Long-term management
A follow-up appointment should be arranged to check for resolution.

Periodontal abscess
A periodontal abscess is defined as ‘a localised accumulation of pus within the gingival wall of a periodontal pocket resulting in the destruction of the collagen fibre attachment and the loss of nearby alveolar bone.’ In a study performed in general practice in the UK, 6-7% of patients treated in one month suffered from a periodontal abscess, which made it the third most prevalent infection demanding emergency treatment after dento-alveolar abscesses and pericoronitis.

Presentation
The most prominent sign is the presence of an ovoid elevation in the gingiva along the lateral part of the root. However, abscesses deep in the periodontium may be less apparent, presenting as a diffuse swelling or simply a red area. The area may be tender on touch. The abscess is usually associated with a deep periodontal pocket with bleeding and tenderness on probing. Suppuration may also occur through a fistula or through the periodontal pocket opening and may be spontaneous or following finger pressure. Increased tooth mobility and tenderness on percussion are common. The patient may also report the tooth feeling “high” in the occlusion. As periodontal abscesses are usually associated with existing periodontal pockets, some degree of bone loss around the tooth in question is likely to be seen radiographically. When performing sensibility testing, the tooth should respond positively.

Aetiology
A periodontal abscess may represent disease exacerbation of existing periodontitis in the presence of...
complex pocket morphology, furcation involvement or a vertical defect, in which marginal closure of the pocket may lead to spread of infection into the surrounding periodontal tissues. Changes in the composition of the subgingival microbiota, with an increase in bacterial virulence, or a decrease in the host defence, may also result in a diminished capacity to drain the increased suppuration. Common examples of when this may occur include:

• Following debridement – calculus fragments may become dislodged and pushed into the periodontal tissues.
• After surgical therapy – associated with the presence of foreign bodies such as membranes or sutures.
• Acute exacerbation of periodontitis.
• Systemic antimicrobial intake without subgingival debridement in severe periodontitis (related to an overgrowth of opportunistic bacteria).

Emergency management
The first phase of treatment involves control of the acute condition to arrest tissue destruction and management of acute symptoms. If the tooth can be saved, drainage needs to be established (through the pocket or with an external incision) and the periodontal pocket should be thoroughly debrided. Occlusal adjustment may also be help to provide immediate relief. Systemic antibiotics are only required if there are clear signs of systemic involvement or spreading infection (e.g. fever and lymphadenopathy). If the tooth has a hopeless prognosis for periodontal or other restorative reasons, the most sensible treatment option may be extraction.

Long-term management
As most periodontal abscesses occur in a pre-existing periodontal pocket, periodontal therapy should be evaluated after resolution of the acute phase. In cases where the patient has not been treated previously, the appropriate periodontal treatment should be provided. If the patient is already within the active phase of therapy, the periodontal therapy should be completed once the acute lesion has been treated. In patients receiving supportive periodontal therapy, careful evaluation of the recurrence of the abscess should be made, as well as assessment of the tissue damage and its implications on the tooth’s long-term prognosis.

Pericoronitis or peri-coronal abscess
Pericoronitis is ‘inflammation of the soft tissues surrounding the crown of a partially erupted tooth.’ A peri-coronal abscess is the ‘localised accumulation of pus within the overlying gingival flap surrounding the crown of an incompletely erupted tooth.’

Presentation
Red, swollen, possibly suppurative lesion that is painful to touch. Swelling of the cheek at the angle of mandible, trismus and a radiating pain to ear are common. The patient may also experience systemic complications such as lymphadenopathy, fever and general malaise.

Aetiology
The partially erupted and impacted mandibular third molar is the site most frequently involved. The overlying operculum is an excellent harbour for the accumulation of debris and bacteria. In addition, insult to the operculum is often produced by trauma from an opposing tooth.

Emergency management
Following anaesthesia, the operculum should be irrigated to remove any debris. In some cases, it may be necessary to excise the operculum and possibly also perform some occlusal adjustment on the opposing tooth to eliminate any sources of trauma. Antibiotics are only indicated if there are systemic signs or spreading infection.

