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Essential Human Disease for Dentists

Chris Sproat
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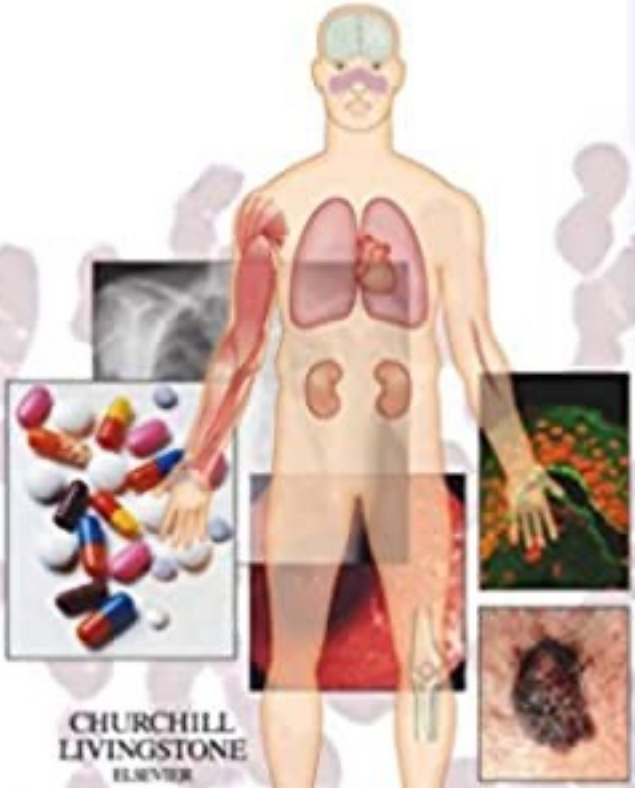

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PREFACE

An understanding of human disease is essential to the practice of dentistry enabling risk assessment prior to treatment and the diagnosis of systemic diseases with oral manifestations. The education of dentists in human disease has become more focused on those aspects with direct relevance to dentistry and it is important for any text to reflect this change in practice.

This book has been designed as a revision aid for undergraduate students studying for the dental human disease examination and as chairside reference for busy general dental practitioners. To enable readers to get quickly to the point we have limited the content to commonly occurring or life threatening conditions.

The information is presented in a concise manner with illustrations and pictures to emphasise important aspects. Relevance to dentistry boxes are included in each chapter for quick reference.

This text is unique in that the majority of contributors are dentally or dual qualified enabling an objective view of what is relevant to practising dentists. The editors and co-authors have a wealth of experience teaching all aspects of human disease to both undergraduate and postgraduate dentists.

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London
2006

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1 ASSESSMENT AND RISK MANAGEMENT

Chris Sproat

Overview

All dental procedures carry inherent risks which must be assessed and dealt with appropriately. The decision to proceed with a course of treatment will depend on two major factors:

1. Professional dental opinion
2. Patient's perceived need.

In order to carry out safe and effective treatment each patient must be assessed to identify and quantify the risks involved. The risk/benefit analysis must be explained to the patient and informed consent obtained.

As dental practitioners we are in the unique situation of regularly reviewing a large cohort of 'healthy' patients who may display oral manifestations of as yet undiagnosed systemic disease. In order to make the correct diagnosis and give appropriate advice it is necessary to have an understanding of diseases that commonly present in the head and neck region.

The safety of dental procedures carried out under local anaesthetic is generally good. The risks of a procedure can be increased by the:

- health status of the patient
- complexity and duration of the case
- degree of invasiveness
- experience and skill of the operator
- addition of sedation or general anaesthetic.

Risk can be minimised by an adequate pre-procedure assessment and working within your own and the practices limits.

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Assessment

This begins when you first see the patient in the waiting room and should continue throughout the treatment into the postoperative phase as you monitor the patient's wellbeing.

Clues to the functional status of the patient can be obtained as they enter the surgery and position themselves in the dental chair, for example, do they have difficulty or require assistance?

The most reliable method of obtaining a patient's medical history is to use a combination of direct questioning and a confidential questionnaire. If further information is required then the general medical practitioner or hospital physician should be contacted. In the case of children the relevant information will often come from the parents but remember to involve the child as appropriate. Patients who are not able give an adequate or reliable history – for example, some psychiatric patients and stroke victims – are often escorted by a carer who may be able to assist in obtaining the necessary history. A typical medical history questionnaire is shown in Figure 1.1.

Patients must be given adequate time and the necessary help to complete the medical history questionnaire. Disabled patients may require special format versions, for example, large print.

During the verbal history it is good practice to use a combination of open questions, for example, 'are you generally well?' and follow this with closed questions about specific diseases, for example, 'have you had rheumatic fever?'. For any conditions detected it is necessary to determine severity and the impact on the patient's functional status; for example, for asthma, how often do they have attacks and does it limit their daily life?

The ASA (American Society of Anesthesiologists) classification of physical status of patients (Table 1.1) is a useful guide to determine the likely risk of a procedure. Patients in ASA groups I and II are normally safe to treat in general dental practice. Those who are ASA III and IV should receive treatment only in specialist centres.

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MEDICAL HISTORY – CONFIDENTIAL

To be completed by Patient, Parent, or Guardian.

No medical problem or infection will exclude you from receiving essential treatment.

Please note that there is no guarantee that a particular operator will carry out your treatment.

No.....
Name.....
D.O.B. .....

	YES	NO	
Are you in good health?			
Have you had:–			
Heart trouble/murmur, high blood pressure, or rheumatic fever?			
Chest trouble or shortness of breath?			
Jaundice or hepatitis?			
Severe bleeding that needed special treatment?			
Is there a family history of bleeding?			
Any operations or serious illness?			
A general anaesthetic?			
ARE YOU suffering of HAVE YOU suffered from:–			
Diabetes?			
Asthma, Hay fever or eczema?			
Fainting attacks, blackouts or epilepsy?			
If appropriate: Could you be pregnant?			
Is there any chance that you have become infected with HIV?			
Are you allergic to penicillin or any other drugs?			
Are you taking any medications, tablets, skin creams, ointments or drugs?			

How much do you smoke per day? ..... How much do you drink per day? .....

FURTHER DETAIL (please add anything of medical importance)

CHECKED BY								
DATE	: :	: :	: :	: :	: :	: :	: :	: :

Fig. 1.1 A typical medical history questionnaire.

The following questions are a useful basis for the verbal medical history:

'Are you in good health?'
Heart problems, e.g. angina or myocardial infarction (MI)?
Chest problems, e.g. asthma or bronchitis?
Other illness, e.g. diabetes, jaundice, hepatitis, epilepsy, rheumatic fever, TB?
Allergies?
Medications?
Recent operations?
Family history of illness?

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Table 1.1
The ASA guidelines used to quantify the impact of systemic disease on the functional status of the patient.

ASA classification	*Functional status of the patient*
I	Healthy
II	Presence of systemic disease with no effect on normal function, e.g. well controlled diabetes
III	Presence of systemic disease which limits function, e.g. poorly controlled epilepsy
IV	Presence of systemic disease which is a constant threat to life, e.g. severe coronary artery disease
V	Patient not expected to survive more than 24 hours

A patient who has recently had an operation under general anaesthetic without complication is unlikely to present a serious risk for dental treatment under local anaesthetic.

All unfamiliar medications should be looked up in the *British National Formulary* (BNF) to check for potential interactions with the common dental drugs and for possible oral side-effects. The patient's list of medications helps to clarify their medical history and give an idea of the severity of any diseases.

All patients should be asked about their 'risk habits':

- smoking
- alcohol consumption.

These pose a significant general health risk and increase the risk of oral cancer, periodontal disease and delay wound healing post-surgery.

Smoking should be quantified in terms of duration and number of cigarettes smoked. Past smoking habits should be recorded. Calculation of pack years (one pack year equals 20 cigarettes per day for 1 year) is a useful guide.

Alcohol consumption should be recorded in terms of the number of units consumed per week with questions about past alcohol consumption included.

An overall examination of the clothed patient in the dental chair should be carried out. The general demeanour of the patient should be assessed to look for signs of anxiety and any manifestation of systemic disease.

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Risk management

Focused risk management strategies looking at patient safety are a key issue in dental practice.

Before embarking on any treatment a risk assessment must be made and equated to the proposed benefit to the patient, forming the basis of our professional opinion. The information should be delivered to the patient in an understandable form so that informed consent can be obtained.

Once the decision has been made that a patient is suitable for routine dental treatment the patient can be placed in one of three broad groups in terms of risk:

1. High risk
2. Medium risk
3. Low risk.

Those in the high-risk group require some form of action to be taken before treatment is carried out, for example, patients with replacement heart valves require antibiotic cover for invasive dental procedures, those on warfarin require an INR (International Normalised Ratio) check pre-operatively.

The medium risk group have a systemic disease that requires monitoring but does not pose an immediate problem for dental treatment, for example, well controlled asthma.

The vast majority of patients form the low-risk group and these are fit and healthy people with no history of systemic disease.

Patients may migrate from one group to another as their health status changes. This requires frequent reassessment of the medical history and examination of the patient. The triad of risk is shown in Figure 1.2.

Most risks can be quantified and treatment modified accordingly; however, there are those emergency situations in which an unpredictable event occurs, for example, an anaphylactic reaction. In this circumstance risk management involves early identification of the problem, the availability of appropriate equipment and adequate training of the dental staff to deal with the situation.

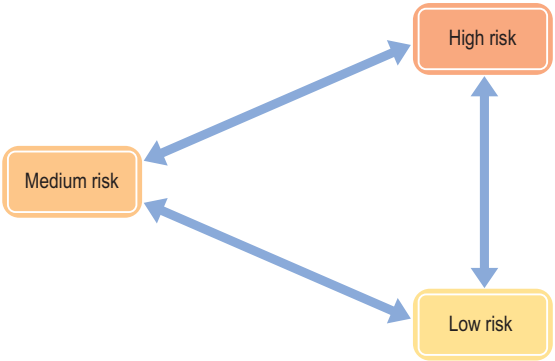


Fig. 1.2 The triad of risk used in assessment of patients for dental treatment.

Consent

Before you examine, treat or care for patients you must obtain their consent. This is not a one-off event but a continual process and the patient can withdraw consent at any time.

In order for consent to be valid the following criteria should be met:

- *Explanation.* An adequate explanation of the proposed treatment, including the risks and benefits, should be discussed in a manner and language acceptable to the patient.
- *Capability.* The patient should be capable of giving consent. All adults are assumed competent unless proved otherwise.
- *Voluntary.* Consent should be given voluntarily by the patient.

Ideally, consent should be obtained by the practitioner performing the treatment. It is possible to obtain consent on behalf of a colleague if you are capable of performing the procedure or if you have been specifically trained to obtain consent for the procedure.

Consent may be given verbally, or non-verbally in writing. The advantage of written consent is that there is evidence that a discussion took place and of the patient's decision.

Children over the age of 16 are able to give consent for themselves. If a younger child is deemed capable of understanding the explanation and consequences then they, too, may give consent (ideally with parental involvement).

Patients have the right to refuse treatment even if it is contrary to their best interests.

Patients who are not competent to give consent may be treated if it is considered to be in their 'best interests'.

Those sectioned under the mental health act can be treated against consent for their mental illness but not for other problems.

Useful web site

www.doh.gov.uk/consent

2 CARDIOVASCULAR DISEASES

Chris Sproat

Cardiovascular history and examination

Cardiovascular history and examination of the clothed patient begins when you first meet in the waiting room and should continue throughout the appointment as you monitor the patient's wellbeing. The vast majority of patients who suffer from cardiovascular disease can be safely treated under local anaesthetic in your surgery.

History

Using a few careful questions it is possible to quickly assess the patient's general status:

'Are you generally fit and well?'
'Do you have any heart problems?'
'Do you have high blood pressure?'
'What medications are you taking?'

You will be happy with most patients' cardiovascular status at this point. However, if you are concerned at this stage then you can ask more specific questions to investigate the problem further, assess their response to previous dental treatment and ask about their current exercise tolerance. If you are still concerned, then you should seek advice from the patient's doctor or cardiologist before embarking on further treatment.

A patient's exercise tolerance is a good indicator of general fitness and you can assess it with a few further questions:

'How far can you walk unaided without stopping?'
'Can you climb stairs?'
'What prevents you from going further?'

Patients whose tolerance is limited should be dealt with cautiously. A favourable response to previous dental treatment may give you reassurance but this is not to be taken as a green light to go ahead without careful evaluation.

Examination (remember: look then feel)

General appearance

Breathless at rest (respiratory rate >12/minute) may indicate heart failure or a respiratory problem. An abnormal appearance may indicate an underlying syndrome associated with congenital heart defects (e.g. Down's syndrome). An apprehensive, sweaty, pained expression may indicate angina or myocardial infarction.

Hands

- Finger clubbing: congenital heart defect
- Pale nail bed: anaemia
- Splinter haemorrhages: bacterial endocarditis.

Face and oral cavity

- Cyanosis (blue discoloration of the lips or palate) indicates poor oxygenation of the blood and may have a cardiac cause.
- Gingival hypertrophy may result from nifedipine antihypertensive medication.
- Xanthelasma (yellow plaques around eyes) indicates elevated cholesterol.

Pulse

Palpate the radial pulse with your index and middle fingers as shown in Figure 2.1 (do not use your thumb).

An irregular pulse indicates a cardiac rhythm abnormality, most commonly atrial fibrillation.

The heart rate is calculated by counting the number of beats over 15 seconds and multiplying by 4. The normal range at rest is 60–100 beats per minute. A rate greater than 100/minute is called tachycardia and less than 60/minute is bradycardia; both may indicate a cardiac problem.

Blood pressure

Blood pressure measurement can be carried out manually or automatically depending on the available equipment. The patient should be as relaxed as possible and sitting upright. You should note from which arm the reading was taken.

Manual technique

Place sphygmomanometer cuff on the arm with about 3 cm of skin visible above the antecubital fossa. (An appropriate

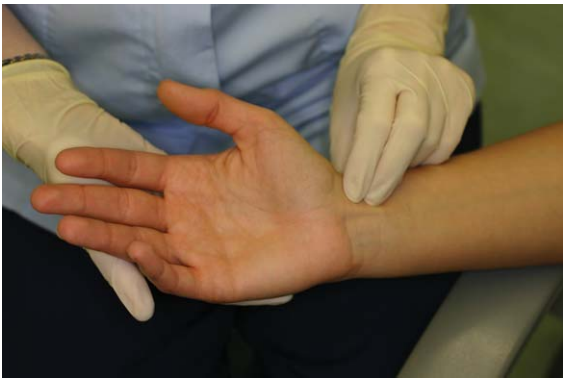


Fig. 2.1 Palpation of the radial pulse is carried out using the middle and index fingers as shown.

cuff size should be chosen. A cuff which is too small on an obese or large, muscular arm falsely elevates the reading; a cuff which is too large on a small arm gives a falsely low reading.)

1. Palpate radial pulse.
2. Inflate cuff until the radial pulse is no longer palpable. (This provides an estimate of the systolic blood pressure.)
3. Deflate cuff slowly while listening with stethoscope over the brachial artery on the skin of the inside of arm below cuff.
4. Record the systolic pressure as the pressure when the first tapping sound (Korotkoff sound) appears.
5. Record the diastolic pressure as the pressure at which the tapping sounds disappear.

The correct placement of a blood-pressure cuff is shown in Figure 2.2.

Automatic technique

Many practitioners prefer this method as it can be carried out reliably by trained dental surgery assistants. Cuff placement is identical to that used in the manual technique. The machine will automatically inflate and deflate the cuff and will give you a reading of diastolic and systolic pressure obtained.

Fig. 2.2 Correct placement of the blood pressure cuff is essential for accurate readings. It should be placed 3 cm above the anticubital fossa as shown.

Hypertension

Hypertension is defined as persistently raised blood pressure (BP) >140/90 mm of mercury (mmHg). Both the diastolic and systolic components are important. In 90% of cases no cause can be found and it is called primary hypertension. Dental procedures may cause a further rise in blood pressure and lead to acute complications.

Epidemiology

Primary hypertension affects 5–10% of the general population and is the most common cause of preventable disease in the developed world. It is normally detected between the ages of 20 and 50 years. There are both genetic and environmental factors involved in its aetiology. The cardiovascular risks are higher for people of African origin.

Aetiology

In primary hypertensives no specific cause can be found and it is likely that the origin is multifactorial. The causes of secondary hypertension are known and include renal disease, pregnancy, coarctation of the aorta, endocrine tumours (e.g. phaeochromocytoma) and drugs (e.g. steroids).

Pathogenesis

There are no initial pathological changes but as the hypertension persists arteriolar sclerosis occurs as the vessels adapt to the raised pressure by increasing smooth muscle and hyaline content of the media. This may in itself increase the blood pressure further by raising the peripheral resistance. The left ventricle enlarges to cope with the extra pressure but eventually it dilates and fails. Increased rate of atheroma formation narrows many arteries and reduces blood supply to vital organs (e.g. the heart, causing ischaemic heart disease). Arteries may become aneurysmal (abnormally dilated) and rupture (e.g. aortic aneurysm). Organs commonly affected by hypertension are shown in Figure 2.3.

Clinical features

Primary hypertension is asymptomatic until complications develop in target organs. In severe hypertension (>180/110 mmHg) there may be dizziness, headache and epistaxis. Any untreated hypertensive patient is at risk of developing left ventricular failure, MI, stroke or renal failure. Coronary artery disease is the most common cause of death among treated hypertensive patients and hypertension is the most important predisposing factor for stroke.

Diagnosis

Diagnosis is by accurate measurement of the blood pressure on at least three occasions over a 3-month period in a relaxed atmosphere (e.g. not your surgery before wisdom tooth extraction). This can be done manually or automatically. As dentists we have a unique opportunity to screen people for hypertension and offer advice on how to seek treatment. Blood pressure should be measured prior to any sedation procedure.

Treatment

If secondary hypertension has occurred then treatment of the cause is possible. Primary hypertension has no cure, but blood pressure can be reduced to an acceptable level by both conservative and medical methods. Advice on lifestyle modification should be given to all newly diagnosed hypertensives, which may reduce blood pressure

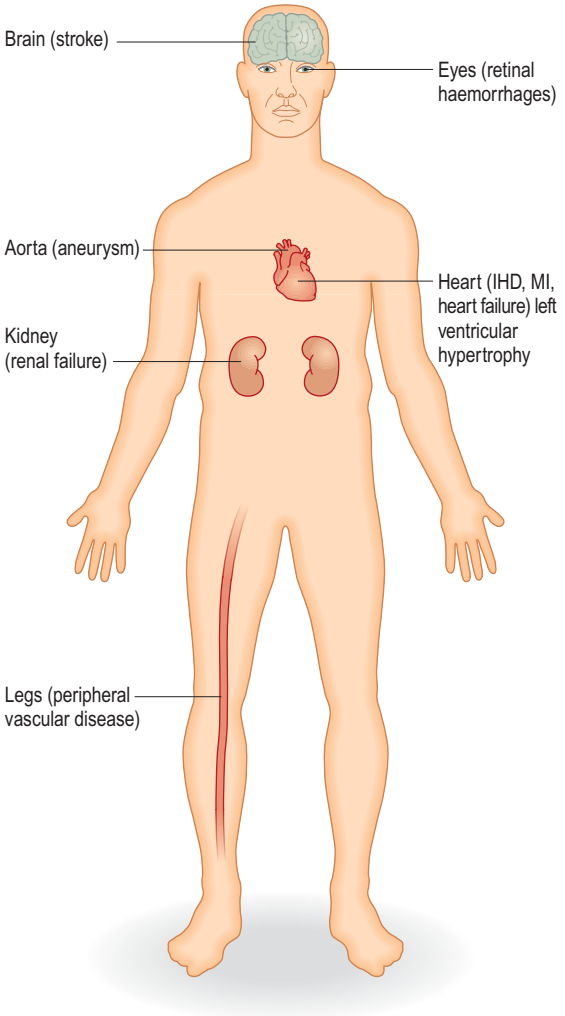


Fig. 2.3 'Target organs' commonly affected by hypertension.

Table 2.1
The common classes of antihypertensive medications and their main action.

Drug class	*Action*
Diuretic	↓ Fluid volume, venodilation
β-Blocker	↓ Cardiac output, ↓ TPR*
Ca antagonist	↓ TPR (vasodilation)
ACE inhibitor	↓ TPR (vasodilation)
Angiotensin II receptor blocker	↓ TPR (vasodilation)
Adrenergic inhibitors	↓ Sympathetic activity

* TPR = total peripheral resistance.

satisfactorily in some cases. This includes weight reduction, increased exercise, and decreased alcohol consumption, stopping smoking and a low salt diet. In the majority of cases medical management is required and a large number of antihypertensive drugs are available. Selection of the appropriate drug or drug combination depends on the patient's age, race, coexisting diseases and side effects. The common classes of antihypertensive medications, and their main mode of action are listed in Table 2.1.

Dental treatment of hypertensive patients

You will be required to treat both known and previously unknown hypertensive patients in dental practice.

All hypertensive patients are best treated under local anaesthetic. Reliable and complete analgesia is desirable to avoid distress to the patients, which will induce increased sympathetic output and further increased blood pressure. The careful use of adrenaline (epinephrine) containing local anaesthetic causes minimal increases in BP so long as it is not given intravascularly or in excessive doses. This type of anaesthetic is more reliable and thus preferred to alternatives.

From a dental perspective it is useful to divide patients into four groups:

1. Normotensive
2. Controlled hypertensive on treatment
3. High blood pressure detected in practice
4. Malignant hypertensive.

Patients in group 2 can be treated as normotensive patients but beware of interactions with, and the oral side-effects of, the antihypertensive drugs in use.

Group 3 patients are those who have a high reading on routine screening in dental practice. They should be referred to their medical practitioner for further investigation and elective treatment deferred.

Group 4 patients with a blood pressure reading greater than 185/110mmHg are at high risk of acute complications and should be referred urgently to their doctor or hospital.

Dental complications in hypertensive patients

There are few direct dental complications of hypertension; however, remember that stressful situations may cause an additional rise in BP which could precipitate systemic problems (e.g. stroke or myocardial infarction). Post-operative bleeding is more likely to complicate surgical procedures in hypertensive patients. There may be interactions between the patient's antihypertensive medication and drugs you wish to prescribe and many antihypertensive medications have oral side-effects.

Oral side-effects of antihypertensive drugs include:

- xerostomia (diuretics)
- gingival hyperplasia (nifedipine)
- salivary gland swelling (clonidine)

■ DENTAL RELEVANCE OF HYPERTENSION

Hypertension is common, affecting between 5 and 10% of the population.

Do not carry out routine treatment on patients whose BP is greater than 160/110.

May be detected first in dental practice.

Increased postoperative bleeding.

Patients may also be taking aspirin as part of their management and thus suffer increased post-operative oozing.

Many antihypertensive medications have oral side-effects.

Check for drug interactions between antihypertensive medications and drugs prescribed as part of dental treatment.

Hypertensive patients are at increased risk of cardiovascular disease (e.g. angina and myocardial infarct).

- lichenoid drug reactions (angiotensin-converting enzyme inhibitors)
- altered taste (acetozolamide).

FURTHER READING

British Hypertension Society guideline for hypertension management 2004 (BHS-IV): Summary, British Medical Journal 2004;328:634–640.

Ischaemic heart disease

Ischaemic heart disease (IHD) occurs when there is an imbalance between supply of blood to the heart muscle and demand. It is the most common cause of death in the western world. Over 20% of males under 60 years of age have ischaemic heart disease and almost all elderly people are affected to some degree. Dental procedures may provoke symptoms or acute complications.

Epidemiology

Ischaemic heart disease is the most common cause of death, accounting for 35% of total mortality, in the western world. Some 3% of adults suffer with angina and 1% have had a myocardial infarction in the last 12 months.

Aetiology

Coronary atheroma is the most common cause of IHD. Occasionally increased tone in the coronary vessels may be the cause. There are a large number of risk factors, some of which are fixed and others modifiable.

Fixed	Age
	Male gender
	Family history
Modifiable (*hard*)	Hyperlipidaemia
	Smoking
	Hypertension
	Diabetes
Modifiable (*soft*)	Obesity
	Lack of exercise
	High intake of alcohol
	Personality
	Oral contraceptive

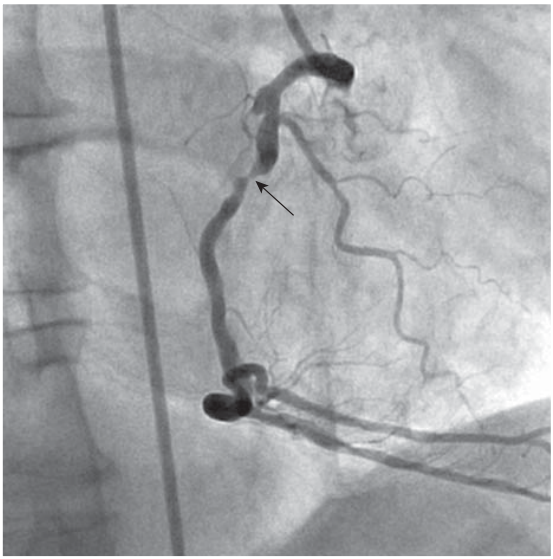


Fig. 2.4 A coronary angiogram showing narrowing of the coronary arteries due to the development of atheromatous plaques.

Figure 2.4 is an angiogram showing narrowing of the coronary arteries.

Pathogenesis

Pathogenesis involves the formation of an atheromatous plaque within the coronary arteries which produces a fixed constriction to blood flow. The plaque consists of a necrotic core containing cholesterol surrounded by increased smooth muscle and fibrous tissue. The endothelial lining is disrupted and thrombus formation occurs due to platelet adhesion and the vessel lumen becomes narrowed. The plaque is at risk of fissuring; if this occurs an acute thrombus may form which can completely occlude the vessel leading to myocardial infarction (MI).

An atheromatous plaque is shown in Figure 2.5.

Clinical features

Clinical features depend on the rate and severity of narrowing of the vessels and on the degree of oxygen demand of the cardiac muscle. *Angina* is the classical symptom

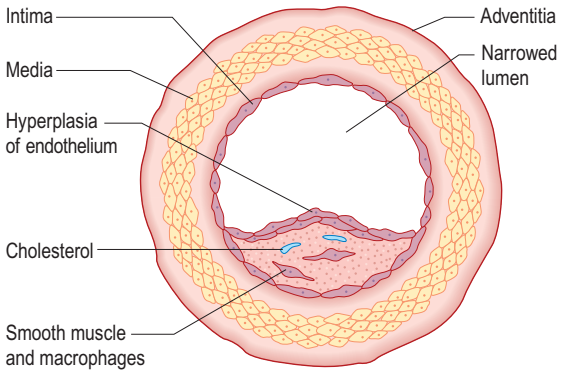


Fig. 2.5 An atheromatous plaque showing narrowing of the lumen due to accumulation of cholesterol and increased smooth muscle in the vessel wall.

consisting of severe, crushing central chest pain that radiates down the left arm. This is usually provoked by increased physical exertion and resolves with rest. IHD does not always cause chest pain; the patient may become breathless, feel nauseous, sweat or complain of pain in the right arm, neck or even jaw. If the symptoms persist for longer than 15 minutes and do not respond to rest or anti-anginal medication then you must consider the possibility of an MI.

Diagnosis

Diagnosis is made from the history and supplemented with ECG examination, coronary angiography and nuclear medicine scans of the myocardium.

Treatment

Treatment consists of preventing further damage to the heart by either increasing the blood supply or decreasing the myocardial demand.

Conservative

Modify risk factors (e.g. cease smoking, lose weight, take sensible exercise).

Medical

Decrease myocardial oxygen demand (e.g. by using nitrates).

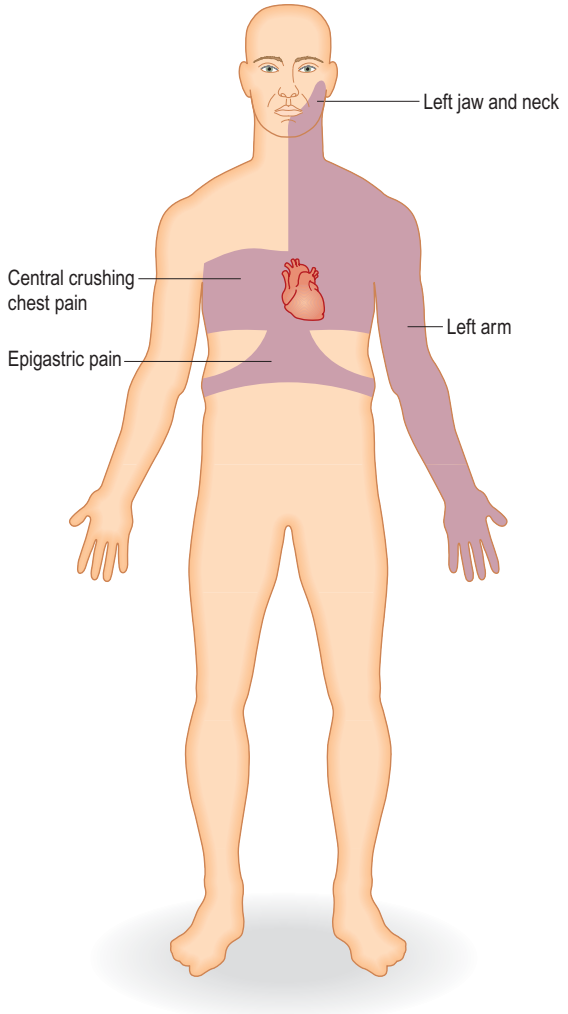


Fig. 2.6 Classical distribution of pain in angina.

Surgical

Dilate affected vessels (angioplasty) and insert stents, or bypass the affected areas with vascular grafts (coronary artery bypass graft).

Dental treatment

Dental treatment may provoke an angina attack or an MI. In all cases liaison with the patient's doctor is sensible. Take a detailed history and record all medication prescribed. You need to establish the severity of the patient's symptoms, provoking factors and whether their angina is stable or unstable. Stable angina is predictable; it occurs under reproducible conditions, responds to rest and medication. Unstable angina occurs in an unpredictable manner; it may get progressively worse and occur at rest. Patients with stable angina can be treated in dental practice under adequate local analgesia (2% lidocaine with adrenaline (epinephrine) 1:80000). It is good practice to request that the patient takes their GTN (glyceryl trinitrate) before you commence treatment, avoid prolonged procedures and minimise stress.

Figure 2.6 shows the classic distribution of pain in angina.

Patients with unstable angina should not be treated until their condition has been brought under control. These patients should be referred to their doctor for management.

Rarely, patients may suffer with decubitus angina which is brought on by lying flat. These patients must not be treated supine.

Emergency treatment in dental practice

If a patient develops central chest pain or other anginal symptoms during treatment you should take the following course of action:

- stop what you are doing
- reassure the patient
- summon help
- place patient in comfortable position
- give GTN (glyceryl trinitrate) sublingually.

If there is no response then consider the possibility of unstable angina or MI:

- call an ambulance
- place oxygen high flow
- repeat GTN sublingual

- give aspirin 300 mg chewed (Fig. 2.7)
- use relative analgesia (set to 50% oxygen 50% nitrous oxide) if available.

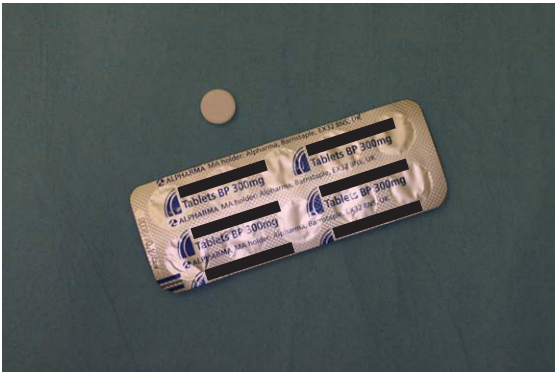


Fig. 2.7 A 300 mg aspirin tablet should be given to the patient to chew if you suspect that he/she is having an MI.

■ DENTAL RELEVANCE OF ISCHAEMIC HEART DISEASE

It is very common within the population, with 20% of males under 60 years affected.

It may present with tooth ache or pain in the jaw.

Dental treatment may provoke symptoms or acute complications.

Determine whether symptoms are stable or unstable; stable patients can be treated in dental practice.

- preventative dentistry
- plan short treatments
- keep stress to minimum
- give GTN pre-treatment
- adequate local analgesia.

Unstable patients should be referred to their doctor before any dental treatment.

In emergency stop treatment, give GTN and consider the possibility of an MI.

There is no requirement for antibiotic cover for coronary artery bypass grafts or coronary artery stents.

Rheumatic fever

Rheumatic fever (RF) is an acute inflammatory disease which primarily affects the joints and the heart. It is an autoimmune disorder that is usually preceded by a streptococcal sore throat. The heart valves can be damaged and become vulnerable to endocarditis.

Epidemiology

Rheumatic fever affects 3% of patients following a group A β-haemolytic streptococcal sore throat. It is most common in childhood between 5 and 15 years of age and is rare in adults. Poor socioeconomic groups are at increased risk. There is a genetic predisposition and it can recur. The incidence has decreased in the west due to better socioeconomic conditions and the use of antibiotics to treat the bacterial sore throat.

Pathogenesis

This is not fully understood but there are four absolute requirements for RF to occur: group A β-haemolytic streptococcal infection, a susceptible host, pharyngeal site and persistence of infection.

RF occurs 2 to 3 weeks after a streptococcal sore throat. It is thought to be an autoimmune condition in which cross-reacting antibodies are produced as a result of the bacterial infection, which then attack various normal body tissues. The connective tissues of the heart, including the valves, are particularly susceptible to damage, which is often permanent and may result in death. The mitral valve is most commonly affected by the formation of rheumatic nodules (Aschoff nodules) which result in incompetence of the valve (inability to close properly) and production of a heart murmur. Immune complexes cause damage to other tissues, including the joints, resulting in arthritis and arthralgia. The skin can also be affected, resulting in a rash and nodule formation.

Clinical features

'RF licks the joints and bites the heart'. Most patients have a fever and a flitting polyarthritis. If the heart is involved

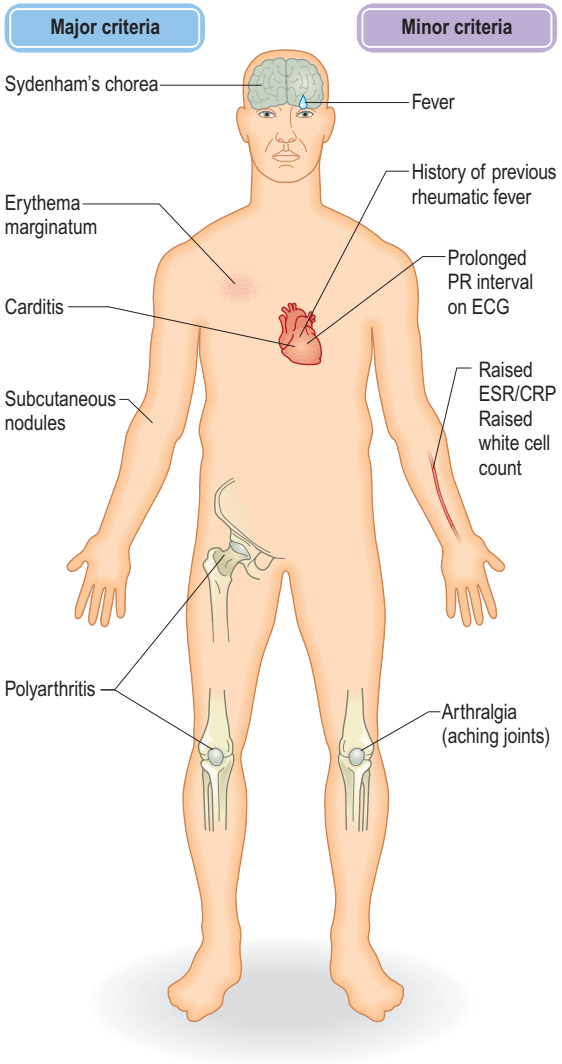


Fig. 2.8 Modified Jones criteria used in the diagnosis of rheumatic fever.

then there may be a new murmur, pericarditic pain or acute heart failure. Rarely the skin is involved; there may be palpable subcutaneous nodules and a characteristic rash with a raised red margin and pale centre (erythema marginatum). Chorea (involuntary movement of the limbs and face) may develop; this is a distressing feature and has been likened to a dance (St Vitus' Dance). The heart is the only tissue to suffer permanent damage and this may lead to endocarditis or heart failure in the future.

Diagnosis

Diagnosis is based on the modified Jones criteria (Fig. 2.8) which are divided into major and minor groups. There must be evidence of streptococcal infection (detected by a throat swab), presence of antistreptolysin antibody or previous scarlet fever. The diagnosis is made if there are two major criteria or one major and two minor criteria.

Treatment

Treatment involves bed rest, analgesia and eradication of the streptococcal infection.

■ DENTAL RELEVANCE OF RHEUMATIC FEVER

Patients with a history of rheumatic fever have an increased risk of developing bacterial endocarditis following invasive dental treatment.

Antibiotic prophylaxis is required prior to invasive dental treatment.

Current antibiotic prophylaxis guidelines should be checked in the BNF prior to treatment in each case.

Invasive dental treatment includes:

- tooth extraction
- tooth reimplantation
- implant placement
- subgingival scaling and probing
- endodontic treatment beyond the apex
- placement of matrix and ortho bands
- intraligamentary local anaesthetic

Infective endocarditis

This is a condition caused by infection of the endocardium and heart valves, which in some cases is fatal. It is most commonly due to blood-borne bacterial infection but may be fungal in the immunocompromised. Some 50% of cases are due to Streptococcus viridans and, as a result, dental treatment is often implicated as the causal event. It is usually a chronic condition but it may follow an acute course with rapid valve destruction. The latter occurs commonly in intravenous drug users.

Epidemiology

Infective endocarditis primarily affects older patients with degenerative valvular heart disease. It used to be common in patients who had suffered valve damage due to rheumatic fever. Now there is an increased incidence in intravenous drug users.

Pathogenesis

Damaged or prosthetic heart valves are usually involved, as are areas affected by abnormal flow jets from congenital heart defects (e.g. ventral septal defect). Normal valves on the right side of the heart can be affected in intravenous drug users. The mitral valve is most often affected, with the formation of adherent vegetations along the damaged valve cusps. These consist of clumps of organisms, fibrin and platelets. Clinical features of bacterial endocarditis are indicated in Figure 2.9.

Clinical features

Clinical features are due to both local and systemic effects. Local valvular damage causes incompetence or stenosis of the valve, producing a new heart murmur that may change as the damage progresses. Distant effects occur as parts of the vegetation break away and pass in the blood stream to lodge at distant sites (e.g. kidney and brain) where they cause further local infection (septic emboli). Immune complexes are deposited at various sites in the body and cause rashes, nail splinter haemorrhages and, rarely, nodules in the skin of the fingers (Osler's nodes). Finger clubbing may

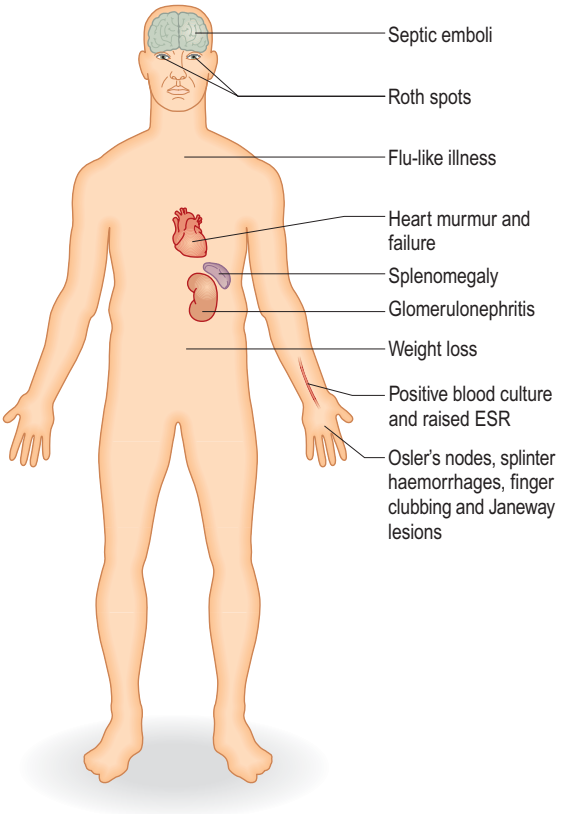


Fig. 2.9 Clinical features of bacterial endocarditis.

also occur later in the process. Systemic effects include 'flu-like' illness and weight loss.

Diagnosis

Diagnosis is made primarily from the history, the results of multiple blood cultures and visualizing the cardiac vegetations using ultrasound.

Prevention

Cases may be prevented by antibiotic prophylaxis: administration of a high dose of antibiotic before any

■ DENTAL RELEVANCE OF INFECTIVE ENDOCARDITIS

10% of cases are thought to follow dental treatment.

50% of cases are due to *Streptococcus viridans*, an oral commensal.

Antibiotic prophylaxis should be given prior to invasive dental treatment on all patients who have had endocarditis.

Antibiotic prophylaxis should be given prior to invasive dental treatment in all patients who are at risk of developing endocarditis.

At-risk patients include those with:

- previous rheumatic fever
- congenital heart disease
- significant heart murmur (check with doctor/cardiologist)
- heart valve replacement
- degenerative valvular disease
- past episode of endocarditis.

Consult the BNF for current antibiotic prophylaxis guidelines prior to treatment in each case.

procedure that may cause a significant bacteraemia (shower of bacteria into the systemic circulation). The majority of cases originally ascribed to dental treatment are now thought to be due to a continuous low-grade bacteraemia from the 'normal' activities of chewing and cleaning of the teeth.

Heart failure

Heart failure occurs when the pumping efficiency of the heart is decreased. It is a common cause of death in the western world. Dental procedures may provoke symptoms or acute complications.

Epidemiology

There is an increased prevalence in the elderly. It affects 1% of people age 50 years increasing to 5% of people by age

75. Two-thirds of patients with heart failure will die within 5 years of diagnosis.

Aetiology

Heart failure is due to a problem with either contraction (systole) or relaxation (diastole) of the heart muscle. There are three common causes:

- hypertension
- valvular heart disease
- ischaemic heart disease.

Pathogenesis

The left or right or both ventricles may be affected, depending on the cause. Failure of the left ventricle decreases the ability to pump blood to the body (poor tissue and organ perfusion) and causes fluid to build up in the lungs (pulmonary congestion). Failure of the right ventricle leads to build up of fluid in the periphery of the body (dependent oedema). Left ventricular failure often leads to right ventricular failure and then it is called congestive cardiac failure.

Clinical features

Clinical features depend on which side of the heart is affected:

- shortness of breath which is worst on laying flat (orthopnoea)
- quick weight gain due to fluid retention (a weight gain of 1 kg in 1 day is possible)
- swelling in ankles (Fig. 2.10), legs and abdomen (dependent oedema)
- fatigue and weakness
- other symptoms, such as nausea, palpitations, chest pain, waking suddenly at night unable to breath and changes in sleep pattern.

Diagnosis

Diagnosis is often confirmed by a chest X-ray which reveals any cardiac enlargement or fluid build up in the lungs. An echocardiogram may be carried out to measure the pumping efficiency of the heart (ejection fraction).

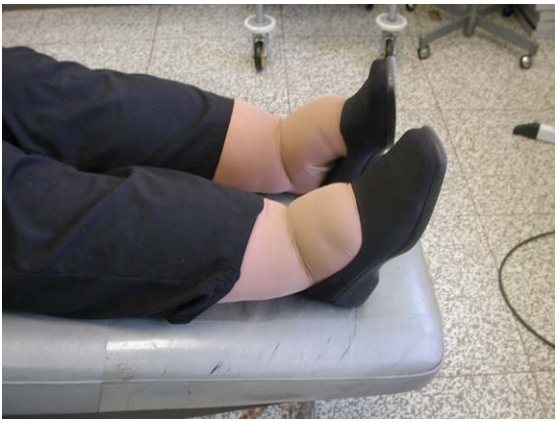


Fig. 2.10 Ankle oedema is a typical sign of right or congestive heart failure.

Treatment

Treatment is by a combination of lifestyle changes, medical treatments and, rarely, surgery:

Lifestyle
- Exercise
- Cessation of smoking
- Low-salt diet
- Weight loss

■ DENTAL RELEVANCE OF HEART FAILURE

Most patients can be treated safely in dental practice under local anaesthetic.

It is very common within the population, with 5% of over-75s affected.

Dental treatment may provoke symptoms and patients may become breathless if laid flat in the dental chair.

Determine whether their symptoms are stable or unstable. Stable patients can be treated in dental practice:

- preventative dentistry
- plan short treatments
- keep stress to minimum.

Unstable patients should be referred to their doctor before any dental treatment.

Use sedation with caution.

Medical	Diuretics to reduce fluid overload
	Inotropic drugs to increase cardiac contractility
	ACE inhibitors to decrease cardiac workload
Surgical	Valvular surgery
	Heart transplant (rare)

Congenital heart defects

Congenital heart defects (CHDs) are structural, functional or positional defects of the heart that are present at birth in approximately 1% of the population. They may manifest at any time after birth or may never be detected at all. Dental treatment of affected individuals should be carried out only after liaison with the patient's cardiologist.

Epidemiology

Some 1% of live births are affected. There are eight common lesions:

1. Ventricular septal defect (VSD)
2. Patent ductus arteriosus (PDA)
3. Atrial septal defect (ASD)
4. Pulmonary valve stenosis
5. Aortic valve stenosis
6. Coarctation of the aorta
7. Tetralogy of Fallot
8. Transposition of the great vessels.

There is a familial tendency, males and females are equally affected, and more than one defect may be present in an individual.

Aetiology

In most cases no specific causes are found and it is likely that the aetiology is multifactorial. Diseases and hazards faced in the first trimester of pregnancy are thought to be important (e.g. maternal rubella resulting in fetal PDA, VSD or ASD).

Pathogenesis

Most heart defects either obstruct blood flow in the heart or great vessels or cause blood to flow through the heart in an abnormal way. They are best divided into four groups:

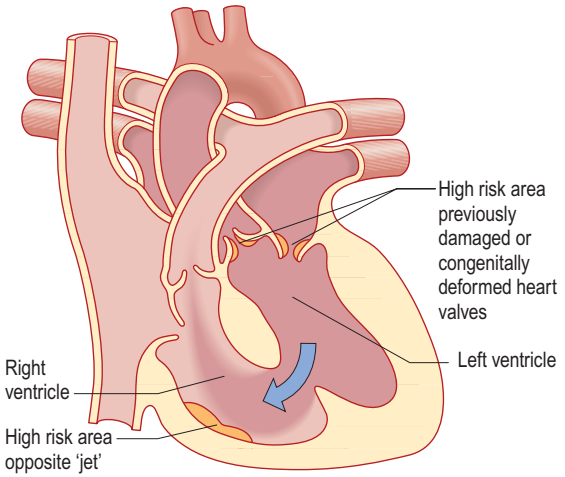


Fig. 2.11 VSD allowing a jet of abnormal blood flow from the left to the right ventricle with the area at increased risk of endocarditis.

Obstructive defects, due to narrowing (stenosis) of the normal blood flow path (e.g. pulmonary/aortic valve stenosis).

Septal defects ('hole in the heart'), in which blood is allowed to flow abnormally between the left and right heart chambers. In a VSD (Fig. 2.11) there is a persistent hole between the left and right ventricle allowing blood to flow from the left to the right side of the heart without passing round the systemic circulation.

Cyanotic defects, in which blood pumped to the body contains less oxygen than normal, causing cyanosis (blue discoloration of the skin and mucous membranes) due to the presence of deoxygenated haemoglobin (e.g. tetralogy of Fallot).

Shunts, in which blood is diverted along an abnormal or persistent fetal passage (e.g. PDA).

Clinical features

Despite the large number of defects that exist there are only a limited range of presentations. The most common include:

- cyanosis if blood is not properly oxygenated
- heart failure due to abnormal pressures or volumes within the heart

■ DENTAL RELEVANCE OF CONGENITAL HEART DEFECTS

These defects are common within the population, affecting 1% of patients.

Many will require antibiotic prophylaxis before high-risk dental procedures (see endocarditis).

Liaise with the patient's doctor before embarking on treatment.

Preventative dental treatment should be a priority in order to avoid excessive antibiotic prescription and reduce dental sepsis.

- heart murmur if there is turbulent flow, i.e. VSD
- growth failure
- chest infections.

Treatment

This normally involves surgical correction.

Dental treatment

Dental treatment should not be undertaken without consultation with the patient's medical practitioner. The majority of affected individuals can be treated in dental practice but may require antibiotic prophylaxis as they have an increased risk of developing endocarditis. At-risk patients include those with uncorrected cyanotic defects, valvular defects or replacement and heart murmurs due to abnormal shunts.

Patients with heart failure may not tolerate the supine position in a dental chair and become breathless.

Deep vein thrombosis

Deep vein thrombosis (DVT) is due to the formation of a blood clot in the deep veins of the lower limb. These clots may release fragments (emboli) which travel to the lungs, causing a pulmonary embolism.

Epidemiology

DVT is a very common but often asymptomatic problem. It can occur at any age but it is more common in those over 60.

Aetiology

This is due to one of three factors, known as Virchow's triad:

1. Stasis of normal blood flow due to:
   - prolonged bed rest
   - general anaesthesia
   - long-distance economy travel
   - pregnancy.
2. Damage to the blood vessel wall due to:
   - trauma
   - inflammation (phlebothrombosis).
3. Composition change of the blood due to:
   - lupus anticoagulant
   - antithrombin III
   - malignancy
   - oral contraceptive.

Pathogenesis

Once formed, the blood clot enlarges and obstructs the local venous blood flow and may release fragments into the systemic circulation (see Fig. 2.12).

Clinical features

Usually only one lower limb is affected and the symptoms include:

- swelling
- tenderness and pain
- erythema of the overlying skin.

