WINSPERT

ORAL MEDICINE PART 2

H.O.T

HIGH-PRIORITY ORGANISED THEORY

NOTES

By Dr. Jigyasa Sharma

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Thank you for your understanding and continued dedication.

Best regards,
WINSPERT TEAM

Table of Contents

ORAL MEDICINE PART 2 H.O.T TOPICS

OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

Osteoporosis is defined as a disease characterised by low bone mass and microarchitectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk.

- It is diagnosed by bone density tests that measure the density at the hip and spine. The result is called a 'T-score,' and will be in the range of normal, osteopenia, or osteoporosis.
- Osteoporotic fractures usually result from a combination of decreased bone strength and injurious falls.
- Osteoporosis affects both women and men, however women are at a greater risk of developing osteoporosis than men, mainly due to the rapid decline in oestrogen levels after menopause. When oestrogen levels decrease, the bones lose calcium (and other minerals) at a much faster rate.

Osteoporosis may be:

- primary osteoporosis, which includes postmenopausal and age -related osteoporosis; or
- secondary osteoporosis, caused by identifiable agents, such as corticosteroids, or disease, such as rheumatoid arthritis.

TABLE ONE: **Some risk factors for osteoporosis**

• Back pain	• Low body weight
• Corticosteroid treatment	• Low calcium intake
• Early menopause (women)	• Low vitamin D levels
• Family history of osteoporosis	• Physical inactivity
• Height loss	• Recurrent falls
• High alcohol intake	• Secondary causes eg. rheumatoid arthritis, chronic kidney disease
• Hypogonadism	
• Hyperthyroidism	• Smoking

For diagnosis, patients need a referral for dual-energy X-ray absorptiometry (DEXA) bone densitometry of the hip and spine to determine bone mineral density (BMD). Additional tests can include:

- plain X-rays—to check for spinal fractures;
- calciumand PTH—to help determine if the parathyroid gland is functioning normally;
- vitamin D (using the 25-hydroxyvitamin D radioimmunoassay) to help determine if there is a deficiency;
- full blood examination; urea, electrolytes and renal function; liver function tests; thyroid function tests; inflammatory markers; serum testosterone.

OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

Key points from the osteoporosis Australia statement on calcium supplements

- In order to reduce or prevent bone loss and fracture, it is vital to have adequate levels of calcium in the body, and calcium intake needs to be high enough to maintain these body levels, particularly in bone.
- Osteoporosis Australia recommends a total daily intake of 1000–1300mgof calciumper day (recommended dietary intake [RDI]), depending on age and gender.Ideally, the RDI should be achieved by consuming a diet rich in calcium; selecting foods that are naturally high in calcium, and including foods that have had calcium added to them—so called ‘calcium enriched’ foods.
- When the RDI cannot be achieved through diet alone, supplements may be required. In these circumstances, Osteoporosis Australia recommends a daily supplement of 500–600mg of elemental calcium. Clinical trials have shown that calcium supplementation, especially when it is combined with vitamin D, reduces the rate of bone loss and fracture in people who are likely to be deficient in dietary calcium, and optimises the effectiveness of osteoporosis medicines including bisphosphonates, denosumab, raloxifene, strontium ranelate, and teriparatide.
- Most people taking calcium supplements do not suffer any serious side-effects, but recent re-analysis of some previous clinical trial results has suggested a possible increase in the rate of heart attacks. Other research does not support these findings.
- A large European study designed to better understand the risks associated with calcium intake has been widely discussed in recent media. This study also appears to show an increased risk of heart attack in people take calcium supplements. However, the researchers did not observe any increase in the risk of heart attack in people who derived their calcium intake through diet alone.
- Further studies have shown that taking a combined vitamin D and calcium supplement, as recommended by Osteoporosis Australia, may be beneficial for general health as well as for reducing fracture risk in people who may not be getting enough calcium through their diet.

- combined calcium and vitamin D supplements remain both safe and effective for the majority of people who require them.

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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

Treatment Options

1. Calcium and vitamin D

Australian Guidelines recommend a daily intake of 1200–1300mg of elemental calcium per day. It is recommended that calcium supplements be taken with at least 800iu of vitamin D (colecalciferol) per day to optimise efficacy and reduce risk.

Recent meta-analyses suggesting an increased risk of cardiovascular events with the use of calcium supplements continue to be the subject of international debate. As a result of uncertainty there are concerns that many people may stop preventative treatment. For most Australians, dairy foods are the main source of calcium. Dairy foods are the most convenient way to obtain adequate calcium because milk, yoghurt and most cheeses are particularly high in calcium. Small amounts of calcium are found in nuts, breads, cereals, fruits, vegetables and fish (salmon and sardines are high). Three servings of dairy products each day will generally provide the recommended daily calcium intake around 1000mg/ day.

2. Vitamin D

The most clearly established effects of vitamin D are to maintain calcium and phosphate homeostasis, and to optimise bone health and muscle function. Vitamin D supplementation can prevent falls, particularly in the vitamin D deficient older person. Adequate vitamin D levels and dietary calcium intake are needed for effective primary fracture prevention, and also for secondary fracture prevention. Vitamin D deficiency should be corrected as well as adequate calcium intake ensured in conjunction with specific anti-osteoporosis treatment. Vitamin D is frequently used as a generic term to describe a number of specific molecules. Vitamin D3 (colecalciferol) is formed through the action of ultraviolet light on precursors in the skin. Vitamin D2 (ergocalciferol) is produced from by ultraviolet irradiation of the plant steroid, ergosterol. In the liver, vitaminD3 andD2 are metabolised to 25-hydroxyvitamin D [25-OHD]—the major circulating form of vitamin D. Both agents have a slow onset (4–8 weeks) and a prolonged duration of action (8–16 weeks). Ergocalciferol is now only available in OTC vitamin and mineral products. Colecalciferol is the usual ‘vitamin D’ supplement as it has been shown to be more effective.

The response to colecalciferol and ergocalciferol depends on the ability of the kidney to hydroxylate it to the physiologically active hormone calcitriol (1.25 dihydroxy vitamin D) so these agents should not be used in severe renal impairment.

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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

Treatment Options

2. Vitamin D

On the review of the available evidence, the position statement defines vitamin D status according to the following levels of serum 25-OHD:

- vitamin D adequacy: ⩾ 50nmol/L at the end of winter (level may need to be10–20nmol/L higher at the end of summer, to allow for seasonal decrease);
- mild vitamin D deficiency: 30–49nmol/L;
- moderate vitamin D deficiency: 12.5–29nmol/L;
- severe vitamin D deficiency: < 12.5nmol/L

Optimal values to prevent adverse musculoskeletal outcomes, including falls and fractures are not clear and levels for prevention of other diseases is not clear but may be higher, in the range of 75–80nmol/L (30–32ng/mL). There is currently limited high-level evidence to support these higher 25-OHD serum levels.
A wide range of diseases have been associated with low circulating levels of serum25-OHD including autoimmune disease, cardiovascular and metabolic disease, some cancers, microbial and respiratory diseases and some neurological and mental health conditions including schizophrenia as well as all-cause cardiovascular mortality.

The most clearly established effects of vitamin D are to maintain calcium and phosphate homeostasis, and to optimise bone health and muscle function.

Vitamin D toxicity:

The main concerns with excessive vitamin D levels are hypercalciuria and hypercalcaemia. Symptoms include nausea, vomiting, constipation, anorexia, apathy, headache, thirst, sweating and polyuria. Hypercalcaemia is not seen until serum25-OHDlevels reach 220nmol/L. There is no evidence of toxicity, based on blood calcium concentrations, at vitamin D doses up to 5000IU per day or 50,000IU per month.

Sources of vitamin D:

The main source of vitamin D is exposure of the skin to sunlight. The amount needed varies with the latitude, season, time of day, clothing and skin pigmentation. For moderately fair skinned people a walk with arms exposed for 6–7 minutes mid-morning or mid-afternoon in summer and7–40minutes in winter depending on latitude at noon in winter is likely to be helpful in maintaining adequate vitamin D levels in the body.

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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

Treatment Options

2. Vitamin D

Sources of vitamin D:

The production of vitamin D in the skin begins with the conversion of 7-dehydrocholesterol by sunlight in the UVB range of wavelengths (320–290nm). UVB light cannot pass through glass so that vitamin D production may be inadequate if persons rely on sunlight passing through a window.
Vitamin D3 is found naturally in small quantities in a few foods, such as wild-caught fatty fish (e.g. North Sea salmon, herring, mackerel). Liver, eggs and fortified foods such as margarine and some low-fat milk products also contain very small amounts of vitamin D3, however, for most people, dietary vitamin D intake is limited.

Prevention with lifestyle changes

Unfortunately, there are no studies reporting fracture or bone density outcomes for interventions for smoking, low body weight, high alcohol intake and sedentary lifestyle although these factors are associated with higher fracture risk.

Pharmacological management

The risk of vertebral fracture is about four times higher in women with a previous vertebral fracture than in those without. Despite this, less than 30%of Australian postmenopausal women with a previous fracture were taking an anti-osteoporotic drug and only 10% of Australian men who were eligible for a PBS-subsidised bisphosphonate were taking (or had taken) one.

Bisphosphonates are suitable for men and women; raloxifene, strontium or denosumab are other options for women. All anti-osteoporotic drugs reduce fracture risk but differ in their specific data and adverse event profiles.
Pharmacological management should be considered in the following patients:

- those with a minimal trauma fracture;
- those aged 70 years or older with a T-score of –3.0 or lower;
- those currently on prolonged (at least 3months) high-dose corticosteroid treatment (at least 7.5mg/day prednisolone or equivalent) and with a T-score of –1.5 or lower.

Choice of pharmacological treatment is influenced by patient gender, menopausal status, medical history, whether it is for primary or secondary fracture prevention, patient preference and eligibility for government subsidy.

OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

First-line medications

Bisphosphonates, including risedronate, alendronate and intravenous zoledronic acid, strontium ranelate and selective oestrogen receptor modulators (SERMs), e.g. raloxifene, have been shown to reduce fracture risk by 30–70% (calculated as relative risk reduction). However, they cannot reverse structural damage.

(Reference: TG)

Antiresorptive drugs available in Australia are:

- alendronate
- ibandronic acid
- pamidronate
- risedronate
- zoledronic acid
- denosumab

→ BISPHOSPHONATES

Antiresorptive drugs have been associated with an increased risk of medication-related osteonecrosis of the jaw; the risk may also be increased with other drugs that have a similar mechanism of action.

Medication-related osteonecrosis of the jaw

Medication-related osteonecrosis of the jaw (MRONJ) is an area of exposed bone in the jaw persisting for more than 8 weeks in a patient currently or previously treated with an antiresorptive or antiangiogenic drug, who has not received radiation therapy to the craniofacial region. Antiresorptive drugs include bisphosphonates and denosumab. Antiangiogenic drugs (e.g. bevacizumab, sunitinib) interfere with the formation of new blood vessels, and are used in the treatment of some malignancies.

Osteonecrosis of the jaw is an uncommon adverse effect of antiresorptive drugs. Severe cases have mainly occurred in patients who had dental surgery during treatment with a high-dose intravenous bisphosphonate for multiple myeloma or metastatic cancer. Current data in oncology populations suggests that the incidence of medication-related osteonecrosis of the jaw is up to 12 222 per 100 000 patient-years for bisphosphonates, and up to 2316 per 100 000 patient-years for denosumab.

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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

Medication-related osteonecrosis of the jaw

The incidence of medication-related osteonecrosis of the jaw in patients treated with antiresorptive drugs for osteoporosis is significantly lower than oncology populations. Based on limited data, the incidence of medication-related osteonecrosis of the jaw in patients taking antiresorptive drugs for osteoporosis may be up to 150 per 100 000 patient-years, which is only slightly higher than in the general population.

Although medication-related osteonecrosis of the jaw has significant consequences for the patient and can be difficult to treat, the **benefits of antiresorptive therapy outweigh the risk of harm in most patients.**

Risk assessment for medication-related osteonecrosis of the jaw

Bone-invasive dental procedures (e.g. tooth extractions, difficult surgical extractions, implant placement, periapical or radicular surgery, periodontal flap surgery) can trigger medication-related osteonecrosis of the jaw. However, medication-related osteonecrosis of the jaw can occur in patients who have not had a bone-invasive dental procedure, such as patients with poorly fitting dentures or exostoses (e.g. tori, mylohyoid ridges). Osteonecrosis that is not related to medication use can also occur at these sites spontaneously.

The risk of medication-related osteonecrosis of the jaw is higher for long durations and high doses of antiresorptive therapy, and may be higher when antiresorptive and antiangiogenic drugs are used concomitantly.

C-terminal telopeptide (CTX) is a breakdown product of bone resorption. The serum C-terminal telopeptide concentration has been proposed as a way of estimating a patient's risk of medication-related osteonecrosis of the jaw. However, there is insufficient evidence to support a threshold serum C-terminal telopeptide concentration beyond which medication-related osteonecrosis of the jaw will not occur. Furthermore, the serum C-terminal telopeptide concentration is affected by many variables, including patient age, gender, medical conditions (e.g. bone disease), menopausal status, drugs (e.g. oral contraceptives, hormone replacement therapy, antiresorptive drugs), and the assay method used. It is therefore difficult to use a patient's C-terminal telopeptide test result to assess the risk of medication-related osteonecrosis of the jaw.

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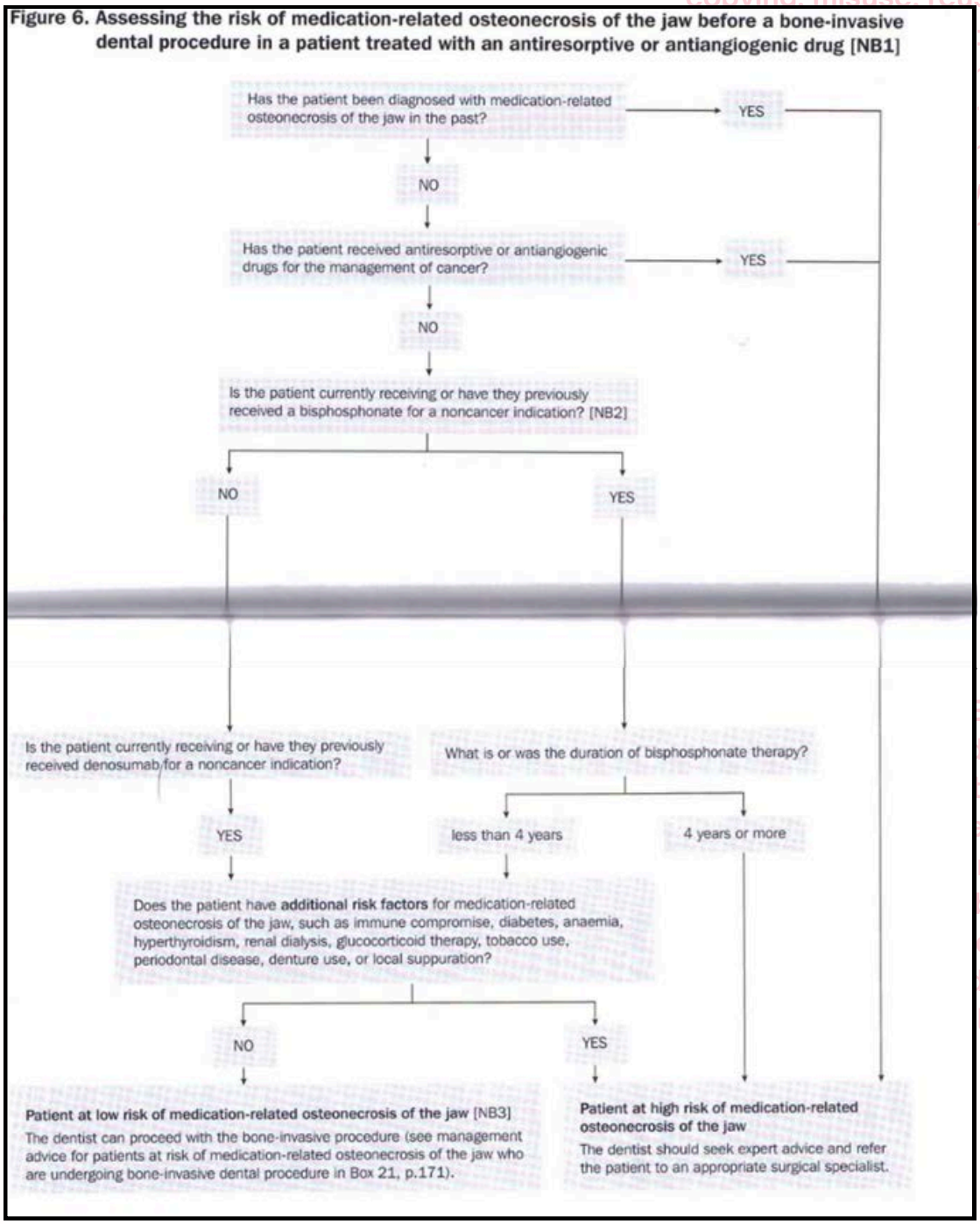
OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

Figure 6. Assessing the risk of medication-related osteonecrosis of the jaw before a bone-invasive dental procedure in a patient treated with an antiresorptive or antiangiogenic drug [NB1]



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Reference: Osteoporosis: prevention and management in older people

Prevention of medication-related osteonecrosis of the jaw

Maintaining optimal oral health

- Ensuring optimal oral health by implementing early dental assessment and initiation of appropriate dental care reduces the incidence of medication-related osteonecrosis of the jaw.
- If a medical practitioner refers a patient for dental assessment before or shortly after starting an antiresorptive drug, the dentist should undertake a comprehensive oral examination including pulp tests and radiographs.
- The dentist should ensure that the patient is dentally fit and unlikely to require extractions in the foreseeable future.
- The dentist should eliminate dental caries (e.g. extractions, restorations), establish a healthy periodontium (e.g. debridement, extractions), and encourage good oral hygiene.
- If possible, any necessary dental treatment should be completed before or shortly after starting antiresorptive therapy for osteoporosis (e.g. within 6 months)—the risk of medication-related osteonecrosis of the jaw in patients with osteoporosis remains low in the early stage of treatment.
- Patients with cancer who are prescribed high doses of antiresorptive drugs are at greater risk of medication-related osteonecrosis of the jaw, so ideally dental procedures should be completed before starting treatment.
- Regular dental review is essential to monitor oral health (e.g. clinical oral examinations, radiographs), particularly if the patient has a history of periodontal disease. Advise patients to seek early management of oral or dental symptoms and, if worn, ensure optimal fit for dentures.
- Effective communication between treating dentists and medical practitioners is essential. Inform and involve the patient in treatment decisions.

Antibiotic prophylaxis is not recommended to reduce the risk of medication-related osteonecrosis of the jaw.

OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

Reference: Osteoporosis: prevention and management in older people

Prevention of medication-related osteonecrosis of the jaw

Management advice for patients at risk of medication-related osteonecrosis of the jaw undergoing a bone-invasive dental procedure

- Inform the patient of the risk of medication-related osteonecrosis of the jaw and obtain consent for the procedure.
- See advice on drug holidays and scheduling of procedures.
- Do not use antibiotic prophylaxis to reduce the risk of medication-related osteonecrosis of the jaw—there is insufficient evidence to support this practice. However, an active infection should be treated.
- Ensure optimal oral hygiene before and after the procedure.
- Reduce the plaque load with mechanical debridement and pre- and post-procedural chlorhexidine mouthwash.
- Minimise trauma and periosteum stripping and close any mucosal flaps that are raised with sutures.
- Monitor the oral wound until it heals—healing may be slow.
- Do not debride nonhealing wounds.
- Refer to a specialist if bone is still visible at 8 weeks.

There is no evidence that drug holidays reduce the risk of medication-related osteonecrosis of the jaw.

Clinical judgment of treating doctors and dentists is required to determine the appropriate management of each patient.

For patients receiving antiresorptive drugs for osteoporosis, the benefits of continued therapy outweigh the low risk of medication-related osteonecrosis of the jaw. Although stopping bisphosphonates for a short period is unlikely to cause harm in a patient at low risk of fracture, there is no evidence that this approach reduces the risk of medication-related osteonecrosis of the jaw.

Denosumab is a reversible antiresorptive administered every 6 months for osteoporosis. If it is possible to delay a bone-invasive dental procedure in a patient taking denosumab for osteoporosis, ideally schedule the procedure just before the next dose of denosumab. It is never appropriate to interrupt or delay the dose of denosumab; withdrawal of denosumab has been associated with an increased risk of spontaneous vertebral fractures.

OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

(REFERENCE: Osteoporosis therapy: treatment duration and when to stop)

As the key objective of osteoporosis treatment is to protect patients from the occurrence of fragility fractures, pharmacists should focus on the long-term benefits in fracture prevention, not just improvement in bone mineral density.

