardiogram (ECG) shows some minor ST/T wave changes. Despite the protestations of the patient, the chances of an acute coronary syndrome are high and this must be excluded.

if the patient is keen to leave the emergency room and go home (➔ Box 1.1). This is particularly common in young and middle-aged males who are presenting for the first time with ischaemic chest pain. Generally patients do not present as emergencies unless they or their spouse, partner, or family have been alarmed by the event which has caused them to come to hospital (see ➔ Key points 2 and 3, p.26). Doctors should be extremely cautious when confronted with the patient who has arrived in the A&E department in the early hours of the morning, who does not have a lengthy past medical history, and then begins to downplay their symptoms. These patients are usually ill.

The doctor has to navigate this complex situation to obtain an accurate history and nowadays may also have the added complication of the patient having already made up their mind about aspects of their illness having consulted the Internet and often arriving in the clinic clutching printouts from their computer. It is important to allow the patient to talk rather than putting words in their mouth, but then the use of judicious direct questioning is needed to refine the history.

It can be helpful at the end of the process for the doctor to summarize the history and repeat it back to the patient and ask them whether they agree. There is always the difficulty in this situation that the patient will agree, simply because they don’t want to upset the doctor, but when done carefully this process can be an extremely powerful tool for finalizing the history and eradicating errors due to misunderstanding.

The basic cardiovascular history

On rare occasions, cardiovascular disease can lead to almost any symptom; however, the most common are listed in ➔ Table 1.2. The basic principles for analysing each

Table 1.2 Cardiovascular history

Past and family history
Risk factors
Employment: may be affected by the presence of cardiac problems, e.g. professional drivers, pilots, divers etc.
Chest pain ± radiation
Shortness of breath/cough
Palpitations: awareness of irregular heartbeat
‘Dizziness’ and unsteadiness
Syncope and falls
Fatigue
Ankle oedema (a symptom and a sign)
Less common symptoms:
Abdominal pain
Vomiting with acute MI
Polyuria associated with tachycardia
Pulsating in the neck associated with tachycardia and with tricuspid regurgitation
Abdominal swelling—ascites
Weakness of legs
Back pain

symptom remains the same, but it is extremely important that a systematic approach is always taken (➔ Table 1.3). Patients may have inaccurate perceptions of time and distance (➔ Boxes 1.2 and 1.3). If a patient has a recurrence of a particular cardiovascular condition their symptoms are usually very similar on each occasion. For example, the patient who in the past experienced most of their ischaemic pain in their arm will tend to do so on another occasion, but beware, this is not always the case. One must always consider that a previous incident ascribed by the patient to the same situation was in fact different and also the diagnosis they were given may have been incorrect. Finally, a history may be so characteristic that a strong provisional diagnosis can be made over the phone (➔ Box 1.4).

Table 1.3 Always establish some basics for all symptoms

Nature and severity of symptom
Duration of symptom:
When did it start?
How long did it last?
How often does it occur?
Precipitating and relieving factors
Similarity to previous incidents
Impact on daily life and job

Box 1.2 Scenario 2: time perception

The patient gives a history that sounds typical of angina, but states the pain lasts for 20min after they stop exercising, therefore making a clinical diagnosis of angina unlikely. Is this really long lasting pain or does the patient have an inaccurate perception of time?

Solution

Ask the patient, who has only been in your office for < 5min, ‘does the pain last as long as you have been in this room?’—the patient will often realise that they have mistakenly over-emphasized the amount of time and actually the pain lasts less time than they have been in the room. They may then revise their answer

Chest pain (↔ Table 1.4)

Because of the prevalence and importance of coronary disease (↔ Chapters 16 and 17) inevitably there is an emphasis on determining whether or not chest pain is likely to have an ischaemic origin (↔ Table 1.5). Cardiac chest pain tends to be described by doctors as being typical or atypical; the term typical referring to whether it is typical of pain usually associated with myocardial ischaemia. The nomenclature of ischaemic chest pain can be slightly confusing. Ischaemic chest pain has similar characteristics but may vary in intensity. In general, chest pain associated with myocardial infarction is the most severe pain, followed by pain associated with acute coronary syndromes (ACSs), and then pain only occurring with a precipitating factor. Ischaemic chest pain induced by precipitating factors is usually referred to as angina while pain which tends to be constant, coming on spontaneously, and often associated with an ACS, is simply referred to as ischaemic chest pain.

Typical anginal chest pain (↔ Tables 1.5 and 1.6) nearly always indicates myocardial ischaemia and in the vast majority of cases this symptom occurs because of coronary

Box 1.3 Scenario 3: distance perception

The patient cannot remember how far they walk before the symptoms start

Solution

Ask the patient if he or she managed to walk from the hospital car park to your clinic without stopping or take some similar example within the route they would have taken to reach your clinic.

Box 1.4 Scenario 4: using the history to be prepared for rapid action

A 65-year-old man with treated hypertension but otherwise well, returned to his car with grocery shopping. As he leant forward to put the shopping bag in to the car he felt an extremely severe burning pain in the centre of the back of his chest and fell to the ground. The pain was so severe and intense he felt that he had somehow been stabbed by a hot object from behind. An ambulance was called who found him lying on the ground, looking extremely pale and in pain but fully conscious. This history was ‘phoned to the hospital by the incoming ambulance. On the basis of this story the duty cardiologist suspected a dissecting aneurysm and mobilized the cardiac surgeons. A transoesophageal echocardiogram was performed immediately he arrived in the emergency department which confirmed a type A dissecting aneurysm and he was transferred immediately to the operating theatre. He was on the operating table within 90min of the incident occurring. The surgery was successful and the patient was discharged home 10 days later.

artery disease. It can, however, occur in patients with normal coronary arteries as a result of aortic stenosis (↔ Chapter 21) (and, occasionally, aortic regurgitation) because the increased amount of cardiac muscle makes oxygen demands that exceed the amount of oxygen that can be delivered even by the large, normal coronary arteries in many of these patients. Occasionally, anginal chest pain may be prominent in patients in whom there is a right-sided cardiac problem, such as in patients with severe pulmonary hypertension (↔ Chapter 24), e.g. primary pulmonary hypertension, Eisenmenger’s syndrome (↔ Chapter 10), and severe mitral stenosis (↔ Chapter 21) in whom blood which may have reduced oxygen content flows down the coronary arteries

Table 1.4 Types of chest pain

Cardiac
Angina (coronary disease)
Pericarditis
Aortic aneurysm
Non-cardiac
Pleuritic
Musculoskeletal
Gastrointestinal—particularly, oesophageal
Other

Table 1.5 Ischaemic chest pain

Location:
Central or slightly to left of centre
May be experienced anywhere between the pubis and the top of the head. Pain may be only present in the areas of radiation and not in the chest
Nature: dull, pressing, constricting (for example 'elephant sitting on chest', 'strangling')
Occasionally described as either burning or stabbing
Body language: patient may indicate that the pain has a constricting nature or may hold a clenched fist in front of their chest. This is remarkably common and indicates the constricting nature of the discomfort.
Radiation of the pain:
Common—left arm, neck, jaw
Less common—right arm (or both), back, abdomen, teeth

to a hypertrophied right ventricle (RV) which has increased demands because of the high pressure that it is generating and increased muscle to supply (RV hypertrophy). A more extreme example of this phenomenon is when there is acute right heart strain in acute massive pulmonary embolism. The combination of severe arterial desaturation and increasing RV work may lead to RV ischaemia and anginal chest pain.

Anginal chest pain rarely occurs without a precipitating factor (⇒ Table 1.6). Chest pain that comes on when the patient is feeling well, unstressed, and is at rest is unlikely to be cardiac chest pain. The exception is when this is the first

Table 1.6 Precipitating factors of ischaemic chest pain

None
Onset of acute coronary syndrome
Coronary spasm
Underlying arrhythmia
Exercise
Mental stress (angina may be more prolonged than with exercise):
Anxiety
Anger
Stress-induced left ventricular dysfunction ('takotsubo')
Occurs on lying down (angina decubitus) often a sign of instability
Angina occurs more easily than usual:
After meals because of blood flow needed to the gastrointestinal tract
Extremely hot or cold weather
Recent onset of anaemia
Thyrotoxicosis
Hypoxia from some other cause, e.g. onset of heart failure.

episode of an ACS (⇒ Chapter 16) which would not need a precipitating factor, or if there is some underlying factor which is not unveiled by the patient's history. The two most likely circumstances are either a cardiac arrhythmia which has not produced any awareness of palpitations or change in level of consciousness, but increases cardiac work, and coronary artery spasm, although quite rare in the European population, which leads to attacks of typical anginal pain without any obvious precipitating factor. The most common precipitating factors for ischaemic chest pain are presented in ⇒ Table 1.6. In most situations where anginal chest pain is precipitated by increased cardiac work the pain will go away 5min or less after the precipitating factor ceases.

It is important to question the patient about the offset of pain as well as the onset of pain. Truly prolonged chest pain, for example after exercise, is usually not angina except in the context of an ACS. A point which may be difficult to appreciate unless the history is taken extremely carefully is that the pain may actually be occurring after exercise rather than during exercise. A common example of this is the patient who carries out some physical work, for example carrying shopping bags, arrives home and then develops chest discomfort which is in fact musculoskeletal due to the chest wall stress produced by the effort of carrying the bags. Such pain often lasts considerably longer than the normal 5min seen with angina, despite the patient resting.

More prolonged pain usually indicates the possibility of an ACS or a non-cardiac cause. An exception to this is pain occurring when patients become angry or anxious. This often has a slow offset because the sensation of anger or anxiety may take a considerable period of time to abate. Angina may sometimes be precipitated by lying flat (angina decubitus); this usually indicates severe coronary disease and is caused by the increased venous return, generated by lying flat, increasing cardiac work. There may also be associated cardiac failure with hypoxia occurring due to incipient pulmonary oedema (⇒ Chapter 23) when this is made worse by the patient lying flat. Pain occurring when the patient lies flat must, however, be distinguished from oesophageal pain, usually due to reflux oesophagitis. This distinction is always difficult and often impossible on clinical grounds. If the pain is instantly relieved by antacids it is probably oesophageal. The same confidence cannot be attached to relief of pain by nitroglycerine (GTN). Immediate relief does favour ischaemic chest pain, but oesophageal pain, if associated with oesophageal spasm, is also relieved by GTN (and calcium-channel blockers) (⇒ Table 1.7).

Angina is often described by the patient as being uncomfortable rather than painful. The patient may simply say

Table 1.7 Response of symptoms to GTN

Anginal chest pain usually resolves with GTN within 1–5min. If response takes longer the diagnosis of angina should be questioned
GTN also relieves shortness of breath (and any associated chest constriction) due to left ventricular failure by reducing venous return
May relieve pain due to any kind of smooth muscle spasm, particularly oesophageal but occasionally gallstone colic
If the patient describes the response to GTN occurring after 5–10min then it is probable this is not a response to GTN but a spontaneous resolution of the pain

they have a discomfort in their chest which is not very painful but is unpleasant, and on occasions this discomfort may be described as breathlessness. The main reason is that the constricting nature of anginal symptoms makes the patient feel that they cannot take a deep breath. This difficulty in distinguishing breathlessness from angina by the history is very common. It may be impossible to make the distinction between angina and dyspnoea clinically, but prominent tightness and difficulty taking a breath because of tightness favours angina, whereas the sensation that they are panting as though they had just run somewhere rapidly favours dyspnoea. Finally, if resting lung function and left ventricular (LV) function on echo are normal, angina becomes much more likely.

One of the most difficult situations in patients presenting with acute chest pain is the patient in whom there is a statistically low chance of serious underlying disease [5]. The classical situation would be a young woman with what appears to be typical angina. Although the chances of anginal-type symptoms described by a woman in their 20s of being due to coronary disease are low, one has to accept there are a few female patients of this age with severe coronary disease and if their condition is ignored the consequences may be serious or even fatal. Therefore, although the chances of a positive diagnosis are low, the consequences of missing it in the individual are so serious that the situation has to be treated as though it is an ACS until proved otherwise.

Chest pain in acute coronary syndrome (also see Chapter 16)

Ischaemic chest pain associated with ACSs is similar to the pain which is frequently described as ‘angina’ but is usually, but not always, more intense, longer lasting, as well as occurring without an obvious precipitating factor. In addition to the severity of the pain the patient may give a history of sweating, nausea, faintness, and feeling systemically more unwell than they would with straightforward exercise-induced chest pain, i.e. angina. Vomiting is not uncommon in patients with myocardial infarction.