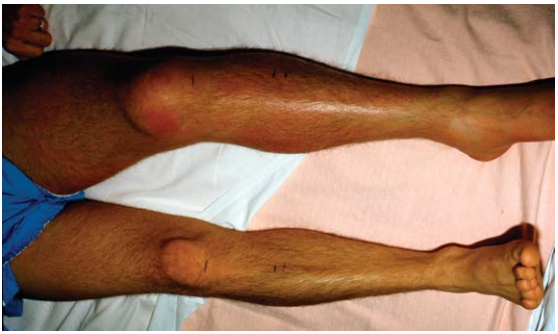


Fig. 2.12 Swollen leg due to deep vein thrombosis.

Prevention

It is important to prevent the intraoperative formation of a DVT and this is carried out by:

1. Identification of high-risk patients and procedures
2. Prophylactic anticoagulation
3. Careful positioning and padding of patients on the operating table
4. Use of compression stockings and boots
5. Adequate fluid replacement
6. Expeditious operating
7. Early postoperative mobilisation.

Treatment

Most patients are anticoagulated using warfarin to facilitate the resorption, prevention of extension and reformation of the DVT.

■ DENTAL RELEVANCE OF DVT

DVT may lead to pulmonary embolism.

Patients are often anti-coagulated, with warfarin, and require special care before invasive dental procedures are carried out (cf. haematology).

3 RESPIRATORY DISEASES

Richard Cook

Respiratory examination

The respiratory history and examination of the clothed patient begins when you first meet in the waiting room and should continue throughout the appointment as you monitor the patient's wellbeing. The vast majority of patients who suffer from respiratory disease can be safely treated under local anaesthetic in your surgery.

History

Using a few careful questions it is possible to quickly assess the patient's general status:

'Are you generally fit and well?'
'Do you have any chest problems?'
'Do you suffer with shortness of breath?'
'What are your current medications?'

You will be happy with most patients' respiratory status at this point. If you are concerned at this point then you can ask more specific questions to investigate the problem further and assess the patient's response to previous dental treatment. If at this stage you are still concerned then you should seek advice from the patient's doctor or respiratory physician before embarking on further treatment.

Remember that many respiratory conditions fluctuate and assessment of their current status is important – meaning that treatment may need to be postponed during acute exacerbations.

Many of the signs and symptoms are non-specific and need to be set into context with the history, examination and medications taken.

Cough is a common and often non-specific symptom which may arise as a result of acute or chronic chest disease, cardiac problems or even psychological conditions.

Haemoptysis is the coughing up of blood from the respiratory tract. There are many causes, including chest infection, inhaled foreign body, pulmonary oedema or lung cancer.

Chest pain is another common symptom which may be due to respiratory or cardiac disease, trauma or psychological conditions. If related to inspiration it is more likely to be of respiratory origin.

Dyspnoea is the sensation of shortness of breath. The speed of onset and duration may indicate the cause. Rapid onset: asthma, pulmonary embolus or inhaled foreign body. Gradual onset: obstructive airway disease or malignancy. If dyspnoea is increased by lying flat (orthopnoea) then this may indicate a cardiac cause.

Examination (remember: look and listen and then feel)

General appearance

Breathlessness at rest (respiratory rate >12/minute) may indicate a respiratory or cardiac problem. When severe, the patient may have difficulty speaking, i.e. not being able to complete their sentences.

The presence of abnormal respiratory sounds may indicate a respiratory abnormality

Wheeze, which is a multi-pitch (polyphonic) expiratory sound, may indicate small-airway obstruction, i.e. asthma.
Stridor is a harsh rasping sound on inspiration and is indicative of upper airway obstruction such as laryngeal oedema due to allergic swelling or a foreign body in the larynx.

Examination of the patient's hands

Finger clubbing – doming of nails and loss of paranychial skin fold angle – may be seen. The respiratory aetiologies are bronchial carcinoma, chronic suppurative lung disease, asbestosis, fibrosing alveolitis and familial. (Others include chronic inflammatory GI and hepatic conditions, endocarditis and cyanotic cardiovascular disease.)

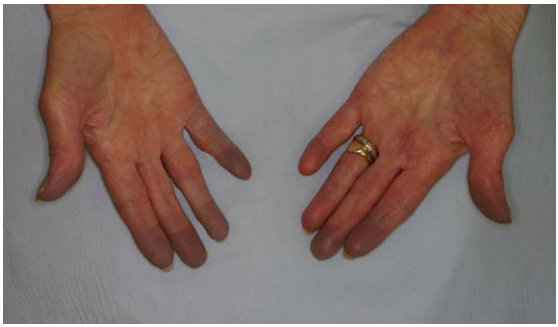


Fig. 3.1 Peripheral cyanosis causes blue discoloration of the extremities.

Examination of the face and oral cavity

Cyanosis refers to blue discoloration of the skin and mucous membranes (>5 g/dl deoxyhaemaglobin). If detectable only in the extremities (Fig. 3.1) it indicates poor perfusion (cold) or cardiac output, whereas if detectable centrally it reflects hypoventilation, parenchymal lung disease, heart failure and right-to-left arterio-venous malformation.

Cervical lymphadenopathy refers to the presence of palpable lymph nodes in the neck; it may be caused by infective or neoplastic lung diseases.

Horner's syndrome, with unilateral drooping of upper eye lid, constriction of the pupil and lack of facial sweating, may be due to infiltrative spread of a lung tumour to the cervical sympathetic chain.

Asthma

Asthma is an increasingly common, reversible, obstructive airway condition currently affecting 5% of the UK population. There are three characteristic features: hyper-responsive airways, inflammation and excess mucus production. Care must be taken not to provoke an acute attack or further suppress respiration during dental treatment by inappropriate prescribing, excessive stress or the indiscriminate use of sedation.

Epidemiology

Asthma affects 5% of the UK population. There is an increasing incidence in children, with >10% affected. It is most commonly diagnosed in the first decade of life with a second peak in the third and fourth decades. There is a similar incidence in males and females. Asthma may indicate an atopic tendency with multiple allergic reactions to other compounds and medications.

Aetiology

Aetiology involves a mixture of intrinsic and extrinsic factors and lifestyle/environmental factors.

Extrinsic	Pollen
	Faeces of house-dust mites
	Salivary proteins
	Pet fur
Intrinsic	Atopy with raised IgE levels
	Asthma gene (chromosome 11)
	Bronchial hyper-reactivity
Lifestyle/environmental	Exercise (>cold air)
	Stress
	Smoking
	Drugs (NSAIDs, β-blockers)
	Pollution
	Viral infections (upper respiratory tract infections)

Pathogenesis

There is a triad of oedema, broncho-constriction and mucous plugging, affecting the bronchioles. This is reversible and causes a variable degree of obstruction. It is caused by mast cell degranulation and release of vasoactive amines (e.g. histamine and prostaglandin).

Clinical features

Clinical features depend on the severity of the disease, ranging from the classic polyphonic expiratory wheeze to an inability to breathe, with cyanosis and coma. In children the only feature may be a nocturnal cough which keeps the child awake. When severe, speech will be affected and the patient may not be able to complete their sentences and will become increasingly anxious and panicked.

Life-threatening features	Cyanosis
	Absent breath sounds
	Confusion
	Exhaustion

Treatment

Treatment is carried out in a series of steps (Fig. 3.2) which can be used as a guide to assess the severity of your patient's disease

Education is concerned with the cause and effect and the regular use of prophylactic medication.

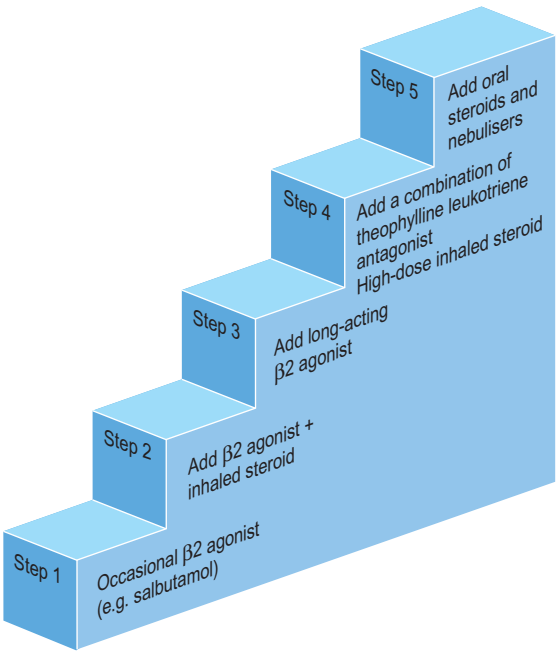


Fig. 3.2 Stepwise treatment of asthma of increasing severity.

Prevention involves the avoidance of house dust, pet fur and cold air and the cessation of smoking.

Treatment is monitored by assessment of symptoms and measurement of the peak expiratory flow rate using a peak flow meter (Fig. 3.3).

Fig. 3.3 Measurement of peak expiratory flow rate using a peak flow meter in an asthmatic patient.

■ DENTAL RELEVANCE OF ASTHMA

Most asthmatics can be treated safely under LA.

Non-steroidal anti-inflammatory drugs may precipitate or worsen an asthma attack in sensitive patients.

Inhaled steroids may cause changes in the oral mucosa, e.g. *Candida* infection in the palate. Advise patients to rinse with warm water after using their steroid inhaler.

Postpone treatment during exacerbations of the condition.

Use sedation with care.

Advise patient to use their inhaler at the beginning of each treatment session.

Patients who are taking steroids no longer need steroid cover for dental treatment under LA.

Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is caused by a group of lung diseases that lead to damage of lung tissue with persistent and progressive limitation of air flow. Chronic bronchitis and emphysema are the two most common causes of COPD and often coexist. Smoking is implicated in 95% of cases.

Epidemiology

In the United Kingdom, COPD currently costs the health service £500 million per year. Between 15 and 20% of long-term smokers will develop the condition.

Aetiology

Four major agents are involved:

- smoking (95% of cases) active and passive
- atmospheric pollution
- respiratory infection
- α_1-antitrypsin deficiency (pulmonary protective protease).

Pathogenesis

In chronic bronchitis there is damage to the respiratory epithelium with ulceration, excess mucus production and variable airway narrowing. In emphysema there is air-space dilation and loss of elastic tissue within the alveolar walls – leading to gas trapping, over inflation and limitation of expiratory airflow.

Clinical features

These are variable and depend on the dominant pathological process. Many patients are not aware of the problem until significant damage has occurred. There is often a chronic cough, production of excess sputum, shortness of breath and an expiratory wheeze. In contrast to asthma, bronchodilators do not offer the same degree of relief. There are number of potential complications as the disease progresses, including:

- respiratory failure
- right heart failure (cor pulmonale)
- pneumothorax (burst bullea).

Treatment

This has many aspects and involves a multidisciplinary team approach. Treatment strategies:

1. Cessation of smoking is one of the most important steps in the treatment of COPD and every effort should be made to help achieve this.
2. Drug therapy – targeted antibiotics, bronchodilators, inhaled and oral steroids.
3. Non-invasive ventilation.
4. Influenza and pneumococcal vaccination.
5. Rapid management of exacerbations.
6. Breathing exercises and chest physiotherapy.
7. Pulmonary rehabilitation.

Many of these patients rely on low oxygen tension to drive their respiration and should not be given high-flow oxygen over long periods of time, i.e. relative analgesia.

Figure 3.4 shows the use of a nebuliser.

In an emergency situation you should use 100% oxygen at a high flow rate.

Fig. 3.4 A patient with COPD using a nebuliser at home.

■ DENTAL RELEVANCE OF COPD

Most patients with COPD can be treated safely under local anaesthetic.

Keep treatment sessions short.

Patients may find it difficult to cope with a rubber dam due to increased airway obstruction.

Inhaled steroids may cause changes in the oral mucosa, i.e. *Candida* infection in the palate. Advise patients to rinse with warm water after using their steroid inhaler to help prevent this occurring.

Postpone treatment during exacerbations of the condition.

Do not sedate these patients or use relative analgesia.

Advise patient to used their inhaler at the beginning of each treatment session.

Lung cancer

Lung cancer is the most common malignancy in the western world and third most common cause of death in the UK. Patients may present to their dental practitioner with head and neck symptoms.

Epidemiology

Lung cancer is a leading cause of cancer death in the UK. The incidence in the male population plateaued in the mid 1980s, while rates in females have increased 150% in the same period. It is rare in those under 45 years of age with median age at diagnosis of 69 years.

Aetiology

Smoking is the main cause of lung cancer, accounting for up to 90% of cases. The risk increases with the intensity, quantity and duration of smoking. There is increasing evidence that passive smoking offers a significant risk to those exposed. Air pollution is known to increase the incidence of lung cancer. There are certain occupational hazards, including exposure to dust from coal and asbestos.

Pathogenesis

There are two major groups of lung cancer, accounting for 90% of cases:

- small-cell lung cancer (SCLC)
- non-small-cell lung cancer (NSCLC).

SCLC commonly occurs centrally in the lungs, has a rapid growth rate and metastasises early.

NSCLC tends to have a slower rate of growth and tends to metastasise later.

Clinical features

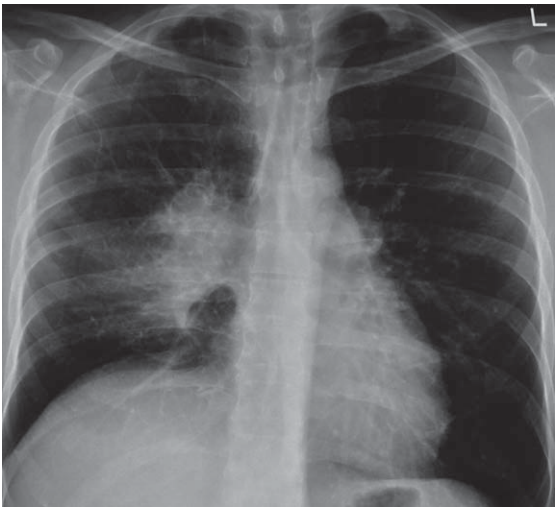
In most patients the diagnosis is made too late for an effective cure. Lung cancers have local, systemic and metastatic effects:

Local	Persistent cough
	Prolonged chest infection
	Chest pain ↑ by breathing and coughing.
	Haemoptysis (coughing up blood)
	Progressive shortness of breath
	Hoarse voice (recurrent nerve invasion)
	Horner's syndrome
	Distended neck veins (superior vena caval obstruction)
Systemic	Finger clubbing
	Weight loss
	Malaise
	Anaemia
	Ectopic hormone production
Metastatic spread to bone, brain and liver, causing:	
	Bone pain
	Neurological deficits
	Jaundice

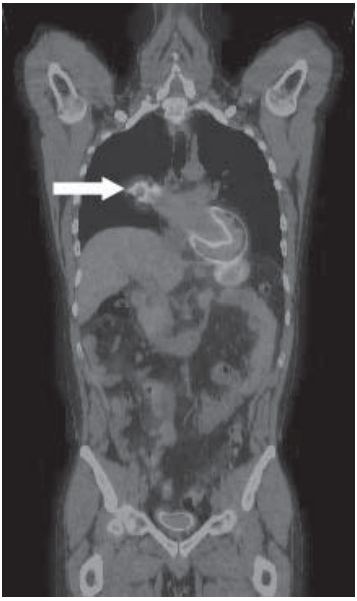
Treatment

Treatment depends on the tumour histotype, location, degree of spread and fitness of the patient. Curative surgical treatment is possible in only 20% of cases. In most patients only palliative treatment can be offered, including radio- and chemotherapy, supportive therapy and analgesia.

Figure 3.5 shows a cancerous growth in the right lung as viewed by X-ray (A) and positron emission tomography (B).



A



B

Fig. 3.5 Chest X-ray (A) and positron emission tomography (B) showing a cancerous growth in the right lung, highlighted by arrow.

■ DENTAL RELEVANCE OF LUNG CANCER

Advice on how to stop smoking, and support, should be given to all smokers.

Lung cancer can present with:

- cervical lymphadenopathy
- distended neck veins
- Horner's syndrome – meiosis, ptosis and anhydrosis.

Inhaled foreign body

There is a risk of inhalation of foreign bodies during dental treatment due to the instrumentation used and the position of the patient during treatment.

Epidemiology

Dental treatment has been reported to cause up to 27% of all foreign body inhalation incidents. It is more common in adult patients and the incidence has increased with the introduction of implant dentistry.

Aetiology

There is an increased risk of inhalation of foreign bodies during dental treatment due to:

- the close proximity between the operative field and the airway
- small instruments used in dentistry
- supine position of the patient
- sedation-induced reduction in reflex airway protection.

Pathogenesis

Typically the object passes down the most direct route from the pharynx into the right main bronchus (Fig. 3.6) and the right lower lobe of the lung. If the object is not retrieved rapidly there is a risk of lung abscess or pneumonia. Impacted objects may irritate the respiratory tract and obstruct the airflow, causing cough, stridor and wheeze.

Clinical features

The majority of foreign bodies will be swallowed and pass into the alimentary tract, carrying a small risk of perfora-

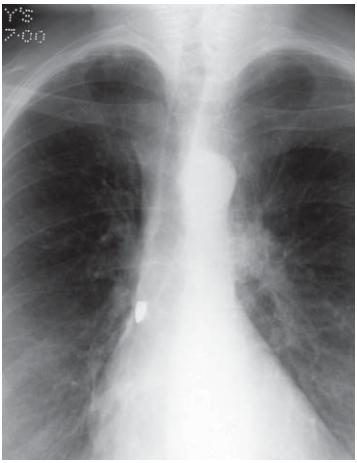


Fig. 3.6 A crown lodged in the right main bronchus.

tion as they pass through. If inhaled, large objects are likely to impact above the vocal cords, whereas smaller objects are more likely to pass into the lung. The patient may cough and choke but this is not always the case and inhalation may occur asymptomatically. Presentation may be delayed, with signs and symptoms of chronic lung infection.

Diagnosis

This depends on a high degree of clinical suspicion and acumen. If you cannot locate a missing object it is mandatory to refer the patient for a chest X-ray in an attempt to exclude inhalation.

Treatment

Having 'lost' an object during treatment you are obliged to locate it. Remember that most 'lost' instruments will be found around the patient or in the oral cavity. If it cannot be found you must consider that it has been inhaled. Ask the patient to cough and turn their head to the side to aid location of the object. Use suction and good lighting to examine the oropharynx and carefully retrieve any objects with forceps. If the patient is in distress the emergency services should be contacted immediately and high-flow oxygen should be given, along with reassurance. The patient may benefit from a sharp blow on the back or the Heimlich manoeuvre (Fig. 3.7). You should not attempt to

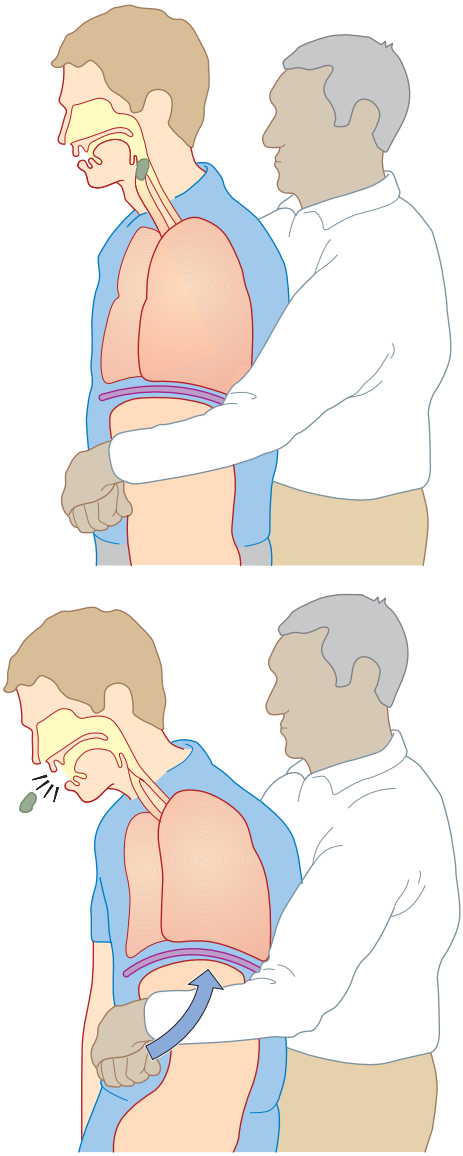


Fig. 3.7 The Heimlich manoeuvre used to aid retrieval of an inhaled foreign body.

■ DENTAL RELEVANCE OF INHALED FOREIGN BODY

Up to 27% of inhalation incidents occur during dental treatment.

The risk of inhalation is increased when consciousness is impaired, e.g. sedation.

Inhalation may occur asymptomatically.

You are obliged to ascertain the location of objects 'lost' during dental treatment.

If you cannot locate a missing object a chest X-ray should be carried out to attempt to exclude inhalation.

If there are signs of airway obstruction the emergency services should be called and high-flow oxygen administered.

Do not attempt a surgical airway unless appropriately trained.

Patients who have ingested sharp objects, e.g. endodontic files, should be referred to hospital for assessment.

create any form of surgical airway unless you have been given the appropriate training.

Inhaled objects can usually be retrieved using a bronchoscope; this becomes more difficult the longer the object has been present.

Prevention

This is best carried out by identification of high-risk procedures and taking the appropriate precautions. Risk is increased by the use of small instruments in patients with reduced reflexes. The airway can be protected by:

- use of rubber dam
- placement of pharyngeal sponges
- restraining cords on instruments
- avoidance of over sedation
- training in instrument handling.

Other pulmonary disease

Pulmonary embolism

This occurs due to the blockage of a portion of the arterial system in the lungs. It is most commonly due to a blood

clot shed from a deep vein thrombosis (DVT) in the patient's lower limb.

Epidemiology

Pulmonary embolism (PE) is detected in up to 60% of post-mortems. In many cases it is minimally symptomatic and thus undiagnosed. However, 10% of PEs are thought to be fatal.

Aetiology

PE is most commonly due to emboli released from blood clots formed in the lower limbs as a result of deep vein thrombosis. Less commonly the emboli may be formed from fat, air or amniotic fluid.

Pathogenesis

The embolus impacts and obstructs a portion of the pulmonary arterial circulation (Fig. 3.8), resulting in collapse of the alveoli in the area and decreasing the efficiency of gas exchange.

Clinical features

These depend on the position and size of the area obstructed and include:

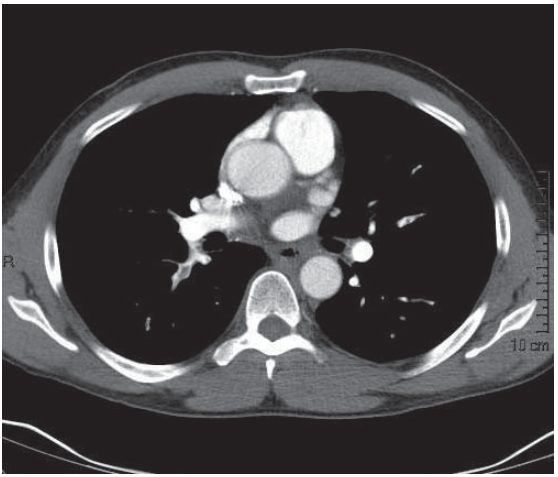


Fig. 3.8 A CT scan of a pulmonary embolus. The arrow indicates the embolus in the right pulmonary artery.

■ DENTAL RELEVANCE OF PULMONARY EMBOLISM

Patients are often anticoagulated with warfarin, and require special care before invasive dental procedures are carried out (cf. haematology).

- sudden onset of chest pain
- acute shortness of breath
- haemoptysis (coughing up blood)
- collapse
- sudden death.

Treatment

In life-threatening cases the embolus may be dissolved using thrombolytic drugs and then further thrombosis is prevented by anti-coagulation, most commonly with warfarin.

Sarcoidosis

Sarcoidosis is a common chronic granulomatous condition of unknown origin that may cause cervical lymph node enlargement and parotid gland swelling.

Sarcoidosis has a worldwide distribution, affecting females more than males with an increased incidence in those of Afro-Caribbean origin. It is most commonly diagnosed between the ages of 20 and 40. The lungs and lymph nodes are most commonly affected, but any tissue or organ may be involved.

Most patients are asymptomatic. Those who develop symptoms usually recover spontaneously; some may follow a relapsing, remitting course.

Only 40% of those patients with symptoms require treatment with immune suppression.

■ DENTAL RELEVANCE OF SARCOIDOSIS

Some 4% of patients with sarcoidosis develop parotid gland enlargement and a dry mouth.

Cervical lymph node enlargement occurs in 15%. Sinus involvement may lead to repeated sinusitis.

Patients may be treated with immune suppressant drugs, increasing their risk of infection.

Table 3.1
Causes of haemoptysis[a].

System	*Nature of cause*	*Example*
Respiratory	Traumatic	Inhaled foreign body, intubation
	Infective	**Bronchitis, pneumonia, TB,** lung abscess, bronchiectasis
	Neoplastic	**Bronchial CA**
	Vascular	**PE,** vasculitis
	Lung tissue	Sarcoidosis, cystic fibrosis
Cardiovascular	Heart failure	Pulmonary oedema
	Valvular disease	Mitral stenosis
	Degenerative	Aortic aneurysm
Bleeding diathesis	Congenital	Haemophilia
	Acquired	Warfarin

[a]The most common causes are highlighted in bold.

Haemoptysis

Haemoptysis is the coughing up of blood most commonly due to respiratory disease. This must be distinguished from haematemesis (vomiting of blood). The blood is usually frothy, alkaline and bright red and there is often a history of chest disease. This is rarely life-threatening but may indicate serious underlying pathology. Table 3.1 lists causes of haemoptysis.

Acute breathlessness

Patients commonly become short of breath during dental treatment. In most cases this is due to anxiety and will respond to reassurance. There are a number of more serious causes which we must be aware of.

Anxiety

Most patients experience a degree of anxiety when attending for dental treatment and this may result in breathlessness. In most cases careful explanation and reassurance is all that is required to prevent any complications. In cases of severe anxiety it may better to carry out the treatment

using a supplemental therapy to aid relaxation, e.g. hypnotherapy, acupuncture, intravenous/oral sedation or relative analgesia.

Hyperventilation

Hyperventilation – extreme over-breathing – may occur in patients who are anxious or phobic. In such cases the pH of the plasma is increased as CO_2 is blown off. This causes the Ca^{2+} in the plasma to be reduced and as a result muscle and nerve transmission are affected causing the patient to experience tingling of their extremities or muscle spasm (carpopedal spasm). This is treated by making the patient re-breathe from a paper bag to raise their CO_2 level. Further treatment may require supplemental relaxation therapy.

Pain

Inadequate analgesia may cause the patient to become short of breath. This is best remedied by supplemental anaesthetic or, on occasion, it may be necessary to delay treatment and give the patient a course of antibiotics to settle any acute infection.

Asthma

Asthma may be provoked by anxiety, stress or the inappropriate prescription of NSAIDs to sensitive patients. It is characterised by difficulty in breathing out with an expiratory wheeze.

COPD

Patients with COPD tend to be chronically short of breath but the situation may be made worse by the use of a rubber dam, for example, which may further restrict air flow. If the patient has developed heart failure they may become acutely breathless in the supine position.

Anaphylaxis

Swelling of the tongue and laryngeal tissues may occur during anaphylaxis, causing acute airway obstruction. In addition to this, bronchospasm causes an asthma-like state with reduced airway patency and an expiratory wheeze.

Inhaled foreign body

The patient will often cough at the time of inhalation and – depending on where the object impacts – may become acutely short of breath.

Heart failure

When affecting the left ventricle, heart failure causes fluid to accumulate in the lungs, leaving the patient short of breath. This is often made worse by laying the patient backwards during treatment.

Angina

Angina may manifest as acute shortness of breath without the classical signs of central chest pain. Patients often have a history of cardiovascular problems, e.g. myocardial infarction.

Myocardial infarction

This condition often causes crushing central chest pain leading to acute shortness of breath. In addition to this the patient may develop sudden left heart failure and pulmonary oedema, making the situation worse.

Pulmonary embolus

This often causes acute shortness of breath with chest pain that is increased by the act of breathing. There may be a recent history of deep vein thrombosis or prolonged immobility, e.g. economy air travel.

Pneumothorax

This is due to the escape of air into the pleural space, causing the affected lung to collapse (Fig. 3.9). This may occur spontaneously or, more commonly, is due to trauma or lung pathology. Those with a history of chronic lung disease, e.g. emphysema, are at a higher risk. Tall patients are at a higher risk of spontaneous pneumothorax.

Lung cancer

This causes progressive shortness of breath but there may be an acute exacerbation if there is an associated pneumothorax or haemorrhage from the tumour leading to acute obstruction of the airway or haemothorax.

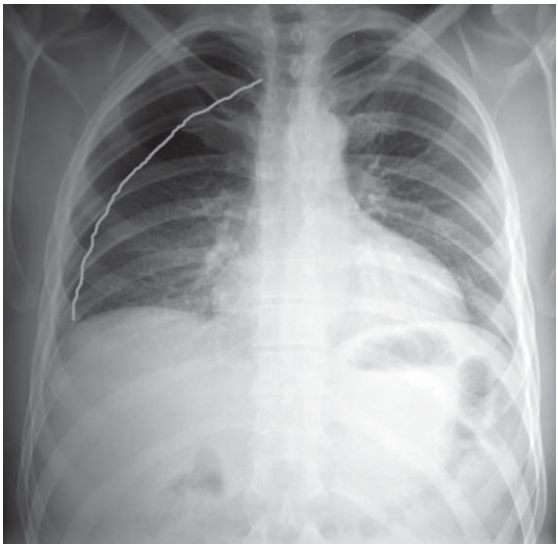


Fig. 3.9 A chest X-ray showing a right-sided pneumothorax. The line indicates the edge of the collapsed lung.

■ DENTAL RELEVANCE OF ACUTE BREATHLESSNESS

If not due to anxiety, sudden onset of shortness of breath is a serious sign.

In all cases stop treatment and assess the patient.

Administration of high-flow oxygen 15L/minute will do no harm.

If there is no immediate resolution the emergency services should be called.

Useful web sites

http//www.brit-thoracic.org.uk
British thoracic society, guidelines for respiratory disease management.
http//www.ginaasthma.com
Global initiative for asthma.
http//www.goldcopd.com
WHO global initiative for COPD management and diagnosis.

4 GASTROENTEROLOGY

Mike Escudier

Gastrointestinal disorders and diseases are common and may be divided into:

Inflammatory: peptic ulcer disease, coeliac, Crohn's and Ulcerative colitis

Neoplastic: carcinoma of the oesophagus, stomach and colon

Functional: achalasia, diverticulosis and irritable bowel syndrome.

Oesophageal disorders

Oesophageal disorders usually present with one or more of the following symptoms:

- *Dysphagia* (difficulty in swallowing). Dysphagia is commonest in the elderly in whom stroke is the leading cause. Causes of dysphagia are listed in Table 4.1.
- *Pain*. Pain may result from acid reflux or spasm and may be impossible to distinguish from cardiac pain.
- *Cough or vomit*. If food or liquids do not pass normally to the stomach they may reflux back to the pharynx, overflow into the lungs or present as a cough.

Pharyngeal pouch

This consists of a herniation of mucosa through a weakness in the pharyngeal constrictor muscles. The pouch hangs down due to gravity and the patient complains of dysphagia combined with a swelling developing in the lower part of the neck, usually on the left side. Undigested food may be regurgitated into the mouth hours after eating. Overflow into the lungs may cause respiratory symptoms.

Table 4.1
Causes of dysphagia.

	Oral and pharyngeal	*Oesophageal*
Obstructive	Tumours, e.g. oral SCC	Tumours, e.g. SCC/adeno CA
	Inflammation, e.g. severe candidosis, Herpes	Inflammation, e.g. acid reflux, drugs, chemical burn
	Fibrosis, e.g. scleroderma	Stricture, e.g. radiation, acid reflux
	Trauma, e.g. fish bones	Swallowed foreign body
	Surgery	Surgery
	Webs	Lymphadenopathy
	Pouches	Goitre
	Deformity of cervical spine	Webs
	Xerostomia, e.g. Sjögrens or drugs	
Neuromuscular	Stroke	Oesophageal spasm
	Parkinson's disease	Achalasia
	MS	
	Myasthenia gravis	
Psychogenic	'Globus hystericus'	

Treatment
Treatment is carried out endoscopically.

Achalasia
Loss of ganglia from intramural plexus leads to a failure of relaxation of the gastro-oesophageal sphincter. This produces a functional obstruction to oesophageal emptying with dysphagia for solids and liquids. Failure of peristalsis leads to progressive dilatation of the oesophagus. Retained oesophageal contents may be regurgitated causing respiratory problems.

Treatment
This can involve drugs, e.g. nifedipine to relax the sphincter, balloon dilatation or cardiomyotomy.

Oesophageal spasm

A descriptive term for spasm due to a variety of causes leading to attacks of dysphagia and pain. *Causes*:

- Atypical achalasia
- Gastro-oesophageal reflux
- Motor disorders
- Symptomatic peristalsis
- Obstruction at the cardia
- Neuromuscular disorders.

This condition is diagnosed by the radiological appearance or by oesophageal pressure reading.

Oesophageal web

This condition was first described in Britain by Paterson and Brown-Kelly and later in the USA by Plummer and Vinson. It is classically seen in middle-aged women and consists of:

- glossitis
- iron deficiency anaemia
- dysphagia
- koilonychia.

The mucosa becomes atrophic and a fibrous stricture forms at the upper end of the oesophagus, seen as a 'web' on a barium swallow. This is pre-malignant.

Treatment

Dilatation of the stricture and correction of the iron deficiency.

Carcinoma of the oesophagus

Epidemiology

Carcinoma of the oesophagus accounts for 3% of cancer deaths in the UK. It is uncommon below the age of 50 years and is commoner in males than in females, reflecting its aetiology. There is a particularly high incidence in China due to fungal contamination of food.

Aetiology

A history of smoking increases the risk fivefold while heavy consumption of alcohol raises it 20-fold. Other promoting factors include food toxins, pharyngeal pouch,

■ DENTAL RELEVANCE OF CARCINOMA OF THE OESOPHAGUS

Patients may present with dysphagia which may be due to malignancy.

Medicines may need to be prescribed in elixir form.

peptic ulcer disease, achalasia of the cardia, coeliac disease and iron deficiency – causing oesophageal web.

Pathogenesis

The majority are squamous cell carcinomas while in the lower third they may be adenocarcinomas. These usually show diffuse infiltration with spread to adjacent structures, including lymph nodes, but may fungate into the lumen. Later the carcinoma metastasises to the liver.

Clinical features

Patients usually present with dysphagia of gradual onset, which is initially to solids and later fluids. They may also present with pain on swallowing or the effects of local spread such as tracheo-oesophageal fistulation or recurrent laryngeal nerve palsy.

Diagnosis

Diagnosis is made from the history, barium swallow, endoscopy and biopsy. A CT scan may be required to stage the disease and plan treatment.

Treatment

Treatment will depend on whether the aim is cure or palliation. In the former case surgery will be required. Palliation may take the form of surgery, radiotherapy (not adenocarcinoma) or stenting. The 5-year survival rate is 3–6% with 75% of patients dead within 1 year.

Peptic ulcer disease and oesophageal reflux

Acid refluxing into the oesophagus may cause pain, ulceration and spasm. Peptic ulcers can affect the oesophagus, stomach or duodenum.

Epidemiology

This is particularly common in the developed world, affecting 10% of the population. The incidence in males is greater than that in females and increases with age.

Aetiology

This is most commonly due to mucosal inflammation caused by acid and pepsin, with *Helicobacter pylori* infection and stress. In the oesophagus ulceration is usually related to acid reflux while in the stomach it occurs due to decreased mucosal resistance, induced by smoking and/or NSAIDs. Duodenal ulceration is related to increased gastric acid production, which the duodenal secretions are unable to neutralise. In all cases stress may play an important role.

Pathogenesis

Ulcers in the oesophagus usually affect the lower part, while the lesser curve and pyloric antrum are common sites in the stomach as well as the first part of the duodenum. The natural history of the ulcers may lead to healing with scarring which may cause strictures. Alternatively, the ulcer may become chronic with episodes of activity alternating with quiescence. Other complications include acute bleeds or perforation.

Clinical features

The classical presenting features are pain, which is relieved by eating. If a stricture develops this may be replaced by vomiting while an acute bleed is a cause of haematemesis (vomiting of blood).

Diagnosis

This is made from the history and supplemented with a barium meal and/or endoscopy.

Treatment

Treatment involves the modification of risk factors and medical therapy with antacids, H_2 blockers, e.g. ranitidine and proton pump inhibitors, e.g. omeprazole. In the case of duodenal ulceration therapy will include eradication of any *Helicobacter pylori* infection.

■ DENTAL RELEVANCE OF PEPTIC ULCER DISEASE

Prescription of NSAIDs must be avoided.

Inflammatory bowel disease

Coeliac disease

Epidemiology

The prevalence is 1:1800.

Aetiology

This is due to sensitivity to gluten (wheat, barley, rye, oats) and has an HLA associated with B8.

Clinical features

This disease classically presents with a change in bowel habit (COBH). There is an increased frequency and bulky, offensive frothy greasy stool which flush with difficulty. There is often associated abdominal colic, weakness and weight loss. If the onset is in childhood this results in short stature and a 'failure to thrive'.

Diagnosis

This is based on the presence of antibodies to gliadin and endomysium. Augmented by an endoscopic biopsy.

Treatment

This is based on adherence to a gluten-free diet.

■ DENTAL RELEVANCE OF COELIAC DISEASE

Malabsorption of B_{12}, folate and iron causing:

- oral ulceration
- glossitis
- angular chelitis
- anaemia.

Bleeding tendency due to malabsorption of vitamin K.

Enamel defects may occur in the permanent dentition if the onset is in childhood.

Crohn's disease

This is a chronic granulomatous disease that may affect any part of the gastrointestinal tract from the mouth to the anus. The terminal ileum is most commonly involved.

Aetiology

Unknown, but usually starts in teens or early twenties with a second peak in old age.

Prevalence

1:1300–1:2500.

Clinical presentation

Intermittent abdominal pain, diarrhoea, abdominal distension (90%), anaemia and weight loss (50%), clubbing (50%), fresh blood or melaena (40%), fever (30%), fistulae and perianal sepsis (20%), uveitis, arthritis, skin rashes (erythema nodosum).

Diagnosis

Based on the appearance seen on barium enema ('rose thorn', 'skip lesions', 'string sign'), endoscopic examination, colonoscopy/ileoscopy and biopsy. The differential diagnosis includes other granulomatous conditions such as tuberculosis and sarcoidosis. Labial swelling in Crohn's disease is shown in Figure 4.1.

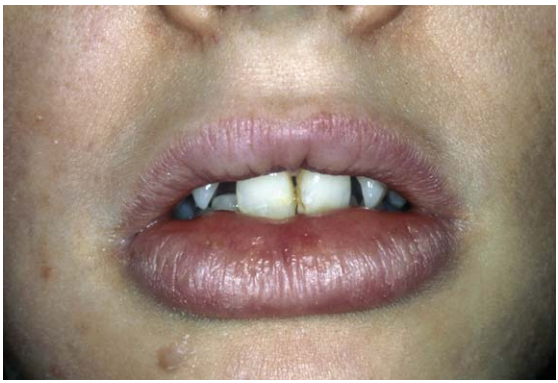


Fig. 4.1 Labial swelling due to Crohn's disease.

■ DENTAL RELEVANCE OF CROHN'S DISEASE

Oral manifestations are likely to occur at some stage in most patients and include:

- ulceration
- cobblestone appearance of the mucosa
- facial and labial swelling
- mucosal tags.

Malabsorption (particularly B_{12}) may lead to oral complications.

Immunosuppression increases the infection risk.

Treatment

Medical management includes correction of any nutritional deficiencies and the use of immunosuppressives such as prednisolone (acutely), azathioprine (long-term and disease-modifying) as well as other immunomodulating medications such as sulphasalazine and mesalazine. Surgical intervention may be necessary, particularly for the management of obstruction, fistulae and adhesions.

Ulcerative colitis

This is a chronic inflammatory bowel disease that affects the colon with backwash involvement of the terminal ileum.

Aetiology

Unknown, but usually presents in the age group 20–40 years. There is a probably a genetic basis with a close link to ankylosing spondylitis and HLA–B27.

Clinical presentation

At presentation 30% are localized to the rectum while 20% are widespread. The presenting symptoms include painless, bloody diarrhoea with mucus which may be associated with fevers and periods of near normality (remissions). Superficial ulceration in the colon is shown in Figure 4.2.

Diagnosis

Diagnosis is from the history and by endoscopic examination (colonoscopy) which reveals superficial ulceration and

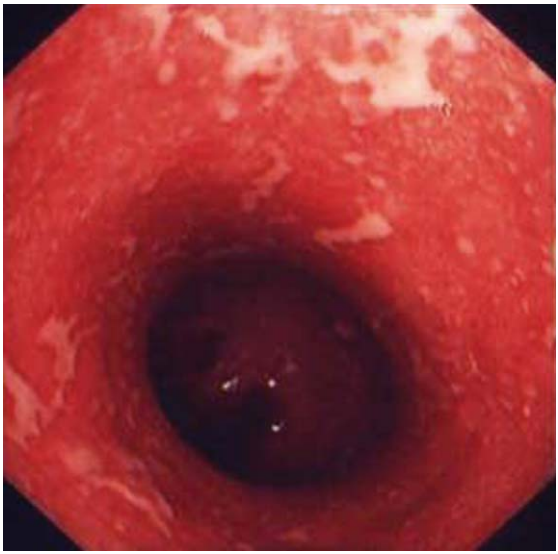


Fig. 4.2 An endoscopic view of the colon showing the superficial ulceration in ulcerative colitis.

contact bleeding. The differential diagnosis includes carcinoma of the colon, food poisoning and pseudomembranous colitis.

Treatment

Medical management includes a high-protein, high-fibre diet and immunomodulation with corticosteroids (acutely), azathioprine (long term), sulphasalazine and mesalazine. Surgical intervention may be required, particularly in the case of a toxic megacolon.

■ DENTAL RELEVANCE OF ULCERATIVE COLITIS

Oral ulceration may occur.

Immunosuppression increases the infection risk.

Carcinoma of the colon

Carcinoma of the colon is the third commonest cancer in the UK with 27 633 cases in 2002.

Predisposing factors

These include neoplastic polyps, long-standing ulcerative colitis (and possibly Crohn's), a positive family history, familial polyposis coli and previous cancer.

Site

Distribution: 45% rectum, 25% sigmoid, 15% caecum and ascending, 10% transverse and 5% descending.

Clinical presentation

Often detected during investigation of unexplained anaemia. In 60% there is a mass on rectal examination. Presentation depends on site of the tumour in the colon:

- *left*: bleeding PR, COBH and tenesmus
- *right*: anaemia, weight loss and abdominal pain
- *both*: obstruction, perforation, haemorrhage or fistula.

Spread is via lymphatics and initially local with later haematogenous spread (liver 75%, lung and bone) and sometimes transcoelomic.

Diagnosis

This relies on colonoscopy and biopsy. CT scan to determine size and liver ultrasound to detect metastasis.

Treatment

Usually by primary surgery with adjuvant chemotherapy.

■ DENTAL RELEVANCE OF CARCINOMA OF THE COLON

Oral manifestations of anaemia may occur.

Other gastrointestinal diseases

Diverticular disease

A diverticulum is a herniation of the bowel mucosa through the bowel wall, which occurs at a weak point where it is pierced by blood vessels. Inflammation of a diverticulum results in diverticulitis which most commonly presents with abdominal pain. Treatment is with a high-fibre diet and may involve surgery if recurrent.

Irritable bowel syndrome (IBS)

IBS is the commonest diagnosis made at gastrointestinal clinics and is characterised by intermittent diarrhoea, abdominal pain and bloating relieved by bowel action. Psychological factors account for most cases while some may relate to specific food intolerances. Women are more commonly affected than men.

Treatment

This involves reassurance and explanation. A high-fibre diet and avoidance of dietary triggers may help.

■ DENTAL RELEVANCE OF IRRITABLE BOWEL SYNDROME

May be associated with psychogenic oral symptoms such as chronic (atypical) facial pain, burning mouth syndrome and temporomandibular joint dysfunction

Haematemesis

This is the vomiting of blood, most commonly from the upper gastrointestinal tract. This must be distinguished from haemoptysis (coughing up blood). Vomited blood is usually partially digested and dark, resembling coffee grounds, and it may be mixed with food. If severe this can be life threatening, e.g. bleeding oesophageal varices. Causes of haematemesis are listed in Table 4.2.

Table 4.2
Causes of haematemesis (coughing up blood).

Cause	
Congenital	Haemophilia, Ehlers–Danlos syndrome, Peutz–Jeghers syndrome
Infective	*Helicobacter pylori*-induced ulceration
Inflammatory	Peptic ulceration, gastritis, oesophagitis
Traumatic	Surgery, swallowed blood from epistaxis
	Mallory–Weiss tear in oesophagus due to excessive vomiting
Venous engorgement	Oesophageal varices
Vascular malformation	Haemangioma
Neoplasia	Oesophageal SCC, gastric CA
Fistula	Aorto-oesophageal fistula
Drug induced	Warfarin, NSAID induced gastric erosion

5 NEUROLOGICAL DISORDERS

Georgina Burke

Neurological disorders may present as dental problems. A cranial nerve examination will help to distinguish the two.

Cranial nerve examination

Examine with the patient seated and facing you.

Smell (cranial nerve I)

The sense of smell is rarely formally tested; however, anosmia may occur after a head injury and so should be documented.

Vision (cranial nerve II)

There are three aspects to optic nerve function:

1. Visual acuity, which is usually assessed using a Snellen chart (Fig. 5.1).
2. Visual field testing to confrontation is a simple screen for homonymous hemianopia (an identical visual field defect due to cerebral hemisphere disease) and for sensory inattention (due to parietal lobe lesions). Sit opposite the patient and ask them to fixate on your nose. Get the patient to count fingers at the edge of each field in each eye in all four quadrants.
3. Fundoscopy takes practice; perform it only if competent with an ophthalmoscope.

Eye movements and ptosis (cranial nerves III, IV, VI)

Look for ptosis (eyelid droop) and then ask the patient to keep the head still and follow your finger. Check both lateral and vertical movements (Fig. 5.2).

The trochlear (IV) nerve supplies the superior oblique (turns the eye down and in). The abducens (VI) nerve supplies lateral rectus (turns the eye out). The oculomotor (III)

Fig. 5.1 A Snellen chart viewed at a specified distance can be used to assess visual acuity; the eyes are tested one at a time.

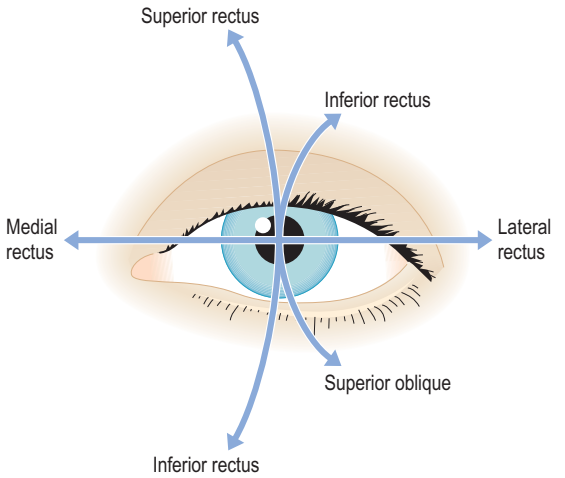


Fig. 5.2 Extra-ocular muscles responsible for eye movement.

■ CAUSES OF UNILATERAL PTOSIS

Unilateral ptosis occurs in:

- Horner's syndrome: with a constricted pupil
- III nerve lesion: with a dilated pupil and loss of adductive (inward) and vertical eye movements.

nerve supplies the other eye muscles, the eyelid muscles and the efferent supply to the pupils. A lesion in any one or more of these cranial nerves will result in diplopia (double vision). Causes of unilateral ptosis are given in the box above.

Pupils should react (constrict) to both light and accommodation (near vision).

Facial sensation and masticatory muscles (cranial nerve V)

Facial sensation in the territories of the trigeminal nerve (frontal V_1, maxillary V_2 and mandibular V_3) can be tested and mapped out using a pin or cotton wool.

The masseter and temporalis muscles can be palpated while the patient clenches their teeth to assess the motor division of the trigeminal nerve. The corneal reflex and jaw jerk also assess V nerve function.

Facial movements (cranial nerve VII)

Facial movements can be tested by asking the patient to 'raise their eyebrows' and to 'smile'. The anatomy of the facial nerve is explained in more detail in the section on Bell's palsy (p. 75). The effects of motor neurone lesions are listed below.

■ VII MOTOR NEURONE LESIONS

VII lower motor neurone lesions affect movement of both forehead and mouth whereas an upper motor neurone lesion affects just the mouth.

Hearing (cranial nerve VIII)

Hearing can be tested by whispering a number in one ear while masking the other. Unilateral or bilateral deafness should be examined further by Weber's and Rinne's tests to determine whether it is due to conductive problems (e.g. middle ear disease) or due to a lesion in the vestibulocochlear nerve.

Speech, swallow and palatal movement (cranial nerves IX, X)

A lesion in either the glossopharyngeal (IX) or vagus (X) nerve may result in dysarthria. Palatal movement can be visualised to test the vagus nerve which is more comfortable than the gag reflex.

Shoulder shrug (cranial nerve XI)

The spinal accessory (XI) nerve supplies the sternocleidomastoids and part of the trapezius. It is rarely tested routinely.

Tongue movement (cranial nerve XII)

The tongue should be observed at rest for wasting or fasciculations. Effects of motor neurone lesions are shown in the box at the top of p. 75.