There is strong and consistent evidence that medications can reduce the incidence of vertebral and other fragility fractures in older people with osteoporosis at moderate to high fracture risk. A range of medications are available with different mechanisms of action and routes of administration. The optimal duration of use of these medications remains to be clearly established, with evidence demonstrating continued gains in bone mineral density (BMD) over 10 years of bisphosphonate treatment. New evidence from the FREEDOM Extension trial shows denosumab is associated with sustained reduction in bone turnover markers (BTMs) and continued improvement in BMD over 8 years.

The key objective of osteoporosis treatment is to protect patients from the occurrence of fragility fractures, not to increase surrogate markers such as BMD and BTMs. So, when evaluating published literature, pharmacists should focus on the long-term benefits in fracture prevention, not just improvement in BMD.

This article discusses three important issues:

- **How long should treatment be continued?**
- **Are there long-term adverse effects with treatment?**
- **Is continued use necessary for sustained fracture prevention?**

TABLE 1: **MEDICATIONS FOR PREVENTION OF OSTEOPOROSIS**

MEDICATION CLASS	MEDICATIONS	ROUTE OF ADMINISTRATION	MECHANISM OF ACTION
Bisphosphonates	Alendronate Ibandronic acid Pamidronate Risedronate Tiludronate Zoledronic acid	Oral Oral or IV IV infusion Oral Oral IV infusion	Decrease bone resorption by inhibiting osteoclasts
RANKL inhibitor	Denosumab	SC	Decreased formation and activity of osteoclasts, thus reducing bone resorption
Selective oestrogen receptor modulator (SERM)	Raloxifene	Oral	Oestrogen agonistic effects on bone mass and lipid
Dual acting bone agent	Strontium	Oral	Increases bone formation and reduces bone resorption
Recombinant human PTH	Teriparatide	IV	Promotes bone formation and increases BMD

OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

(REFERENCE: Osteoporosis therapy: treatment duration and when to stop)

Bisphosphonates

The risk of ONJ increases with the duration of exposure to bisphosphonates, commencing within the first two years, and increasing four-fold beyond two years of treatment.

Conversely, the incidence of adverse events with denosumab does not appear to increase over time. Recent results from the FREEDOM and its Extension trials demonstrate the safety profile of denosumab remained consistent over eight years of treatment.

Unlike all the other drugs used to treat osteoporosis, the effects of bisphosphonates on bone remodelling persist after stopping therapy. Evidence suggests that with 3–5 years of treatment, protection against fracture persists for 1–2 years and then gradually wanes with discontinuation of therapy.

There is no clear consensus on the optimal bisphosphonate duration of treatment. It probably will vary in patients depending on their age, bone turnover, renal function, absolute fracture risk and other clinical factors.

The current body of evidence suggest patients should take bisphosphonates for 3–5 years. Studies confirm that therapy for one year or less is not long enough. After this period, whether to continue or temporarily discontinue bisphosphonates should be considered.

Denosumab

Denosumab is a potent anti-resorptive agent. It inhibits osteoclast-mediated bone resorption and significantly reduces the risk of vertebral, non-vertebral, and hip fractures. Denosumab appears to be about equally effective as bisphosphonates in clinical trials in reducing the risk of fragility fractures. In these trials, bisphosphonates and denosumab were taken with calcium and vitamin D supplements.

In contrast to bisphosphonates, denosumab is not bound to bone, so its anti-resorptive effects decline rapidly at the end of the six-month interval.

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OSTEOPOROSIS MANAGEMENT AND DENTAL RELEVANCE

(REFERENCE: Osteoporosis therapy: treatment duration and when to stop)

Drug holiday

US guidelines recommend a 'drug holiday' of 1–2 years after completion of 4–5 years of alendronate therapy in patients at moderate fracture risk.
Patients with higher BMDs and no fracture history should be considered candidates for a 'drug holiday', during which their bone health should be monitored at regular intervals.
Patients at mild risk of fractures might stop treatment after 5 years and remain off as long as bone mineral density is stable, and no fractures occur.
Higher risk patients should probably be treated for 10 years, have a holiday of no more than a year or two, and perhaps be on a non-bisphosphonate treatment during that time.

TABLE 4: STRATEGIES FOR BISPHOSPHONATE DISCONTINUATION BASED ON FRACTURE RISK

FRACTURE RISK	STRATEGY
Mild fracture risk	Stop treatment after five years and remain off as long as BMD stable and no fractures occur
Moderate fracture risk	Treat for 5–10 years, stop treatment for 2–3 years (or less if BMD decreases or fracture occurs)
High fracture risk	Stop treatment after 10 years, drug holiday for 1–2 years plus non-bisphosphonate treatment (raloxifene, teriparatide)

The optimal length of a 'drug holiday' has not been established but existing data suggest up to five years with alendronate, three years with zoledronate and one year with risedronate.

Conclusions

Long-term use of bisphosphonates may be associated with an increased risk of adverse effects, although the risk is low. A 'drug holiday' can be justified in patients at low fracture risk, after completion of 3–5 years of continuous bisphosphonate therapy.
Anti-fracture efficacy is sustained for at least 1–2 years. Denosumab, and other anti-osteoporosis medications such as oestrogens and raloxifene, do not have skeletal retention and therefore the benefits do not continue after cessation.

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

(REFERENCE: Oral fungal infections: an update for the general practitioner)

Oral candidosis is the most common fungal infection encountered in general dental practice. It manifests in a variety of clinical presentations which may mimic more sinister diseases and can occasionally be refractory to treatment requiring the attention of an oral medicine specialist. Management of oral candidosis should always include a thorough investigation of underlying predisposing conditions, as the disease often presents when the patient is systemically compromised.

Oral candidiasis and candida - associated lesions

REFERENCE: TG

Candida species are a commensal organism of the oral cavity. Oral candidiasis is an opportunistic infection that is uncommon in healthy individuals; however, it occurs relatively commonly in neonates.
If any 'red flag' features of oral mucosal disease are present, refer to an appropriate specialist.
Management of oral candidiasis in immunocompromised patients requires specialist advice. Patients with undiagnosed HIV infection may present initially with oral candidiasis.

Common risk factors for oral candidiasis

Local Factors	Systemic Factors
• dentures • salivary gland hypofunction • corticosteroid inhalers • poor oral hygiene • smoking	• immune compromise leg poorly controlled diabetes) • drugs (e.g. systemic corticosteroids, antibiotics)

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Pseudomembranous candidiasis

Clinical Features

- creamy white curd, papules and plaques that are sometimes removable
- red, raw and often bleeding base
- generally asymptomatic
- may affect the oropharynx
- if the dorsal tongue is affected, autoinoculation of the palate may occur

Management

- address predisposing factors
- use topical antifungal therapy for oral candidiasis
- if the infection affects the oropharynx, refer for specialist management

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Pseudomembranous candidiasis

(REFERENCE: Oral candidiasis and the therapeutic use of antifungal agents in dentistry)

Extensive white pseudomembranous consisting of desquamated epithelial cells, fibrin and fungal hyphae. These white patches occur on the surface of the labial and buccal mucosa, hard and soft palate, tongue, periodontal tissues and oropharynx (Fig 2). The membrane can usually be scraped off with a swab to expose an underlying erythematous mucosa. In infants, the infection is usually superficial and easy to manage, but in debilitated patients it may be widespread with angular cheilosis and oesophageal involvement.

This form of candidiasis is common amongst patients:

- **Who are immunocompromised, in particular: those taking anti-mitotic drugs and/or corticosteroids; those patients with HIV infection;**
- **extremes of age;**
- **those with uncontrolled diabetes mellitus;**
- **patients who have taken long-term broad-spectrum antibiotics and psychotropic drugs;**
- **patients who are terminally ill.**

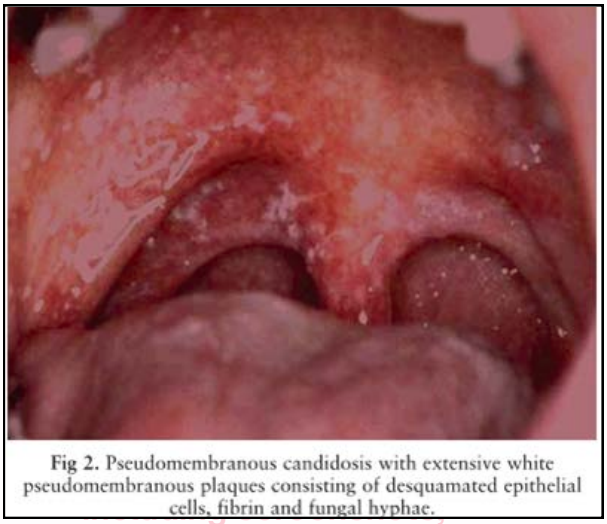

Fig 2. Pseudomembranous candidosis with extensive white pseudomembranous plaques consisting of desquamated epithelial cells, fibrin and fungal hyphae.

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Erythematous candidiasis

(REFERENCE: TG)

Clinical Features

- sensitive red lesions commonly affecting the palate and tongue
- the tongue may appear de-papillated and smooth

Management

- address predisposing factors
- use topical antifungal therapy for oral candidiasis

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Erythematous candidiasis

(REFERENCE: Oral candidiasis and the therapeutic use of antifungal agents in dentistry)

Two forms of this condition can be recognized:

- **symptomatic**
- **asymptomatic**

The symptomatic form may be associated with a burning sensation in the mouth or on the tongue. The tongue may be bright red, similar to that seen with a low serum B12, folate and iron. Diagnosis may be difficult but should be considered in the differential diagnosis of a sore tongue, especially in a frail older patient with dentures who has received antibiotic therapy or who is on inhaled steroids.

The asymptomatic form is much more common and is characterized by localized chronic erythema of tissues covered by dentures and is often referred to as denture-induced stomatitis (Fig 1). Lesions usually occur on the palate and upper jaw but may also affect mandibular tissue. It is quite common with incidence rates of up to 65 per cent reported. Trauma from an ill-fitting denture (usually a full maxillary, acrylic denture) is a major contributing factor, because this provides a suitable environment for candida proliferation.

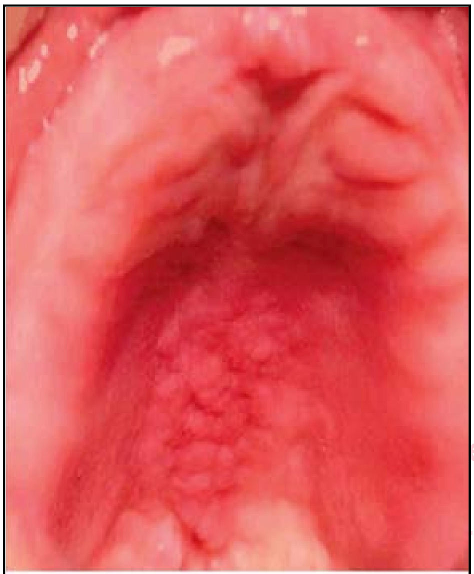

Fig 1. Erythematous candidosis, showing localized erythema of tissues covered by dentures, often referred to as denture-induced stomatitis.

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Hyperplastic candidiasis

(REFERENCE: TG)

Clinical Features

- asymptomatic, nonremovable white plaques that may appear nodular
- usually affects the retro-commissures, anterior buccal mucosa and lateral tongue
- may be bilateral
- may resemble oral leucoplakia or oral cancer

Management

- address predisposing factors
- may be associated with epithelial dysplasia—refer to a specialist for biopsy and management

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Hyperplastic candidiasis

(REFERENCE: Oral candidiasis and the therapeutic use of antifungal agents in dentistry)

Hyperplastic candidosis occurs in several different forms. The first of these is a form of oral candidosis occurring as a well demarcated white patch anywhere on the oral mucosa but frequently on the post- commissural cheek mucosa in patients over 35 years of age who smoke tobacco (Fig 4). These white patches cannot be wiped off. It is important to recognize this condition because hyperplastic candidosis has a higher incidence of dysplasia and malignant transformation than an otherwise similar white keratotic patch of the oral mucosa which is not infected with Candida.

Fig 4. Hyperplastic candidosis presenting as a well demarcated white patch on the retro-commissural cheek mucosa.

A further presentation of hyperplastic candidosis is as part of a more widespread mucocutaneous candidosis. There are a number of forms of mucocutaneous candidiasis, but all have skin as well as mucosal involvement and the oral component is first noticed early in life, usually when the patient is younger than 10 years. The oral lesions often precede skin and nail involvement.

Finally, a form of hyperplastic candidosis can be seen in the posterior hard palate, immediately posterior to full upper dentures in patients who incorrectly use steroid inhalers (Fig 5). These lesions are very difficult to treat without changing the use of the steroid inhaler.

Fig 5. Hyperplastic candidosis presenting immediately posterior to full upper dentures.

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Angular cheilitis (angular stomatitis)

(REFERENCE: TG)

Clinical Features

- painful erythema and fissuring of the corners of the mouth
- usually caused by a mixed infection of Candida, Staphylococcus aureus and Streptococcus species
- often associated with intraoral candidiasis

Predisposing Factotrs

- deep skin folds around the mouth (associated with worn down teeth, ill-fitting dentures or not wearing dentures)
- iron, folate or vitamin E3,2 deficiency
- Crohn disease
- granulomatous disease
- atopic and seborrhoeic dermatitis

Management

- dental review to assess dental or denture-related causes
- address predisposing factors
- use topical antifungal therapy for angular cheilitis
- treat oral candidiasis if present, with topical antifungal therapy for oral candidiasis

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Angular cheilitis (angular stomatitis)

(REFERENCE: Oral candidiasis and the therapeutic use of antifungal agents in dentistry)

An erythematous fissuring at one or both corners of the mouth, usually associated with an intra-oral candidal infection. Other organisms implicated are staphylococci and streptococci. Facial wrinkling at the corners of the mouth and along the nasolabial fold leads to a chronically moist environment that predisposes to this lesion. Other factors implicated are iron deficiency, anaemia and vitamin B12 deficiency.

(REFERENCE: Oral fungal infections: an update for the general practitioner)

It is often symptomatic and bilateral. This is seen commonly in denture wearing patients with reduced vertical occlusal dimension. While nutritional factors have an important aetiologic role in the development of these lesions.

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Denture-associated erythematous stomatitis (denture stomatitis)

(REFERENCE: TG)

Clinical Features

- sensitive erythematous lesions confined to denture bearing areas, particularly the palate
- may appear punctate, or smooth and red
- nodular hyperplasia may be observed

Predisposing Factotrs

- ill-fitting dentures
- suboptimal oral and denture hygiene
- dietary factors

Management

- advise patient to optimise denture hygiene and to remove dentures at night, clean them, then store them dry overnight
- dental review to assess fit of dentures
- if symptoms do not resolve after 1 month of optimal oral and denture hygiene, use topical antifungal therapy for oral candidiasis, applied inside the mouth and to the dentures

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Denture-associated erythematous stomatitis (denture stomatitis)

(REFERENCE: Oral fungal infections: an update for the general practitioner)

Denture-associated erythematous candidosis (Fig 4) is a common inflammatory oral mucosal lesion that occurs on mucosa in contact with the fitting surface of a denture. Classically, the lesion presents as erythema and oedema restricted to the denture supporting area. Lesions are frequently asymptomatic, how- ever, patients may complain of slight soreness or burning sensations. Though most lesions are associated with a Candidal infection, other aetiologic factors include poor oral and denture hygiene, nocturnal denture wearing, and ill-fitting dentures.

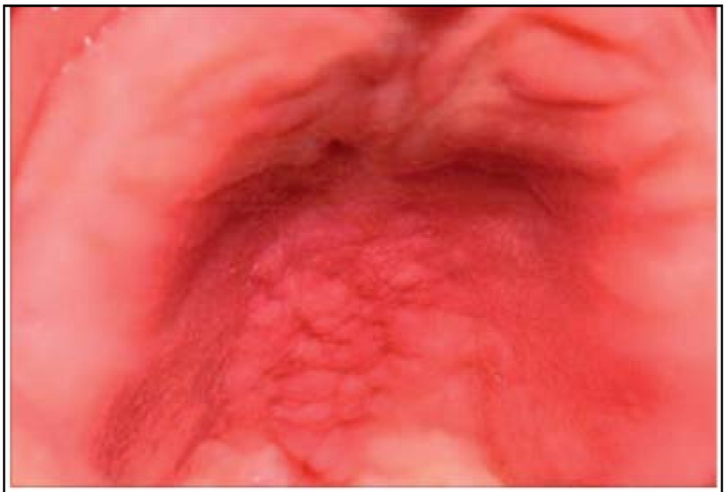

Fig 4. Denture-associated erythematous candidosis.

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Overview of oral candidiasis and Candida associated lesions

Median rhomboid glossitis

(REFERENCE: TG)

Clinical Features

- rhomboid area of depapillation and erythema in the midline of the dorsal tongue
- may be fissured or nodular
- autoinoculation of the palate may occur
- usually asymptomatic although it may sting or burn

Management

- address predisposing factors
- use topical antifungal therapy for oral candidiasis

(REFERENCE: Oral candidiasis and the therapeutic use of antifungal agents in dentistry)

A chronic symmetrical area on the tongue anterior to the circumvallate papillae (Fig 3). It is made up of atrophic filiform papillae. The exact cause is not fully understood; however, biopsy of this area usually yields Candida hyphae in over 85 per cent of cases. It tends to be associated with smoking and the use of inhaled steroids.

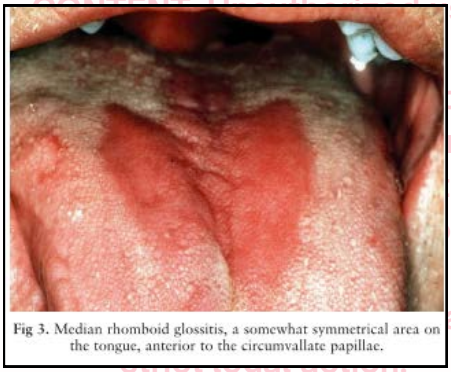

Fig 3. Median rhomboid glossitis, a somewhat symmetrical area on the tongue, anterior to the circumvallate papillae.

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Antifungal Therapy for Oral candidiasis

(REFERENCE: TG)

Management of oral candidiasis in immunocompromised patients require specialist advice.

Antifungal therapy is not always necessary for oral candidiasis and Candida-associated lesions. Successful treatment relies on adequate contact time between the antifungal and the affected mucosa, so advise patients not to eat or drink directly after administration.

Treat oral candidiasis in neonates and children younger than 2 years with a topical antifungal. In infants, apply the dose in the front of the mouth to avoid choking.

Use:

- **nystatin 100 000 units/mL suspension 1 mL topically (then swallowed), 4 times daily, after feeding, for 7 to 14 days; continue treatment for 2 to 3 days after symptoms resolve***

OR

- **miconazole 2% gel 1.25 mL topically (then swallowed), 4 times daily, after feeding, for 7 to 14 days; continue treatment for at least 7 days after symptoms resolve.**

Treat oral candidiasis in adults and children 2 years and older with:

- **miconazole 2% gel 2.5 mL topically (then swallowed), 4 times daily, after food, for 7 to 14 days; continue treatment for at least 7 days after symptoms resolve**

OR

- **amphotericin B 10 mg lozenge sucked (then swallowed), 4 times daily, after food, for 7 to 14 days; continue treatment for 2 to 3 days after symptoms resolve**

OR

- **nystatin 100 000 units/mL suspension 1 mL topically (then swallowed), 4 times daily, after food, for 7 to 14 days; continue treatment for 2 to 3 days after symptoms resolve.**

Advise patients with dentures to use miconazole or nystatin, because the antifungal should be applied to the fitting surface of the dentures at least twice a day. Dentures should be removed from the mouth at night, then cleaned and stored in a dry environment overnight.

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Antifungal Therapy for Angular cheilitis

Treat angular cheilitis with a topical antifungal cream; use:

- **clotrimazole 1% cream topically to the angles of the mouth, twice daily for at least 14 days; continue treatment for 14 days after symptoms resolve**

OR

- **miconazole 2% cream topically to the angles of the mouth, twice daily for at least 14 days; continue treatment for 14 days after symptoms resolve.**

A mild topical corticosteroid can be used in addition to the topical anti-fungal, to treat the associated inflammatory dermatitis; use:

- **hydrocortisone 1% cream topically to the angles of the mouth, twice daily until inflammation subsides.**

Combination products containing a topical corticosteroid and antifungal are available but should only be used until inflammation subsides. Treatment should be completed with a topical antifungal alone, for 14 days after symptoms resolve.

Seek specialist advice for persistent angular cheilitis. Treatment for mixed Infection may be required or it may be a symptom of a systemic condition, such as Crohn disease or granulomatous disease.

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ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Antifungal Therapy for Angular cheilitis

(REFERENCE: Oral candidiasis and the therapeutic use of antifungal agents in dentistry)

Therapeutic use of antifungal agents Diagnosis: smear, culture, biopsy

Correct diagnosis should be based on history and thorough examination. It should be treated based on clinical features but confirmation by one of following methods should be undertaken if initial therapy is unsuccessful.

