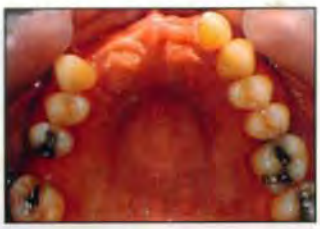
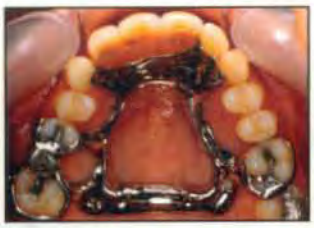
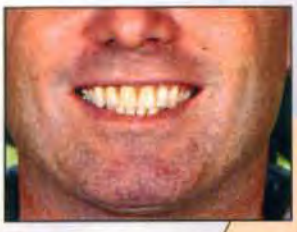
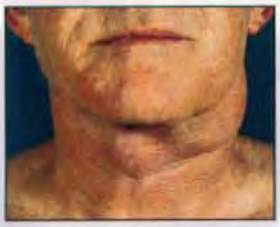
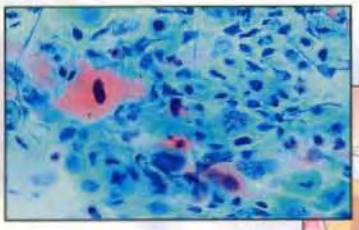
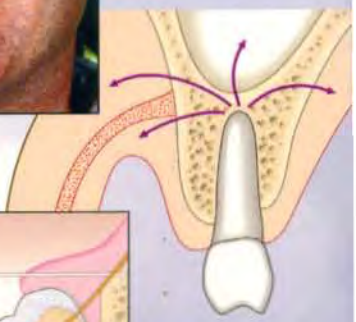
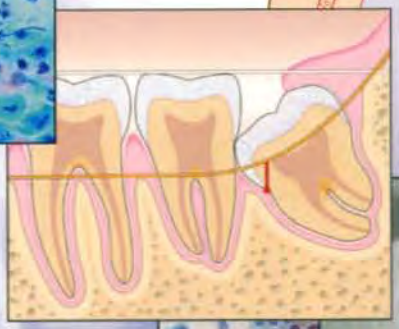
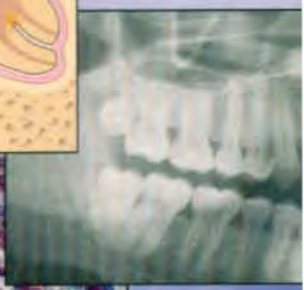
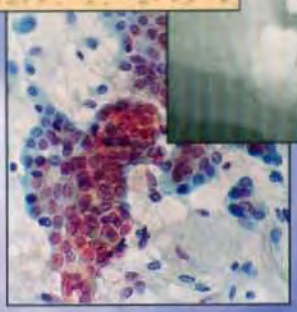
SECOND EDITION

Clinical Problem Solving in Dentistry

EDITED BY
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CHURCHILL LIVINGSTONE

1 A high caries rate

Summary

A 17-year-old sixth-form college student presents at your general dental surgery with several carious lesions, one of which is very large. How should you stabilize his condition?

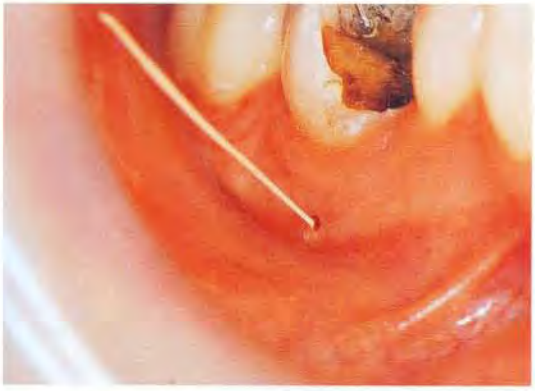


Fig. 1.1 The lower right first molar. The gutta percha point indicates a sinus opening.

HISTORY

Complaint

He complains that a filling has fallen out of a tooth on the lower right side and has left a sharp edge that irritates his tongue. He is otherwise asymptomatic.

History of complaint

The filling was placed about a year ago at a casual visit to the dentist precipitated by acute toothache triggered by hot and cold food and drink. He did not return to complete a course of treatment. He lost contact when he moved house and is not registered with a dental practitioner.

Medical history

The patient is otherwise fit and well.

EXAMINATION

Extraoral examination

He is a fit and healthy-looking adolescent. No submental, submandibular or other cervical lymph nodes are palpable and the temporomandibular joints appear normal.

Intraoral examination

The lower right quadrant is shown in Figure 1.1. The oral mucosa is healthy and the oral hygiene is reasonable. There is gingivitis in areas but no calculus is visible and probing depths are 3 mm or less. The mandibular right first molar is grossly carious and a sinus is discharging buccally. There are no other restorations in any teeth. No teeth have been extracted and the third molars are not visible. A small cavity is present on the occlusal surface of the mandibular right second molar.

■ *What further examination would you carry out?*

Test of tooth vitality of the teeth in the region of the sinus. Even though the first molar is the most likely cause, the adjacent teeth should be tested because more than one tooth might be nonvital. The results should be compared with those of the teeth on the opposite side. Both hot/cold methods and electric pulp testing could be used because extensive reactionary dentine may moderate the response.

The first molar fails to respond to any test. All other teeth appear vital.

INVESTIGATIONS

■ *What radiographs would you take? Explain why each view is required.*

Radiograph	Reason taken
Bitewing radiographs	Primarily to detect approximal surface caries, and in this case also required to detect occlusal caries.
Periapical radiograph of the lower right first molar tooth, preferably taken with a paralleling technique	Preoperative assessment for endodontic treatment or for extraction should it be necessary.
Panoramic tomograph	Might be useful as a general survey view in a new patient and to determine the presence and position of third molars.

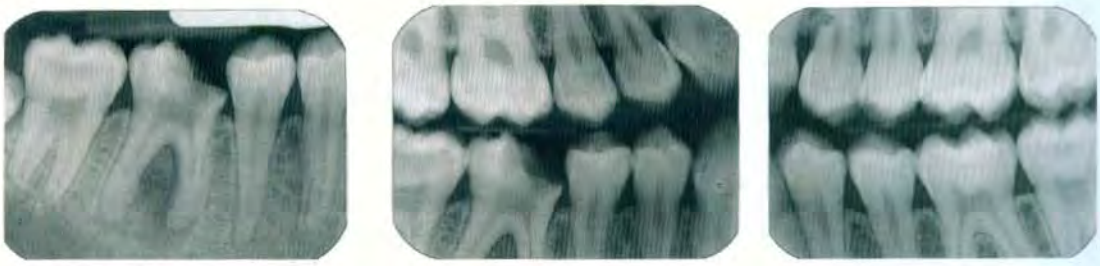


Fig. 1.2 Periapical and bitewing films.

■ ***What problems are inherent in the diagnosis of caries in this patient?***

Occlusal lesions are now the predominant form of caries in adolescents following the reduction in caries incidence over the past decades. Occlusal caries may go undetected on visual examination for two reasons. First, it starts on the fissure walls and is obscured by sound superficial enamel, and secondly lesions cavitate late, if at all, probably because fluoride strengthens the overlying enamel. Superimposition of sound enamel also masks small and medium-sized lesions on bitewing radiographs. The small occlusal cavity in the second molar arouses suspicion that other pits and fissures in the molars will be carious. Unless lesions are very large, extending into the middle third of dentine, they may not be detected on bitewing radiographs.

■ ***The radiographs are shown in Figure 1.2. What do you see?***

The periapical radiograph shows the carious lesion in the crown of the lower right first molar to be extensive, involving the pulp cavity. The mesial contact has been completely destroyed and the molar has drifted mesially and tilted. There are periapical radiolucencies at the apices of both roots, that on the mesial root being larger. The radiolucencies are in continuity with the periodontal ligament and there is loss of most of the lamina dura in the bifurcation and around the apices.

The bitewing radiographs confirm the carious exposure and in addition reveal occlusal caries in all the maxillary and mandibular molars with the exception of the upper right first molar. No approximal caries is present.

■ ***If two or more teeth were possible causes of the sinus, how might you decide which was the cause?***

A gutta percha point could be inserted into the sinus prior to taking the radiograph, as shown in Figure 1.1. A medium- or fine-sized point is flexible but resilient enough to pass along the sinus tract if twisted slightly on insertion. Points are radiopaque and can be seen on a radiograph extending to the source of the infection, as shown in another case in Figure 1.3.

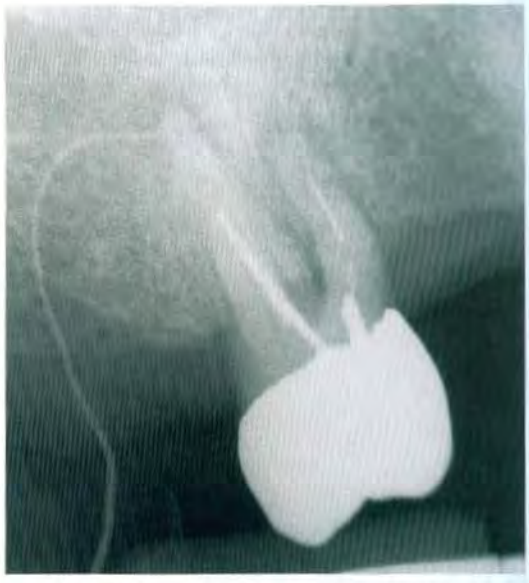


Fig. 1.3 Another case, showing gutta percha point tracing the path of a sinus.

DIAGNOSIS

■ ***What is your diagnosis?***

The patient has a nonvital lower first molar with a periapical abscess. In addition he has a very high caries rate in a previously almost caries-free dentition.

TREATMENT

The patient is horrified to discover that his dentition is in such a poor state, having experienced only one episode of toothache in the past. He is keen to do all that can be done to save all teeth and a decision is made to try to restore the lower molar.

Table 1.3 Dietary advice

Aims	Methods
Reduce the amount of sugar	Check manufacturers' labels and avoid foods with sugars such as sucrose, glucose and fructose listed early in the ingredients. Natural sugars (e.g. honey, brown sugar) are as cariogenic as purified or added sugars. When sweet foods are required, choose those containing sweetening agents such as saccharin, acesulfame-K and aspartame. Diet formulations contain less sugar than their standard counterparts. Reduce the sweetness of drinks and foods. Become accustomed to a less sweet diet overall.
Restrict frequency of sugar intakes to meal times as far as possible	Try to reduce snacking. When snacks are required select 'safe snacks' such as cheese, crisps, fruit or sugar-free sweets, such as mints or chewing gum (which not only has no sugar but also stimulates salivary flow and increases plaque pH). Use artificial sweeteners in drinks taken between meals.
Speed clearance of sugars from the mouth	Never finish meals with a sugary food or drink. Follow sugary foods with a sugar-free drink, chewing gum or a protective food such as cheese.

The patient should be advised to use a fluoride-containing toothpaste. During the period of dietary change it would also be beneficial to use a weekly fluoride rinse as well. This could be continued for as long as the diet is felt to be unsafe.

Oral hygiene instruction is also important, but may be emphasized in a later phase of treatment. It will not stop caries progression, which is critical for this patient, and there is only a mild gingivitis.

■ *Assuming good compliance and motivation, how will you restore the teeth permanently?*

The mandibular right first molar requires orthograde endodontic treatment and replacement of the temporary restoration with a core. Retention for the core can be provided by residual tooth tissue, provided carious destruction is not gross. The restorative material may be packed into the pulp chamber and the first 2–3 mm of the root canal. If insufficient natural crown remains, it may be supplemented with a preformed post in the distal canals. The distal canal is not ideal, being further from the most extensively destroyed area, but it is larger.

The other molar teeth will need to have their temporary restorations replaced by definitive restorations. Caries involved only the occlusal surface but removal of these large lesions has probably left little more than an enamel shell. Restoration of such teeth with amalgam would require removal of all the unsupported, undermined enamel leaving little more than a root stump and a few spurs of tooth tissue. Restoration could be better achieved with a radiopaque glass ionomer and composite hybrid restoration. The glass ionomer used to replace the missing dentine must be radiopaque so that it is not confused with residual or secondary caries on radiographs. A composite linked to dentine with a bonding agent would be an alternative to the glass ionomer.

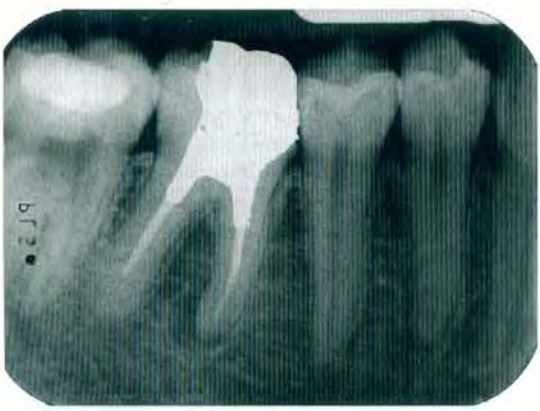


Fig. 1.5 Periapical radiograph of the restored lower first molar.

■ *Figure 1.5 shows the restored lower first molar 2 months after endodontic treatment. What do you see and what long-term problem is evident?*

There is good bone healing around the apices and in the bifurcation. Complete healing would be expected after 6 months to 1 year at which time the success of root treatment can be judged.

As noted in the initial radiographs, the lower right first molar has lost its mesial contact, drifted and tilted. This makes it impossible to restore the normal contour of the mesial surface and contact point. The mesial surface is flat and there is no defined contact point. In the long term there is a risk of caries of the distal surface of the second premolar, and the caries is likely to affect a wider area of tooth and extend further gingivally than caries below a normal contact. The area will also be difficult to clean and there is a risk of localized periodontitis. Tilting of the occlusal surface may also favour food packing into the contact unless the contour of the restoration includes an artificially enhanced marginal ridge.

Feature of lesion	Radiographic finding
Site	Posterior body, angle and ramus of the right mandible.
Size	Large, about 10 × 8 cm, extending from the second premolar, back to the angle and involving all of the ramus up to the sigmoid notch, and from the expanded upper border of the alveolar bone down to the inferior dental canal.
Shape	Multilocular, producing the *soap bubble* appearance.
Outline/edge	Smooth, well defined and mostly well corticated.
Relative radiodensity	Radiolucent with distinct radiopaque septa producing the multilocular appearance. There is no evidence of separate areas of calcification within the lesion.
Effects on adjacent structures	Gross lingual expansion of mandible, expansion buccally is only seen well in the occlusal films. Marked expansion of the superior margin of the alveolar bone and the anterior margin of the ascending ramus. The involved teeth have also been displaced superiorly. The roots of the involved teeth are slightly resorbed, but not as markedly as suggested by the periapical view. The cortex does not appear to be perforated.

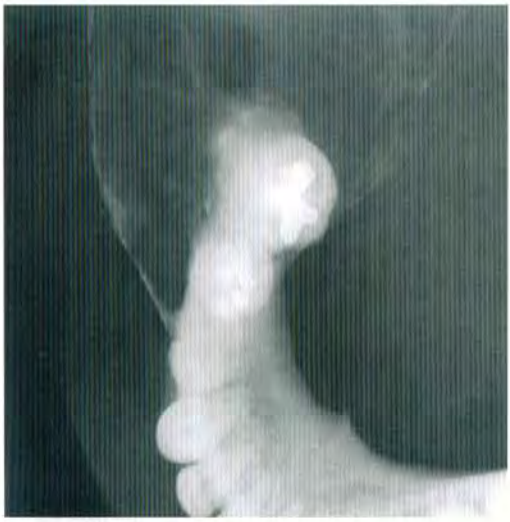


Fig. 2.4 Lower true occlusal view.

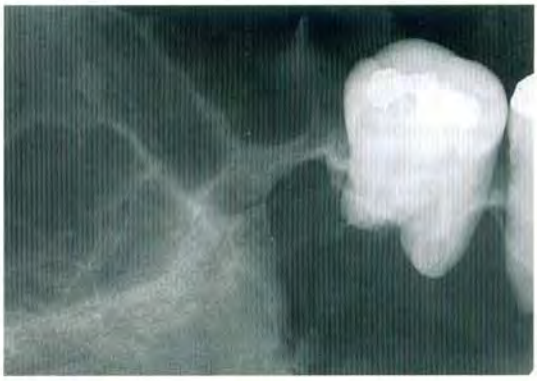


Fig. 2.5 Periapical view of the lower right first permanent molar.

RADIOLOGICAL DIFFERENTIAL DIAGNOSIS

■ *What is your principal differential diagnosis?*

1. Ameloblastoma
2. Giant cell lesion

■ *Justify this differential diagnosis.*

Ameloblastoma classically produces an expansile multilocular radiolucency at the angle of the mandible.

As noted above, it most commonly presents at the age of this patient and is commoner in his racial group. The radiographs show the typical multilocular radiolucency, containing several large cystic spaces separated by bony septa, and the root resorption, tooth displacement and marked expansion are all consistent with an ameloblastoma of this size.

A giant cell lesion. A central giant cell granuloma is possible. Lesions can arise at almost any age but the radiological features and site are slightly different, making ameloblastoma the preferred diagnosis. Central giant cell granuloma produces an expansile and sometimes apparently multilocular radiolucency, but there would be no root resorption and the lesion may be less radiolucent (because it consists of solid tissue rather than cystic neoplasm), often containing wispy osteoid or fine bone septa subdividing the lesion into a *honeycomb*-like pattern. However, these typical features are not always seen. The spectrum of radiological apearances ranges from lesions which mimic odontogenic and solitary bone cysts to those which appear identical to ameloblastoma or other odontogenic tumours. The aneurysmal bone cyst is another giant cell lesion which could produce this radiographic appearance with prominent expansion. Adjacent teeth are usually displaced but rarely resorbed. However, aneurysmal bone cyst is much rarer than central giant cell granuloma in the jaws.

■ *What types of lesion are less likely and why?*

Several lesions remain possible but are less likely either on the basis of their features or relative rarity.

2 A multilocular radiolucency

Summary

A 45-year-old African man presents in the accident and emergency department with an enlarged jaw. You must make a diagnosis and decide on treatment.

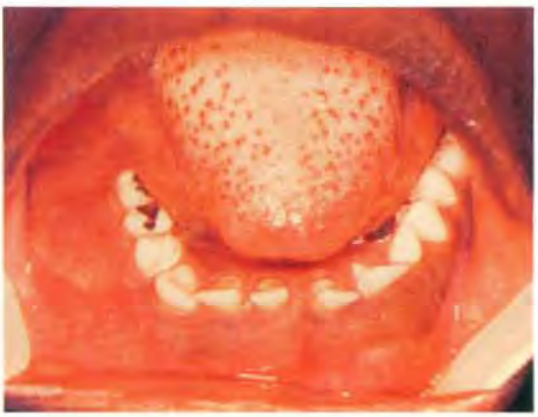


Fig. 2.1 The patient on presentation.

HISTORY

Complaint

The patient's main complaint is that his lower back teeth on the right side are loose and that his jaw on the right feels enlarged.

History of complaint

The patient has been aware of the teeth slowly becoming looser over the previous 6 months. They seem to be 'moving' and are now at a different height from his front teeth, making eating difficult. He is also concerned that his jaw is enlarged and there seems to be reduced space for his tongue. He has recently had the lower second molar on the right extracted. It was also loose but extraction does not seem to have cured the swelling. Although not in pain, he has finally decided to seek treatment.

Medical history

He is otherwise fit and healthy.

EXAMINATION

Extraoral examination

He is a fit-looking man with no obvious facial asymmetry but a slight fullness of the mandible on the right. Palpation reveals a smooth rounded bony hard enlargement on the buccal and lingual aspects. Deep cervical lymph nodes are palpable on the right side. They are only slightly enlarged, soft, not tender and freely mobile.

Intraoral examination

■ *What do you see in Figure 2.1?*

There is a large swelling of the right posterior mandible visible in the buccal sulcus, its anterior margin relatively well defined and level with the first premolar. The lingual aspect is not visible but the tongue appears displaced upwards and medially suggesting significant lingual expansion. The mucosa over the swelling is of normal colour, without evidence of inflammation or infection. There are two relatively small amalgams in the lower right molar and second premolar

If you could examine the patient you would find that all his upper right posterior teeth are extracted and that the lower molar and premolars are 2–3 mm above the height of the occlusal plane. Both teeth are grade 3 mobile but both are vital.

■ *What are the red spots on the patient's tongue?*

Fungiform papillae. They appear more prominent when the tongue is furred, as here, for instance when the diet is not very abrasive.

■ *On the basis of what you know so far, what types of condition would you consider to be present here?*

The history suggests a relatively slow-growing lesion, which is therefore likely to be benign. While this is not a definitive relationship, there are no specific features suggesting malignancy, such as perforation of the cortex, soft tissue mass, ulceration of the mucosa, numbness of the lip or devitalization of teeth. The character of the lymph node enlargement does not suggest malignancy.

The commonest jaw lesions which cause expansion are the odontogenic cysts. The commonest odontogenic cysts are the radicular (apical inflammatory) cyst, dentigerous cyst and odontogenic keratocyst. If this is a radicular cyst it could have arisen from the first molar, though the occlusal amalgam is relatively small and there seems no reason to

Radiograph view	Reason
Panoramic tomograph or an oblique lateral	To show the lesion from the lateral aspect. The oblique lateral would provide the better resolution but might not cover the anterior extent of this large lesion. The panoramic tomograph would provide a useful survey of the rest of the jaws but only that part of this expansile lesion in the line of the arch will be in focus. An oblique lateral view was taken.
A posterior-anterior (PA) of the jaws	To show the extent of mediolateral expansion of the posterior body, angle or ramus.
A lower true (90°) occlusal	To show the lingual expansion which will not be visible in the PA jaws view because of superimposition of the anterior body of the mandible.
A periapical of the lower right second premolar and the first molar	To assess bone support and possible root resorption.

suspect that the tooth is nonvital. A residual radicular cyst arising on the extracted second or third molar would be a possibility. A dentigerous cyst could be the cause if the third molar is unerupted. The possibility of an odontogenic keratocyst seems unlikely, because these cysts do not normally cause much expansion. An odontogenic tumour is a possible cause and an ameloblastoma would be the most likely one, because it is the commonest, and arises most frequently at this site and in this age group. There is a higher incidence in Africans. An ameloblastoma is much more likely than an odontogenic cyst to displace the teeth and make them grossly mobile. A giant cell granuloma and numerous other lesions are possibilities but are all less likely.

INVESTIGATIONS

■ ***Radiographs are obviously indicated. Which views would you choose? Why?***

Several different views are necessary to show the full extent of the lesion. These are listed in the 'Radiograph view' box above.

■ ***These four different views are shown in Figures 2.2–2.5. Describe the radiographic features of the lesion (shown in 'Feature of lesion' box on p. 11).***

■ ***Why do the roots of the first molar and second premolar appear to be so resorbed in the periapical view when the oblique lateral view shows minimal root resorption?***

The teeth are foreshortened because they lie at an angle to the film. This film has been taken using the bisected angle technique and several factors contribute to the distortion:

- the teeth have been displaced by the lesion, so the crowns lie more lingually, and the roots more buccally;
- the lingual expansion of the jaw makes film packet placement difficult, so it has had to be severely angulated away from the root apices;
- failure to take account of these two factors when positioning and angling the X-ray tubehead.

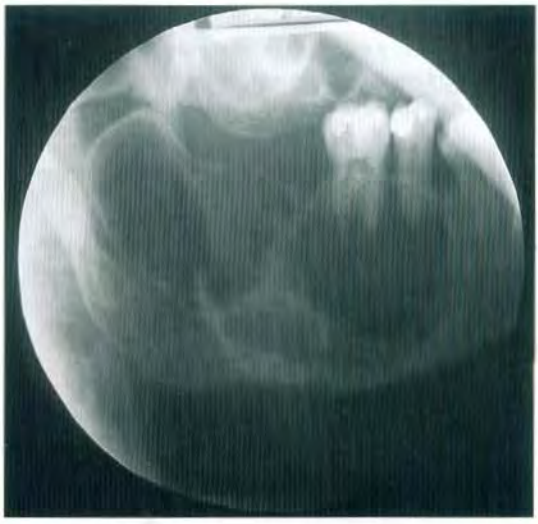


Fig. 2.2 Oblique lateral view.

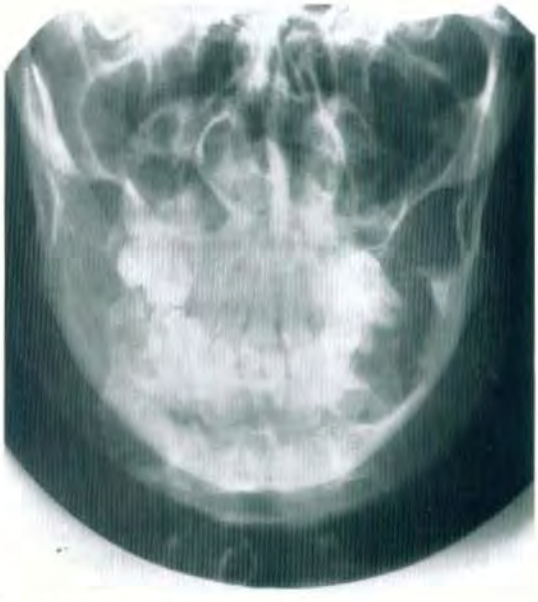


Fig. 2.3 Posterior-anterior view of the jaws.

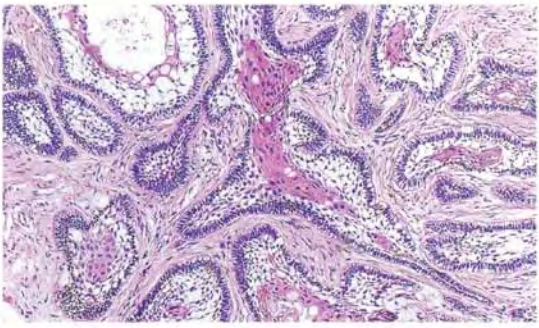


Fig. 2.6 Histological appearance of biopsy at low power.

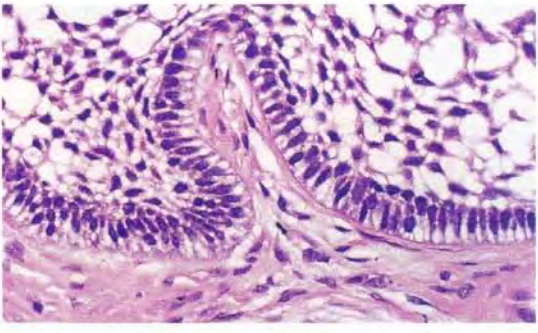


Fig. 2.7 Histological appearance of biopsy at high power.

TREATMENT

■ *What treatment will be required?*

The ameloblastoma is classified as a benign neoplasm. However, it is locally invasive and in some cases permeates the medullary cavity around the main tumour margin. Ameloblastoma should be excised with a 1 cm margin of normal bone and around any suspected perforations in the cortex. If ameloblastoma has escaped from the medullary cavity, it may spread extensively in the soft tissues and requires excision with an even larger margin. The lower border of the mandible may be intact and is sometimes left in place to avoid the need for full thickness resection of the mandible and a bone graft. This causes a low risk of recurrence, but such recurrences are slow growing and may be dealt with conservatively after the main portion of the mandible has healed. The fact that the ameloblastoma is of the follicular pattern is of no significance for treatment.

■ *What other imaging investigations would be appropriate for this patient?*

In order to plan the resection accurately, the extent of the tumour and any cortical perforations must be identified. Computed tomography (CT) and/or magnetic resonance imaging (MRI) would show the full extent of the lesion in bone and surrounding soft tissue respectively.

3 An unpleasant surprise

Summary

A 30-year-old lady develops acute shortness of breath following administration of amoxicillin. What would you do?

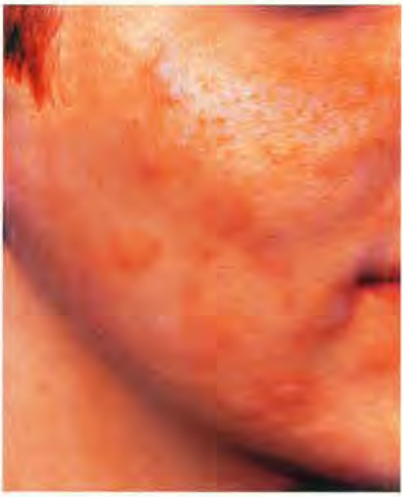


Fig. 3.1 The patient's face as she starts to feel unwell.

HISTORY

Complaint

The patient complains that she feels unwell, hot and breathless.

History of complaint

The patient has an appointment for routine dental treatment involving scaling and a restoration under local anaesthesia and antibiotic prophylaxis. She took a 3 g oral dose of amoxicillin 45 minutes ago.

Medical history

You checked the medical history before administering the amoxicillin and so you know that the patient is a well-controlled asthmatic taking salbutamol on occasions. She also suffers from eczema, as do her mother and her two children, and uses a topical steroid cream as required. The patient has a confirmed heart murmur requiring antibiotic cover.

Dental history

The patient has been a regular attender for a number of years but has not previously received antibiotic cover for dental treatment. She has had previous courses of penicillin from her general medical practitioner for chest infections.

■ *What is the likely diagnosis?*

Anaphylaxis, arising from hypersensitivity to the amoxicillin.

EXAMINATION

■ *The patient's face is shown in Figure 3.1 What do you see?*

There is patchy erythema. In the most inflamed areas there are well-defined raised oedematous weals, for instance at the corner of the mouth and on the side of the chin. This is a typical urticarial rash and indicates a type 1 hypersensitivity reaction.

■ *What would you do immediately?*

- Reassure the patient.
- Assess the vital signs including blood pressure, pulse and respiratory rate.
- Call for help.
- Obtain oxygen and your practice emergency drug box.

■ *What are the signs and symptoms of anaphylaxis?*

The signs and symptoms vary with severity. The classical picture is of:

- a red urticarial rash
- oedema that may obstruct the airway
- hypotension due to reduced peripheral resistance
- hypovolaemia due to the movement of fluid out of the circulation into the tissues
- small airways obstruction.

■ *What does urticarial mean?*

The word urticarial comes from the Latin for nettle rash. An urticarial rash has superficial oedema that may form separate flat raised blister-like patches (as in Fig. 3.1) or be diffuse. In the head and neck it is often diffuse because the tissues are lax. Markedly oedematous areas may become pale by compression of their blood supply but the background is erythematous. Patients often know an urticarial rash by the lay term *hives*.

■ *What is the pathogenesis of anaphylaxis?*

Anaphylaxis is an acute type 1 hypersensitivity reaction triggered in a sensitized individual by an allergen. The allergen enters the tissues and binds to immunoglobulin E (IgE) that is already bound to the surface of mast cells, present in almost all tissues. Binding of allergen to IgE induces degranulation and the release of large amounts of inflammatory mediators, particularly histamine. This causes the vasodilatation, increased capillary permeability and bronchospasm.

■ *Type 1 hypersensitivity is also known as immediate hypersensitivity but onset was delayed for 45 minutes. Why?*

Acute anaphylactic reactions may occur within seconds or may be delayed for up to an hour depending on the nature of the allergen and the route of exposure. It takes time for an oral dose of antibiotic to be absorbed and pass through the circulation to the tissues, in this case 45 minutes. The reaction would be expected about 30 minutes after intramuscular administration of an allergen but almost instantaneously after intravascular administration. The time of onset is unpredictable. Some allergens such as peanuts and latex can cause rapid reactions despite being applied topically. The variability in onset of reactions explains why patients should be observed for an hour after administration of antibiotic cover.

On examining for the signs noted above you discover that the patient is breathless and a wheeze can be heard during both inspiration and expiration indicating small airways obstruction. She feels hot and has a pulse rate of 120 beats per minute and blood pressure of 120/80 mmHg. She is conscious but the effects are becoming more severe and the rash now affects all the face and neck region and has spread onto the upper aspect of the thorax. The appearance of one arm is shown in Figure 3.2.

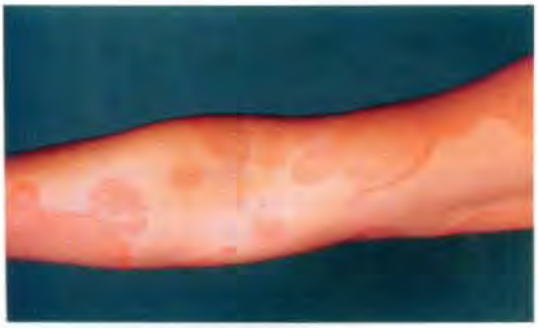


Fig. 3.2 The patient's arm 5 minutes later.

TREATMENT

■ *What treatment would you perform?*

Allow the patient to adopt the most comfortable position for breathing and give oxygen (5 litres per minute) by facemask.

Because there is bronchospasm, give the following drugs in order:

Adrenaline (epinephrine) 1:1000, 500 micrograms intramuscularly. The easiest form to administer is a preloaded 'EpiPen' or 'Anapen', which are available for both adults (300 micrograms/dose) and children (150 micrograms/dose). Alternatively, a Min-I-Jet prepacked syringe and needle assembly or a standard vial of adrenaline solution, both containing 1 milligram in 1 millilitre (1:1000), may be used. However, both of these latter methods require a delay in administration to prepare the injection. You need to be familiar with whichever form is held in your practice as delay in calculating doses and volumes is clearly undesirable. Adrenaline (epinephrine) may also be given subcutaneously but the absorption is slower and this route is no longer recommended. Note that autoinjectors are designed for self-administration and so provide a slightly lower dose than is recommended.

Chlorphenamine (chlorpheniramine) 10 mg intravenously will counteract the effects of histamine.

Hydrocortisone 100–200 mg intravenously or intramuscularly.

Intravenous fluid. Only required if hypotension develops. A suitable regime would be 1 litre of normal saline infused over 5 minutes with continuous monitoring of the vital signs.

The presentation of drugs useful for anaphylaxis is shown in Figure 3.3.

■ *Why must the drugs be given in this order?*

Adrenaline is the life-saving drug and must be given straight away, before circulatory collapse. It is rapidly acting. Chlorphenamine (chlorpheniramine) is less potent and slower acting and cannot alone counteract pulmonary oedema or bronchospasm, which indicate a severe reaction. Hydrocortisone is the lowest priority; it takes up to 6 hours to act and is not immediately life saving.

■ *After giving all three drugs, the patient recovers. What would you do next?*

- Abandon dental treatment.
- Continue to monitor the vital signs.
- Continue to administer oxygen.
- Arrange transfer of the patient to an appropriate secondary care facility.
- Advise the patient of the need for formal investigation of their probable allergy.

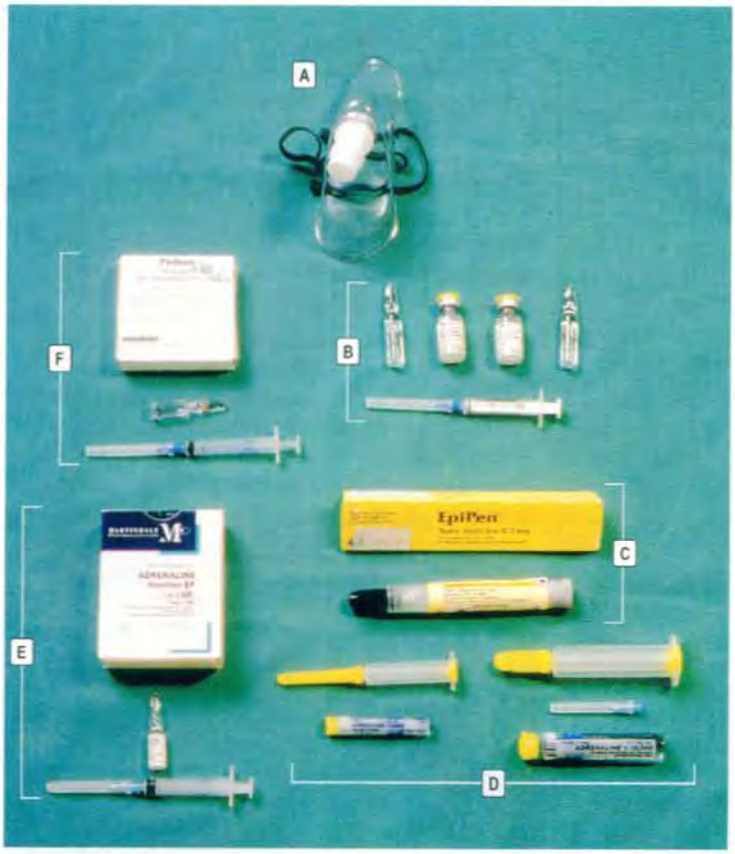


Fig. 3.3 Typical presentations of drugs used to treat anaphylaxis.
A. Oxygen mask.
B. Hydrocortisone. Vials of lyophilised powder for reconstitution in water for injection, NOT saline. Adminster with a conventional syringe and needle.
C. Adrenaline* in an Epipen disposable autoinjector spring-loaded syringe, boxed, and below with the plastic covers removed from each end. Press directly onto the skin and the spring-loaded needle is unsheathed and the drug is injected automatically. A similar device, the Anapen, has a spring-loaded needle that springs out when a button at the opposite end is pressed. Both deliver 300 micrograms of adrenaline.
D. Adrenaline in Min-I-Jet format. The yellow plastic cover is removed from the back (right hand end) of the syringe barrel and front of the glass cartridge and the cartridge is screwed into the syringe barrel. Available in two types, with needle fitted (left, recommended) and with luer lock fitting for a conventional needle (slower to use). After removing front cover and fitting needle, if required, use as a conventional syringe. Versions with finer needles for subcutaneous administration are available but the intramuscular route is preferred and the version with the larger 21 gauge needle should be used.
E. Adrenaline as traditional ampoule, ready to inject with a conventional syringe.
F. Chlorpheniramine as traditional ampoule, ready to inject with a conventional syringe.

**Note that epinephrine is now the recommended name for adrenaline internationally but that adrenaline is still the most widely used name in the UK.*

■ *Can you relax now the immediate crisis is over?*

No, definitely not. The response of the patient needs to be closely observed. Adrenaline (epinephrine) is highly effective but has a very short half-life. Recurrence of bronchospasm, a drop in blood pressure or worsening oedema indicates a need for further adrenaline (epinephrine). This is likely to be needed about 5 minutes after the previous administration and it can be repeated again as often as necessary. However, the chlorphenamine (chlorpheniramine) will start to become effective and no more than two doses of adrenaline (epinephrine) should be necessary.

Late relapse, hours later, is also possible. Mast cells also release other potent inflammatory mediators and some have long half-lives.The hydrocortisone prevents this late relapse.

■ *Can an anaphylactic reaction be controlled without adrenaline (epinephrine)?*

If the only features are a rash and mild swelling not involving the airway it may be appropriate to give chlorphenamine (chlorpheniramine) and hydrocortisone in the first instance and observe the response. However, if bronchospasm, hypotension or oedema around the airway develops, adrenaline (epinephrine) will be needed. Adrenaline (epinephrine) should be administered as early as possible to be effective and it is better not to delay unless the signs and symptoms are very mild.

FURTHER POINTS

■ *Why is adrenaline (epinephrine) effective?*

Adrenaline (epinephrine) is the prototypical adrenergic agonist and has both alpha and beta receptor activity. Alpha receptor-mediated action on arterioles causes vasoconstriction and thus reverses oedema. Beta receptor-mediated actions include increasing the cardiac output by increasing the force of contraction and heart rate (beta 1) and bronchodilatation (beta 2). Mast cell degranulation is also suppressed.

4 Gingival recession

Summary

A 30-year-old woman has gingival recession. Assess her condition and discuss treatment options.

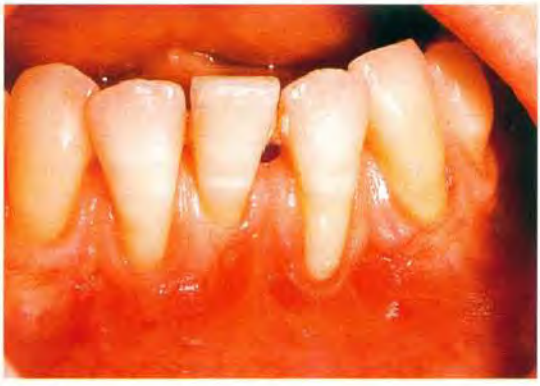


Fig. 4.1. The appearance of the lower incisors.

HISTORY

Complaint

The patient is worried about the gingival recession around her lower front teeth, which she feels is worsening.

History of complaint

She remembers noticing the recession for at least the previous 5 years. She thinks it has worsened over the last 12 months. There has recently been some sensitivity to hot and cold and gingival soreness, most noticeably on toothbrushing or eating ice cream.

Dental history

The patient has been a patient of your practice for about 10 years and you have discussed her recession at previous visits and reassured her. She has a low caries rate and generally good oral hygiene.

Medical history

She is a fit and healthy individual and is not a smoker.

■ *What further specific questions would you ask to help identify a possible cause?*

How often do you brush your teeth? Provided brushing is effective, cleaning once a day is sufficient to maintain gingival health. However, most patients clean two or three times each day and some brush excessively in terms of frequency, duration and force used. Trauma from brushing is considered a factor in some patients' recession, and recession may indicate a need to reduce the frequency and duration of cleaning while maintaining its effectiveness. In this instance the patient has a normal toothbrushing habit but should clean no more than twice each day and for a sensible period of time.

Have you had orthodontic treatment? A lower incisor is missing, suggesting that some intervention may have taken place. Fixed orthodontics in the lower labial segment is occasionally associated with gingival recession in patients with thin buccal gingiva, narrow alveolar processes and correction of severe crowding. Plaque control may be compromised during the wearing of an orthodontic appliance and, even over a relatively short period, this can contribute to the problem. In this instance the patient had undergone extraction of the incisor but had not worn an appliance.

EXAMINATION

Intraoral examination

■ *The appearance of the lower incisors is shown in Figure 4.1. What do you see?*

— Missing lower left central incisor.
— Unrestored teeth.
— No plaque is visible except for a small amount at the cervical margin of the lower left lateral incisor.
— Gingival recession affecting all lower incisors and, to a lesser extent, the lower canines.
— Apart from the abnormal contour, the buccal gingivae are pink and healthy and the interdental papillae are normal.
— Reduction in width of keratinized (cornified) attached gingival epithelium. In places, attached gingiva appears absent.

■ *What clinical assessments would you make, how would you make them and why are they important?*

See Table 4.1.

Table 4.2 Alternative treatment

Treatment	Effectiveness
Mucogingival surgery to correct the recession, either a lateral pedicle graft, double papilla flap, or a coronally repositioned flap. These may be used in conjunction with an interpositional (subepithelial) connective tissue graft. These are essentially cosmetic operations.	May be effective in carefully selected cases. The presence of adjacent interdental papillae and suitable donor sites is essential. Total root coverage is difficult to achieve and unpredictable, especially in the long term.
Mucogingival surgery to provide a wider and functional zone of attached gingiva. This therapeutic procedure provides a zone of thicker tissue which is more resistant to further recession and less prone to soreness with normal brushing. A free gingival graft is the treatment of choice.	Highly effective. Grafting palatal mucosa into the alveolar mucosa prevents the lip pulling the gingiva from the teeth. Even if the gingival margin has little attached gingiva, it can remain healthy if protected from displacement or other trauma.
Provision of a thin acrylic gingival stent or veneer.	Can provide an excellent cosmetic result if well made, but only considered for extensive recession in highly visible areas. The usual indication is the upper incisors following periodontal surgery with loss of papillae. Rarely used and not applicable to this case.

casts are very helpful and should be repeated at intervals.

- Treat the dentine hypersensitivity. Recession alone should not be painful. Ensure that the exposed root surface is suffering neither early caries nor erosion. Check the diet for sugars, acid drinks and foods and apply topical antihypersensitivity agents. This is another reason to perfect the cleaning of these teeth.

In this case the patient maintained good plaque control but the recession worsened slowly over a period of several years until there was a lack of functional attached gingiva.

■ ***What other treatments might be possible? Are they effective?***

Table 4.2 shows alternative treatments.

In this case a free gingival graft was placed and the result is shown in Figure 4.2

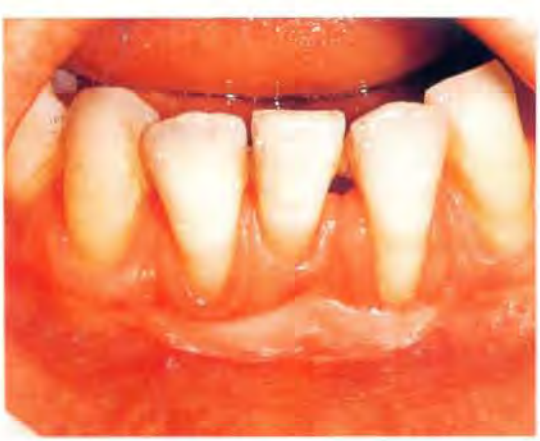


Fig. 4.2 Appearance of the free gingival graft 6 months after placement.

■ ***What do you see; is the graft successful?***

Yes, the graft appears successful. Palatal connective tissue and overlying epithelium has been placed apical to the lower incisor gingival margin to provide a wider zone of attached keratinized gingiva. Because the palatal connective tissue is transferred the epithelium retains its keratinized palate structure.

■ ***Does the graft need to lie at the gingival margin?***

No. The graft forms the gingival margin on the lower left lateral incisor but elsewhere lies below the margin. Provided the graft is firmly bound down to the underlying tissue it will stabilize the gingival margin against displacement on lip movement.

■ ***Why not place the graft over the root as well and correct the recession?***

As noted in Table 4.2, surgery to correct the recession itself is difficult to achieve and unpredictable, especially in the long term. The root surface does not provide a nutrient bed on which the free graft can survive. Grafts in this situation would have to be pedicled to ensure their nutrient supply and also need to be placed so that they receive some nutrient from an adjacent exposed connective tissue bed. A more predictable result may be obtained by using an interpositional (subepithelial) connective tissue graft. A free graft is most unlikely to be successful if simply placed over the root surface.

■ ***Figure 4.3 shows a different patient with recession. What does the appearance tell you?***

There is approximately 4 mm of recession buccally on the lower right canine. Apical to the gingival margin there is a hole in the gingival tissue. Plaque and subgingival calculus

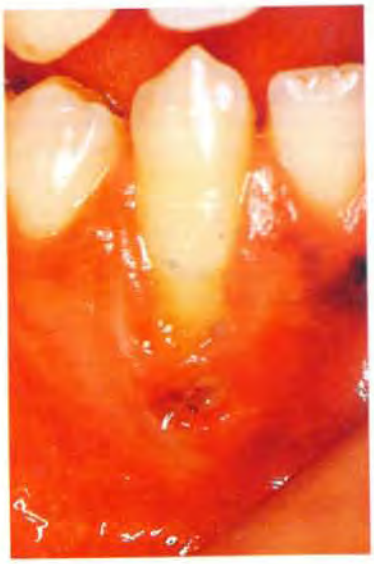


Fig. 4.3 A different patient.

(formed within a periodontal pocket) are visible and the tissue is inflamed. The small 'bridge' of tissue at the gingival margin is not attached to the tooth surface and will eventually break down. In this case the recession is secondary to pocket formation in a plaque-induced periodontitis. Inflammation associated with subgingival calculus has caused loss of much of the buccal bone.

■ ***How would treatment of this patient's recession differ?***

It would differ only in the early stages. Inflammation must be treated by oral hygiene improvement and subgingival debridement. If, after a period to allow healing, there is resolution of inflammation, the situation is very similar to that in the first case and assessment and treatment would be identical. There would be no value in attempting to surgically correct the fenestration in the attached gingiva. As discussed above, grafting onto the root surface is technically complex and success is unpredictable.

Summary

A 9-year-old boy is referred to you in the orthodontic department with an unerupted upper left central incisor. What is the cause and how may it be treated?

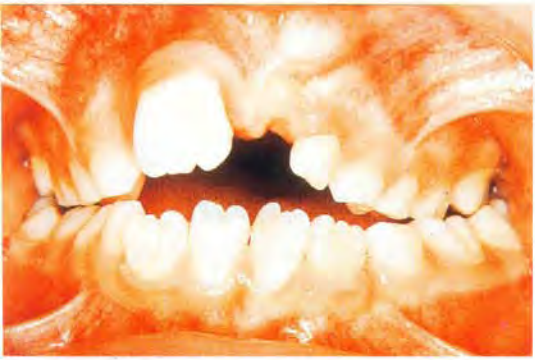


Fig. 5.1 The appearance of the patient on presentation.

HISTORY

Complaint

The patient's upper left central incisor has not erupted although he is 9 years old. His mother is very concerned about her son's appearance and is anxious for him to be treated.

History of complaint

The upper left deciduous predecessor had been present until about 4 months ago. It was extracted by the patient's general dental practitioner in an attempt to speed up the eruption of the permanent successor. Despite this, there has been no change in appearance. The upper permanent central incisor on the opposite side erupted normally at 7 years of age.

Medical history

The patient has suffered from asthma since he was 4 years old. This is controlled using salbutamol (Ventolin).

■ ***The appearance of the mouth is shown in Figure 5.1. What do you see?***

The patient is in the early mixed dentition stage and the teeth present are:

6EDCB1	BCDE6
6EDC21	12 DE6

No upper left central incisor is present, but there is a pale swelling high in the upper labial sulcus above the edentulous space and the upper left B. There has been some loss of space in the region of the absent upper central incisor.

There is a tendency to an anterior open bite which is slightly more pronounced on the right.

There is mild upper and lower arch crowding and a unilateral crossbite on the left. If you were able to examine the patient you would discover that this is associated with a lateral displacement of the mandibular position. The lower centre line is shifted to the left.

There are no restorations but the mouth is not very clean.

■ ***What are the possible causes of an apparently absent upper central incisor?***

The incisor may be missing or have failed to erupt. Possible causes include the following:

Missing	Developmentally absent Extracted Avulsed
Failure to erupt	Dilaceration and/or displacement as a result of trauma Scar tissue preventing eruption Supernumerary tooth preventing eruption Insufficient space as a result of crowding Pathological lesion (e.g. cyst or odontogenic tumour)

■ ***What specific questions would you ask the parents?***

The most important questions are related to trauma. Avulsion or dilaceration would follow significant trauma which is likely to be recalled by the parent. The parent should be asked whether the deciduous predecessor was discoloured. If it was this would provide evidence of loss of vitality, perhaps related to trauma. Extraction would be unusual and a cause should be readily obtained in the history.

Table 5.1 Radiographic views and their purposes

View	Reason
Dental panoramic tomograph	To provide a general view of the developing dentition and establish the presence or absence of the permanent teeth.
Upper standard occlusal or periapicals of the edentulous area, taken with a paralleling technique	To provide a more detailed view of the region, in particular the root morphology and any adjacent structures such as supernumerary teeth or pathological lesions. These may lie outside the focal trough of the tomograph or be obscured by superimposition of other structures in the panoramic view. If periapical views are taken they should include the adjacent teeth in case these were damaged in the original accident. In addition the standard occlusal and panoramic view can be used together to establish the relationship of unerupted structures relative to the dental arch, using the principle of (vertical) parallax. Objects lying nearer to the X-ray tube (labially positioned) appear to move in the opposite direction to the tube relative to a fixed point. Those further away (palatally positioned) appear to move in the same direction as the tube.
Lateral view	Confirms the presence of any distortion of the tooth, if dilacerated, and confirms the relationship of the tooth to the labial swelling in a third dimension.

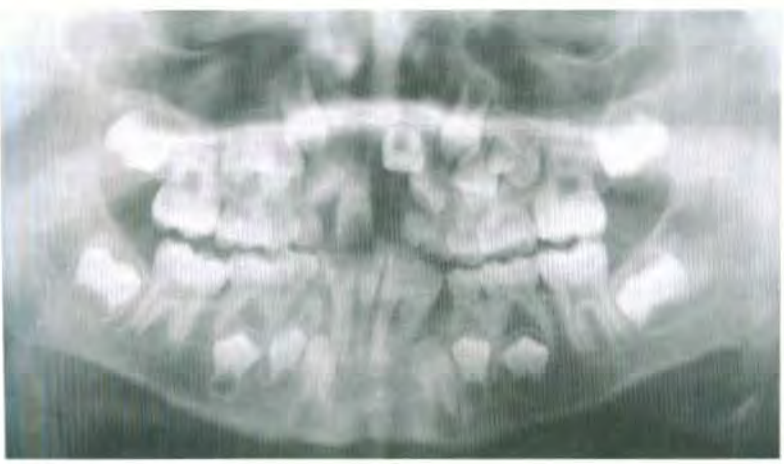


Fig 5.2 Dental panoramic tomograph.

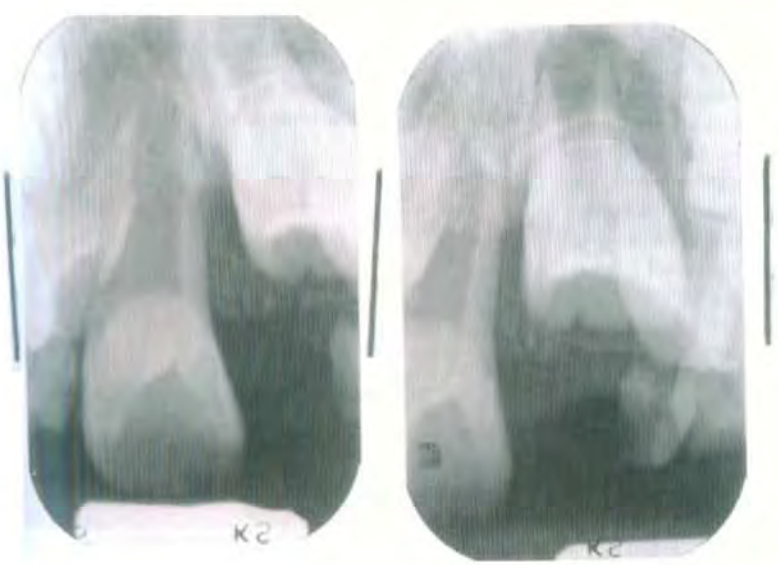


Fig 5.3 Periapical views.

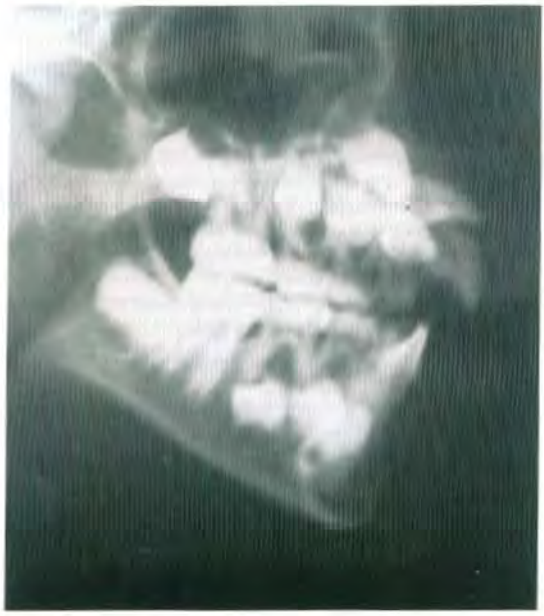


Fig 5.4 Lateral view.

■ *What is your final diagnosis?*

The upper left central incisor is dilacerated, probably as a result of intrusion of the deciduous predecessor in the injury sustained in infancy.

TREATMENT

■ *What are the options for treatment?*

If the dilaceration were severe, the tooth would require extraction. Then either of the following options could be selected:

1. Align the adjacent teeth, ideally with fixed appliances, using the central incisor space. The lateral incisor would replace the central incisor and could be masked to simulate it. In the short term this could be accomplished by adhesive restoration but in the longer term a permanent restoration would be necessary. The canine might also need restoration or masking so that it would not appear incongruous, especially in a patient with slender lateral incisors. This option is not ideal because the final appearance is often poor.
2. Immediate replacement of the extracted central incisor by a denture or adhesive bridge with permanent restoration or possibly a single tooth implant in adulthood (see Case 30).

