The Lips, Mouth, Tongue and Teeth: Part II

25

Omar Sheikh and Michael Perry

25.1 Swellings and Lumps in and Around the Mouth

Swelling refers to an abnormal, generalised enlargement of a part of the body, often due to the accumulation of fluid. They are usually obvious, being diffuse or localised, soft or firm, tender or non-tender and acute or chronic. It is important to identify the chronicity of any swelling and to determine whether it primarily involves the soft tissues or the underlying bones. Patients presenting with swelling of the lips should always be checked for simultaneous swelling intraorally, notably in the tongue, floor of the mouth and the throat. If swelling if advanced and involves these sites, the patient should be assessed further regarding their swallowing, speech and breathing. In severe infection, such as Ludwig's Angina, potentially life-threatening swelling can occur rapidly over a few hours. By way of contrast, a lump is usually smaller and more discrete. It can be soft tissue or bony in nature. These can occur anywhere in the mouth. Recent onset of pain and increased growth in any lump or swelling suggests the possibility of infection or malignancy.

25.1.1 Normal Anatomical 'Lumps'

Several prominences are normally found in the oral cavity. These are nearly always bilateral, symmetrical and non-inflamed, but they can result in concern in both patients and clinicians alike. These include:

(i) Parotid papilla. The opening of the parotid (Stenson's) duct is located inside the buccal mucosa adjacent to the upper second molar tooth, at the level of the

O. Sheikh (✉)
University College London Hospital, London, UK
e-mail: osheikh@nhs.net

M. Perry
London Northwest University Hospital, Harrow, Middlesex, UK

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occlusal plane. Bimanual palpation of the cheek will usually express saliva from the duct. This should be clear. If it is not, consider pathology.

(ii) Submandibular ducts. The openings of the left and right submandibular ducts are located either side of the fraenum, in the anterior floor of mouth. Saliva can usually be milked from the ducts.

(iii) The tongue is covered in tastebuds and the lingual tonsils are sited posteriorly. These can sometimes be mistaken for warts or growths. They tend to be symmetrical in distribution.

(iv) Lymph nodes in the buccal mucosa are usually not palpable. Sometimes they become enlarged secondary to viral or bacterial infection. A common site is the bucco-facial lymph node, which may be palpable both intraorally and extraorally. Typically this is found just above the lower border of the mandible, immediately in front of the anterior border of the masseter muscle. Ectopic lymphoid tissue is also common and can appear as a yellow nodule or multinodular lump. This can be found in the floor of the mouth and the soft palate.

(v) Exostoses are benign localised overgrowths of the cortical bone. They commonly occur in the maxilla (Torus Palatinus) and the lingual cortex of the mandible in the premolar region. These present as very hard, smooth, discrete bony lumps.

(vi) Prominent genial tubercles may be seen in the edentulous lower jaw, following complete resorption of the alveolar bone. As a result the tubercle, which is normally hidden from view, becomes more obvious, appearing as a hard swelling in the midline of the floor of the mouth. This can often be confused with an infiltrating tumour.

Pathological swellings and lumps within the oral cavity include cysts (dental and minor salivary gland), granulation tissue, inflammation, abscesses and connective-tissue proliferations. Rarely, a tumour may present as a swelling (sarcoma/lymphoma). From a clinical perspective the three most important features of any soft-tissue swelling are its location, colour and palpable characteristics. Certain conditions tend to occur in specific sites, for example mucoceles are commonly seen in the lower lip and buccal mucosa. The colour of a lesion depends on its composition and its depth. Yellow-appearing lesions are usually made of lymphoid or adipose tissue, red swellings are vascular, blue swellings are mucinous or venous and brown swellings contain melanin or blood pigments. Lesions with a normal pink mucosal surface are usually composed of fibrous tissues or are deep in the tissues. Firm movable masses are usually neoplasms or granulomas, soft mobile masses are fatty or myxoid tumours, fluctuant masses are cysts, abscesses or vascular and indurated fixed masses are probably malignant (most likely carcinomas, salivary tumours, lymphomas, and sarcomas).

25.1.2 Infections/Abscesses

Specific infections are discussed in the chapters on the lower jaw and the front of the neck. The most common causes of acute swelling in the oral cavity are trauma and infection, the latter often arising from a dental source. Other possible causes include an infected fracture (or its fixation plate), infected salivary glands, infected wounds and osteomyelitis. Cellulitis may soon develop in the neck, resulting in diffuse swelling, redness and pain. Patients usually present with localised, tender swelling that develops over 24–48 h. An urgent OPG should be taken to assess the status of the teeth and bones. Patients with a lip abscess may have a prior history of skin infection, dental pain, or recent piercing. If an oral, facial, dental or lip abscess is suspected, urgent treatment is required. Abscesses must be drained. Although antibiotics are commonly given, they are not sufficient alone to treat abscesses. Drainage of pus and removal of the underlying cause is essential. Untreated, infections can spread resulting in cellulitis and Ludwig's Angina (see the neck). This requires urgent surgical decompression and airway support. Patients will be systemically unwell, febrile, with an accompanying leukocytosis and elevated CRP. CT scanning of the neck and chest may demonstrate fascial space collections along with mediastinitis. These are very late and imminently life-threatening complications (Figs. 25.1, 25.2, 25.3, 25.4, and 25.5).

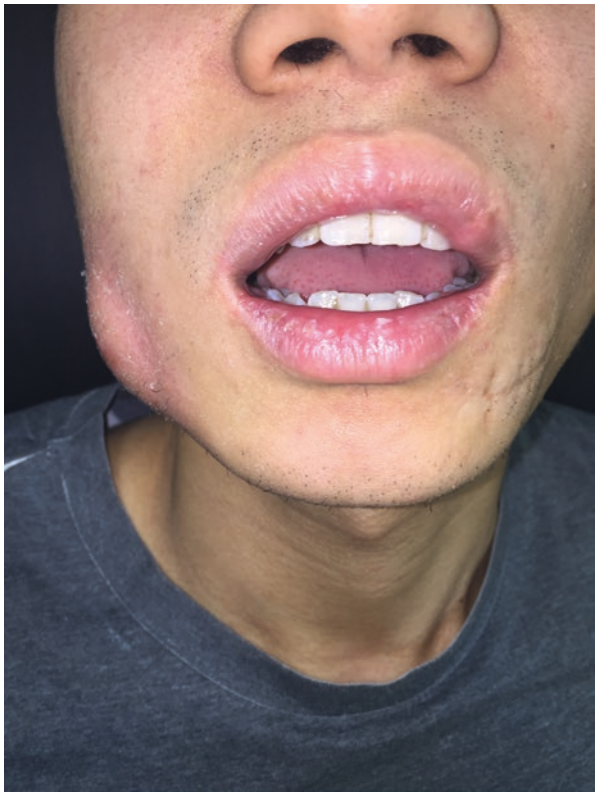

Fig. 25.1 Extraoral swelling—consider all possible causes (a good exam question)

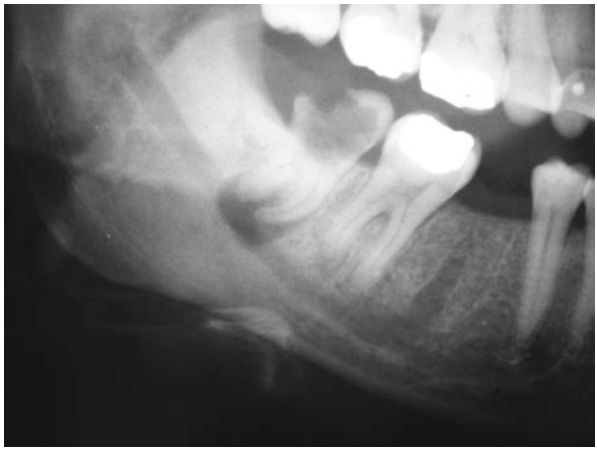

Fig. 25.2 Dental sepsis is a common cause of facial swelling

25.1.3 Surgical Emphysema

This is more commonly seen in the face and neck following facial fractures or other injuries to gas containing structures. It can also be a sign of serious infection (gas forming organisms). On rare occasions it can occur in the mouth. Air may be forcefully introduced between tissue planes during dental procedures where compressed air has been used to clean or dry a tooth. The trapped air results in diffuse oral and facial swelling and crepitus. This is a serious complication. Not only is there risk of infection, but air embolism can occur. Eventually, the air is absorbed by the tissues.

25.1.4 Anaphylaxis

Anaphylaxis is a clinical emergency, and all healthcare professionals should be familiar with its recognition and management. It results from Ig-E mediated degranulation of mast cells in response to an allergen, commonly food, latex or medication. Anaphylaxis may present as an itchy rash, swelling in the throat or tongue, shortness of breath, vomiting, lightheadedness and low blood pressure. Swelling in the mouth, throat (larynx) and neck can threaten the airway. Anaphylactic shock is characterised by hypotension, tachycardia and warm peripheries. Patients should be given high flow oxygen and if known, the allergen must be removed. Treatment includes adrenaline, IV fluids, anti-histamines and steroids as per local protocols. The oral swelling tends to resolve as the anaphylaxis settles.

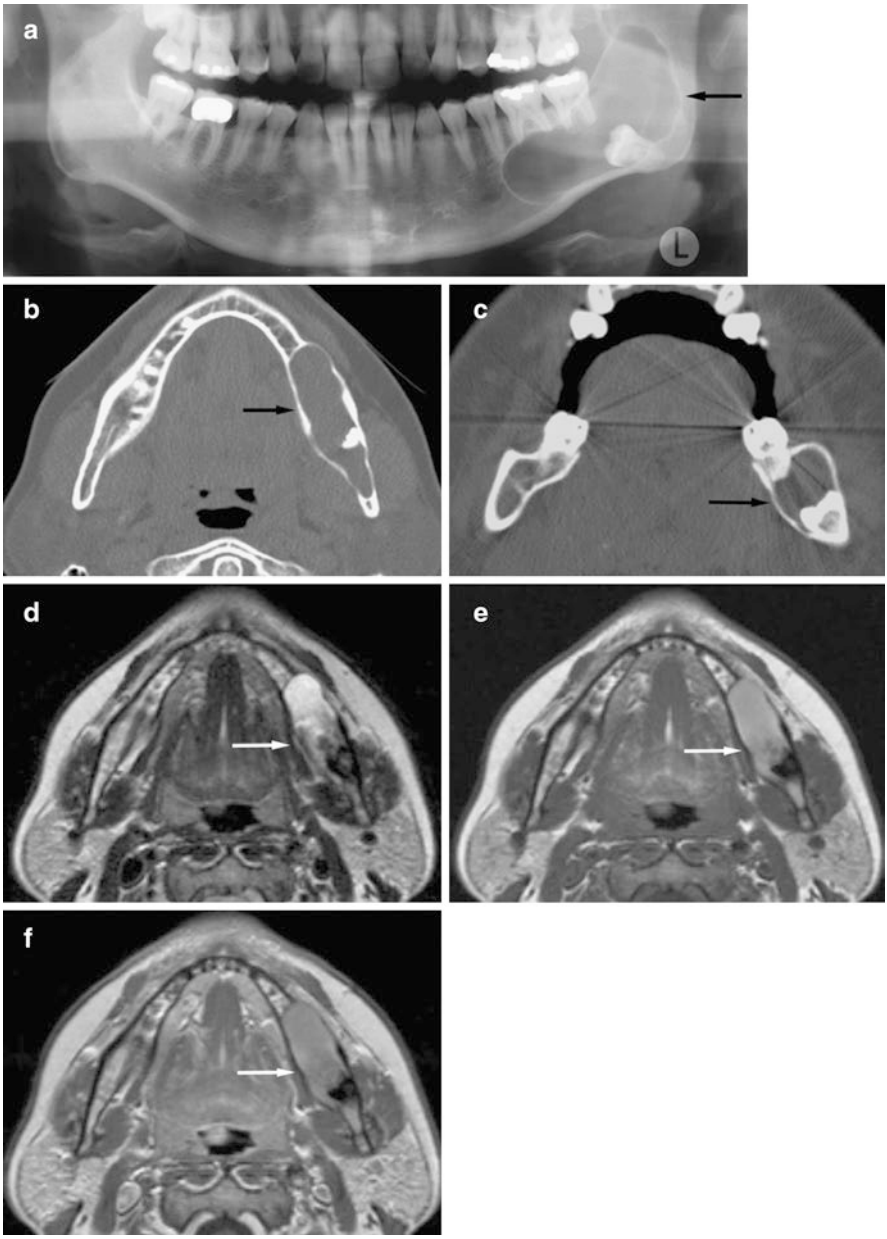

Fig. 25.3 Follicular cyst

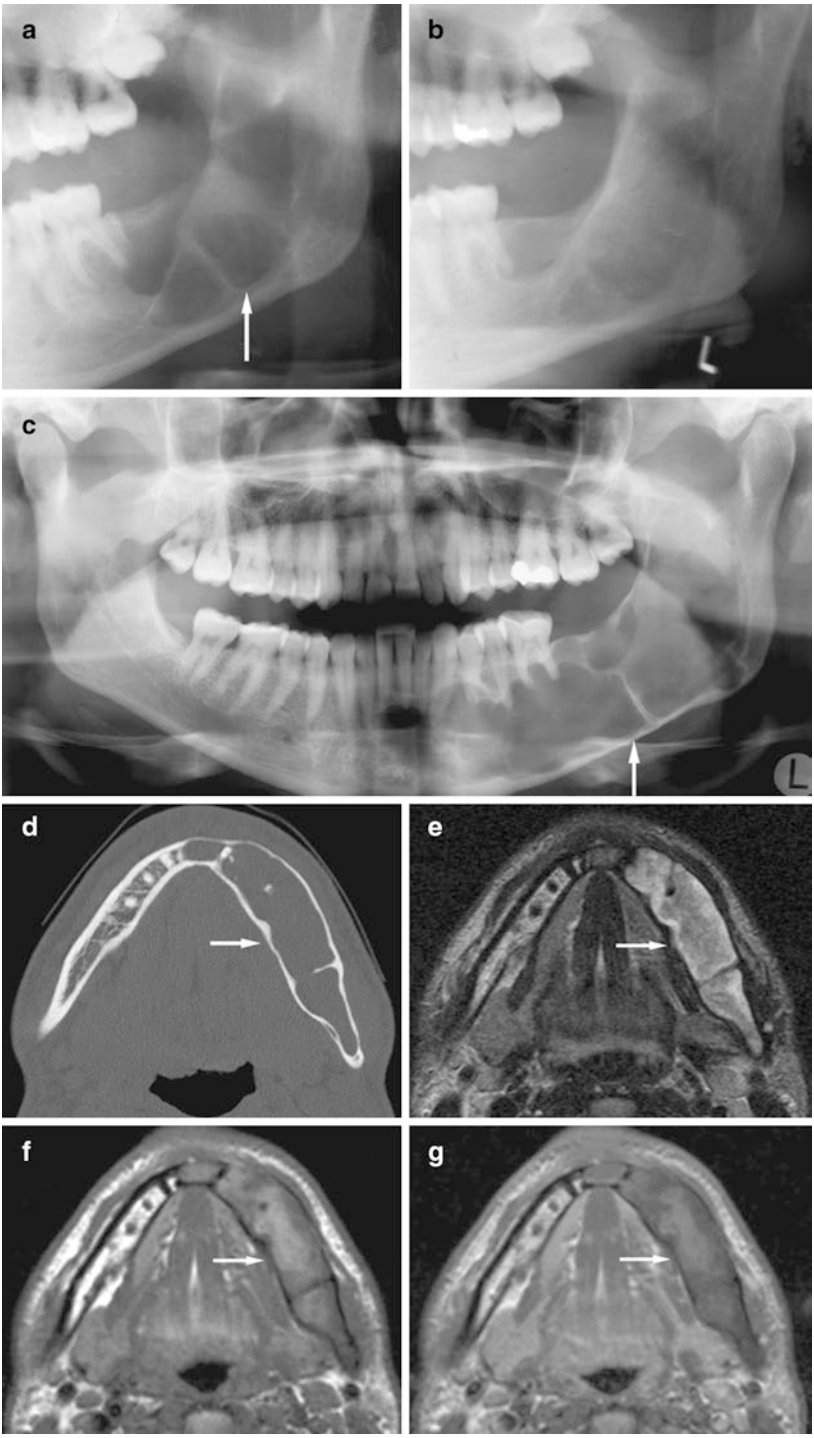

Fig. 25.4 Keratocystic odontogenic tumor, mandible

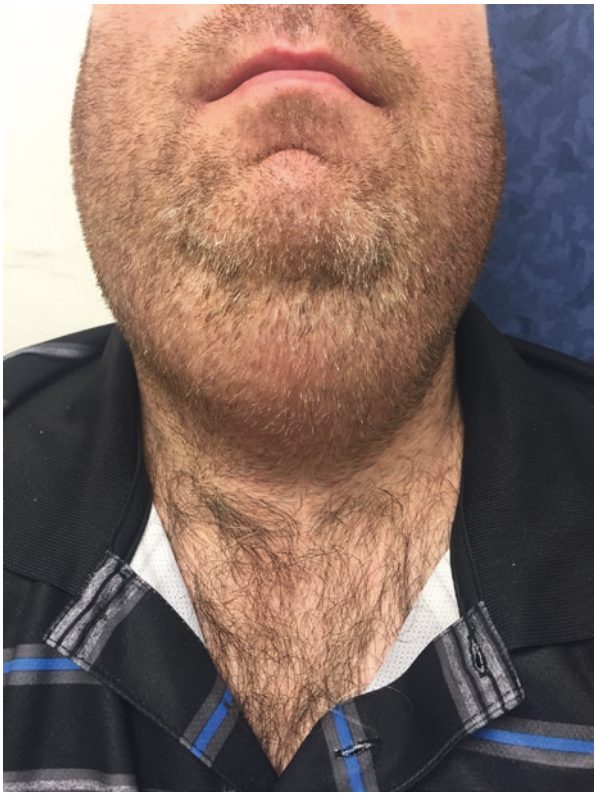

Fig. 25.5 Submental swelling

25.1.5 Angioedema

Angioedema (Quincke's oedema) is rapid swelling that occurs within the soft tissues (notably subcutaneous and submucosal). It is very similar to urticaria, but this latter condition is confined to the skin. The term angioneurotic oedema was previously used, (due to the belief that the nervous system was involved in some way), but this is no longer the case. Angioedema is classified as either hereditary or acquired. Acquired angioedema can be immunologic, non-immunologic or idiopathic. It is usually caused by an allergy and occurs along with other symptoms of allergy and urticaria. It can occur as a side effect to certain medications. Hereditary angioedema (HAE) exists in several subtypes. These result in a deficiency of C1 esterase inhibitor, or coagulation protein factor XII, both of which are components in the complement cascade. The end result is increased vascular permeability and subsequent tissue oedema.

In both types of angioedema the skin of the face (normally around the mouth) and the mucosa of the mouth, tongue and throat quickly swell, sometimes over a period of just a few minutes. In severe cases there may be stridor and wheezing. Itchy swelling may also occur in the hands and urticaria (hives) may develop.

Episodes may be triggered by allergens (commonly peanuts), stress, surgery and dental treatment. However, in many cases the cause is unknown or is only weakly associated to a particular allergen. Patients with HAE often have other gastrointestinal symptoms such as abdominal pain and diarrhoea.

Diagnosis is often based on the clinical picture, plus blood tests. Complement levels may indicate a deficiency of C1-inhibitor. Rapidly progressing angioedema is a medical emergency and should be treated as anaphylaxis. Airway obstruction can occur. Adrenaline (Epinephrine) may be life-saving. If this is allergic in nature. Avoidance of the allergen and use of antihistamines may prevent future attacks. Severe angioedema may require desensitisation to the allergen. However with hereditary angioedema, treatment may not be as effective. Prior to surgical or dental treatment, C1-inhibitor concentrate, or fresh frozen plasma can be given prophylactically. Needless to say, this condition requires specialist expertise in its management. Medications used in the management of angioedema include:

(i) Alpha- and beta-adrenergic agonist agents (e.g., Adrenaline)
(ii) Antihistamines (e.g., chlorpheniramine cetirizine, loratadine)
(iii) Histamine H2 antagonists (e.g., ranitidine, cimetidine)
(iv) Leukotriene receptor antagonists (e.g., montelukast, zafirlukast)
(v) Corticosteroids (e.g., prednisone, methylprednisolone)
(vi) Androgen derivatives (e.g., danazol, oxandrolone), progesterone-based birth control pills.
(vii) Antifibrinolytic agents (e.g., aminocaproic acid, tranexamic acid)
(viii) Immunomodulators (e.g., cyclosporine, mycophenolate, methotrexate)

25.1.6 Swelling Secondary to Injury

Localised lip and oral swelling is very common following trauma, in which case there is usually a clear history of injury. If there is a wound consider the possibility of a foreign body. A traumatic fibroma (a misnomer) is a localised fibrous overgrowth, which can occur following chronic trauma. Common sites include the lips, commissures, buccal mucosa, and tongue. Poorly fitting dentures are often a cause. Friction from an overextended denture flange can irritate the tissues resulting in multiple, flabby ridges, commonly in the anterior maxillary and mandibular vestibules. Irritation of the hard palate from a denture can also cause denture hyperplasia, which can become secondarily infected. Treatment is to trim the denture (or better still, leave it out for several weeks). If the fibrous tissue persists it can be excised.

Fibroepithelial polyps are discrete smooth, pedunculated soft tissue lumps that typically arise following trauma, most commonly due to cheek biting. They are most commonly found inside the buccal mucosa, but can be found on lips and the side of the tongue. These polyps are comprised of normal, non-inflamed, non-ulcerated tissue. There is no risk of malignant transformation. With recurrent trauma, the polyps can become progressively larger and are then more likely to be bitten and bleed. Treatment is excision.

25.1.6.1 Sublingual Haematoma

Sublingual haematoma is bleeding or swelling under the tongue. It usually occurs secondary to trauma (where it is highly suggestive of a fracture of the mandible), or dental treatment. Clinically it may resemble a large soft bruise in the floor of the mouth, with distension of the sublingual tissues and displacement of the tongue. Patients may experience problems with moving the tongue, speaking and swallowing. Occasionally it may result in airway obstruction. Large sublingual haematomas such as these are usually the result of significant trauma. Patients should be assessed carefully for a fractures of the mandible. Beware also patients taking anticoagulants—the swelling may continue to enlarge. There have been several reported cases of such haematomas in the upper airway in anticoagulated patients. Haematoma into the tongue itself has also been reported after the use of streptokinase, heparin, tissue-type plasminogen activator administration and in haemophilia. CT may be required if the diagnosis is not clear and to assess involvement of the airway. Spontaneous sublingual haematoma is a rare subtype, which is thought to be due to aneurismal changes in the facial or lingual arteries. Only a few reports of this have been reported in the literature, occurring in elderly hypertensive patients.

25.1.7 Mucocele (Mucus Retention Cyst)

Minor salivary glands are located throughout the entire oral cavity with the exception of the anterior dorsal tongue and the attached gingiva. A mucocele is a localised collection of mucus that occurs following rupture or blockage of one of the gland's duct. This usually arises following trauma to the area, usually a minor bite. The salivary duct becomes blocked, causing saliva to collect and distending the mucosal surface. This presents as a cystic swelling on the mucosal surface. When superficial they have a blueish look, which can be confused with a venous malformation. If fibrosed or deep they may feel more solid and have normal mucosal coloration. Treatment is usually excision and removal of the underlying minor glands, although some may resolve spontaneously (Fig. 25.6).

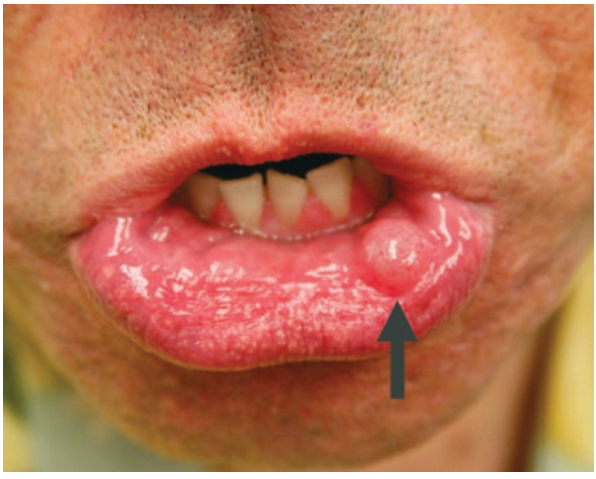

Fig. 25.6 Clinical photograph of mucocele on lower lip

25.1.8 Ranula

A ranula is a large mucocele arising in the floor of the mouth. It is occurs following rupture of one of the sublingual glands and usually presents as a soft, translucent, compressible swelling in the anterior floor of mouth. The name is taken from the latin word for frog, as this resembles the distended throat of some species. Ranulas may be simple or plunging. Following rupture (or blockage) of the gland, saliva is unable to drain freely and begins to collect and distend the surrounding tissues. As it enlarges the ranula extends along sublingual space. This can eventually pass through or behind the mylohyoid muscle and enter the submandibular or submental spaces—a 'plunging' ranula. There are reported cases of extension inferiorly as far down as the supraclavicular area and upper mediastinum and posteriorly into the retropharyngeal space—but this is very rare. US, CT or MRI imaging may therefore be necessary to define the extent of the ranula.

Surgery is the main treatment. This include incision and drainage, excision of the ranula, marsupialisation (this has fallen out of favour due to recurrence) and complete excision of the sublingual gland (Figs. 25.7 and 25.8).