Long-term management
Once the acute phase of the infection has subsided, a decision needs to be made on whether the tooth

### TABLE 1
KEY FEATURES OF THE GINGIVAL, PERIODONTAL, PERI-CORONAL AND PERI-ENDO ABSCESS

<table>
<thead>
<tr>
<th></th>
<th>Gingival abscess</th>
<th>Periodontal abscess</th>
<th>Peri-coronal abscess</th>
<th>Perio-endo abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>Painful, especially to touch</td>
<td>Painful, tooth may feel more mobile and ‘high’ in occlusion</td>
<td>Painful to touch, extraoral swelling, difficulty opening fully, radiating pain to the ear</td>
<td>Painful, tooth tender to bite on and may feel more mobile</td>
</tr>
<tr>
<td>Site</td>
<td>Marginal gingiva/interdental papilla</td>
<td>Attached gingiva/mucosa</td>
<td>Partially erupted/impacted tooth</td>
<td>Attached gingiva/mucosa usually close to the root apex</td>
</tr>
<tr>
<td>Peridontal</td>
<td>Periodontally healthy</td>
<td>Deep pocket depths</td>
<td>False pocketing possible around partially erupted tooth but adjacent teeth with no increased probing pocket depths</td>
<td>Deep pocket depths</td>
</tr>
<tr>
<td>Sensibility testing</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
<td>Negative</td>
</tr>
</tbody>
</table>
PERIODONTAL EMERGENCIES IN GENERAL PRACTICE

requires extraction. If the tooth is retained, optimal oral hygiene measures are necessary to help prevent further acute pericoronitis episodes. The evidence suggests that ‘the first episode of pericoronitis, unless particularly severe, should not be considered as an indication for surgery. Second or subsequent episodes should be considered appropriate indication for surgery.’

Extraction might also be indicated if the tooth is associated with extensive caries or root resorption.

Perio-endo lesion or abscess
Combined periodontal/endodontic lesions are localised, circumscribed areas of infection originating in the periodontal and/or pulpal tissues. They are essentially a result of a communication between the periodontal pocket and the pulp.

Presentation
The abscess will be associated with a deep pocket surrounding a non-vital tooth. The pocket may circumscribe a large part of the tooth or be a localised narrow deep lesion. A smooth, shiny swelling of the gingiva or mucosa will be present. The swelling may have purulent exudate or fistula and will be tender to palpation. The tooth may be tender to percussion and mobile. A typical radiographic appearance is shown in Figure 2.

Aetiology
The infection may arise primarily from pulp inflammatory disease expressed through the periodontal ligament or the alveolar bone to the oral cavity, or it may be initiated from a periodontal pocket communicating to the pulp apically or through accessory canals. Vertical root fracture of a tooth (which may not be heavily restored or root canal treated) may also present in a similar manner. Rapid loss of the periodontal attachment and peri-radicular tissues may ensue.

Emergency management
Considerations include establishing drainage by debriding the pocket and/or extirpating the pulp. The abscess may be incised if it is fluctuant and pointing. Other treatments may include limited occlusal adjustment, the administration of antimicrobials if there are signs of spreading infection and management of patient comfort.

Long-term management
Primary endodontic lesions often respond well to root canal therapy alone. If the lesion is primary periodontal or truly combined, there is often a very poor prognosis and this is usually a good indication for extraction in single rooted teeth. In multi-rooted teeth, root resection may be a possible consideration following endodontic therapy. It is also necessary to ensure that there has not

### Table 2

**KEY DIFFERENCES BETWEEN NECROTISING GINGIVITIS AND ACUTE HERPETIC GINGIVOSTOMATITIS**

<table>
<thead>
<tr>
<th>Ulcers</th>
<th>Necrotising gingivitis</th>
<th>Acute herpetic gingivostomatitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site</td>
<td>Interdental papilla, marginal gingiva</td>
<td>Gingiva, entire oral mucosa</td>
</tr>
<tr>
<td>Characteristics</td>
<td>Punched-out, covered with yellow/white/grey slough, bleed readily, painful on palpation</td>
<td>Multiple vesicles that coalesce and form shallow, regular shaped ulcers. No marked tendency to bleed and may/may not be tender on palpation</td>
</tr>
<tr>
<td>Duration</td>
<td>Short-lived with appropriate management</td>
<td>More than 1 week</td>
</tr>
<tr>
<td>Local Symptoms</td>
<td>Painful gums, bad taste/breath</td>
<td>Sore mouth</td>
</tr>
<tr>
<td>Systemic Symptoms</td>
<td>Not usually feverish</td>
<td>Feverish</td>
</tr>
</tbody>
</table>

Figure 3: Necrotising gingivitis
Figure 4: Necrotising periodontitis
Figure 5: Acute herpetic gingivostomatitis
been a vertical fracture, since this may often render a tooth unsavable.