If, on the radiographs, the dilaceration does not appear to be too severe or lies in the apical portion of the root, consideration could be given to aligning the tooth orthodontically. This would involve regaining any lost space followed by localized surgical exposure of the crown of the tooth and applying extrusive traction with an orthodontic appliance.

■ *What factors affect the selection of a particular treatment?*

- Position and severity of the dilaceration (see above)
- The size of overjet
- Degree of crowding
- Position and condition of the other permanent teeth
- The general condition of the mouth
- The attitude of the child and parent

■ *Assuming none of these factors prevents the ideal treatment, what would you recommend for this case?*

In this case the ideal treatment is to extrude and align the dilacerated tooth into the arch.

The dilaceration appears to be in the root and relatively mild. Therefore, an attempt should be made to regain the lost space to accommodate the central incisor crown. This would be best achieved by extraction of both upper Cs and the upper left B to encourage eruption of permanent lateral incisors. Some months later the dilacerated tooth should be surgically exposed and an orthodontic attachment with a length of gold chain placed on its palatal surface for extrusion.

■ *Should a fixed or removable appliance be used?*

As the tooth movements are relatively simple an upper removable appliance can be used at this stage. More control and more accurate tooth positioning would be achieved with a fixed appliance. However, the patient will probably require further fixed appliance treatment at a later age and the fine adjustment of tooth position could be performed then.

■ *Design a suitable removable appliance.*

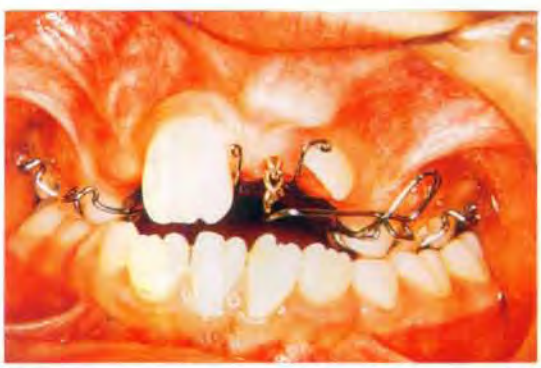


Fig. 5.5 The fitted extrusion appliance.

The appliance consists of:
— cribs on D|D (0.6-mm wire)
— cribs on 6|6 (0.7-mm wire)
— finger springs on 1| and |2 (0.5-mm wire) to retract and regain the space for the|1.
— a buccal arm to extrude |1 (0.7-mm wire) attached to the gold chain bonded to |1.

■ ***Figure 5.6 shows the position of the dilacerated tooth after approximately 18 months of active treatment. What further treatment may be necessary at a later stage of dental development?***

Ideally it would be appropriate to relieve the crowding in the permanent dentition and align the teeth, correcting the unilateral posterior crossbite and eliminating the mandibular displacement. Details of appropriate treatment cannot be finalized until the patient passes from mixed dentition to permanent dentition at about 10–12 years of age.

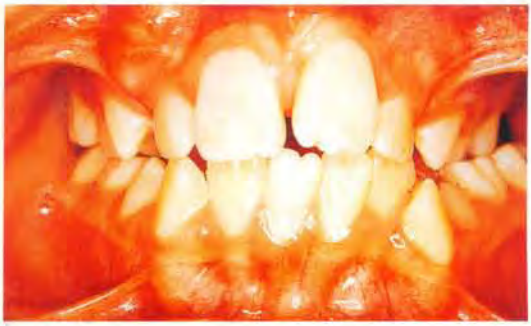


Fig. 5.6 After 18 months of treatment.

6 A dry mouth

Summary

A 50-year-old lady presents to you in your hospital dental department complaining of dry mouth. Identify the cause and plan treatment.

HISTORY

Complaint

She complains of dryness which makes many aspects of her life a misery. The dryness is both uncomfortable and renders eating and speech difficult. She is forced to keep a bottle of water by her side at all times.

History of complaint

She first noticed the dry mouth about 4 or 5 years ago though it may have been present for longer. At first it was only an intermittent problem but over the last 3 years or so the dryness has become constant. Recently the mouth has become sore as well as dry.

Medical history

The patient describes herself as generally fit and well but has had to attend her medical practitioner for poor circulation in her fingers. They blanch rapidly in the cold and are painful on rewarming. She has also used artificial tears for dry eyes for the last 2 years but takes no other medication.

EXAMINATION

Extraoral examination

She is a well-looking lady without detectable cervical lymphadenopathy. There is no facial asymmetry or enlargement of the parotid glands and the submandibular glands appear normal on bimanual palpation. Her eyes and fingers appear normal.

Intraoral examination

■ *The appearance of the patient's mouth is shown in Figures 6.1 and 6.2. What do you see? How do you interpret the findings?*

The alveolar mucosa appears 'glazed' and translucent or thin (atrophic) suggesting long-standing xerostomia. Some oral debris adheres between the teeth, again suggesting dryness, which causes plaque to be thicker and more tenacious. There are carious lesions and restorations at the cervical margins of the lower anterior teeth, indicating a high caries rate. The tongue is lobulated and fissured. Both features suggest a lack of saliva.

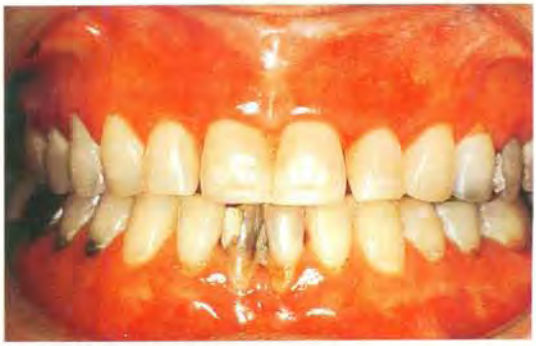


Fig. 6.1 Appearance of the patient's anterior teeth.

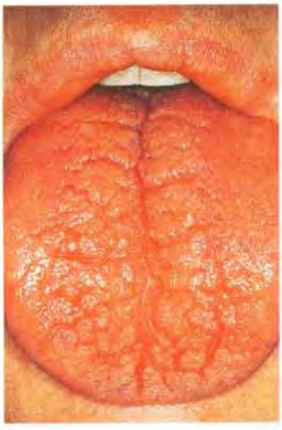


Fig. 6.2 Appearance of the patient's tongue.

If you were able to examine the patient you would find that her mouth does feel dry. Gloved fingers and mirror adhere to the mucosa making examination uncomfortable. Parts of the mucosa, especially the palate and dorsal tongue appear redder than normal. No saliva is pooling in the floor of the mouth and what saliva can be identified is frothy and thick. Small amounts of clear but viscid saliva can be expressed from all four main salivary ducts.

■ *What are the common and important causes of xerostomia and how are they subdivided?*

'In true xerostomia the salivary flow is reduced. The term 'false xerostomia' describes the sensation of dryness despite normal salivary output.

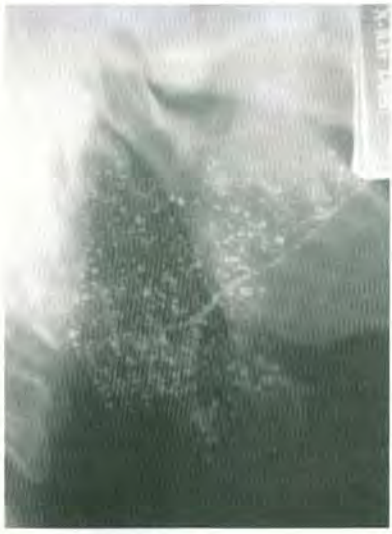


Fig. 6.3 Parotid sialogram.

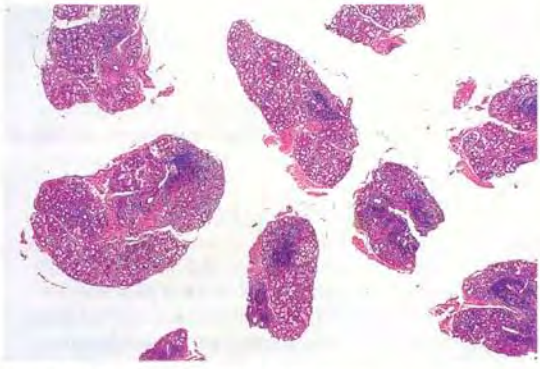


Fig. 6.4 Minor salivary gland biopsy; low power.

■ *The minor salivary gland biopsy is shown in Figures 6.4 and 6.5. What do you see?*

The low power view shows several minor salivary glands. A minimum of 6–8 glands is required for reliable diagnosis and this sample is sufficient. Even at this low magnification, dark foci of inflammatory cells are visible (though they cannot be identified as such) and it can be seen that the lobular structure of the glands is largely intact.

The high power view shows one gland lobule. Centrally there are three small ducts surrounded by a dense lymphocytic infiltrate. The infiltrate is sharply defined and within the lymphocytic focus there is complete loss of acinar cells (acinar atrophy). Around the lymphocytes there is a zone of essentially normal uninflamed mucous salivary gland.

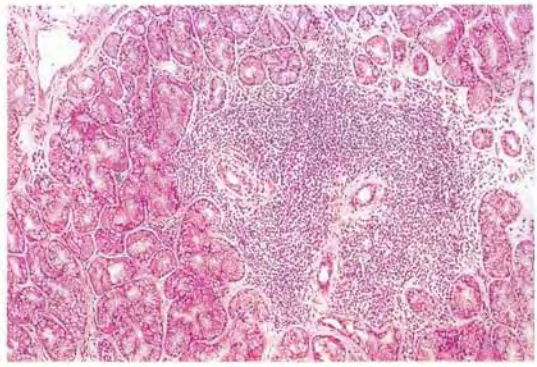


Fig. 6.5 Minor salivary gland biopsy; high power.

■ *How do you interpret these histological appearances?*

The focal lymphocytic sialadenitis centred on ducts and concentric sharply defined zones of acinar atrophy surrounded by normal acini are characteristic of Sjögren's syndrome.

DIAGNOSIS

■ *What is your final diagnosis?*

The patient has primary Sjögren's syndrome. The diagnosis was suspected on the basis of history and examination, and is confirmed by the characteristic sialogram and biopsy findings. The primary form of Sjögren's syndrome is indicated by the lack of autoimmune/connective tissue disease and the positivity for ssA and ssB autoantibodies. The presence of Raynaud's phenomenon, the severity of the xerostomia and dryness of the eyes are also more consistent with the primary form. In addition the patient has candidosis which is the probable cause of the soreness.

TREATMENT

■ *How could you contribute to the management of this patient?*

Control of the underlying disease is not possible but the patient requires treatment for complications and continued follow up:

- Treat candidosis and follow up regularly for recurrence.
- Preserve what salivary secretion remains; saliva is more effective than saliva substitutes.
  - — Sip water rather than drinking it, so as to expand remaining saliva and not wash it from the mouth.
  - — Whenever possible avoid drugs which cause xerostomia.

7 Painful trismus

Summary

A 27-year-old woman is unable to open her mouth normally. What is the diagnosis and how should she be managed?

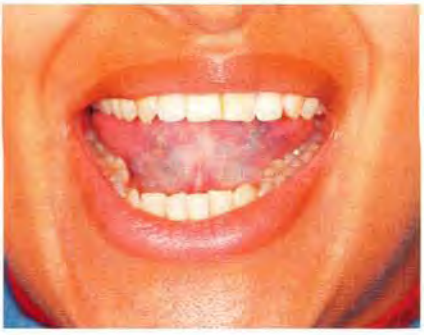


Fig. 7.1 The patient on presentation showing maximal opening.

HISTORY

Complaint

The patient is unable to open her mouth more than half the normal distance.

History of complaint

She has had sporadic painless clicks from her right jaw joint for many years. Recently the click has become louder and painful. On occasions there has been some hesitancy of opening just at the position where the click would normally be felt. Three days ago, while eating a particularly chewy piece of meat, she felt a sudden pain in front of the right ear and since that time she has been unable to open her mouth more than about half way.

Medical history

The patient is otherwise well but she has suffered from previous episodes of knee pain and was seen by a rheumatologist who diagnosed generalized hypermobility of her joints.

Social history

The patient used to be a keen and successful gymnast as a teenager.

EXAMINATION

Extraoral examination

The patient is apyrexial and appears well. There is no facial swelling and the skin colour over the preauricular regions is normal. There is tenderness on palpation over the right condyle but no tenderness on the left side. There is generalized muscular tenderness, particularly of the right masseter and the right lateral pterygoid muscles. Examination of the fingers, wrists and elbows shows an increased range of joint movement.

Intraoral examination

■ *The patient's appearance is shown in Figure 7.1. She is trying to open her mouth to the maximum extent. What do you see?*

There is limited mouth opening and a deviation towards the right side. If you were able to examine the patient you would find that the opening, measured between the tips of the incisor teeth, is 23 mm. Lateral excursions of the mandible were measured at 8 mm to the right and 1 mm to the left. The patient readily achieved a normal position of maximum intercuspation between upper and lower teeth.

DIFFERENTIAL DIAGNOSIS

■ *The patient has trismus. What is trismus?*

The definition of trismus is reduced opening caused by spasm of the muscles of mastication but the term is used loosely for all causes of limited opening. True trismus is usually temporary.

■ *What are the causes of trismus?*

Causes of limitation of opening include:

Intra-articular causes	Internal derangement of the joint
	Fractured condyle
	Traumatic synovitis
	Septic arthritis
	Osteoarthrosis
	Inflammatory arthritis, (e.g. rheumatoid or psoriatic)
	Ankylosis (secondary to trauma or infection)
	Lesions of the condylar head (e.g. osteochondroma)
Extra-articular causes	Trauma (e.g. fractured mandible not involving the condyle)
	Postsurgical removal of impacted lower third molar

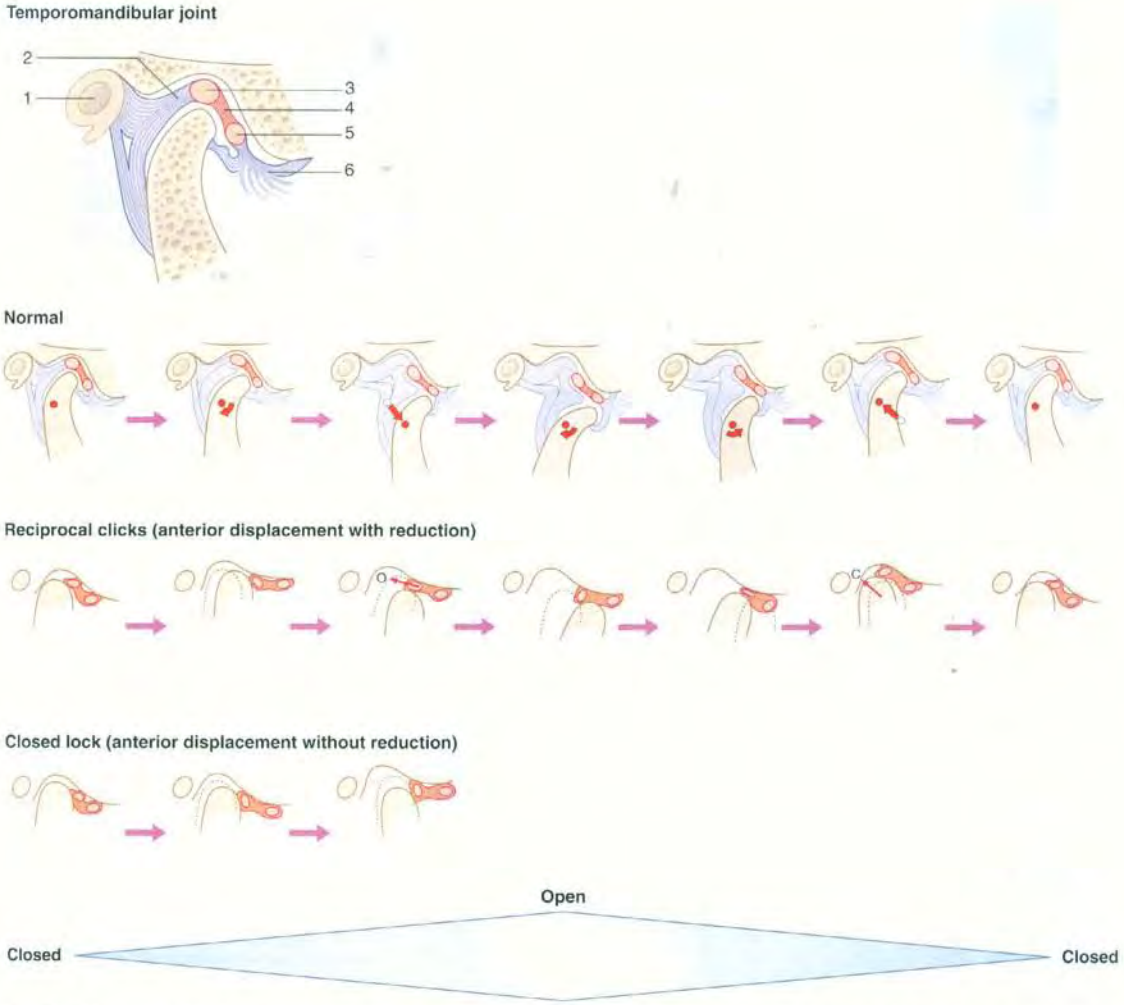


Fig. 7.2 Movements of the temporomandibular joint during the normal opening and closing cycle, with reciprocal clicking and in closed lock. The structure of the normal temporomandibuar joint is shown in the upper panel, with the components of the articular disc and joint capsule. The top row shows the normal opening and closing cycle. Rotation occurs in the lower joint compartment and translation in the upper. The mechanism of reciprocal clicking is shown in the middle row with arrows indicating the sudden movements of disc and condyle that cause opening (O) and closing (C) clicks respectively. The bottom row shows partial opening in a patient with closed lock as a result of anterior displacement of the disc with reduced translation and opening. 1, External auditory meatus; 2, bilaminar region of disc; 3, posterior band of disc; 4, intermediate zone of disc; 5, anterior band of disc; 6, insertion of lateral pterygoid.

space and is required for opening and lateral excursion. Thus, in intra-articular causes of trismus there is usually limitation of movement *in all directions*, as in the present case.

Movements possible in intra- and extra-articular trismus and locking are shown in Figure 7.3.

■ *What is the most likely cause?*

There is no history of surgery or trauma, no suggestion of fracture, no inflammation visible over the joint to suggest arthritis and no systemic cause for arthritis. Traumatic synovitis is a possibility but does not usually cause selective impairment of movement; all joint movements are painful. This leaves internal derangement involving the intra-articular disc as the most likely cause. The progression of clicking to locking with pain and intra-articular trismus of rapid onset is typical of closed lock and fits with the pattern of symptoms and signs seen in this case.

In this case the patient is still able to translate the left condyle forward, causing deviation to the right on opening. Lateral excursion to the right was normal at 8 mm. Therefore the cause of the restricted opening is internal derangement of the right joint.

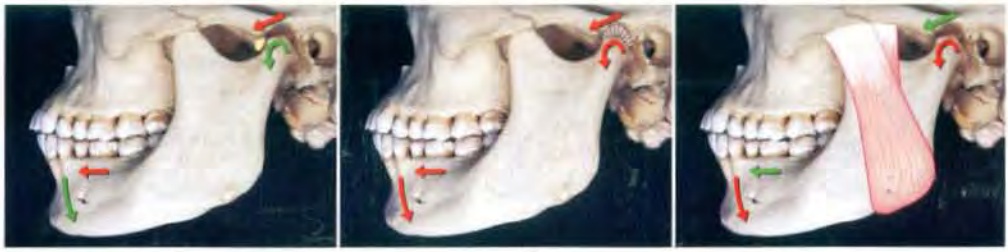


Fig. 7.3 Movements possible in intra and extra articular trismus. Green arrows indicate movements that are possible and red arrows those which are impossible. Left, intra-articular trismus: closed lock caused by an anteriorly displaced disc (yellow). Middle, intra-articular trismus: ankylosis (red). Right extra-articular trismus: spasm or fibrosis of masseter muscle (red).

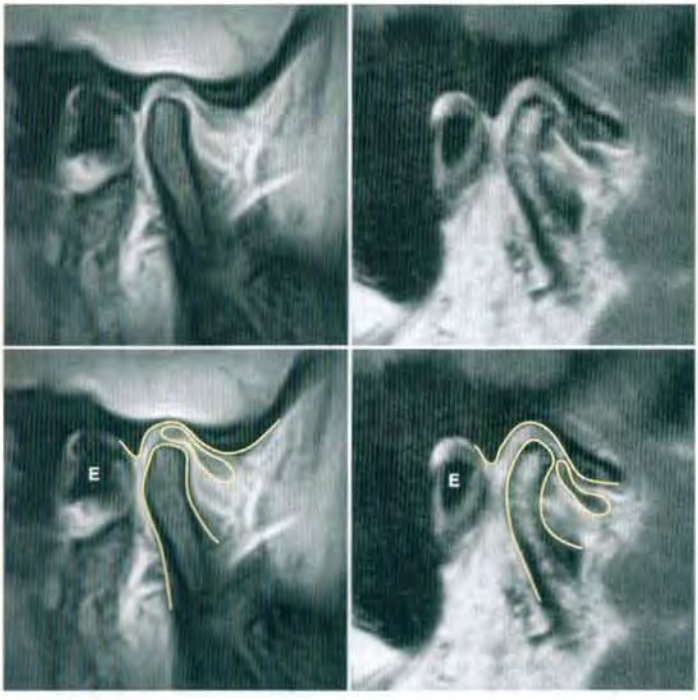


Fig. 7.4 T1 weighted magnetic resonance imagin of the right temporomandibular joint. A normal join is shown on the left and the patient's joint on the right with the external auditory meatus labeled E. I this technique air, dense cortical bone and the disc all appear dark. In the lower panels the condyle, temporal bone and disc are outlined.

INVESTIGATIONS

What investigations may help?

Plain radiographs will probably show no abnormality because there is no change in the bony structures of the joint. If a pathological process other than internal derangement is suspected then radiography may be helpful. A dental panoramic tomogram is usually the first view of choice with other tomographic projections including spiral tomography and CT giving additional information. Alternatively transpharyngeal or transcranial projections give clearer views but with a higher radiation dose.

Magnetic resonance imaging (MRI) would show the malpositioned disc and this may sometimes be helpful in diagnosis. Images from this patient's magnetic resonance scan are shown in Figure 7.4.

Arthrography, – radiography with a contrast medium injected into the joint – is possible. Lower joint space arthrograms are more helpful but it is more difficult to inje medium into the lower joint space.

Serology for rheumatoid factor and an autoantibody profi may be indicated if a polyarthropathy is suspected. However, some causes of arthritis are seronegative, for instance psoriasis and ankylosing spondylitis.

* In this, and most other cases, the clinical picture is sufficiently clear to make the diagnosis and these investigations are not normally required.

8 A lump on the gingiva

Summary

A 48-year-old man presents to you in general dental practice with a gingival swelling. What is the cause and what would you do?

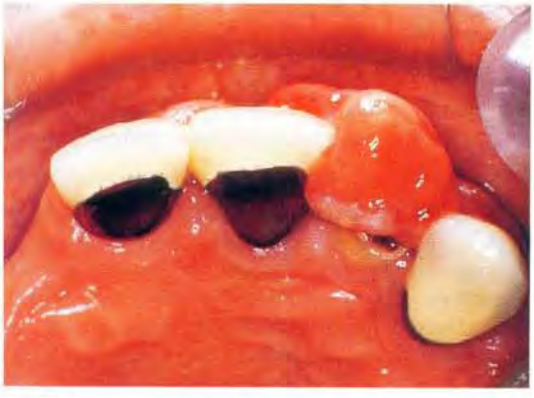


Fig. 8.1 Appearance of the swelling.

HISTORY

Complaint

The patient complains of a lump on the gum at the front of his mouth on the left side. It sometimes bleeds, usually after brushing or eating hard food but it is not painful.

History of complaint

The swelling has been present for 4 months and has grown slowly during this period. It was never painful but now looks unsightly. The patient gives no history of other mucosal or skin lesions.

Medical history

The patient has hypertension, controlled with atenolol 50 mg daily.

EXAMINATION

Extraoral examination

He is healthy looking but slightly overweight. There are no palpable cervical lymph nodes.

Intraoral examination

The patient is partially dentate and has relatively few and extensively restored teeth. He wears an upper partial denture. The root of the upper lateral incisor is present and its carious surface lies at the level of the alveolar ridge. The teeth on each side of the lesion are restored with metal–ceramic crowns.

There is a mild degree of marginal gingivitis. Most of the interdental papillae are rounded and marginal inflammation is present around crowns. Flecks of subgingival calculus are visible.

■ *The appearance of the lesion is shown in Figure 8.1. Describe its features.*

Feature	Appearance
Site	Appears to arise from the gingival margin of the lateral incisor root or the interdental papilla mesially
Size	Approximately 10 × 7 mm
Shape and contour	Irregular rounded nodule. It is not possible to say whether it is pedunculated or sessile, though from its size and the fact that it overlies the lateral incisor root, it is probably pedunculated
Colour	Patchy red and pink with a thin grey translucent sheen. The surface is almost certainly ulcerated

If you were able to palpate the lesion you would find that it is fleshy and soft and attached by a thin base to the gingival margin. It bleeds readily from between the tooth and lesion when pressed with an instrument but it is not tender.

■ *From the information in the history and examination so far, what is your differential diagnosis?*

Likely:

— Pyogenic granuloma (if the patient had been female, pregnancy epulis would have been considered)

— Fibrous epulis

Less likely:

— Peripheral giant cell granuloma

— Sinus papilla (parulis)

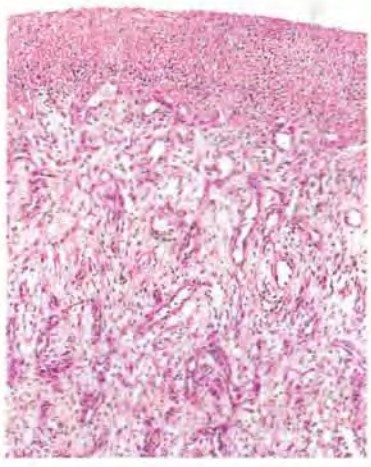


Fig. 8.2 Histological appearance of the surface layers of the excision specimen.

The lesion is a nodule of ulcerated maturing granulation and fibrous tissue.

■ *What is the diagnosis?*

Pyogenic granuloma.

OTHER POSSIBILITIES

■ *Is a more conservative approach to treatment ever justified?*

Yes: elimination of the causative factors may induce considerable resolution. However, the degree of resolution varies; softer more vascular lesions shrink most and firmer more fibrous lesions hardly at all. Removal of calculus and improved oral hygiene may cause partial resolution and leave a smaller lesion which is easier to excise and bleeds much less. Such a course of action is often appropriate for treatment of pregnancy epulis, both because of the wish to avoid the procedure during pregnancy and because excision during pregnancy carries a risk of recurrence. Definitive excision may then be delayed until after parturition. Occasionally resolution is almost complete and no further treatment is required.

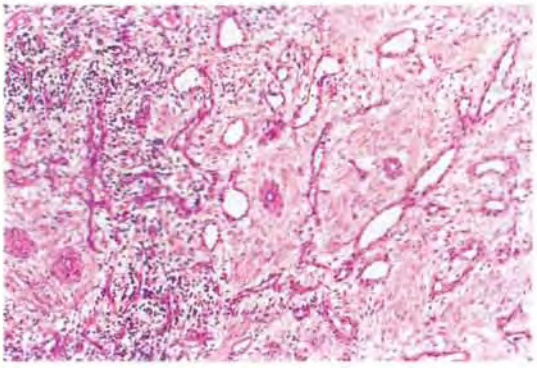


Fig 8.3 The deeper tissue of the specimen.

■ *If, on removing the lesion, you felt bone within it, what would this signify?*

Woven and lamellar bone, sometimes quite large pieces, can lie within fibrous epulides and pyogenic granulomas. Bone may be noted on excision or on histological examination. Sometimes such lesions are referred to as mineralizing epulides (or peripheral ossifying fibroma in the US). The presence of bone seems to be of no great significance and it may indicate that such lesions arise by proliferation of the deep fibrous tissue of the periosteum. Some consider lesions containing bone more likely to recur than those without but there is no good evidence to support this belief.

9 Pain on biting

Summary

A 32-year-old man presents at your general dental practice surgery with intermittent pain on biting. Identify the cause and discuss treatment options.

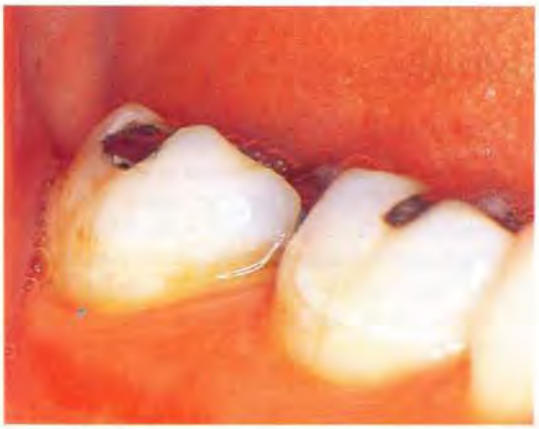


Fig. 9.1 The teeth in the lower right quadrant.

HISTORY

Complaint

He complains of pain on biting which is unpredictable, extremely painful and sharp but poorly localized. It originates in the lower right quadrant and lasts a very short time, only as long as the teeth are in contact, and is so painful that he has become accustomed to eating on the left. The pain only arises on biting hard foods or deliberately clenching his teeth. Apart from these sharp electric shock-like pains he has no other symptoms.

History of complaint

The pain is a recent phenomenon, having been first noticed a month or two ago. At first it was frequent but it has become less of a problem now that he has learnt to avoid triggering the pain. He has not noticed the pain being provoked by hot or cold.

Dental history

The patient has been a regular attender at your practice since childhood. He has a small number of relatively small restorations. At his last appointment, some 4 months ago you placed an amalgam restoration in the lower right second molar.

■ *Based on what you know already what are the likely causes? Explain why.*

A pulpal pain is the most likely cause because the pain appears to originate in a tooth and is poorly localized. Pain of periodontal ligament origin should be well localized. However, pulpitis appears not be present because there is no sensitivity to hot or cold. Pulpitis caused by placement of the recent amalgams and pain due to caries or exposed dentine can be excluded for the same reasons.

A crack in the tooth or electrogalvanic pain are possible causes suggested by pain on biting. Both are triggered by tooth–tooth contact.

Trigeminal neuralgia should be considered as an unlikely nondental cause. It causes paroxysmal stabbing or electric shock-like facial pain in distributions of the trigeminal nerve and may be initiated by touching or moving trigger zones. It usually affects the middle-aged or elderly. The history of pain on biting is almost conclusive of a dental cause but it can be difficult to exclude trigeminal neuralgia in some patients, particularly when trigger zones lie in the mouth or attacks are triggered by eating. If no dental cause is found, the possibility of trigeminal neuralgia may need further investigation.

Acute periodontitis caused by an occlusal high spot on the recently placed amalgam needs to be considered. However, although this could cause great tenderness on biting it would be expected that the pain from the bruised periodontium would be present at other times. Also, such periodontally-sensed pain would be well localized.

■ *What additional questions would you ask? Why?*

The patient should be asked about clenching or bruxing of the teeth because the additional occlusal load can cause fracture and will determine treatment options.

The patient describes a habit of nocturnal bruxism with some tenderness of masticatory muscles at times of stress.

EXAMINATION

Extraoral examination

There is a suspicion of hypertrophy of the masseter muscles on clenching.

10 A defective denture base

Summary

The acrylic denture base and cobalt–chromium casting shown both have defects caused by similar mechanisms. Can you identify the problem and its causes, which are different in the two examples.

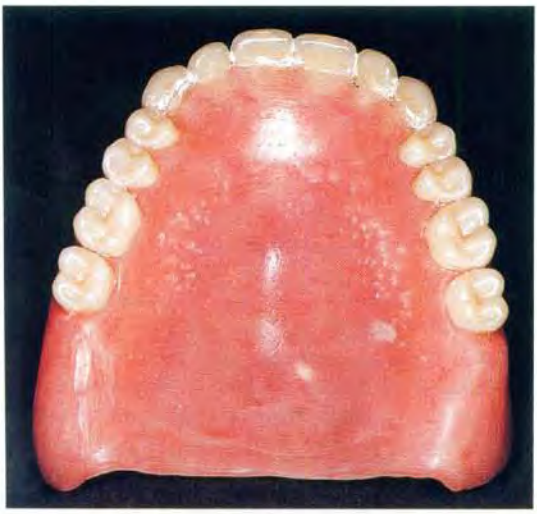


Fig. 10.1 The heat-processed 'acrylic', poly(methylmethacrylate) denture base.

ACRYLIC COMPLETE DENTURE

■ ***A heat-processed 'acrylic', poly(methylmethacrylate) denture base is shown in Figure 10.1. What do you see and how do you interpret these observations?***

The denture base has a cluster of small round holes in a horseshoe-shaped distribution just inside the teeth. The defects are more frequent in areas of thicker acrylic. Each defect appears to be round, some are completely enclosed in acrylic, while others communicate with the surface via sharply defined holes.

Table 10.1 Types of porosity

Defect	Manifestation	Cause
Contraction porosity	Porosity throughout the denture. The denture may be the incorrect shape.	Insufficient material packed into the flask, or inadequate flasking pressure. Correct use of the trial packing stage should eliminate this.
Gaseous porosity	Porosity in a localized area of the denture base, particularly in the thicker parts. Each defect is round and sharply defined.	Vaporization of monomer during processing.
Granular porosity	Porosity appears in thin sections of the denture, which often have a 'white and frosty' appearance.	Incorrect polymer: monomer ratio when producing the dough, or failing to pack the flask at the dough stage.

The presence of numerous small holes or defects within the acrylic is known as porosity.

■ ***What are the types of porosity? How do they manifest and what are their causes?***

The types of porosity are presented in Table 10.1.

This denture has suffered from gaseous porosity and the appearances are typical but more extensive than usually seen.

■ ***What causes monomer to vaporize during processing?***

The boiling point of methylmethacrylate is 100.3°C at standard temperature and pressure. If the boiling point is exceeded then the methylmethacrylate vaporizes and bubbles produce porous defects. The polymerization of methylmethacrylate is exothermic and will contribute to vaporization if precautions are not taken to reduce the temperature. Because the process is heat-dependent, it is most likely to develop in thick sections of the denture and in the last portions to be polymerized.

■ ***How is gaseous porosity normally prevented?***

Methylmethacrylate should be polymerized at a low temperature and under pressure. Packing the dough under pressure raises the boiling point of the methylmethacrylate, and polymerization at 72°C for 16 hours (or 72°C for 2 hours and 100°C for a further 2 hours) followed by slow cooling gives time for the heat of the exothermic reaction to dissipate.

COBALT–CHROMIUM CASTING

■ ***A cobalt–chromium denture framework is shown in Figure 10.2. What do you see and how do you interpret these changes?***

The metal has numerous small perforating holes. They are of various sizes and some have coalesced to form large defects.

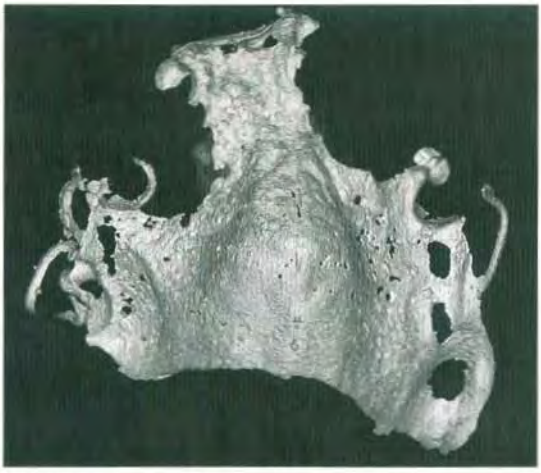


Fig. 10.2 The cobalt–chromium partial denture casting.

■ ***What are the common defects in cobalt–chromium casting? How may they be prevented?***

See Table 10.2.

■ ***Which of these defects affects the present casting? Explain why.***

The casting defects are small and round, like those in the acrylic denture, and also appear to be caused by gas bubble formation. This is another example of porosity but it is much more extensive than is seen when the investment is too thick or gas dissolves in the alloy. In this case a more fundamental mistake must have been made and the cause is probably use of the wrong investment material.

If a framework is invested in a gypsum-bonded investment, the investment will break down at a lower temperature than the melting point of the alloy. The $CaSO_4$ binder reacts with the SiO_2 refractory to produce SO_3 gas, bubbles of which cause porosity in the casting. Gypsum-bonded investments are used for gold-based alloys and phosphate-bonded investments must be used for Co–Cr based alloys.

Table 10.2 Common defects in cobalt–chromium castings

Defect	Cause	Preventive measure
Porosity: spherical voids	Investment too thick Gases dissolve in the alloy and form bubbles on cooling	Use the correct powder: liquid ratio Do not overheat the alloy
Porosity: irregular voids	Casting shrinkage Turbulent flow of the alloy	Ensure sprues are of the correct diameter Ensure sprues are in the correct position
Incomplete cast: rounded margins	Back pressure of air in the mould	Use a porous investment or include vents
Incomplete cast: short casting	Insufficient alloy Mould too cold when cast Insufficient casting force	Use sufficient alloy Ensure the correct operating temperature Ensure the machine is correctly set up
Fins	Investment cracking	Use the correct investment and do not heat too rapidly
Rough surface	Investment breakdown Air bubbles on wax pattern	Use the correct investment and do not overheat Use a wetting agent
Distortion	Stress relief of the wax pattern	Warm the wax thoroughly before making the pattern
Cast too small	Insufficient investment expansion	Use the correct operating temperature
Cast too large	Too much investment expansion	Use the correct investment for the alloy, and the correct operating temperature

mother's knees, all of which are more familiar than the dental chair. Place the mother where your dental light can be used if the child will tolerate it. If the child does not allow her teeth to be examined in these positions then you must consider an examination under more controlled conditions.

■ ***How could you safely restrain the child without frightening her further?***

If you decide to perform a full examination on a reluctant child it must be done in a controlled, caring and confident manner with experienced nursing support and with the consent and cooperation of the mother. You must explain to the mother exactly what you are going to do and repeat to the child that you are just going to count teeth. Then:

- Align the chair in a fairly upright position.
- Ask the mother to sit in the chair as if she is being examined – the child will probably come with her.
- Ask the child to sit on the mother's lap.
- Explain to the child what you are going to do.
- Ask the mother to turn the child so that she is sitting across the mother with her head at the 10 o'clock position.
- Ask the mother to control the arms and hands.
- Your nurse will gently control the feet.
- Slowly and calmly lower the child's shoulders and head onto your lap, explaining what you are doing.

The technique is shown in Figure 12.2. Note how hands and legs are gently restrained and the child remains in close contact and able to see her mother. This position is useful not only for mild degrees of anxiety but also to examine severely frightened children in pain who are determined to resist. Understandably the child sometimes cries, but not always. Although not ideal, crying often allows good access to the mouth. If the child will not open their mouth, your nurse tickling their stomach will usually have the required effect. Your nurse will need to position the operating light carefully as light in the child's eyes is often upsetting.

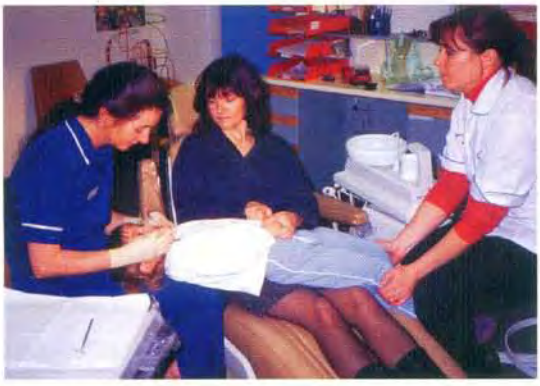


Fig. 12.2 Controlled examination technique.

Keep the examination short and immediately afterwards, whether the child is upset or not, reward her with words, (a 'soft' reward) and a sticker or balloon, (a 'hard' reward). This will encourage the child to allow examination at the next visit. However, be careful not to give inappropriate praise for poor behaviour and inadvertently give the impression that bad behaviour is acceptable.

In your brief examination you see the appearances shown in Figure 12.3.

■ ***The appearances on examination are shown in Figure 12.3. What do you see and what do the appearances indicate?***

- Caries in the occlusal pits and fissures of the second primary molar
- Caries in the distal of the first primary molar, the marginal ridge has collapsed
- Reasonable gingival condition with some interdental marginal inflammation

The key feature is the collapse of the marginal ridge of the first primary molar. The pulp is either directly involved by caries or compromised in the great majority of primary molars once the ridge collapses. This tooth will require a pulpotomy or extraction. Following pulpotomy, a preformed metal crown will be the restoration of choice because they have been shown to be more durable than an intracoronal restoration. However, in an anxious child with limited cooperation it might be appropriate to stabilize the lesion with a temporary intracoronal restoration and delay the definitive restoration for a few months.

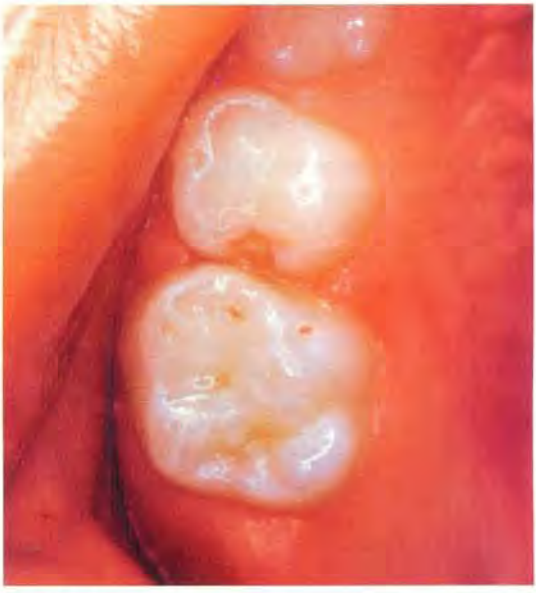


Fig. 12.3 Appearances on examination.

13 Pain after extraction

Summary

A 36 year old lady presents with severe pain a few days after tooth extraction. What is the cause and what can be done?

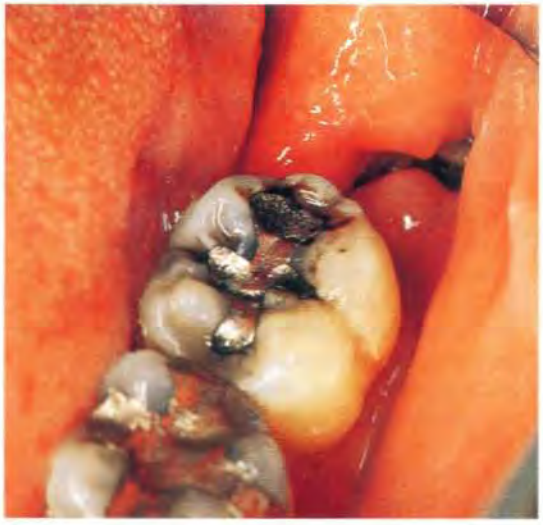


Fig. 13.1 The appearance of the socket.

HISTORY

Complaint

She complains of a distressingly severe pain from an extraction socket in the left side of her mandible. The pain is localized to the extraction socket and is not sensitive to hot or cold. It is a constant, dull, boring pain unrelieved by aspirin or paracetamol preparations. It prevents the patient performing any normal activity and kept her awake last night.

History of complaint

The patient underwent surgical removal of the lower left third molar tooth at her dentist 4 days ago. The extraction had proved more difficult than expected and involved repeated attempted elevation and a small amount of bone removal using a bur. Following the extraction, bleeding stopped normally. The extraction site had been tender but apparently was healing slowly until the pain started yesterday. Since then she has also noticed halitosis and a bad taste.

Medical history

The patient is otherwise fit and well. She is taking an oral contraceptive and no other positive findings were revealed by the medical history.

EXAMINATION

Extraoral examination

The patient has moderate extraoral swelling of the facial soft tissues overlying the extraction socket and some early discoloration of the skin by ecchymosis. There is trismus and she is able to open her mouth to only 22 mm interincisal clearance. There are no palpable lymph nodes in the deep cervical chain or submandibular triangle.

Intraoral examination

Halitosis is noticeable. The appearance of the socket is shown in Figure 13.1. The lower left third molar socket contains no tissue, only food debris. The surrounding soft tissues are slightly swollen but not significantly inflamed as judged by redness.

DIAGNOSIS

■ *Based on what you know already, what is the most likely diagnosis?*

The diagnosis is most likely to be a dry socket (alveolar osteitis). The history of severe and persistent pain localized to the tooth socket appearing 3-5 days after extraction, particularly a traumatic one, is characteristic. The lack of local inflammation or enlarged lymph nodes is compatible with this diagnosis and argues against post extraction infection either in the bone or soft tissue.

The diagnosis is confirmed by the examination which shows that the blood clot has been lost from the socket. In severe cases the bone of the socket may be exposed, and the bone of the distal lip of the socket can be seen in Figure 13.1.

Halitosis is the result of food debris in the socket being degraded by a partially anaerobic bacterial flora. The trismus is almost certainly related to the surgical trauma of extraction.

Table 14.1 Investigations to be carried out

Test	Reason	Problems
Vitality test	To check the vitality of all four upper and lower incisors and canines (excluding any known root-filled teeth). Late loss of vitality is a complication of trauma and any one of these teeth could have periapical infection and be the cause of the bad taste. The vitality of the lateral incisor needs to be known, to plan treatment once the diagnosis is established.	Electric pulp tests are notoriously difficult to perform on crowned teeth and the results must be interpreted with caution. The lateral incisor has a metal ceramic bonded crown and the ceramic will insulate the tooth while the metal layer will diffuse the applied voltage and conduct the stimulus to the gingiva. The patient may mistake a gingival sensation for a vitality response.
Periapical radiograph	To detect the possible causes and assess bone levels around the teeth. To determine the pulp canal morphology in case root canal treatment is required, and the root morphology in case extraction is necessary.	Root fractures may be difficult to identify if the fragments are not separated. A second view at a slightly different angle may allow detection of a root fracture invisible in the first. However, this tooth is so mobile that any root fracture should be readily identified.

would appear to be independent of the original trauma. Teeth which suffer coronal fractures do not usually suffer root fractures as well because less of the energy of the blow is transmitted to the root. However, if a root fracture had been present for the last 4 years it might have triggered slow resorption, combining both possible causative factors.

An unsuspected lesion has destroyed the bone and/or the tooth root apically, leaving support only coronally; this is a remote possibility. The tooth would then be mobile about the remaining intact periodontal ligament. The commonest lesion to do this would be a radicular cyst arising on a nonvital tooth.

However this seems most unlikely as there is no expansion and the adjacent teeth are not displaced or mobile. A different lesion remains a remote possibility.

INVESTIGATIONS

■ *What investigations would you carry out? Why? What are the potential problems?*

See Table 14.1.

On performing the tests of tooth vitality you find that it is impossible to obtain a response from the upper left central and lateral incisors. All other anterior teeth appear vital.

■ *The periapical radiograph is shown in Figure 14.1 What do you see?*

The left lateral incisor is crowned but not root filled. A large oval radiolucency fills the middle third of the root and extends laterally to replace the full width of the root and communicate with the periodontal ligament. The margins of the defect are smooth and sharply defined. The lamina dura around the apex appears intact. The bone level mesially and distally is coronal to the defect and there is no evidence of either horizontal or vertical bone loss. Very little root dentine remains below the crown and gingival margin.

The upper left central incisor is root filled. The filling appears well condensed and extends very close to the ideal level. The root appears to have a curve at the apex. There is a poorly defined radiolucency around the apex mostly on its mesial side, where the lamina dura is missing.

The canine has mesial caries and its apical lamina dura is indistinct. However no obvious apical radiolucency is present.

■ *What is wrong with the radiograph in Figure 14.1?*

A regular pattern is superimposed over the whole film. This is a developing artefact caused by some film processors (e.g. Velopex) which use woven nylon bands to transport

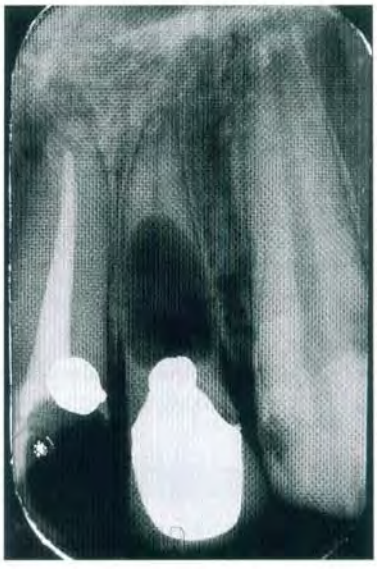


Fig. 14.1 Periapical radiograph of the mobile lateral incisor.

15 Oroantral fistula

Summary

A 42 year-old man presents with pain following extraction of an upper first molar. What is the cause and how will you treat him?

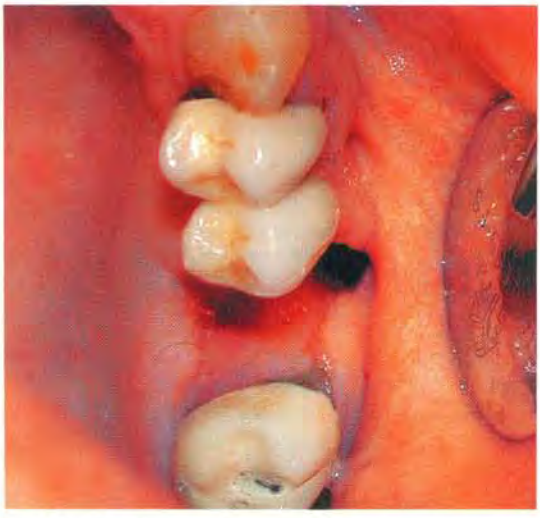


Fig. 15.1 The extraction socket on presentation

HISTORY

Complaint

The patient is suffering dull throbbing pain in his upper jaw and face on the left side only. Pressure below his eye is painful and all his upper teeth on the left are tender on biting. He has a nasal discharge and blocked nose on the left.

History of complaint

He has had the pain for 2 weeks following extraction of the upper left first molar by his dentist. The extraction was difficult and the roots required surgical removal. There was little pain immediately afterwards but pain has slowly developed so that it is now preventing him from sleeping. The pain is constant.

Medical history

He gives a history of smoking 20 cigarettes a day for 24 years but considers himself fit and healthy.

EXAMINATION

Extraoral examination

He is a healthy looking man with no facial swelling or lymphadenopathy. There is a lightly blood-stained discharge from the left nares and halitosis.

Intraoral examination

■ *The appearances on presentation are shown in Figure 15.1. What do you see and how do you interpret the features?*

There is a large defect in the alveolus at the site of the first molar socket, the socket appears empty and the oral mucosa has grown to line the visible sides of the socket. After 2 weeks the oral epithelium should have proliferated to cover the socket mouth but there must have been a failure of clot formation and/or organization. One possibility is that the patient has a dry socket (see Case 13). No bone is visible in the socket but it could be exposed apically. However, dry socket is rare in the maxilla and it is more likely that the socket is communicating with the maxillary antrum.

DIFFERENTIAL DIAGNOSIS

■ *What causes for this pain are possible and why?*

Sinusitis secondary to oroantral fistula. An oroantral communication itself causes little or no discomfort but usually induces a degree of sinusitis. The nature and distribution of pain and presence of nasal discharge are typical of sinusitis. This seems the most likely diagnosis. Fistula formation is most commonly associated with extraction of maxillary first and second molars.

Dental pain. Before jumping to conclusions, it is worth considering whether the wrong tooth may have been removed. If the extraction was performed for pulpitis (which is often poorly localized), it is possible that at least some of the symptoms may arise from the adjacent teeth. You will need to check whether additional symptoms suggest pain of dental origin.

■ *What is an oroantral fistula?*

An oroantral fistula (OAF) is a persistent epithelialized communication between the maxillary antrum and the mouth, present for more than 48 hours. The epithelial lining of the fistula grows from the gingival epithelium, periodontal pocket lining or the antral lining and it may take up to 7

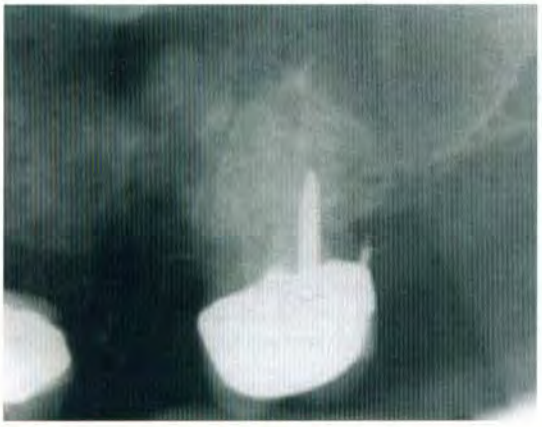


Fig. 15.2 Periapical view of the socket on presentation.

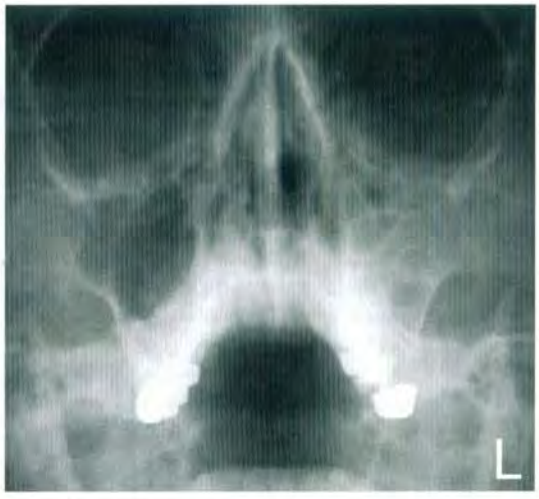


Fig. 15.3 Part of the occipitomental view

Fig. 15.7). The second molar has an inadequate root canal treatment, probably associated with loss of apical lamina dura and a small periapical radiolucency. A pin has perforated the distal root.

■ *What else do you need to know about the root fragment?*

Whether it is loose in the antrum or trapped under the sinus lining or in granulation tissue. The root will have to be removed and if it is under the lining or trapped, it should be possible to remove it through the socket. If it is loose in the antrum, removal in this way may prove impossible, necessitating a later elective surgical procedure such as a Caldwell–Luc approach.

■ *How will you decide where the root is and whether it is mobile?*

If the fistula opening is large you might try to visualize the fragment directly. If not, a second radiograph at right angles to the periapical, such as an occipitomental view, would help to localize it. A further view with the patient's head tilted would reveal whether or not the root moves.

■ *A section of the occipitomental view is shown in Figure 15.3. What do you see and how do you interpret the appearances?*

The sinuses and facial bones are symmetrical and there is no expansion of the maxillary antrum. However, the sinus on the left is much more radiopaque than that on the right indicating oedema and thickening of the sinus lining or exudates within it. There is no fluid level visible. The root fragment is not visible because it lies on the sinus floor and is obscured by the superimposed alveolus.

DIAGNOSIS

■ *What is your final diagnosis?*

Sinusitis secondary to oroantral fistula caused by extraction of the upper first molar. A root fragment has been displaced into the sinus. Apical periodontitis of the second molar may also contribute to the sinusitis but this is a chronic problem and a lower priority for treatment.

TREATMENT

■ *How would you treat this patient?*

- If pus is present in the fistula or if symptoms are severe, consider treating the sinusitis first and closing the fistula later after the sinusitis has partially resolved (it will not resolve completely until the fistula is closed). If there is long-standing infective sinusitis, this must be treated prior to surgical closure otherwise healing will be compromised.
- excise the fistula otherwise remnants of the epithelial lining may proliferate to reform the tract.
- Remove the root fragment from the sinus.
- Close the oroantral communication surgically.

■ *Would you treat this patient in general practice?*

Provided you are confident of your ability to remove the root fragment, there is no reason why this cannot be dealt with in a general practice situation. However, if the root is mobile in the antrum, the patient should be referred to hospital.

