1 Cardiovascular conditions

Atheroma

Atheroma (atherosclerosis) is characterised by the accumulation of cholesterol and lipids in the arterial intimal surface. Atheroma has a patchy distribution and, depending on the site and extent of disease, can give rise to a variety of clinical presentations (Table 1.1). A platelet–fibrin thrombus (clot) may form, break up and travel in the bloodstream (thrombo-embolism) with potentially life-threatening consequences. Alternatively, atheromatous plaques may rupture and 'heal' spontaneously.

Table 1.1 Common sites of atheroma and clinical presentation

Artery site	Clinical presentation
Cerebral	Cerebrovascular accident – stroke Transient ischaemic attacks (TIAs)
Coronary	Coronary artery disease – chest pain (angina pectoris) Arrhythmias Myocardial infarction
Peripheral	Intermittent claudication Resting leg pain Infarction leading to gangrene

Coronary artery disease

Coronary artery disease (CAD) is caused by atheroma. It is the leading cause of death in the UK and results from a combination of genetic and lifestyle factors. Irreversible (fixed) risk factors include:

- Increasing age
- Gender: men are at greater risk than premenopausal women
- Family history of CAD.

Potentially reversible (modifiable) risk factors for CAD include:

- Cigarette smoking
- High blood cholesterol level: low density lipoproteins (LDL) are associated with a high risk of CAD, whilst high density lipoproteins (HDL) appear to be anti-atherogenic

- Hypertension
- Diabetes mellitus
- Obesity and lack of exercise.

Clinical features

The clinical presentation of CAD is reflective of the degree and duration of impaired coronary blood flow. Features include dizziness, shortness of breath, decreased exercise tolerance, chest pain (angina pectoris) and sometimes sudden death due to a catastrophic myocardial infarction (irreversible damage to cardiac muscle). Xanthelasmata may signify hyperlipidaemia. A chronically reduced blood supply to the myocardium progressively damages the heart muscle and may lead to cardiac arrhythmias and cardiac failure.

Clinical signs

Distended neck veins (Figure 1.1) due to increased jugular venous pressure (JVP), are a classic sign of right-sided cardiac failure, although it may also be seen in hypervolaemic states, superior vena cava obstruction and cardiac tamponade. The causes of cardiac failure may also include cardiac valvular disease

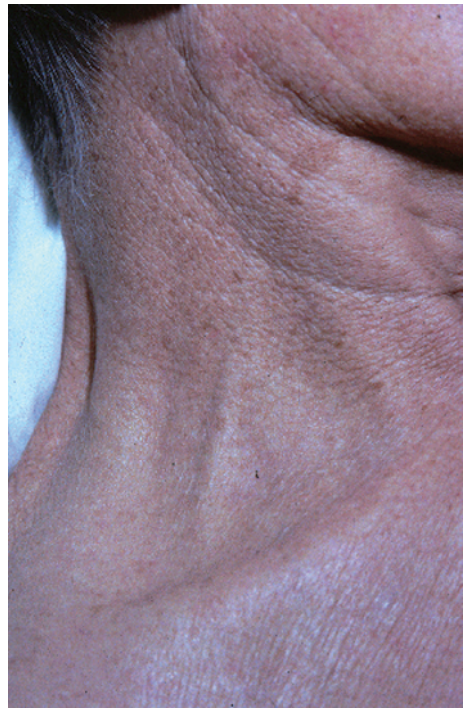

Figure 1.1 Distended neck veins (raised JVP) are a classic sign of right-sided cardiac failure

and chronic obstructive pulmonary disease. Pitting oedema may be demonstrated by applying firm digital pressure over the lower legs or ankles.

Hyperlipidaemia may predispose to CAD [sometimes premature]. The combination of corneal arcus with xanthelasma (Figure 1.2) should suggest the possibility of hyperlipidaemia. This is especially the case in young people where autosomal dominant familial hypercholesterolaemia may be the underlying cause. Other causes of xanthelasma (but not corneal arcus) include hypothyroidism and primary biliary cirrhosis.

Figure 1.2 Hyperlipidaemia is suggested by the combination of corneal arcus with xanthelasma

It is thought that if an individual has diagonal creases (Figure 1.3) on both ear lobes, there may be some benefit in undergoing screening to exclude the possibility of cardiovascular disease. The actual cause of earlobe creasing is unknown but it is possible that chronic circulatory problems allow the vascular bed in the earlobe to collapse and the telltale earlobe crease to appear. In one study the presence of a unilateral earlobe crease was associated with a 33% increase in the risk of a myocardial infarct; the risk increased to 77% when the earlobe crease appeared bilaterally.