Direct examination of a smear

The affected area is scraped with a spatula and the smear spread onto a glass slide. The slide is air dried, fixed in alcohol and stained with periodic acid Schiff reagent (PAS), after which examination by light microscopy is undertaken for the presence of yeast and hyphae. The dorsum of the tongue, a reservoir for oral yeast, should be smeared at the same time, along with the fitting surface of the denture, if one is worn.

Culture

Whole saliva, or a 10ml mouth-swill of sterile water, is collected and the presence and number of candidal colonies noted after culture on Sabouraud's agar.

Biopsy

Biopsy is particularly useful for the diagnosis of hyperplastic candidosis. Histopathological examination will reveal epithelial parakeratosis with polymorpho-nuclear leukocytes in the superficial layers. PAS stained slides will show the presence of candidal hyphae in this area. As this form may mimic other lesions, such as squamous cell carcinoma, a biopsy is recommended in addition to empirical therapy.

Principles of management: denture adequacy and hygiene; medical considerations (B12, folate, iron deficiency; diabetes; medication; immune deficiency)

Confirm the diagnosis. Investigate/eliminate underlying causes:

- **check adequacy of dentures;**
- **exclude deficiency states (e.g., iron, folate or vitamin B12 deficiency);**
- **exclude diabetes mellitus;**
- **check drug history (e.g., antimicrobials, corticosteroids);**
- **check medical history (e.g., immune deficiency)**

ORAL FUNGAL INFECTIONS (ORAL CANDIDIASIS)

Recommended treatment protocol

- Recognize predisposing factors and eliminate as many as possible. Particular attention should be paid to denture hygiene and, if the dentures are inadequate, they should be replaced.
- Possible underlying systemic disorders should be investigated if local factors such as dentures are not contributing.
- Administer topical antifungal agents, e.g., amphotericin lozenges – alone if the patient does not wear dentures, or in conjunction with nystatin ointment or miconazole if dentures are worn – for a period of three weeks. The ointment should be liberally applied to the fitting surface of the denture four times daily.
- At night, dentures should be removed and cleaned thoroughly and soaked twice weekly in a diluted solution (approximately 1:20) of either sodium hypochlorite (common bleach) or white vinegar. This latter step is most important as the denture will act as a reservoir of infection if not treated in this way.
- If there are still signs of infection after three weeks, ensure that the patient is complying with the prescribed regimen, that the diagnosis is correct and that any predisposing factor has not been overlooked. If necessary, prescribe another course of medication.
- Occasionally, it is necessary to prescribe another form of antifungal, e.g., change to miconazole oral gel. Systemic antifungal agents should be reserved for candidal infections in debilitated or immuno- compromised patients.
- Treatment of immunocompromised patients with oral candidal infection using the drugs mentioned above will usually resolve or significantly diminish the signs and symptoms, but recurrences can be expected, and regular reviews and repeated antifungal therapy are usually required.
- Refer any patient who does not respond to the treatment outlined above to an oral medicine specialist or an oral and maxillofacial surgeon.

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ORO FACIAL PAIN

(REFERENCE: The differential diagnosis of toothache from other orofacial pains in clinical practice)

Patients with non-dental causes of orofacial pain will also present seeking a dental solution to symptoms which may closely mimic toothache. Correct identification of such patients may be difficult and may occur only after extended irreversible and expensive treatment. Dentists need to carefully evaluate all toothache patients to ensure that the diagnosis is correct prior to the initiation of irreversible treatment.

Usually, the patient has self-diagnosed the problem as toothache and expects quick and efficient resolution of their problem. The unwary dentist may feel obligated to do something to help the patient without first carefully evaluating the patient's pain history, carrying out thorough facial and intra-oral examinations and completing the required diagnostic tests. If the findings do not establish a tooth-related diagnosis or if initial treatment is unsuccessful, then specialist referral is indicated.

Table 1A. Diagnostic categories – endodontic

Condition	Characteristics	Investigations
Reversible pulpitis	– Short duration – Reacts to cold and/or heat stimulus – not tender to percussion – no radiographic change	– Check for caries, cracks, restoration breakdown, etc – Bitewing and periapical radiographs – Reproduce symptoms (with appropriate stimuli applied under rubber dam isolation) – Pulp sensibility test – Normal – Periodontal probing
Irreversible pulpitis	– Tendency to have lingering pain to cold and or heat – Not usually tender to percussion – Pain: initially sharp pain then dull, throb, poorly localized, can be spontaneous, wakes at night	– As above – Pulp sensibility test may provoke lingering pain
Pulp necrosis with infection	– May/may not be painful – Can be lingering pain to heat that is relieved by cold	– As above – No response to pulp sensibility tests
Chronic apical periodontitis	– No or minimal symptoms – Only sign is periapical radiolucency – Can have acute exacerbation	– As above – No response to pulp sensibility tests – Periapical radiograph shows periapical radiolucency
Acute apical periodontitis	– Tender to percussion +/- palpation – Pain with chewing – May/may not be pulp symptoms (only if acute irreversible pulpitis)	– As above – Pulp sensibility tests may vary – depends whether acute irreversible pulpitis or a necrotic, infected pulp – Periapical radiograph may/may not show periapical changes
Acute abscess	– Pus accumulation in periapical tissues – Acute tenderness to percussion and palpation – Often intra-oral swelling – Pain with chewing	– As for chronic apical periodontitis
Cellulitis	– Pain often decreased – Facial swelling: indurated, red diffuse – Possible fever, lethargy	– As for chronic apical periodontitis – No response to pulp sensibility tests – Palpate intra-orally and extra-orally to check for deep space infections – Temperature increased

Table 1B. Diagnostic categories – orofacial pain – non-odontogenic

Condition	Characteristics	Investigations
Muscular (MPD, muscle tension headaches, neck pain, whiplash, fibromyalgia)	Chronic dull ache following muscular distribution Muscular dysfunction	Tender muscles Imaging normal Diagnostic block no effect
Arthralgia (Internal derangement, osteoarthritis)	Pain, clicking, locking related to the TM joint	Radiograph or CT may show bone morphology changes MRI show disc abnormality
Psychogenic Atypical facial pain Atypical odontalgia	Abnormal often exaggerate description of symptoms Abnormal response to treatment	Objective tests normal Subjective tests atypical Known previous psychiatric history and treatment
Pathology Chronic infection – Sinusitis – Osteomyelitis	Deep constant pains Signs of inflammation	Abnormal imaging Abnormal blood test – white cell shift – C-reactive protein
Malignancy – Oral SCC – Brain tumour	Usually painless unless advanced Neuralgic pain	Abnormal CT – biopsy
Neuromuscular Dystonia Dystinesia	Abnormal involuntary movements with muscle pain	Abnormal EMG

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ORO FACIAL PAIN

Patients with a muscular problem (which is commonly myofascial pain dysfunction – MPD), muscle tension headaches and/or chronic neck problems, usually have referred pain to the teeth and alveolus.5,6 Their histories usually indicate multiple teeth, in particular the last one in the arch, are tender to lateral pressure. This is often more so than to percussion. The results of pulp sensibility tests and radiographic examination are usually normal. Often, when asked, the patient will admit to head or neck pains, but then frequently will tell the dentist that these have "nothing to do with their toothache". Examination of the masticatory and cervical muscles will usually confirm tenderness and dysfunction.

A subgroup of head and neck pain patients is those with fibromyalgia. This is a diffuse musculoskeletal syndrome involving either all of the muscles in the body or unilaterally so. They usually seek, but respond poorly to, physical, non-surgical treatment for their jaw pains and they have a number of features in common with the psychiatric group. A wide range of medical treatments has been reported for fibromyalgia, but there is no effective single treatment.

Neuralgic pains may closely mimic acute toothache of pulpal origin. Careful examination will confirm that soft tissues and not hard tissues are the triggering points for the pain. The extent of triggering away from the mouth to the lower eyelid and lateral nose for maxillary neuralgias and to the tongue and gingivae for mandibular neuralgias are important diagnostic clues. Interestingly, neuralgias rarely wake patients once they are asleep, whereas toothache is no respecter of the time of day since acute irreversible pulpitis will often wake a patient during the night. The longer the pain history, then usually the easier it is to make a diagnosis that the pain is primarily neuralgic. There is an important subgroup where there is a history of injury to the infra-orbital nerve from cheekbone fractures or to the mandibular nerve from mandibular fracture or removal of wisdom teeth. This is important to elicit. Neuralgic pains are abolished by diagnostic blocks, which can be useful as a diagnostic aid.

Patients with a psychiatric background are easy to misdiagnose if the dentist has not spent some time interacting with the patient before proceeding to dental treatment. The history of pain will usually be unusual and the response to treatment often markedly atypical. Other clues to the correct diagnosis are the past and present medications that the patient has been taking and sometimes there is an unwillingness to divulge aspects of their health history. Failure to correctly diagnose such patients will result in over-treatment or unnecessary treatment

ORO FACIAL PAIN

Patients with intra-articular temporomandibular joint problems, which present as pain, clicking and locking of the jaw, are less likely to receive dental treatment in an attempt to resolve their problem. Conversely, dental treatment may frequently be cited by the patient as either the cause of or the exacerbating factor for their jaw joint problems. Consideration should always be given to using a bite block to support the jaw, particularly for those with a previous history of muscular or arthralgia jaw problems. This reduces any overstrain of the masticatory system.

A migraine is a severe debilitating unilateral headache, usually following the temporal artery, but sometimes the facial artery. There is nausea, tendency to vomit, photophobia and the patient is generally non- functional for several hours or days. Between attacks they may have a background muscular pain. It is common for patients to tell the dentist that they suffer from migraines, but on simple questioning it is clear that usually they are talking about bilateral muscle tension headaches.

Temporal arteritis is an uncommon but severe deeply boring headache, which simulates MPD. It most commonly involves the temple, but may also occur following the facial or lingual arteries. Prompt diagnosis by blood tests for raised ESR (above 80 is diagnostic) and arterial biopsy is required as, if untreated, permanent unilateral blindness may occur.

Patients with an unresolved, deep-seated pain commonly fear that they have cancer. In the early stages, cancer is not usually painful and only becomes so when nerves are involved or superficial tissues become infected. The fear of cancer is best addressed directly by exclusion with CT scans or other images and biopsy of any lesions.

The first step in managing a patient with orofacial pain is to diagnose the nature of the pain and this must be done prior to initiating any treatment. This must include taking a detailed history of the pain from the whole head and neck, not just a tooth or dental, perspective. Practitioners should be aware of any prior history of pains, both generally and in the specific region. Detailed examination of the jaw muscles, jaw joints and sensory nerves should be performed prior to examination of the teeth. A full dental examination should include visual examination, palpation, pulp sensibility tests, and radiographs. The effect of diagnostic or therapeutic blocks should also be considered.

CONCLUSIONS

If there are no clear signs of dental disease which is consistent with toothache, then referral to a specialist is strongly recommended prior to initiating any treatment.

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ORO FACIAL PAIN

Orofacial pain has many causes, only some of which are dental. If the patient's pain is not of dental origin, refer them for medical assessment. If the patient's pain is of dental origin, assess the type of pain (nociceptive, neuropathic or nociplastic) and its duration (acute or chronic), because these factors affect the optimal treatment strategy.

Pain managed by dentists is usually acute dental pain. Such pain:

- is usually nociceptive
- usually resolves rapidly with appropriate dental treatment, but may require short-term use of analgesics
- serves a protective biological function—it alerts the body to a potential threat, prevents the body from further harm, and can teach the body to avoid similar harm in the future.

Chronic pain (also referred to as persistent pain) may be associated with ongoing pathology; however, often the originating pathology is no longer evident and pain persists because of lasting changes within the nervous system. Such pain:

- may be nociceptive, neuropathic, nociplastic or mixed
- can be difficult to diagnose because there may not be any obvious pathology
- may result in anxiety, fear, depression, loss of sleep and impaired social functioning; these factors may also affect a patient's pain experience
- requires a sociopsychobiological (biopsychosocial) approach to assessment and management; Analgesics have a limited role.

ORO FACIAL PAIN

Indications for analgesics for acute dental pain

Analgesics modify the sensation of pain but do not address its cause. Therefore, for acute dental pain, analgesics should only be used as an adjunct to dental treatment in the following circumstances:

- when the patient's pain cannot be eliminated or adequately controlled by dental treatment and other required drugs (e.g. antibiotic therapy for spreading infection)
- following surgical procedures that cause postoperative pain
- when a patient has presented to a medical practitioner and is unable to see a dentist promptly—interim analgesics should be offered for certain indications and the patient should be reminded that dental treatment is necessary to treat the cause of pain.

Analgesics should only be used as an adjunct to appropriate dental treatment.

Can the patient take medications orally?

Oral administration is preferred. If this is not possible (i.e. if the patient has difficulty swallowing or gastrointestinal absorption is likely to be significantly reduced), consider suppositories or injections.

Although nonsteroidal anti-inflammatory drugs (NSAIDs) are preferred for acute nociceptive dental pain because they are anti-inflammatory, their adverse effect profile limits their use in some patients. Before prescribing an NSAID for a patient, determine whether NSAID use is appropriate based on their comorbidities and risk factors. If NSAID use is appropriate, ibuprofen is preferred because of good efficacy with limited toxicity, and widespread experience with its use. However, other NSAIDs have different safety profiles and may be preferred in some patients.

Paracetamol can be combined with an NSAID or used alone in patients who cannot take NSAIDs.

Opioids can be used for acute nociceptive dental pain in adults, when nonopioid analgesics and nonpharmacological measures (e.g. dental treatment) have failed to provide adequate pain relief or are unlikely to do so (e.g. severe pain). Oxycodone is preferred; do not use codeine.

Combining analgesics from different classes can result in enhanced pain management, or synergistic analgesia (e.g. combining ibuprofen and paracetamol provides greater pain relief than either drug alone).

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ORO FACIAL PAIN

Guide to differentiating and managing acute dental pain

Description of pain & associated features	Likely cause & suggested management
intermittent dental pain that is experienced when the tooth is exposed to a stimulus (e.g. hot, cold or sweet food or drinks) and resolves once the stimulus is removed	**Likely cause:** reversible pulpitis **Initial management by medical practitioners:** advise the patient to avoid food or drink that provokes pain, cover any obvious cavity with an inert material (e.g. chewing gum, Blu Tack) advise the patient to see a dentist as soon as possible analgesics and antibiotic therapy are not indicated **Dental treatment:** simple restoration or desensitisation treatment is required
severe dental pain that is experienced when the tooth is exposed to a stimulus (e.g. hot, cold or sweet food or drinks) pain persists as a dull throbbing ache after the stimulus is removed, and can become continuous	**Likely cause:** irreversible pulpitis **Initial management by medical practitioners:** advise the patient to avoid food or drink that provokes pain offer analgesics—NSAIDs are preferred if the patient can use them. cover any obvious cavity with an inert material (e.g. chewing gum, Blu Tack) if symptoms are severe, consider local anaesthesia of the affected tooth for temporary pain relief advise the patient to see a dentist as soon as possible antibiotic therapy is not indicated. **Dental treatment:** endodontic treatment (root canal) or extraction is usually needed
dental pain that presents as a dull throbbing ache, and is not triggered by a stimulus such as hot, cold or sweet food or drinks tooth may be sore to bite on.	**Likely cause:** infected root canal system with acute periapical inflammation (apical periodontitis) **Initial management by medical practitioners:** offer analgesics—NSAIDs are preferred if the patient can use them, advise the patient to see a dentist urgently antibiotic therapy is not indicated for a localised odontogenic infection; however, if dental treatment is not likely to be received within 24 hours, start antibiotic therapy as for spreading odontogenic infection without severe or systemic feature. **Dental treatment:** endodontic treatment (root canal) or extraction is needed.
tenderness of the tooth to pressure and on biting	**Likely cause:** fractured or cracked tooth or localised odontogenic infection. **Initial management by medical practitioners:** advise the patient to see a dentist urgently; it is difficult for medical practitioners to differentiate a fractured tooth from a Localised odontogenic infection (even with imaging) without a visible abscess or pus to indicate infection offer analgesics—NSAIDs are preferred if the patient can use them. antibiotic therapy is only indicated if a localised odontogenic infection is confirmed and dental treatment is not likely to be received within 24 hours. **Dental treatment:** restoration, endodontic treatment (root canal) or extraction is needed.

ORO FACIAL PAIN

Guide to differentiating and managing acute dental pain

Description of pain & associated features	Likely cause & suggested management
facial swelling and pain following a toothache **without any of the following**: significant facial swelling and pain, trismus, neck swelling, difficulty swallowing, difficulty breathing, airway compromise or systemic features of infection	**Likely cause:** spreading odontogenic infection without severe or systemic features **Initial management by medical practitioners:** offer analgesics—NSAIDs are preferred if the patient can use them, if dental treatment is not likely to be received within 24 hours, start antibiotic therapy, otherwise antibiotic therapy is not indicated. advise the patient to see a dentist urgently. **Dental treatment:** endodontic treatment (root canal) or extraction is needed
swelling and pain following a toothache **with any of the following**: significant facial swelling and pain, trismus, neck swelling, difficulty swallowing, difficulty breathing, airway compromise or systemic features of infection	**Likely cause:** spreading odontogenic infection with severe or systemic features **Initial management by medical practitioners:** provide appropriate support of airway, breathing and circulation arrange urgent transfer to a hospital with an oral and maxillofacial surgeon or other appropriate expert. **Dental treatment:** surgical intervention and intravenous antibiotic therapy is needed.
dental pain worsening 1 to 4 days after tooth extraction	**Likely cause:** alveolar osteitis (dry socket) **Initial management by medical practitioners:** flush the socket with warm sterile saline until all debris is removed from the socket offer analgesics—NSAIDs are preferred if the patient can use them advise the patient to see the practitioner who performed the extraction urgently antibiotic therapy is not indicated. **Dental treatment:** further socket irrigation and analgesia may be needed an obtundent dressing may relieve pain
acute onset of severe pain throughout the mouth associated with gingival bleeding and necrosis or ulcers of the interdental papillae halitosis is usually present	**Likely cause:** necrotising gingivitis (previously known as acute necrotising ulcerative gingivitis) **Initial management by medical practitioners:** offer analgesics (to select an appropriate analgesic regimen for acute dental pain. chlorhexidine mouthwash or hydrogen peroxide solution may be used if pain limits the patient's ability to mechanically clean their teeth advise the patient to see a dentist urgently for further management advice **Dental treatment:** thorough local debridement of the gingiva, local irrigation and antibiotic therapy are needed

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Guide to differentiating and managing acute dental pain

Description of pain & associated features	Likely cause & suggested management
Acute unilateral or bilateral pre-auricular pain mouth opening may be restricted.	**Likely cause:** temporomandibular disorders **Initial management by medical practitioners:** Advice the patient to rest the jaw (e.g. eat only soft foods) and avoid extreme jaw movements (e.g. yawning) Advice the patients to apply cold or warm compresses, as indicated offer analgesic-NSAIDs are preferred if the patient can use them, advice the patient to see the dentist as soon as possible for future management advice. **Dental treatment:** If conservative measures fail, referral to an oral medicine specialist or oral and maxillofacial surgeon may be required.
Dental pain that worsens when the head is tilted forward	**Likely cause:** maxillary sinusitis **Initial management by medical practitioners:** Therapy is recommended and antibiotics are rarely needed. **Dental treatment:** is not required.

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Analgesic regimens for mild to moderate acute dental pain in adults

Nonopioid analgesics (NSAIDs and paracetamol) should be taken regularly, rather than as required, to achieve continuous pain relief.

If adjunctive analgesia is required for mild to moderate acute nociceptive dental pain use:

- ibuprofen 400 mg orally, 6- to 8-hourly for the shortest duration possible and no more than 5 days without review

PLUS

- paracetamol 1000 mg orally, 4- to 6-hourly (to a maximum of 4 g in 24 hours) for the shortest duration possible.

Avoid fixed-dose combination products because they do not allow the daily dose of each drug to be maximised. As the tissue heals, stop ibuprofen and use paracetamol as a single drug.

If a COX-2-selective NSAID is preferred based on the patient's risk factors use:

- celecoxib 100 mg orally, twice daily for the shortest duration possible and no more than 5 days without review

PLUS

- paracetamol 1000 mg orally, 4- to 6-hourly (to a maximum of 4 g in 24 hours) for the shortest duration possible.

As the tissue heals, stop celecoxib and use paracetamol as a single drug.

If NSAIDs are contraindicated use:

- paracetamol 1000 mg orally, 4- to 6-hourly (to a maximum of 4 g in 24 hours) for the shortest duration possible.

If analgesics are used after a surgical procedure that causes postoperative pain, inform the patient of the usual course of pain (e.g. pain is worst 48 to 72 hours after surgery, then improves). Advise the patient to return to the dentist for review if pain persists.