■ XII MOTOR NEURONE LESIONS

Hypoglossal (XII) lower motor neurone lesions cause the tongue to be wasted and to deviate to the side of the lesion. In bilateral upper motor neurone lesions the tongue becomes spastic and its movements slow and limited. Alternating tongue movements will be slowed and irregular with a cerebellar lesion.

Bulbar palsy and pseudobulbar palsy are lower motor neurone (LMN) and upper motor neurone (UMN) disorders respectively resulting from paralysis of the lower cranial nerves. This causes slurred speech, difficulty swallowing, choking and a hoarse voice.

LMN bulbar palsy may be a feature of motor neurone disease, myasthenia gravis or a tumour in the medulla. Here the tongue is wasted.

UMN bulbar palsy is due to bilateral lesions (stroke, multiple sclerosis, motor neurone disease). Here the tongue is not wasted but movements are slow and stiff.

Bell's palsy

Bell's palsy is the most common cause of facial nerve paralysis.

Epidemiology

Annual incidence is about 20 per 100 000. The risk of recurrence is 10%. It is more common in pregnant women, diabetics and the elderly.

Pathogenesis

This is not fully understood but the condition is probably caused by latent Herpes viruses (Herpes simplex virus type 1 and Herpes zoster virus), which are reactivated from cranial nerve ganglia. Inflammation of the nerve results in nerve damage and a lower motor neurone (LMN) weakness that involves the whole musculature of one side of the face. By contrast, upper motor neurone (UMN) weakness spares the forehead muscles and is usually due to a stroke or tumour of the cerebral hemisphere. Other causes of LMN facial palsy are given in box at top of next page.

■ OTHER CAUSES OF LOWER MOTOR NEURONE FACIAL PALSY

- Tumours of the eighth nerve or skull base
- Parotid tumour, inflammation, injury
- Sarcoidosis (often bilateral weakness)
- Middle ear or mastoid infections
- Pontine lesions (demyelination or infarction)
- The Ramsay Hunt syndrome due to herpes zoster infection of the geniculate ganglion produces severe facial palsy with a painful vesicular eruption on the palate and external auditory meatus; aciclovir should be given.
- Melkersson's syndrome: association of recurrent facial nerve palsy, inflammatory swelling of the face and a geographical tongue.
- Traumatic injury (parotid surgery and skull base fractures).

Clinical features

Ear pain may precede the attack then facial weakness develops over 1 to 5 days and is often complete. The face feels stiff and the patient has difficulty closing the eyelids, and difficulty with eating and smiling. Altered taste can also occur due to involvement of the chorda tympani nerve. Hyperacusis (distortion of sound in the ipsilateral ear) indicates involvement of the nerve supplying the stapedius muscle. A branch also innervates the lacrimal glands and decreased production of tears on the affected side may result.

After recovery of a proximal lesion there may be synkinesia (a blink causes ipsilateral movement of the lips) or crocodile tears (lacrimation accompanies salivation during eating) due to aberrant innervation. Right-sided Bell's palsy is shown in Figure 5.3.

Treatment

Eighty percent fully recover in 1–2 months, but a small number, particularly diabetics and the elderly, have permanent facial weakness. The risks of this may be reduced by a short course of high-dose prednisolone and aciclovir if started within the first few days of symptom onset. To prevent drying and other corneal injury, lubricating eye

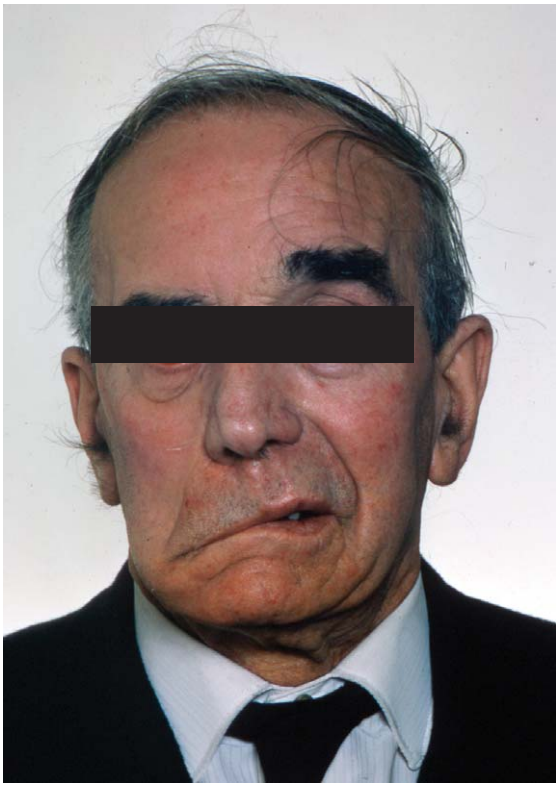


Fig. 5.3 Right-sided Bell's palsy.

drops may be necessary and the eyelids should be taped closed during sleep.

■ DENTAL RELEVANCE OF BELL'S PALSY

A parotid tumour may present as facial palsy.

Occasionally dental treatment may cause facial nerve palsy. Accidental injection into the parotid gland during a misdirected mandibular block may anaesthetize the facial nerve. The condition will only last for the duration of the anaesthetic, but the eye must be protected with an eye patch.

Anatomy

The facial nerve is predominantly motor and the nucleus is in the pons. It leaves the brainstem to pass through the cerebello-pontine angle into the internal auditory canal (with the VIII cranial nerve). It lies close to the inner and middle ear in the temporal bone. It leaves the skull via the stylomastoid foramen to supply the muscles of facial expression and platysma.

Trigeminal neuralgia

Trigeminal neuralgia is a sudden, severe, brief, stabbing, recurrent pain in the distribution of one of the divisions of the trigeminal nerve (see Fig. 5.4).

Epidemiology

Usually occurs in the elderly; in younger people it can be secondary to multiple sclerosis.

Pathophysiology

This remains unknown but it is thought to be related to compression of the trigeminal nerve by blood vessels. Differential diagnosis is shown in box at top of next page.

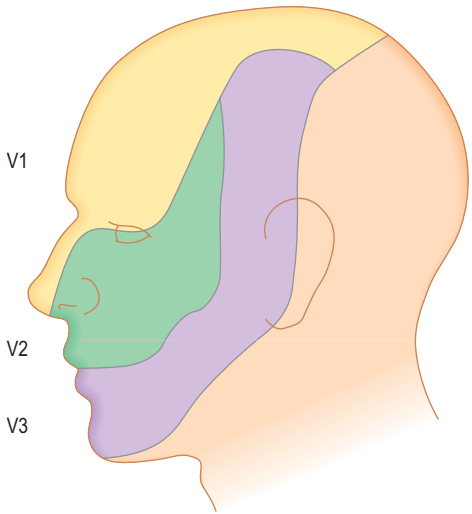


Fig. 5.4 Divisions of the trigeminal nerve.

■ DIFFERENTIAL DIAGNOSIS OF TRIGEMINAL NEURALGIA

- Dental: exposed dentine, fractured tooth, periodontitis, osteomyelitis
- Trigeminal neuropathy (e.g. compression of trigeminal roots from tumours or aberrant vessels)
- Glossopharyngeal neuralgia
- Postherpetic neuralgia
- Cluster headache
- Cranial arteritis
- Temporomandibular joint disorders
- Atypical facial pain.

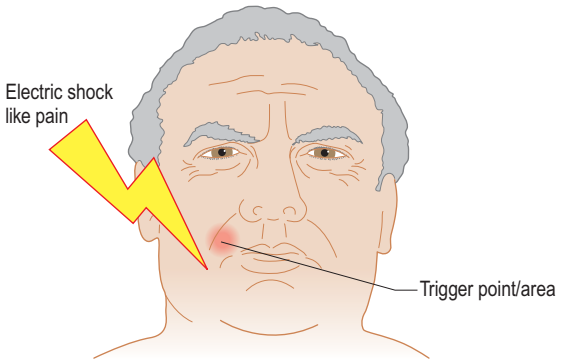


Fig. 5.5 Clinical features of trigeminal neuralgia. Note the electric-shock-like nature of the pain and the orofacial trigger point.

Clinical features

Spasms of pain occur in the face, usually radiating from the corner of the mouth or from the gingivae towards the cheek and ear. It is often triggered by touch, chewing, shaving or even cold wind. The pain is severe and occurs hundreds of times a day. Facial sensation and the other cranial nerves are normal. Clinical features are illustrated in Figure 5.5.

■ DENTAL RELEVANCE OF TRIGEMINAL NEURALGIA

Trigeminal neuralgia may occur after dental treatment.

Many patients attribute their pain to dental causes and will initially seek dental treatment.

The pain can be confused with dental pain. A marcaine block can be very successful at producing immediate and even long-term relief from this painful condition.

Dental care may be difficult not only due to the pain experienced by the patient but also because of depression related to chronic pain and diminished salivation due to the use of tricyclic antidepressants.

Trigeminal neuralgia is rare in young people.

Treatment

Carbamazepine is usually effective in controlling the pain, although dosages associated with drowsiness are often needed. Alternatives include phenytoin, lamotrigine, valproate and gabapentin. If the condition fails to respond to medical treatment, various surgical interventions are available, including cryotherapy, microvascular decompression, percutaneous balloon compression, radiofrequency thermocoagulation, and glycerol injection.

Stroke

A stroke is a sudden neurological disturbance due to blockage or rupture of a brain blood vessel.

Epidemiology

The inciden ce of stroke is declining in Western countries yet it remains a huge cause of long-term disability.

Classification

There are three main types of stroke:

1. Ischaemic stroke (85% of all strokes) due to blockage of a cerebral artery by an embolus or by thrombosis. If the neurological deficit lasts for less than 24 hours it is called a transient ischaemic attack (TIA).
2. Haemorrhagic stroke due to intracerebral bleed.

Table 5.1
Types of ischaemic stroke.

Artery occluded	*Symptoms*
Middle cerebral	Contralateral hemiparesis Aphasia (if left hemisphere)
Posterior cerebral	Contralateral homonymous hemianopia
Basilar	Diplopia, ataxia, facial weakness and altered sensation
Posterior inferior cerebellar	Vertigo and ipsilateral cerebellar ataxia

3. Subarachnoid haemorrhage due to rupture of a blood vessel (often an aneurysm or arteriovenous malformation) into the cerebrospinal fluid within the subarachnoid space.

Pathogenesis of ischaemic stroke

Most ischaemic stroke is due to emboli arising from an internal carotid artery stenosed (narrowed) by atheroma. Occasionally emboli come from the heart affected by mitral valve disease, right to left shunts, bacterial endocarditis, or a mural thrombosis following myocardial infarction or atrial fibrillation. Table 5.1 lists the types of ischaemic stroke.

■ RISK FACTORS FOR STROKE

- Age > 60 years
- Diabetes mellitus
- Cardiac disease
- Smoking
- Peripheral vascular disease
- Oral contraceptive pill and pregnancy
- Crack and cocaine
- Hypertension
- Polycythaemia
- Hyperlipidaemia.

Diagnosis

Diagnosis is by CT (see Fig. 5.6) or MRI. Carotid stenosis is imaged by Doppler ultrasound.

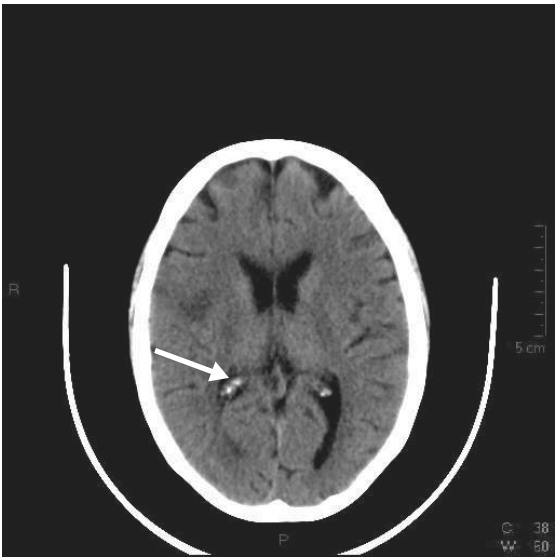


Fig. 5.6 An acute stroke can often be visualised by CT imaging.

Treatment of ischaemic stroke

This involves reduction of risk factors and rehabilitation. Aspirin 150 mg daily should be given and surgical endarterectomy considered for symptomatic carotid artery stenosis > 70%. The risk factors for stroke are shown in box previous page.

Haemorrhagic stroke

This is much less common than ischaemic stroke and is immediately visible on CT scan. It usually occurs in hypertensives or those with coagulation disorders.

Subarachnoid haemorrhage

This presents with a severe headache 'like being hit on the back of the head with a cricket bat'. It may be provoked by exertion, sexual intercourse or while straining. About 1/3 become unconscious and have a high risk of death or disability.

■ DENTAL RELEVANCE OF STROKE

Patients will often be taking anti-platelet medication or be anticoagulated.

Oral hygiene may be compromised due to weakness of the facial area or paralysis of extremities.

Swallowing may be compromised; adjust head position and ensure thorough, constant evacuation during dental procedures.

Communication may be difficult as speech and/or understanding may be affected.

Mobility may be affected.

Epilepsy

Epilepsy is a central nervous system disorder in which seizures recur, usually spontaneously.

Epidemiology

Epilepsy is one of the most common neurological disorders; about 1% of the population has epilepsy and about 3% of the population will experience a seizure during their lifetime. It is more common at either extreme of age.

Classification

This involves differentiating between attacks that start all over the brain (generalized) and those that start in a focal area (see box on p. 85). A focal seizure can remain localized or may spread to involve the whole brain (secondary generalized).

Pathogenesis

This is not fully understood but patients with primary generalized epilepsy are more likely to have a family history of the disorder. Focal epileptic attacks arise from brain abnormalities such as tumours, infections, infarction, after head injury, or from hippocampal sclerosis associated with frequent childhood febrile convulsions.

Isolated non-recurrent seizures are commonly caused by metabolic disturbances (hypoglycaemia, hyponatraemia, uraemia, and liver failure), hypotension, alcohol and drugs.

■ CLASSIFICATION OF SEIZURES

Generalized seizures
- Tonic-clonic (*grand mal*)
- Absence (*petit mal*) – common in children.
- Myoclonic: shock-like jerks of the limb

Focal seizures
- Simple – consciousness not impaired
- Complex – consciousness impaired.

Diagnosis

An eyewitness description of a typical convulsion is diagnostic of epilepsy. Convulsions can be tonic (generalized stiffness), or clonic (repetitive shaking of the limbs); most seizures are tonic then clonic. Other features that point strongly to a diagnosis of epilepsy are:

1. Postictal (after fitting) confusion lasting 5–20 minutes
2. Incontinence of urine
3. Biting the tongue or cheek
4. Inability to remember the onset
5. Stereotyped auras (hallucinations).

Treatment

Antiepileptic drugs (AEDs) are indicated for recurrent seizures. The choice of drug depends on the type of epilepsy and the side-effect profile of the drug.

- Cosmetic side-effects include weight gain, acne, hair loss, and gingival hypertrophy.
- There are many drug interactions, e.g. phenytoin and carbamazepine are enzyme inducers.
- Most AEDs cause delayed healing and increased risk of microbial infections.
- Excessive bleeding can occur with carbamazepine and sodium valproate.

Phenytoin-induced gingival hypertrophy is shown in Figure 5.7.

Most seizures will terminate on their own, but prolonged seizures (>5–10 minutes) should be terminated using intravenous or rectal diazepam. An alternative, Epistatus®, has recently been developed that is a buccal preparation of midazolam (10 mg in 1 mL of liquid).

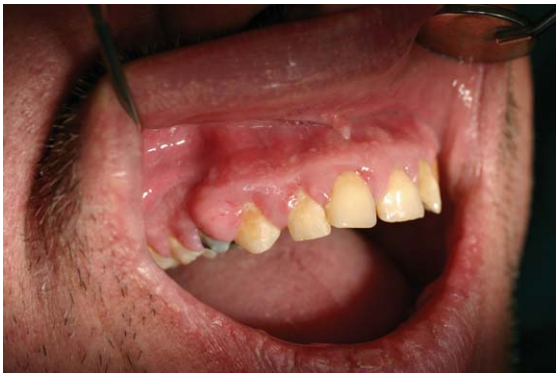


Fig. 5.7 Phenytoin-induced gingival hypertrophy.

■ DENTAL RELEVANCE OF EPILEPSY

Patients who have seizures need referral to specialist services and must stop driving.

Stress may precipitate seizures.

Drug interactions are common with antiepileptic medications; always check in the BNF.

Most antiepileptic drugs cause delayed healing and increased risk of infection, and some cause excessive bleeding. Oral manifestations from the use of AEDs include:

- gingival hypertrophy (phenytoin)
- xerostomia (carbamazepine).

In the event of a seizure:

- protect the airway
- protect the patient from the surrounding environment but do not restrain.

Most seizures will stop on their own. Once the seizure has stopped the patient is likely to be drowsy so do not continue their dental treatment but briefly examine for dental trauma. They will need an escort home.

Dementia

Dementia is characterised by gradual deterioration of intellect, memory and cognitive function in the absence of a disturbance of consciousness.

Epidemiology

Dementia is mainly a disease of the elderly and affects 5% of those >65 years of age.

Aetiology

This is usually multifactorial. The most common cause of dementia is Alzheimer's disease.

Pathogenesis

There are many causes of dementia (see box below). Alzheimer's disease is associated with the formation of neurofibrillary tangles and amyloid plaques in the brain.

Clinical features

These depend on the area of cortex affected but may result in impairment of:

- intellect: e.g. impaired reasoning and calculation
- language: e.g. difficulty reading, writing and disordered speech

■ CAUSES OF DEMENTIA

- Degenerative disease (e.g. Alzheimer's disease)
- Genetic (e.g. Huntington's chorea)
- Vascular (e.g. multi-infarct dementia)
- Metabolic (e.g. Wilson's disease)
- Toxic (e.g. alcoholic dementia)
- Deficiency (e.g. Wernicke–Korsakoff syndrome in thiamine deficiency)
- Mass lesion (e.g. cerebral tumour)
- Infection (e.g. CJD, AIDS)
- Inflammation (e.g. SLE)
- Trauma
- Hydrocephalus.

- social function: e.g. loss of personality, inability to work, withdrawn
- visuospatial function
- memory and concentration: e.g. inattentive, difficulty retaining new information.

Dementia is NOT associated with impaired consciousness.

Treatment

Treatment depends on the cause of dementia but in all cases social support for patients and their families is essential.

■ DENTAL RELEVANCE OF DEMENTIA

Dental treatment is challenging in patients with dementia as they often cannot understand the environment around them or what is being done to them.

Parkinson's disease

Parkinson's disease is an idiopathic disorder characterised by:

- *tremor (pill-rolling)*
- *rigidity (cog-wheel)*
- *akinesia (slowness of movement)*
- *postural abnormalities.*

Epidemiology

The overall incidence is 1–2/1000, and the peak age of onset is between 55 and 70. There is a slight male preponderance.

Pathology

Pathology demonstrates degeneration of the nigrostriatal pathway with loss of dopaminergic input into the striatum. The substantia nigra loses pigment and Lewy bodies can be found. Clinical features are given in box below.

Treatment

This involves replacement therapy for the dopamine deficiency. The most effective treatment is l-dopa which is

■ CLINICAL FEATURES OF PARKINSON'S DISEASE

- Rest tremor, initially just one limb
- Progressive micrographia (small writing)
- Loss of facial expression
- Slowness in initiating movements
- Delayed swallowing
- Monotonous weak voice
- Loss of arm-swing while walking
- Hunched posture and loss of postural adjustment mechanisms (may fall)
- Depression and dementia.

converted in the brain by dopa-decarboxylase to dopamine. In the rest of the body, conversion of l-dopa to dopamine gives rise to postural hypotension, nausea and vomiting. To reduce these side-effects, a dopa-decarboxylase inhibitor, which cannot cross the blood–brain barrier, is combined with the l-dopa (e.g. Sinemet or Madopar). Long-term side-effects include confusion, hallucinations, dyskinesias and fluctuations in the effectiveness of the drug as the disease advances. Alternatives to l-dopa include:

- dopamine agonists: often used initially so that l-dopa can be 'saved' for later
- amantadine: promotes the synthesis and release of dopamine
- anticholinergics: may be better for tremor; side-effects include dry mouth, constipation, urinary retention and confusion.

■ DENTAL RELEVANCE OF PARKINSON'S DISEASE

Do not interrupt medication as patients are sensitive to even the smallest changes in their drug regimen.

Avoid prescribing dopamine antagonists (e.g. metoclopramide) as these can make parkinsonian symptoms dramatically worse.

In advanced PD, patients may have difficulty swallowing and are at risk of aspiration.

Oral hygiene is often impaired with the consequent increase in dental decay.

Multiple sclerosis

Multiple sclerosis (MS) causes attacks of neurological disturbance resulting from immune-mediated demyelination of the central nervous system.

Epidemiology

MS is a major cause of disability and has a prevalence of 1:1000 in the UK. It is more common in temperate regions than in the tropics.

Pathogenesis

The cause of MS is unknown but it is generally believed to be immune-mediated, involving both environmental and genetic factors. There have been some claims but no scientific evidence that mercury leaking from amalgam dental fillings can lead to the development of MS.

Clinical picture

A huge variety of neurological disturbance can occur but the commonest manifestations are as follows:

- optic neuritis resulting in loss of vision.
- spinal cord lesions (myelitis) causing limb weakness or numbness/tingling. Sphincter control can also be affected.
- brainstem lesions resulting in vertigo and/or unsteadiness (ataxia). Eye movement abnormalities (nystagmus, gaze palsies and internuclear ophthalmoplegia) are also common. The types of MS are shown in box below.

■ TYPES OF MULTIPLE SCLEROSIS

- Relapsing remitting MS (RRMS)

Patients experience an attack followed by complete or partial remission. Patients may have several attacks per year.

- Primary progressive MS

This is characterised by a gradual decline in function with no clear periods of remission.

- Secondary progressive MS

Many patients with RRMS will go on to develop the secondary progressive phase of the disease where disability accumulates and there are no periods of remission.

Diagnosis

Diagnosis of MS uses the McDonald criteria which rely on the objective evidence of dissemination of demyelinating plaques in both time and space. Clinical features and MRI are integrated with other diagnostic tests (e.g. CSF oligoclonal bands, delayed visual evoked potentials).

■ DISEASE-MODIFYING DRUGS IN MULTIPLE SCLEROSIS

- Interferon-β: reduces the relapse rate by 1/3 but it is not certain whether it prevents disability
- Azathioprine and mitoxantrone: reduce disability but have toxic side-effects.

Treatment

Acute disabling relapses are treated with intravenous or oral steroids. This speeds up recovery but probably doesn't improve the eventual extent of the recovery. Muscle spasms can be treated with baclofen, diazepam or botulinum toxin injections. Anticholinergics are useful for urinary symptoms but many eventually self-catheterise. Physiotherapy and occupational therapy are important aspects of maintaining function in patients with MS. Disease-modifying drugs are shown in box above.

■ DENTAL RELEVANCE OF MULTIPLE SCLEROSIS

Infections and stress can make MS symptoms worse and should be avoided.

The most frequent oro-facial symptoms include trigeminal neuralgia, trigeminal sensory neuropathy (paraesthesia) and facial palsy.

Avoid NSAIDs if the patient is taking corticosteroids due to the risk of peptic ulceration.

Patients may be taking long-term interferon-β; oral manifestations include: chelitis, gingivitis, stomatitis, xerostomia, and candidiasis.

Polypharmacy is extremely common in these patients; check for drug interactions.

Patients may be taking corticosteroids or other immunosuppressants which increases their risk of infection.

Head injury and conscious level

Head injury is a common problem and may lead to brain death, brain damage or epilepsy.

The *level of consciousness* is formally assessed using the Glasgow Coma Scale (GCS) (box below); the lower the score the higher the risk of intracranial complications.

Extradural haematoma

Blood accumulates between the skull and dura mater due to laceration of meningeal blood vessels. It is more likely if a skull fracture has occurred. (The temporal region is particularly vulnerable due to the intra-boney path of the mid-dle meningeal artery.) The ipsilateral pupil may be

■ THE GLASGOW COMA SCALE (GCS)

GCS is used to assess patients with head injury. It is scored out of 15; patients with a score of 8/15 or less require intubation as breathing may be compromised.

Best eye-opening response	
• spontaneous	4
• to speech	3
• to pain	2
• none	1
Best verbal response	
• orientated	5
• confused conversation	4
• short inappropriate words	3
• incomprehensible	2
• none	1
Best motor response	
• obeys verbal commands	6
• localises painful stimulus	5
• limb flexion in response to pain	4
• flexion abnormality (decorticate)	3
• abnormal extension (decerebrate)	2
• none	1
Total	/15

dilated due to a III nerve palsy as a false localising sign. Diagnosis is with a brain CT scan and treatment is surgical.

Subdural haematoma

Blood collects between the dural and arachnoid layers of the meninges. It may occur acutely after head injury or chronically (particularly in the elderly, alcoholics, and in those taking anticoagulants). MRI is more sensitive than a CT scan for detecting a subdural haematoma. Small subdurals can be managed conservatively but large ones will require surgical drainage.

Figure 5.8 shows CT scans of the two types of haematoma.

Basal skull fracture should be suspected if there is haemotypanum (blood behind the tympanic membrane), 'raccoon eyes', CSF otorrhoea or Battle's sign (subcutaneous haemorrhage behind the ear).

Cervical spine immobilisation should be attempted in patients who have sustained a head injury and have any of the following:

- GCS < 15
- neck pain or tenderness
- focal neurological deficit
- paraesthesia in the peripheries.

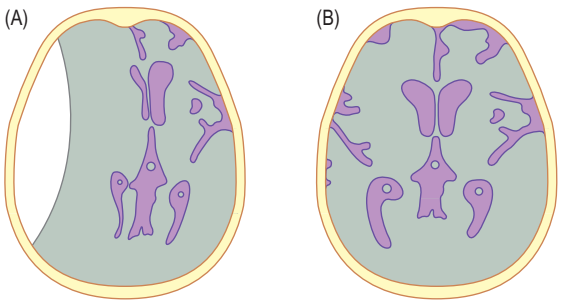


Fig. 5.8 CT scans in head injury. An extradural haematoma (A) has a biconvex shape with a well defined margin, whereas a subdural haematoma (B) is often spread more widely in the subdural space and has a more irregular inner margin.

Other causes of reduced consciousness/coma include:

- *metabolic*: too much or too little glucose, hypoxia/hypoperfusion, liver or kidney failure, hypothyroidism, hypothermia
- *toxic*: drugs (benzodiazepines, opiates, barbiturates, tricyclics) and alcohol
- *stroke or subarachnoid haemorrhage*
- *infections*: meningitis, encephalitis, malaria
- *epilepsy*: during or after a seizure.

■ DENTAL RELEVANCE OF HEAD INJURIES

Dental injuries commonly occur with head trauma.

Custom-made mouthguards can reduce the rate of concussion as well as dental and mandibular injuries during sport.

NICE guidelines for dental practitioners (criteria for referral to A&E after a head injury):

- GCS < 15 at any time
- any loss of consciousness
- any focal neurological deficit
- any suspicion of a skull fracture or penetrating head injury
- amnesia
- persistent headache
- vomiting (especially if >12 years of age)
- seizure
- previous cranial neurosurgery
- a high-energy head injury
- history of bleeding or clotting disorder
- current anticoagulation
- drug or alcohol intoxication
- age ≥ 65 years
- suspicion of non-accidental injury
- irritability or altered behaviour.

All of the above are associated with increased risk of intracranial complications.

For further advice see www.nice.org.uk

Headache

Headache is a common problem; most headaches are benign.

Headache of raised intracranial pressure

Classically this is a dull frontal headache, worse in the mornings and associated with nausea. It can be exacerbated by Valsalva manoeuvres (e.g. coughing). Papilloedema is a late sign.

Cranial arteritis

This headache, arising from arterial inflammation, rarely occurs in people under the age of 50 years. The main clue to the diagnosis is that the scalp is tender. The ESR is invariably raised and provides a good, rapid, screening test. Treatment is with a course of high-dose corticosteroids.

Migraine

This headache (Fig. 5.9) disorder usually starts in adolescence and there is often a family history of migraine. There are two types: migraine with aura (classical) and migraine without aura (common). In both, the headache is usually unilateral, throbbing and usually severe. It is associated with nausea and vomiting, photophobia and phonophobia. The attack usually lasts several hours. Migraine often responds to analgesics if taken soon after the start of the headache but many take prophylactic medication (e.g. propanolol or pizotifen). Stress and certain foods (e.g. cheese, chocolate, red wine) can precipitate migraine and so should be avoided.

Cluster headache

This is a specific headache disorder consisting of severe pain over one eye (Fig. 5.10) that lasts between 20 and 60 minutes and tends to re-occur at the same time each day for up to 6 weeks.

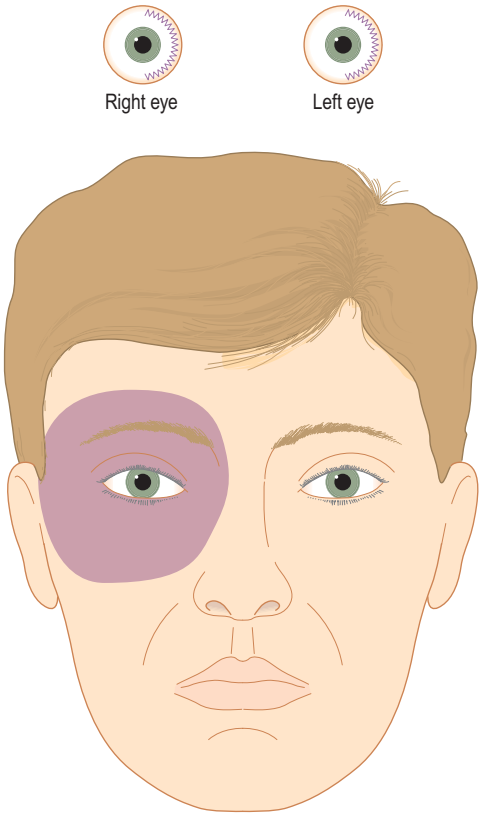


Fig. 5.9 Migraine is often preceded by a visual aura in the form of zigzag lines (fortification field). The headache is severe and unilateral.

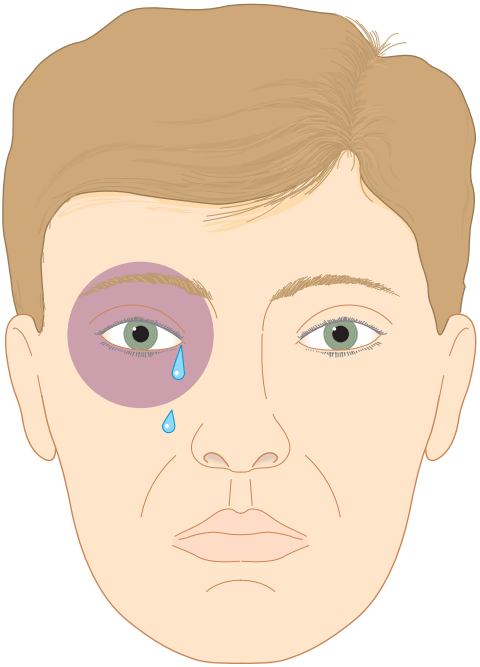


Fig. 5.10 Cluster headache is centred around one eye, which often waters. More common in young males.

Tension-type headache

Classically this non-specific headache consists of a band of pain around the head (Fig. 5.11). Paracetamol and NSAIDs are frequently used by patients, but are not very effective and often lead to chronic daily headache. Reassurance and low-dose amitriptyline may be effective in these patients.

Chronic daily headache

The major cause for a headache occurring almost daily is drug misuse. Minor analgesics and opiates are the common culprits. Treatment involves explanation to the

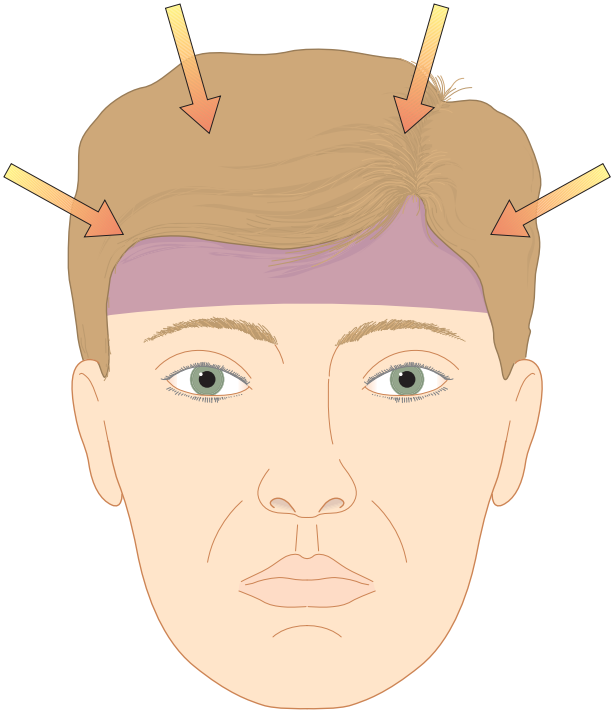


Fig. 5.11 Tension headache presents as a band of pain around the head.

patient and gradual withdrawal of the excessive drug usage.

■ DENTAL RELEVANCE OF HEADACHE

Headaches are common and are a cause for facial pain.

It is important to recognise and treat cranial arteritis, as arterial inflammation can lead to blindness.

6 ENDOCRINE DISEASE

Georgina Burke

Thyroid disease

The thyroid gland lies in the anterior midline of the neck below the thyroid cartilage. It is butterfly shaped, with two lobes and a central isthmus. Its main function is to regulate metabolic rate by production of thyroxine (T4), and triiodothyronine (T3). By production of calcitonin it has a role in calcium homeostasis. Over- or under-activity of the thyroid gland is the commonest of all endocrine problems. Figure 6.1 shows the hypothalamic–pituitary–thyroid feedback system.

Hypothyroidism

Under-activity of the thyroid gland may arise from disease of the thyroid or secondary to pituitary disease (reduced TSH drive). Causes of hypothyroidism are given in box on next page.

Clinical features of hypothyroidism

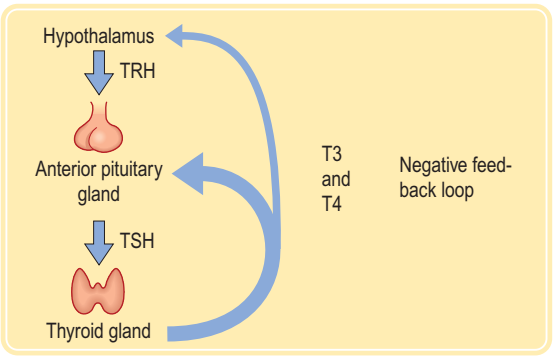
The clinical features are shown in Figure 6.2. Remember, everything slows up apart from menstruation!

Diagnosis

Diagnosis is made from thyroid function tests: free T4 level is low; a high TSH confirms primary hypothyroidism.

Treatment

Treatment is with life-long replacement therapy (thyroxine).



T3 and T4 feed back on the pituitary and perhaps hypothalamus to reduce thyroid releasing hormone (TRH) and thyroid stimulating hormone (TSH) levels.

Fig. 6.1 The hypothalamic–pituitary–thyroid feedback system.

Primary Causes of Hyperthyroidism

- Congenital
- Defects of hormone synthesis (e.g. iodine deficiency, drugs such as lithium or amiodarone)
- Autoimmune (atrophic or Hashimoto's)
- Infective
- Iatrogenic (post-surgery or irradiation)
- Infiltration (tumour).

Secondary

- Hypopituitarism.

■ DENTAL RELEVANCE OF HYPOTHYROIDISM

Children with hypothyroidism may have delayed dental development and an increased risk of decay and periodontal disease.

Adults with hypothyroidism may have an enlarged tongue, delayed tooth eruption, variable periodontal health, delayed wound healing and changed taste sensitivity.

Dental treatment should be avoided in patients with severe untreated hypothyroidism as a myxoedema coma can be precipitated by the use of CNS depressants (e.g. narcotics, sedation), surgical procedures and infections.

Patients with hypothyroidism may be sensitive to sedatives and opioid analgesics. However, they may also have a lower pain threshold.

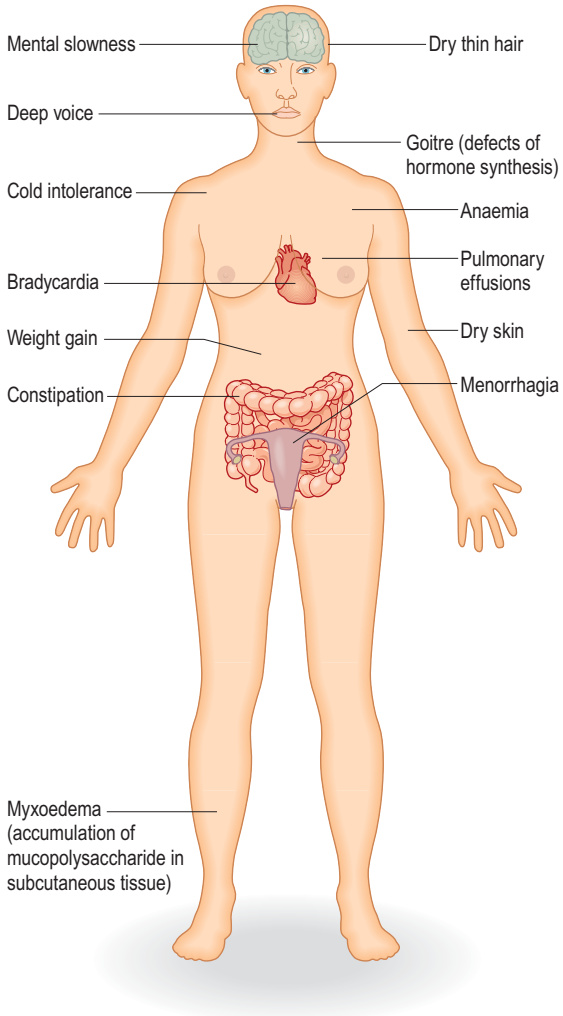


Fig. 6.2 Clinical features of hypothyroidism.

■ CAUSES OF HYPERTHYROIDISM

- Graves' disease
- Solitary toxic nodule/adenoma
- Toxic multinodular goitre
- Rarer causes include: acute thyroiditis (viral/autoimmune/post-irradiation) and drugs (e.g. amiodarone).

Hyperthyroidism

This is common, affecting 2–5% of all females with a sex ratio of 5:1. Nearly all cases are caused by intrinsic thyroid disease. Causes of hyperthyroidism are given in box above. Figure 6.3 lists the clinical features of hyperthyroidism, while Figure 6.4 shows proptosis in Graves' disease.

Diagnosis

Diagnosis is made from thyroid function tests: serum TSH is suppressed with a raised T3 or T4.

Treatment

There are three possibilities available for treatment:

1. Antithyroid drugs (e.g. carbimazole) inhibit thyroid hormone synthesis and are often given with beta-blockers as many of the manifestations of hyperthyroidism are mediated via the sympathetic nervous system.
2. Radioactive iodine (iodine-131) accumulates in the thyroid and destroys the gland by local radiation. Again, patients must be rendered euthyroid before treatment.
3. Surgery (subtotal thyroidectomy) should be performed only in those patients who have previously been rendered euthyroid. Surgery is particularly indicated for large goitres.

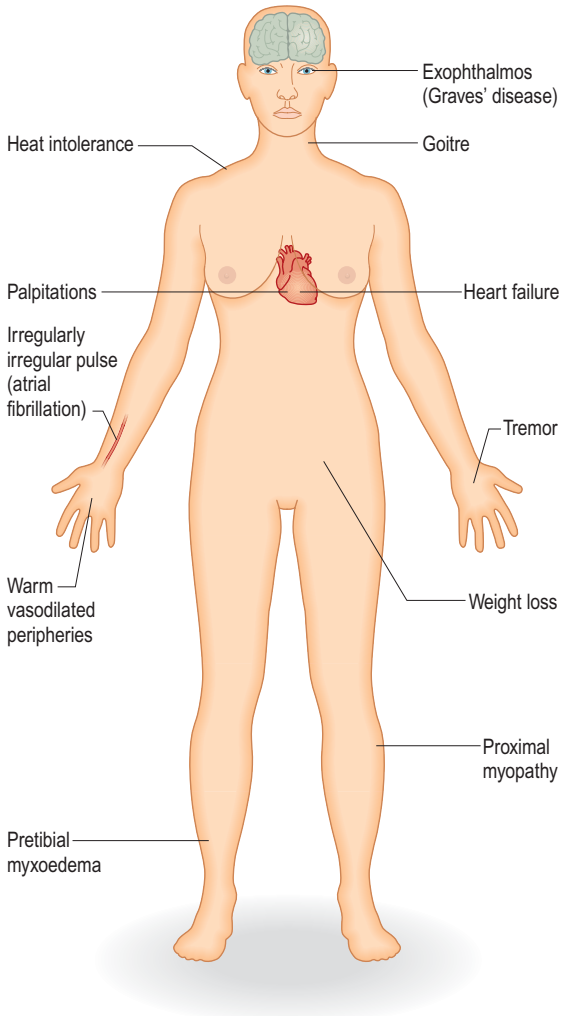


Fig. 6.3 Clinical features of hyperthyroidism.

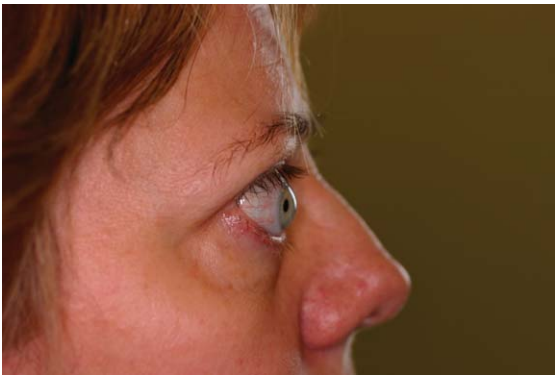


Fig. 6.4 Proptosis seen in Graves' disease.

■ DENTAL RELEVANCE OF HYPERTHYROIDISM

Hyperthyroidism may accelerate periodontal disease. Premature tooth eruption occurs and oral and facial bones may be weakened.

Dental treatment should be avoided in patients with severe untreated hyperthyroidism, as a thyrotoxic crisis can be precipitated by stress, surgical procedures and infections. This is a medical emergency and carries a mortality of 10%.

Patients with hyperthyroidism may be sensitive to adrenaline (epinephrine) (contained in local anaesthetics).

Antithyroid drugs may predispose patients to infections and poor wound healing.

Thyroid lumps

Disease of the thyroid often causes lumps or nodules to develop in the gland. Fortunately 95% of these are benign and only 10% are active 'hot', producing thyroid hormone. Approximately 8% of females and 4% of males will develop thyroid nodules. Once visible a thyroid swelling is called a goitre.

Clinical features

Most thyroid lumps are asymptomatic and detected by the patient, dentist or doctor during a routine examination. Rarely there may be pain, difficulty in swallowing, the feeling of a 'lump in the throat', stridor (difficulty inhaling), symptoms of hyperthyroidism or hoarseness of the voice. Differential diagnosis of thyroid nodules is given in box below.

The thyroid gland develops from the foramen caecum at the tongue base and descends to its final position in the neck during embryogenesis. Thus thyroid swellings may be detected at any point along this course of descent.

The classic feature of thyroid swellings is that they move on swallowing which should be checked for when examining any midline neck swelling.

Diffuse enlargement of the gland is most likely to be due to thyroiditis in the UK. If there are multiple nodules this is most likely to be a benign multinodular goitre. Single nodules are more sinister, but the majority of these will turn out to be benign in nature.

DIFFERENTIAL DIAGNOSIS OF THYROID NODULES

- Adenoma
- Cyst
- Carcinoma
- Multinodular goitre
- Hashimoto's thyroiditis
- Effect of previous operation or radioiodine therapy
- Parathyroid cyst or adenoma
- Thyroglossal cyst
- Aneurysm
- Laryngocele

Only 5% of nodules are malignant; this risk is increased by:

- past history of neck radiation
- family history of thyroid cancer
- associated lymphadenopathy
- high rate of growth

- male
- age <20 or >60
- pain
- fixation to underlying muscles
- hoarseness of the voice
- 'cold' nodule on scan.

Investigation

This is carried out by a combination of fine-needle aspiration cytology, ultrasound scan, thyroid function tests and autoantibody detection.

Treatment

Asymptomatic benign nodules may just require close observation. Toxic (thyroid hormone-producing) nodules can be removed surgically or treated with radioiodine. Non-toxic nodules which are symptomatic or suspected malignant should be surgically removed. Thyroid cysts can be aspirated. Thyroid surgery is usually carried out through a necklace incision (see Fig. 6.5).

Figure 6.5 shows a scar resulting from thyroid surgery.

The dental relevance of thyroid nodules is shown in the box.

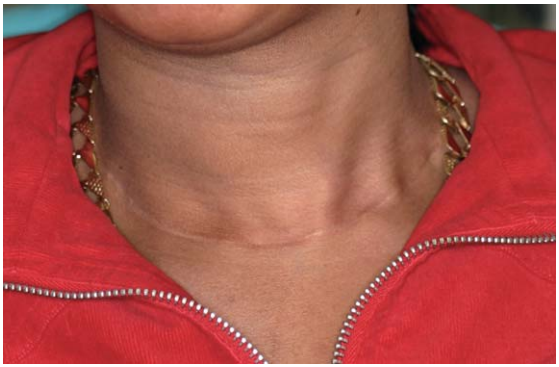


Fig. 6.5 A necklace scar as a result of thyroid surgery.

■ DENTAL RELEVANCE OF THYROID NODULES

Thyroid swelling may be picked up during routine examination of the neck.

Swelling at the base of the tongue may be due to accessory thyroid tissue left at the foramen caecum.

Parathyroid disease

There are normally four parathyroid glands, one located at each pole of the thyroid. They are central to calcium regulation.

Hypoparathyroidism

This condition is usually caused as a result of post-thyroid surgery or as idiopathic/autoimmune cases although there are rarer genetic causes (e.g. Di George syndrome). Clinical features are related to the hypocalcaemia which causes neuromuscular instability:

- Chvostek sign: circumoral twitching secondary to gentle tapping of the facial nerve
- Trousseau's sign: carpal spasm when blood pressure cuff is >20 mmHg higher than systolic blood pressure
- Seizures
- Laryngospasm and bronchospasm
- Circumoral paraesthesia.

■ DENTAL RELEVANCE OF HYPOPARATHYROIDISM

Hypoparathyroidism may be part of the polyglandular syndrome type 1 which is also associated with Addison's disease; oral candidiasis is often a difficult problem to treat.

Pseudo-hypoparathyroidism is due to resistance to parathyroid hormone and causes functional hypoparathyroidism. Magnesium levels are characteristically extremely low. It is usually due to Crohn's disease or renal tubular disease.

Hyperparathyroidism

This condition has an incidence of 1 per 1000 and is more common in females and in the fifth decade. Eighty percent of cases are due to a parathyroid adenoma; the majority of the rest arise from parathyroid hyperplasia.

Hyperparathyroidism is usually asymptomatic but a few patients have the classic 'bones, stones, groans and abdominal moans' symptoms related to hypercalcaemia. Causes of hypercalcaemia are listed in box below, and the clinical features of hyperparathyroidism are given in Figure 6.6.

■ CAUSES OF HYPERCALCAEMIA

- Primary hyperparathyroidism
- Carcinoma
- Abnormal vitamin D metabolism
- Immobilisation
- Other endocrine disorders
- Drugs: e.g. thiazide diuretics
- Renal disease.

■ DENTAL RELEVANCE OF HYPERPARATHYROIDISM

- May present with Brown tumours of the mandible or maxilla.
- Loss of lamina dura around the teeth is pathognomonic.
- Hyperparathyroidism may be associated with other autoimmune diseases.

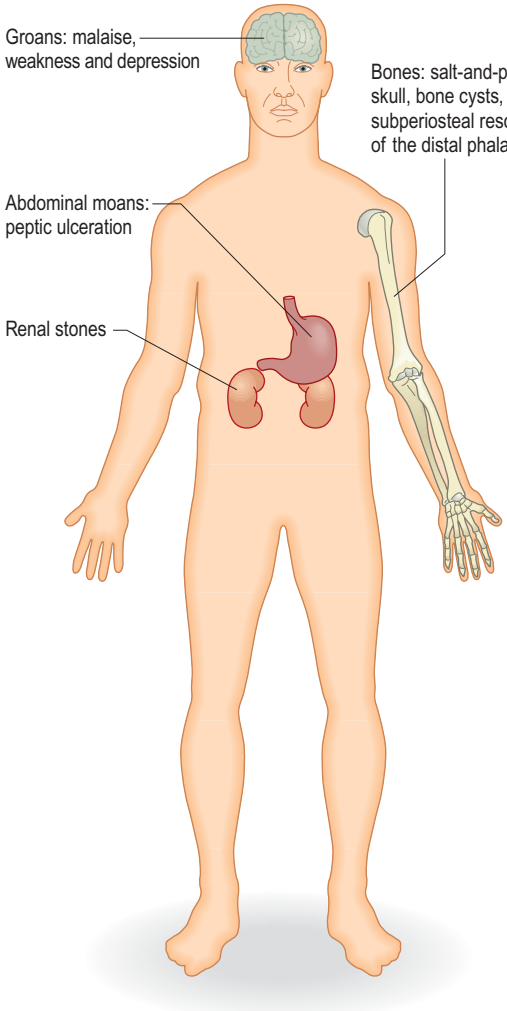


Fig. 6.6 Clinical features of hyperparathyroidism.

Pituitary disease

Pituitary tumours are the commonest cause of pituitary disease. As with most endocrine disease, problems may arise due to excess hormone secretion, by local effects of the tumour, or by inadequate hormone production by the remaining normal pituitary (hypopituitarism). See box below for size and frequency of pituitary tumours.