Musculoskeletal chest pain

Pain of musculoskeletal origin occurring from around any areas of the chest is often the most difficult condition to distinguish from ischaemic chest pain, and, as with oesophageal pain, clinical distinction may be impossible. Although it may be exercise induced, some distinction can be made in taking the history by defining the precipitating factors, the duration, and localization. Musculoskeletal chest pain may have no precipitating factors or may come on during or after exercise and a history of antecedent injury or exertion should be sought. It may be made worse by breathing or by movement. It is frequently of longer duration than ischaemic chest pain and often localized to small areas of the chest; the patient may well be able to point with one finger to the site of the pain when asked where the pain is, whereas the patient who has myocardial ischaemia generally cannot identify the site of the pain as precisely and it frequently occupies an area of at least several cm² and often much more. Although the history may definitely make the diagnosis of musculoskeletal pain, for example episodes of very sharp pain lasting a matter of seconds, localized to a small area between the ribs, are almost never cardiac in origin, unfortunately many episodes of chest pain of musculoskeletal origin are not easily distinguished from cardiac pain and in the end it may be necessary to depend on investigations to help distinguish the pain from ischaemic cardiac pain.

Physicians frequently palpate the chest, and apply pressure to try to identify whether the pain is made worse by these manoeuvres [6]. This can be extremely helpful if the pain produced in this way is typical, but it is an approach which can be fraught with danger. A robust young cardiologist may induce pain in the chest of a frail, elderly patient which they then have difficulty distinguishing from their normal pain. In circumstances where the pain may be ischaemic pain it is not safe to dismiss it on the basis that the doctor believes that they can reproduce the pain by pressing on the chest. Finally, pain of pleuritic or pericardial origins can be made worse by pressing on the chest as this deforms the structures beneath and creates pain.

Other non-cardiac pain

Patients suffering from anxiety and/or depression often are aware of chest tightness which may sound very like angina; this may be associated with hyperventilation. It tends to be prolonged over hours, have little or no association with exercise, and indeed may be more prominent when the patient is relaxing and also often does not limit exercise. Although the history can help, it may remain difficult to be sure whether

there is ischaemic heart disease. These patients often perform poorly on the treadmill and also find coronary angiography very stressful. Often stress imaging to look for ischaemia is the ideal first investigation to see if there is any evidence of ischaemia. (See ↻Key point 4, p.27.)

Pericardial pain

Pain from the pericardium (↻Chapter 19) occurs because the inflamed surfaces of the pericardium move over each other with each heartbeat and also with physical movement and respiration. The pain is nearly always extremely sharp and although may be constant it is usually made worse by moving, breathing, and by pressure over the sternum and anterior chest. Characteristically it is relieved by the patient leaning forward and sometimes this makes the diagnosis immediately recognizable when the patient is seen sitting in their bed, leaning forward over a table trying to relieve the pain.

Other cardiac pain

Pain from myocarditis may have an element of pericardial pain, but also tends to produce a deep, aching pain in the chest with some similarities to the pain occurring with ACSs, but often not as intense.

Aneurysms

Aneurysms of the thoracic aorta (↻Chapter 31) are usually painless, but if they begin to expand rapidly they may produce a non-specific chest discomfort which the patient may find hard to describe and which is difficult to diagnose. Dissecting aneurysm produces one of the most intense pains ever experienced by patients. It is often of very sudden onset and indeed the patient may believe that they have been hit in the chest or back and the pain is often described as tearing. The pain may be in the front of the body, but often in the back. The pain frequently radiates and may go down the legs and can be in the abdomen. The onset may be associated with collapse. The patient may not lose consciousness but simply falls to the ground because of the suddenness and severity of the onset of the symptoms. There may be additional truly ischaemic chest pain if the dissection has involved the origin of a coronary artery (most often the right). There may also be associated neurological symptoms if a major vessel to the head and neck has been compromised by the dissection.

Shortness of breath (dyspnoea)

The terms ‘shortness of breath’ and ‘dyspnoea’ are interchangeable. They imply that the patient has difficulty getting their breath and that this difficulty in getting breath

is in some way unpleasant. This symptom must be distinguished from hyperpnoea which is increased breathing and from angina. The increased breathing associated with exercise in normal people at a moderate level is an example of hyperpnoea, but if a patient then pushes themselves beyond their limit then breathing becomes uncomfortable and the sensation can be described as being dyspnoea. Hyperpnoea does not usually feel unpleasant to the patient and is associated with conditions that lead to increased respiratory drive, e.g. the acidosis in diabetic ketoacidosis. (Also see ↻Key point 5, p.27)

The aim of the history is to begin to distinguish between the common causes of dyspnoea [7]. The most important are a cardiac cause usually associated with incipient or pre-existing left heart failure (↻Chapter 23), respiratory disease, and when there is an unreasonable demand on the normal heart and lungs. This latter situation is very often seen in patients who are severely overweight but severe anaemia leads to the same situation. In the real world patients often have more than one cause of dyspnoea. A common situation is the patient with mild heart failure secondary to ischaemic heart disease, chronic bronchitis due to smoking, and significant obesity. In such patients it is often difficult to determine how much of the dyspnoea can be attributed to each of the factors. The only way is to treat everything that is treatable and see what happens. Certain features in the history may help to distinguish the cause. Classically cardiac breathlessness is made worse by lying flat (orthopnoea) (see ↻Table 1.8). The degree of orthopnoea is usually defined by the number of pillows that the patient uses, but the physician must determine how many pillows that patient has always used. It is a change in the use of pillows that is important, rather than the actual number. Some people who have no cardiac disease like to sleep with a large number of pillows in a semi-recumbent position. Patients may also sleep sitting up because of other symptoms of which the most common is oesophageal reflux. Although

Table 1.8 Breathlessness lying flat and patients who prefer to sleep sitting up

Heart failure—orthopnoea
Angina—angina decubitus
Gastro-oesophageal reflux disease (GORD)
Lung disease—using accessory muscles and therefore sit up to fix shoulder girdle
Obesity/ascites
Pregnancy
Habit

orthopnoea points towards a cardiac cause, patients who have severe respiratory disease also find it difficult to lie flat and often tend to sit up and fix their shoulder girdle so that they can use the accessory muscles of respiration to aid their impaired respiration. Furthermore, obese patients have great difficulty lying flat because the contents of their abdomen tend to push upwards on to the diaphragm when they are lying flat. A useful line of questioning while taking the history can be to ask the patient what happens if they slip off the pillows while they are asleep. Patients with true orthopnoea tend to wake up distressed because of breathlessness, but other patients may simply continue to sleep in a flatter position than the one they started the night in.

Paroxysmal nocturnal dyspnoea

This dramatic symptom must be sought in taking the cardiovascular history and distinguished, if possible, from orthopnoea. When paroxysmal nocturnal dyspnoea (PND) occurs the patient wakes up with severe breathlessness, has to sit up and usually gets out of bed and stands up. They frequently go to the window because they believe that they can improve the situation by inhaling fresh air which they believe contains ‘more oxygen’. Clearly this is not true, but the effect of standing up dramatically reduces venous return and thereby reduces right heart output and relieves pulmonary congestion. PND is a very specific symptom and indicates severe cardiac dysfunction. It may be associated with coughing which produces pink, frothy sputum. This is the stage of left heart failure just before overt pulmonary oedema (➔ Chapter 23).

Cheyne–Stokes respiration

The intermittent breathing that is usually the result of a low cardiac output is really a physical sign. It is often noticed by the patient’s partner during the night when breathing slows down and stops for some seconds before starting again. The patient may also be aware of it when they drift off to sleep, as the breathing slows and stops and then they wake up with a start as they begin to breathe again

Sleep apnoea

The patient is usually obese and may be plethoric and their main complaint is of somnolence during the day and they may develop heart failure as a complication. However, in common with Cheyne–Stokes respiration, the patient’s partner is the best source for the history. Their sleep is ruined by their partner’s loud snoring and they notice periods when breathing stops and then choking, snoring, and snorting start again

Cough

Although cough is usually regarded as a symptom denoting a respiratory origin to the patient’s breathing problems, there are circumstances where coughing may point to a cardiac problem [8]. Coughing, particularly at night, may be caused by the early stages of pulmonary congestion and be a sign of impending left heart failure. Nocturnal coughing can also be a symptom either of asthma or of gastrointestinal reflux.

Palpitation(s) (cardiac arrhythmias)

Patients usually perceive cardiac arrhythmias (➔ Chapters 28–30) as an awareness of their heartbeat and they may feel thumping in the chest but this may also be described as the heart racing or pounding or fluttering in the chest. This can be one of the most difficult areas in which to obtain a precise history. The history can be difficult both because the patient may be frightened by the symptoms and, furthermore, find them very difficult to describe. The age-old method of asking the patient to tap out the rhythm on the desk or on their knee is extremely helpful. Frequently patients, when asked to do so, say they are unable to reproduce the arrhythmia but if they have a demonstration from the examining doctor of the possibilities then they soon get the idea and are able to do this. The doctor can produce a variety of examples, for example rapid and regular, rapid and irregular, etc., and ask the patient to choose which most closely resembles their arrhythmia. It is also important to distinguish whether the onset of the palpitation is sudden or builds up slowly. In most cases of genuine cardiac arrhythmia the onset is extremely sudden. Theoretically, the offset should also be sudden but sometimes it is not. Although the patient may describe the classical sudden start they become used to the rhythm and the sensation of the cessation is often not as dramatic as the start. The actual offset may be sudden on the ECG tracing but the patient senses the end of the episode as a tailing off.

It is critical to establish what—if anything—precipitates the arrhythmia (➔ Box 1.5). Careful questioning is required because patients may be embarrassed, e.g. symptoms during sexual intercourse, or may not have made a connection, e.g. chronic emotional stress of all types. The doctor must try to distinguish whether an arrhythmia is regular or irregular and this is often demonstrated by the tapping exercise described earlier and also to determine whether or not there are associated symptoms. In some patients there may be lightheadedness or even syncope, while in others the rapid arrhythmia may bring out myocardial ischaemia and be accompanied by ischaemic chest pain. Conversely, in a patient who has

Box 1.5 Scenario 5: palpitation triggers

A 37-year-old, slightly overweight patient complains of sudden-onset, rapid, regular palpitations occurring only while playing football and associated with dyspnoea and mild faintness. Twenty-four-hour ECG monitoring when not playing football revealed a few ectopics. The patient was reassured, but developed a similar but more prolonged episode while playing football a few months later. ECG revealed ventricular tachycardia and the patient was found to have an underlying cardiomyopathy. An ICD was implanted.

Lesson

Always use careful questioning to establish precipitating factors. Attempt to test when precipitating factors are present and continue testing until you have caught an episode. An exercise test could have been performed before reassuring the patient.

unexplained lightheadedness or syncope (➔ Chapter 26) or chest pain, it is important to question the patient as to whether or not there is an associated palpitation, which could represent an arrhythmia which is causing the problem.

Perhaps the commonest symptoms are those due to ventricular or atrial ectopic beats. Patients variously describe the sensation as the heart skipping a beat or stumbling or giving a heavy thump which occurs because the post-ectopic beat is more forceful than usual as the heart has longer to fill before it occurs.

Some patients who describe palpitation are in fact describing a normal cardiac rhythm. The heartbeat may be made more forceful because of anxiety or for no obvious reason and the patient then perceives an acceleration of their normal heart rhythm. Sinus tachycardia is usually relatively slow compared with a true tachyarrhythmia, often at a rate of about 110 beats per minute. Sinus tachycardia usually builds up over a matter of some minutes and then goes off slowly and does not have a sudden onset. Like many less significant arrhythmias it may be most prominent if the patient is lying in bed on their left side; this brings the heart in contact with the chest wall. Similarly a slow regular thumping or heavy beat may occasionally occur and can be due to ventricular bigeminy or, rarely, due to sinus bradycardia.

Presyncope and syncope (also see ➔ Chapter 26)

The main distinction to be made by the history is between a cardiovascular and a neurological cause (➔ Table 1.9).

Table 1.9 History in syncope: cardiovascular versus neurological cause of loss of consciousness

Cardiovascular	Neurological
No warning	Warning (aura)
–	Incontinence—faecal or urinary
–	Tongue biting
Patient usually silent	Grunting and involuntary noises
Pallor ++	Often cyanosed and suffused with (particularly in tonic phase)
No movement	Convulsions (may not be particularly marked)
<i>Afterwards:</i> usually tired but not confused with fairly quick recovery	<i>Afterwards:</i> confusion common and a feeling of being hungover and also possible residual paralysis

This can be extremely difficult, both for the patient and for the doctor [9, 10]. Primary neurological problems often have a rotational element to them and unsteadiness and problems with balance without an associated feeling that consciousness is about to be lost. There may also be associated nausea. Cardiovascular presyncope does not usually have a rotational element, although the patient may still describe a symptom as dizziness which is really a feeling of impending loss of consciousness due to hypotension. If the patient does not notice that the room is revolving, and if they feel as though they are close to losing consciousness, then the problem is more likely to be cardiovascular. (Also see ➔ Key points 6 and 7, p.27.)

