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# 73 Tropical Oral Health

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**KEY POINTS**

- Oral health poses a major health burden for many countries, and some forms of oral disease are specific to tropical countries.
- Oral diseases are the most common non-communicable diseases and share risk factors with many other non-communicable diseases.
- Some 90% of the world's population is affected by tooth decay.
- A high proportion suffer from periodontal disease.
- Oral cancer rates are high among smokers and tobacco users.

## Introduction

The importance of oral health as part of general health is now well established and this is true not only in industrialized countries but also tropical and subtropical climates. Global Oral Health as a specific discipline is beginning to take shape, with support from the World Health Organization (WHO) and World Dental Federation. The focus for the early part of the twenty-first century, for international oral health, will be dental caries, and the commitment to eradicate dental cavitation (or at least confine to <10%) in the child cohort born in 2026.[1]

It is also important to recognize that tropical dentistry is not just dentistry (oral health) in the tropics, but with migration and global travel, oral diseases traditionally restricted in some developing countries have manifested themselves within all areas of the global community. This chapter will cover oral conditions common in all parts of the world, as well as tropical region-specific conditions and risk factors.

## Dental Caries

Together with the common cold, dental caries is perhaps the most prevalent disease of modern man, but unlike the cold, its effects leave behind defects that are permanent.[1] The general consensus of international epidemiological studies is that non-milk extrinsic sugars are the most important dietary factor in the aetiology of dental caries. The role of nutrition during tooth development is considered to be minimal in industrialized countries.[2,3] However, in tropical and subtropical areas where malnutrition is evident, delayed tooth eruption is observed, especially in the primary dentition,[4] but there is inconclusive evidence that malnutrition during tooth development can influence subsequent levels of dental caries.[5]

In the last few decades, there has been enormous progress in development, with decreased infant mortality and increased life expectancy, literacy and sanitation in developing countries. Such development is all too often coupled with increasing access to sugars, commonly in the form of confectionery or carbonated drinks. The WHO's global databank on oral health established in 1969, and continuing to monitor dental caries levels across different countries, demonstrates two clear trends: first the ongoing decline in dental caries for the industrialized world and, second, the increasing prevalence of caries in the developing world.[6]

The treatment of dental caries has not essentially changed over the past few decades, although tooth cavity design and filling materials have changed the practical approach to dental restorative treatment. The Atraumatic Restorative Technique (ART) has produced promising results in developing countries, especially those with a shortage of suitably qualified manpower.[7]

There have been a number of studies that have demonstrated significant caries reduction as a result of fluoride toothpaste. The major barrier to the implementation of fluoride toothpaste to the developing world has been cost; however, the new WHO programmes to introduce locally produced affordable fluoridated toothpaste to many developing countries are producing encouraging results.[8]

The evidence base for addressing dental caries in children has been documented and the predictive models and policy options to managing this clarified.[1] It is clear that each health economy needs to document its fluoride policy and preferably adopt either a water fluoridation or improving the child oral health.[1] Cultural habits such as the use of smokeless tobacco are known to have an effect on dental caries.[9] A significantly higher number of cariogenic bacteria are found at the site of smokeless tobacco placement, attributed to the sugar content in these products which enhance the growth of bacteria such as *Streptococcus mutans*.[10] Although there is some evidence of an association between smokeless tobacco and dental caries, a cause and effect relationship cannot be conclusively established.

## Periodontal Disease

There is no evidence that inflammatory periodontal disease in developed and developing countries is in principle different in character.[11] There are indeed more similarities in periodontal conditions globally than differences. Evidence shows however, that periodontal diseases are more prevalent in developing countries in terms of poorer oral hygiene and greater calculus retention but not in terms of periodontal destruction in adults.[11]

The WHO has published guidelines on prevention.[12] Limited resources, in many developing countries, often inhibit the purchase of toothbrushes, and traditional cleaning materials such

as the miswak chewing stick which is still widely used and has been shown to be an effective tooth-cleaning agent, comparable in effectiveness with some agents used in Western societies.[13] In addition, tobacco products (powder and paste), charcoal and ash have been used as cleaning agents in parts of southern Asia and Africa. They are used by applying tobacco to the teeth and gums, usually with the index finger. Other specific tobacco products used as dentifrices are Gul, Mishri, Bajjar, Gudakhu and tobacco water (used as a mouthwash).[14]

## Oral Cancer

Most oral cancer is squamous cell carcinoma (SCC), and it is customary to include cancers of the lip (ICD 140), tongue (ICD 141), gum (ICD 143), floor of the mouth (ICD 144) and unspecified parts of the mouth (ICD 145).[15] There is clear inter-country variation in both the incidence and mortality from oral cancer and also ethnic differences, which are attributed mainly to specific risk factors such as alcohol and tobacco (smoking and smokeless), betel use, and, in the case of lip cancer also sunlight exposure, but dietary factors as well as the existence of genetic predisposition may play a part. Variations in availability and access to care services are also evident.[16]

The incidence of oral cancer varies widely between countries and geographical areas of the world and is generally most common in developing countries, particularly in Asia, though Eastern Europe and France have some of the highest recorded rates globally. Mouth cancer worldwide is the 12th most common cancer but it is the 8th most common in males.[17] Annually, there are 197 000 deaths worldwide from cancer of the mouth and pharynx, with the highest mortality from mouth cancer in Melanesia and South-Central Asia. The gender ratio is 2 : 0 (M : F). Mouth cancer in men is most common in Eastern Europe, South Asia, Melanesia, southern Africa and Australia/NZ. In females, it is most common in South-Central Asia, Melanesia and Australia/NZ. Lip cancer is particularly common in white Caucasians in the tropics and subtropics.[17]

The aetiology of oral cancer has been attributed to specific risk factors: tobacco[18] and/or alcohol in southern Africa, and betel quid in people of South-Central Asian and Melanesian cultures.[19] Smokeless (chewing) tobacco use is an important factor for South Asian populations. The areca (betel) nut habit is important in the development of oral submucous fibrosis and of mouth cancer. Some chew the nut only and others prefer 'paan', which includes tobacco, and sometimes lime and catechu. Studies from India have confirmed the association between 'paan' tobacco chewing and oral cancer, particularly cancer of the buccal and labial mucosa. There is growing evidence associating increased alcohol consumption with risk of oral cancer. The role of alcohol drinking is observed in a negative social class gradient and for many countries, follows a similar pattern to tobacco use. Finally, there is an increasing predisposition of younger (<45 years) people to oral and pharyngeal cancer in many countries, and in this respect, human papillomaviruses (HPV) are increasingly linked.[20]

The molecular changes found in oral carcinomas from Western countries (UK, USA, Australia), particularly p53 mutations, are infrequent in the East (India, South-east Asia), where the involvement of ras oncogenes, including mutation, loss of heterozygosity (H-ras) and amplification (K- and N-ras) are common, suggesting genetic differences. It is also evident that there can be genetic differences in the metabolism of pro-carcinogens and carcinogens by xenometabolizing enzymes or ability to repair the DNA damage in different ethnic groups.