Cranial diabetes insipidus

This is a rare disease caused by lack of vasopressin, and results in polyuria, nocturia and compensatory increased thirst. Daily urine output may reach over 10 litres, leading to dehydration. Diabetes insipidus (DI) may also arise as a consequence of renal disease (nephrogenic DI); here the renal tubules are resistant to vasopressin.

Table 6.1
Hormones affected in panhypopituitarism.

Deficiency	*Level*	*Features*
Gonadotrophin (LH and FSH)	↓	Loss of libido, amenorrhoea, impotence
Prolactin	↑	Galactorrhoea and hypogonadism
Growth hormone	↓	Retarded growth in children
TSH	↓	Hypothyroidism
ACTH	↓	Adrenal failure

■ SIZE AND FREQUENCY OF PITUITARY TUMOURS

- Large: non-secreting (50%)
  prolactinomas (25%)
- Medium: acromegaly (12%)
- Small: Cushing's disease (5–10%)
  TSH-secreting (1%)

Acromegaly

This is uncommon (prevalence 30–40 per million) and is almost always due to a pituitary tumour secreting growth hormone. It can be diagnosed by the failure of GH to suppress to <2 mU/L in the glucose tolerance test. Clinical

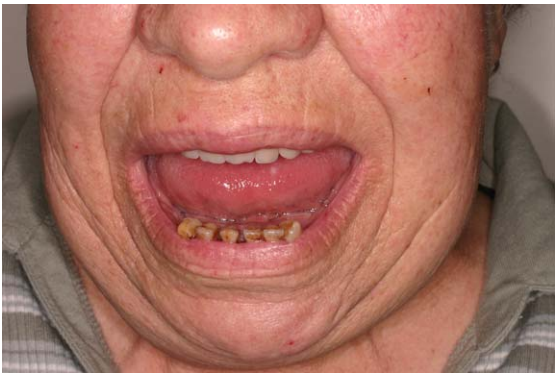


Fig. 6.7 Patient with acromegaly showing the coarse facial features, macroglossia and interdental separation typically seen in this condition.

features include hand and foot enlargement, coarse facial features, overbite of the lower jaw, splaying of the teeth, hypertension and diabetes. Features of acromegaly are shown in Figure 6.7.

Hypopituitarism

Deficiency of hypothalamic releasing hormones or of pituitary trophic hormones can either be selective or multiple. Panhypopituitarism refers to deficiency of all anterior pituitary hormones (see Table 6.1); it is most commonly caused by pituitary tumours, surgery or radiotherapy.

Clinical features depend on the extent of deficiency. Rather than prolactin deficiency, hyperprolactinaemia occurs due to the loss of the normal inhibitory control by dopamine.

Steroid and thyroid hormones are essential for life and must be given as oral replacement drugs. Other hormones are replaced for symptomatic control.

■ DENTAL RELEVANCE OF PITUITARY DISEASE

Growth in facial structures in acromegaly may affect dentures, bridges and orthodontic work, as well as restorations. Extractions may be difficult due to bony ankylosis. Patients are also not as medically robust as they appear.

Dental management may be complicated by multiple endocrine problems, hypertension, cardiac complications and arthritis.

Adrenal disease

The adrenal gland produces steroid hormones (from the cortex) and secretes catecholamines (from the medulla). Three classes of steroid hormones are produced:

- glucocorticoids (wide ranging effects on metabolism, fluid balance and the immune response)
- mineralocorticoids (affect sodium and potassium balance)
- androgens.

Addison's disease

Primary hypoadrenalism due to destruction of the adrenal cortex is rare. It usually occurs in females and is most often caused by autoimmune disease (80%). Clinical features are shown in Figure 6.8.

Diagnosis

Diagnosis is made by measuring plasma cortisol and/or an ACTH stimulation test can be performed.

Treatment

Treatment is with long-term glucocorticoid and mineralocorticoid replacement.

Cushing's syndrome

This condition arises from sustained over-production of glucocorticoid (cortisol). It occurs most often following therapeutic administration of exogenous steroids (e.g. prednisolone). For clinical features see Figure 6.9.

Diagnosis

Diagnosis is made by confirming hypercortisolism (loss of diurnal variation in cortisol or dexamethasone suppression test) and then localising the cause. For causes of this syndrome see box below.

■ CAUSES OF CUSHING'S SYNDROME

- Exogenous steroid intake
- Adrenal tumour
- Pituitary tumour (Cushing's disease)
- Ectopic production of ACTH (e.g. lung cancer).

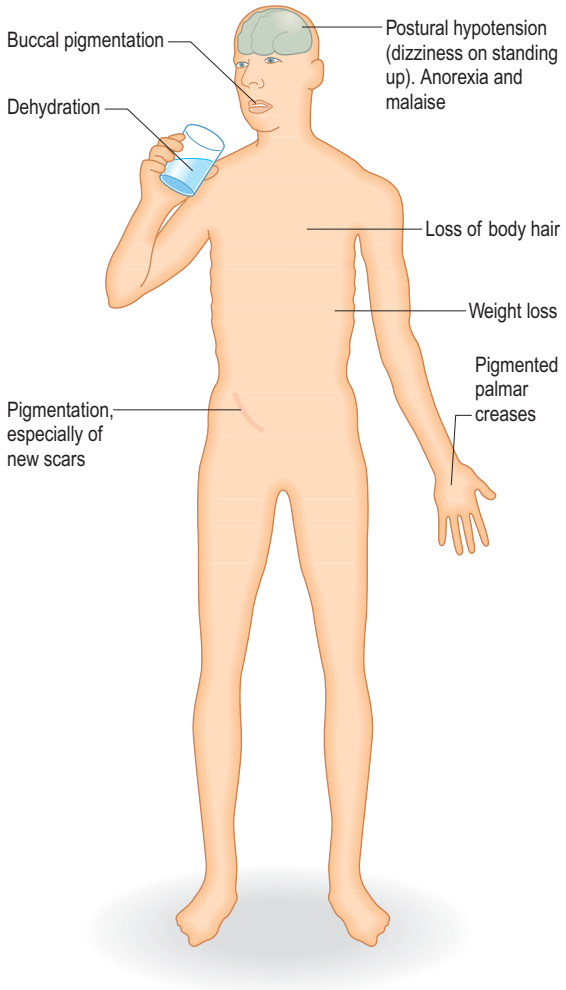


Fig. 6.8 Clinical features of Addison's disease.

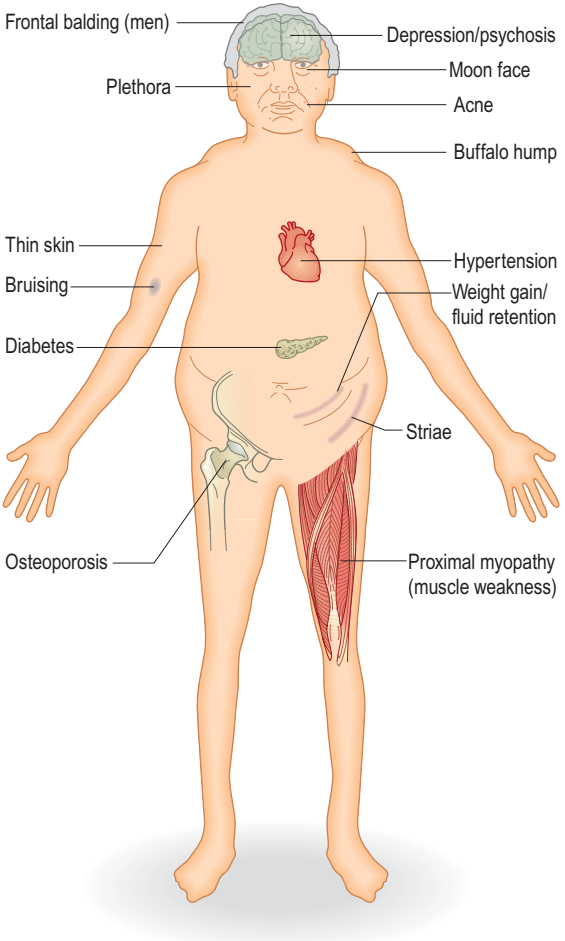


Fig. 6.9 Clinical features of Cushing's syndrome.

Steroid cover for dental procedures

It has been recommended in the past that a steroid booster (steroid cover) should be given prior to dental treatment in all patients taking long-term oral steroid medication to prevent a steroid crisis and collapse. There is little evidence to support this practice. It is no longer necessary to give steroid cover for dental treatment under local anaesthetic or sedation. It may still be necessary to give steroid cover for treatment carried out under general anaesthetic on recommendation of the anaesthetist.

■ DENTAL RELEVANCE OF ADRENAL DISEASE

Patients taking oral steroids are prone to infection so antibiotics should be considered for surgical procedures.

In Addison's disease pigmentation of the oral mucous membranes is a common finding.

Phaeochromocytomas

These are rare tumours of the adrenal medulla. Clinical features are due to hypersecretion of catecholamines and include hypertension (particularly episodic), headache, sweating, cardiomyopathy and weight loss.

- 90% are benign tumours
- 90% arise in the adrenal medulla
- 90% are unilateral.

Diabetes mellitus

Diabetes mellitus is a syndrome characterised by high blood glucose levels (hyperglycaemia) and deranged metabolism resulting from defects in insulin secretion and/or insulin action.

Epidemiology

Diabetes is a common problem and affects 1.8 million people in the UK. Although the condition can affect all ages, it is rare in infants and becomes more common with age. For classification of diabetes mellitus see box below.

■ CLASSIFICATION OF DIABETES MELLITUS

Type 1 diabetes (5–10%)
- Autoimmune destruction of the pancreatic β cells
- Commonly occurs in childhood and adolescence
- Patients are prone to other autoimmune disorders.

Type 2 diabetes (90–95%)
- Impaired insulin function
- Risk increases with age, obesity and lack of exercise
- Strong genetic predisposition.

Other causes of diabetes
- Gestational = onset during pregnancy
- Endocrinopathies = ↑ cortisol, glucagon, GH
- Drugs = steroids, thiazides
- Pancreatic diseases
- Genetic defects of β cell function or insulin action.

■ DIAGNOSIS OF DIABETES MELLITUS

- Random glucose ≥11.1 mmol with symptoms of diabetes
- Fasting glucose ≥7.0 mmol
- Oral glucose tolerance test 11.1 mmol at the 2-hour interval.

Aetiology

Insulin is synthesised in pancreatic β cells and is secreted in response to raised blood sugar. It promotes glucose uptake into cells and its storage in the liver as glucagon. Insulin also promotes uptake of fatty acids and amino acids and their conversion into triglycerides and protein stores.

When there is a lack of insulin, cells are unable to use blood glucose as an energy source so triglycerides are broken down into fatty acids. These are used as an alternative source of fuel leading to the production of ketones. For diagnosis see box above.

Clinical features

The main features of diabetes are a direct consequence of hyperglycaemia. The excess glucose in the blood is excreted via the kidneys (glycosuria) which causes an osmotic diuresis (polyuria) leading to dehydration and thirst (polydipsia) with subsequent weight loss.

Acute complications of diabetes

- Hypoglycaemia
- Autonomic symptoms (sweating, palpitations, shaking)
- Neurological symptoms (confusion, seizures, coma)
- Diabetic ketoacidosis (seen mainly in type 1 DM)
- Hyperosmolar non-ketotic coma (seen mainly in type 2 DM).

Chronic complications of diabetes

These are mainly due to vascular problems related to accelerated atheroma formation.

Macrovascular (*large vessel*):

- 2× risk of stroke
- 3.5× risk of MI
- 50× risk of foot amputation.

Microvascular (*small vessel*):

- Retina with loss of vision
- Kidney leading to renal failure
- Nerves causing glove and stocking numbness and autonomic nervous system problems.

Other:

- Increased infection risk due to dysfunctional polymorphs, high glucose and poor blood supply
- Susceptibility to periodontal disease, especially if poorly controlled diabetes
- Salivary gland dysfunction leading to dry mouth
- Burning mouth syndrome
- Increased prevalence of lichen planus
- More active dental caries
- Traumatic oral ulcers.

Oral candidiasis is often present (Fig. 6.10).

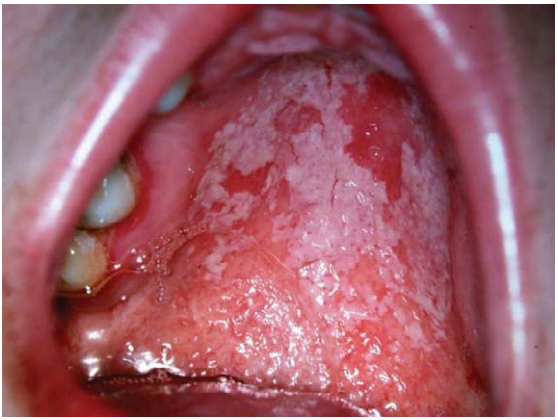


Fig. 6.10 Oral candidiasis.

■ COMPONENTS OF DIABETES MANAGEMENT

- Diet control
- Exercise
- Frequent self-monitoring of blood glucose levels
- Insulin and/or oral hypoglycaemic medications
- Diagnosis and aggressive treatment of complications.

Treatment

Treatment involves a multidisciplinary approach. The objective is to maintain blood glucose levels as close to normal as possible in order to delay the onset or progression of complications. Management: box top of next page.

Type 1 diabetes is treated by injections of insulin; formulations of different durations are available.

Type 2 diabetes is usually treated with oral hypoglycaemic drugs including sulphonylureas (stimulate insulin secretion and increase insulin sensitivity) and biguanides (increase insulin sensitivity only).

The glycated haemoglobin assay ($HbA1_c$) reflects mean glycaemia levels over the preceding 2–3 months and is used to assess glycaemic control.

■ DENTAL RELEVANCE OF DIABETES

Diabetes is a disease that affects the whole body, particularly the cardiovascular system. Oral manifestations include:

- rapidly progressing periodontal disease
- gingivitis
- xerostomia
- oral candidiasis
- poor wound healing
- burning mouth and/or tongue.

For invasive procedures, prophylactic antibiotics are often given as postoperative infections are common in diabetics.

Dental procedures involving local anaesthesia should avoid disruption to normal eating patterns as hypoglycaemia can rapidly develop in diabetics if they miss a meal but have taken their usual hypoglycaemic medication. For this reason they are usually scheduled first AM appointments or operations.

Avoid sedation as it may mask the symptoms of hypoglycaemia (see p. 281).

7 MUSCULOSKELETAL DISEASES

Chris Sproat

Rheumatoid arthritis (RA)

This is a common systemic disease that predominantly affects the joints, resulting in a severely disabling and symmetrical polyarthritis.

Epidemiology

RA affects about 1% of the UK population, with a female-to-male ratio of 3 to 1. In a quarter of patients the temporomandibular joint (TMJ) is affected but this is often asymptomatic. The cervical spine involvement occurs in 40% of patients.

Aetiology

RA is a chronic inflammatory disease with a genetic predisposition the cause of which remains unknown.

Clinical features

These can be divided into two groups:

1. Intra-articular (within the synovial joints) where there is inflammation of the synovium, destruction of the joint cartilage, soft tissues and adjacent bone. This leads to impaired movement, deformity (Fig. 7.1), pain and swelling. The hands and wrists are most commonly affected. The atlanto-axial joint of the cervical spine is often affected, which may make the neck vulnerable to damage during dental treatment.
2. Extra-articular (systemic features) which includes rheumatoid nodules, secondary Sjögren's syndrome, vasculitis, pulmonary fibrosis, pericarditis and carpal tunnel syndrome.

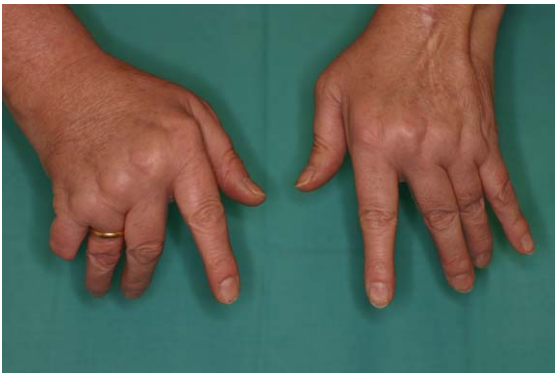


Fig. 7.1 Deformity as a result of RA affecting the hands. Note the ulnar deviation of the wrist and deformity of the fingers.

Diagnosis

This is made by the presence of at least four out of seven of the criteria of the American Rhematology Society:

1. Morning stiffness
2. Arthritis in > 3 joint areas
3. Arthritis of the hands
4. Symmetric arthritis
5. Presence of rheumatoid nodules
6. Positive rheumatoid factor
7. Radiographic changes in the hand.

Treatment

The treatment of RA involves a multidisciplinary team approach involving education, support, medical and surgical treatment.

Two groups of drugs are used:

1. Anti-inflammatory drugs, which give symptomatic relief, i.e. aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), and COX-2 inhibitors.
2. Disease-modifying anti-rheumatic drugs (DMARDs), which modify the fundamental pathological process, i.e. steroids, tumour necrosis factor inhibitors and immune suppressants.

Dental treatment patients with RA pose a number of problems for the dental surgeon which are best thought of in terms of their physical, social and psychological aspects.

■ DENTAL RELEVANCE OF RHEUMATOID ARTHRITIS

Physical

Arthritis-related

- Is the neck stable for treatment?
- Is the TMJ symptomatically involved?

Haematological

- The associated anaemia may lead to glossitis, burning mouth and angular chelitis
- Altered liver function may affect drug metabolism

Drugs

- NSAIDs are often prescribed at high doses
- Immune suppressants may increase the risk of infection
- Aphthous ulceration may occur

Social

Mobility

- Access to treatment may be restricted
- Domiciliary visits may be required
- Disabled facilities may be required
- ***The patient may not be able to hold a conventional toothbrush***

Psychological

- Pain, lack of mobility, dry eyes and mouth associated with Sjögren's syndrome may lead to depression

Other musculoskeletal conditions

Ankylosing spondylitis

This is a form of arthritis in which the sacroiliac joints and spine become ossified ('bamboo spine'). It usually manifests in the early twenties and rarely after the age of 35. Its origin is multifactorial with an increased risk in those people who are HLA-B27 positive. There is often back pain and progressive restriction of movement. In about 20% of cases complete rigidity of the spine and pelvis occurs, affecting the patient's ability to walk and move.

■ DENTAL RELEVANCE OF ANKYLOSING SPONDYLITIS

- Spinal deformity may make access for treatment difficult.
- Patients may be prescribed NSAIDs for symptomatic relief.

Polymyalgia rheumatica and giant cell arteritis

These conditions represent the opposite ends of the spectrum of the same disease, involving granulomatous inflammation. Polymyalgia rheumatica (PMR) commonly affects the musculature of the pelvic and shoulder girdles, causing stiffness and pain. In giant cell arteritis there is granulomatous inflammation within the arteries of the head and neck (Fig. 7.2), leading to headache and scalp tenderness, most commonly in the temporal region.

Both conditions are more common in the over-60 age group and there are often systemic features of tiredness, weight loss and fever. In both conditions the erythrocyte sedimentation rate (ESR) is often raised.

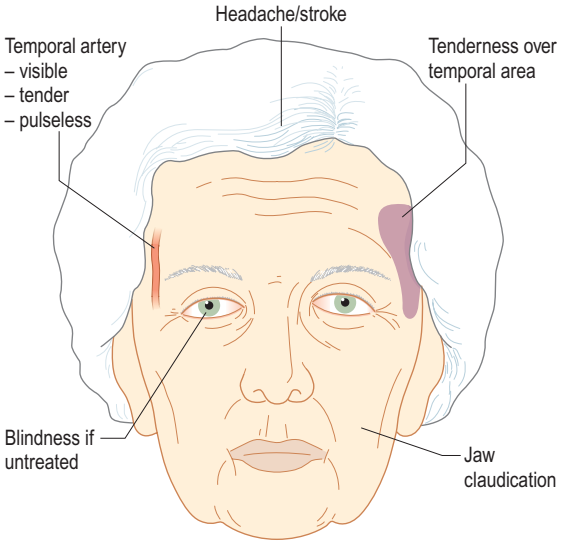


Fig. 7.2 Signs and symptoms of giant cell arteritis in the head and neck.

■ DENTAL RELEVANCE OF GIANT CELL ARTERITIS

- If untreated giant cell arteritis can lead to irreversible blindness.
- Jaw claudication may indicate the presence of giant cell arteritis.
- Patients with PMR have an increased risk of giant cell arteritis.

If giant cell arteritis is suspected the patient should be referred urgently to their general medical practitioner for investigation and treatment to avoid irreversible blindness.

Osteoporosis

Osteoporosis is a common condition affecting one in three women and one in 12 men over the age of 50. There is a decrease in mineral density of normally mineralised bone (Fig. 7.3). The whole skeleton is affected with significant weakening of the structure leading to fractures of the hip and wrist. Fractures of the vertebral bodies lead to collapse of the spine and deformity. Osteoporosis is classified as primary if no predisposing or causative disease can be

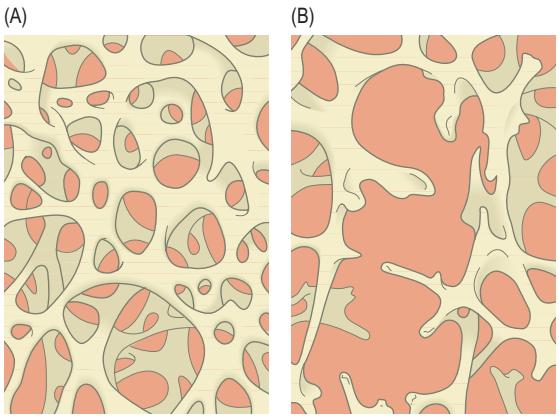


Fig. 7.3 Comparison between normal bone (A) and osteoporotic bone (B). There is decreased bone density in osteoporosis.

■ DENTAL RELEVANCE OF OSTEOPOROSIS

- There are no significant dental implications of primary osteoporosis.
- Dental implants may take longer to integrate in osteoporotic bone.

found or secondary if there is an identifiable cause, e.g. steroid therapy or renal disease. Post-menopausal women are at the greatest risk and some protection can be offered by hormone replacement therapy.

Osteoarthritis

This is the most common form of arthritis and is due to degenerative destruction of the joint cartilage and underlying bone (Fig. 7.4). Unlike rheumatoid arthritis it is limited to the joints and does not affect other tissues. The large weight-bearing joints hips and knees are most commonly affected but the hands, feet and spine may also be involved. Pain after repetitive use is the main symptom with decreased range of movement in severe cases. In primary osteoarthritis there is no detectable cause and this is usually age-related. In secondary osteoarthritis a cause can be found, e.g. trauma, surgery or obesity.

The main aim of treatment is to reduce pain and restore function. This often requires regular analgesia and joint replacement.

■ DENTAL RELEVANCE OF OSTEOARTHRITIS

- Patients may be taking regular NSAIDs so avoid overdose.
- Antibiotic prophylaxis for invasive dental procedures in those with prosthetic joint replacement is controversial.

Paget's disease

Paget's disease is chronic disease of bone in which there is disorganised breakdown and reformation of bone leading to deformity and altered function (Fig. 7.5). The cause is unknown but may be related to a slow virus infection. About 3% of the population are affected, males and females are at equal risk and it is rarely diagnosed under

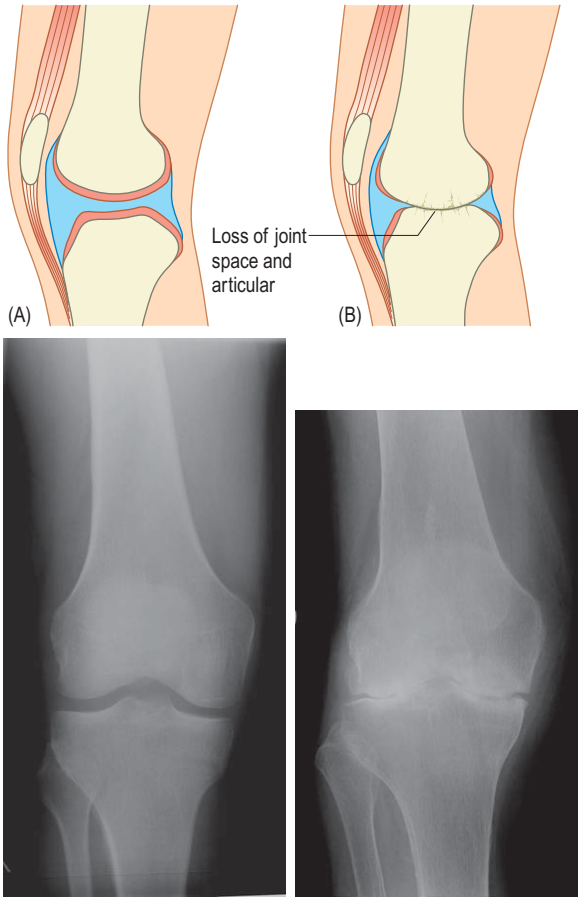


Fig. 7.4 Diagrams and radiographs showing the differences between a normal joint (A) and an osteoarthritic joint (B). Note the destruction of the joint cartilage and reduced joint space in the case of osteoarthritis.

the age of 40. There are random phases of bone resorption and deposition leading to disorganised bone structure. The affected bones become weakened and susceptible to fracture. Bony foraminae become narrowed, putting pressure on the underlying nerves.

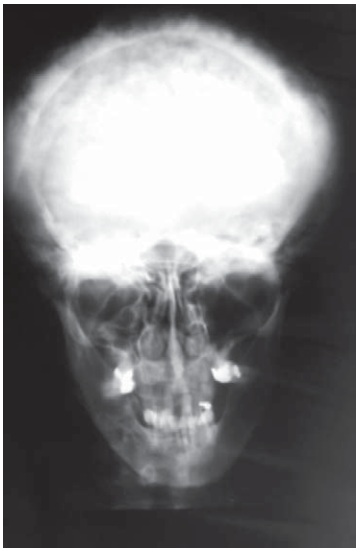


Fig. 7.5 Skull X-ray showing the classical features of Paget's disease with 'cotton wool' appearance of the bone.

Symptoms:
- Deformity
- Bone pain
- Headaches
- Hearing loss.

Treatment includes calcium supplementation, drugs to reduce the rate of bone turnover, i.e. bisphosphonates, and occasionally surgery.

■ DENTAL RELEVANCE OF PAGET'S DISEASE

If the jaw bones are affected there may be:
- mobility of the teeth
- occlusal derangement
- difficult tooth extraction
- hypercementosis
- osteomyelitis
- increased incidence of facial pain.

8 LIVER AND RENAL DISEASE

Mike Escudier

Liver disease

The liver is the largest organ in the body and is involved with almost all of the biochemical pathways that per-mit growth, fight disease, supply nutrients, provide energy and carry out detoxification. Liver disease and its consequences can pose multiple problems in dental practice.

Anatomy

The liver gains 70% of its blood supply from the portal vein and 30% from the hepatic artery. Almost all the nutrients and drugs absorbed from the stomach and intestine are directed to the liver for processing (Fig. 8.1). This so-called first-pass metabolism can be avoided when drugs are given intravenously (IV) or sublingually.

Functions of the liver

- *Protein metabolism*. Synthesis of all proteins apart from gamma-globulins
- *Carbohydrate metabolism*. Glucose homeostasis
- *Lipid metabolism*. Metabolism of lipoproteins
- *Bile acid metabolism*. Conjugation of bilirubin and production of bile
- *Drug and hormone metabolism*. Catabolism and breakdown of a large number of drugs, hormones and vitamins
- *Immunological*. Kupffer's cells act as a sieve for bacteria and other antigens carried to it via the portal system.

Epidemiology

Liver disease is relatively uncommon in the western world. About 600 liver transplants are carried out per year for end-stage liver disease in the UK. Two aspects

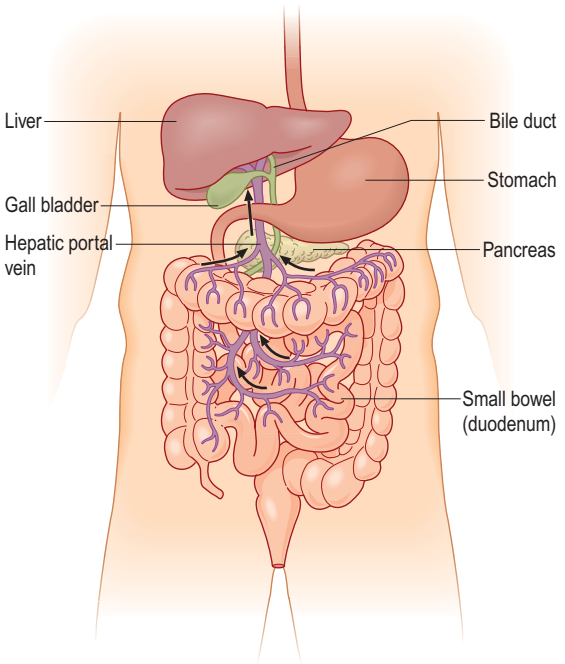


Fig. 8.1 Relationship of the liver to the gastrointestinal tract. Note the hepatic portal vein transporting absorbed nutrients and drugs to the liver for processing.

of liver disease are most important in relation to dentistry:

1. Hepatitis
2. Cirrhosis.

Aetiology

Alcohol consumption is the most common cause of liver disease in the UK. Other less common causes are viral infections, autoimmunity, prescription drugs and genetic disease. The liver is also affected by disease of the biliary tree, particularly gallstones.

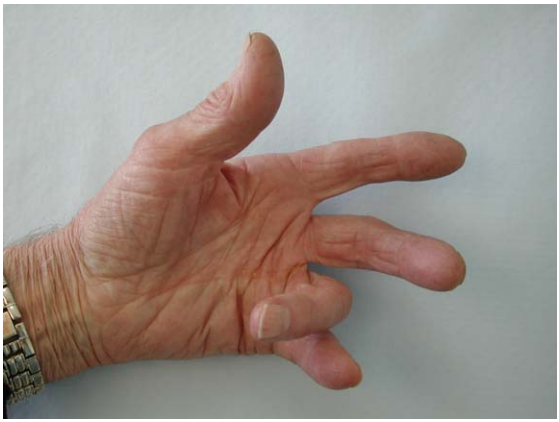


Fig. 8.2 Dupuytren's contracture affecting the ring and little fingers. Notice the scar from attempted surgical correction.

Clinical features

The general features of liver disease that may be detected in dental practice include:

- *Jaundice.* This is a yellow pigmentation of the skin, sclerae and oral mucosa due to deposition of bilirubin in tissues. It is detectable when the bilirubin is above 30–60 mmol/L.
- *Spider naevi.* These small arterial dilations may be detected in the skin of the face and neck.
- *Palmar erythema.* Redness of the palms of the hand.
- *Dupuytren's contracture.* Fixed flexion of the little and sometimes ring finger due to thickening of the palmar fascia (Fig. 8.2).
- *Finger clubbing.* Loss of the normal angle at the bed of the nails.
- *Multiple bruises.* These occur on areas exposed to trauma due to the underlying clotting defect.
- *Delayed healing.* This is due to the decreased synthesis of protein and immunoglobulins.
- *Confusion.* This may occur in severe cases due to unmetabolised toxins reaching the CNS.

Hepatitis

This is inflammation of the liver which may be acute or chronic, causing enlargement, tenderness and deranged function. Viral infection with hepatitis A, B, C, D or E is the most common cause of inflammation of the liver and can present a significant cross-infection risk as well as liver damage. Other viruses, including CMV, EBV, Varicella, Rubella, Toxoplasma and Coxsackie virus and HIV can also cause hepatits (cf. infectious disease section). Alcoholic hepatitis can occur even with moderate intake in susceptible individuals. In rare circumstances acute hepatitis can lead to hepatic failure and death, for example paracetamol overdose.

Cirrhosis

This results from necrosis of liver cells followed by fibrosis and nodule formation. This causes disruption of blood flow through the liver and loss of function. The diagnosis is a histological one and requires a biopsy although the severity of cirrhosis is determined clinically.

If the cirrhosis is asymptomatic the long-term prognosis is usually good, provided the causative factor is under control. Alcohol-induced cirrhosis has a worse prognosis. Patients must abstain from alcohol whatever the cause of their cirrhosis. In long-standing cases there is a small risk of developing hepatocelluar carcinoma.

The liver has a huge reserve capacity and the ability to regenerate so the effect of hepatitis or cirrhosis depends on whether the liver is compensated (coping with reduced capacity) or decompensated (not coping with reduced capacity). Patients with decompensated disease are at a greater risk from any intervention, including dental treatment. Signs of decompensated disease include jaundice, ascites and neurological impairment.

Dental treatment in liver disease

Most of the drugs administered during dental treatment are metabolised by the liver and may affect liver enzyme function (see Table 8.1).

In decompensated disease treatment should be postponed until the failure has been dealt with as there is a high risk of accelerating the rate of failure.

Table 8.1
Drugs to be avoided or altered during dental treatment of patients with liver disease.

Drug name	*Effect or alteration required*
Paracetamol	Limit daily dose to 2 grams in decompensated disease
NSAIDs	Avoid in decompensated disease
Amoxicillin	No change
Metronidazole	Reduce dose to 1/3 and frequency to once daily in decompensated disease
Clindamycin	Reduce dose
Tetracycline	Avoid
Miconazole	Avoid
Lignocaine	Avoid in decompensated disease
Halothane	Avoid
Midazolam	Avoid as may cause coma

■ DENTAL RELEVANCE OF LIVER DISEASE

Post-operative haemorrhage due to deficiency of clotting factors

Altered drug metabolism (see Table 8.1 and check Appendix II of the BNF)

Cross-infection risk in patients infected with hepatitis B, C and HIV

Delayed healing due to protein and immunoglobulin deficiency

Avoid intravenous sedation due to risk of coma

Liver transplant patients require antibiotic cover for invasive dental procedures

Liaise with the physician concerned before treatment.

Renal disease

The kidneys receive approximately 25% of the cardiac output per minute normally producing 1–2 litres of urine per day. Most drugs along with other waste products are excreted by the kidneys and they have an important role in homoeostasis and hormone synthesis.

The functions of the kidney

- Elimination of waste material
- Maintenance of blood pressure
- Maintenance of the composition of the body fluid:
  - regulation of electrolyte balance
  - regulation of acid–base balance
  - regulation of calcium balance
- Endocrine
  - erythropoietin secretion
  - renin–angiotensin system
  - vitamin D metabolism.

Epidemiology

Renal disease is common within the population. The incidence of chronic renal failure increases with age; it is more common in men and those of Asian or Afro-Caribbean origin. 1700 renal transplants were carried out in the UK in 2004. There are about 20000 people in the UK with a functioning renal transplant.

Aetiology

Diabetes is the main cause of end-stage renal failure (ESRF) accounting for 40% of cases. The kidneys may also be damaged by hypertension, ascending infection and immunological mechanisms.

Three aspects of renal disease are most important in relation to dentistry:

1. Renal failure
2. Dialysis
3. Transplantation.

Renal failure

This is said to occur when the kidneys fail to maintain excretory function as a result of a reduced glomerular filtration rate. This may be acute or chronic.

Acute renal failure (ARF)

This is associated with a decline in renal function over a few hours or days.

Aetiology may be pre-renal (poor perfusion), renal (glomerulonephritis, SLE, acute tubular necrosis) or post-renal (obstruction).

Clinical features vary depending on the level of uraemia and range from none, through oliguria (polyuria) to weakness, fatigue, lassitude to pruritis, breathlessness and eventually confusion, fits and even coma.

Treatment is based on identification and treatment of the underlying cause, careful maintenance of fluid balance and dialysis where the level of toxic wastes needs to be reduced.

Chronic renal failure (CRF)

This represents end-stage renal disease.

Aetiology: diabetes (40%), hypertension (25%) and glomerulonephritis (12%).

Clinical features: patients may be asymptomatic but may suffer from, anaemia, nausea, pruritis, hypertension, disturbed urine production, vomiting, oedema, dyspnoea, neuropathy, confusion, fits and coma.

Oral manifestations:

- Ulceration
- Candidiasis
- Parotitis
- Fetor (ammonia-containing breath)
- Lytic lesions in the jaw bone.

Drugs to be avoided or altered during dental treatment of patients with renal failure are listed in Table 8.2.

Table 8.2
Drugs to be avoided or altered during dental treatment of patients with renal failure.

Drug name	*Effect or alteration required*
Paracetamol	Short courses only
NSAIDs	Avoid if possible
Amoxicillin	Reduce dose
Metronidazole	No change
Clindamycin	No changes if short course
Tetracycline	Avoid
Miconazole	Reduce dose
Lignocaine	No change
Halothane	No change
Midazolam	Avoid if possible, use lower dose

Fig. 8.3 An arterio-venous fistula in a diabetic patient with chronic renal failure. Note the scarring from multiple needle insertions.

Treatment. Dialysis is often required with a view to transplantation.

Dialysis allows removal of waste products from the blood when the kidneys have failed. Toxins diffuse across a semi-permeable membrane towards a low concentration present in the dialysis fluid. The two common techniques are haemodialysis and peritoneal dialysis.

Haemodialysis requires a blood flow of 200 mL per minute via a surgically created arterio-venous (AV) fistula in the forearm (see Fig. 8.3). The process takes 4–5 hours three times per week during which the patient is heparinised to minimise the risk of clotting.

Peritoneal dialysis may be either continuous ambulatory (via a permanent catheter) or intermittent (patient remains in bed) which is usually used in ARF. The most common complication of both techniques is peritonitis.

Transplantation is the treatment of choice for end-stage renal failure (see Ch. 17).

■ DENTAL RELEVANCE OF RENAL DISEASE

Preventative dentistry, the key to management.

Minimise the drugs used and alter doses as required (see Table 8.2 for commonly prescribed drugs).

Treat under local anaesthetic.

Treat infections aggressively as patients are often immunosuppressed by the disease or treatment.

High risk of hypertension and its complications.

Screen for bleeding tendencies in CRF before invasive dental procedures.

Lytic lesions may occur in the jaw bones due to secondary hyperparathyroidism.

Dialysis patients:

- Treat on non-dialysis days to avoid bleeding tendency from heparin administration
- Antibiotic cover for AV fistulas
- Increased risk of hepatitis B, C and HIV carriage.

Transplant patients:

- See Ch. 17.

9 HAEMATOLOGY

Georgina Burke

Anaemias

Anaemia is a reduction in the oxygen-carrying capacity of the blood and is defined by a low value for haemoglobin (see box below).

Epidemiology

Anaemia is a major world health problem. In the UK anaemia is most prevalent in women between the ages of 15 and 44 years. Particularly susceptible groups are children under the age of 5 years, pregnant women and those in social classes IV and V.

Classification

This is based on the red-cell mean corpuscle volume (MCV):

- microcytic (small) MCV <80 fl
- normocytic MCV 80–96 fl
- macrocytic (large) MCV >96 fl.

Pathogenesis

Anaemia may be caused by a number of disease states or secondary to drug therapy. Any of the following mechanisms may result in anaemia:

1. Reduced red-cell production:
   (a) defects in haemoglobin function
   (b) decreased production (e.g. deficiency state or bone marrow aplasia)

■ ANAEMIA IS DEFINED BY A LOW VALUE FOR HAEMOGLOBIN

<13.5 g/dl for men

<11.5 g/dl for women.

2. Increased red-cell destruction (haemolysis)
3. Loss of red cells from the circulation (bleeding)
4. Dilutional effect from increased plasma volume (e.g. pregnancy).

Clinical features

Clinical features are varied and depend on the speed of onset of anaemia.

After acute blood loss the symptoms are mainly related to volume depletion with collapse, breathlessness, tachycardia, a poor volume pulse, reduced blood pressure and marked peripheral vasoconstriction.

With anaemia of insidious onset, compensatory mechanisms alter the clinical picture:

Mild anaemia may be asymptomatic or may be associated with lethargy and pallor, particularly of the mucous membranes.

Severe anaemia (Hb <7.0 g/dl) is associated with widespread organ dysfunction:

- cardiorespiratory effects: exertional dyspnoea, tachycardia, palpitations, angina, claudication and cardiac failure
- neuromuscular effects: headache, vertigo, lightheadedness, faintness, tinnitus and increased sensitivity to the cold
- gastrointestinal effects: e.g. loss of appetite, nausea and bowel disturbance
- genitourinary effects: menstrual irregularities, urinary frequency, and loss of libido.

The dental relevance of anaemia is shown in box below.

Microcytic anaemia (MCV <80 fl)

Microcytic anaemia is most commonly due to iron deficiency as a result of blood loss. Other causes include thalassaemia, sideroblastic anaemia and lead poisoning.

■ DENTAL RELEVANCE OF ANAEMIA

There is a marked reduction in the oxygen-carrying capacity of the blood in severe anaemia (Hb <7.0 g/dl) resulting in poor wound healing and widespread organ dysfunction; caution with sedation and drug prescribing.

Iron deficiency anaemia *(Fig. 9.1)*

Causes:

- Increased blood loss (e.g. menorrhagia, gastrointestinal, malignancy)
- Increased iron requirement (e.g. pregnancy)
- Inadequate intake (e.g. vegetarian)
- Impaired absorption (e.g. coeliac disease).

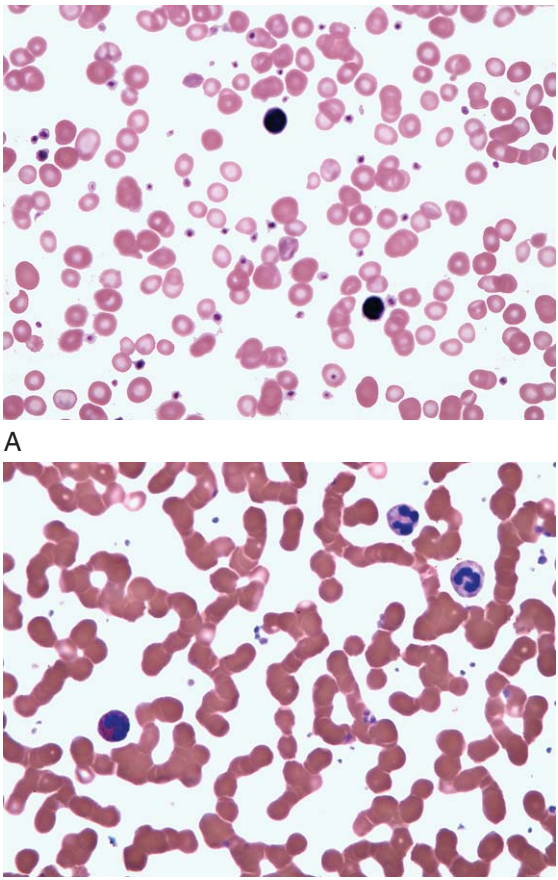


Fig. 9.1 Blood films. (A) Blood from a patient with iron deficiency anaemia showing hypochromic (pale), microcytic (small) red blood cells. (B) Blood from a normal control.

Hook worm is the major cause outside the western world.

The Plummer–Vinson syndrome is a pre-malignant condition associated with chronic atrophic glossitis, angular cheilitis, koilonychia (spoon-shaped nails), achlorhydria and oesophageal web (causes dysphagia). (See also box below and Figure 9.2.)

Iron deficiency anaemia is treated by removing the cause wherever possible and by replacing the body's stores with iron supplements (e.g. oral ferrous sulphate 200 mg t.d.s.).

■ ORAL MANIFESTATIONS OF IRON DEFICIENCY ANAEMIA

- Angular stomatitis
- Glossitis
- Oesophageal web
- Burning mouth.

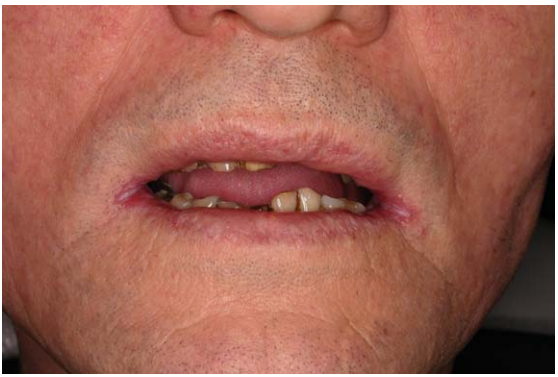


Fig. 9.2 Angular stomatitis can occur in iron deficiency anaemia.

Thalassaemia

The thalassaemias, genetically inherited disorders of haemoglobin, may be found in patients of Asian, Mediterranean and Middle-East origin. The production of either the α or the β chains of haemoglobin may be reduced, resulting in α and β thalassaemia respectively. The composition of normal haemoglobin is shown in box at top of next page.

■ COMPOSITION OF NORMAL HAEMOGLOBIN

Normal haemoglobin is composed of four globin chains.

- HbA ($\alpha_2\beta_2$) = majority adult haemoglobin
- HbA2 ($\alpha_2\delta_2$) = ~2% adult haemoglobin
- HbF ($\alpha_2\gamma_2$) = predominates in fetal life.

Table 9.1
Haematology parameters in iron deficiency anaemia and thalassaemia.

	Iron deficiency	*Thalassaemia*
Iron	↓	Normal
Ferritin	↓	Normal
Hb	HbA	↑ Hb A_2 HbF
Red cell count	↓	↑
Iron supplements	Benefit	No benefit

Thalassaemia major is a severe transfusion-dependent disorder resulting from defective synthesis of β globin (↑ HbF, no HbA). It presents from infancy with failure to thrive, intermittent infections and anaemia. It is often associated with hepatosplenomegaly and bone marrow expansion leading to deformities of the skull with marked frontal bossing and prominent maxillae giving rise to the classical mongoloid facial appearance.

Thalassaemia minor. Carriers for β thalassaemia (HbA2 >3.5%) are usually symptom-free except in periods of stress such as pregnancy, when they may become more anaemic. Thalassaemia trait is believed to offer resistance against falciparum malaria.

The α *thalassaemias* are commoner on a global basis than the β thalassaemias but they pose less of a public health problem because the severe, homozygous forms cause death in utero or in the neonatal period and the milder forms do not produce major clinical problems.

Table 9.1 compares parameters of iron deficiency anaemia and thalassaemia.

The dental relevance of thalassaemia is shown in box at top of next page.

■ DENTAL RELEVANCE OF THALASSAEMIA

- Bony abnormalities may occur.
- Regular blood transfusions can lead to iron overload, resulting in cardiac failure.
- Patients are prone to recurrent infections.
- Patients may have severe anaemia (see above).

Macrocytic anaemia (MCV >96 fl)

Macrocytic anaemia is most commonly due to a deficiency of vitamin B_{12} or folate; both result in megaloblastic haemopoiesis on bone marrow examination. Normoblastic haemopoiesis is seen with other causes of macrocytic anaemia, such as alcoholic liver disease and hypothyroidism.

Vitamin B_{12} deficiency

Vitamin B_{12} is involved in DNA synthesis, so that a deficiency results in abnormal cell growth and maturation.

Causes:

- Inadequate intake (e.g. vegans)
- Impaired absorption (e.g. pernicious anaemia, Crohn's disease).

The absorption of B_{12} depends on two factors: the secretion of intrinsic factor by gastric parietal cells and the ability of the terminal ileum to absorb the B_{12}–intrinsic factor complex. A Schilling test will help to determine whether the B_{12} deficiency is due to lack of intrinsic factor or malabsorption.

Pernicious anaemia is due to lack of intrinsic factor as a result of autoimmune damage to the gastric mucosa.

Complications of vitamin B_{12} deficiency include neurological symptoms (subacute combined degeneration of the cord, peripheral neuropathy) in addition to oral manifestations (glossitis and angular stomatitis) and anaemia.

Treatment is with parenteral B_{12}, 1000 μg every 3 months.

The dental relevance of B_{12} deficiency is shown in the box.

■ DENTAL RELEVANCE OF VITAMIN B_{12} DEFICIENCY

Burning tongue may occur due to glossitis.

The tongue may appear smooth and red due to depapilation ('beefy tongue').

Folate deficiency

Folate is required for the normal synthesis of red cells. A deficiency results in a macrocytic anaemia similar to that occurring with vitamin B_{12} deficiency.

Causes:

- Increased folate requirement (e.g. pregnancy)
- Inadequate intake (especially in old age)
- Impaired absorption
- Drugs (e.g. methotrexate).

Complications include fetal neural tube defects.

Treatment is with 5–10 mg of oral folate (exclude and treat vitamin B_{12} deficiency first to prevent precipitation of neurological complications).

The dental relevance of folate deficiency is shown in the box.

Other causes of macrocytic anaemia are listed in the box at bottom of page.

■ DENTAL RELEVANCE OF FOLATE DEFICIENCY

Increased incidence of oral candidiasis.

■ OTHER CAUSES OF MACROCYTIC ANAEMIA

- Liver disease (especially alcohol-related)
- Marrow infiltration
- Drug treatment (e.g. azathioprine)
- Hypothyroidism
- Pregnancy.

Normocytic anaemia (MCV normal)

This is the anaemia of chronic disease associated with:

- chronic infections (e.g. tuberculosis)
- chronic diseases (e.g. rheumatoid arthritis, renal failure)
- cancer (e.g. carcinoma, lymphoma).

The cause is not fully understood but is probably related to the production of inflammatory mediators. There is no specific treatment for this form of anaemia.

Haemolytic anaemias

The MCV may be reduced (e.g. thalassaemia) or increased (due to increased erythropoiesis).

Haemolytic anaemias are characterised by:

- shortening of the normal red cell life span (i.e. less than 120 days)
- accumulation of the products of haemoglobin metabolism
- a marked increase in bone marrow erythropoiesis.

They may arise because of intrinsic (defective red cells) or extrinsic causes.

Intrinsic causes:

Hereditary

- disorders of red cell membrane (e.g. hereditary spherocytosis)
- disorders of haemoglobin synthesis (e.g. sickle cell disease, thalassaemia)
- deficiencies of red cell enzymes (e.g. glucose-6-phosphate dehydrogenase).

Acquired

- red cell membrane defect (e.g. paroxysmal nocturnal haemoglobinuria).

Extrinsic causes:

Immune

- iso-immune incompatibility (e.g. blood transfusion reactions)
- autoimmune haemolytic anaemia.