25.1.9 Haemangioma and Varices

The use of the terms haemangioma and malformation can be confusing. The term haemangioma is often a generic one, commonly used to describe congenital hamartomas and vascular malformations. In general, true haemangiomas are developmental and are often recognised at an early stage. A true haemangioma is a benign vascular hamartoma of endothelial lined blood vessels. It is relatively common in infancy in the head and neck region and affects 5–10% of infants. They are rare in the oral cavity but may occur on the tongue, lips, buccal mucosa, gingiva and palatal mucosa. Clinically, they appear as a smooth or lobulated soft sessile mass which may vary in size from a few millimetres to several centimetres. They are usually

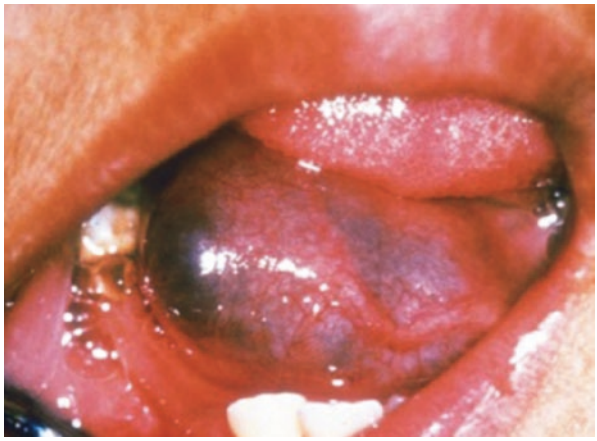

Fig. 25.7 Ranula

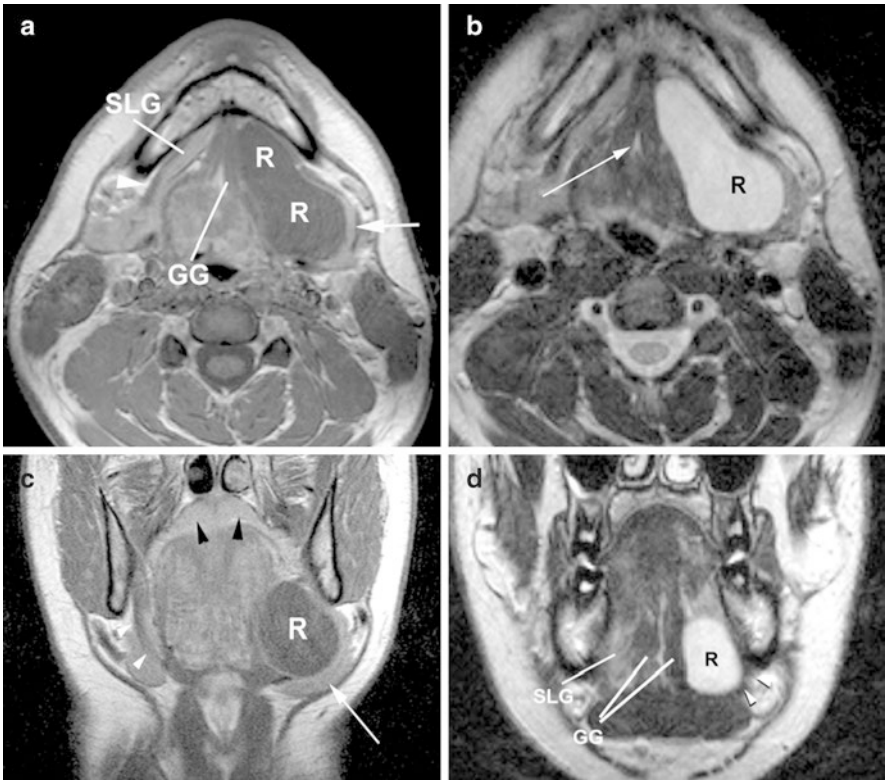

Fig. 25.8 Ranula, MRI

deep red in colour and may blanch on pressure. If large they can interfere with eating. Pedunculated haemangiomas of the oral cavity are rare. Appearances may mimic several other lesions include pyogenic granuloma, chronic inflammatory hyperplasia, epulis granulomatosa, telangiectasia and angiosarcoma. Histologically they are benign vascular proliferations composed of densely packed capillaries with endothelial cells and pericytes expanding in a lobular pattern.

If bitten or traumatised haemangiomas may bleed. In contrast to vascular malformations, infantile haemangiomas are usually absent at birth but then undergo a period of remarkably rapid postnatal proliferation. Haemangiomas follows a unique life-cycle of rapid growth, called the proliferating phase, followed by a slow spontaneous involuting phase. However it is still difficult to predict progression and some haemangiomas, even small ones, may result in aesthetic or functional handicaps. For this reason some clinicians suggest treatment should commence at the early stage. Treatment depends on the size of the lesion. If small (<1 cm) many can be successfully excised. If larger, treatment options include cryotherapy, sclerotherapy or embolisation. In children until recently, the mainstay of treatment was oral corticosteroid therapy. Propranolol has recently been shown to reduced these in infants. Other treatments include interferon, bleomycin or vincristine.

There is also an additional neoplastic form of haemangioma that appears in middle life or in older people. In the oral cavity these may occur in any area, at any age, without any racial or gender predilection. However more than half are found in patients over 40 years of age. These require surgical removal. A varix is another 'vascular' swelling common to the oral cavity. This is dilated vein. It occurs most commonly on the lip, buccal mucosa and ventral surface of the tongue. These are more common with increasing age and are possibly the consequence of trauma to the submucosal vessels. Lesions may be flat or raised, blue or purple and often blanch on direct pressure. Sometimes tiny pulsations can be palpated or observed. Those that fail to blanch are often thrombosed. The labial artery can also develop a small aneurysmal dilatation that appears as a pulsatile nodule. Treatment of both is by excision.

25.1.10 Orofacial Granulomatosis

Orofacial granulomatosis (OFG) is a condition in which the patient develops gradual, painless enlargement of the lips and peri-oral tissues, typically around adolescence. It can occur in isolation as granulomatous cheilitis, or it may also be linked to other granulomatous conditions, the most common being Crohn's Disease. Oral lesions may be the first presenting sign in patients with Crohn's Disease in 5 to 10% of cases. OFG can also present as a triad of symptoms which includes facial nerve weakness, lip swelling and fissured or furrowed tongue, referred to as Melkersson–Rosenthal syndrome. However, its precise pathogenesis has yet to be defined. Granulomatous cheilitis is a persistent relapsing-remitting, idiopathic condition in which the non-tender lips often feel firm and rubbery.

The cause of orofacial granulomatosis is still unknown, although several theories have been suggested, including infection, genetic predisposition, and allergy. Numerous aetiological agents, such as food substances, food additives, dental material microbiological agents have been proposed, but their pathogenesis is uncertain. Delayed hypersensitivity reactions may play a role, although the antigen inducing the reaction seems to vary in patients. Patients should be questioned regarding gastrointestinal symptoms and referred for a Gastroenterology opinion. Diagnosis of OFG is made by histological identification of non-caseating granulomas. Treatment tends to be conservative, involving dietary restriction (avoiding cinnamon and benzoate preservatives). Some toothpastes have also been implicated. Occasionally intralesion steroids, infliximab or debulking can be performed for massive and persistent swelling. Tumour necrosis factor-α (TNF-α) inhibitors have also been reported to be of benefit.

25.1.11 Amyloidosis

An increasing number of diseases are recognised to result from a failure of proteins to adopt their correct functional conformational states. These pathologic conditions are generally referred to as protein misfolding diseases. The largest group of

misfolding diseases is associated with the conversion of peptides or proteins from their soluble functional states into highly organized fibrillar structures, termed "amyloid." One resulting condition—amyloidosis—comprises a group of diseases in which this abnormal protein (amyloid) normally produced in the bone marrow, builds up in the soft tissues. To date, at least 28 different proteins have been identified as causative agents of amyloid diseases, ranging from localised cerebral amyloidosis in neurodegenerative conditions such as Alzheimer's and Creutzfeldt-Jakob diseases, to systemic amyloidoses such as immunoglobulin (Ig) monoclonal light chain amyloidosis (AL).

Secondary localised amyloidosis in the head and neck is a rare and generally benign condition. The most common sites of involvement are the thyroid, the larynx and subglottis, whereas in the oral cavity, amyloidosis usually tends to results in gradual swelling or involve the tongue or buccal mucosa. Systemic amyloidosis can involve the kidneys and heart and can present with fatigue, weight loss or congestive cardiac failure. Oral amyloidosis tends to appear as benign soft nodules with an overlying yellow, purple or blue mucosal discolouration. Diagnosis can only be made by biopsy. Once diagnosed, treatment is targeted to the organs involved (e.g., diuretics and dialysis), whilst surgery and laser excision may help to minimise localised symptoms, including dysphagia. Treatment may include high dose melphalan, a chemotherapy agent, followed by stem cell transplantation, although this is not always possible, depending on the age of the patient, progression of the disease and haematological markers. Combinations of chemotherapy and immunomodulatory agents are alternative treatments. Prognosis is variable with treatment, but if untreated the median survival is 1–2 years. Poor prognosis is associated with cardiac and liver involvement, neuropathy, or presence of underlying plasma-cell disease (i.e. multiple myeloma). In contrast, the prognosis of localised amyloidosis is much better.

25.1.12 Dermal Fillers

The use of fillers in the cosmetic industry is now common practice with the aim of increasing the volume of the injected area. The lips are therefore a commonly injected site. Dermal fillers come in many forms, from resorbable to permanent. In experienced hands the result are often very satisfactory and pleasing for the patient. However the process is not without risk. The most common side-effects include:

(i) Bruising and bleeding
(ii) Itching
(iii) Skin discolouration
(iv) Viral/bacterial infection
(v) Redness and swelling at the region of injection
(vi) Allergic reactions
(vii) Lumps under the skin
(viii) Skin ulceration in the injected site

(ix) Blanching and necrosis
(x) Migration of the filler
(xi) Hypertrophic scar

Most of these are uncommon. Allergic reactions are rare. Referral back to the original practitioner may be appropriate, however if the patient presents with infection and swelling or necrosis urgent referral to an on call 'facial' team (maxillofacial, plastics, dermatology) may be warranted. If there is abscess formation this will need to be drained. Migration of some fillers may also occur. If these are permanent this can result in palpable collections within the tissues of the lips (and sometimes cheeks).

25.1.13 Fibrous Dysplasia

This is a disorder of bone growth of unknown origin, in which normal bone and marrow are replaced by immature fibrous bone. This results in the formation of bone that is immature, weak and prone to expansion. It can occur in any part of the skeleton but the skull and face are commonly involved. Intra oral involvement in isolation is rare, but bony expansion in the upper jaw may encroach into to the mouth. Patients present with a smooth hard swelling or deformity usually in childhood or early adulthood, although it can continue throughout adult life. During the growth spurt this may become painful. Two types of FD are described.

- Monostotic (Involving a single bone, or adjacent bones, such as the upper and lower jaw
- Polyostotic (Involving many bones).

The most severe form of polyostotic fibrous dysplasia is known as McCune-Albright syndrome, which includes endocrine diseases (growth, sex, thyroid and steroid hormones) and skin pigmentation (café au lait macules). Fibrous dysplasia may also be associated with neurofibromatosis. The condition is said to burn itself out during puberty but exceptions are well known. The exact cause of fibrous dysplasia is unknown, but it is not a tumour. Diagnosis is usually made following by CT and bone biopsy. Treatment of fibrous dysplasia is mainly symptomatic. Bone pain can be treated symptomatically and in extreme cases surgical debulking may be required. Bisphosphonates have also been used with varying success with bone pain.

25.1.14 Paget's Disease

Paget's disease is a chronic condition of bone characterised by an abnormality in the normal bone remodeling process. This ability to 'turnover' bone is important for maintaining the normal calcium levels in our blood. Paget's disease typically begins with excessive bone resorption followed by an increase in bone formation. Two

types are described—monostotic and polyostotic. Usually the bones become fragile and misshapen. Patients may mention that previously well-fitting dentures (or hats) now seem too tight. Clinical examination may reveal excessive warmth from hypervascularity and deformity. Bone sarcoma can occur in less than 1 percent of people with Paget's disease.

Diagnosis of Paget's disease is made following imaging and laboratory tests. Urinalysis may show markers of bone turnover. There are also elevated levels of urinary and serum hydroxyproline. Bone scan will reveal "hot spots" and serum Alkaline phosphatase will be elevated. Treatment is not always required if asymptomatic. However, if the disease is active (indicated by an elevated alkaline phosphatase level) and is affecting high-risk sites, such as the skull or spine, bisphosphonates may be prescribed. Calcitonin may also be required. The outlook is generally good, particularly if treatment is given before major changes have occurred.

25.1.15 Dermoid Cyst

A dermoid cyst is an embryological malformation that can develop anywhere where fusion of tissues occurs during foetal development. The most popular theory regarding their aetiology is that they are derived from fragments of ectodermal tissue that become entrapped during fusion of the branchial arches. This explains their propensity to occur close to the midline and at other specific sites. They are however uncommon lesions in the head and neck and usually affect the periorbital region, floor of the mouth (FOM) and submental region, or the nose. The lateral eyebrow is the commonest site in the head and neck, but dermoid cysts in the oral cavity can also occur. These are most frequently located in the midline of the floor of mouth and are most likely caused by the retention of epithelium during development of the mandible and branchial arches. Cysts typically present during the second or third decade of life, although they can present shortly after birth. CT or MRI maybe needed to define the extent of the lesion. Complete surgical excision is the definitive treatment. Both ranulae and dermoid cysts commonly occur in the floor of the mouth. Clinically dermoid cysts are usually firmer than ranula. They do not normally transilluminate. The teratoid cyst is very rare in the head and neck but is important to recognise because of its malignant potential (Figs. 25.9 and 25.10).

25.1.16 Calculi (Stones)

Stoney-hard lumps in the floor of the mouth or buccal mucosa are most likely to be calculi within the duct of the submandibular or parotid gland. These have typical features—they feel like stones. However further back in the mouth, where the duct is deeper, this may not be so easy to determine. Many, but not all stones are radioopaque and can be seen on a plain X-ray. Other investigations include US or

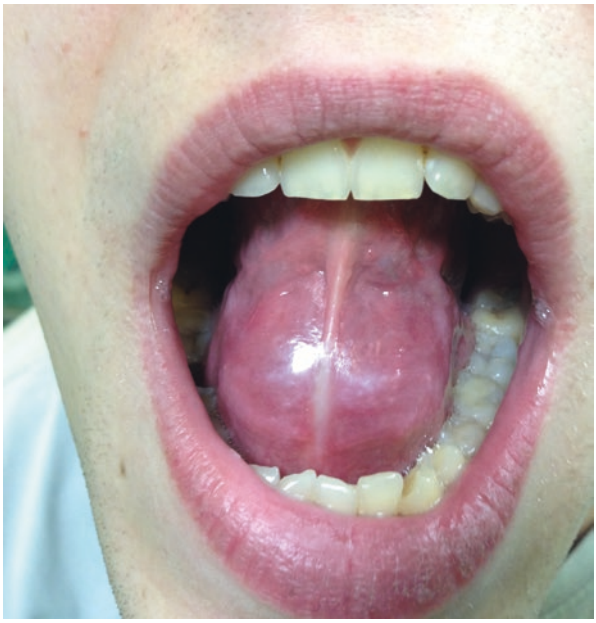

Fig. 25.9 Large dermoid cyst floor of mouth

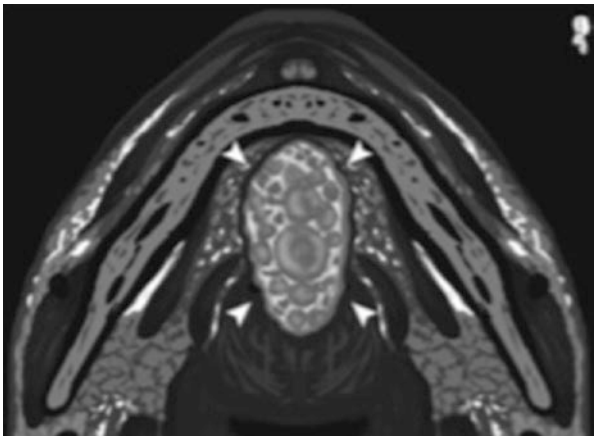

Fig. 25.10 Axial TI-weighted MR illustration shows a medial isointense cystic mass containing multiple mildly hyperintense small masses. This appearance is typically seen in dermoid and epidermoid cysts resulting in a "sack of marbles" appearance (*arrowheads*)

sialogram. Treatment involves removal, either surgically or endoscopically. Small stones may occasionally pass spontaneously (Figs. 25.11 and 25.12).

25.1.17 Submucosal Fibroma/Lipoma

Submucosal fibroma or lipomas are similar to fibroepithelial polyps in that they present as discrete, smooth, mobile lumps, typically within the substance of the lip, cheek or tongue. However, these lumps are not pedunculated due to their deeper location.

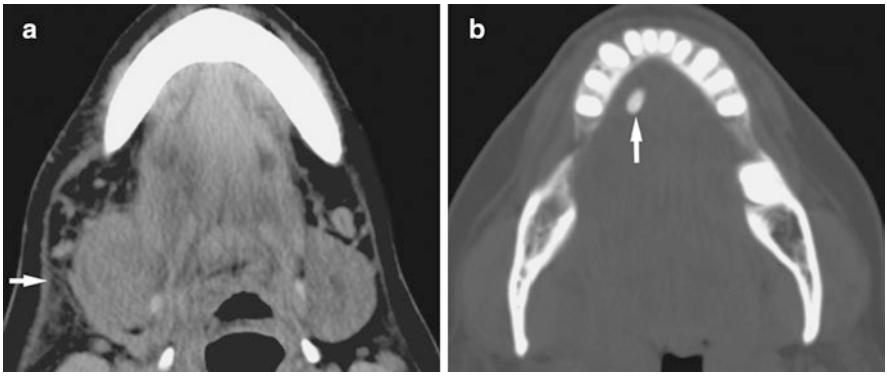

Fig. 25.11 Submandibular sialoadenitis due to duct stone; 59-year-old female with right submandibular swelling. (**a**) Axial post-contrast CT image shows enlarged submandibular gland with stranding and reticulation of periglandular fat (arrow). (**b**) Axial CT image shows stone in anterior part of Wharton s duct (*arrow*)

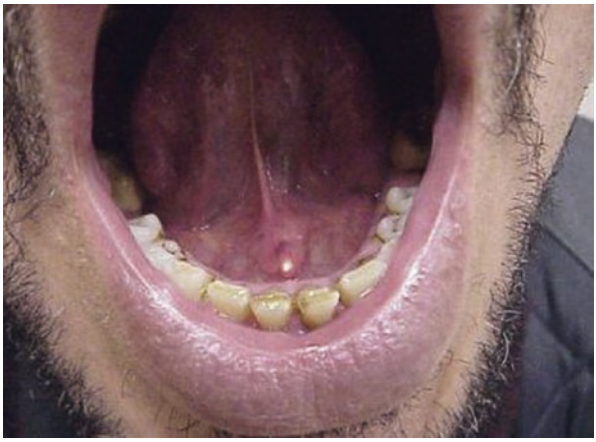

Fig. 25.12 Stone in submandibular duct

Approximately 15% to 20% of lipomas are found in the head and neck with 1% to 5% affecting the oral region including the buccal sulcus, lips, cheek, floor of mouth (FOM), tongue, and retro- molar trigone (RMT). Treatment is excision.

25.1.18 Papilloma

These are benign pedunculated warts which can occur anywhere on the body, including the fingers, genitalia, and oral cavity. They often arise following viral infection with human papillomavirus (HPV). Oral papillomata are generally found on the tongue, buccal mucosa, palate and oropharynx. Malignant transformation is very rare. If desired, treatment is excision. Other alternatives can include cryotherapy and laser ablation, particularly with multiple lesions.

25.1.19 Epulis

An epulis is any enlargement or tumour arising from the gum. They a typically enlargements on the gingiva. The three types are fibromatous, ossifying and acanthomatous.

25.1.19.1 Pyogenic Granuloma

A type of epulis, pyogenic granuloma is a misnomer because it is neither produces pus, nor are there granuloma. It is instead a vascular rich lesion that occurs on both mucosa and skin. It can presents as an inflamed lump, often on the lip or gingiva, particularly the interdental papillae. This develops as a result of irritation. It is common during pregnancy when hormonal changes trigger an exaggerated inflammatory response to dental plaque. Pyogenic granuloma can bleed profusely if traumatised and as such may warrant removal.

The prognosis is good, although recurrence may occur in up to 10% of patients (Fig. 25.13).

25.1.19.2 Pregnancy Epulis

Pregnancy tumour or granuloma gravidarum is identical to a pyogenic granuloma in all respects apart from the fact that it occurs exclusively in pregnancy. Usually there is also pregnancy gingivitis. If occurring during pregnancy is prudent to wait until after delivery. If there is no spontaneous regression then surgical removal is indicated.

25.1.19.3 Fibrous Epulis

Most commonly occurring on the gingiva near between two anterior teeth, it may be sessile or pedunculated. It is composed of fibrosed granulation tissue. They are firm and rubbery with a pale pink colour.

25.1.19.4 Ossifying Fibroid Epulis

Over time bone may form within a fibrous epulis lesion. This is then termed an ossifying fibroid epulis or peripheral ossifying fibroma.

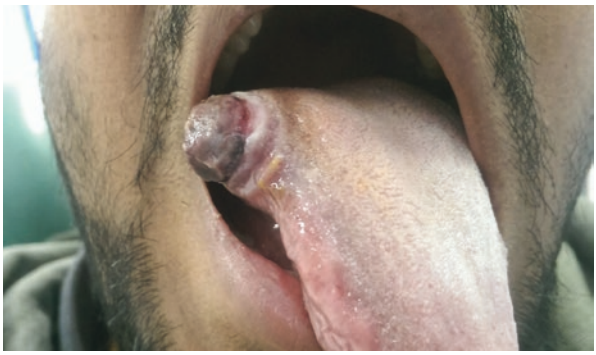

Fig. 25.13 Huge pyogenic granuloma following repeated trauma

25.1.19.5 Giant Cell Epulis

If an epulis contains giant cells it is termed a giant cell epulis or a peripheral giant cell granuloma.

25.1.19.6 Congenital Epulis

Congenital granular cell tumour or Neumann's tumour is a rare benign oral tumour which presents in the newborn. It usually appears as a single pedunculated or sessile, smooth or lobulated, rounded, firm, elastic lump with normal overlying mucosa, though occasionally several lesions may be present. The appearance can be distressing to the family of the newborn and presence can cause obstruction to the airway and feeding. CE may spontaneously regress with time though where size causes disturbance prompt surgical removal is required (Figs. 25.14 and 25.15).

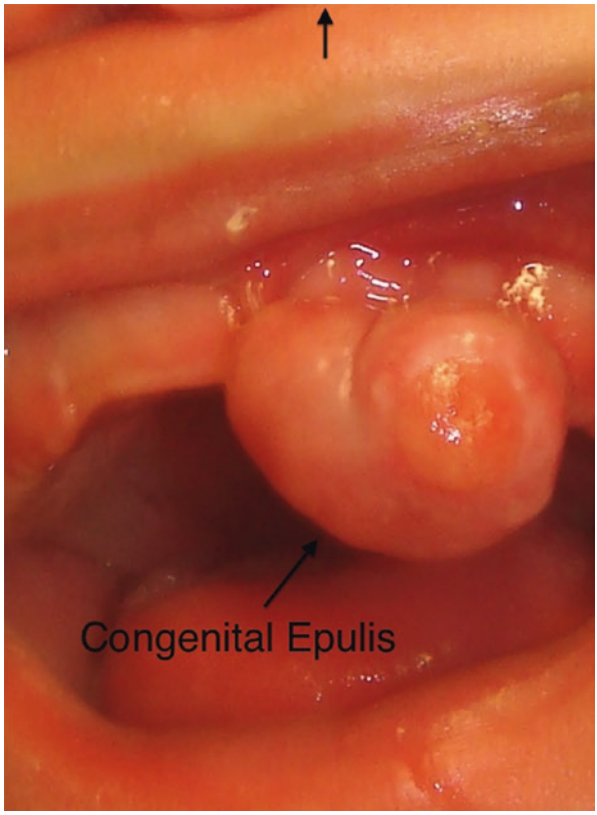

Fig. 25.14 Congenital epulis

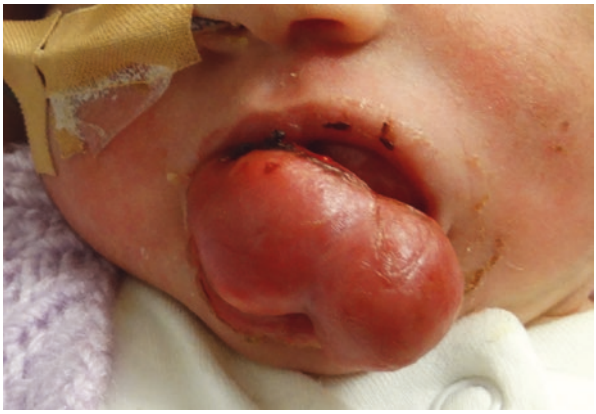

Fig. 25.15 Congenital epulis

25.1.20 Gingival Hyperplasia

Generalised gingival hyperplasia presents as a diffuse overgrowth and thickening of the gums and is classified according to its aetiology:

(i) Inflammatory
(ii) Drug induced
(iii) Enlargement associated with systemic diseases or conditions
(iv) Neoplastic
(v) Familial

Inflammatory enlargement occurs in response to the build up of plaque and poor oral hygiene. Drug induced enlargement is particularly associated with calcium channel blockers, anticonvulsants and cyclosporine. If the medications can be safely discontinued, the excess gingival tissue can be surgically trimmed. Enlargement associated with systemic diseases include vitamin C deficiency, pregnancy, puberty, leukaemia, granulomatous disease and neoplastic disease. Familial gingival enlargement is a rare inherited disorder. The enlarged gingivae are firmly fibrotic and not inflamed. Also termed hereditary gingival fibromatosis it may be associated with learning difficulty, epilepsy and hypertrichosis. It involves the full width of the attached gingivae (Fig. 25.16).

25.1.21 Salivary Gland Tumours

These occasionally present as a slowly growing lump in the palate or upper lip. Although they are solid, palatal tumours do not feel bony hard. This helps distinguish them from palatal tori. Tori also tend to occur in the midline of the palate. The differential diagnosis includes odontogenic cysts and other tumours. Salivary glands

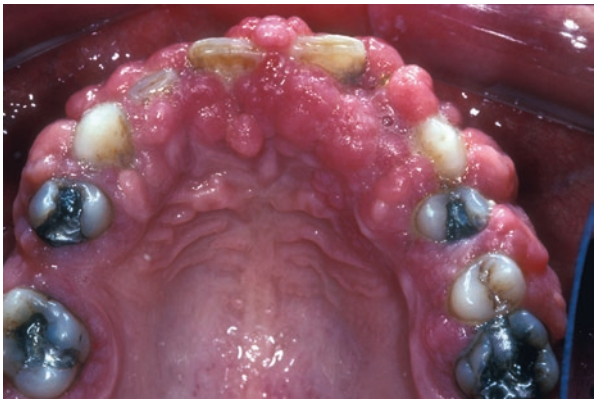

Fig. 25.16 Drug induced gingival enlargement

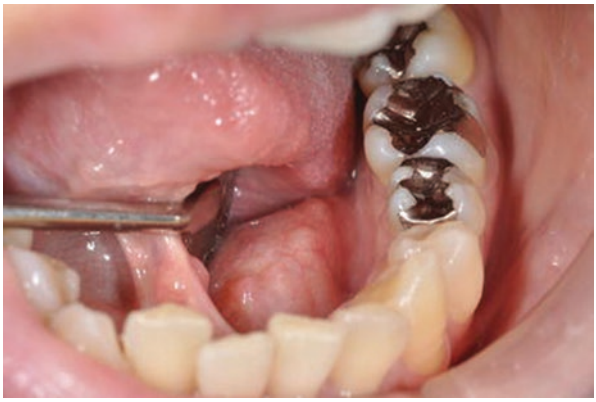

Fig. 25.17 An adenoid cystic carcinoma of the sublingual gland

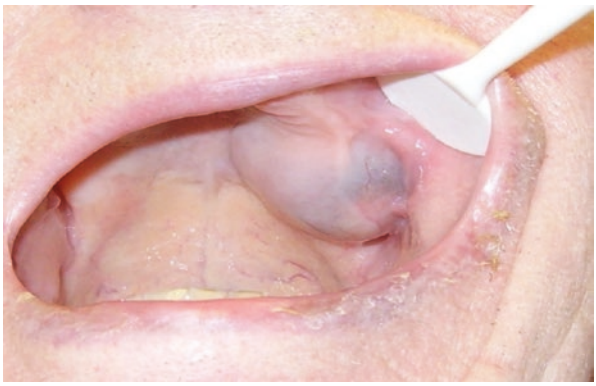

Fig. 25.18 Mucoepidrmoid tumour

tumours arising from the minor salivary glands are more likely to be malignant, so urgent referral is required. Management is surgical excision (Figs. 25.17, 25.18, 25.19, 25.20, and 25.21).