If postoperative pain persists for longer than expected, advise patients to return to the dentist for review.

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Analgesic regimens for mild to moderate acute dental pain in adults

Nonopioid analgesics (NSAIDs and paracetamol) should be taken regularly, rather than as required, to achieve continuous pain relief.

For severe acute nociceptive dental pain (e.g. after dental surgery) in patients who can use NSAIDs, as a three-drug regimen, consider:

- ibuprofen 400 mg orally, 6- to 8-hourly for the shortest duration possible and no more than 5 days without review

OR

- celecoxib 100 mg orally, twice daily for the shortest duration possible and no more than 5 days without review

PLUS

- paracetamol 1000 mg orally, 4- to 6-hourly (to a maximum of 4 g in 24 hours) for the shortest duration possible

PLUS

- oxycodone immediate-release 5 mg orally, every 4 to 6 hours as necessary, for the shortest duration possible and no more than 3 days. Use a lower dose in elderly or frail patients because they are particularly vulnerable to adverse effects. Prescribe small quantities to avoid inappropriate use in the community.

- **In patients who cannot use NSAIDs, use paracetamol plus oxycodone.**

Always consider the benefits, harms and regulatory requirements of prescribing an opioid. Prescribe the lowest effective dose and advise patients to take a dose only when necessary. Ensure the patient understands the intended duration of opioid use and when to stop taking the opioid or return for review (e.g. if pain persists for longer than expected). This is of particular importance because long-term opioid use often starts with the use of opioids to treat acute pain. As the tissue heals and the patient requires less analgesia, use a stepwise approach to tapering and stopping analgesics. First, stop oxycodone, then stop ibuprofen or celecoxib, and lastly, stop paracetamol.

Ensure patients understand the intended duration of opioid use.

If opioids have not been required in hospital or pain can be successfully managed with nonopioid analgesia, do not prescribe opioids on discharge. Do not use modified-release opioids for acute dental pain.

Administering local anaesthetics by infiltration or regional block is an alternative or additional state' for the management of severe acute dental pain, provided the clinician is competent in these methods.

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Analgesic regimens for acute dental pain in children

Do not use aspirin in people younger than 16 years because of the risk of Reye syndrome. Do not use opioids for acute dental pain in children outside the specialist setting.

For acute dental pain in children 3 months or older, use ibuprofen or paracetamol; ibuprofen and paracetamol can be combined for enhanced pain management. Give doses regularly, rather than as required, to achieve continuous pain relief. Use:

- ibuprofen 5 to 10 mg/kg (use the lowest effective dose) up to 400 mg orally, 3 times daily, at 6- to 8-hour intervals. Continue treatment for the shortest duration possible and no more than 3 days without review

OR

- paracetamol 15 mg/kg up to 1000 mg orally, 4 times daily, at 4- to 6-hour intervals. See Table 14 (p.143) for calculated doses. Continue treatment for the shortest duration possible.

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Temporomandibular disorders

Temporomandibular disorders (TMD) is a collective term for a number of clinical problems that involve the masticatory muscles, or the temporomandibular joints (TMJ) and associated structures.

They can be classified into subtypes such as:

- temporomandibular joint disorders
- masticatory muscle disorders

Accurate diagnosis of a temporomandibular disorder requires appropriate history, examination and imaging. Table 15 (below) outlines signs and symptoms of temporomandibular disorders.

Table 15. Signs and symptoms of temporomandibular disorders

Common symptoms	pain and restriction with mandibular function
	pain in and around the ears and masticatory muscles
	headache
	neck pain
	limited jaw opening
	joint sounds
	clenching of teeth (bruxism; see p.149)
Uncommon symptoms	reduced hearing
	changes in occlusion
	toothache
	altered sensation in the face (eg paraesthesia, a feeling of swelling)
Signs	tenderness of the temporomandibular joints and masticatory muscles during movement
	temporomandibular joint sounds (clicking, clunking or crepitus) during repetitive opening and closing of the mouth, as well as lateral and protrusive movements [NB1]
	deviation on opening of the jaw

Temporomandibular joint sounds are not an indication for treatment, unless associated with pain or dysfunction.

Risk factors for temporomandibular disorders include direct trauma or indirect trauma (e.g. acceleration—deceleration injury) and parafunctional activities (e.g. tooth grinding or Clenching teeth). Psychosocial issues (e.g. stress, anxiety) may also contribute to the development of temporomandibular disorders. Malocclusion has not been shown to be a risk factor for temporomandibular disorders. Habits such as lip and cheek biting, biting fingernails, chewing gum and smoking can perpetuate an existing temporomandibular disorder. Chronic temporomandibular disorders are often associated with psychological disorders.

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Management of temporomandibular disorders

The aim of temporomandibular disorder management is to control the patient's symptoms rather than achieve a cure. Treatment goals should be tailored to the specific diagnosis, and may include reducing pain and adverse loading, restoring mandibular function and resuming normal daily activities.

Management strategies are conservative and can include:

- patient education and reassurance
- jaw rest, using strategies such as dietary modification to minimise chewing (e.g. eating only soft foods)
- avoidance of extreme jaw movements (e.g. yawning, chewing gum, singing)
- massage and application of warm packs to the temporomandibular joints and cheeks several times per day. old packs can be useful in the presence of redness and swelling
- behavioural modification (e.g. identifying and managing sources of stress, which may be facilitated by individual or group counselling)
- regular treatment (gentle muscle stretching and massaging) by a physiotherapist familiar in the management of temporomandibular disorders
- use of custom-made full-coverage intraoral occlusal splints* to reduce joint loading, muscle activity and pain—splints are generally worn at night and protect teeth from the effects of bruxism. They should constitute only one part of a broader management approach
- short-term use of drugs—discourage patients from relying on drugs alone (particularly drugs of dependence) to treat the symptoms of temporomandibular disorders because of their chronic nature. Analgesics, muscle relaxants, anxiolytics, anticonvulsants, corticosteroids and antidepressants have been used with variable success.

An acute exacerbation of chronic temporomandibular disorder can be treated with ibuprofen and/or paracetamol.
If conservative measures are inadequate and pain and dysfunction become severe or chronic, refer the patient to an oral medicine specialist or oral and maxillofacial surgeon.

There is some evidence for the use of botulinum toxin to manage the symptoms of temporomandibular disorders when conservative measures are inadequate. Ensure patients understand that botulinum toxin is not a cure for temporomandibular disorders but may be used as part of the overall management strategy. The use of botulinum toxin for temporomandibular disorders is off label. Dentists require additional training to administer botulinum toxin.

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Management of temporomandibular disorders

If the recommended doses and protocols are adhered to, the incidence of adverse effects is low. Local complications include stinging during injections, bruising at the site of injection and excessive muscle weakness. Adverse effects associated with inadvertent injection of botulinum toxin into nontarget tissues are rare but can include alteration in smile and temporary dry mouth. Systemic adverse effects include an influenza-like syndrome that is transient and hypersensitivity reactions.

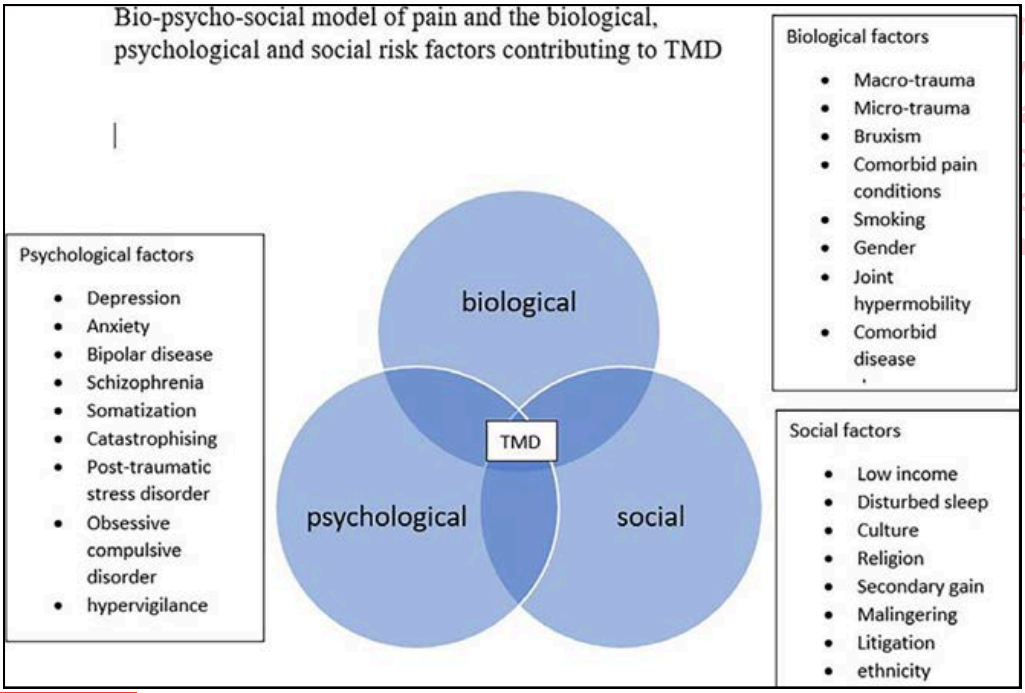
Surgery for temporomandibular disorders is rarely required. Only consider referring the patient for a surgical assessment if symptoms have not responded to conservative management and there is definitive evidence of internal joint derangement or other joint pathology on imaging.

(REFERENCE: Temporomandibular Disorder: a practical guide for dental practitioners in diagnosis and management)

Temporomandibular disorder (TMD) is the most common reason for seeking dental care other than dental pain. Dental practitioners, therefore, need to know the risk profile for those patients most vulnerable to TMD, to accurately diagnose TMD and know the most appropriate management pathways for optimal outcomes.

Temporomandibular disorder (TMD) is a broad term encompassing pain and/or dysfunction of the masticatory musculature and the temporomandibular joints. 1 The most important feature is pain, followed by restricted or limited jaw movement, and joint noises during jaw movement.

WHO IS AT RISK OF DEVELOPING A TMD?



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The 'biopsychosocial' theory of pain/TMD and the biological, psychological and social risk factors for TMD.

Macro-trauma refers to events resulting in head trauma including accidents, falls, sports injuries, forceful intubation, physical abuse, removal of third molars and other long dental procedures.

Micro- trauma includes awake and sleep bruxism and other parafunctional habits including chewing gum, nail biting, lip and cheek biting. Trauma can be both a pre-disposing and initiating factor in the development of TMD.

Table 1. TMD signs and symptoms

Common symptoms	Less common symptoms	Common signs
Facial pain	Reduced hearing or Feeling of blocked ears	Temporomandibular joint sounds
Restricted jaw movement	Tooth ache/sensitivity/ Tooth mobility	Restricted opening or jaw deviation upon opening
Headache	Sleep disturbance	masseteric hypertrophy
Pre-auricular pain	Paresthesia/swelling of face	Tenderness muscle of mastication
Difficulty eating	Tinnitus	Tenderness TMJ
Bruxing/ clenching	Occlusal disturbance	tenderness to percussion of teeth
Ear ache	Pain with swallowing	Bruxing/clenching
Joint sounds	Sharp pain in ear	
Neck/shoulder pain	Retro-orbital pain	

While the incidence rate of TMD is only slightly greater in women than men, up to four times as many women seek treatment in addition to having a greater overall pain sensitivity. TMD is also more likely to persist in women. It is interesting to note that adverse experiences in childhood may be associated with greater risk for the development or maintenance of chronic pain in youth, particularly chronic head-aches and musculoskeletal pain.

Patients with TMD and chronic pain should be assessed for psychological factors such as depression or anxiety, catastrophizing, distress, fear avoidance beliefs and post-traumatic stress disorder (PTSD)

Chronic TMD can be challenging to manage and consequently such patients should be referred to a specialist dental practitioner for a detailed assessment and management, with pain education forming an important component of the consultation.

Bruxism may be a risk factor for some negative oral health consequences such as masticatory muscle pain, temporomandibular joint pain and extreme tooth wear. Bruxism and TMD can exist independently. Signs of bruxism (parafunction) include cracked teeth, multiple failed restorations, chipping of incisal edges, wear facets, mobile teeth, masseteric hypertrophy, buccal mucosal ridging and scalloping of the lateral border of the tongue.

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HIGH-RISK DENTAL PROCEDURES

Certain dental procedures may increase the likelihood of either initiating or perpetuating a TMD.

- **Dental appointments that are predicted to be more than 30 min long,**
- **procedures on posterior teeth where rubber dam is used**
- **procedures where force is used such as dental extractions, may initiate a TMD.**
- **Patient reported fatigue or jaw and muscle pain, should also be taken into account by the dental practitioner.**

Table 2. TMD Diagnosis and subtype

	Myalgia	Arthralgia	Intra-articular joint disorder	Degenerative joint disorder
Information from patient history	Pain in the masticatory musculature, modified jaw movement, function or parafunction	Pain in the masticatory structure, modified by jaw movement, function or parafunction	TMJ noise(s) present or TMJ noises present and jaw locks with limited opening	TMJ noises present
Clinical examination findings	Reported pain on palpation of the muscles of mastication, or pain reported with maximum mouth opening	Patient reported pain on palpation of TMJ or patient reported pain upon maximum unassisted or assisted opening, right, left, lateral or protrusive movements	Clicking, popping, or snapping noise present with either opening and closing or opening and closing and lateral or protrusive movements	Crepitus (crackling sounds) present in TMJ during maximum active opening, passive opening, right lateral, left lateral or protrusive movements

(REFERENCE: Temporomandibular Disorders: Current Concepts and Controversies in Diagnosis and Management)

Table 1. Common diagnoses of temporomandibular disorders (TMD) and their clinical findings.

Painful Conditions	Clinical Findings
Myalgia	Familiar pain in the masseter or temporalis upon palpation or mouth opening
Local Myalgia	Familiar pain in the masseter or temporalis localized to the site of palpation
Myofascial pain	Pain in the masseter or temporalis spreading beyond the site of palpation but within the confines of the muscle
Myofascial pain with referral	Pain in the masseter or temporalis beyond the confines of the muscle being palpated
Arthralgia	Familiar pain in the TMJ upon palpation or during function
Headache attributed to TMD	Headache in the temple upon palpation of the temporalis muscle or during function

Non-Painful Conditions	Clinical Findings
Disc displacement with reduction	Clicking in the TMJ upon function
Disc displacement with reduction with intermittent locking	Clicking in the TMJ with reported episodes of limited mouth opening
Disc displacement without reduction with limited opening	Limited mouth opening affecting function, with maximum assisted opening < 40mm
Disc displacement without reduction without limited opening	Limited mouth opening affecting function, with maximum assisted opening of ≥ 40mm
Degenerative joint disease	Crepitus of the TMJ upon function
Subluxation	History of jaw locking in an open mouth position, cannot close without a self-maneuver

Modified from Schiffman et al., 2014 [31]

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Table 2. Some less common diagnoses of temporomandibular disorders (TMD).

I. TMJ
A. Joint pain
1. Arthritis
B. Joint disorders
1. Hypomobility disorders other than disc disorders
a. Adhesions/Adherence
b. Ankylosis (Fibrous or Osseous)
2. TMJ dislocations
C. Joint diseases
1. Systemic arthritides
2. Condylysis/Idiopathic condylar resorption
3. Osteochondritis dissecans
4. Osteonecrosis
5. Neoplasm
6. Synovial Chondromatosis
D. Fractures
E. Congenital/Developmental disorders
1. Aplasia
2. Hypoplasia
3. Hyperplasia
II. Masticatory Muscles
A. Muscle pain
1. Tendonitis
2. Myositis
3. Spasm
B. Contracture
C. Hypertrophy
D. Neoplasm
E. Movement Disorders
1. Orofacial dyskinesia
2. Oromandibular dystonia
F. Masticatory muscle pain related to central/systemic pain disorder
1. Fibromyalgia/widespread pain
III. Associated Structures
A. Coronoid hyperplasia

Modified from Peck et al., 2014 [32].

Table 3. Differential diagnosis of temporomandibular disorders (TMD).

Neuropathic Pain
Trigeminal neuralgia
Glossopharyngeal neuralgia
Postherpetic neuralgia
Traumatic neuralgia
Burning mouth syndrome
Atypical odontalgia
Atypical facial pain
Odontogenic Pain
Dental caries
Periodontal disease
Dental abscess
Dental sensitivity
Cracked tooth syndrome
Periocoronitis

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Intracranial Pain
Tumours
Aneurysms
Bleeding
Infection
Pain from Other Adjacent Structures
Ear
Nose
Throat
Eyes
Sinus
Salivary glands
Lymph nodes
Vasculature
Cervical region
Headaches not Attributed to TMD
Migraine
Cluster headache
Tension-type headache
Temporal arteritis
Referred Pain
Psychogenic Pain

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(REFERENCE: Temporomandibular Disorder: a practical guide for dental practitioners in diagnosis and management)

CLINICAL EXAMINATION

01. Observation of the patient

- Assessment of facial symmetry
- muscular hypertrophy, i.e. masseteric hyper- trophy
- evidence of nail biting
- evidence of other habits, such as jaw posturing, movements

02. Jaw movements

- mouth opening, limitations, trismus
- deviations of the mandible with opening or closing

03. TMJ

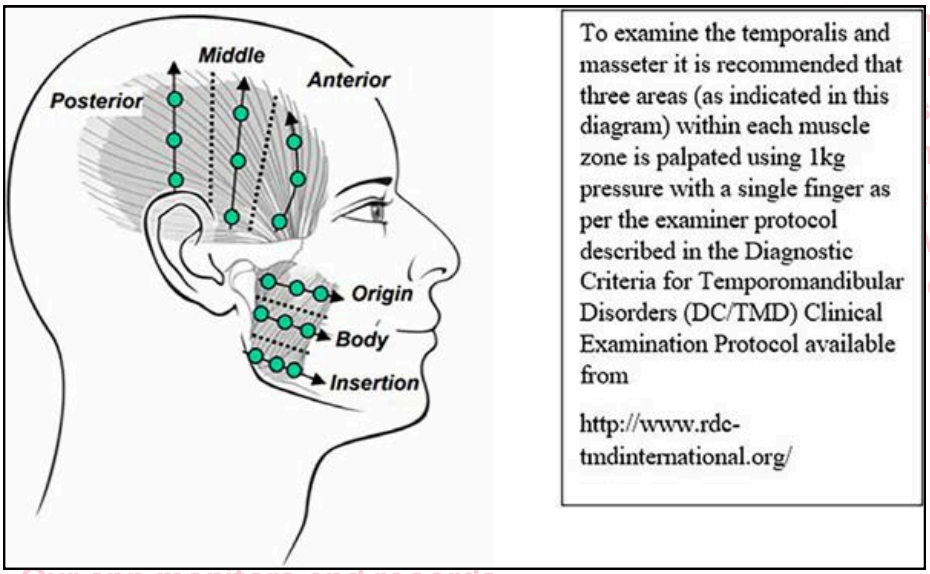
- palpate the TMJ in both the open and closed position
- feel for irregularities in joint movement
- listen for joint sounds

04. Muscles of mastication

05. Examination of dentition

- evidence of attrition, wear facets, cracks, craze lines, fractured teeth or restorations
- periodontal mobility, fremitus
- reduced occlusal vertical dimension
- loss of posterior molar support
- assessment of oral health and hygiene

06. Radiographic assessment: OPG or other images might be indicated



To examine the temporalis and masseter it is recommended that three areas (as indicated in this diagram) within each muscle zone is palpated using 1kg pressure with a single finger as per the examiner protocol described in the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) Clinical Examination Protocol available from

http://www.rdc-tmdinternational.org/

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MANAGEMENT OF TMD

TMD may resolve or improve over time plays an important role in patient management. In simple language a discussion about jaw rest avoiding initiating and perpetuating behaviour, such as:

- clenching and excessive movement
- limiting wide mouth opening
- some gentle muscle massages
- application of heat to the musculature may be therapeutic
- the use of anti-inflammatory medications

might be indicated for some patients and can offer significant relief of symptoms.

The use of an oral appliance may be warranted in some situations where nocturnal bruxing is implicated. Oral appliances can protect teeth from the effects of tooth grinding but their role in reducing the habit has not been conclusive.

They have been shown however to be an efficacious treatment modality for some TMD. Previously, the role of Oral appliances was mainly considered to be mechanistic and related to occlusal disharmony and skeletal disparity.

Oral appliances if used should be full coverage and patients should be reviewed at regular intervals. After a thorough assessment patients should be made aware of events that might trigger another episode of TMD for example:

- long dental appointments
- intubation for general anaesthesia
- micro and macro trauma.

Dental appointments should be kept short, mouth opening restricted and the use of a bite-block during dental procedures might be warranted. During dental appointments, frequent rest periods are indicated.

It is widely accepted now that psychosocial factors play an important role in the management of the TMD patient, along with other factors such as the doctor-patient relationship and placebo effect. Most individuals will indicate on their medical history questionnaire if they have a psychiatric diagnosis or are receiving counselling. The patients' list of medications may include anti-depressants and/or anti-psychotics which can prompt further discussion about their use.