Non-immune

- red cell fragmentation (e.g. in patients with prosthetic valves)
- sequestration (e.g. hypersplenism)
- infections (e.g. malaria)
- chemicals (e.g. lead).

Table 9.2 lists investigations carried out for haemolytic anaemias.

Sickle cell anaemia

Sickle cell anaemia is an autosomal recessive, chronic haemolytic disorder associated with intermittent acute crises. It is caused by a point mutation (substitution of valine for glutamine) at the sixth position of the β-globin gene. The haemoglobin formed is 80–99% HbS, with most of the remainder being HbF. Sickle cell anaemia is particularly common in West Africans and Afro-Caribbeans.

When exposed to low oxygen tensions or acidaemia, HbS polymerises, resulting in distortion and sickling of red cells (Fig. 9.3) leading to premature death and blockage of the microcirculation (deformed red blood cells get stuck in small vessels causing tissue ischaemia and death).

Clinical features:

- Progressive anaemia
- Exogenous erythropoiesis, with frontal bossing
- Repeated splenic infarction, resulting in hyposplenism and an increased rate of infection.

Table 9.2
Investigations for haemolytic anaemia.

fbc	Reticulocytosis (↑ rbc destruction)
Blood film	MCV, spherocytes or sickle cells
Bilirubin	↑ unconjugated bilirubin (↑ rbc destruction)
LDH	↑ (↑ rbc destruction)
Urine	↑ urinobilogen (↑ rbc destruction), haemoglobinuria indicates intravascular rbc destruction
Coombe's test	Autoimmune haemolytic anaemia

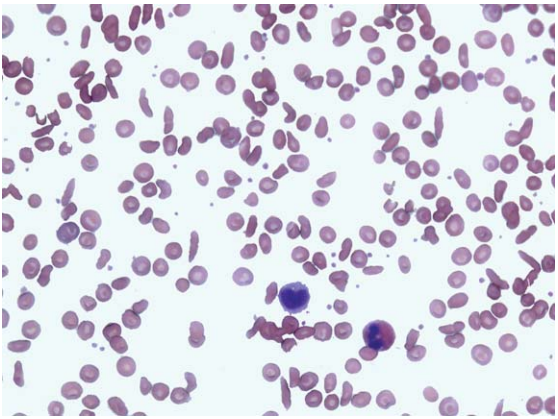


Fig. 9.3 Blood film illustrating sickle-shaped red cells in a patient with sickle cell disease.

■ COMPLICATIONS OF SICKLE CELL DISEASE

- Urinary pigment stones
- Folate deficiency
- Hyposplenism due to splenic atrophy leading to increased risk of pneumoccocal septicaemia
- Stroke
- Salmonella osteomyelitis
- Chronic leg ulceration
- Impaired renal concentrating ability (nephrogenic diabetes insipidus).

Three types of acute crisis are recognized:

- thrombotic (painful)
- aplastic
- sequestration (lung, abdominal).

Sickle cell crises rarely occur in heterozygotes (those with sickle cell trait).

Complications of sickle cell disease are given in box above and the relevance of this disease to dentistry is shown in box which follows.

■ DENTAL RELEVANCE OF SICKLE CELL ANAEMIA

It is safe to treat patients with sickle cell disease in dental practice under local anaesthetic.

Sickle-positive patients are at risk of acute crises if they become:

- dehydrated
- hypoxic
- hypothermic.

Sedation in dental practice should be avoided.

General anaesthesia poses the greatest risk to sickle-positive patients and should be carried out only when absolutely necessary and by a specialist anaesthetist.

Sickle cell crises rarely occur in those with sickle cell trait.

Haemostasis and bleeding disorders

Haemostasis prevents blood loss from vascular injury and is the process of blood clotting followed by dissolution of the clot after tissue repair.

Haemostasis is composed of four events:

1. Vasoconstriction to limit the blood flow
2. Activation and aggregation of platelets by thrombin and fibrinogen to form a platelet plug
3. Activation of the coagulation cascades resulting in a fibrin clot
4. Fibrin degradation by plasmin resulting in clot dissolution.

Platelet activation and von Willebrand factor (vWF)

The adhesion of platelets to collagen exposed after vascular injury is mediated by vWF, which is missing in the inherited von Willebrand's disease (vWD). This glycoprotein also stabilises factor VIII in the intrinsic coagulation cascade. Thus, in vWD, the most common inherited bleeding disorder in males and females affecting about 1% of the UK population, defective platelet adhesion and a secondary deficiency of factor VIII, can cause bleeding that

■ **DENTAL RELEVANCE OF VON WILLEBRAND'S DISEASE**

May present with gingival bleeding or prolonged bleeding after tooth extraction.

May have associated factor VIII deficiency (see haemophilia section).

resembles platelet dysfunction or haemophilia. The dental relevance of this condition is shown in the box above.

Haemophilia

Haemophilia is an X-linked recessive bleeding disorder in which certain clotting factors are missing. The disease usually affects only males but some female carriers can also have low factor levels.

- Haemophilia A = deficiency factor VIII
- Haemophilia B (Christmas disease) = deficiency factor IX.

Both disorders cause bleeding into joints and soft tissues that can be spontaneous.

Haemophilias A and B are classified as mild/moderate/severe depending on the level of clotting factor (<2% is severe). Although patients with mild haemophilia (levels 10–50%) have few problems, **they will require treatment before tooth extraction**, surgery or following trauma.

Treatment involves replacing the missing clotting factors, usually by giving genetically engineered recombinant factors +/– desmopressin (stimulates release of factor VIII).

The dental relevance of haemophilia is given in the box on next page.

Platelet disorders

Thrombocytopenia (reduced platelet numbers) and functional platelet abnormalities result in a bleeding tendency. Thrombocytopenia can arise as a result of reduced platelet production (e.g. bone marrow infiltration or aplasia), increased platelet destruction (e.g. ITP, DIC, sepsis) or sequestration (e.g. hypersplenism). Bleeding generally does not occur until the platelet count has fallen below $10–20 \times 10^9$/L.

■ DENTAL RELEVANCE OF HAEMOPHILIA

Dental treatment in haemophiliacs should be carried out only in liaison with specialist haematology services.

General guidelines are:

- infiltration of local anaesthesia requires no cover with coagulation factor concentrate
- scaling and polishing of teeth usually requires no cover with coagulation factor concentrate
- most haemophilia centres will have a protocol to cover both extractions and following administration of an inferior dental nerve block and will usually recommend an overnight stay.

Potential hazards from dental work include delayed bleeding in the retropharyngeal space after inferior dental nerve block and bleeds into the tongue after cuts from dental instruments.

Many of the adult patients are known to be HIV- and/or hepatitis C-positive, infected from contaminated blood products in the 1970s and early 1980s, and thus precautions should be taken to prevent cross-infection. All blood donations are now screened for these viruses.

Table 9.3
Drugs affecting platelet function.

NSAIDs (aspirin)	Inhibit cyclo-oxygenase resulting in reduced platelet aggregation
Clopidogrel	Inhibit platelet ADP
Gycoprotein IIb/IIIa inhibitors	Inhibit the platelet receptor for fibrinogen thus blocking platelet aggregation
β-lactam antibiotics	Bind and/or modify platelet membrane resulting in abnormal aggregation
Nitrates and β-blockers	Inhibit platelet aggregation

Clinical features:

- Spontaneous cutaneous purpura (bruising)
- Mucous membrane bleeding and nosebleeds
- Menorrhagia and postpartum haemorrhage
- Retinal or subconjunctival haemorrhage
- GI or intracranial bleeding.

Many drugs can also cause thrombocytopenia (e.g. heparin, sulfonamides, quinidine) and also affect platelet function; a few examples are given in Table 9.3.

Idiopathic thrombocytopenic purpura (ITP)

Acute self-limiting ITP is the usual form in children and often arises post-virally. Chronic ITP is an autoimmune disease of adults associated with antibodies against platelet glycoprotein complexes. Initial treatment is with high-dose corticosteroids; splenectomy may be necessary for persistent thrombocytopenia.

Warfarin

More than 450 000 people are taking warfarin in the UK. It is prescribed to prevent blood clots forming or becoming larger. It acts by inhibiting the vitamin K-dependent reactions of the coagulation cascade. This leads to a decrease in thrombin, factors VII, IX, X and proteins C and S.

Indications for warfarin use include:

- prophylaxis of embolisation in rheumatic heart disease and atrial fibrillation
- prophylaxis and treatment of venous thombosis and pulmonary embolism
- prosthetic heart valves
- stroke prophylaxis in patients with transient ischaemic attacks.

The International Normalised Ratio (INR) is used as a measure of anticoagulation:

INR = patient's PT : control PT

in which PT is prothrombin time. The INR is usually maintained between 2.0 and 4.5 when an anticoagulant effect is required. A portable INR-measuring machine is shown in Fig. 9.4. (See also Fig. 9.5.)

Side-effects of warfarin include:

- rash
- liver disorders and jaundice
- alopecia
- skin necrosis
- bruising and an increased bleeding tendency
- gastrointestinal upset
- pancreatitis.

Fig. 9.4 Portable INR measuring machine.

Fig. 9.5 Patients on warfarin usually hold a record of their most recent INR values, the reason for treatment and target range.

Many drugs interact with warfarin – those that potentiate the effects of warfarin will increase the INR (and the risk of bleeding); antagonists will have the opposite effect and reduce the INR (and increase the risk of blood clots).

Drug interactions with warfarin are shown in Table 9.4.

Table 9.4
Drug interactions with warfarin; for a more extensive list check the BNF.

Drug class	*Increased warfarin activity*	*Reduced warfarin activity*
Antibiotics	✓	
Antiepileptics	✓	✓
Antifungals	✓	✓
Hormones and steroids	✓	
Anti-arrhythmics	✓	✓
Analgesics	✓	✓
Allopurinol	✓	✓
Peptic ulcer medication	✓	✓
Alcohol	✓	
Barbiturates		✓

■ DENTAL RELEVANCE OF WARFARINISATION

Pre-treatment evaluation:

- Check need for endocarditis prophylaxis
- Schedule an appointment within 24 hours of INR measurement
- Schedule treatment for first a.m.
- Avoid block injections
- Beware of patients with fluctuating INR readings.

Patients with an INR <4.0 can have most dental treatments (multiple extractions should be carried out in stages) with additional haemostatic measures:

- atraumatic surgery
- packing of the socket with haemostatic gauze e.g. surgicel
- careful suturing of all sockets
- additional pressure application
- careful check for haemostasis.

NSAID analgesia should be avoided due to the risk of peptic ulceration and the additional anti-platelet effect.

Haematological malignancies

In adults, haemopoiesis (blood formation: Fig. 9.6) occurs in the bone marrow of the axial skeleton (vertebrae, ribs, sternum and pelvis). All blood cells are derived from a common (pluripotent) stem cell. Stems cells not only renew themselves but give rise to a series of progenitor cells, one for each lineage.

Acute leukaemia

Acute leukaemias are malignant tumours of haemopoietic precursor cells. There are two types based on origin: acute myeloid leukaemia (AML) and acute lymphoblastic leukaemia (ALL).

Epidemiology

AML affects 1 in 10 000 per year and is more common with increasing age. ALL is the commonest malignancy in childhood and is rare in adults.

Aetiology

The aetiology of acute leukaemia remains unknown but factors that damage DNA (e.g. radiation) can be involved and there is a hereditary predisposition in Down's syndrome.

Classification

This is morphological; there are eight subtypes of AML (M0–M7) and three subtypes of ALL (L1–L3).

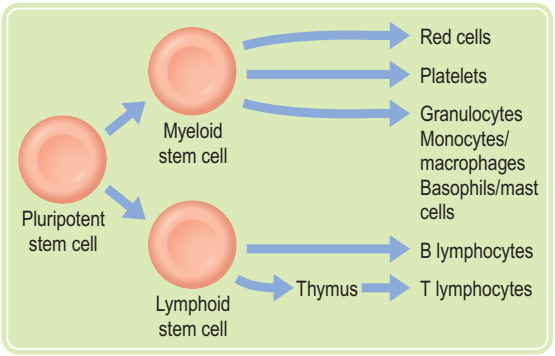


Fig. 9.6 Haemopoiesis.

Clinical features

In acute leukaemias, rapid presentation is common and patients are seriously ill. Clinical features are listed in Table 9.5.

Diagnosis

This is made from the full blood count, blood film and bone marrow examination.

Treatment

Treatment involves intensive chemotherapy to eradicate the leukaemic cells and supportive measures (e.g. blood and platelet transfusions, prophylactic antibiotics). Bone marrow transplantation may be appropriate.

Prognosis

This is good for childhood ALL (cure rate >70%). The prognosis for AML varies depending on age (worst if >60 years) and type of AML.

Chronic leukaemia

Epidemiology

Chronic myeloid leukaemia (CML) is a rare disease (incidence 1/100 000/year) that is most common in middle age. Chronic lymphocytic leukaemia is the commonest adult leukaemia (incidence 20/100 000/year) and is predominantly a disease of the elderly, and of males.

Aetiology

This is unknown for CLL but ionizing radiation and chemicals (e.g. benzene) are associated with CML.

Table 9.5
Clinical features of acute leukaemia.

Bone marrow suppression	Anaemia (lack of rbc)
	Purpura and bleeding (lack of platelets)
	Infection (lack of wbc)
Organomegaly	Lymph nodes
	Spleen
	Liver
Systemic symptoms	Malaise
	Sweats
	Weight loss/anorexia

Clinical features

Many patients with chronic leukaemias are asymptomatic and are discovered incidentally on the full blood count performed for unrelated reasons:

- massive organomegaly: particularly hepatosplenomegaly in CML and lymphadenopathy in CLL
- anaemia due to bone marrow suppression
- systemic symptoms: malaise, sweats, weight loss
- recurrent infections due to immunodeficiency are common in CLL and there is a high incidence of haematological autoimmune disease (e.g. 5–10% develop autoimmune haemolytic anaemia).

Treatment

This is mainly supportive for CLL (e.g. immunoglobulin for recurrent infections) with chemotherapy being reserved for symptomatic or progressive disease. Hydroxyurea is helpful in CML but the disease is incurable without allogenic transplantation.

Prognosis

Most patients with early stage, asymptomatic CLL die of unrelated causes. Prognosis in CML is variable but overall median survival is 5.5 years. The relevance of leukaemia to dentistry is shown in the following box.

■ DENTAL RELEVANCE OF LEUKAEMIA

Patients may present first to the dentist:

1. ALL is the commonest childhood malignancy and may present with gingival bleeding, oral ulceration, sore mouth and increased susceptibility to infections (e.g. oral candidiasis).
2. CLL is the commonest leukaemia in adults and may present with cervical lymphadenopathy or Herpes zoster infections.

Prophylactic antibiotics are recommended for invasive procedures, and infections should be treated promptly and aggressively.

Patients who have undergone transplantation may have additional complications (see Ch. 17).

Extensive chemotherapy regimens often leave patients with reduced reserves in many systems.

Gingival infiltration can occur with some subtypes of AML (M4, M5).

Lymphoma

Lymphomas are tumours of lymph nodes (Hodgkin's disease) or tumours of lymphoreticular tissue derived from malignant B or T cells (non-Hodgkin's lymphoma).

Epidemiology

Non-Hodgkin's lymphoma (NHL) is an increasingly common cause for cancer mortality in young adults. Hodgkin's disease is uncommon in children; median age of onset is 28 years with a smaller second peak in the elderly.

Aetiology

This is unknown for NHL but the Epstein–Barr virus (EBV) may play a role in Hodgkin's disease.

Clinical features

These reflect whether the tumour is low-grade (indolent course, presents with widespread lymphadenopathy) or high-grade (short history of localised rapidly enlarging lymphadenopathy). Hodgkin's disease typically presents as painless cervical and supraclavicular lymphadenopathy. Systemic features (weight loss, night sweats, fever or pruritus) can occur with both types of lymphoma. Figure 9.7 illustrates organs that may be involved in lymphoma, and Figure 9.8 shows gingival lymphoma.

Diagnosis

This is histological and is usually made from a lymph node biopsy. Imaging is used to document the extent of disease for staging.

FBC = normochromic normocytic anaemia
ESR = raised
LDH = raised, indicates poor prognosis in NHL
LFTs = abnormality indicates liver involvement
Ca^{2+} = raised with bone involvement.

Treatment

Treatment depends on the extent of involvement. Radiotherapy is used for localised disease, chemotherapy for generalised disease.

Prognosis

Low-grade NHL = prolonged survival
High-grade NHL = 50% 2-year survival

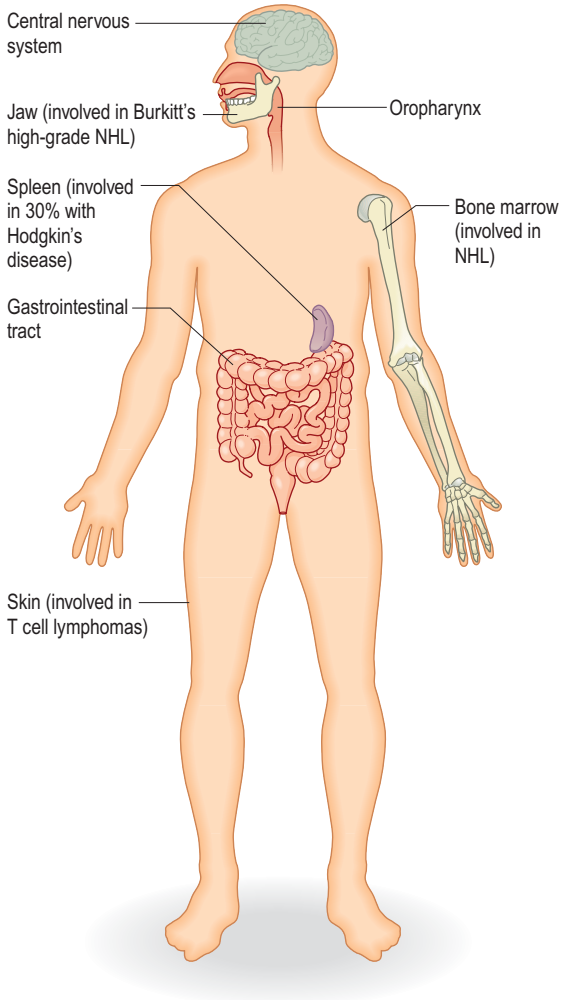


Fig. 9.7 Organs that may be involved in lymphoma.

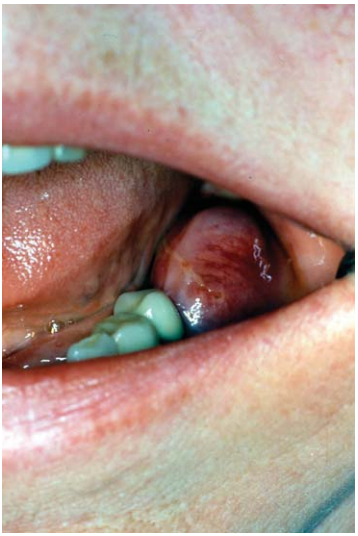


Fig. 9.8 Gingival lymphoma.

■ DENTAL RELEVANCE OF LYMPHOMA

NHL is common in young adults and may present with oropharyngeal involvement of Waldeyer's ring, causing a sore throat or noisy breathing.

Lesions may be found in the salivary glands.

Burkitt's lymphoma is found in patients from EBV-endemic areas and commonly involves the jaw.

Patients who have undergone transplantation may have additional complications (see Ch. 17).

Hodgkin's disease = 5 year survival varies from 90% to 25% depending on the stage and the presence of systemic symptoms.

The relevance of lymphoma to dentistry is shown in the box above.

Multiple myeloma

Multiple myeloma is characterised by malignant proliferation of a single clone of bone marrow plasma cells and is the most common type of paraproteinaemia with a prevalence of 3/100 000.

Paraproteins are monoclonal immunoglobulins. Often there is an excess of light-chain synthesis, which may appear in the urine as Bence–Jones proteins. Primary amyloidosis develops in 5–10% of patients.

Epidemiology

Peak incidence is in the 7th decade. Multiple myeloma is more common in males and Afro-Caribbeans.

Aetiology

This remains unknown.

Clinical features

These result from:

- tumour replacement of bone marrow causing bone marrow suppression: anaemia, leucopenia and thrombocytopenia
- production of paraprotein causing an increased viscosity of the blood and amyloid
- lytic lesions in the bone causing bone pain, pathological fractures and hypercalcaemia
- renal impairment due to dehydration, light-chain deposition and hypercalcaemia
- infections of the oral cavity (fungal and bacterial) and respiratory tract; the latter are a major cause of death.

Treatment

This involves supportive measures (e.g. analgesia, rehydration and treatment of infection) and median survival is 3–4 years. Chemotherapy and plasma exchange may be required for symptomatic hyperviscosity. Selected patients may undergo autologous transplantation.

The relevance of multiple myeloma to dentistry is shown in the box.

■ DENTAL RELEVANCE OF MULTIPLE MYELOMA

Multiple myeloma is a cause of lytic lesions in the jaw and skull.

Patients may present with anaemia or bleeding problems.

Patients are prone to oral candidiasis.

Caution with NSAIDs if renal impairment exists.

Patients who have undergone transplantation may have additional complications (see Ch. 17).

10 SKIN DISEASES

Jane Setterfield

History and examination, including structure and function of the skin

The skin is one of the largest organs of the body. It comprises distinct layers which together provide several vital functions.

The functions of skin:
1. Protection from the environment
2. Thermoregulation
3. Neuroreceptor
4. Antigen processing
5. Metabolises vitamin D
6. Provides cosmetic functions.

The structure of human skin is shown in Figure 10.1.

The epidermis is composed of layers of keratinocytes. When intact it provides a relatively impermeable barrier to inward penetration by chemicals, microorganisms and allergens. It provides some protection against the carcinogenic effects of UV radiation. Furthermore it has an immunological role via Langerhans cells, which are antigen-presenting cells. These are implicated in contact dermatitis through a type 4 hypersensitivity reaction.

The basement membrane zone is a complex structure containing many interlinking components which have an important structural role by maintaining firm adhesion between the epidermis and dermis. It acts as a barrier to inflammatory and neoplastic cells and is involved in cell signalling pathways.

The dermis provides nourishment to the epidermis and interacts with the epidermis during embryogenesis and wound repair.

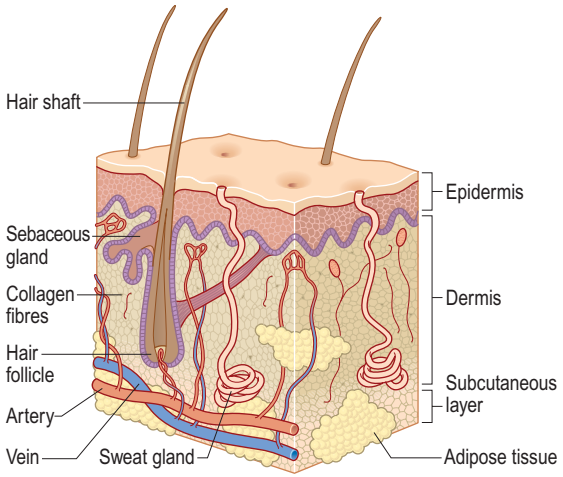


Fig. 10.1 Structure of human skin.

Connective tissue comprises collagen, elastin and ground substance which provides strength and elasticity. Blood vessels and eccrine glands play a vital role in thermoregulation. A complex innervation of skin and hair follicles comprise the neuroreceptor sense organ. Apocrine glands secrete a viscous fluid which produces distinctive body odours

Assessment of a patient

History

- Age
- Occupation
- History of presenting complaint, e.g. precipitating cause, such as association with new medication, itch, pain, blistering
- Relevant systems review, e.g. joint pains, gastrointestinal problems
- Relevant past medical history or family history.

Examination

- Skin (ideally whole surface including the scalp)
- Mucous membranes (eyes, mouth and genitalia if indicated).

It is important to be able to describe the appearance of skin lesions accurately.

Primary lesions

Macule: a circumscribed flat change in the colour of skin which is less than 1 cm in diameter
Papule: circumscribed palpable elevation of the skin < 1 cm in diameter
Nodule: circumscribed palpable elevation of the skin > 1 cm in diameter
Patch: a flat lesion > 1 cm in diameter
Plaque: a slightly raised lesion > 1 cm diameter
Vesicle: a raised lesion < 0.5 cm containing clear fluid
Bulla: a vesicle > 0.5 cm diameter
Wheal: a transient pink or red swelling of the skin, often with central pallor
Telangiectasia: dilatation of capillaries.

Secondary lesions

Crust: a dried exudate which may have been serous, purulent or haemorrhagic
Excoriation: a shallow haemorrhagic excavation resulting from scratching. It may be linear or discrete.
Lichenification: a thickening of the skin with exaggeration of skin creases
Scar: the final stage of healing of a destructive process which involves the deeper dermis resulting in a smooth shiny lesion
Erosion: partial break in the epidermis which heals without scarring unless secondary infection occurs
Ulcer: full-thickness loss of the epidermis which heals with scarring
Atrophy: a thinning and translucency of the skin with loss of skin markings.

Dermatitis and psoriasis

Dermatitis (*syn.* excema)

There are several clinical sub-types based upon the underlying aetiology which are listed below:

1. Atopic dermatitis
2. Seborrheic dermatitis

3. Lichen simplex
4. Contact allergic dermatitis
5. Contact irritant dermatitis.

Atopic dermatitis

This is a chronic, itchy disorder with a strong genetic predisposition. There is often an association with asthma or rhinitis. Onset is in infancy in 60% patients with 80% clear between the ages of 2 and 5 years.

Clinical findings. Characteristic patterns of skin involvement:

- extensor surfaces and the face in infancy
- flexures of the knees and arms in childhood
- a more generalized involvement in adults or discoid (coin-shaped) lesions.

In the acute form there is erythema, weeping, papules, vesiculation (pompholyx). Chronic eczema is associated with dryness, scaling, lichenification, hyperpigmentation. There may be secondary infection with *Staphylococcus aureus* 'impetiginisation' or with herpes simplex virus 'eczema herpeticum'. (See Fig. 10.2.)

Investigations:

- Detection of raised serum IgE elevated in 80% patients and frequent positive radioimmunoassay to house-dust mite, animal dander and pollens.

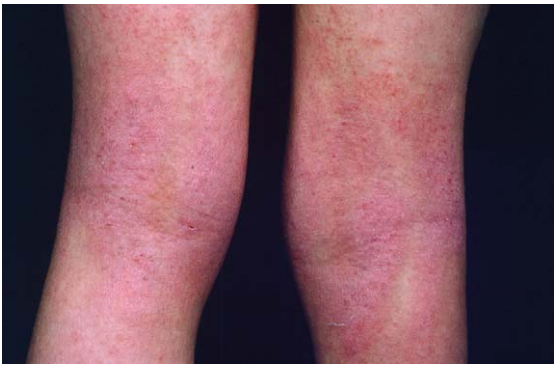


Fig. 10.2 Flexural eczema showing erythema and lichenification.

Management:

- Avoidance of known allergens, e.g. pets, house-dust mite etc
- Regular emollients and 'wet wrapping' the limbs in children with moistened bandages
- Topical steroids and/or tacrolimus ointment
- Antibiotics
- Sedating antihistamines
- Occasionally phototherapy or systemic immunomodulating therapy, e.g. prednisolone, azathioprine, ciclosporin.

Contact allergic dermatitis

This is becoming increasingly common among dentists who are exposed to high-risk dental materials, e.g. latex.

- Type IV hypersensitivity reaction (see Ch. 13)
- Contact exposure to allergen in previously sensitised individual
- Occupational risk/hobbies, e.g. dentists, nurses, hairdressers, gardening, DIY, photography
- Affected site may suggest a contact allergy, e.g. face/neck – nail varnish, cosmetics, perfumes; ears, wrist, umbilicus – nickel allergy (jewellery, watch strap, buckle); scalp – hair dyes.

Investigations:

- Patch testing.

Management involves avoidance of known allergens by use of latex-free gloves for example.

Other subtypes of dermatitis include:

- *Seborrhoeic eczema*, which predominantly affects the central face, scalp, anterior chest
- *Lichen simplex*, a localized area often on the hands, ankles or neck precipitated by chronic scratching
- *Irritant contact dermatitis*, which is not an immunological reaction and does not require prior sensitisation. At-risk patients are those with frequent contact with irritants and wetting of hands, e.g. healthcare workers, cleaners etc.

Dental relevance of dermatitis is shown in the following box.

■ DENTAL RELEVANCE OF DERMATITIS

- Patient may be on corticosteroids.
- Eczema may affect the lips (cheilitis).
- Dentists and allied professionals are at high risk of developing allergic contact dermatitis and should always protect themselves from high-risk dental materials.

Psoriasis

This is a common chronic benign hyperproliferative condition of the skin that affects 2% of the population.

There is a family history in approximately one-third of cases. It is characterized by symmetrical well-defined erythematous plaques with a thick silvery scale. There may be nail changes (pitting) and arthropathy.

Sub-types:

- Chronic plaque psoriasis: the commonest form (Fig. 10.3)
- Guttate: common disorder of adolescents and young adults; acute eruption which may appear after a streptococcal throat infection

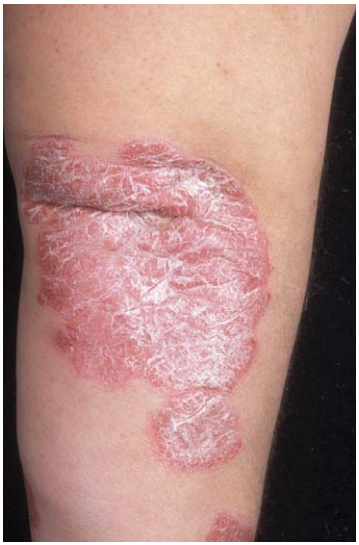


Fig. 10.3 Chronic plaque psoriasis.

- Erythrodermic
- Pustular: e.g. palms and soles or rarely generalised.

Histology:

- Capillary dilatation, polymorphonuclear infiltration, parakeratosis.

Treatment is varied, and depends on the severity of the disease and effect on the patient's lifestyle:

- Topical preparations
- Emollients, corticosteroids, tar products, dithranol, vitamin D_3
- Phototherapy UVB, PUVA
- Systemic therapies, including methotrexate, cyclosporin, retinoids, infliximab.

The box shows the relevance of psoriasis to dentistry.

■ DENTAL RELEVANCE OF PSORIASIS

- Joint involvement may impair oral hygiene.
- Rarely oral changes may be seen, e.g. erythema migrans-like appearance.
- Patients may be immunosuppressed.

Mucocutaneous diseases

Lichen planus

This is a relatively common pruritic autoimmune disorder of unknown aetiology. Onset usually 30–60 years, with similar incidence in males and females. Always consider lichenoid drug eruption, e.g. gold, beta-blockers, thiazides.

Clinical variants

- Hypertrophic, atrophic, linear (Koebner's phenomenon)
- Acute generalised, annular, palmar or plantar, bullous
- Oral (several patterns), actinic (check lips)
- Genital (often associated with oral)

- Lichen planopilaris (scalp) may result in scarring alopecia
- Nail.

Figure 10.4 shows lichen planus on the elbow.

Examination

- Flat-topped, shiny papules in symmetrical distribution with a predilection for flexor surfaces and lower back
- Hypertrophic plaques
- Atrophic lesions – flexures
- Scalp – may result in scarring alopecia
- Nails – linear grooves +/– scarring (pterygium).

Histology: hyperkeratosis, 'saw-tooth' rete ridges, degeneration of the basal layer and band-like lymphocytic infiltrate in papillary dermis.

Treatment depends on symptoms and extent:

- topical: corticosteroids, emollients
- systemic: prednisolone, azathioprine, cyclosporin, PUVA.

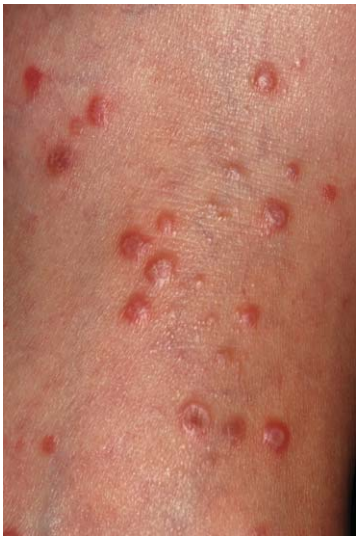


Fig. 10.4 Lichen planus affecting the flexor surface of the elbow.

Prognosis: cutaneous LP often remits in 1–2 years; mucosal LP may persist for several years.

The relevance of lichen planus to dentistry is shown in the box.

■ DENTAL RELEVANCE OF LICHEN PLANUS

- Oral lesions often present and are painful if atrophic or erosive.
- Consider other mucosal site involvement, e.g. oesophagus and eyes.
- The patient may be taking corticosteroids and may be at increased risk of infection.

Autoimmune blistering diseases

These are an autoantibody-mediated group of disorders characterised by the appearance of blisters or erosions of skin and/or mucosal sites, e.g. the mouth, eyes, nasopharynx, genitalia, oesophagus or larynx. Blistering occurs at different levels of the skin/mucous membrane.

Subepidermal blister:

- Bullous pemphigoid
- Mucous membrane pemphigoid
- Linear IgA disease
- Epidermolysis bullosa acquisita.

Intercellular blister:

- Pemphigus vulgaris (Fig. 10.5).

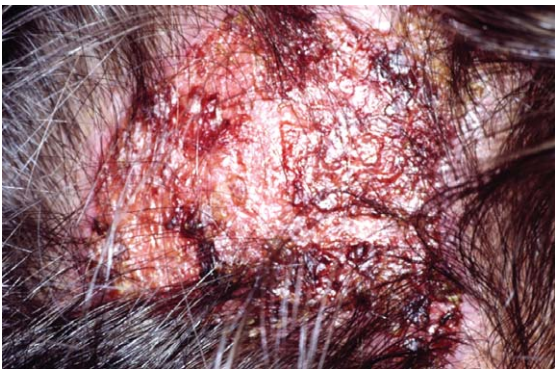


Fig. 10.5 Erosions on the scalp in pemphigus.

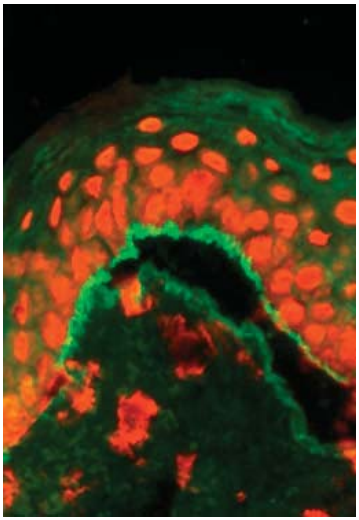


Fig. 10.6 Direct immunofluorescence showing linear IgG at the basement membrane in mucous membrane pemphigoid.

Investigations

- Diagnosis is confirmed by characteristic histology and direct immunofluorescence (IF).
- The subepidermal group demonstrate linear fluorescent staining (IgG, IgA, C3) at the dermoepidermal junction (see Fig. 10.6).
- Intraepidermal IgG is consistent with the pemphigus group.

Management

This usually combines topical corticosteroid preparations with immunosuppressive agents, e.g. dapsone or prednisolone combined with a steroid-sparing agent such as azathioprine or mycophenolate mofetil.

Skin tumours

Basal cell epithelioma (basal cell carcinoma)

This is the commonest malignant neoplasm of skin arising from the basal layer of keratinocytes. It is most commonly

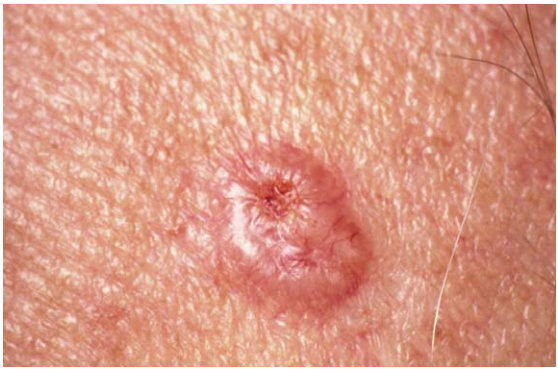


Fig. 10.7 'Classical' appearance of a basal cell carcinoma as a raised nodule with a pearly edge.

seen on sun-exposed sites, e.g. the face, particularly around the eyes, nose, scalp and trunk. The tumour enlarges slowly and is locally destructive but rarely metastasises. There is a variety of presentations; however, the commonest is an erythematous nodular lesion with a pearly opalescent edge and superficial dilated capillaries (Fig. 10.7).

Treatment depends on the type of tumour, site, size and general health of the patient. The most definitive therapeutic interventions are surgical excision occasionally followed by radiotherapy. Small lesions in the elderly may be curetted but will require follow-up since the relapse rate is much higher.

Squamous cell carcinoma

These tumours also arise from keratinocytes but have potential for metastatic spread either via lymphatics or more rarely haematogenous dissemination. They characteristically arise de novo in sun-exposed sites, e.g. lower lip; however, they may arise from pre-existing lesions such as actinic keratoses or Bowen's disease. Immunocompromised patients are at higher risk, e.g. transplant recipients. The tumour may be nodular or ulcerative and is often painful.

Treatment is by excision or radiotherapy.

Melanoma

This tumour is derived from melanocytes in the basal layer of the epidermis and may arise either de novo or from a pre-existing pigmented lesion, e.g. an intradermal naevus. It often metastasizes early therefore early detection and excision is critical. The incidence is rising and is currently approximately 1 in a 100 in Europe and the USA.

Risk factors: excess sun exposure, particularly in early childhood, white skin, a family history of melanoma and multiple naevi.

Frequent sites include the leg in females, the back in males and the face in older patients of either sex.

Prognosis is primarily based upon the depth of invasion within the dermis.

Melanoma should be considered if the history suggests:

- a change in the shape, size, colour of a mole
- a new naevus which is enlarging
- itching or bleeding from a mole
- a naevus greater than 5 mm in size.

Four histological sub-types are recognized:

1. Superficial spreading malignant melanoma – commonest sub-type (80% of cases in caucasians) presenting as an expanding flat irregularly pigmented lesion
2. Nodular melanoma – rapidly-growing black or red nodule
3. Lentigo maligna melanoma – usually a slow-growing lesions on the face of elderly patients
4. Acral melanoma (palms and soles including subungual melanoma) – the rarest site in caucasians but the most frequent site in Asian skin.

A nodular melanoma is shown in Figure 10.8.

Treatment is by excision.

The relevance of skin tumours to dentistry is shown in the box.

■ DENTAL RELEVANCE OF SKIN TUMOURS

Skin tumours commonly present on the face.

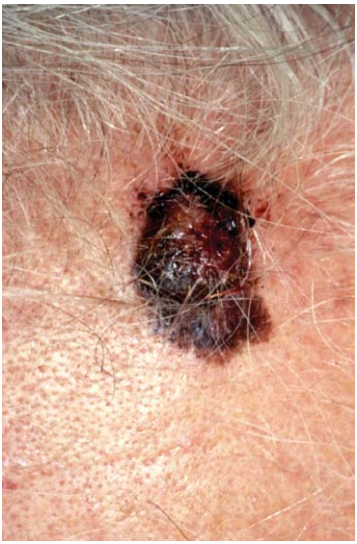


Fig. 10.8 A nodular melanoma on the skin of the forehead.

Cutaneous manifestations of systemic diseases

Many systemic diseases have cutaneous manifestations. The most common are listed in the box.

■ CUTANEOUS MANIFESTATIONS OF SYSTEMIC DISEASE

Thyroid disorders
Hypothyroidism

- dry, coarse hair
- pruritus

Grave's disease

- pretibial myxoedema
- pruritus
- hyperhidrosis
- periorbital soft-tissue swelling

Cont'd

Adrenocortical dysfunction

Addison's disease

- hyperpigmentation of skin and mucous membranes

Cushing's syndrome

- buffalo hump
- truncal obesity
- striae
- acne vulgaris
- hirsutism

Diabetes

- granuloma annulare – localised or generalised
- necrobiosis lipoidica
- recurrent or persistent fungal and bacterial infections of skin
- pruritus
- vascular disease – large and small vessel ischaemia
- lipoatrophy

Acromegaly

- soft tissue hypertrophy
- greasy skin

Crohn's and ulcerative colitis

- pyoderma gangrenosum (also associated with rheumatoid arthritis, and haematological disorders, e.g. paraproteinaemia, myeloma)
- perianal ulceration, skin tags, fistulae
- oral – aphthae and 'cobblestone' appearance of mucosa

Connective tissue diseases

Systemic lupus erythematosus

- malar rash on face
- vasculitic lesions
- Raynaud's phenomenon
- alopecia
- scarring skin/scalp lesions in discoid lupus

Scleroderma

- skin thickening and contractures

11 INFECTIOUS DISEASES

Judith Jones

Background

Infectious diseases may have an oral source or cause oral lesions. There may be a risk of cross infection. Oral infections may indicate an underlying disease process or condition. Dental staff should be immunised against the common infective organisms.

The *severity* of an infection will depend on a number of variables, including:

- host resistance
- pathogenicity and virulence of the infecting agent
- environmental factors.

The routes of infection are shown in the box below.

Manifestations of infection

Fever is due to an increase in body temperature which helps to prevent the multiplication of many pathogens. Its adverse effects include delirium and febrile convulsions. Fever may make pre-existing disorders worse, e.g. a relapse in multiple sclerosis.

Inflammation results from the release of inflammatory mediators and increases vascular permeability, allowing cells of the immune system to reach areas of infection. It is characterised by erythema, oedema, pain and heat in affected area.

■ ROUTES OF TRANSMISSION OF INFECTION

Fomites – inanimate objects

Vectors – living creatures, e.g. mosquito, flea, louse

- Direct contact
- Inhalation
- Ingestion
- Inoculation.

Bacterial infections

Tuberculosis (causal bacterium: *Mycobacterium tuberculosis*)

Epidemiology. Approximately 7000 cases were reported in the UK in 2004, with an increase in notifications among immigrants and high-risk populations, e.g. HIV/AIDS.

Spread is by inhalation of infected sputum.

Types of infection. Tuberculosis (TB) most commonly occurs in the lungs (pulmonary) but may involve many other systems or organs (non-pulmonary), including the CNS, spine, renal and gastrointestinal tracts. The infection may be latent or active (see Table 11.1).

Clinical features are often non-specific and include:

- cough >2 weeks
- pain in chest
- haemoptysis
- weakness and fatigue
- weight loss
- fever and night sweats.

Diagnosis is based on clinical examination, chest X-ray (Fig. 11.1), tuberculin testing (Heaf or Mantoux), microbiology and histological examination of specimens.

Treatment requires prolonged use (>6 months) of multiple antibiotics to avoid resistance. Regimes are tailored depending on the host status and organism(s) involved.

Table 11.1
Features of latent and active tuberculosis.

Latent tuberculosis	*Active tuberculosis*
TB alive in body but inactive	TB alive and active in body
No symptoms of disease	Symptoms of disease
Positive skin reaction	Positive skin test
Normal chest X-ray and sputum test	Abnormal chest X-ray and sputum test
Not contagious	Contagious
May develop TB later	

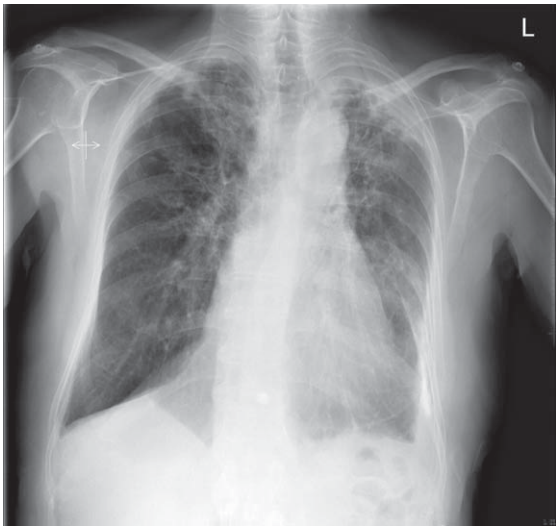


Fig. 11.1 Chest X-ray of a patient with pulmonary TB.

■ DENTAL RELEVANCE OF TUBERCULOSIS (TB)

All dental staff should be vaccinated.

Painful ragged ulcers can occur in the mouth, usually secondary to pulmonary TB.

Cervical lymphadenopathy may occur.

Patients with open pulmonary TB pose a high cross-infection risk: delay treatment if possible.

Syphilis (causal bacterium: *Treponema pallidum*)

Epidemiology. The incidence of syphilis in the UK increased by 144% to 600 cases between 2000 and 2001.

Spread is by direct contact.

Clinical features. The disease occurs in stages:

- *primary syphilis* – painless, indurated ulcer, at the site of infection (chancre)
- *secondary syphilis* – fever, rash and snail-track ulcers in mouth or genital regions due to systemic spread of the bacterium
- *latent period* – may last up to 2 years, during which slow tissue damage occurs

- *tertiary syphilis* – gumma formation which affects skin, mucosa and bone. Degeneration of the spinal cord may lead to dementia, pains and ataxia – 'general paralysis of the insane'.

Diagnosis. Dark-ground microscopy of a smear from a chancre, spirochaetes visible, enzyme immunoassay (EIA) to detect IgM and IgG.

Management. Procaine penicillin (erythromycin or tetracycline if the patient is allergic).

Complications. Infection of fetus may cause miscarriage or lead to characteristic features, including saddle-shape deformity of the bridge of the nose and destruction of nasal cartilages, papules at the commissures of the mouth which scar on healing, Hutchinson's incisors (tapering and notched incisors), and Moon molars (rounded or domed first permanent molars).

■ DENTAL RELEVANCE OF SYPHILIS

- Oral ulceration – during primary and secondary stages
- Oral gumma – tertiary syphilis
- Involvement of neck lymph nodes
- Congenital abnormalities of teeth, Hutchinson incisors, Moon molars
- Papules at commissures
- Cardiovascular defects.

MRSA

Methicillin-resistant *Staphylococcus aureus* (MRSA) is resistant to certain antibiotics, including methicillin, oxacillin, penicillin and amoxicillin. Severe staphylococcal infections, including those mediated by MRSA, occur most frequently among in-patients who have weakened immune systems.

Many healthy people are colonised with MRSA and have no ill effect from the bacteria; these can be treated safely in general dental practice. Normal cross-infection control procedures apply.

■ DENTAL RELEVANCE OF MRSA

If dental staff are found to be colonised with MRSA this must be eradicated before any further patient contact occurs.

Universal cross-infection measures are adequate.

Viral infections

Hepatitis (hepatitis A, D, C, E, G)

Hepatitis A

Hepatitis A virus (HAV) is an RNA hepatovirus of the family Picornaviridae.

Epidemiology. Hepatitis A is a highly infectious common disease. Its incidence is increased by poor sanitation, so many children in the developing world are infected either clinically or subclinically and have immunity.

Spread is via the orofaecal route.

■ DENTAL RELEVANCE OF HEPATITIS A

Nil, except to distinguish it from other types of hepatitis – see below.

Hepatitis B

Hepatitis B virus (HBV) is a double-stranded DNA virus.

Epidemiology. Hepatitis B is uncommon in UK, about 500 cases per year, which occur mostly in the high-risk groups.

Spread: mainly parental, sexually and perinatally.

Symptoms:

- Incubation period: 3–6 months, prodromal period of 1–2 weeks
- Malaise
- Anorexia
- Jaundice and tender, enlarged liver
- Nausea
- Muscle pains, arthritis; a rash can occur.

Diagnosis. Serological tests for antigens and antibodies.

Antigens. A large amount of HbsAG is produced during replication of the virus, which is detectable in the serum 2–8 weeks before clinical illness. HbeAG then appears. Antibodies to viral proteins appear in the blood as recovery progresses.

Management:

- Prevention by immunisation with recombinant DNA HbsAg vaccine
- Treatment – bed rest, avoidance of hepatotoxins (e.g. alcohol)

Complications. Carrier state. Chronic liver disease, death from which occurs in 15–25% of chronically infected people. Rarely hepatocellular carcinoma.

■ DENTAL RELEVANCE OF HEPATITIS B

- Immunisation of all healthcare workers
- Cross-infection control
- Abnormal drug metabolism in liver
- Abnormal clotting factors.

Hepatitis C, D

Epidemiology. It is estimated that 200 000 people are infected with hepatitis C in the UK.
Spread. Same as for hepatitis B.
Symptoms. Same as for hepatitis B.
Diagnosis. Serological tests.

Management:

- No vaccination available
- Avoid hepatotoxins, e.g. alcohol
- Interferon and ribavirin used for patients with chronic liver disease.

Complications. Hepatocellular carcinoma.

■ DENTAL RELEVANCE OF HEPATITIS C

- Cross-infection control
- Abnormal drug metabolism in liver
- Abnormal clotting factors.

Herpes virus infections

Herpes simplex (types 1 and 2)

Traditionally, type 1 infected the mouth and upper body and type 2 the genital region, although the distinction is no longer clear cut.

Acute herpetic gingivostomatitis

Epidemiology. Primary infection is often seen in childhood and adolescence.

Spread. Direct contact.

Symptoms. Infection is often subclinical but may cause:

- pyrexia
- malaise
- lymphadenopathy
- oral vesicles which break down to form ulcers, painful, lasts 10 days.

Diagnosis. Usually clinical.

Figure 11.2 shows acute herpetic gingivostomatitis of lower lip and tongue.

Management is symptomatic:

- hydration
- soft diet

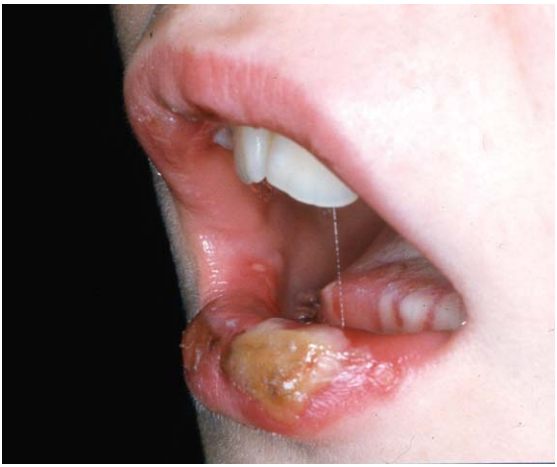


Fig. 11.2 Acute herpetic gingivostomatitis affecting the lower lip and tongue.