Vertex baldness also appears to be a valid marker for an increased risk of cardiovascular disease, particularly when clustered with other factors such as hypertension or hypercholesterolaemia. Other factors include being short and having an 'apple-shaped' physique.

The American Academy of Periodontology recently showed that people with periodontal disease are 200–300% more likely to experience a heart attack than those with healthy periodontium, making periodontal disease a possible risk for cardiovascular disease.

Atheroma, trauma, orbital apex disease, cavernous sinus disease, aneurysm of the posterior communicating artery, raised intracranial pressure and diabetes (often a partial palsy) are all possible causes of a third nerve (oculomotor) palsy

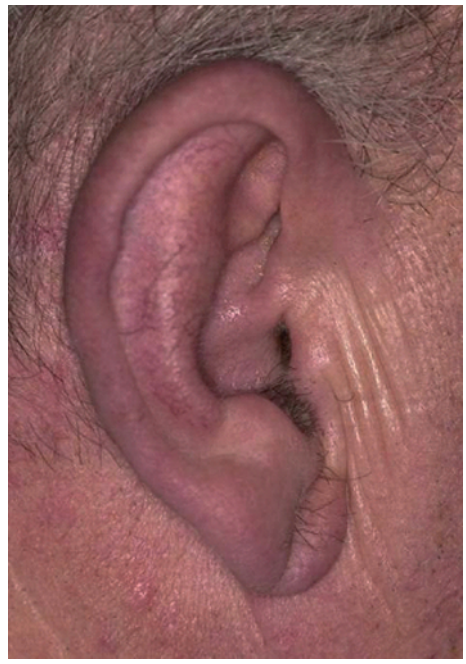

Figure 1.3 Creases in the earlobe may indicate cardiovascular disease

Figure 1.4 A left-sided third cranial nerve palsy

(Figure 1.4). The third nerve supplies all of the muscles of the orbit apart from the superior oblique (IV cranial nerve) and the lateral rectus (VI). Unopposed action of these muscles leads to the eye pointing 'down and out'. It is also the muscle that raises the eyelid (levator palpebrae), the ciliary muscle and constrictor of the pupil, hence there is a complete ptosis (drooping of the eyelid) and dilatation of the pupil. The left eye would be looking inferolaterally ('down and out') giving a divergent squint and the pupil would be dilated (complete paralysis) or normal ('partial third nerve palsy').

Diagnosis

- Clinical history.
- Electrocardiogram (ECG): the resting ECG may be normal and so an exercise ECG is also indicated.
- Myocardial perfusion scans (thallium-201) show ischaemic areas as 'cold spots' during exercise.
- Coronary angiography assesses the coronary artery anatomy and patency.

Management

Emphasis should be on lifestyle changes with the primary aim to prevent, or reduce progression of, coronary atheroma. These include:

- Dietary modification: reduction of cholesterol and saturated fat intake
- Regular exercise
- Weight loss
- Smoking cessation.

Pharmacological measures for the management of CAD include:

- Anti-platelet drugs (aspirin or clopidogrel)
- Anti-hypertensive treatment with beta-blockers (atenolol), diuretics (furosemide) and angiotensin converting enzyme (ACE) inhibitors (enalapril)
- Cholesterol lowering drugs such as statins (simvastatin)
- Good control of blood glucose levels if diabetic.

When CAD is extensive and an individual's symptoms are worsening despite general measures and optimal medical management, cardiac revascularisation techniques that should be considered include:

- Coronary angioplasty: stents may be placed percutaneously (percutaneous coronary intervention [PCI]), to re-establish coronary blood flow and improve myocardial perfusion
- Coronary artery bypass grafts (CABGs) to bridge severe obstructions in the coronary blood vessels.

Angina pectoris

Angina pectoris is the name given to episodes of chest pain caused by myocardial ischaemia secondary to CAD. Angina affects around 1% of the adult population and its prevalence rises with increasing age. The severity and prognosis of angina depends upon the degree of coronary artery narrowing and has a varied clinical presentation. The average annual mortality rate in the UK is about 4% per year.