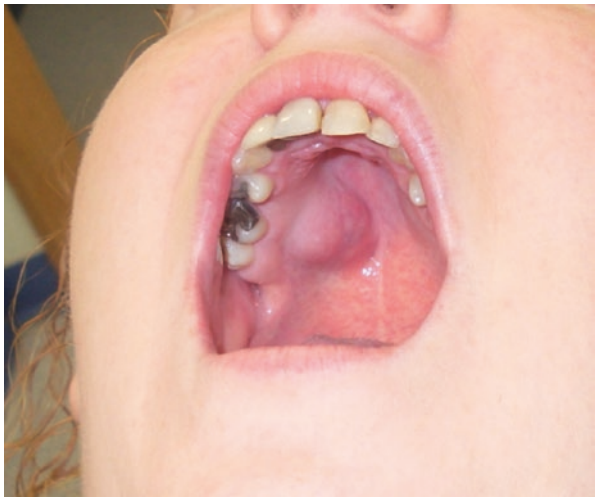

Fig. 25.19 PSA palate

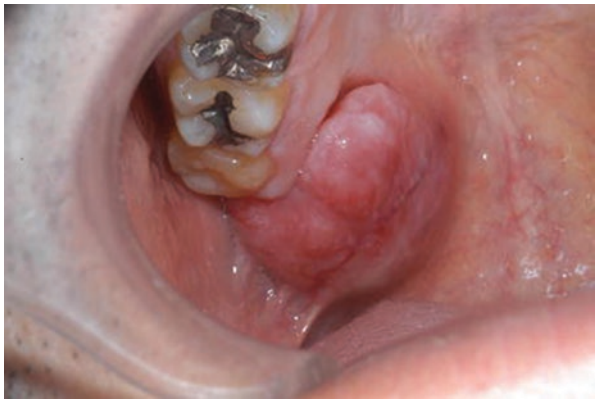

Fig. 25.20 A mucoepidermoid carcinoma (poorly differentiated type with high grade malignancy) of the palatine gland

25.1.22 Unerupted Teeth

Firm lumps under the gingivae may be due to unerupted teeth. This is the commonest cause of a lump in children. Wisdom teeth may also present as unerupted lumps. Supernumerary or ectopic teeth (such as unerupted palatal canines) may also be palpable. Diagnosis is best achieved radiographically with an OPG. This will show the presence and location of unerupted teeth. If the teeth do not have sufficient space to erupt into spontaneously, extraction or surgical exposure may be indicated. In children, this treatment plan is usually decided by an orthodontist in conjunction with an oral and maxillofacial surgeon. It is important to note that unerupted teeth can sometimes undergo cystic change and may become infected (Fig. 25.22).

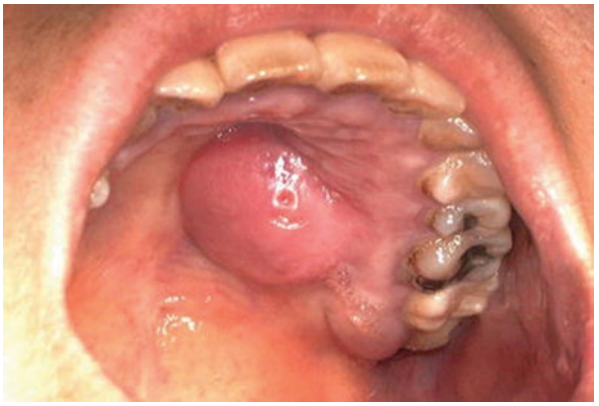

Fig. 25.21 A pleomorphic adenoma of the hard palate with small ulceration of the covering mucosa

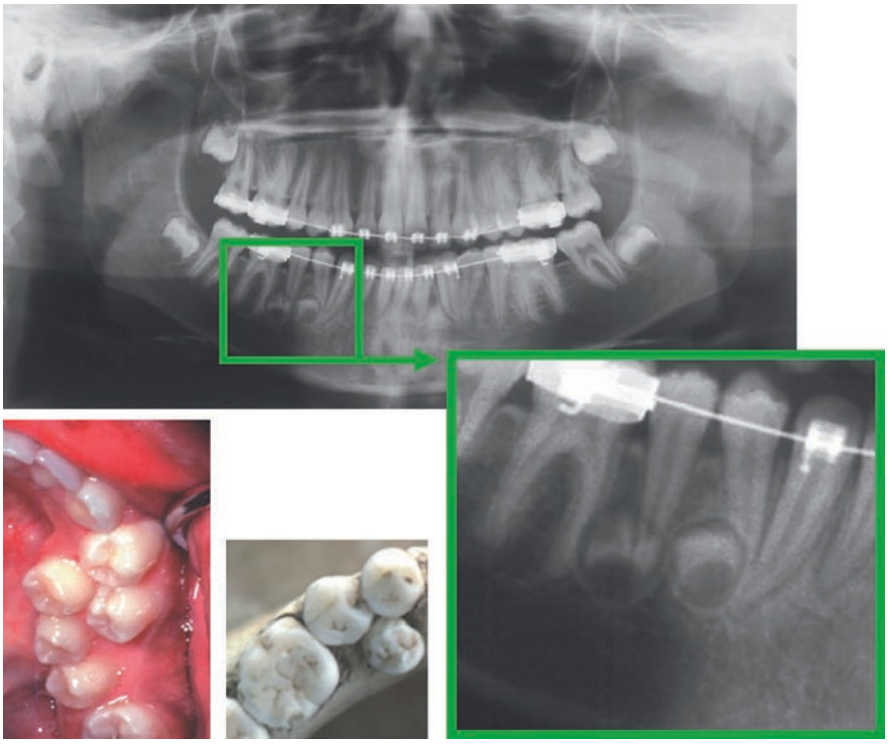

Fig. 25.22 Post-dentition supplemental supernumerary premolars are illustrated in the panoramic radiograph. The clinical photograph shows dental malocclusion occurring in a patient having three such supplemental teeth that have erupted. The dried jaw specimen is of an ancient Indian jaw more than 1,000 years old (Mississippian) showing an erupted supplemental premolar tooth

25.2 Bleeding from the Mouth

Non-traumatic bleeding from the mouth is common, typically mild and most likely to be coming from the gums (gingivae). The most common cause of oral bleeding is gingivitis. Minor bleeding may be noticed on brushing the teeth in the morning. This sort of bleeding rarely presents to the emergency department. However, it is important to be aware of rare but serious causes, notably local and haematological malignancies. In these disorders, bleeding can be an early or presenting sign.

25.2.1 Gingivitis/Periodontitis

Periodontology is the study of the tooth-supporting tissues, the "periodontium". This functional structure is comprised of the tissues that surround and anchor each tooth to the supporting alveolar bone. These include

(i) Gingiva
(ii) Periodontal Ligament
(iii) Root Cementum and
(iv) Alveolar bone

Many diseases affect the periodontium. By far the most common is plaque-associated gingivitis and periodontitis. Indeed, these have been described as the most common diseases in the world, affecting (to varying extents) anyone who has teeth. Gingivitis is inflammation of the gingiva. This is a clinical diagnosis. Untreated this results in redness and swelling of the gums, especially the interdental papillae. It can be classified by its appearance into ulcerative, haemorrhagic, necrotising and purulent. Many well known conditions or states can predispose to gingivitis including (i) drug-induced, (ii) hormonal, (iii) nutritional, (iv) infections, (v) plaque-induced and (vi) smoking. The most common type of gingivitis is a chronic form induced by plaque, but acute gingivitis can also occur. Gingivitis typically presents with diffusely inflamed gums, which bleed on tooth brushing or spontaneously. It is limited to the superficial structures and is usually diagnosed when there is bleeding on probing the gingival crevice. Many patients report a bad taste in the mouth in the morning. Inflammation can be exacerbated during pregnancy as a result of the hormonal changes that occur (Fig. 25.23).

Periodontitis can develop from pre-existing gingivitis, especially in immunocompromised patients. Smoking and stress are well known risk factors. Inflammation in the gingiva extends into the deeper tooth-supporting structures. Destruction of the collagen in the periodontal ligament and gradual loss of the surrounding alveolar bone results in "pocketing", which extends down the root. This pocket then acts as a reservoir for bacteria, which sustain the inflammatory processes. Gingival recession occurs later with exposure of the root and hypersensitivity of the tooth.

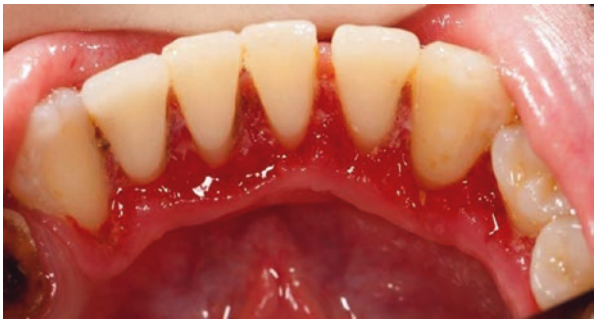

Fig. 25.23 Gingival erosion and bleeding

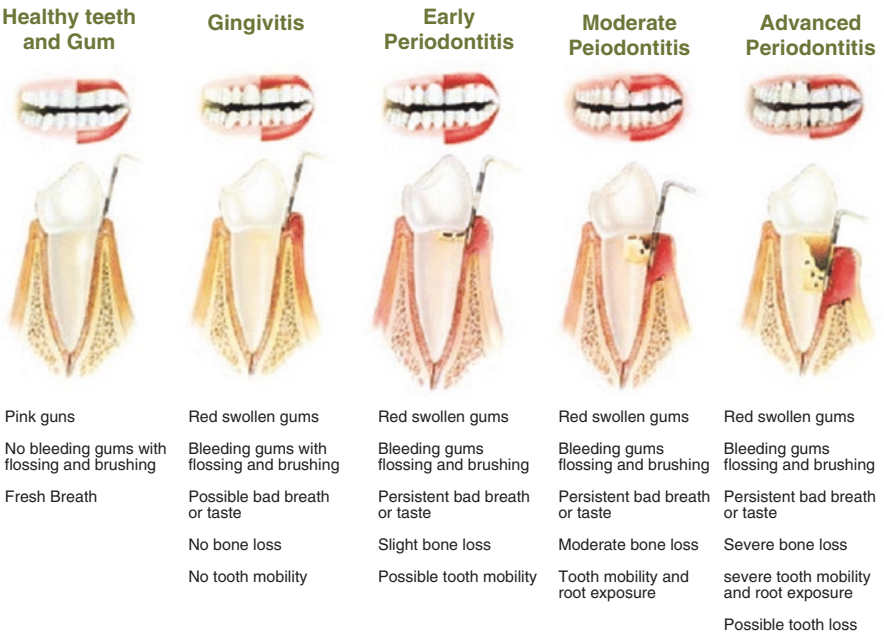

Fig. 25.24 The different stages highlighting the progression of periodontal disease (Reprinted with permission from the Advanced Institute of Oral Health, Brentwood, TN)

Periodontitis may be classified as chronic or aggressive, with varying degrees of severity. Approximately 90% of cases are chronic periodontitis. In the early stages, chronic periodontitis has few symptoms. Over time the following develop

(i) Redness or bleeding from the gums, especially when brushing teeth
(ii) Gingival swelling
(iii) Halitosis, or a persistent metallic taste in the mouth
(iv) Gingival recession.
(v) Loose teeth (Figs. 25.24 and 25.25)

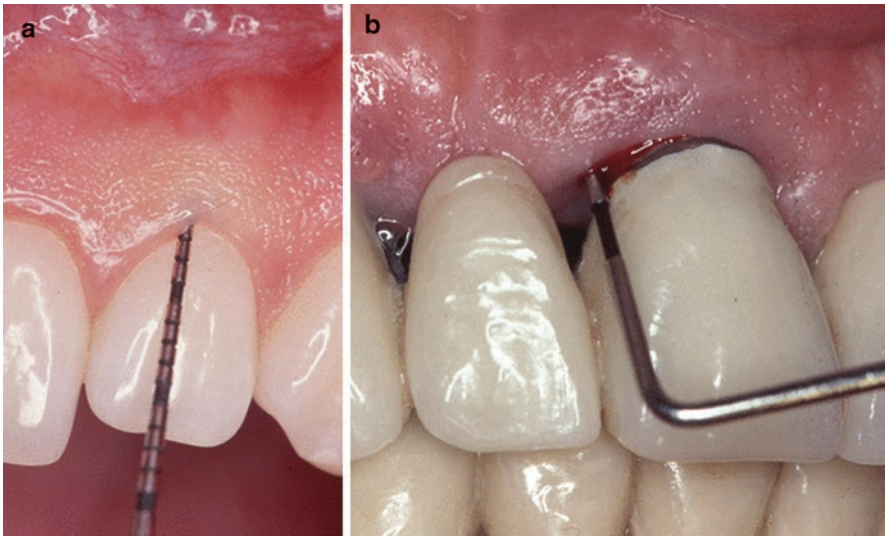

Fig. 25.25 The clinical evaluation of periodontal disease by probing. (**a**) Healthy gum and (**b**) diseased gum, bleeding upon probing (Reprinted with permission from the *Journal of the California Dental Association*, 2013 February; 41(2): 119–24)

Treatment of both gingivitis and periodontitis is requires the skills of a dental hygienist, dentist or periodontist. This initially involves intensive scaling, oral hygiene instruction and chlorhexidine mouthrinse, but may require more complex treatments, or extraction of any unsalvageable teeth.

25.2.2 Desquamative Gingivitis

Despite the name "gingivitis", this is an entirely different pathological problem. In some patients desquamative gingivitis can be a clinical manifestation of one of several important systemic diseases. The gingiva develops a number of characteristic appearances, including a "fiery red", glazed, atrophic or eroded form. Its superficial mucosa can desquamate following minimal trauma, hence the name. In contrast to simple gingivitis, desquamative gingivitis is often painful, affecting the buccal/labial gingiva predominantly. Many cases are secondary to mucocutaneous conditions, such as lichen planus, pemphigoid and pemphigus. Other causes include allergic reactions to toothpastes/mouth rinses, Crohn's disease, psoriasis, linear IgA disease and chronic ulcerative stomatitis.

25.2.2.1 Lichen Planus

This is described in greater detail under "White patches". Diagnosis of oral lichen planus can be difficult if the gingiva is the only site of involvement. Careful examination of the erythematous gingiva may reveal faint keratotic lines. If lichen planus

is suspected the rest of the oral mucosa and skin should be examined (looking for pruritic, purple papules on the flexural surface of the wrist). Involvement of the genital mucosa and the gingiva, especially in females has also been reported (vulvovaginal-gingival syndrome). Treatment includes careful oral hygiene and topical corticosteroids. This can be applied in many forms, including topical beclomethasone spray. Systemic tetracycline and recently topical cyclosporin have also been reported.

25.2.2.2 Immune-Mediated Blistering Diseases (Vesiculobullous Disorders)

These are traditionally divided into intra-epithelial and subepithelial disorders, based on the histological depth of the bullae. The most common presentation of mucous membrane pemphigoid (MMP) intraorally is desquamative gingivitis. Mucous membrane pemphigoid is defined a group of chronic autoimmune diseases, in which there is inflammation and subepithelial blistering, predominantly affecting mucous membranes. Antibodies are produced against antigens in the basement membrane, resulting in the epithelium becoming detached from the underlying lamina propria. Mucous membrane pemphigoid mainly affects the oral cavity, larynx, oesophagus and ocular membranes (hence the name). Rarely does it involve the skin. It usually affects females in their sixth decade, although males and other age groups can also be affected. Superficial painful erosions develop on the gingiva, and sometimes the buccal mucosa, palate, alveolar ridge and tongue. Rarely blood-filled bullae may be seen on the palate. These lesions tend to heal without scarring. However other mucosal sites (notably the conjunctiva) do not. The eyes can be affected in up to 80% of cases with 15% becoming blind. Strictures may also develop in the larynx, oesophagus and genital mucosa. Diagnosis of MMP usually requires a biopsy. In some cases stroking the epithelium induces a blister (Nikolsky's sign). Because the gingiva can be the only site involved, this can result in delayed diagnosis. Once the diagnosis is confirmed, an ophthalmologic opinion should be sought, even if patients have no ocular symptoms. Oral lesions are usually mild and can be controlled with topical corticosteroids. If other mucosal sites are involved, more potent medication may be necessary (systemic corticosteroids, azathioprine, or dapsone).

Pemphigus, linear IgA disease, dermatitis herpetiformis and erythema multiform may also present with desquamative gingivitis. Pemphigus (vulgaris) is rare, but is a potentially fatal mucocutaneous disease characterised by intra-epithelial bullous formation. It usually affects females in their fourth to fifth decade of life and the mouth can be the first sign of presentation. Bullae develop, but quickly rupture resulting in irregular painful erosions. Any site within the mouth subjected to trauma may be involved, especially the palate, tongue and buccal mucosa. There may be severe desquamation of the gingiva, even bullae. Other sites that can be involved include the oesophagus, pharynx, larynx, nasal and genital mucosae. Although ocular lesions can occur in pemphigus, they are uncommon and tend to be transient. Before the use of steroids the mortality was high, at around 30%, secondary to electrolyte loss and sepsis. Definitive

diagnosis requires a biopsy of intact epithelium. This can be difficult as the epithelium is usually very friable. Early diagnosis is important as this is a serious condition. Oral lesions can be quickly followed by the skin or other mucosae. Urgent referral to a dermatologist is therefore important. Systemic steroids are the usual treatment. Once the disease is under control, these can be withdrawn and 'steroid-sparing' drugs used (azathioprine, cyclosporin, cyclophosphamide and methotrexate). Treatment may need to be lifelong. Isolated desquamative gingivitis can be treated with topical steroids.

Linear IgA disease is a rare vesiculobullous disease characterised by deposition of IgA autoantibodies in a linear pattern (hence the name) along the basement membrane zone. Oral lesions are similar to mucous membrane pemphigoid. The gingiva can develop diffuse desquamation with non-specific ulcers on the palate, tongue and buccal mucosa. Treatments include topical or systemic steroids, dapsone or sulphapyridine. Chronic ulcerative stomatitis and plasma cell gingivitis are rare conditions which can affect the gingiva.

25.2.3 Infections

Dental and oral infections can also present with discrete areas of intraoral bleeding. This is discussed later. Diagnosis is usually self evident as the patient often complains of swelling, pain and may have a fever. Take an OPG to exclude intra bony causes, or pathology. Treatment depends on the source of the infection and may include surgical drainage and antibiotics. These are discussed elsewhere in relation to the various pathologies.

25.2.4 Oral Cancer

Oral cancer is common and is usually seen in heavy smokers and drinkers. This is discussed elsewhere in this chapter. Most cancers are squamous cell carcinoma (SCC), which may be confined solely to the mucosa, or may invade the deeper tissues. When this occurs, bleeding is more likely to occur. Due to the friability of the affected mucosa, these lesions also tend to bleed spontaneously or easily on examination. Most early cancers present as an indurated or friable looking painless ulcer, which may be raised or have rolled edges. However appearances can vary widely. Nevertheless, in advanced cases, the diagnosis is usually relatively obvious on examination. Biopsy of course is still required. The tissues can often be very friable and can result in a lot of trouble some bleeding as a result of local irritation or following biopsy. Lesions may ooze for hours following a biopsy due to the difficulty in suturing fragile tissue. Most oral cancers occur around the ventrolateral surface of the tongue, floor of mouth, retromolar region and alveolus (Figs. 25.26, 25.27, and 25.28).

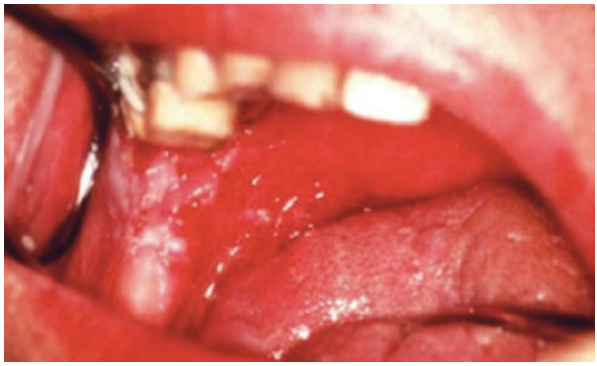

Fig. 25.26 Cancer of the retromolar trigone

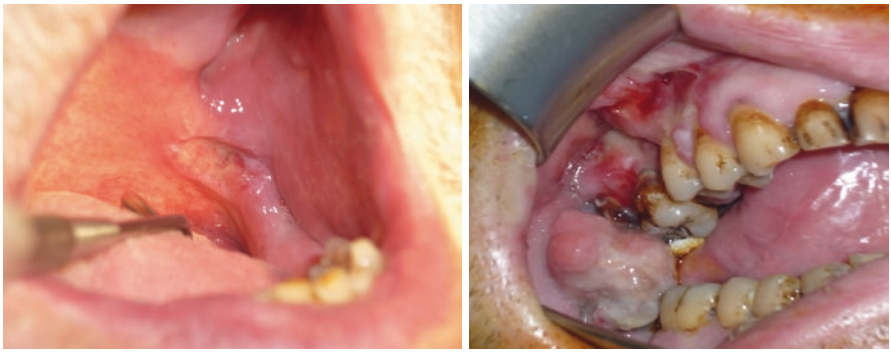

Fig. 25.27 Typical features of malignant ulcer

25.2.5 Antiplatelet and Anticoagulant Medication

Patients are often prescribed aspirin or other types of anticoagulants for a variety of reasons, including prophylactic measures against stroke. Therefore consider the possibility of anticoagulant induced bleeding in any patient with a complex medical history, or any elderly patient presenting with unexpected or significant oral breeding. If the patient is discovered to be taking such medication, a coagulation screen should be performed. Unfortunately, with some of the newer anticoagulant drugs, measurements of therapeutic levels are not possible. This is discussed further in chapter en general assessment. Overdose or dietary/drug interactions can result in enhanced effects of anticoagulants and spontaneous bleeding. Remember also that over the counter herbal remedies can also interact with prescribed medication. In warfarinised patients with a significantly elevated INR, discuss urgent management with haematology. Vitamin K may be required, but this should be decided on a case-by-case basis, due to its prolonged affects on the INR. Preoperatively, an INR of less than 4.0 is usually low enough to safely perform simple tooth extractions, so long as

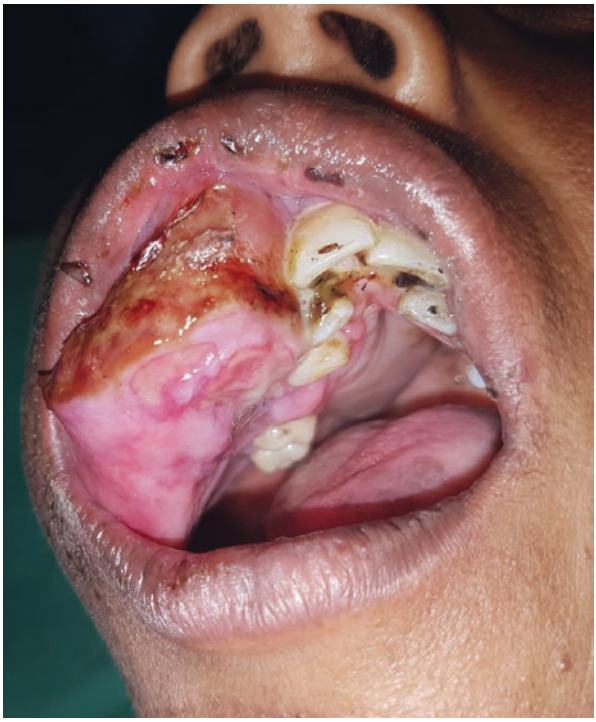

Fig. 25.28 Neglected tumour

local measures to achieve haemostasis (such as surgicel in the extraction socket and suturing of the associated gingivae) are undertaken. Injection of adrenaline containing local anaesthetic can sometimes help control bleeding from a discrete area. Biting on a tranexamic acid soaked gauze swab is also often effective. Some patients may require a tranexamic acid oral rinse, which can then be swallowed. Advice from a haematologist may be necessary for the new novel anticoagulant medications.

25.2.6 Haematological Disorders

Many patients with these disorders who present with oral bleeding, will be aware of their condition and already have management in place. Local measures to control bleeding should be used and discussion with a haematologist necessary. However in some patients, especially children, spontaneous bleeding, or disproportionate bleeding from an injury, may be the first sign of an underlying condition. These include, leukaemia, thrombocytopenia, haemophilia, Von Willebrand disease and other factor deficiency states. If oral bleeding is from an unknown source, enquire about unintentional weight loss, malaise and night sweats. If a haematological malignancy is suspected, look for anaemia, lymphadenopathy and hepatosplenomegaly. Inspect the skin for purple bruises (purpura)—this may be seen in Idiopathic thrombocytopenic purpura (ITP). If a coagulopathy is suspected, the patient should have FBC,

LFTs and a coagulation screen. In males where Haemophilia is suspected, testing of Factor VIII and IX levels should be requested.

Other conditions which can result in oral bleeding are discussed in the section on Ulceration and blistering.

25.2.7 Management of Oral Bleeding

Carefully assess the patient with a good light to find the site of bleeding. If you are uncertain ask the patient to rinse their mouth. In most cases bleeding can be controlled with pressure from a gauze swab, or tranexamic acid soaked swab. Bleeding is usually from multiple small vessels. If it is pulsatile in nature and not as a result of trauma, suspect and AV malformation. Management of this require immediate specialist investigations and input. This is rare. In all other cases tranexamic acid mouthwash is often very useful. Tranexamic acid acts as an antifibrinolytic. It inhibits activation of plasminogin to plasmin thereby preventing clot degradation. If this does not control bleeding after 40 min of pressure, further investigations as previously noted may be necessary. Consider anticoagulant overdose, haematological disorders and undiagnosed malignancy. Very rarely bleeding from the gums can occur as a result of nutritional deficiencies, notably scurvy.

Bleeding following tooth extraction may require additional local measures. Following removal of any soft clot, place haemocollagen or surgicel into the tooth socket and suture the gingivae to apply pressure. Replace the gauze and re-apply pressure. In the case of gingival or post biopsy bleeding consider the use of cauterisation either with silver nitrate or monopolar/diathermy. Desmopressin (DDAVP) is commonly used in patients with Von Willebrand Disease, Haemophilia, or Thrombocytopenia. Thrombocytopenia is also treated by steroids and very occasionally, if necessary, with platelet transfusion. Haemophilia may require appropriate factor replacement therapy. Management of bleeding patients taking anticoagulation or with haematological disease should only be undertaken following local protocols or specialist advice. All unexpected cases of oral bleeding, or those without an obvious diagnosis should be discussed with an appropriate oral specialist. Patients often require investigation to look for a underlying cause.

