Chapter 1

Taking a cardiovascular history

Introduction

In an era when imaging and other diagnostic techniques have become more numerous and sophisticated, there is a tendency for the clinical assessment of the patient to seem less relevant. This is wrong, for the importance of the clinical examination cannot be overemphasized. However much information is gathered by other means, decisions about the correct management of patients still rely fundamentally on a good overall clinical assessment. It is bad practice to put patients through more than the minimum number of investigations needed to make a diagnosis and allow patient management to be planned, some of which may be uncomfortable and have associated risks. The good clinician selects investigations carefully and sparingly, always balancing the potential value of any information that may be obtained against the risks, cost and discomfort to the patient.

Establishing a good rapport and obtaining the confidence of a patient start at the time of the clinical assessment and are extremely important later, when sometimes distressing information has to be discussed and difficult decisions made about potential treatment. The clinical assessment will often involve obtaining information from relatives or friends of the patient, who may be able to judge more objectively than the patient the extent of their limitations in daily activities.

The clinical assessment consists of obtaining a history to elicit symptoms, and performing a physical examination to observe signs, of cardiovascular disease. In this chapter, broad principles will be covered, but symptoms and signs relating to specific conditions will be detailed in the chapters relating to those conditions.

The art of good history taking involves allowing the patient to tell their story whilst, at the same time, through the questions asked, directing their attention to those aspects of their clinical presentation which are most likely to provide information that is relevant to making a diagnosis and determining treatment. Often a patient will unwittingly give vitally important clinical information almost as an aside remark, information that may never have come to light if the history taking consisted merely of asking numerous predetermined questions or, worse, providing a questionnaire for the patient to complete. Taking time to obtain a clear history, in patients’ own words and in their own time, is an important part of clinical training. As the clinician’s expertise increases, this process can be achieved in relatively short periods of time. With experience, helpful information can also be gleaned from other sources, such as the patient’s attitude, demeanour, emotional state and dress.

Specific symptoms (see Boxes 1.1 and 1.2)

Because cardiac work is so closely related to exercise, many cardiac symptoms are worse during exertion, the effect of which should be enquired about specifically.
Dyspnoea

Defined as an abnormal and uncomfortable awareness of breathing, dyspnoea is a common symptom of both cardiac and respiratory disease and is most commonly observed on exertion. This is unlike the breathlessness associated with anxiety where a heightened awareness of respiration progresses to hyperventilation, and where the sensation of dyspnoea is often worse at rest or in stressful situations. Hyperventilation also causes other symptoms (many of which are due to the fall in arterial $PCO_2$ and alkalosis), such as peri-oral and peripheral paraesthesiae, clouding of consciousness, stabbing left infra-mammary chest pain and, in extreme cases, tetany. As the underlying cardiac condition progresses, the sensation of dyspnoea becomes present at ever lower levels of exertion, and ultimately occurs at rest (see Box 1.3 for causes of dyspnoea).

Dyspnoea due to cardiac disease arises due to pulmonary venous congestion. Left atrial pressure, and hence pulmonary venous pressure, is normally around 5mmHg. When it rises, as will occur with mitral and aortic valve disease or left ventricular dysfunction, the pulmonary veins become distended and the bronchial walls congested and oedematous, causing an irritating non-productive cough and wheeze. As pulmonary venous pressure rises further and the plasma oncotic pressure (around 25 mmHg) is exceeded, so the lung tissue becomes stiffer due to interstitial oedema (increasing the muscular work required to inflate the lungs and the sensation of dyspnoea), a transudate collects in the alveoli, and pulmonary oedema results. As this worsens, frothy sputum is expectorated, which may be pink due to ruptured small bronchial vessels bleeding into the oedema fluid.