Clinical features

Angina is often unmistakable because the pain is precipitated by physical exertion, particularly in cold weather, and is relieved by rest. Affected individuals

Figure 1.5 Severe oral ulceration caused by nicorandil

may describe a sense of tightness, heaviness, compression or constriction of the chest, sometimes radiating to the left arm or jaw. Emotion (anger or anxiety) and stress (fear or pain) can induce angina by leading to the release of catecholamines (epinephrine [adrenaline] and norepinephrine [noradrenaline]) from the adrenal cortex. These hormones result in an increased heart rate (tachycardia), a raised blood pressure (reactive hypertension), and vasoconstriction of the coronary circulation. Consequently an increased cardiac workload is accompanied by a paradoxical drop in blood flow and myocardial ischaemia occurs – resulting in angina.

Variants of angina include:

- Stable angina: pain only on exertion and relieved in a few minutes by rest and sublingual glyceryl trinitrate (GTN)
- Decubitus angina: pain on lying down
- Vasospastic (variant or Prinzmetal) angina: caused by coronary artery spasm
- Acute coronary syndrome (unstable angina): angina at rest or of sudden onset with a rapid increase in severity. This is due to a transient subtotal obstruction of a coronary vessel and is a medical emergency
- Cardiac syndrome X: clinical features of angina but normal coronary arteries on angiogram. It is thought to be due to a functional abnormality of the coronary microcirculation.

Clinical signs

Some drugs such as nicorandil used in the management of unstable angina, can produce severe oral ulceration (Figure 1.5).

Diagnosis

The diagnosis of angina is primarily a clinical one. Physical examination and

investigations may be normal. The individual's risk factors for CAD should be carefully assessed. Investigations may include:

- Resting electrocardiogram (ECG): during pain there may be ST segment depression with a flat or inverted T-wave. The ECG is usually normal between episodes of angina
- Exercise ECG testing: positive in approximately 75% of people with severe CAD
- Myocardial perfusion scans (thallium-201): to highlight ischaemic myocardium
- Coronary angiography: to assess coronary blood flow in diagnostically challenging cases. Occasionally gastro-oesophageal reflux disease (GORD) and chest wall disease may mimic angina.

Management

Risk factors for CAD (cigarette smoking, physical inactivity, obesity, hypertension, diabetes mellitus, hypercholesterolaemia) should be identified and corrected. Prognostic therapies for angina include:

- Aspirin: inhibits platelet aggregation by preventing the synthesis of thromboxane A_2
- Glycoprotein IIb/IIIa receptor inhibitors: prevent adherence of fibrinogen to platelets and reduce thrombus formation, and are used in 'high-risk' individuals and patients with acute coronary syndrome
- Lipid-lowering drugs (e.g. statins): have been shown to lower mortality rates in patients with CAD.

During acute episodes of angina, pain is relieved by administering oxygen, sublingual GTN and reducing anxiety. When angina occurs more frequently long-acting nitrates (isosorbide mononitrate), β-adrenergic blocking drugs (atenolol), and calcium antagonists (amlodipine) are used to reduce cardiac oxygen demands. For angina that fails to respond to medical measures, cardiac revascularisation techniques should be considered:

- Percutaneous transluminal coronary angioplasty (PTCA): stents (miniature wire coils) may be inserted into the coronary arteries to re-establish blood flow
- Coronary artery bypass grafts: to bridge severe obstructions in patients with extensive CAD.

Myocardial infarction

Myocardial infarction (MI) results from the complete occlusion (blockage) of one or more coronary arteries. It arises when atherosclerotic plaques rupture causing platelet activation, adhesion and aggregation with subsequent thrombus formation within the coronary circulation. Angina may progress to MI, but fewer than 50% of patients with MI have any preceding symptoms.

Clinical features

Myocardial infarction most commonly presents with central chest pain similar to that of angina. Unlike angina it is not relieved by rest or with sublingual nitrates. Vomiting, nausea, restlessness, sweating, shortness of breath, and a feeling of 'impending doom' are common. Approximately 10–20% of individuals have silent (painless) infarctions and the first sign may be the catastrophic onset of left ventricular failure, shock, loss of consciousness and death. Up to 50% of patients die within the first hour of MI and a further 10–20% within the next few days. Fatal cardiac arrhythmias (ventricular fibrillation), valvular dysfunction, cardiac failure and myocardial rupture may complicate an MI.