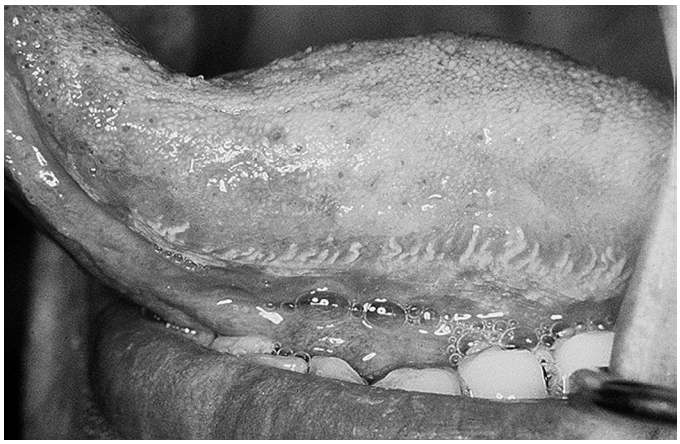

**Figure 73.1** Hairy leukoplakia associated with HIV.

Carcinomas present anywhere in the oral cavity, commonly on the posterolateral margin of the tongue and floor of the mouth – the 'coffin' or 'graveyard' area – and in the buccal mucosa in betel users. It is crucial, therefore, not only to examine visually and manually the whole oral cavity, but also to take particular care to inspect and palpate the posterolateral margins of the tongue and the floor of the mouth (Figure 73.1). There is usually solitary chronic:

- Ulceration
- Red lesion
- White lesion
- Indurated lump
- Fissure
- Cervical lymph node enlargement.

Anterior cervical lymph node enlargement may be detectable by palpation. Of presenting patients, 30% present with palpably enlarged nodes containing metastases and, of those who do not, a further 25% will go on to develop nodal metastases within 2 years.

Lip carcinoma presents with thickening, crusting or ulceration, usually of the lower lip. Potentially malignant lesions or conditions may include actinic cheilitis, erythroplasias, dysplastic leukoplakias (about 50% of oral carcinomas have associated leukoplakia), lichen planus, oral submucous fibrosis and chronic immunosuppression.

Too many patients with oral SCC present or are detected late, with advanced disease and lymph node metastases. With early detection and treatment, the cosmetic and functional results and survival are better. There should be a high index of suspicion, especially of a solitary lesion present for over 3 weeks, particularly if it is indurated, there is cervical lymphadenopathy and the patient is in a high-risk group.

It is essential to confirm the diagnosis, and determine whether cervical lymph nodes are involved or there are other primary tumours, or metastases (Figure 73.2). Therefore, almost invariably indicated are:

- Lesional biopsy: an incisional biopsy is usually indicated but an oral brush biopsy is now available mainly for cases where there are widespread potentially malignant lesions, and for revealing malignancy in lesions of more benign appearance

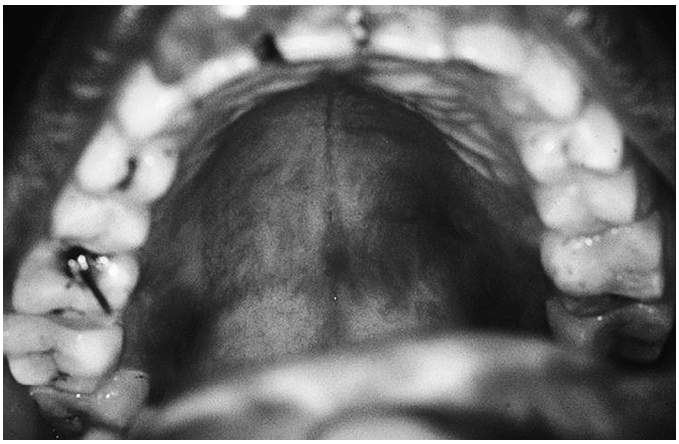

**Figure 73.2** Kaposi's sarcoma associated with HIV.

- Jaw radiography
- Chest radiography and endoscopy of the upper aerodigestive tract to exclude second primary tumours
- Fine needle aspiration biopsy of palpable cervical lymph nodes; full blood count and liver function tests, in some indications.

Oral cancer is still treated largely by surgery and/or irradiation, though chemotherapy is an emerging modality, especially using inhibitors of epidermal growth factor receptor.[21] There have been few unequivocal controlled trials of the conventional treatment modalities. Multidisciplinary clinics, with surgeons, oncologists and support staff, usually have an agreed treatment policy and offer the best outcomes. However, mortality rates for oral cancer have substantially increased in many countries. Although the efficacy of screening for oral cancer to increase survival and reduce mortality remains unproven, it is believed that Cuba's ongoing oral cancer screening programme has resulted in a higher proportion of cancers being localized at diagnosis and a comparatively high survival rate.[22] The WHO has published guidelines on prevention,[23] and studies have shown reduction or reversal in pre-cancerous lesions by primary prevention or abstinence from tobacco. There is a considerable body of evidence indicating a protective effect on oral cancer and pre-cancer, of diets rich in fresh fruits and vegetables and of vitamin A in particular. The prognosis is very site-dependent. For intra-oral carcinoma, 5-year survival may be as low as 30% for posterior lesions presenting late, as they often do, while for lip carcinoma, there is often more than a 70% 5-year survival.

## Erythroplasia (Erythroplakia)

Erythroplasia is a rare, isolated, red, velvety lesion which affects patients mainly in the 6th and 7th decades. Erythroplasia usually involves the floor of the mouth, the ventrum of the tongue or the soft palate. This is one of the most important oral lesions because 75–90% of lesions prove to be carcinoma or carcinoma in situ, or are severely dysplastic. The incidence of malignant change is 17 times higher in erythroplasia than in leukoplakia. Erythroplasia should be excised and sent for histological examination. Prevention is by avoidance of lifestyle habits of tobacco, betel and alcohol use.[24]

## Leukoplakia

All oral white lesions were formerly called leukoplakia and believed often to be potentially malignant. The term leukoplakia is now restricted to white lesions of unknown cause. Most white lesions are innocuous keratoses caused by cheek biting, friction or tobacco, but can be:

- Infections (e.g. candidosis, syphilis, and hairy leukoplakia)
- Dermatoses (usually lichen planus)
- Neoplastic disorders (e.g. leukoplakias and carcinomas).

Other conditions must be excluded, usually by biopsy.

Keratoses are most commonly uniformly white plaques (homogeneous leukoplakia), prevalent in the buccal (cheek) mucosae, and usually of low malignant potential. More serious are nodular and, especially, speckled leukoplakias, which consist of white patches or nodules in a red, often eroded, area of mucosa. The presence of severe epithelial dysplasia indicates a considerable risk of malignant development. The overall prevalence of malignant change is 3–33% over 10 years, but a percentage (about 15%) regress clinically.

It can be difficult to be certain of the precise diagnosis of a white patch, as even carcinoma can present as a white lesion. Incisional biopsy is indicated, sampling indurated, red, erosive or ulcerated areas rather than the more obvious whiter hyperkeratinized areas; staining with toluidine blue may help highlight the most appropriate area.

Management can be difficult, especially in extensive lesions of leukoplakia, and those with areas of erythroplasia. Obvious predisposing factors need to be reduced or eliminated. Prevention is by avoidance of lifestyle habits of tobacco and alcohol use.[25] Dysplastic lesions should certainly be excised and the patient should then be followed-up regularly at intervals of 3–6 months. Unfortunately, more than one-third recur.