For complex patients or those with chronic TMD, management can be more complicated and a referral to a specialist should be considered. The role of the general dentist is essential in the diagnosis and ongoing support for these patients; however, the management of TMD for this cohort, is likely beyond the scope of general dentistry.

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MANAGEMENT OF TMD

Specialist management may involve:

- further reassurance
- imaging (where indicated)
- pain education
- oral appliance therapy
- physiotherapy
- pharmacotherapy
- meditation/relaxation strategies
- education about sleep hygiene
- psychological/cognitive behavioural therapy
- botulinum injections
- hypnotherapy;
- biofeedback
- and in some cases, TMJ surgery

While there is some evidence for the efficacy of botulinum toxin in the treatment of TMD, it's use is 'off-label' and dentists must undertake additional training in order to administrate it.

Management of TMD should always include a detailed discussion with the patient and be augmented with a home care programme providing the patient some ownership of the treatment.

Patients who do not have any predisposing factors or a diagnosed TMD who are undergoing a high-risk dental procedure should be informed of the risk of developing a TMD. In such cases appropriate clinical review is recommended and should a TMD develop then referral to a specialist might be warranted.

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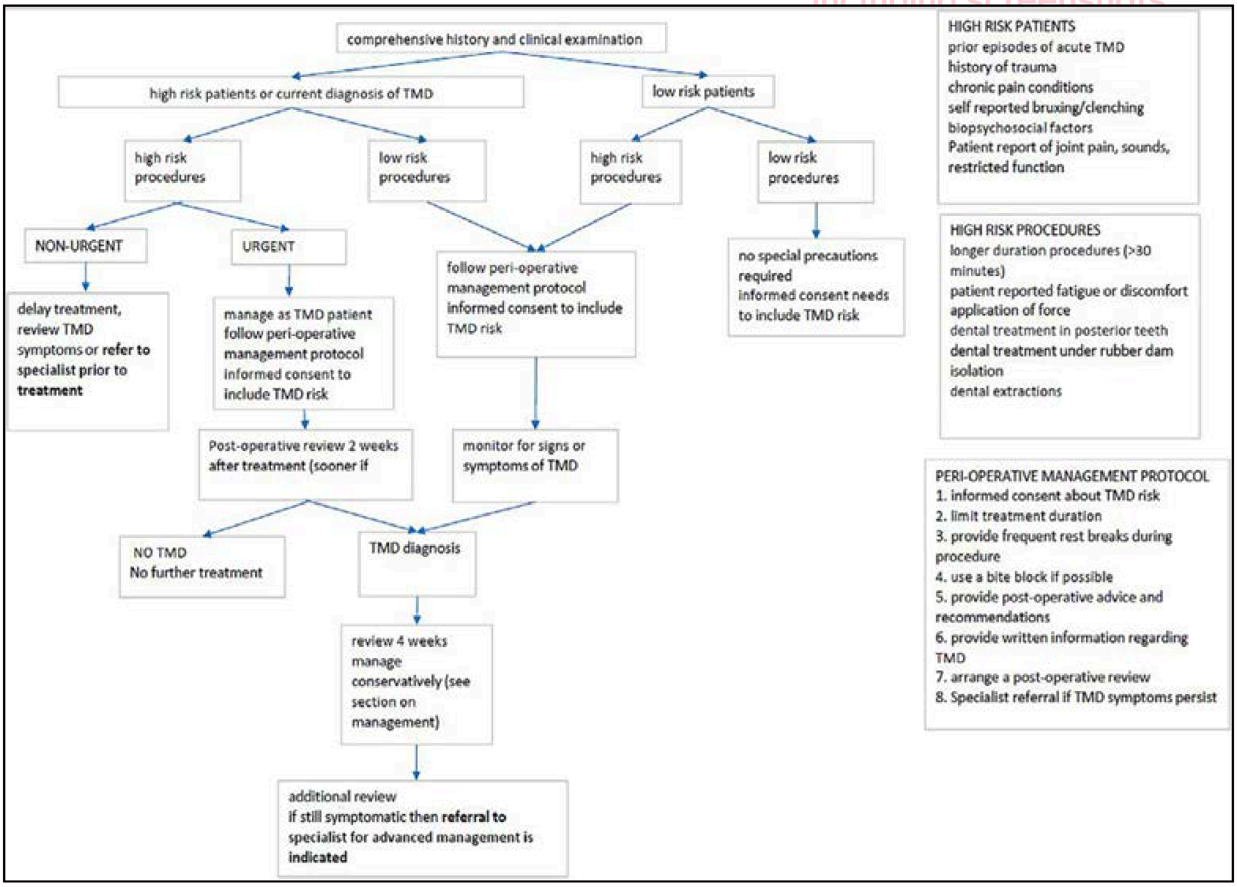
MANAGEMENT OF TMD



ACTINIC CHEILITIS

(REFERENCE: Actinic cheilitis in dental practice)

Actinic cheilitis is a potentially premalignant condition involving predominantly the vermilion of the lower lip. The action of actinic radiation is restricted to the skin and vermilion (lip-stick surface) of the lip and the interactions and boundaries of these are important in clinical assessment.

Understanding the geographic variation of the lip is the first step in identifying change. The second is recognition that the lips will often undergo a rapid and permanent change with disease.

Patients will often relate clinically apparent sun damage to lips to a single significant exposure with sunburn whilst others seem resistant to the same apparent exposure.

This is not uncommon on the lip and an example is actinic cheilitis and angular cheilitis. These are very different conditions, often contributed to but not primarily caused by similar factors, e.g., lip anatomy, but treated clinically in a very different manner. It is also appropriate at this early stage in the discussion to remove the often-used term of photo-ageing. There must be a recognition of age- related change but the use of ‘‘photo-ageing’’ dilutes the significance of sun-induced change.

Actinic cheilitis attracts a number of clinical definitions varying from premalignant to relatively bland descriptors of the sun-damaged lip.

Almost all lip carcinomas are associated with pre-existing actinic cheilitis and the particular histological type of actinic cheilitis does not correlate with progression to squamous cell carcinoma.

The expected demographic of a patient with actinic cheilitis is usually fair skinned, middle aged and with a history of accumulated sun exposure to the lower lip where the vermilion receives a high dose of ultraviolet radiation because it lies at right angles to the midday sun. It is also poorly protected by melanocytes or keratin. The upper lip is rarely affected.

Actinic cheilitis is more often seen in men in their fourth to eighth decade of life. The patient may have a past history of actinic keratoses on the skin, non-melanoma skin cancer or malignant melanoma.

Actinic cheilitis develops slowly over an extended period and with a greater prevalence in outdoors workers. This is also the group most likely to be regular cigarette users and the coincident application of radiation and chemical carcinogens to the one site is of concern with a synergistic effect amplifying the summed individual effects of ultraviolet radiation and cigarette use.

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ACTINIC CHEILITIS

(REFERENCE: Actinic cheilitis in dental practice)

Hyperkeratosis is often cited as the dominant finding and a diagnostic feature of actinic cheilitis but in fact dryness and atrophy are a more frequent clinical finding. The early presence of atrophy is significant as it identifies the earliest sign the clinician should be aware of and explains the significance of allowing actinic cheilitis to develop. A typical description of actinic cheilitis is a white lip plaque but this is a late reactive change occurring after atrophy and, given the tissue fragility inherent in atrophy, it is potentially "useful" for the integrity of the lip epithelium. Such hyperkeratotic plaques however, are frequently progressive and the early homogeneity often changes to an opaque non-homogeneous plaque requiring at least biopsy and histological assessment.

Table 1. Clinical signs associated with actinic cheilitis[8]

Sign	% of patients
Dryness	100
Atrophy	72
Scaly sites	65
Swelling	62
Erythema	59
Ulceration	59
Vermilion border indistinct	59
Transverse fissures	48
White plaques	41
Crusting	34
Blotchiness	28
Tissue pallor	17

Actinic cheilitis is a persistent chronic condition that is present in all seasons of the year, not just summer where acute sun exposure can cause congestion, erythema, vesicles, bullae, desquamation and even ulceration. The vermilion edge loses its plasticity which can be noted by the formation of wrinkles perpendicular to the long axis of the lip.

Although squamous cell carcinoma may develop without ulcer formation, it should always be considered if an ulcer develops within an area of actinic cheilitis. Other clinical signs that may indicate malignant change include: recurrent ulceration and failure to heal; red and white blotchy appearance with loss of the vermilion border; persistent crusting and flaking; generalized atrophic appearance with focal areas of opaque white thickening; and focal induration or nodule formation.

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ACTINIC CHEILITIS

Table 2. Histological features of actinic cheilitis[8]

Feature	% of cases
Dysplasia	100
Solar elastosis	100
Inflammation	100
Vasodilatation	100
Hyperplasia	86
Hyperkeratosis	59
Epithelial atrophy	55

There is an acknowledgement among clinicians that lip squamous cell carcinoma is often reported alone with no coincident diagnosis of actinic cheilitis. There is also the possibility that lip squamous cell carcinoma is reported as non-melanoma skin cancer and so the registry numbers for this condition are likely to be under-reported.

Assessment and recording clinical information

The main current recording instruments are:

- a detailed clinical description
- clinical measurement with a ruler
- precise diagrams
- clinical photography

It is important to carry out this assessment with adequate lighting, magnification if available, and with gloves so the lip surface can be palpated.

In a balanced assessment, this clinical examination must be combined with a range of other factors including:

- patient age
- skin type
- past sun exposure
- immune status
- anticipated sun exposure
- lifestyle factors (tobacco and alcohol)
- current clinical presentation
- dual or multiple pathoses
- medium and long-term management

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ACTINIC CHEILITIS

Fig 1. Early presentation of actinic cheilitis in a young patient. The vermilion presents generalized changes with a midline transverse fissure and melanotic macule.

- Figure 1 shows an adolescent patient with a broad exposed vermilion on the lower lip and clear evidence of dryness and tissue fragility from heavy sun exposure.
- He has a transverse midline fissure that has ulcerated on a number of occasions and currently presents a very fragile zone highly likely to continue to break down in the future.
- There is a recent presentation of a melanotic macule on the right vermilion and, as a coincidental finding, shallow commissural pits.
- His pale freckled skin, outdoor lifestyle and active participation in sport provides a challenge both in immediate clinical decision-making and particularly in the medium to long term.
- He currently presents as a mild actinic cheilitis and the potential in this patient for significant progression is very concerning.
- Requiring early active management and introduce a plan to improve the current presentation of the lip epithelium as well as provide long-term protection from further ultraviolet damage.

A suggested plan might include:

- initial discussion with the patient and family about the diagnosis and the potential for development of neoplastic lesions in the future
- limiting sun exposure between 10 am and 2 pm (or 11 am and 3 pm in daylight saving time), wearing a protective broadbrimmed hat, using an SPF30+ broad spectrum and water-resistant sunscreen lip balm applied generously and reapplied at least every two hours when outdoors
- the prescription of an emollient to moisturize the lip
- early review of the lip fissure to ensure progress is satisfactory
- a decision on the melanotic macule, review or excise.

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ACTINIC CHEILITIS

Fig 2. (a) Actinic cheilitis in a young patient with generalized atrophy and a non-homogeneous keratotic plaque with the right lateral aspect showing a granular surface. (b) A second younger patient with bilateral keratotic plaques overlying the generalized atrophy of actinic cheilitis.

Figures 2a and 2b show not uncommon initial presentations of actinic cheilitis. This does not equate to the early manifestations of actinic cheilitis but merely the time at which it was noted. Both patients are fair skinned, young and with a past history of significant sun exposure but relatively limited current exposure. The typical features of epithelial atrophy, hyperkeratotic plaques, blotchy erythema and indistinctness of the vermilion border are present.

The initial clinical assessment of hyperkeratotic lip plaques should always result in a clear decision to review, biopsy or excise. The assessment criteria are simple but clinically demanding and focus on the determination of either homogeneity or non-homogeneity.

Keratotic plaques that are homogeneous are consistent across the lesion in all clinical parameters. They do not show any variation in appearance, texture (induration) or tissue mobility on the underlying dermis (fixation compared with adjacent or contralateral normal tissues). Any doubt during the clinical examination places the lesion in the non-homogeneous category. Some lesions are visibly homogeneous but difficult to examine due to the thickness of keratin and again they should be regarded as non-homogeneous and addressed as such.

ACTINIC CHEILITIS

Figure 2a shows an extensive keratotic plaque extending from the left of midline to involve over half of the right vermilion. The central zone carries a focal erosion but the overall presentation of this area is non-homogeneous and any latitude given to this site should be limited with early review in two weeks. The lateral aspect of the keratotic area on the right vermilion is perhaps blander in presentation but of greater clinical concern. The surface presents a granular keratosis that is difficult to examine and requires histology for a confident diagnosis. Overall, this lip plaque is non-homogeneous and the immediate requirement for histology should be discussed with the patient but in context with the medium to long-term value of vermilionectomy, either partial or total. This patient is an excellent example of the value of intervention providing a very favourable long-term outcome.

The treatment must be supplemented with the ongoing requirement for:

- limiting sun exposure
- broad-brimmed hat
- generous application of SPF30+ broad spectrum
- water resistant sunscreen applied two hourly when outdoors.

This is standard advice applicable to all patients with actinic cheilitis and sound advice in the prevention of this condition in those without actinic cheilitis, particularly outdoor workers and sportspersons.

Figure 2b shows a not dissimilar presentation but with keratotic plaques on both sides of the vermilion. The keratin density on the right side made clinical examination difficult and the patient made an informed decision and underwent excision of this area with an excellent cosmetic result. Her current level of sun exposure is virtually nil and she decided to continue review of the lip with daily attention with an emollient sunscreen and the routine sun hygiene steps listed above.

ACTINIC CHEILITIS

Fig 3. (a) Generalized actinic cheilitis in an older patient with advanced non-homogeneous plaque formation. (b) A second older patient and tobacco user with a small squamous cell carcinoma on the left vermilion in a background of actinic cheilitis.

Figures 3a and 3b show cases with clearly non-homogeneous keratotic plaques overlying an actinic cheilitis. They both require histology for a definitive diagnosis. The precise features of non-homogeneity require documentation in the patient records but it is the presence of non-homogeneity that demands histology rather than the specific features that make a lesion non-homogeneous, e.g., crusting, nodularity and surface contour. In addition, the physical examination to determine induration and fixation may override the visible features as they strongly suggest malignancy.

Biopsy of both keratotic sites seen in Fig 3a is required, preferably excisional with an anticipated diagnosis of dysplasia associated with the actinic cheilitis, which was confirmed.

The case in Fig 3b has the additional complication that the patient is a cigarette user. Clinical examination of the keratotic plaque readily confirms the presence of induration (loss of normal tissue texture and hardness compared with adjacent or contralateral sites) and fixation (loss of normal tissue mobility due to an infiltrative process) and the clinical diagnosis of squamous cell carcinoma was confirmed histologically. An appropriate treatment for this patient focused on the urgent management of the squamous cell carcinoma with surgical excision. The medium-term options were also reviewed for the actinic cheilitis and the choices were to review or to include a vermilionectomy in the resection procedure.

ACTINIC CHEILITIS

Treatment of actinic cheilitis

- A thorough assessment of the patient. This is to ensure that any required short-term treatment is followed by a reasonable medium to long-term management plan.
- It is so important to gather comprehensive patient information encompassing previous sun exposure history, current status, anticipated lifestyle changes, and likely compliance with intervention and preventive strategies. The plans for an office worker with limited exposure will clearly be very different from that prepared for a professional outdoor sportsperson.
- Squamous cell carcinoma is the most common cancer of the oral cavity and this includes the lip. As with actinic keratoses, the exact rate of transition of actinic cheilitis to squamous cell carcinoma is not known.
- Various treatment methods have been used in the management of actinic cheilitis and these can be both surgical and non-surgical and include:
  1. cryotherapy
  2. electrosurgery
  3. topical retinoids
  4. 5-fluorouracil cream
  5. imiquimod cream
  6. photodynamic therapy
  7. carbon dioxide laser ablation
  8. surgical vermilionectomy
- All patients with actinic cheilitis, whether mild, moderate or severe should be advised to limit further sun exposure especially between 10 am and 2 pm (or 11 am and 3 pm in daylight saving time), wear a protective broad-brimmed hat, use a SPF30+ broad spectrum sunscreen lip balm regularly (every morning) with re-application every two hours when outdoors.
- Mild cases can be reviewed regularly and if there is any indication of clinical progression, they should be referred to the patient's medical practitioner, dermatologist or specialist in oral medicine for further assessment, investigation and possible treatment. Any patient with more significant disease should be referred immediately.

Dental practitioners are in an ideal position to provide a regular screening service to patients as a routine part of the general soft tissue examination. It adds minimally to a dental recall appointment and has the potential to provide a significant service to patients.

Dental practitioners have an opportunity to support medical colleagues and whilst many currently undertake this responsibility with their patients, there remains a valuable opportunity for further expansion into dental practice.

ORAL HERPES

(REFERENCE: TG)

Primary oral mucocutaneous herpes simplex virus (HSV) infection (herpetic gingivostomatitis) often occurs in childhood with fever, painful intraoral lesions, systemic symptoms (e.g. malaise, lethargy) and cervical lymphadenopathy.

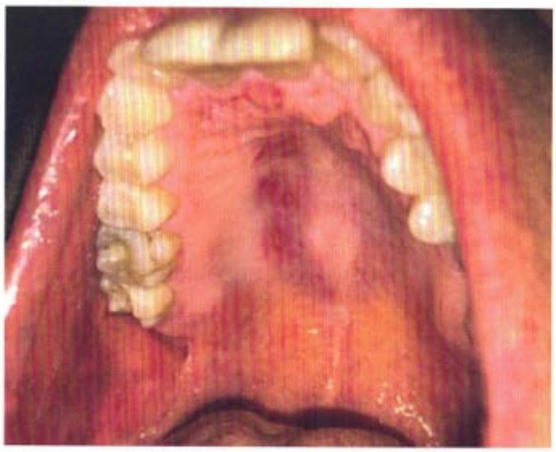
Intraoral herpes simplex virus lesions begin as blisters an ulcerate rapidly. Healing occurs within several day;
in infants, but can take up to 2 weeks in older children. During this time, it may be difficult to eat and drink and hospital admission may be required.

Herpetic gingivostomatitis is rare in adults, but can be severe and present with dehydration due to severe odynophagia.

While herpes simplex virus is the most common virus to cause mouth ulcers, other viruses (e.g. varicella zoster virus, coxsackie virus, cytomegalovirus) may be the cause. Intraoral herpes simplex virus lesions may resemble those seen in necrotising gingivitis.

However, necrotising gingivitis is rare in children and is confined to the gingival tissues, while herpetic gingivostomatitis lesion: are widespread and affect all soft tissues in the mouth.

Photo 15. Intraoral lesions caused by the herpes simplex virus



To reduce the risk of virus transmission, advise patients with an active herpes simplex virus infection to avoid direct contact of the lesion with other people.

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ORAL HERPES

Management:

1) Treat minor primary oral mucocutaneous herpes with supportive management (i.e. oral fluids, antipyretic drugs and analgesia).
2) Apply a topical anaesthetic or analgesic, such as:
   - benzydamine 1% gel (adult and child 6 years or older), topically to the lesions, 2- to 3-hourly as necessary.
3) If this is not available, lidocaine viscous solution is an alternative topical anaesthetic for hospital settings (but be aware of the higher cost):
   - lidocaine 2% viscous solution adult: use the lowest dose necessary up to 15 mL, rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 8 doses in 24 hours
   - child 3 years or older: use the lowest dose necessary up to 0.2 ml./kg (maximum 5 mL), rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 4 doses in 24 hours
   - child younger than 3 years: use the lowest dose necessary up to 0.2 ml/kg (maximum 1.25 mL), applied to the affected areas with a cotton swab, 3-hourly as necessary; maximum 4 doses in 24 hours.
4) lithe pharynx is affected in adults and children older than 12 years, lidocaine viscous solution can be gargled and swallowed.

Management of primary oral mucocutaneous herpes differs for the following patients, so referral to a medical practitioner is required:

- patients with severe presentations
- immunocompromised patients
- patients with HIV.

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ORAL HERPES

Recurrent oral mucocutaneous herpes (Herpes labialis)

(REFERENCE: The patient with recurrent oral ulceration)

Recurrent intraoral viral infection is usually limited to secondary herpes simplex virus. The recurrences are most commonly due to human herpes virus-1 (HHV-1) (which usually causes orofacial infections). Recurrent oral HHV-2 lesions (which are usually associated with genital infections) are rare. Clinically, the initial presentation is of fluid-filled vesicles which rapidly break down to form a cluster of small ulcers with ragged margins.