Cardiac dyspnoea is worse when lying flat (orthopnoea), may wake the patient from sleep in the early hours of the morning associated with sweating and anxiety (paroxysmal nocturnal dyspnoea) and tends to be relieved by sitting upright or standing. Systemic venous return to the right heart is increased in the recumbent position, especially in the early hours of the morning when
Chest pain

Chest pain or choking discomfort due to myocardial ischaemia (angina) typically has certain characteristics: a tight, constricting, band-like, or sometimes burning, retrosternal discomfort, occurring principally on exertion and relieved within minutes by rest or sublingual nitrates. Patients usually describe this as an uncomfortable rather than a truly painful sensation. The discomfort may radiate to either arm (most commonly the left), to the neck and jaw, or through to the back or abdomen. An attack is normally short lived, lasting up to 20 min. Angina is sometimes atypical, causing neck, throat, jaw, back or abdominal discomfort without chest symptoms.

Angina is due to an imbalance between myocardial oxygen supply (coronary blood flow) and demand (myocardial oxygen consumption). The most common cause of angina is therefore coronary artery disease, but it may occur even with normal coronary arteries in conditions of severe left ventricular hypertrophy or dilatation where myocardial O₂ demand is high (see Chapter 9). Angina that occurs at rest or is rapidly worsening is termed ‘unstable angina’, and usually indicates critical coronary disease. Anginal pain lasting more than 30 min, and especially if associated with sweating, nausea and vomiting, should make one suspicious of myocardial infarction. Stabbing pain or episodes of pain lasting only seconds suggests a musculoskeletal cause.

Patients may describe exertional breathlessness rather than chest pain but, when pressed to be more precise, it is often the sensation of heaviness of the mid-chest (angina) that gives rise to a feeling of difficulty in expanding the chest. Breathlessness may, however, be due to the associated left ventricular dysfunction that occurs with myocardial ischaemia. Other symptoms that may be associated with myocardial ischaemia include belching, indigestion, nausea and dizziness, although their association with exertion is usually a consistent feature. Symptoms occurring above the mandible and below the umbilicus are very unlikely to be due to myocardial ischaemia. See Box 1.5 for the

**Box 1.4 New York Heart Association (NYHA) classification of heart failure**

Describes the degree of disability from dyspnoea due to cardiac disease:
- Class I: no limitation in physical activity
- Class II: slight limitation of exercise (fatigue, dyspnoea)
- Class III: marked limitation of activity (comfortable at rest but slight exertion causes symptoms)
- Class IV: symptoms even at rest

**Box 1.5 Canadian Cardiovascular Society (CCS) classification**

Describes the degree of disability caused by angina:
- Class 1 — Angina only on strenuous or prolonged exertion
- Class 2 — Slight limitation due to angina with normal activities
- Class 3 — Marked limitation due to angina with ordinary activity
- Class 4 — Unable to undertake any physical activity. Angina at rest
Canadian Cardiovascular Society (CCS) classification: the most commonly used classification to describe the degree of disability caused by angina.

Almost any structure in the chest may also cause chest pain, but some of the more common are given in Box 1.6. Usually a careful history will allow these causes to be differentiated. Perhaps the most common differentiation lies between cardiac pain and oesophageal spasm or reflux. The character of the pain caused by oesophageal spasm may be indistinguishable from angina and may be relieved by vasodilators, such as the nitrates. Oesophageal causes of pain often last longer than anginal episodes, are rarely related to exertion, and oesophageal reflux tends to be worse on bending or lying down. Functional or psychogenic chest pain (Da Costa syndrome) may occur in patients with a fear of heart disease, for instance due to a family history of myocardial infarction. It manifests as a dull, persistent ache in the area of the cardiac apex lasting hours or days, and is often interspersed with more intense stabbing episodes. This pain may be associated with hyperventilation, palpitation and panic attacks. Chest pain is also well described in patients with mitral valve prolapse, although the reason for this is not known.