Clinical signs

- An ECG shows typical changes of an anterior inferior infarct (Figures 1.6 and 1.7).

Diagnosis

The diagnosis of an MI is based mainly on clinical features supported by characteristic ECG changes (ST segment elevation, T-wave inversion, pathological

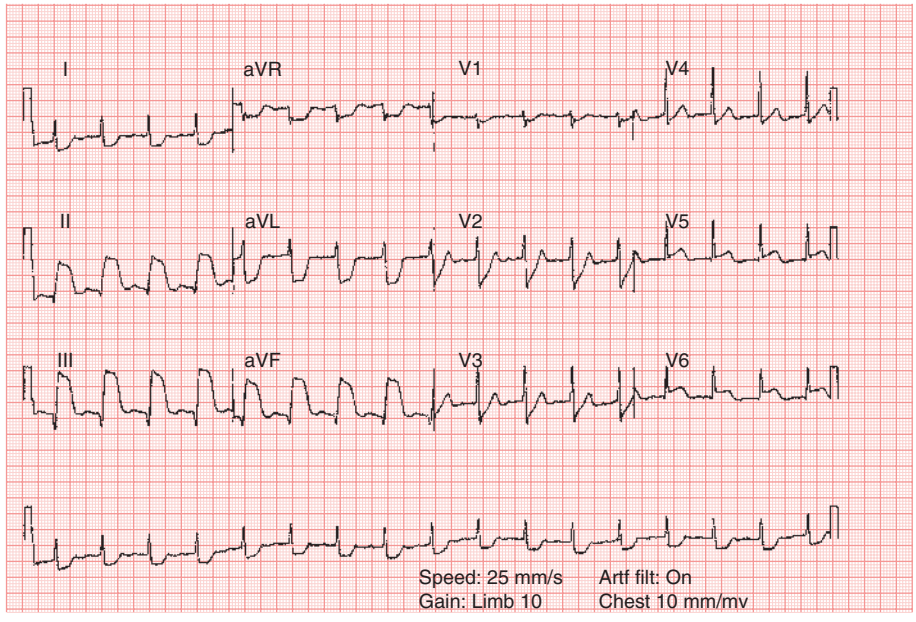

Figure 1.6 This ECG shows massive ST segment elevation in the inferior leads (II, III and aVF) with lesser elevation in the lateral chest leads (V5 and V6). There is so-called reciprocal ST depression in leads I, aVL, V2 and V3. These changes are consistent with an acute apical myocardial infarction

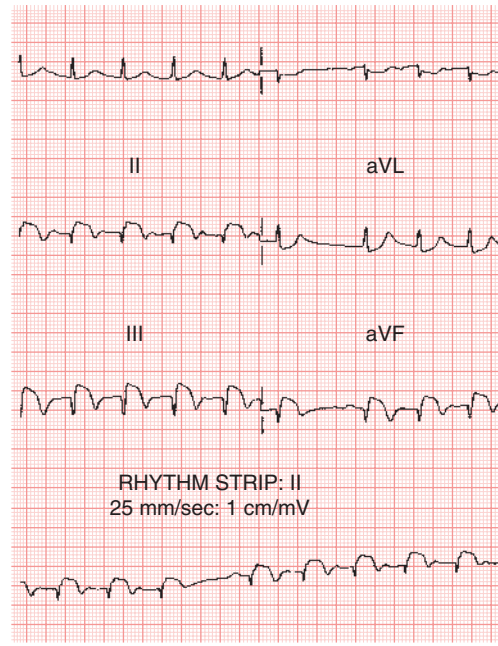

Figure 1.7 There is elevation of the ST segments in the inferior leads (II, III and aVF) along with T-wave inversion and Q wave formation. There is also prolongation of the PR interval eventually leading to a dropped beat, typical of Wenckebach phenomenon (second degree heart block). These changes indicate an acute inferior myocardial infarction

Q waves). Damaged (infarcted) cardiac muscle releases several enzymes and proteins into the circulation – troponin (an early marker of heart damage), cardiac specific creatine kinase (CK-MB), aspartate transaminase (AST) and lactate dehydrogenase (LDH). Serial blood measurements of these enzymes allows the time course of the MI to be followed.