After primary infection, the virus is not eliminated from the body but migrates along nerve fibres to the trigeminal ganglion, where it lies dormant. In perhaps one-third of individuals the virus can be reactivated by non-specific stimuli, e.g., illnesses associated with fever, when it travels back to the periphery to cause secondary oral mucosal lesions. The lesions usually resolve in about 7 to 10 days in healthy individuals, but in immunocompromised patients' secondary herpetic lesions can be widespread, very slow to heal and refractory to treatment.

(REEFRENCE: TG)

Recurrent oral mucocutaneous herpes simplex virus (HSV) infection follows latent reactivation of the virus. Lesions usually occur on the lips (herpes simplex labialis or cold sores) but can also occur on the intraoral mucosa or other areas of skin.

Lesions are usually preceded by the prodromal stage, lasting several hours to days, which features pain, burning, tingling or itching.

Recurrences are usually mild and infrequent, and their frequency can be minimised with sun protection. Herpes simplex virus reactivation may be complicated by erythema multiforme.

A herpes simplex virus lesion affecting the oral mucosa cannot be differentiated from an aphthous or traumatic ulcer using microbiological testing—most adults will have positive serology for the herpes simplex virus from previous exposure, and viral DNA may be detected on swabs of aphthous or traumatic ulcers. Instead, diagnosis requires a thorough history and clinical examination.

To reduce the risk of virus transmission, advise patients with an active herpes simplex virus infection to avoid direct contact of the lesion with other people.

ORAL HERPES

(REFERENCE: Treatment of herpes labialis (cold sores)

Herpes labialis, or cold sores, is characterised by the eruption of groups of small blisters on the lip or around the mouth. The skin around the blisters is often red, swollen and sore.

Lesions are usually preceded by a set of prodromal symptoms (prodromal stage) which can include pain, burning, tingling, itching, inflammation, or redness, lasting for several hours to days. The blister stage follows during which the characteristic blisters emerge as a crop on the upper or lower lip. The blisters are filled with clear, yellow fluid and smaller blisters may coalesce into larger blisters as the infection progresses.

After around two days the blisters begin to rupture, to produce a red open wound and release highly infectious clear-yellow fluid (the weeping stage). The wound becomes covered with yellowish crusts in the scabbing stage, during which the skin beneath the scabs may be painful and itchy. This stage can last a further two to three days before the patient enters the healing stage. As the infection heals, scabs become progressively smaller and pink skin develops beneath the scabs to gradually assume the appearance of surrounding tissue. The healing stage may last up to one week.

The usual duration of an outbreak of cold sores is between seven to ten days, but sometimes may last up to two weeks.

Five stages of cold sore progression are:

1) Prodromal stage
2) Blister stage
3) Weeping stage
4) Scabbing stage
5) Healing stage

Initial infection with HSV-1 usually occurs in childhood from direct contact with an infected person; the initial infection can produce a variety of symptoms. In some cases, the primary infection may be asymptomatic, or it may produce a range of oral and systemic symptoms including oral lesions (gingivostomatitis), fever, and discomfort when eating or drinking.

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ORAL HERPES

TABLE 1: TRIGGERS OF HSV VIRUS ACTIVATION THAT MAY LEAD TO A RECURRENT HERPES LABIALIS INFECTION
• Sunburn or excessive exposure to UV light (including solaria)
• Lip trauma or abrasion
• Fever
• Menstrual periods
• Fatigue
• Stress – emotional or physical
• Immunosuppression
• Common cold infection or other infection
• Exposure to wind
• Dental treatment
• Travellers' diarrhoea
• Pregnancy

Assessment of condition

Recurrent cold sore infection (herpes labialis) is generally straightforward to identify. Classical presentation is of red blisters located on the lips, containing clear fluid which may have erupted and may be beginning to form crusts. The patient may be able to describe prodromal symptoms and sometimes identify a trigger factor.

Pharmacists should differentiate this condition from other lesions including impetigo, angular chelitis, acne, ringworm, mouth ulcers and other incidental lesions. Asking specific questions of the patient may assist this process, see Table 2.

TABLE 2: QUESTIONS TO ASK A PATIENT WHO PRESENTS WITH SYMPTOMS SUGGESTIVE OF ORAL HERPES
• **Describe the onset of the condition. When did the symptoms first appear?** Patients with cold sores may have symptoms prior to the emergence of the blisters. No warning symptoms occur with many other conditions. If the condition has been present for longer than 14 days medical assessment is recommended.
• **Where are the lesions located?** Cold sores typically occur around the mouth, especially where the lip meets regular skin (the vermilion border). Impetigo is much more likely to spread to other areas of the face or body. Angular chelitis occurs in the corners of the mouth.
• **What do you think may have triggered the condition?** Patients may be able to identify a trigger factor such as stress, ill health, injury or sunlight exposure.
• **Have you experienced these symptoms previously? How often do you experience these symptoms?** Patients with cold sores will generally have experienced these symptoms previously. However patients who experience oral herpes frequently, e.g. monthly, may benefit from a trial of products to prevent recurrence.
• **Are you pregnant or breastfeeding?** This may affect your choice of medicines to treat the condition.

ORAL HERPES

(REFERENCE: Oral viral infections and the therapeutic use of antiviral agents in dentistry)

Fig 2. Herpes labialis occurs on the mucocutaneous junction of the lip or on the skin adjacent to the nostril. These lesions are often preceded by a prickling sensation before blisters form, which then enlarge, coalesce, rupture and become crusted before healing.

Secondary herpes simplex lesions (Herpes labialis) develop in susceptible people most often at the mucocutaneous junction of the lip or on the skin adjacent to the nostril. The development of these lesions is often preceded by a prickling sensation before blisters form. These blisters then enlarge, coalesce, rupture and become crusted before healing (Fig 2). This usually takes 7-10 days in healthy individuals, but in immunocompromised patients' secondary herpetic lesions can be widespread, very slow to heal and refractory to treatment.

(REFERENCE: TG)

Management:

For a minor recurrence of oral mucocutaneous herpes, episodic antiviral therapy may reduce its duration; use:

- **aciclovir (adult and child older than 3 months) 5% cream topically, 5 times daily (every 4 hours while awake) for 5 days, started at the first sign of recurrence or during the prodromal stage**

OR

- **famciclovir (adult) 1500 mg orally, as a single dose, taken at the first sign of recurrence or during the prodromal stage.**

Management of recurrent oral mucocutaneous herpes differs for the following patients, so referral to a medical practitioner is required:

- **patients with severe recurrences of herpes (with systemic signs and symptoms, or if the patient has difficulty eating or swallowing)**
- **patients with generalised or chronic herpes infection (with crusted lesions and ulceration)**
- **immunocompromised patients**
- **patients with HIV**

ORAL HERPES

(REFERENCE: Treatment of herpes labialis (cold sores)

Complementary and alternative therapies

Some customers may wish to consider complementary or alternative therapies to relieve their condition. Options for these customers include:

- Ice – direct application of ice can relieve the burning, stinging and pain associated with herpes labialis infection.
- Propolis extract – sourced from bees, the product sponsor makes a claim that it reduces healing time and associated pain.
- Products containing menthol, camphor or melaleuca oil (which are, or can be, derived from plant sources) may be considered suitable by patients seeking "natural" products for the treatment of herpes labialis.

Use in pregnancy, breastfeeding and children

Pregnancy and breastfeeding risk categorisation, where available, is provided in Table 3. Categorisation information for many Listed products is unavailable, pharmacists should be cautious about recommending the use of such products in pregnancy, breastfeeding or children until adequate data are provided.

TABLE 3: **PREGNANCY AND BREASTFEEDING RISK CATEGORISATION**

PRODUCT	PREGNANCY CATEGORISATION	USE IN BREASTFEEDING	AGE RESTRICTIONS
Aciclovir 5% cream[14]	*B3*	No data available, although dose likely to be received by infant is considered negligible.[7] AMH states safe to use.[15]	3 months or older
Famciclovir 3×500mg tablets[9]	*B1*	No data available. Not recommended by product sponsor. AMH states safe to use.[15]	18 years and older
Idoxuridine, lignocaine, benzalkonium chloride[15]	*B1*	Product sponsor states product should not be used by breastfeeding women.	12 years and older
Penciclovir 1% cream[8]	*B1*	No data available. Product sponsor advises caution. AMH states safe to use.[15]	12 years and older
Povidone-iodine paint, ointment[15]	*Not categorised. Product sponsor advises caution.*	Product sponsor advises caution.	Appropriate to use in infants if large areas are not involved.
Propolis cream	*No information available.*	No information available.	No information available.

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ORAL HERPES

Lifestyle measures to assist management of herpes labialis

- The virus that causes cold sores (herpes simplex) is most infectious during the blister and crusting stages when large amounts of HSV is released by the body. To reduce the risk of transmission of the infection, people with active symptoms of oral herpes should avoid oral contact with others and sharing objects (cups, glasses, cutlery, toothbrushes, straws, lip balms) that have contact with saliva.
- They should also abstain from oral sex, to avoid transmitting herpes virus to the genitals of a sexual partner.
- To minimise incidence of cold sore episodes patients should avoid known trigger factors where appropriate, e.g. use sun protection to avoid sunburn, moisturisers to limit wind damage, minimise stress where possible, and discuss options to manage menstrual cycle with a doctor.
- To reduce the chance of secondary bacterial infection patients should avoid touching lesions with their hands or picking at scabs. Patients should wash hands before application of topical products.

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RECURRENT APHTHOUS ULCERATIVE DISEASE

(REFERENCE: The patient with recurrent oral ulceration)

Recurrent aphthous ulceration (RAU) is the most common form of recurrent oral ulceration, reportedly affecting up to 20% of the population. In most patients, the ulcers first appear in childhood or adolescence; there may be a slight female predisposition and, in some patients, there is a family history of similar ulceration which suggests a genetic factor. The aetiology of the condition is not completely understood but is thought to be immunologically based.

Recurrent aphthous ulceration can occur in three forms:

1) Minor recurrent aphthous ulceration

This is the most common form, accounting for approximately 80–90% of cases. The ulcers are usually round or oval and occur on the non-keratinized oral mucosa. Thus, they tend to occur on the lip and cheek mucosa and lateral margins of the tongue, sparing the dorsum of the tongue, palate and gingivae. In the buccal or labial sulcus, the ulcers may be linear (Fig 2). One to five ulcers usually occur at a time and they are approximately 5 mm in diameter. The ulcers heal without scarring after 1 to 2 weeks and then recur, usually at intervals of a few weeks or months, although some patients are rarely without ulcers.

Fig 2. Minor aphthous ulcers in the maxillary buccal sulcus. Note the erythematous margin.

RECURRENT APHTHOUS ULCERATIVE DISEASE

2) Major recurrent aphthous ulceration

This form is much less common and accounts for about 5–10% of cases. The ulcers are similar to those of minor recurrent aphthous ulceration, but occur on any part of the oral mucosa including keratinized regions such as the hard palate and dorsum of the tongue as well as the oropharynx and can be larger than 10 mm in diameter (Fig 3). One or two ulcers generally occur at any one time. They tend to be persistent, lasting for at least one month, heal with scarring, and then recur.

Fig 3. A major aphthous ulcer on the soft palate. The ulcer is large and irregular in shape.

3) Herpetiform ulceration

This has a similar prevalence to major RAU. This form of ulceration begins as small round ulcers, approximately 1 mm in diameter (Fig 4), which are present in large numbers (up to 100). These coalesce to produce larger ulcers with irregular margins. They usually occur on the non-keratinized mucosa, but any part of the oral mucosa may be affected. The ulcers can take up to two weeks to heal (without scarring) and later recur.

Fig 4. Herpetiform ulceration on the lower lip mucosa.

Some patients have ulceration which is intermediate between minor and major RAU, sometimes termed severe minor RAU. Although these ulcers commonly develop in childhood, some patients develop them later in life.

All forms of aphthous ulceration produce significant discomfort and patients with severe minor aphthae, major aphthae or herpetiform ulceration may have difficulty eating and talking.

RECURRENT APHTHOUS ULCERATIVE DISEASE

(REFERENCE: TG)

Recurrent aphthous ulcerative disease is the most common cause of nontraumatic ulcers of the oral mucosa. The disease has an immune-mediated pathogenesis and is characterised by the periodic eruption of painful ulceration of the oral 'mucosa.

Aphthous ulcers can occur acutely with smoking cessation, but these usually resolve with time, or can be triggered by trauma (e.g. toothbrushing, orthodontic appliances)

The ulcers usually occur on the mucosa of the cheek, lip and floor of the mouth, but can occasionally affect the mucosa of the gingivae and hard palate.

Three forms of aphthous ulcers are recognised:

Minor aphthous ulcers	Major aphthous ulceration	Herpetiform aphthous ulceration
• most common form • presents as smaller lesions (usually 2 to 4 mm in diameter) • occur a few at a time • heal within 7 to 10 days.	• less common form • presents as larger lesions (10 mm or more in diameter) • can persist for up to 6 weeks • (and occasionally months) • heal with submucosal scarring.	• rare • presents as recurrent crops of non-vesicular small ulcers (1 to • 2 mm in diameter) that coalesce to form larger ulcers • heal within 1 to 2 weeks • not caused by the herpes virus, so do not have a cluster pattern.

Photo 14. Minor aphthous ulcer of the mandibular labial mucosa

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RECURRENT APHTHOUS ULCERATIVE DISEASE

(REFERENCE: Recurrent aphthous ulcerative disease: presentation and management)

Recurrent aphthous ulceration (RAU) represents the second most common type of oral ulceration after traumatic ulceration. It is, however, the most common type of recurrent ulcerative disease in the mouth.

Definitions:

An ulcer is any breach of the oral mucosa causing exposure of the lamina propria. They may be classified on a number of bases and the most common are listed in Table 1. Aphthous ulceration is defined by the authors as a recurrent, non-infectious, non-vesicular and immunologically mediated oral mucosal disease.

Table 1. Common causes and examples of ulceration of the oral mucosa

Aetiology	Examples
Trauma	acute or chronic
Aphthous ulcerative disease	minor, major, herpetiform
Viral	herpetic gingivostomatitis, herpangina
Mucocutaneous/immunological	RAU, lichen, pemphigus, pemphigoid
Neoplasia	squamous cell carcinoma
Ischaemia	necrotizing sialometaplasia

Aetiology

The trigger of an episode of RAU is unknown, but extensive investigations in large patient series have identified a range of:

- **Local**
- **haematological**
- **gastrointestinal**
- **immunologic**
- **genetic**
- **nutritional**
- **allergic**
- **psychological**

medication reactions as probable triggers in some RAU patients.

However, in the majority of patients there is no consistent event coincident with an exacerbation. In the small percentage of patients where a systemic association can be made, e.g., inflammatory bowel disease, then the activity of RAU tends to reflect the activity of the associated condition. These conditions are listed in Table 2.

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RECURRENT APHTHOUS ULCERATIVE DISEASE

Table 2. Conditions related to the onset of RAU in some patients

Physical trauma	toothbrushing, orthodontic brackets
Chemical trauma	chemical burns
Medications	NSAIDS, cardioselective Beta$_1$-blockers
Psychological	personal/work-related stress
Nutritional	iron deficiency
	folate deficiency (related to GIT disease)
	vitamin B_1, B_2, B_6, B_{12}
Gastrointestinal disease	mal-absorption syndromes
	regional enteropathy (Crohn's disease)
	gluten sensitive enteropathy (Coeliac disease)
	ulcerative colitis
Endocrinological	premenstrual
Haematological	cyclic neutropenia
	anaemias
	haematological malignancies
Immunological	immunodeficiency states, HIV infection
Allergy/hypersensitivity	foods (tomatoes, chocolates, nuts, dairy, wheat)
	metals (nickle-based oral appliances)
Microbiological	streptococci, herpes viruses, Epstein Barr virus
Syndromal associations	Behcets Disease (multi-organ involvement including oral, genital and eye lesions)
	PFAPA/Marshall's Syndrome (periodic fever, aphthae, pharyngitis, cervical adenitis)
	MAGIC Syndrome (mouth and genital ulcers with inflamed cartilage/relapsing polychondritis)

Clinical Presentation

Clinically, patients and clinicians are often able to map the sequence of presentation through to resolution into the following stages:
(1) prodromal – symptoms but without any visible clinical sign
(2) pre-ulcerative – initial presentation, usually erythema and slight oedema
(3) ulcerative – formation of the epithelial defect
(4) healing – symptom abatement and progressive healing
(5) remission – no evidence of lesions.

The prodromal stage is infrequent and transient and heralds the early ingress of lymphocytes from the peripheral blood (recirculating lymphocyte pool).
This is the period when application of a topical corticosteroid may inhibit the further development of the lesion or at least minimize the severity and duration of any lesion that does progress to the ulcerative stage.

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RECURRENT APHTHOUS ULCERATIVE DISEASE

The pre-ulcerative stage is also transient and again is an ideal time to apply a topical corticosteroid. It represents the period when the local vascular response has allowed the clinical development of redness and swelling. Histologically, lymphocytes have now entered the basal epithelial layers and are initiating the cytotoxic process that will lead to epithelial cell death and progression to the ulcerative stage.

The ulcerative stage is the dominant stage and noted particularly by the patient due to local pain. Early epithelial destruction and breakthrough causes a small clinical ulcer which rapidly progresses to the full size determined for that particular lesion, most frequently 0.3–0.5cm in diameter. The lesion is umbilicated or crateriform with clear sharp raised margins and surrounding erythema and oedema. The lesion is generally round to oval and the depressed central zone carries a pseudomembrane that corresponds to a scab on a similar skin lesion. inflammatory component. The ulcerative stage lasts for three to seven days.

The healing stage is identified by the abrupt cessation of pain and the appearance of granulations within the decreasing surface exudate. Healing progresses by secondary intention with ingrowth of marginal epithelium and gradual centripetal closure of the defect.

The remission stage identifies the ulcer-free periods. This may be prolonged or short, regular or irregular, apparently spontaneous in progression to a pre-ulcerative stage or triggered by an identifiable and sometimes predictable event, for example some dietary items and occasionally in the premenstrual phase.

RECURRENT APHTHOUS ULCERATIVE DISEASE

(REFERENCE: ODELL)

TABLE 19.1 Features of Ulcers

Feature	Details
Site of ulcer	Recurrent aphthous stomatitis (RAS): Typically nonkeratinized mucosa. Erythema multiforme: Vermilion border of lip, buccal and labial mucosa. Traumatic ulceration: Usually recurs at the same site, often close to a sharp tooth. Crohn's disease: Typically affects buccal sulcus.
Size of ulcer	Minor RAS: 3–8 mm in diameter. Major RAS: greater than 1 cm in diameter (up to 3–4 cm). Herpetiform RAS: 0.2–3 mm in diameter.
Duration of each ulcer	Minor RAS: 7–10 days. Major RAS: May take weeks to months. Ulcers heal with scarring. Herpetiform RAS: 7–10 days. Erythema multiforme: Variable (10–21 days).
Number of ulcers	Minor RAS: Single ulcers or small crops of 2–5 ulcers Major RAS: One or two ulcers at a time. Herpetiform RAS: 30–100 ulcers at a time, which can coalesce such that it becomes difficult to count individual ulcers.
Frequency of attacks	RAS: Variable frequency; some patients may have ulcers continuously, whilst others experience ulcers just once per year. Erythema multiforme: Ulcers may recur at 6–8 week intervals in severe cases; other patients experience one-two attacks per year. Nutritional deficiencies/inflammatory conditions: Patients may have continuous ulceration.
Shape of ulcer	RAS: Usually round/oval and sharply defined; may become more irregular with healing. Herpetiform ulcers coalesce to form irregular shapes. Erythema multiforme: Irregular and ragged, merging with inflamed surrounding mucosa. Those on the lips are often covered by bloody fibrin sloughs.
Are the ulcers preceded by vesicles?	The presence of vesicles indicates possible viral infection or immunobullous disease. This fact may be helpful in the differential diagnosis of herpetiform ulcers, which resemble viral ulcers but are not preceded by vesicles.
Age of onset	RAS: Usually before or around adolescence. Erythema multiforme/inflammatory bowel disease: Typically second or third decade. Immunobullous disease: Typically, fourth decade onwards.
Family history	RAS: May have family history of ulceration. Coeliac disease/inflammatory bowel disease: May have positive family history. Erythema multiforme: Typically no family history. Traumatic ulceration: No family history.
Exacerbating or relieving factors	Ulcers can develop at a site of minor trauma in both RAS and immunobullous disease. Stress and menstruation can precipitate attacks of RAS. Erythema multiforme may be triggered by a drug, viral or other infection, although often no trigger is identified.

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RECURRENT APHTHOUS ULCERATIVE DISEASE

(REFERENCE: TG)

Assessment of aphthous ulcers involves taking a thorough history and examination; see also 'red flag' features of oral mucosal disease.