Occasionally, a fall in cerebral perfusion due to a cerebrovascular cause can trigger a seizure. In a patient who complains of blacking out, it is very important to establish whether they are actually losing consciousness. Some patients may say they are blacking out, when in fact they simply feel very distant from surrounding events. Such a feeling of ‘being disembodied but remaining fully conscious’ is often associated with anxiety or other psychological symptoms. Another area in which confusion occurs is when the patient says they have lost consciousness when in fact they have fallen to the ground without losing consciousness. If the patient remembers hitting the ground this potential differential diagnosis has to be considered. There are a particular group of patients whose legs simply give way on them and there is no cardiovascular cause for this. These are the so-called true ‘drop attacks’.

It is crucially important to discover whether there have been any eye witnesses to episodes of syncope and if so to make contact with them and question them, particularly about the onset, offset, and duration of the attack as well as the patient’s colour and their behaviour, breathing pattern,

and the speed of recovery. Sometimes the witness may also have taken the pulse. It may be possible, in the age of the mobile phone, to telephone the witness while the patient is actually in the clinic. In addition to trying to elicit the symptoms that may distinguish between a cardiovascular and a cerebrovascular cause, it is also necessary to establish the circumstances under which an episode of syncope occurs. This information may be helpful in making a diagnosis, particularly when the episodes occur as a result of either fear, emotion, prolonged standing, micturition, defecation, etc. (➔ Box 1.6). Most of these circumstances are known to lead to a high level of vagal tone and lead on to vasovagal syncope. Such episodes are often known as ‘situational’. They frequently occur in young patients without any other illness, although problems with voiding, i.e. micturition and defecation syncope, are much more likely to occur in the elderly. Such situational attacks which are vasovagal must be distinguished from the rare attack that occurs in patients particularly with the long QT syndrome, when loud noises and alarming circumstances may precipitate serious arrhythmias.

If the patient describes injury associated with syncope then this usually denotes a lack of warning and suggests a significant underlying problem and a sudden onset. A warning favours a neurological cause, particularly if this takes the form of an aura, i.e. an unusual sensation which precedes the episode of loss of consciousness. This should not be confused with a brief period of presyncope prior to a true syncopal episode. Following an episode of cardiovascular syncope, patients may feel tired, but do not usually feel particularly unwell, whereas patients who have experienced a convulsion often feel extremely unwell for a long period of time with headache, lethargy, and what

they would describe as a hangover. Residual brief periods of paralysis (Todd’s paralysis) are more likely to be present after a neurological event.

Occasionally patients may give a history of collapsing with syncope and then coming round after a short period and being aware of a rapid tachycardia. This may be because whatever caused the syncope also produced a tachycardia. However, more often it is because the sudden drop in cardiac output caused by the tachycardia and an unprepared dilated peripheral circulation causes a severe fall in blood pressure and syncope. Then protective constrictive reflexes raise the blood pressure despite the continuing tachycardia and the patient regains consciousness.

Oedema and ascites

Although oedema and ascites are physical signs, they are also described by patients as symptoms.

Fatigue

Fatigue is probably very common but hard to define as it has so many causes. It is most striking when a patient has a successful treatment and suddenly realizes how tired they were before. If it occurs intermittently it may have a definite underlying cause although this may be difficult to track down (➔ Box 1.7).

Less common cardiological symptoms

Vomiting

Patients in the early stage of an acute myocardial infarction may vomit profusely. It may be difficult to be sure whether the stress and dehydration of the vomiting precipitates the infarction, or vice versa [11, 12].

Box 1.6 Scenario 6: effect of position

A middle-aged woman with no known cardiac problems notices a fast heartbeat at the end of eating a heavy meal in a hot restaurant. She decided that she must go to the lavatory, stands, and starts to walk to the door but loses consciousness after a few yards.

Explanation

The arrhythmia initially causes a moderate fall in cardiac output but while she is sitting the circulation can compensate. As soon as she stands up the gravitational effect on the circulation reduces the cardiac output further and loss of consciousness ensues.

Box 1.7 Scenario 7: tiredness—a difficult symptom

A 55-year-old male, who was a successful veteran cycle racer at national level, noticed that occasionally while racing he suddenly and unexpectedly ran out of energy and became tired. This caused him to slow down and abandon the race. When he did so the symptoms usually resolved within a few minutes and he felt normal again. He had no other symptoms. Holter monitoring during a cycle race showed that this loss of energy was due to the sudden occurrence of atrial flutter with 1:1 conduction. This has been cured by ablation and he is now racing successfully again.

Polyuria [13, 14]

Patients with supraventricular tachycardia (☞ Chapter 28) may describe striking polyuria which starts a few minutes after the onset of an attack. This is very characteristic and is probably due to a raised intra-atrial pressure causing a release of ANP leading to natriuresis and diuresis. It is particularly common in atrial fibrillation (☞ Chapter 29), and paroxysmal atrioventricular (AV) nodal re-entry tachycardia.

Fullness in the neck ± right upper quadrant abdominal pain [15]

Patients with high right atrial (RA) pressures may notice this being transmitted in to the neck as a feeling of fullness. It is particularly common in patients who have severe tricuspid regurgitation. In addition to this, venous back pressure into the liver stretches the liver capsule and causes discomfort.

Pulsation in the neck associated with tachycardia

A feeling of pulsation in the neck associated with a rapid regular tachycardia suggests an AV nodal re-entry tachycardia [16]. This is because the atrium contracts at the same time as the ventricle, i.e. when the tricuspid valve is closed and so the wave form is transmitted upward into the neck rather than forward into the RV.

Unusual noises in the chest

A very unusual but striking symptom is when the patient complains of hearing a squeaking noise in their chest, often associated with symptoms. This may occur in the context of an arrhythmia in a patient with mitral valve prolapse (☞ Chapter 21) and can also be described by patients who have suddenly developed a severe degree of mitral regurgitation due to chordal rupture or when the cusp of a prosthetic tissue valve tears spontaneously.

A further example is in a patient with a mechanical valve (☞ Chapter 21) in whom there is sudden cessation of clicks associated with symptoms and then the clicks resume. This is very rare, but may indicate a mechanical fault with the valve with a component sticking. When the part of the valve sticks the noises generated by the valve cease.

Using the cardiovascular history to identify danger areas

There is a high potential for harm if a serious cardiovascular diagnosis is missed and if a patient who is either in the clinic or emergency department is then sent home without further investigation. If the differential diagnosis lies between a serious cardiovascular problem, such as an ACS, and a much less serious diagnosis, such as gastrointestinal

reflux, it is essential to err on the side of caution and provisionally make the more serious diagnosis while recognizing that tests must be carried out to either confirm or refute this diagnosis. Many doctors are averse to making the more serious of two potential diagnoses and have an inclination to reassure the patient unjustifiably. This is not good practice and must be resisted. It is crucial to remember that differentiation of two such diagnoses may be impossible using the history alone.

Some cardiovascular histories which require urgent attention

The patient who wants to go home

The patient in the emergency room who feels perfectly well at the time when their history is taken but has presented with an episode of chest discomfort that could well be of cardiac origin. The patient ascribes these symptoms to indigestion. The ECG is normal. This situation cannot be resolved by the history and requires a period of observation and further investigation. If the patient is in the hospital and the condition progresses, appropriate therapy, including early revascularization, can be provided or resuscitation can be given should they develop a cardiac arrest. After leaving the hospital then neither of these options is feasible and out-of-hospital resuscitation has a very low success rate. It is no disgrace to admit a patient for one night, decide the diagnosis is not myocardial ischaemia, and send the patient home. It is a disaster to send home a patient who dies because of a misinterpretation of their cardiac history.

Unexplained syncope of recent onset

A patient presents with a completely unexplained sudden blackout, usually occurring for the first time. This is a particularly difficult situation. Many of these patients will have a relatively benign cause for their symptoms, but a small number will have extremely severe underlying disease which requires immediate treatment if the patient is to survive. In such patients it is important to look for ancillary information. For example, a patient who has a massive pulmonary embolism may quickly recover after the first embolic event, but has at the time of the collapse a brief period of breathlessness and a reduced oxygen saturation if measured. These observations may have been made in the ambulance bringing the patient to hospital. It is therefore crucial in any acute patient to obtain a history from the relatives and ambulance staff, and also information from the ambulance records. Another example is the patient who has an aortic dissection with an episode of collapse, sudden pain in the back, but quick resolution so that by the time

they arrive in the emergency department the patient feels perfectly well. Again it is the associated symptoms that act as the telltale for a serious underlying condition.

The patient with valvular heart disease

Intermittent symptoms of shortness of breath or episodes of presyncope or syncope in a patient with a mechanical prosthesis

These could be due to intermittent valve malfunction. This is a particularly difficult area in which to establish a diagnosis, but if a patient with a mechanical valve notices the clicks have stopped or changed in character the diagnosis of a malfunctioning valve must be seriously considered.

A patient with unexplained deterioration of valvular heart disease and non-specific symptoms

When a patient describes being generally unwell, with widespread aches and pains, weight loss, and fever, infective endocarditis must be considered. The consequences of missing it are very serious. In such patients inflammatory markers should be measured, an echocardiogram performed, and blood cultures taken if there is any suspicion of this diagnosis.

Examination

Introduction

Examination remains a key component of cardiology (☞ Table 1.10). A comprehensive cardiovascular examination can be time consuming and often a rapid assessment of the patient is required, especially in acute situations. Time pressures even in an out-patient environment often make a full examination impractical. This ‘scenario examination’ technique can save valuable time in an acute or emergency situation. Examples of this approach are discussed later in this chapter. It is important to understanding the relevance of both the presence and the absence of particular clinical signs.

General examination

If possible, clinical examination should be performed in a quiet, warm, comfortable environment with good lighting, although clinical circumstances often dictate the need to perform an examination under suboptimal conditions. Putting the patient at ease from the outset helps examination and it is important to respect their privacy and dignity at all times.

Examination begins before the patient is settled on an examination couch and valuable insights can be gained

Table 1.10 Cardiovascular examination checklist: the essentials

Cardiovascular exam checklist	Comments
Watch patient enter the room/get on couch	May be dyspnoeic or get pain
Correct position	45°—to see JVP
General exam	
Scars/rash	Previous surgery
Hands	Splinters/peripheral cyanosis
Face/eyes	Pallor, cyanosis, jaundice
Ocular fundi	Diabetic and hypertensive changes, endocarditis
Pulse	
Radial (both)	Feel the vessel, wall, and rhythm (catheter site)
Carotid	Best for pulse character and timing, JVP, and murmurs
Femoral	Decreased/unequal/bruit—PVD Delayed compared to radial—coarctation Catheter site
Foot pulses	PVD
Blood pressure	
Take it yourself	Use correct cuff Use point of disappearance <i>not</i> muffling as diastolic pressure Compare at different sites if dissection possible
JVP	
If invisible: ◆ Press on liver to fill JVP ◆ Sit up in case it is invisible because very high	Need good light, head turned slightly away
Normal < 4cm above sternal notch	Left usually more reliable than right JVP Gentle digital pressure stops pulsation
Chest	
Inspect	Scars, deformity, movement
Palpate	Body habitus has major effect on palpation and auscultation
Listen for murmurs and normal and abnormal sounds	Locate apex—feel <i>all over chest</i> for heaves, thrills etc. If necessary assess effect of: ◆ Position ◆ Respiration ◆ Exercise
Quiet environment needed	If pulse irregular compare with heart rhythm on auscultation

JVP, jugular venous pulse; PVD, peripheral vascular disease.

during general observations of gait and mobility, and associated symptoms such as breathlessness and discomfort as the patient enters the room and gets on to the couch; the presence of confusion or distress may become apparent at this stage. General observations such as height, obesity, obvious skeletal deformities such as kyphoscoliosis may also be more obvious as the patient enters the room (see ➔ Key points 8 and 9, p.27).

The history should be expanded and refined during examination and if unexpected physical signs are found this may prompt further questioning of the patient. If the sign is mentioned, for example the presence of clubbing, the patient may then respond that it has been known for a long time or has been noticed by other doctors.

There are many clinical signs that might be elicited during a general physical examination which have relevance to the cardiovascular system (➔ Table 1.11).

Scars from previous surgery are important especially in the unconscious or confused patient. Although a median sternotomy scar can relate to many types of cardiac surgery it may also indicate other past mediastinal problems, e.g. thymus (consider myasthenia gravis) or thyroid. A left thoracotomy scar may indicate aortic surgery, particularly previous surgery for coarctation of the aorta, and a right thoracotomy may indicate previous mitral surgery. Always look at the legs and arms for scars indicating that a vascular conduit has been harvested, strongly suggesting a previous coronary artery bypass graft. Chest scars may be due to previous lung surgery which may shift the mediastinum and with it the heart, making interpretation of physical signs more difficult.