■ *How would you excise the fistula and remove the root?*

Under local anaesthesia, incise around the edge of the socket from gingiva right down into the antrum, removing all the soft tissue in the socket as a cylinder or cone-shaped piece and draw it into the mouth. Depending on the size of the bony defect and the amount of bone resorption (usually greater in long-standing fistulae) this opens up a large hole into the sinus. With suction, good light and direct vision try to identify the root fragment and remove it with fine forceps, sucker tip or other instrument. Take care not to displace it into the sinus. If it becomes displaced it may be possible to wash it out by flushing saline into the sinus. Alternatively better surgical access to the sinus may be achieved using a Caldwell–Luc approach under general anaesthesia. This is the main reason for referring patients with mobile fragments to hospital.

Send the excised tract for histopathological examination in case of unexpected underlying lesions.

■ *How will you close the defect?*

The buccal mucoperiosteal flap with advancement (buccal advancement flap) is the most commonly used technique and it has more than a 90% success rate. The technique is shown in Figure 15.4. After excising the fistula, as above, proceed as follows:

Make two incisions buccally, anterior and posterior to the socket, passing parallel up the attached gingiva and then splaying to provide a wider base to ensure a good blood supply for the flap. The line of the incisions must be compatible with the flap sliding palatally to cover the defect in the alveolus.

Elevate the mucoperiosteal flap you have outlined by lifting the soft tissues in the plane beneath the periosteum.

Advance the flap. The flap cannot yet be pulled across the defect because the periosteum cannot be stretched. Fold the flap back to expose its periosteal surface and make several shallow parallel incisions across the flap that penetrate only the periosteum (about 0.25–0.5 mm in depth). This must be done very carefully with the flap under slight tension. As the periosteum is incised the flap will be felt to stretch. Do not perforate the flap or it will either be cut off or have a compromised blood supply. Make sufficient incisions to lengthen the flap so that it can reach across to the palatal side of the defect with minimal tension.

Prepare the palatal aspect of the alveolar defect. Refresh the margin of the palatal side to expose a narrow bony rim at least 1 mm wide, preferably 2 mm. The flap must be sutured into place at a site that has bony support.

Suture the flap in place using slowly resorbable sutures (for instance 3/0 vicryl) or nonresorbable sutures. Place several sutures around the apex of the flap (mesial, distal and central) and buccally. The flap must not be under tension and sufficient sutures must be placed to ensure an airtight and watertight seal supported by underlying bone. Ensure haemostasis. The sutures must remain in place for 10–14 days.

■ *What alternative flap designs are possible?*

The buccal advancement flap may not be possible when the bony defect is very large or when a previous attempt at repair has failed.

A number of other flaps are possible including the palatal island flap, submucosal palatal island flap, combined buccal and palatal flaps and even pedicled grafts from the tongue. However, most of these techniques are complex and have been superseded by the buccal fat pad technique. Diagrams of the rotated palatal flap technique, the second most commonly used method, are shown in Figure 15.5. Alternative methods to close oroantral fistulae are noted in the 'Local flap design' box on p. 73.

■ *What postoperative instructions are required?*

In addition to the routine instructions given after extraction, the patient must be placed on an *antral regime* to reduce

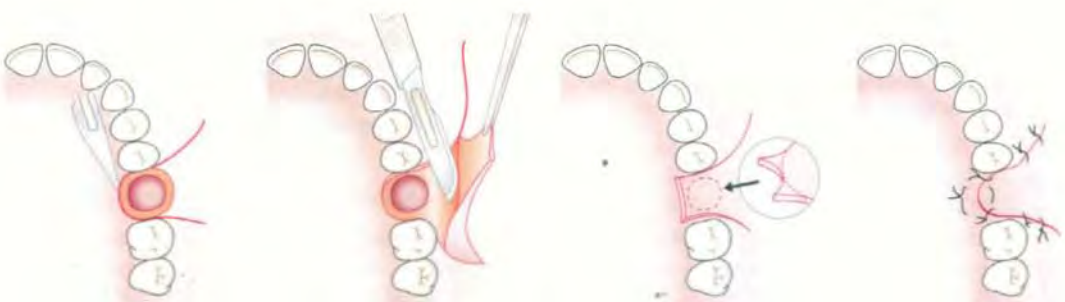


Fig. 15.4 The buccal advancement flap. Note how the relieving incisions buccally flare widely, shallow incision of the periosteum, trimming of the flap apex to ensure a good fit palatally and the rim of palatal bone on which it lies.

Local flap design	Indications/advantages	Contraindications/disadvantages
Buccal advancement flap (see Figure 15.4)	Relatively simple, no flap donor site to heal, suitable for local analgesia	Not ideal for large defect, advancing the flap reduces the depth of the residual buccal sulcus. Alveolar rather than masticatory mucosa advanced onto the ridge. Flap may break down if under tension.
Buccal fat pad transfer. As above and the buccal fat pad is dissected from under the buccal flap on a pedicle and secure in the socket.	As above, able to fill a larger defect	General anaesthesia required for fat pad dissection, sulcus loss.
Palatal flap	Possible when buccal flap has failed or would have insufficient length to cover a palatally placed bone defect. Covers the defect with masticatory mucosa.	The flap is of thick tissue and is difficult to mobilize. The donor site is left to granulate and this is painful until healed.

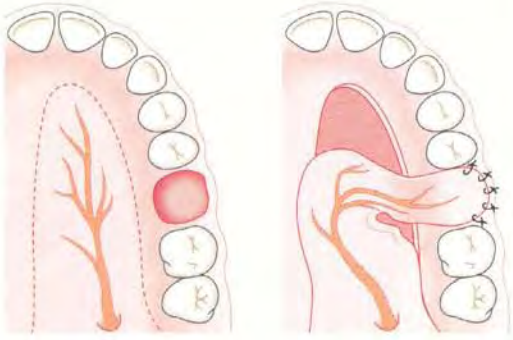


Fig. 15.5 The palatal rotation flap. Note how the flap derives its blood supply from the palatal artery within it. The difficulty of folding the thick flap is clear. The exposed bone will granulate and should be covered with a pack during healing.

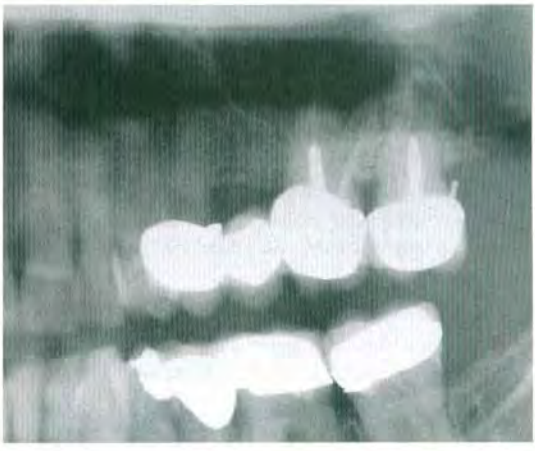


Fig. 15.6 Preoperative radiograph.

inflammation and prevent a rise in air pressure in the antrum. The upper first molar is situated in the lowest point of the maxillary sinus. Persistent infection or inflammation will induce exudates that will drain to this point and cause breakdown of the clot. Increased air pressure in the sinus, for instance from sneezing or blowing the nose, will force air or exudate through the alveolar defect as well as physically disrupting the clot and flap. Decongestants maintain the patency of the opening of the sinus to favour drainage to the nose.

A suitable antral regime would be:

- an absolute ban on blowing the nose for 48 hours
- sneeze allowing pressure to escape through the mouth
- nasal decongestant (such as ephedrine nasal spray 0.5%)
- decongestant inhalant (e.g.Karvol).

In addition, chlorhexidine mouthwash should be given. The repair will fail if there is leakage of saliva and bacteria past the flap from the oral aspect. No rinsing should be performed for 24 hours.

OTHER POSSIBILITIES

■ *How might formation of oroantral communication be prevented?*

The risk of oroantral communication should be assessed routinely on a radiograph before extraction of upper molars. If the risk is high, an experienced surgeon should remove the tooth. Surgical extraction, possibly with elective sectioning of the tooth, reduces the chances of disrupting the maxillary floor.

■ *The preoperative radiograph is shown in Figure 15.6. What do you see?*

Several features in the list of risk factors above are evident. There is a low antral floor in contact with the roots, there is little alveolar bone height and there is loss of lamina dura around the tooth root apices.

■ ***How could an oroantral communication be confirmed at the time of extraction? How might this help?***

If an antral communication is present, an echoing 'wind tunnel sound' will be heard if a small suction tip is held in the socket, the result of air being sucked from the antrum as well as the mouth. If the communication is large you may be able to see into the antrum or identify nasal regurgitation of your irrigation fluids or blood from the extraction site. Do not ask patient to blow through their nose while holding it. The sinus lining may still be intact but would be burst by the pressure and a small communication might be enlarged.

If a communication is suspected, stabilization of the clot, closure of the socket with resorbable sutures and appropriate warnings to the patient about blowing the nose should prevent a fistula developing. This is likely to be effective if the diameter of the communication is 4 mm or less. If it is larger, it should be repaired immediately using a suitable flap technique to avoid sinusitis and infection developing.

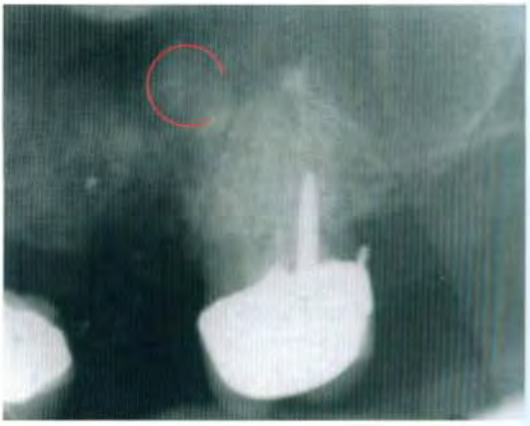


Fig. 15.7 The root fragment outlined on Figure 15.2.

16 Troublesome mouth ulcers

Summary

A 38-year-old woman with mouth ulcers has noticed a recent exacerbation in their severity. You need to make a diagnosis and decide on suitable investigations and treatment.

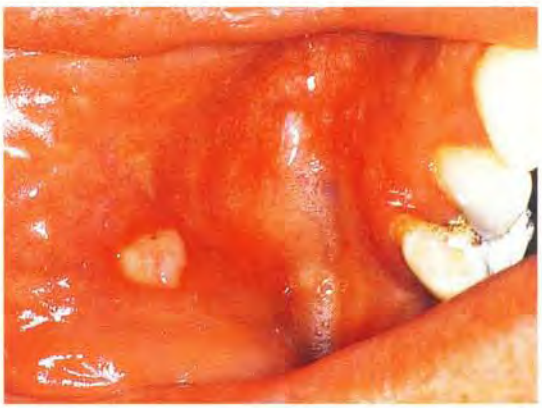


Fig 16.1 The appearance of one ulcer.

HISTORY

Complaint

The patient complains of mouth ulcers which have been troubling her recently.

History of complaint

She has suffered from occasional mouth ulcers, usually one at a time, over a period of more than 20 years. However, recently they seem to have become worse and she now has several. Normally she ignores them but, because she was attending your surgery for a filling, she thought she would ask whether anything could be done.

Medical history

The patient is otherwise fit and well.

■ *The patient has already provided several pieces of information of value for differential diagnosis. How do you assess her ulcers on the basis of the information available?*

The patient has noted an outset of ulceration early in life with recurrent attacks of single ulcers or small crops of ulcers. There are very many causes of oral ulceration but these ulcers appear to be **recurrent,** that is they appear periodically and heal completely between attacks. Recurrent ulceration has relatively few common causes.

■ *What are the common causes of recurrent oral ulceration?*

- Recurrent aphthous stomatitis (RAS)
  - — Minor type
  - — Herpetiform type
  - — Major type
- Erythema multiforme
- Occasional cases of traumatic ulceration
- Ulcers associated with gastrointestinal disease.

■ *How will you differentiate between these conditions?*

Almost entirely on the basis of the findings in the history. Some features of the examination, blood tests or a biopsy may be helpful in certain cases, but the history is most important.

■ *What features of the ulceration would you ask about to determine the diagnosis? Explain why for each.*

See Table16.1 This patient's answers are shown in the right-hand column.

■ *How are major and minor RAS differentiated?*

By severity rather than by any one feature alone. RAS may be labelled as major because of the size of the ulcers, their long duration or because they develop scarring on healing.

■ *From which type of ulcers does the patient appear to be suffering?*

She would appear to have typical minor RAS which has increased in severity recently.

EXAMINATION

Intraoral examination

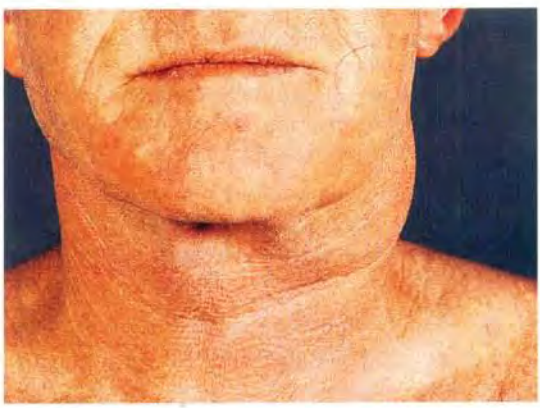
■ *The appearance of one ulcer is shown in Figure 16.1. What do you see?*

There is an obvious ulcer on the anterior buccal mucosa. It is shallow, a few millimetres in diameter and has a slightly irregular but well-defined margin. The surrounding mucosa

17 A lump in the neck

Summary

A 55-year-old man presents to your oral and maxillofacial surgery department clinic with a lump on the left side of the neck. You must make a diagnosis.



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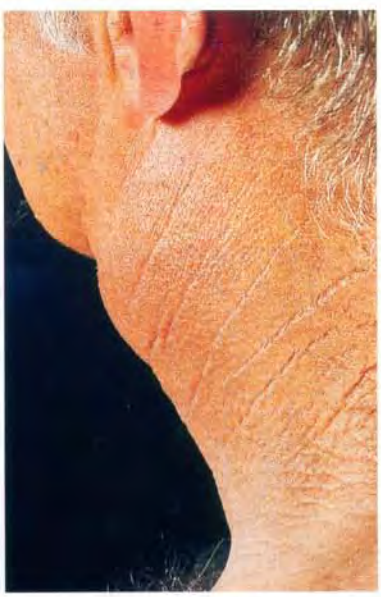


b

Fig. 17.1 **a** and **b** The appearance of the swelling.

HISTORY

Complaint

The patient complains of the lump and notices some discomfort on swallowing, as if something is stuck in his throat. He assumes the lump is the cause.

History of present complaint

He thinks he first noticed the lump about 3 months ago. It has always been painless and is slowly enlarging. The discomfort on swallowing is of recent onset.

Medical history

The patient is otherwise fit and well. He smokes 20 cigarettes per day and drinks 10 units of alcohol each week as beer.

EXAMINATION

Extraoral examination

The appearance of the swelling is shown in Figure 17.1.

■ *What do you see? What is the likely origin of the mass?*

There is a swelling just anterior to the anterior border of the sternomastoid muscle and below and behind the angle of the mandible. It is several centimetres in diameter and extends forwards below the angle of the mandible towards the submandibular region. The overlying skin does not appear to be inflamed.

The lesion lies over the deep cervical lymph node chain and could well arise from a cervical lymph node. It is too low and too far posterior to be arising from the submandibular gland and probably too low to have arisen in the lower pole of the parotid gland. Other soft tissues of the neck could be the origin, but a lymph node is the most likely cause.

If you could palpate the lesion you would find that it is approximately 8 cm by 6 cm in size and feels firm on palpation, possibly slightly fluctuant. It is mobile, not fixed to the overlying skin or deep structures. The patient does not notice any tenderness on palpation. There are no other swellings or enlarged lymph nodes palpable on either side of the neck.

Intraoral examination

The submandibular glands are palpable bimanually and appear symmetrical. Both are mobile and clearly separate from the swelling which lies posterior to the gland.

The patient's mouth has been well restored in the past but suffers from recent neglect and several carious cavities are visible. There is no significant periodontal

disease with most probing depths less than 5 mm and no mobile teeth. The lower left first permanent molar has lost a large restoration and has extensive caries. There is no soft tissue swelling, sinus or tenderness in the sulcus adjacent to the apices of the roots. The tooth is not tender to percussion. The oral mucosa appears normal, and the tonsils appear to be symmetrical.

DIFFERENTIAL DIAGNOSIS

■ ***What are the most likely causes of the lump and why?***

Metastatic malignancy appears likely and this lesion is so typical of a cervical lymph node metastasis that it must be considered to be malignant until proved otherwise. The combination of features suggestive of metastasis is the patient's age (should be considered a possible cause in any patient aged over 45), the site (consistent with a cervical lymph node), the firm consistency and lack of tenderness. Fixation to the skin or other structures would be almost conclusive of malignancy but is a late sign. The patient is a smoker and drinker and so has an increased risk of malignancy. Either a squamous carcinoma or adenocarcinoma is likely. Melanoma and other malignancies are further possible causes.

Lymphadenitis secondary to a local cause is common and so must be considered. However there is no tenderness on palpation to suggest an inflammatory cause. If this were a reactive inflammatory enlargement, the most likely source of infection would be a dental, pharyngeal or skin infection. The patient has a potential source of dental infection in the lower left first permanent molar but the tooth is not tender to percussion nor associated with overt infection, making it an unlikely cause.

Tuberculosis needs to be considered both as a possible diagnosis and as a factor affecting management. Most patients with cervical lymph node enlargement caused by tuberculosis have reactivation ('secondary' or post-primary) tuberculosis in which a previous quiescent infection becomes reactivated. This localized infection may or may not be accompanied by pulmonary disease though there may be radiologial evidence of past tuberculosis on chest radiograph. Cervical tuberculous lymphadenitis is common in those from the Indian subcontinent. Atypical mycobacterial infection is a disease which often affects the cervical lymph nodes but is almost always seen in children or the immunosuppressed.

■ ***Which additional but less likely causes need to be considered whenever a patient complains of an enlargement at this site? Why are they unlikely causes in this case?***

Numerous lesions could arise at this site and it is not useful to list them all. A number of possible causes (Table 17.1) merit consideration, because they are common, easily excluded or cause significant morbidity.

INVESTIGATIONS

■ ***What is the most important investigation? Which methods might be used and what are their advantages and disadvantages?***

The critical requirement when malignancy is suspected is to obtain tissue speedily for microscopic diagnosis. All other investigations are less important at this stage. Two techniques are in common use; the fine-needle aspiration biopsy and the surgical incisional biopsy (Table 17.2).

■ ***What other investigations might be performed, either now or at a later date? why?***

See Table 17.3.

In this case a suitable combination of investigations would be fine-needle aspiration, dental radiographs, vitality tests and possibly ultrasound scan. The sialogram would have been performed if a salivary origin had been thought possible after clinical examination.

The lower first molar was nonvital and a periapical radiograph revealed apical radiolucency. The smear from a fine-needle aspirate is shown in Figure. 17.2.

■ ***What does the fine-needle aspirate show and how do you interpret the appearances?***

The aspirate shows cells from the lesion spread as a single layer and stained with the Papanicolaou stain. This stains nuclei dark blue, keratin orange and the cytoplasm of nonkeratinized epithelial cells turquoise. The cells are almost all epithelial as shown by their prominent cytoplasm and by the presence of keratinization (arrowed A) in some of them. The larger cells

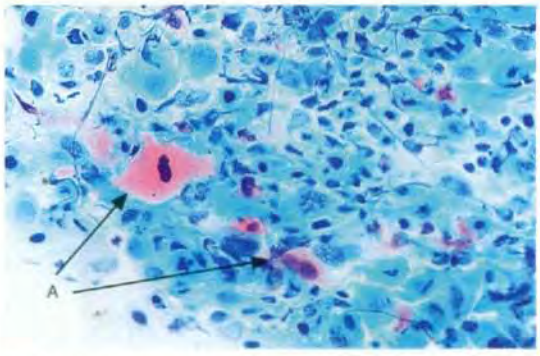


Fig. 17.2 Fine-needle aspirate from the lesion.

18 Trauma to an immature incisor

Summary

An 8-year-old girl has fractured her upper right permanent central incisor tooth.

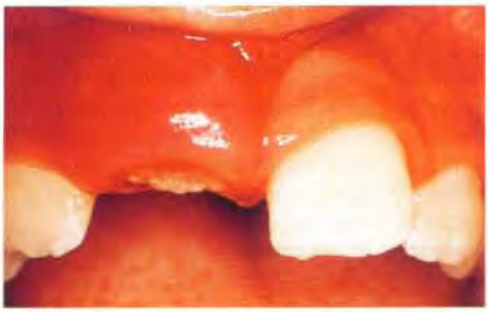


Fig. 18.1 The patient's anterior dentition on presentation.

HISTORY

Complaint

The child is brought in as an emergency by her mother, complaining of a broken front tooth.

History of complaint

Two hours prior to presentation the child had slipped at school, hitting her mouth. One front tooth appears to be broken.

Medical history

The child suffers mild asthma, but is otherwise healthy.

Dental history

The child has attended the dentist irregularly but has had no caries and has no experience of operative dentistry. Her mother states that the broken tooth had not appeared normal and may have been decayed.

■ ***What additional questions would you ask and why?***

Did the patient lose consciousness? This would indicate a relatively severe blow to the head and might indicate significant intracranial trauma. If the patient lost consciousness, even for a short period, they should be referred to hospital where they would almost certainly be admitted for 24 hours of observation. In this case the patient did not lose consciouness.

Was a piece of the tooth broken off and was it found? Missing fragments of teeth may have been inhaled, swallowed, embedded in the lip or lost. If a fragment has been found it must be matched to the fracture to determine whether other pieces remain missing and the patient investigated to locate and remove the pieces. In this case no fragment was found.

Has the patient suffered trauma previously? Previous trauma to this tooth could have resulted in arrested root development, disturbed crown formation or pathological mobility prior to this incident, depending on the age and stage of dental development at the time. Such changes could affect treatment and might explain the parent's observation that the tooth was not normal. In this case no previous trauma could be recalled by the parent.

Was the damaged tooth fully erupted before the accident? In early mixed dentition, incisors on opposite sides of the mouth may be at different stages of eruption. At this age it would be expected that eruption would be complete but there is wide variation in eruption date and rate. It would be possible to misinterpret incomplete eruption as an intrusion injury if the original degree of eruption were not known. In this case, the child's mother reported that both front teeth were fully erupted.

What object or surface did the child hit with her mouth? Injury on surfaces such as playgrounds, roads and pavements carries the risk of contaminating the wound with dirty particulate material. Sometimes such foreign material even enters intraoral wounds. Thorough debridement would then be required. It would also be necessary to check the child's immunization status for tetanus prophylaxis and arrange a booster dose if required. In this case, the child hit the edge of a table.

EXAMINATION

Extraoral examination

The child is distressed but is readily examined. There is some slight swelling of the upper lip but no external abrasions or lacerations.

Intraoral examination

■ ***The appearances of the teeth are shown in Figure 18.1. What do you see?***

The gingival tissues labial to the upper right permanent central incisor are erythematous and swollen. The crown of the tooth appears to be missing and less than 1 mm of the

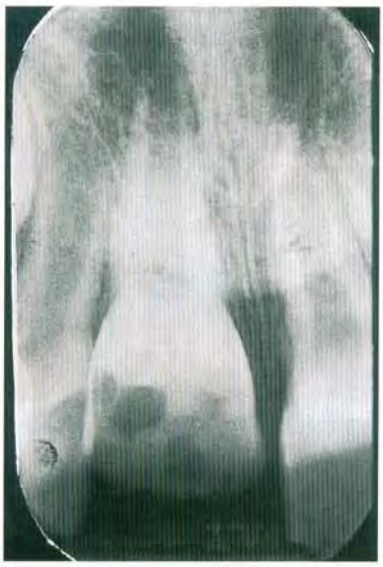


Fig. 18.2 Periapical radiograph.

decayed and causes predating the current injury are the most likely. A number might be considered:

Cause	Merits as a diagnosis
Loss of enamel/dentine as a direct result of the trauma	Such an injury would be unusual and the circular pattern is difficult to explain.
Dental caries	Caries in this distribution also seems unlikely. Labial surface caries is usually found following the gingival margin in individuals with poor oral hygiene. This child is caries free and has good oral hygiene.
Enamel hypoplasia or hypomineralization	Could affect single or multiple teeth. Hypomineralization was noted in the incisal third of the enamel of the adjacent teeth. Hypomineralization could not result from the present injury because the teeth were erupted.
Resorption defects	These are a possibility but the distribution would be unusual and some cause, such as previous injury, might be expected. Resorption could not occur so rapidly after the current intrusion but may be seen on the root surface some months following an intrusive luxation.

Enamel hypoplasia or hypomineralization would appear to be the most likely cause.

■ *Why is there a horizontal dark line on the radiograph across the crown of the upper left central and upper right lateral incisor?*

It is the edge of the soft tissue shadow of the upper lip.

DIAGNOSIS

■ *What is your final diagnosis?*

The patient has an intrusive luxation to the permanent central incisor. This tooth also has several discrete hypoplastic enamel defects which were present before the accident.

TREATMENT

■ *What types of tissue injury result from intrusion and what are their complications?*

Injury	Complication
Crushing and rupture of the periodontal fibres	Bacterial infection or inflammation tracking along the periodontal ligament. Increased risk of root resorption. Weakened periodontal attachment.
Crushing, devitalization and scraping off of cementum	Transient surface root resorption with the possibility of more extensive external resorption and ankylosis in the longer term.
Crushing of the apical neurovascular bundle (and the pulp itself in immature teeth)	Loss of pulp vitality.

■ *Will the tooth re-erupt or should it be surgically repositioned?*

All mature teeth (closed apex) and over 60% of immature teeth become nonvital as a result of intrusive luxation. Therefore, it is advisable to reposition the tooth as rapidly as possible so that access to the pulp chamber can be facilitated before pulp necrosis occurs. Intruded teeth with open apices do have the potential for re-eruption, but if this has not commenced within 1 week, intervention is required. There is at present no evidence to indicate the optimal treatment for the intrusive luxation of permanent teeth. Given sufficient cooperation, immediate surgical repositioning of the tooth will immediately restore the appearance. This should be followed by a short period of splinting of 7–10 days. This option may, however, increase the likelihood of external root resorption and loss of marginal bone support. Relatively rapid orthodontic extrusion over a period of 3–4 weeks is considered less traumatic and less likely to induce resorption.

■ *What immediate treatment is indicated?*

Immediate treatment aims to prevent subsequent external root resorption, preserve marginal bone support and

prevent sepsis. Teeth with a closed apex should be treated by immediate pulp extirpation and placement of a non-setting calcium hydroxide root canal dressing. Immature teeth should be monitored for spontaneous re-eruption and loss of vitality. A 5-day course of systemic antibiotics should be prescribed, and the false gingival pocket surrounding the intruded crown gently irrigated with chlorhexidine.

■ *What follow up should you arrange?*

Follow-up period	Reason
1 week	To monitor spontaneous eruption and vitality of immature teeth, or to remove the splint and change the calcium hydroxide paste in a tooth treated by immediate repositioning.
3 weeks and 6 months	To continue monitoring spontaneous re-eruption of an immature tooth. Replace calcium hydroxide dressing. Radiograph.
6-monthly and then annually for several years	To observe for delayed onset of external root resorption.

In this case, spontaneous eruption was awaited, but was very slow. Electric pulp testing indicated early pulp necrosis. The tooth was then extruded rapidly with a simple orthodontic appliance engaged on to a bracket attached to the labial surface of the intruded tooth. As soon as there was adequate access to the pulp chamber, the necrotic pulp was extirpated, the canal cleaned and obturated with non-setting calcium hydroxide paste. The appearance of the extruded tooth is shown in Figure 18.3 and it confirms the diagnosis of enamel hypoplasia made radiographically.

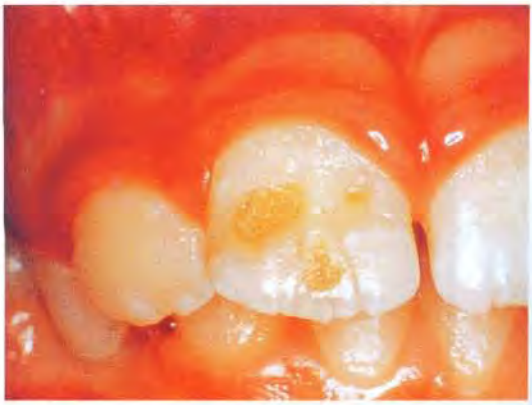


Fig. 18.3 Appearance of the extruded upper right central incisor.

■ *How would your management have differed if the patient had been a 3-year-old child with an intruded primary incisor?*

Mild intrusive luxation injuries in the primary dentition may be treated with reassurance and observation though parents should always be warned that damage to the permanent successor is common. Partial or sometimes total re-eruption over the following months is usual.

However, extraction should be performed without delay if a combination of periapical and lateral radiographs demonstrate that the deciduous tooth has impinged on the follicle of the underlying tooth or if there is subsequent loss of vitality. As in the permanent dentition, vitality must be monitored carefully if the apex is closed at the time of injury. Pulp tests in young children are often unreliable because of lack of understanding, and a close watch must be kept for the slightest colour change.

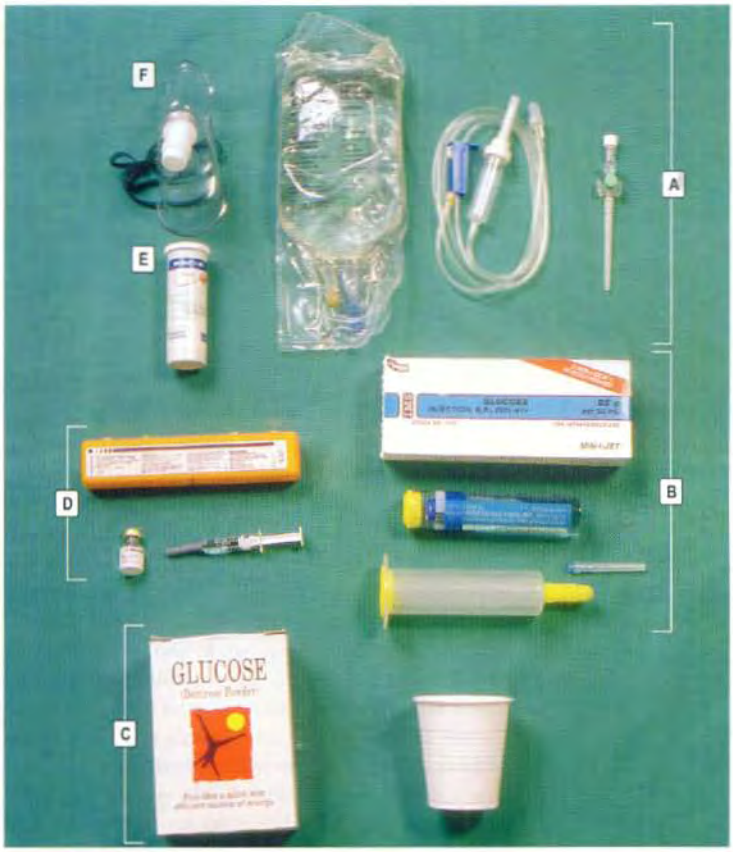


Fig. 19.1 Typical presentations of drugs used to treat hypoglycaemia.
A. Infusion set with 20% glucose.
B. Glucose 50% in Min-I-Jet format. The yellow plastic cover is removed from the back (left hand end) of the syringe barrel and front of the glass cartridge and the cartridge is screwed into the syringe barrel. Available in two types, with needle fitted and with luer lock fitting for a conventional needle (shown). After removing front cover and fitting needle, if required, use as a conventional syringe.
C. Glucose powder, dissolve 20 grams in up to one cup of water.
D. Glucagon emergency set with vial of lyophilised powder. Dissolve by injecting water for injection already in the syringe, and draw up for injection.
E. Blood glucose dipstick test strips.
F. Oxygen mask, give 5 litres/minute.

third molars are partially erupted and appear vertically orientated and there is mild inflammation of the attached gingivae surrounding both crowns. The upper third molars are overerupted and nonfunctional. The patient has a pronounced gag reflex when the teeth are examined.

INVESTIGATIONS

■ *Would you take radiographs? If so, which views would you take and why?*

Yes, radiographs are required to assess root morphology, degree of bone impaction, proximity to inferior dental nerve and the possibility of associated disease (e.g. cysts, hypercementosis and temporomandibular joint problems).

The views to be possibly taken are listed, with their advantages and disadvantages, in Table 21.2.

There is little to choose between these radiographic views in terms of radiation dose, provided fast films and appropriate intensifying screens are used.

In this case the patient's gag reflex prevented the taking of paralleling technique periapicals and so a panoramic tomograph was taken. It is shown in Figure 21.1.

■ *What does the radiograph show?*

The patient is fully dentate with no restoration or caries visible on the film. The lower third molars are vertically

Table 21.2 The radiographic views

View	Advantages	Disadvantages
Periapicals of upper and lower third molars	Provided the periapicals can be taken with a paralleling technique these are the ideal views. They provide a geometrically accurate projection with true relationships to the adjacent structures. They are also convenient for single extractions. These views are the first choice.	Unfortunately it may not be possible to obtain films using the paralleling technique because of patient tolerance. Placement of the film in the ideal position, showing the teeth and inferior dental nerve canal, is uncomfortable. If films are angled then a degree of distortion is inevitable.
Oblique laterals	Readily taken without specialized equipment. Show both upper and lower third molars without superimposition. Give a good view of the surrounding bone when adjacent lesions (e.g. cysts) are present. It is the second-best option.	Suffer a degree of distortion as the beam is angled upwards, so that the relationship to adjacent structures is not accurate.
Panoramic tomograph	Convenient survey film if equipment available. Gives a good view of the surrounding bone when adjacent lesions (e.g. cysts) are present. Though only third choice on technical merit, panoramic films are often used and in practice usually provide sufficient information to assess extractions.	Poor image quality because the view is a tomograph. In addition there is superimposition of the opposite angle of mandible over upper and lower third molars. The upward beam angle distorts the relationship between teeth and adjacent structures and the image is magnified. Root morphology often cannot be assessed on panoramic films.
Lower oblique occlusal	Useful when the lower third molar lies horizontally and is seen end-on in a periapical view. Provides information on buccolingual orientation. Useful if tooth lies out of the line of arch. Used only rarely.	

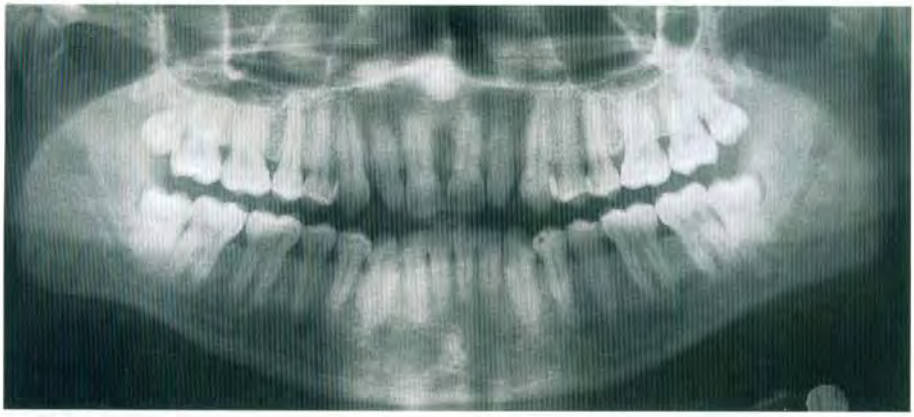


Fig. 21.1 Panoramic tomograph.

orientated and impacted against soft tissue rather than the second molars. The impacted teeth are of normal size and the surrounding bone appears to be of normal density. The roots of both teeth appear to be closely related to the inferior dental nerve canal, there is darkening but no narrowing or deflection of the bony wall of the canal, suggesting that it does not contact or pass through the tooth root.

■ ***You now have sufficient information to decide whether the third molars should be removed or not. What are the indications for removal?***

There has been much debate about indications for removal of third molars, and those for removal of asymptomatic third molars are particularly contentious. Mandibular impacted third molars (MITMs) are very common, affecting approximately 75% of 20–30-year-old patients. Surgery is unpleasant, carries risks and is expensive to state or patient; thus, following accepted guidelines is essential.

The suggested indications for removal are:

- Recurrent pericoronitis and pericoronitis with acute spreading infection.
- Unrestorable caries of MITM or adjacent teeth.
- Untreatable periapical inflammation.
- Periodontal disease associated with the MITM or adjacent teeth.
- Internal or external resorption of MITM or adjacent teeth.
- MITM in fracture line.
- Associated cysts or neoplasia.
- For tooth reimplantation.
- For orthognathic surgery or restorative treatment.
- Prophylactic removal may be advised in specific medical conditions.

■ ***Should this patient's teeth be removed and why?***

Yes, he has suffered two episodes of pericoronitis. There is a greater risk of future episodes as the number of attacks increases, and they are likely to become more frequent and more severe.

■ ***How will you decide whether extraction of this patient's third molars is within your ability?***

Easier extraction	More difficult extraction
Young patient	Patient aged over 30
Female patient	Male patient
Caucasoid/Mongoloid racial stock	Negroid racial stock
Superficial impaction	Deeply buried
Mesioangular or vertical impaction	Distoangular or horizontal impaction
Small crown	Wide crown
Conical root	Multirooted, divergent roots
Lying buccally in relation to line of arch	Lying lingually in relation to line of arch
Clear path of delivery, usually forward and upward	Vertical or distal path of removal required, possibly requiring tooth section
External oblique ridge well posterior to tooth	External oblique ridge overlies tooth
Sound second molar	Crowned, root-treated or heavily restored adjacent molar
Normal second molar root morphology	Conical root (risks accidental elevation)
Distant from inferior dental nerve	Adjacent to inferior dental nerve
Large dental follicle	Narrow dental follicle or ankylosis
Good access	Poor access (e.g. due to trismus)
Not impacted or soft tissue impacted	Impacted against bone or root of second molar
	History of complex or difficult extraction

This is a matter of judgement. You must judge your own ability and experience against the likely difficulty and also consider your ability to manage any complications. In general the following factors should be considered: The most important of these factors may be remembered using the mnemonic WHARFE:

W angulation using **W**inter's lines
H **H**eight of mandible
A **A**ngle of second molar
R **R**oot form and development
F size of **F**ollicular sac
E **E**xit path of tooth to be extracted

■ ***What are 'Winter's lines' and how might they help assess difficulty?***

To apply Winter's lines, three imaginary lines are drawn on the radiographic image (Fig. 21.2). For descriptive

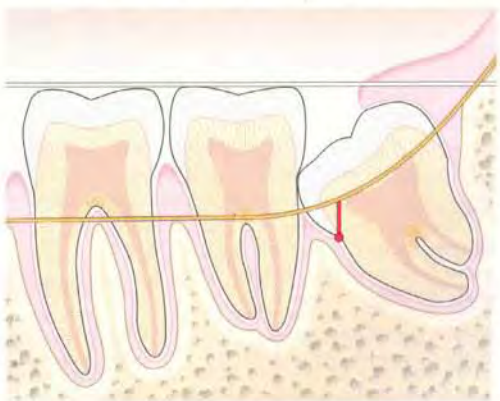


Fig. 21.2 Example of the application of Winter's lines.

22 A phone call from school

Summary

An 11-year-old schoolboy has avulsed a permanent incisor tooth. What would you do?

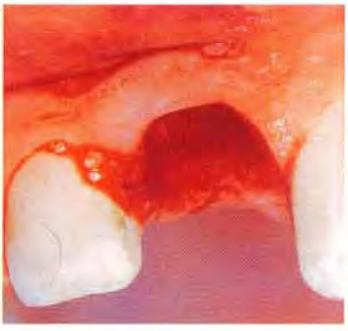


Fig. 22.1 The patient on presentation.

HISTORY

Complaint

The school nurse from a nearby primary school telephones your general dental practice to ask for advice. A child has slipped in the school playground and knocked out a front tooth. The accident happened less than 5 minutes ago, the tooth has been found and it is wrapped in a tissue.

■ *What information would you want from the school nurse?*

Are there any other injuries? It should be established whether there has been any loss of consciousness or signs of concussion. Disorientation and impaired response to simple questions may indicate a brain injury that would require immediate hospital assessment. Limb extremity injuries are common in playground accidents, and the school nurse should be asked to establish whether there is any suspicion of limb fracture or lacerations that require suturing. Dealing with these might be a higher priority than the avulsed tooth.

Is there any relevant medical history? Reimplantation of an avulsed tooth is contraindicated in a child predisposed to infective endocarditis. Any known clotting defect could result in problematic bleeding from a tooth socket.

Has the child an up-to-date tetanus immunization? The school should have a record of immunization status, with particular reference to antitetanus immunization.

Have the parents been informed? Ideally the parents should be notified of the injury and of any intended treatment prior to it being carried out. If at all possible, a parent or legal guardian should be encouraged to either accompany the child or to meet at your practice (or hospital if indicated by other injuries).

What age is the child? The age will determine whether the tooth is primary or permanent and, if permanent, the likely stage of root development.

Is the tooth intact? This is difficult for a lay person to ascertain, but you should ask the caller to carefully examine the tooth, ideally without touching it, or at least without touching the root. A crown fracture would be easier to describe than a root fracture, but if a tapering root of approximately 1.5 cm in length is present, the tooth is probably intact.

Is the root contaminated with dirt or debris? The root surface must be visibly clean prior to any attempt at reimplantation. Dirt on the root must be cleaned off though any asphalt or gravel 'tattoo' on the incisal edge is not relevant at this stage.

You are told that the child is healthy and that their only other injuries are minor grazes on knees and palms of both hands. These are no signs of head injury.

■ *What advice can you safely give over the telephone?*

Keep the tooth wet. The first advice should be to place the tooth in a cup of cold fresh milk. Storage in tap water is undesirable, as its hypotonicity reduces viability of the periodontal ligament cells adhering to the root.

Clean off visible contaminants. Rinse the root gently under cold running water for a maximum of 10 seconds to remove dirt from the root surface. The tooth will be slippery to hold but it should be held by the crown only, and under no circumstances should the root surface be scrubbed or scraped. Debris that does not rinse off may be carefully dabbed off with a clean, ideally sterile, cotton-wool bud.

If you can, reimplant the tooth straight away. The speed with which the tooth is replanted is the most important factor in determining a favourable outcome following replantation. A responsible adult should reimplant

extirpation risks external resorption and discolouration of the crown.

Endodontic treatment should be commenced before the splint is removed. The necrotic pulp tissue is extirpated, the root canal cleaned and irrigated with sodium hypochlorite and a non-setting calcium hydroxide paste is placed in the canal and sealed appropriately. A definitive gutta percha root canal filling is usually delayed for 6–12 months.

■ *Why not extirpate the pulp and root fill the tooth while it is avulsed?*

Even if pulp extirpation is required, it should never be carried out extraorally as the need to manipulate the tooth would almost certainly cause more damage to the periodontal ligament and any delay reduces the chances of a successful outcome.

■ *What are the main complications of replantation of an avulsed permanent tooth? How are they managed?*

FOLLOW UP

■ *What are the aims of treatment and chances of success?*

The aim of replantation is to maintain the tooth for as long as possible in the child patient thus avoiding the need for prosthetic replacement for as long as possible. While there is no frank sepsis or overt discomfort to the patient, the original tooth is the ideal space maintainer.

A failing tooth should be preserved until the patient has reached skeletal maturity, when they could be assessed for an osseointegrated implant. However, in the case of severe resorption or infraocclusion, extraction and immediate replacement with a space maintainer are required.

In one large prospective study of 400 replanted teeth followed up for 5 years, 30% were eventually lost. Immature teeth have a worse prognosis than mature teeth. The chances of successful revascularization of immature teeth is low at 34%. The periodontal ligament heals in 90%

Complication	Features	Management
External inflammatory root resorption, thought to be the result of bacteria or their products passing from the pulp chamber through the dentinal tubules to induce inflammation in the periodontal ligament and resorption of root and bone.	Recognized radiographically, irregular crescentic areas of resorption on the external root surface and radiolucent areas in the adjacent alveolar bone. Prompt diagnosis and treatment are essential as the process may be very rapid, especially in immature teeth.	The infected necrotic pulp tissue or existing root filling must be removed as soon as possible and the root canal should be dressed with calcium hydroxide. A definitive root canal filling should not be placed until the inflammatory resorption has arrested.
External replacement resorption. Following ankylosis, the tooth becomes fused to bone and is remodelled and progressively replaced by bone as part of the process of normal bone turnover.	The ankylosed tooth has a high percussion sound, reduced mobility and eventually becomes infraoccluded as a result of reduced alveolar growth. Radiographically there is loss of definition of periodontal ligament space and dentine. Replacement resorption takes place more rapidly in the growing child.	There is no treatment for external replacement root resorption.

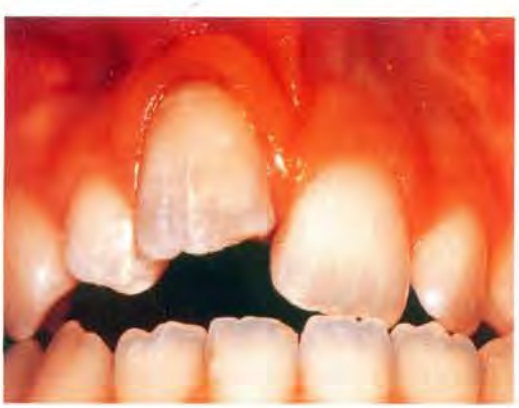


Fig. 22.2 The patient aged 14 years.

Fig. 22.3 Periapical radiograph at age 14 years

23 Discoloured anterior teeth

Summary

A 22-year-old woman presents at your general dental practice surgery complaining of the poor appearance of her teeth. What is the cause and what treatment is appropriate?

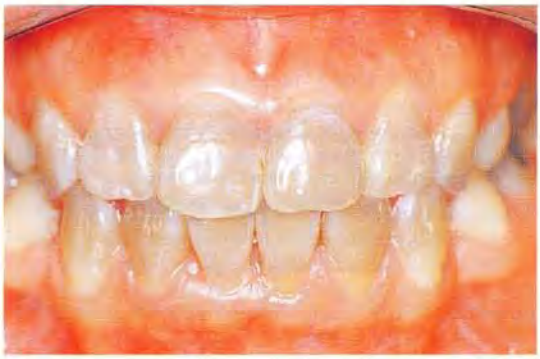


Fig. 23.1 The appearance of the teeth.

HISTORY

Complaint

She is unhappy with the colour of the teeth which she feels are becoming darker. She is very conscious of them and realizes that she is reluctant to smile because of their appearance.

History of complaint

The teeth looked grey on eruption but they have slowly darkened.

Dental history

The patient has had very little dental treatment but received regular preventive care from your practice until the age of 16. Your notes record that she was given oral fluoride supplementation as a child. This was provided as fluoride drops at a dose of 0.25 mg daily from birth to 2 years and 0.5 mg daily as tablets from 2 to 4 years, rising to 1 mg daily from 4 to 12 years of age.

Medical history

The patient is fit and healthy with no relevant medical conditions noted on her medical history questionnaire.

■ *What are the possible causes of discolouration of teeth? What features of each cause aid differential diagnosis?*

The possible causes and relevant features are presented in Table 23.1.

■ *What specific questions would you ask this patient? Explain why.*

Did she suffer any illness between birth and 6 years? This might account directly for the discolouration or could have been the reason for antibiotic treatment with tetracyclines. Further information on chronological hypoplasia will be found in Case 47.

What toothpaste was used during fluoride supplementation? The fluoride supplementation regimen provided for this patient was recommended during her childhood, but the doses would now be considered too high. On these doses, a small proportion of patients would be expected to show mild fluorosis. More severe fluorosis would be associated with a second source of fluoride. The most probable additional source would be ingestion of adult-formula fluoride toothpaste, though living in an area with fluoridated water should also be excluded.

Is there a family history of tooth discolouration or tooth loss? A positive family history aids diagnosis of inherited defects and is essential for diagnosis of some types of amelogenesis imperfecta.

In response to your questioning the patient tells you that she remembers taking many courses of antibiotics as a child for chest infections. She cannot remember what toothpaste she used before the age of 6, but for as long as she can remember she has used an adult paste. She has no family history of similar defects.

EXAMINATION

Intraoral examination

On examination the oral mucosa is healthy and oral hygiene is good. The dentition is unrestored.

■ *The appearance of the anterior teeth is shown in Figure 23.1. What do you see? How do you interpret the appearance?*

The morphology of the tooth crowns is normal and the incisors, canines and premolars are a grey-brown colour. There are some areas which appear less affected and

Table 23.2 Treatment options

Option	Advantages	Disadvantages
Vital bleaching agents using carbamide (or urea) peroxide	Work best with extrinsic stains and quite well for many intrinsic stains. Easily applied in custom trays, nondestructive and easily repeated if necessary. Does not alter the underlying tooth shade or translucency. If sufficient and even lightening of the shade is achieved, bleaching produces the best appearance of all options. Can also be used to mask severe staining before a veneer is placed. This prevents the dark enamel showing through and allows a more translucent veneer to be used, improving the final appearance.	Unpredictable effectiveness with tetracycline staining, often leaving a dark zone cervically where the stained root shows through the thin cervical enamel. However, almost always some improvement and this may satisfy the patient. Only appropriate when there are minimal or no restorations in the teeth. Restorations are not bleached and there is a theoretical concern that bleaching agents might track to the pulp along the margins of restorations. Some over-the-counter formulations are acidic and others may cause local soft tissue irritation, and should not be encouraged. Licensing regulations vary between countries.
Nonvital bleach	Allows bleaching of deeper dentine than a vital bleach, producing greater effect.	Only possible in nonvital teeth and so usually inappropriate for multiple teeth. To bleach dentine below the cervical enamel the bleaching agent must be applied to the cervical part of the root canal as well as the pulp chamber.
Direct composite, indirect composite or porcelain veneers	Good appearance possible, can be as good as crowns but much less destructive.	Some tooth preparation is required, the amount varying slightly between types. The 'emergence profile' or contour at the gingival margin must be maintained by removing cervical enamel, to avoid a plaque trap. When placed over darkly stained teeth, veneer and cement must be opaque. This reduces translucency and produces a 'flat' artificial colour to the finished restoration. Expensive.
Crowns	Strong and retentive, a variety of bonded or reinforced crowns are available if the occlusion is a problem. Very darkly stained teeth are best crowned. The porcelain is thicker than veneers so that opaque materials are not required. If necessary, metal-bonded crowns completely mask the underlying colour. Usually the best alternative if the teeth contain extensive restorations. Appearance can be excellent.	Destructive of tooth tissue. Margins may compromise periodontal health. Expensive.

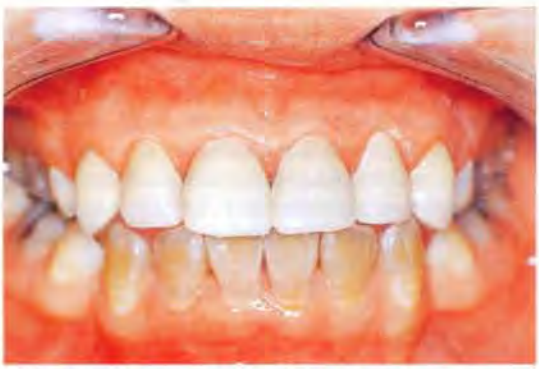


Fig. 23.2 The completed porcelain veneers immediately after cementation.

of having to use opaque veneers is well shown. However, the patient was very happy with this result.

■ ***What is the long-term prognosis for these veneers?***

The veneers on the upper right canine and lateral incisor are in crossbite with the lower canine and almost edge to edge on the lateral incisor. On the upper left the same teeth are edge to edge. There is a risk of chipping the incisal edges and debonding.

PREVENTION

■ ***Tetracycline should no longer be prescribed to those below the age of 12. Presumably tetracycline staining should no longer be seen?***

This is true, but unfortunately courses of tetracycline are still occasionally prescribed for children. There are some specific

24 A very painful mouth

Summary

A 20-year-old man presents to you in your general dental practice, feeling ill and with a very sore mouth.

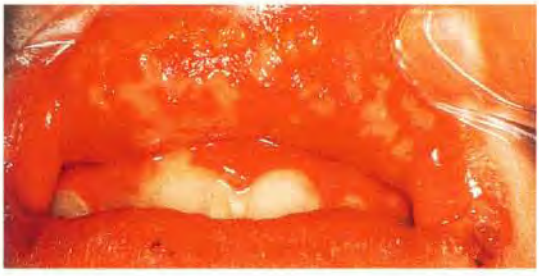


Fig. 24.1 Appearance of the patient's mouth.

HISTORY

Complaint

The patient complains of pain which is preventing eating and hampering drinking. He also feels unwell.

History of complaint

He first noticed feeling unwell 4 days previously and thought he had 'flu. He was slightly feverish and developed a headache. His mouth was sore but it was not until about 1 day later that it became very painful. Because he felt unable to take time off work, he took the remains of a course of an unknown oral antibiotic which had been prescribed for his brother who had an infected cut on his arm. This did not appear to have led to any improvement. He has had no similar attacks before.

Medical history

The patient is otherwise fit and well.

EXAMINATION

Extraoral examination

The patient has enlarged cervical lymph nodes which are slightly tender, mobile but soft or firm rather than hard. Apart from this finding no abnormalities are found in a routine examination of the head, neck and hands.

Intraoral examination

■ *What do you see in Figure 24.1?*

There are numerous ulcers on the labial mucosa which have the following characteristics:

Site	Labial mucosa and attached gingiva
Size	A few millimetres in diameter
Shape	Well defined, rounded, sometimes coalescing to form larger irregular ulcers
Colour	Covered by a yellow-grey fibrin ulcer slough, no well-defined rim of periulcer erythema
Background	The surrounding mucosa appears uniformly inflamed

In addition, one large ulcer lies at the commissure and there are small bloodstained crusts around the lips.

If you were able to examine the patient you would discover that more ulcers affect much of the oral mucosa, including the gingivae, palate and tongue, and that they extend back into the oropharynx.

■ *Give a differential diagnosis on the basis of the information you have so far.*

- Primary herpetic gingivostomatitis
- Erythema multiforme

■ *Justify this differential diagnosis.*

Primary herpetic gingivostomatitis and other oral viral infections typically cause multiple round small ulcers of acute onset, sometimes coalescing, on a background of inflamed mucosa. The patient feels unwell and has enlarged tender lymph nodes suggesting infection. Primary *Herpes simplex* infection usually affects much of the mucosa and has a predilection for the keratinized masticatory mucosa of the gingiva. The patient is older than is normally expected for a primary infection. However, the average age of patients with this infection has increased over the last few decades because improved living conditions have resulted in fewer individuals coming into contact with the virus during their childhood.

Erythema multiforme (Stevens–Johnson syndrome) is possible. The acute onset and bloody crusts on the lips suggest this diagnosis and the age of the patient is compatible. However, the distribution of ulcers is not particularly suggestive of this condition. Erythema multiforme usually primarily affects the lips and nonkeratinized lining mucosa of the anterior mouth, and the

- To determine all sources of error and allow their correction
- To increase efficiency
- To reduce costs
- To reduce the radiation dose to patients and staff

■ ***What areas or topics should a quality assurance programme address?***

There are six main areas or topics of concern that should form the basis of any QA programme. The essential procedures for each should be laid down in writing and should be the responsibility of a named person.

- Personnel and training
- Image quality
- Working procedures
- Patient dose and X-ray equipment
- Darkroom, films and processing
- Audit

■ ***How can image quality be easily assessed in general dental practice?***

One recommended method of assessing image quality is to visually compare every radiograph with a good quality standard reference film and to subjectively rate the quality using published criteria such as the example shown below, which comes from the *Guidance Notes for Dental Practitioners on the Safe Use of X-ray Equipment* (Department of Health, UK, 2001).

Rating	Quality	Basis
1	Excellent	No errors of patient preparation, exposure, positioning, processing or film handling
2	Diagnostically acceptable	Some errors of patient preparation, exposure, positioning, processing or film handling, but which do not detract from the diagnostic utility of the radiograph
3	Unacceptable	Errors of patient preparation, exposure, positioning, processing or film handling, which render the radiograph diagnostically unacceptable

■ ***What should be done with the 'unacceptable' category 3 films?***

They should be collected over a suitable time period (e.g. a week or a month) and then analysed. This analysis is often referred to as *Film Reject Analysis*. The category 3 films are assessed as to why they have been rejected and to identify the causative error. Changes to practice can then be instituted to avoid the error being repeated.