25.3 Dental Caries, Toothache and Dental Abscesses

In all mouths, bacteria can quickly proliferate in dental plaque as a “biofilm”. Even following professional cleaning this is seen to start within minutes. Over time, lactic acid and other enzymes are produced, which slowly break down the calcified tissues of the teeth. Although the mouth contains a wide variety of bacterial species, only a few of these are known to cause decay (caries). Sugars are the primary food source for bacteria, which is why diets high in sugar are a risk factor. This is in essence the cause of dental decay. “Stephan’s curve”, well known to dentists, shows that a sudden decrease in plaque pH occurs following a glucose mouth rinse. This returns to

normal after 30–60 min. When the pH falls below 5.5 demineralisation of calcified dental tissue occurs. Untreated, caries eventually erodes through the enamel and dentine of the tooth into the pulp cavity and the ingress of bacterial results in pulpitis. Because the pulp chamber is a confined rigid space, a tiny compartment syndrome effectively occurs, resulting in considerable pain and eventually necrosis of the pulp. Infected material can then pass through the apex of the root(s) of the tooth into the periodontal and periapical tissues and spread to the alveolar bone—resulting in an periapical abscess.

In its early stages, caries is symptom free. Only when the dentine is exposed (which has fine channels—dentinal tubules- passing to the pulp) does symptoms occur. These include pain, especially on drinking hot, cold or sweet drinks. With the eventual death of the pulp tissue, the tooth looses this sensitivity, but may remain tender to pressure. Pus from the tooth may then extend into the surrounding bone resulting in further pain. Treatment of caries involves removal of all the decayed tissue and restoration of the tooth with dental material (a "filling") (Fig. 25.29).

Dentin hypersensitivity is a relatively common condition. This is dental pain which arises from exposed dentin. It usually occurs when stimulated by hot, cold, pressure (including tooth brushing) or chemicals (acidy drinks). The pain is usually sudden and sharp and short in duration. Receding gums, acid erosion, dental bleaching and smoking all expose the microscopic dentinal tubules, which are normally covered and protected by the gingiva. Changes that then occur in the flow of fluid within the tubules irritates the nerves in the pulp, resulting in pain. Inflammation of the dental pulp (pulpitis), is classified as reversible and irreversible—the latter occurs when inflammation irreversibly progresses to necrosis. With irreversible pulpitis severe pain is poorly localised and continues after the stimulus has been removed. Spontaneous pain may also occur.

A dental abscess (dentoalveolar abscess), is a localised collection of pus related to the tooth. This can take several forms. It is commonly seen at the tip of a root (periapical abscess) following pulp necrosis and pulpitis. The tooth is usually non-vital. Periodontal abscesses arise in the periodontal ligament. A pericoronal abscess (or pericoronitis) is inflammation involving the soft tissues surrounding the crown of a partially erupted tooth (usually a lower wisdom tooth). In all these forms the abscess may be acute or chronic, painful or painless. The quality of the pain can

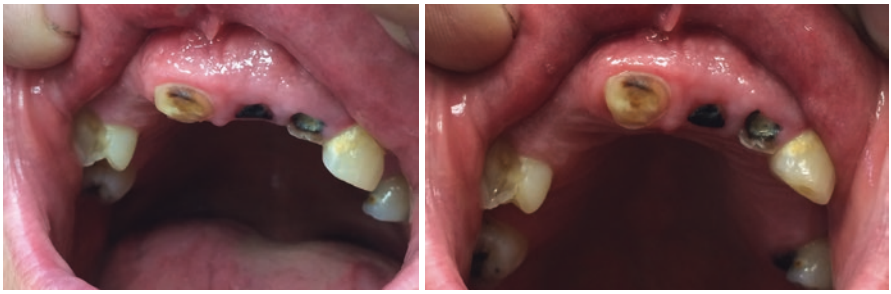

Fig. 25.29 Severe caries with loss of crowns

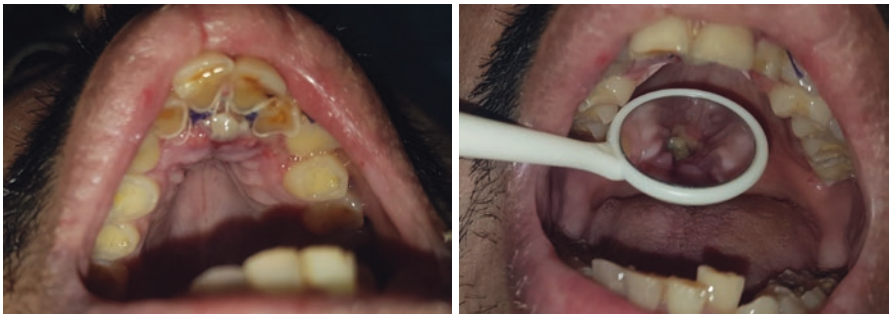

Fig. 25.30 Chronic dental abscess

vary significantly. Tapping on the tooth may induce extreme pain. In some patients, the abscess may perforate the overlying bone and drain into the surrounding tissues, forming a chronic fistula. Lymphadenopathy may also occur. If left untreated, abscesses can become chronic, or they can flare up resulting in a fascial space infection. Both osteomyelitis and cellulitis can also occur. Depending on the severity of the infection, the patient may feel only mildly ill, or may require admission and urgent drainage. This is discussed further in the chapter on the lower jaw (Fig. 25.30).

25.3.1 Draining Sinus

Commonly referred to as a gum boil, any "boil" or painful lump, next to a tooth is often a sign of dental infection. Pus from the periapical area of a chronically infected tooth passes through the bone to eventually discharge into the mouth. In rare cases this can present extraorally as a persistent pimple or sinus. Surgical removal of the fistula will only result in recurrence if the causative tooth remains untreated. Treatment of the offending tooth is usually extraction or root canal treatment. An OPG X-ray will show the infected tooth associated with a periapical radiolucency. At times the draining sinus may not be in direct proximity with the causative tooth. When this is the case a rubber gutta percha point can be placed in to the sinus until resistance is met. A periapical radiograph can then be taken and the radiopaque gutta percha point will have advanced towards the root of the causative tooth.

25.3.2 Progression of Infection

Whilst extensive progression of infection is relatively uncommon with most dental abscesses, if left untreated or occurring in immunocompromised patients, extension within and beyond the jaw is possible. Dental infection and tonsillitis are the two commonest causes of cervical fascial space infections (discussed in the chapter on the front of the neck) and dental infection is the commonest cause of Ludwig's angina (also discussed in the chapter on the lower jaw). Therefore when assessing a

patient presenting with oral infections and facial swelling it is important not to forget the markers that may suggest the patient is septic, or has a systemic inflammatory response syndrome (SIRS). Two of the following would make a patient positive for this:

(i) Temperature below 36 °C or above 38 °C
(ii) Heart rate greater than 90
(iii) White cell count less than 4 or greater than 12
(iv) Respiratory rate greater than 20

Other and newer definitions for sepsis and septic shock have also been proposed. The Sepsis 3 consensus provides a new screening tool for sepsis. Quick Sequential Organ Failure Assessment (qSOFA) has been proposed to predict the likelihood of poor outcome in out-of-intensive care unit patients with clinical suspicion of sepsis. If the patient is positive for SIRS with a history or signs of infection then he/she should be considered to be septic. If this is the case oxygen should be administered, blood cultures taken, haemoglobin and lactate checked as well as routine bloods. Intravenous antibiotics and fluid therapy should be commenced and the patient urgently referred.

25.3.3 Dental Pain

For the non-dentally qualified practitioner the diagnosis and triage of toothache can be very difficult. Determining the need for antibiotics requires careful consideration, as not all types of dental pain require these. It is important to remember that dental pain usually requires active treatment from a dentist and antibiotics on their own are only the first step. Some dental abscesses may also require surgical drainage or extraction of the tooth. It is also worth remembering that dental pain can be severe and patients may unintentionally overdose, particularly with paracetamol containing drugs. A detailed drug history should therefore be taken in any patient presenting with toothache. Occasionally diffuse jaw pain may be confused with myocardial ischaemia. Dental pain can come in many forms.

25.3.3.1 Pulpitis

This is a common cause of pain and is typically severe. It arises as a result of inflammation within the dental pulp and is effectively a tiny compartment syndrome within the tooth. This is usually caused by infection (caries) which penetrates through the dentin to reach the pulp. However it can also occur following trauma, or a thermal insult (from prolonged dental drilling) or chemically following application of acidic dental filling materials to exposed dentinal tubules. Two types of pulpitis are typically described—(i) Reversible—the pulp is vital and will recover if treated and (ii) Irreversible—where the pulp is irreversibly damaged and cannot recover.

Pain is usually described as prolonged, intense and throbbing, which often wakes the patient. It is poorly localised and made worse by hot and cold drinks. Therefore

diagnosing the offending tooth can be difficult. Of note, the pain does not cross the midline. Often there will be a fractured tooth, or a tooth with gross caries or a large restoration. Sensitivity to hot and cold stimuli and localised pain on biting, help to localise the offending tooth. Pulp sensitivity tests are now routinely used in dental practice to diagnose dental disease. These can be thermal (ethyl chloride sprayed onto a small ball of cotton wool) and electrical (electric pulp testing—EPT). However these require careful interpretation and cannot be used in patients with orthodontic bands or crowned teeth. Radiographs are also required to make sure there is no extension of the infection into the surrounding bone (periapical abscess). Treatment by a dentist includes root canal treatment or extraction of the offending tooth. Opiate analgesia is no more effective than paracetamol with ibuprofen.

25.3.3.2 Periodontal/Periapical Abscess

These are potentially severe conditions which untreated can result in serious complications. Although these are two distinct conditions, they often present similarly with pain, swelling, halitosis, a tender or mobile tooth and sometimes discharge of pus. Pain is usually well localised to the tooth. With untreated or extensive abscesses, patients may develop fever, trismus, dysphagia or stridor, all of which require emergency management. Clinically there may be trismus, an obvious swelling and a tender tooth. Pus may be visible around the gum of the tooth. Radiographs will often show a grossly decayed tooth with periapical radiolucency/bone loss. Treatment involves root canal treatment or extraction of the offending tooth and if necessary incision and drainage of the associated abscess. Antibiotics are usually required. Severe cases need urgent referral and admission.

25.3.3.3 Recent Dental Treatment

Patients may present within a few days of dental treatment if they have had a large or root canal fillings. Typically they present with symptoms of pulpitis, or a generalised discomfort in the jaw. They should be prescribed appropriate analgesia and referred back to their dentist for further assessment. Symptoms usually subside spontaneously if there is no infective complication.

25.3.3.4 Dentine Hypersensitivity

This is a common cause of toothache and is often caused by chronic, enthusiastic, over brushing of the teeth. Patients often have very good oral hygiene. Recession of the gums exposes the dentine in the root of the tooth, which becomes exquisitely sensitive to hot cold sweet or acid drinks. One or several teeth maybe involved. Patients should be reassured and advised to see the dentist. Various coating agents can be placed to occlude the dentine tubules. Desensitising toothpastes are also available.

25.3.3.5 Cracked Tooth

Teeth with very large fillings can sometimes develop fine cracks in the remaining natural tooth. This can result in flexing of the cusp of a tooth—the central filling acts like a wedge on biting. Over time a fracture can propagate and the cusp can

eventually break off. Flexing of the cusp can result in localised discomfort on biting. Diagnosis can therefore be made by asking the patient to bite on a tongue depressor, or cotton bud, to apply a localised force to the tooth. Pain is typically worse on releasing the bite. Various treatments are available depending on the size of the filling and health of the tooth.

25.3.3.6 Referred Pain

A number of non-dental conditions can mimic toothache. These are described elsewhere in this book but include

(i) Sinusitis—this is usually affects the upper molar teeth resulting in a constant dull throbbing pain which may be confused with pulpitis. OPG will be required to assess the upper teeth. This may show a fluid level or opacity in the maxillary sinus.
(ii) Overuse or heavy clenching of the masseter and temporalis muscles can sometimes result in referred pain in the molar teeth. The teeth themselves may also become painful as a result of heavy occlusal forces.
(iii) Temporomandibular joint parafunction can result in vague facial pain often confused with toothache
(iv) Trigeminal neuralgia—often precipitated by triggers such as touch, cold or wind.
(v) Cardiac—Rarely, angina may present as dental pain.
(vi) Psychological disorders

25.3.4 The Wisdom Teeth and Pericoronitis

Patients can present acutely with painful wisdom teeth for a number of reasons. These include eruption pain, pericoronitis, dental caries, pulpitis and infected dentigerous cysts. Wisdom teeth ("third molars") typically erupt between the ages of 18–25, provided there is sufficient space. This may be accompanied by some discomfort, but this usually requires more than simple analgesia. If a tooth cannot erupt fully because of insufficient space, it is said to be impacted. In such cases the tooth may only partially erupt and part of it remains covered by the overlaying gum, which forms a flap called an operculum. This can trap food debris and bacteria and become infected—pericoronitis. Pericoronitis can therefore occur in any partially erupted tooth, but is far more common with lower wisdom teeth. Patients usually present with localised pain, swelling and sometimes trismus. On examination pus may be seen extruding around the tooth. OPG may show a small amount of bone resorption around the crown of the tooth. Treatment in the initial stages involves irrigation and antibiotics to settle the condition. Repeated, or severe episodes of pericoronitis may indicate removal of the offending wisdom tooth (Fig. 25.31).

If the patient describes a shooting or electric-type sensation involving the lower lip, or tongue, following dental treatment this suggests iatrogenic nerve injury. This can occur following removal of impacted lower wisdom teeth, the roots of which

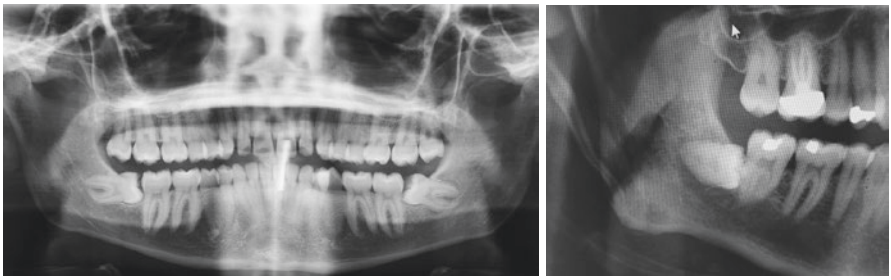

Fig. 25.31 Impacted third molars

can lie in close proximity to the inferior alveolar nerve. Occasionally patients can develop dysaesthesia. In many cases no intervention is required and nerve eventually recovers. However in some cases this can become chronic and patients may require medication such as amitriptyline or gabapentin. Always consider the possibility of a retained root in any socket which fails to heal uneventfully.

25.4 Ulceration and Blistering of the Mouth and Lips

An ulcer is a full thickness discontinuation in the epithelium of the skin or mucous membrane. If only the superficial layers are lost, it is called an erosion. Ulcers can extend into the underlying tissues. A blister is a rounded fluid-filled elevation of the skin/mucosa, caused by a separation either between the layers of the epidermis, or between the epidermis and the underlaying dermis. In the mouth the mucosa may separate from the lamina propria. Blisters are classified as vesicles if they are 0.5 cm or less in diameter and as bullae if they are larger than 0.5 cm. Whilst most lip and mouth ulcers are usually benign, it is important not to miss an oral cancer. Usually the history and appearances of the ulcer are enough to establish the diagnosis in most cases. Traumatic, aphthous and herpetic ulcers often have typical features and usually resolve spontaneously, within a few weeks. Malignant ulcers tend to present as a slowly progressive, non-healing ulcer, which is often painless. These ulcers often look malignant from the outset, although traumatic ulcers can sometimes appear malignant. Ulcers in the depths of the vestibular sulci can occur as a result of chronic irritation from dentures. Usually the patient has had the same set of dentures for many years. As the alveolar bone has resorbed naturally, the age of the denture has pressed further into the sulcus, resulting in a craggy-looking and indurated lesion. These can often be confused as malignant and may require biopsy, but they often heal very quickly if the patient stops wearing the offending denture. Similarly, nicorandil induced ulcers can look very much like malignant ulcers, but resolve quite quickly following cessation of the drug. If the ulcers and other associated lesions include areas beyond the oral cavity and involve other mucocutaneous sites, consider the possibility of pemphigoid, pemphigus, and Stevens-Johnson Syndrome. A useful classification of ulcers includes

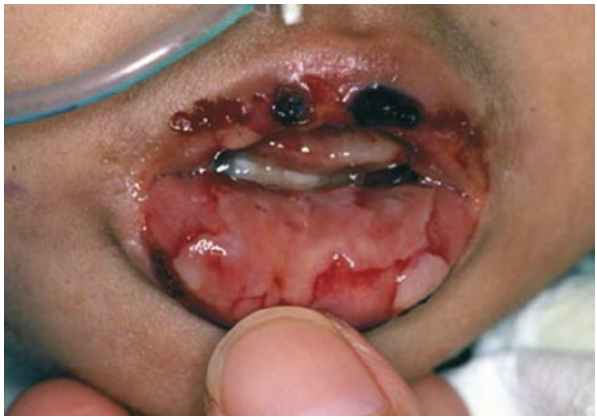

Fig. 25.32 Widespread mucosal ulceration

Multiple Acute Oral Ulcers

(i) Primary Herpes Simplex Virus Infections
(ii) Coxsackievirus Infections
(iii) Varicella-Zoster Virus Infection
(iv) Erythema Multiform
(v) Contact allergic stomatitis
(vi) Ulcers secondary to cancer chemotherapy
(vii) Acute necrotizing ulcerative gingivitis (ANUG) (Fig. 25.32)

Recurring Oral Ulcers

(i) Recurrent aphthous stomatitis
(ii) Behçet's syndrome
(iii) Recurrent Herpes Simplex Virus Infection

Chronic Multiple Oral Ulcers

(i) Pemphigus
(ii) Subepithelial Bullous Dermatoses
(iii) Herpes Simplex Virus Infection in Immunosuppressed Patients

Solitary Oral Ulcer

(i) Oral cancer
(ii) Traumatic ulcer (Fig. 25.33)
(iii) Nicorandil induced
(iv) MRONJ
(v) Fungal invasion (Histoplasmosis, Blastomycosis, Mucormycosis)

Systemic Causes of Oral Ulceration

(i) Bacterial infections—tuberculosis, secondary syphilis
(ii) Behçet's disease
(iii) Candida (usually in the immunocompromised)

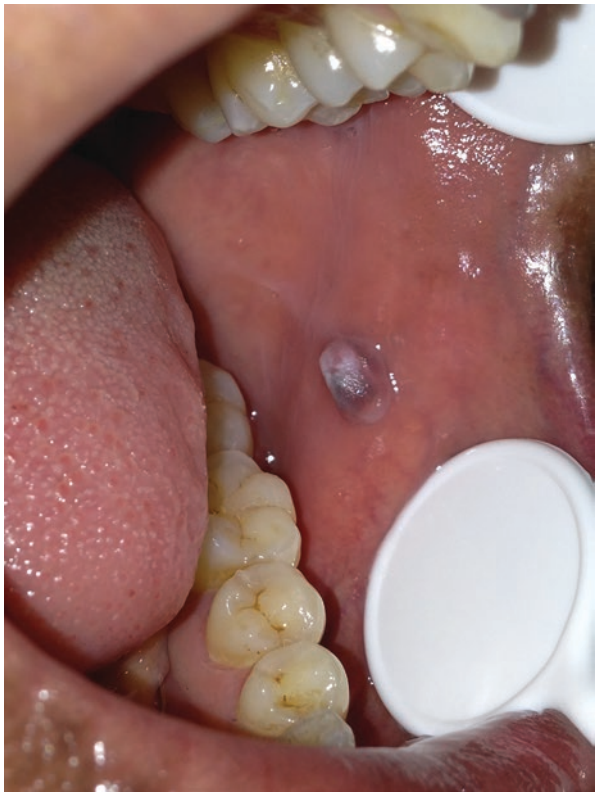

Fig. 25.33 Traumatic lesion

(iv) Chemotherapeutic agents
(v) Chronic renal failure
(vi) Crohn's disease
(vii) Dermatitis herpetiformis
(viii) Epidermolysis bullosa
(ix) Haematological disease—vitamin B12, folate and iron deficiencies
(x) Kawasaki disease
(xi) Lichen planus
(xii) Linear IgA disease
(xiii) Nicorandil
(xiv) Reiter's syndrome
(xv) Strachan's syndrome (Amblyopia, painful neuropathy, and orogenital dermatitis. Seen in undernourished patients)
(xvi) Stevens-Johnson syndrome
(xvii) Systemic lupus erythematosus (SLE)
(xviii) Sweet's syndrome (also known as acute febrile neutrophilic dermatosis)—a rare skin disorder characterised by a fever, malaise, arthralgia, myalgia, mouth ulcers, sore red eyes and the appearance of tender red lumps on the skin. Its cause is unknown.

(xix) Viral infections—HSV, HZV, Coxsackievirus, Epstein-Barr, hand, foot and mouth disease, HIV
(xx) Wegener's granulomatosis
(xxi) Cyclic neutropenia
(xxii) Selected anaemias
(xxiii) Inflammatory bowel diseases
(xxiv) Gluten-sensitive enteropathy (celiac sprue)
(xxv) Relapsing polychondritis syndromes (including the so-called "MAGIC" syndrome, which consists of mouth and genital ulcers with inflamed cartilage)
(xxvi) FAPA syndrome (recurring fevers, aphthous stomatitis, pharyngitis, and lymphadenopathy)

25.4.1 Traumatic Ulcers and Burns

Usually trauma or burns to the mouth or lips (usually from hot food or drinks) is self evident from the history. Traumatic ulcers commonly occur in sites which are prone to biting, such as the inner surface of the lips, the buccal mucosa along the occlusal plane and the side of the tongue. Patients with loose orthodontic appliances can you also injure the buccal mucosa and lips. Burns are more commonly seen on the palate and tongue, often after the patient has taken a mouth full of hot fluid without realising. Both are often painful. Caustic burns may also as a result of accidental or deliberate swallowing of caustic solutions. These are often much more extensive and are discussed in the chapters of the throat and the front of the neck. Other causes of burns include prolonged application of topical drugs, particularly aspirin, which the patient may hold next to a painful tooth. Ulcers and burns can also present as blisters—a sloughy white surface membrane sheds to reveal an erythematous or bleeding base. These lesions tend to look inflamed around the periphery.

Ulcers which are caused as a result of chronic trauma (such as from the edge of a broken tooth, or an over extended denture flange) can be sometimes be confused with oral cancer. These tend to have a white or keratinised appearance, whilst malignancies are usually more indurated, friable and may have exophytic margins. The two may also coexist. Palpation is therefore an important part of the examination, but in many cases a biopsy is still required. Another important differentiating factor is progression over time—traumatic ulcers and burns will usually heal spontaneously within 2–3 weeks, once the cause of trauma has been removed. In contrast, malignancy continues to evolve. Factitious ulceration (self induced) oral lesions are rare. Underlying mental illness is usually well concealed. Features suggestive of factitious ulceration include lack of correlation with any recognisable disease, bizarre configuration with sharp outlines, in an otherwise healthy person. Treatment of traumatic ulcers and burns involves removal of the

cause and symptomatic relief. Patients should be advised to see their Dentist to smooth any rough teeth that may be causing irritation. If necessary, topical local anaesthetic gels and mouthrinses can be prescribed. During healing, a bland diet and avoidance of irritating substances (such as salt, vinegar, citrus and chilli) is advised. Very hot food and drinks should be avoided. Chlorhexidine mouthrinse may help in preventing bacterial superinfection. Patients should always be reviewed to ensure the lesion has resolved. All non-healing ulcers should be regarded with suspicion and need urgent referral.

25.4.2 Acute Necrotising Ulcerative Gingivitis (Trenchmouth)

Acute necrotising ulcerative gingivitis (ANUG) is a non-contagious opportunistic infection of the gingiva. It usually presents with painful, bleeding gums and ulceration, which classically affects the inter-dental papillae (the triangular pieces of gum between adjacent teeth). The gums often bleed profusely following probing or wiping, or can bleed spontaneously. The patient has very marked halitosis. However, malaise, fever and/or cervical lymph node enlargement are rare. Untreated, ANUG can spread into the tissues beyond the gingiva to become necrotising periodontitis, followed by necrotising stomatitis. Severe pain is characteristic, which helps distinguish this from the chronic periodontitis. Predisposing factors for ANUG include poor oral hygiene, smoking, malnutrition, stress and immunosuppression. The causative organisms are mostly anaerobic bacteria, notably Fusobacteria and Spirocaetes. Treatment involves gentle debridement and antibiotics (usually metronidazole). Inflammation often resolves quickly and does no serious harm. However if neglected (as seen in some third world countries) this can progress to necrotising stomatitis. This is highly destructive, the most extreme form being cancrum oris.

25.4.3 Herpes Infection (Primary Herpetic Stomatitis, Cold Sores)

The herpes family of viruses is a large group which commonly infects the head and neck. Currently there are eight identified members (some reports now state 9).

(i) Herpes simplex type I (HSV-1)
(ii) Herpes simplex type II (HSV-2)
(iii) Varicella-zoster virus (VZV/HHV-3) (Human herpes virus)
(iv) Epstein-Barr virus (EBV/HHV-4)
(v) Cytomegalovirus (CMV/HHV-5)
(vi) Herpesvirus type 6 (HBLV/HHV-6)
(vii) Herpesvirus type 7 (HHV-7)
(viii) Kaposi's sarcoma herpesvirus (KSHV/HHV-8).

These viruses are ubiquitous and well adapted pathogens. Indeed, their name comes from the Greek 'herpein'—to creep—relating to their chronic, latent or recurrent nature. The viruses can be classified into the following three groups:

(i) Alpha-herpesviruses: HSV-1 and HSV-2, VZV—these have a relatively short reproductive cycle, variable host range and result in latent infections in sensory ganglia.
(ii) Beta-herpesviruses: CMV, HHV-6 and HHV-7—these have a long reproductive cycle and a restricted host range. Latency can occur in the white cells, kidneys, secretory glands and other tissues.
(iii) Gamma-herpesviruses: EBV and HHV-8—these are specific for either T or B lymphocytes and can become latent in lymphoid tissue.

Widespread oral ulceration and blistering is often seen following herpetic viral infection (primary herpetiform stomatitis or secondary cold sores). Primary infection is usually caused by the Herpes simplex virus type1 (HSV-1). Although HSV-2 primarily infects the genital area, the lips and oral mucosa can also be affected. However treatment is the same. Most infections are subclinical, often occurring in children, but in older patients and the non-immune it can present as an acute vesicular stomatitis. Subsequent reactivation of the infection can result in herpes labialise (cold sores). Although sporadically seen in otherwise healthy patients, in the immunocompromised this can become persistent or recurrent.