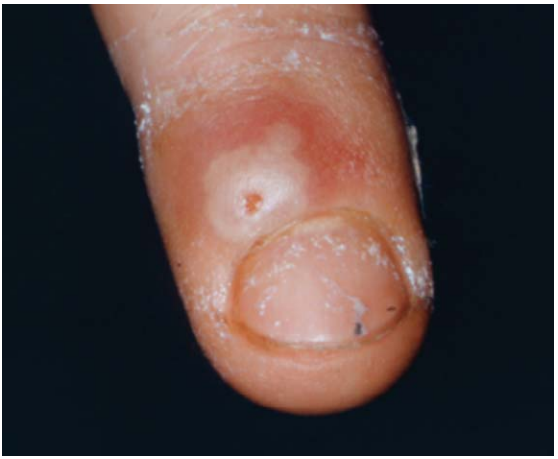


Fig. 11.3 Herpetic whitlow – this acutely painful lesion is due to infection of the finger with Herpes simplex.

■ DENTAL RELEVANCE OF HERPES INFECTION

Herpetic whitlow – finger infection with Herpes virus by direct contact with lesions.

Oral lesions – virus may become latent in the trigeminal ganglion or basal ganglia of the brain. May be reactivated to cause a secondary infection.

- analgesia
- topical and systemic acyclovir 200 mg 5 times a day for 5 days.

Complications. Herpes simplex encephalitis.

Virus becomes latent in the trigeminal ganglion or basal ganglia of the brain. It may then be reactivated to cause a secondary infection (see below).

Figure 11.3 shows a herpetic whitlow.

Herpes labialis/recurrent Herpes

Reactivation of latent virus following primary Herpes infection (approx. 50–70% individuals affected with primary Herpes) or following subclinical primary infection (Fig. 11.4).

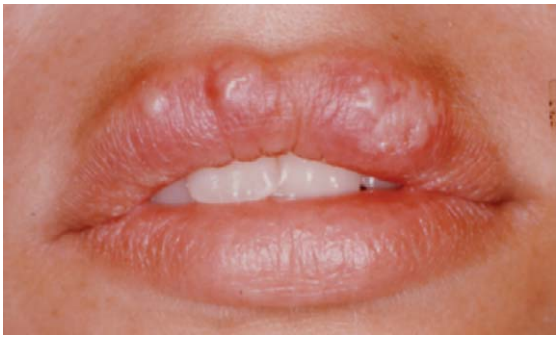


Fig. 11.4 Herpes labialis due to reactivation of Herpes virus from the trigeminal ganglion.

■ DENTAL RELEVANCE OF HERPES LABIALIS INFECTION

Lesions on the lips.

Reactivating factors. Include trauma, chemicals, sunlight, hormones, stress, immunosuppression and concurrent infection.

Symptoms. During the prodromal 24-hour period, prickling sensation on the lip is followed by vesicle formation; then the lesion crusts over. There may be severe pain and lymphadenopathy.

Diagnosis. Clinical.

Managment. Acyclovir topical paste, applied five times daily for 5 days. This needs to be applied in the prodromal phase to be effective.

Varicella zoster virus (VZV)

Chickenpox

Epidemiology. Common childhood infection.

Spread. Direct contact with patients with chickenpox or shingles (herpes zoster).

Symptoms:

- Fever
- Malaise
- Lymphadenopathy

- Itchy vesicular rash which starts on trunk or head and spreads across body. Mucosal lesions also occur.

Diagnosis. Usually clinical.

Management. Supportive, with analgesics and antihistamines for rash. In adults and severe childhood cases acyclovir 800 mg five times daily for 5 days can be used, but to be most effective it must be started at the first sign of the rash.

Complications. Reactivation of dormant virus in nerve tissue to give Herpes zoster or shingles.

■ DENTAL RELEVANCE OF CHICKENPOX

Oral lesions – ulcers.

Herpes zoster

Reactivation of VZV in a dermatomal distribution which can include regions of the trigeminal nerve.

Symptoms. Severe pain, vesicles and ulceration (Fig. 11.5).

Diagnosis. Usually clinical.

Management: Acyclovir 800 mg five times daily for 7 days, but needs to be given within 72 hours of onset to be effective.

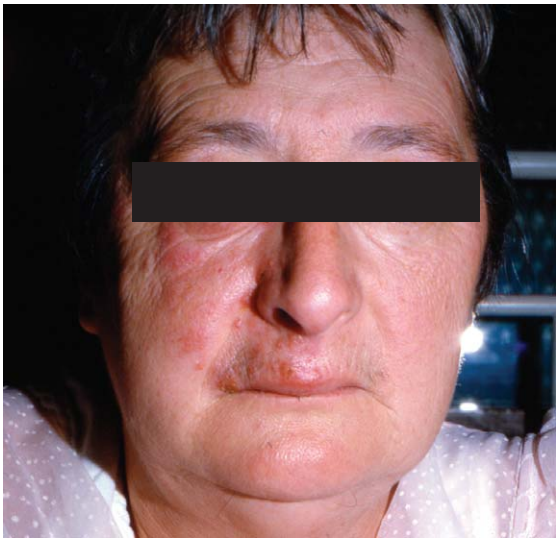
Complications. If the ophthalmic division of the trigeminal nerve is involved there is risk of corneal involvement and permanent scarring. Post-herpetic neuralgia occurs in up to 50% of elderly patients following resolution of the infectious process. Its incidence is reduced by use of acyclovir during the infective period. Once established neuralgia is

■ DENTAL RELEVANCE OF HERPES ZOSTER INFECTION

Oral lesions – distribution depends on branch of nerve affected (unilateral).

Pain – if V2 or V3 branches of trigeminal nerve involved may be misdiagnosed as toothache.

Post-herpetic neuralgia leads to chronic facial pain.



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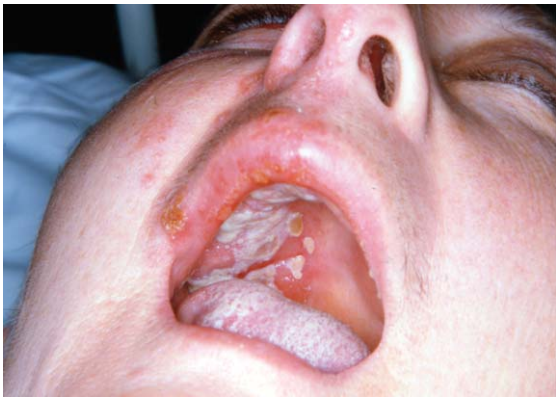


B

Fig. 11.5 Herpes zoster infection of the maxillary division of the right trigeminal nerve. Note the involvement of half of the palate.

difficult to treat; gabapentin and tricyclic antidepressants are effective in some cases.

Epstein–Barr virus (EBV)

The virus infects B lymphocytes and causes antibody production, although not necessarily against EBV.

Epidemiology. Occurs worldwide and most people become infected at sometime during their lives.

Primary infection. Infectious mononucleosis/glandular fever ('kissing disease').

Symptoms:

- Fever
- Malaise
- Lymphadenopathy
- Intra-oral petechial rash at the junction of the hard and soft palate
- White exudate over tonsils
- Pharyngeal swelling.

The duration of the fever varies, from a few weeks to several months.

Diagnosis. Clinical, Monospot (Paul–Bunnell) test and PCR (polymerase chain reaction).

Management. Symptomatic.

Other conditions caused by EBV:

- Hairy leukoplakia, on the lateral borders of the tongue
- Burkitt's lymphoma seen in African children
- Nasopharyngeal carcinoma in the Far East.

■ DENTAL RELEVANCE OF EBV INFECTION

- Cervical lymphadenopathy
- Creamy exudate over pharynx.

Human immunodeficiency virus (HIV)

The virus infects CD4 lymphocytes, causing severe deficiency of T helper cells resulting in immunodeficiency, which in the absence of treatment, leads to death by opportunistic infection.

Epidemiology. Over 30 million adults are currently infected with HIV worldwide, with 53000 cases in the UK.

Spread is by exchange of virus-infected body fluids. In Africa this is mainly via heterosexual intercourse followed by vertical transmission to infants. In the developed world it is mainly via homosexual intercourse and sharing of infected intravenous drug-using equipment. Risk from blood transfusions has been reduced by screening donors and treatment of donated blood.

Clinical features. Following infection there are a number of stages in the progression of HIV:

- primary HIV infection (acute HIV infection) 6–8 weeks
- seroconversion
- clinical latent period with or without persistent generalised lymphadenopathy (PGL) 15 months to 18 years
- early symptomatic HIV infection
- AIDS (CD4 cell count below 200/mm^3)
- advanced HIV infection characterised by a CD4 cell count below 50/mm^3.

Disease progression is monitored using a combination of clinical features, CD4 count and viral load.

Management involves a multidisciplinary team approach with:

- prevention of transmission by education
- counselling
- prevention of opportunistic infections
- early detection and treatment of neoplasms
- antiretroviral therapy.

Treatment is by combination antiretroviral therapy to avoid resistance:

Asymptomatic HIV

- CD4 >350 mm^3 no treatment
- CD4 200–350 mm^3 consider treatment
- CD4 <200 mm^3 treat immediately

Symptomatic HIV and AIDS

- Treat immediately.

In *needlestick injury* the chance of seroconversion is about 4:1000. This can be reduced by 80% if post-exposure

■ DENTAL RELEVANCE OF HIV INFECTION

Consider PEP (post-exposure prophylaxis) for needlestick incidents from high-risk patients:

1. HIV + ve
2. High-risk behaviour.

Universal cross-infection control measures

Patient more prone to infections

Oral manifestations include:

- *Candida*
- Hairy leukoplakia of tongue
- Kaposi sarcoma
- Aggressive periodontal disease
- Oral papilloma
- Squamous cell carcinoma
- Parotitis
- Cervical lymphadenopathy
- Lymphoma.

prophylaxis (PEP) is given within the first hour; the benefit decreases with time.

Rubivirus

German measles/rubella

Spread. Droplet infection.

Incubation period: 2–3 weeks.

Symptoms. Sore throat, conjunctivitis, fever, macular rash appears on day 3 which coalesces to form a 'blush' which fades in about 5 days, cervical lymphadenopathy and arthralgia.

Diagnosis. Usually clinical.

Management. Immunisation of children as part of MMR. Treatment of the infection is supportive – bed rest and analgesics. Children must be kept off school for 5 days following the onset of the rash.

Complications. If infection occurs within the first 4 months of pregnancy high risk of congenital defects. Thrombocytopenic purpura and encephalitis.

■ DENTAL RELEVANCE OF GERMAN MEASLES

- Enlarged posterior cervical lymph nodes
- Pharyngitis
- Facial rash.

Paramyxovirus

Measles

Epidemiology. Common childhood infection.

Spread. Droplet, childhood infection.

Incubation period: 10–14 days.

Symptoms. URTI, irritability, fever and febrile convulsions, conjunctivitis, Koplik spots (rash in buccal mucosa), maculopapular skin rash appears after 3–4 days behind ears and spreads to face and trunk; by the time the rash reaches the legs the fever has started to resolve.

Diagnosis. Clinical, but it is an uncommon condition so laboratory tests may be used.

Management. Prevention by immunisation of children aged 12–15 months by live attenuated vaccine (usually as part of MMR). Treatment of the infection is supportive with bed rest, analgesics and fluids.

■ DENTAL RELEVANCE OF MEASLES

- Koplik spots in buccal mucosa
- Pharyngitis
- Facial rash.

Mumps

Epidemiology. Common childhood infection.

Spread. Droplet spread, usually children affected.

Incubation period: 2–3 weeks.

Symptoms. Affects exocrine glands, commonly resulting in tender bilateral parotid swelling (Fig. 11.6). Orchitits (more common in adult men), pancreatitis, mastoiditis and

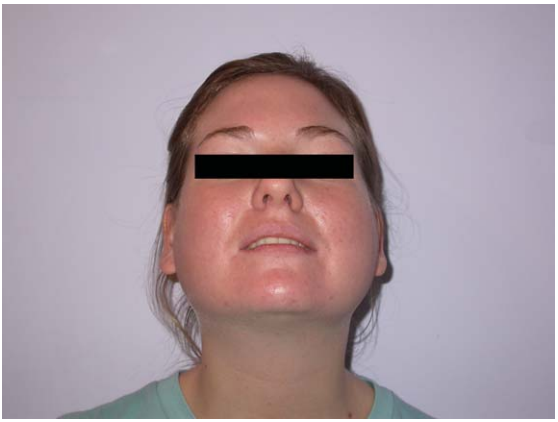


Fig. 11.6 Bilateral parotid swellings in mumps.

■ DENTAL RELEVANCE OF MUMPS

Enlarged and tender parotid glands.

oophoritis can occur. Meningitis/meningoencephalitits, and arthritis are rare.

Diagnosis. Usually clinical.

Management. Prevention by immunisation of children aged 12–15 months with a live attenuated vaccine (usually as part of MMR). Treatment is supportive, analgesics, bed rest and fluids. In orchitis, steroids can be used to reduce swelling and associated pain.

Fungal infections

Candidal infections

Candida albicans is a normal commensal of skin and mucosa. Infection is usually opportunistic following a predisposing local or systemic cause, e.g. denture wearing, dry mouth, antibiotic, steroid or cytotoxic therapy, immunosuppression, iron and folate deficiency and diabetes.

Symptoms. Depending on the area infected, there is often reddened mucosa or skin with creamy exudates. Infection of the palate: see Figure 11.7.

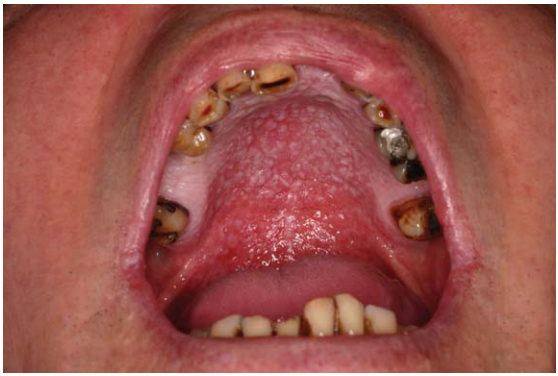


Fig. 11.7 Severe candidal infection of the palate with angular cheilitis in a poorly controlled diabetic patient.

■ DENTAL RELEVANCE OF CANDIDA

- Oral cavity commonly infected
- Often a sign of underlying disease
- Causes angular cheilitis at commissure of lips.

Diagnosis. Smear from lesion will show yeasts.

Management. Treat predisposing cause and administer topical or systemic antifungals.

Prion infections

Bovine spongiform encephalopathy and new variant Creutzfeldt–Jakob disease (vCJD)

Epidemiology. There were 146 definite and probable cases of vCJD deaths up to November 2004 in the UK.

Spread. Consumption of cattle products that are infected with the causative agent of BSE. It is thought that this agent is a modified form of a normal cell-surface component known as prion protein, which is highly resistant to enzyme degradation.

■ DENTAL RELEVANCE OF CJD

Low risk of CJD transmission in dental practice.

Universal cross-infection measures must be used. Instrument decontamination is particularly important.

Symptoms. Sensory and psychiatric abnormalities in early stages, ataxia, neurological abnormalities, dementia as the disease progresses.

Diagnosis. Biopsy of brain tissue or autopsy.

Management. Supportive, as disease is fatal.

Useful websites

CJD advice www.dh.gov.uk/cjd/

The British HIV Association www.bhiva.org

12 ENT DISORDERS

Mark McGurk

Symptoms arising from dental disorders can overlap with diseases in the ear, nose and throat. This can lead to misdiagnosis and delay in treatment. The separation of pathology in the mouth from that in ear, nose and throat is somewhat artificial.

Overview

Minor disorders of the ear, nose and throat (ENT) are common. It is reputed that 25% of all attendances in general medical practice are for such complaints.

The character of common ENT complaints varies with age. In childhood the main problem arises from congestion of the oro/nasopharynx. This anatomical compartment is filled by hypertrophied and active lymphoid tissue. The congestion leads to tonsillitis and/or Eustachian tube dysfunction, glue ear with the attendant infection and obstructive hearing.

In adolescence there is a switch of symptoms to allergic condition of the nose. The days of chronic sinusitis or mastoiditis have passed. Middle age is not marked by a single symptom complex but a variety of complaints, none forming a dominant theme. In old age there is the inevitable appearance of presbycusis, vertigo and an increased risk of neoplasia.

Nose and paranasal sinuses

Epistaxis

The nose and paranasal sinuses are extremely well perfused and epistaxis can be a frightening experience. Childhood haemorrhage is different to that occurring in the elderly. In childhood the bleeding arises from 'Little's area' at the cordial end of the septum (see Fig. 12.1).

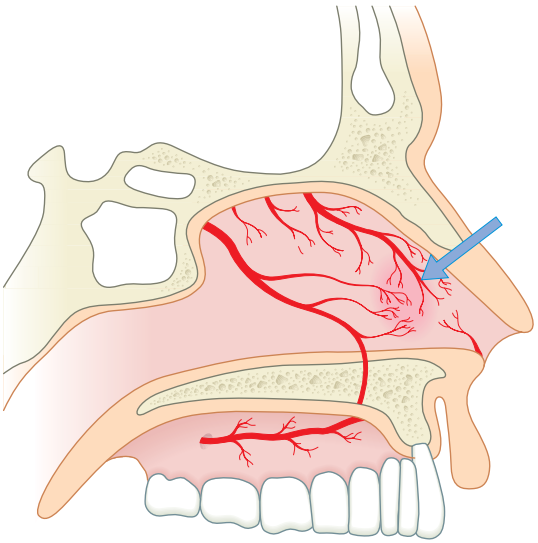


Fig. 12.1 Little's area indicated by the arrow is the common site for epistaxis in children.

Pinching the nose between the fingers with the head held forward resolves the immediate problem. In the elderly, haemorrhage arises from the rostral end of the septum and is associated with hypertension and thinning of the nasal mucosa. This area of the nose is well perfused by the ethmoidal and spheno-palatine arteries, the bleeding site is inaccessible and in addition the blood tends to flow into the throat which leads to coughing so aggravating the situation. The management of such a case is to pack the nose with cotton wool or ribbon gauze soaked in vasoconstrictor (cocaine (10%) or lidocaine/adrenaline (epinephrine) solution). The pack must be placed high in the nasal cavity. Epistaxis in children is a minor event but it is not uncommon to have adults admitted to hospital. (See Fig. 12.2.)

Allergic rhinitis

This condition tends to appear in adolescence and leads to oedema of the nasal mucosa with obstruction of the nose. Over time the patient develops a characteristic nasal pattern of speech. Swollen mucosa can be pendulous and

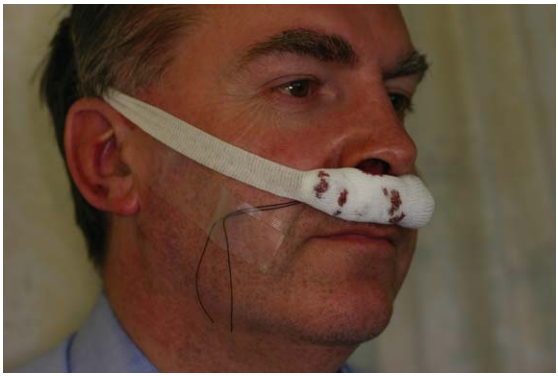


Fig. 12.2 A patient with nasal bolster and pack after a brisk epistaxis.

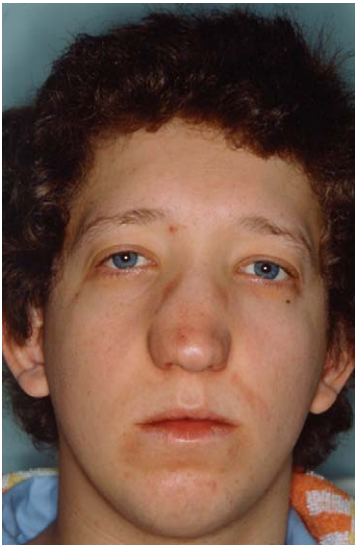


Fig. 12.3 Nasal deformity due to the presence of nasal polyps.

start to form polyps. These can be of such a size as to deform the nose (see Fig. 12.3).

These polyps normally arise from the ethmoidal air cells and first fill the upper compartment of the nose which contains the receptors for smell. Patients therefore

typically complain of loss of taste. Polyps can descend into the lower airway and obstruct the nasal air flow. Occasionally mucosal cysts can form in the maxillary antrum and on dental radiographs are seen as well-defined radiopaque spheres. An antral polyp can sometimes be washed out of the osteum to hang behind the soft palate as an 'antrochoanal polyp'. Most nasal polyps have no malignant potential but are prone to recur.

Chronic sinus infection

This is much less common today than 40 years ago and, if present, a dental cause should be excluded. This can have medico-legal implications if infection is due to an unobserved oro-antral fistula or retained dental root.

Acute sinusitis

This condition usually follows a viral infection. In its evolution sinusitis can masquerade as toothache in the upper molar teeth for the upper alveolar nerve serving these teeth runs through the antrum. Care should be taken to distinguish between sinusitis and dental decay to avoid unnecessary dental treatment. (See Fig. 12.4.)

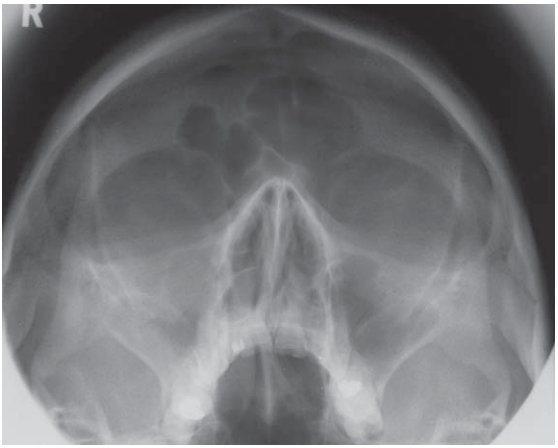


Fig. 12.4 An occipitomental radiograph of sinusitis. Note the opacity of the right sinus due to thickening of the lining mucosa.

■ **DENTAL RELEVANCE OF SINUS PROBLEMS**

An evolving sinusitis can present as an ill-defined toothache in the maxilla.

Chronic sinus infection may have a dental cause.

Cancer of the paranasal sinuses is rare, hidden within the recesses of the face, and is usually advanced when diagnosed. It can be heralded by vague, unrelenting and progressive pain.

Trauma

The nose occupies a prominent position in the face and it is a common site of trauma. The injury can be as innocent as a lively 2-year-old child inadvertently butting his mother when held in her arms. In most instances the injury is trivial but even a slight displacement of the bones can be obvious, although at first masked by oedema. The septum itself can buckle or fracture and occasionally the injury is complicated by a haematoma beneath the periosteum of the septum which can lead to infection and perforation. The diagnosis is made on clinical features and surgical repositioning of the bones can be undertaken up to 2 weeks post-injury. Late repair of serious nasal fractures is by way of rhinoplasty. Chemical or physical trauma to the septum can lead to septal perforation.

Malignant disease

Malignant disease of the nasal cavity and para-nasal sinuses is uncommon, constituting only 3% of all head and neck cancers. The two most common sites are the nasal cavity (32%) and the maxillary sinus (58%).

Tumours of the sinuses are hidden within bony cavities where they grow silently protected from prying eyes or fingers. Late presentation is the norm. The most frequent type of cancer is squamous cell carcinoma which occurs mainly in the maxillary antrum. Adenocarcinoma arises mainly from the minor mucus secreting cells that line the ethmoidal sinuses. Finally, the upper echelon of the nose can be affected by rare neural tumours. An important radiological feature suggestive of antral cancer is unilateral opacity of the maxillary sinus. Treatment is surgically based supported by radiotherapy.

The ear

Congenital abnormalities and developmental disorders of the ear commonly involve the adjacent structures such as the temporomandibular joint, mandibular ramus and the zygomatic complex. Management of ear deformities has been transformed in the last decade. The helix of the ear can be rebuilt from local tissue and external auditory meatus re-fashioned if a normal ossicular system is present in the middle ear. If only the auditory nerve is functional then bone anchored hearing aids can restore the patient's ability to hear. The implants are sited in the temporal bone and sound energy is conducted through the bone to the cochlear. Cochlear implants stimulate the auditory nerve directly but the perceived sound has much less clarity.

Infections

The common disorder of childhood is glue ear. The condition is due to Eustachian tube dysfunction and the disorder resolves naturally as the child passes into adolescence. Mucus retention within the middle ear (glue ear) reduces hearing by 10–20 decibels. If necessary, the mucus can be aspirated and patency of the cavity maintained with a grommet. If the symptoms persist treatment can be augmented by adenoidectomy.

Middle ear infection (otitis media)

This can result in perforation of the ear drum. Repeat infections lead to scarring and restriction in movement of the ossicular chain or, on occasion, ischaemic necrosis of the ossicles themselves. If unchecked the situation results in permanent conductive hearing loss. A perforated ear drum opens a portal for bacterial contamination of the middle ear which is then vulnerable to repeat infection. The perforations can be closed surgically.

Otitis externa

This condition is common in adolescent patients and those in middle age. Eczema or dry scaly skin are predisposing factors for infection, as is self-induced trauma (ear cleaning, hearing aids, pencils and fingers). The ear is designed to be self-cleansing. The epithelium adjacent to the drum

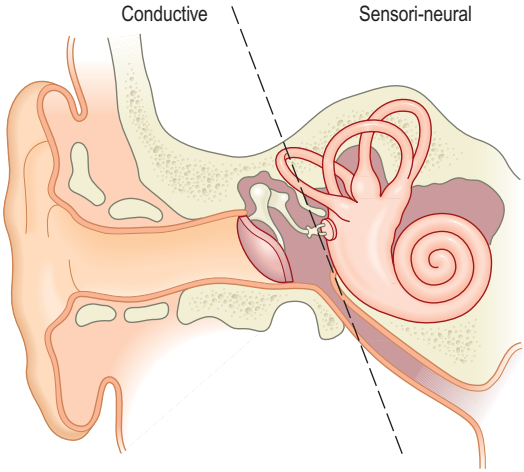


Fig. 12.5 The division between conductive and sensori-neural deafness.

migrates towards the osteum carrying wax and debris with it. Treatment is based on a combination of regular debridement (suction) and topical antibacterial or anti-fungal medication.

The hearing loss is categorised as *sensory neural* where the defect is attributable to malfunction of the auditory nerve or *conductive* where the sound energy is dampened in its passage from the eardrum to the cochlear (Fig. 12.5).

In the past (40 years ago) infections of the middle ear that extended into the mastoid air cells were encountered frequently (mastoiditis). The condition is now uncommon presumably due to improvement in personal hygiene and consequent alterations in bacterial flora. Historically these infections could have serious complications and at least one king of France (Francis II, 1560) died of a brain abscess secondary to ear infection.

Cholesteatomas

These are vaginations of epithelium and a loose dental analogy would be a keratocyst. The epithelial cavity accumulates debris and slowly enlarges to such a degree that

it can extent as far as the dura. Surgical excision can be difficult and may lead to loss of auditory and vestibular systems.

The temporomandibular joint (TMJ)

This traverses the boundaries of dentistry and otology. Swelling and effusion of the joints can be interpreted by patients as fullness of the ear and it is quite possible that some inflammatory proteins cross the divide between the joint and the ear cavity. Thus it is not uncommon for patients with temporomandibular joint dysfunction to present to ENT surgeons.

Vertigo

The semicircular canals are aligned in three planes and are important for the maintenance of balance. Vestibular receptors consist of banks of tiny hairs, each supporting a calcified cap within a fluid-filled tube. The fluid moves in continuity with the head. A complication of facial trauma is dislodgement of these calcified caps which then may move independently and collide with the hairs, resulting in positional vertigo. In old age, ischaemic changes in the microvasculature of the ear lead to vestibular nerve dysfunction. Eventually the vestibular system fails and in the elderly balance is maintained with eye contact.

Tinnitus

This is a neurological phenomenon not related to physical dysfunction of the auditory membrane. Most patients can overlook the ringing sensation. The noise cannot be eliminated but the strategy is to mask the perceived sound. Tinnitus should be distinguished from a bruit as the latter may signify a glomous tumour (vascular tumour originating from the jugular vein).

Malignant disease

Very rarely a patient will present with acute pain – almost neuralgic in origin – in the region of the temporomandibular joint for which no cause can be discerned. It may represent the first symptoms of an early adenocystic carcinoma in the parotid or nasopharyngeal area. These tumours are difficult to diagnose. The features that help to distinguish

■ DENTAL RELEVANCE OF EAR PROBLEMS

Patients with TMJ dysfunction sometimes present to the ENT surgeon with complaints of fullness in the ear.

Cancers of the mouth and throat produce referred pain to the ear. A complaint of a sore throat or dysphagia with unilateral earache is a cancer until proven otherwise.

this condition are unilateral, progressive and unrelenting pain. Such patients require detailed evaluation.

Another important pattern of symptoms, particularly in elderly patients, is the combination of a sore throat and pain radiating to one ear. This is indicative of a cancer (squamous cell carcinoma) involving the oropharynx or larynx and is due to referred pain from the glossopharyngeal nerve.

The cancer that most affects the ear is the basal or squamous cell carcinoma of the helix. Squamous carcinoma of the middle ear is rare and it normally follows years of chronic ear infection.

The pharynx, larynx obstruction and sleep apnoea

The common cause of mild airway obstruction is enlarged tonsils.

The threshold for tonsillectomy is much higher today than 20 years ago for it is appreciated that tonsils atrophy with age. Large pitted tonsils can collect debris and may be responsible for halitosis.

Severe obstructions can also occur in children secondary to micrognathia or TMJ ankylosis. This is because the mandible does not develop sufficiently to draw the tongue forward and as a result the growing tongue obstructs the airway.

In adults minor upper airway obstruction is evidenced by snoring. The condition is linked to obesity and other contributing factors such as alcohol (relaxant), posture and micronasia. Sleep apnoea, if severe, can lead to hypertension, fatigue and poor mental performance.

Some children are susceptible to papilloma virus which results in multiple laryngeal polyps that can obstruct the

airway. The condition can be sufficiently severe to require a tracheostomy. New papilloma virus vaccines may control this condition. With respect to a tracheostomy or open tracheostome following laryngectomy, it should be remembered that a person cannot strain without closing the glottis (vocal cords). Constipation can therefore be a real problem in patients with tracheostomies. Morphine-based analgesia produces constipation!

Malignant disease

Cancers in the pharynx can be easily overlooked. In the oral cavity squamous cell carcinomas produce a visible ulcer. But the pharynx is covered by a layer of lymphoid tissue (Waltyer's ring) and squamous cell carcinoma tends to burrow beneath this layer and remain hidden from view. The sites at risk are tonsil and base of tongue. Tumours can be diagnosed from symptoms of unremitting progressive pain usually radiating to ear and possible evidence of asymmetrical enlargement of the tonsils or tongue base. Palpation is the most valuable diagnostic tool as it frequently reveals a hard mass within the substance of the tissues. Tumours of the larynx tend to be identified early because they interfere with the tone of the voice. A persistent hoarse voice in a middle-aged smoker requires investigation.

■ DENTAL RELEVANCE OF MALIGNANT DISEASE

Malignant disease in the oropharynx (tonsil and base of tongue) is frequently hidden so diagnosis can be delayed. This is a common source of litigation.

Unilateral earache, progressive, unremitting pain are symptoms indicative of cancer. Palpate the tongue or tonsil for the presence of a hard mass.

13 IMMUNOLOGICALLY MEDIATED DISEASES

Georgina Burke

The immune system has evolved to protect individuals from infections. However, the system of immune recognition and response can break down, leading to the following problems:

Autoimmune disease occurs when lymphocytes react against self antigens (i.e. antigens on the individual's own tissues).

Immunodeficiency occurs when the immune system is unable to make an adequate immune response to control pathogens.

Hypersensitivity occurs when there is an exaggerated response of the immune system to an antigen.

Autoimmune diseases

Autoimmune disease may be limited to one organ of the body (e.g. pernicious anaemia) or involve widely distributed antigens (e.g. systemic lupus erythematosus).

Aetiology

This is probably multifactorial, although genetic factors are thought to play a role; autoimmune diseases often run in families and are more common in women. Types of autoimmunity are shown in Figure 13.1, and the spectrum of autoimmune diseases is given in the box on p. 207.

Treatment

Treatment varies depending on the autoimmune disease involved but often involves immunosuppression with corticosteroids and other immunosuppressant agents. While on immunosuppression live or attenuated vaccines should be avoided. Also, Herpes zoster infections may be severe

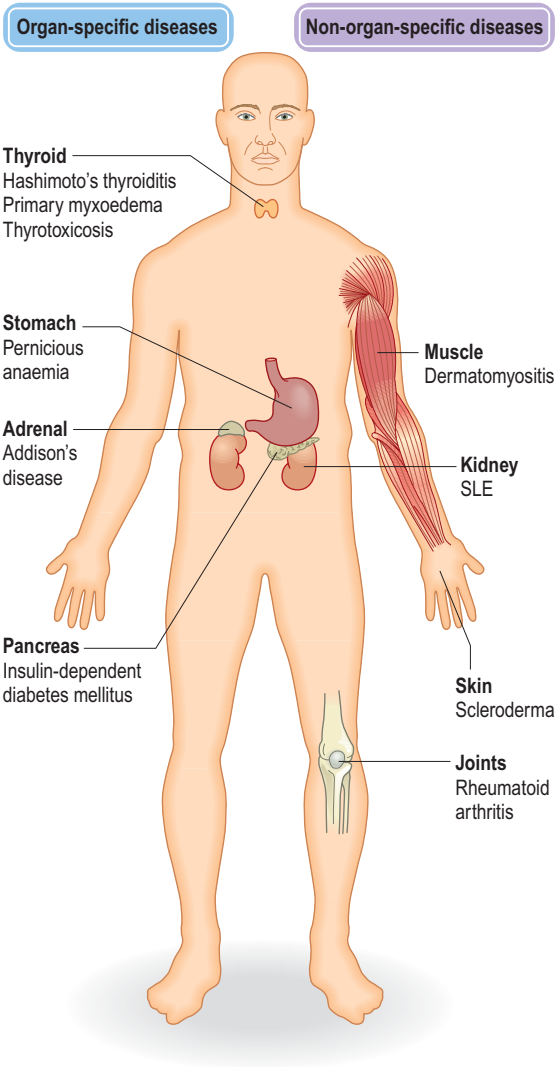


Fig. 13.1 Spectrum of autoimmune disease.

and so patients should seek immediate medical attention after contact with individuals with chickenpox or shingles. Complications of corticosteroid treatment are shown in the box p. 206, and a steroid treatment card is shown in Figure 13.2.

■ SPECTRUM OF AUTOIMMUNE DISEASES

Organ-specific diseases

- Thyroid: Hashimoto's thyroiditis, Graves' disease (see **p. 99**)
- Stomach: pernicious anaemia (see **p. 142**)
- Adrenal: Addison's disease (see **p. 112**)
- Pancreas: insulin-dependent diabetes mellitus (see **p. 115**)
- Neuromuscular junction: myasthenia gravis.

Non-organ-specific diseases

- Muscle: dermatomyositis
- Kidney: SLE
- Skin: systemic sclerosis, Behçet's disease
- Joints: rheumatoid arthritis (see **p. 119**).

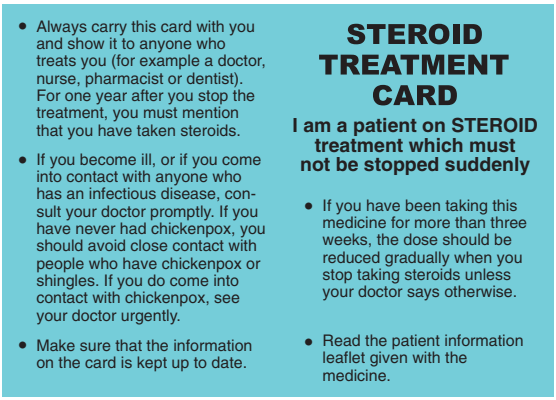


Fig. 13.2 Steroid treatment card.

Systemic lupus erythematosus (SLE)

SLE is a multisystem disease with a wide spectrum of clinical manifestations.

Epidemiology

SLE is predominantly a disease of young women, particularly of Afro-Caribbean descent, and has a prevalence of 1/1000–6000.

Pathogenesis

Anti-DNA antibodies are characteristic of SLE but other antibodies directed against the cell nucleus and immune complexes are found. Drug-induced lupus may occur in

■ COMPLICATIONS OF CORTICOSTEROID TREATMENT

Short-term complications

- Mood disturbance
- Insomnia.

Long-term complications

- Weight gain
- Cushing's syndrome (moon face, striae, acne, see **p. 112**)
- Cataracts
- Hypertension
- Osteoporosis and avascular necrosis
- Muscle wasting
- Diabetes
- Peptic ulceration
- Delayed wound healing
- Increased susceptibility to infections.

Marked adrenal suppression occurs when doses >30 mg/day are taken for more than 2–3 weeks. It is therefore vital than patients do not abruptly stop treatment otherwise they will develop symptoms of acute adrenal insufficiency that may be life-threatening (see **p. 285**). In addition, patients may be unable to initiate a sufficient adrenocortical response with illness or surgery and may require additional steroid cover. For this reason patients are advised to carry a 'steroid treatment card'.

slow acetylators on taking hydralazine, procainamide, penicillamine and some anticonvulsants and antibiotics.

Clinical features

These are illustrated in Figure 13.3, and mucosal ulceration is shown in Figure 13.4.

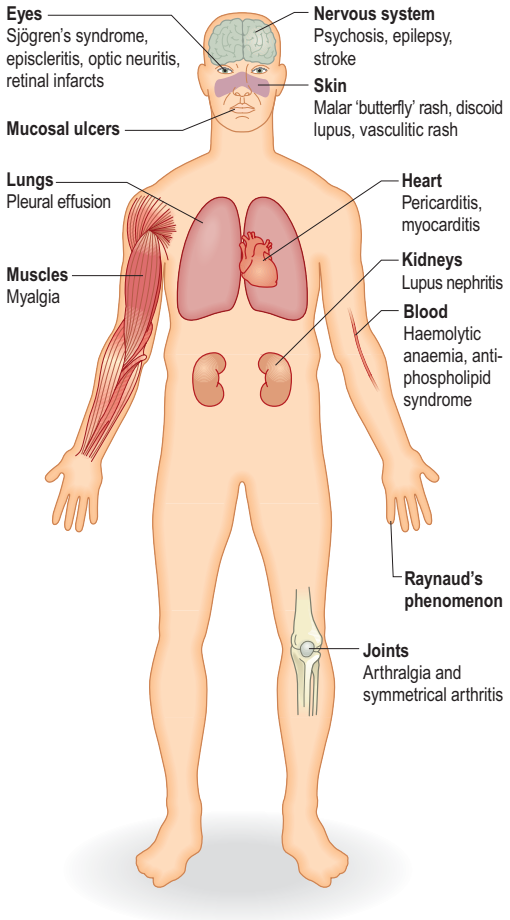


Fig. 13.3 Clinical features of SLE.

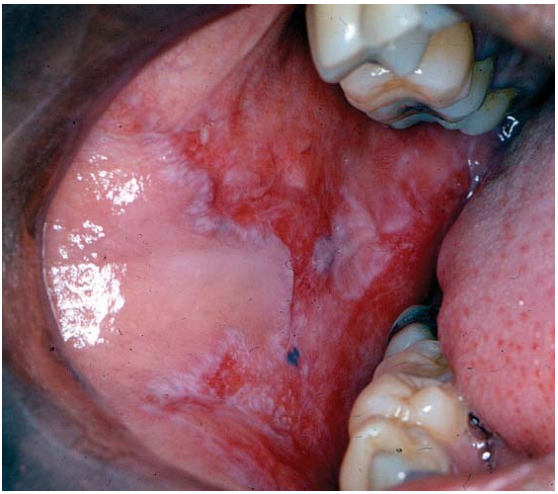


Fig. 13.4 SLE is a cause of mucosal ulceration.

The antiphospholipid syndrome is characterised by recurrent abortions, venous and arterial thromboses and thrombocytopenia and is associated with anti-cardiolipin antibody and lupus anticoagulant.

Prognosis

Complete remission is rare but 10-year survival is 90%. Infection and renal failure account for most of the mortality.

Behçet's disease

Behçet's disease is a multisystem disorder that may present with oral ulceration.

Epidemiology

Behçet's disease affects young adults with a prevalence of 1/500 000 but it is more common in Turks and Japanese.

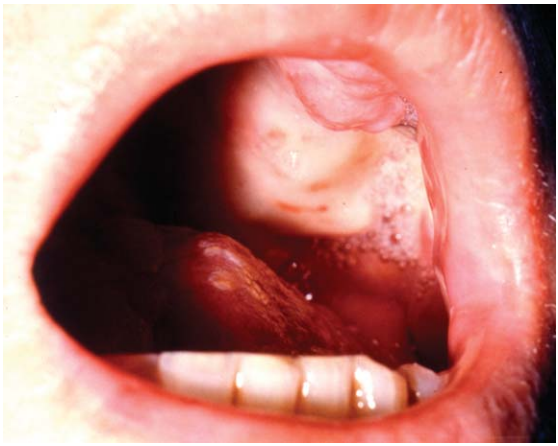


Fig. 13.5 Oral ulceration in Behçet's disease.

Pathogenesis
This is not entirely clear but it is associated with vasculitis, autoantibodies to the oral mucous membrane and immune complexes.

Clinical features
Behçet's disease presents mainly with recurrent orogenital ulceration (Fig. 13.5) and uveitis.

Treatment
Symptomatic treatment includes analgesia and NSAIDs but immunosuppression is required for severe disease. Colchicine may be useful for oral ulceration.

Systemic sclerosis

Systemic sclerosis is associated with inflammation, fibrosis and vascular damage to the skin and internal organs.

Epidemiology
This is a rare disease that usually affects women aged 30–50 years.

■ DENTAL RELEVANCE OF AUTOIMMUNE DISEASES

Autoimmune disease may present first in the mouth (e.g. oral ulcers in Behçet's disease).

Patients may be taking corticosteroids and thus prone to delayed healing and increased susceptibility to infection. Furthermore, additional steroid cover may be required for surgical procedures under general anaesthesia.

Pathogenesis

Systemic sclerosis is characterised by fibrosis (scleroderma); thickening of the skin with tethering to subcutaneous tissue. Autoantibodies, especially ANA are present.

Clinical features

Raynaud's phenomenon and skin involvement (scleroderma, telangiectasia, capillary nailbed changes) are commonly found. The gastrointestinal tract, heart, lungs and kidneys may also be involved.

Treatment

This is supportive as no drug has been shown to halt progression of the disease.

The dental relevance of autoimmune diseases is shown in the box p. 211.

Immunodeficiency

Failure of the immune system often results in increased susceptibility to infections.

Immunodeficiencies are classified as primary (congenital) or secondary (acquired, e.g. HIV see **p. 186**). They can arise from deficiencies in lymphoid cells, phagocytes and complement.

Patients with immunodeficiency are prone to opportunistic infections from pathogens that normally do not cause problems in immunocompetent individuals (see box p. 211).

Deficiencies of other cells of the immune response (e.g. B cells, complement) are associated with staphylococcal and streptococcal infections.

■ **OPPORTUNISTIC INFECTIONS IN IMMUNOCOMPROMISED PATIENTS ARE COMMONLY ASSOCIATED WITH T-CELL IMMUNODEFICIENCY**

Fungal infections, e.g. *Candida albicans* infecting the mucous membranes, hair, skin and nails

Parasitic infections, e.g. *Pneumocystis carinii* producing pneumonia or *Toxoplasma gondii* producing CNS cysts

Viral infections, e.g. Herpes zoster and Herpes simplex causing severe localised ulceration; CMV may affect many organs, including lungs (pneumonia) and brain (encephalitis)

Bacterial infections, e.g. mycobacterial infection of internal organs (lung, CNS).

Hypersensitivity

Hypersensitivity occurs when there is an exaggerated immune response to an antigen. This results in local tissue injury and/or systemic symptoms, including shock or death.

There are four major types of hypersensitivity; one or several types may occur with an immune-mediated disease.

Type I hypersensitivity (anaphylaxis)

This occurs when an allergen interacts with a specific IgE antibody that is present on mast cells. Degranulation of the mast cell releases histamine and other vasoactive mediators that activate an inflammatory response.

Type 1 hypersensitivity reactions usually occur within minutes of exposure to the antigen and may cause mild symptoms or anaphylactic shock. Examples include allergic rhinitis (hayfever), allergic asthma and food allergy.

Type II hypersensitivity (antibody-dependent)

Here, cell-bound antigens interact with specific IgG or IgM antibodies to form in situ complexes that activate complement, leading to cell lysis and death. These reactions

take many hours to develop and occur in transfusion reactions and some autoimmune diseases (e.g. myasthenia gravis).

Type III hypersensitivity (immune complex)

In type III hypersensitivity, soluble immune complexes form in the circulation and are deposited in various tissues where they activate complement. Systemic lupus erythematosus is an example of type III hypersensitivity.

Type IV hypersensitivity (cell-mediated)

This is initiated by the interaction of T cells with antigen when it is complexed with MHC molecules. T-cell-mediated immunity arises over several days and occurs with granulomatous disease (e.g. tuberculosis), contact dermatitis, Hashimoto's thyroiditis and organ allograft rejection.

Latex allergy

Epidemiology

Latex allergy is common and reports suggest that it affects 1–6% of the population. The risk increases with latex exposure and so healthcare workers are at particular risk. (See Fig. 13.6.)

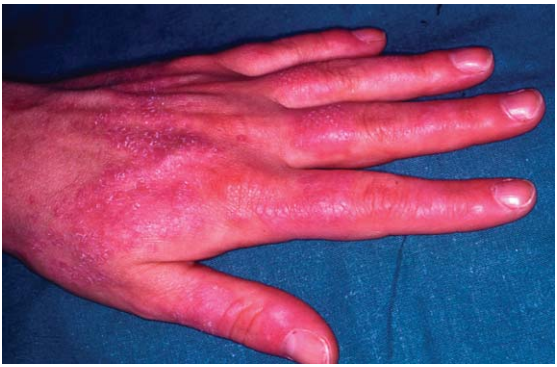


Fig. 13.6 Contact dermatitis as a result of wearing latex gloves.

Aetiology

Latex is a complex product of the Brazilian rubber tree *Hevea brasiliensis*, which is grown mainly in the Pacific Rim countries. It can cause many types of reaction:

1. Irritant contact dermatitis is the most common type of latex allergy, resulting in dry, itchy, irritated areas of skin.
2. Type IV hypersensitivity results from exposure to chemicals added to latex during harvesting, processing or manufacturing. A rash usually begins 24 to 48 hours after contact, and may progress to urticarial skin blisters.
3. Type I hypersensitivity reactions are potentially life-threatening and so patients must avoid all contact with latex.

All patients should be screened for latex allergy. The following questions can be useful:

'Have you experienced hives, wheezing, rashes, coughing, or difficulty in breathing when handling rubber items like balloons?'

'Have you experienced any of these symptoms after contact with medical or dental products like rubber gloves?'

'Have you ever worked in a health care setting? In the rubber industry?'

Diagnosis

Diagnosis is based on a history of latex exposure and reactions, physical signs of latex hypersensitivity, and a positive blood test (RAST) or skin test for IgE antibodies to latex allergens. This is performed under controlled conditions as even a skin test can provoke an anaphylactic reaction.

Treatment

This primarily involves avoiding contact with latex. Thus, treatment modification may be required for patients with latex allergy. Anaphylactic shock is a medical emergency; treatment is covered on **page 284**.

Dental items that may contain latex are listed below and the following box gives the relevance of latex allergy to dentistry.

■ DENTAL ITEMS THAT MAY CONTAIN LATEX

- Anaesthetic cartridge
- Bite block
- Blood pressure cuff
- Endodontic stops
- Gloves
- Hoses
- Instrument bands
- Mixing bowls
- Nitrous oxide masks and hoses
- Orthodontic bands and elastics
- Polishing wheels and points
- Rubber dams.

■ DENTAL RELEVANCE OF LATEX ALLERGY

Latex allergy is a common and potentially life-threatening problem and should be screened for in all patients.

The risk of latex allergy is related to exposure: dentists should wear powder-free, latex-free gloves to minimise exposure.

Patients with latex allergy must avoid all contact with latex. They should be the first patient on the list (low 'latex dust').

14 PSYCHIATRIC DISORDERS

Matthew Smith

Classification and history

Psychiatric illness has a huge impact on society due to its prevalence and consequences. Approximately 25% of all hospital beds are used for psychiatric treatment. The prevalence of psychiatric illness in the community is about 35%. Dentists are likely to encounter a patient with a psychiatric illness every working day.

Classification

Psychiatric illnesses are classified according to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM IV). Those with the greatest impact on dentistry are:

- anxiety disorders
- mood disorders
- somatoform disorders
- schizophrenia and other psychotic disorders
- substance-related disorders
- eating disorders.

In most psychiatric disorders there is a complex interrelationship between biological, social and psychiatric elements which can be difficult to interpret.

There are three main groups of factors:

1. *Predisposing* determine an individual's vulnerability to psychiatric problems, e.g. personality, chronic illness etc.
2. *Precipitating* occur shortly before the onset of the illness, e.g. a stressful life event.
3. *Perpetuating* maintain the process once it has started, e.g. lack of social support.

History

History is taken in the same way as any other condition but there is more emphasis on the patient's appearance and behaviour, past psychiatric problems, social situation, family and personal history. The routine medical history and examination must not be omitted because there may be a physical cause for the problem which can easily be overlooked.