Certain chest deformities can have cardiovascular significance. Pectus excavatum (depressed sternum) can produce a false impression of cardiomegaly by displacing the heart to the left and may also distort the RV producing an innocent murmur. Pectus excavatum and also pectus carinatum (prominent sternum) can be seen in Marfan syndrome (➔ Chapter 31) [17]. In patients with cardiomegaly since early childhood the chest wall may bulge out over the enlarged heart.

Cardiovascular examination

Pulse

The rate, rhythm, pulse character, and the nature of the vessel wall can usually be assessed from the radial pulse. Although the pulse pattern may suggest a diagnosis for the underlying rhythm an ECG is always necessary for confirmation. An easily palpable radial vessel wall is usually an indication of abnormal thickening and possibly calcification, and suggests more generalized vascular disease (see ➔ Key point 10, p.27).

Table 1.11 Physical signs on general examination

	Causes	Comments
Common signs		
Anaemia	Blood loss Infective endocarditis Hypothyroidism	Many causes of anaemia can cause sinus tachycardia, heart failure
Central cyanosis	Intracardiac or extracardiac shunting, reduced oxygen	Any situation where venous–arterial admixture occurs
Peripheral cyanosis	Vasoconstriction Low cardiac output	Can be environment dependent, e.g. Raynaud's
Corneal arcus	Aging Hyperlipidaemia	
Xanthelasma (➔ Fig. 1.1)	Hyperlipidaemia	Often occur as normal variant
Splinter haemorrhages (➔ Fig. 1.2)	Infective endocarditis Local trauma Vasculitis	One or two splinters may be present without disease
Tremor	Hyperthyroidism Alcohol withdrawal Drug induced	
Capillary pulsations	Severe aortic regurgitation	Quinke's sign
Rash and petechiae	Vasculitis, endocarditis, rheumatic fever	Very non-specific but extremely helpful. Often associated with arthralgia or arthropathy
Uncommon signs		
Finger clubbing (and toes)	Infective endocarditis Cyanotic congenital heart disease Idiopathic—familial	Also in lung cancer and other lung disease
Malar flush	Mitral stenosis	Also seen in systemic lupus erythematosus
Roth spots	Infective endocarditis	Retinal haemorrhage with central white spots
Janeway lesions	Infective endocarditis	Raised haemorrhagic lesions on hands and feet
Facial dysmorphism	Down, Turner, Noonan syndromes	A variety of congenital chromosomal abnormalities
Blue sclera	Pseudoxanthoma elasticum Ehlers–Danlos syndrome	Aortopathy main cardiovascular problem Most commonly occurs in osteogenesis imperfect
Cushingoid facies	Cushing's syndrome Steroid therapy	Hypertension Fluid retention
Non-pitting oedema	Hypothyroidism	
Arachnodactyly Arm span height Lens dislocation High arch palate	Marfan syndrome	Associated with aortopathy and mitral valve prolapse



Figure 1.1 Xanthelasma. Typical appearance of xanthelasma in patient with severe hypercholesterolaemia. Note the yellowish deposits surrounding the eyes.

The character of the pulse is a valuable sign but must usually be assessed using a central pulse and the carotid is usually best although the brachial may be helpful. One exception is the collapsing pulse of severe aortic regurgitation which, although detected at the carotid, is often best appreciated by palpating the radial pulse with the arm raised above the head when the pulse acquires a very sharp tapping quality.

Some typical pulse characters are illustrated in Fig. 1.3. A slow rising pulse (Figs. 1.3B and 1.4) is most commonly associated with significant aortic stenosis [18] (Chapter 21). There is a time delay to peak systolic pressure which gets later as stenosis gets worse and the pulse volume also falls. It is, however, a myth that a normal or raised systolic blood pressure *excludes* severe aortic stenosis. The LV has a remarkable ability to generate pressure which may occasionally get as high as 300mmHg. In this situation a 100mmHg gradient across the aortic valve still leaves a systolic pressure of 200mmHg! The typical slow rising pulse can be mimicked by local disease in the carotid and checking both carotids may clarify the situation. Patients can also have severe aortic stenosis without a slow rising pulse. This is because these patients are often elderly with



Figure 1.2 Splinter haemorrhages. Splinter haemorrhages are noted as linear haemorrhagic lesions at the distal nail bed. One or two splinters can occur in normal individuals but multiple splinters are typical of infective endocarditis or vasculitis (Chapter 22).

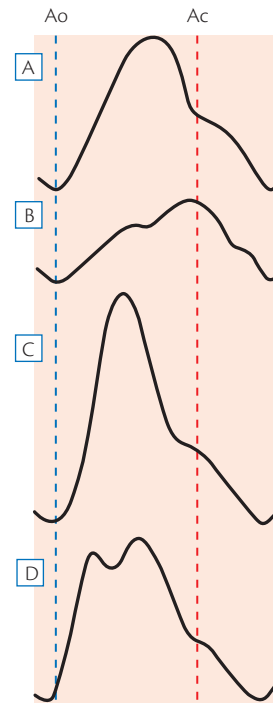


Figure 1.3 Pulse character. (A) Normal pulse. Following Ao there is a fairly rapid upstroke of the pulse to its peak, then a more gradual descent which includes an impalpable 'dicrotic notch' created as a result of aortic valve closure. (B) Slow rising pulse. The upstroke is more gradual, the peak reduced and delayed. Usually associated with aortic stenosis. (C) Collapsing pulse commonly associated with severe aortic regurgitation. There is a rapid rise in systolic and a rapid decline of pressure after the peak. (D) Bisferiens pulse. Often associated with combined aortic stenosis and regurgitation, it is characterized by twin peaks (percussion wave and tidal wave) separated by a mid-systolic dip. Ac, aortic (valve) closure; Ao, aortic (valve) opening.

hard sclerotic vessels which amplify the pulse pressure and mask the slow rising pulse.

Figs. 1.3C and 1.4 illustrate a collapsing pulse typically resulting from severe aortic regurgitation (Chapter 21). With each beat the LV has to eject both the forward cardiac output and the amount of blood that will leak back into the ventricle in the next early diastole. This large volume of blood is ejected forcefully into the aorta producing the sharp upstroke and then a large volume immediately falls back into the LV producing the 'collapse'. These pulse characteristics also occur in any situation when there is a large volume systolic leak from the central circulation, e.g. with a large AV fistula or an uncorrected patent ductus arteriosus (Chapter 10). In conditions where there is an increased cardiac output, e.g. pregnancy, fever, anaemia, Paget's disease of bone, the pulse will be of large volume and is often described as 'bounding'. The pulse in these conditions has some similarities to the collapsing pulse but there are subtle differences. The upstroke and particularly the downstroke are not as abrupt since there is not a sudden diastolic leak of blood from the central circulation

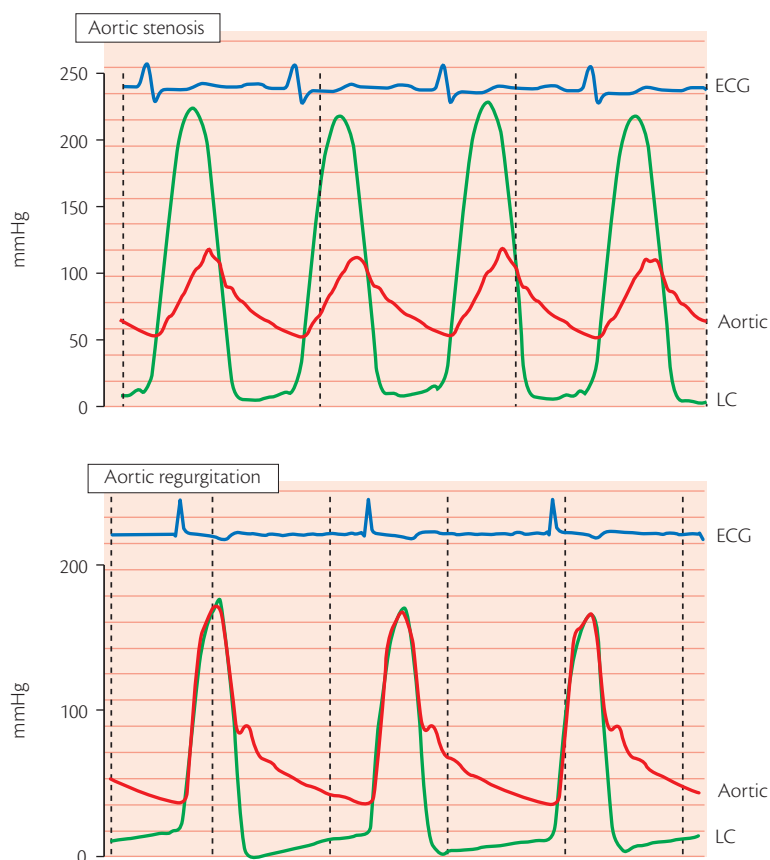






Figure 1.4 Pulse pressure waveform. Illustration of the aortic pressure waveform in aortic stenosis and aortic regurgitation compared to the simultaneous left ventricular pressure. In aortic stenosis the aortic pressure rises slowly to its peak in late systole and there is a significant systolic pressure difference between the aorta and left ventricle. In aortic regurgitation the aortic and left ventricular pressures are identical during systole but the rapid fall in aortic pressure in diastole results in a wide pulse pressure and the characteristic collapsing pulse.

Finally,  Fig. 1.3D illustrates the ‘bisferiens’ pulse which usually occurs when aortic regurgitation is combined with aortic stenosis. There are two peaks (percussion wave and tidal wave) separated by a mid-systolic dip. The bisferiens pulse is rare but very striking when it is encountered in clinical practice.

Pulsus paradoxus

Pulsus paradoxus ( Fig. 1.5) occurs when the pulse pressure falls by $> 10\text{mmHg}$ with each inspiration [19]. Clinically it is difficult to detect by palpation until this decrease exceeds 20mmHg . The sign is elicited and measured by slowly deflating the blood pressure cuff while listening to the blood pressure and observing the patient breathing. The commonest cause is pericardial tamponade ( Chapter 19). The physiological mechanism is complicated but the main effect is that the heart cannot expand because of the surrounding,

compressing pericardial fluid and consequently when blood is sucked into the RV by inspiration, left-sided cardiac filling from the pulmonary circulation is reduced and the output from each beat during inspiration falls. Inspiration also pulls the heart down and the globular shape of the heart becomes more cylindrical. This restricts cardiac volume further as the volume of a cylinder is smaller than that of a sphere with the same surface area. In constrictive pericarditis ( Chapter 19) the pericardium has the same limiting effect on cardiac volume. In patients with extreme dyspnoea, e.g. due to asthma, the very striking swings in intrathoracic pressure also produce pulsus paradoxus [20].

Pulsus alternans

This is when there are alternate strong and weak pulses in a basically regular pulse. This is a sign of severely impaired LV function and may be exacerbated by the presence of

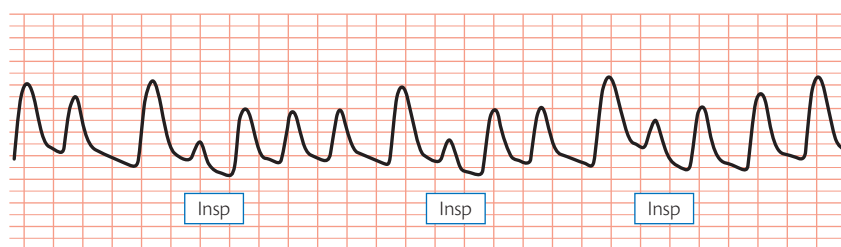


Figure 1.5 Pulsus paradoxus. Aortic pressure waveform demonstrating variation in pulse amplitude with respiration, the pressure decreasing with inspiration (insp).

hypovolaemia. It has to be distinguished from ‘pulses bigeminus’. This is when there are alternate strong and weak beats due to ventricular (or occasionally atrial) ectopic beats alternating with normal sinus beats. As well as there being a weak and strong pulse as in pulsus alternans, there is also an irregularity in the underlying pulse rhythm which is not present with alternans. The ECG may also show electrical alternans, i.e. variation in the voltage of alternate QRS complexes in this situation, whereas in pulsus bigeminus the ectopic beats are revealed.

Other pulses

Differences in the volume and timing of major pulses may indicate either localized vascular disease or disease at the origin of the vessels, sometimes caused by dissecting aneurysm. Differences in pulses may be appreciated better if the blood pressure is taken in more than one site and the pressures compared.