25.4.3.1 Primary HSV (Herpes Simplex Virus)

This usually presents in children and teenagers as widespread painful blisters—primary herpetic gingivostomatitis. Patients may become unwell with a fever and lymphadenopathy. Small (2–3 mm) dome shaped vesicles can affect any part of the oral mucosa, but the hard palate and the tongue are the most common sites. The vesicles soon rupture leaving well defined, shallow ulcers with red margins. If widespread, this can be confused with erythema multiforme, Steven's Johnson syndrome and pemphigus vulgaris. During this time, if unprotected fingers with small breaks in the skin, come into contact with the mucosa, a herpetic whitlow may develop. Inoculation of the eyes can also occur following rubbing with HSV-infected hands. This can result in serious infection (See the chapter on the eye).

Diagnosis is usually clinical, but can be confirmed with viral swabs. Rising antibody titres can be detected in the primary infection. Treatment is directed towards symptom relief. If diagnosed early, antiviral agents can be helpful. HSV (and HZV) usually respond well to acyclovir if prescribed early. This can be given as a suspension, which is first used as a mouthwash and then swallowed. Supportive treatments include rest, analgesia and hydration. Rarely, hospital admission is required for severe dehydration and nutritional support. Prolonged or severe infection which fails to respond to Aciclovir should be investigated for immunodeficiency. Ulceration

persisting for more than a month is an 'aids-defining illness'. Potentially lethal herpetic encephalitis is a rare complication.

25.4.3.2 Secondary HSV (Recurrent Herpes Labialis, or Cold Sores)

Following primary infection, the virus can migrate to the trigeminal ganglion, where it can remain latent permanently. Reactivation can subsequently occur during times of stress or immunocompromise. The virions then travel down the branches of the nerve to exit the nerve endings and form new vesicles. This presents as localised perioral vesicles, which often follow an initial period of tingling and pain. These are highly contagious. The differential diagnosis includes shingles (Herpes zoster virus) which can also involve the oral cavity. However, this is usually confined to one side and follows a dermatomal distribution, whereas HSV is more widespread. Asymptomatic shedding of the virus in saliva can also occur and has been reported to be relatively common in ICU patients.

25.4.4 Hand, Foot and Mouth Disease

Hand, foot and mouth disease is caused by several members of the Coxsackievirus (CSV) group. This results in vesicles that breakdown into small ulcers throughout the mouth except the hard palate or gingiva (ie keratinised epithelium). Skin lesions also appear on the elbows, knees, hands and feet. These are painful and pruritic, but resolve in 10–12 days. Treatment, is supportive. Coxsackievirus (CSV) can occasionally cause myocarditis or severe diarrhoea (Fig. 25.34).

25.4.5 Herpangina

This is another Coxsackievirus infection. Lesions resemble herpes infections and are very painful (hence the name). The vesicles are localised posterior to the

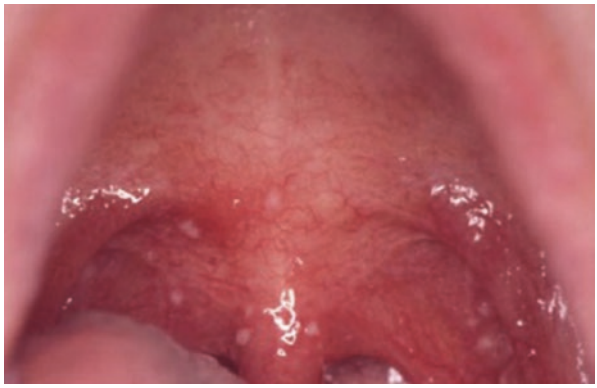

Fig. 25.34 Herpangina

junction of the hard and soft palate, usually involving the soft palate, uvula, and fauces. Multiple scattered vesicles rupture to leave small ulcers. This combination of pharyngitis and ulceration can cause confusion with streptococcal disease (strep throat). Treatment, is supportive. Enteroviruses can also cause oral vesiculo-ulcerative lesions, but these are uncommon, self-limited and do not recur.

25.4.6 Stevens-Johnson Syndrome (SJS)

Stevens-Johnson syndrome (also known as Toxic Epidermal Necrolysis-TEN) is a serious medical emergency which carries a high mortality. It is relatively uncommon but is still seen from time to time. This disorder should be considered in all patients that present with acutely painful, extensive oral lesions without an obvious cause. Pathologically it is a delayed hypersensitivity reaction in which there is bullous detachment of the epidermis and mucous membranes from the underlying dermis. This results in areas of extensive necrosis. The cause of the hypersensitivity is usually following ingestion of a drug (notably antibiotics), but this can also arise following a few specific infections (such as HSV, influenza and mumps). Loss of the skin places the patient at risk of dehydration and with the lost of its protective immunological barrier function, infection and sepsis. If the mucous membranes are involved this can result in gastrointestinal haemorrhage, respiratory failure and genitourinary complications. Ocular complications are also common and patients present with red, sore eyes and profuse lacrimation. If extensive this can result in scarring inside the eyelids, ocular ulceration and can threaten vision. SJS thus requires prompt recognition and withdrawal of any identified causative agent. Treatment is largely supportive until the epithelium regenerates. This includes isolation, IV fluids and electrolytes, nutritional support, pain relief and if the skin is involved, protective dressings. Patients should be referred early to an ophthalmologist even if ocular involvement is minimal. Treatment with corticosteroids is controversial and should not be prescribed until a viral infection has been excluded.

25.4.7 Behcet's Disease

Behcet's disease (or syndrome) is a triad of oral ulceration, genital ulceration and uveitis. This occurs mostly in young adult males and is associated with HLA-B5 and HLA-B51. It is most common in Mediterranean Countries, South East Asia, Japan and Turkey. The disease is not infectious, contagious or sexually transmitted. Rather, the histological changes are similar to recurrent aphthous stomatitis, suggesting an immune-complex aetiology. However there is some evidence to suggest that some viruses (possibly herpes simplex) and bacteria (*E. coli*, Klebsiella and Mycoplasma) are somehow associated. Symptoms can occasionally be life-threatening. Initially non-specific symptoms (malaise, anorexia, weight loss),

precede the onset of the mucous membrane ulceration. This can be by many months to several years. The entire syndrome has many features. These include

(i) Recurrent aphthous stomatitis (RAS). This is seen in 90–100% of cases. It is the most common initial manifestation of Behcet's disease.
(ii) Ocular inflammation—this occurs in about 70% of patients, usually following oral ulceration. The most common problem relapsing iridocyclitis. Uveitis, uveitis with conjunctivitis, hypopyon, retinal vasculitis and optic atrophy can also occur. Both eyes are usually involved, although uniocular disease has been reported.
(iii) Genital Ulcers—Recurrent painful ulcers that heal with scarring occurs in up to 90% of patients. They are common in females. In males they often involve the scrotum and penis. In both sexes they can occur around the anus.
(iv) Skin lesions occur in about two-thirds of cases and include erythema nodosum, pustular lesions and acneiform nodules.
(v) Large joints (arthralgia), heart, intestinal tract, vascular system (aneurysms) and most other systems may also be involved. Large vein thrombosis (of inferior vena cava and cranial venous sinuses) can be life threatening.

Definitive diagnostic tests for Behcet's disease are currently not available. Diagnosis is therefore based on the clinical picture (any two of the oral, genital and ocular features) and immunological findings. The differential diagnosis includes Erythema multiforme, Pemphigoid, Pemphigus and Reiter's syndrome. Management can be difficult. Many treatments have been tried with varying results. These include corticosteroids, Azathioprine, Colchicine, Thalidomide, Interferons, cyclophosphamide, dapsone, methotrexate, pentoxifylline and chlorambucil. Most recently there has been interest in anti-TNF alpha monoclonal antibody (Infliximab). Topical corticosteroids may be useful in the management of oral ulceration. An ophthalmological opinion should be obtained early, since ocular involvement can result in blindness.

25.4.8 Pemphigus and Mucous Membrane Pemphigoid

These have previously been discussed. Pemphigus is often divided into three major groups: (1) pemphigus vulgaris (PV), with pemphigus vegetans representing a rare variant; (2) pemphigus foliaceus, with pemphigus erythematosus representing an unusual localised variant, and fogo selvagem, an endemic form; and (3) paraneoplastic pemphigus. Additional subtypes include the two forms of IgA pemphigus and drug-induced pemphigus.

PV is the most common and the more aggressive, being associated with significant morbidity and mortality. In this form, patients have circulating IgG autoantibodies that bind to the cell surface of keratinocytes in the skin and mucous membranes. This leads to a disruption of the transmembrane proteins that are a

component of desmosomes and play an important role in cell–cell adhesion. As a result the decrease in cell–cell adhesion leads to separation of individual keratinocytes from one another (acantholysis) and splitting within the epidermis or mucosal epithelium, just above the basal layer. Thus, pemphigus vulgaris results in intraepidermal blistering which is potentially life-threatening. Patients may have poorly defined, irregular shaped, painful ulcers on the gingiva, buccal mucosa and palate. Intact bullae are rarely seen, but Nikolksy's sign can often be elicited. Other involved mucosal surfaces include conjunctiva, oesophagus, vagina, cervix, penis, urethra, nasal mucosa and anus. Diagnosis requires biopsy. Immediate treatment is essential to prevent sepsis from infection. This initially involves high-dose steroids and antibiotics. Adjuvant therapy includes azathioprine, methotrexate, cyclophosphamide, cyclosporin and dapsone. More recently, newer therapies have included the use of monoclonal antibodies (Rituximab), intravenous immunoglobulin, plasmapheresis and TNF-antagonists. Avoidance of any known antigen (environmental or medications such as penicillamine or captopril) may prevent further onset. Referral for an ophthalmic assessment is important.

The less common variants of pemphigus include paraneoplastic pemphigus, drug induced pemphigus and IgA pemphigus. In addition to SJS, pemphigus vulgaris and mucous membrane pemphigus should be considered in any patient presenting with an acute onset of widespread blistering and ulceration without an obvious cause. Mucous membrane pemphigoid is another autoimmune blistering disease, which tends to occur in older patients. This is due to autoantibodies that bind several components of the basement membrane zone (BMZ) of the skin and mucosae, most often BP180 and laminin 332. Although the condition heterogenous is its presentation, a tendency to scarring is typically seen. Blisters are typically restricted to mucosal surfaces, notably the gingivae, sinuses and genitourinary tract. Conjunctival involvement can result in erosions, scarring with symblepharon formation and blindness. Rarely it can involve the nasopharynx, larynx, and esophagus. The disorder can be detected clinically by a positive Nikolsky's Sign, but diagnosis requires a biopsy and immunofluorescence studies. Management is more localised than for pemphigus, with soft diet, oral hygiene, and topical steroids. Occasionally intralesional corticosteroids, dapsone, cyclophosphamide (for severe or progressive ocular disease) and rituximab may be indicated.

25.4.9 Aphthous Ulceration

Recurrent aphthous stomatitis (RAS) is a common oral condition which is seen worldwide. It is characterised by the repeated formation of benign and non-contagious mouth ulcers (aphthae) in otherwise healthy individuals. Although it is one of the most common recurrent oral ulcerative conditions of adults and children, it is also one of the least understood oral diseases. It usually presents in childhood or adolescence. Up to 20% of the population may be affected at some point in their lives. Patients present with recurrent episodes of painful ulcers. These are

non-infective and mostly occur in the labial and buccal mucosa, side of the tongue and floor of the mouth. Three main types of ulcer are described—(i) minor (MiRAS), (ii) major (MaRAS) and (iii) herpetiform (HU) ulcers. MiRAS is by far the commonest. Ulcers appear in crops of typically no more than 5, on non-keratinised mucosa. These are well circumscribed, small and ovoid with erythematous margins. Most ulcers resolve within 2–3 weeks of onset. Major RAS (MaRAS) is a more severe form which comprises about 10% of patients with RAS. These ulcers are 1 cm or more in diameter and often occur on the lips, soft palate and fauces. They persist longer (up to 6 weeks) and often heal with scarring. Herpetiform ulceration arises in up to 10% of patients with RAS. This is characterised by multiple recurrent crops of widespread, small, painful ulcers. There may be up to 100 ulcers at any given time, each measuring just a few mm in diameter. Despite its name, there is no association with herpes viruses.

Triggering factors that precipitate recurrent episodes of ulceration are diverse and can be unique to the affected individuals. They include nutritional deficiencies, local trauma, stress, hormonal influences and allergies. Haematinic deficiencies (Vitamin B12, folate and iron), immunocompromise and stress are well known associated factors. The cause is not completely understood, but involves a T cell-mediated immune response triggered by a variety of factors. A genetic predisposition has also been suggested, possibly with HLA-B51. Interestingly, aphthous stomatitis is uncommon in people who smoke, possibly because tobacco use is associated with an increase in keratinisation of the oral mucosa. Possible allergens include certain foods (e.g., chocolate, coffee, strawberries, eggs, nuts, tomatoes, cheese, citrus fruits, benzoates, cinnamaldehyde, and highly acidic foods), toothpastes, and mouthwashes. Systemic disorders have also been associated with aphthous-like ulceration. These include

- Behçet's disease (mouth ulcers, genital ulcers and anterior uveitis)
- Celiac disease. The link between gastrointestinal disorders and aphthous stomatitis is believed to be related to nutritional deficiencies caused by malabsorption.
- Cyclic neutropenia
- Nutritional deficiencies
- IgA deficiency
- Immunocompromised states, e.g. HIV/AIDS
- Inflammatory bowel disease
- MAGIC syndrome—a possible variant of Behçet disease—"mouth and genital ulcers with inflamed cartilage" (relapsing polychondritis)
- PFAPA syndrome—a rare condition in children. The name stands for "periodic fever, aphthae, pharyngitis (sore throat) and cervical adenitis". This is believed to be immunologic in nature.
- Reactive arthritis
- Sweet's syndrome
- Ulcus vulvae acutum

In such cases the ulcers are clinically and histopathologically identical to those of aphthous stomatitis, although they are not considered to be true aphthous ulcers. Ulceration on other mucosal surfaces may also occur, such as the conjunctiva or the genital mucous membranes. Some of these systemic diseases are discussed elsewhere. Resolution of the systemic condition often leads to decreased frequency and severity of the oral ulceration.

Diagnosis is usually clinical, but blood tests may be required to exclude deficiency states. Treatment is mostly symptomatic, similar to traumatic ulcers and burns. Vitamin B12, folate and iron supplementation may help to prevent recurrence. A bland diet during episodes of ulceration helps reduce pain and irritation. In resistant or severe cases topical steroids may help. Commonly used preparations include hydrocortisone pellets, triamcinolone acetonide in carboxymethyl cellulose paste (Adcortyl in orabase, although now discontinued in some countries) and betamethasone sodium phosphate dissolved in 15 mL of water as a mouth rinse. Only very occasionally is ulceration severe enough to require admission for IV hydration and systemic steroids. If this is required Behçet disease, auto-inflammatory syndromes, gastrointestinal disease, or immune defects such as HIV/AIDS should be considered. Several immunomodulating and anti-inflammatory drugs, including colchicine, cyclosporine, pentoxyfylline, azelastine and dapsone have shown some effectiveness for treatment of RAU. However, most of these drugs require further research. Recently, thalidomide, a potent immunosuppressive agent, has been found to be effective for treating HIV-infected patients with severe RAS and oral aphthae in immunocompetent individuals with Crohn's and Behcet's disease.

25.4.10 Angina Bullosa Haemorrhagica

Angina bullosa hemorrhagica (ABH) is a condition which affects middle-aged and elderly patients. No children have been reported to have this condition. The earliest reported age of onset is 22 years. The term ABH is descriptive, used to describe the sudden development of isolated, benign, subepithelial oral mucosal blisters. These are often filled with blood. Solitary blood blisters occur usually in the palate but may worsen progressively, leading to multiple lesions in other areas. The majority of ABH lesions occur at the junctions of the hard and soft palates, although lesions may also arise in the mucosa of cheeks and lips and lateral margins of the tongue. Rare sites include the epiglottis, arytenoids and pharyngeal walls. In one case blisters have been reported to extend into the oesophagus. Blisters vary in diameter from millimeters to centimetres—up to 3.0–4.0 cm in diameter. These subsequently rupture spontaneously and the sites heal uneventfully within a few days to a few weeks. The aetiology of ABH is not known. It has not been found to be associated with any known systemic or haematological disorders. Suggested factors include trauma, long-term use of steroids, diabetes and possibly a hereditary predilection. Blisters often occur following mild trauma. They may last from a few minutes to several days but they eventually rupture, releasing blood and then heal without scarring. A sensation of stinging or burning may precede the onset of a blister, but

otherwise patients are generally asymptomatic. Blisters may appear intermittently or at regular intervals. Diagnosis is based on exclusion of other conditions and the history of blood within the blister. No treatment is usually required, although incision and de-roofing of large palatal blood blisters has been recommended to prevent further extension of the lesion. Rinsing the mouth with chlorhexidine gluconate may also promote healing and prevent secondary infection. Some specialists have suggested that ascorbic acid/citroflavoronoid (200 mg twice daily) can help to prevent relapse of ABH. Alternatively, a short course of topical steroids may help.

25.4.11 Wegener's Granulomatosis

Granulomatosis with polyangiitis (GPA), also known as Wegener's granulomatosis, is one of the inflammatory vessel disorders—the vasculitides. It is a rare systemic autoimmune disease of unknown aetiology, characterised by necrotising granulomatous inflammation and vasculitis involving small and medium-sized blood vessels. This commonly involves the nose, sinuses, throat, lungs and kidneys. Initial manifestations can occur in the nasopharynx or mouth. Ulceration of the palate, which erodes into the nasal cavity is highly suggestive. Septal necrosis may also occur (Fig. 25.35).

Main features include ulceration, pain and a foul-smelling oral or nasal discharge. Other signs and symptoms may develop suddenly, or over several months. These include

(i) Runny nose, stuffiness, sinus infections and nosebleeds
(ii) Coughing/haemoptysis
(iii) Shortness of breath or wheeze
(iv) Fever and fatigue with general aches and pains
(v) Weight loss
(vi) Numbness in the fingers or toes
(vii) Hematuria
(viii) Ocular symptoms
(ix) Ear pain and infections

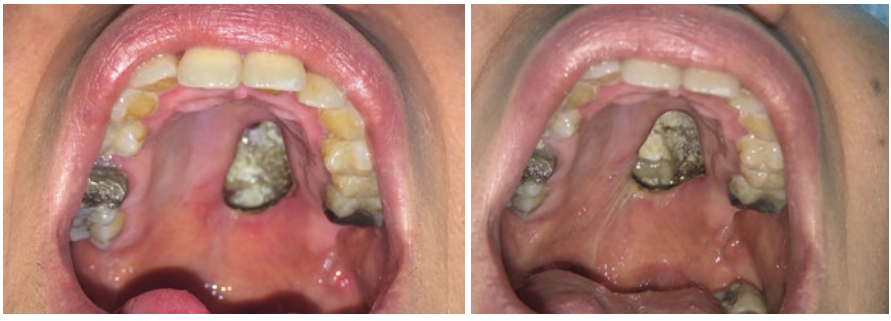

Fig. 25.35 Palatal necrosis from GPA

GPA can occur at any age, but most often affects people between the ages of 40 and 65. Its cause is unknown, but it appears to develop after an infection or some other inflammation-causing event. This triggers an abnormal reaction in the immune system, resulting in inflamed blood vessels and necrotising granulomas. The granulomas can destroy healthy tissue. Diagnosis can be difficult in view of the wide range of symptoms. Investigations include

(i) Blood tests—Anaemia, raised C-reactive protein or ESR, anti-neutrophil cytoplasmic antibodies. Hypergammaglobulinemia may also be present.
(ii) Urine tests. Proteinuria and hematuria
(iii) Chest X-ray, CT and MRI
(iv) Biopsy.

Early diagnosis and treatment may lead to a full recovery. Without treatment, GPA can be fatal. Commonly prescribed drugs include immunosuppressants such as corticosteroids cyclophosphamide, azathioprine, Rituximab and methotrexate. Some drugs may need to continued long term to prevent relapse.

25.5 Malignancies of the Mouth and Lips

Early intraoral cancer usually presents as a painless non-healing ulcer, with raised rolled edges. When advanced, other symptoms include pain, spontaneous bleeding, paraesthesia, trismus, dysarthria and loosening of teeth. Dysphagia and otalgia (pain referred to the ear) indicate involvement of the pharynx (oropharyngeal cancer—see the chapter on the throat). Very rarely the patient may present with a pathological fracture of the mandible. Risk factors for squamous cell carcinoma (SCC) include smoking, alcohol, chewing tobacco, betel leaf and areca nut (paan). Whilst oral SCCs can occur anywhere in the mouth, the most common sites are the floor of mouth and the posterolateral tongue. Other less common types of intraoral cancer include salivary gland malignancy (consider this in any palatal swelling or non-healing palatal ulcer), malignant melanoma and lymphoma.

In some countries today, the leading cause of oral and oropharyngeal SCC is human papilloma virus (HPV). This is a double-stranded DNA virus that can infect epithelial cells of skin and mucosa (such as the mouth, throat, tongue, tonsils, vagina, cervix, vulva, urethra and anus). Transmission of the virus occurs by direct contact, including sexual contact, both ‘conventional’ and oral. The HPV family contains almost 200 strains, but only nine are associated with cancers. Of these, only one is strongly associated with oropharyngeal cancer, HPV16. A few others are associated with benign warts. Any ulcer which persists longer than 2 weeks, should be referred for urgent biopsy to exclude dysplasia or malignancy.

25.5.1 Squamous Cell Carcinoma

This is the commonest cancer in the mouth. As it advances, it spreads to the cervical lymph nodes. This follows a predictable pattern, depending on where the primary tumour is sited. The nodes most commonly involved are in the submental, submandibular and deep jugular chain (See the chapter on the Front of the neck). The neck should therefore be carefully examined. The likelihood of lymph node spread is related to the size of the primary tumour and its depth of invasion. Tumours greater than 4 mm in thickness have a much greater chance of nodal involvement. Early detection is therefore important as the presence of cervical node metastasis is the single most important prognostic factor, reducing survival by nearly 50%. Clinical features suggestive of a malignant ulcer include

(i) Fixation
(ii) Induration
(iii) Non-healing
(iv) Painless
(v) Friable
(vi) Round/rolled/heaped margins.

Many cancers are believed to have a genetic and environmental basis, in which a series of changes occurs during cell division, resulting eventually in malignant change—the 'multistep hypothesis'. These cancers therefore often arise in a pre-existing "premalignant" lesion, although a more correct term is 'potentially malignant' lesion (malignant change does not always occur). Whilst some oral SCC's may also arise in apparently normal mucosa, most are preceded by recognisable potentially malignant lesions, such as erythroplakia (red patch), leukoplakia (white patch), speckled leukoplakia (red and white patch), or verrucous leukoplakia. Biopsy is thus often required. The rate of malignant changes can be as high as 35% when moderate or severe dysplasia is present (Figs. 25.36, 25.37, and 25.38).

Once diagnosed, imaging is required to define the nature and extent of the tumour. An OPG may show signs of bony invasion and document the patient's periodontal status (which is important for planning treatment). Contrast-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) are also required, based on local protocols. MRI is generally more precise in examining the extent of soft-tissue invasion and identifying perivascular and perineural spread. CT with contrast can provide better bony detail. Positron emission tomography (PET) is now also used to provide data on tumour metabolism before and after chemotherapy. Elevated or rising PET activity after chemoradiotherapy strongly suggest persistent or recurrent disease that may not be detectable by CT or MRI (Figs. 25.39, 25.40, and 25.41).

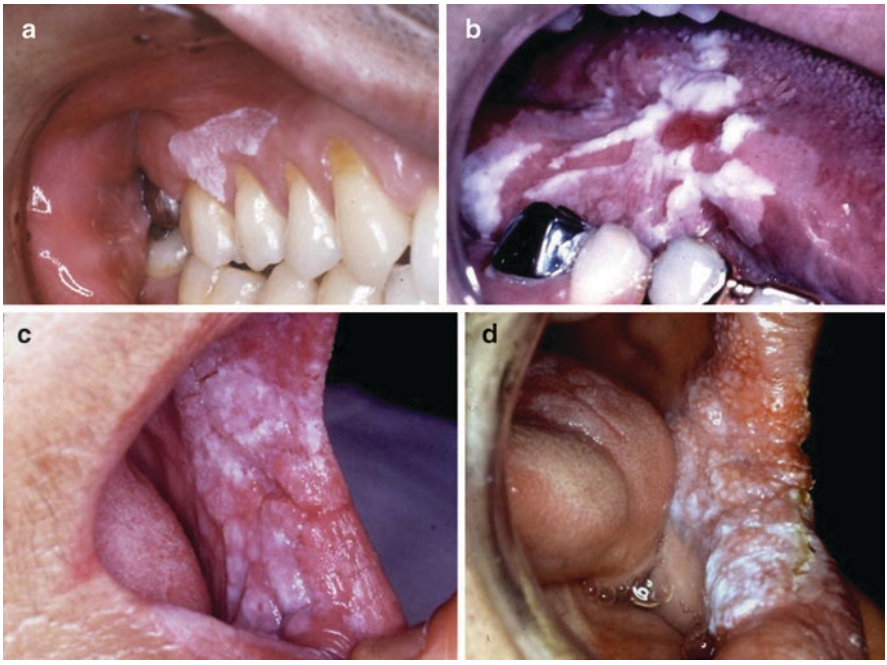

Fig. 25.36 Clinical features of leukoplakia can be classified into four types: type 1 (**a**) flat white patch on the of the maxillary gingival, type II (**b**) white patch and plaque with erosion on the tongue, type III (**c**) slightly elevated white plaque on the buccal mucosa, and type IV (**d**) markedly elevated white plaque with partly granular appearance on the buccal mucosa [10]

Up to 15% of patients with oral cancer may have a second primary tumour nearby (a cancer that is separate and not connected to the original primary). Careful examination of all mucosal surfaces is therefore important. Synchronous tumours are defined as those developing at the same time, or within 6 months of the primary tumour. After 6 months they are referred to as metachronous. Most occur within the head and neck, but up to one-third may be seen in the lungs or oesophagus. Management of oral SCC depends largely on how advanced the tumour is (its stage). As a general rule, surgery is followed by irradiation for most 'curable' cancers of the mouth. Surgical resection for advanced disease may also be indicated for palliative reasons and with pathological fractures of the lower jaw. The role of chemotherapy in head and neck cancer has historically been palliative. However, this has evolved to now play a role in primary combined-modality treatment.