## Oral Submucous Fibrosis

Oral submucous fibrosis (OSMF), though not regarded as a connective tissue disease, has pathological changes closely similar to those of scleroderma. Unlike the latter, which has severe effects on the skin but minimal effects on the oral mucosa, OSMF causes severe and often disabling fibrosis of the oral tissues alone.

OSMF affects virtually only those from the Indian subcontinent.[26] There is some evidence it is premalignant. The condition appears to be related to the chewing of areca nut and the 5A genotype of matrix metalloproteinase 3 (MMP3) promoter is associated with the risk of OSMF.[27] Iron-deficiency anaemia may be present but this is not uncommon in Asians in the absence of submucous fibrosis.[28]

Clinically, OSMF causes symmetrical fibrosis of such sites as the cheeks, soft palate or inner aspects of the lips. The fibrosis is often so severe that the affected area is almost white and so hard that it literally cannot be indented with the finger. Frequently the buccal fibrosis causes such severe restriction of opening that dental treatment becomes increasingly difficult and finally impossible. Ultimately, tube feeding may become necessary.

Intralesional corticosteroids and regular stretching of the oral soft tissues with an interdental screw or 'therabite' may delay fixation in the closed position. Medical therapies range from topical medication (e.g. with COX-2 inhibitors); to intralesionally injected medicaments such as corticosteroids,

collagenase, or hyaluronidase; to systemic medication with lycopene or pentoxifylline. Surgical therapies range from laser release to excision of the bands and split skin, radial forearm or other flap repair.

## Tobacco and Areca Nut Use

### SMOKELESS TOBACCO

Tobacco (both smoking and smokeless) had been a part of the Native American culture even before Columbus discovered America, after which this habit has spread worldwide. In the Middle Ages, tobacco was used as medication in ointments, mouth rinses and poultices. Some communities continue to believe in its perceived medicinal value.

Smokeless tobacco use is on the increase among young individuals, particularly, after the smoking ban, and the general misconception that it is less harmful than smoking tobacco. There is an ever-increasing market of smokeless tobacco products, which have diverse and varied patterns of use,[29] e.g. insufflation (of snuff) or mastication and sucking (of gutka). Smokeless products are currently used in many parts of the world, including America and Sweden, however, the most harmful products are available from southern Asia, particularly, the Indian subcontinent. The habit is most prevalent in the south Asian communities and, despite substance control via point of sale legislation, is culturally acceptable among all ages, with the habit commencing from as young as 5 years.[30,31]

Data on the prevalence of this habit in the UK are limited. The Health Survey of England 2004 observed that smokeless tobacco was most widespread among the Bangladeshi community, with a prevalence of 9% in men and 16% in women, followed by Indians (mainly Punjabi and Gujarati; 1–4% in men and 1% in women) and Pakistanis (1–2% in men and 1% in women).[32]

This habit is associated with an increase in cardiovascular risk factors including hypertension, increased heart rate, brain damage from stroke and cancers of the pancreas, stomach, bladder and lungs, decrease in sperm quality, increased risk of stillbirth, decreased gestational age at birth and decreased birth weight independent of gestational age in pregnancy.[33] Different smokeless products have a range of health risks, depending on the ingredients used, varying widely from minor reddish-orange discolouration/staining of teeth to more serious effects including oral birth defects like cleft palate and cancer of the oral cavity. These products contain variable quantities of proven carcinogens such as tobacco-specific nitrosamines, benzo(a) pyrene and toxic metals. Although some users associate it with oral cancers, knowledge of precancerous lesions, such as leukoplakia and oral submucous fibrosis, are relatively low.[34] The risk of pre-cancerous lesions and mouth cancers are known to increase when used as a 'night quid', i.e. placed in the cheek pouch overnight, combination with other habits such as alcohol and cigarette smoking has a synergic effect, further increasing these risks.[35]

Typical lesions, i.e. 'the Indian Oral Cancer' refer to cancer in the buccal mucosal, gingival and retromolar trigonal region, caused by placement of smokeless products in this area. As the name suggests, these patterns are particularly prevalent in southern Asia. Poor oral health is another trait often seen among users, characterized by increased periodontal pocket depth, gingival inflammation and bleeding.[36] While insoluble particulate matter in smokeless products leads to attrition and abrasion of dental hard tissues, there is also some indication of association between smokeless tobacco and dental caries, although, a cause and effect relationship cannot be conclusively established.

Recent smoking bans have highlighted an increased incidence of smokeless use, as an alternative to smoking. Although, interventions proven to be effective for smoking cessation, such as nicotine replacement and bupropion SR are ineffective among smokeless users, behavioural interventions have had a positive impact.[37,38]

### ARECA NUT

Areca nut, the fourth most common addiction globally following tobacco, alcohol and caffeine, is estimated to be used by 600 million people, particularly among South, East and South-east Asian communities. It is considered to be a 'fruit of divine origin' in Hindu religious ceremonies, as it is a vital ingredient of idol worship. In southern Asia, it is perceived to have medicinal values, including as an aphrodisiac, breath-freshener and with digestive properties. It is common practice to offer these products to guests in important social gatherings, weddings and other religious events.[39] This habit is widely accepted among all strata of society, including vulnerable populations such as women and children, making this habit a significant part of the cultural and ethnic identity.

Although there are regional variations, the habit is generally practiced by chewing or holding areca nut typically with slaked lime (calcium hydroxide), spices and other flavouring agents wrapped in a betel pepper leaf between the gum and cheek. The saliva produced can be either swallowed or spat out. While habitual users add tobacco to the quid in parts of the Indian subcontinent, this is not the norm in other countries such as Taiwan and Papua New Guinea.[40] In the last few decades, an estimated 20–40% of the Indian, Nepali and Pakistani populations were reported to be habitual users. While trends appear to be changing, with a decrease in the habit in Thailand, this habit is shockingly high in other countries, such as Palauans, where 70–80% of the population use areca nut. Areca nut is not only common throughout Asia but is becoming increasingly widespread in the Western society where these communities have settled. There has been a reported increase in the prevalence of areca nut among the minority immigrant population in the USA and UK.[41]

Areca nut with/without tobacco is carcinogenic to humans. It contains about 11–26% tannins and 0.15–0.67% alkaloids, known to be both cytotoxic and genotoxic. It is associated with the development of potentially malignant disorders with high potential for malignant transformation, including oral lichen planus, oral leukoplakia and oral submucous fibrosis. The risk of malignancy further increases with the addition of smokeless tobacco.[42]

Lime used in the quid has high concentrations of arsenic, a toxic metal also known to be carcinogenic.[43] Other additional ingredients used are known to cause lichenoid lesions, the premalignant potential of which is not fully understood. While areca nut consumption is associated with oral, pharyngeal and oesophagus cancers (due to p53 gene mutation), and to some extent liver cancers, it is also linked to metabolic syndrome, obesity, cardiovascular disease, diabetes, chronic kidney disease, liver cirrhosis and low-birth-weight infants.[44–48]

Areca nut users have poor oral hygiene, halitosis, poor periodontal health with an increase in gingival lesions, recession, periodontal pockets and bleeding of gums. Mild to severe attrition is another common finding among users, in severe cases it can be accompanied by periapical periodontitis and alveolar bone resorption. The severity of attrition is directly proportional to length and frequency of the habit. Although there appears to be little evidence on effective interventions, cessation advice along with behavioural support and counselling may be effective for abstinence from areca nut use.[49]

### NASWAR

Naswar (Nass, niswar, kap) is a form of smokeless tobacco containing powdered tobacco, slaked lime, indigo, cardamom and menthol. It is practised primarily among Central Asian, Afghani, Pakistani, Iranian and also in some Swedish and South African communities. Typically, it is used by sniffing through the nose, but is also used orally by placing a pinch under the tongue or in the buccal vestibule.