Examining the leg and foot pulses helps evaluate peripheral vascular disease (➔Chapter 36). The presence or absence of femoral pulses is also important when cardiac catheterization is considered. In a busy clinic where it can be very time consuming to examine the femoral pulses of an elderly patient, a careful examination of the foot pulses may be a reasonable substitute. If all foot pulses (dorsalis pedis and posterior tibial on both sides) are strong and present it is very unlikely that that patient will be suffering from coarctation, particularly if they have a normal blood pressure. In the presence of hypertension the femoral pulses must be examined to exclude coarctation (➔Chapter 10), for although it is very rare for this condition to be undiagnosed in adults, occasionally patients do appear. The pulse should arrive at the femoral arteries and the radial arteries at about the same time as they are approximately the same distance from the heart. In coarctation, when there is severe obstruction at the site of coarctation the blood has to traverse collaterals and the femoral pulse is delayed.

Blood pressure

Often the blood pressures (➔Chapter 13) taken initially by the nurse are simply transferred to the notes written by the doctor, without the doctor checking them; this is bad practice. This has the disadvantage that an inaccuracy in the previous blood pressure may be perpetuated, and the blood pressure may have changed in the time between the two examinations. This is an increasing problem, especially with electronic medical records, where whole sections are often cut and pasted from one consultation to the next. With the progressively more obese population it is important to use a large cuff on big arms, otherwise there is an overestimation of blood pressure levels [21].

Jugular venous pulse (➔Table 1.12)

Assessing the jugular venous pulse (JVP) can be one of the most difficult clinical signs in cardiology [22, 23]. The JVP results from transmission of the pulsation from the internal jugular vein to the skin surface, and it is the pulsation rather than the vein itself that is visible. The two aspects of the JVP to be assessed are its level and its character (waveform). Since no valve intervenes, the pressure in the internal jugular is the same as that in the RA. The JVP is often invisible especially in obese patients. If it cannot definitely be seen, record it as ‘not seen’ rather than ‘normal’. Engorged superficial veins may be due to kinking of the vein at the thoracic inlet and do not indicate the level of the JVP.

The JVP is examined with the patient semi-recumbent at an angle of about 45° with the patient’s head partially rotated to one side. The JVP is normally just visible at or above the level of the sternal notch behind the sternal heads of the sternomastoid muscle. It is best to examine both sides of the neck when evaluating the JVP. One potential problem is that sometimes the left-sided JVP is higher than the right-sided JVP because there is some obstruction of the innominate vein as it crosses the chest which artificially raises the left JVP. If the JVP is not visible, it is useful to lie the patient flatter to accentuate a low JVP and pressing on the liver may accentuate it further. The ‘hepatojugular reflex’ (sometimes stated as ‘reflux’) does not in our experience generally contribute anything else further to the examination although some authorities believe it to be helpful in diagnosing heart failure [24]. Similarly, the pulsation of the JVP may not be visible because the top of the pulsation is above the angle of the jaw and only appears when the patient sits more upright. Pulsation from the carotid artery can be transmitted to the surface and has to be distinguished from the JVP. Firstly the waveform is often different. The carotid pulse is usually a single wave. Secondly the JVP can usually be abolished by gentle digital pressure and it is not possible to feel the pulsation with the finger while doing this, whereas

Table 1.12 Causes of an elevated JVP

Right heart failure
Right ventricular infarction
Pulmonary hypertension
Hypervolaemia
SVC compression
Tricuspid stenosis
Tricuspid regurgitation
Reduced right ventricular compliance
Pericardial constriction/tamponade

carotid pulsation is always palpable and cannot be abolished by gentle digital pressure. The one occasion when the JVP may be palpable is when there is severe tricuspid regurgitation producing a large V wave in the JVP which is caused by direct transmission of RV systolic contraction into the RA and then up the internal jugular vein. This can often be felt in the neck and may also be transmitted to the liver.

Jugular venous pulse character

The normal JVP waveform is illustrated in Fig. 1.6. The 'a' wave results from an increased pressure caused by atrial contraction and is the dominant wave in the normal JVP. It is accentuated in the presence of RV hypertrophy, pulmonary hypertension, or tricuspid stenosis (Chapter 21) because RA contraction is powerful but lost in atrial fibrillation. It occurs just before the carotid pulse. In the normal JVP, following the 'a' wave pressure decreases (the x descent) due to atrial relaxation and RV systole which causes descent of the tricuspid valve towards the apex as a result of longitudinal shortening of the ventricle. Next comes the V wave which is due to passive filling of the RA while the tricuspid valve is closed by RV contraction. In tricuspid regurgitation, the v wave peaks earlier, is accentuated and becomes the dominant waveform (Fig. 1.7). This is because the 'v' wave in this situation has a completely different genesis but confusingly still carries the same name. It is due to the direct transmission of the systolic contraction of the RV into the RA and up the internal jugular rather than the passive filling of the atrium with the tricuspid valve closed. When the tricuspid valve opens, pressure falls (y descent) until continued passive filling of the atrium begins

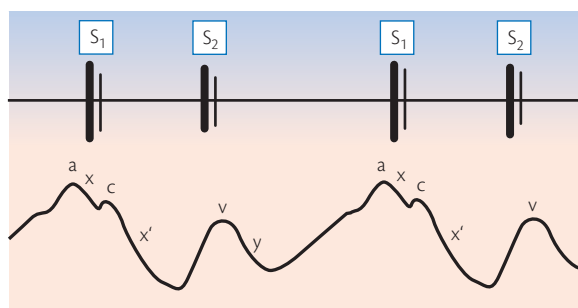


Figure 1.6 JVP Character. 'a' wave: results from an increased pressure caused by atrial contraction and is the dominant wave in the normal JVP. It is accentuated in the presence of right ventricular hypertrophy, pulmonary hypertension, or tricuspid stenosis but lost in atrial fibrillation. 'x' descent: follows the 'a' wave and is due to atrial relaxation and right ventricular systole causing descent of the tricuspid valve towards the apex as a result of longitudinal shortening of the ventricle with systolic contraction. 'c' wave: interrupts the 'x' descent as a transmitted carotid pulsation. 'v' wave: increasing pressure due to passive filling of the atrium while the tricuspid valve is closed by right ventricular systole. 'y' descent: fall in atrial pressure due to opening of the tricuspid valve; followed by passive filling of the atrium culminating in a further 'a' wave. The 'y' descent, as a result of tricuspid valve opening, occurs after the second heart sound (S_2), a useful reference for timing of the JVP waveform.

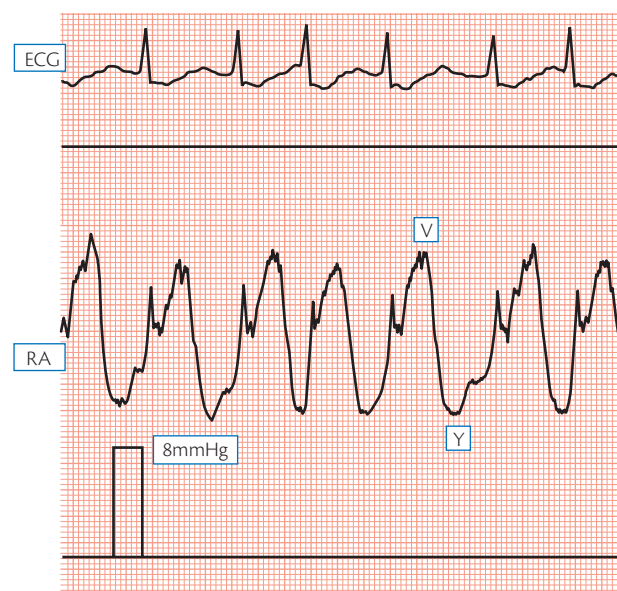


Figure 1.7 Tricuspid regurgitation. Right atrial pressure trace in tricuspid regurgitation illustrating the early, accentuated, and dominant 'v' wave.

to increase venous pressure again, ending with the next atrial contraction producing the 'a' wave. Since the 'y' descent results from tricuspid valve opening it occurs after the second heart sound (S_2), a useful reference for timing of the JVP waveform. The 'y' descent becomes particularly prominent in constrictive pericarditis where venous filling of the heart is predominantly an early diastolic phenomenon. A useful sign is that the JVP elevates with inspiration (Kussmaul's sign) [25], a reversal of the normal situation where the decreased intrathoracic pressure sends blood into the chest with a resultant decrease in venous pressure in the JVP. This can also occur in some other clinical situations such as right ventricular infarction [26].

If the RA and RV contract at the same time, as occurs intermittently with AV dissociation (e.g. complete heart block, see Chapter 27), blood pumped by the atrium cannot go into the RV because the tricuspid valve is held shut by RV contraction and so shoots up the internal jugular to produce a 'cannon wave'. Cannon waves can also sometimes occur as a result of a ventricular ectopic beat that closes the tricuspid valve but is not electrically conducted to the atrium, which therefore is excited in the normal way. The resulting RA contraction shoots blood up the neck. This can also occur in patients with ventricular tachycardia without VA conduction. Regular cannon waves occur in ventricular tachycardia with intact retrograde conduction and also in AV node re-entrant tachycardia (Chapter 28).

Cardiac palpation

The cardiac examination always involves thorough palpation all over the front of the chest. This will pick up abnormal

movement of the chest wall related to the contraction of the underlying heart, and thrills which are palpable murmurs. Abnormalities may be brought out by sitting the patient forward and feeling the chest in expiration, and also rolling the patient onto their left side. Thrills are palpable murmurs and the associated murmur is nearly always loud. They are usually best felt in the areas where the murmur is best heard. In a patient in whom coarctation (➔Chapter 10) is suspected, palpation of the back may reveal widespread diffuse pulsation secondary to large collaterals running in the muscles of the back. This indicates a very severe degree of coarctation.

In pulmonary hypertension (➔Chapter 24) the pulmonary artery dilates and produces an impulse in the second left intercostal space and the loud pulmonary component of the S_2 may also be appreciated as a sharp snapping feeling in this area.

Apical impulse (apex beat)

The lowest and most lateral position on the chest wall where a cardiac impulse can be felt is known as the apex beat. Some physicians refer to the point of maximal impulse (PMI). This can be confusing as sometimes the most laterally felt impulse is not the 'maximal impulse'. The apical impulse or apex beat is usually located in the fifth intercostal space at the level of, or just medial to the mid-clavicular line. Chest deformity, lung disease, and obesity all reduce the intensity of the apex beat or render it impalpable. In these situations, rotating the patient to a left lateral decubitus position tips the heart towards the chest wall and makes the apex beat easier to feel.

Abnormalities of the apical impulse

The most common abnormalities of the apex impulse are as follows:

- ◆ A forceful or 'thrusting' apex, either in the normal position or slightly displaced to the left. This is usually due to concentric LV hypertrophy as a result of conditions such as aortic stenosis or hypertension.
- ◆ The apical impulse may be displaced to the left and have a more diffuse heaving nature. This is usually when there is volume overload, e.g. in mitral or aortic regurgitation. A similar type of apex beat is often seen when there is severe LV dysfunction and the ventricle is enlarged. If the patient has an audible gallop rhythm this can sometimes also be palpated with a hand placed over the cardiac apex [27, 28].
- ◆ Mitral stenosis (➔Chapter 21) produces a particularly characteristic cardiac apical impulse. The apical impulse often has a sharp tapping nature. This is because the first heart sound (S_1) is loud because of forceful closure of the mitral valve and this forceful closure is transmitted to the chest wall as a 'tap'.

- ◆ Constrictive pericarditis (➔Chapter 19), which is rare in many developed countries, but common in the developing world, can produce in-drawing of the intercostal spaces during systole because the LV is tethered to the chest wall by the diseased pericardium [29].

Left parasternal (right ventricular) heave

A significantly hypertrophied and/or dilated RV will produce an abnormal impulse at the lower end of the sternum, usually to the left side. In a patient with lung disease and an abnormal RV this physical sign may be absent because the over-inflated lung acts as a cushion between the heart and the chest wall and therefore prevents the impulse being transmitted to the surface.

Auscultation

This still remains an important aspect of the clinical cardiovascular examination but auscultatory skills are decreasing with the almost universal availability of echocardiography in the developed world. Heart sounds and murmurs are often not difficult to time in the cardiac cycle but if there is doubt, palpation of the carotid pulse is extremely useful. Systolic events tend to occur at the same time as the carotid pulse since as there is only a short distance between the aortic valve and the carotid artery, the systolic pulse wave in the carotid artery occurs only a matter of milliseconds after the ejection phase of LV systole. Diastolic events occur between the palpable pulses.

Normal heart sounds

S_1 and S_2 are usually the only heart sounds heard on auscultation of a normal heart (➔Fig. 1.8A), although in young and athletic subjects a soft third sound (S_3) may be present.