■ ***The following four intraoral radiographs have been rejected as diagnostically unacceptable (category 3). What is wrong with each and what are the possible explanations?***

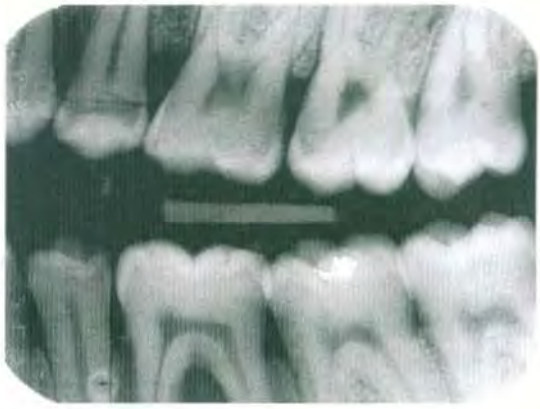


Fig. 25.2 A bitewing film.

■ ***What is wrong with the film in Figure 25.2?***

The film is too dark. There is contrast between enamel and dentine but it is not possible to detect the subtler features of the teeth or to see the margin or internal structure in the alveolar bone.

■ ***How might this error have been caused?***

The film can be too dark for three reasons, each of which has a number of possible explanations:

Reason	Possible causes
Overexposure	Usually the time of the exposure is too great because the incorrect exposure setting has been selected by the operator. The X-ray set timer may be faulty.
Over-development	The developer solution could be too hot or too concentrated. The film could have been left in the developer for too long.
Fogged film	Light leakage in the darkroom, faulty safe lighting or poor film storage. Use of old film as a result of poor stock control.

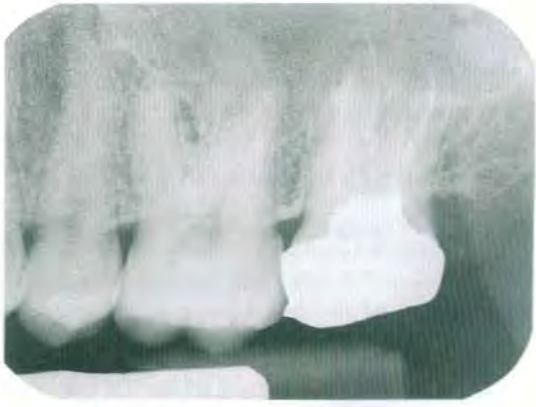


Fig. 25.3 Periapical film of upper molars.

■ *What is wrong with the film in Figure 25.3 (p. 121)?*

The periapical film is too pale. There is insufficient contrast between enamel, dentine and bone and the background is not sufficiently black.

■ *How might this error have been caused?*

The film can be too pale for two reasons, each of which has a number of possible explanations:

Reason	Possible causes
Under exposure	Usually the time of the exposure is too short because the incorrect exposure setting has been selected or the timer switch has not been depressed throughout the exposure.
	The X-ray set timer may be faulty.
Underdevelopment	The developer solution could be too cold, too dilute or too old.
	The film could have been left in the developer for too short a time.

■ *What is wrong with the film in Figure 25.4?*

The bitewing image is blurred or unsharp and the molar teeth have been 'coned-off' i.e. the corner of the film has not been exposed.

Fig. 25.4 Another bitewing film.

■ *How might these errors have been caused?*

Error	Possible causes
Blurring	Patient movement during the exposure.
'Coning-off'	The X-ray tubehead has been placed too high and was not aiming directly at the film packet. The straight edge of a rectangular collimator/spacer cone has prevented X-rays reaching part of the film.

■ *What is wrong with the film in Figure 25.5?*

The periapical image is geometrically distorted and has been elongated to such a degree that the apices of the lateral incisor and canine are not shown.

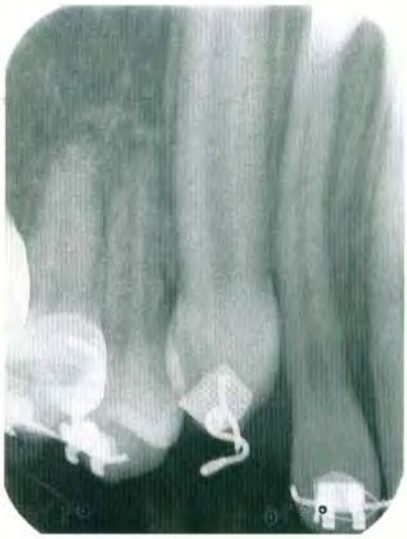


Fig. 25.5 Periapical film of upper canine.

■ *How might this error have been caused?*

Error	Possible causes
Elongation	The film has been taken using the bisected angle technique and the X-ray tubehead has been positioned at too shallow an angle with respect to the teeth.
	The film could have been bent in the mouth by excessive pressure from the patient's finger supporting the film packet.

■ *Where are the regulations governing dental radiography and radiology for UK dental practitioners published?*

In the UK the 2001 *Guidance Notes for Dental Practitioners on the Safe Use of X-ray Equipment* is essential reading. This booklet brings together guidelines on good practice and the legislative requirements of the Ionising Radiation Regulations 1999 and the Ionising Radiation (Medical Exposure) Regulations 2000 (IR(ME)R 2002) as they apply to dentistry. These regulations encompass the principles of the International Commission on Radiological Protection (ICRP) of **Justification, Optimization** and **Limitation.**

26 Ouch!

Summary

You sustain a needlestick injury. What should you do next?

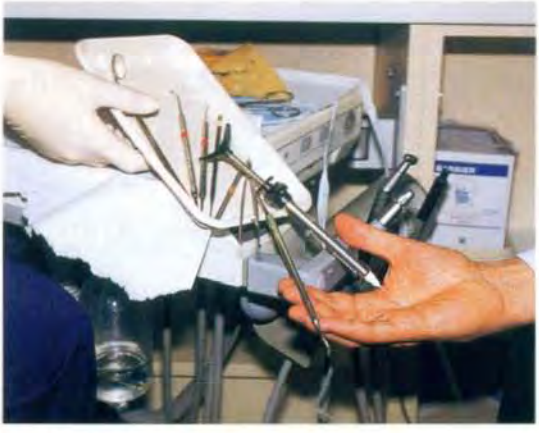


Fig. 26.1 An unfortunate reflex reaction.

Problem

You are writing up the notes after your last patient of the day. Out of the corner of your eye you see your nurse tip an instrument tray over the edge of the bracket table and instinctively lean over and put out your hand to catch it. The local anaesthetic syringe falls onto your hand. The needle sheath has not been replaced and the needle penetrates about 5 mm into your palm. When you pull out the needle the injury bleeds.

■ *What diseases of significance may be transferred by needlestick injury?*

Most infectious diseases can be transmitted by needlestick injury but the main concerns are hepatitis B, hepatitis C and HIV infection.

■ *What would you do immediately?*

- Encourage bleeding at the injury site and wash it with either 70% alcohol, antiseptic handwash or soap and water. Do not scrub the injury.
- Send the nurse or receptionist to ask the patient to return to the surgery as a matter of urgency.

■ *What is the most urgent priority and why?*

The most urgent priority is to assess whether there is a significant risk of transmission of HIV infection. Postexposure prophylaxis with antiretroviral drugs can significantly reduce the chance of transmission of HIV, but for maximum effectiveness it is recommended that it is administered within 1 hour.

■ *How could you obtain postexposure prophylaxis if required?*

Postexposure prophylaxis is only available following a formal risk assessment for each individual injury. This involves determining the severity of the injury and the risk that the patient is carrying HIV infection.

The procedure for obtaining a formal risk assessment varies with local circumstances. In hospitals, the infection control consultant(s), hospital casualty or occupational health department will perform the risk assessment and provide the appropriate medication. Those in general practice must contact their local hospital casualty department who will follow their local guidelines. Each dental practitioner should know the contact number and name/position of the appropriate person.

When you phone you will be asked details of the injury and patient. You will then be told whether or not the injury is sufficient to carry a risk of transmission and whether a risk assessment of the patient is required. In general, a needlestick injury after a dental injection is sufficiently serious to warrant a formal risk assessment.

■ *What is the risk of developing HIV infection following a needlestick injury?*

On average the risk is approximately 0.3% if the needle has been used on an infected patient, but the exact risk varies.

■ *What factors affect the risk of transmission?*

The risk depends on the type of injury, the degree of contamination and the infectivity of the material transferred.

The risk of transmission is greatest with a penetrating injury. The risk rises if the needle has entered an artery or a vein because this allows a greater amount of blood to be transferred to the recipient. Dental local anaesthetic needles have a very fine bore and transmit very little fluid to the recipient of a needlestick injury, providing no pressure is being applied to the plunger. It is unusual for a needle to enter a blood vessel during local analgesia but because almost all dental syringes aspirate, potentially infectious blood or tissue fluid must be assumed to lie in the needle and local anaesthetic cartridge.

Occurrences Regulations 1995). Note the time of injury carefully. Evidence that procedures have been followed correctly would be important if a claim were to be made for Industrial Injury Benefit or insurance purposes in the event that you do contract an illness from the needlestick injury. Any incident in which acute ill health results from occupational exposure to isolated pathogens or infected material must be reported centrally.

■ ***This injury could have been avoided if the needle had been resheathed. What are the advantages and disadvantages of resheathing needles?***

In general, needles should be disposed of immediately after use and this is the usual practice in most areas of medicine. When disposable syringes are used with needles they may be easily disposed of together.

In dentistry, resheathing a needle has the advantage that it may be reused for the same patient later in the appointment and at the same time it is made 'safe' until disposal. If a needle is to be left ready for reuse it must be resheathed. Resheathing also reduces the risk of injury when removing the needle from the sterilizable syringe body, though it does not eliminate the risk completely.

Several methods exist for safely resheathing needles. In general simple methods are best and one-handed resheathing (picking up the sheath on the needle while holding the syringe in one hand) has this advantage. The sheath must still be pressed into place by hand to ensure that it does not fall off again. Holders can be used to support the needle sheath upright and these avoid both the need to seat it by hand and to chase the sheath around the bracket table with the needle. An example is shown in Figure 26.2. Alternative methods include proprietary devices where a sheath is an integral part of the needle or syringe. However, these still need to be used correctly if they are to be effective.

Fig 26.2 A simple needle sheath holder. The holder is not intended to hold the syringe upright, only to hold the sheath during resheathing.

■ ***This injury has ruined your day. This has all proved so complex that next time you might just wash the injury and ignore it. Why not?***

The main reason is the worry that you might contract HIV infection from an unsuspected carrier. The effectiveness of postexposure prophylaxis, reducing the risk of transmission by 70%, cannot be ignored. Also, it would be unethical for a dentist not to follow up the possibility of developing an infection which could jeopardize patients' wellbeing. There would also be a risk of transmission to the dentist's sexual partner(s).

27 A swollen face and pericoronitis

Summary

A 23-year-old woman presents in your hospital casualty department with a painful swelling of the right side of the face and neck. What is the cause and what treatment would you provide?

Further information on the diagnosis of soft tissue infection will be found in case 41.

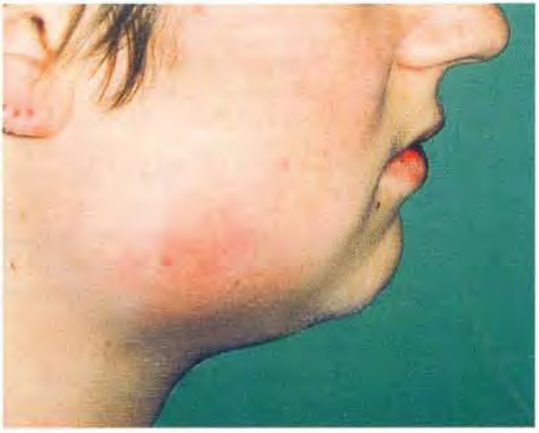


Fig. 27.1 The patient on presentation.

HISTORY

History of complaint

The patient has suffered worsening pain 'from her wisdom tooth' on the lower right side for 5 days. There has been some swelling of the gum around the tooth and she has been unable to bite together for a couple of days. Yesterday she noticed pain in the floor of her mouth and found that moving her tongue was painful. Today she awoke to find the facial swelling, she feels unwell and has difficulty eating, swallowing and opening her mouth.

She had an episode of pericoronitis a few months ago and is on her local hospital waiting list to have all third molars extracted. Until the swelling developed, she thought this was just another attack of pain from her wisdom teeth. She has not had facial swelling before and has come straight into hospital.

Medical history

The patient is otherwise fit and well.

EXAMINATION

Extraoral examination

There are palpable tender lymph nodes in the upper deep cervical chain and submandibular triangle. Opening is limited to 15 mm interincisal distance.

There is swelling below and around the lower border and angle of the mandible and extending back towards the neck. The swelling is hot, tender and very firm and a dusky red colour centrally. The swelling is not pointing to the skin. There is a marked halitosis.

Intraoral examination

Trismus hampers examination. The lower right third molar can be seen to be partially erupted, the operculum is swollen and pus exudes from below it on gentle probing. The second and third molars appear caries-free.

The floor of the mouth is very tender and firm on the right side.

■ *What additional examinations or investigations would you perform? Explain why.*

It is extremely important to take the patient's temperature to determine whether the infection is exerting systemic effects. She has a temperature of 37.8°C (normal temperature 36.8°C) and is, therefore, pyrexic.

There is a need to confirm that pericoronitis is the cause. It would be prudent to exclude the possibility that this is infection from a nonvital molar and tests of vitality should be performed. If there were a suggestion from the examination that a lower molar was nonvital, a radiograph might be indicated, otherwise radiographs would provide little useful information for diagnosis unless another lesion were present.

DIAGNOSIS

■ *What do these findings tell you?*

The combination of inflammation (swelling, pain, redness and heat) together with local lymphadenitis and pus seen intraorally indicate an infection. Pericoronitis is present and this appears to be the primary source of the infection. Trismus is an important sign, indicating that the infection or inflammation has spread to involve the muscles of mastication.

The patient is pyrexic and feels unwell. These features indicate that the infection is exerting a systemic effect. Infection appears to be spreading relatively fast because the swelling has appeared overnight and there are already systemic signs.

■ *Which type of infection is this?*

It is difficult to tell because the tissues involved are deeply sited. Pus is draining from under the operculum indicating abscess formation, but this might extend into a soft tissue space or be limited to the tissues around the unerupted tooth. The rapid spread, firmness and tenderness of the tissues ('brawny' swelling) indicate cellulitis. This might continue to spread or develop into an abscess. There is probably a mixed infection with a local pericoronal abscess and a spreading cellulitis.

■ *To which tissue spaces may infection spread from a lower third molar? What are the boundaries of these spaces?*

Pus from lower third molars may track to many spaces and spread is unpredictable, depending on many factors including the angulation of the tooth, the size of the follicle, relationship to the second molar, degree of bone loss around both teeth and the anatomical relationships between the teeth, bone and muscle attachments in the region. Pus may drain into the mouth from under an operculum, into the buccal or lingual sulcus or into one or more tissue spaces. The routes of spread to tissue spaces are shown in Figure 27.2 and are described in Table 27.1.

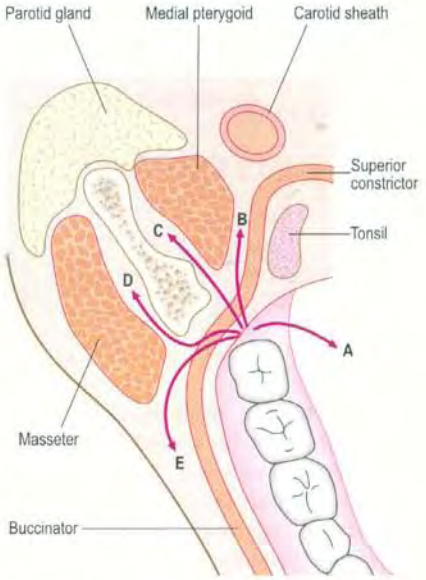


Fig. 27.2 Paths of spread of infection into tissue spaces from third molars: **A,** into the sublingual and submandibular space; **B,** into the parapharyngeal space; **C,** into the pterygomandibular space leading to the infratemporal fossa; **D,** into the submasseteric space; **E,** into the buccal space.

■ *In what tissue spaces is the present infection tracking and why?*

This swelling appears to be in the submandibular space. The main infected tissue is not visible and lies around the submandibular gland deep to the body of the mandible. The

Table 27.1 Paths of spread of infection from lower third molars

Direction of spread	Tissue space	Boundaries
Medially above the attachment of mylohyoid	Sublingual space, A	Lies between the floor mouth and mylohyoid muscle with the body of the mandible laterally.
Medially below attachment of mylohyoid	Submandibular space, A	Lies between mylohyoid muscle and platysma, with the hyoid bone medially and the lower border of the mandible laterally. Contains the submandibular gland.
Posterior and medial to mandibular ramus, medial to lateral pterygoid muscle	Parapharyngeal space, B	Lies between superior constrictor muscle and the pterygoid muscles with the pterygoid plates.
Posterior and superior, between mandibular ramus and lateral pterygoid muscle	Infratemporal space via C which communicates with the cavernous sinus	Base of skull superiorly, laterally sigmoid notch of mandible and temporalis muscle, medially lateral and posterior wall of maxilla.
Posterior and medial to mandibular ramus, lateral to lateral pterygoid muscle	Pterygomandibular space, C (and potentially on into the infratemporal space)	Lies between lateral and medial pterygoid muscles and the ascending ramus of mandible. Extends up to base of skull.
Posterior and lateral to mandible ramus	Submasseteric space, D	Lies between masseter muscle and the ascending ramus of the mandible.
Posterior and superiorly, lateral to buccinator	Buccal space, E	Between the buccinator muscle and skin

28 First permanent molars

Summary

A 7-year-old girl has pain from a first permanent molar. What is the cause and how might it affect her dental development?

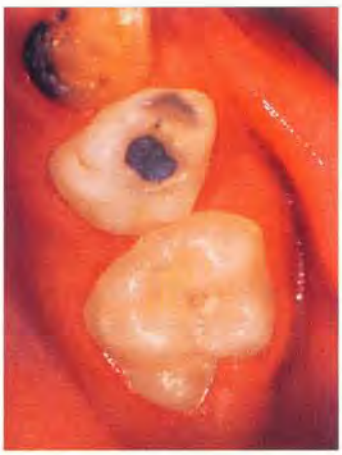


Fig. 28.1 The appearance on presentation.

HISTORY

Complaint

The patient's mother reports that the child suffers intermittent spontaneous discomfort from the upper left quadrant.

History of complaint

The symptoms have been vague, no sleep has been lost and there has been no facial swelling. The patient has complained of the pain three or four times over the last month.

Medical history

The child is fit and well.

Dental history

The child has been a regular patient since the age of 3. She has required restorations in four primary molars, one requiring local analgesia. Despite intensive preventive advice and diet analysis, new carious lesions have been present at each recall visit.

EXAMINATION

You ask the child to point to the painful tooth and she points to an apparently sound upper left primary canine.

■ *The appearance of the upper left quadrant is shown in Figure 28.1. What do you see?*

- An amalgam restoration with ditched or raised margins on the palatal aspect of the first primary molar.
- An apparently sound amalgam restoration in the second primary molar.
- Possible caries in an occlusal pit on the second primary molar.
- An erupting first permanent molar with the occlusal surface not fully through the mucosa.
- A small occlusal cavity in the confluence of the mesial fissures of the permanent molar.
- Plaque or food debris in the fissures.

■ *How do you interpret the information so far and what are the likely diagnoses?*

The child is probably pointing at the wrong tooth. The canine appears intact and children are often poor historians. They often have difficulty in localizing the source of pain if the pain is not present at the time of examination.

Pulpitis appears likely because the pain appears poorly localized and is relatively intermittent. A history of hot or cold or sweet exacerbating factors would point to this diagnosis. The likely causes are caries beneath a restoration or carious or traumatic pulpal exposure in one of the primary molars. Any primary molar with an unrestored carious cavity or even a clinically sound restoration should be examined closely for signs of pulpal necrosis.

■ *What features might suggest a necrotic pulp?*

- Extension of caries or fracture into the pulp
- Discolouration of the crown
- Swelling or tenderness in the buccal sulcus adjacent to the tooth
- Pus draining from a sinus in the mucosa, usually buccally but occasionally lingually or palatally

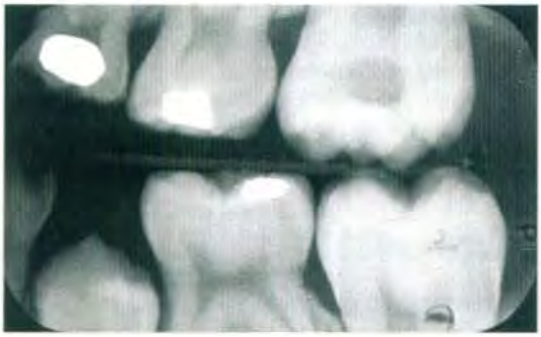


Fig. 28.2 The left bitewing radiograph.

- Pus draining from the gingival margin
- Facial swelling
- Well-localized pain

None of these symptoms and signs is present. Pulpitis seems likely.

INVESTIGATIONS

■ *What investigations are indicated? Why?*

- Bitewing radiographs to check the proximity of restorations to the pulps, the extent of the occlusal caries in the permanent molar and to detect small proximal surface carious lesions.
- Clinical examination of the other permanent molars for caries.
- Tests of vitality of primary and permanent molars are unlikely to help because the results are unreliable in children.

■ *The left bitewing radiograph is shown in Figure 28.2. What do you see?*

DIAGNOSIS

■ *What is your final diagnosis?*

Pulpitis in the upper left first permanent molar, in response to a deep occlusal carious lesion. On the basis that pain has been intermittent, the pulpitis is probably reversible but the rapid progression of the caries and proximity to the pulp may herald involvement of the pulp. Caries must be excavated to discover the true extent of the caries.

■ *How can caries be so extensive beneath such a small enamel cavity?*

This pattern of caries is sometimes referred to as *occult* or *hidden* caries. This presentation was much less common prior to the 1970s, when caries was more prevalent, lesions larger and fluoride becoming ubiquitous. It has been speculated that the widespread use of fluoride toothpaste has resulted in enamel that is resistant to smooth surface caries. Fluoride in toothpaste has little effect on the progression of dentine caries and is less effective against fissure caries. Carious lesions in fissures that progress to dentine might well be able to enlarge greatly before the occlusal enamel cavitates significantly. In a partly erupted tooth such as this there is no significant occlusal force to fracture away any unsupported enamel. This hypothesis seems feasible but there is no evidence that the prevalence of occult lesions is affected by fluoride exposure. An alternative hypothesis is that there is external resorption of the crown before eruption, hollowing out the crown through a small occlusal defect. After eruption the cavity would become carious. Coronal resorption is usually considered unusual but it is difficult to prove that it was not present after caries supervenes without pre-eruption radiographs. Radiographs are required to diagnose occult lesions.

■ *Does this discovery have significance for other teeth?*

Yes, it will be necessary to examine all the other permanent molars clinically again. Carious lesions may have been

Table 28.1 Features seen in Figure 28.2

Tooth	Features
First primary molar	The restoration appears to be in the pulp chamber but reinspection of the tooth clinically will remind you that the restoration is on the palatal aspect of the tooth and is only superimposed on the pulp chamber. No proximal surface caries is present.
Second primary molar	The restoration appears sound, with no radiolucency between the base of the restoration and the pulp to suggest caries. There is sufficient sound dentine (approximately 1–2 mm) between the restoration and the pulp to make pulpal exposure unlikely. No proximal surface caries is present.
First permanent molar	There is an extensive dentine radiolucency under the small occlusal cavity indicating caries. The deep aspect of the lesion is ill-defined, indicating rapid progression. The lesion extends close to the distobuccal pulp horn and there may be involvement of the pulp.

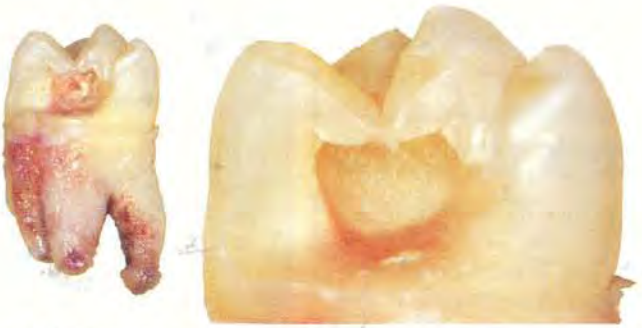


Fig. 28.3 The extracted tooth.

dental development rather than the chronological age is important. The crucial factor is the stage of development of the lower second molar, because this determines how satisfactorily space will close in the lower arch. The range of acceptable development is from crown completion to mineralization of one-third of the root. One authority quotes the appearance of an inter-radicular crescent of bone in the second molar as the optimal stage for first molar extraction (at approximately 9½ years of age). Radiography is required to confirm the stage of development, and ensure that third molars and second premolars are present and normal.

■ ***Does extraction of first permanent molars complicate orthodontic treatment?***

It used to be said that extraction of all four first permanent molars doubled orthodontic treatment time and reduced the prognosis. This saying held good only for removable appliance treatment and does not apply to fixed appliance treatment. However, closure of residual lower first molar spaces does pose complex anchorage problems and it will sometimes be preferable to delay the extraction of compromised but symptom-free first molars until the second permanent molars have erupted. This is a further reason why a full orthodontic assessment is required.

FURTHER INFORMATION

■ ***The extracted tooth is shown in Figure 28.3 What do you see?***

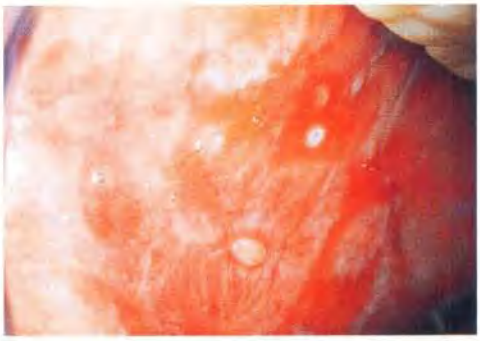
Part of the crown has been removed to expose the carious cavity. On the left you can see a small periapical granuloma at the apex of the palatal root. The pulp was therefore nonvital despite the lack of symptoms. On the right you can see a large defect extending from the enamel to the pulp. The central occlusal enamel is completely unsupported but has not fractured. The carious dentine is hardly discoloured because it is rapidly progressing.

29 A sore mouth

Summary

A 55-year-old gentleman presents to you in general practice complaining of a sore mouth. You must make a diagnosis and institute treatment.

a



b

Fig. 29.1 a, b The patient's right and left buccal mucosa on presentation.

HISTORY

Complaint

He complains of an extremely sore mouth and the recent appearance of white patches on his cheeks. He thinks he may be allergic to his dentures.

History of complaint

The patient was fitted with a new set of complete dentures 3 weeks ago and since then his mouth has become progressively more sore. In recent days he has noticed the appearance of white patches on his cheeks. He had not noticed these before.

Medical history

One year ago the patient was diagnosed as a non-insulin-dependent diabetic and he has a history of peptic ulceration. Current medications are metformin and ranitidine. He is otherwise fit and well.

EXAMINATION

Extraoral examination

The patient appears fit and well. No cervical lymph nodes are palpable.

Intraoral examination

The patient is edentulous and his complete dentures are stable and retentive. The appearance of the right and left buccal mucosa is shown in Figure 29.1. Despite its abnormal appearance the mucosa is freely mobile with no evidence of tethering or scarring. Other parts of the oral mucosa appear healthy and the mouth is well lubricated by saliva.

■ *Describe what you see on the buccal mucosa.*

The buccal mucosa is affected bilaterally by poorly defined ulcerated red and white lesions. These extend from the commissural region to the retromolar area, as well as vertically into the upper and lower buccal sulci. The white areas are arranged as diffuse zones but some have reticular keratotic striae within them and around their borders. Irregularly shaped erythematous zones lie around the white areas and some have ulcers centrally. There are two large oval/linear ulcers approximately a centimetre in length on the left and one smaller ulcer on the right. The ulcers have yellow fibrinous sloughs on their surfaces and appear relatively superficial and flat rather than deep or punched out. No bleeding is evident.

■ *Suggest a differential diagnosis.*

1. Lichen planus
2. Lichenoid drug reaction
3. Lupus erythematosus

■ *Justify this differential diagnosis.*

The combination of white, red and ulcerated areas alone is highly suggestive of one of these three conditions, though it could also be seen in a number of other mucosal diseases

evidence to implicate a drug and this can be helpful when deciding whether or not to stop or adjust the dose of an important medication. Though not present in this case, lichen planus can form plaque-type lesions and these must be sampled to exclude dysplasia. Patients with high alcohol or tobacco consumption should have a biopsy to exclude dysplasia because lichen planus has a very low risk of malignant transformation. For this patient, an incisional biopsy is indicated.

■ *Which part of the lesion would you remove for biopsy?*

The centre of ulcers must be avoided because inflammation may mask histological features. However a sample of the ulcer margin may be useful and a piece including ulcer margin and red and white areas should be selected. Ideally some normal mucosa is always included in biopsy specimens, but in this case almost all the mucosa is affected. The specimen should be elliptical, about 1 cm long, 5–6 mm wide and an even 3–4 mm in depth. A biopsy specimen was removed from the left buccal mucosa and is shown later in Figure 29.3.

■ *What other investigations would you perform?*

Microbiological tests. When lichen planus or a lichenoid reaction become symptomatic or extensively ulcerated the possibility of additional candidal infection should be considered. The thick keratotic epithelium is more prone than normal epithelium to infection. The patient is further predisposed to candidal infection by non-insulin-dependent diabetes. A smear from the surface of the lesions on each side is an ideal investigation. Saliva sampling for candidal counts may also be helpful. This has the advantage that the organism is cultured for complete identification and sensitivity testing to antifungal agents. The disadvantage is that it does not specifically sample the lesion. In this case the patient is a complete denture wearer and is therefore likely to have an elevated salivary candida count. A smear is the better choice in this case and one was taken from the left buccal mucosa. It is shown in Figure 29.2.

Fig. 29.2 Periodic acid-Schiff (PAS) stained smear from buccal mucosa.

Autoantibody screen. If lupus erythematosus is a possibility, an autoantibody screen may provide evidence to support the diagnosis. A serum sample should be sent for antinuclear antibody (ANA) determination. Four-fifths of patients with systemic disease are ANA-positive, often having high titres. A high titre of anti-double-stranded DNA (dsDNA) antibody is almost exclusive to SLE but is positive in only 50% of cases. In discoid lupus erythematosus this is less helpful in diagnosis because only a quarter of patients have antinuclear antibodies. Individuals with lichen planus or lichenoid reaction should have no antinuclear antibody. In this case the autoantibody screen was negative.

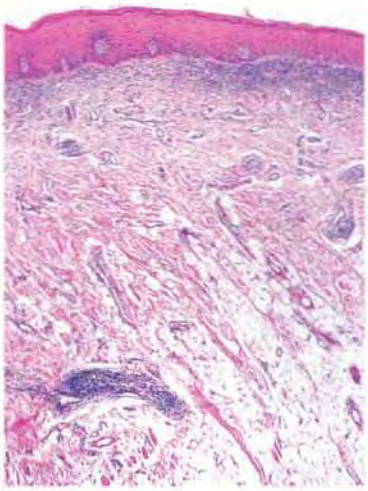
■ *The smear is shown in Figure 29.2. What do you see and how do you interpret the features?*

The smear is stained with periodic acid–Schiff (PAS), which stains the carbohydrate in fungal cell walls a magenta colour. Gram stain may also be used to detect fungi; *Candida* stains strongly Gram-positive. A sheet of pale pink-stained buccal epithelial cells is present, together with a few dispersed cells. Numerous dark pink branching fungal hyphae are growing in and around the epithelial cells. There are also several small round blastospores budding from the hyphae. The fungus is dimorphic and branching, and the size and appearance are typical of *Candida* sp. The patient has candidosis.

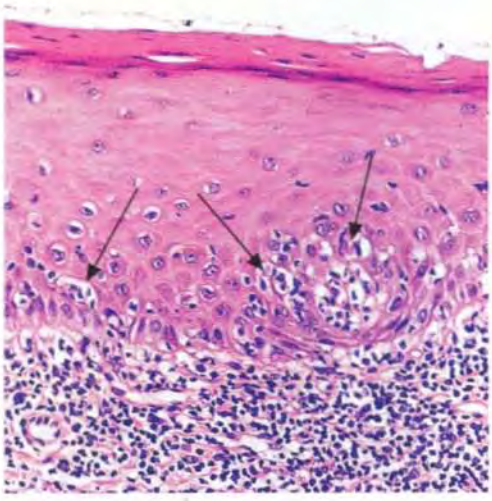
■ *The biopsy is shown in Figure 29.3. What do you see?*

The low power view shows mucosa with underlying fat. The surface epithelium is slightly thinner than normal buccal epithelium and has a surface layer of keratin. There is a well demarcated inflammatory infiltrate in a band immediately below the epithelium in the superficial connective tissue. The band is denser towards each side of the picture. (At this magnification the cells cannot be definitely identified as inflammatory cells but this is the most likely explanation for the very cellular zone.) There are also several foci of inflammatory cells in the deeper tissues, one particularly large one associated with a vessel near the bottom of the picture. The basement membrane is prominent.

The higher power view shows the interface between the epithelium and connective tissue. The very cellular layer can be seen to be composed of lymphocytes. Lymphocytes have infiltrated into the basal and suprabasal layers of the epithelium and caused the basal epithelial cells to undergo apoptosis. Apoptotic cells are visible as shrunken very pink cells with nuclear remnants (arrowed). There is no



a



b

Fig. 29.3 Buccal biopsy; haematoxylin and eosin. **a,** Low power view; **b,** higher power view.

remaining clearly defined basal layer of small darkly stained cells and the cells lying at the basement membrane have the appearance of prickle cells. The surface is parakeratinized. Buccal epithelium is normally nonkeratinized, though a thinner layer than this may be present along the occlusal line as a result of friction.

■ *How do you interpret the histological findings?*

The dense band-like infiltrate of lymphocytes and lymphocytic infiltration of the basal cells with focal basal cell degeneration, apoptosis, loss of basal cells and a thickened basement membrane are typical of lichen planus. The deeper infiltrates of inflammatory cells around blood vessels suggest that this is the result of a systemic process rather than one localized to the epithelial–connective tissue interface. This suggests a lichenoid reaction rather than lichen planus as a cause. However, it is not usually possible to differentiate lichen planus and lichenoid reactions on histological grounds alone, For this reason the biopsy diagnosis is 'consistent with lichen planus or a lichenoid reaction'. The dentist must ensure that this histological diagnosis is compatible with the clinical features and results of any other investigations before finalizing the diagnosis.

DIAGNOSIS

■ *What is your final diagnosis? Explain why.*

Lichenoid drug reaction with superimposed candidosis. The clinical presentation is typical of a lichenoid reaction or a severe atrophic lichen planus, the diagnosis is supported by biopsy and the patient is taking a drug known to cause such reactions. The clinical appearance does not suggest lupus erythematosus and the autoantibody screen was negative.

■ *What drugs can cause lichenoid reactions?*

A very large number of drugs may be associated with the development of a lichenoid reaction. Reactions to gold injection may be particularly severe and prolonged. Drugs of the following types cause lichenoid reactions:

- allopurinol
- captopril
- chloroquine antimalarials
- gold
- beta blockers
- methyldopa and related antihypertensives
- nonsteroidal anti-inflammatory drugs
- oral hypoglycaemic agents
- penicillamine
- some antidepressants
- occasionally other drugs.

■ *What treatment or advice would you recommend?*

Firstly the candidal infection must be treated. Denture hygiene must be checked and night wear ceased if appropriate. In view of the mucosal inflammation and ulceration, an antifungal agent should be prescribed and amphotericin or nystatin would be appropriate. Subsequently, intermittent chlorhexidine mouthwashes may help prevent repeated attacks of candidosis.

Corticosteroid preparations would be helpful for the underlying lichenoid reaction. The mode of corticosteroid

30 A failed bridge

Summary

A 40-year-old man has a missing upper incisor replaced by a spring cantilever bridge. This has become decemented and you must assess options for replacement.

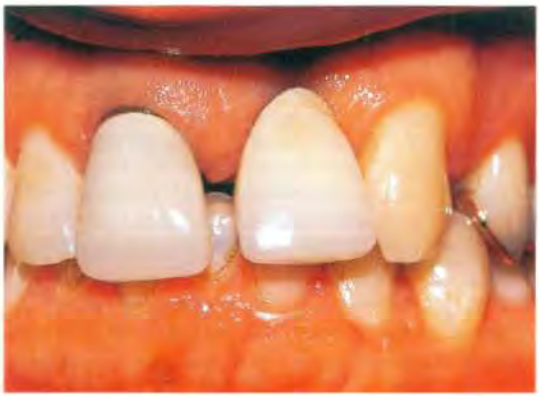


Fig. 30.1 The patient at presentation with the bridge which replaced the upper left central incisor reinserted.

HISTORY

Complaint

The patient complains that his anterior bridge has become detached. He would like it recemented or replaced.

History of complaint

The bridge had been satisfactory for many years but detached about 2 years ago. It was recemented and had been firm until yesterday when it fell off the teeth without warning.

Dental history

The upper left central incisor had been lost as a result of a bicycle accident when the patient was aged 16. It was completely avulsed and the adjacent upper right central incisor was fractured. The missing central incisor was initially replaced with a simple spoon denture and then a few years later by a spring cantilever bridge attached to full coverage crowns on the left first and second premolars. The other upper central incisor was root treated and a post crown fitted. The present bridge is a replacement made about 8 years ago after the cantilever spring fractured. The patient has never had an upper left lateral incisor.

EXAMINATION

Intraoral examination

The dentition is in good condition with few carious lesions and a small number of restorations. The upper left premolars are the abutment teeth and have relatively conservative crown preparations. There is superficial caries over much of the surface of the first premolar crown and a larger cavity at the distal gingival margin. The mesial surface of the second premolar is also slightly carious. Both abutment teeth are vital. The gingival condition is good except for bleeding on probing between the abutment premolars. Here the probing depth is 4 mm. The bridge can be replaced and the appearance with it fully seated is shown in Figure 30.1. The caries in the first premolar is exposed below the crown margin.

■ *What is the prognosis for this bridge? Why?*

Hopeless. Figure 30.1 shows that the cosmetic result is not good. The bridge pontic has moved buccally and upwards, probably a combined result of alveolar ridge resorption and distortion of the spring cantilever. It also appears to have moved distally increasing the median diastema. The abutment teeth will both require re-restoration and the first premolar appears to be very carious. In the long term, both abutment teeth are compromised by the risk of further caries and periodontitis.

■ *Why was this method of replacing the central incisor chosen originally?*

Although a well-designed partial denture should not compromise the health of the remaining dentition, most patients prefer a fixed prosthesis without palatal coverage for a single tooth replacement.

The spring cantilever design was considered suitable for this case for the following reasons.

- It allows diastemas between adjacent crowns. Diastemas would have been present because the lateral incisor on that side was developmentally absent. A replacement crown which filled the available space would be too wide.
- The upper right central incisor was not a suitable abutment tooth for conventional fixed bridgework, having

TREATMENT

■ ***What precautions must be taken when inserting an implant?***

The implant is inserted into a hole which must be made with a drill matched to the implant. The surgical stent mentioned in Table 30.1 is used to ensure the correct angulation and depth so that the implant does not perforate the cortex. Careful sterile technique is essential and particular care must be taken to ensure that the bone is not damaged by overheating. Copious irrigation and slow drill speeds are required. The exact method of insertion and subsequent restoration depends on the type of implant and manufacturer. The implant must be left in place for 3–6 months to allow osseointegration before loading. A spoon denture would provide a satisfactory temporary replacement during this period.

■ ***Figure 30.2 shows the result 3 years after completion and Figure 30.3 the corresponding radiograph. Is the implant osseointegrated and successful? How can you tell?***

Osseointegration is the direct structural and functional connection between living bone and the surface of a load-carrying implant. This implant was firm and symptomless and appears successful radiographically. There is bone in close apposition to the implant surface along its whole length. The end of the implant is level with, or only just above, the surrounding bone. Failure would be indicated by mobility, peri-implant radiolucency or progressive marginal bone loss.

■ ***What factors are important in achieving and maintaining osseointegration and why?***

See Table 30.2.

■ ***At each review, how would you determine whether the implant is successful?***

- The implant is immobile when tested clinically.
- A periapical radiograph does not reveal any peri-implant radiolucency.
- Radiographs taken at annual visits should reveal a steady crestal bone level after the first year of loading. This can only be assessed using periapical radiographs taken by the paralleling technique.
- Vertical bone loss, assessed radiographically, should be less than 0.2 mm annually in subsequent years.
- Absence of signs and symptoms such as pain, infections, or, for mandibular implants, neuropathies, paraesthesia or a violation of the mandibular canal.
- The cosmetic result remains acceptable.

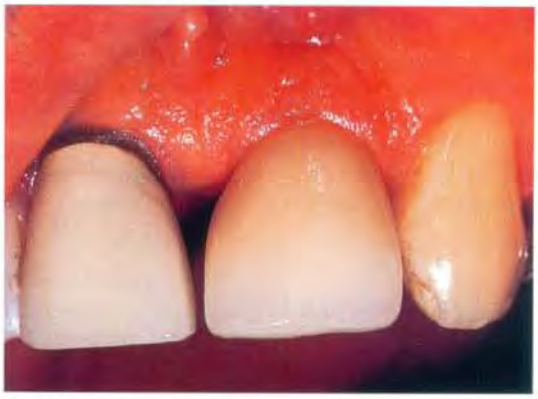


Fig. 30.2 The final result 3 years after completion.

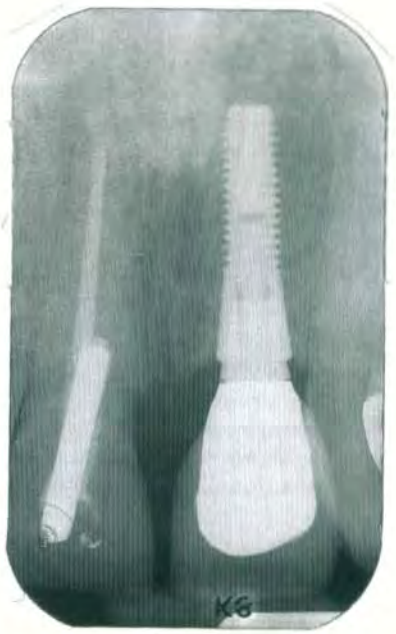


Fig. 30.3 Periapical radiograph taken 3 years after completion.

Table 32.2 Radiographic views

View	Advantages	Disadvantages
Dental panoramic tomograph (DPT)	This is the appropriate initial view for periodontal assessment; also as a general survey for a new patient and to identify the number of implants present and their approximate site.	Distortion and poor resolution make detailed assessment of periodontal bone support and caries difficult, especially around incisors.
Full mouth periapical films	Ideal for assessing bone loss if taken with a paralleling technique. Selected films, based on the panoramic view, would be ideal and still involve a lower dose than full mouth films because the panoramic provides a dose equivalent to only 4 E-speed periapical films.	Not necessary: probing depths around the upper and lower anterior teeth were normal or only slightly increased. Full mouth films cannot be justified as an initial investigation on the basis of the radiation dose.
Vertical bitewings	Ideal for molars and premolars when there is no more than moderate bone loss.	The missing upper molars would have made the positioning of the films difficult, if not impossible.

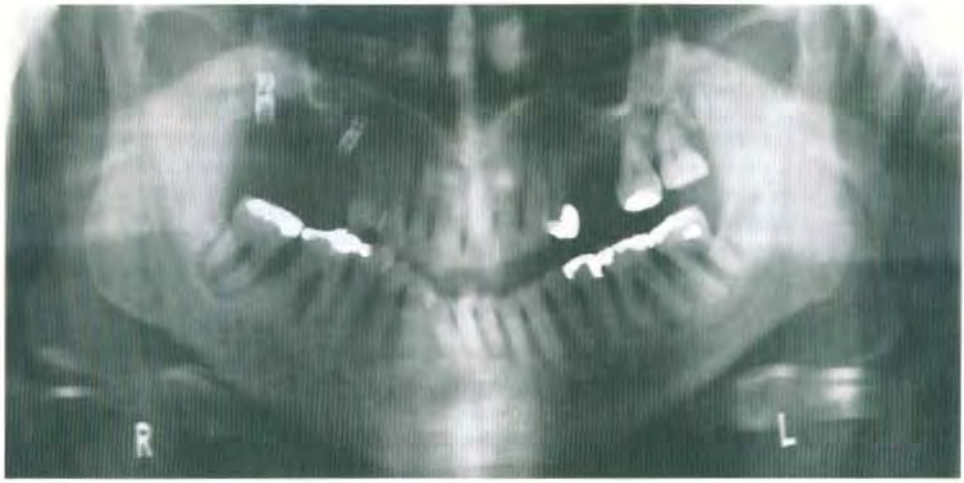


Fig. 32.2 Dental panoramic tomograph of the patient at presentation.

poor positioning and the teeth in the midline appear out of focus because of superimposition. The head was also twisted, enlarging one side of the film. This can be seen most easily by looking at the molar crowns which are wider on the right than on the left. The patient's postoperative film in Figure 32.3 (p. 156) shows what the film should have looked like.

The radiograph shows extensive bone loss around the lower right and left second molars. The lower left second molar has bone loss and caries in the furcation. Furcation involvement was also evident on both lower first molars which were not as mobile.

■ ***Where are the implants? How might you localize them more accurately?***

The panoramic view is insufficient to localize the implants accurately. They appear in focus but the focal trough in the molar region is quite wide so that this gives no clue as to their buccolingual position. Their position and angulation will be critical in determining whether they can be used to support restorations.

If the end of the implant cannot be identified in the mouth, the implants could only be accurately localized using a tomographic technique, either CT scanning or multidirectional tomography. CT scanning is expensive and requires special software to prevent 'star artefact' shadows on the film. Multidirectional (spiral or epicycloid) tomography performed in machines such as the Scanora or Tomax produces cross-sectional images of any part of the jaws much more easily and would be the best method. If the implants were misplaced, the film might be taken with radiopaque markers on the ridge to aid localization. Alternatively it could be assumed that the implants are in an appropriate position and a flap could be raised.

■ ***Are any other investigations necessary?***

Yes, a urine glucose test to exclude diabetes would be prudent. This was negative.

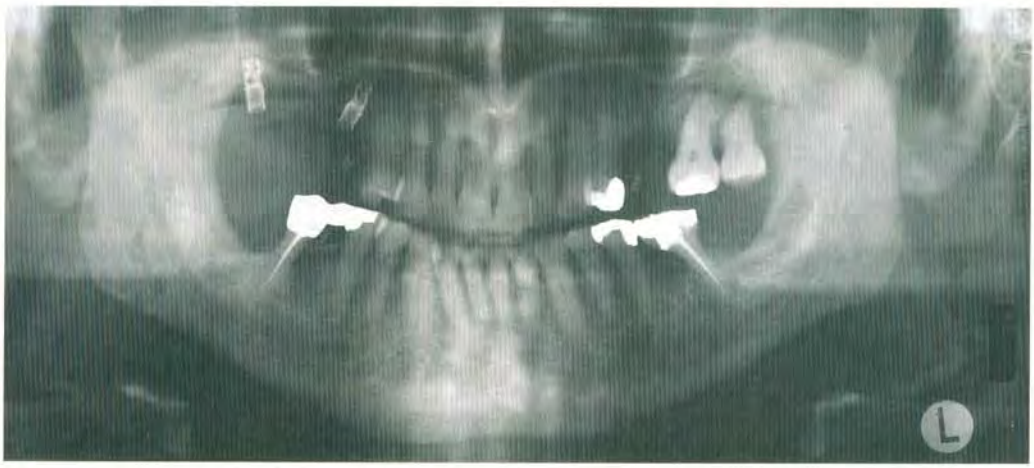


Fig. 32.3 Dental panoramic tomograph showing the result of treatment.

on the lower left molar and hemisection on the right. This eliminates the furcation and enables cleaning. The inflammation around the roots resolves.

■ ***Do the hemisected or root-resected teeth require restoration?***

Ideally, yes. The large area of exposed dentine and risk of fracture of the overhanging crown after root resection really demand full coverage restorations. However, complex and expensive treatment is often avoided because hemisected and root-resected teeth are compromised.

However, hemisected or root-resected teeth which have proved themselves stable over a period of months or years are best restored. In this case the hemisected molar root was linked to the premolar with a fixed movable bridge. Care must be taken that the design of the bridge does not overload the periodontal support of the root. Both teeth remain in function and are excellent semipermanent solutions to this patient's problem.

Restoration, root treatment and surgery add up to a huge investment in time and money spent on one very compromised tooth root. If a definite need for a bridge or denture had been identified at the outset, an implant would have provided the support required at lower cost and with a better long-term prognosis.

■ ***How do you assess the potential usefulness of the implants?***

The position of the implants is not favourable. Even on the panoramic view it can be seen that the fixtures are not parallel making them unsuitable for a fixed prosthesis. The fixtures are small, of different types and partially integrated. The mesial implant would appear to have less bone supporting it, and it is unclear whether it could support significant occlusal load.

33 Fractured incisors

Summary

A 38-year-old man presents to you in your local hospital accident and emergency department. He has fractured his front teeth. You must manage the injury and outline a treatment plan for restoration.

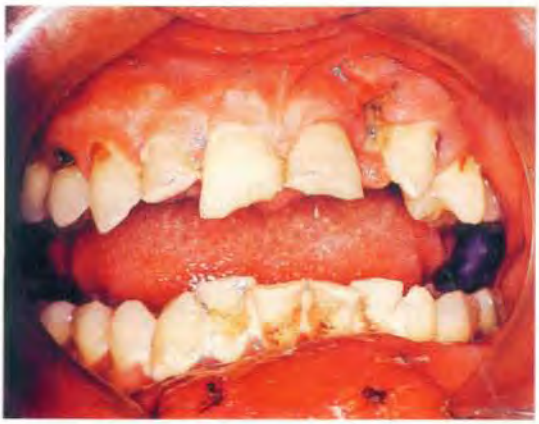


Fig. 33.1 The appearance of the anterior teeth on presentation.

HISTORY

Complaint

The patient's front teeth have been fractured and they are all loose. One tooth was knocked out and he feels pain when he bites.

History of complaint

The car accident occurred yesterday. The patient was sitting in the driver's seat when another car drove into his. He was stationary and not wearing a seat belt and was thrown forward, his lower face hitting the steering wheel. He did not lose consciousness and was taken to a local accident and emergency department where a laceration of his lower lip was sutured and no other injuries were found. At that time his teeth and jaws were not examined or radiographed and he has returned for a follow-up appointment with you.

Medical history

Prior to the accident the patient was fit and healthy, with only allergy to penicillins and erythromycin noted on his medical history questionnaire.

EXAMINATION

Extraoral examination

■ *How will you assess the possibility of a mandibular fracture?*

Fracture is suggested by:

- pain, swelling and tenderness at the fracture site
- bleeding, bruising or haematoma at fracture site
- displacement, step deformity
- change in occlusion
- mobility of fragments or of teeth
- difficulty opening the mouth or in lateral excursion
- paraesthesia or anaesthesia in the distribution of nerves involved in the fracture.

■ *How will you assess the possibility of a fracture of the zygomatic arch or facial skeleton?*

In addition to the features noted above, fracture at these sites may produce:

- facial asymmetry and flattening of facial contour (may be masked by swelling for a few days)
- step deformity along infraorbital margin
- anaesthesia or paraesthesia of cheek, nose, upper lip and teeth
- unilateral epistaxis
- subconjunctival haemorrhage with no definable posterior limit
- restricted eye movements and diplopia.

On extraoral examination you cannot identify a mandibular or facial fracture. The lower lip is swollen and lacerated to the left of the midline. There is no restriction or pain on opening, nor swelling associated with the temporomandibular joint.

Intraoral examination

■ *The anterior teeth are shown in Figure 33.1. What do you see?*

The swollen lower lip is just visible. The upper right lateral and both central incisors have been fractured. The upper left lateral incisor appears to be missing. The upper left canine is not fractured but has caries buccally and is mesially inclined. This inclination could predate the injury, in which case the lateral incisor may have been buccally positioned, or it could be a result of injury.

The oral hygiene is poor. This does not appear to be a result of injury because there are large accumulations of

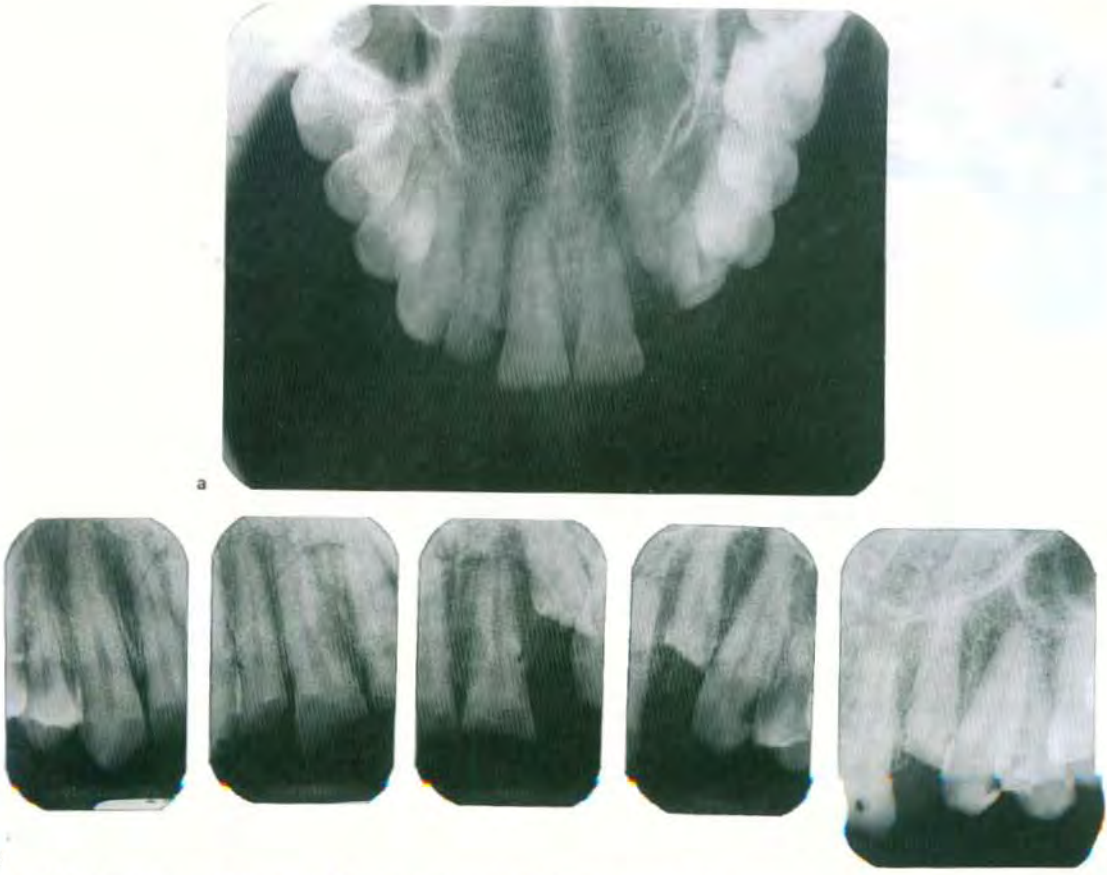


Fig. 33.2 **a** Upper standard occlusal, and **b** periapical radiographs of the upper incisors and canine teeth.

INVESTIGATIONS

■ *What investigations would you perform?*

Intraoral radiographs are required and should include an upper standard occlusal and periapical radiographs of all the upper and lower anterior teeth. The occlusal view and periapicals of the upper teeth are shown in Figure 33.2.