Table 1 summarises the key features of the gingival, periodontal, peri-coronal and perio-endo abscesses.

Necrotising periodontal diseases
Necrotising periodontal diseases are considered the most severe inflammatory lesions associated with the oral biofilm. These include necrotising gingivitis (NG) and necrotising periodontitis (NP). NG describes a scenario where only the gingival tissues are affected (see Figure 3). In NP, the necrosis progresses into the periodontal ligament and alveolar bone, leading to attachment loss (see Figure 4). It has been suggested that these conditions may be different stages of the same disease.

Presentation
The mandibular anterior teeth are most commonly affected. NG will be associated with necrosis and ulcers in the free gingiva. These lesions start at the interdental papilla and typically have a ‘punched out’ appearance. Marginal erythema may be present, and necrotic lesions can progress to the marginal gingiva. A pseudo-membrane may form over the necrotic area. When this ‘membrane’ is removed, the underlying connective tissue becomes exposed and bleeds. The severity of pain experienced by the patient is dependent on the severity and extension of the lesions. The bouts of pain usually increase with eating and oral hygiene practices. Other less common symptoms include halitosis, fever and malaise. NP will have the features described for NG but in addition the necrosis will affect the periodontal ligament and alveolar bone, leading to attachment loss. As the disease progresses, an interproximal crater will divide the buccal and lingual/palatal portion of the papilla. If these craters are deep, the interdental crestal bone may become denuded. When interproximal necrotic areas spread laterally and merge, this results in the creation of an extensive zone of destruction. In severe cases, bone sequestration may occur. In patients with NG, there may be associated risk factors such as high stress levels, heavy smoking and poor nutrition. Both NG and NP may be associated with untreated HIV/AIDS or other diseases and drugs that may, directly or indirectly, have an immunosuppressant effect, such as chemotherapy or anti-rejection medication in transplant patients.

Aetiology
Necrotising periodontal diseases are caused by infectious but commensal organisms. However, predisposing factors including a compromised host immune response are key in facilitating bacterial pathogenicity. Knowledge of the pathogenesis of this condition is limited but the spirochetes and fusiform bacteria described in the necrotic lesions have been shown to have the capacity to invade the epithelium and connective tissue, as well as release endotoxins, which may cause periodontal tissue destruction through modification of the host response.

Emergency management
Superficial debridement to remove soft and mineralised deposits should be carefully performed. Ultrasonic rather than hand instruments are recommended to ensure minimum pressure over the ulcerated soft tissue. The debridement may be performed daily, getting deeper as the patient’s tolerance improves, lasting for as long as the acute phase lasts (usually 2-4 days). Mechanical oral hygiene measures such as brushing should be limited to avoid pain and ensure healing. The patient should be advised to use chemical plaque control agents, such as chlorhexidine mouthwash (0.2% twice daily) instead. Other agents, such as 3% hydrogen peroxide diluted in 1:1 warm water and other oxygen-releasing agents, provide an additional antibacterial effect against anaerobes through the release of oxygen. If there is no improvement in the periodontal condition following debridement, the use of systemic antimicrobials should be considered. Metronidazole (400 mg three times a day for five days) is usually the first choice, due to its action against strict anaerobes.

Long-term management
These patients require close monitoring and support. As signs and symptoms improve, strict oral hygiene measures should be enforced and debridement should be completed where necessary. Once the acute phase has been controlled, treatment of any pre-existing periodontal disease and control of systemic risk factors should be addressed. Management may also occasionally involve attempts to try to disguise soft tissue asymmetry, which may be present following secondary surgery.

REFERENCES
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resolution of lesions. However, not all cases (eg. class IV recession defects) will be amenable to predictable soft tissue augmentation.

**Acute herpetic gingivostomatitis**
Herpetic gingivostomatitis is the most common viral infection of the oral mucosa (see Figure 5).

**Presentation**
This condition is characterised by small ulcers with elevated margins that may be dispersed throughout the mouth on both attached and unattached mucosal surfaces. The patient may experience generalised pain in the gingiva and/or oral mucosa. Systemic signs such as lymphadenopathy, fever and malaise may also be present. Lesions normally last for 7–10 days and heal without scarring. This condition is more frequently observed in children aged two to five years of age.