25.5.1.1 Oral Submucous Fibrosis

Oral submucous fibrosis is a chronic debilitating disease of the oral cavity characterised by inflammation and progressive fibrosis of the submucosal tissues. It causes progressive limitation of mouth opening which if untreated will progress to a total inability to open. The buccal mucosa is the most commonly involved site, but any part of the oral cavity can be involved, including the pharynx. The condition is well

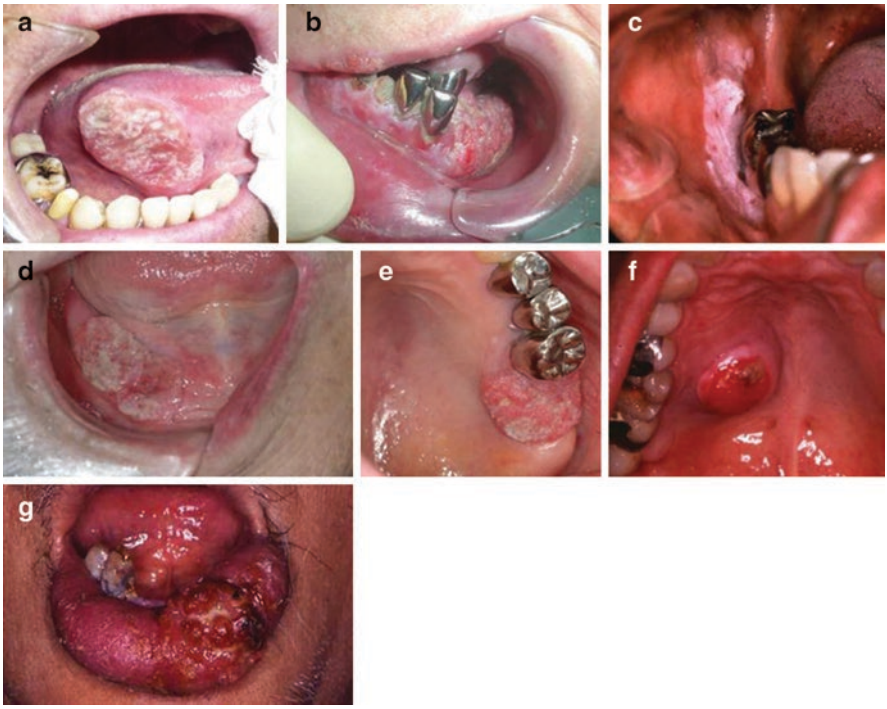

Fig. 25.37 Site of oral cancer, (**a**) Tongue cancer, (**b**) Mandibular gingival cancer, (**c**) Buccal mucosa cancer, (**d**) Oral floor cancer, (**e**) Maxillary gingival cancer, (**f**) Palate cancer, (**g**) Lower lip cancer

recognised for its malignant potential and is particularly associated with areca nut chewing, the main component of betel quid. It can be diagnosed by the marbled appearance of buccal mucosa and the palpation of tight fibrous bands in the buccal mucosa. Although chronic use of betel nut is the most likely cause of the condition, chemical or thermal burns can also result in similar appearances.

25.5.2 Mucosal Malignant Melanoma

This is rare and the prognosis is much worse than with SCC. These tumours represent less than 2.0% of all malignant melanomas. Pre-existing melanocytic lesions may be seen in about one-third of mucosal melanomas of the head and neck. Although the most common site appears to be the nasal cavity, oral sites include the hard palate, gingiva, buccal mucosa, lip and floor of the mouth. Some tumours may be amelanotic, making the diagnosis especially difficult. In contrast to cutaneous melanoma, melanomas involving the head and neck mucosa are more aggressive. Like SCC, regional lymph node metastases also plays an important role in prognosis. Any pigmented lesion other than an amalgam tattoo, racial pigmentation, or

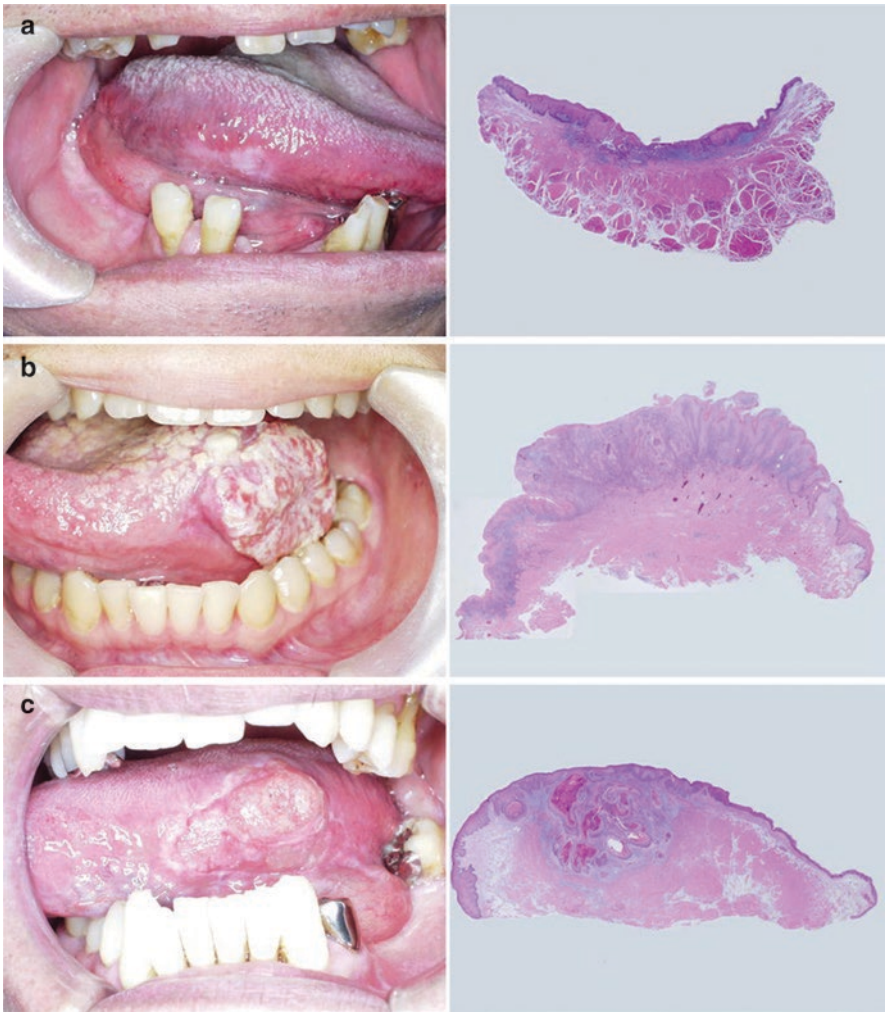

Fig. 25.38 Clinical types, (**a**) Superficial type, (**b**) Exophytic type, (**c**) Endo

physiologic pigmentation should be excised and submitted for urgent histopathologic evaluation. Following staging and MDT assessment, treatment of most oral mucosal melanomas generally involves surgical removal. Immunotherapy using interferon-alpha2b (IFN-alpha 2b) may be used as adjuvant treatment for advanced-stage melanoma. Prognosis is generally poor with a reported 5-year survival rate of less than 20% for all oral melanomas (Fig. 25.42).

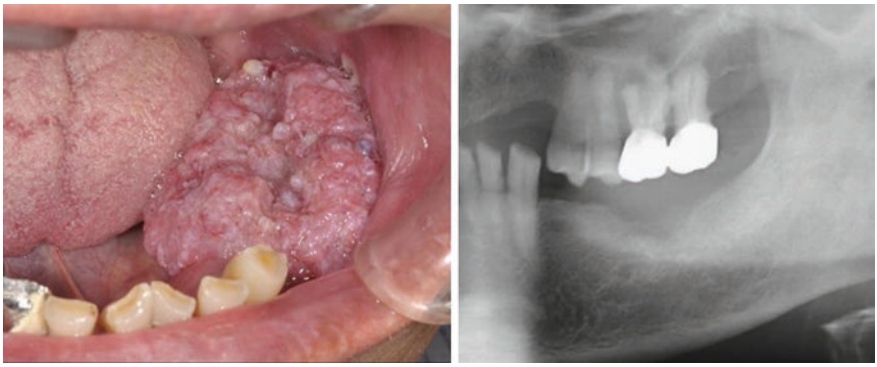

Fig. 25.39 A squamous cell carcinoma of the lower gingiva showing the granular type of observational findings, the exophytic type of clinical growth pattern, and the pressure type of bone resorption

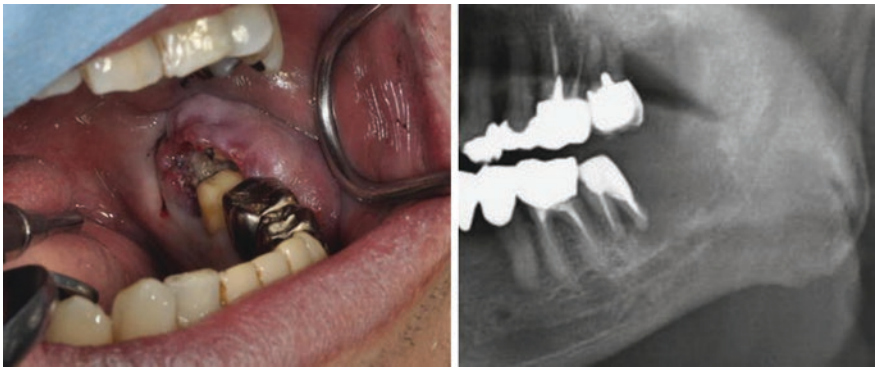

Fig. 25.40 A squamous cell carcinoma of the lower gingiva showing the indurative type of observational findings, the endophytic type of clinical growth pattern, and the moth-eaten type of bone resorption

25.5.3 Lymphomas

These are a mixed group of malignant lymphoproliferative neoplasms that arise following uncontrolled clonal expansion of B and/or T lymphocytes. These are generally grouped into non-Hodgkin (NHL) and Hodgkin lymphomas (Hodgkin's disease). They are the most common non-epithelial neoplasm in the head and neck region, accounting for about 5% of head and neck cancers. Approximately 15% of NHLs arise in the head and neck region, commonly presenting as painless cervical lymphadenopathy. NHL can also present at extranodal sites, sometimes presenting

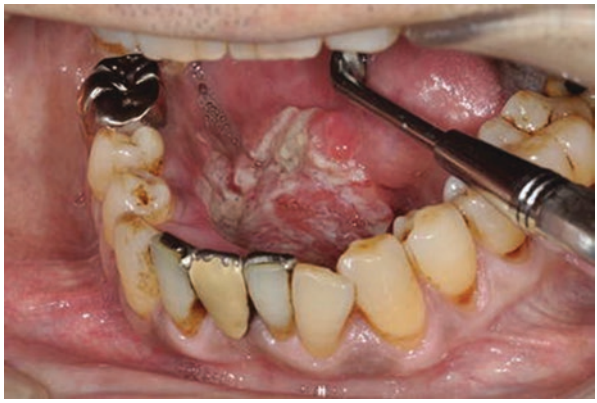

Fig. 25.41 A squamous cell carcinoma of the floor of the mouth showing the ulcerative type of observational findings and the endophytic type of clinical growth pattern

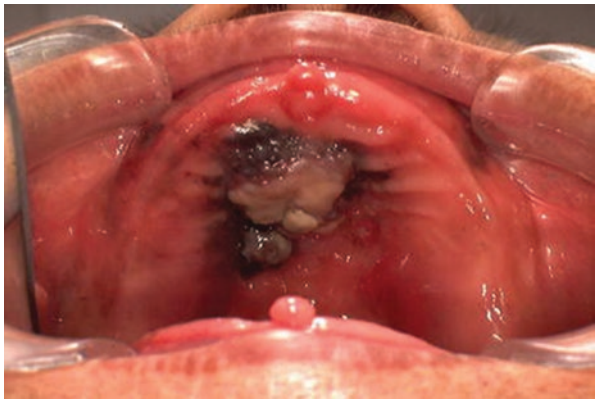

Fig. 25.42 A malignant melanoma of the hard palate

as a palatal swelling. This should be included in the differential diagnosis of any soft tissue palatal lump. Other sites include Waldeyer's ring (esp the palatine tonsil), the nose and paranasal sinuses. Clinical presentation may be similar to squamous cell carcinoma. Biopsy and subsequent staging are therefore important. Chemotherapy is the mainstay of treatment for most patients.

25.5.4 Kaposi's Sarcoma

Kaposi's Sarcoma is a rare malignancy of endothelial cell origin, often presenting in HIV positive patients. It was first described involving the skin in elderly males of Mediterranean or Jewish origin, but is also endemic in black African children and adults. More recently it has been recognised as a feature in immunosuppressed patients, secondary to organ transplantation or human immunodeficiency virus (HIV) infection. Kaposi's Sarcoma can therefore be an 'aids-defining illness'. Kaposi sarcoma herpesvirus (KSHV), is a human virus that has co-evolved with human populations, and has been shown to be common

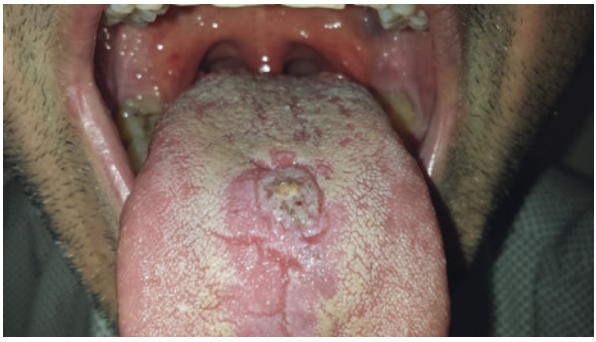

Fig. 25.43 Kaposi's sarcoma

in sub-Saharan Africa, around the Mediterranean Sea, parts of South America and in a few ethnic communities. It has been suggested as an important cause in three human malignancies, (i) Kaposi sarcoma, (ii) primary effusion lymphoma and (iii) many cases of the plasmablastic form of multicentric Castleman's disease (MCD). It has also been linked to rare cases of bone marrow failure and hepatitis. Since the virus has co-existed with humans for many thousand years, immune deficiencies are required in order for it to become pathogenic (Fig. 25.43).

Patients present with a violaceous (dark red, blue or black) macule on the palate which then ulcerates and becomes painful. Other clinical manifestations can range from slowly progressive lesions confined to the skin of the legs, to a more aggressive state involving several visceral organs. Biopsy is recommended to distinguish Kaposi's sarcoma from other skin conditions that can look similar (including non-Hodgkin lymphoma and cutaneous fungal or bacterial infections). The lesions tends to be highly vascular. Kaposi's Sarcoma cannot be cured. Since the introduction of highly active antiretroviral therapy (HAART), there has been a decline in its incidence. However, Kaposi's sarcoma continues to be diagnosed in HIV-infected patients. Other treatment options include surgery, radiation and chemotherapy. Generally, more widespread disease, or disease affecting internal organs, is treated with systemic therapy (including interferon alpha, liposomal anthracyclines, thalidomide and paclitaxel). Transplant associated KS responds to immune reconstitution, although lowering the immunosuppressive effects, risks graft rejection. Prognosis is related to three main characteristics—(i) extent of the tumour, (ii) Immune status of the patient and (iii) Severity of systemic symptoms.

25.5.5 Necrotising Sialometaplasia

This is mentioned here because it frequently has the clinical appearances of malignancy. It is however, a benign self-limiting disorder. The lesion often presents itself as a deep-seated palatal ulcer with clinical and histological features similar to those of a malignant neoplasm. Involvement of other oral sites such as

major salivary glands, lateral border of the tongue, sino nasal mucosa and larynx, is a very rare occurrence, with only few cases reported. It is three times more common in males than in females and in heavy smokers. Most patients are over 40 years of age. Necrotising sialometaplasia is believed to arise as a result of necrosis of the minor salivary glands, sometimes following a minor injury. The most widely accepted theory is ischaemia of the blood vessels, leading to infarction of the glandular tissues. Factors believed to lead to ischaemia are trauma, administration of local anaesthetics, smoking, alcohol and cocaine use. Intubation and surgical procedures have also precipitated onset. Necrotising sialometaplasia has also been reported in a patient with sickle cell disease, Buerger's disease and Raynaud's phenomenon but it can arise seemingly spontaneously. Histologically five stages are described—infarction, sequestration, ulceration, reparative and healed. Clinically, appearances may often be confused with malignancy, and therefore biopsy is nearly always necessary. However once diagnosed, no treatment is required. The lesion heal spontaneously over 6- to 10-weeks. The ulcer fills in with granulation tissue and completely epithelialises its surface. Recurrence is virtually unknown.

25.5.6 Melanotic Neuroectodermal Tumour of Infancy

This is a rare benign tumour which probably arises from neural crest cells. It typically appears as a rapidly growing mass in the anterior maxilla during the first year of life. The tumour may begin as a small pink or red-purple nodule that resembles an eruption cyst. Radiographs usually show localised and irregular destruction of the underlying alveolar bone and a primary tooth bud floating in a soft tissue mass. Urinary levels of vanilmandelic acid are elevated. Treatment is conservative excision. Histologic examination often shows melanin pigmentation. Recurrence and metastasis rarely occur.

25.5.7 Lip Cancer

This is one of the more common cancers of the head and neck region and is often easily diagnosed. It generally has a good prognosis. The behaviour of lip cancer resembles skin cancer more than carcinoma of the oral cavity. However, in some individuals, it can behave aggressively and recur. Mortality has been reported in up to 15% of patients following late presentation. The most common cancer of the lip is squamous cell carcinoma (SCC). Basal cell carcinoma accounts for only 1% of all lip cancers. Other malignancies have been reported but are less common (Fig. 25.44).

The lower lip is the most common site for lip cancer, with only a few percent of cases arising in the upper lip and 4% at the oral commissures. Approximately one-third of patients with lip cancer have outdoor occupations, suggesting that sun exposure may be an important causative factor. Because of its prominence, the lower lip is more exposed to the sun, compared with the upper lip. Carcinoma of

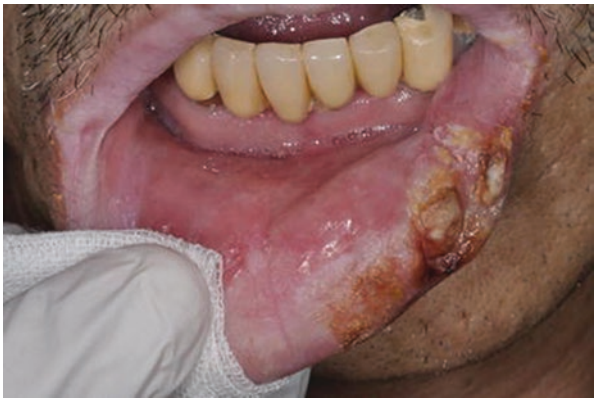

Fig. 25.44 A squamous cell carcinoma of the lip with scabbing ulcer

the lip also tends to affect individuals with fair skin complexions. Other suggested risk factors include tobacco use, pipe smoking, thermal injury, exposure to chemicals, immunosuppression and chronic infections. Cervical metastasis from lip cancer occurs in less than 10% of patients with cancer of the lower lip but in up to 20% in cancers of the upper lip and commissure. Upper lip lymphatics drain into the submandibular lymph nodes but may also drain into the preauricular or parotid lymph nodes.

Clinical presentation of lip carcinomas is usually quite characteristic, with an exophytic or ulcerated lesion along the vermilion border, together with a variable degree of infiltration into the underlying muscle or adjacent skin. Hyperkeratosis and leukoplakia may be present. Biopsy is almost always required. Management involves surgical resection with reconstruction in most cases. At the same time a lip shave (vermilionectomy) may be undertaken to remove adjacent 'premalignant conditions' such as leukoplakia, actinic keratosis and carcinoma in situ. With extensive and neglected tumours, invasion of the mandible, involvement of the mental nerve and regional lymph node metastasis may occur. These tumours then require a more aggressive resection, including neck node dissection.

25.5.8 Erythroplakia

Erythroplakia is a clinical term to describe any erythematous area on the mucosa, that cannot be attributed to any other pathology. It particularly involves the floor of mouth, tongue or palate and signifies a potentially malignant lesion. Typically it has a 'velvety red' surface and may or may not be associated with leukoplakia. Erythroplakia should always be biopsied, as it is associated with dysplasia or carcinoma in many cases. Some studies have reported carcinoma to be found in almost 40% of patients with erythroplakia.

Surgical excision of the lesion is occasionally required depending on the histology. Recurrence is common and long-term monitoring with repeat biopsy is often needed.

25.6 White and Red Lesions in the Lips, Tongue and Mouth

25.6.1 Physiological White Patches

White patches inside the mouth can sometimes be quite normal. A thin white line inside the cheeks at the level of the occlusal plane can often be seen. This is called the Linea Alba and is due to mild keratinisation, secondary to minor cheek irritation from the occluding teeth. Multiple small creamy white spots line the buccal mucosa. These are intraoral sebaceous glands, termed Fordyce's Spots. Neither of these require biopsy or treatment.

25.6.2 Leukoplakia

The term 'leukoplakia' is simply a descriptive term for a firmly adhered white patch on the skin or a mucous membrane. It does not indicate any specific condition. There are many causes for this. Most are harmless. However in all cases it is important to consider and if necessary, exclude malignancy (Fig. 25.45).

25.6.3 Actinic Keratosis

White patches on the lips or vermillion border in sun damaged skin is known as actinic keratosis. This is typically seen on the lower lip (as well as the scalp, face and ears), due to chronic exposure to the sun. This should be biopsied and monitored as it may become dysplastic. Patients with this condition typically have a history of excessive sun exposure, either occupationally or recreationally.

25.6.4 Nicotine Stomatitis of the Palate

Nicotine stomatitis presents is a diffuse white patch on the palate, which is thought to represent thermal-induced keratosis from pipe and cigarette smoking. Whilst malignant transformation is uncommon, the area may need to be monitored and biopsied if it undergoes a change in appearance, especially if it becomes ulcerated. Smoking cessation and sucking a peach stone may help reduce the area.

25.6.5 Oral Lichen Planus (OLP)

Lichen planus (LP) is a chronic inflammatory disease of the skin, mucous membranes and nails. Several subtypes are described (i) reticular, (ii) papular, (iii) plaque (iv) atrophic, (v) erosive, (vi) vesiculo-bullous and (vii) desquamative. Its pathogenesis is not known and there is no known cure. It is currently thought to be a chronic autoimmune-related disorder (delayed-type IV hypersensitivity reaction), resulting

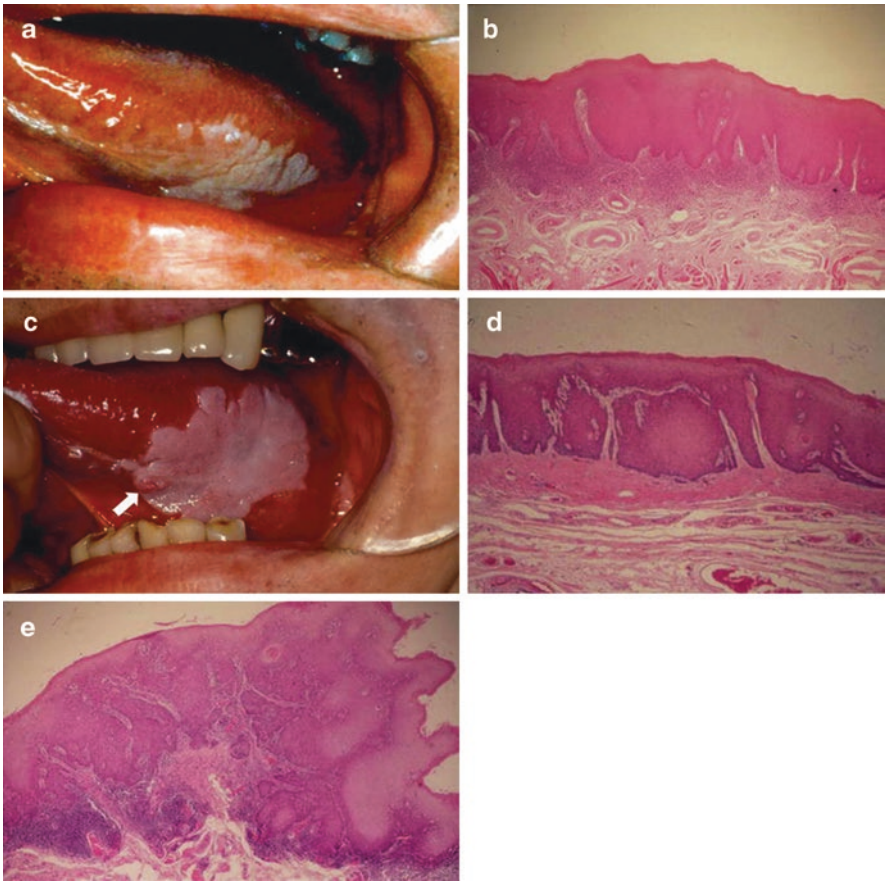

Fig. 25.45 Leukoplakia

in damage to the basal cells of the epithelium. Precipitating antigens may include foods, some toothpastes, drugs, viruses and possibly trauma.

Within the mouth, the most common presentation is the appearance of lacy white lines—reticular LP, commonly seen on the buccal mucosa and side of the tongue. Atrophic, papular or plaque-like forms are also common. Vesiculo-bullous lesions are rare, but may be confused with other bullous conditions involving the mouth. Oral lichen planus is often asymptomatic, but can present with inflammation and stinging, made worse by spicy or acidic foods. Erosive lichen planus is a more severe form of lichen planus, which presents with severe pain and ulceration. This can sometimes be very extensive and disabling for the patient.

Diagnosis is usually clinical, but often requires a biopsy to exclude other or co-existing conditions. The risk of malignant transformation is controversial and estimated at a 1% lifetime risk. In some patients the condition persists many years, in others it inexplicably "burns out". Treatment is often symptomatic and includes

analgesic mouthwashes (Difflam), avoidance of spicy foods, and topical steroids for acute symptoms. Systemic steroids are rarely required. Antifungal medication may occasionally be indicated, especially if the patient taking long-term steroids, or fungal growth is seen following biopsy. Retinoids and monoclonal antibodies have also been reported to be effective. However, these should only be prescribed by specialists in the condition. Referral for to a dermatologist and sometimes gynaecologist should be considered, depending on the sites of involvement.

25.6.6 Lichenoid Reaction

Lichenoid reactions tend to look virtually the same as oral lichen planus, although they tend to be more localised. Lichenoid reactions are often the result of inflammation from a nearby amalgam filling, or metallic denture clasp. Drugs may also result in a lichenoid reaction. Treatment is removal of the offending cause.

25.6.7 Frictional Keratosis

Frictional keratosis often presents as a non-inflamed white patch, located over an area of repeated low grade trauma (cheek biting or rubbing). It is commonly seen on the buccal mucosa, lateral tongue, gingivae and lips. It is not associated with ulceration or induration and is not at risk of malignancy. If in doubt, biopsy can be used to confirm the diagnosis. Treatment is not usually required and the site may spontaneously regress if the cause is removed. The patient should be referred back to their dentist for smoothing of any sharp teeth.

25.6.8 Candidiasis

Fungal diseases involving the oral cavity can be superficial or deep. Most are opportunistic in nature. Deep fungal infections, including histoplasmosis, coccidioidomycosis, blastomycosis and cryptococcosis, present as exophytic ulcerated granulomatous lesions that may appear malignant. Persistent or severe infection should therefore always raise suspicion of immunosuppression.