Naswar is chiefly manufactured in Pakistan and is available as green, grey and black variants. Like most smokeless products, it contains unionized nicotine (13.2 mg/g), carcinogenic tobacco-specific N-nitrosamines (TSNAs), a relatively high pH of 9.0, potential to induce cell mutations due to the presence of cyclic aromatic compound as well as toxic metals including cadmium, arsenic and lead which contributes to the adverse effects on both oral and general health.[50] While it is used as herbal medicines, especially in children, naswar is associated with peptic ulcers, potentially malignant disorders such as oral submucous fibrosis and cancers of the mouth and oesophagus.[51] Although users associate this habit to poor oral health (toothache, dental caries as well as bleeding gums), many fail to link its use to more serious life-threatening conditions such as oral cancers.

There are limited data on effective interventions and hence, culturally acceptable education materials, to increase the awareness of the adverse health effects associated with this habit, as well as behavioural interventions, to support the long-term abstinence, should be used for cessation interventions among these communities.

### HOOKAH

Hookah, a type of smoking tobacco, is increasing in prevalence among young individuals and is considered to be a global epidemic. Although the use of hookah is culturally associated with eastern Mediterranean and western Asia, it is increasingly becoming popular worldwide, including developed countries such as the UK and the USA. Depending on the region of the world, this habit is also known as shisha, waterpipe, nirghile, arghile, hubble-bubble and galyan. Each hookah smoking session last about 20–80 minutes and releases the same quantity of smoke as an individual smoking 100 cigarettes, exposing hookah users to an eightfold increase in carbon monoxide, increasing the adverse effects of this habit.

This habit is perceived to be a less harmful alternative to smoking; however, its use has both oral and general health implications. Users are at a significantly increased risk of lung cancer, heart disease, respiratory illness, periodontal disease and adverse effects during pregnancy such as low birth weight. Although there is some evidence of its association with hepatitis C and tuberculosis, particularly because it is a shared habit, this evidence is inconclusive. While there is a dearth in the current evidence-base for prevention and treatment of the long-term abstinence of this habit, evidence suggests that hookah users should be made aware of its addictive and harmful effects in order to influence behaviour change.[52]

## Coca Chewing

Despite legal restriction, coca, a plant native to South America, is a significant part of the Andean culture and religious identity, as it is believed to be a protector of health. The coca plant is popularly known as the plant responsible for the production of cocaine, globally. The habit of chewing coca leaves has been prevalent since before the Middle Ages and the plant is grown in Peru, Bolivia, Colombia, Argentina, Ecuador and Chile.[53] While these leaves have religious connotations and are used as offerings among indigenous communities in the Andean regions, coca leaves are also infused in water and imbibed as tea. Coca users adapt to cold, fatigue, hunger and are able to perform prolonged periods of strenuous physical activity, making the habit more prevalent among manual workers, including farmers and mine workers as well as at high altitudes.[53]

Traditionally, the coca leaves are used as a stimulant, as well as for various ailments including gastrointestinal problems, altitude sickness, depression, toothache (for its anaesthetic effects) and obesity. Some of the associated oral ill-effects of coca use are an increased risk of dental attrition, cervical root caries and periapical abscess.[54,55]

Although coca is one of the drugs on the UN's list of prohibited drugs, South American countries such as Bolivia are keen on protecting the indigenous cultural practices among the Andean communities. The use of coca by the indigenous people is comparable with caffeine consumption in Western culture and despite the increased dental health risks, and international disapproval, prohibition of coca is currently under dispute to protect the cultural identity and heritage of these communities.

## Dental Mutilation

Dental mutilation, including chipping and filing of teeth as well as intentional extraction of teeth, is a common traditional practice in many African countries, including Tanzania, Uganda, Sudan, Ethiopia and Kenya. Adult dental mutilations, primarily involving the maxillary incisors and canines, have been observed among many African tribes. Evidence from Cameroon suggests six patterns of adult dental mutilation – loss of incisors; inverted V-shaped central incisors; V-shaped central incisors; T-shaped anteriors; rectangular and hourglass-shape of at least one of the anteriors.[56] The tribe-specific adult dental mutilation is a cultural form of beautification and is associated with puberty ceremonies in both sexes, where the inverted V-shaped pattern is particularly observed. Clinical implications of these traditional practices are pulpal exposure, radicular cysts and extensive bone loss of the labial cortical plate.[56]

Infant dental mutilations differ from adult dental mutilations, as they are performed for their perceived medicinal benefits, however, they can have devastating health implications leading to death due to sepsis. Two patterns of infant dental mutilations have been observed – extraction of mandibular primary canines; and rarely extraction of permanent mandibular incisors. The strong cultural belief that during infancy, the

**TABLE 73.1 Odontogenic Infections**

| Condition | In Non-Allergic Individuals, Antimicrobial for >3 Days or until Symptoms Resolve | Comments |
|---|---|---|
| Acute necrotizing gingivitis | Metronidazole or amoxicillin | Only if systemic involvement |
| Bites | Co-amoxiclav | |
| Cellulitis | Benzyl penicillin plus flucloxacillin | |
| Periapical abscess | Amoxicillin or metronidazole for 5 days | Only if systemic involvement or cellulitis |
| Pericoronitis | Metronidazole or amoxicillin | Only if systemic involvement or trismus |
| Periodontal abscess | Amoxicillin or metronidazole for 5 days | Only if systemic involvement or cellulitis |
| Periodontitis | Metronidazole or doxycycline | Only for severe disease |
| Sinusitis | Amoxicillin, or doxycycline or erythromycin for 7 days | Only for symptoms >7 days |

swelling in the area associated with unerupted primary canines is the cause of persistent fever, vomiting, loss of appetite, diarrhoea and gastroenteritis, make this procedure acceptable. Children as young as 1 month undergo extractions of primary teeth by traditional healers, without topical anaesthetics. Tools used for these traditional customs are not sterilized and include knives, metal blades, bicycle spokes as well as sharp fingernails. A salt or herbal pack is sometimes placed over the operated area.