S_1 results from closure of the mitral and tricuspid valves and has two components in close proximity [30]. Clinically this splitting is usually narrow and is difficult to hear unless there is right bundle branch block which accentuates this splitting by delaying the onset of RV contraction and therefore tricuspid closure. This sign has no clinical significance.

S_2 results from closure of the aortic and pulmonary valves (A_2 and P_2) [31], and is also normally split, the dominant aortic component occurring first. This splitting is usually accentuated by inspiration when right heart filling is increased and can be detected in most patients. Pulmonary hypertension (➔Chapter 24) increases the intensity of P_2 and systemic hypertension (➔Chapter 13) may make A_2 louder [32].

The splitting of the S_2 requires both the aortic and pulmonary valves to be mobile so they can contribute their

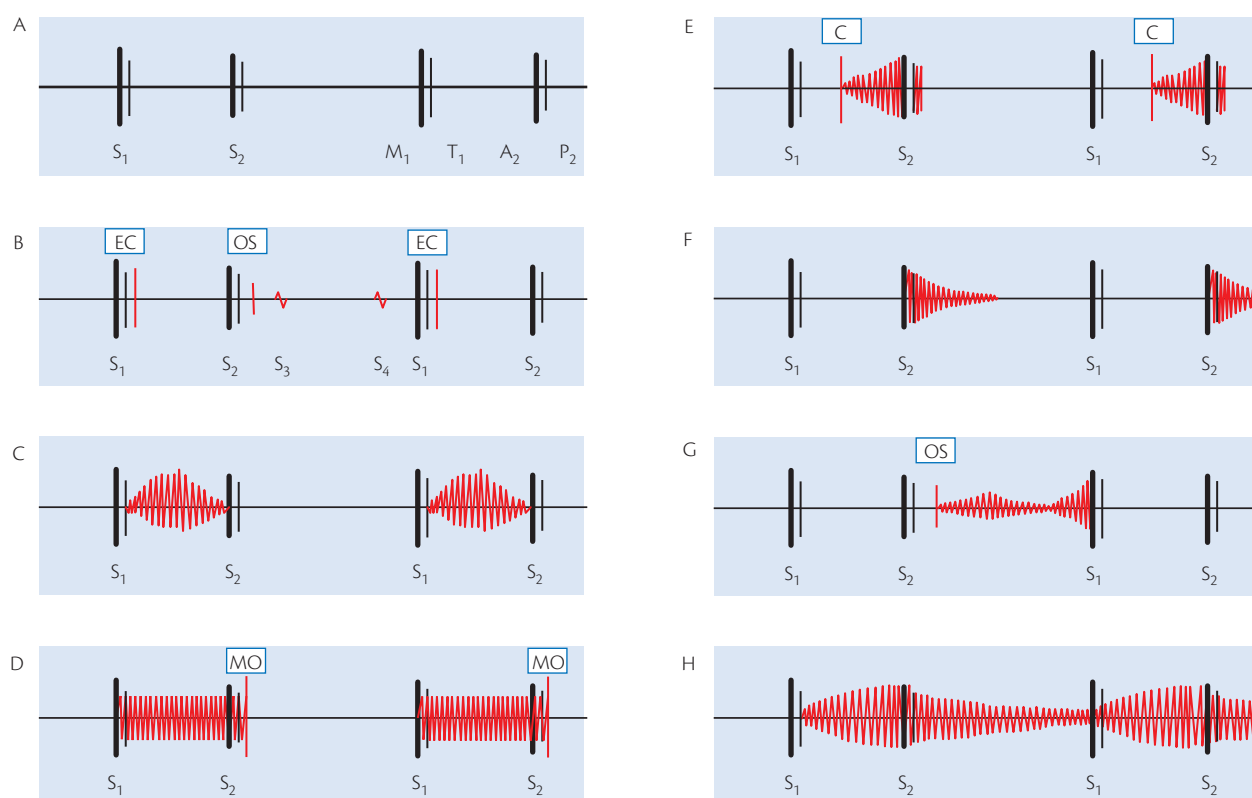


Figure 1.8 Heart sounds and murmurs. (A) Timing of normal heart sounds. S_1 , first heart sound; S_2 , second heart sound. S_1 has two components, mitral closure (M_1) and tricuspid closure (T_1); M_1 is louder. S_2 also has two components, due to aortic valve closure (A_2) and pulmonary valve closure (P_2) with A_2 being the louder. (B) Additional heart sounds. Third heart sound (S_3) in early diastole due to early ventricular filling. Fourth heart sound (S_4) in late diastole due to reduced ventricular compliance. EC, ejection click, (or ejection sound) an abrupt, high frequency, systolic sound associated with an abnormal (usually congenital) aortic or pulmonary valve; OS, opening snap, associated with opening of the valve in mitral stenosis. (C) Mid (ejection) systolic murmur. Typical of aortic stenosis, the murmur increases in early systole and decreases in late systole (crescendo–decrescendo pattern) and occurs only when the valve is open and ejection is occurring. As such, it starts just after S_1 and stops at S_2 . (D) Pan- (holo-) systolic murmur. Typical of mitral regurgitation, the murmur intensity varies little throughout systole (plateau pattern). It starts with S_1 and continues through (and masks) S_2 , stopping with mitral valve opening (MO). (E) Late systolic murmur. Typical of mitral valve prolapsed, the murmur commences in mid-late systole, often preceded by an ejection click (C). As with (D) it continues through and masks S_2 , stopping with mitral valve opening. (F) Early diastolic murmur—typical of aortic or pulmonary regurgitation. It commences immediately following S_2 and decreases in intensity during diastole as the pressure difference between the respective great vessel and ventricle decreases. (G) Mid-diastolic murmur with presystolic accentuation. Typical of mitral stenosis, the murmur is a low intensity ‘rumble’ in mid diastole. It can be preceded by an OS when the valve is pliable and, in sinus rhythm, increases in intensity at end-diastole as a result of atrial contraction, stopping with the associated loud mitral component of S_1 . (H) Continuous murmur. Murmur in systole which continues through S_2 into some or all of diastole. This results from a communication between the left- and right-sided circulations so that there is a pressure drop across the pathological structure throughout systole and diastole, hence the continuous nature of the murmur.

component. Usually the splitting widens with inspiration, mainly because blood is drawn into the right heart by negative intrathoracic pressure, increasing RV filling. It takes longer for this extra blood to be ejected thereby delaying P_2 and widening the splitting. ➔ Table 1.13 shows the commonest abnormalities of the S_2 and an illustration of wide, fixed splitting is shown in ➔ Fig. 1.9.

Further effects of respiration on auscultation

In practical terms:

- ◆ If a murmur or sound (e.g. ejection click) is made louder by inspiration it is nearly always right sided since right heart blood flow is increased in inspiration.
- ◆ If a murmur is made louder by expiration it may be left sided; however, this is not definite since expelling air from the lungs decreases the amount of air between the heart and chest walls and may increase the intensity of any event whether its source is right or left sided.

Additional heart sounds (➔ Fig 1.8B)

Heart sounds other than S_1 and S_2 are usually abnormal but S_3 and an ejection sound can occur in normal subjects (➔ Table 1.14).

- ◆ S_3 : the S_3 coincides with rapid filling of the LV [33] and in fit young subjects and the athlete (usually young people), often with a slow heart rate and compliant ventricle,

Table 1.13 The second heart sound: common abnormalities

Abnormality	Condition	Explanation
Wide normal splitting	RBBB (Chapter 2)	Delayed RV contraction delays P ₂
Reversed splitting (widening with expiration)	LBBB (Chapter 2) Impaired LV Severe AS (Chapter 21)	Delayed LV ejection makes P ₂ occur after A ₂ . Inspiration makes P ₂ late as usual and narrows splitting
Single S ₂ (no split heard)	Diseased aortic or pulmonary valve (Chapter 21)	No sound from diseased, immobile cusp
Wide fixed splitting (Fig. 1.9)	ASD—moderate or large (Chapter 10)	The ASD allows blood to flow between atria and thus inspiration does not increase RV filling and delays P ₂ . Also partial RBBB delays P ₂

AS, aortic stenosis; ASD, atrial septal defect; LBBB, left bundle branch block; LV, left ventricular; RBBB, right bundle branch block; RV, right ventricular.

LV filling is fast enough to produce a dull thudding third sound. In disease, S₃ is heard either when the LV is abnormal and usually dilated with reduced compliance [34] or when there is rapid filling of the LV, as in severe mitral regurgitation when the normal stroke volume and the blood regurgitated into the left atrium (LA) in the last systole returns to the LV together in early diastole.

- ◆ **Ejection sound:** an ejection sound occurs as the aortic or pulmonary valve opens and is close to S₁ and may be misinterpreted for a split S₁. Ejection sounds are sometimes heard in normal subjects but the most common cause in an asymptomatic patient is a bicuspid aortic valve (Chapter 21).
- ◆ **S₄:** this corresponds to atrial contraction and may be present whenever atrial contraction is powerful, usually secondary to ventricular dysfunction or hypertrophy.
- ◆ **Gallop rhythm (triple rhythm):** an additional sound combined with the normal heart sounds may produce a

sound resembling galloping horses. This is most marked when there is also sinus tachycardia; in this situation of tachycardia a relatively soft S₃ and S₄ may occur at the same time and add to each other to become audible or produce a ‘summation gallop’.

- ◆ **Openingsnap (mitral or tricuspid stenosis) (Chapter 21):** an opening snap occurs when the mitral valve snaps open due to high atrial pressure. An opening snap is louder and earlier when mitral stenosis is more severe and the LA pressure is therefore higher. Its presence denotes a pliable valve and means valvuloplasty is likely to be feasible.
- ◆ **Mid-systolic click(s):** the prolapsing mitral valve tenses in mid/late systole and this produces single or multiple clicks [35]. Their presence or absence plays very little part in assessing this condition.
- ◆ **Prosthetic valves (Chapter 21):** mechanical prosthetic valves usually produce additional sharp clicking opening sounds which correspond to an ejection sound and an opening snap. The closing sound corresponds to the component of the heart sounds contributed by that valve, i.e. mitral component of S₁ for mitral valve and aortic component of S₂ for an aortic valve. As there is normally no sound from the opening of normal heart valves, the high intensity, abrupt metallic click of mechanical valve opening produce additional opening sounds which in the case of the aortic valve has the same timing as an ejection sound, and in the case of the mitral valve occurs when an opening snap would occur. Tissue valves do not normally produce additional sounds. Both mechanical and tissue aortic valve usually causes turbulent flow and an ejection systolic murmur.
- ◆ **Pericardial knock:** in constrictive pericarditis (Chapter 19), a pericardial ‘knock’ may be heard in early diastole and is due to the high pressure atrium rapidly decompressing into a restricted LV producing an audible reverberation.

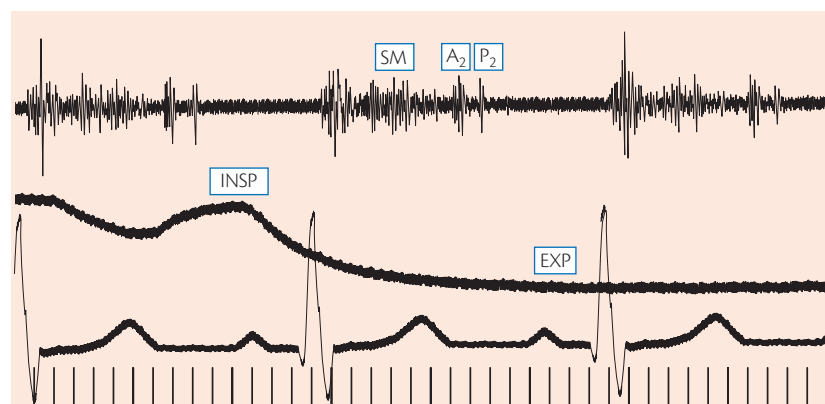


Figure 1.9 Wide, fixed splitting of the second heart sound (S₂). This illustrates that the aortic (A₂) and pulmonary (P₂) components of S₂ are widely split. Note that there is no variation in the splitting with respiration. SM, systolic murmur.

Table 1.14 Clinical implications of additional heart sounds

S ₃	LV impairment or increased filling May be palpable at apex
S ₄	LV impairment or hypertrophy
Gallop rhythm	The heart under stress Often palpable at apex
Ejection sound	Aortic or pulmonary valve opening suggests abnormal valve May occur in hypertension. Commonest cause bicuspid aortic valve
Opening snap	Mitral stenosis (and tricuspid stenosis—rare). High pressure opening of valve Longer the interval from the S ₂ the milder the mitral stenosis since it depends on LA pressure
Mechanical valve clicks	Absent or reduced sounds = <i>danger</i> Mitral valve prosthesis—gap from S ₂ to opening sound is guide to LA pressure

LA, left atrial; LV, left ventricular.