Oral candidiasis (candidosis or 'thrush') is a fungal infection due to one of several types of Candida, a yeast-like fungus. The organism is a normal commensal in the mouths of about 70% of the world's population. It presents as thick white plaques, which in contrast to most other leukoplakias, can be easily scraped off to reveal a bleeding base. More than 20 types of Candida can cause infection, with *Candida albicans* being the most common. Oral candidiasis is usually classified as acute or chronic, with various subtypes (such as acute pseudomembranous candidiasis). However, a more practical classification separates primary candidiasis (where the infection is confined to the mouth and lips) from secondary (in which there is involvement of other parts of the body).

Candidiasis is most commonly seen on the soft palate of asthmatics who use steroid inhalers. It can also occur under dentures in patients who constantly wear them. Three main types are described (i) pseudomembranous, (ii) erythematous (atrophic) and (iii) hyperplastic.

(i) Acute pseudomembranous candidiasis (thrush) is the most common type, which sometimes looks like "curdled milk". It is commonly seen in infants and people taking antibiotics or immunosuppressant medication. Extension into the oesophagus is an opportunistic infection and should raise suspicion for severe immunosuppression (leukaemia, HIV, malnourishment and diabetes).
(ii) Antiobiotic-induced stomatitis is a subtype of erythematous candidiasis. This appears as a red, raw-looking lesion, sometimes called "antibiotic sore mouth" or "antibiotic sore tongue".
(iii) Denture stomatitis is infection of the mucosa beneath a denture, usually the upper one. It is common in the elderly and those who don't take their dentures out at night. Often there are no symptoms.
(iv) Median rhomboid glossitis is an oval (or rhomboid) shaped lesion in the centre of the dorsal tongue, just anterior of the circumvallate papillae. This appears smooth and red and is rarely painful.
(v) Chronic mucocutaneous candidiasis is a rare group of syndromes, characterised by chronic infections in the skin, mouth and other mucous membranes. It is a secondary oral candidiasis.
(vi) Angular cheilitis is characterised by painful cracked fissures and inflammation at the corners of the mouth. This is commonly due to a mixture of Candida and Staphylococcus species. Symptoms include soreness, erythema, and fissuring of one or both commissures. Angular cheilitis generally occurs in elderly people and is often associated with denture stomatitis. In some patients it may signify a nutritional deficiency, such as iron, B12 or folate. Diagnosis is usually clinical but may be confirmed with fungal swabs, or preferably by biopsy (Fig. 25.46).

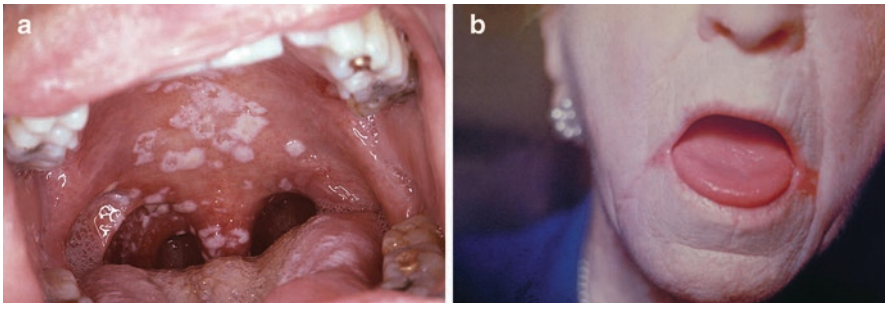

Fig. 25.46 *Candida* causing (**a**) pseudomembranous thrush, and (**b**) atrophic (or erythematous) thrush. Angular cheilitis (irritation at the comers of the mouth) due to thrush may be present as well, as in (**b**). Courtesy of Centers for Disease Control and Prevention (CDQ), Public Health Image Library

Treatment of candidiasis involves treating any predisposing factors (diabetes and haematinic deficiencies) and oral (or topical for angular cheilitis) antifungal medication (such as clotrimazole, amphotericin B, ketoconazole, or nystatin cream). Some antifungals such as miconazole also have antibacterial action so are helpful in angular cheilitis. In the edentulous patient construction of new dentures may help.

25.6.9 Hairy Tongue of HIV

This is a benign and otherwise asymptomatic white patch seen on the lateral tongue which is caused by an opportunistic infection from the Epstein-Barr Virus. This usually occurs in association with immunosuppression and may therefore indicate HIV infection. The lesion itself is said to appears as a homogenous leukoplakia with a characteristic 'shaggy' or hairy appearance. No treatment is required, but its presence should prompt investigation for immunosuppression.

25.6.10 Anaemia

Generalised paleness of the oral mucosa, such as the lips, tongue and gums may indicate the possibility of anaemia. Patients should be screened accordingly.

25.6.11 Red Patches in the Mouth

There are many causes of red patches. The vast majority are benign and apart from vascular malformations, are often inflammatory in nature. Many are discussed elsewhere in this chapter.

25.6.12 Erythema Multiforme

Erythema multiforme is a hypersensitivity reaction. It can occur at any age and varies from mild to severe. Inflammation of the skin and oral mucosa occurs following ingestion of a drug, usually antibiotics. Herpes simplex virus (HSV) is a rare but another recognised cause. Lesions may occur independently or combine to involve the mouth, lips, and skin. These appear suddenly as nonspecific irregular red patches with painful ulceration and crusting. The lips are commonly involved. Severe cases require admission, IV steroids and IV fluids. The differential diagnosis is an acute viral infection (HSV).

25.6.13 Lupus Erythematosus

Lupus erythematosus is an autoimmune disease that is characterised by the presence of circulating antibodies against the cell nucleus. Anti-nuclear antibodies are therefore often found, although many other antibodies may be present on testing. These various autoantibodies form immune complexes, which are responsible for the diverse vascular and renal lesions that occur. Lesions of lupus erythematosus are rarely found in the mouth. When present, they appear as nonspecific, chronic, red-white, erosive lesions. Diagnosis is therefore based upon clinical suspicion, involvement of skin and other organs and positive serology. Management includes topical and/or systemic corticosteroids. Prognosis depends upon the extent of systemic involvement of the disease. For the more isolated discoid disease, it is good.

25.6.14 Erythroleukoplakia (Speckled Leukoplakia)

This lesion is so called because it combines the clinical features of both leukoplakia and erythroplakia i.e. both red (atrophic) and white (keratotic) features are seen with a lesion. It often occurs on the buccal mucosa in the commissural area (just inside the cheek at the corners of the mouth), although any part of the mouth may be affected. Although it is rare, speckled leukoplakia has the highest malignant potential among the various types of leukoplakia. Therefore timely diagnosis is required for the early detection.

25.7 Pigmented and Discoloured Lesions

Pigmented lesions of oral mucosa can be considered as generalised (diffuse and multifocal), or localised involving one or several locations. Most pigmentation seen within the mouth is benign and normal. Oral tissues are normally pink but can become pale, brown, red, purple, blue, grey or black. Blue, grey, and black lesions usually occur as a result of exogenous materials (amalgam tattoos) or melanin. Brown lesions are caused by melanin or haemosiderin. Yellow discolouration can occasionally be seen as a result of bilirubin deposition or the ingestion of large amounts of β-carotene. Blue and purple lesions are generally vascular in nature. All these lesions are in themselves benign. Rarely localised discolouration can be a sign of malignancy (melanoma, oral cancer or dysplasia), or if more widespread may be a sign of systemic disease (such as anaemia, Addison's disease or cyanosis). As with the skin, brown pigmentation within the mouth can occur as a result of melanin deposition and benign nevi can develop. However, multiple small peri-oral freckles may represent Peutz-Jehgers disease, with a small small risk of internal malignancy. As with the skin, any change in a pigmented lesion may require biopsy to exclude malignancy (Fig. 25.47).

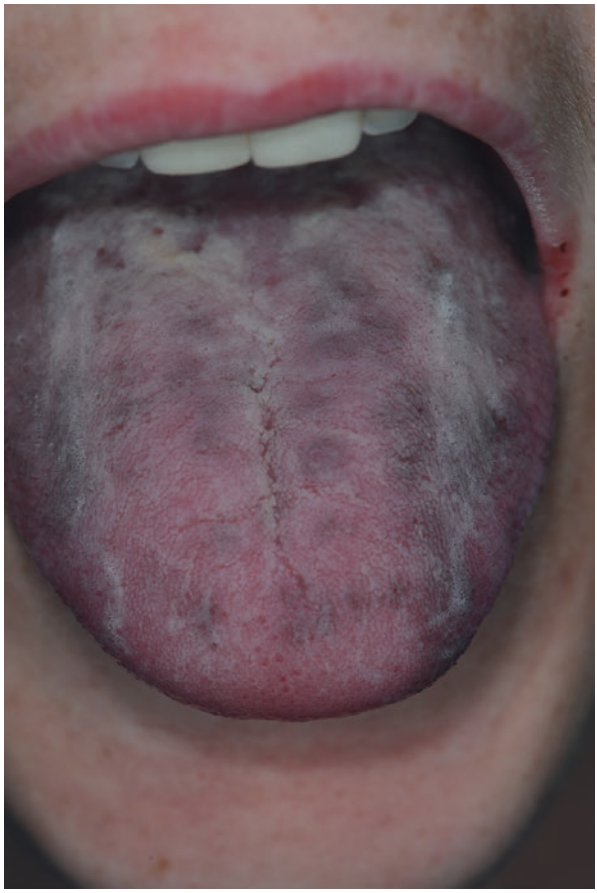

Fig. 25.47 Melanosis of the tongue

25.7.1 Racial Pigmentation and Pigmentation in Pregnancy

The most common type of generalised pigmentation is hereditary or racial. Patients with heavily pigmented skin will often have pigmentation within the mouth. This commonly involves the lips and gingiva. Oral pigmentation tends to be macular and widely distributed. No investigations or treatment are required. Melasma, sometimes referred to as the "pregnancy mask", is physiological pigmentation in the facial skin that occurs in the third trimester on pregnancy. Chloasma is a clinically identical phenomena which occurs in patients taking birth control pills. These melanoses can also involve the lips. Following delivery (or cessation of birth- control), the lesions slowly involute.

25.7.2 Oral Melanotic Macules and Nevi

Small melanotic macules can occur anywhere, including the oral cavity. These are well-circumscribed flat macules which can be gray, brown, blue, or black. Most are 1 to 3 mm in diameter. Common sites are the vermilion border of the lip, gingiva and buccal mucosa. If small, flat and uniform in appearance this requires no further treatment other than simple observation. True nevi are relatively uncommon in the mouth. These are usually small and are most commonly seen on the hard palate and buccal mucosa. Most are raised and thickened, but can also be flat. Lentigo maligna (Hutchinson's melanotic freckle), is a slow-growing, premalignant lesion. A few cases have been reported in the oral cavity, half of which became malignant. These should all be referred urgently.

25.7.3 Extrinsic Staining: Drugs, Food, Betel, Nicotine

All patients presenting with abnormal pigmentation should be asked about their occupation and diet. Industrial exposure to heavy metals, such as copper, lead, mercury or arsenic can result in discolouration of the oral mucosa, which presents as a blue-black line, around the gingivae. Blood levels may be required to look for heavy metal toxicity. Certain medications, particularly metronidazole and chlorhexidine mouth rinse are well known to discolour the tongue. This may develop a black coating, which should resolve spontaneously following cessation of the drug. If it does not sucking on a peach stone may help. Anti-malarials and chemotherapeutic agents can also present with blue-black pigmentation of the hard palate. This will slowly resolve after cessation of the offending medication.

Many different foods and drinks also contain stains. These include food dyes, some spices, tea, coffee and soy sauce. The most common site involved is the dorsum of the tongue. If oral hygiene is good these often result in temporary discolouration. However prolonged use can result in more permanent discolouration. Black or brown hairy tongue occurs when there is elongation of the filiform papillae which become discoloured by pigments contained within foods and produced by bacteria. In most cases discolouration is benign, but two important sources are nicotine and betel nut. These are both well known to be carcinogenic. Smokers melanosis is a variant of this in which tobacco results in pigmentation of the gingiva. This is most commonly seen around the gums of the lower teeth.

Another common cause of exogenous pigmentation is amalgam tattooing. This is seen in up to 1% of the general population. During placement of an amalgam restoration fine particles of the metal can become deposited in the surrounding soft tissues. The most common sites include the buccal mucosa and gingivae adjacent to the tooth. Over time the silver particles in the metal slowly leach out and stain the tissues. This can present as a Blue, Grey or black irregular lesion, often near a heavily restored tooth. Amalgam tattoos are completely benign and the diagnosis is made clinically. However they are a common source of anxiety due to concerns of malignant melanoma.

25.7.4 Diffuse Pigmentation

Many causes for this exist. They include

(i) Hereditary (racial)
(ii) Pregnancy (melasma)
(iii) Smoking (smoker's melanosis)
(iv) Medications (Antimalarials, Oral contraceptives, Cyclophosphamide, Bleomycin Phenytoin, Phenothiazines, Minocycline)
(v) Heavy metals (Bismuth, Lead, Silver, Gold, Arsenic, Mercury)
(vi) Syndromes and systemic diseases (Peutz-Jeghers syndrome, Addison's disease Neurofibromatosis, Albright's syndrome)

The development of widespread pigmentation within the oral cavity can sometimes be an early sign of Addison's disease. As secretion from the adrenal glands falls, the anterior pituitary glands compensates by producing more ACTH. This stimulates melanocytes to produce more pigmentation. All patients that present with increasing pigmentation in both the mouth and the skin should therefore be investigated. Other causes of diffuse pigmentation include Acanthosis nigricans, Cushing syndrome, McCune-Albright syndrome and Neurofibromatosis (Café au lait spots). Some drugs such as Minocycline can also result in increased pigmentation (Fig. 25.48).

Fine peri-oral freckling can be seen in Peutz-Jeghers syndrome an uncommon condition in which there is the association of intestinal polyps and an increased risk of gastrointestinal malignancy. Whilst the peri-oral freckling itself does not require treatment, the patient should be referred to a gastrointestinal specialist.

25.7.5 Malignant Melanoma

Malignant melanoma has previously been discussed. This is an aggressive disease with a relatively poor prognosis when compared to other skin cancers. It is very rare within the oral cavity (approximately 1% of all melanomas). When it does occur it

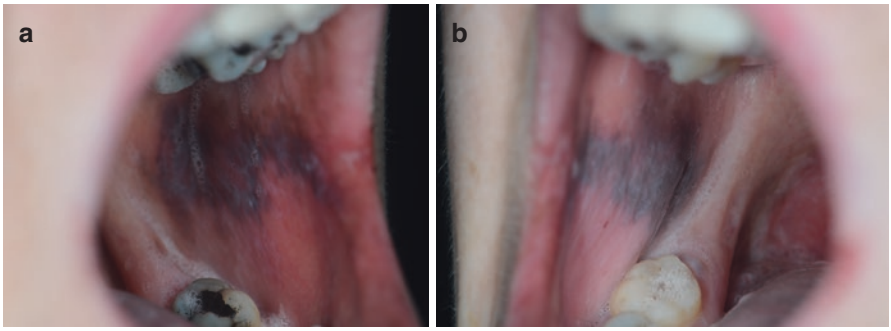

Fig. 25.48 Peutz-Jeghers syndrome

presents in the palate and maxillary gingiva as a pigmented nodule, or an area of deep pigmentation. Very rarely an amelanotic lesion maybe seen. As a rule of thumb, any symptomatic, asymmetric, irregular, variegate (different shades of different colours), ulcerative or large (>10 mm) lesion should be viewed with suspicion and urgently referred. Change in appearance or behaviour is the key to diagnosis. Treatment consists of radical surgery, often followed by radiation. Vaccines are being developed but these are currently experimental. Local, regional and distant metastases are common.

25.7.6 Haemangioma/Arteriovenous Malformation (AVM)

Haemangiomas, varices and arteriovenous malformation (AVM) are usually raised nodules or multinodular lesions that may be red, blue or purple. They are most commonly found on the lips, cheeks or tongue. They blanch with pressure. Treatment is indicated if there are problems with bleeding or cosmesis. Small lesions may be treated using sclerotherapy or laser. Very large arteriovenous malformations may require injection of sclerosing agents or embolisation. In children, treatment is generally withheld, since most haemangiomas spontaneously regress after puberty. These should not be confused with Kaposi sarcoma, a rare form of HIV-associated angiosarcoma (Fig. 25.49).

25.7.7 Discoloured Teeth

Patients presenting with discoloured teeth should be questioned about the duration and extent. The most common cause is extrinsic staining. This tends to affect all teeth and is more intense in the fissures, grooves and any surface irregularities which are harder to clean. Staining is typically brown to black in colour. The most common causes of extrinsic staining are nicotine, wine, coffee, tea, soy sauce and chlorhexidine mouthrinse. Certain conditions may predispose patients to extrinsic

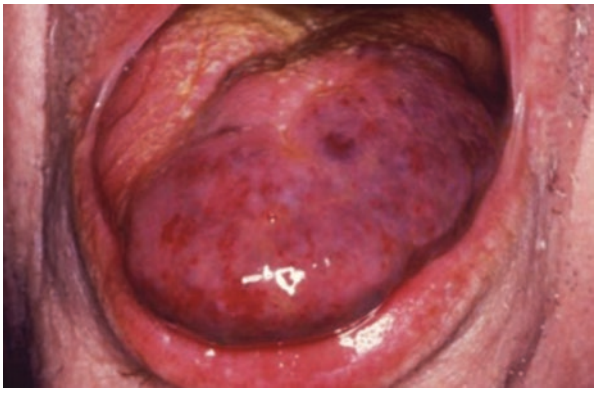

Fig. 25.49 Hemangioma of the tongue

stains, such as enamel defects, salivary dysfunction and poor oral hygiene. Treatment is usually professional scaling and polishing of teeth. Patients may also undergo tooth whitening (bleaching).

Intrinsic tooth discolouration has a number of causes. This can either be localised, (1 or 2 teeth), regional or generalised. Both primary and secondary teeth can be affected. Patients complaining of darkening of a single tooth, with a past history of dental trauma, are more likely to have a devitalised tooth with pulp necrosis. These patients need referral to their own dentist for endodontic treatment (root canal treatment), followed by bleaching to restore the colour of the crown. A tooth may also become discoloured following endodontic treatment as the root filling itself may be discoloured. Other dental materials may cause discolouration, particularly amalgam which can generate corrosion products such as silver sulfide, leaving a gray-black color in the tooth. As the patient ages the dentition progressively becomes more grey and yellow. Age-related colour changes may be due to progressive loss in enamel from tooth wear (attrition) (Fig. 25.50).

Chalky white discolouration may be a sign of dental caries and secondary hypocalcification of the enamel. In rare cases, discolouration can occur during tooth development. Tetracyclines given in the first year of life can present as a band of brown-grey discolouration affecting the permanent incisors and first molars. If given later it can affect the canine and premolar teeth. Tetracyclines are known to diffuse through the developing dentine to the enamel interface, where they bind to the hydroxyapatite matrix. Once tooth formation is complete tetracycline use can no longer lead to discolouration. Genetic defects in enamel or dentin formation which can also cause discolouration and changes in the shape of teeth include amelogenesis imperfecta, dentinogenesis imperfecta and dentinal dysplasia.

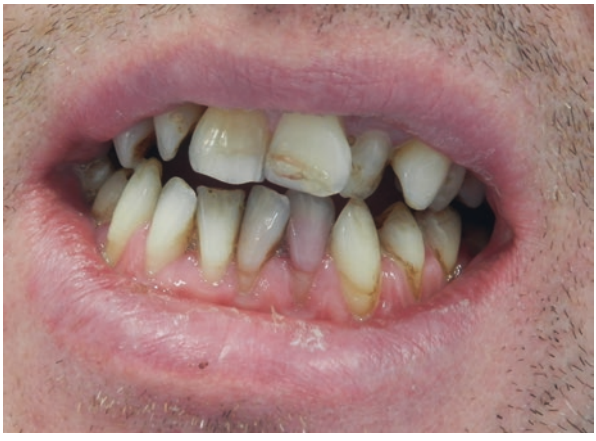

Fig. 25.50 Intrinsic tooth discolouration has a number of causes

25.8 Exposed Bone in the Mouth

25.8.1 Dry Socket (Alveolar Osteitis)

A painful extraction socket can have several causes, most commonly normal post-operative pain or an infection. Patients who have undergone surgical removal of a tooth, particularly the lower wisdom teeth are likely to have a fair amount of post-operative pain if they have not been prescribed adequate analgesia. In those patients who develop worsening symptoms after the first 1–3 days, alveolar osteitis (dry socket), retained roots, or infection should be considered. An X-ray should be taken to check for residual root fragments and rule out any pathological fracture of the mandible. It is important to exclude an abscess, cellulitis, fever, lymphadenopathy and trismus, as these may require admission for intravenous antibiotics.

Dry socket is amongst the most commonly encountered complications following extraction of teeth. It is the most common cause of exposed bone in the oral cavity. The incidence following removal of mandibular third molars is around 10–20% and is believed to occur as a result of breakdown of the initial blood clot, possibly as a result of a low-grade infection. The underlying bone then becomes exposed and very painful. This is more likely to occur in patients who smoke and in female patients who take oral contraceptives. Dry socket typically presents at 24–72 h following dental extraction, usually a lower wisdom tooth. Pain is usually an intense, throbbing, constant ache, often with some degree of trismus and swelling. Exposed bone in the socket can be seen and there may be halitosis and a foul taste in the mouth. If the patient is systemically well management involves irrigation of the socket, gentle débridement, placement of an sedative dressing and antibiotics. Alvogyl is the most common dressing. This contains butamben (anaesthetic), eugenol (analgesic) and iodoform (antimicrobial). In the majority of cases these local measures are satisfactory and the socket heals in 2–4 weeks. If there are symptoms or signs of more extensive or systemic infection (fever, lymphadenopathy, severe trismus, dysphagia etc.) it is probably safer to admit the patient for IV antibiotics. Whilst dry socket is not a serious infection, if this spreads to invoke the surrounding soft tissues and mandible this becomes significant.

25.8.2 Osteomyelitis

This is discussed further in the chapter on the lower jaw. Osteomyelitis may be defined as an infection in bone *with a tendency to progression*. Patients most at risk include diabetics, immunocompromised, the elderly and smokers. It can involve both the cancellous bone and the cortical plates of the mandible, extending into the subperiosteal and surrounding tissues. Today, osteomyelitis is relatively uncommon due to the early use of antibiotics. However, antimicrobials have been reported to be less effective, as a result of increasing bacterial resistance. Bone is quite resistant to bacterial colonisation, however trauma, surgery, or the presence of foreign bodies (fixation plates) can predispose it to infection. Infection is more common in the

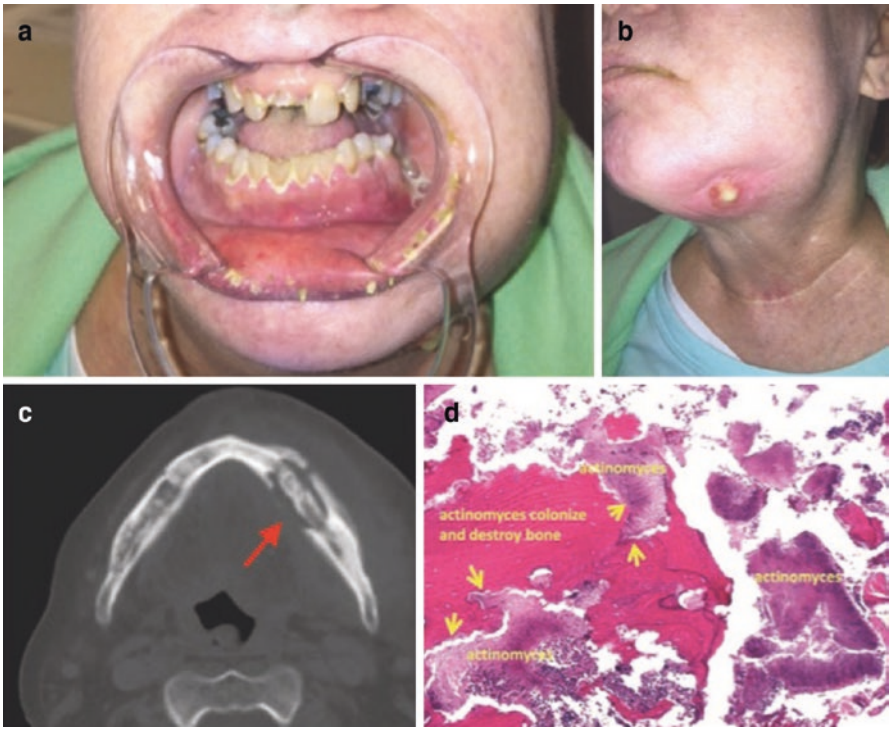

Fig. 25.51 (**a**–**d**) Chronic osteomyelitis

lower jaw, as the upper jaw has a relatively better blood supply, compared to the mandible's dense and less vascularised cortical plates. Osteomyelitis can also result from haematogenous spread, although this is rare in the jaws. Acute osteomyelitis of the jaw tends to present as dull, severe throbbing pain, with swelling, redness and lymphadenopathy. The teeth may become loose or tender to percussion. In chronic osteomyelitis, patients may present with persistent fistulas. An OPG will show a 'moth eaten' appearance and possibly the cause, such as a fracture, or infected tooth. Treatment requires antibiotics and debridement (Fig. 25.51).

25.8.3 Radiation: Osteoradionecrosis (ORN)

This is also discussed further in the chapter on the lower jaw. Irradiation for malignant lesions of the upper aerodigestive tract and salivary glands commonly includes the jaws and associated structures. Such radiation results in a reduction of cellularity and vascularity in these structures, secondary hypoxia and poor wound healing. This is most commonly seen in the mandible. Poor healing can occur following extraction of a tooth. Removal of teeth, especially third molars is the most common cause of ORN in the oral cavity. For this reason all patients undergoing radiation

therapy to the head and neck need a thorough dental assessment and removal of at-risk teeth prior to treatment. ORN can result in large necrotic areas of exposed bone that may be painful, suppurative, and result in fistula formation and even fracture. Treatment is difficult and described elsewhere.

25.8.4 Medication Related Osteonecrosis of the Jaws (MRONJ)

This is defined as the presence of exposed bone that does not heal within 8 weeks. Previously known as BRONJ (bisphosphonate related osteonecrosis of the jaw), it is now recognised that jaw necrosis can occur as a complication with a number of other drugs, including denosumab (a human monoclonal antibody) and anti-angiogenic agents. However bisphosphonates still remain the most common cause of MRONJ. These are commonly prescribed for osteoporosis, multiple myeloma and painful bony metastases. Intravenous bisphosphonates are much more potent than oral. The drug binds irreversibly to bone, inhibiting osteoclasts and bone remodelling. Diagnosis of MRONJ is based on three characteristics (i) current or previous treatment with antiresorptive or antiangiogenic agents (ii) Visibly exposed bone (or bone that can be probed through a fistula) for more than 8 weeks and (iii) no history of irradiation or metastatic disease in the jaws.