The traditional healers extract the primary teeth after which a hot knife is inserted into the socket to destroy the permanent tooth. The procedure is successful if the 'toothworm' (dental follicle of the permanent tooth) is removed along with the primary teeth. The consequences of infant oral mutilation are mainly due to the damage of the permanent teeth during this procedure; these range from enamel defects, loss of teeth, decreased mandibular arch size, hypoplasia of adjacent primary and permanent teeth, retention of primary teeth, malpositioned, displaced or impacted permanent successors, odontomas, to serious infection and death from sepsis.[57,58]

Although there have been efforts made to reduce the prevalence and associated adverse health consequences of infant dental mutilations, it is still practised in many African countries due to the strong cultural beliefs associated to it.[59]

## Infections

Infections are suspected and/or antimicrobial therapy is indicated in immunocompetent patients with fascial space infections in the neck, necrotizing fasciitis (surgical osteomyelitis, removal of affected tissue is mandatory) or other serious, or life-threatening infections. In ill or immunocompromised persons, antimicrobial therapy should be considered in acute sinusitis, acute ulcerative gingivitis, dental abscess, dry socket, pericoronitis and to cover oral surgery.

### ODONTOGENIC INFECTIONS

Odontogenic infections are mainly a consequence of pulpitis, leading initially to periapical infection and a dental abscess. Most odontogenic (and many orofacial) infections arise from the commensal oral mixed flora, with a substantial proportion of anaerobes. Most odontogenic and orofacial infections respond to drainage, either by endodontic treatment, incision, or tooth extraction. Analgesics also may be required. Antimicrobials may be indicated in a number of circumstances.

Most odontogenic infections respond well to penicillin or metronidazole, but increasing rates of resistance due to production of β-lactamase enzymes, which degrade penicillins, have lowered the usefulness of penicillins (Table 73.1). Co-amoxiclav and clindamycin, because of broad spectrum of activity and resistance to beta-lactamase, are increasingly first-line antimicrobials. Other bacterial infections are shown in Table 73.2.

#### *Acute Necrotizing Ulcerative Gingivitis*

Acute necrotizing ulcerative gingivitis (ANUG) is characterized by painful ulceration of the gum between the teeth (interdental papillae) (Figure 73.3), a pronounced tendency to gingival bleeding and halitosis. Anaerobic fusiform bacteria and spirochaetes are implicated, predisposing factors including poor oral hygiene, smoking, malnutrition and immune defects including HIV and other viral infections and leukaemias. ANUG not infrequently follows a respiratory tract infection presumably being predisposed by the transient immune defect consequent upon some such infections, particularly viral. ANUG is increasingly seen in viral infections such as HIV disease; in some other persons with ANUG only more subtle immune defects, such as reduced salivary immunoglobulin A and neutrophil dysfunction, have been described. However, there are patients who suffer from ANUG in the absence of any clear immune defect, malnutrition or other systemic factor and, in these, poor oral hygiene and tobacco-smoking may be factors. It is seen primarily in early childhood, young adults and HIV disease.[60]

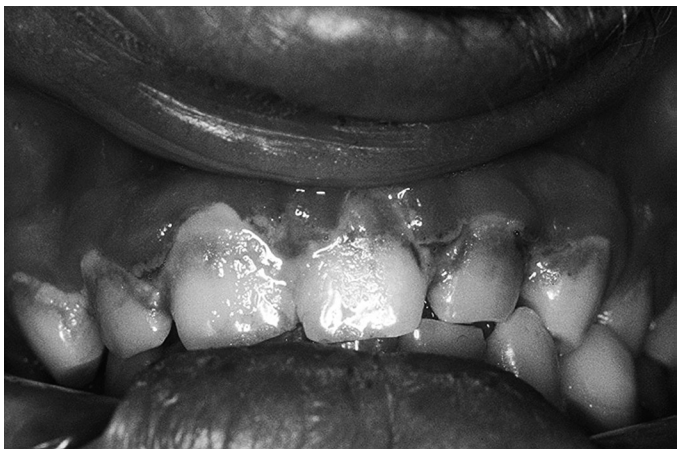

**Figure 73.3** Acute necrotizing ulcerative gingivitis.

**TABLE 73.2 Bacterial Infections which May Have Implications on Oral Health**

| Infecting Organism | Main Features | Orofacial Lesions |
|---|---|---|
| *Treponema pallidum* | Bejel | Mucous patches/sores in the mouth, destruction of palate in late stages |
| *Treponema pallidum carateum* | Pinta | Flattened, red, itchy patches on the face |
| *Treponema pallidum* | Yaws | Soft nodules on the face, which may ulcerate, destructive, disfiguring growths (gangosa), especially around the nose, mouth, and palate |
| *Klebsiella granulomatis* | Granuloma inguinale | Secondary to genital lesions, chronic, granulomatous ulceration in the oral cavity |
| *Chlamydia trachomatis* | Lymphogranuloma venereum | |
| *Bacillus anthracis* | Anthrax | Painful or ulcerated swellings mainly on palate |
| *Brucella, melitensis, suis* and *abortus* | Brucellosis | Rare infections or cranial nerve palsies |
| *Clostridium botulinum* | Botulism | Xerostomia, parotitis, muscle weakness |
| *Clostridium perfringens (Cl. welchii), Cl. sporogenes, Cl. oedematiens, Cl. septicum* | Gas gangrene | Gas gangrene |
| *Escherichia coli* | Enteric infections mainly<br>Also urinary tract, wound, and other infection | Found in some oral infections, especially in denture-wearers and immunocompromised |
| *Francisella tularensis* | Tularaemia | Pharyngitis<br>Stomatitis (often ulcerative)<br>Faucial membrane<br>Cervical lymphadenopathy |
| *Mycobacterium leprae* | Leprosy | In lepromatous leprosy, yellow-red, soft to hard, sessile lesions that ulcerate and heal to form fibrous scars, seen on gingiva, palate and tongue |
| *Mycoplasma hominis* and *pneumoniae* | Pneumonia | Rare infections or cranial nerve palsies?<br>Reiter's syndrome |
| *Neisseria meningitidis* | Meningitis<br>Septicaemia | Petechiae<br>Occasionally: herpes labialis<br>Facial palsy |
| *Nocardia asteroides, brasiliensis* and *caviae* | Nocardiosis | Ulceration<br>Cheek or gingivae |
| *Proteus vulgaris* | | Occasional infections |
| *Pseudomonas mallei* | Glanders (acute pneumonia) | Ulceration from nasal glanders<br>Ulcers |
| *Pseudomonas pseudomallei* | Melioidosis (lung or other localized infections or septicaemia) | Oral abscesses, or other infections<br>Parotitis |
| *Rickettsia ricketssiae* | Rocky mountain spotted fever | Faucial gangrene |
| *Rickettsia akari* | Rickettsialpox | Vesicles |
| *Salmonellae typhi, paratyphi, choleraesuis* and *enteritidis* | Typhoid and paratyphoid fever | Occasional infections |

ANUG is typically seen where plaque control is poor. A mixed flora dominated by fusobacteria and spirochaetes such as *Treponema* species, *Bacteroides* (*Porphyromonas*) *melaninogenicus* species intermedius, *Fusobacterium* species, *Selenomonas* species and *Borrelia vincentii* is invariably present and the condition improves dramatically when treated with penicillin or metronidazole, suggesting a significant role for these bacteria. Viruses may play a role,[61] possibly also by inducing immune suppression.

Management includes oral debridement and hygiene instruction, peroxide or perborate mouthwashes and metronidazole 200 mg three times a day for 3–5 days.