In children, investigate for systemic causes of nontraumatic ulceration. In adults who have additional symptoms, investigate for a systemic cause. Systemic causes of aphthous ulcers include:

- **iron, vitamin B12, folate or zinc deficiency**
- **coeliac disease**
- **ulcerative colitis**
- **Bechet syndrome**
- **PFAPA (periodic fever, aphthous stomatitis, pharyngitis, cervical adenitis) syndrome in children**

Deficiencies should be treated only on laboratory confirmation.

Management of recurrent aphthous ulcerative disease

If an aphthae-like ulcer occurs in a child, refer for further investigation because it could be a sign of systemic disease.

Topical corticosteroid treatment can produce rapid healing of minor aphthous ulcers, particularly if used in the prodromal or pre-ulcerative stage.

The aim is to treat the lesion rather than prevent further outbreaks; for adults, use:

- **hydrocortisone 1% cream or ointment topically to the lesions, 2 to 3 times daily after meals.**
- **If pain relief is required for minor aphthous ulcers in adults, apply a topical anaesthetic or analgesic to the ulcers, such as:**
  - **benzydamine 1% gel topically to the lesions, 2- to 3-hourly as necessary.**
- **Lidocaine viscous solution is an alternative topical anaesthetic for hospital settings (but be aware of the higher cost); for adults, use:**
  - **lidocaine 2% viscous solution, use the lowest dose necessary up to 15 mL, rinsed in the mouth for 30 seconds then spat out, 3-hourly as necessary; maximum 8 doses in 24 hours.**

Ulcers that are not improving after 2 weeks are potentially malignant—refer to a specialist for management and biopsy. Seek specialist advice for patients with major or herpetiform aphthous ulceration or immunocompromised patients with neutropenic ulceration.

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ORAL LICHEN PLANUS

(REFERENCE: Oral lichen planus: Causes, diagnosis and management)

Clinical features of oral lichen planus Oral lichen planus present as white striations (Fig 1), white papules, white plaques, erythema, erosions (Fig 2) or blisters affecting predominantly the buccal mucosa, tongue and gingivae, although other sites are occasionally involved.

Fig 1. Reticular pattern of fine white keratotic striae typical of oral lichen planus (OLP) on the posterior buccal mucosa. This baseline presentation is found in almost all OLP patients somewhere on the oral or gingival mucosae.

Fig 2. Linear erosive lesion on the ventral surface of the tongue. The surrounding mucosa is atrophic and erythematous with very faint reticular striae.

ORAL LICHEN PLANUS

- Oral lichen planus affects 1-2 per cent of the general adult population and is the most common non-infectious oral mucosal disease in patients referred to oral medicine and oral pathology clinics.
- Oral lichen planus affects women more than men (1.4:1). Oral lichen planus occurs predominantly in adults over 40, although younger adults and children may be affected.
- Lesions are typically bilateral and often appear as a mixture of clinical subtypes. White or grey streaks may form a linear or reticular pattern on an erythematous background.
- Alternatively, there may be a central area of shallow ulceration (erosion) with a yellowish surface (fibrinous exudate) surrounded by an area of erythema. Notwithstanding the multiple oral manifestations that form the basis of most current clinical classifications of OLP, the major issue is to arrive at a correct diagnosis.
- Almost all cases of OLP present with reticular keratotic striae in some area of the oral mucosa. Therefore, all cases of oral mucosal disease should be examined carefully for fine striae both peripherally around atrophic and/or erosive sites and on the buccal mucosa, ventral tongue, lateral tongue and gingivae (Fig 3).

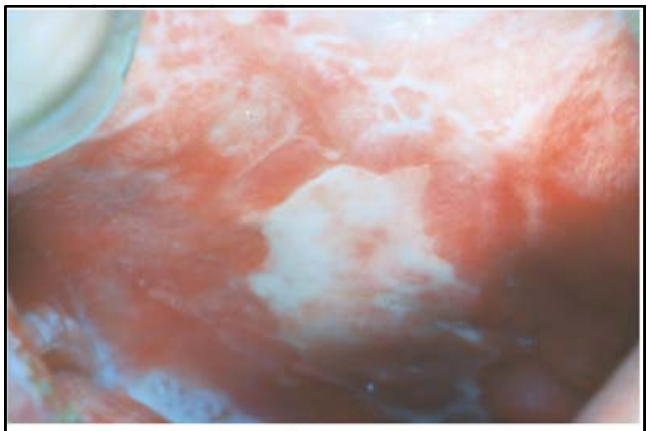

Fig 3. A more extensive area of erosion overlying a delicate atrophic mucosa. The typical striae are seen anterior to the erosion.

ORAL LICHEN PLANUS

- Gingival lesions frequently present as fiery red erythema affecting the entire width of the attached gingiva, a condition previously termed “desquamative gingivitis” (Fig 4).

Fig 4. Gingival OLP may present with the typical fine reticular striae or, as in this case, with generalized erythema and fragility. The striae can usually be seen as a very fine pattern on the tips of the interdental papillae. This pattern is frequently and incorrectly referred to as desquamative gingivitis and may be misdiagnosed as mucous membrane pemphigoid.

- As discussed previously, OLP lesions may be associated with patchy brown melanin deposits in the oral mucosa (inflammatory melanosis), although this is uncommon in fair-skinned people (Fig 5).

Fig 5. Oral lichen planus involving the lateral hard palate with typical reticular striae and secondary melanosis.

- The dorsal surface of the tongue also carries the striae in some patients, but a frequent alternative is an annular pattern in which the keratotic lines form circles of varying size (Fig 6).

Fig 6. Oral lichen planus lesions on the dorsum of the tongue showing interlacing striae and, in particular, the annular keratotic striae seen most frequently although not exclusively on the tongue.

ORAL LICHEN PLANUS

- General dental practitioners may reasonably examine a patient's wrists, scalp and nails. The detection of lesions at these sites may expedite appropriate referral and management.
- Approximately two thirds of OLP patients report oral discomfort. Most cases of symptomatic OLP are associated with atrophic (erythematous) or erosive (ulcerative) lesions. Symptoms vary from mucosal sensitivity to continuous debilitating pain.
- Oral lichen planus lesions usually persist for many years with periods of exacerbation and quiescence.
- During periods of exacerbation, there is increased erythema or ulceration with increased pain and sensitivity.
- During periods of quiescence, there is a decrease in the extent of erythema or ulceration with decreased pain and sensitivity. Patients are often unaware of quiescent OLP that presents typically as faint white striations, papules or plaques.
- Exacerbation of OLP has been linked to periods of psychological stress and anxiety, a predictable correlation with any condition that is related to an immune system imbalance.

Oral lichen planus and oral cancer

There is ongoing concern that OLP may be premalignant. Malignant transformation of OLP remains a very controversial issue. OLP patients are at increased risk of oral cancer. However, less than 5 per cent of OLP patients who do not use tobacco products develop oral SCC, most frequently in atrophic, erosive and plaque lesions. It is therefore unlikely that OLP is inherently premalignant.

The cause of increased oral cancer risk in OLP patients is unknown, although the oral mucosa affected by OLP may be compromised to the extent of being more sensitive to exogenous mutagens in tobacco, alcohol, betel quid and Candida albicans. Alternatively, the chronic inflammatory response and simultaneous epithelial wound healing response in OLP may increase the likelihood of cancer-forming gene mutations.

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ORAL LICHEN PLANUS

Diagnosis of oral lichen planus

- **Biopsy** is required to differentiate between OLP and other chronic white or ulcerative oral lesions including reactive keratoses, chronic hyperplastic candidiasis, epithelial dysplasia, discoid lupus erythematosus, gastro-intestinal disease (including oral Crohn's disease) or anaemic states. Malignancy must also be excluded.
- **Direct immunofluorescence** can help distinguish erosive, ulcerative or the very rare bullous form of OLP from pemphigus vulgaris, benign mucous membrane pemphigoid, dermatitis herpetiformis and linear IgA bullous dermatosis.
- There are no consistent **serological changes** associated with OLP but some patients do present an elevated ANA titre.
- **Blood biochemistry and FBE** should also be included in the full patient workup.
- Although cytological changes may be detected in OLP, the use of **exfoliative cytology** is not recommended.

Management of oral lichen planus patients

- Many **systemic drugs may trigger** oral LDRs and therefore clinicians should be suspicious of any systemic drug in patients with oral lichenoid lesions. Suspected oral LDRs should be managed, in part, by the prescription of alternative drugs by the patient's physician.
- Oral lichenoid **contact sensitivity reactions** may be triggered by many contact allergens. Dental restorations and prostheses associated with oral lesions should be replaced and cinnamate-flavoured toothpaste should be discontinued if contact sensitivity is suspected. Skin patch testing may help to identify contact allergens in some OLP patients. The current recommendation is to use a standard series, a dental prosthesis series and a metal salt series including gold, mercury and palladium salts as well as other salts of metals used in dental restorations.
- Oral lichenoid lesions may be **triggered by mechanical trauma** (Koebner phenomenon) from calculus deposits, sharp teeth, rough surfaces of dental restorations or prostheses, cheek or tongue biting and oral surgical procedures.
- Some studies have shown an increased **incidence of C. albicans** infection in OLP. As discussed, OLP is associated with an increased risk of oral cancer and chronic oral C. albicans infection is recognized as an oral cancer risk factor. Hence, candidal culture or smear should be undertaken periodically and C. albicans superinfection should be controlled with topical polyene or azole antimycotics.

ORAL LICHEN PLANUS

Management of oral lichen planus patients

- Oral lichenoid lesions may arise in habitual chewers of betel quid. Betel quid chewing is also recognized as an oral cancer risk factor and patients should be advised to eliminate the habit.
- Some studies have shown an association between OLP, hepatitis C virus (HCV) infection, chronic active hepatitis and primary biliary cirrhosis. A causal role for HCV infection in OLP has not been identified although liver function tests should be considered in all OLP patients.
- Exacerbation of OLP has been linked to periods of psychological stress and anxiety. Psychological assessment may be beneficial in some cases.
- Oral lichenoid lesions are seen within the spectrum of chronic graft-versus-host disease following allogeneic bone marrow transplantation. These lesions are usually effectively controlled with systemic immunosuppressive therapy.

Medical treatment of oral lichen planus

- The aims of current OLP therapy are to eliminate mucosal erythema and ulceration, alleviate symptoms and reduce the risk of oral cancer in OLP patients. In this context, medical treatment is restricted currently to atrophic (erythematous), erosive (ulcerated), bullous (blistering) or symptomatic OLP lesions.
- Corticosteroids are the mainstay of OLP therapy because of their activity in dampening cell mediated immune activity and are administered topically, intra-lesionally or systemically. The combination of systemic and topical steroid therapy is often very effective.
- Localized oral lesions are treated with topical ointment, applied two to four times daily after meals. The strength and specific preparations used need to be balanced carefully with the individual patient's needs.
- Generalized oral lesions are often treated effectively with a steroid mouth rinse twice daily after meals.
- C. albicans superinfection, which may accompany any immunosuppressive therapy, should be controlled with topical antimycotics especially in risk groups such as xerostomics. Some patients should be managed prophylactically to prevent secondary candidal overgrowth and a careful examination should be undertaken in denture wearers to ensure they do not have a pre-existing atrophic candidiasis.
- Intralesional and perilesional injection of steroids is useful for persistent localized erosive OLP lesions but should be used with due caution. Most protocols using topical and intralesional corticosteroids cause some adrenal suppression and clinicians need to be aware of the precise amounts of these medications being used on a regular or irregular basis.

ORAL LICHEN PLANUS

Medical treatment of oral lichen planus

- Generalized atrophic or erosive oral lesions that do not respond to topical therapy may be treated with a short course of systemic corticosteroids.
- Hypersensitivity, hypertension, viral infection, tuberculosis, diabetes mellitus, pregnancy, stomach ulcers or a family history of early osteoporosis may contra-indicate the use of corticosteroid preparations.
- Treatment of OLP with cyclosporin, azathioprine, levamisole, griseofulvin, retinoids, hydroxychloroquine sulphate, dapsone and psoralen/UVA has been reported.
- The main concerns with these and other current therapies are local and systemic side effects and lesion recurrence following withdrawal of treatment.
- Oral lichen planus patients should be reviewed every month during active treatment and lesions monitored for reduction in mucosal erythema and ulceration and alleviation of symptoms.
- Active treatment should be continued, and alternative therapies tried until erythema, ulceration and symptoms are controlled. The elimination of mucosal erythema and ulceration leaving residual asymptomatic reticular or papular lesions may be considered an endpoint of current OLP therapy.
- Thereafter, due to the chronicity of OLP and the increased risk of oral cancer, OLP patients should be reviewed at least six-monthly and lesions re-biopsied as indicated by their clinical presentation and previous histological findings.
- Oral lichen planus patients should be advised to attend whenever there is an exacerbation of symptoms or a change in lesion presentation.

ORAL LICHEN PLANUS

Decreasing the risk of oral cancer in oral lichen planus patients

- As discussed, OLP patients are at increased risk of oral cancer. Patients with OLP should be advised to eliminate smoking and alcohol consumption.
- Most oral cancers arising in OLP patients are associated with erosive, atrophic and plaque lesions. Erosive and atrophic lesions can be converted into reticular lesions using topical steroids. Hence, treatment of erosive and atrophic lesions with topical steroids may reduce the risk of oral cancer.
- Oral lichen planus patients should be advised that a nutritious diet including fresh fruit and vegetables may help reduce the risk of oral cancer.
- Due to the potential role of C. albicans in the development of oral SCC, fungal superinfection should be eliminated with topical antimycotics.
- Oral lichen planus patients should attend whenever there is an exacerbation of symptoms or a change in lesion presentation. Such changes most often indicate a phase of increased disease activity, although neoplasia must be excluded.
- Oral lichen planus patients should be reviewed at least six-monthly and lesions re-biopsied as required.
- Although OLP may in many cases be diagnosed clinically, appropriate specialist referral is required for:
  - histological diagnosis
  - assessment of causative/exacerbating factors, associated diseases and oral cancer risk
  - patient education and management
  - medical treatment
  - long-term review and re-biopsy as required.

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ORAL LICHEN PLANUS

Table 1. Oral lichen planus (OLP) patient information

1. Oral lichen planus (OLP) is a mouth rash of unknown cause
2. OLP is not contagious and not inherited
3. OLP presents as white lines or spots, redness or ulceration, often in combination
4. OLP may be painless or cause mucosal sensitivity or pain
5. OLP may be more severe at times of psychological stress
6. OLP may eventually disappear, although this can take many years
7. The skin, nails and scalp may be affected
8. Lesions similar to OLP may be caused or aggravated by medicines, tooth filling materials or toothpastes
9. A biopsy is usually necessary to confirm the diagnosis
10. The aims of OLP treatment are to eliminate mucosal redness, ulceration, pain and sensitivity
11. OLP is usually effectively treated with steroids, although responses vary and several treatments may need to be trialled
12. The teeth should be scaled and patients instructed in thorough oral hygiene
13. It may be necessary to have teeth, fillings and dentures smoothed and polished
14. OLP patients are at increased risk of oral cancer
15. Oral cancer develops in less than 5 per cent of OLP patients who do not use tobacco products
16. OLP patients should eliminate smoking and alcohol consumption
17. OLP patients should eat a nutritious diet including fresh fruit and vegetables
18. OLP patients are reviewed at regular intervals and lesions may occasionally require re-biopsy
19. OLP patients should attend whenever there is a change in the look or feel of lesions
20. On-line OLP patient information, including a web-based OLP chat group, is available currently at URL: 'http://www.tambcd.edu/lichen/'. Accessed January 2002.

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ORAL LICHEN PLANUS

(REFERENCE: TG)

Lichen planus is an uncommon idiopathic immune-mediated condition that can affect the skin, hair, nails, and oral and genital mucosae.

Oral lichen planus typically occurs on the buccal mucosa, tongue and gingivae. In the nonerosive form of the disease, the lesions consist of a characteristic reticular pattern of white striations or plaques.

Photo 8. Oral lichen planus of the left buccal mucosa showing characteristic white striations

Erosive oral lichen planus presents as erythematous, ulcerated or eroded areas of mucosa, which are often painful. Symptoms include stinging or burning, especially with spicy or acidic food.

Oral lichen planus is associated with an increased risk of oral squamous cell carcinoma.

Refer patients with suspected oral lichen planus to a specialist for biopsy, definitive diagnosis and management.
Differential diagnosis should exclude oral lichenoid lesions.
If lichen planus occurs on the gingival tissues, management includes improving oral hygiene and periodontal health.
Patients with oral lichen planus require ongoing review by an oral medicine specialist because of the chronic nature of the condition and the potential for malignant transformation.

Biopsy-proven oral lichen planus becomes symptomatic, treat with:

- betamethasone dipropionate 0.05% cream or ointment topically to the lesions, twice daily after meals, until symptoms resolve.
- Advise patients to stop using topical corticosteroids once symptoms have resolved.
- If the patient's symptoms have not improved after 3 weeks of topical corticosteroids, the symptoms change or the appearance or texture of the lesion changes, advise patients to return to their treating specialist.

ORAL LICHEN PLANUS

(REFERENCE: Oral mucosal diseases: the inflammatory dermatoses M Schifter* S-C Yeoh Coleman* A Georgiou*)

There are six recognized oral presentations of lichen planus:

- reticular
- papular
- plaque-form
- atrophic
- ulcerative(erosive) and rare
- bullous form.

These latter three forms can be associated with significant discomfort requiring either topical and/or systemic immunosuppressive therapy.

The cause(s) of the various oral lichenoid lesions, ranging from idiopathic oral lichen planus (OLP) to the "contact" lesion, is not understood, but all the lesions are characterized histologically by a typical "lichenoid tissue reaction" featuring a band-like lymphohistiocytic infiltrate within the lamina propria and liquefaction degeneration of the basal keratinocytes. These reactions may be the result of several diverse possible triggers, but all culminate in a common pathologic process, that of T-lymphocyte directed, immune-mediated, damage to the oral epithelial basal cells.

OLP most frequently presents in women, by a ratio of approximately 3:1 to 3:2 compared with men, aged 40 years and above.

Mucosal lesions are usually multiple and almost always have a bilateral, symmetrical distribution. They commonly take the form of minute white papules that gradually enlarge and coalesce to form either a reticular, annular, or plaque-like pattern. A characteristic feature is the presence of slender white lines (Wickham's striae) radiating from the papules. In the reticular form, there is a lace-like network of slightly raised white lines, often interspersed with papules or rings. The plaque-like form may be difficult to distinguish from leucoplakia.

Involvement of the gingivae is described clinically as desquamative gingivitis, but is not unique to OLP and may feature in the presentation of other oral dermatoses, especially pemphigoid and pemphigus.

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ORAL LICHEN PLANUS

(REFERENCE: ODELL)

Lichen Planus.

This condition is the commonest of the three possible diagnoses and most prevalent in women in their 5th and 6th decades of life. The striae and lacy patterns on the buccal mucosa are distinctive of lichen planus and are typically bilateral. Lichen planus can also cause white patches on the lip and palate as in this patient, although this is less common.

Subject	Questions and Reasons
Rash	Is a rash present? The patient should be questioned about any skin, nail or scalp problems and about any genital symptoms. Ask about and examine the flexor surface of the wrist and the extensor surface of the shins. These are common sites for skin lesions of lichen planus and lichenoid reactions. The typical skin lesions of lichen planus are purplish polygonal papules with faint striae (Wickham's striae). They are usually very itchy. Severe lichenoid reactions may be accompanied by an extensive erythematous rash. Only a minority of cases with oral lichen planus or lichenoid reaction will have skin lesions on presentation. This is because the skin lesions often resolve spontaneously after a few years or with topical steroid treatment. In contrast, oral lesions may persist for many years and are often resistant to treatment. Therefore, ask about previous rashes. The skin lesions of SLE are different from those of lichen planus and may be of several different types, including 'discoid' rashes, erythema nodosum and other more nonspecific papular, erythematous and blistering rashes. Rashes in DLE are often confined to the face, neck and scalp and present as well-demarcated, round or oval, red, scaly patches (discoid rash). The dorsum of the hands may also be affected, with sparing of the knuckles. Typically the face is affected by a butterfly rash on the malar region of the face. Rashes elsewhere on the body tend to be photosensitive so are worse on sun-exposed areas.

Treatment Option	Indications and Use
Analgesic relief	Topical benzydamine (a nonsteroidal anti-inflammatory agent) provides temporary analgesia to the oral mucosa. It can also be useful prior to toothbrushing to help maintain good oral hygiene.
Topical medium-potency corticosteroids	These can be prescribed in the form of mouthwashes, sprays, ointments or in the form of aerosol inhalers used for asthma. This option would be the mainstay treatment for most patients with mild to moderate symptoms. Options include beclomethasone inhaler and betamethasone mouthwash.
Topical high-potency corticosteroids	Topical high-potency drugs such as clobetasol ointment should be considered for more severe or generalized conditions. In general, this option should be provided in a secondary or tertiary care setting.
Systemic corticosteroids and disease-modifying agents	Systemic corticosteroids (e.g. prednisolone) and disease-modifying agents (e.g. hydroxychloroquine, azathioprine and mycophenolate mofetil) are used for severe cases that do not respond to topical treatments and should only be used in a secondary or tertiary care setting. Regular monitoring for the adverse effects of these drugs is required.