MRONJ can occur spontaneously, but is more likely following trauma to the bone, such as following a dental extraction, especially if surgical. Patients presents with areas of exposed and necrotic bone. This can be asymptomatic, but symptoms can develop if the surrounding soft tissues become inflamed. These include prolonged jaw pain, tooth mobility, bony enlargement, gingival swelling, erythema and ulceration. Untreated, fistulae may develop if the necrotic bone becomes secondarily infected. This may then mimic osteomyelitis. With maxillary involvement chronic maxillary sinusitis can occur. Investigations include OPG/CT and sometimes biopsy. Management involves cessation of the drug (if possible), control of pain and secondary infection, and prevention of extension. Treatment is conservative wherever possible, with chlorhexidine mouthwash and antibiotics. Areas of necrotic bone that cause soft tissue irritation and loose sequestrae can be removed or recontoured to allow soft tissue healing. If the area of necrotic bone becomes extensive, resection and reconstruction may be required. Because MRONJ can cause such problems, prescribers of these drugs should refer patients for dental assessment and extractions prior to commencement.

25.8.5 Malignant Invasion

Exposed bone in the jaw can also be a sign of malignancy. The most common oral cancer involving bone is primary SCC with local bony invasion. Primary bone tumours (such as osteosarcoma) and metastases from breast, lung and renal cell carcinoma can also occur but are rare. In these cases patients may present with exposed necrotic bone, surrounded by soft tissue ulceration, induration, and

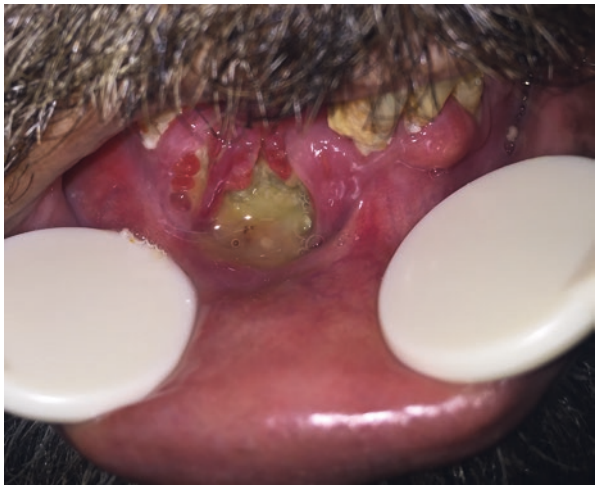

Fig. 25.52 Chronic infection with discharge

erythema. Symptoms can occasionally mimic other conditions such as dental infection, osteonecrosis and osteomyelitis (Fig. 25.52).

25.9 Generalised Pain and Alteration of Sensation Within the Mouth

25.9.1 Xerostomia (Dry Mouth)

A dry mouth can be either a subjective feeling, in which the mouth appears moist, or an objective finding during examination. Even in the absence of major salivary gland disease, function can still be abnormal. Ageing itself does not cause significant deterioration of salivary gland function. However, postmenopausal women have the highest incidence of Sjögren's syndrome and most frequently complain of a sore or dry mouth. Diagnosing subjective dryness can often be very difficult. Many patients who complain specifically of a dry mouth are usually found to have normal salivary flow production. Causes of xerostomia include the following

(i) Depression and chronic anxiety states—However no correlation has been found between the degree of xerostomia and the severity or type of depression. Patients with chronic fatigue syndrome may also complain of dry mouth. Anxiety states with significant sympathetic overactivity and drying of the mouth may affect public speakers. In such cases, a beta-blocking agent may be helpful.

(ii) Drugs—There are now over 400 medications known to cause dry mouth as a side effect, making this the commonest cause after anxiety (physiological xerostomia). Tricyclic antidepressants and phenothiazine neuroleptics are among the most troublesome drugs. These have particularly strong antimusca-

rinic effects and are generally used over long periods. Some antihypertensive drugs also cause xerostomia.

(iii) Salivary gland disease—Sjögren's syndrome, radiation damage, sarcoidosis, HIV infection, amyloidosis, hyperlipoproteinaemia can all result in the feeling of dry mouth.

(iv) Dehydration—Loss of body fluids secondary to diarrhoea or chronic vomiting, polyuria (diabetes and diuretics) should be considered.

(v) Radiotherapy

(vi) Mouth breathing (Figs. 25.53 and 25.54)

Patients with objectively impaired salivary flow may not necessarily complain of a dry mouth. Rather, they may complain of difficulties in eating dry foods. In severe cases, xerostomia can affect the patient's speech as a result of intra oral stickiness. Other changes suggestive of a dry mouth include accelerated dental decay, poor oral hygiene and candidiasis. In severe cases, the mucosa appears wrinkled and parchment-like. Suppurative parotitis can also occur. Management involves relief of symptoms and control of the complications. Patients should take frequent sips of water, especially at meal times. Sugar-free chewing-gum is also helpful as mastication increases salivary flow. Artificial salivas and cholinergic drugs, such as pilocarpine, have been reported to be of value but functional salivary tissue must be present for the latter. Attention to the diet, strict oral hygiene and application of fluorides are also important. All patients with dry mouth should be asked if they have dry/gritty eyes. If Sjögren's syndrome is suspected (discussed elsewhere in this book), early

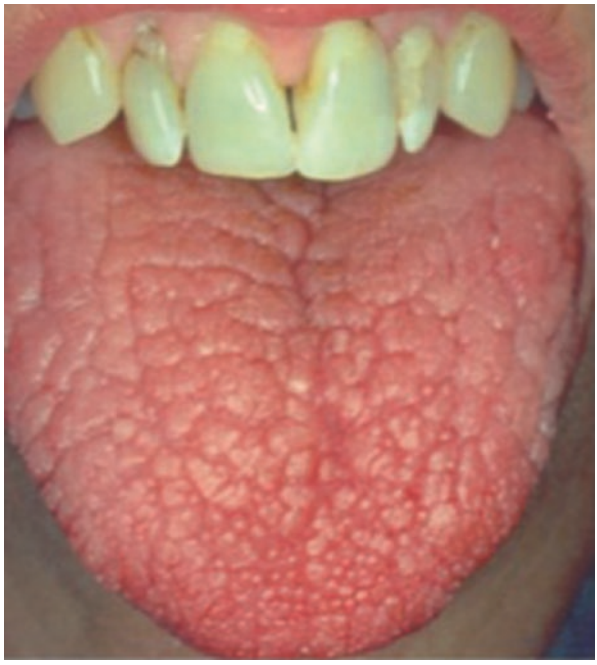

Fig. 25.53 Mucosal alterations in Sjögren's syndrome. Dry, furrowed, cobblestone appearance of the tongue of a patient with severe salivary gland dysfunction from Sjögren's syndrome. Note also the extensive restored dental caries

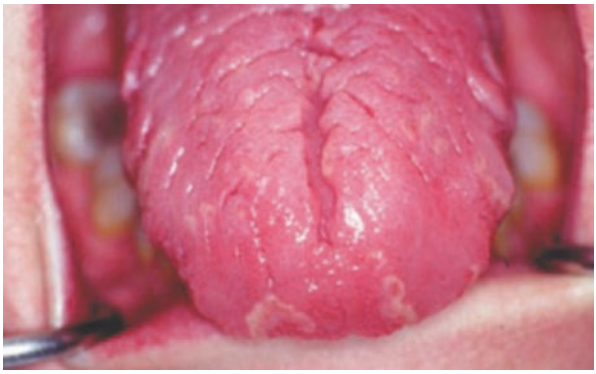

Fig. 25.54 Radiation-induced xerostomia of the oral tongue with fissuring of the dorsal anatomy, ere nations, and leukoplakia

referral to ophthalmology should be made. Keratoconjunctivitis sicca is often asymptomatic and should be excluded.

25.9.1.1 Radiation Induced Xerostomia

This is a common complication following radiotherapy in head and neck cancer patients, where the parotid glands have been included in the radiation fields. Fibrosis, necrosis and severe dysfunction may develop months or years after treatment. Acute xerostomia can also occur during radiotherapy. This is often complicated by candidiasis and mucositis. Pilocarpine, a muscarinic agonist which stimulates salivary production, helps reduce the severity of acute symptoms but cannot prevent radiation induced dysfunction.

25.9.2 Mucositis

Mucositis is another common complication of radiotherapy and chemotherapy, notably with 5-fluorouracil (5-FU), cisplatin, etoposide and melphalan and in haemopoietic stem cell transplant patients. It is characterised by ulceration of the oral, oesophageal and gastrointestinal mucosae, resulting in pain, dysphagia, diarrhoea and malabsorption. Oral mucositis results in severe discomfort and a burning sensation which impairs the patients ability to eat, swallow and talk. This can develops within a few days of receiving chemotherapy, or when radiation doses reach 10 Gy. Ulcers soon develop. Mucositis can predispose to bacteraemia, septicaemia and fungaemia which can be life-threatening. Streptococci and Candida species are the most common organisms. Chemotherapy-induced mucositis lasts about 1 week and usually heals spontaneously over 1 month. Radiation-induced mucositis last longer. Chronic mucositis can occasionally develop. Management includes topical benzydamine, amifostine, Leucovorin and Granulocyte-macrophage colony stimulating factor. Recombinant human keratinocyte growth factor (KGF) has also been reported to reduce severity.

25.9.3 Trigeminal Neuralgia (See Also the Lower Jaw)

Trigeminal neuralgia (TN) can present with oral pain, either in isolation, or more commonly with pain in the lower jaw and lower lip. The pain is often described as a sudden, electric shock type pain localised to one or more divisions of the trigeminal nerve. It is almost always unilateral and may be bought on by 'triggers' such as toothbrushing, shaving, drinking cold or hot fluids, talking, chewing, or cold air. If symptoms are bilateral, systemic disease should be suspected, such as multiple sclerosis and the patient referred to a neurologist. Diagnosis of trigeminal neuralgia is often clinical, based largely on the symptoms and their dermatomal distribution. CT or MRI are usually undertaken to exclude alternative diagnoses and to assess for microvascular compression. Useful diagnostic criteria for TN as defined by the International Headache Society includes

(i) Paroxysmal attacks of pain lasting from a fraction of a second to 2 min, affecting 1 or more divisions of the trigeminal nerve and fulfilling criteria (ii) and (iii)
(ii) Pain has at least 1 of the following characteristics: (i) intense, sharp, superficial or stabbing or (ii) precipitated from trigger areas or by trigger factors
(iii) Attacks are the same in any individual patient
(iv) No clinically evident neurologic deficit
(v) Cannot be attributed to any other disorder

Treatment for trigeminal neuralgia includes, pharmacologic therapy (gabapentin, carbamazepine, oxcarbazepine or amitriptyline), percutaneous procedures (percutaneous retrogasserian glycerol rhizotomy), surgery (microvascular decompression) and radiation therapy (gamma knife surgery). A full blood count and liver function tests are required if therapy with carbamazepine is started. Oxcarbazepine can also cause hyponatremia. With acute pain a local anaesthetic block can often bring temporary relief.

25.9.4 Burning Mouth Syndrome

Burning Mouth Syndrome (BMS) is a common condition characterised by a burning, tingly discomfort inside the mouth usually in the absence of clinical and laboratory findings.

Affected patients often present with multiple oral complaints, including burning, dryness and taste alterations. BMS predominantly affects the tongue but can involve the mucosa and lips. It is most prevalent in postmenopausal women. Symptoms often arise in more than one site, commonly the anterior two thirds of the tongue, the anterior hard palate and the mucosa of the lower lip. It is often accompanied by gustatory disturbances (dysgeusia, parageusia) and subjective xerostomia. Rare cases have also reported of patients losing their sense of smell. However of note is that pain is absent during the night. It progressively increases throughout the day. Known associations include

(i) Personality traits, especially anxiety and depression. These have been consistently demonstrated in patients with burning mouth syndrome and have been used to suggest that the disorder is a psychogenic problem. The effectiveness of tricyclic antidepressants and some benzodiazepines may be partly related to this, as well as their analgesic properties and effects on taste-pain pathways.
(ii) Systemic factors. Although burning mouth syndrome has not been linked to any specific medical condition, associations with a wide variety of conditions have been reported. These include high blood glucose levels, hypothyroidism and nutritional deficiencies (iron, folic acid, vitamins B1, B2 and B6, zinc.) Hormonal changes are believed to play a role. Approximately 90% of women in some studies have been postmenopausal.
(iii) Dry mouth.
(iv) Chronic low-grade trauma secondary to parafunctional habits (e.g. rubbing the tongue against the teeth, or pressing it against the palate) may be involved. BMS is more common in persons with Parkinson's disease.
(v) Taste. The role of taste in burning mouth syndrome is unclear but there an apparent group of "supertasters" (persons with enhanced abilities to detect taste) among patients with burning mouth syndrome.
(vi) Case reports have also linked burning mouth symptoms to the use of angiotensin-converting enzyme (ACE) inhibitors.
(vii) Candidal infections are also reported to cause burning mouth syndrome.

Investigations are therefore quite diverse but often negative. Currently no definitive treatment exists. Patients may experience temporary relief from topical local analgesia. Antidepressants (e.g., amitriptyline) and neuropathic pain agents (gabapentin) are commonly prescribed. Given in low dosages, benzodiazepines, tricyclic antidepressants or anticonvulsants may be effective. Topical capsaicin (the active ingredient in Pepper spray or 'Mace') has been used as a desensitising agent in some patients, but may not be palatable in others. Cognitive behavioural therapy may also be helpful.

25.9.5 Reflux Oesophagitis

Detailed discussion of this condition falls outside the scope of this book. Nevertheless this can result in oral discomfort. Usually the diagnosis of any oral symptoms is straightforward as patients present with a history of burning epigastric pain which extends into the mouth. There may also be other findings including halitosis, indigestion, or vomiting. If severe this can result in dental erosions, typically seen on the palatal surfaces of the upper teeth. Patients should be referred for investigations such as Helicobacter pylori and may require treatment with proton pump inhibitors and antacids. Spicy food, smoking and alcohol should be avoided.

25.9.6 Tumours

Malignancy and large lesions may cause oral and facial pain or paraesthesia as a result of compressing or invasion of nerves. Consider these in patients presenting with a numb lower lip. On examination a neoplastic mass may not be evident clinically, and so diagnosis is often confirmed by CT or MRI.

25.9.7 Iatrogenic Injury to the Inferior Alveolar and Lingual Nerve

Of the three major divisions that emerge from the trigeminal ganglion, the mandibular is the largest. This is both a sensory and motor nerve. The inferior alveolar nerves enter the mandible to innervate the lower teeth, emerging from the mental foramen to innervate the skin of the lower lip. Any mechanical injury to the mandibular division can result in a form of compression or entrapment neuropathy. Common sites include the infra temporal fossa, (just below the middle cranial fossa), the pharynx and the mandibular ramus. Irritation of the nerve from the maxillary artery or local pathology can therefore result in numbness of the lower lip. The mandibular nerve can also be compressed when passing between the medial and lateral pterygoid muscles. When these contract, both the Inferior alveolar and lingual nerves may be compressed. Other important causes to note are cysts and tumours arising within the mandible. Numbness of the lower lip may thus be the first indication there is a serious problem.

Nerve injuries are an inherent risk of many surgical procedure in nearly all areas of the body, and the oral cavity is no exception. They may occur despite all efforts being taken to prevent it—surgery is never risk-free. Injury to the inferior alveolar and lingual nerves are known complications in dentistry and jaw related surgical procedures. These procedures include third molar (wisdom teeth) removal, endodontic (root canal) procedures, dental implant placement and facial trauma surgery. Injury to the lingual nerve also occur as a result of crushing or transection following surgical procedures in floor of the mouth. This can result in dysaesthesia, paraesthesia, or even anesthesia. Patients may also experience dysgeusia (difficulty in chewing) and loss of gustatory function on the side of the injury. Speech articulation can also be affected.

The most common procedure resulting in injury to both these nerves is wisdom tooth removal. This is due to the proximity of the roots of some wisdom teeth to the inferior alveolar nerve. The lingual nerve is also nearby. These nerves can therefore be injured directly at the time of surgery or affected temporarily afterwards, as a result of swelling. Dental injections (mandibular nerve blocks) can also result in injuries to the IAN and LN injuries. This has been reported to occur in about 3.5% of patients following injection. Direct neural trauma, local anaesthetic toxicity and a focal haematoma have all been suggested as possible causes. Symptoms vary and include numbness, tingling, burning, electric shock-type, or hypersensitivity of the

affected area—painful anaesthesia (anesthesia dolorosa). These may interfere with normal function and can range from minor to quite troublesome. Persistence beyond several months following the initial injury, may indicate that an injury will not resolve and should be evaluated further. Early steroid therapy and/or nerve exploration may be beneficial in selected cases. Other possible iatrogenic causes of injury include jaw (orthognathic) surgery, inadvertent screw placement following mandibular fracture repair, other surgical procedures to the mandible, endodontic and periapical surgery to the mandibular molar and premolar teeth and placement of implants. Spontaneous numbness however, should always be viewed with suspicion.

25.10 Some Denture and Orthodontic Related Problems

25.10.1 Denture Related Problems

For the vast majority of denture problems, patients should be redirected to their local dentist for assessment and further treatment. Most problems are transient and may be essentially disregarded by the patient, but occasionally they may be serious enough to result in the patient being unable to tolerate denture wear. Problems may be considered as.

- Adverse intra-oral anatomical factors eg atrophic mucosa.
- Clinical factors eg poor denture stability.
- Technical factors
- Poor patient adaptation.

Ulceration can be caused by loose old, or tight new dentures, rubbing against the adjacent soft tissues. Tongue biting may occur if the denture is too loose or bulky. This is usually obvious on clinical examination. Patients should be advised not to wear the denture and may benefit from topical lignocaine gel to affected areas and chlorhexidine mouthwash to prevent infection. The patient should see their dentist for denture adjustment. Ulcers typically resolve within 2 weeks following removal of the traumatic stimulus. Any ulcer which fails to heal requires a biopsy to exclude malignancy.

25.10.1.1 Loose or Broken Dentures

As a result of continued resorption and remodelling of the supporting alveolar bone, dentures tend to loosen with age. Patients with severely loose or broken dentures should not wear them, and instead see their dentist for repair or relining of the prosthesis.

25.10.1.2 Denture Stomatitis

Patients with denture stomatitis present with persisting redness of the tissues underneath the denture, most commonly the palate. There is usually a characteristic line demarcating normal mucosa from the inflamed tissue. It is often seen in patients

who do not remove their dentures at night, or who do not clean them adequately. Although harmless, denture stomatitis often represents candidal infection. Diagnosis is often clinical, but swabs can be taken to confirm the presence of candidal hyphae. Treatment tends to involve a combination of antimicrobials, such as nystatin or miconazole, together with advising the patient to remove the denture at night, and cleaning it with an appropriate denture cleansing solution.

25.10.1.3 Soft Tissue Changes: Denture Hyperplasia, Flabby Ridge, Denture Fibroma

Patients with long-standing, poorly-fitting dentures can develop firm fibrous swellings under the denture. These can occur as discrete lumps (denture fibroma) of a more generalised thickening (denture hyperplasia). If there are no natural teeth this is often called a 'flabby ridge'. These are effectively the oral equivalent to calluses if poorly fitting shoes are worn. The thickened tissues are usually non-inflamed, firm and have no sinister signs. Treatment involves surgical resection of the offending tissue and relining or remaking the dentures to ensure a snug fit.

25.10.1.4 Dentures Too Tight

It is very unusual for patients to present with dentures which become tighter over time. If this occurs consider bone-related conditions such as acromegaly or Paget's Disease.

25.10.2 Orthodontic Appliances

Orthodontic appliances can be fixed or removable. Fixed appliances are commonly referred to as braces. Orthodontic appliances are essentially used to straighten teeth. Indications for orthodontic treatment include aiding oral hygiene, preventing periodontal disease with crowded teeth, improving the bite and for cosmetic reasons. Issues that may be encountered in the emergency setting include:

(i) Pain and sensitivity—usually after recent adjustment
(ii) Staining/demineralisation of the teeth
(iii) Gingivitis
(iv) Bad Breath
(v) Broken/loose appliance

These patients should referred back to their orthodontist or regular dentist. Broken appliance may lacerate the soft tissues, which should be managed first. Don't try and remove these, unless they are very loose. Orthodontic wax can be applied to sharp edges of wire and brackets as a short term measure. If this does not work and the wire is very loose it may need to be cut with a wire cutter. If brackets have to be removed, make a note of how many are. If there has been significant trauma or a history of loss of consciousness consider the possibility of foreign body aspiration.

25.10.3 Restorative Dentistry Related Issues

These all relate to issues that are best managed by a dentist. Issues that may present following treatment in the emergency setting include:

(i) Pain and swelling during endondontic (root canal) treatment is common following instrumentation of the dental pulp. Patients may present for pain relief or antibiotics. If pain or swelling is significant then help from the on call Maxillofacial team may be needed.
(ii) Patients undergoing prothodontic treatment (crowns, bridges dentures) may present mid treatment or following treatment. The commonest complaint is loss of the prosthesis. Many patients will have found the prosthesis and will need to be told to see their Dentist to replace/re-cement this in the mouth. If following trauma where there has been loss of consciousness or where a patient does not remember dropping the prosthesis a chest X-ray and soft tissue view of the neck may be indicated to assess for aspiration.

25.11 Miscellaneous Infections of Oral Mucosa

25.11.1 Syphilis

Syphilis is a venereal infection caused by the spirochete Treponema pallidum. It can also be transmitted by transfused blood and crosses the placental barrier from mother to foetus. The initial site of infection (primary syphilis) typically forms an area of ulceration (chancre), with regional lymphadenopathy. These persist for 3–10 weeks and then resolve spontaneously. Secondary syphilis subsequently develops after a latent period of several weeks. This results in fever, malaise, a maculopapular rash and multiple ulcerations on mucosal surfaces. If the patient is still untreated, the disease may enter a prolonged latent period which can last months or years. Tertiary syphilis develops in only a few patients. This can present in many ways, owing to the extensive involvement of differing systems. Central nervous system involvement can present as generalised paralysis or tabes dorsalis. Inflammation of the vascular system can result in aneurysms. Intraoral manifestations include granulomatous proliferations (gumma), and a generalised glossitis. Diagnosis of syphilis is usually made following serologic studies (Venereal Disease Research Laboratory and fluorescent treponemal antibody absorption tests). Treatment is high dose penicillin. For patients allergic to penicillin, erythromycin or tetracycline may be used. The Jarisch-Herxheimer reaction is an acute febrile reaction accompanied by headache, myalgia, fever and other symptoms. This usually occurs within the first 24 h of treatment.

25.11.2 Gonorrhea

Gonorrhea is caused by *Neisseria gonorrhoeae*, a gram-negative diplococcus. Transmission is usually venereal and can involve genital, oral or pharyngeal mucosa. The incubation period is about 1 week with initial symptoms of mucosal ulcers (more commonly pharyngeal) and regional lymphadenopathy. Thus, consider gonorrheal infection in any patient presenting with chronic aphthous-like ulcers predominantly involving the pharynx. Diagnosis is based on isolation of the organism and immunofluorescent antibody. Treatment is with penicillin.

25.11.3 Measles

This begins with high fever, conjunctivitis, photophobia, cough and nasal discharge. Red vesicles with white centres (Koplik's spots) appear on the buccal mucosa, followed by an erythematous maculopapular skin rash first on the face and then the trunk and extremities. Management is symptomatic. It is usually a self-limited disease but can have serious complications, including croup, bacterial pneumonia, otitis media, and encephalitis.

25.11.4 Rubella

German measles is a mild infectious disease, but it can cause serious foetal malformations when it occurs during pregnancy. A prodrome of malaise, fever, mild conjunctivitis, and lymphadenopathy can be followed by oral vesicles, ulcers, arthralgia and a maculopapular skin rash which begins on the face and spreads to the trunk and extremities. It usually lasts for about 3 days.

25.11.5 HIV

This destroys helper T lymphocytes, resulting in immunosuppression which predisposes to opportunistic infections and tumours. The virus is transmitted by sexual intercourse, contact with blood/blood products and perinatally. It is also found in saliva. HIV infection presents with malaise, fever and lymphadenopathy. Neurologic disorders are common and range from subtle memory loss to dementia. Opportunistic infections and malignant neoplasms are part of acquired immunodeficiency syndrome (AIDS). Many of these can be present in the oral cavity. (i) Candidosis may present in HIV patients. This responds to antimycotic medications, but is chronic and recurrent. (ii) Hairy leukoplakia is most commonly found on the lateral surface of the tongue. It is pathognomonic of HIV infection and is highly predictive that AIDS will develop. (iii) Herpes simplex and herpes zoster are also more frequent

and severe in HIV patients. (iv) A unique form of periodontal disease can develop with chronic gingival erythema, severe pain, soft tissue necrosis and rapid destruction of alveolar bone and the periodontal attachment. This "HIV periodontitis" does not respond to conventional therapy alone. (v) The most common malignant neoplasms involving the oral cavity in HIV patients are Kaposi's sarcoma, non-Hodgkin's lymphoma, and squamous cell carcinoma. Other oral manifestations of HIV infection include salivary gland enlargement, xerostomia and ulcerations similar to aphthous ulcers.

25.12 Recreational Drug Use and the Oral Cavity

An important cause of oral ulceration is recreational drug use. This includes placing cocaine in the buccal mucosa causing a white or erythematous burn to amphetamine use which causes users to involuntarily chew their lips leading to the white or keratinised appearance described above. In some cases this involuntary chewing has been so exaggerated that patients have unduly chewed their own lip to such an extent that a portion is either eaten or lost. This poses considerable difficulty to the oral and maxillofacial surgeon as simple primary closure may not be possible, or even if possible with result in a dog ear or considerable asymmetry when the patient smiles. Common affects of amphetamines are dry mouth, bruxism and jaw clenching. The thirst may be quenched with sugary drinks and since an amphetamine induced trip can be up to 36 h dependant on the agent used users can have signs of myofacial and temporomandibular joint pain and significant tooth surface loss (Fig. 25.55).

Significant users of recreational drugs will also show a tolerance to local anaesthetics and may require greater quantities to achieve pain free treatment. Opioid users, particularly heroin users have increased incidence of tooth decay linked either directly or indirectly to heroin use. Heroin users can hold the drug or methadone in their buccal mucosa increasing contact time with teeth. Sugar free forms of methadone have now been developed.

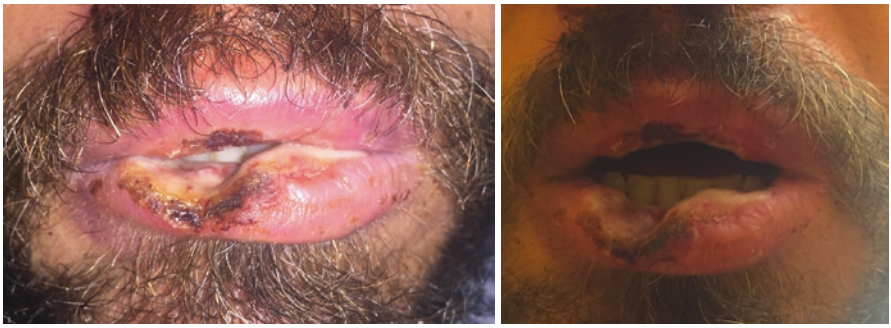

Fig. 25.55 Lip trauma following recreational drug use