### *Gangrenous Stomatitis* (Cancrum Oris; *Noma*)

Noma is derived from the Greek *nomein*, which means to 'devour'; essentially it is a gangrenous stomatitis, which starts in the mouth as a benign oral lesion and rapidly destroys both the soft and hard tissues of the mouth and face (Figure 73.4). Most noma sufferers are under 6 years of age and it has been estimated that the case-fatality rate is probably between 70% and 90%. It is estimated that 100 000 African children under the

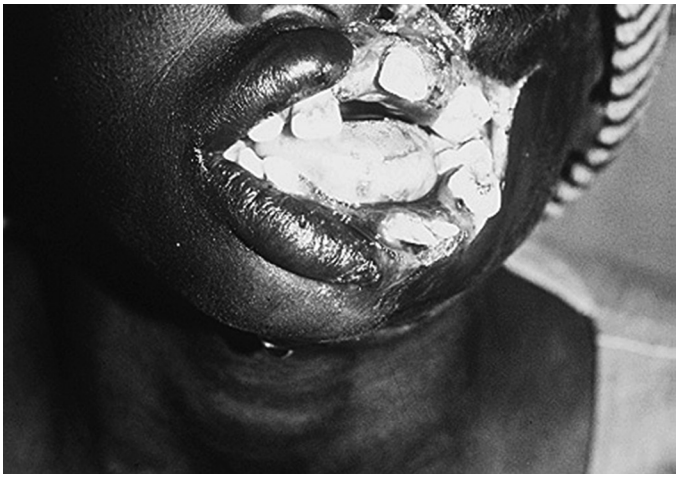

**Figure 73.4** Noma.

age of 6 years contract noma every year.[62] Factors which predispose to the development of gangrenous stomatitis include protein–energy malnutrition and deficiencies of vitamins A, B, C, iron or magnesium. Therefore, poor living environment, exposure to debilitating childhood diseases, poor oral hygiene and malnutrition all appear to put children at risk for noma. In the developed world, gangrenous stomatitis is rare, and typically seen in immunocompromised persons such as those with HIV infection, leukaemia and diabetes.[63] The condition is seen especially in sub-Saharan Africa. Nigeria probably has the highest incidence, although The Gambia, Algeria, Uganda, Senegal, Madagascar, South Africa, Sudan and Egypt are also areas of high prevalence, as are Afghanistan, India, the Philippines, China, Vietnam, Papua New Guinea and South America.

Anaerobes, particularly *Bacteroides* (*Porphyromonas*) species, *Fusobacterium necrophorum* (an animal pathogen), *Prevotella intermedia*, Actinomyces and alpha haemolytic streptococci, have been implicated. In cases following ANUG, *Streptococcus anginosus* and *Abiotrophia* spp. are predominant species. In early noma, predominant species include *Ochrobactrum anthropi*, *Stenotrophomonas maltophilia*, an uncharacterized species of *Dialister*, and an uncultivated phylotype of *Leptotrichia*. A range of species or phylotypes are found in advanced noma, including *Propionibacterium acnes*, *Staphylococcus* spp., *Stenotrophomonas maltophilia*, *Ochrobactrum anthropi*, *Achromobacter* spp., *Afipia* spp., *Brevundimonas diminuta*, *Capnocytophaga* spp., *Cardiobacterium* spp., *Eikenella corrodens*, *Fusobacterium* spp., *Gemella haemolysans*, and *Neisseria* spp. Phylotypes unique to noma infections include those in the genera *Eubacterium*, *Flavobacterium*, *Kocuria*, *Microbacterium*, and *Porphyromonas*, and the related *Streptococcus salivarius* and genera *Sphingomonas* and *Treponema*. Spreading necrosis penetrates the buccal mucosa, leading to gangrene and an orocutaneous fistula and scarring.

The presenting feature may be a painful red or purplish-red spot (an indurated papule), usually on the gingiva in the premolar–molar region, which enlarges and ulcerates rapidly and spreading to the labiogingival or mucobuccal fold, and exposing the underlying bone. There is pain and often fetor. A blue-black area of discoloration appears on the skin and leads to a perforating wound. Sequestration of the exposed bone and loss of teeth are rapid and then the wound heals slowly by secondary intention, often leaving a defect. In former times, noma was often a lethal condition.

Gangrenous stomatitis does not respond readily to treatment unless the underlying disease is controlled, especially nutritional rehabilitation. The wound should be cleaned regularly with chlorhexidine and/or saline and/or hydrogen peroxide. A soft cotton gauze or tulle gras dressing may be used but changed frequently. Any loose slough, loose teeth and bony fragments should be removed. Parenteral fluids should be given to correct any dehydration and electrolyte imbalance. Management includes improving nutrition, systemic antibiotics (clindamycin, penicillin, tetracyclines or metronidazole) and plastic surgery. Folic acid, iron, ascorbic acid and vitamin B complex may be required.

### *Syphilis (Venereal Treponematosis)*

In 1995, it was estimated that there were approximately 12 million new cases of syphilis among adults worldwide, with the greatest number of cases occurring in South and South-east Asia, followed by sub-Saharan Africa.