Anxiety disorders

Anxiety is a normal response to a threat or an unpredicted change in one's environment. It is a non-specific symptom and occurs in a wide range of normal situations as well as in psychiatric illness. A patient with anxiety disorder is overwhelmed by their anxiety. Those with a phobia experience extreme anxiety, but only in certain circumstances.

Epidemiology

Mood and anxiety disorders are said to affect 1 in 6 at some time and cost the UK economy in excess of £10 billion pa.

Aetiology

This is multifactorial. There is often a precipitating life event superimposed on a genetic predisposition. Life events may be positive or negative, e.g. a marriage or bereavement. Tests should be carried out to eliminate organic causes as anxiety may be the manifestation of a disease process, e.g. hyperthyroidism or hypoglycaemia.

Clinical features of anxiety

The clinical features are divided into two groups, physical and psychological:

Physical

- Tachycardia and chest tightness
- Dizziness and hyperventilation
- Diarrhoea
- Dry mouth and bruxism
- Myofacial pain as a result of jaw clenching, posturing or nail biting
- Swallowing difficulty – 'globus hystericus'.

Psychological

- Aggression
- Lack of concentration and poor memory
- Reduced pain threshold
- Exaggerated reaction to stimuli
- Sleep loss.

Diagnosis

Diagnosis is made from the history and examination after investigating for any possible organic causes.

Types of anxiety disorder often encountered in dental practice include:

1. *Phobias.* These are due to irrational fear of an object or situation leading to avoidance behaviour. Phobia of dental treatment is relatively common and may cause the patient to avoid treatment and demand general anaesthesia on every occasion.
2. *Panic disorder,* which is characterized by brief episodes of intense anxiety or panic. Panic attacks may be precipitated by dental treatment and lead to hyperventilation and loss of control (see Fig. 14.1).

Fig. 14.1 Re-breathing from a paper bag can be useful when dealing with hyperventilation during a panic attack. This prevents the patient from becoming alkalotic and developing muscular spasm.

■ DENTAL RELEVANCE OF ANXIETY

An aggressive or unfriendly patient may just be anxious.

A sympathetic and empathetic approach to anxious patients is essential.

Remember that sweating and tachycardia may be due to hypoglycaemia in a diabetic patient, for example, and not anxiety.

An up-to-date drug history is an imperative. Check all interactions in the BNF before treatment.

Dry mouth may be explained by anxiety or the drugs used for therapy.

3. *Generalised anxiety disorder*, which is a state of persistent anxiety not due to a specific situation or object.

Treatment

In the dental setting a sympathetic ear, empathy, reassurance and giving the patient some control ('put your hand up if you need me to stop') are very useful when treating anxious patients. Very anxious and phobic patients may benefit from sedation or other relaxation techniques to facilitate dental treatment.

More severe cases require treatment under the care of a psychiatrist. Therapies include cognitive behavioural therapy, counselling and medical therapy (β-blockers, antidepressants, minor tranquillisers).

Mood disorders

There are two broad groups of mood disorder; depressive and bipolar. In the former there is sustained depression of mood that is accompanied by various features such as sleeplessness, loss of appetite and suicidal thoughts. In the latter there are alternating episodes of depression and euphoria.

Epidemiology

Every year in the UK about 10% of the population will experience clinical depressive illness; 70% are female.

Onset is usually in the late 20s to mid-30s. An increased incidence in first-degree relatives indicates a genetic component.

Aetiology

Classically described as 'reactive' when it is triggered by a life event(s) such as bereavement, promotion etc. or 'endogenous' when there is a predisposition to the illness without an obvious trigger. Depression may also be secondary to a pathological process – for example, an endocrine abnormality or a side-effect of certain drugs, such as steroids.

Clinical features

Clinical features can be divided into two groups:

Biological

- Appetite disturbance (most commonly loss of appetite)
- Weight disturbance (most commonly weight loss)
- Early morning waking
- Decreased energy
- Loss of concentration
- Reduced sex drive.

Psychological

- Persistently depressed mood
- Anhedonia (loss of pleasure)
- Feelings of guilt, worthlessness and self blame
- Recurrent thoughts of death (suicidal ideation).

It is common to experience some or all of the above features during normal life; however, to be classified as depression they must be persistent (>2 weeks) and pervasive.

There is an association with 'atypical syndromes' such as IBS (irritable bowel syndrome), BMS (burning mouth syndrome), ME (myalgic encephalomyelitis – more accurately known as chronic fatigue syndrome), TMJ PDS (TMJ pain dysfunction syndrome) and fibromyalgia.

Diagnosis

This is based on history and must exclude organic causes such as hypothyroidism or substance abuse.

Treatment

Many studies show that cognitive behavioural therapy, psychotherapy, exercise and various types of counselling are as effective as the antidepressant drugs that are often prescribed (Fig. 14.2).

Electroconvulsive (ECT) therapy can produce a rapid and life-altering improvement in severe depression where there is a high risk of suicide.

■ DENTAL RELEVANCE OF MOOD DISORDERS

Mood disorders affect 10% of the population.

The dental profession is a high-risk group for suicide and mood disorder.

Check interactions and side-effects of prescribed drugs (many antidepressants cause dry mouth).

Adequate informed consent can be difficult to obtain.

Understanding and empathy is helpful when dealing with these patients.

There is an increased incidence of TMJ pain dysfunction syndrome and atypical facial pain in this group.

Fig. 14.2 Many patients are treated with selective serotonin re-uptake inhibitors (SSRIs). However, other forms of treatment are often as effective.

Somatoform disorders

Somatoform disorders are characterized by the presence of physical symptoms that cannot be adequately explained by a medical condition or substance taken. Patients express their inner conflicts and distress as physical symptoms.

Epidemiology

Between 3 and 13% of the population are affected. There is no age association. Females are affected more commonly than males.

Aetiology

Most patients are found to have an underlying anxiety or, more commonly, mood disorder.

Clinical features

Patients present with a variety of physical symptoms, including pain, paraesthesia, burning mouth, and temporomandibular joint (TMJ) dysfunction. The symptoms have often been present for years, cross normal anatomical boundaries and have not responded to previous physical treatment. There is frequently a history of multiple consultations and negative investigation results. In dysmorphic disorder the patient may feel that their teeth are deformed and demand unrealistic treatment for apparently normal dentition.

Atypical facial pain is a common manifestation in dental practice. It is characterised by a persistent dull pain that has changed little over time. It is more common in the upper jaw and crosses anatomical boundaries. Older women are most commonly affected, who may in extreme cases have all of their teeth, in the affected quadrant, extracted with no relief.

Treatment

Early identification of these patients and avoidance of multiple investigation and physical treatment is paramount. Referral to specialist clinics is often required where the underlying psychological disorder can be investigated and treated. Enabling the patient to make the link between their emotional and physical symptoms is the key to success.

■ DENTAL RELEVANCE OF SOMATOFORM DISORDERS

Somatoform disorders affect up to 13% of the population.

Attempt to rule out physical cause without excessive investigation.

Common dental presentations are:

- atypical facial pain
- temporomandibular joint dysfunction
- burning mouth
- paraesthesia.

Understanding and empathy is helpful when dealing with these patients.

Atypical facial pain often responds well to tricyclic antidepressant medication.

Schizophrenia

This is an incredibly complex and controversial area in medicine. Schizophrenia is a psychosis and is characterised by hallucinations (auditory, somatic and sometimes visual), delusions and disorders of thought. Schizophrenics find the world a place of bewildering and vivid personal significance.

Epidemiology

The annual prevalence is about 0.3%. The lifetime risk of developing schizophrenia is about 1%, but for first-degree relatives of schizophrenics this figure is about 12%. The age of onset is commonly between 15 and 45.

Aetiology

There are various theories and these include a genetic predisposition, a biochemical imbalance in the brain (dopamine), brain damage (perhaps by viruses and drugs such as marijuana) and structural brain abnormalities.

Clinical features

The key features are hallucinations, delusions and thought disorders.

Hallucination is a false sensory perception in the absence of an external stimulus. It may be:

- auditory
- visual
- tactile
- gustatory.

Delusion is an unshakable belief in something untrue which may be:

- persecutory, e.g. believing that people 'are out to get you'
- grandiose, e.g. believing that you have special powers
- reference, e.g. thinking that special messages are being transmitted to you from the television.

Thought disorder:

- thought insertion: 'Someone's putting thoughts into my head'
- thought broadcasting: 'Others can hear my thoughts'
- thought withdrawal: 'People are taking thoughts out of my head'
- feelings of passivity: 'Others can control me and make me do things'.

These features are all real to the patient and they are adamant in their belief. They are often said to lack insight. The patient may also hold paranoid beliefs and be socially withdrawn and difficult to communicate with. Some exhibit odd movements. Severe dental neglect in a schizophrenic is shown in Figure 14.3.

Diagnosis

The clinical features referred to above are called 'first-rank symptoms' after Schneider (1959). The identification of these symptoms is necessary for a diagnosis of schizophrenia to be made by a psychiatrist.

Treatment

If the patient is a danger to himself or others then hospital treatment is necessary.

Acute symptoms are treated with major tranquillisers that have an anti-psychotic/neuroleptic effect. Additional therapy includes psychotherapy and support to help the patient lead as normal a life as possible. Some 20% of schizophrenics are said to make a complete recovery.

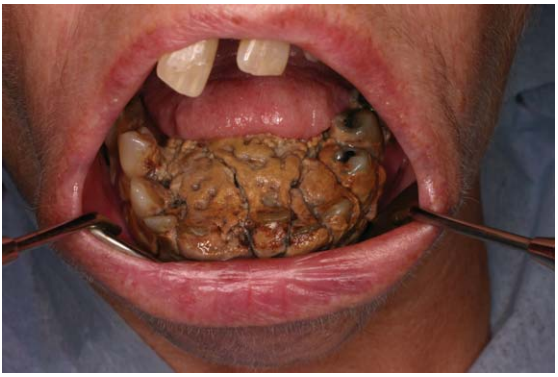


Fig. 14.3 Severe dental neglect in a schizophrenic patient.

■ DENTAL RELEVANCE OF SCHIZOPHRENIA

Most schizophrenics can be treated safely in general dental practice.

Drug treatment may lead to dry mouth and uncontrolled facial movements making treatment difficult, affecting speech and swallowing and denture wear problematic.

There is often severe dental neglect with increased caries and periodontal disease.

Adequate informed consent may be difficult or impossible to obtain in some circumstances.

Substance-related disorders

Alcohol-related problems

Abuse of alcohol is a significant issue in the UK. Problems range from social and economic to medical. Medical problems due to alcohol abuse are not necessarily associated with alcohol dependence.

Epidemiology. There are at least 500000 people in the UK with significant medical problems as a result of alcohol abuse. Alcohol abuse puts a huge burden on the National Health Service (NHS). A significant number of those attending Accident and Emergency (A&E) have injuries as a consequence of alcohol abuse. Heaviest drinking occurs in late teens and early 20s.

Aetiology. This is complex, involving genetic and environmental factors. Social and occupational pressures, personality and psychiatric problems are all involved.

Features of alcohol abuse

There are physical, social and psychological features:

Physical

- Liver disease: *cirrhosis, hepatitis*
- GI disease: *ulcers and varices, pancreatitis*
- CNS: *encephalopathy*
- Cardiac disease: *cardiomyopathy*
- Vitamin deficiencies
- Weight gain
- Repeated traumatic injury.

Social

- Deterioration of relationships
- Problems with work and finance
- Increased criminal activity
- Decreased social standing.

Psychological

- Mood disorders
- Anxiety disorders
- Deliberate self-harm
- Sexual problems
- Morbid jealousy
- Alcoholic psychosis.

Alcohol withdrawal can cause life-threatening delirium tremens characterised by fits, hallucinations, tachycardia and hypotension.

Figure 14.4 shows the effects of alcohol abuse.

Diagnosis. The CAGE questionnaire is helpful to identify those with an alcohol problem:

1. Have you felt you need to **C**ut down?
2. Ever been **A**nnoyed if others criticise your drinking habits?
3. Ever felt **G**uilty about your drinking?
4. Ever drink first thing in the morning – an **E**ye opener?

Two or more positive answers indicate problem drinking.

Blood test results including a high gamma-GT and macrocytosis may suggest alcohol abuse.

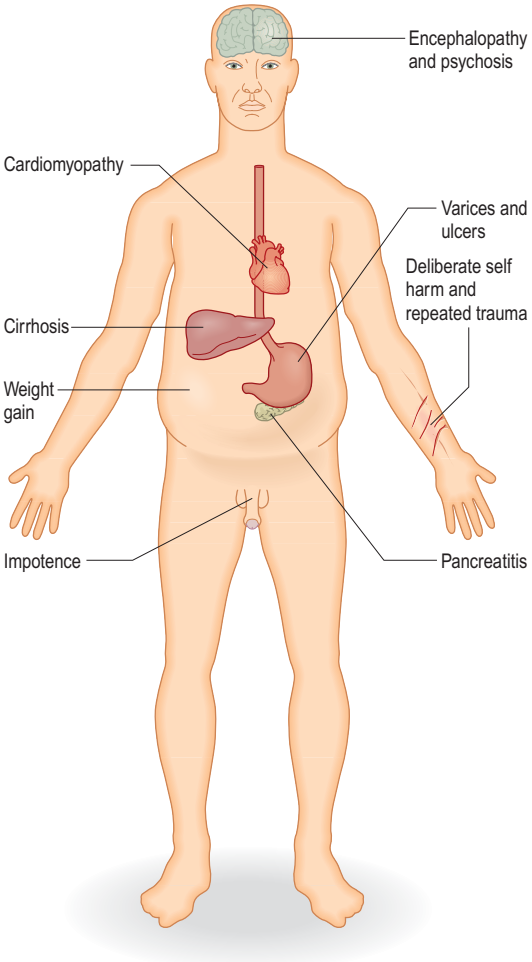


Fig. 14.4 Effects of alcohol abuse.

Treatment will work only if the patient is motivated to change and has adequate social support. Group therapy and counselling play an important role. Drugs such as disulfiram can be used to produce a very unpleasant reaction even if small quantities of alcohol are ingested. The

■ DENTAL RELEVANCE OF ALCOHOL-RELATED PROBLEMS

There is a tendency towards harmful drinking within the dental profession.

Increased risk of oral cancer, particularly if smoking as well.

Organ damage:

Liver disease:	drug metabolism, bleeding tendency
GIT:	gastritis and oesophagitis – avoid NSAIDs
Cardiac:	risk of arrhythmia with LA
CNS:	poor comprehension of treatment.

Poor compliance with treatment.

Adequate informed consent may be impossible to obtain from someone who is under the influence of alcohol.

Avoid metronidazole as it may cause a disulfiram reaction.

Repeated traumatic injuries to teeth are common.

Dangerously hypoglycaemic patients may appear to be drunk.

If you have concerns then seek advice from a physician before starting treatment.

patient must abstain from alcohol for the rest of their life and even alcohol-containing mouthwashes should be avoided.

Drug dependence

Drug dependence (addiction) is the compulsive use of a substance despite negative consequences. It implies that if the person stops taking the drug they suffer physical and/or psychological withdrawal symptoms and signs.

Commonly used drugs of dependence, apart from alcohol and nicotine, include:

Stimulants:	Cocaine and derivatives
	Amphetamines and derivatives
Hallucinogens:	Cannabis
	Solvents
	LSD
Narcotics:	Opioids such as morphine, heroin, pethidine and codeine
Hypnotics:	Barbiturates
	Benzodiazepines

■ DENTAL RELEVANCE OF DRUG DEPENDENCE

Tobacco and cannabis use are risk factors for head and neck cancers.

Drug users may be manipulative and demand repeated prescriptions of opioid analgesics.

Intravenous drug users have an increased incidence of endocarditis.

There is an increased risk of cross-infection due to HIV and hepatitis B and C among intravenous drug users.

Drug tolerance is common and patients may react unexpectedly to intravenous sedation.

Refer to hospital if you have concerns.

Epidemiology. Absolute figures of drug abusers and those who are drug dependent are unknown. Drug dependence and abuse (including alcohol, tobacco and cannabis) have huge social, health and economic effects.

Features of drug dependence depend on the drug involved and the abuser's reaction to it. Some drugs produce specific and characteristic signs and symptoms. There may be odd behaviour and criminal activity to fund the habit.

Physical signs can include needle tracks, nasal discharge and self-neglect.

Drug withdrawal is characterised by restlessness, aggression, hallucinations and other signs associated with mental illness.

Eating disorders

These are characterised by a severe disturbance in eating behaviour. The two main disorders are anorexia nervosa and bulimia nervosa.

Anorexia nervosa

In anorexia nervosa there is voluntary starvation and exercise stress with a refusal to maintain a minimum body weight. Individuals with anorexia nervosa often believe that they are overweight even though they are grossly underweight.

Epidemiology. Affects about 2% of schoolgirls in the UK. It is 20 times more prevalent in women than men. The peak incidence occurs in girls aged 10–19 years (younger in boys). The incidence of suicide is very high at about 10% of sufferers.

Aetiology. This is largely unknown, but there is a complex interplay of genetic, hormonal, psychological and social factors. Patients often have low self-esteem and a desire to gain control over their existence.

Definition. There are four main diagnostic criteria:
1. Avoidance of normal weight
2. Disturbance of weight perception
3. Fear and loathing of obesity – will diet even when very underweight
4. Amenorrhoea in women.

Clinical features. Dieting which often starts as an adolescent fad spirals out of control leading to:
- relentless dieting and exercise
- low body mass (>15% below expected)
- laxative and diuretic abuse
- habitual vomiting, amenorrhoea
- languno hair over body (very fine)
- low BP and a postural drop in BP
- depression and suicide.

Treatment must address the underlying psychological factors involving cognitive behavioural therapy and social support. In severe cases the patient may have to be admitted to hospital under the Mental Health Act for nourishment and intensive therapy.

■ DENTAL RELEVANCE OF ANOREXIA NERVOSA

Tooth erosion due to vomiting (especially palatal surfaces of upper incisors)

Salivary gland enlargement due to vomiting

The low body mass may require drug dose alterations

Dizziness when sitting up due to postural hypotension.

Bulimia nervosa

This is characterised by binge eating, self-induced vomiting and generally normal body weight or weight loss, not as severe as in anorexia.

Epidemiology. Bulimia is far more common than anorexia and has been known to reach almost epidemic proportions in female college students. It is thought to affect up to 10% of women in the age group 20–30 years.

Aetiology. Similar to anorexia with low self esteem and social pressure to be thin.

Clinical features:

- Binge eating and vomiting with generally normal weight
- Russell's sign of calluses on the back of the hand due to pushing fingers down the throat to induce vomiting.
- Laxative, diuretic, thyroxine and purgative abuse
- Fear of becoming overweight, but generally happy with their present weight.

Figure 14.5 shows a common feature of bulimia: erosion of teeth.

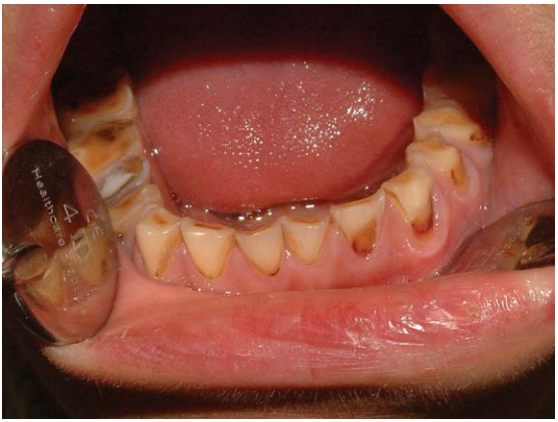


Fig. 14.5 Erosion of teeth due to repeated vomiting is a common feature in bulimia.

Treatment. Many bulimics keep their illness secret and eventually 'grow out of it'. Cognitive behavioural therapy can help those who wish to be helped.

■ DENTAL RELEVANCE OF BULIMIA NERVOSA

Tooth erosion due to vomiting.

15 EXTREMES OF AGE

Chris Sproat

The elderly

The UK population is ageing, with 16% over the age of 65. This group of patients will place increasing demands on the dental services as their numbers continue to increase and expectations of treatment rise. Maintenance of a healthy dentition into old age is important for a good quality of life and general wellbeing.

Epidemiology. In the last 30 years the number of people aged 65 or over has increased by 28%. This is mainly due to the number of births in the post-war years with a lesser contribution from decreased mortality.

As people live longer the gap between life expectancy and healthy life expectancy is increasing. This means that we will experience more years of poor health.

Ageing is not just a physical process: there are important social and psychological aspects which must be appreciated in order to deliver dental care appropriately.

To think of patients in terms of their biological age rather than the chronological age is often useful when assessing an individual's suitability for treatment.

General issues of ageing

Diseases. The most commonly reported conditions among those aged 65 and over are heart and circulatory diseases, musculoskeletal ailments and respiratory conditions such as bronchitis and emphysema. Many patients have multiple problems which can have an additive deleterious effect on their general health.

Reduced physical reserve means that patients may not be able to cope with interventions that upset their normal physical balance and routine.

Healing and repair are generally reduced so that patients need increased healing time and careful observation after invasive procedures.

Polypharmacy (Fig. 15.1) can produce many problems due to oral side-effects of drugs and increased risk of drug interactions (see Table 15.1).

Fig. 15.1 Polypharmacy is common in the elderly and increases the risk of side-effects and interactions with dental medication.

Table 15.1
Oral side-effects of commonly prescribed drugs or class of drug.

Oral side-effect	*Drug/class*
Bleeding	NSAIDs warfarin, heparin
Increased infection risk	Immunosuppressants, steroids
Erythema multiforme	Antibiotics, carbamazepine, phenytoin
Gingival hyperplasia	Nifedipine, ciclosporin, phenytion
Lichenoid reactions	Antibiotics, diuretics, NSAIDs, β-blockers, gold salts, penicillamine
Oral ulceration	Cytotoxics
Altered taste	ACE inhibitors, inhalers, metronidazole
Dry mouth (xerostomia)	Anti-cholinergics, anti-depressants, phenothiazines, anti-parkinsonians, anti-histamines, anti-hypertensives, tranquillisers

Disability/impaired functional status is common, with 50% of those over 75 classified as disabled. This may affect access to services, ability for self care and ability to cope with the demands of treatment. If memory is impaired this will have an impact on consent for treatment and understanding/retention of the treatment options discussed.

Social circumstances need to be investigated as many elderly patients have little social support and may feel isolated. If living alone they may not be able to cope with recovery from surgical procedures for example.

Psychological. Decreased perceived need for treatment is a major problem in the over-65s, many of whom seek treatment only when they are in desperate need.

There is an increased level of anxiety among the elderly towards all aspects of treatment which leads to avoidance behaviour.

Motivation to seek help and maintain health is often reduced in the elderly. This is due to many factors, including accessibility, functional status, physical illness and psychiatric disorders.

The elderly have an increased incidence of psychiatric disease, particularly mood disorder, which may lead to reduced interest in self care and their desire to seek treatment.

Oral diseases in the elderly

There are a number of oral conditions which are more common in the elderly:

Tooth loss

One in four of the over-65 age group have lost all of their teeth. Despite this more patients are retaining some or all of their teeth into old age and therefore require more complex restorative treatment. Teeth that require extraction are likely to be heavily restored and more likely to require a surgical approach.

Periodontal disease

The incidence of periodontal disease increases with age; half of those over 55 have periodontal disease.

Oral cancer

There is an increased incidence of oral cancer with age, especially if there is a past history of smoking and excess alcohol consumption.

Dry mouth

This is most commonly due to side-effects of medications taken (see Table 15.1). Other causes include:

- salivary gland disease, e.g. secondary Sjögrens syndrome
- jaw irradiation for treatment of malignant disease
- dehydration.

Management

In order to treat elderly patients in an appropriate and safe manner it is important to:

- Appreciate the specific needs of this group of patients.
- Assess each individual in terms of their physical, social and psychological capability.
- Be aware of the oral manifestations of systemic diseases and the impact they may have on the treatment plan.
- Avoid interactions with and look for the oral side-effects of drugs taken. Remember that patients may have difficulty in swallowing tablets so be prepared to prescribe medicines in elixir forms.
- Take a multidisciplinary approach and liaise with other individuals responsible for each patients care, e.g. general medical practitioner, carer, physiotherapist, occupational therapist, family, etc.

■ SAFE DENTAL TREATMENT OF THE ELDERLY

Most elderly patients can be treated safely using local anaesthetic in dental practice.

To maximise safety:

- avoid sedation as there may be increased sensitivity to benzodiazepines and concurrent airway disease.
- be aware of the risks of polypharmacy.

Patients may have multiple medical problems so close liaison with the general medical practitioner or physician is often required.

- Be aware of accessibility issues, including:
  - transport difficulties
  - access to the surgery
  - communication problems.
- Place the safety of treatment first and seek advice or refer the patient if there is any concern about this issue.

The young

Children under the age of 16 make up 20% of the UK population and they present a number of unique management issues, from behavioural problems to suspected abuse, which a practising dentist will have to deal with on a daily basis.

Most children have erupted teeth from the age of 6 months and may present for dental treatment.

Behaviour management is the main issue for dentists in the treatment of children; there are, however, specific childhood conditions and illnesses that should be understood in order to deliver safe, effective treatment and advice.

The presentation of illness in children differs to that in adults and depends on:

- age
- understanding
- communication skills.

In babies, for example, there may be inconsolable crying, fever, convulsions, refusal to feed or failure to thrive.

Childhood illnesses

These are common and often the result of self-limiting viral infections. In modern western society, with the aid of mass vaccination (Table 15.2), most of the serious infectious diseases are rare (see infection chapter). There are, however, cases where vaccination has been omitted or refused, putting the individual child at increased risk.

Autism

It is estimated that up to 1 in 100 children are affected by autism spectrum disorders in the UK. It is a developmental

Table 15.2
The recommended UK vaccination schedule (2004).

Age	*Vaccination*
2, 3 and 4 months	Diphtheria, tetanus, pertussis, polio, Hib, and meningitis c
13 months	Measles, mumps, rubella (MMR)
Pre-school	Diphtheria, tetanus, pertussis, polio and MMR
10–14 years old	BCG (tuberculosis)
13–18 years old	Tetanus, diphtheria and polio

■ DENTAL RELEVANCE OF AUTISM

- Repeated traumatic injury to teeth due to fall and seizures
- Self-induced mucosal injury and bruxism
- Caries due to increased intake of sugar-containing foods and difficulty performing routine oral hygiene.

disability characterised by severely impaired social interaction and communication skills.

There is a variable degree of functional and intellectual impairment which can affect communication, understanding and the ability to perform tasks. Behaviour may be erratic with a tendency to frustration and violent acts. Autistic patients like to be in a stable environment and follow routine, so when faced with a new challenge, for example dental treatment, they tend to become hyperresponsive and overreact to stimuli. There may also be unpredictable body movement which can make treatment difficult and even dangerous. Seizures are common and many patients are treated with anti-epileptic medication.

Cerebral palsy

This is characterized by abnormal body movement and muscle coordination due to non-progressive lesions in the immature brain. The aetiology is not fully understood but birth asphyxia, family history, maternal exposure to toxins and infection during pregnancy have all been implicated. Incidence is 2–2.5/1000 live births.

There is a spectrum of clinical features from subtle to profound. Three types are described based on the type of movement abnormality:

- spastic (60%) – hypertonic
- ataxic – short jerky
- dyskinetic – slow writhing

IQ may be normal but many patients have learning difficulties. There is an increased incidence of visual and hearing problems, seizures and disorders of speech.

Depending on the severity, treatment can be carried out in general practice. Involuntary movement may make this impossible and necessitate treatment under GA in a specialist centre.

■ DENTAL RELEVANCE OF CEREBRAL PALSY

- Speech, chewing and swallowing difficulties
- Increased incidence of caries
- Drooling
- Malocclusion.

Hyperkinetic disorder (attention deficit hyperkinetic disorder (ADHD))

This is characterised by inattention, hyperactivity and impulsiveness. Affected children have an inability to focus or maintain attention. About 1% of school-age children are affected with a male:female ratio of 4:1.

The exaggerated behaviour affects school and social life, often leading to underachievement and emotional problems.

The aetiology is poorly understood but there appears to be a strong genetic component; in some cases certain foods make the situation worse and should be avoided.

■ DENTAL RELEVANCE OF HYPERKINETIC DISORDER

Behavioural problems during treatment.

Down's syndrome

Down's syndrome is most commonly due to trisomy of chromosome 21 (95%). There is an incidence of 1 in 1000 births which increases with maternal age to 9:1000 by the age of 40.

Facial features. Down's patients have flattened facial profile due to hypoplasia of the mid-face. Prominent epicanthic folds, vertical creases from the inner canthus to nasal bridge, give the classic mongoloid appearance. There is a relative macroglossia due to the reduced size of the mid-face and oral cavity. The neck is short and broad. There is a flattened area over the occiput (brachycephaly).

Complications include:

- Cardiac abnormality (40%)
- T-cell deficiency
- Learning difficulties
- Associated medical problems, e.g. epilepsy, diabetes and hypothyroidism
- Decreased airway patency/obstructive sleep apnoea due to decreased muscle tone.

Most Down's patients can be treated in general dental practice.

■ DENTAL RELEVANCE OF DOWN'S SYNDROME

Cardiac abnormalities may need antibiotic cover.

- Malocclusion
- Aphthous ulcers
- *Candida*
- Perio/ANUG due to immune defect
- Relative macroglossia
- Mouth breathing (dry mouth)
- Delayed tooth eruption.

Safeguarding children

Dental practitioners have a duty to safeguard and promote the welfare of children.

Child abuse can take a number of forms:

- physical
- emotional
- sexual
- neglect.

Signs of abuse are detectable in the head and neck region in about 50% of cases. Dentists should be able to recognise the signs of abuse and respond appropriately.

Clinical features

The investigation of abuse has huge implications for the child and parents and is a sensitive and emotional issue. Children often suffer traumatic injury in an accidental manner; features that increase the likelihood of abuse are:

- delayed presentation
- inconsistent history
- unexplained injury
- unusual injury pattern
- repeated injury
- abnormal child and parent relationship
- information from a third party
- child on the protection register.

Head and neck signs that may indicate abuse include:

- torn labial frenum
- contusions on face or in the palate
- jaw fracture
- oral STDs (sexually transmitted diseases)
- subconjunctival haemorrhage
- bite marks
- burn marks
- scalds.

If there is concern that a child may be at risk or suffering abuse it is sensible to discuss the situation with senior colleagues. If further advice or action is required then the following professions or organisations can be contacted:

- General medical practitioner
- Social services
- Police
- NSPCC.

Contact by phone and follow-up in writing within 48 hours. There should be an appropriate response within 3 days, if not you must find out what action has been taken. Keep detailed notes as you may be required to give evidence in any child abuse investigation.

Sharing information (confidentiality issues)

There are three legal aspects to the disclosure of confidential information about a child whom you suspect is suffering or at risk of abuse:

- Children's Act 1989
- Common law duty of confidence
- Data Protection Act 1998.

The law will not prevent the sharing of information with other colleagues or professions if:

- the information is not confidential
- those concerned consent to disclosure
- the public interest in safeguarding a child's welfare overrides confidentiality
- disclosure is required by a court.

■ DENTAL RELEVANCE OF CHILDREN'S WELFARE

It is part of duty of care to safeguard and promote the welfare of children.

Up to 50% of abuse cases have head and neck signs and symptoms.

Prescribing for the young

Do not deal with children as if they are little adults and use the adult dose of a drug to estimate the paediatric dose. All prescriptions should be checked by reference to the British National Formulary (BNF). (See Table 15.3.)

Table 15.3
Drugs commonly prescribed during dentistry to be avoided or altered during the treatment of children.

Drug name/type	*Effect or alteration required*
Paracetamol	Reduce dose
NSAIDs	Do not use aspirin in under 12s due to risk of Reye's syndrome
Amoxycillin	Reduce dose
Metronidazole	Reduce dose
Clindamycin	Reduce dose
Tetracycline	Do not use in under-12s due to risk of tooth staining and enamel hypoplasia
Miconazole	Reduce dose
Nystatin	No change
Local anaesthetic	Reduce maximum quantity
Midazolam	Avoid in under-16s or emotionally immature due to unpredictable reaction
Adcortyl/Corlan	Maximum of 5-day course

Drugs often have to be given in a form acceptable to the child. This will often mean prescription in the form of an elixir.

Many drug formulations contain the following excipients which should be avoided:

- sugar
- alcohol
- dyes.

Compliance with prescribed medication is influenced by formulation, taste and appearance of the drug and often depends on parental administration. This will be improved if the benefits and likely side-effects have been explained carefully to both the parent and the child.

16 PREGNANCY

Tara Renton

Pregnancy is common; before embarking on dental treatment the possibility of pregnancy should be considered.

Considerations for dental treatment should be made throughout the phases of pregnancy and subsequent breast feeding.

The first trimester is when most of the baby's organs are formed; this period is the most crucial for baby's development, so if the treatment is urgent it is best to plan procedures during the second trimester to minimise any potential risk.

Dental work is not recommended during the third trimester because the dental chair tends to be too uncomfortable for the mother. Furthermore, in certain positions the enlarged uterus may obstruct flow in the inferior vena cava.

Dental local anaesthetics such as novocaine or lidocaine are considered safe for use in pregnant women, at the doses used in most dental procedures. Citanest should be avoided as the vasoconstrictor, octopressin, may induce labour.

Antibiotics such as penicillin, amoxicillin and clindamycin are acceptable, but avoid tetracycline, which can cause discoloration of the child's deciduous and permanent dentition. Metronidazole has teratogenic potential and should be avoided in pregnant patients.

Analgesics. Paracetamol is the preferred analgesic for pregnant women, and products containing acetaminophen, such as Tylenol, are approved. You should avoid non-steroidal anti-inflammatory drugs (aspirin or ibuprofen) which may induce allergy in the unborn fetus, and opioid/opiate medications for dental pain.

Anaesthesia. Dental procedures requiring general anaesthesia or sedation should also be avoided due to the risk of fetal hypoxia. An acute toothache may be considered a dental emergency but can be treated conservatively.

■ DENTAL RELEVANCE OF PREGNANCY

Avoid unnecessary dental treatments, particularly in the first and third trimesters.

Avoid X-rays.

Avoid aspirin and NSAIDs, sedatives and hypnotics as these may have deleterious effects on the fetus.

Local anaesthetics are safe to use in pregnancy, but combinations containing vasopressin should be avoided.

Some antimicrobials (e.g. tetracycline and metronidazole) should be avoided during pregnancy.

Amalgam toxicity

Pregnant women, in particular, express a heightened concern about mercury toxicity from amalgam restorations. Unnecessary removal or replacement of amalgam should always be avoided; however, numerous scientific studies conducted over the last 150 years show that mercury toxicity is not linked to dental amalgam. If your patient is uncomfortable about having amalgam restorations placed, then consider using composite restorative materials.

Radiation – radiographs

A recent study that suggests an association between women's exposure to dental radiographs and low-weight births reinforces the ADA's long-standing recommendation that dentists use both abdominal aprons and thyroid collars, whenever practical, to minimise radiation exposure. Therefore, a degree of caution should be used when prescribing X-rays during pregnancy, i.e. only if there is a dental emergency.

Oral manifestations

Pregnancy is associated with gingivitis (Fig. 16.1) for the following reasons:

- High plasma oestrogen and progesterone increase gingival blood flow and cause an exaggerated inflammatory response to plaque.

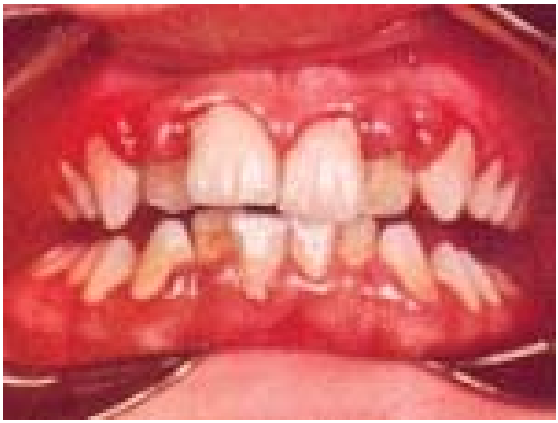


Fig. 16.1 Pregnancy gingivitis.

- Alteration in dental plaque.
- Negligence.

The WHO advises that, from the 4th month of pregnancy, women should avoid the intake of sugar, so that the fetus does not develop an exaggerated attraction for these types of foods, thus being susceptible to caries.

There is also contradictory new research that suggests a link between pre-term, low-birth-weight babies and gingivitis. The bacteria responsible for the gingivitis may travel via the bloodstream to the uterus where they result in prostaglandin production thereby inducing premature labour.

Pregnancy epulis (pyogenic granuloma: Fig. 16.2) are rare, usually painless gingival lesions that develop in response to plaque. They usually subside shortly after childbirth but may still cause a significant cosmetic problem and require surgical removal.

Pre-eclampsia

Pre-eclampsia is a disorder that affects 5% of pregnancies and is a rapidly progressive condition that affects the mother and the unborn child.

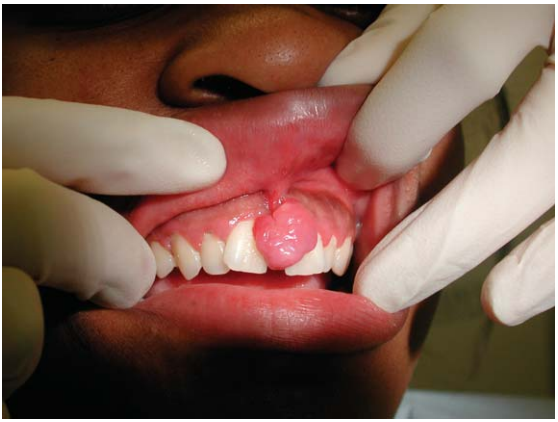


Fig. 16.2 Pyogenic granulomas are associated with pregnancy.

■ RISK FACTORS FOR PRE-ECLAMPSIA

- First pregnancy in women under age 17 or over age 35
- A family history of high blood pressure
- Multiple birth pregnancy
- Poor diets during pregnancy
- Obesity
- Smokers
- Other health problems (blood vessel conditions, kidney disease, diabetes).

Pre-eclampsia is defined as:

1. A rise in blood pressure of more than 15 mmHg diastolic or more than 30 mmHg systolic from measurement in early pregnancy; or to more than 140/90 mmHg in late pregnancy.
2. Proteinuria of more than 3 g per 24 hours.
3. And/or oedema.

The risk factors for pre-eclampsia are shown in the box above.

Typically, high blood pressure occurs after the 24th week of pregnancy and it can cause a decrease in the blood and oxygen supply available to mother and baby. In the mother it can lead to kidney problems, breathing problems,

seizures, strokes and even death in very rare cases. Babies may have problems with growing, getting enough oxygen and other complications.

High blood pressure will resolve postpartem for most women.

Gestational diabetes

Gestational diabetes mellitis (diabetes type III) is defined as diabetes that develops during pregnancy. It is less serious than other forms of diabetes and tends to resolve after birth. Symptoms include thirst, frequent urination, weight loss, lethargy and weakness. Gestational diabetes is diagnosed through routine blood and urine tests which are carried out in all pregnancies. Untreated, it can lead to problems for the mother (e.g. pre-eclampsia) and can lead to fetal abnormalities such as cleft palate. Risk factors for gestational diabetes are shown in the box.

Treatment includes:

- Regular blood glucose monitoring
- A diet high in fibre and carbohydrates, and low in sugar and fat
- Exercise
- Insulin injections in some cases.

■ RISK FACTORS FOR GESTATIONAL DIABETES

- Increasing age
- Obesity
- Family history of diabetes
- Previous large baby
- Previous perinatal loss.

Anaemia

Anaemia in pregnancy occurs when haemoglobin concentration is less than 10 gm/dl. In some populations 80% of pregnant women are anaemic. Symptoms include weakness, fatigue, shortness of breath and headaches. If the reason for anaemia is an inheritable condition, the baby

■ **RISK FACTORS FOR ANAEMIA DURING PREGNANCY**

- Lack of iron. Less often it is caused by folic acid deficiency.
- Women from low socio-economic groups, and teenagers.

may also have it. Risk factors for anaemia are shown in Box 16.4.

Patients with severe anaemia are more likely to deliver early and have small babies. Therefore, iron and folate supplements are given during pregnancy and for several months after delivery in order to help the body replace the lost blood cells and iron stores. Breast-feeding women may also need to take iron because iron is lost in breast milk.

Infectious diseases

Measles (rubella)

Pre-term labour and birth defects are associated with measles infection during pregnancy (the risk being highest in the first 3 months). Rubella vaccine is recommended for non-immune women who are considering starting a family. It takes 3 months to build up the immunity and during this time it is unwise to risk exposing a developing baby to the rubella virus.

Varicella zoster virus

Pregnant women exposed to chickenpox usually experience a benign, self-limited infection. Chickenpox occurs in pregnancy in about 3 per 1000 women in the UK. About 90% of women have antibodies to varicella zoster virus and therefore the fetus is not at risk of chickenpox even if the mother develops shingles during pregnancy. The antibodies are transferred to the infant through the placenta throughout the pregnancy. Therefore, immune pregnant women who are exposed to someone with chickenpox do not need to worry about complications for themselves or their infant.

In the non-immune pregnant woman, chickenpox is associated with increased fetal and maternal morbidity and mortality.

17 TRANSPLANT PATIENTS

Chris Sproat

The incidence of solid-organ transplantation is increasing rapidly in the UK. Many patients will present for pre-transplantation dental assessment and for routine treatment postoperatively.

Epidemiology

In the UK in 2005 a total of 2241 solid-organ transplants were performed. The waiting list for transplantation has increased 34.5% in the last 10 years and it is likely to carry on increasing in the future as the population ages and scientific advances permit transplantation in an ever expanding group of patients. The most common transplants and numbers performed in 2004 are:

- Cornea 2328
- Kidney 1673
- Liver 640
- Heart 154
- Lung 118
- Kidney/pancreas 79

There are currently 6146 patients waiting for an organ transplant with 12 160 261 or 20% of the population on the NHS donor register.

Aetiology

As the population ages the incidence of organ failure increases as does the demand on the transplant service. The majority of transplants carried out in the UK are from deceased donors; however, a significant number are carried out from living donors.

History

The term transplant was coined by John Hunter in 1778. Early attempts at transplantation failed due to a lack of understanding of the immunological processes

involved. Those that did succeed were between identical twins.

Two major advances allowed transplantation to proceed:

1. The discovery that each individual has a unique set of tissue antigens, encoded by the major histocompatibility complex (MHC) on chromosome 6, which must be closely matched to prevent rejection.
2. The development of immunosuppressive drugs to reduce the incidence of rejection.

Types of transplant

- *Autograft* from one part of the body to another, i.e. iliac crest bone to jaw
- *Allograft* between individuals of the same species, most common type
- *Zenografts* between different species, i.e. porcine heart valves to humans.

Types of donor

There is a large discrepancy between the number of organ donors and patients requiring transplants. This is due to the number of people registered as donors, difficulty gaining consent from relatives of recently deceased patients and compatibility between donors and recipients. The increased use of zenografts would help to ease the situation but there are increased compatibility problems and the potential risk of spread of infection from the donor species.

Figure 17.1 shows the numbers of cadaveric donors and transplants in the UK between 1994 and 2003.

Immune suppressant medication

Most transplantation patients rely on immune suppression for life. As a result there is an increased risk of infection, small risk of skin cancer and haematological malignancy and side-effects of the drugs prescribed. A tailored combination of drugs is used for each patient to spare the use of steroids and keep the doses and side-effects of the individual drugs as low as possible (see Table 17.1 and Fig. 17.2).

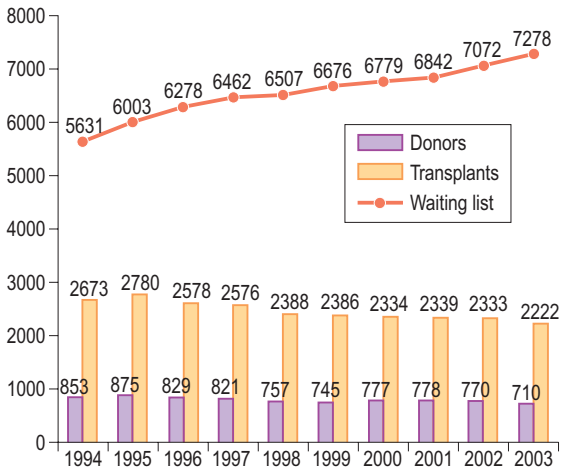


Fig. 17.1 Number of cadaveric donors and transplants 1994–2003. The number of patients on the waiting list for transplantation at 31st December each year (from NHS UK transplant data).

Table 17.1
Commonly used immunosuppressant drugs and their side effects.

Drug	*Side effect*
Ciclosporin	Gingival hyperplasia, diabetes, hypertension
Tacrolimus	Nephrotoxicity, hair loss, hypertension
Azathioprin	Leucopenia, hair loss
Mycophenolate	GI upset, leucopenia
Steroids	Hypertension, osteoporosis, diabetes, cushingoid face
General	Infections, malignancy

Survival and cost effectiveness

Success rates for transplants are high, with a 5-year survival of between 60 and 90% depending on the organ, the donor type and the pre-transplant status of the patient. Renal transplantation saves £21 000 per year per patient

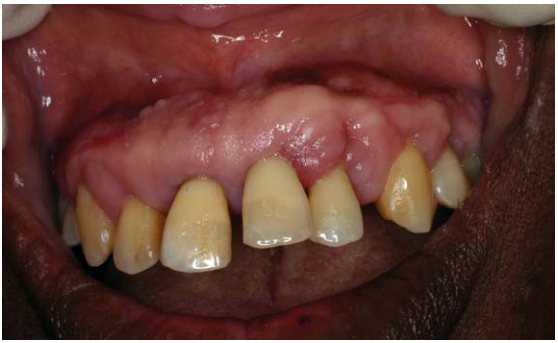


Fig. 17.2 Ciclosporin-induced gingival hyperplasia in a renal transplant patient.

when compared to dialysis. The total cost benefit in terms of the physical, social and psychological aspects is difficult to quantify but far outweighs the monetary value.

Dental implications of transplantation

There has been little research into implications of transplantation to dentistry. It appears that due to pre-existing organ failure, the patients are often at a higher risk from dental treatment before their transplant is carried out.

There are, however, implications for dental treatment, mainly due to the immunosuppression required for transplantation. It is reasonable to give antibiotic cover for invasive dental procedures in view of healing problems and tendency for wound infection. It is also of paramount importance to reinforce oral hygiene and carry out preventative measures. Any dental infections should be treated aggressively. Ciclosporin-induced gingival hyperplasia may need to be reduced by gingivectomy, for cosmetic reasons.

If you are in any doubt about dental treatment of an individual you should contact the transplant team for advice.

■ DENTAL RELEVANCE OF TRANSPLANTATION

Preventive dentistry is important.

It is reasonable to give antibiotic cover for invasive dental procedures.

There is increased susceptibility to infection.

High risk of hepatitis B/C and cross-infection.

Increased incidence of hypertension and IHD.

Oral effects of immune suppressant drugs:

- Candidiasis
- Herpes simplex
- Ulceration
- Increased incidence of malignancy
- Gingival hyperplasia (ciclosporin)

If the transplanted organ is failing there may be altered drug metabolism.

Useful web site

www.uktransplant.org.uk

18 MALIGNANT DISEASE

Mark McGurk

The risk of developing cancer is increased in patients who have already had a cancer. Do not ignore unusual symptoms in this population of patients.

Epidemiology

Approximately 270 000 new cases of cancer are reported each year in the UK where this disease is now the commonest cause of death (26% of all deaths in the UK) (see Fig. 18.1). Cancer can be viewed as a degenerative disorder as the incidence of neoplasia increases markedly with age.

Against this background different types of cancer have their own profile, some occurring only in childhood (retinoblastoma), others having a bimodal presentation – such as Hodgkin's lymphoma. Cancers that occur in children tend to arise from discrete genetic defects whereas those in the elderly occur through gradual accumulation of non-specific genetic damage over time.

Aetiology

Aetiological factors that predispose to neoplasia vary between the different types of tumour and some relationships are complex, although they can have a common theme. Breast cancer is related to the reproductive cycle. The risk of disease increases with the duration of time a woman is fertile whereas pregnancy has a protective effect so long as it commences before 35 years of age.

Tobacco and alcohol are recognised carcinogens and are incriminated in the development of mouth, throat, lung, oesophagus, pancreas and bladder cancer. Radiation is associated with haematological disorders and thyroid cancer. Infective agents such as papilloma virus and hepatitis B are linked to cervical and liver cancer respectively whereas solar radiation is responsible for cancer of the skin (squamous cell carcinoma, basal cell carcinoma, melanoma) which together are numerically by far the most

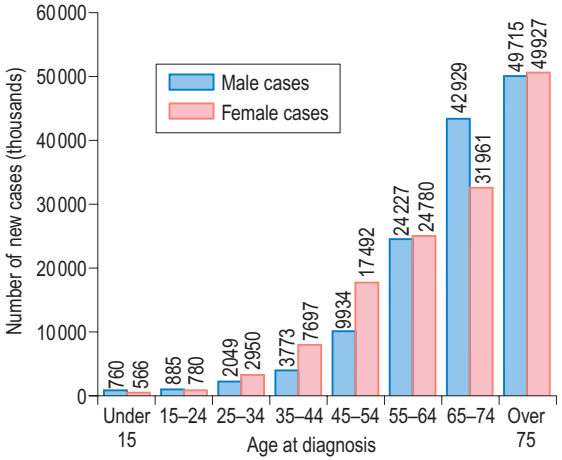


Fig. 18.1 New cases of cancer by age and sex. The number of new malignancies can be seen to rise rapidly with age.

common cancers in the body (109000 cases per year in the UK).

It is apparent that most of the common cancers are related to lifestyle and environmental factors. It has been estimated that up to 90% of human cancer is potentially avoidable by changes in lifestyle.