**Syncope**

Loss of consciousness may be caused by a number of cardiovascular causes, but their final common pathway is a reduction in cerebral blood flow. A careful history will often suggest the underlying cause, but there are a number of patients who have transient dizzy or syncopal episodes that defy cardiological and neurological diagnosis. See Box 1.7 for cardiovascular causes of syncope and

### Box 1.7 Cardiovascular causes of syncope and presyncope

- **Aortic stenosis (AS):** usually exertional, or at rest with the onset of AF (atrial fibrillation) or heart block
- **Left ventricular outflow tract obstruction (LVOTO):** as occurs with hypertrophic obstructive cardiomyopathy (HOCM). Syncope may be due to associated arrhythmias as well as LVOTO
- **Tachyarrhythmias:** may be associated with awareness of palpitation
- **Heart block:** patient may be aware of bradycardia or pauses
- **Hypotensive drugs:** symptoms often postural
- **Vasovagal syndrome:** often occurs in painful situations, after standing up or prolonged standing, with emotional stress. Attacks often occur over many years
- **Carotid sinus syndrome:** sensitivity of carotid sinus to neck movement or palpation results in vagal stimulation, causing bradycardia and hypotension
- **Myocardial ischaemia:** rarely causes syncope in the absence of other cardiac disease (e.g. aortic stenosis) except if left main stem coronary artery is stenosed
- **Severe pulmonary hypertension:** usually exertional. Mechanism comparable with hypertension or AS. Fixed obstruction to circulatory blood flow
- **Acute pulmonary embolism:** only when massive embolism produces circulatory obstruction
- **Subclavian steal syndrome:** due to severe subclavian artery stenosis or occlusion causing ‘steal’ of blood by retrograde flow down the vertebral artery. Occurs with ipsilateral arm movement
- **Cerebrovascular disease:** often causes dizzy spells (transient ischaemic episodes), and mainly in the elderly
- **Atrial myxoma:** rare. Symptoms may be posturally related. Produces intermittent mitral valve obstruction

### Box 1.6 Non-myocardial causes of chest pain

**Acute**

- **Oesophageal spasm:** very similar to angina but more prolonged and unrelated to exertion
- **Thoracic aortic dissection:** usually felt inter-scapularly
- **Pneumonia:** usually pleuritic but may be more diffuse ache
- **Pneumothorax:** localized, intense, pleuritic
- **Pulmonary embolus:** either pleuritic and localized or dull central discomfort
- **Pericarditis:** varies with position and respiration

**Chronic**

- **Costochondritis (Tietze syndrome):** localized, tender area of chest wall
- **Peptic ulceration**
- **Gall bladder disease:** usually abdominal symptoms also present
- **Pancreatic disease**
- **Cervical or thoracic spine disease:** related to movement
presyncope. Cardiac syncope is usually of rapid onset, without an aura, and is usually not associated with convulsions or incontinence. Recovery is typically rapid (unlike the slower recovery of neurological causes which may cause postsyncopeal confusion), and may be associated with profound vasodilatation as blood supply is restored to arterioles that have become vasodilated by the accumulation of local metabolites. A gradual reduction of consciousness is more suggestive of vasodepressor syncope or postural hypotension.

**Palpitation** (see also Chapter 13)

This is a common symptom and is defined as an unpleasant awareness of the heart beating. At the outset it is important to determine exactly what sensation the patient is describing. It may be an awareness of the heart beating more forcefully than usual, more rapidly, more slowly, erratically, or a combination of these.

- An awareness of a forceful beat may suggest an increased stroke volume (e.g. aortic or mitral regurgitation) or may just represent an individual’s heightened awareness of their heart.
- Rapid palpitation suggests a tachycardia.
- An erratic palpitation may be fast, as in atrial fibrillation, or slower, as in an awareness of ectopic beats.
- With ectopics the patient may be aware of the prematurely occurring ‘extra beat’ (ectopic), the compensatory pause after the ectopic which may give the sensation of a ‘missed beat’, or of the post-ectopic beat which is accentuated and felt as a ‘more forceful’ beat because, occurring later, it has a larger stroke volume than the preceding sinus or ectopic beats.

Palpitation associated with a slow rate may be due to atrioventricular block or sinus node disease. Rapid palpitations usually start and stop suddenly, and imply an atrial, atrioventricular nodal or ventricular tachycardia. A gradual termination of the palpitation is more in keeping with a sinus tachycardia.

**Oedema**

An elevation in right heart pressure increases systemic venous pressure in the inferior and superior venae cavae, and this will be greatest in the most dependent parts of the body, most usually the feet and ankles, but will be the sacral region in those confined to bed. Oedema occurs when plasma oncotic pressure is exceeded by the raised intravascular pressure, a situation which is exacerbated in hypoalbuminaemic states.