Management

Myocardial infarction requires immediate hospital admission. Early treatment halves the mortality rate. Management aims include adequate pain relief, limitation of the extent of damage to the myocardium (infarct size) and prevention/early treatment of complications. Immediate treatment of an MI includes:

- Alert emergency services (if in community) or cardiac arrest team (if in hospital)
- Aspirin (300 mg): to be chewed
- Pain relief: opioid analgesia (diamorphine) is usually necessary
- Oxygen or nitrous oxide with at least 28% oxygen
- Thrombolytic agent (streptokinase/tissue plasminogen activator [t-PA]) to dissolve the coronary thrombus provided the patient is not at risk of a life-threatening haemorrhage
- Glyceryl trinitrate infusion to relieve pain and prevent pulmonary oedema (fluid accumulation within the lungs)

- Prompt treatment of complications, particularly cardiac arrhythmias, including the initiation of cardiopulmonary resuscitation.

Subsequent management of an MI includes initiation of secondary prevention therapy including:

- An ACE inhibitor: cardioprotective against subsequent events
- Early mobilisation
- Cardiac rehabilitation programme
- Correction of risk factors for CAD (as in the management of angina).

Oral health care relevance

- Angina can be precipitated by the stress and anxiety associated with dental treatment. Appointment times should therefore be kept as short as possible and the dental team should be empathetic and reassuring at all times.
- Preoperative oral temazepam (e.g. 10 mg at night prior to the day of the appointment and a further 10 mg on the morning of the appointment) may be helpful in reducing anxiety.
- Appointments should be scheduled to avoid early mornings as endogenous adrenaline [epinephrine] levels are higher at this time, potentially increasing the risk of adverse cardiac events.
- The use of adrenaline-containing local anaesthetics is not contraindicated. Profound anaesthesia is most important and an effective aspirating technique is necessary to avoid intravascular injections.
- Conscious sedation in the primary care setting is best avoided in patients with a recent history (6–12 months) of myocardial infarction or angina, and also in patients with unstable angina. The reader is reminded of The Department of Health's Standing Dental Advisory Committee document 'Conscious Sedation in the Provision of Dental Care, 2003'. This states that conscious sedation in the primary care setting should only be administered to patients in ASA (American Society of Anaesthesiologists) groups I and II (i.e. normal healthy individuals or a patient with systemic disease but no functional impairment).
- The patient's GTN medication should be readily available in case of an angina attack. Continuous chest pain for more than 3 minutes following its administration and giving oxygen is suggestive of a myocardial infarction. Oxygen (100%) should be continued, the patient given chewable aspirin and help summoned. 'Entonox' (1:1 nitrous oxide:oxygen) may be given to allay anxiety and provide analgesia.
- Patients with a history of myocardial infarction within the previous 6 months are regarded as being at greater risk of developing a further infarct during this time. Therefore, more complex procedures are best deferred.
- Angina can mimic dental pain spreading along the lower teeth and jaw.
- The medication of patients with ischaemic heart disease must be considered as they can cause oral manifestations (e.g. nicorandil used in the management of angina can produce severe oral ulceration). Potential drug

interactions must also be taken into account (e.g. concurrent use of triazole and imidazole anti-fungal agents should be avoided with the statins as this increases the risk of myopathy).

A stable cardiac patient receiving atraumatic treatment under local anaesthesia should be manageable in dental practice. A patient requiring complex surgery, a general anaesthetic or who is unstable with dyspnoea on minimal exertion, cyanosis, frequent angina or a recent MI, requires treatment in hospital.

Hypertension

Hypertension is a persistently raised blood pressure (BP). The BP is measured with a sphygmomanometer, in units of millimetres of mercury (mmHg). Hypertension may be defined as an elevated blood pressure of at least 140/90 mmHg, based on at least two readings on separate occasions. The BP will vary depending on age, gender, ethnicity, environment, emotional state and activity. The BP tends to increase with age but in more than 90% of people with hypertension the cause is unknown. This is termed 'primary' or 'essential' hypertension. Aetiological factors include:

- Genetic predisposition
- High alcohol intake
- High salt intake
- Smoking
- High body mass index (BMI)
- Impaired tissue response to insulin (insulin resistance)
- Sympathetic overactivity: approximately 40% of hypertensive patients have raised levels of circulating catecholamines (epinephrine [adrenaline] and norepinephrine [noradrenaline]).