Murmurs

Detecting and interpreting cardiac murmurs is difficult and needs a combination of physiological and cardiological knowledge and experience. In reality, in the developed world if a murmur is heard then it is investigated further with an echocardiogram (➔ Chapter 4). Murmurs are audible vibrations produced by turbulent flow through the heart. They are described using a number of characteristics as shown in ➔ Table 1.15 and the grading of murmur intensity (loudness) is described in ➔ Table 1.16.

Innocent murmurs

Not all murmurs are pathological and ‘innocent’ murmurs are common, occurring in situations where the circulation is hyperdynamic, e.g. normal children [36], but also in pregnancy, fever, anaemia, and hyperthyroidism. It may require

Table 1.15 Description of murmurs

Intensity (loudness)	Graded as 1–6 (or 1–4) (➔ Table 1.16)
Duration	Short to long
Pattern (shape)	Crescendo, decrescendo, variable, plateau, crescendo–decrescendo
Timing	Relation to cardiac cycle, e.g. mid-systolic, ejection systolic, pansystolic, late systolic, early diastolic
Frequency	High or low pitched
Character	E.g. blowing, harsh, rasping, cooing, scraping etc.
Position	Precordial position of maximum intensity
Radiation	Precordial and other (e.g. carotids) radiation of murmur
Variation	With respiration

Table 1.16 Grading of murmur intensity

Grade 1–6	Grade 1–4	Description
1	1	Very faint murmur. Usually only heard with specialist training
2	2	Soft but easily distinguishable murmur
3	3	Loud murmur without an associated thrill (palpable murmur)
4	4	Loud murmur with barely palpable thrill
5	4	Loud murmur with easily palpable thrill
6	4	Loud murmur with thrill, and audible with stethoscope removed from chest wall

an echocardiogram (➔ Chapter 4) to be sure that murmurs really are innocent but they are always systolic, usually soft or moderate in intensity, as well as having a musical rather than either harsh or blowing quality.

Systolic murmurs [37]

The flow of blood through or across a pathological structure generates the murmur and this flow is determined by the pressure difference on opposite sides of the responsible pathology (abnormal valve, septal defect, coarctation, etc.). The sound generated is louder when the pressure difference across the pathological structure is greater and higher velocity of flow and greater turbulence is generated. For example, ➔ Fig. 1.8C illustrates the murmur generated in aortic stenosis (➔ Chapter 21). The murmur does not start until ejection begins and peaks when blood flow is greatest, and consequently, as stenosis becomes more severe, the murmur peaks later in systole. The murmur stops before S₂ since ejection is finished. Thus the murmur has a crescendo/decrescendo character and is contained well within the heart sounds. This type of murmur is described as an ejection murmur. The flow dependence of murmurs means that the murmur gets softer and may disappear if transvalvar flow starts to fall when a lesion is very severe and causes heart failure. Regurgitant systolic murmurs through the AV valves, e.g. mitral regurgitation [38] (➔ Chapter 21), may start immediately isovolumic contraction begins (➔ Fig. 1.8D), i.e. before ejection, since the leak occurs as soon as the pressure rises in the ventricle and continues up until S₂ or slightly beyond. This is because there is a continuing pressure difference between the LV and the LA during this period of time. Often the S₂ is swamped by the end of the murmur. Murmurs of this type that fill the whole of systole are described as pan- or holosystolic. Pansystolic murmurs also occur with ventricular septal defect (VSD), see ➔ Chapter 10. In many patients with mitral regurgitation, however, the valve does not become incompetent until, for example, it has prolapsed

and then the murmur begins in mid- or even late systole and then continues up to and slightly beyond the second heart sounds (➔ Fig. 1.8E). Late systolic murmurs may have a crescendo rather similar to an ejection systolic murmur but are much later in the cycle and run into the S_2 and stop abruptly. This is easily appreciated by the experienced auscultator, particularly if the heart rate is not fast but sometimes a mid- or late systolic click is mistaken for S_2 and the murmur placed erroneously in diastole (see ➔ Key point 11, p.27).

Diastolic murmurs (➔ Figs. 1.8F and 1.8G)

Diastolic murmurs from the AV valves can be extremely difficult to hear. They are often very low pitched and rumbling and the inexperienced auscultator simply thinks they are ambient noise. Typically they are produced by mitral stenosis (➔ Chapter 10) (and, in rare cases, tricuspid stenosis) and these conditions are becoming much less common in the developed world. A mitral diastolic murmur may be accentuated quite considerably by turning the patient on their left side and listening at the cardiac apex with the bell of the stethoscope and/or getting the patient to exercise and then listening again. Mid-diastolic murmurs are accentuated just before the next systole as blood flow across the mitral valve is increased by atrial contraction (➔ Table 1.17). This presystolic accentuation usually disappears when the atrial fibrillation develops but on occasions may persist [39].

Early diastolic murmur

Early diastolic murmurs occur from regurgitation through aortic or pulmonary valves. They are decrescendo and follow the S_2 . This is because the biggest pressure difference between the outflow vessel and the ventricle is at the beginning of diastole. Mild aortic regurgitation (➔ Chapter 21) produces a short, soft, early diastolic murmur which is difficult to hear but this can often be elicited by leaning the patient forward and getting them to breathe out. This brings the heart closer to the chest wall and makes the regurgitation audible. Increasing intensity of the murmur tends to suggest the lesion is becoming more severe, but sometimes there is a paradoxical situation with early diastolic murmurs. When chronic aortic regurgitation is very severe the backflow into the ventricle from the aorta occurs quickly and so the murmur, although loud, is not very long. This is even more striking when there is acute aortic regurgitation due to sudden disruption of the aortic valve by endocarditis, dissecting aneurysm, or trauma. The LV immediately prior to the regurgitation developing is of normal size and the sudden torrential aortic regurgitation fills the ventricle to its full capacity almost instantaneously, and at the time slams the mitral valve shut. This leads to an extremely low cardiac output and a very short murmur [40]. The predominant signs are of a patient with cardiovascular collapse, a sinus tachycardia, and what sounds like a gallop rhythm. The experienced

Table 1.17 Important systolic murmurs: some ways of distinguishing them

Murmur	Lesion	Site	Hints
Ejection systolic	Aortic stenosis	Left upper SB—also often at apex. Radiates to carotids.	Slow rising carotid but often not obvious in the elderly. Apex if palpable forceful but not displaced Young patients' murmur preceded by an ejection click. S_2 variable and often single when the valve is heavily calcified
	Pulmonary stenosis	Right upper SB	Increases with inspiration Ejection click, delayed P_2 may be soft
	ASD		Fixed split S_2 Heaving volume-loaded RV felt at left lower SB if large shunt
	Innocent	All areas Musical quality	May just appear when cardiac output is high
Pansystolic	Mitral regurgitation	Apex and radiates to axilla	Very variable but with valvar mitral regurgitation often blowing and goes up to and into S_2 . Apex heaving. May be MDM and S_3 is severe
	Tricuspid regurgitation	Left sternal border	Increase with inspiration, prominent v wave in JVP, possibly pulsatile liver. Also left parasternal heave—often also signs of pulmonary hypertension
	Ventricular septal defect	Left sternal border	Often harsh and thrill frequently present. Single S_2 with large defect.
Late systolic	Subvalvar mitral regurgitation (MVP, chordal rupture)	Apex—radiates to axilla but also may go to the back, head or neck	May be harsh and preceded by systolic click or clicks at apex. Apex heaving and an MDM and S_3 if MR severe. May be mistaken for early diastolic murmur if preceded by a late click which is mistaken for S_2
Presystolic	Mitral stenosis (and also tricuspid stenosis—very rare)	Apex to left SB	Can be difficult to time and may mistakenly be placed in systole and ascribed to mitral regurgitation. Needs meticulous timing against the carotid

ASD, atrial septal defect; SB, sternal border; MVP, mitral valve prolapse; MDM, mid-diastolic murmur.

cardiologist will immediately recognize acute severe aortic regurgitation as a possible cause of this problem and arrange suitable investigations, including immediate echocardiography. Often aortic valve surgery is lifesaving but if the condition is not recognized it will prove fatal. Pulmonary hypertension (➔Chapter 24) produces an early diastolic murmur which tends to be slightly lower pitched than the aortic murmur. The early diastolic murmur is heard at the upper left sternal border and follows the loud pulmonary component of the S₂ due to pulmonary hypertension.

Continuous murmurs

Continuous murmurs are rare in adults. They are exactly what they say they are, a murmur which continues throughout the cardiac cycle (➔Fig. 1.8H) [41]. The systolic component is usually louder than the diastolic component but the overall effect is that there is no break in the sound and the term ‘machinery murmur’ is extremely appropriate, as it sounds like heavy machinery working in the background. This may be due to a patent ductus arteriosus (➔Chapter 10) that has been missed in childhood, but most commonly in adults it is due to some acute communication developing between the right and left side of the heart through which flow occurs both in systole and diastole. The commonest situation is a ruptured sinus of Valsalva although infective endocarditis (➔Chapter 22) can result in arteriovenous and right/left heart communications.

Bruits and conducted murmurs in the carotids

A systolic noise over the carotids may:

- 1) be conducted from the heart—usually from aortic valve although anteriorly directed loud mitral murmurs may also go to the neck. The same noise will be heard in neck and chest;
- 2) originate from disease in the carotid in which case it is only heard in the neck.

Clearly it may be impossible to be sure if there is both carotid and valve disease or valve disease alone in situation (1).

The radiation of cardiac murmurs

The radiation of cardiac murmurs is complex and in essence any cardiac murmur from any structure can be heard anywhere in the chest. There are, however, typical areas—apical/mitral, pulmonary, aortic, and tricuspid areas, with radiation to the carotid, back, and/or axilla. It should be remembered that loud murmurs from mitral valve prolapse and ruptured chordae can radiate anywhere and can go into the neck and sound very like aortic stenosis murmurs. Furthermore, it is common for the murmur of aortic stenosis in elderly patients to be louder at the cardiac apex than it is over the aortic area, which is its classical site.

This is because in elderly patients with hyperinflated lungs there is more lung between the upper part of the heart and the chest wall, i.e. around the aortic area, than there is at the cardiac apex where the heart usually remains in contact with the chest wall. Aortic murmurs that are heard only at the cardiac apex often radiate into the neck and can be heard over the carotids.

Other features of auscultation

A ‘pericardial rub’ is associated with pericarditis (➔Chapter 19) and is due to the inflamed visceral and parietal pericardium rubbing together with each heartbeat, producing an intermittent scratchy sound with systolic and diastolic components usually best heard with the patient recumbent, and can, on occasions, disappear when the patient sits forward, a position that also tends to relieve the discomfort of pericardial pain. Always think of the diagnosis if you see a patient leaning forward in bed.

Examination of other systems

The presence of pulmonary oedema and/or pleural effusion on chest examination is an integral part of assessing the cardiac patient, as is examination of the abdomen for ascites, splenomegaly, hepatomegaly (pulsatile and non-pulsatile), and the presence of abdominal aortic aneurysm or abdominal bruits which may indicate the presence of more extensive vascular disease or renal artery stenosis (➔Chapter 13).

Examination scenarios

Although it would be nice to have ideal conditions and unlimited time available to examine all patients in a thorough and systematic way, clinical reality is often very different and we need to tailor our examination to circumstances dictated by the clinical situation

Acute chest pain

Chest pain is the commonest reason for acute cardiac hospital admission. Performing an ECG to determine if there is ST elevation should take priority unless the patient is moribund.

ST elevation myocardial infarction (➔Table 1.18)

If a diagnosis of ST elevation myocardial infarction (STEMI) is confirmed the first priority is rapid reperfusion by thrombolysis or primary percutaneous coronary intervention. History and examination should concentrate on establishing the safety and feasibility of reperfusion and the presence of complications of acute myocardial infarction (e.g. ventricular septal defect or a ruptured papillary muscle). In patients with ST elevation alternative diagnoses

Table 1.18 Important examination points in STEMI

General examination
Responsiveness of patient
Breathing
Peripheral vasoconstriction
Sweating
Bruising/bleeding
Pulses in both arms
Aortic dissection
Blood pressure
Hypotension requiring circulatory support
Hypertension contraindicating thrombolysis
JVP
Right heart failure
Right ventricular infarction
Systolic murmur
Post-infarction ventricular septal defect
Acute mitral regurgitation
Aortic stenosis
Chest auscultation
Pulmonary oedema
Femoral pulses
Primary percutaneous coronary intervention patients
Gross neurology (in case a cerebral complication of treatment occurs)

must be considered, especially where some aspect of the history and examination does not fit well with a presumed STEMI. Pericarditis may masquerade as STEMI although the ECG is often quite different. A pericardial rub does not contribute much as it may also be secondary to an MI. The echocardiogram may show pericardial fluid in pericarditis but more importantly in the patient with a STEMI there will be reduced LV contraction in the infarcting area. A history of sudden, searing chest pain, combined with differential pulses and/or blood pressure would suggest the possibility of acute aortic dissection, including the origin of the coronary arteries.