- Elevation of right heart pressure may be secondary to left heart disease (left ventricular failure, mitral or aortic valve disease) or may be due to right heart failure as a consequence of pulmonary hypertension, right ventricular or constrictive pericardial disease.
- Oedema due to superior vena cava obstruction (usually caused by malignancy) is obviously confined to the head, neck and arms.
- A history of periorbital oedema is characteristic of renal disease (nephrotic and nephritic syndromes).
- Unilateral oedema of a limb implies local vascular or lymphatic obstruction, as occurs following deep venous thrombosis or chronic venous insufficiency due to varicose veins.
- Other causes of oedema include the cyclical oedema that may occur perimenstrually and angioneurotic oedema that occurs as an allergic reaction to various stimuli, including seafood.

**Fatigue**

This is a non-specific but common symptom in cardiac disease. It may arise due to a low cardiac output or an inability to raise cardiac output sufficiently on exercise. Drug therapy may cause fatigue, either directly as in the case of β-blockers, or indirectly such as that due to hypokalaemia caused by diuretic therapy.

**Cyanosis**

As well as being a sign which should be sought on examination, patients may complain of a bluish discoloration of the skin and mucous membranes, and thus cyanosis may also be a presenting symptom. The blue discoloration arises as a result of the
presence of increased amounts of deoxygenated haemoglobin in the blood perfusing the tissues. Cyanosis can be divided into ‘peripheral’ and ‘central’, terms which indicate the cause of the cyanosis rather than where it is observed.

● **Peripheral cyanosis** is usually due to cutaneous vasoconstriction, because of either exposure to cold or Raynaud’s phenomenon. Cyanosis is most readily seen when cardiac output is reduced for any reason. Whereas cyanosis of central origin usually worsens on exercise, peripheral cyanosis is usually unchanged if cardiac output is poor, or may improve with reflex vasodilatation if the principal abnormality is vasoconstriction.

● **Central cyanosis** is characterized by decreased arterial oxygen saturation, due to central venous-arterial admixture of blood in conditions causing right-to-left shunting, or due to pulmonary disease causing impaired arterial oxygen uptake. Right-to-left shunting may be intra-cardiac in origin (cyanotic congenital heart disease involving absence of or defects in ventricular or atrial septa) or may be extra-cardiac (pulmonary arteriovenous malformations). Central cyanosis is best observed by examining the oral mucous membranes and is clinically apparent when >40 g/L of deoxygenated (reduced) haemoglobin is present. Cyanosis of central origin is usually not improved by giving higher concentrations of inspired O₂, whereas peripheral cyanosis may be. In darker skinned individuals, cyanosis may not be observed until greater levels of reduced haemoglobin are present. More rarely cyanosis may be due to the presence of abnormal haemoglobin pigments, such as methaemoglobin. When there is a central cause for the cyanosis, peripheral cyanosis must also be present, whereas cyanosis due to a peripheral cause will not result in cyanosis of the mucous membranes.

**Claudication**

This aching discomfort in the legs, usually the calves, occurs after varying amounts of exercise, and is due to skeletal muscle ischaemia as a consequence of peripheral vascular disease. Since this is almost always atheromatous, the presence of claudication should alert one to the probability that the patient also has underlying coronary artery disease.

**Additional history**

A full medical history should be obtained from the patient. Box 1.8 details what this should include.

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**Box 1.8 Patient history**

A full medical history for the patient should include:

● **Systems review**: for urinary, menstrual and gastrointestinal symptoms

● **Drug history**: specific cardiovascular drugs, contraceptive pill, other medication (e.g. treatment for indigestion)

● **Past medical history**: for tuberculosis, rheumatic fever, diabetes mellitus, hypertension, stroke, venereal or tropical diseases, thyroid disease, asthma, previous operations

● **Social history**: exercise, occupation, smoking, alcohol consumption, family/partner

● **Family history**: for any cardiovascular or other possibly genetically linked disease