■ *What do the radiographs show?*

The occlusal radiograph shows that the upper left lateral incisor is fractured and intruded rather than avulsed. There is a curved alveolar fracture line running across the premaxilla, extending from the upper right to upper left lateral incisor. It is most obvious where it crosses the roots of the central incisors just below their apices. No root fractures are evident. The upper left canine has suffered a lateral luxation injury; the outline of the original socket can be seen most clearly on the mesial side of the apex. There is also caries distally in the canine.

■ *What emergency treatment would you provide?*

The fractured alveolar process must be immobilized to alleviate pain and promote healing, and this is most easily achieved with an orthodontic wire and composite splint. The splint used in this case is shown in Figure 33.3. No displacement was present and so no reduction was required. The laterally luxated upper left canine requires repositioning before splinting either by manual manipulation or surgically. Manual manipulation was not possible and forceps were used to move the root past a bony obstruction and back into its correct position.

If it is accessible without disturbing the fracture, the upper left lateral incisor root should be surgically extracted as soon as possible.

The upper left canine has a closed apex and either the luxation injury itself or the surgical repositioning will almost certainly cause loss of pulp vitality. Therefore, the pulp must be extirpated from this tooth as soon as possible and a calcium hydroxide dressing placed. Calcium hydroxide has the potential to reduce the risk of root resorption. In

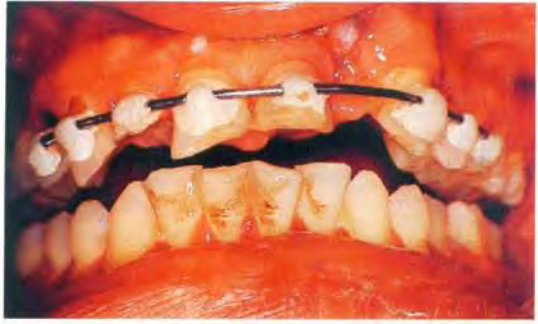


Fig. 33.3 Wire and composite splint in position.

addition the pulps must also be removed from the fractured incisors. These also have closed apices, the pulps have been exposed to infection for 24 hours and the exposures are large. In addition, infection in the fracture line must be avoided. Again, the root canals can be dressed with calcium hydroxide paste and the teeth restored temporarily with composite, carefully checking the occlusion so as not to precipitate further trauma.

A chlorhexidine mouthwash should be prescribed for use until the tissues have healed sufficiently to allow oral hygiene procedures. There is no indication to prescribe antibiotics unless the lip wound was contaminated.

■ ***How long should the splint remain in place? Wh should be done in this period?***

The composite splint should remain in place for about 4 weeks. This splinting period should be adequate for both healing of the alveolar fracture and stabilization of the luxation injury. Some authorities suggest that lateral luxation injuries should be splinted for slightly longer. This can be achieved by selectively separating individual teeth from the splint with a bur. The canine should not be left unsplinted if there is mobility or pain. It is desirable to remove the teeth from splinting as soon as is practicable because it is difficult to isolate them with a rubber dam fo root filling. Permanent endodontics must wait until the spli is removed. If still in place, the upper left lateral incisor ro may now be surgically extracted without disturbing the fracture and replaced with a prosthesis for 6 months to allow alveolar bone remodelling.

Throughout this period, oral hygiene instruction and dietary advice should be given. Success of treatment during this period will determine the long-term options for restoration.

Table 33.1 Types of temporary replacement to be considered

Type of prosthesis	Advantages	Disadvantages
Partial acrylic denture of Every type	Minimal gingival coverage, easy to make and cheap. Does not interfere with orthograde root fillings of adjacent teeth. Acrylic flange masks bone defect following alveolar remodelling.	Patients dislike removable prostheses and they may be difficult for some patients to tolerate. Will require relining following alveolar remodelling.
Composite or denture tooth bonded to adjacent teeth with composite	Fixed replacement with no gingival coverage. No laboratory stage, simple chairside technique. Allows orthograde root filling of adjacent teeth.	Bond may fail because enamel had already been bonded for the etch-retained splint. Difficult to mask space formed beneath pontic following resorption.
Rochette-type adhesive bridge	Fixed replacement with no gingival coverage. Simple cantilever design possible, easily removed by dentist for permanent restoration.	Thickness of wing required may conflict with deep overbite causing occlusal trauma in already compromised teeth. Bond may fail because enamel has already been bonded for the etch-retained splint. Difficult to mask space formed beneath pontic following resorption. Orthograde root filling of abutment tooth not possible.
Adhesive bridge cemented with an adhesive cement (e.g. Panavia 21)	Fixed replacement with no gingival coverage. Simple cantilever design possible. Metal wing thinner than Rochette	Bond may fail because enamel has already been bonded for the etch-retained splint. Difficult to mask space formed beneath pontic following resorption. More difficult to remove, may require ultrasonics. Orthograde root filling of abutment tooth not possible.
Heat-cured acrylic conventional type bridge	Fixed replacement avoiding gingival margins of teeth other than the abutments. Greater range of designs possible including simple cantilever or fixed – fixed design. Best appearance and restores the coronal fractures of abutment tooth/teeth	Difficult to mask space formed beneath pontic following resorption but bridge is easier to remove and modify with composite and replace. Requires tooth preparation and commits patient to a permanent conventional bridge. More destructive than adhesive designs though teeth are already badly fractured. Slightly more difficult to isolate with rubber dam for orthograde root filling of abutment teeth; bridge might need to be removed for treatment.

■ ***What types of temporary replacement would you consider for the upper lateral incisor? What are their advantages and disadvantages?***

See Table 33.1.

In this case a temporary fixed–fixed conventional bridge was chosen, and this is usually the restoration of choice when several teeth are badly fractured. The upper right central and lateral incisor were also crowned, primarily because of the better appearance, although composite restorations would have been possible and might have been preferred if the oral hygiene had not improved markedly. The appearance of the final restorations is shown in Figure 33.4.

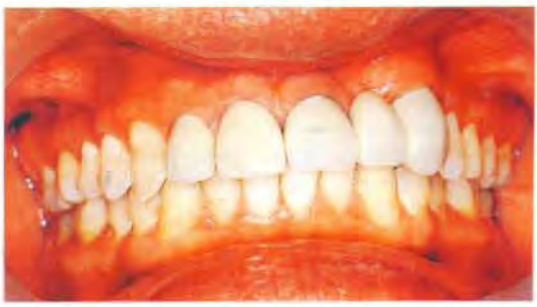


Fig. 33.4 The final restorations in place.

■ ***What complications of the injury require follow up?***

In the short to medium term the other anterior teeth should be monitored for late loss of vitality. The main long-term problem is resorption, either of inflammatory type (following unsuccessful root treatment or persistent inflammation on lateral canals) or replacement resorption (without inflammation) which can lead to ankylosis. These processes start on the outer surfaces of root-filled teeth and must be excluded by occasional radiographs. The risk of resorption can be reduced by removing the splint as soon as possible to encourage early physiological tooth movement. Further features of resorption are covered in Case 14.

35 A blister on the cheek

Summary

A 58-year-old lady patient of your general dental practice complains of a sore mouth with blisters. Identify the cause and outline appropriate management.

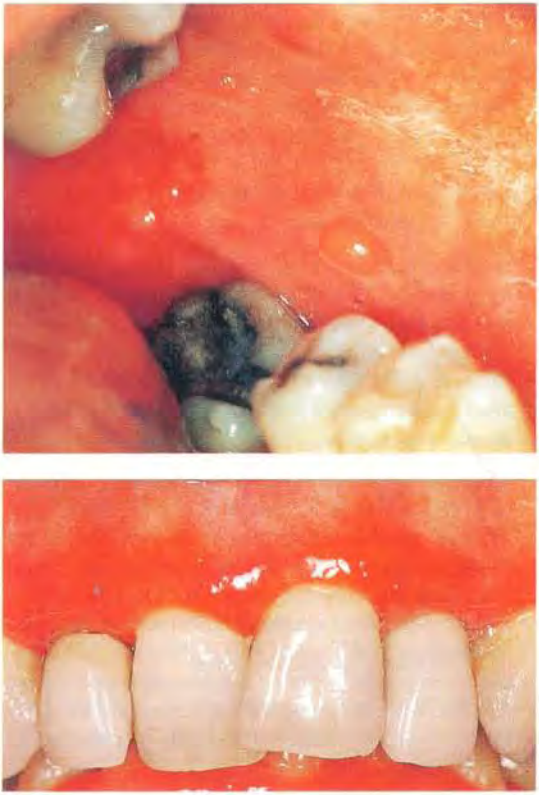


Fig. 35.1 **a, b** The patient on presentation.

HISTORY

Complaint

The patient complains of a very sore mouth. She describes blisters which last a few hours before bursting to release a clear fluid, or sometimes blood. The palate is particularly affected though lesions may develop anywhere in the mouth and often follow minor trauma. Each blister heals very slowly and the area is painful until healing is complete. She often finds she cannot brush her teeth.

History of complaint

The symptoms started about 1 year ago and are worsening.

Medical history

She has had hypertension for many years and her elderly medical practitioner has been treating her with methyldopa.

EXAMINATION

Extraoral examination

A fit-looking woman with a blood pressure of 140/90 when sitting. Visible skin and nails appear normal.

Intraoral examination

■ *The appearance of the buccal mucosa and gingivae is presented in Figure 35.1. What do you see?*

The buccal mucosa has an extensive area of red atrophic mucosa posteriorly, possibly with small ulcers towards the anterior edge. The red area has an irregular margin. A small blister, a few millimetres across, lies near the centre of the buccal mucosa, just above the buccal cusp of the second premolar in the photograph.

The gingivae are also red but no blisters are present. The red area extends from the gingival margin across the mucogingival junction to involve the adjacent alveolar mucosa. The margin is poorly defined. The gingivae around all visible teeth are involved and the distribution of inflammation is not consistent with plaque accumulation as the cause.

DIFFERENTIAL DIAGNOSIS

■ *Which conditions cause oral blisters?*

- Mucous membrane pemphigoid
- Pemphigus vulgaris
- Lichen planus
- Erythema multiforme
- Angina bullosa haemorrhagica
- Epidermolysis bullosa
- Dermatitis herpetiformis
- Viral infections
- Trauma

alone during the first year but develop skin lesions later. Lichen planus may be accompanied by a rash on the wrists, shins or back. Though the rash may resolve long before the oral lesions, it should be specifically sought in the history if lichen planus is a possibility. Erythema multiforme may be accompanied by the typical target lesions, though these usually signify typical severe oral erythema multiforme (Stevens–Johnson syndrome), which is unlike this patient's presentation. A history of onset following specific triggers (e.g. cold sore) would also suggest erythema multiforme.

Do you have any lesions anywhere else? Mucous membrane pemphigoid may be accompanied by ocular and vaginal lesions, the former leading to scarring and, if untreated, sometimes blindness. Eye and vaginal symptoms should be sought by questioning and, if appropriate, by examination.

If you were able to examine the patient you would find that she has no skin lesions and gives no history of a rash.

INVESTIGATIONS

■ ***What special tests would you perform and what is their significance? Are any special procedures required?***

See Table 35.1.

■ ***The biopsy specimen is shown in Figure 35.2. What do you see?***

The epithelium has separated cleanly from the underlying connective tissue in the plane of the basement membrane. A few erythrocytes lie in the cleft between the two. No cause for the separation is evident. The epithelium appears almost normal and there are only a few inflammatory cells in the lamina propria.

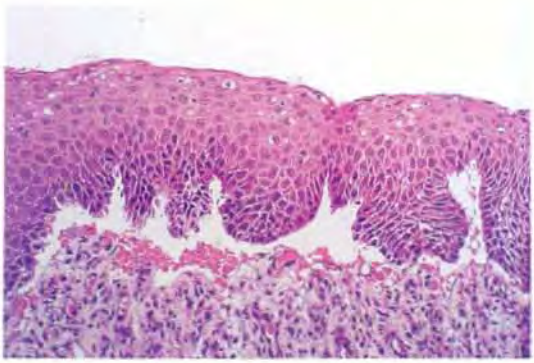


Fig. 35.2 Histological appearances of the biopsy specimen stained with haematoxylin and eosin.

■ ***The immunofluorescence stain for complement C3 is shown in Figure 35.3. What do you see?***

The immunofluorescence staining has been carried out on a separate part of the specimen in which there is no epithelial separation. A bright line of fluorescence runs along the basement membrane, outlining the rete processes of the epithelium. Immunofluorescence for IgG gave an identical result.

■ ***How do you interpret these histological features?***

Separation of the full thickness of the epithelium at the level of the basement membrane, without epithelial damage, almost certainly signifies pemphigoid. Pemphigus is excluded by the lack of acantholysis and the level of separation. Lichen planus is excluded by the lack of basal cell degeneration and lymphocytic infiltration of the epithelium. The direct immunofluorescence indicates

Table 35.1 Tests and special procedures

Test	Significance/special procedures
Nikolsky's sign	Some clinicians attempt to elicit Nikolsky's sign, in which gentle lateral pressure on apparently unaffected mucosa or skin (not rubbing the surface) raises a bulla. This is positive in vesiculobullous diseases but is somewhat unpredictable. In pemphigus the epithelium tends to disintegrate rather than form a bulla. If no lesions are present on examination it may be a useful way of demonstrating reduced epithelial adhesion, but it is often not necessary for diagnosis. Unsurprisingly, it is also unpopular with patients who are left with a large new ulcer which may take weeks to heal.
Biopsy	An incisional biopsy is indicated and it will almost certainly need to be investigated by immunofluorescence to differentiate the autoimmune blistering conditions. An incisional specimen removed from a vesicle or bulla margin or from apparently normal perilesional mucosa is best. Skin may also be sampled if involved. The biopsy must include epithelium and may be difficult to perform because the mucosa may disintegrate on slight trauma. The specimen should be either taken fresh to the laboratory immediately, frozen in liquid nitrogen at the chairside or placed in a special transport medium. Tissue fixed in formalin is useless for immunofluorescence.
Serum autoantibody determination	A sample of clotted blood should also be sent for indirect immunofluorescence to detect circulating pemphigus or pemphigoid autoantibody.

Fig. 35.3 The biopsy specimen after immunofluorescence staining for complement component C3.

binding of IgG at the basement membrane and activation of complement. This indicates pemphigoid in which IgG autoantibody binds and fixes complement. Taken together, these features indicate pemphigoid.

DIAGNOSIS

The patient has pemphigoid. There are different variants of pemphigoid but, as there is no skin involvement, mucous membrane pemphigoid is almost certainly the diagnosis. Bullous pemphigoid, linear IgA disease and epidermolysis bullosa acquisita are pemphigoid variants which very rarely affect the mouth.

TREATMENT

■ ***How should this patient be managed?***

The patient should preferably be treated in a hospital environment, at least initially. This will probably be necessary in order to perform the immunofluorescence tests. Treatment of pemphigoid requires more potent steroids than are usually considered appropriate in a general dental practice setting. However, there is no reason why routine dental treatment should be transferred to hospital.

The patient should be referred to an ophthalmologist to identify and manage any ocular lesions that may be present.

Lesions limited to the mouth and of relatively minor severity can be treated with topical steroids. Potent steroids are required, such as betamethasone 0.5 mg qds used as a mouthwash. Patients must be warned not to swallow such potent steroids and must be regularly checked for adverse effects. However, if oral lesions are widespread from the outset, if there are eye signs or if topical steroids fail, dapsone is the drug of choice. If this proves ineffective, systemic steroids, sometimes with azathioprine, are required. After the disease is brought under control, topical steroids may be sufficient for maintenance.

All patients using systemic or topical steroids are predisposed to oral candidal infection and this should be monitored at subsequent visits.

36 Bridge design

Summary

A 28-year-old woman presents to you in your general dental practice with an edentulous premolar space on the upper left. She would like this space filled. What are the options?

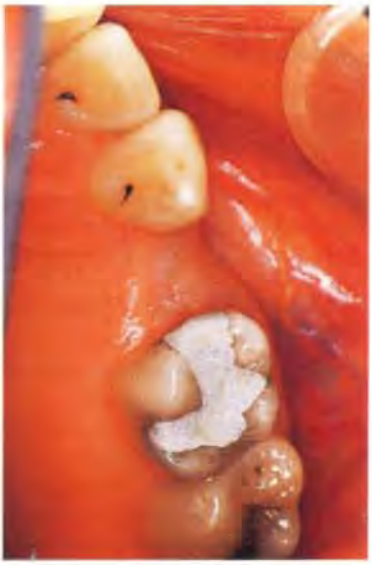


Fig. 36.1 The premolar space on presentation.

HISTORY

Complaint

Her complaint is the appearance of the gap. She would like it filled in time for her wedding in a few months time and requests a bridge.

History of complaint

The patient had all four first premolars extracted for orthodontic treatment in her early teens. After treatment with fixed appliances the premolar space was closed and the result had been stable. However, she then lost the upper left second premolar because of a combination of caries and root fracture following root canal treatment. This was about 2 years ago and she has had no replacement since.

Dental history

The patient first came to your practice 18 months ago, shortly after having had the second premolar extracted. You have made her dentally fit and instituted preventive treatment which appears to have been successful. No caries is present in any teeth and the gingival condition is good. The patient consumes a low sugar diet and has good oral hygiene.

Medical history

The patient is fit and well with no medical problems.

EXAMINATION

Extraoral examination

No abnormalities are present on extraoral examination. The premolar space is visible during speech.

Intraoral examination

The patient has an almost complete and well restored dentition with small or medium amalgam restorations. Although two premolars are missing, the gap is only a single premolar-sized unit of space because of the orthodontic treatment. This is her only missing tooth.

There is a mesio-occlusal restoration in the upper left first molar tooth. The first molar and the incisor teeth are in class I occlusion, with canine guidance in left lateral excursion. The orthodontic treatment has left the canine and molar vertically aligned and there has been no significant mesial drift of the first molar in the 2 years since extraction. The features are shown in Figure 36.1.

■ *What alternatives are there for replacing the missing tooth and what are their relative advantages and disadvantages?*

The options are shown in Table 36.1.

■ *What specific features of importance with regard to restoration would you examine? Explain why.*

The degree of bone loss of the edentulous alveolar ridge is important. If this is extensively resorbed, an elongated pontic would be necessary to hide the bone loss. This might well be unacceptable if the pontic is easily seen during talking or smiling. This problem can be overcome with ridge augmentation prior to placement of the bridge, but this would prolong the treatment and make it considerably more complex. A diagnostic wax-up may help

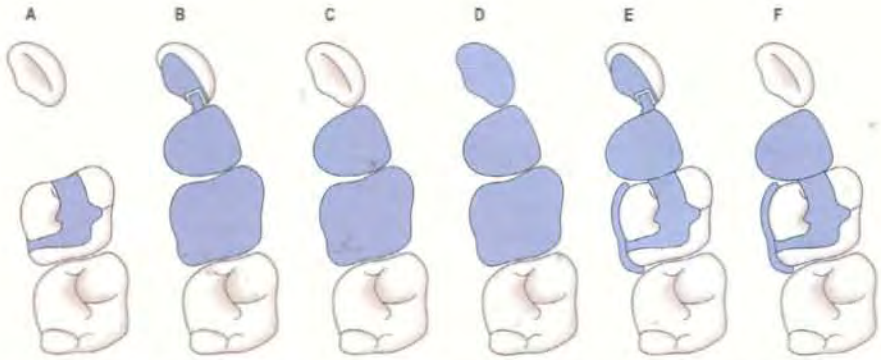


Fig. 36.2 Possible bridge designs. The gap can be left and provided overeruption of the opposing teeth does not occur, the existing situation would be stable (A). A possible fixed-movable design (B) would be a minor retainer covering part of the canine and secured to the tooth with an adhesive cement and a conventionally prepared full coverage retainer on the molar. The molar can be conventionally prepared for a full coverage restoration, either for a simple cantilever (C), or in addition the canine can be prepared producing a fixed-fixed design (D). A minimal preparation bridge is also possible. The existing restoration can be partly removed to secure the retainer, and the canine can be either included in a fixed-movable design (E) or avoided to produce a simple cantilever design (F).

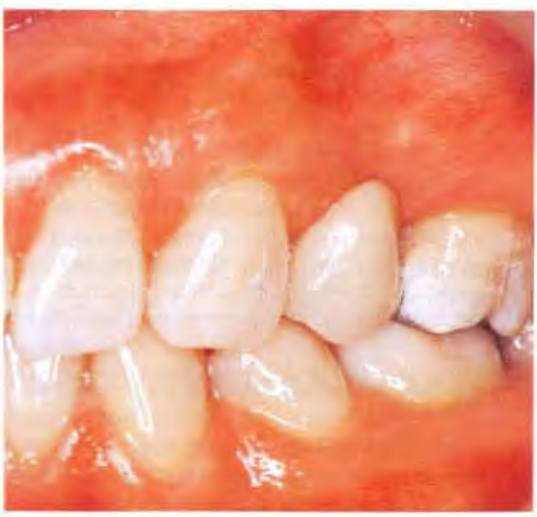


Fig. 36.3 The completed bridge.

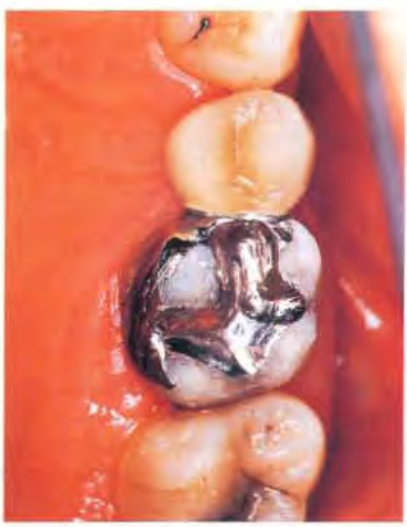


Fig. 36.4 The completed bridge showing retainer design.

should be wrapped around the abutment tooth as far as possible without encroaching on the contact point. The prepared area should be either within enamel or just into dentine. Modern luting cements bond to dentine and placing part of the preparation in dentine reduces the reliance on the enamel bond. Including the existing cavity also helps, by providing a dentine surface for bonding. The pontic is usually made of porcelain bonded to the metal.

■ ***What would you do if the bridge fails through debonding?***

If the bridge decements shortly after placement, it is acceptable to recement the bridge and ensure that there is no occlusal interference. If the problem persists, a conventional bridge would then be indicated, probably using the same abutment teeth for conventional crowns.

■ ***What investigations are required and why?***

The premolars and molars should be checked for vitality. The first premolar is vital but both second premolar and first molar do not respond to testing with an electric pulp tester.

A radiograph is required in order to decide whether the molar is restorable, i.e. to gauge the extent of caries and determine the success of the root filling. If extraction turns out to be required, a radiograph will be necessary to assess the difficulty of the extraction. This is particularly important in a patient who may suffer prolonged bleeding. A periapical view is the ideal view.

■ ***The periapical view is shown in Figure 37.1. What does it show?***

The first permanent molar is extensively carious. A root filling is present but only one gutta percha or silver point is visible, in the palatal canal. It extends beyond the apex by approximately 2 mm. The buccal roots are not clearly visible but appear to contain no root filling. The overextended root filling lies close to the antrum and the antrum extends down between the roots of the first molar and second premolar. There is no apical radiolucency. The second premolar is also root-filled. The root filling appears to stop just short of the anatomical apex at an appropriate point but a small apical radiolucency is present, surrounded by the lamina dura of the tooth socket. The caries below the crown on the first premolar is visible and the second molar contains a large pinned amalgam.

■ ***What is your diagnosis? Explain your diagnosis.***

The patient's pain is caused by periapical periodontitis of the first permanent molar.

The patient points clearly to this tooth as the cause of his pain. This and the tenderness on percussion indicate inflammation of the periodontal ligament and the overfilled root canal provides a likely cause. No radiolucency is shown on the radiograph. However, none is required for diagnosis in the presence of typical signs and symptoms. There may be either only a small apical lesion or one on th apices of the buccal roots or in the trifurcation, both of which are superimposed on the film.

The second premolar has an unsuccessful root filling and granuloma or a small radicular cyst at the apex. However, it is not tender to percussion and is not felt by the patient to be the cause of his pain. The first premolar i vital and the caries would produce pain of pulpitis type without tenderness on biting or percussion.

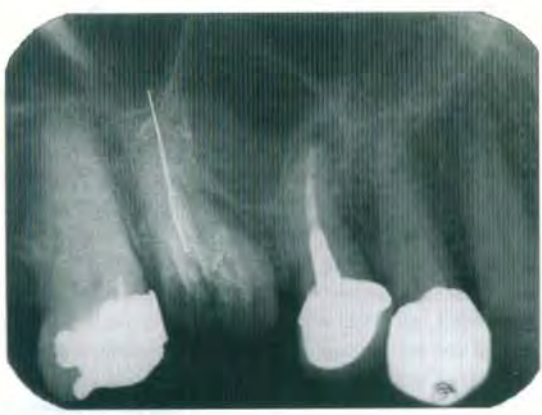


Fig. 37.1 Periapical radiograph of the upper first molar.

■ ***What treatment would you recommend?***

The primary consideration should be that the patient is at risk of infective endocarditis and potential sources of infection should be eradicated. The first molar cannot be restored without another root filling and extensive preparation. The patient prefers extraction and this is the appropriate course of action. The first premolar is more problematic. It is apparently symptom-free and the apical granuloma has probably been present for some time. This lesion is a further potential source of infection and it must be eliminated, either by extraction or another root filling. The success of the new root filling must be monitored to ensure that it is successful, and if not either apicectomy or extraction will need to be considered. Elements of this treatment would require antibiotic cover and/or adjustment of anticoagulation, and so these items must be incorporated into a complete treatment plan that takes the rest of the dentition into account.

■ ***Is the extraction of the first molar likely to be straightforward?***

No. The tooth is broken down and root-filled and there is little or no bone loss from periodontitis. It will be difficult to grasp with forceps and may be brittle. The roots extend close to the antrum and there is a risk of creating a surgical oroantral fistula.

A simple forceps extraction might turn out to be possible, but extraction may well require a mucoperiosteal flap and bone removal. A simple forceps extraction would be preferable in an anticoagulated patient because bleeding is more easily controlled when it is limited to a socket and surrounding gingiva. A surgical extraction would be less traumatic overall and separating the roots and elevating them singly would reduce the chances of creating an oroantral fistula.

■ ***Would you expect this patient to suffer prolonged bleeding after a dental extraction?***

Potentially yes and, if untreated, such bleeding could require hospital admission. Untreated prolonged haemorrhage could be fatal.

38 A white patch on the tongue

Summary

A 52-year old woman has a white patch on her tongue. Make a diagnosis and decide on appropriate treatment.

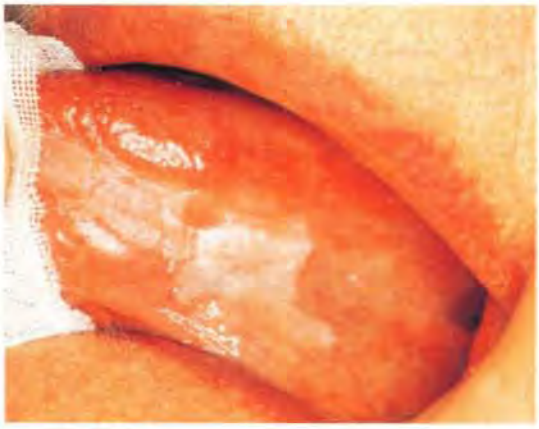


Fig. 38.1 The patient's tongue.

HISTORY

Complaint

The patient has no complaint.

History of complaint

You have just noticed the lesion in a patient attending for the first time for several years. There is no written record of the white patch in her notes. The patient had noticed the lesion but has ignored it. She thinks it has probably been there for several years.

Medical history

The patient is otherwise fit and well. She smokes 4 cigarettes a day and drinks 4–8 units of alcohol each week.

EXAMINATION

Extraoral examination

No lymph nodes are palpable in the neck and there are no abnormal findings on extraoral examination.

Intraoral examination

Apart from this lesion, the remainder of the oral mucosa is normal.

■ ***The appearance of the lesion is shown in Figure 38.1. What do you see?***

There is a flat and homogeneous white patch on the left lateral border and ventral tongue mucosa. It is well defined and varies slightly in whiteness.

If you were able to feel the lesion you would find that it is soft and feels no different from the surrounding mucosa.

DIFFERENTIAL DIAGNOSIS

■ ***What are the common or important white patches in the mouth? How are they caused?***

Almost all oral white patches are caused by increased keratinization of the epithelium. Keratin absorbs water and appears white, brighter white where it is thicker. The exception is a chemical burn where the white surface layer is caused by necrosis or ulceration.

Type of lesion	White lesion(s)
Normal mucosal variants	Leukoedema Fordyce spots/granules
Inherited epithelial disorders	White sponge naevus Pachyonychia congenita
Traumatic lesions	Frictional keratosis Chemical burn Cheek and tongue biting
Infections	Thrush (acute hyperplastic candidosis) Chronic hyperplastic candidosis (candidal 'leukoplakia') Chronic mucocutaneous candidosis Hairy leukoplakia Syphilitic leukoplakia
Lichen planus and similar conditions	Lichen planus Lichenoid reaction (topical and systemic) Lupus erythematosus
Unknown or smoking-related	Idiopathic keratosis (*leukoplakia*), including: Homogeneous leukoplakia Verrucous/nodular leukoplakia Sublingual keratosis Smoker's keratosis Speckled leukoplakia Stomatitis nicotina (smoker's palate)
Neoplastic	Squamous cell carcinoma

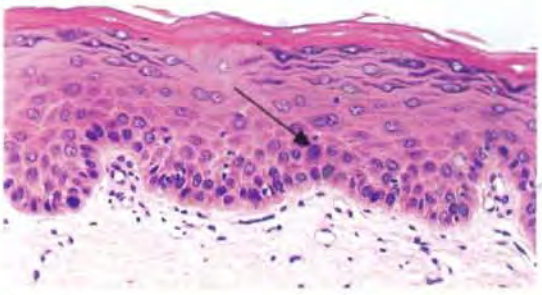


Fig. 38.2 The histological appearances of the biopsy sample.

■ ***The histological features of the lesion are shown in Figure 38.2. What do you see and how do you interpret the changes?***

The surface is covered by a regular orthokeratinized stratified squamous epithelium. The basement membrane is almost flat with a few short dermal papillae extending up into the epithelium. The epithelium is abnormal for either lateral border or ventral tongue, neither of which shows even orthokeratosis.

There are minimal signs of dysplasia. The epithelium shows good stratification with well-organized basal, prickle cell, granular cell and keratin layers, each composed of cells at the same stage of maturation. The basal cell layer is slightly disorganized. Instead of a well-defined single layer of small dark cells there is a slightly irregular layer of cells whose nuclei vary a little in size and staining intensity. Near the centre there is one darkly staining cell in a suprabasal position (arrowed). No candida was found in a section stained with PAS stain.

The changes of abnormal keratinization and slight basal cell irregularity are not very marked. The epithelial cells form a well-organized epithelium; there are only occasional abnormal single cells, minimal nuclear abnormalities and no evidence of increased growth. These signs might be graded as either nondysplastic or mildly dysplastic depending on the pathologist. The final diagnosis given in this case was keratosis with mild dysplasia.

DIAGNOSIS

■ ***What is the final diagnosis? Is this a risk lesion for malignant transformation?***

The diagnosis is idiopathic white patch (or keratosis) with mild dysplasia. This is a risk lesion for malignant transformation.

TREATMENT

■ ***What treatment is indicated?***

The following principles of treatment apply to all idiopathic keratoses. The patient should stop smoking and moderate their alcohol intake. If candidal infection had been detected

Feature	Risk of malignant transformation
Dysplasia	The degree of dysplasia is the best predictor and it may change, either progressing or regressing, with time.
Site	White lesions in the floor of mouth, posterior and lateral tongue and retromolar area carry the highest risk. Those on the hard palate and dorsum of tongue carry no significant risk. This distribution matches the distribution of oral squamous cell carcinoma.
Colour	Development of red areas carries a high risk and is usually associated with severe dysplasia histologically.
Surface	Development of verrucous or nodular areas.
Tobacco use	Smoking indicates increased risk. However, smoking also causes many white patches with no dysplasia and so, statistically, patches in nonsmokers carry an even higher risk.
Age	The risk of malignant transformation rises with age.
Sex	Female patients are at higher risk (despite the fact that oral carcinoma is commoner in men).
Size	Larger lesions have a higher risk of malignant transformation.
Duration	Patches present for a longer time have a higher risk of malignant transformation.
Family history of carcinoma in upper aerodigestive tract	Indicates increased risk.
Candidal infection in presence of dysplasia	Indicates a small increase in risk.
Change in clinical appearance	Changes apart from colour, such as size, nodularity or development of a verrucous surface, indicate a higher risk.
Underlying conditions	Conditions which predispose to oral carcinoma, such as submucous fibrosis, raise the relative risk of malignant transformation.

39 Another white patch on the tongue

Summary

A 39-year-old woman has a white patch on the lateral margin of her tongue. What is the cause and what are the treatment options?

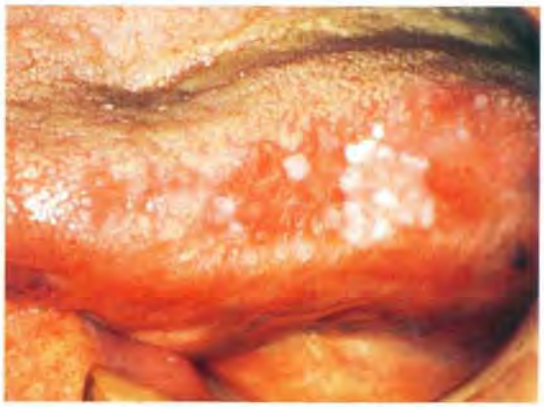


Fig. 39.1 The patient's tongue.

HISTORY

Complaint

The patient has no symptoms.

History of complaint

The patient is an infrequent dental attender and has not been to the dentist for at least 5 years. Following an oral cancer awareness week she inspected her mouth and became nervous about her tongue. She would like it checked.

Medical history

She has had cervical dysplasia treated in the previous year by cone biopsy and this has left her very worried about cancer. She is otherwise fit and well.

She has smoked 40 cigarettes daily since the age of 18 years and drinks 14 units of alcohol per week as spirits.

EXAMINATION

Extraoral examination

She seems a healthy woman with no obvious skin, nail or eye lesions present on visible skin. No lymph nodes are palpable in the neck.

Intraoral examination

The oral mucosa appears normal, except for the tongue which is shown in Figure 39.1.

■ ***Describe the appearance of the tongue lesion.***

Site	Right lateral border of tongue
Size	1 × 3 cm approximately
Shape	Ill defined ellipse
Colour	Mixture of white and red components
Surface	Appears nodular or irregular

Palpation reveals the lesion to be firmer than the adjacent mucosa. The white component of the area cannot be rubbed away. The tongue is freely mobile.

DIFFERENTIAL DIAGNOSIS

■ ***What are the causes of mixed red and white patches in the mouth?***

The causes of white patches are discussed more fully in Case 38. Several may also be associated with red areas.

Cause	Red and white lesion(s)
Trauma	Chemical burn Cheek biting
Infection	Thrush (acute hyperplastic candidosis) Chronic hyperplastic candidosis (candidal 'leukoplakia')
Lichen planus and similar conditions	Lichen planus Lichenoid reaction (topical and systemic) Lupus erythematosus
Idiopathic or smoking	Idiopathic keratosis *(leukoplakia)* including: Sublingual keratosis Smoker's keratosis Speckled leukoplakia Stomatitis nicotina (smoker's palate)
Neoplasia	Squamous cell carcinoma

■ ***Which of the above lesions would you include in the differential diagnosis for this particular lesion?***

1. Squamous cell carcinoma
2. Idiopathic white patch with or without dysplasia including speckled leukoplakia

■ *Would you perform this biopsy in general practice?*

No, definitely not. Although removing a sample of the tissue is well within the capability of the general dental practitioner, it would be unwise to do so. The patient will return for the result and dental practitioners are not usually the appropriate person to break the news of malignant disease. There is also a theoretical risk that biopsy of the wrong site or removal of the whole of a small lesion might compromise subsequent treatment but this is a largely theoretical problem. In a practice environment the patient should be referred urgently, preferably the same day, to the centre where definitive treatment is likely to be provided. This will allow the most appropriate biopsy to be performed. No other special investigations are indicated at this stage.

■ *Which part of the lesion should be removed for biopsy?*

The specimen should include those areas most likely to be malignant, the red and speckled parts. Some normal tissue should also be included and the sample should be about 1 cm long, 4–5 mm wide, and an even depth including underlying muscle. Larger malignancies are often friable and if the specimen is too small it may disintegrate on removal. No attempt should be made to excise the whole lesion until a diagnosis is obtained.

■ *The biopsy specimen is shown in Figure 39.2. What are the microscopic features and how do you interpret them?*

The lower power view (Fig. 39.2a) shows tongue mucosa with underlying muscle. The overlying epithelium is very irregular and instead of being an even and well organized layer it forms an irregular series of rete processes which penetrate deeply into the underlying tissue. The deepest epithelium is breaking off into apparently separate islands and strands and these extend deeply between muscle bundles. The higher power (Fig. 39.2b) view is taken from the deep surface and shows the deepest epithelium invading muscle. The epithelium is disorganized, with keratin forming in the centre of islands and an irregular darkly stained basal cell layer around the edge. This epithelium has lost its ordered maturation and stratification and is invading the underlying muscle. These features indicate malignancy and the malignant epithelium shows squamous differentiation.

DIAGNOSIS

■ *What is the diagnosis?*

The patient has a squamous cell carcinoma. It is only superficially invasive and probably an early lesion.

TREATMENT

■ *What types of treatment are possible and what is the prognosis?*

The lesion appears to have been diagnosed at a much earlier stage than most oral carcinomas. Treatment may be by radiotherapy (implant or external beam), by surgery or both in combination. The final decision will depend on the results of investigations to stage the carcinoma (determine

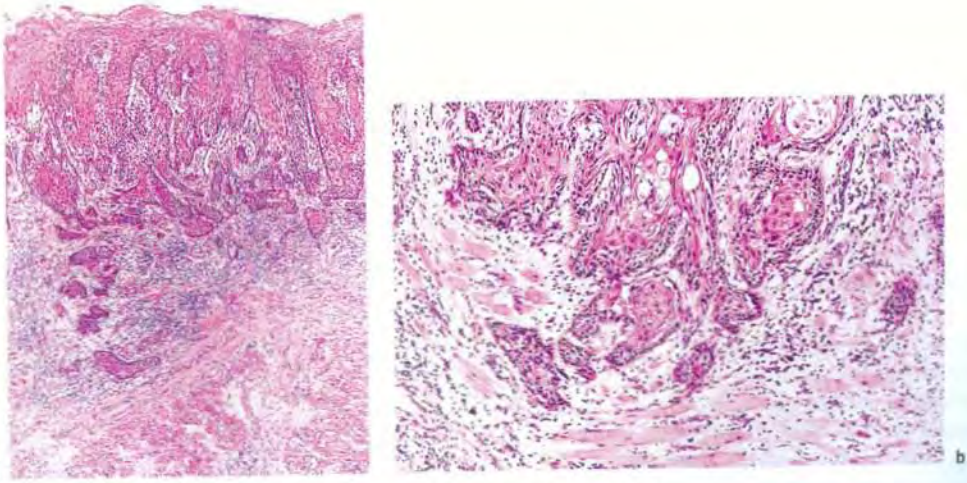


Fig. 39.2 The histological appearances of the lesion, **a** Lower power view; **b** higher power view.

brain. For this reason they are sometimes referred to as sensitivity tests.

It is assumed that pulp without sensory innervation is devitalized but this is not necessarily so. Inflammation can alter sensation and sensation may be incorrectly localized (see Case 1). Conversely, a positive response does not guarantee the health of the pulp.

Tests sometimes indicate a hypersensitive pulp and this is a more useful piece of information because detection of hypersensitivity is not prone to false results.

■ ***What are the causes of misleading electric pulp test results?***

Both false positive and false negative sensitivity responses can occur. Causes are listed in the table below, together with precautions that minimize the risk of a spurious result.

On performing these tests you discover that the lower second molar gives a hypersensitive response to hot gutta percha. The pain lasted until a local anaesthetic was given.

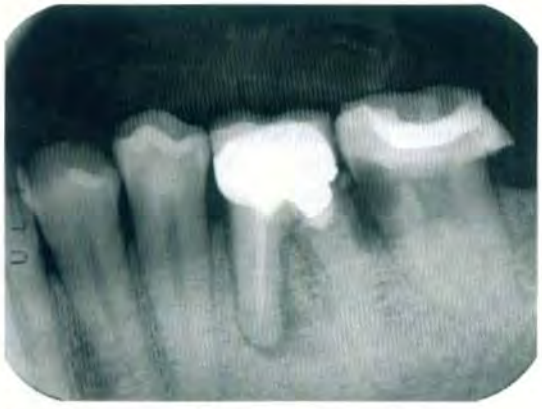


Fig. 40.1 Periapical radiograph on presentation.

■ ***The periapical radiograph is shown in Figure 40.1 What do you see?***

The lower premolar teeth are unrestored and there is no caries. The molar teeth show several changes:

DIAGNOSIS

■ ***What is the most likely diagnosis?***

Irreversible pulpitis in the lower left second molar tooth caused by the large mesial carious lesion. When the pain started 8 months ago the carious lesion would have been smaller and the pulpitis reversible.

TREATMENT

■ ***What emergency treatment would you provide at the first appointment?***

The first molar is unrestorable because caries involves the furcation. This tooth will require extraction but this is not an immediate problem. The periradicular lesion is chronic and painless and the second molar, as the cause of the pain, is the first priority.

Loss of the first molar makes it desirable to conserve the second if possible. Caries is extensive in the second molar and extension down the mesial root is a potential problem. Definitive restoration will require either a deep subgingival restoration or a crown lengthening procedure. The possibilities will become clearer after excavating the caries, but it is possible that extraction will be required or requested by the patient.

Assuming the tooth is to be conserved, the priority is relief of pain. Having assessed the pulpitis as irreversible, extirpation of the pulp is the only appropriate treatment and this will require cleaning and shaping of the root canals to ensure no remnants remain. Extirpation with barbed broach alone risks leaving fragments of inflamed vital pulp that could cause pain after treatment. A root canal dressing of calcium hydroxide will inhibit bacterial growth. This must be sealed from the mouth with a suitable temporary dressing material to prevent bacterial ingress pending definitive treatment.

Pulpotomy, with removal of the coronal pulp, or partial pulpectomy, with removal of pulp from the widest canal, is sometimes advocated in multirooted teeth but should be avoided. The apical extent of inflammation in the pulp is unknown and pain relief cannot be guaranteed.

First molar	Second molar
The tooth is crowned and the distal margin is defective and carious. Caries extends into the furcation.	The tooth has a disto-occlusal restoration with a large ledge distally.
The mesial root has a root canal filling present, short of the radiographic apex and a periradicular radiolucency.	There is also a large carious lesion mesially that extends close to the mesial pulp horns.
There is no evidence of a root canal filling in the distal root. The canal is not visible radiographically and may be sclerosed.	No periradicular radiolucency is visible.
There is a suggestion of periradicular radiolucency within the furcation.	

■ *What definitive treatment does the patient require for the second molar?*

The patient should return for obturation of the root canals and a definitive coronal restoration. A cuspal coverage indirect restoration should be considered to give protection against occlusal forces, either an onlay or core with a full coverage crown. Post crowns in molar teeth are to be avoided because of the incidence of perforations and root fractures. Retention should be sufficient from the undercut shape of the pulp chamber and the remaining coronal tooth structure (see Case 1).

After discussing the possibility of restoring or extracting the second molar, the patient opts for root canal treatment. You carry out the first stage of treatment successfully. However, the patient fails to return to complete the treatment for 6 months. When he returns you discover that the tooth has remained asymptomatic but the coronal temporary restoration has been lost and the lingual cusp has fractured off. The pulp chamber is open to the oral cavity but the tooth is still restorable.

■ *What effect will this have on the long-term prognosis for this tooth?*

Loss of the coronal seal will have allowed microbial invasion of the root canals and dentinal tubules. Bacteria and their metabolic and breakdown products are major irritants and will penetrate apically and along lateral canals to induce or maintain periradicular inflammation. In addition, the flora in the canal will change and a more mixed oral flora with anaerobes will become established in the canal. This may be more difficult to eradicate and be more likely to penetrate through the apex and induce an acute abscess. Fracture of the lingual cusp further weakens the tooth and complicates building up a core to support a definitive restoration.

■ *What should be the next stage of treatment?*

Root canal treatment must be recommenced as soon as possible. The tooth should be isolated with rubber dam and the root canals cleaned and shaped, using appropriate files and copious irrigation with sodium hypochlorite. Sodium hypochlorite is antibacterial but some bacteria may survive in lateral canals and dentinal tubules that are blocked by the smear layer produced by instrumentation. The smear layer must be removed by occasionally irrigating with either citric acid or EDTA solution. Hypochlorite will then be able to penetrate the dentine and lateral canals. It is important that coronal root canal preparation is carried out first to reduce the bacterial load and improve access to the apical portion of the root canal. The working length can then be determined and confirmed with a working length radiograph (Fig. 40.2).

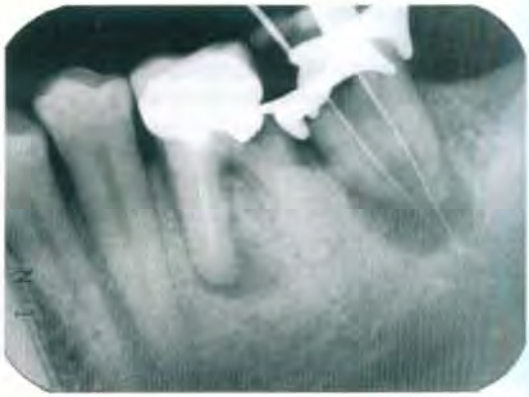


Fig. 40.2 The working length radiograph.

■ *The working length radiograph is shown in Figure 40.2 What do you see and what does it mean?*

There is a large periradicular radiolucency that was not present in Figure 40.1; the lesion has developed as a result of the canals being left open. This is almost certainly a granuloma rather than a radicular cyst, as cysts take some time to develop.

The working length files have been placed beyond the root apices. This should be avoided as debris, irrigants, medicaments or bacteria can be introduced into the periradicular tissues, delaying the healing process.

A further film is required to confirm the working length using larger ISO-size files. Determining the working length has clearly been a problem and an apex locator may help locate the apical constriction, which is 1–2 mm short of the radiographic apex and often not visible on a periapical film.

A further dressing of non-setting calcium hydroxide paste for 1 week is required to kill any bacteria remaining within the root canal system. If the tooth remains asymptomatic, obturation can be carried out at the following visit. The definitive restoration should be placed without delay to prevent coronal leakage and to avoid further fracture of tooth structure. The final appearance after obturation is shown in Figure 40.3.

■ *How long should this tooth be reviewed after completion of root canal treatment? What are the criteria for success?*

Root canal treatment should be reviewed for at least 4 years because complete healing may require considerable time. Outcome must be evaluated clinically and radiographically. The radiographic assessment must be made by comparing the appearances with previous films, taken under as near standardized conditions as possible.

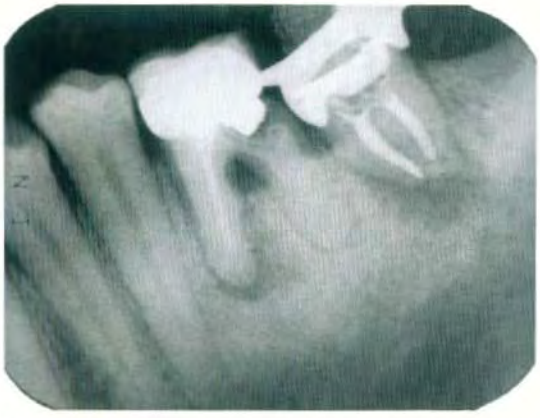


Figure. 40.3. The completed root canal filling.

Criteria for success and failure are shown below:

Success	No symptoms No tenderness on percussion or increased mobility No sinus Width and contour of periodontal ligament normal Slight radiolucency around excess filling material allowable
Uncertain	No symptoms No tenderness on percussion or increased mobility No sinus Residual radiolucency at 4 years that is smaller than seen on completion of root canal filling. Some authorities suggest that under these circumstances a further period of 3 years' healing time should be given
Failure	Symptoms Tenderness on percussion or increased mobility Sinus Unchanged or enlarged periradicular radiolucency Development of new radiolucency at another site on the tooth

41 A swollen face

Summary

A 30-year-old man is referred to your dental hospital by his general practitioner with a painful swelling of the right side of the face. What is the cause and what treatment would you provide?

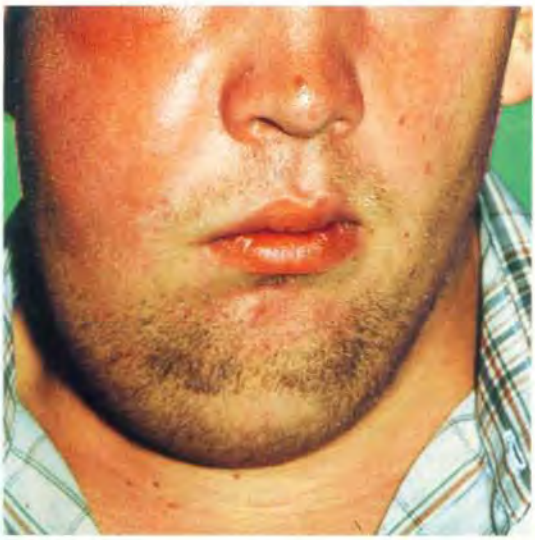


Fig. 41.1 The patient on presentation.

HISTORY

History of complaint

The patient has had toothache intermittently for many months. A few weeks ago the pain became excruciating and did not respond to analgesics. Then, suddenly, it reduced in severity and the patient thought it had resolved.

However, about 10 days ago a different pain developed. A tooth on the upper right has become very tender and he has not been able to bite on it. The swelling suddenly enlarged yesterday.

Over the last few months the patient has been prescribed several courses of antibiotics and he finished a course of oral erythromycin 2 days ago.

Medical history

The patient is otherwise fit and well.

EXAMINATION

Extraoral examination

The patient is shown in Figure 41.1. The swelling is hot, tender and firm centrally but peripherally it is almost painless and softer. It extends from the nose to the anterior border of the masseter and the lower eyelid is very oedematous and contains blood pigment as if bruised.

The swelling is not pointing extraorally. There are palpable tender lymph nodes in the upper deep cervical chain.

Intraoral examination

The patient has slight limitation of opening which does not significantly hamper examination. The sulcus adjacent to the upper first molar and both premolars is tender and slightly reduced in depth by a firm swelling. The upper first and second premolars and first molar have large amalgam restorations. However the patient indicates clearly that the second premolar is the cause of the pain and this tooth is slightly mobile and raised in its socket. It is very tender on percussion and nonvital on testing with ethyl chloride. The first molar and first premolar appear vital.

INVESTIGATIONS

■ ***Which additional investigation is critically important? Why?***

Taking the patient's temperature. This gives a good indication of the systemic effects of the infection and reflects the amount of pus in abscesses and/or the tendency of the infection to spread. The patient has a temperature of 37.2°C.

■ ***Would you take a radiograph?***

In this case a radiograph is not a useful investigation. Tests of vitality are much more likely to identify the causative tooth and, in any case, there appears to be no doubt about the diagnosis.

However there are good reasons why taking a radiograph may not help or even be counterproductive:

1. It takes up to 3 weeks for radiographic changes to develop at the root apex after pulp necrosis.
2. The radiographic features may mislead if you attempt to use them to diagnose loss of vitality. When root apices are radiographed with the maxillary antrum superimposed, the normal periodontal ligament appears wider. This may be confused with early apical changes of infection.

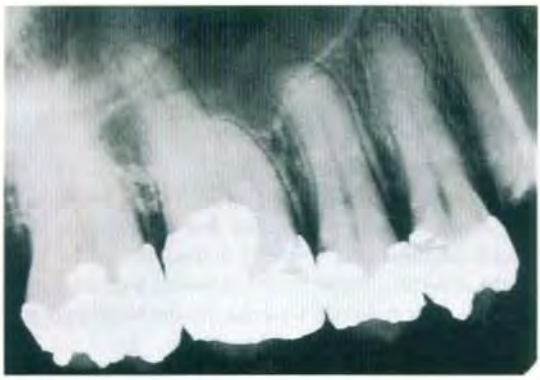


Fig. 41.2 Periapical radiograph showing the causative premolar.

Both these problems are appreciated in Figure 41.2, the periapical radiograph of this patient, which is completely normal.

DIAGNOSIS

■ *What do these findings tell you?*

The combination of inflammation, the nonvital tooth and adjacent probable abscess indicate an odontogenic soft tissue infection. The history of severe toothache which suddenly resolved suggests pulpitis subsequently relieved by necrosis of the pulp. The subsequent pain of a different character with a tender tooth suggests an apical abscess. The patient points clearly to the second premolar and this is almost certainly the cause of the pain because pain involving the periodontal ligament is well localized.

Trismus is an important sign, indicating that infection or inflammation has spread to involve muscles of mastication. However, trismus is not severe and probably results from inflammation and oedema of the buccinator and the anterior fibres of the masseter which lie at the posterior border of the swelling.

The infection has induced minimal systemic effects and the patient is not significantly pyrexic. Luckily, the infection appears to be localized. The firm centre to the swelling and the swelling in the sulcus will contain pus.

■ *What types of soft tissue infection arising from teeth cause facial swelling? How may they be distinguished and what is the relevance of doing so?*

Facial swelling may be the result of oedema, abscess formation, cellulitis or their combination.

It is important to determine which of these types of infection are present because the treatment and sequelae are different. Abscesses require drainage. Cellulitis requires aggressive treatment, usually including antibiotics, and oedema requires no direct treatment but resolves when the causative tooth is removed or the pulp treated.

Despite the fact that these terms are convenient, in practice most odontogenic soft tissue infections are caused by a mixed microbial flora and do not fall neatly into one category or another. It is not unusual to find an abscess with a surrounding zone of cellulitis and a degree of oedema is always present. Which type of infection develops is determined by the virulence of the pathogens (and synergy between species in the mixed flora), the resistance of the host and the anatomical constraints on the infection.

Cause of swelling	Features
Oedema	Soft, not very red or hot, not tender on palpation and not painful. Compressible with slow continuous pressure. Often accounts for much of the facial swelling in children with odontogenic infection.
Abscess	Localized collection of pus which feels hard if small, tense or covered by a thick layer of tissues. If large it may be softer and exhibit fluctuance. Pointing to the skin or mucosa indicates abscess formation.
Cellulitis	Brawny, poorly localized swelling with marked tenderness and dusky redness. May contain small collections of pus but no large localized abscesses. Spreads, sometimes rapidly, through tissues. Usually associated with systemic symptoms, pyrexia, malaise, leucocytosis and lymphadenitis.

■ *If infections are not easily characterized, what are the important features on which treatment must be based?*

The critical factors which must be determined are whether:

- an abscess cavity is present (palpation, eliciting fluctuation)
- there is evidence of systemic effects (malaise, pyrexia, a toxic-shocked appearance)
- the infection is spreading rapidly (judged by the history and observation during treatment)
- the patient is predisposed to infection (from the medical history).

■ *Which type of infection is this?*

This appears to be primarily an abscess with surrounding oedema.

■ *In what tissue space(s) is the infection tracking/localizing? What are the boundaries of this space?*

This abscess appears to be in the upper part of the buccal space. This is a potential space between the buccinator

muscle and the facial muscles and parotid fascia, filled normally with loose connective tissue. Posteriorly it communicates with the masseter muscle and around the front of the ramus to the pterygoid space. Oedema spreads beyond the buccal space to involve the lower eyelid and anterior cheek in the canine fossa. The abscess is not yet pointing to the skin.

■ *Why has the infection localized here? Will it remain localized here?*

Abscesses arising from the canine, premolar and molar teeth which perforate the buccal plate of alveolar bone will spill out into the soft tissues either above or below the attachment of the buccinator. The attachment of the buccinator usually runs below the apices of the upper teeth so that infection is likely to pass superficially to the buccinator and into the cheek. If it passes below the attachment, an alveolar abscess or sinus will develop. Paths of spread of infection from an upper premolar are shown in Figure 41.3.