In a patient with extreme pallor and hypotension following thrombolysis the possibility of occult bleeding should be seriously considered (clinical scenario) (see ↻Key point 12, p.27).

Non-ST elevation myocardial infarction chest pain: ?cause

If there is no ST elevation on an ECG, history and examination will be targeted at diagnosis and there will be less time pressure. More subtle aspects of cardiac examination

such as the presence of a dyskinetic apex beat or a S_4 might suggest coronary artery disease whereas a raised JVP, dyspnoea, and tachycardia might point to the possibility of pulmonary embolism. The presence of a pericardial rub may suggest pericarditis alone but it is worth remembering that this can be secondary to myocardial infarction. Pain elicited by pressure on the chest can be very misleading (see ↻Key point 13, p.27).

Acute cardiovascular collapse

There is often little clinical history from the patient, and examination is crucial in pointing to the correct diagnosis and initiating potentially life-saving treatment. Do not forget to talk to the family and the ambulance crew. The clinician must examine the patient swiftly and accurately and be able to multitask, combining examination with initial treatment while assimilating all the available information from history, examination, initial investigations, and responses to initial treatment. The clinician must give both leadership and guidance to the team.

Initial examination is similar to that for a STEMI patient (↻Table 1.18). Tachycardia may result from an arrhythmia such as ventricular tachycardia or simply sinus tachycardia as a response to acute problem. Bradycardia may indicate the presence of heart block or sinus arrest and immediate external pacing may be necessary. Most acutely unwell patients will be hypotensive, but the presence of hypertension may suggest aortic dissection either because it is the underlying cause or the renal arteries are involved, pulmonary oedema, or a non-cardiac (cerebral) cause; patients with subarachnoid haemorrhage may have striking ECG changes, high blood pressures, and altered levels of consciousness. Vasodilatation suggests infection whereas vasoconstriction may suggest pump failure and different initial circulatory support requirements.

A patient with pulmonary oedema will want to sit up whereas a patient with cardiac rupture (↻Chapter 16) or tamponade or acute pulmonary embolus (↻Chapter 37) may be unable to do so because they become hypotensive. Oxygen saturation, although strictly an investigation, is a valuable part of initial examination. It can also be useful to know the early response to any treatment, such as oxygen therapy administered by paramedics prior to admission, so discussion with paramedics and examination of the paramedic charts may reveal a rapidly changing situation.

Examination is much more productive if the possible diagnoses are running through the cardiologist's mind as they examine the patient (see ↻Key point 13, p.27).

Patient with hypotension following invasive cardiac investigations or percutaneous coronary intervention

The situation that must be considered immediately is cardiac tamponade due to a perforation of a cardiac chamber or a coronary artery during the procedure. It is essential to rule this out by immediate echocardiography. The physical signs of tamponade may be present, but they may be very difficult to elicit in the acutely distressed hypotensive patient. Having said this, by far the most common cause of this problem is a vasovagal reaction, often attributable to a degree of dehydration prior to the procedure, and fear and discomfort. There is often bradycardia associated with this which will respond to atropine and in addition a quick fluid load and raising the foot of the bed will often recover the situation within a short period of time. Other possibilities are acute myocardial infarction due to an embolic event, cerebrovascular problems, retroperitoneal bleeding, and catheter-induced arterial dissection. An early ECG is an essential investigation.

Patients with multiple injuries (e.g. road traffic accident)

Major cerebral or orthopaedic injuries may seem most prominent but associated cardiac injury may be life threatening and should be considered in all patient with chest trauma. Three key areas of concern from a cardiovascular standpoint are direct traumatic injury, cardiac tamponade, and aortic transection or rupture.

Physical examination may detect cardiac tamponade and there may be missing pulses due to aortic injury. The other circulatory consequences of major trauma are more likely to be recognized with appropriate imaging (echocardiography, X-ray, or computed tomography) in response to a high level of suspicion. All patients with chest trauma need an ECG which may identify myocardial contusion or even transection of the left anterior descending coronary artery with STEMI.

Conclusion

The history and the examination are the everyday tools of the clinical cardiologist and used correctly often allow rapid diagnosis, and at other times point investigations in the correct direction from an early stage. Effective use of these tools requires thoughtful application and considerable experience. There are some golden rules:

- ◆ Listen to the patient.
- ◆ Speak to a witness.

- ◆ Record both positive and negative features of the history and examination accurately.
- ◆ If a sign cannot be elicited, e.g. a JVP cannot be seen, record it as 'not seen' instead of 'normal'.
- ◆ Tailor the history and examination to the clinical situation. In a real emergency multitasking is essential, i.e. the history must be taken while the patient is examined and while first-line resuscitative treatment is begun.
- ◆ Always seek to confirm or refute definitively the more serious of two possible diagnoses. Do not assume the less serious diagnosis is the cause of the problem and discharge the patient.
- ◆ Repeat the history and examination as appropriate. Examination may need to be frequent, e.g. in the acutely ill patient with a myocardial infarction or with endocarditis. The findings must be recorded on each occasion accurately.
- ◆ Know how reliable physical signs are (↻ Table 1.19). If one of the 'top signs' is present do not be reassured by other features, e.g. the patient who feels reasonably well but has a persistent sinus tachycardia and a reduced oxygen saturation may well be on the edge of cardiovascular collapse. Young patients in particular accommodate a serious illness remarkably well until the moment when they collapse.

Table 1.19 Significance of some important physical signs

Top signs (signs that must not be ignored and are usually easy to elicit)	Difficult signs (often need considerable experience to detect and understand their significance)
◆ Pallor and sweating	◆ JVP level and character
◆ Hypotension	◆ Carotid pulse character
◆ Tachycardia or bradycardia	◆ Minor irregularities of the pulse
◆ Dyspnoea at rest and raised respiratory rate	◆ Subtle added heart sounds
◆ Low pulse volume	◆ Soft murmur
◆ Pulsus paradoxus	◆ Splinter haemorrhages
◆ Loud systolic murmur	
◆ Unequal pulses	
◆ Reduced oxygen saturation (cyanosis)	

Personal perspective

History taking and physical examination are basic cardiological skills that have changed very little over the years and require to be honed by frequent use. Technology, particularly in the form of echocardiography, has allowed validation of many of the auscultatory signs which are the most difficult in cardiology. Consequently the fine detail of auscultation has, to some extent, become less

important as long as the echocardiogram is available. The more widespread use of small handheld echocardiography machines is likely to be a very valuable adjunct to examination for the clinician, particularly in high-volume clinics. However, all the technology in the world is no substitute for clinical experience and for being able to answer the question as to whether the patient is ill or not. The most important question is usually not ‘what do the tests show?’ but ‘how well is the patient?’.

Table 1.20 Popular examination eponyms in cardiology

Eponym	Condition	Description
Austin Flint murmur	Aortic regurgitation	Mid-diastolic murmur due to partial closure of mitral valve by jet of aortic regurgitation
Becker's sign	Aortic regurgitation	Pulsation of the retinal vessels on fundoscopy
Broadbent's sign	Constrictive pericarditis	Intercostal indrawing during systole
Carey–Coombs murmur	Rheumatic fever	Early diastolic murmur associated with acute mitral valve inflammation
Carvalho's sign	Tricuspid regurgitation	Increasing systolic murmur intensity with inspiration
Cheyne–Stokes respiration	Heart failure	Periodic or cyclic respiration pattern
Corrigan's pulse	Aortic regurgitation	Collapsing pulse with rapid upstroke and decline, typical of aortic regurgitation
De Musset's sign	Aortic regurgitation	'Head nodding' sign in time with cardiac cycle associated with excessive pulsation from aortic regurgitation
Duroziez's sign	Aortic regurgitation	Diastolic murmur heard over femoral pulses when partly occluded below the stethoscope
Graham Steell murmur	Pulmonary regurgitation	Murmur of pulmonary regurgitation when caused by pulmonary hypertension
Janeway lesions	Infective endocarditis	Slightly raised, non-tender haemorrhagic lesions of palms of hands and/or soles of feet
Kussmaul's sign	Constrictive pericarditis	Elevation of the JVP with inspiration
Mueller's sign	Aortic regurgitation	Cyclic pulsation of the uvula in aortic regurgitation
Osler's nodes	Infective endocarditis	Small tender, purple, erythematous skin lesions due to infective emboli usually seen on fingers and toes or palms of hands/soles of feet
Quincke's sign	Aortic regurgitation	Capillary pulsation in the nail beds due to aortic regurgitation
Roth spots	Infective endocarditis	Retinal haemorrhages with central white spots, usually near the optic disc
Still's murmur	Innocent murmur	Rare but most commonly seen in children and due to vibration of normal pulmonary valve leaflets
Traube's sign	Aortic regurgitation	'Pistol shot' systolic sound in femoral arteries on auscultation

Key points

Key point 1

Let the patient talk and tell the story in their own words.

Key point 2

Use all the information available:

- ◆ Look at all ambulance records in acute patients, e.g. the ambulance crew may have observed a low oxygen

saturation while the patient was in the ambulance which has resolved by the time they arrive in the hospital. They may also have detected transient hypotension, tachycardia, or bradycardia.

- ◆ Look at the nursing records.

Key point 3

Patients who present as emergencies are usually ill even if the symptoms are lessening by the time they see the doctor

Key point 4

Beware chest pain induced by chest pressure—it does not exclude cardiac chest pain. Acute musculoskeletal chest pain, severe enough to bring a patient to hospital, usually has an identifiable trigger, e.g. a fall with bruising.

Key point 5

Patients in middle and old age who have not had previous psychiatric problems do not suddenly develop hyperventilation as a cause for their symptoms. Ascribing symptoms to hyperventilation can be extremely dangerous.

Key point 6

Presyncope: most patients have had a similar feeling to cardiovascular presyncope when standing up quickly, particularly from bending or squatting on a hot day. If the patient is reminded of this they may then make the connection.

Key point 7

Palpitations and syncope:

- ◆ If there are eye witnesses speak to them.
- ◆ Rotational element to symptoms strongly suggests a neurological cause.
- ◆ Tell the patient to ‘tap out’ the arrhythmia.
- ◆ The patient who has unpredictable syncope and who has driven to the clinic should not be allowed to drive home.

Key point 8

Always think of these conditions during general examination: anaemia, diabetes mellitus, thyrotoxicosis, hypothyroidism, infective endocarditis

Key point 9

Observing the patient entering the consulting room and getting onto the examining couch often reveals important clinical information.

Key point 10

A persistent tachycardia in isolation may be an ‘early warning sign’ of circulatory stress, e.g.

- ◆ Impaired LV function.
- ◆ Pulmonary thromboembolism.

Key point 11

Severe valve lesion and little or no murmur.

- ◆ Critical aortic stenosis and low output.
- ◆ Severe paraprothhetic mitral regurgitation.
- ◆ Sudden acute aortic regurgitation, e.g. endocarditis or dissection.
- ◆ Severe mitral stenosis (occasionally).

Key point 12

Always consider possible alternative diagnoses:

- ◆ Aortic dissection.
- ◆ Coronary embolism.
- ◆ Pericarditis—thrombolysis may produce haemopericardium.
- ◆ ST elevation not new—other causes of chest pain.

Key point 13

Always consider the following in a patient with cardiovascular collapse

- ◆ Cardiac arrhythmia.
- ◆ Aortic dissection.
- ◆ Pulmonary embolism.
- ◆ Pericardial tamponade.
- ◆ Contained cardiac rupture.
- ◆ Post infarct ventricular septal defect/mitral regurgitation.
- ◆ Infective endocarditis or other form of sepsis.
- ◆ Cardiogenic shock due to myocardial infarction.
- ◆ Acute severe aortic/mitral regurgitation.
- ◆ Prosthetic valve dysfunction.

Further reading

Douglas G, Nicol F, Robertson C (eds.). *Macleod's Clinical Examination*, 11th edn., 2005. Edinburgh: Elsevier Churchill Livingstone.


Epstein O, Perkin DG, Cookson J, et al. *Clinical Examination*, 4th edn., 2008. London: Mosby Elsevier.


White PD. *Heart Disease*, 1931. New York: The Macmillan Company.

Wood P. *Diseases of the Heart and Circulation*, 1950. London: Eyre and Spottiswoode.

Online resources


The following websites are useful sources of medical images:

 <http://images.google.com/>

 <http://www.healcentral.org/index.jsp>

 <http://www.images.md/users/index.asp>

 <http://www.omnimedicalsearch.com/images.html>

 **For full references and multimedia materials please visit the online version of the book (<http://esctextbook.oxfordonline.com>).**