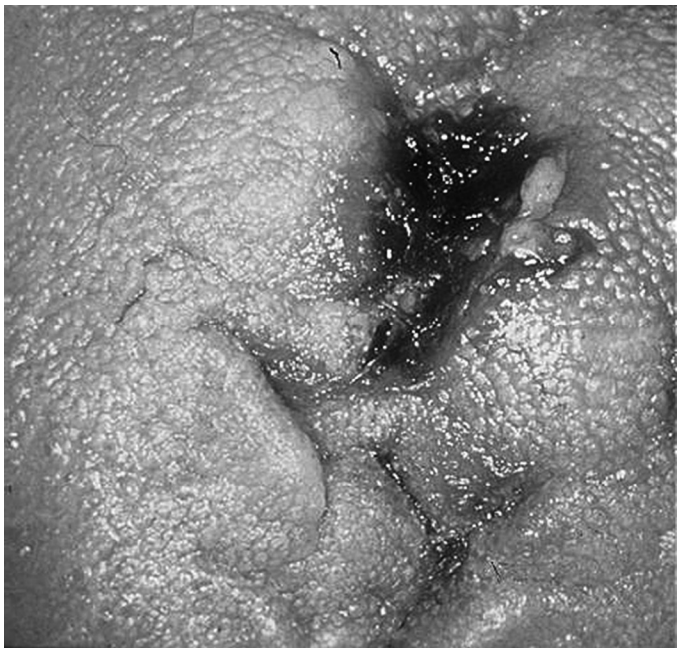

**Figure 73.5** Oral lesion associated with syphilis.

The lip is the most common extragenital site of primary infection with *Treponema pallidum*. It causes a chancre (primary, hard or Hunterian chancre) which begins as a small, firm, pink macule, changes to a papule and then ulcerates to form a painless round ulcer with a raised margin and indurated base (Figure 73.5). About 60% of oral cases affect the lip or may present at the angles of the mouth.[64] Other oral sites affected may include the tongue and to a lesser extent the gingivae and fauces. Lymph nodes in the submaxillary, submental and cervical regions are usually enlarged. Chancres heal spontaneously within 3–8 weeks. Secondary syphilis follows the primary stage after 6–8 weeks but a healing chancre may still be present. As in the primary stage, the mucosal lesions are highly infectious. The typical signs and symptoms are fever, headache, malaise, a rash (characteristically symmetrically distributed coppery maculopapules or lesions on the palms) and generalized painless lymph node enlargement. It is this stage that classically causes oral lesions. Painless oral ulcers (mucous patches and snail-track ulcers) are the typical lesions and are slightly raised, greyish white, glistening patches seen on the fauces, soft palate, tongue, buccal mucosa and, rarely, gingivae. Cervical nodes are enlarged and 'rubbery' in consistency. Latent syphilis follows secondary syphilis and persists until late syphilis (tertiary syphilis) develops. The characteristic lesion of tertiary syphilis is a localized midline granuloma ('gumma') varying in size from millimetres to several centimetres, which breaks down to form a deep punched-out painless ulcer. The most common oral site for a gumma is the hard palate although the soft palate, lips or tongue are commonly involved. The gumma starts as a small, pale, raised area which ulcerates and rapidly progresses to a large zone of necrosis with denudation of bone and, in the case of a palatal gumma, may eventually perforate into the nasal cavity.[65]

The presence of clinical manifestations together with a history of contact may suggest the diagnosis but serodiagnostic tests, and sometimes dark-field microscopy is required for confirmation. There is no specific oral management except general palliative care if there is soreness of oral soft lesions, but the

general management is straightforward: procaine penicillin intramuscularly for 10 days (erythromycin for 14 days) should be given.

### *Gonorrhoea*

Oral, pharyngeal and tonsil involvement is being reported with increasing frequency particularly among homosexuals and heterosexuals practising oral sex. Infection of these sites is acquired primarily by fellatio and infrequently by cunnilingus.[66] The tonsils become red and swollen with a greyish exudate and there is cervical lymphadenitis. Lesions in other parts of the mouth are described as showing fiery erythema and sometimes oedematous, perhaps with painful superficial ulceration of the tongue, gingiva, buccal mucosa, hard or soft palate. The inflamed mucosa may also be covered with a yellowish or greyish exudate, which when detached may leave a bleeding surface.

A throat swab should be taken for Gram staining to show polymorphs containing Gram-negative diplococci. Confirmation is by culture and sugar fermentation to aid differentiation of species. Rapid identification of gonococci by fluorescent antibody techniques is possible.

Penicillin is the drug of choice, given as 2 g ampicillin plus 1 g probenecid as a single oral dose. Patients hypersensitive to penicillin can be treated with co-trimoxazole. Many strains are resistant to penicillin in parts of Africa and the Far East. Tetracycline or cefazolin–probenecid and streptomycin or spectinomycin may be used.

### *Actinomycosis*

A breach in the continuity of mucosa caused either by trauma or surgery is the prerequisite for the majority of actinomycotic infections. Cervicofacial actinomycosis occurs predominantly in adult males following trauma either accidentally or rarely from dental treatment such as exodontia or endodontics.[67] Rarely, a periodontal pocket with suitable anaerobic conditions predisposes to the disease. The perimandibular area appears to be the commonest site. A relatively painless reddish-purple indurated mass appears at the angle of the jaw or in the vicinity of the parotid gland. It may drain through sinuses, the material containing the so-called sulphur granules. Actinomycosis may rarely involve the oral cavity, tongue, mandible, maxilla, paranasal sinuses, eye, ear, face, neck or salivary glands.

Sulphur granules may be seen by direct vision or after staining with Gram stain. Actinomycosis should be confirmed by the isolation of *A. israelii* in anaerobic culture. Penicillin is the first-choice antimicrobial. Alternatives include cephalosporin, clindamycin and lincomycin.

### *Tuberculosis*

It is estimated that over 1.5 million tuberculosis cases per year occur in sub-Saharan Africa. HIV and tuberculosis speed each other's progress, with the latter contributing about 15% of AIDS death worldwide.

Oral lesions are seen mainly in pulmonary tuberculosis although systemic symptoms suggestive of lung disease are by no means always present.[68] Apart from pain, typically the main symptom of tuberculosis is chronic ulcers or granular masses. These are usually on the dorsum/base of the tongue, gingivae or occasionally in the buccal mucosa, floor of the mouth, lips and the hard and soft palates.

Primary oral lesions develop when bacilli are directly inoculated into the oral tissues of a person who has not acquired immunity. Primary tuberculosis of the mouth is more common in children and adolescents than adults. It usually presents as a single painless indolent ulcer commonly on the gingiva with enlarged cervical lymph nodes, or the gingivae, tooth extraction sockets and the buccal folds. Occasional cases of primary jaw tuberculosis have been reported, usually resulting from extension of a gingival lesion, from an infected post-extraction socket, from an extension from a tuberculous granuloma at the apex of the tooth or haematogenous spread. Tuberculous osteomyelitis may involve the maxilla particularly, or the mandible. The same general pattern as seen in other affected bones is common, with a slow rarefying osteitis resulting in sequestration of bone. Pain is not a prominent early feature but is seen later. Secondary infection may lead to difficulty in making a diagnosis. Tuberculous involvement of the mandible causes symptoms of pain, swelling, difficulty in eating, trismus, paraesthesia of the lower lip and enlargement of the regional lymph nodes. The infection may spread throughout the jaw, producing multiple sinuses, which drain intra- or extra-orally. The posterior mandible and ascending ramus are typically affected, and radiographical appearances include irregular linear calcifications along the lower border and irregular radiolucencies within the jawbone. In the maxilla the infra-orbital region, particularly in the young, is the usual site affected. Typically, a cold abscess develops and may eventually drain through fistulae but occasionally a firm intra-bony lesion may be present. TB in AIDS may affect the salivary glands.[69–71]

The diagnosis of pulmonary tuberculosis, suggested by a chronic cough, haemoptysis, loss of weight, night sweats and fever, is confirmed by physical examination, chest radiography, sputum smears and culture, and tuberculin testing (Mantoux or Heaf test). A lesional biopsy should be examined histologically, and with acid-fast stains and culture of the organism is the absolute proof of the disease.

Conventional chemotherapy of tuberculosis consists of administering two or more active drugs for 18 months to 2 years. Isoniazid in combination with ethambutol, thiacetazone or para-aminosalicylic acid and, depending on the severity of the disease, streptomycin intramuscularly for a period of the first 2–3 months, may be necessary. Other available drugs include rifampin, pyrazinamide and ethionamide. In tropical countries, directly observed treatment strategies may be adopted which use multiple drugs for shorter durations.

## NON-TUBERCULOUS MYCOBACTERIAL INFECTIONS

Non-tuberculous (atypical) mycobacteria (NTM) include *Mycobacterium avium* and *M. intracellulare* (*M. avium-intracellulare* complex: MAC), *M. scrofulaceum* and *M. haemophilum*. Infections with NTM are being increasingly reported, especially in immunocompromised individuals. Cervical lymphadenopathy is occasionally caused by NTM but oral lesions are rare.

Atypical mycobacteria may be resistant to conventional antituberculous chemotherapy, although in children with cervical lymphadenitis caused by NTM conventional drug therapy alone or cycloserine for very resistant cases may be effective, and only occasionally is surgical excision necessary.[72,73]

## VIRAL INFECTIONS

Viral infections affecting the oral region are briefly reviewed in Table 73.3.