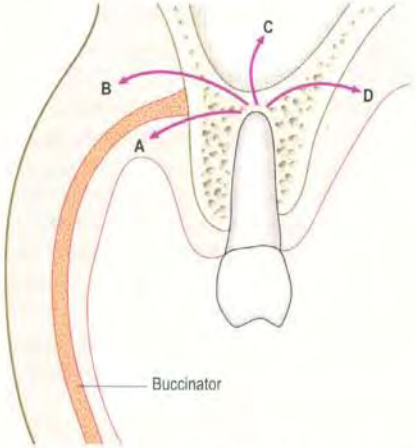


Fig. 41.3 Coronal section showing the paths of spread of infection from upper molars and premolars. Infection may pass buccally below the buccinator muscle into the sulcus or cheek intraorally (A), above the buccinator into the buccal space (B), into the sinus (C) or into the palate (D).

Despite being a thin muscle, the buccinator is a significant barrier to the spread of infection. It is unlikely that the infection would be able to perforate the muscle and develop a sinus into the mouth. Several sequelae are possible. Pus would be most likely to gravitate and spread through the whole buccal space down to the lower border of the mandible; it could point and then drain to the skin or spread laterally around the buccinator to involve other areas of the face, tissue around the masseter muscle or the pterygoid space. Its future course cannot be predicted.

■ *Is this a potentially life-threatening infection? If so, why?*

Not yet. Infection appears localized, the spread is not particularly rapid and there are no significant systemic symptoms. If a more spreading infection developed, the situation would change.

Involvement of the tissues around the eyelid is worrying. At present the swelling here is caused by oedema, but if infection were to spread to the upper lid or medial canthus of the eye the patient would be at risk of cavernous sinus thrombosis. This is a very rare but potentially fatal complication.

It would also be possible for the infection to spread posteriorly into the pterygomandibular space and infratemporal fossa. From here the infection could spread via veins to the cavernous sinus or middle cranial fossa.

■ *What is cavernous sinus thrombosis and what are its features?*

Thrombosis of the cavernous sinus follows spread of odontogenic infection along two main venous pathways. Bacteria and infected emboli travel posteriorly from the upper lip and face via the anterior facial vein. This connects via the ophthalmic veins to the cavernous sinus without valves which might otherwise prevent this retrograde flow. Alternatively, infection may spread from the pterygoid space via the pterygoid plexus of veins which connect directly to the cavernous sinus via the foramen ovale.

The local features are seen on one side at first but the signs become bilateral as the thrombus grows. The features are:

Local effects	Marked oedema of the eyelids
	Pulsating exophthalmos caused by venous obstruction
	A dilated facial vein
	Inhibition of movement of the eye
	Papilloedema and retinal haemorrhage
Systemic effects	Rapid pulse
	Marked pyrexia
	Severe malaise

In addition to treatment for the infection, thrombosis requires anticoagulation. The mortality rate is high.

TREATMENT

■ *What are the general principles of treatment for all odontogenic infections of the soft tissues?*

Treatment should be started rapidly. Infection may spread quickly and in some cases progress to a life-threatening situation with great rapidity. Identify patients with a risk of significant complications. Those at risk of

42 Missing upper lateral incisors

Summary

A 15-year-old boy presents to you in general dental practice requesting closure of the spaces between his upper front teeth. What is the cause and how can a better appearance be achieved?

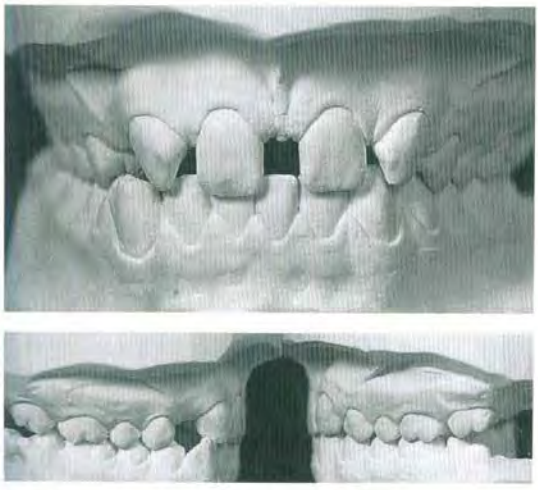


Fig. 42.1 Study models taken at presentation.

HISTORY

Complaint

The patient does not wish to have gaps between his upper front teeth.

History of complaint

His permanent teeth erupted at a normal age with large spaces between them. The primary predecessors had all been present and were exfoliated normally. None of the permanent teeth has been extracted.

Medical history

The patient is fit and well.

Family history

The patient's mother had a number of teeth missing. They had been replaced with a partial denture at an early age.

EXAMINATION

Extraoral examination

The patient has a skeletal class I appearance without facial asymmetry. There is a slight deviation of the mandible to the patient's left-hand side on opening, but no limitation of opening, temporomandibular joint clicks or crepitus or masticatory muscle tenderness.

Intraoral examination

The patient's soft tissues are healthy and his oral hygiene is good, with no calculus deposits, gingival inflammation or bleeding on probing. The teeth appear sound, with the exception of a buccal amalgam restoration in the lower left first molar.

Study models taken for treatment planning are shown in Figure 42.1.

■ *What features relevant to treatment do the study models show?*

Both upper lateral incisors are absent. From the front the upper central incisors are upright and separated by a large midline diastema. There is a mild class III incisor relationship, with a normal overjet but a reduced and complete overbite. The upper canines are mesially inclined and mesiolabially rotated, that on the left being more prominent. The lower right canine is labially placed, slightly distally inclined and in crossbite with the upper canine. There is mild lower labial crowding. The posterior teeth are well aligned and the first molars on the right-hand side are in a class I relationship and on the left-hand side in a half a unit class II relationship.

■ *What are the possible causes for the absent lateral incisors? What is the cause in this case?*

Missing	Developmentally absent, possibly associated with cleft lip or palate or other craniofacial syndrome
	Extracted
	Avulsed
Failure to erupt	Dilaceration and/or displacement as a result of trauma
	Scar tissue preventing eruption
	Supernumerary tooth preventing eruption
	Insufficient space as a result of crowding
	Pathological lesion (e.g. cyst or odontogenic tumour) preventing eruption

In this case the most likely cause for the missing lateral incisors is genetic absence. Genetic absence of some teeth is found in 3–7% of the population. The teeth most commonly missing are, in descending order of frequency, third molars, maxillary lateral incisors and second premolars. The absence of maxillary lateral incisors is a hereditary trait in about 1–2% of the population. The fact that the patient's mother wore a denture to replace missing teeth from an early age suggests a possible familial aetiology. Trauma or extraction and their related sequelae are readily excluded by questioning. The other causes are discussed in Case 5.

INVESTIGATIONS

■ *What special investigations are required? Explain why for each.*

Investigation	Reason
Tests of vitality of the upper anterior teeth	To exclude incidental loss of vitality, to ensure that endodontic treatment is not required and that unsuspected loss of vitality does not compromise the subsequent treatment plan.
Radiographs	To determine whether the lateral incisors are present and unerupted and to exclude underlying lesions such as supernumerary teeth or cysts. Examination for this case should include a panoramic tomograph to provide a survey, exclude significant periodontal bone loss and confirm the presence or absence of third molars. In addition periapical views or an upper standard occlusal view are required for detailed analysis of the incisor region which suffers from superimposition in the panoramic view. Further films may be required to define the caries status.
The study models should be mounted on an articulator	To assess the occlusion and produce a diagnosis wax-up if required.

In this case all the upper anterior teeth responded to tests of vitality by ethyl chloride and an electric pulp tester.

■ *The panoramic tomograph is displayed in Figure 42.2. What does it show?*

The dental panoramic tomograph shows that the upper lateral incisors are missing with no evidence of supernumerary teeth or other lesions in this region. All other teeth are present including the unerupted third molars. This confirms the diagnosis that the upper lateral incisors are developmentally absent.

TREATMENT

■ *What are the main treatment options? What are their advantages and disadvantages?*

Option	Advantages and disadvantages
Space closure with adhesive restorations	Composite restorations added to the approximal surfaces of the central incisors and canines could reduce the spaces. This is the most conservative option, technically straightforward and might be acceptable as a provisional solution. However, complete closure could not be achieved with such wide diastemas and each tooth would look unacceptably wide.

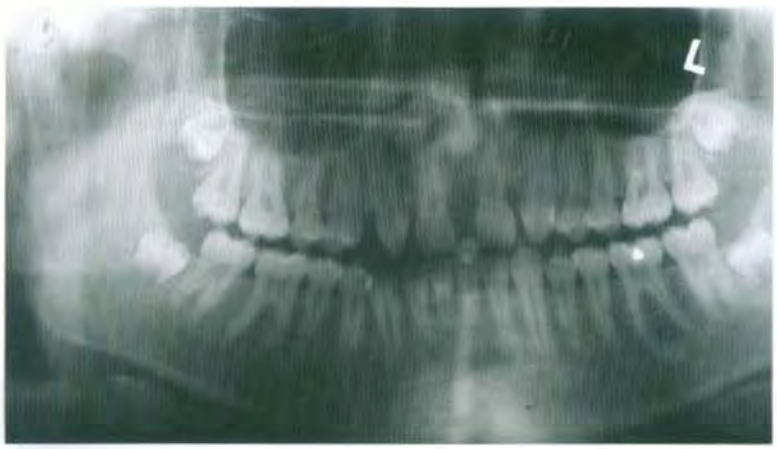


Fig. 42.2 Panoramic tomograph.

Option	Advantages and disadvantages
Orthodontic space closure	This would bring the canine into the position of the lateral incisor requiring the shape of the canine to be modified by selective grinding of the tip and placement of composite to disguise it as a lateral incisor. However, the darker colour of the canine would be difficult to conceal, as would the gingival contour because of the canine eminence. The palatal cusp of the first premolar tooth is frequently visible and compromises the appearance. When the difficulty of complete space closure is taken into account, it is clear that this option is rarely ideal. It frequently produces a poor result despite being a time-consuming and costly procedure.
Create space for lateral incisors	Space creation by orthodontic treatment followed by provision of lateral incisors with a prosthesis involves a protracted phase of orthodontics and is costly. However it would produce the best appearance.

■ *The patient's main concern is his appearance. How would you demonstrate the possible results to him?*

The patient is considering committing himself to a long and complex treatment so the result of each of the treatment plans should be assessed with study models and diagnostic wax-ups. The possibility of the orthodontic treatment can be visualized by cutting the teeth off duplicate study models and fixing them in an orthodontically achievable position, the so-called Kessling set-up. Patient and dentist can then see what might be achieved by each treatment option.

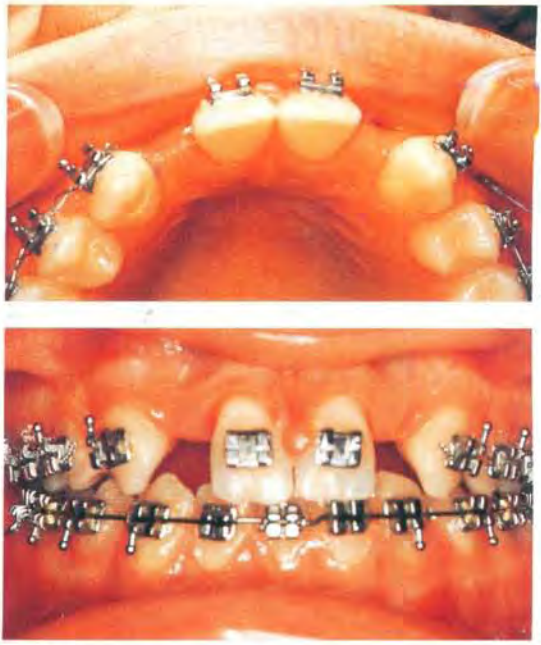
Following discussion, the patient opts for the third treatment plan.

■ *How would you carry out the orthodontic treatment?*

The tooth movement demands fixed appliance treatment. Tooth tilting using a removable appliance would result in a poor appearance in the midline and produce spaces which are difficult to fill with a prosthetic replacement. If a fixed appliance is used the incisors may be more accurately positioned and derotation of the canines is possible. The orthodontic result for this patient can be seen in Figure 42.3.

■ *How would you now replace the missing lateral incisors?*

Prosthetic treatment should be as conservative as possible because the upper anterior teeth are vital and sound, and the patient is young. The teeth can be replaced with fixed



a

b

Fig. 42.3 The final orthodontic result.

or removable prostheses but the treatment of choice would be a minimum preparation bridge or bridges. Possible designs are shown in Figure 42.4.

Normally a fixed-fixed design in a minimum preparation bridge should be avoided. This is because debonding of one retainer will create an area of stagnation below it and risk caries. A typical minimum preparation bridge to replace a lateral incisor would be a cantilever design retained on the canine or central incisor.

However canine abutments (option A) would have a major disadvantage in this case. The canines were originally mesiolabially rotated and the orthodontic result is potentially unstable. Relapse would result in the pontics swinging out labially. An alternative might appear to be a cantilever design retained on a central incisor (option B) which has the advantage of a greater enamel area for bonding. However, two separate cantilever bridges retained on the central incisors would also enable the orthodontic result to relapse and the midline diastema to reappear. Linking the central incisors together (option C) would prevent this but could not prevent the canines from relapsing to their original position.

A degree of orthodontic retention must be designed into the prosthesis and only a fixed-fixed bridge extending from canine to canine is suitable (option D). The potentially unstable orthodontic result may in itself favour debonding or one of more of the wings. Regular recall will be essential to detect this early. If debonding is a repeated problem, replacement with a conventional bridge may have to be

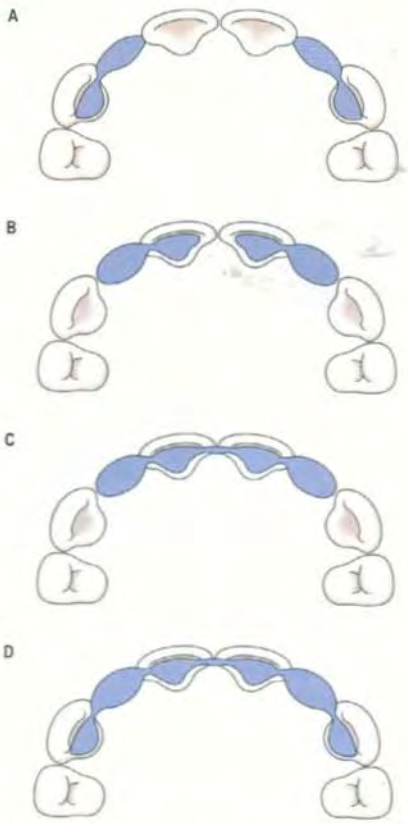


Fig. 42.4 Possible designs for minimum preparation bridge(s).

considered. The need for orthodontic retention is the main reason that an implant retained solution is not appropriate.

The final bridge design and appearance are shown in Figure 42.5. Note how the orthodontic treatment plan must take into account the occlusal clearance required to cover the palatal surfaces of the canines.

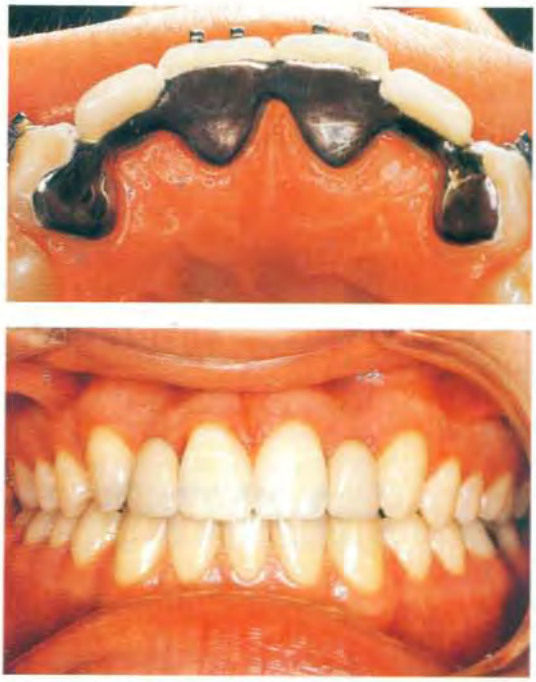


Fig. 42.5 The final result.

■ ***What else has been done to improve the appearance of the final result? Look closely and compare Figure 42.5b with Figure 42.1.***

The lower arch has been treated orthodontically. One lower incisor has been extracted and the space gained has been used to align the lower incisors and the lower right canine, which was in crossbite. This has made a significant contribution to the final appearance.

43 Anterior crossbite

Summary

An 8-year-old girl is referred to you for an orthodontic opinion. She has an anterior crossbite. What is the cause and how would you treat it?

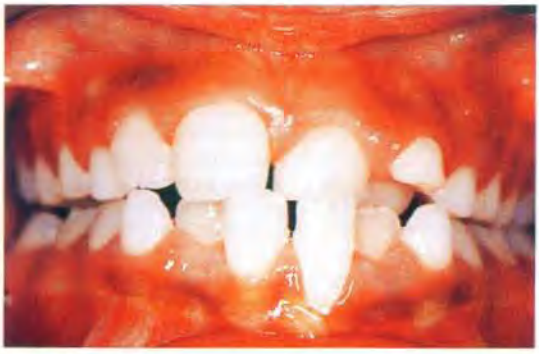


Fig. 43.1 The patient's appearance on presentation.

HISTORY

Complaint

The mother of the patient noticed the crossbite and is very anxious about her daughter's appearance. She requested a referral from the family's general dental practitioner.

History of complaint

The incisors erupted into their present positions and there is no history of trauma.

Medical history

The patient is fit and healthy.

EXAMINATION

Extraoral examination

There is no facial asymmetry and no clicks, locking or crepitus are present on examination of the temporomandibular joints.

Intraoral examination

■ *The appearance of the teeth on presentation is shown in Figure 43.1. What do you see?*

The patient is in the early mixed dentition stage and the teeth present are:

6EDC21	12BCDE6
6EDC21	12 CDE6

The upper and lower incisors are crowded and the upper left central and lateral incisors are in crossbite. The lower left central incisor is labially placed and there is gingival recession and loss of attached gingiva to the mucogingival junction on its labial aspect. The oral hygiene is reasonable though mild interdental gingivitis is present around the poorly aligned incisors. The dental health is good.

■ *What specific feature would you check in your examination? Explain why for each.*

See Table 43.1.

Table 43.1 Features to be examined

Feature	Reason
Can the patient achieve an edge-to-edge incisor relationship when closing on hinge axis?	On closing in a retruded position the patient makes initial contact on the lower left central incisor. If left untreated this could result in continued excessive occlusal loading on this tooth, causing further loss of support. The ability to achieve incisal contact is regarded as favourable because it indicates that minimal tooth movement should be required to correct the crossbite.
If so, is there an associated forward displacement of the mandible?	The initial contact on the central incisors displaces her mandible forwards into the intercuspal position shown in Figure 43.1. Early correction of the displacement activity may prevent possible temporomandibular joint dysfunction in later life. There is not yet significant wear faceting on the incisors. However, if they are left untreated, considerable attritional wear may develop.
How mobile is the lower left central incisor? Are probing depths increased?	Mobility is limited (grade 1) and probing depths are less than 2 mm. This would suggest that the prognosis for the tooth is good. If there were significant mobility or periodontal destruction, extraction of the incisor might have to be considered as part of an orthodontic treatment plan.
How might space be provided to relieve the incisor crowding?	At the present stage of dental development, sufficient space would be provided by the extraction of deciduous teeth.

DIAGNOSIS

■ *What is your diagnosis?*

The diagnosis of crossbite has already been made by the patient's mother. The incisor crowding, gingival recession and anterior displacement of the mandible are the other significant factors requiring recognition.

■ *How would you assess the long-term prognosis for the lower left central incisor?*

At this early stage the recession may be reversible. The crossbite and premature contact are producing movement of the central incisor, which is in danger of being pushed beyond the alveolus and losing its labial bone. The soft tissue defect is difficult to assess in the presence of slight inflammation. Some attached gingiva is almost certainly present labially and the recession seems to stop just short of the alveolar mucosal reflection. This patient has only a narrow band of attached gingiva as can be seen on the opposite side which is normal.

If the oral hygiene is improved and the crossbite corrected, the recession may also improve considerably. No additional attached gingiva will develop but further damage will be prevented. Further discussion of gingival recession around the lower anterior teeth will be found in Case 4.

■ *What is the cause of the crossbite?*

Ectopic eruption of the upper incisors in association with crowding.

INVESTIGATIONS

■ *What special investigations would you require? Explain why.*

Radiographs are the most useful investigation for any orthodontic assessment. The following radiographic views are indicated:

View	Reason
Dental panoramic tomograph	As a general survey. Primarily to assess the presence or absence of permanent successors and any supernumerary teeth.
Upper standard occlusal	When considering active movement of the incisors, an upper standard occlusal may be useful in order to give more detail of the area around the incisor roots. When incisors are misaligned this view may reveal supernumerary teeth or odontomes or dilaceration as rare causes.
Periapical view	If you are concerned about the prognosis of the lower left central incisor a periapical film may help. This should be taken using a paralleling technique to assess bone loss. However, as noted in Case 4, labial bone loss will not be visible in this view.

■ *The dental panoramic tomograph is shown in Figure 43.2. What does it show?*

The tomograph shows a normal dentition. The developmental age matches the patient's chronological age. All permanent successors are present and appear to be in

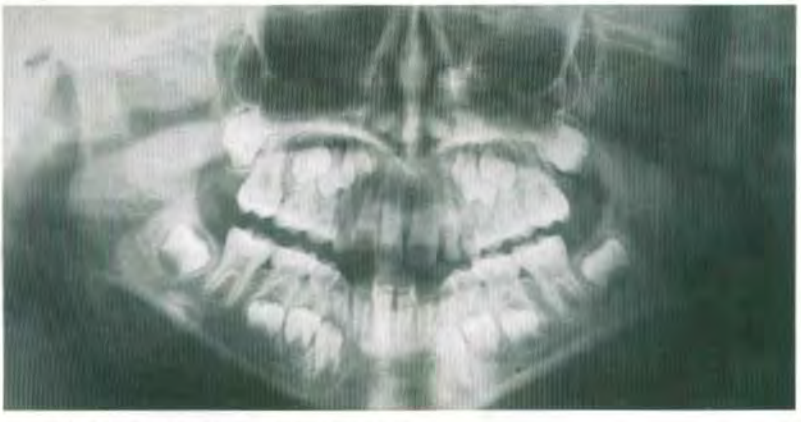


Fig. 43.2 Dental panoramic tomograph of the patient.

favourable positions. There is not yet any evidence of third molar development, as is normal at this age. Though the panoramic view is not suitable for detailed diagnosis, there seems to be some mesial bone loss on the lower left central incisor.

TREATMENT

■ ***What treatment plan would you propose?***

The incisor crossbite should be treated immediately to prevent further damage to the periodontium and attrition. Some authorities consider that early treatment will also reduce the possibility of temporomandibular joint pain dysfunction (myofascial pain) syndrome in later life. However, the evidence to support this contention is by no means conclusive.

To provide space for relief of crowding and to allow the active tooth movements required to correct the crossbite, all deciduous canines and the deciduous upper left lateral incisor should be extracted.

An upper removable appliance can then be fitted to correct the incisor crossbite.

■ ***Design a suitable removable appliance to correct the crossbite.***

Only simple tilting tooth movements are required and these can be achieved most easily with a removable appliance. There would be no advantages to the use of a fixed appliance in such a case.

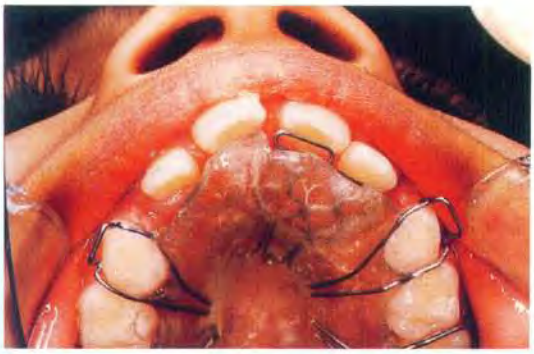


Fig. 43.3 The removable appliance used to correct the crossbite.

A suitable removable appliance is shown in Figure 43.3. It consists of:

- cribs on both upper Ds (0.6-mm wire)
- cribs on both upper first permanent molars (0.7-mm wire)
- T springs on the upper left central and lateral incisors (0.5-mm wire).

Treatment should take no more than 3–4 months as the amount of tooth movement required to correct the crossbite is minimal.

■ ***Why is no posterior capping included on the appliance?***

Posterior capping would normally be considered beneficial when correcting a crossbite. Unless the teeth are held apart during treatment, the upper incisors cannot easily cross over the incisal edges of the lower incisors. Either an anterior bite plane or posterior capping would allow this.

In this particular case it was decided not to incorporate posterior capping as there is a reduced overbite and therefore minimal occlusal interference to the tooth movements. An anterior bite plane would not be indicated in such a case because eruption of the molars would further reduce or even eliminate the already small overbite.

■ ***Figure 43.4 shows the patient at the end of active treatment. What do you see?***

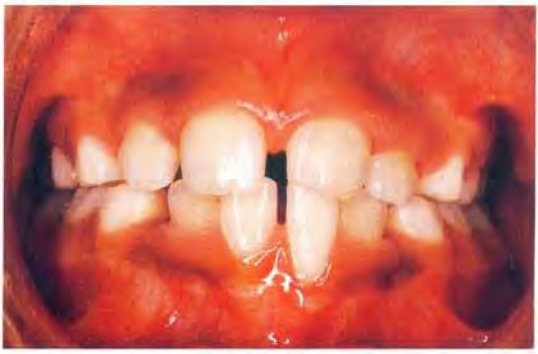


Fig. 43.4 The result after correction of the crossbite.

There has been an improvement in the incisor alignment and the crossbite has been eliminated. If you were able to examine the patient you would find that the mandibular displacement has disappeared.

The patient's oral hygiene has improved and the swollen rounded gingival contour seen in Figure 43.1 has resolved. There is still some slight gingival inflammation in the area of recession. It is difficult to judge whether there is a band of attached gingiva around the lower left central incisor. However, at this age the incisor is not fully erupted and enamel rather than root is exposed. The lower incisor has suffered premature gingival regression rather than recession. Follow up is required to check that sufficient attached gingiva remains until the patient is mature. It appears that no permanent damage has been suffered.

44 Unexpected findings

Summary

A 14-year-old boy presents with toothache and a slightly swollen cheek. What is the diagnosis and how will you treat him?

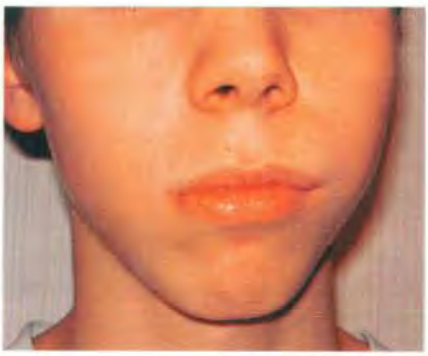


Fig. 44.1 The patient's appearance on presentation

HISTORY

Complaint

The patient complains of intermittent toothache on the left side of his face, which he feels is coming from an upper tooth.

History of complaint

He has been aware of intermittent pain and discomfort from an upper back tooth when eating, especially anything very hot or cold, for several months. The pain is gradually getting worse.

Medical history

The patient is otherwise fit and well.

Dental history

He has never been to a dentist before.

EXAMINATION

Extraoral examination

The patient is a fit and healthy looking boy. His left cheek appears slightly swollen but there is little extraoral asymmetry. The cheek is not tender or inflamed and both the patient and his parents say that he has always looked like this. No lymph nodes are palpable and his temporomandibular joints appear normal.

Intraoral examination

The upper left first molar has heavily stained fissures and the whole crown is discoloured. The other teeth appear sound. The alveolus in the upper left quadrant is enlarged, with reduction of depth of the buccal sulcus. The swelling affects the buccal and palatal aspects, is smooth, uninflamed and is not tender to palpation.

In addition, the upper left second premolar appears missing and there is a small space between the first premolar and molar tooth. Several supernumerary teeth are evident.

■ ***How do you interpret the history and examination so far?***

There could be several explanations for the presentation. The history of the pain, exacerbated by hot and cold, and poorly localized almost certainly indicates pulpitis. The obvious cause would appear to be caries in the upper first molar. The whole crown is discoloured and there may be extensive caries despite the intact occlusal surface.

The enlargement of the alveolus has expanded into the buccal sulcus and could account for the slight extraoral swelling. The commonest cause of smooth uninflamed expansion of the alveolus is an odontogenic cyst. Further investigation is required.

The absent second premolar may be unerupted or absent. Missing premolar teeth is a relatively common developmental anomaly but the patient also has supernumerary teeth and it would be unusual to have missing and supernumerary teeth in the same patient. In addition, if the premolar had never developed, the space between the first premolar and molar would have been likely to have closed completely. The tooth is probably unerupted and relatively superficial in the alveolus, holding the teeth apart.

INVESTIGATIONS

■ ***What investigations would you now undertake and why?***

Vitality tests. The vitality of the upper first molar needs to be determined and on testing it you discover that it appears vital, as are the adjacent teeth.

Radiographs. Right and left bitewings for caries assessment and a panoramic radiograph to assess the overall dentition are indicated. These views should provide sufficient information to explain the missing upper left second premolar, assess any further unerupted supernumerary teeth and to investigate the swelling of the left maxilla. If required, further views including periapicals, oblique upper occlusal views or antral views may be required later but are not indicated at this stage.

■ *The panoramic radiograph is shown in Figure 44.2. Look carefully. What do you see?*

The panoramic radiograph shows:

- a large carious cavity in upper first molar
- right and left supplemental maxillary canines
- peg-shaped supernumerary overlying upper right lateral incisor and right canine
- upper left second premolar present, unerupted and inverted
- developing third molars in all four quadrants
- increased opacity in the region of the left maxillary sinus.

■ *What terms are used to describe extra teeth? What do they mean?*

Term	Definition
Supernumerary tooth	Any tooth over and above the normal complement of teeth.
Supplemental tooth	Supernumerary tooth with the morphology of a normal tooth, usually an additional tooth in a series, for instance additional lateral incisor, third premolar or fourth molar.
Mesiodens	Supernumerary tooth in the upper midline, may be conical (forms early and rarely interferes with eruption of incisors) or tuberculate (with a wide crown with cusps, forms late and often interferes with eruption). See Case 5.

■ *What is the cause of supernumerary teeth?*

The cause is unknown but some clues are available. Supernumerary teeth can be caused by mutations in single genes, for instance the CBFA-1 gene. Mutations of this gene cause cleidocranial dysplasia, in which multiple supernumeraries are a prominent feature. More is known about congenitally **missing** teeth and only a single gene mutation is sufficient to cause teeth to be absent. For instance, mutations in the skeletal patterning gene MSX-1 are associated with missing second premolars and third molars and mutations in PAX-9 are associated with missing molars and lower incisors. It seems likely that supernumerary teeth in normal patients will turn out to have similar relatively simple genetic causes.

■ *The section of the panoramic radiograph showing the left maxilla is enlarged in Figure 44.3. What else does it show?*

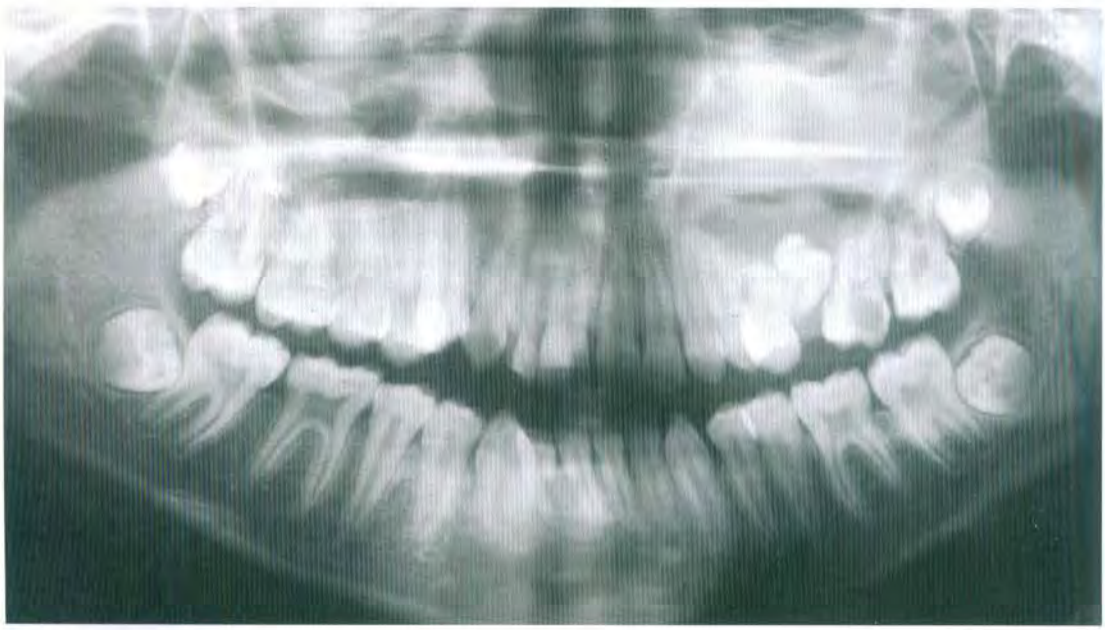


Fig. 44.2 Panoramic radiograph

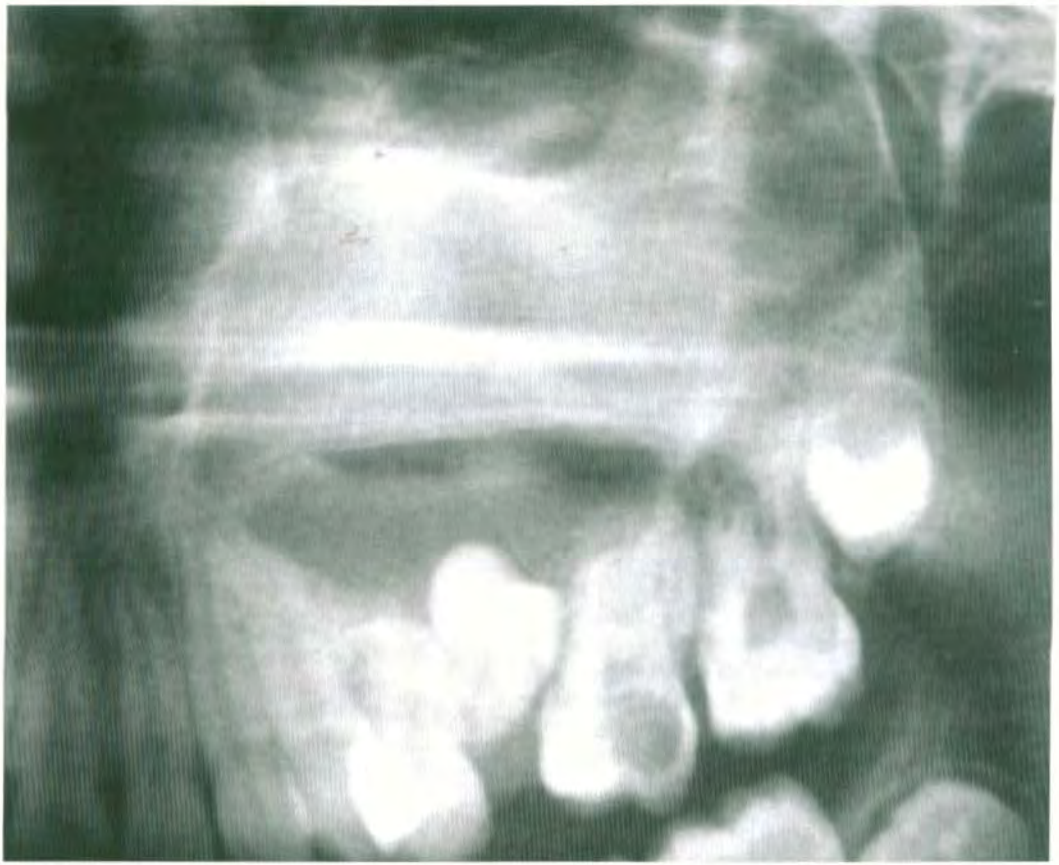


Fig. 44.3 Left maxilla enlarged from Figure 44.2.

There is loss of the thin radiopaque (white) line of the maxillary cortex forming the bony floor of the maxillary antrum. This is clearly visible on the patient's right side in Figure 44.2. A domed relatively radiopaque lesion occupies most of the maxilla and antrum. It has a very thin radiopaque margin at its upper limit.

The panoramic radiograph is shown again in Figure 44.4 with the features including the supernumerary teeth and the margin of the lesion in the left maxilla indicated.

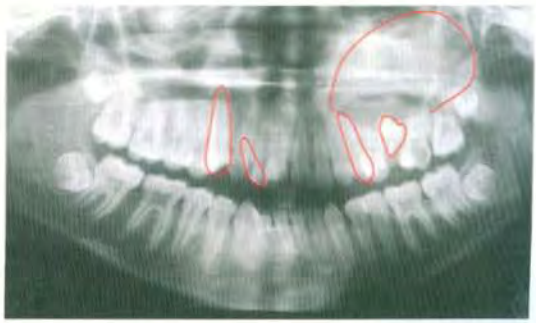


Fig.44.4 The panoramic radiograph with the features outlined.

DIFFERENTIAL DIAGNOSIS

■ ***What is the cause of the patient's pain?***

Pulpitis. As noted above, the symptoms fit pulpitis. The first permanent molar is the most likely source; it is vital and has extensive caries. Periapical periodontitis can be eliminated as a cause because the pain would be well localized and because there are no nonvital teeth in the quadrant.

■ ***Give a differential diagnosis for the lesion in the left maxilla. Explain which cause is most likely and why.***

The unilocular dome-shaped lesion with a thin bony margin of expanded periosteal new bone and the overall round shape are highly suggestive of a cyst. The commonest cysts in the alveolus and maxilla are odontogenic. There are no radiological features of malignancy.

■ *What treatment would you recommend for this particular cyst?*

The inverted second premolar cannot erupt and needs to be removed. The cyst extends around the apices of adjacent teeth and marsupialization would have the advantage that their vitality could be preserved. However, washing out the cavity after marsupialization would be difficult and in a child it would be better to perform enucleation and complete the treatment in one episode. If adjacent teeth were devitalized they would require root treatment, unless orthodontic assessment for the crowding suggested gaining space by extraction. In practice it will probably prove possible to enucleate without devitalizing the adjacent teeth.

A further possibility when enucleating the cyst is to break down the bony wall separating the cyst from the antrum, *remove the cyst lining and extract the unerupted tooth.* This effectively reforms the antrum immediately. A nasal anterostomy (opening from the sinus through the lower part of the lateral wall of the nose) would be required to ensure drainage from the sinus until the antral healing is complete.

Whichever procedure is carried out, a sample, preferably all, of the cyst lining should be taken for histological examination to confirm the diagnosis.

FURTHER INVESTIGATIONS

■ *Figure 44.5 shows the histological appearances of the cyst after enucleation. What do you see and how do you interpret the appearances?*

The left-hand figure shows a length of fibrous cyst wall (W) lined on its inner aspect by epithelium of regular thickness (E). In the bottom right-hand corner there is a focus of inflammation, seen as dark nuclei of inflammatory cells (I), and above it a large pink mural nodule of cholesterol clefts in loose tissue (C) protruding into the lumen. The cholesterol elicits a foreign body giant cell reaction and, although they are not clear at this magnification, the very dark angulate areas among the cholesterol crystals are foreign body giant cells. Haemorrhage (H) is present in and around the mural nodule, visible most easily as red cells on the left of the nodule between it and the epithelial lining. The cholesterol is derived from breakdown of cell membranes of erythrocytes and inflammatory cells that die in the cyst. Two less inflamed areas are shown on the right. The rest of the cyst was lined by similar nonkeratinizing epithelium, often very thin and without rete processes.

Taken together with the radiological features the *appearances indicate dentigerous cyst. Dentigerous cysts* are lined by nonkeratinizing stratified epithelium, though this is not in itself a diagnostic feature. In the early stages the epithelium is characteristically only two cells thick because it is reduced enamel epithelium that has separated from the tooth crown. As the cyst enlarges and impinges on mucosa or the antrum it becomes inflamed. The inflammation causes the epithelium to undergo hyperplasia, thicken and develop rete processes. Inflammation causes cholesterol to form in the wall and it ulcerates into the lumen to form the mural nodules. The appearances can then be very like a radicular cyst and the histopathology is not diagnostic. Therefore, the main reason for submitting the surgical specimen for examination is to exclude an unexpected diagnosis such as odontogenic keratocyst or ameloblastoma. These alternatives do have characteristic histological appearances.

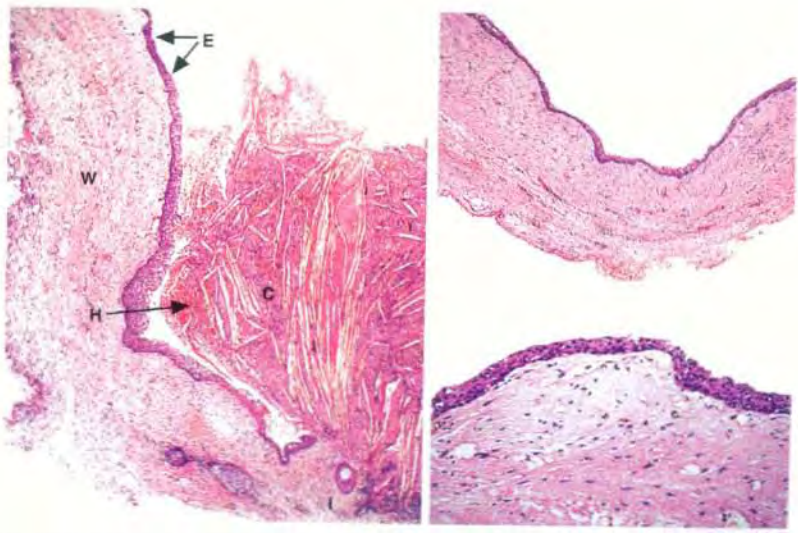


Fig. 44.5 Appearances of the cyst lining after removal, sections stained with haematoxylin and eosin. The width of the cyst wall is 1–1.5mm.

45 A gap between the front teeth

Summary

A 35-year-old man has noticed a gap appearing between two incisor teeth. What is the cause and how can you treat him?

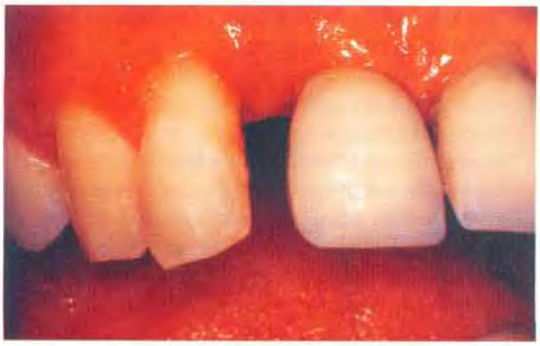


Fig. 45.1 The patient's anterior teeth on presentation

HISTORY

Complaint

The patient is concerned about the gap between the crowned upper right central incisor tooth and lateral incisor.

History of complaint

He noticed the gap about 9 months ago and feels that it has enlarged, that the teeth have drifted forwards and that the crowns are now loose. He has had no symptoms from these or any other teeth.

Medical history

He has mild asthma controlled with an inhaled steroid and salbutamol. He does not smoke.

Dental history

The patient is new to your practice. He attended the dentist regularly, previously going every 6 months but now less frequently. The crowns are approximately 6 years old and were required to replace discoloured class III composite restorations.

■ *The patient's anterior teeth are shown in Figure 45.1. What do you see and how do you interpret the appearances?*

The upper right lateral incisor is rotated mesiolabially and the central incisor distolabially. There is recession on both central and lateral incisors. The gingival tissues have some rounding of the margin and no obvious gingivitis, though there is loss of stippling and contour. The appearances suggest labial drifting as a result of loss of periodontal support. Pocketing is probably present even though it is not obvious.

EXAMINATION

Extraoral examination

There is no lymphadenopathy and no temporomandibular joint signs. The two crowns, although prominent and rather light in shade, are under control of the lower lip associated with a competent lip seal. The patient has a broad smile and moderately high lip line.

Intraoral examination

The oral mucosa is healthy, there is no caries and only a few amalgam restorations but there is focal marginal

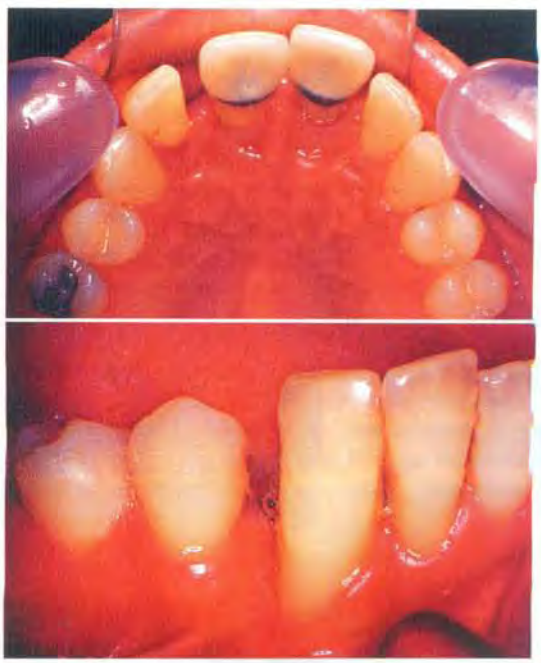


Fig. 45.2 Further views showing the appearances on presentation

inflammation and plaque lying interdentally. The metal ceramic crowns on the two upper central incisors have good margins but there is 3 mm recession palatally and both teeth are grade 2 mobile. A further diastema is present in the opposing arch between the lower right canine and first premolar (Fig. 45.2) and the adjacent lateral incisor has grade 3 mobility.

The gap between the upper central incisor and the lateral incisor is at least 3 mm and on gently probing the area there is profuse bleeding. The gingival tissue in that area was red, inflamed and had lost any contouring or stippling. There are deep probing depths on several teeth and mobility. Probing depths and gingival bleeding are shown in Figure 45.3. For details of indices and periodontal examination see Case 32.

The crowns of the upper incisors are rather bulky palatally, probably as a result of inadequate tooth preparation. On closing to intercuspal position the lower teeth occlude on the crowns in premature contact and displace the teeth labially. In both instances the entire tooth is loose, not just the crown.

INVESTIGATIONS

All teeth are vital.

The choice of radiographs for periodontal diagnosis is reviewed in Case 32. Periapical radiographs for this patient are shown in Figure 45.4.

DIAGNOSIS

■ *What is your diagnosis?*

The patient has chronic gingivitis and localized periodontitis. The periodontitis has reduced the bony support for the upper and lower right anterior teeth and they have drifted labially. Bone loss extends close to their apices and they would have drifted further had they not been retained by the high lower lip line and competent lips. Recession is also the result of the periodontitis (see Case 4).

However, the pattern of periodontitis is unusual. The patient is only 35 years old but has severe localized attachment loss.

■ *What causes severe localized periodontitis?*

The causes are usually local factors affecting the distribution of chronic periodontitis, such as:

- food packing and diastemas
- overhanging and poorly contoured restorations
- subgingival calculus
- destructive habits and self-inflicted injury
- perio-endo lesions
- root fractures
- high fraenal attachment
- localized aggressive periodontitis.

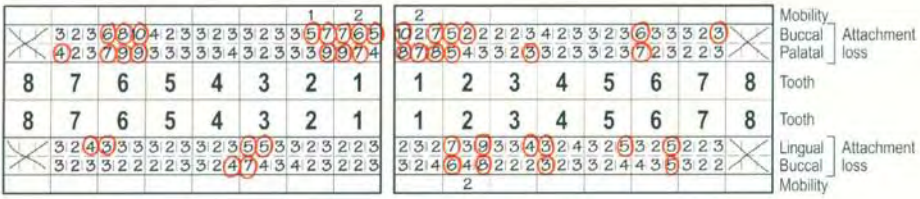


Fig. 45.3 The probing depths (in mm) at six points, bleeding sites (ringed in red) and mobility (grade), recorded for each tooth

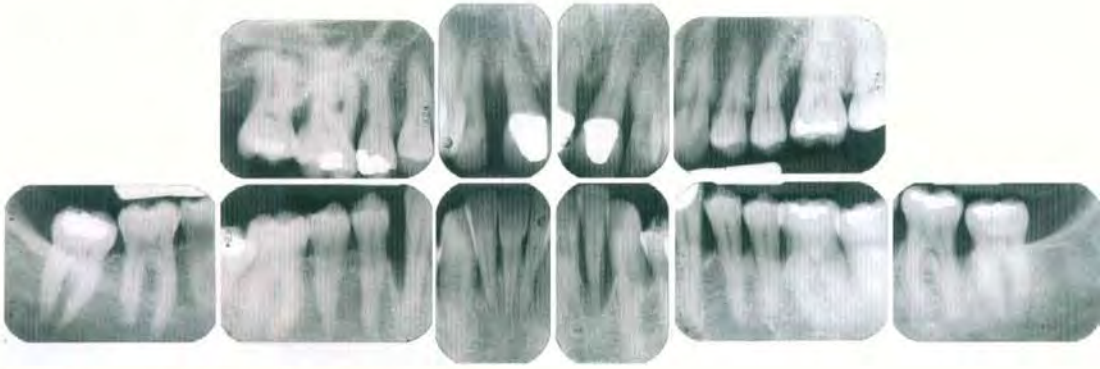


Fig. 45.4 Periapical radiographs of the patient on presentation.

bone and implants would require autogenous bone grafts or guided regeneration techniques with membranes. No immediate insertion technique would be successful.

■ *What are the options for an immediate replacement prosthesis in the lower arch?*

Immediate insertion removable acrylic denture. In the lower arch this is a less advantageous option. A simple partial denture will be relatively bulky and could not be designed to avoid gingival margins. It would therefore be much more likely than an upper denture to compromise plaque control and risks being poorly tolerated.

Immediate insertion minimal preparation bridge. This could comprise a cantilever pontic retained on the central incisor or the canine. The canine is the obvious choice because of its more favourable crown:root ratio but you will need maximum wrap-around to maximize retention. The quality of laboratory work will be critical because the restoration cannot be temporized if the fit is not satisfactory; if the immediate replacement does not fit after the extraction, the patient will be left with an unsightly gap. In the immediate insertion option, moisture control and haemostasis will be of critical importance if the bond is to be sufficiently strong. The design of the pontic must be optimized to favour plaque control in view of the patient's susceptibility to periodontitis. Probably the best option if a replacement is indicated.

No replacement. In the short term it is quite possible that the patient might accept the appearance of the gap. This would be the best option for the periodontal treatment and the situation could be reassessed when the prognosis of the lower teeth becomes clearer.

The patient opts for an upper immediate insertion removable acrylic denture and a lower immediate insertion minimal preparation bridge. He completes the first eight items of your treatment plan and no further extractions are necessary. After 6 months the patient has responded very well to plaque control and scaling/root planing. There has been marked gingival shrinkage around the lower canine and there is no bleeding on probing and a pocket depth of 4 mm. The excellent response to treatment is, of course, largely down to the effort of your hygienist who has motivated and educated the patient and performed the treatment.

The patient is 'managing' with the denture but dislikes it. It is now becoming less retentive and the anterior teeth have dropped on a couple of occasions. He would very much like to discuss options for a more permanent replacement.

The appearances of the patient's mouth after this initial treatment are shown in Figure 45.5. Bone levels around the remaining teeth are more or less as they were on presentation in Figure 45.4.

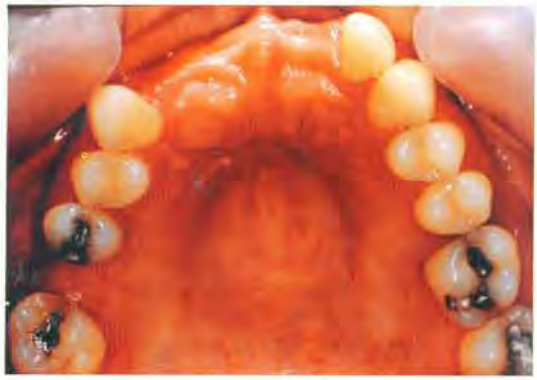


Fig. 45.5 The upper arch six months after extractions and periodontal treatment

■ *What are the options for definitive treatment?*

A bridge. This would be possible but there are some difficulties. The extensive bone loss from periodontitis has resulted in fairly marked shrinkage of the ridge after extraction. This would require the pontic to be placed more palatally than is ideal and, in combination with the high lip line and reduced lip support, could make the appearance of a bridge less than ideal. This problem would be overcome by a removable prosthesis with a labial flange.

The span is quite long and one of the abutment teeth is a lateral incisor. It is unrestored but lateral incisors are never abutments of choice because of their small root, thin coronal dentine and angulation. In addition Figure 45.4 shows bone loss of half the root length. Although this tooth is currently stable there must remain doubts about long-term maintenance and it is not suitable to take the load required of a bridge abutment, even if the adjacent canine were prepared as well. If a bridge were to be provided it would be wiser to extract the compromised upper left lateral incisor and construct a fixed–fixed bridge using both canines as abutments. This would allow better control of the appearance. Had the tooth been less compromised it could have been included in a double abutment with the canine. A bridge would also involve considerable expense.

Implants. An implant-retained bridge is possible. The periodontitis would need to be fully treated and maintained and bone graft would be necessary for ideal pontic placement.

A new permanent removable acrylic prosthesis. If the patient was not managing to control the periodontitis or the prognosis of other teeth was questionable, a replacement denture with the major connector constructed from acrylic resin could be considered. This prosthesis would derive

some support from rests on the cingula of the canine teeth and by covering the vault of the palate. Teeth with uncertain prognosis could be added later and, in the meantime, would not be clasped for direct retention. Clasps of either wrought stainless steel or gold could be used on teeth with a favourable prognosis.

Removable prosthesis with chrome–cobalt framework. This is probably the best option. It is less expensive than a bridge or implants, much simpler and can be designed to favour oral hygiene procedures and for addition of further teeth in the future.

The patient is very happy with the appearance of the acrylic partial denture and found it well retained until recently. After discussing the options the patient opts for a new cobalt–chromium-based removable prosthesis. An additional factor not visible in the pictures is that the lower incisors have overerupted as a result of the drifting of the upper teeth. The occlusion makes the option of a bridge untenable without adjustment of the lower incisors.

■ *Who should design the prosthesis, you or the laboratory? Why?*

The design of any denture has to be a team effort between you and the laboratory, with you as the dentist taking the final responsibility for the restoration. In an ideal environment the dentist should have access to a model surveyor and use the skills learnt in dental school surveying and designing regularly. However, with increasing emphasis on the role of the team, it should be possible to devolve the detailed design to your laboratory technician. As a minimum you will need to discuss which teeth need to be replaced and identify any teeth with doubtful prognosis.

■ *What are the general principles to consider when designing any partial denture?*

Decide the teeth to be replaced and outline the saddles.

Plan support for the denture. Is the denture going to be tooth supported, tooth and mucosa supported or just mucosa supported?

Plan the path of insertion on a model surveyor, identify guide planes and consider their preparation. As this patient has lost anterior teeth and has a high lip line, the acrylic flange has to engage the labial soft tissue undercut so the model will have a tilt to the posterior teeth on the surveyor table.

Plan direct retention normally provided with clasps. Consider engaging 0.25 mm undercuts with cast cobalt-chromium occlusal approaching clasps and 0.5 mm with gingival approaching clasps.

Is there adequate direct retention or will you have to provide indirect retention?