In 1–2% of hypertensive patients an underlying cause is present and this is termed 'secondary' hypertension. Causes include:

- Renal disease: responsible for over 80% of cases
- Endocrine disorders
- Pregnancy
- Drugs (oral contraceptive pill)
- Narrowing of the aorta (coarctation).

Accelerated (malignant) hypertension is uncommon and seen mainly in people of African descent. It can have an acute onset or may develop in individuals with pre-existing essential hypertension. It is potentially life threatening and should be regarded as a medical emergency.

Clinical features

About 20% of the population are hypertensive with the majority being asymptomatic, and about one third of individuals being unaware that they have the condition. Longstanding hypertension accelerates atheroma (atherosclerosis) and predisposes to damage to the:

- Heart (coronary artery disease)
- Brain (cerebrovascular disease), particularly stroke
- Kidneys (chronic renal failure)
- Hands and feet, rarely (peripheral vascular disease)
- Eyes (hypertensive retinopathy), leading to blindness.

Accelerated hypertension may present with headaches, visual impairment, nausea, vomiting, fits (seizures) or acute cardiac failure.

Clinical signs

Anti-hypertensive medication can produce a variety of orofacial side effects such as gingival overgrowth from calcium channel blockers e.g. nifedipine, amlodipine (Figure 1.8).

Figure 1.8 Gingival overgrowth caused by calcium channel blockers

Diagnosis

Hypertension is diagnosed by standardised serial BP measurements. Investigations to identify a 'secondary' cause and assess end-organ damage include:

- Chest radiograph: increased cardiac size (cardiomegaly) is suggestive of hypertensive heart disease
- Electrocardiogram (ECG): may indicate ischaemic heart disease and show changes of left ventricular hypertrophy
- Serum urea and electrolytes: deranged in hypertensive renal disease and endocrine causes of secondary hypertension
- Urine dipstick testing: blood and protein in the urine suggests renal disease.

Management

Treatment of hypertension reduces the risk of stroke, heart failure and renal

failure. It has less of an effect on ischaemic cardiac events. General measures that lower BP include:

- Relaxation
- Smoking cessation
- Restricting alcohol intake
- Low salt diet
- High fibre diet
- Weight reduction
- Regular exercise.

A large number of drugs are currently available for the treatment of hypertension (Table 1.2).

Table 1.2 Anti-hypertensive drugs

Anti-hypertensive agents	Examples
Diuretics	Bendroflumethiazide, furosemide
Angiotensin converting enzyme (ACE) inhibitors	Enalapril, ramipril, perindopril
Angiotensinogen II receptor blockers (ARB)	Candesartan, losartan, telmisartan
β-adrenergic blockers	Atenolol, propanolol
Calcium channel blockers	Amlodipine, nifedipine
α-adrenergic blockers	Doxazosin
	Moxonidine

A single agent (monotherapy) is used initially, but combination therapy may be needed in more resistant cases for adequate control of the BP. The most effective drug combinations include:

- An ACE inhibitor with a diuretic
- An ARB with a calcium antagonist.

Life-threatening accelerated hypertension requires urgent hospital admission with the aim to reduce the BP slowly with oral anti-hypertensives. Rarely, intravenous anti-hypertensives (e.g. sodium nitroprusside) are used, but a sudden drop in BP may result in a stroke (cerebral infarction).

Oral health care relevance

- Essential hypertension is not in itself associated with orofacial manifestations. There may, however, be a plethoric facial complexion in patients with very elevated blood pressure.
- Anti-hypertensive medication can produce a variety of orofacial side effects
  - beta blockers: xerostomia and lichenoid reactions
  - calcium channel blockers: gingival overgrowth (Figure 1.8)
  - angiotensin converting inhibitors: lichenoid reactions, burning sensations of the oral mucosa
  - angiotensin II receptor blockers: lichenoid reactions, burning sensations of the oral mucosa.

- The provision of dental care should be as stress free as possible. It may be advantageous to avoid early morning appointments as this is when blood pressure is highest and adverse cardiac events more likely to occur.
- Local anaesthetics containing adrenaline (epinephrine) are not contraindicated in patients with hypertension, although it is prudent to use the minimal amount consistent with obtaining satisfactory anaesthesia. An exception is in those patients with severe (diastolic pressure greater than 140 mm/Hg) or malignant hypertension. Both of these conditions require urgent hospital referral.
- Following lengthy dental treatment in the supine position, the patient should be returned to the upright position slowly to minimise the risk of postural hypotension.
- Long term, inadequately managed hypertension may lead to cardiac, cerebrovascular and renal complications, which may in turn impact on the provision of dental treatment (see appropriate sections).