**TABLE 73.3 Viral Infections with Orofacial Manifestations**

| Virus | Orofacial Lesions |
|---|---|
| Cytomegalovirus | Oral ulcers with necrotic borders |
| Epstein–Barr virus | Oral hairy leukoplakia |
| Hepatitis B | Lichenoid lesions |
| Hepatitis C | Lichen planus |
| Herpes simplex | Primary infections, gingivitis, oral ulcers |
| | Recurrent oral |
| | Recurrent peri-oral, herpes labialis |
| HIV | Opportunistic fungal and viral infections, bacterial periodontitis, neoplasms (Kaposi's sarcoma, lymphoma), salivary gland disease and xerostomia |
| Influenza | Vesicles, erosive lesions, particularly in children |
| Papillomaviruses | Leukoplakia, oral cancers |
| Severe acute respiratory syndrome | Lesions not common, site of early replication |
| Varicella zoster | Chickenpox |
| | Zoster |
| | Zoster in immunocompromised |

## FUNGAL INFECTIONS

### *Superficial Mycoses*

**Candidosis.** Candidosis (candidiasis) is the most common oral superficial mycosis. Caused mainly by *Candida albicans*, the condition typically reflects an underlying change in oral flora, depressed salivation, or immune defect. Increasingly, infections with variants of *C. albicans*, with other and sometimes new *Candida* species and of organisms resistant to antifungal agents, are now seen especially in immunocompromised persons.[74]

Pseudomembranous candidosis or thrush may be seen in neonates and among terminally ill patients, particularly in association with immunocompromising conditions (Figure 73.6).[75] Thrush is characterized by white patches on the surface of the oral mucosa, tongue, gingivae and elsewhere. The lesions form confluent plaques that resemble milk curds and can be wiped off the mucosa with gauze. Oral candidosis in the form of thrush is classically an acute infection, but it may recur for many months or even years in patients using corticosteroids topically or by aerosol, in HIV-infected individuals and in other immunocompromised patients. The term chronic pseudomembranous candidosis has been used for chronic recurrence. Erythematous or atrophic candidosis is an uncommon and poorly understood condition. It may arise as a consequence of persistent acute pseudomembranous candidosis, when the pseudomembranes are shed, or in HIV infection may precede pseudomembranous candidosis. Erythematous areas are seen mainly on the dorsum of the tongue, palate, gingivae or buccal mucosa. Lesions on the dorsum of the tongue present as depapillated areas. Midline or median rhomboid glossitis, or glossal central papillary atrophy, is characterized by an area of papillary atrophy that is rhomboid in shape, symmetrically placed centrally at the midline of the tongue, anterior to the circumvallate papillae. Red areas are often seen in the palate in HIV disease. Hyperplastic candidosis (*Candida* leukoplakia) is typified by chronic, discrete raised lesions that are typically found at the commissures, rarely on the gingivae. Angular stomatitis (perlèche, angular cheilitis) is a clinical diagnosis of lesions that affect, and are restricted to, the angles of the mouth, characterized by soreness, erythema and fissuring, and is commonly associated with denture-induced stomatitis. Both yeasts and bacteria are involved, as interacting, predisposing factors. It is occasionally an isolated initial sign of anaemia or vitamin deficiency, and resolves when the underlying disease has been treated. Angular stomatitis may also be seen in HIV disease and Crohn's disease.

Chronic multifocal oral candidosis is a term given when there are several lesions in the absence of predisposing drugs (except tobacco smoking) or medical conditions, typically angular stomatitis that is unilateral or bilateral, retrocommissural leukoplakia, which is the most constant component of the tetrad, median rhomboid glossitis, and palatal lesions where the lesions are of more than 1 month duration.

Clinical diagnosis can be supported by culture from saliva or an oral rinse. Antifungal therapy is initially with topical agents, especially the polyenes (nystatin, amphotericin), except in immunocompromised persons in whom the azoles, especially fluconazole, may be required systemically.[76]

**Systemic (Deep) Mycoses.** The systemic mycoses are potentially serious, sometimes lethal fungal infections seen mainly in the developing world, or in those who have visited endemic areas. Cases have been recorded as long as 34 years after visits to endemic areas.[77] Infections are increasingly seen in immunocompromised persons, especially in HIV infection.[78–80] In otherwise healthy persons, infection with these fungi is typically subclinical although some have pulmonary infection. The increase in mycoses in immunocompromised persons is accompanied by significant morbidity and mortality and 'new' opportunists are appearing.

Orofacial lesions are mainly chronic ulcers or maxillary sinus infection, which are typically associated with respiratory lesions. Most of the mycoses may mimic carcinoma or tuberculosis, and are diagnosed on the basis of a history of travel to endemic areas, or an immunocompromising state, confirmed by taking a smear, biopsy or culture of the affected tissues. Serodiagnosis, physical examination and chest radiographs may be indicated. Most systemic mycoses can be treated with systemic amphotericin or azoles.

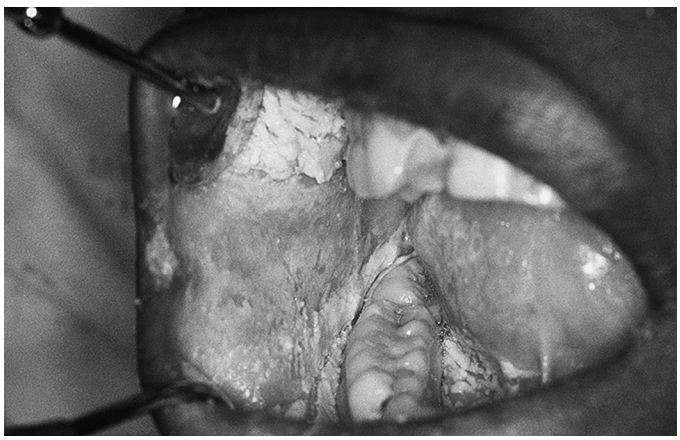

**Figure 73.6** Thrush associated with HIV.

## PARASITIC INFECTIONS

Malaria is the most important parasitic disease of man, and like many parasitic infestations has few oral complications. However, the lack of reporting of oral lesions in parasitic infestations may simply be a reflection of their under-diagnosis.

### *Myiasis*

Myiasis is caused when fly maggots invade living tissue or when they are harboured in the intestine or bladder. In oral lesions they are seen mainly in the anterior maxillary or mandibular gingivae.[81,82] An opening burrow is usually patent, with induration of the marginal tissues and is raised, forming a dome-shaped 'warble', or an extraction wound may be effected. Often several larvae are present and there is a severe inflammatory reaction in the surrounding tissues.

Larvae can be seen with the naked eye. A few drops of turpentine oil or chloroform in light vegetable oil should be instilled in the lesion and the larvae removed with blunt tweezers. It may be prudent to give an antibiotic, as there is often a superimposed secondary infection. Ivermectin may be effective in some cases.[83]

## Ciguatera Poisoning

Ciguatera, the most common form of fish poisoning, occurring in most tropical and subtropical seas, may result in oral or perioral paraesthesiae or dysaesthesiae.[84,85]

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