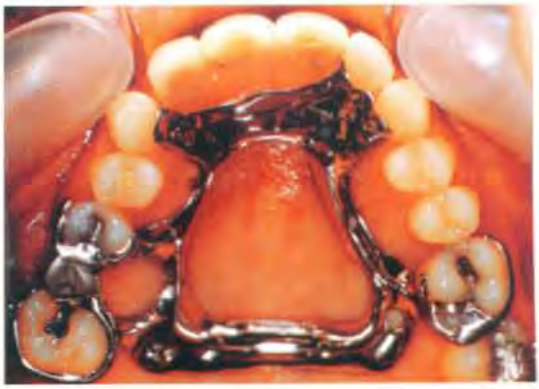


Fig. 45.6 The final design.

Consider major and minor connectors. These together with proper base extension will help to provide stability against lateral and anteroposterior displacing forces. Advantages of different connectors are given in Table 45.1.

Review design for three aspects: simplicity, biological acceptability and appearance. Is as much gingival tissue as possible left uncovered? Are there unsightly anterior clasps or prominent occlusal rests that are really unnecessary?

What tooth preparation is required? This should include rest seat preparation either cutting into the tooth or adding composite resin, guide planes and altering the height of survey lines to allow correctly constructed clasps.

■ *The final design is shown in Figure 45.6. What are the design features in this particular case?*

- The path of insertion was determined by the soft tissue profile of the anterior edentulous saddle. When a large labial undercut is present the model must be tilted down posteriorly during surveying to ensure that the flange fits well and has the best possible appearance.
- Support was derived from rests placed on the upper right canine, second premolar and first molar and left lateral incisor, canine and first molar. This is probably more than necessary but the design has not become overcomplicated. The rest on the left canine is necessary in case the compromised lateral incisor is extracted in future. Without it the denture would have no left anterior support after the extraction. This demonstrates the importance of planning for possible failure and also good communication with the laboratory technician. This rest on the canine might not have been included otherwise.
- Direct retention was provided by cast cobalt–chromium occlusal approaching clasps placed on posterior teeth because of the patient's high smile line. The clasps face into the main saddle. However, the recurved clasp on the upper right premolar is still visible (see Fig. 45.7). The clasp tips are placed in undercuts relative to the planned

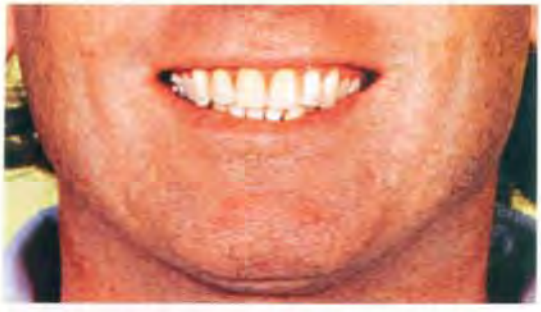


Fig. 45.7 The finished result

path of insertion. There is sufficient direct retention that indirect retention is unnecessary.

- The major connector of anterior and posterior palatal bar connectors has at least 5 mm clearance from the gingival margin (see Table 45.1). This type of connector does not provide soft tissue support so that a fully tooth-supported design is necessary. If the denture had included a posterior saddle as well, an element of tissue support would have been necessary and full palatal coverage would have been required
- Minor connectors have been placed to avoid the interdental gingival tissues, the areas most susceptible to chronic periodontitis. This has been achieved with *mid-unit minor connectors* on the posterior teeth.
- The pontic design chosen for the upper right molar was a metal sanitary pontic. The tilting of the second molar had reduced the space. Using a sanitary pontic has avoided placing any components in the periodontally vulnerable interdental area.

■ ***What is indirect retention and why is none required for this denture?***

If a partial denture has two clasps the denture will tend to rotate away from the soft tissues about an axis between the two clasps. Indirect retention is provided by a rigid component placed on the teeth opposite to the saddle so that the saddle cannot fall away from the ridge. Indirect retention is usually required for free end saddles (distal extension) and in Kennedy class IV situations where direct retention from anterior clasps at each end of the saddle would be unsightly. In a single free end saddle situation the denture will rotate around the distal clasps and a rest must be placed on a tooth opposite the saddle and as far away from the axis of rotation as possible. In the present case direct retention from clasps is sufficient.

■ ***The appearances of the finished denture are shown in Figure 45.7.***

The final result, a restoration that the patient was delighted with and that you should be pleased with. One posterior clasp is visible but the overall result is excellent. This case effectively illustrates how a relatively simple treatment plan produces the most predictable result.

Table 45.1 Major and minor connectors – advantages and disadvantages

Connector	Indications	Advantages	Disadvantages	Key to clinical success
Mandibular lingual plate	Commonly used For use where the lingual sulcus is shallow	Teeth can be relatively easily added With prepared rest seats can provide excellent indirect retention	Covers much gingival tissue Has to be fenestrated if diastemas are present	Has to be well adapted to the lingual aspects of the incisor teeth
Mandibular lingual bar	The other most commonly used mandibular connector	Minimal tissue coverage	Teeth cannot be added subsequently Requires minor connectors to connect to rests for support and indirect retention in Kennedy class I and class II situations	Half pear shape in profile Needs at least 8 mm clearance between the gingival margin and the raised floor of the mouth
Mandibular sublingual bar	Where a bar connector is required but the lingual sulcus is shallow	Minimal tissue coverage	Can be bulky due to profile, projects out towards the tongue and risks being poorly tolerated	Very accurate functional impression of the raised floor of the mouth is required
Mandibular continuous bar (cingulum bar)	Where a bar connector is required but the lingual sulcus is shallow	Minimal tissue coverage Teeth can be added	Needs to be bulky on the cingula of the teeth for rigidity Poor aesthetics if diastemas are present	Needs to be well supported and needs good adaptation to the teeth Can be used with a lingual bar (Kennedy connector) and both components can be made with reduced dimensions

46 A lump in the palate

Summary

A 32-year-old lady is referred to your hospital oral and maxillofacial surgery department by her general dental practitioner because of a swelling in her palate. What is the cause and what treatment is appropriate?

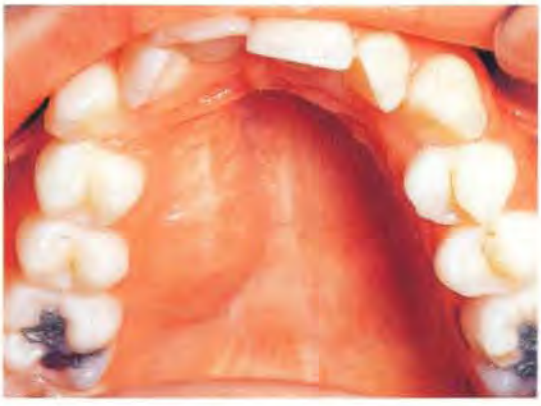


Fig. 46.1 The patient's palate on presentation.

HISTORY

Complaint

The patient has noticed a lump but has experienced no pain.

History of complaint

The patient thinks that the lump has been present for at least a year, possibly two. It has enlarged slowly and is now starting to be a nuisance.

Medical history

Her medical history reveals no illness. She has recently given up smoking but previously smoked two or three cigarettes a day.

EXAMINATION

Extraoral examination

She is a fit and well-looking woman. No cervical lymph nodes are palpable and the temporomandibular joints appear normal. There in no facial asymmetry.

Intraoral examination

The appearance of the palate is shown in Figure 46.1. There is a swelling of the right side of the palate and maxillary alveolus. There is no caries and only a few relatively small amalgam restorations.

■ *What are the features of the swelling?*

The swelling has the following characteristics:

Site	Molar to central incisor region
Size	2 × 3 cm approximately
Shape	Oval
Surface	No ulceration
Colour	Overlying mucosa normal. Has a slight blue tinge No evidence of inflammation, not pointing
Contour	Regular, rounded

If you were able to palpate the lesion and the patient's neck you would discover the following:

Lesion consistency	Firm, not fluctuant
Lesion mobility	Fixed
Cervical lymph nodes	No submandibular or cervical lymph nodes palpable

DIFFERENTIAL DIAGNOSIS

■ *On the basis of what you know so far, what types of lesion would you include in your differential diagnosis?*

1. Benign neoplasm of palatal salivary gland, most probably a pleomorphic adenoma
2. Malignant salivary tumour, and if so:
   — most probably a mucoepidermoid carcinoma, *or*
   — polymorphous low grade adenocarcinoma,
   — but possibly an adenoid cystic carcinoma
3. Odontogenic causes:
   — *either* an abscess
   — *or* an odontogenic cyst, probably a radicular cyst
4. Mucous retention cyst
5. Antral or nasal lesion bulging into the mouth (e.g. a carcinoma)
6. Miscellaneous other possibilities

Table 46.2 Possible biopsy types

Biopsy type	Advantages and disadvantages
Fine-needle aspiration cytology (FNA/FNAC)	Quick and accurate in most cases though a minority of such lesions will not be amenable to diagnosis by cytology. Nevertheless, usually accurate enough to give the definitive diagnosis and can be performed in conjunction with other biopsy types if necessary. Result usually available in 1 day.
Trucut or wide-needle biopsy	These provide a small tissue sample. Wide-needle biopsy is now little used because FNA is easier and more accurate. There is a risk of damaging important structures, though this is more of a problem in the parotid gland than in the palate.
Incisional biopsy	As a general rule, incisional biopsy risks spreading salivary neoplasms into the tissues. Pleomorphic adenomas, the commonest benign neoplasms, are often mucinous in texture and can spread into the fascial planes of the neck and up to the skull base or down to the mediastinum when incised for biopsy. However, this is more of a problem in submandibular or parotid glands. In these sites incisional biopsy should not be performed unless the lesion is thought to be malignant. Only then will the diagnosis influence treatment. In the present lesion, spread is not a particular concern. The top of the lesion is accessible and the entire biopsy site could be excised during definitive surgery if the lesion turns out to be malignant. There is no risk of spread to tissue spaces. An incisional biopsy could be performed, but would not be required if a fine-needle aspirate provided a diagnosis.
Excisional biopsy	If the lesion were smaller then an excisional biopsy might be considered appropriate. A small margin of normal tissue could be excised on the assumption that the lesion is a pleomorphic adenoma. This would not be ideal. FNA or incisional biopsy are readily performed and it would be better to determine whether the lesion is benign or malignant before excision, to ensure that an appropriate margin is taken.

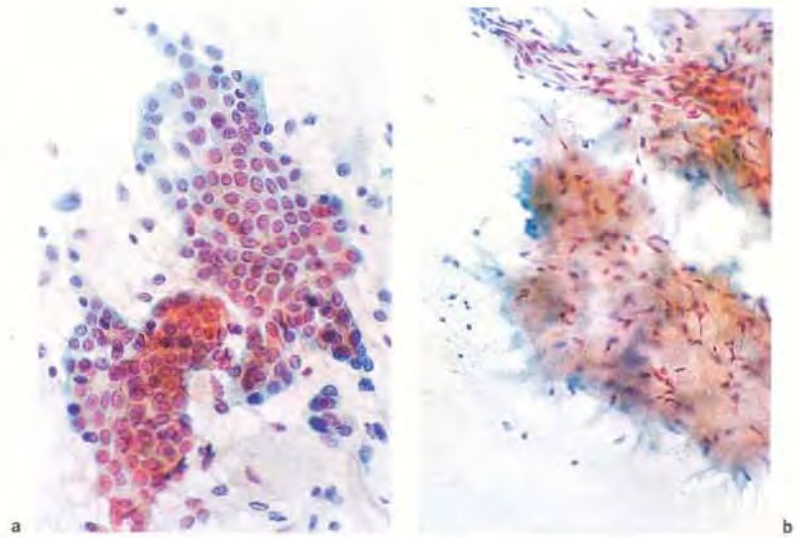


Fig. 46.2 a, b The appearances of the smear of cells taken by fine-needle aspiration.

Either an incisional biopsy or fine-needle aspiration should be performed, and the choice will depend on the availability of cytology services in the clinic and the surgeon's preference.

■ ***A fine-needle aspiration of the lesion was performed and the appearances of the aspirate are shown in Figure 46.2. What do you see?***

The cells are stained with the Papanicolaou stain which stains nuclei blue and cytoplasm green, or orange if keratinized. Figure 46.2a shows a sheet of uniform cells with moderate amounts of cytoplasm. Their cytoplasm, polygonal shape and cohesive growth indicate that these are epithelial cells and their uniform nuclei suggest they are benign. The second field, shown in Figure 46.2b, shows smaller numbers of spindle cells without significant cytoplasm in a myxoid matrix. Taken together, these appearances are characteristic of pleomorphic adenoma.

DIAGNOSIS

The diagnosis is pleomorphic adenoma.

TREATMENT

■ *How should this lesion be treated?*

The appropriate treatment is excision with a small margin, at least a few millimetres. The pleomorphic adenoma is benign but often incompletely encapsulated and simple enucleation often results in recurrence. The defect could be closed with either a temporary acrylic plate or local surgical flaps depending on the surgeon's preference. Radiotherapy is ineffective as a primary treatment and would not be indicated for a benign neoplasm. It is sometimes used for widespread recurrences of parotid pleomorphic adenomas.

■ *Would you like further investigations before carrying out treatment?*

Although the pleomorphic adenoma is benign it can resorb bone by pressure. Before excising the lesion it is important to know whether the palate has been perforated because any postsurgical oronasal or oroantral fistula would have to be repaired. This lesion is relatively small and this step might be omitted, but with a larger tumour it would be an appropriate reason for carrying out a CT scan.

■ *The histological appearances of the excision specimen are shown in Figure 46.3. What do you see?*

Very different appearances are seen in different areas of the tumour (it is pleomorphic). Figure 46.3a shows sheets of epithelial cells in which there are small ducts containing eosinophilic material. At the edges of the sheets, cells separate and progressively merge with more dispersed cells in a myxoid stroma. These sheets and dispersed cells are the same cell types as those seen in the smear made from the fine-needle aspirate shown in Figure 46.2. Here they are seen in section, in their correct relationship to each other, whereas the smear is of whole cells spread onto a glass slide, hence their different morphology.

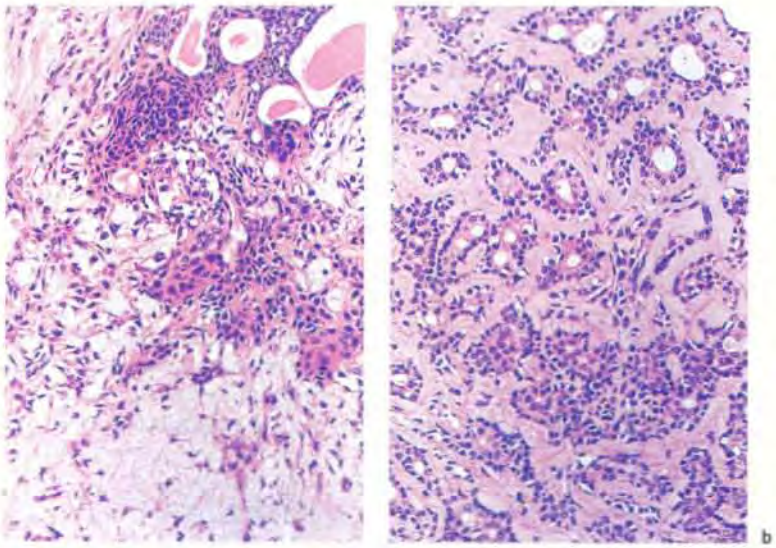
Figure 46.3b shows numerous small duct-like clusters of cells separated by a hyaline fibrous stroma. Many of the ducts have a bilayered structure with a partial outer layer of cells with clear cytoplasm.

Other fields show incomplete encapsulation but a well-demarcated periphery. These features are typical and diagnostic of pleomorphic adenoma. If the lesion appears excised histologically, cure is expected.

PROGNOSIS

■ *Are there any significant complications of pleomorphic adenoma? Are you concerned for this patient?*

Yes, carcinoma may arise in a long-standing pleomorphic adenoma and these *carcinomas ex pleomorphic adenoma* carry a very poor prognosis. However, this process takes many years, usually 10 or more, and is therefore seen mostly in elderly patients. This is an unlikely risk in this young patient with a short history. There is nothing in the history or examination to suggest malignancy and fine-needle aspiration or biopsy and examination of the excised specimen have excluded it.



a b

Fig. 46.3a, b The histological appearances of the excision specimen.

47 Rapid breakdown of first permanent molars

Summary

A 7-year-old boy presents with first permanent molar teeth which his parents say have decayed rapidly, starting immediately on eruption. Identify the cause and discuss the treatment options.

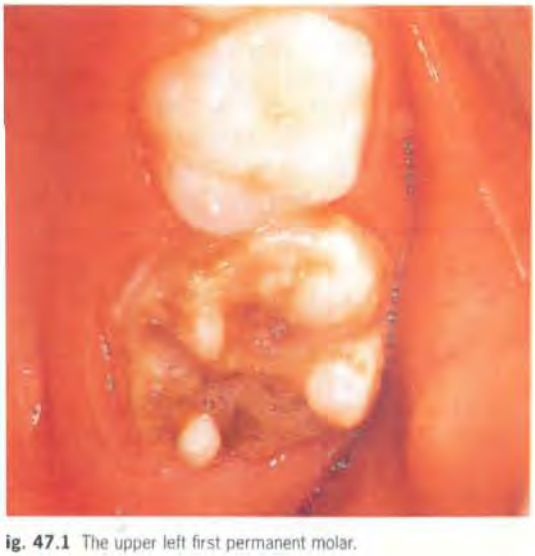


Fig. 47.1 The upper left first permanent molar.

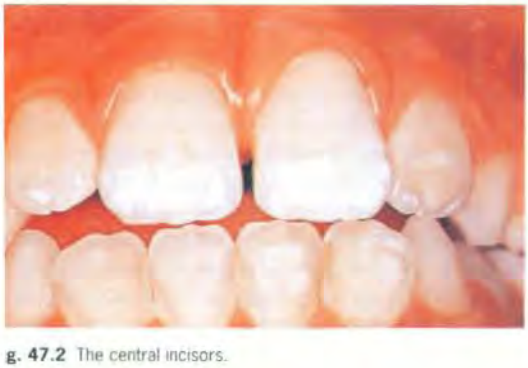


Fig. 47.2 The central incisors.

HISTORY

Complaint

The child complains of pain from his back teeth on both sides. The pain is worse with sweet foods and cold liquids and persists for several minutes after stimuli. Tooth brushing with cold water is also painful but the teeth do not cause pain on biting.

History of complaint

The pain has been present for a few months and has increased in severity over the last month. The child now reports that one of his back teeth feels broken. The first permanent molars erupted on time and his mother noticed that some of them appeared to crumble as soon as they emerged through the gum. She has read in magazines that fluoride can damage teeth and has switched to a toothpaste without fluoride on the assumption that this is the cause.

Medical history

The patient is a healthy child, the only history of note being neonatal jaundice.

Dental history

The child has no experience of operative dental care. A diet history reveals a reasonably well-balanced diet, with limited consumption of refined carbohydrates and carbonated beverages. Toothbrushing has been performed with adult-formula fluoride-containing toothpaste, starting at approximately 1 year and continuing until 7 years of age.

EXAMINATION

Extraoral examination

The child has no facial swelling or asymmetry and no lymph nodes are palpable.

Intraoral examination

He is in the early mixed dentition stage. All four first permanent molars have areas of brown, rough, irregular coronal enamel. The severity varies between the teeth and the worst affected are the maxillary molars whose enamel appears to be completely absent in some areas. These teeth have soft dentine exposed occlusally. The lower right first permanent molar is the least severely affected with only a small localized brown enamel defect on the buccal aspect. This is hard on probing. In addition, there are areas of white enamel opacity in the incisal third of the labial surface of all permanent central and lateral incisors, which are most pronounced in the maxillary central incisors. The remaining primary dentition is caries free, and appears normal in structure and morphology. Oral

hygiene appears good. The appearances of the dentition are shown in Figures 47.1 and 47.2.

■ ***On the basis of what you know already, what do you suspect?***

The defects appear to be hypoplasia of the enamel which has either become carious or taken up extrinsic stains. The molars are so severely affected that diagnosis is difficult, but the opaque white zones on the central incisors are characteristic of enamel hypoplasia. This term is often incorrectly used to include enamel hypoplasia (in which less enamel is formed) and enamel hypocalcification (in which the enamel is not fully mineralized).

■ ***Do the enamel defects follow a chronological pattern, and if so, at what time was the affected enamel formed?***

Yes, the incisal and occlusal parts of the permanent central incisors and first molars form at about the same time, starting to mineralize just before birth. The affected enamel would have been formed after birth and during the first 1-2 years of life. This may be seen by consulting Figure 47.3. This distribution of hypoplasia is often referred to as molar–incisor hypoplasia.

■ ***What additional questions would you ask, and why?***

The chronological pattern suggests systemic illness which may be identifiable in the history. Defining a possible cause may allow others such as fluorosis to be excluded. You need to ask further details about the prenatal and perinatal medical history. The following conditions may be relevant and should be specifically sought:

- Preterm birth or low birth-weight baby
- Rhesus incompatibility
- Intubation as neonate
- Maternal vitamin D deficiency.

These disturbances may manifest as enamel defects distributed along the enamel formed around birth. You should also enquire about all severe systemic disturbances in the first 2 years of life, for example meningitis, encephalitis, severe measles or pneumonia.

DIFFERENTIAL DIAGNOSIS

■ ***What is the likely cause of the child's pain?***

The hot and cold sensitivity is characteristic of pain mediated by a vital pulp. It could be a result of caries in the dentine or exposed occlusal dentine.

■ ***What is your initial differential diagnosis for the enamel hypoplasia?***

Dental caries is the commonest cause of destruction of first permanent molars and should be considered, even though the appearances would be very unusual. Enamel hypoplasia is more likely and developmental and acquired forms and/or generalized and localized forms are recognized. The most likely cause is enamel hypoplasia due to neonatal illness. Other causes which might be considered are amelogenesis imperfecta, fluorosis and cytotoxic chemotherapy for malignant disease. In some cases no cause is found and the term idiopathic enamel hypoplasia is used.

■ ***Justify this differential diagnosis.***

Dental caries is the commonest cause of destruction of the dental hard tissues. Newly erupted teeth are particularly prone to dental caries until their enamel maturation is completed in the oral environment. First permanent molars are also prone to early caries because of their deep fissures. However the possibility of caries seems unlikely. Although there is no guarantee that the diet history elicited is truly representative of the child's actual diet, there are no restorations or caries in the deciduous dentition. The molars have discoloured or absent enamel over a wide

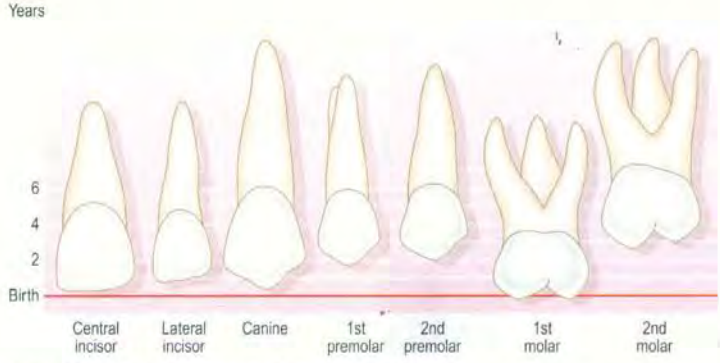


Fig. 47.3 Time of mineralization of the permanent dentition.

area. This is not typical of dental caries unless carbohydrate intake is excessive or the teeth have some other predisposing factor such as enamel hypoplasia. The soft dentine indicates that some caries is present but the pattern of destruction suggests that this caries is secondary. The zones of opacity on the incisors look like early 'white spot' demineralization but are at a site that is almost never affected by caries.

Enamel hypomineralization and/or hypoplasia is the most common developmental disorder observed in teeth. This child has a generalized defect with a chronological pattern. The history of neonatal jaundice deserves some consideration. It is capable of affecting amelogenesis but rarely causes clinically evident hypoplasia and then only in very severely affected individuals. Neonatal jaundice is a very common condition and in almost all cases can be excluded as a cause of enamel hypoplasia. In this child the position of the enamel defects is inconsistent with a short period of jaundice at birth.

Fluorosis where it is endemic, is a common cause of enamel opacities and enamel hypoplasia. The deciduous dentition is usually much more mildly affected. The level of fluoride required to cause enamel defects depends on concentration, period and age of ingestion.

Fluorosis is almost certainly not the cause of the current problem. The defects in the molars would result from only very high fluoride concentrations, in excess of 25 parts per million (p.p.m.). Such levels do not occur in the UK. In addition, fluorosis of this severity is endemic and should not follow a chronological pattern.

Mild fluorosis may be seen as a result of supplementation and this is presumably what the parent has read. In such cases there are usually fine opaque white lines following the perikymata and small irregular enamel opacities or flecks with or without staining. Such mild defects are also common in normal teeth and increase in frequency when the fluoride level is lower than 0.7 p.p.m. Although the severity of the defects and distribution are incompatible with the diagnosis of fluorosis, it needs to be considered because the child has been using adult-formula fluoride-containing toothpaste from an early age. Ingestion of adult-formula fluoride toothpaste would be the most likely cause of fluorosis. Fluoride toothpastes should not be used by children until they can rinse and spit out, at about the age of 7 years.

Amelogenesis imperfecta must be considered even though it is rare. Amelogenesis imperfecta can cause enamel hypoplasia, hypocalcification or hypomaturation and either of the first two conditions could lead to the appearances seen in the molar teeth. However, several factors suggest that this is not amelogenesis imperfecta. There appears to be no family history, the pattern appears chronological rather than affecting all surfaces of all teeth equally and the deciduous dentition is unaffected. While these features are not conclusive because of the wide range of clinical presentations seen in the many different types of this disease, they do make the diagnosis most unlikely.

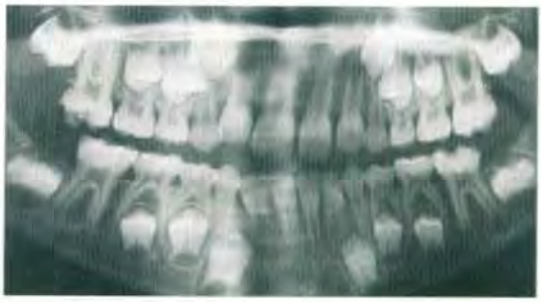


Fig. 47.4 Panoramic tomograph.

Idiopathic molar–incisor hypoplasia is a convenient term used to describe cases of enamel hypoplasia with this distribution for which no cause can be ascertained.

INVESTIGATIONS

■ *What investigations are indicated and why?*

Intraoral radiographs are indicated to assess the proximity of the coronal defects to the dental pulp. A panoramic tomograph is indicated, to ascertain the presence and stage of development of the remaining permanent dentition, in view of the possibly poor long-term prognosis of some of the first permanent molars. The panoramic tomograph is shown in Figure 47.4.

If there were extensive softening of the occlusal dentine of the molars or if radiographs indicated deep caries, tests of vitality would be required.

■ *What does the panoramic tomograph show?*

All permanent teeth with the exception of the third permanent molars are present and the dental age is consistent with the patient's chronological age. The gross structural defect in the first permanent molars is reflected in their radiographic appearance. The worst affected teeth – the maxillary molars – have irregular enamel outlines and there is reactionary dentine in the distal pulp horns. The view is unsuitable for detailed examination of the teeth, but no large carious lesions are evident and the unerupted permanent second molars appear to be of normal shape and to have a normal enamel structure.

DIAGNOSIS

■ *What is your diagnosis?*

The patient has enamel hypoplasia in a chronological pattern, and in the absence of a known insult to account for

48 Oral cancer

Summary

A 72-year-old man with squamous cell carcinoma is referred to your cancer treatment centre. How should he be managed?

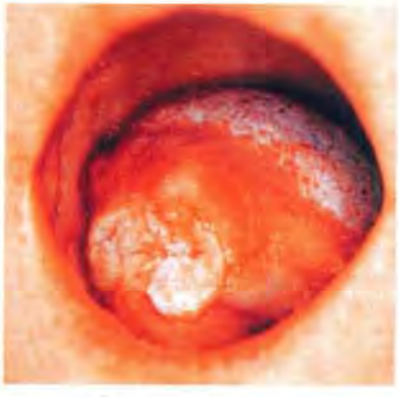


Fig. 48.1 The patient's appearance on presentation

HISTORY

Complaint

The patient complains only of discomfort on wearing his dentures and earache on the right, despite the large lesion.

History of complaint

He first noticed symptoms 6 weeks ago and has sought treatment because of the earache. He had noticed a 'crack' in his tongue that has not healed for some time but this has always been painless.

Medical history

He had a small myocardial infarction 7 years ago and has mild hypertension for which he takes amlodipine and aspirin. He has smoked 20 or more cigarettes a day for the last 55 years and drinks a quarter bottle of whisky per day.

EXAMINATION

Extraoral examination

The examination of the neck is normal *apart from a* possible mass just below the right angle of the jaw. It is difficult to ascertain whether or not the mass is hard. It is deeply situated and approximately 15 mm in diameter.

Intraoral examination

■ *The appearance of the ulcer is shown in Figure 48.1 What do you see?*

There is a large fungating ulcer arising on the right lateral border of the tongue. It is approximately 30 mm in size and has raised everted borders. Towards the dorsal surface the mucosa bulges, as if there is a mass beneath the intact epithelium, and there is a white patch affecting the mucosa over it.

If you could palpate this ulcer you would find that it is indurated (firm or hard on palpation) and extends down towards the floor of the mouth and up beneath the dorsal mucosa towards the midline. The lesion is clearly much larger than it looks. The patient has reduced tongue mobility.

DIAGNOSIS

In this case the diagnosis has already been made at the referring hospital. The patient has squamous cell carcinoma and the appearance and presentation are typical. The patient knows the diagnosis.

■ *Why might the patient have earache?*

Ear pain is thought to result from malignant infiltration (or other damage) of nerves that pass through or supply the ear on their way to the periphery. Thus in the tongue or pharynx, pain may be generated by involvement of the lingual nerve, which contains fibres from the chorda tympani from the ear and the auriculotemporal nerve (branch of the mandibular division of the trigeminal nerve), which also supplies the external auditory meatus. Similar referred pain may result from involvement of the glossopharyngeal nerve.

PRINCIPLES OF TREATMENT

■ *What are the principles of treatment for oral squamous cell carcinoma?*

Patients with oral carcinoma often present too late for cure and some may not benefit from treatment. Three treatment options are possible:

complete removal of the carcinoma because the lymph nodes are removed covered by fat or other tissues.

Excision of the sternomastoid muscle sacrifices the spinal accessory nerve, which passes through it. This causes a postoperative droop of the shoulder because it supplies trapezius as well as sternomastoid. For this reason prophylactic neck dissections are more conservative and either part or all of sternomastoid and the internal jugular vein may be left behind.

TREATMENT

The recommended treatment for this case is to excise the carcinoma at the primary site and perform a prophylactic neck dissection en-bloc (in one continuous specimen). Postoperative (adjuvant) radiotherapy is planned. The defect in the mouth will be reconstructed with a radial forearm flap. The patient opts for this treatment and makes a good recovery following surgery.

■ ***Pictures from the pathological examination of the resection specimen are shown in Figure 48.3. What do you see?***

The surgical specimen is shown on the left. At the top is a hemiglossectomy (HG), below it the contents of the submandibular triangle (SMT) and submental tissue (SM). The sternomastoid muscle has been preserved and the fat surrounding the jugular vein and cervical lymph nodes is on the left (F). The omohyoid muscle extends across the back of the specimen (O). In the centre the hemiglossectomy is shown at higher power. You can see the ulcer with its everted margin on the lateral border of tongue and extending into the floor of mouth. On the right is a vertical coronal section across the tongue with the midline on the left. The specimen has lost its colour after fixation in formalin. The ulcer (U) and the mass visible clinically towards the dorsum (M) are marked. Carcinoma appears white (because it contains keratin). Note how deeply the carcinoma has penetrated, to within a few millimetres of the midline excision margin (arrowed). Luckily, carcinoma extends upwards and medially rather than down into the floor of the mouth, where excision would have been more difficult.

■ ***The microscopic appearances are shown in Figure 48.4. What do you see?***

On the left, at medium power, is typical squamous carcinoma comprising islands of epithelium invading around and between muscle fibres. The carcinoma is forming keratin and, in this area, is well or moderately well differentiated. The next panel shows a more poorly differentiated area at high power. Pleomorphic cells with irregular nuclei (anisonucleosis) and darkly staining nuclei (hyperchromatism) are infiltrating along a pink muscle fibre running from top left to bottom right (MF). There is invasion as single cells (S). The third panel, at high power, shows a small vein (V) filled by carcinoma and inset is a cluster of carcinoma cells in a lymphatic. Invasion as single cells and lymphatic and vascular permeation are poor prognostic factors and indicate a high risk of metastasis. Two of the lymph nodes from the neck dissection are shown on the right with a scale in millimetres. Metastases (MT) are seen as round pink areas because of the keratin in them. There is a compressed rim of residual blue lymph node tissue at the periphery (L).

The surgical result is reviewed at a meeting of the multidisciplinary team. The closest margin of excision was 5 mm and metastatic carcinoma was found in 2 of 28 lymph nodes found in the neck dissection. The patient is doing well postoperatively.

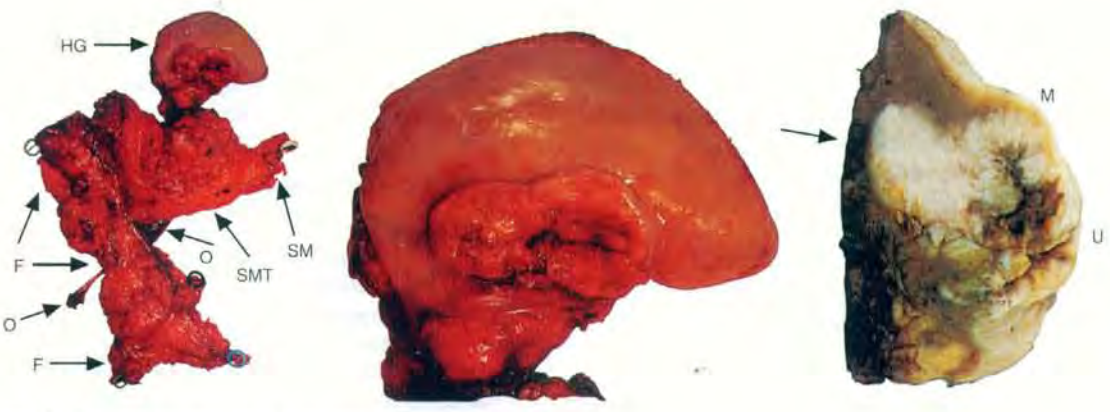


Fig. 48.3 The pathology specimen, macroscopic views.

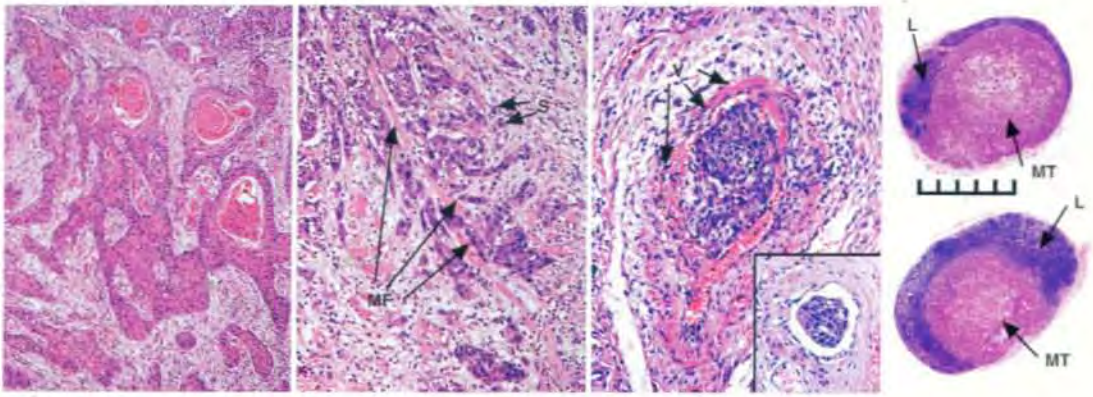


Fig. 48.4 The pathology specimen, microscopic views.

■ *Are these excision margins adequate?*

A margin of 10 mm of normal tissue would be ideal but in many cases the excision margins are only a few millimetres. If no radiotherapy had been planned, a margin less than 5 mm would be an indication for postoperative radiotherapy and the treatment plan would be changed. This patient has a poorly differentiated carcinoma and metastases. Postoperative radiotherapy can compensate for close or incomplete excision and so a full course of postoperative radiotherapy to the primary site and neck is recommended.

■ *What is the procedure for radiotherapy?*

Radiotherapy must start within 6 weeks of surgery for best results. Once the patient has recovered from the operation and swelling has settled, the preparation for radiotherapy can begin. The radiation will be given in many small doses or fractions to reduce side-effects. To ensure that the patient is reproducibly positioned in the X-ray beams, a close fitting acrylic mask including alignment markers is made for the patient's face, on a model cast from an impression.

The radiotherapy treatment plan is made on a computer, calculating the doses absorbed by the tissues in and around the tumour site. Radiation is delivered by one or more beams of radiation and metal wedges shield sensitive tissues such as the eyes. In the UK radiation is usually given at a rate of 1 fraction per weekday. Recently, hyperfractionation protocols (2 fractions per day) have been shown to be equally effective and have fewer side-effects. This patient will receive 66 Gy over 6.5 weeks.

■ *After completing treatment, what follow-up is required and why?*

The patient must be reviewed regularly for years. The greatest risk of recurrence is in the first 3 years and initially he will be seen every few weeks for a year, gradually extending review periods to a few months. As well as checking for recurrence and metastasis it must be remembered that all oral carcinoma patients have a 1 in 10 risk of developing a second primary carcinoma somewhere in the upper aerodigestive tract.

■ *What is the role of the dentist in care?*

The dentist in the hospital cancer team will have examined the patient at diagnosis and arranged for extractions of teeth of poor prognosis and restoration of caries. Teeth in the radiotherapy field used to be routinely extracted but are now often conserved because of advances in radiotherapy. There may also be a need for obturator or facial prosthesis postoperatively. Ideally the cancer team dentist would continue to care for the patient for a period after treatment.

General dental practitioners will care for patients in the longer term and need to provide intensive preventive advice, ensure oral health and prevent infection, particularly around teeth in irradiated bone. There is a need for continued surveillance of the mucosa for recurrence and new lesions and also to provide reassurance and support for patients and carers.

■ *After 21 months the patient develops a recurrence in the mouth. What can be done?*

If the recurrence is small and accessible it can be resected. It may be possible to provide radiotherapy but only if the site has not already been given a full course.

If no curative treatment can be provided, the patient will require palliative care. Palliative care is the control of symptoms, allowing the patient to end their life with dignity. The patient may be cared for at home by family, district or Macmillan nurses and the local hospice may coordinate treatment and organize short admissions to give some respite to carers. Pain is controlled with strong analgesics

49 A complicated extraction

Summary

A 35-year-old man attends your general dental practice surgery requesting extraction of a tooth.

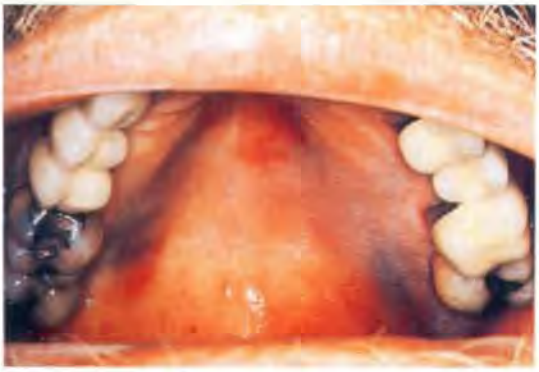


Fig. 49.1 The appearance of the patient's palate.

HISTORY

Complaint

He points to the lower left second premolar and says that the tooth is very tender to touch.

History of complaint

The tooth has been tender for some months and root canal treatment at another surgery was initially successful but has proved ineffective in the longer term.

Medical history

The patient's medical questionnaire indicates no relevant medical conditions.

EXAMINATION

Extraoral examination

The extraoral examination is normal except for a few palpable but normal-sized lymph nodes in his right and left cervical chain.

Intraoral examination

You immediately notice that the patient's oral mucosa is not normal. The appearance of the palate is shown in Figure 49.1.

The lower left second premolar has a large amalgam restoration and is tender to percussion. A sinus is present over the apex and the tooth does not respond to a test for vitality.

■ *What do you see in the patient's mouth?*

- The palate appears bruised with two purple-coloured lesions, one on each side of the palate extending from the gingival margin up the sides of the vault. Neither lesion appears to be raised above the surface.
- There is a discrete sharply defined slightly red patch in the anterior palate, just to the left of the midline.
- There are a few scattered red spots on the soft palate.

■ *What do these changes tell you?*

Individually none of these lesions can be diagnosed on the basis of the appearance alone. However in combination the appearances are almost diagnostic. The purple lesions appear vascular and could be haemangiomas or another blood vessel lesion including Kaposi sarcomas. The red patch has the characteristic appearance of erythematous/chronic atrophic candidosis. Both lesions are associated with immunosuppression, and you should immediately suspect HIV infection because Kaposi sarcoma is extremely rare with other causes of immunosuppression. In this clinical setting the palpable lymph nodes also support this diagnosis. It is almost certain that the patient is HIV-positive.

■ *What do you need to know? What would you say to the patient?*

You need to identify whether the patient is aware of his HIV infection but has chosen not to tell you the full medical history, or whether he is completely unaware of it. Almost all patients who know that they are HIV-positive will tell their dentist provided they are asked in an appropriate manner. Sometimes patients withhold the information because of previous insensitive management, a dentist having refused to provide treatment or because they are worried that practice confidentiality cannot be relied on. However, your questions need to be phrased to take account of the fact that the patient may be unaware of his HIV infection. It is very important that you approach this matter with sensitivity. You could adopt the following line of questioning.

- Tell the patient that he has some unusual signs in his mouth which you cannot easily explain. Ask whether he has noticed them.

50 Difficulty in opening the mouth

Summary

A 40-year-old Indian man presents to you in your general dental practice with limitation of mouth opening. You must identify the cause and institute appropriate follow up.

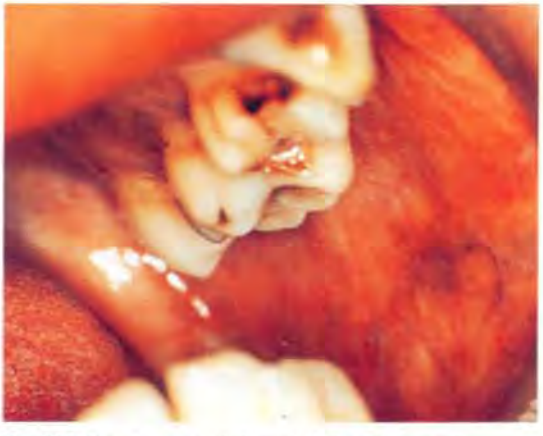


Fig. 50.1 Buccal mucosa, photographed at maximum mouth opening.

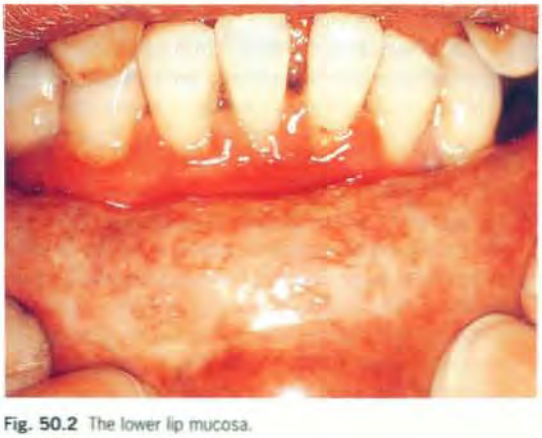


Fig. 50.2 The lower lip mucosa.

HISTORY

Complaint

The patient complains of difficulty in eating. He cannot open his mouth widely enough to place a proper mouthful of food inside and also has difficulty chewing.

History of complaint

He has noticed the reduction of his mouth opening over a period of several years but has never sought advice. It has not been painful though he has felt a burning sensation from his oral mucosa on eating during the same period. This varies in intensity.

Medical history

The patient is otherwise fit and well.

■ *What are the causes of limitation of mouth opening and how may they be classified?*

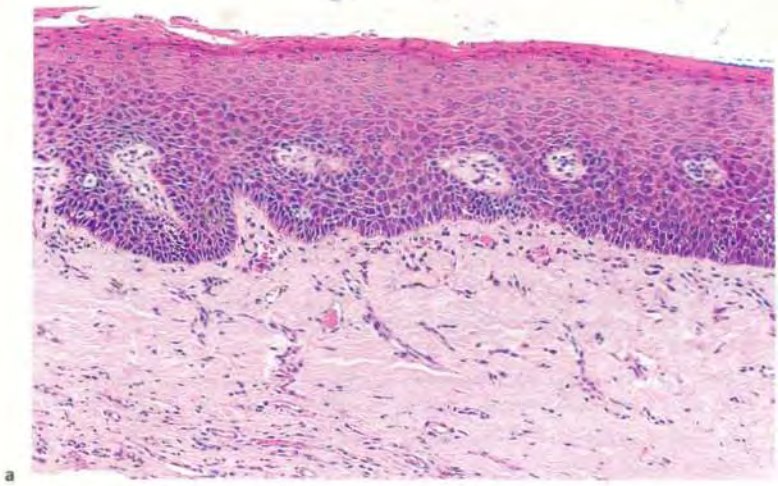
Limitation of opening is most frequently caused by trismus. By definition, trismus is caused by spasm of the muscles of mastication, though the term is often used loosely when opening is prevented by oedema or inflammation of the muscles or joint. Trismus is usually temporary.

Permanent limitation of opening may be caused by scarring of the soft tissues around the joint or mandible or by fusion of the condyle to the glenoid fossa (ankylosis). The causes may be divided as follows:

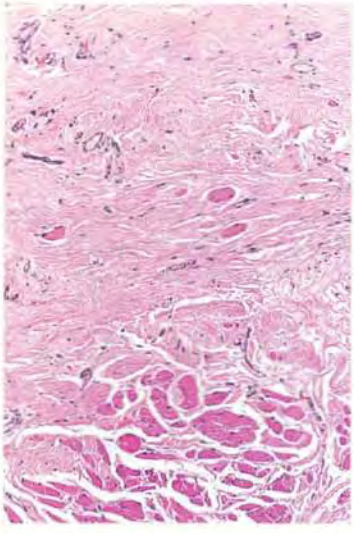
Trismus	Inflammation in and around the temporomandibular joint
	Trauma (fractures and/or soft tissue injury)
	Tetanus and tetany
	Temporomandibular joint (myofascial) pain dysfunction syndrome
	Soft tissue infection around the jaws or joint (usually dental in origin)
Permanent limitation of opening	*A. Extra-articular causes*
	Fibrosis due to burns or irradiation
	Oral submucous fibrosis
	Mucosal scarring (e.g. in epidermolysis bullosa)
	B. Intra-articular causes
	Congenital ankylosis
	Traumatic ankylosis
	Ankylosis following pyogenic arthritis
	Ankylosis following juvenile arthritis
	Neoplasms and other causes of enlargement of the condyle

■ *What questions would you ask?*

The patient should be asked whether there has been trauma or irradiation to the skull, temporomandibular joint or face and whether there have been any episodes of swelling of the face or around the joint. He should also be asked whether he uses betel quid (*pan* or *paan*).



a



b

Fig. 50.3 Biopsy from the quid site: **a,** buccal mucosa; **b,** underlying tissue.

with submucous fibrosis have oral white patches and dysplasia is present in the mucosa of up to 16%. Malignant transformation to squamous cell carcinoma occurs in between 5 and 8% of cases.

The second significant feature is the restricted opening. This is often progressive and responds poorly to treatment. In the late stages of disease the patient may be unable to open the mouth at all and incisor extractions may be required to allow feeding. Limited opening is a major handicap for diagnosis and treatment of malignancy and premalignant lesions. It makes examination, detection and treatment extremely difficult and the prognosis for oral carcinoma in a patient with submucous fibrosis is very poor, mostly as a result of late diagnosis.

INVESTIGATIONS

■ *What investigations are required? Explain why.*

Biopsy is required to assess dysplasia. If there are lesions suspicious of malignancy, red or white patches or areas of otherwise abnormal mucosa they should be sampled for microscopy. More than one biopsy may be required. If no particular part of the mucosa is suspect, a sample should be taken from the area where the quid is held in the mouth.

The biopsy will probably also provide evidence to support the diagnosis of oral submucous fibrosis. However, in such a typical case biopsy for this purpose alone would not be justified. It might be considered in an early case where the diagnosis is in doubt.

■ *Would you perform this patient's biopsy in a general practice setting?*

No. This patient is at high risk of developing an oral carcinoma and the biopsy and further recall should be carried out in a specialist centre. Further discussion about when to biopsy potentially dysplastic mucosa will be found in Case 38.

■ *A biopsy from the quid site is shown in Figure 50.3. What do you see and how do you interpret the findings?*

The mucosa is covered by epithelium which is atrophic and parakeratinized. The thickness of the normal buccal mucosa is about twice the thickness from the surface to the dermal papillae in this specimen. It is normally nonkeratinized except for a thin layer near the occlusal line. The epithelium is largely well organized and stratification and maturation are not particularly disordered; the epithelial

51 Tooth wear

Summary

A 35-year-old policeman presents having noticed that his anterior teeth are becoming shorter. Identify the cause and outline options for management.

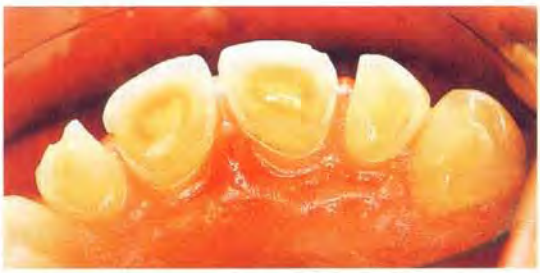


Fig. 51.1 Palatal view of the upper anterior teeth

HISTORY

Complaint

The patient has become increasingly aware of his shortening front teeth. He is not greatly concerned about the appearance but feels that continued wear will eventually destroy the teeth completely.

History of complaint

He has noticed that his teeth have become worse over the last 3–5 years but cannot remember when he first noticed the signs. The patient has always attended a dentist regularly and has relatively few, small restorations and good oral hygiene.

Medical history

The patient is generally fit and well. He drinks about 10–20 units of alcohol each week.

EXAMINATION

Extraoral examination

The patient is a fit-looking man and slightly overweight. No submandibular or cervical lymph nodes are palpable. The temporomandibular joints appear normal and there is no evidence of hypertrophy of the masseter muscles.

Intraoral examination

■ *The appearance of the anterior teeth is shown in Figure 51.1. What do you see?*

The palatal surfaces and incisal edges of the upper incisor teeth are worn. The wear involves the enamel and dentine but not the pulp. The palatal surfaces of the teeth appear smooth and unstained. The incisal edges are rough, small chips of unsupported labial enamel having fractured away.

If you were able to examine the patient, you would find that some of the upper and lower anterior teeth do not contact each other in the retruded contact and the intercuspal positions. All other teeth appear normal and the palatal surfaces of the upper posterior teeth are unaffected.

■ *What does this appearance signify?*

This is *tooth wear*, the loss of dental tissues through the processes of erosion, attrition and abrasion. Although each process may act alone, significant tooth wear is usually the result of a combination of these processes and erosion is often dominant.

The smooth surfaces suggest that erosion is a factor in this case and the distribution of enamel loss suggests that regurgitation of gastric acid may be the cause. Dietary acids are usually associated with erosion on the buccal or labial surfaces of the upper anterior teeth but if the patient rinses or swills acidic drinks in the palatal vault prior to swallowing, the pattern of erosion is very similar to that seen when gastric acid is regurgitated. Either source of acid might be the cause.

■ *Define erosion, abrasion and attrition.*

Erosion is the chemical dissolution of teeth by acids.

Attrition is the wear of tooth against tooth. Mild degrees of attrition are normal.

Abrasion is the wear of teeth by physical means other than the teeth.

DIFFERENTIAL DIAGNOSIS

■ *What is your differential diagnosis for this patient?*

1. Dental erosion caused by gastric acid combined with attrition
2. Dental erosion caused by dietary acids combined with attrition

52 Worn front teeth

Summary

A 60-year-old man presents at your general dental practice saying that his teeth have worn down. What is the cause and how should he be managed?

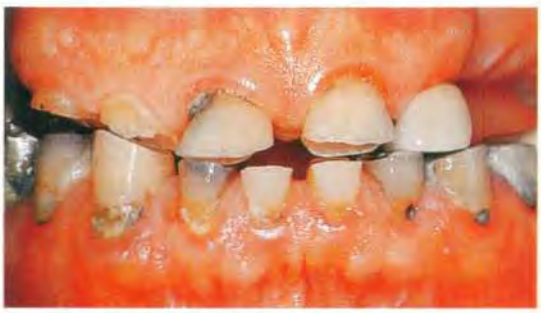


Fig. 52.1 The patient's appearance at presentation.

HISTORY

Complaint

The patient is unhappy about his short and discoloured upper anterior teeth. He is also finding that he has difficulty eating. The appearance of his teeth has recently become more important to him because he has taken a job in which he deals with the public. He wishes primarily to improve his appearance and appears to be sufficiently motivated to complete a course of complex dental treatment.

History of complaint

The patient has only recently started to attend his dentist regularly, after a 10-year period without treatment. Most of his posterior teeth were extracted before the age of 45 and he has worn his present upper partial denture for at least 12 years.

EXAMINATION

Extraoral examination

No submandibular or cervical lymph nodes are palpable, the temporomandibular joints appear normal and there is no tenderness around the muscles of mastication. Despite the anterior tooth wear there is no evidence of loss of occlusal vertical dimension.

Intraoral examination

The oral mucosa is healthy. In the mandible all teeth between the left and right second premolars are present but in the maxilla there are only five anterior teeth remaining. The appearances are shown in Figure 52.1

Wear of the incisal edges of upper and lower anterior teeth has produced short clinical crowns and the upper right lateral incisor and canine are worn almost to gingival level. There are deposits of plaque around the cervical margins of his teeth and a number of teeth have cervical caries. Despite the plaque, there is only occasional interdental bleeding on probing and no significant increase in pocket depths. The upper ridges are not extensively resorbed and are broad and well defined. When asked to bite his teeth together the patient adopts the forward mandibular posture shown in Figure 52.1.

Both lower lateral incisors and the lower left canine and first premolar appear discoloured. Only the lower left second premolar fails to respond to an electronic test of vitality.

The patient produces his acrylic upper partial denture from his pocket. It is poorly retentive and of indifferent fit.

■ ***What is your differential diagnosis? What features suggest each possibility?***

The patient is suffering tooth wear. This is usually caused by a combination of three basic underlying processes: erosion, attrition and abrasion. In this case it seems likely that the cause is predominantly erosion with attrition as a secondary factor.

Aetiological factor	Features
Erosion	Erosion is usually caused by excessive dietary acid or regurgitation. Both possibilities must be excluded by careful questioning and dietary analysis (see also Case 51). The appearance of the wear facets suggests erosion as the major cause. Although the teeth interdigitate on incisal enamel, the dentine has been lost from an area which is not in contact with the opposing teeth.
Attrition	Attrition is usually caused by occlusal wear and a minor degree is normal. Bruxism and other parafunctional habits may have caused increased attrition. There is no evidence of masticatory muscle tenderness or hypertrophy to suggest that such habits contribute.

■ *Which approach is necessary in this case and how would you manage the vertical dimension?*

In this case a reorganized approach should be adopted. This is because the crowns of the unrestored teeth are very short. To crown these teeth the natural crown would need to be reduced in height to provide occlusal clearance and this would result in shorter unretentive preparations. It is therefore necessary to gain space for the new tooth height.

■ *How might this problem be overcome?*

Surgical crown lengthening with alveolar bone remodelling can be used to overcome this problem. The gingival margin is repositioned apically to create a longer tooth, shown in Figure 52.2, which can be prepared for a crown in the conventional manner, shown in Figure 52.3. Crown lengthening does not alter the occlusal vertical dimension because the additional crown length needed for preparation is obtained by exposing the root. Surgical crown lengthening is preferable to electrosurgery which merely alters the gingival contour.