Congenital heart disease

Congenital lesions may involve the heart or adjacent great vessels either in isolation or in a variety of combinations. They are the most common type of heart disease in children and in developed countries, affecting about 1 in 1000 live births. A variety of factors may give rise to congenital heart disease (CHD). These include:

- Congenital rubella and cytomegalovirus infection
- Maternal drug and alcohol abuse
- Single gene mutations
- As a feature of hereditary syndromes (Down syndrome).

Congenital heart disease may be cyanotic – 'blue babies' (Table 1.3), where there is right-to-left shunting and in general more severe defects, or acyanotic (Table 1.4).

Clinical features

In cyanotic CHD reduced oxygen carriage (chronic hypoxaemia) leads to impaired development; polycythaemia and gross clubbing of fingers and toes may result. Patients may crouch in an attempt to improve venous return of the

Table 1.3 Cyanotic congenital heart disease (CHD)

Congenital heart lesion	Nature of defect
Transposition of the great vessels	Reversal of the origins of the pulmonary artery and aorta
Tetralogy of Fallot	Ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy and an aorta that overrides both ventricles
Eisenmenger syndrome	Right to left shunting of blood flow through the heart

Table 1.4 Acyanotic congenital heart disease (CHD)

Congenital heart lesion	Nature of defect
Mitral valve prolapse (floppy mitral valve)	The most common cardiac defect, 20% of the population
Ventricular septal defect (VSD)	Common. Usually a single opening in the interventricular septum
Patent ductus arteriosus (PDA)	A persistent opening between the aorta and pulmonary artery
Pulmonary valve stenosis	Narrowing of the pulmonary valve
Atrial septal defect (ASD)	An opening in the atrial septum. Many different forms ranging from a simple primum defect to a complex atrioventricular septal defect
Coarctation of the aorta	Narrowing of the aorta usually sited beyond the origin of the subclavian arteries. The blood supply to the head, neck and upper body are normal but the circulation to the lower part of the body is restricted
Aortic valve stenosis	Narrowing of the aortic valve, most commonly secondary to a congenital bicuspid valve

blood to the heart. Both haemorrhagic and thrombotic tendencies may arise. Cyanotic congenital heart defects invariably result in heart failure, and in the absence of treatment 40% of individuals die within the first 5 years. Approximately 20% of patients with CHD have other congenital anomalies.

Clinical signs

Central cyanosis and a malar flush (Figure 1.9) can be congenital or acquired. Atrial fibrillation (AF) occurs in 40% and reactive pulmonary hypertension in 25% of patients with severe stenosis, acute pulmonary oedema, infective endocarditis, bronchitis and recurrent pulmonary embolism; the lady shown in Figure 1.9 suffers from orthopnoea and paroxysmal nocturnal dyspnoea.

Congenital mitral stenosis is usually associated with other lesions causing left ventricular outflow obstruction including atrial stenosis, subaortic stenosis, coarctation and atrial myxoma. In 99% of patients, this is acquired and due to rheumatic heart disease. Other rare causes include infective endocarditis,

Figure 1.9 Patient with central cyanosis and a malar flush

granulomatous infiltration in association with eosinophilia, calcified mitral valve ring and systemic lupus erythematosus.

Diagnosis

- Antenatal ultrasound: allows early diagnosis
- Chest radiograph: may show cardiomegaly
- Electrocardiogram: may demonstrate an abnormal cardiac axis, ventricular hypertrophy and strain depending on the lesion present
- Echocardiography: allows the majority of defects to be diagnosed non-invasively and has now superseded intracardiac catheter studies.

Management

Early correction of the congenital defect is the treatment of choice and an increasing number of lesions are now treated by transvenous catheter techniques thereby avoiding the need for invasive surgery. More complex defects may require an operation. Although surgery has enormously improved the prognosis for patients with CHD, residual defects can predispose to infective endocarditis. Medical treatment may also be needed for the management of:

- Pulmonary oedema
- Heart failure
- Polycythaemia
- Infection
- Emotional disturbances.

Children with CHD receiving modern surgical and medical care now often survive into adult life, so-called 'grown-up' congenital heart disease.