Treatment

Treatment regimes for different cancers cannot be discussed in detail but therapy can be divided into local and systemic. Also, cancers can be separated into solid or haematogenous. Local therapy depends mainly on surgical excision and radiotherapy and is usually the mainstay of treatment for solid tumours. Systemic therapy is the more complex and depends on a range of cytotoxic drugs that are usually administered in combination. Haematological malignancies (leukaemia, lymphomas, myelomas) are more responsive to chemotherapeutic agents than are solid tumours. In general tumour is dose-dependent and the challenge has been to devise ways of delivering increasingly higher doses of toxic drugs but at the same time rescuing normal tissues. Escalating doses of chemotherapy can be used to advantage, particularly in haematological

Table 18.1
New therapeutic modalities in the treatment of malignancy.

Agent	*Tumour treated*
Interferon α	Melanoma
EGF receptor blocker	Squamous cell carcinoma
Papilloma virus vaccine	Cervical cancer
Angiogenesis inhibition	Solid tumours
Haematogenous growth factors	Leukaemias
Hormone blocking drugs	Breast cancer

malignancies and childhood cancers. Techniques include autologous stem cell rescue and marrow transplantation. However, success is achieved at a price that includes subtle persistent diminution in function of remaining normal body tissues. The systems particularly at risk are the nervous system, heart, lungs and bone marrow. There is a diminution of reserve which can manifest itself as an inability to respond adequately when the body systems are severely stressed such as following major trauma or extensive surgery.

In addition to these mainstays of therapy new modalities are continually being developed. Gene therapy is still in its infancy but molecular engineering has produced designer drugs in the form of monoclonal antibodies to facilitate bone marrow clearance in leukaemia, receptor blockers for growth promotion epidermal growth factor) and tissue cytokins (see also Table 18.1).

The new therapeutic agents have side-effects which take the form of fevers, malaise and hypertension but their long-term effects on body function seem minimal. Rather it is cytotoxic drugs that carry their legacy into adulthood.

Childhood malignancy

Approximately 1500 new cases of childhood cancers (at <14 years) are recorded each year in the UK.

The most common cancer of childhood is leukaemia, of which 80% of cases are acute lymphoblastic type. Over 90% of these children achieve remission for over 20 years based on multiple-agent chemotherapy. Relapses can be treated by marrow transplantation if histo-compatible sibling donors are available. Common cancers of childhood are shown in Figure 18.2.

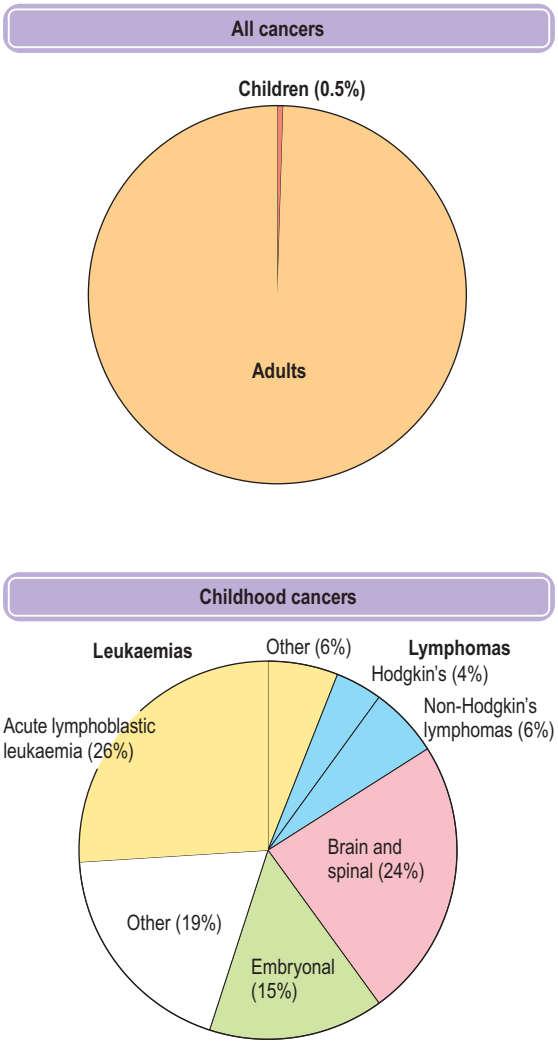


Fig. 18.2 Contribution of childhood malignancy to total malignancy and the relative frequency of the different types of malignancy in children.

Head and neck tumours in children

These cancers are rare and usually consist of sarcomas or retinoblastoma.

Local therapy in the form of surgery or radiotherapy can have a significant impact on dento-facial development. Focal growth retardation is the norm which leads to facial deformity as the child grows. The teeth may not develop or may form with short roots, wide pulp cavities and altered crown morphology. This places the teeth at risk of dental decay. There is also the long-term effects of radiation on bone healing which make these patients at potential risk of osteoradionecrosis. Childhood tumours tend not to occur in the mouth and so the full force of radiation is not normally delivered to the jaws. Consequently the risk of radionecrosis is low following treatment of childhood cancer unless the cancer was sited in the oral cavity.

■ DENTAL RELEVANCE OF CHILDHOOD CANCER

The majority of children with cancer receive chemotherapy as a part of their treatment.

Local treatment in the head and neck (surgery and radiotherapy) can lead to facial deformity, altered tooth morphology, malocclusion.

Systemic chemotherapy may have long-term effects in the form of growth retardation, cardiac toxicity, lung fibrosis and diminished bone marrow function.

Also, there is a risk of the patient developing second primary cancers in childhood.

Unless the individual received a course of radiotherapy directed at the jaws (which is uncommon) the risk of bone infection is low.

Dental treatment does not pose a particular risk to the patient (see childhood cancer).

Adult malignancy

Cancers of the breast, lung, large bowel and prostate account for over half of all the new cancers diagnosed each year in the UK (see Fig. 18.3).

The majority of neoplasms occur after the 6th decade of life and so management of late side-effects of therapy is less of an issue than in children. Also in adults, solid

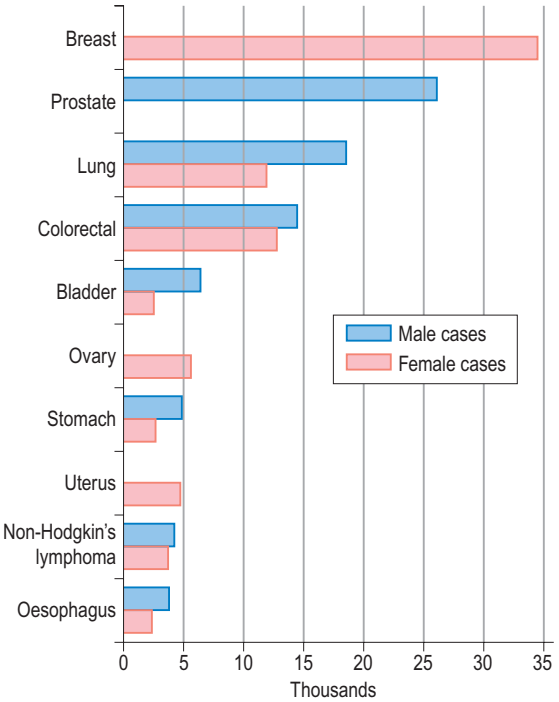


Fig. 18.3 Incidence of the most common cancers affecting adults by sex.

tumours predominate and usually with these cancers chemotherapy is not usually used with such intensity as for haematologically tumours.

However, adult haematological malignancies are still treated aggressively apart from the sub-population of patients with chronic leukaemias. This is an indolent disease that can persist for years without symptoms. Nevertheless, these patients may have subtle alteration in immune competence which is revealed only in the face of an infective challenge. Dental sepsis may be such a challenge and the risk of infection should be kept to a minimum.

Cancers in the head and neck

Cancers of the skin surface are particularly common especially in light-skinned subjects in tropical climates. These

tumours (squamous cell carcinoma and basal cell carcinoma) are relatively indolent. However, a small proportion (1–3%) of the squamous cell carcinomas of skin have the propensity to metastasise. So, in Australia, with its hot climate and Caucasian population, the commonest cause of a parotid lump is metastasis from a primary skin cancer in the scalp.

Mouth and throat cancer have an incidence of 80–120 cases per million per annum in the UK. It is a disease of the elderly and the median age of presentation is about 60 years. Salivary and thyroid tumours are much less common (7–15 cases annually/1 000 000 population). The peak incidence of disease is a decade earlier (50 years of age).

The treatment of head and neck tumours is usually by surgery and/or radiotherapy. But there is now an increasing contribution made to treatment from chemotherapy. In mouth and throat cancer it is used in conjunction with radiotherapy and improves outcome by about 7–10%.

Occasionally tumours from distant primary sites can metastasise to the jaws. These tumours migrate in the blood stream and tend to alight at areas of high vascularity such as the temporomandibular joint or where the inferior dental artery enters and exits the mandible. Paraesthesia is an important sign that should never be ignored. Tumours that tend to metastasise to the jaw are those from the breast, kidney, prostate, lung and thyroid.

■ DENTAL RELEVANCE OF HEAD AND NECK CANCERS

The dental implications of treatment of solid tumours outside the head and neck is minimal.

Dental implications of cancer therapy pertain mainly to cancer of the mouth and to a lesser extent to salivary gland. This is because the full force of treatment is focused on the jaws. Those patients who continue to smoke and drink have a 2% accumulative risk of a new cancer each year. Growth retardation is not a problem in the adult population. Surgery can lead to trismus and poor access to the oral cavity. A liquid diet increases the risk of dental caries. Similarly, radiation therapy reduces salivary secretions, alters the oral flora and even with excellent oral hygiene patients are still at significant risk of root caries. Dental extractions, particularly in the mandible, place the patient at risk of osteoradionecrosis. It is recommended that all extractions in the mandible post-radiotherapy should be undertaken at specialist centres.

19 DISABILITY

Chris Sproat

Disability is defined by the World Health Organisation as 'a restriction or lack of ability to perform an activity in the manner or within the range considered normal for a human being'. It is well known that people with a disability may not have an equal opportunity to receive normal dental care.

Epidemiology. Using the current UK criteria, approximately 10 million people are classified as being disabled. The proportion of people with long-term illness or disability increases with age (Fig. 19.1). Types of disability, as described in the Disability Discrimination Act, include:

- physical
- intellectual
- psychiatric
- sensory
- neurological
- physical disfigurement
- presence in the body of organisms causing or capable of causing disease.

Barriers to health care exist at many different levels, including:

- *Physical*. Patients may not be able to travel to the dental surgery, gain access to the building or treatment room.
- *Psychological*. There tends to be an increased level of anxiety in both the patients and providers of dental care when disability is involved.
- *Availability*. The availability of routine NHS treatment is decreasing and may be difficult to find in the local area. Specialist services are often not able to cope with the demand. There may be a lack of domiciliary services.
- *Training*. There is often reduced awareness of disability and its implications for oral health among dental health providers.

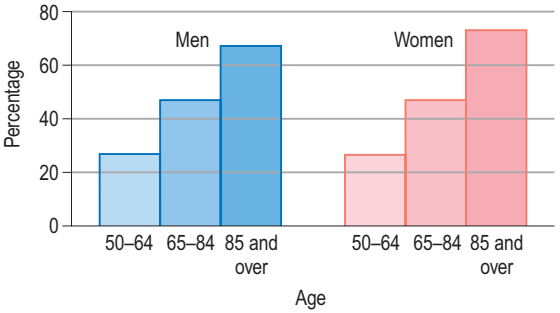


Fig. 19.1 Long-term illness or disability which restricts daily activities: by sex and age, April 2001, Great Britain (Household Survey 2001).

The *Disability Discrimination Act* (DDA) is designed to ensure that services are equally accessible to disabled and non-disabled people. This act covers all aspects of delivery of dental care and includes both patients and employees. New providers have to comply with the act and existing providers have to make reasonable adjustments to comply. The act covers those who have had a disability for greater than 12 months.

Oral implications of disability are related to the disability itself and to the side-effects of treatments used. In addition to reduced access to normal dental care many patients are not able to maintain their own oral health and require assistance to perform regular tasks, e.g. tooth brushing. Those with severe neuromuscular, neurological or learning difficulties often suffer with incontinence of saliva and bruxism, increasing tooth wear. Patients with seizure disorders may suffer frequent traumatic injury to their teeth and oral soft tissues. In Down's syndrome there is an increased susceptibility to periodontal disease. Many drugs used for treatment of disabling conditions, e.g. anti-Parkinson's or antidepressants, cause dry mouth as a side-effect, leading to swallowing and speech difficulties and increased susceptibility to caries. In disorders that affect muscle control and movement, denture wear and routine dental treatment are often difficult.

Management of disability often requires a multi-disciplinary approach with close liaison between the dental team and those involved in the medical and social

Post-Sedation advice sheet

Following intravenous sedation you will not be discharged until we are happy that it is safe for you to leave.

You must be accompanied by a responsible adult escort for your journey home and for the following eight hours.

You must not drink alcohol, drive a car, operate machinery or sign important documents for at least eight hours after you leave the surgery.

Remember your mouth will remain numb for up to four hours after the procedure so you must be careful when eating or drinking to avoid biting and burning your lips and mouth.

To aid rapid healing do not smoke for 10 days or drink alcohol for 48 hours.

Reduce discomfort by taking normal headache type pain killers, for example Ibuprofen, regularly for the first few days after the treatment. Always read the instructions on the box before taking any medication and do not exceed the maximum dose.

If you experience persistent problems or are concerned do not hesitate to contact us.

Fig. 19.2 Large print information leaflets are required for patients with visual impairment.

care of the patient. The most helpful strategy is removal of the barriers to dental care, allowing the vast majority of disabled patients to be treated in general practice.

Physical alterations to building may be required but equally important is the availability of information in formats understandable by all patients, e.g. large print (Fig. 19.2) and Braille. Training of staff in the awareness of the problems posed by disability is useful.

Patients with severe disabilities may require specialist treatment and the availability of general anaesthesia.

■ DENTAL RELEVANCE OF DISABILITY

The vast majority of disabled people can be treated safely in dental practice.

Disability is common, with 16% of the population currently classified as disabled.

Disabled patients and staff should be able to gain easy access to all the services in dental practice.

Domiciliary treatment may be required.

There is an increased incidence of oral disease in disabled patients.

Useful website

www.disability.gov.uk

20 HEALTH PROMOTION

Chris Sproat

Background

The health of the UK population has improved dramatically over the last century. The dominance of infectious disease has declined and has been replaced by degenerative diseases, cancer and coronary heart disease. The health gap between the rich and poor has increased.

The aims of the government

- To reduce mortality rate
- To reduce health inequalities
- To tackle the determinants of ill health and health inequalities.

Factors affecting health can be modifiable or fixed. The modifiable factors are divided into those under individual control (lifestyle) and those requiring general legislation (Table 20.1).

Priority areas for action

1. Reducing the number of people who smoke
2. Reducing obesity and improving diet and nutrition
3. Increasing exercise
4. Encouraging and supporting sensible drinking
5. Improving sexual health
6. Improving mental health.

The mechanism. Rather than attempting to control all aspects of health the main thrust is to promote and support healthy lifestyle choices through:

- education to increase awareness of the benefits of a healthy lifestyle and the risks associated with less healthy alternatives.
- legislation to improve those factors not under individual control, e.g. access to services, food labelling, etc.

Table 20.1
Factors affecting health.

Fixed	*Modifiable*			
	Lifestyle	*Social/ economic*	*Environment*	*Services*
Sex Genes Age	Smoking Diet Activity Alcohol Sexual behaviour Illicit drugs	Poverty Employment Social exclusion	Housing Water quality Air quality Social environment	NHS Education Social services Transport Leisure

The role of dentists in health promotion is important. It is related to the unique position dentists hold within the health care system, having regular access to a large cohort of the population not seen by other health care professionals.

The majority of the priority areas for action have a direct impact on oral health for which dentists take a lead role in patient education.

Smoking

Smoking causes the death of 120 000 people per year in the UK, or 20% of all deaths, and it is preventable. One in 10 of those in the age group 11–15 years smokes; this is the age when most smokers begin the habit. The government aim is to reduce the number of adults over 16 who smoke from 26% (currently) to 21% or less by 2010. (See also Fig. 20.1.)

Smoking has been implicated in over 50 disease processes; however, there are three common diseases that smokers die from:

- cancer
- chronic obstructive lung disease
- coronary heart disease.

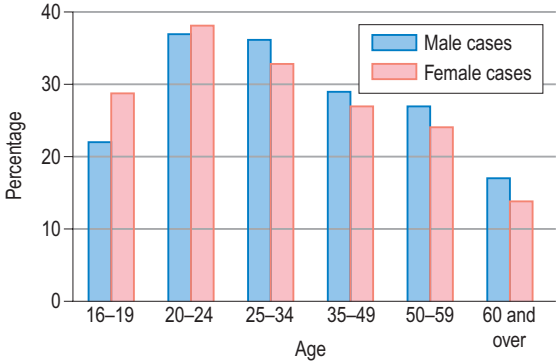


Fig. 20.1 Prevalence of smoking in the UK by age and sex 2002/2003. It can be seen that the prevalence of smoking is greatest in those aged 20–24. For the first time, in the 16–24 age group, the prevalence of smoking is higher in females than in males, which is a worrying trend for the future (from the Office of National Statistics).

Unlike much of the rest of the body, where the effects of smoking are often hidden, there are visible effects on the oral cavity. A thorough dental examination should detect most oral changes and facilitate the delivery of advice on how to stop smoking as well as support along with early treatment. (See also Fig. 20.2.)

Oral effects of smoking include:

1. Oral cancer and pre cancer, where smoking is the leading cause
2. Periodontal disease
3. Delayed wound healing due to peripheral vasoconstriction
4. Increased failure of dental implants
5. Halitosis
6. Poor aesthetics due to tooth staining.

Cessation of smoking

Two-thirds of smokers would like to give up but only 8% are currently successful.

The dental team should follow the 'four-A' approach, with regular review of progress:

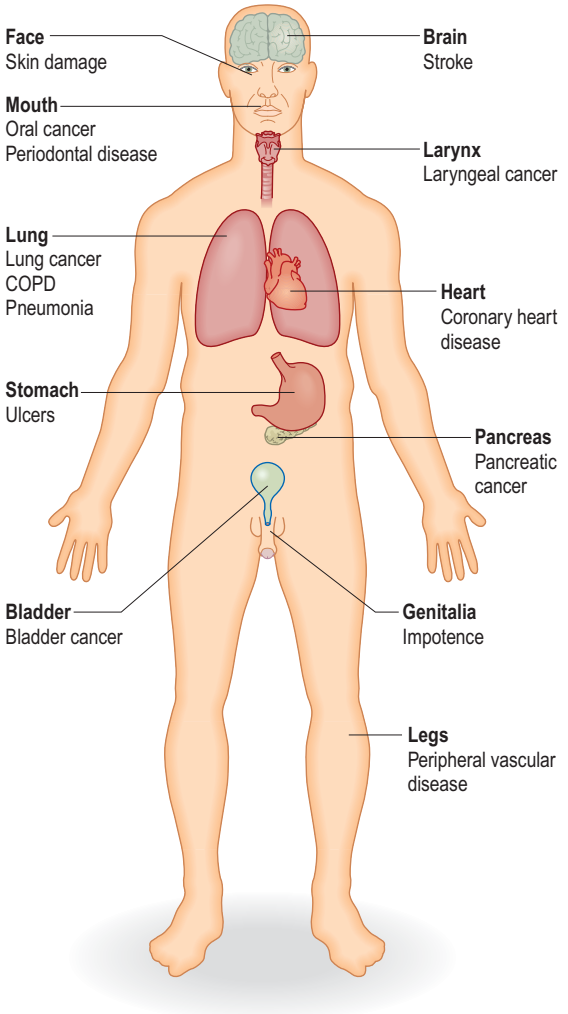


Fig. 20.2 Common smoking-related cancers and diseases. It can be seen that smoking has harmful effects on most body systems.

■ DENTAL RELEVANCE OF SMOKING

The duty of dentists as members of the NHS team is to advise patients of the risks of smoking and to support those who want to quit.

Smoking has serious oral consequences, not least being the leading cause of oral cancer.

- *Ask* about smoking habits and desire to quit.
- *Advise* on the general and oral harmful effects of smoking and the facilities available to aid cessation of smoking.
- *Arrange* to refer smokers to the local NHS 'stop smoking' service or other local services.
- *Assist* by encouraging the cessation of smoking.

How to stop smoking

- Set a quit date.
- Throw away all cigarettes, ash trays, etc.
- Consider; Zyban or nicotine replacement, acupuncture, hypnosis, support groups, i.e. NHS 'stop smoking' clinic.
- Enlist help and support of family and friends.
- Change routine to avoid smoking environments.
- Take exercise and care with your diet.
- Treat yourself with the money you save.

Obesity, diet and exercise

Obesity is a huge problem in the UK which has trebled in the last 20 years. Currently over half of the women and nearly two-thirds of men are overweight.

The upward trend in obesity is due to changes in eating patterns, with increased consumption of 'fast and pre-packed food' combined with a lack of exercise and sedentary life style.

Obesity is determined by the person's body mass index (BMI) – weight in kilograms/height in metres^2 (kg/m^2)

BMI categories:

- underweight <18.5
- normal weight 18.5–24.9

■ DENTAL RELEVANCE OF OBESITY

The risk of adverse events occurring during dental treatment is increased by the associated medical conditions:

- diabetes
- heart disease
- hypertension.

General anaesthetic should be avoided in obese patients.

- overweight 25–29.9
- obesity >30

Obesity may be secondary to endocrine and renal problems but is usually due to eating in excess of necessity.

High BMI is associated with type II diabetes, heart disease, hypertension, depression, osteoarthritis and reduced life expectancy.

Primary prevention is the most effective way to combat the obesity, involving:

1. Raising public awareness of the causes and problems of obesity
2. Facilitating healthy diet, e.g. improved food labelling and school meals
3. Promoting physical exercise for all age groups
4. Improved access to services that support weight loss.

Secondary prevention for those overweight includes:

1. Increased exercise
2. Dieting
3. Drug therapy
4. Surgery (only in extreme cases).

Sensible drinking

Alcohol consumption is endemic in UK, with 90% of adults admitting to drinking. Binge drinking accounts for greater than 40% of alcohol consumption in males and 22% in females.

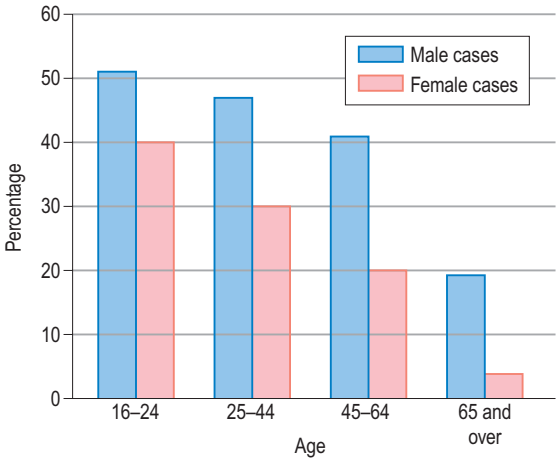


Fig. 20.3 Adults exceeding recommended daily limits on at least 1 day during the preceding week: 2003/2004. It can be seen that harmful drinking is widespread and is commonest in the 16–24 age group, decreasing with age (from the Office of National Statistics).

Sensible drinking implies less than 4 units per day for males and less than 3 units per day for females. (It is more useful to look at daily rather than weekly consumption.) (See also Fig. 20.3.)

Effects of harmful drinking (see Ch. 14: alcohol abuse section).

Sensible drinking strategies

In addition to raising awareness of the harmful effects of alcohol, including binge drinking, a voluntary social responsibility scheme has been set up with producers and suppliers. This aims to protect the young from alcohol and will ask for a sensible approach to advertising by not targeting the younger age group, tightened control on sale of alcohol to those under 18, and improved labelling of containers and bottles to increase public awareness of the harmful effects of alcohol.

■ DENTAL RELEVANCE OF ALCOHOL

An alcohol history should be taken from all patients who drink. The average number of units per day should be recorded.

Patients should be made aware of the recommended safe limits:

Male: 4 units/day (28 units/week)

Female: 3 units/day (21 units/week)

Alcohol can affect multiple organ systems (see psychiatry section) and increase the risk of adverse events occurring during dental treatment.

Useful websites

www.quit.org.uk
www.dh.gov.uk

21 MEDICAL EMERGENCIES IN DENTAL PRACTICE

Mark McGurk

Being aware of medical emergencies is not synonymous with being prepared to manage an emergency. Most emergencies occur unexpectedly.

Background

Which dental factors affect the risk of encountering an emergency?

The risk of encountering a medical emergency depends in part on the type (specialisation) of dental practice, and also the characteristics of the population being treated. Serious medical emergencies are unlikely to be encountered in a young population treated by orthodontists. Provision of dental treatment under general anaesthetic has now ceased but dental practices still specialise in provision of intravenous sedation. The risk of a medical emergency in a practice specialising in intravenous or inhalation sedation is 8.5 and 6 times that expected in a traditional dental practice.

How often does a medical emergency occur?

It is estimated that, in a working life of 40 years, each dentist will have encountered six emergency situations, an event every 4.6 years (Table 21.1).

It is the rarity of events that is the underlying problem. The events are so infrequent that the dentist becomes lulled into a false sense of security and so when an emergency does occur the dental team is frequently unprepared. The answer is to be proactive and try to identify situations where an emergency is likely and then identify the type of emergency and make preparation.

Table 21.1
Number of emergencies a dental practitioner is likely to encounter in a working lifetime of 40 years in general practice.

Event	*Number of cases*
Fits/seizures	2.75
Swallowed foreign body	1.52
Asthma	1.31
Diabetic	1.02
Angina	0.98
Drug reactions	0.89
Other events	0.24
Cardiac arrest	0.13
Myocardial infarction	0.11
Stroke	0.09
Inhaled foreign body	0.06
Total	6.32

What emergencies are likely to occur?

The most common adverse events in general dental practice are:

- fits and seizures
- swallowed foreign body
- asthma attacks
- diabetic emergencies
- drug reactions.

Specific emergencies

Fits and seizures

Occurrence: three times in a working lifetime of 40 years.

This category of emergency does not relate solely to patients with epilepsy but rather is dominated by patients who faint in the dental chair. In the process of a faint the blood pressure falls, reducing the flow of blood to the brain. If this is not restored by quickly placing the patient in supine position, the faint may be accompanied by jerky body movements similar to a seizure. The commonest medical emergency in dental practice is a faint.

Syncope (faint)

Signs and symptoms:
- Light headedness
- Nausea and pallor
- Brief loss of consciousness
- May fit if not laid flat
- Initially rapid, faint pulse and then slow and full.

Treatment:
- Lay patient flat with legs raised
- Loosen tight clothing around neck
- Open windows to relieve stuffiness.

Epileptic seizure

Signs and symptoms:
- Sudden loss of consciousness or lack of response
- Initial rigidity and then jerking of limbs
- Urinary incontinence may occur
- Post-fit drowsiness and confusion
- Most patients recover spontaneously.

Treatment:
- Make sure patient is safe
- Do not attempt to recover objects from mouth
- Only attempt treatment if the fitting is prolonged >5 minutes
- Give oxygen 15 L/minute
- Intravenous diazepam 10 mg can be used with care
- Call emergency services.

Swallowed foreign body

Occurrence: two times in a practice lifetime of 40 years.

The loss of a foreign body into the upper aerodigestive tract is a complication inherent to dentistry. Factors that compound the risk are the supine position of the patient, loss of sensation with local anaesthetic, the use of small delicate equipment within the oral cavity such as screws, files, crowns that can easily slip out of the operator's fingers. Also, a sedated patient, by definition, has reduced awareness and co-ordination. **If a foreign body is lost down the throat it cannot be assumed that because the**

patient does not cough the object has not passed into lung. Unless the patient is absolutely sure a foreign body has been swallowed the patient should be assessed in hospital. This also applies to ingestion of sharp objects.

Signs and symptoms:
- Coughing or choking but not always
- Wheeze or stridor
- Cyanosis in severe cases.

Treatment:
- Ask patient to cough
- Attempt retrieval if visible within oral cavity
- Shoulder blows/Heimlich manoeuvre
- Oxygen 15 L/minute
- **Do not attempt a surgical airway unless you have been trained to do so.**

Asthma attack

Occurrence: 1.3 times in a working lifetime of 40 years.

Approximately 5.1 million people suffer from asthma in the UK population. In the majority of incidences the condition is mild and controlled easily by the patient. A small proportion of patients are prone to acute attacks of asthma that are frightening to the patient and dentist alike. About 1500 patients die from asthma each year, including 25 children and 500 adults under the age of 65.

Patients prone to status asthmaticus should be identified and preparation made prior to attendance to deal with an emergency situation if it occurred (ready access to appropriate drugs and oxygen). Status asthmaticus can occur quickly and may catch the dental team unawares.

Signs and symptoms:
- Wheezing
- If severe silent chest
- Cyanosis.

Treatment:
- Two puffs of salbutamol inhaler
- Oxygen 15 L/minute
- If no response further inhaler/salbutamol nebuliser
- Call emergency services.

Diabetic events

Incidence: one per working lifetime of 40 years.

Hyperglycaemia does not usually present as an emergency. It develops insidiously over a protracted period and most diabetic patients are aware when their blood sugar is elevated. The emergency situation arises with episodes of hypoglycaemia as it can occur precipitously, ushered in by what appears to be an aggressive patient talking incoherently and staggering as if intoxicated. Most diabetics are well controlled and not at risk of hypoglycaemic attacks; rather it is the brittle diabetic whose blood sugar swings markedly through the day. This group of patients should be distinguished and preparations made to deal with a possible emergency prior to the patient attending for treatment.

Signs and symptoms:

- Confusion, disorientation, aggression, slurred speech, loss of co-ordination, sweating and loss of consciousness
- Patient is often aware of the problem and asks for glucose.

Treatment:

- If conscious, oral glucose drink
- If unconscious 1 mg glucagon intramuscularly followed by glucose drink when consciousness regained
- Intravenous glucose can be given but this requires a large cannula in a large vein as it is like treacle.

Angina pectoris

Occurrence: one per working lifetime of 40 years.

The mean age of the UK population is slowly increasing and at the same time a greater proportion of the population are retaining their natural dentition. Consequently the population at risk of developing angina during dental treatment is increasing. Patients with angina are familiar with their symptoms and usually can control them easily with medication prescribed. Those with unstable angina and symptoms that are easily provoked should be identified. Urgent referral is indicated in patients where symptoms of angina are not responding to

medication or the patient is experiencing symptoms for the first time for they may herald a myocardial infarction in evolution.

Signs and symptoms:
- Crushing central chest pain with radiation to left arm, neck and jaw
- Shortness of breath.

Treatment:
- Glyceryl trinitrate (GTN) sublingual
- Oxygen 15 L/minute
- If no response consider possibility of an MI and give aspirin 300 mg to be chewed.

Adverse drug reaction

Occurrence: 0.9 times in a working lifetime of 40 years

Adverse drug reactions account for 7.5% of all emergency events in general dental practice, half of which are either some form of anaphylactic reaction or adverse reaction to local anaesthetic. No deaths were reported in a sample of 1469 emergencies.

Signs and symptoms:
- Rash, itching, swelling of tongue, tingling of face.
- Wheeze and stridor
- Collapse.

Treatment (*depends on severity of reaction*):

Minor reaction
- Chlorpheniramine 10 mg intramuscularly, then continued orally for 24 hours – but keep under review. Anaphylaxis can be delayed up to an hour.
- Hydrocortisone 100 mg intravenously
- Medical review.

Severe reaction
- Adrenaline (epinephrine) 0.5–1 mL of 1 : 1000 (IM or SC – not IV). This may need to be repeated at 5-minute intervals
- Oxygen 15 L/minute
- Chlorpheniramine 10 mg IM or slow IV
- Hydrocortisone 100 mg IV
- Call emergency services.

Cardiac arrest, myocardial infarction, stroke

Occurrence: 0.1 times, respectively, in a working lifetime of 40 years.

In a series of 1341 emergency events reported by 701 dentists covering a working period of 6062 years, 10 deaths were encountered. This would suggest that about 0.7% of emergencies in the UK result in death. A death in the dental surgery can be expected once every 758 practice years. This translates to a 1:19 risk of having to manage a fatal event in one's practising lifetime. Causes of deaths are shown in Table 21.2.

Cardiac arrest

Signs (Fig. 21.1):

- Unresponsive
- No respiratory effort
- Pulseless.

Treatment:

- Basic life support (Fig. 21.2) until patient recovers or emergency services take over.

Table 21.2
Cause of death in dental surgery: a survey of 1341 medical emergencies.

Cardiac arrest	5
Stroke	2
Myocardial infarction	3

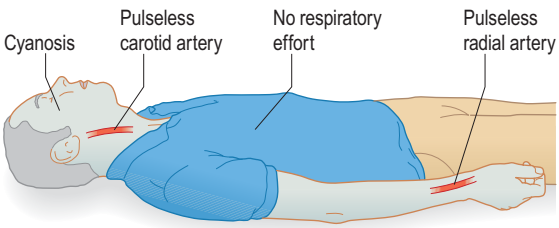


Fig. 21.1 Signs of a cardiac arrest.

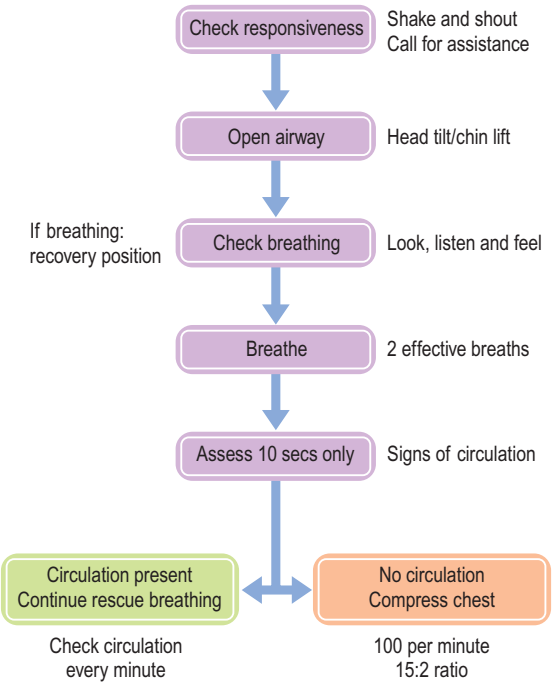


Fig. 21.2 Basic life support algorithm.

Myocardial infarction

Signs and symptoms:

- Prolonged crushing central chest pain, worse than angina, with radiation to left arm, neck and jaw
- Sweating, pallor and anxiety
- Shortness of breath, severe if acute heart failure.

Treatment:

- Sit patient in comfortable position, likely to be upright
- Oxygen 15 L/minute
- Aspirin 300 mg dissolved or chewed

- Give relative analgesia if available set at 50% nitrous oxide 50% oxygen
- Call emergency services.

Stroke

Signs and symptoms:
- Lack of response, difficulty speaking
- Tingling or weakness of one side of the body.

Treatment:
- Place patient in a safe comfortable position
- Oxygen 15 L/minute
- Call emergency services.

Addisonian collapse (adrenal insufficiency)

This is an extremely rare incident in dental practice caused by suppression of the pituitary hypothalamic axis due to administration of steroids. At times of surgical stress the body is not able to maintain blood pressure and collapse occurs.

Signs and symptoms:
- History of long-term steroid use
- Collapse
- Low blood pressure.

Treatment:
- Lay patient flat
- Hydrocortisone 100 mg IV
- Intravenous fluids if available
- Call emergency services.

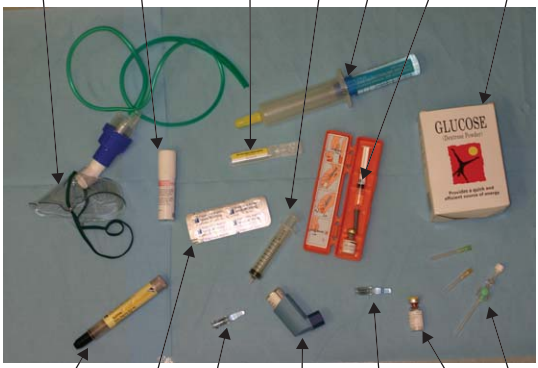
When do emergency events occur?

The majority of emergencies occur when the patient is in the dental surgery itself (80%); in 15% of cases the patient may be elsewhere in the dental practice and in 5% the emergency occurs outside the premises. It cannot be assumed that the emergency is a direct result of treatment, for about 20% of emergencies occur before treatment and another 20% at injection, 36% during treatment and 16% after treatment is complete.

■ DENTAL RELEVANCE OF MEDICAL EMERGENCIES

1. Being aware that a complication may occur is not the same as being ready to manage the emergency.
2. Apart from being generally prepared the dentist should be able to identify high-risk situations and make preparation in the event of an emergency.
3. Situations where the dentist may make a telling impact on outcome is status asthmaticus, the brittle diabetic, the patient with unstable angina and those with severe allergic reaction.
4. The occurrence of life-threatening events such as cardiac arrest, stroke and myocardial infarction cannot be predicted but their management is part of standard resuscitation training with which a dental team should be familiar.

22 EMERGENCY KIT



23 INTRAVENOUS CANNULATION AND INTRAMUSCULAR INJECTION

Chris Sproat

Intravenous cannulation

Intravenous cannulation is a procedure that all dentists should be able to perform in order to administer emergency and sedative drugs and prophylactic antibiotics.

Choice of cannula

There are a number of different types and sizes of cannula available. The common ones available in dental practice are:

- venflons
- Y cans
- butterfly cannula.

Most cannulae are universally colour-coded as a quick guide to their size and the maximum flow rate attainable (Table 23.1).

In general, the size of cannula is determined by the rate at which the particular drug or fluid needs to be given. In emergency situations this is usually as quickly as possible so a green or grey cannula should be used. The following rule is a good one to remember: 'Short and thick does the trick'.

For routine administration of sedative drugs the rate of administration does not need to be so high so a blue or pink cannula is usually adequate.

The larger cannulae are more difficult to insert and cause more discomfort as they are sited.

Where should the cannula be sited?

The choice of site for cannulation depends on a number of factors:

Table 23.1
Colour coding of cannulae.

Colour	*Size (gauge)*	*Max flow mL/min*
Blue	22G	31
Pink	20G	54
Green	18G	80
Grey	16G	180

1. Ease of access to the site during treatment. This often makes the dorsal surface of the hand the position of choice for dental procedures (Fig. 23.1).
2. Size of cannula to be inserted. It is more difficult and painful to place a large cannula into small veins. This makes the forearm the site of choice (Fig. 23.2).
3. Toxicity of fluid to be given, e.g. intravenous glucose is viscous and relatively toxic due to its osmolarity, so it should be given through a large cannula inserted into a large vein to enable rapid dilution by the blood.
4. Duration of cannulation. For most dental procedures this is short, so it is not so important to keep away from areas of movement, e.g. the joint areas.
5. Availability of veins. This is often the overriding factor in many cases and compromises have to be made in order to gain any form of IV access.
6. Condition of the skin. Do not insert a cannula through skin that is damaged or infected.

Equipment required

Assemble all the necessary equipment before you start. An extra cannula is a good idea, in case you miss on the first attempt:

- gloves
- cannula(s)
- 70% alcohol swab
- saline flush and syringe
- adhesive dressing
- gauze
- tourniquet.

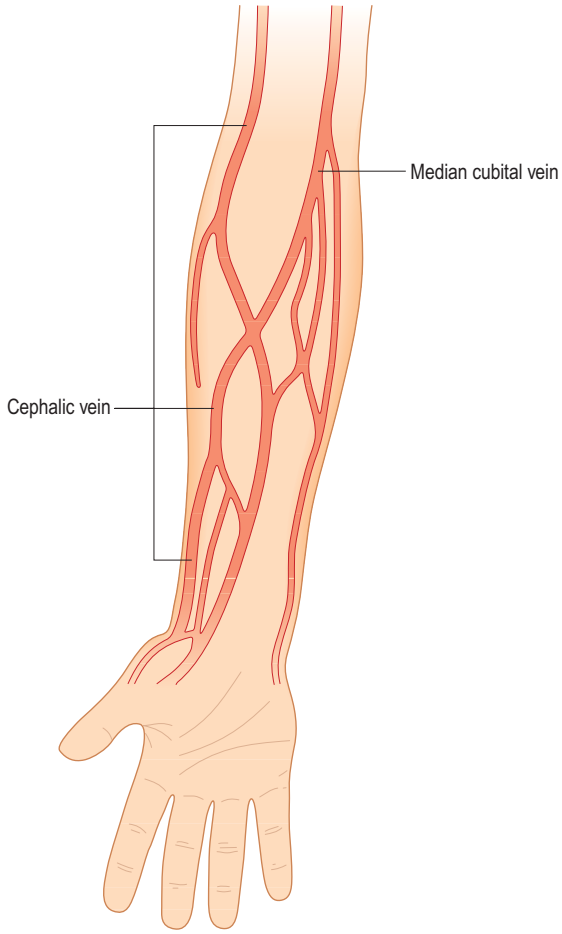


Fig. 23.1 Cannulation of the dorsal venous network or dorsal metacarpal veins of the hand is often convenient for dental procedures but the veins may be small and the insertion is relatively uncomfortable.

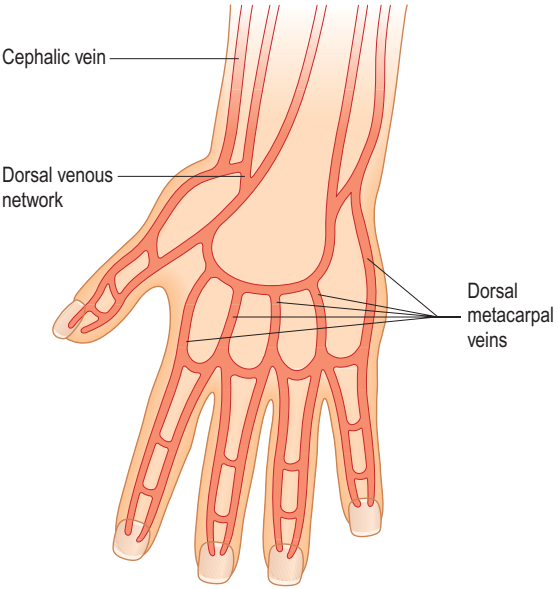


Fig. 23.2 The cephalic vein and median cubital vein of the forearm are suited to larger cannulae and high flow rates, and insertion is more comfortable for the patient. The joint areas are not ideal for long-term cannulation owing to movement and cannula displacement.

Technique

Inspect both hands and arms to select a suitable site. Remember that superficial small veins are often difficult to cannulate.

Apply the tourniquet 20 cm above the proposed insertion point. (This needs to be tight enough to stop venous flow but not arterial flow.) Ask the patient to lower their arm and open clench the fist to activate the muscle pump and fill the veins.

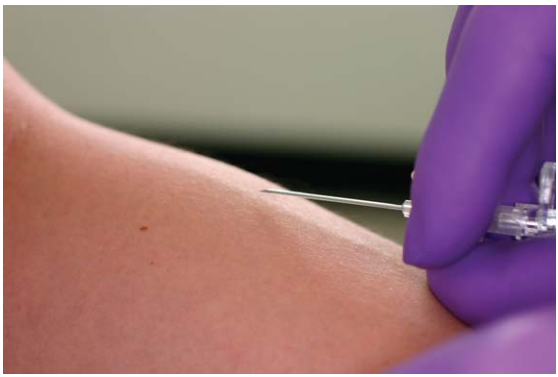
Palpate the veins carefully with your finger tips to evaluate their position, direction, turgidity and mobility. This will also allow you to distinguish springy veins from tendons.

Once you have located the likely site, don clean gloves and clean the area with a 70% alcohol swab. You must allow adequate time for the alcohol to dry otherwise it increases discomfort for the patient and is not effective in reducing bacterial contamination.

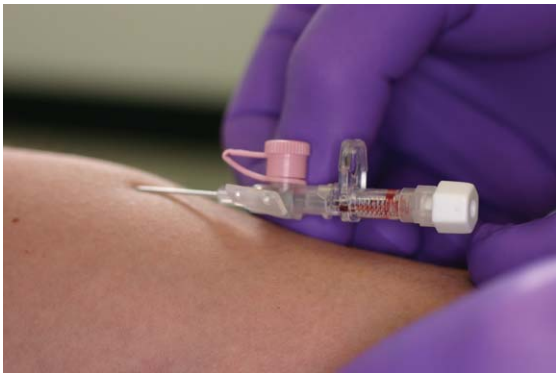
Apply traction to the skin a few centimetres below the insertion site to stabilise the vein (Fig. 23.3A).

Hold the cannula between your thumb, index and middle finger. Insert the cannula in a confident manner through the skin at approximately 30–40° to the surface.

You will often feel the cannula pass through the vein wall and see a flash back of blood in the chamber or connecting tube. At this point stop insertion and withdraw the introducing needle slightly to prevent the cannula passing out of the far side of the vein. Continue to advance the cannula along the vein until fully inserted (Fig. 23.3B).

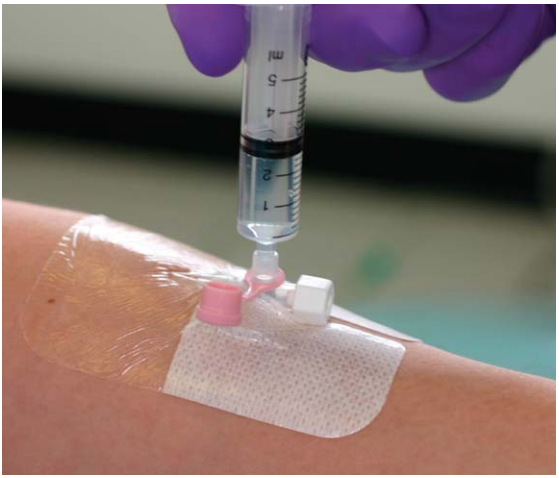


A



B

Fig. 23.3 (A–C) Technique for the insertion of an intravenous cannula into the arm.



C

Fig. 23.3 Cont'd.

Withdraw the introducing needle (dispose of safely in the sharps bin) and in the case of a venflon seal the open port with the plastic stopper provided.

Flatten the wings of the cannula and secure with tape or adhesive dressing to skin (Fig. 23.3C).

As the cannula is flushed with sterile saline look for signs of extravascular injection: pain, swelling and resistance to injection.

The cannula is now ready for use.

Intramuscular injection

It is occasionally necessary to give intramuscular injections of antibiotics and emergency drugs.

Where should you inject?

The best site for use in dental practice is the deltoid muscle of the upper arm. Other sites include the upper outer quadrant of the buttock and the thigh.

Equipment

- Gloves
- Syringe with orange or smaller needle; many of the emergency drugs come pre-assembled with a needle
- 70% alcohol swab
- Gauze
- Adhesive plaster.

Technique

Expose the area to be injected.

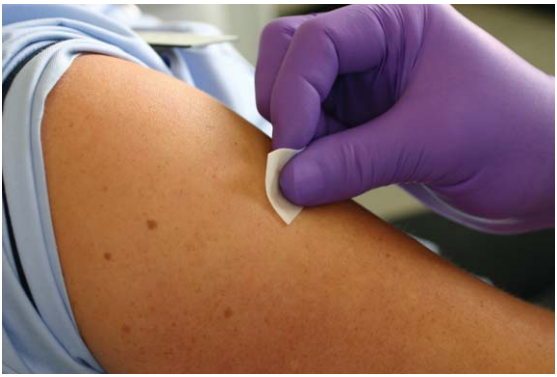
Assemble the syringe and draw up drugs, checking that you have the correct formulation and it is in date.

Wipe the skin with 70% alcohol. You must allow adequate time for the alcohol to evaporate otherwise it increases discomfort for the patient and is not effective in reducing bacterial contamination (Fig. 23.4A).

Squeeze the deltoid muscle between the thumb and fingers of the non-dominant hand. Hold the syringe in the dominant hand for insertion of the needle (Fig. 23.4B).

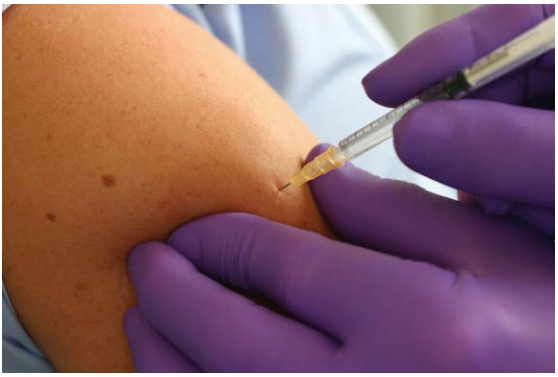
Insert the needle at 90° to skin surface in a confident manner (Fig. 23.4C).

Aspirate to confirm that you are not intravascular before you inject the drug. If there is a flashback of blood into the syringe discard the needle and syringe and start again.

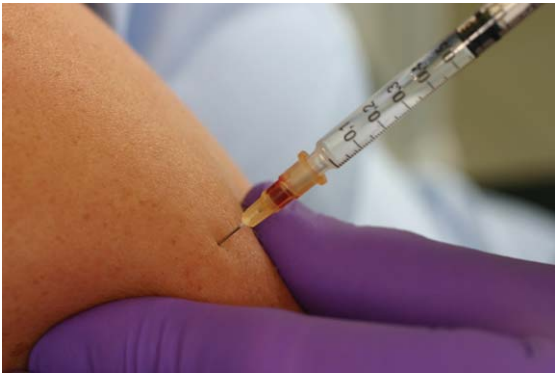


A

Fig. 23.4 (A–C) Technique for an intramuscular injection in the arm.



B



C

Fig. 23.4 Cont'd.

Inject slowly to minimise discomfort. Once the injection is complete remove the needle and apply pressure to the area with gauze. Finally, place an adhesive plaster over the site.

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