The alternative is to accept the height of the teeth and the gingival contour and create vertical space by adding to the incisal height of the artificial crowns.

In this case the teeth are so short that a combination of both techniques is required.

■ *How is surgical crown lengthening achieved?*

Flaps are raised buccally and palatally and crestal bone is removed with a bur. Both the height of the bone and its width must be adapted, remodelling the alveolar contour so that the soft tissues will return to their new apical position but be able to retain the normal shape of the gingival margin. Bone is removed palatally and buccally. The amount removed must be judged so that sufficient crown length is produced to allow a retentive preparation but support from the root is not compromised. The optimum distance from the crest of the alveolar bone to the gingival margin is 3–4 mm. Sufficient bone must be removed to preserve this distance or the gingival tissues will regrow to their original position.

■ *What are the disadvantages of crown lengthening?*

- Crown lengthening results in the crown margins lying on the root surfaces of the teeth. The cross-sectional area of the root is smaller than the crown so that the preparation is rather tall and narrow (and therefore weak) and the final restoration is more triangular in shape (as in the provisional restorations in Figure 52.3).
- Some patients develop significant sensitivity from the exposed dentine.
- Part of the periodontium is removed. The support of teeth with short roots may be compromised as a result.
- The procedure is uncomfortable for the patient and time is needed for healing and for the new gingival contour to stabilize.

■ *Are there any alternatives to crown lengthening?*

An alternative approach to surgical crown lengthening would be to accept the clinical crown height but gain additional retention by placing a post in the root canal. However, elective root treatment should be avoided whenever possible in cases of tooth wear. This is especially so when there is a significant element of attrition, for instance from a parafunctional habit. The additional occlusal loading may result in decementation of the post or fracture of the root.

Secondary dentine formation below the wear may also complicate root treatment by causing sclerosis of the canal.

■ *The patient clearly dislikes his acrylic partial denture. Will restoration require a replacement?*

Ideally, yes. When the anterior teeth are restored the patient will need sufficient occlusal table posteriorly to masticate effectively.

In the short term an upper partial denture is required preferably a tooth-supported chrome–cobalt denture. In the longer term other options such as an implant-retained bridge might be considered. However, such complex treatment should not be provided until caries activity has been controlled.

TREATMENT

In this case crown-lengthening surgery was performed on all upper and lower teeth and the vertical dimension was increased to provide additional space for the new crowns. The effect of crown lengthening is shown in Figure 52.2 and the provisonal restorations in Figure 52.3.

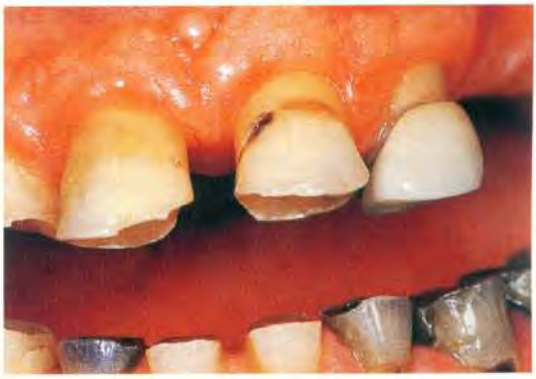


Fig. 52.2 The upper anterior teeth following surgical crown lengthening.

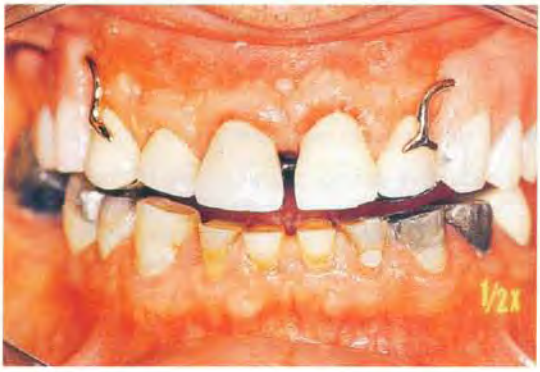


Fig. 52.3 Provisional restorations after crown lengthening.

■ *How should the stages of treatment be organized into a treatment plan culminating in the permanent restoration? Why is each stage required?*

See Table 52.1.

■ *How would your treatment differ for the patient shown in Figure 52.4 who has a complete dentition?*

When more teeth are present the extra vertical space needed to make the crowns can be created orthodontically. An anterior bite plane (Dahl appliance) is cemented to the teeth with a relatively weak cement such as a glass ionomer. This allows the posterior teeth to overerupt and also intrudes the anterior teeth so that tooth movement rather than tooth reduction provides the crown length necessary for retentive crown preparations. The appliance is worn for about 3–6 months, depending on the rate of eruption. The patient's appliance is shown in Figure 52.5 holding the posterior teeth apart at the start of treatment.

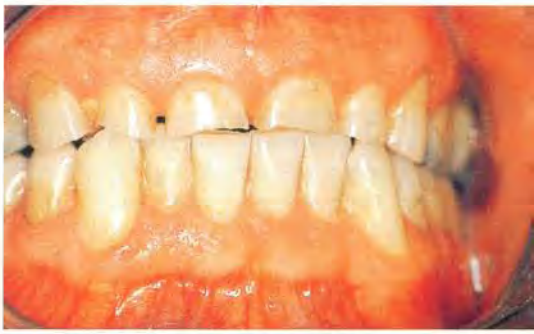


Fig. 52.4 Another patient with marked upper anterior tooth wear.

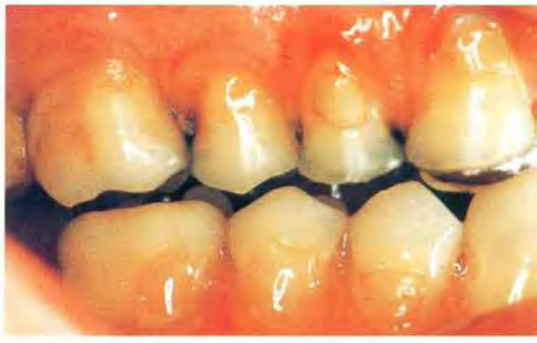


Fig. 52.5 Posterior teeth immediately after insertion of an anterior bite plane.

Table 52.1 Stages of treatment plan

Stage	Reason
Diagnostic wax-up	To show the patient the eventual shape and relationships of the planned crowns. It can also be used to produce the provisional restorations.
Crown-lengthening surgery in upper arch	To gain length for retention. Usually, the upper arch is treated first because it is technically easier. In addition the upper provisional crowns establish the new anterior guidance which can be copied in the definitive restorations once the patient is comfortable.
Healing period	The time delay between the periodontal surgery and placement of provisional crowns should be in the order of a few weeks as there is some evidence that the tissues can heal back towards their original position. Definitive crowns can be made once gingival contour is stable, around 3–4 months post surgery.
Provisional restorations, upper arch	These are made shortly after the crown lengthening and the anterior guidance is adjusted so that the patient is comfortable. The restorations should be worn for at least a few weeks to assess the patient's compliance (Fig. 52.3).
New denture and definitive upper restoration	The new crowns are made first and then the denture around them. Some clinicians will make the crowns, try them in and recement the provisional restoration. This allows the denture to be constructed to fit the final crowns in the laboratory. Others prefer to cement the new crowns in place and take a new impression to construct the denture.
Assessment period	Allows time for the patient to decide whether they wish to have the lower arch restored.
Lower arch crown lengthening and new crowns	The same process is used in the lower arch once the surgical procedure is completed. It is unlikely that the patient needs lower teeth posterior to the second premolars.

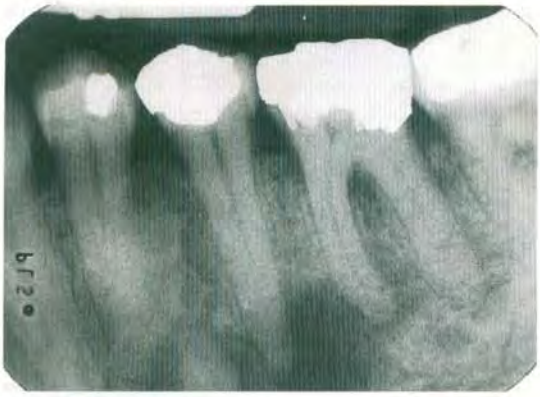


Fig. 53.1 Periapical of both lower premolars and first molar.

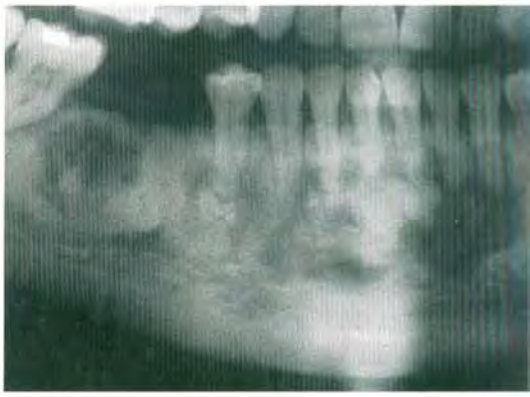


Fig. 53.2 Section from the dental panoramic tomograph.

■ *The periapical radiograph is shown in Figure 53.1. What do you see?*

The periapical radiograph shows:

- A small restoration in the first premolar.
- Large restorations in the second premolar and first and second molars.
- No radiographic evidence of dental caries.
- Early bifurcation bone loss associated with the first molar.
- A radiolucent area centred on the apex of second premolar which appears to extend to involve the mesial root of the first molar.
- Loss of lamina dura around the apex of the root of the second premolar and the first molar mesial root.
- An irregular but relatively well-defined radiopaque zone distal to the first premolar root.

■ *What would you do next and why?*

Further radiographic views are required. The radiograph has not aided diagnosis of the dental pain as no unsuspected cause for the pulpitis has been identified. However it has revealed an apical radiolucency on the second premolar and first molar which is not compatible with an uncomplicated periapical granuloma, infection or cyst. The presence of an apical radiolucency on the second premolar is also incompatible with the history and examination which indicate that this tooth is vital.

The presence of both radiopacity and radiolucency requires consideration of a wider differential diagnosis which would include fibro-cemento-osseous lesions, odontogenic tumours and a variety of bone disorders. The margins of the radiolucent lesion are not visible in the film and need to be defined before a more accurate differential diagnosis can be proposed. Because some fibro-cemento-osseous lesions may be bilateral, appropriate views would be a dental panoramic tomograph or right and left oblique laterals. These will also allow all the teeth and their supporting structures to be assessed because the patient is being seen in the practice for the first time. Bitewings to assess caries would also be appropriate in a new patient with several heavily restored teeth if there is clinical suspicion of caries.

■ *Part of the dental panoramic tomograph is shown in Figure 53.2. What do you see?*

The additional radiograph shows several features including:

- The lower right second premolar and first molar are absent, presumed extracted.
- A small occlusal restoration in the lower left second molar which has tipped slightly mesially.
- An extensive lesion of mixed radiodensity involving the central body of the mandible from the mesial root of the second molar across the midline to join that shown previously in the left.
- The lesion appears to be composed of several radiolucencies often with a central opacity centred on the root apices.
- There is little or no expansion of the bone despite the extensive lesion.
- The lesion has not displaced teeth or inferior dental nerve canal.
- There are no lesions in the maxilla (not seen in figure).

DIAGNOSIS

■ *What are the causes of a mixed radiolucency such as this in the jaws?*

- Cemento-osseous dysplasias
  - — periapical
  - — focal
  - — florid
- Chronic osteomyelitis

- Paget's disease of bone
- Fibrous dysplasia
- Metastatic malignancy

■ *What is the most likely diagnosis? Explain why.*

One of the cemento-osseous dysplasias is the cause of the patient's jaw lesions. The diagnosis may be made on the radiographic appearances alone. No other condition produces multiple lesions centred on the apices of the teeth, each with a central radiopacity and a variable and poorly defined radiolucent rim. As disease progresses this pattern may become less distinct, but it is clearly visible in several of this patient's lesions. This patient has the florid form of the disease in which one or more quadrants are affected. The periapical form affects a few teeth, usually the lower incisors, and the focal form gives rise to one large lesion but all are part of a spectrum of disease severity. The diagnosis is supported by the patient's race, these conditions being more prevalent in those of negroid or mongoloid racial stock. The lesion(s) are normally asymptomatic.

■ *What diagnoses have you excluded? Explain why.*

Chronic osteomyelitis produces a patchy mixed radiolucency but would give symptoms of dull boring central bone pain quite distinct from those reported. Sinuses or other signs of infection would probably be present. However, this diagnosis should not be completely excluded without a further consideration, because the sclerotic bone of fibro-cemento-osseous lesions such as florid cemento-osseous dysplasia is prone to infection, particularly dental infection, and in the past the condition was thought to be a form of osteomyelitis. A biopsy to confirm the presumed diagnosis is contraindicated because of the risk of osteomyelitis.

Paget's disease of bone may be confidently excluded because it almost never affects the mandible without producing obvious lesions, signs and symptoms in other bones. If this were Paget's disease the maxilla would be affected. Paget's disease affects predominantly elderly Caucasians.

Fibrous dysplasia might be considered as a cause of patchy and poorly defined radiolucency but presents with expansion of the jaw, usually the maxilla, during the first or second decade.

Metastatic malignancy might also be considered as a further cause. Most cancers cause purely radiolucent lesions but some, notably prostate and breast, may cause bony sclerosis and radiopacity. However the site is usually at the angle of the mandible, and the radiological appearances are sufficiently characteristic of florid cemento-osseous dysplasia to exclude this sinister diagnosis.

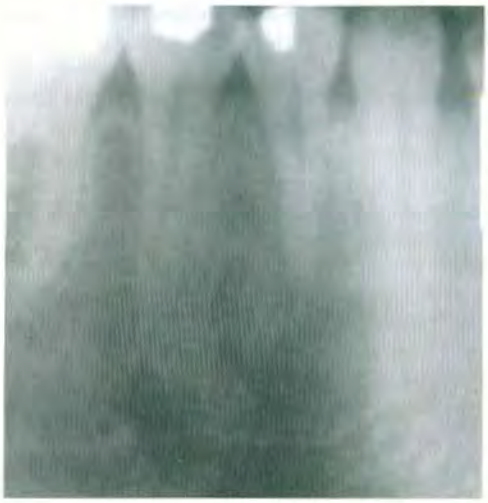


Fig. 53.3 Section from the dental panoramic tomograph taken 11 years previously.

■ *How might you confirm the diagnosis without biopsy?*

Any previous radiographs should be reviewed to determine whether the lesion has been present and slowly progressing for several years. This would confirm the diagnosis. A previous dental practitioner was contacted and provided the radiograph seen in Figure 53.3, which had been taken 11 years previously.

The radiograph shows the lower left quadrant. The lower left second premolar and first molar contain smaller restorations than at present and there is probable caries in the second premolar. However the first premolar appears to contain the same restoration as at present, and at its apex there is a lesion typical of early cemento-osseous dysplasia comprising a radiolucency with a central opacity at the root apex. This early stage of the lesion provides conclusive evidence for the proposed diagnosis.

■ *What would you do about the patient's pain?*

The causative tooth must be identified. Vitality must be accurately established and the most effective way to do this is to perform a 'test cavity' in the first molar without local anaesthetic. When this was done the tooth was found to be nonvital and the second premolar was found to be vital. The first molar requires root treatment or extraction. The apical radioluceny on the second premolar is an early radiolucent lesion of cemento-osseous dysplasia and some years later radiographs revealed that the lesion had developed a zone of radiopacity centrally.

54 A child with a swollen face

Summary

A-5-year-old boy has painless bilateral facial swellings. Identify the cause and recommend treatment.

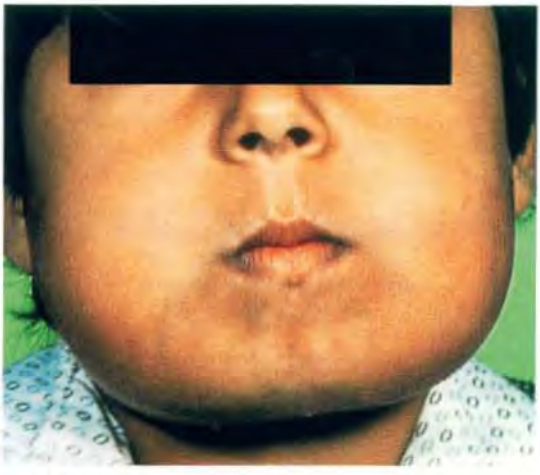


Fig. 54.1 The patient's appearance at presentation.

HISTORY

Complaint

The patient is brought by his parents who have noticed that his face has become fat. They are concerned about his appearance and say that he is being teased and bullied at school.

History of complaint

His parents say that the patient has had a chubby face since he was a toddler but that the swelling has become more noticeable over the last 2 years. He is in no pain.

Medical history

He is otherwise fit and well, has had all recommended immunizations and amongst the childhood illnesses has suffered only chicken pox. His medical practitioner has given him a general examination and found no systemic illness but has referred him to you for a further opinion.

EXAMINATION

Extraoral examination

The appearance of the child is shown in Figure 54.1. He appears healthy but has obvious bilateral enlargement of the side of the face. The temporomandibular joints appear normal on palpation. Some upper deep cervical lymph nodes are palpable bilaterally. They are only slightly enlarged, not tender and are freely mobile.

■ ***On the basis of what you know, what types of lesion would you consider?***

From this view alone it is difficult to tell whether the swelling originates in the salivary glands, mandible or soft tissues. Each site would have different possible causes:

Condition	Possible causes
Soft tissue enlargement	Masseteric hypertrophy is possible. Bruxism is common in children though significant masseteric hypertrophy is rare.
Salivary gland enlargement	Rare in children. HIV salivary cystic disease is seen in HIV infection. Mumps can be excluded. Mumps is acute and, in addition, the child would have had mumps vaccine with the rest of the routine childhood vaccinations.
Enlargement of the mandible	A few rare inherited disorders of bone could cause bilateral expansion of the ramus.
A developmental syndrome	Many syndromes have craniofacial signs and this is a possibility which should be borne in mind. There appear to be no associated features.

Intraoral examination

Intraoral examination reveals a minimally restored dentition and healthy oral mucosa. Palpation of the mandibular rami shows that they are the source of the enlargement. There is obvious rounded swelling of the posterior body and ramus of the mandible. The lower right second deciduous molar is missing.

INVESTIGATIONS

■ ***A radiograph is obviously required. Which view(s) would you choose?***

A dental panoramic tomograph is the investigation of choice as an initial view. The whole of the swellings will be

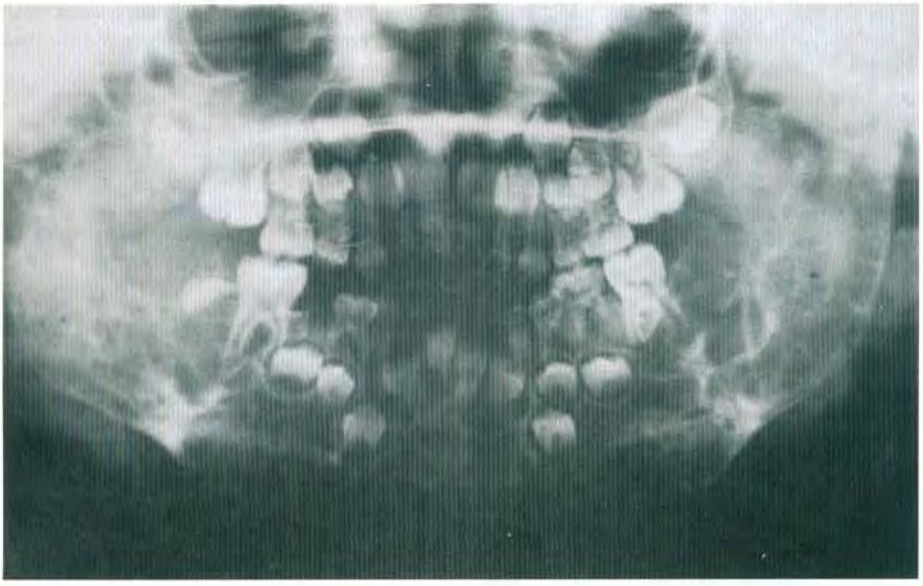


Fig. 54.2 Dental panoramic tomograph.

visible and the left and right can be easily compared. A posterior–anterior view of the jaws would also be useful, providing a second view at right angles to the ramus in the panoramic view. It would allow mediolateral expansion to be assessed.

■ *The radiographic appearance is shown in Figure 54.2 What are the radiographic features of the lesions?*

See Table 54.1

Table 54.1 Radiographic features

Site	Bilaterally in the posterior body, angle and rami of the mandible.
Size	Relatively large, about 5 × 8 cm.
Shape	Lesions on both sides are multilocular.
Type of outline/edge	Smooth, well defined and well corticated.
Relative radiodensity	Radiolucent with internal radiopaque septa producing a multilocular appearance. There are no dense radiopaque inclusions.
Effects on adjacent structures	Gross displacement of the developing permanent second molars. The lower right second primary molar has been lost, presumably by exfoliation. There has been extensive expansion of the height of the body of the mandible. The condyles are not affected.

DIFFERENTIAL DIAGNOSIS

■ *Give a differential diagnosis. Explain which is the most likely cause and why.*

Only a very short differential diagnosis is possible for this case.

Diagnosis	Similarity to present case
Cherubism	Causes bilateral radiolucencies in the mandibular rami and maxilla. Enlargement starts in children before the age of 5 years. The lesions appear multilocular and radiolucent and disrupt the dentition. The radiographic and facial appearances in this case are characteristic.
Other possible causes	There are a few very rare bone diseases and syndromes which may need to be considered if the most likely diagnosis of cherubism cannot be confirmed. Almost all other causes have prominent signs elsewhere in the body and none has been noted in this case.

■ *What further questions might help confirm your diagnosis?*

Did either parent have a similar problem? Cherubism is inherited in an autosomal dominant fashion. Radiographs of both parents may reveal unsuspected healed lesions and this would aid diagnosis.

Are any brothers or sisters affected? For similar reasons, siblings would be expected to show similar signs.

How was the lower second deciduous molar lost? Cherubism may cause early exfoliation of teeth.

■ ***Would any further radiographs help confirm the diagnosis?***

More detailed radiographic examination with intraoral films would be helpful for the following reasons.

- To demonstrate involvement of the maxilla. More severely affected patients usually have lesions in the maxilla, usually centred on the tuberosity but sometimes extending to distort the orbit. These can easily be missed on extraoral films but, if present, confirm the diagnosis.
- To identify displacement or destruction of teeth. As noted above, cherubism often destroys tooth germs and displaces teeth.

■ ***Is a biopsy required?***

In a classical case of cherubism, the diagnosis may be made with certainty on the basis of family history, clinical and radiographic features. In a new case such as this, or if there were no family history, it would be prudent to confirm that the lesions are histologically compatible with cherubism.

■ ***A biospy specimen was removed from the expanded alveolar ridge. The histological appearances are shown in Figure 54.3. What do you see?***

The lesion is composed of cellular fibrous tissue which appears loose and oedematous with spaces rather than dense collagen between the cells. Scattered in the fibrous tissue are multinucleate giant cells. These are relatively small giant cells and have only 4–8 nuclei each.

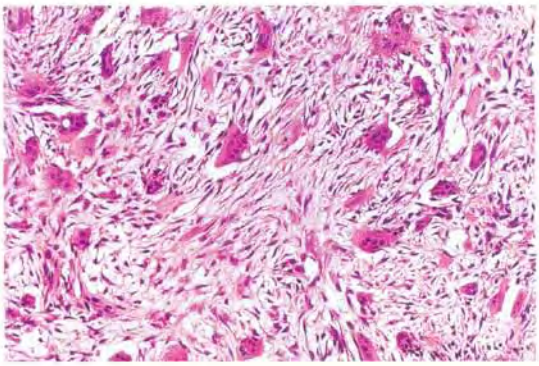


Fig. 54.3 The histological appearance of the biopsy specimen.

■ ***How do you interpret these appearances? Are they consistent with cherubism?***

Lesions with many giant cells fall into two broad categories, those with granulomas, such as tuberculosis, sarcoidosis and foreign body reactions, and the *giant-cell lesions*. No granulomas are present and these appearances indicate a giant-cell lesion, the causes of which are:

- central giant-cell granuloma
- brown tumour of hyperparathyroidism
- aneurysmal bone cyst
- cherubism.

These conditions cannot be distinguished from one another on histological grounds alone. However, the only one which matches the clinical and radiographic findings is cherubism.

DIAGNOSIS

Taken together, the evidence supports a diagnosis of cherubism and this is a typical case.

AETIOLOGY

■ ***What is the cause of cherubism?***

Cherubism is caused by any one of several mutations in the gene for SH3BP2, a regulator of the C-Abl oncogene, a poorly understood signalling molecule involved in regulation of cell division and many other cell functions.

The condition is usually inherited in an autosomal dominant fashion. It would be expected that one parent would be similarly affected. Females are often less severely affected and cases may appear to be sporadic.

TREATMENT

■ ***What treatment would you recommend? What other advice would you give to the parents?***

No treatment is required though the parents and child may need reassurance. The parents can be told that lesions of cherubism usually grow fastest before the age of 5. Although there will be further growth during the next few years, the lesions will stop growing spontaneously and start to regress around the age of puberty. The swelling should have completely resolved by the age of 25 and only radiographic changes will remain into the fourth decade.

Surgical intervention is not usually necessary but may be performed for cosmetic reasons if lesions resolve slowly. Some teeth will be lost through the disease process. The parents should also be warned that future children and siblings are likely to be affected. Genetic counselling would be appropriate.

55 A pain in the neck

Summary

A 51-year-old man has a recurrent swelling in his neck at mealtimes. What is the cause and how may he be treated?

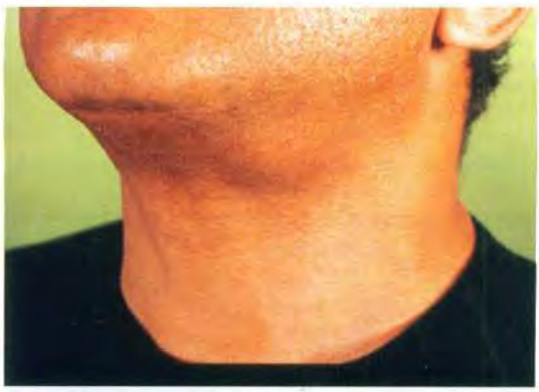


Fig. 55.1 The patient on presentation.

HISTORY

Complaint

The patient has a swelling in the left neck, below the angle of the jaw in the submandibular region. It appears suddenly on eating and is painful, lasts 1 or 2 hours and then slowly subsides. He reports that the swellings are worse when eating citrus fruit. The swelling is still present following his last meal.

History of complaint

The swelling first appeared suddenly during a meal 4 months ago. It resolved within 2 hours but recurred once during the following week. In the following weeks the swelling appeared more frequently and then, on one occasion stayed swollen for 3 days. This swelling appeared different; the swelling was warm to touch and more painful. He consulted his medical practitioner and was prescribed amoxicillin and the swelling resolved shortly afterwards. After that episode the recurrent swelling did not appear for nearly 2 months but it now appears daily. The patient's medical practitioner has suggested that he see a dentist to exclude a dental cause for the swelling.

Medical history

The patient is an otherwise fit and healthy man.

■ *How do you interpret the information you have so far?*

The patient is almost certainly suffering from the symptom known as *mealtime syndrome*, swelling of a salivary gland associated with eating or the thought of eating. Provided the swelling is in the correct site to be a salivary gland, this will be the underlying process. The cause of mealtime syndrome is obstruction. Between meals the low flow of saliva leaks around a partial obstruction. When flow is stimulated the saliva is held back, causing the gland to swell and inducing pain because the gland is restricted within a capsule.

This accounts for the recurrent swelling but the patient's episode of persistent swelling appears to be infectious. The classical signs of inflammation were present and the swelling resolved on antibiotics (though the causative link between the resolution and antibiotic is conjectural). When salivary flow is reduced the gland is prone to ascending infection by oral commensal bacteria and opportunistic pathogens so the two causes of swelling could well be linked.

■ *Are there any specific questions you would ask? Why?*

Whether the patient has noticed any abnormality of salivary flow such as a foul tasting saliva or sudden resolution of the swelling following a sudden discharge of saliva or salty fluid into the mouth. The first would suggest infection and the second would confirm intermittent obstruction.

EXAMINATION

Extraoral examination

■ *The appearance of the swelling is shown in Figure 55.1. What do you see?*

The patient has a round swelling of approximately 3 cm in diameter in the region of the left submandibular gland, below the lower border of the mandible on the left side and just anterior to the angle of the mandible. If you were able to palpate the neck you would feel no cervical lymphadenopathy and find that the swelling is firm, mobile and tender.

Intraoral examination

■ *What features of the intraoral examination are important?*

Bimanual palpation of the submandibular gland will allow the gland to be steadied for examination. Palpation from only one side displaces the mobile gland, making differentiation of the gland from an enlarged lymph node or swelling of the gland from swelling *in* the gland difficult.

Expression of saliva from the salivary gland ducts. Massaging the duct should produce a free flow of clear saliva. Cloudy saliva indicates infection or inflammation. No saliva may indicate reduced secretion or blockage. Pus indicates infection and a sample should be taken for culture and sensitivity.

When you examine the patient you discover that the oral mucosa and dentition appear normal. A small amount of clear saliva can be expressed from the orifice of the left submandibular duct. Bimanual palpation reveals a 3 cm diameter hard mass in the floor of mouth near the posterior free border of mylohyoid muscle. The mass is freely mobile vertically and appears to be the submandibular gland.

DIAGNOSIS

The patient has mealtime syndrome and probably obstruction. The gland is hard because of recurrent sialadenitis, a result of ascending low-grade infection.

■ *What are the possible causes of obstruction?*

Type	Cause
Intraductal causes (common)	Sialolithiasis (stone) Mucous plug Stricture
Extraductal causes (rare)	Trauma Pressure from adjacent neoplasm or other swelling

■ *Are there any other causes of intermittent swelling that need to be considered?*

Yes. Intermittent swelling is associated with overvigorous rinsing (particularly with chlorhexidine) or in wind instrument players who force air into the gland. A number of other chronic conditions that cause persistent swelling may cause intermittent swelling in their early stages. These include sialadenitis (usually the result of obstruction), primary Sjögren's syndrome, sialosis, drug-induced salivary gland swelling (caused by iodine-containing compounds, phenylbutazone and other drugs). However, this last group do not cause such obvious and short-lived swelling as obstruction.

INVESTIGATIONS

■ *What investigations are required? Why?*

Investigations are required to plan treatment rather than confirm the diagnosis. The information required is the cause of the blockage, the site of the blockage and the degree of damage sustained by the gland and ducts from sialadenitis. Useful investigations are shown in Table 55.1.

In this case a plain film and an ultrasound scan were performed as initial investigations.

■ *The plain film is shown in Figure 55.2. What do you see?*

The plain film shows a radiopacity in the region of the left submandibular gland hilum. The stone is outlined in Figure 55.9.

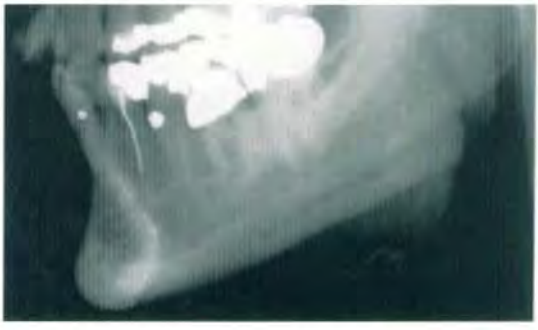


Fig. 55.2 Lateral radiographic view at presentation.

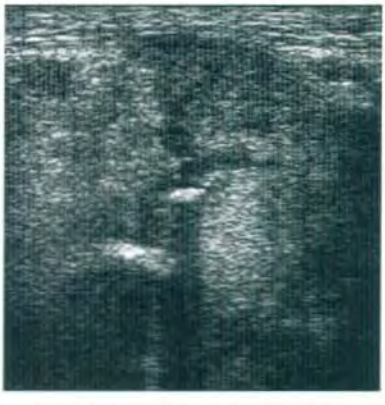


Fig. 55.3 Ultrasound scan performed at presentation.

Table 55.1 Investigations for salivary obstruction

Investigation	Rationale and role
Plain radiograph	Simple to perform, suitable for detection of radiopaque stones in ducts or the submandibular gland; a lower true occlusal film for the submandibular duct or a lateral view of the upper neck, oblique lateral or panoramic tomogram for the gland itself. The parotid duct may be visualized in a posteroanterior jaws view with the cheek blown out. Useful for submandibular calculi because 60–80% are radiopaque but of less value in the parotid where only 20–40% of calculi are radiopaque and other tissues are superimposed on the image. Radiolucent 'stones' are unmineralized or poorly mineralized masses of inspissated mucin.
Sialogram	Usually the most useful investigation but more complex, requiring cannulation of the duct. Helpful because it shows: • whether the obstruction is due to a stone or stricture • the size and position of a stone or stricture • the diameter and length of the duct distal to the obstruction, down which the stone or stone pieces will have to pass if the stone can be disrupted • the degree of disruption of the duct system in the gland, which reflects the damage and degeneration present • an estimate of salivary flow if an emptying film is taken to show how rapidly the contrast is expelled. Sialograms are performed by passing radiopaque contrast media into the duct. The commonly used materials contain iodine and are contraindicated in those with iodine hypersensitivity.
Ultrasound	Ultrasound is a noninvasive, harmless investigation useful to show the gland parenchyma and stones over 2 mm diameter. It is relatively quick and sometimes useful to distinguish stones and sialadenitis from enlarged lymph nodes. It is not as good as sialography for demonstrating duct architecture.
Computerised tomography	Particularly sensitive for detecting small calculi, 10 times more sensitive to calcification than plain film. However, not good for assessing the fine detail of duct morphology and so not helpful in assessing the degree of damage in the gland.
Magnetic resonance imaging	Gives excellent soft tissue contrast and is the technique of choice for examining the possibility of a neoplasm within the gland and to differentiate gland from enlarged lymph nodes.
Radioisotope scan	Indicates salivary gland activity but has low resolution and cannot detect stones or the anatomy of an abnormality. Used mainly to investigate xerostomia.
Salivary endoscopy	New technique available in only a few centres. Uses ultrafine endoscopes between 0.9 and 1.3 mm diameter that can be passed down the salivary duct accompanied by saline irrigation. This allows direct vision of the duct lumen and is used to diagnose stones, strictures and inflammatory conditions.

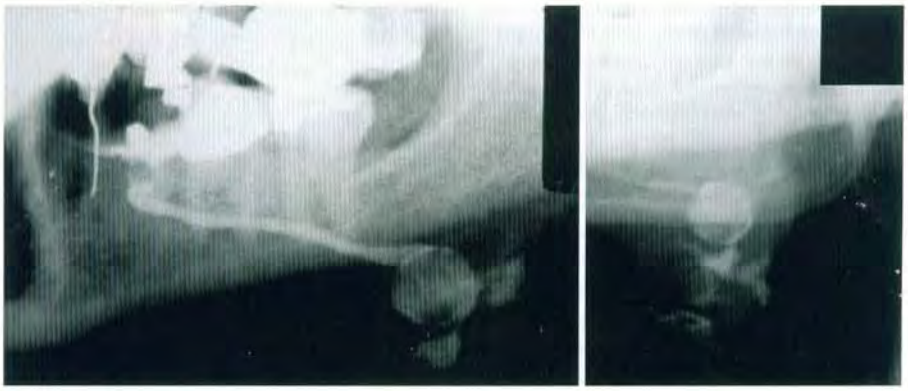


Fig. 55.4 Sialogram. On the left immediately after filling, on the right 15 minutes later.

■ ***The ultrasound is shown in Figure 55.3. What do you see?***

Interpreting a single ultrasound picture is difficult, the investigation is dynamic and the ultrasound operator may observe the screen for several minutes, altering the angle of the ultrasound transducer (transmitter/detector) to provide views at different angles. Ultrasound scans are conventionally orientated with the surface of the patient (ultrasound transducer) at the top. Objects that reflect ultrasound, such as stones, appear white and cast a dark *acoustic shadow* below them. Dilated ducts proximal to the

Technique	Details, indications and contraindications
Extracorporeal lithotripsy	Ultrasonic waves are directed at the stone through the soft tissues of the neck to shatter the stone. The pieces should then pass down the duct and be expelled in saliva. Suitable for stones up to 7 mm in diameter. May require one long or several short visits to reduce the whole stone to fragments that can be passed in saliva. Successful in one-third of suitable cases, (see Fig. 55.5).
Radiologically guided interventional techniques such as Dormia basket extraction of stones	A Dormia basket or balloon catheter can be passed into the salivary duct under X-ray guidance (see Fig. 55.6). Baskets are passed alongside the stone, opened to trap it and then closed around it for withdrawal. Quite large stones can be manoeuvred into a basket but the size of the duct will determine whether or not it can be withdrawn. An incision can be made to allow final delivery into the mouth. In practice stones up to 10 mm diameter are suitable for basket retrieval. Stones must be mobile in the duct for basket removal. Successful in 75% of suitable cases. A balloon can be passed into a stricture and then inflated to dilate it.
Endoscopically guided interventional techniques including intracorporeal lithotripsy	Ultrafine endoscopes are inserted into the duct using saline to expand the duct as the endoscope is slid forwards. Baskets and balloon catheters can be passed along a hollow 'working channel' in the endoscope endoscope to remove stones or dilate strictures under direct vision. Time consuming and has variable success. In intracorporeal lithotripsy the endoscope can be used to deliver ultrasound from a miniature ultrasound tip. Success claimed in up to two-thirds of cases but a relatively new technique. Risk of perforating the duct. Laser light may also be directed down the endoscope to shatter stones.
Intraoral surgery to the hilum of the submandibular gland	For larger stones located at the entrance to the hilum of the submandibular gland, an intraoral surgical approach may be made, carefully dissecting out the stones from the gland while identifying and protecting the adjacent lingual nerve. The stone must be palpable intraorally. High success rate (95%) but only for submandibular gland and has the major advantage that local anaesthesia can be used. A general anaesthetic would be required for gland removal.

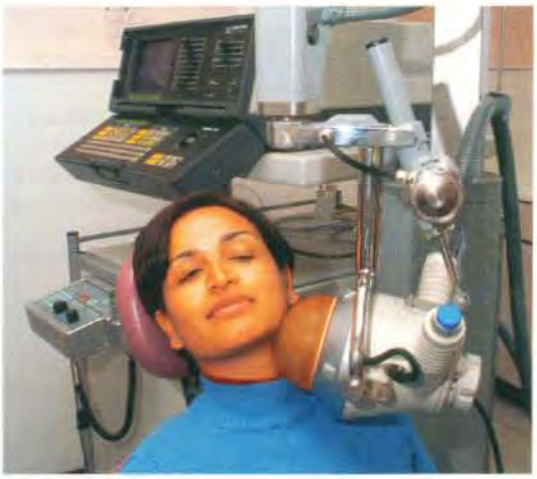


Fig. 55.5 Patient undergoing extracorporeal lithotripsy. The stone is visualised on the ultrasound scanner in the background. Aiming marks on the scan are used to align the ultrasound waves emitted by the transducer head, which is kept in close contact with the skin and as close to the stone as possible.

■ ***Is the present case suitable for treatment by minimally invasive techniques?***

Yes, but surgery cannot be completely avoided. The stone is more than 7 mm in diameter and so is unsuitable for extracorporeal lithotripsy. It is not mobile in the duct and so is unsuitable for basket removal. However, minimal surgery is possible provided the stone can be palpated under the tongue and an intraoral surgical removal is possible. This was carried out successfully for this patient under local analgesia.

ANOTHER POSSIBILITY

■ ***If the obstruction had been caused by a stricture, how would your management have differed?***

Most strictures are caused by fibrosis around stones, though the stones may be passed spontaneously leaving only the stricture. The duct proximal to a stricture often dilates and a series of strictures and dilatations gives rise to the 'string of sausages' or 'string of beads' appearance on a sialogram. This appearance indicates inflammation around the duct or *sialodochitis*.

Strictures located at or near a duct orifice may be dilated by introduction of graded lacrymal duct dilators. The more proximally located stones and strictures are inaccessible and present a more difficult management problem – traditionally treated by surgical resection of the gland, but more recently by dilatation of strictures by balloon catheter. This is often successful in the short term but in the long term the strictures often relapse.

■ ***The submandibular gland from another patient with sialolithiasis is shown in Figure 55.7. What do you see?***

The gland has been sliced along its long axis and opened to reveal a stone in the hilum. The stone is in a cavity with a smooth lining, a dilated duct, seen around the lower left

Fig. 55.6 Ultrafine endoscope for salivary gland endoscopy (left) and Dormia baskets (right). Inset, the basket open to allow the stone to pass between the wires and closed holding a stone (centimetre scale).

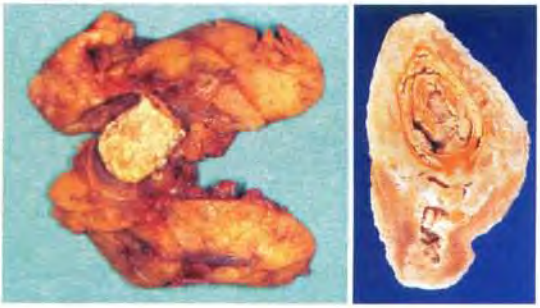


Fig. 55.7 A submandibular gland removed from another patient.

of the stone. On the right is a stone approximately 15 mm long sectioned to reveal the incremental lines that indicate intermittent formation of the stone in concentric layers during periods of low saliva flow or stasis. Somewhere in the centre is a nidus, probably a small crystal, shed duct lining cell or similar, possibly foreign, particle that is the nucleus of the stone.

■ ***The histological appearances of a gland that contained a stone are shown in Figure 55.8. What do you see and how do you interpret the findings?***

On the left is normal submandibular gland. Note how the gland tissue is composed of densely packed acini and ducts with very little space between them. Most of the acini are serous acini and are composed of granular, darkly stained cells. Below the centre of the picture is a cluster of bubbly pale-stained mucous cells. The ducts have a prominent eosinophilic (pink) lining epithelium and are striated ducts. In the normal gland the mucin washes out of the ducts when the section is prepared.

In the centre is a gland excised because of a stone. In the middle is a collecting duct that is very dilated and contains inspissated mucin that cannot be washed away, a result of reduced flow and blockage. Inflammatory cells are visible as tiny blue dots within the mucin reflecting emigration into the duct in response to ascending infection and bacteria around the stone.

On the right is a higher power view of several lobules.

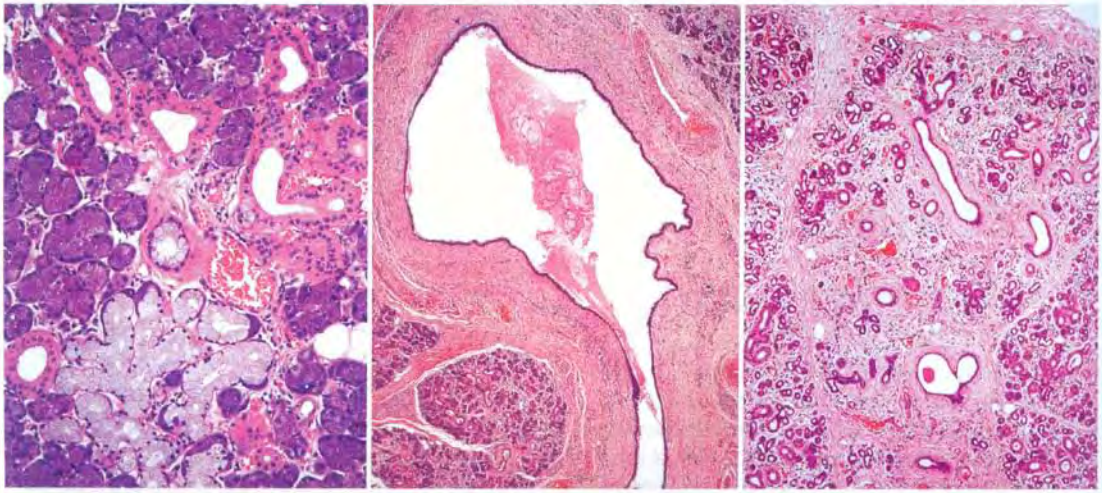


Fig. 55.8 Histological appearances of a normal submandibular gland and one excised for sialadenitis secondary to sialolithiasis.

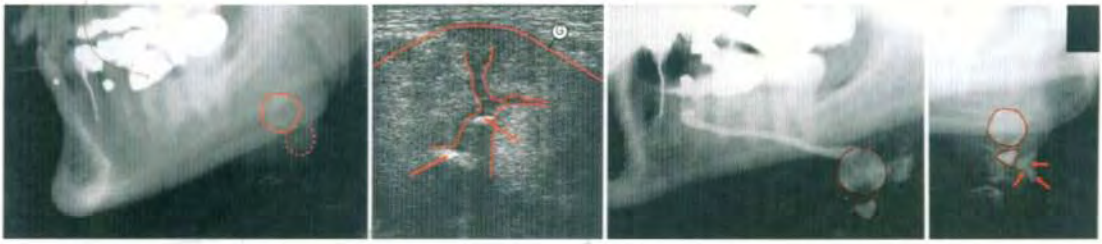


Fig. 55.9 Plain film (Fig. 55.2) with main stone outlined and second stone ringed by a dotted line. Ultrasound scan (Fig. 55.3) with the gland surface outlined (G) two stones arrowed and the dilated proximal duct outlined. Sialogram (Fig. 55.4) with the stones ringed and strictures in the main ducts arrowed.

The acinar cells have almost all been destroyed and only small ducts remain. There are large spaces between the acini and ducts filled with fibrous tissue, seen as a pink collagen cuff around the ducts. This is scarring as a result of chronic inflammation. The scarring is felt as induration on palpation of the whole gland. Fibrosis also destroys the gland structure and prevents recovery, even if the stone were to be removed.

56 Failed endodontic treatment

Summary

A 40-year-old lady presents to you with apical infection on a root-treated and crowned upper incisor. What are the treatment options and their likely chances of success?

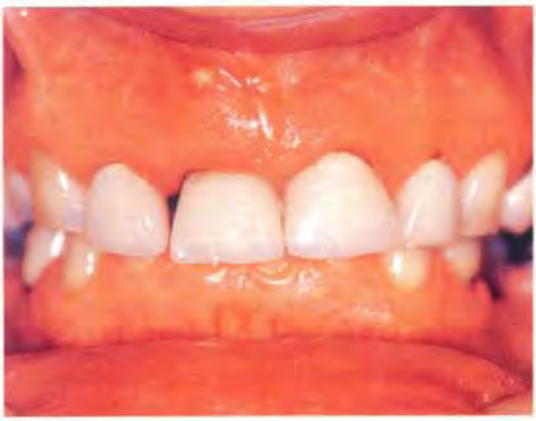


Fig. 56.1 The upper anterior teeth on presentation. The crown on the upper right lateral incisor had fallen out and has been replaced for the photograph.

HISTORY

Complaint

The patient, pointing to her upper right lateral incisor, complains that one of her crowns has fallen out. She is in no pain.

History of complaint

The crown fell out a few days ago. It has not fallen out before, though the adjacent crown on the upper right central incisor requires regular recementing.

Dental history

The patient has always attended regularly for dental treatment, but with a series of different practitioners. Some of the upper incisors were root treated about 10 years ago and all are crowned. Treatment was successful initially but she has suffered several episodes of pain over the last 5 years, for which she has been prescribed a number of courses of antibiotics. Periradicular surgery (apicectomy with root end filling) has been carried out on four occasions and she has suffered intermittent bouts of tenderness from most of the upper incisors since the last surgical procedure.

Medical history

The patient is otherwise fit and well.

EXAMINATION

Extraoral examination

The temporomandibular joints appear normal and no submental, submandibular or cervical lymph nodes are palpable.

Intraoral examination

The patient hands you a porcelain crown from the upper right lateral incisor with a cast gold post still cemented into it.

The appearances of the dentition with the crown temporarily reseated in the lateral incisor are shown in Figure 56.1 and in two occlusal views in Figure 56.2.

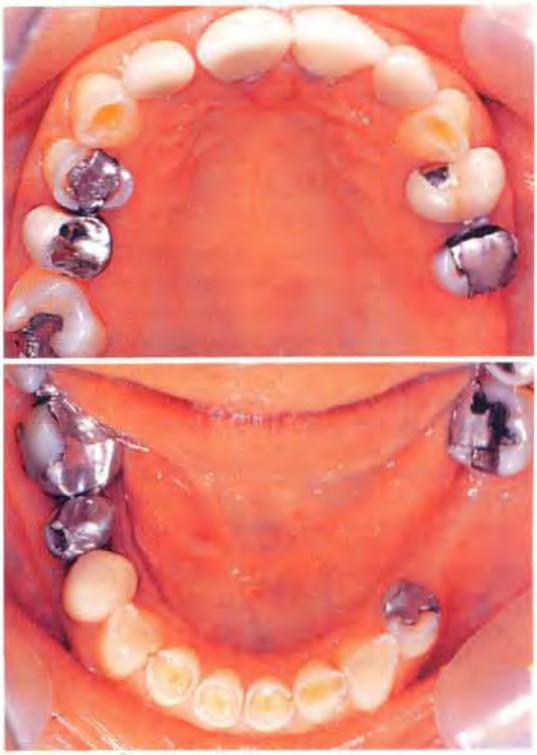


Fig. 56.2 Occlusal views of lower and upper arch at presentation.

In this case the sinus opening lies immediately over the apex of the upper right central incisor, indicating the likely source of infection. If this were less clear-cut, a thin gutta percha cone could be inserted through the sinus opening and it would track to the source of the infection (see Case 1).

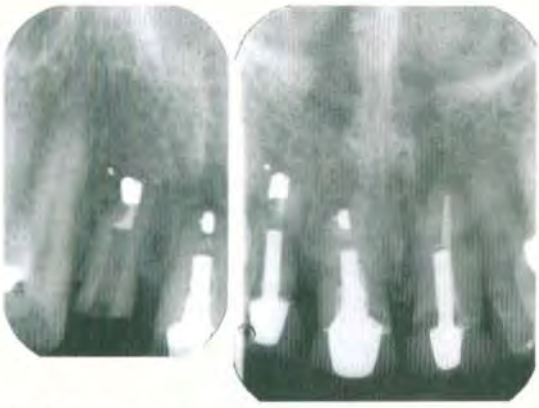


Fig. 56.3 Periapical radiographs taken on presentation. That on the left was taken with the post crown on the upper right lateral incisor removed and that on the right with it temporarily reseated.

■ ***The periapical radiographs are shown in Figure 56.3. What do you see?***

See Table 56.1.

DIAGNOSIS

■ ***What are your provisional diagnoses?***

See Table 56.2.

TREATMENT

■ ***How would you manage these failed restorations?***

The posts and crowns need to be removed from all three teeth to allow further investigation for root fracture and to assess the possibility of re-root filling the teeth by an orthograde approach.

■ ***How will you remove the posts and crowns? What are the advantages and disadvantages of the methods?***

The crowns must be sectioned and removed to gain access to the core and root face.

Table 56.1 Radiographic investigation

Upper right lateral incisor	Upper right central incisor	Upper left central incisor
The cast post does not fill the prepared post space, leaving a void between it and the small amount of gutta percha root filling apically. A root end filling of amalgam has been placed but it does not conform to the apical morphology of the root and extends into the periradicular tissue. Despite these defects there is no periradicular radiolucency. The crown margins are poorly adapted to the root face.	This tooth also has a poorly fitting cast post and core and a thick cement lute can be seen around the post. There is no evidence of a conventional orthograde root canal filling and a root end filling of amalgam is present. A periradicular radiolucency is evident with widening of the periodontal ligament at the apex, even though the lamina dura appears intact across it. No root fracture can be detected. The crown margins are poorly adapted to the root face.	In this tooth the cast post and core deviate from the original line of the root canal and weaken the root mesially. Distally some residual gutta percha lies alongside the post. There is an adequate length of gutta percha root filling apical to the post; however, there is a periradicular radiolucency and loss of lamina dura around the apex. The crown margins are poorly adapted to the root face.

Table 56.2 Provisional diagnoses

Upper right lateral incisor	Upper right central incisor	Upper left central incisor
Failure of cementation of the post crown, predisposed to by inadequate post length. There is additional root length available to provide a longer post Despite its suboptimal appearance, the apical amalgam appears successful and there is no evidence of apical periodontitis	A vertical root fracture is probably present as evidenced by the gingival inflammation, infection, pattern of pocketing and the history of repeated decementation of the post and crown. Root fracture is predisposed to by overpreparation of the root and lateral forces resulting from inadequate post length. Infection of the fractured root. Bacteria have probably entered via the gingival crevice and pocket and repeated decementation of the post. The void below the post provides an additional cause for failure of root canal treatment. Bacteria can colonize it after loss of the post and extend to the apical seal. Chronic suppurative periradicular periodontitis, indicating failure of the apical seal.	The root filling is inadequate. There is no coronal seal – the crown is poorly adapted and gutta percha extends alongside the post, potentially allowing bacterial ingress to the root apex. There is chronic periapical periodontitis indicating failure of coronal and apical seal and persistence of bacteria within the root.

Three methods are available for removing the posts:

1. Application of an ultrasonic instrument, with copious water coolant, around the core at different angles. Ultrasonic vibration will shatter the cement lute and allow removal. This is time consuming and some authorities suggest that the technique must be used continually for 10 minutes before admitting failure. However, it can take much longer and patients may find the prolonged procedure uncomfortable.
2. The Masseran kit, a series of trephines of different diameters that are used to cut around the post. Care must be used to select the smallest trephine that fits over the post. The trephine cuts a cylindrical hole and is thus not conservative of root dentine, especially if the post is large when lateral perforation by the trephine is a risk. Also the core must be cut away before the trephine can be used, limiting other options if the trephine fails.
3. The Egglers or similar clamp/screw devices that grip the core and pull the post out. The core must be prepared to provide retention for the clamp and threaded posts cannot be removed with a pull action.

Rotary instruments cannot ever be used to drill out metal posts. The relative hardness of the post means that rotary instruments will always slip and remove root dentine, risking perforation of the root.

That there is a risk of root fracture should be explained to the patient. However, this rarely results from post removal unless the root is already split.

In this instance all posts were easily removed using an ultrasonic scaler. On removing the posts you discover a vertical root fracture in the upper right central incisor as expected. This tooth is therefore unrestorable and will require extraction. You continue to open the root canals of the other teeth under rubber dam. The single cone gutta percha root filling in the left central incisor was easily removed and, under copious irrigation with sodium hypochlorite, the working length was established.

Fig. 56.4 Working length radiograph for the upper left central incisor. The rubber dam is retained on the premolar teeth for better access and no clamp is visible.

■ ***The working length radiograph is seen in Figure 56.4. What can you deduce?***

The file used to take the working length radiograph is wide. This is because the previous root canal preparation was excessive and only a large file binds against the root canal walls. The file is approximately 2 mm short of the correct working length. The apical part of the root canal has been overprepared and this now poses a problem as the anatomical apical constriction has been destroyed and extrusion of the root canal filling through the apex is likely.

This is exactly what happened, as can be seen in Figure 56.5.

Fig. 56.5 Completed root filling in the upper left central incisor.

■ ***The teeth are now stabilized. What are the longer term options?***

The upper right lateral incisor has no active apical inflammation and a new post and temporary crown can be considered. However, the apical amalgam is less than ideal and the tooth is compromised as an abutment for a fixed bridge to replace the right central incisor.

The upper right central incisor is unrestorable and following extraction will need to be replaced with an upper acrylic immediate partial (removable) prosthesis for a 6 month period to allow for ridge resorption to take place. At this stage a definitive replacement can be considered.

The upper left central incisor may now be symptomless and the apical area may resolve. Attempts to remove the extruded material via the root canal are typically unsuccessful, pushing the material further into the tissues. Therefore it is advisable to place a new post and core straight away to establish a good coronal seal . A laboratory-made temporary crown can be placed for the 6 month observation period required for the right central incisor. Extrusion of gutta percha compromises the long-term prognosis and makes this tooth unsuitable as an

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