Oral health care relevance

The National Institute for Health and Clinical Excellence (NICE) clinical guideline 64, 'Prophylaxis Against Infective Endocarditis', was published in March 2008. This stated that antibiotic prophylaxis against infective endocarditis for invasive dental procedures in patients previously considered as being 'at risk' is no longer recommended. Additionally NICE stated that chlorhexidine gluconate mouth rinses should not be offered to such patients. The recommendations of NICE are based on the lack of supporting evidence that dental treatment predisposes patients to infective endocarditis. There is also evidence that the risk of serious adverse events to antibiotics is substantially greater than the risk of causing infective endocarditis from dental procedures in 'susceptible' patients.

Infective endocarditis

Infective endocarditis (IE) is a rare but potentially life-threatening infection, predominantly affecting damaged heart valves. Platelet–fibrin deposits may

form along the free margins of damaged valves, where there is turbulent blood flow. These sterile vegetations (aseptic thrombotic endocarditis) may become infected with organisms resulting in large friable vegetations. Cardiac lesions that predispose to infective endocarditis include:

- Congenital or acquired valvular defects
- Prosthetic heart valves
- Atrial and ventricular septal defects
- Patent ductus arteriosus
- Complex congenital heart disease (e.g. tetralogy of Fallot)
- Surgically constructed systemic–pulmonary shunts.

Individuals who have had uncomplicated myocardial infarcts, coronary angioplasty, coronary artery bypass grafts and cardiac pacemakers inserted do not have an increased risk of developing IE.

Oral viridans streptococci (*Streptococcus mutans* and *S. sanguis*) have complex attachment mechanisms that enable them to adhere to damaged endocardium, and they are responsible for approximately 50% of cases of IE. Viridans streptococci enter the bloodstream (bacteraemia) during tooth extractions and other oral procedures, including toothbrushing and scaling. The majority of bacteraemias are transient, self-limiting and not associated with any systemic complications. The factors that determine the development of IE are complex, but a susceptible cardiac surface (damaged endocardium) and high bacterial loads within the circulation appear to be important.

Clinical features

The clinical features of IE are highly variable, often with an insidious onset, but should be considered in any individual presenting with fever and a new or changing heart murmur. Symptoms and signs reflect:

- Progressive heart damage (valve destruction and heart failure)
- Infection (fever, malaise, night sweats and weight loss)
- Embolic damage of organs (brain, lungs, spleen and kidneys)
- Immune complex formation (leading to vasculitis, arthritis and renal and retinal damage).

Clinical signs

Splinter haemorrhages (Figure 1.10) are seen in approximately 10% of patients with infective endocarditis. Other signs may include:

- Petechial spots (small and red with a pale centre, often seen in the pharynx and conjunctivae; when present on the retina they are known as Roth spots)
- Osler nodes (hard, tender subcutaneous swellings in the hands and feet)
- Janeway lesions (small, flat, red, non-tender macules on the thenar eminences)
- Pallor (due to anaemia)
- Finger clubbing may be a late feature.

Figure 1.10 Splinter haemorrhages

Diagnosis

- Clinical history and presentation
- At least three sets of blood cultures over 24 hours before starting antibiotics
- Electrocardiogram (ECG): may show conduction abnormalities
- Echocardiography (ECHO): may identify vegetations and enables assessment of valvular and cardiac function
- Urine dipstick testing to detect microscopic haematuria
- Serological testing to identify atypical organisms (e.g. Legionella).

Management

Without treatment, IE is fatal in approximately 30% of patients, so the patient should be admitted to hospital for intravenous antibiotic therapy, usually benzylpenicillin and gentamicin. If staphylococcal endocarditis is suspected vancomycin should be substituted in place of penicillin. In severe cases, such as prosthetic valve endocarditis, early removal of the infected valve and insertion of a sterile replacement may be needed.

Patients at risk of endocarditis should receive intensive preventive dental care to minimise the need for dental intervention. In many countries there are national guidelines on the use of antimicrobial prophylaxis against IE if dental interventions are needed. Although the efficacy of such antimicrobial prophylaxis may be questionable, individuals with susceptible cardiac lesions (as outlined above) were typically given antibiotic therapy prior to undergoing procedures likely to result in bacteraemia.

- NICE no longer recommends antibiotic prophylaxis for patients with a previous history of infective endocarditis who are to receive invasive dental treatment (see also *Congenital heart disease* section).