**Aetiology**
Herpetic gingivostomatitis is caused by the herpes simplex virus (HSV-1). It is the initial presentation, during the primary herpes simplex infection, and is of greater severity than herpes labialis, which is often the subsequent presentation.

**Emergency management**
As the condition is self-limiting, no treatment is usually indicated beyond hydration and management of symptoms. Therapy to relieve pain must be initiated to allow the patient to eat and drink. Paracetamol is commonly used and the recommended dosage for a child aged two to four years is 180 mg every 4-6 hours.\(^\text{22}\)
If the condition is severe and presents in immune-compromised patients, referral to hospital is advised as they may require intravenous antiviral drug treatment.\(^\text{23}\)

**Long-term management**
A two-week follow-up appointment should be arranged to check for resolution.

The key differences between necrotising gingivitis and acute herpetic gingivostomatitis are summarised in Table 2.

**Acute physical, chemical or thermal injury**
This group of acute periodontal lesions are not aetiologically associated with the oral biofilm. However, it is important to be familiar with these as they are not uncommon, and accurate diagnosis will ensure appropriate therapeutic management.

**Presentation**
- Physical injuries may appear as erosions or ulcers, and can be associated with gingival recession. Less frequently, they can present as hyperkeratosis, vesicles or bullae.
- If the physical trauma is limited but continuous over time, the lesion may be hyperkeratotic. If the trauma is more aggressive, superficial laceration

Figure 7: Physical trauma from a silver toothpick

Figure 8: Self-induced physical injury – gingivitis artefacta


Aetiology

- Physical injuries are most commonly caused by inappropriate oral hygiene habits, traumatic injuries or parafunction.24-26 For example, the use of an abrasive dentifrice and an overzealous horizontal brushing technique might result in an ulcer or erosion in the gingiva. The incorrect use of dental floss or other interdental devices may also result in gingival ulceration, inflammation and recession (see Figure 7). Physical injuries can be self-induced, and examples of traumatic agents include fingers, nails or items such as pencils (see Figure 8). Self-induced injuries are more frequent in children or teenagers, sometimes with psychological conditions.27,28 Traumatic injuries might also be related to fractured teeth, orthodontic appliances or oral piercings.29,30

- Common causes of chemical trauma are oral bleaching agents, either due to inappropriate use by the patient or poorly fitting trays. Other agents include etchants and less commonly used dental products.31-33

Emergency management

Therapeutic intervention will depend upon the diagnosis and cause. As well as clinical examination, an accurate patient history will be important in determining the source of the trauma. Treatment will include elimination of the initiating factor (if required) and symptomatic management of the pain.

Long-term management

Lesions usually heal without further intervention, but on some occasions additional treatment may be required. It is important to distinguish this group from lesions related to mucocutaneous diseases where there may not be a clear initiating factor, hence it may be necessary to withhold mechanical oral hygiene procedures, supplemented by the use of chemical plaque control, to help establish aetiology.

Subgingival root fractures

A fracture of a tooth extending from the supragingival oral environment in an apical direction subgingivally can give rise to acute pain and periodontal infection.

Presentation

Magnification and good illumination can help to visualise fracture lines. A “Tooth Slooth” or similar device may also aid diagnosis by applying occlusal loads to individual cusps. Even if a fracture line cannot be visualised, fractured teeth are characteristically associated with a localised deep pocket depth and possibly an abscess. The tooth/fractured cusp may be extremely tender to percussion, or pain may only be felt on release of biting load. Fractures may be vertical along the root axis, or at an angle with varying degrees of root involvement.

Aetiology

The patient may or may not be aware of a specific traumatic event during chewing. Heavily restored teeth without cuspal coverage and bruxism are key risk factors. This is also not uncommon in patients with a reduced periodontium due to an unfavourable crown-root ratio.34

Emergency management

Management depends on the vitality of the tooth as well as the location and extent of the fracture. It may be necessary to remove existing restorations followed by careful assessment of the tooth to confirm diagnosis. A periodontal flap can be useful in visualising the fracture. Crown lengthening can help to expose the most apical extent of the fracture. Initial care to relieve acute pain may involve endodontic treatment. In some cases, the tooth will be deemed untreatable and will require extraction (see Figure 9).

Long-term management

Endodontic treatment should be followed by a full coverage restoration if the tooth is restorable.

If you are unsure of the management in any of the above cases, it may be most appropriate to seek advice from a professional colleague either locally or in secondary care.