ORAL LICHEN PLANUS

(REFERENCE: Oral lichen planus: Causes, diagnosis and management)

Oral lichenoid lesion

Oral mucosal lichenoid lesions may follow the administration of a systemic drug, with a variable lag period.
These lichenoid drug reactions (LDR) may be unilateral but usually appear as idiopathic OLP.
Drugs that have been implicated in oral LDR include non-steroidal anti-inflammatory drugs, angiotensin-converting enzyme inhibitors and beta-blockers, although there are many others (Fig 7).

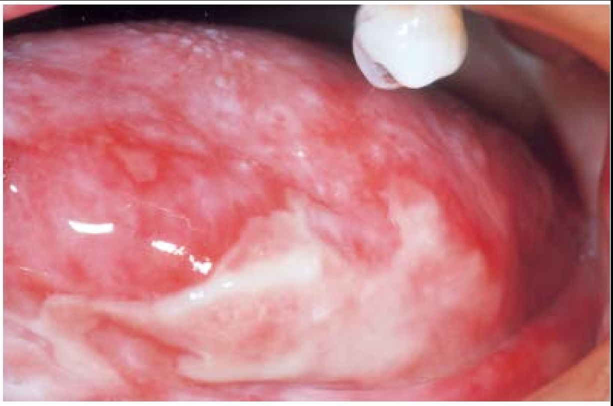

Fig 7. Extensive erosive OLP in a patient on long-term anti-inflammatory medications. In this case the NSAIDS exacerbated a previously existing OLP by introducing the erosive component that had not previously been problematic. Withdrawal of the agent gave rapid resolution to the erosion although the original OLP remained.

Oral mucosal lichenoid lesions may follow the placement of a dental restoration or provision of a denture, again with a variable lag period. These lichenoid reactions are usually the result of a contact sensitivity or irritant contact response to an amalgam or composite resin dental restoration or a denture component in close proximity to the oral mucosa (Fig 8, 9).

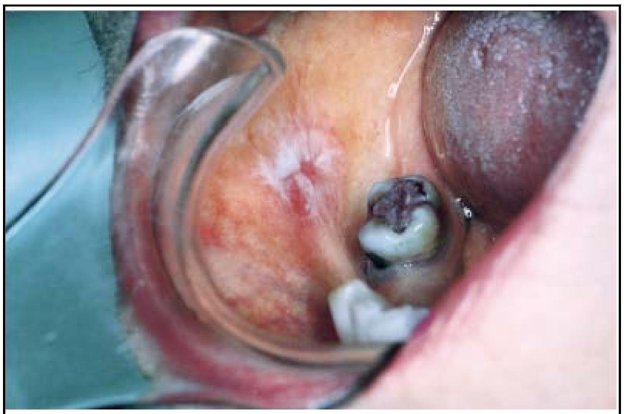

Fig 8. Lichenoid reaction to the amalgam restoration on the buccal aspect of the molar tooth. This is an isolated response without the symmetrical distribution seen in typical OLP.

ORAL LICHEN PLANUS

(REFERENCE: Oral lichen planus: Causes, diagnosis and management)

Oral lichenoid lesion

Fig 9. Lichenoid reaction on the inner aspect of the lip to a composite resin restoration in the adjacent incisor tooth. Replacement of the restoration gave full resolution to the lesion.

Toothpaste flavourings, especially cinnamates, may also trigger lichenoid contact sensitivity reactions.

Oral mucosal lichenoid lesions are also seen within the spectrum of chronic graft-versus-host disease following allogeneic bone marrow transplantation.
In many cases, a cause for the oral lichenoid lesions cannot be identified and the diagnosis by exclusion is "idiopathic OLP".

(REFERENCE: TG)

Lichenoid mucosal reactions can be caused by:

- **contact hypersensitivity to dental restorations**
- **hypersensitivity reactions to drugs, particularly:**
  - **i. Drugs that lower blood pressure (e.g. beta blockers, angiotensininhibitors, diuretics [particularly hydrochlorothiazide)**
  - **ii. nonsteroidal anti-inflammatory drugs (NSAIDs)**
  - **iii. drugs that treat thyroid disorders**
- **medical conditions:**
  - **i. hepatitis C infection, particularly in patients with the human leukocyte antigen HLA-DR6 allele (which is common in people of Mediterranean descent)**
  - **ii. thyroid disorders**
  - **iii. chronic graft-versus-host disease.**

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ORAL LICHEN PLANUS

Photo 9. Oral lichenoid lesion due to contact hypersensitivity to an amalgam filling

Refer patients with a suspected oral lichenoid lesion to an appropriate specialist for definitive diagnosis and management.
In the case of contact hypersensitivity to an amalgam filling, replacement of the implicated amalgam filling may result in partial or full resolution of the lesion. However, removal of all amalgam fillings is not recommended.

(REFERENCE: ODELL)

Lichenoid Reaction

Oral lichenoid reactions may be localized (in response to topical triggers such as restorations) or more widespread, in which case they are usually caused by medication. Some features that point to a lichenoid drug reaction rather than lichen planus include acute onset, extensive ulceration, asymmetrical distribution and severe involvement of the dorsum of the tongue. Lesions may also affect sites that are less commonly affected by lichen planus such as the floor of the mouth. Lichenoid reactions may be clinically indistinguishable from lichen planus. In this case, a lichenoid drug reaction can probably be ruled out because the patient is not on any medication, although double-checking recently discontinued prescriptions and over-the-counter drugs is necessary to be certain. The location and distribution of the lesion do not correspond to any dental restorations so a topical lichenoid reaction can be ruled out.

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

(Sources: Oral cancer Article-ADJ, Oral cancer awareness for general practitioners- ADJ, A chronic oral frictional keratosis (American Dental Association), Features of reactive white lesions of oral mucosa)

White lesions

- **White lesion on the oral cavity is quite common and have a variety of etiologies, (benign, potentially malignant, malignant).**
- **Most oral lesion that appears white are benign.**

Table 3. LESIONS THAT MAY APPEAR WHITE

Developmental
Cannon white sponge nevus
Hereditary benign intraepithelial dyskeratosis
Dyskeratosis congenita
Pachyonychia congenita
Other oral lesions in genodermatoses associated with dyskeratosis
Inflammatory/reactive
Frictional/factitial
Morsicatio mucosae oris
Benign alveolar ridge keratosis
Mouth-wash induced desquamation
Infectious
Oral viral (hairy) leukoplakia
Candidiasis
Immune-mediated
Lichen planus/lichenoid stomatitis
Chronic graft-versus-host disease
Tobacco-associated
Smokeless tobacco keratosis
Nicotinic stomatitis
Autoimmune
Lupus erythematosus
Others
Nondysplastic leukoplakia
Premalignant and malignant
Dysplastic leukoplakia
Verrucous leukoplakia
Proliferative leukoplakia
Squamous cell carcinoma

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

(Sources: Oral cancer Article-ADJ, Oral cancer awareness for general practitioners- ADJ, A chronic oral frictional keratosis (American Dental Association), Features of reactive white lesions of oral mucosa)

A) Benign White lesions

- **The reactive white oral lesions include frictional keratoses, irritant contact dermatitis, smokeless tobacco keratoses,**

1) Frictional Keratosis:

- **White patch which is caused due to chronic and low-grade parafunctional habit (like chewing, constant rubbing, or sucking of oral mucosa against the teeth)**
- **The clinical appearance of a frictional keratotic lesion varies depending on the degree of trauma.**
- **One variation is Linea Alba which is used to describe a white keratotic line on the buccal mucosa approximating the occlusal plane. It can be unilateral or bilateral and varies in color intensity and thickness.**

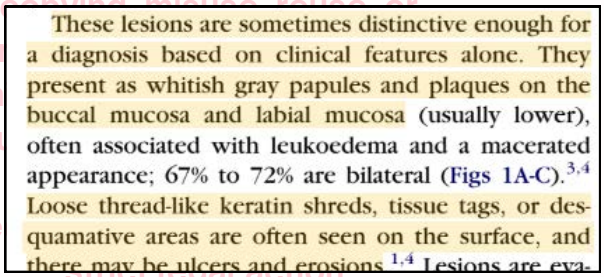
Clinical Appearance of frictional keratotic lesions:

- **Surface is rough with irregular tags. (pts. Trying to remove tags results in more tags formation).**
- **Can be extensive involving entire cheek and even lips.**
- **Clinically, an ill-defined area of gray or white papules and plaques is seen.**
- **If the bite trauma is extensive, it may be associated with erosions and ulcers.**
- **Macerated appearance with shredding and peeling may be seen.**

Fig: Irregular, shaggy macerated appearance typical for cheek biting (morsicatio mucosae)

Morsicatio Mucosae Oris:

- **It is a form of chronic oral frictional keratosis that has no malignant potential**
- **There is diffuse, poorly demarcated, peeling threading appearance. However, may sometimes appear as distinct, well demarcated plaques.**



These lesions are sometimes distinctive enough for a diagnosis based on clinical features alone. They present as whitish gray papules and plaques on the buccal mucosa and labial mucosa (usually lower), often associated with leukoedema and a macerated appearance; 67% to 72% are bilateral (Figs 1A-C).[3,4] Loose thread-like keratin shreds, tissue tags, or desquamative areas are often seen on the surface, and there may be ulcers and erosions.[1,4] Lesions are eva-

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

Morsicatio Mucosae Oris:

- **MMO** is a common chronic mucosal frictional keratosis characterized by poorly demarcated, rough, shaggy, peeling, white plaquesand papules on the buccal mucosa, lateral border of the tongue or the lower labial mucosa easily traumatized by the teeth.
- **Chronic frictional** injury of gingiva or alveolar ridge mucosa, especially of the retromolar pad, presents as benign alveolar ridge keratosis (BARK) with different histological features, namely, hyper orthokeratosis and acanthosis with slight papillomatosis.

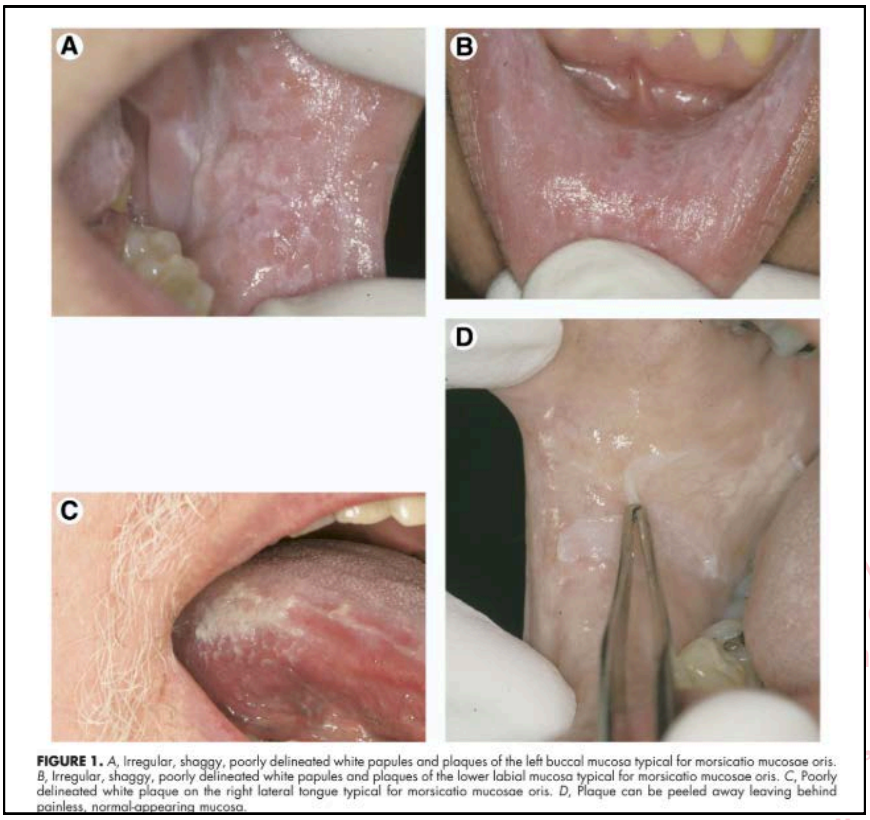


FIGURE 1. *A*, Irregular, shaggy, poorly delineated white papules and plaques of the left buccal mucosa typical for morsicatio mucosae oris. *B*, Irregular, shaggy, poorly delineated white papules and plaques of the lower labial mucosa typical for morsicatio mucosae oris. *C*, Poorly delineated white plaque on the right lateral tongue typical for morsicatio mucosae oris. *D*, Plaque can be peeled away leaving behind painless, normal-appearing mucosa.

Diagnosis:

- Generally, the clinical findings are adequate in determining the etiology of white lesions and confirmatory biopsy is not needed.
- In instances where **etiology is unknown** OR **keratotic lesion is in high-risk area**, then it is prudent to sample any questionable lesion to rule out Oral Potentially Malignant Disorder (OPMD).

For example:

> lesion is in a high-risk area for OPMDs. This is particularly true when the lesion presents on the lateral border of the tongue, which is the most common location for oral cavity squamous cell carcinoma [7]. Therefore, it is prudent to sample any questionable lesion to rule out OPMD.

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

Diagnosis:

- **Care should be made in diagnosis of keratosis of alveolar ridge and most importantly the gingiva of tooth bearing areas where limited clinical information is available.**

ited clinical information is available. Toothbrush trauma is often cited as the etiology of keratotic lesions of the gingiva. However, most traumatized gingiva of the tooth bearing area expresses as erythema, ulceration, or other reactive lesions such as pyogenic granuloma. The clinical findings are critical in helping to distinguish between reactive keratosis and OPMD. There is both clinical and histologic overlap in the

- ***If etiology is present provisional diagnosis cannot be leukoplakia.**

Histological features of frictional keratosis:

- **The histological features of Frictional keratoses from tongue, lip or buccal mucosa can vary depending upon the site of biopsy.**
- **But marked Hyper parakeratosis with shaggy or shredded keratin surface is noted.**
- **Bacteria is usually present on the keratin surfaces in biopsies from tongue but not as often as on buccal mucosa or lip. (Most bacteria seen in BM and lip)**

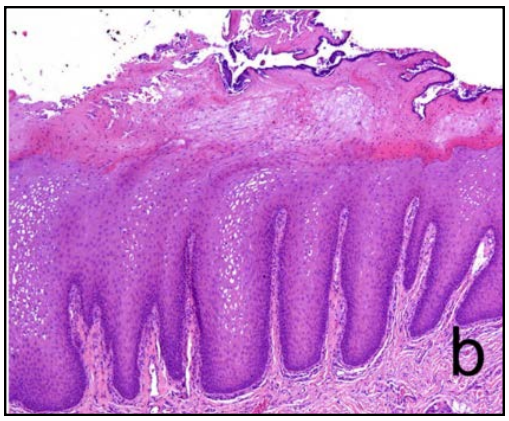


Fig: Marked hyperkeratosis with shaggy appearance with surface fissures and clefts

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ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

Conditions that have overlapping features with Frictional keratosis:

a) Proliferative Verrucous Leukoplakia (PVL)

- The clinical and histological features of benign keratosis and keratosis associated with proliferative verrucous leukoplakia (PVL) overlap.The latter is an OMPD.
- Proliferative Verrucous leukoplakia (PVL) is of unknown etiology and is associated with high recurrence and malignant transformation.

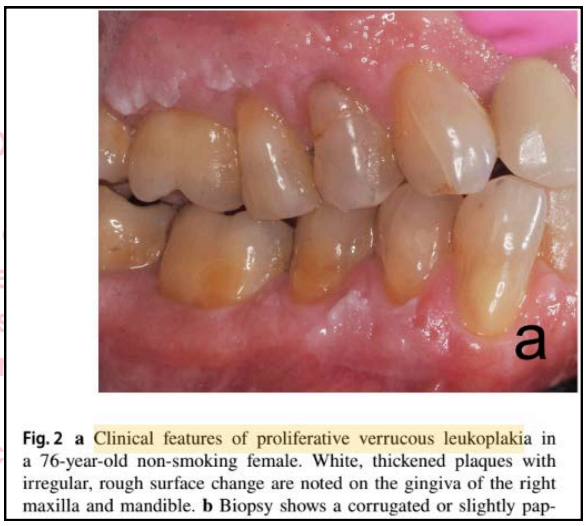

Fig. 2 a Clinical features of proliferative verrucous leukoplakia in a 76-year-old non-smoking female. White, thickened plaques with irregular, rough surface change are noted on the gingiva of the right maxilla and mandible. b Biopsy shows a corrugated or slightly pap-

- The gingiva is the most common site for PVL, and also the most common site for malignant transformation.

b) Leukoedema

- Leukoedema is a common, asymptomatic buccal mucosal finding of unknown etiology.
- It is considered as a normal variation of oral mucosa.
- It affects bilateral labial and buccal mucosa and appears as an opalescent, film gray to white lesion that characteristically diminishes upon stretching of the mucosa.
- This feature distinguishes leukoedema from frictional keratosis, lichen planus and leukoplakia.

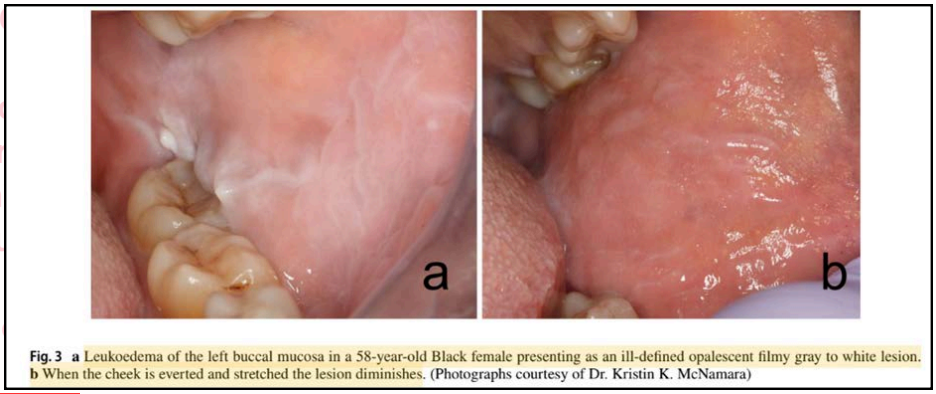

Fig. 3 a Leukoedema of the left buccal mucosa in a 58-year-old Black female presenting as an ill-defined opalescent filmy gray to white lesion. b When the cheek is everted and stretched the lesion diminishes. (Photographs courtesy of Dr. Kristin K. McNamara)

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

Conditions that have overlapping features with Frictional keratosis:

c) White Sponge nevus (WSN)

- It is a rare geno-dermatoses that can have clinical overlap with frictional keratosis but are histologically distinct.
- It is inherited as an autosomal dominant trait.
- It presents as asymptomatic thickened soft white plaques most commonly on the buccal mucosa. Other mucosal sites involved are nasal, esophageal and anogenital.
- Generally first noted on childhood, the lesions wax and wane over time.
- Histologically they are distinct with prominent parakeratosis and acanthosis and clearing of the spinous cell layer.

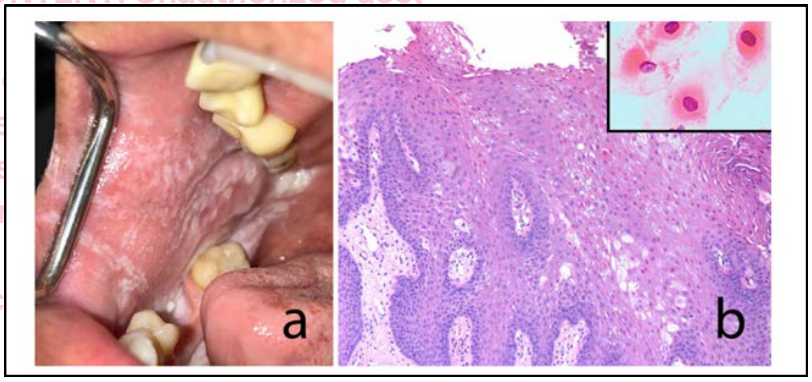

Fig. 4 **a** White sponge nevus of the right buccal mucosa in a 36-year-old Black man. Although there are clinical similarities to frictional keratoses the histology is distinct. **b** Photomicrograph of white sponge nevus exhibiting prominent hyperparakeratosis and acanthosis with vacuolation of the spinous cell layer. Within the spinous layer

d) Hereditary Benign Intraepithelial Dyskeratosis (HBID):

- It is a rare autosomal dominant disorder initially described in tri-racial Native tribe in North Carolina.
- Similar to White spongy nevus, it presents as white spongy plaques in buccal mucosa and tongue. But in addition, HBID has ocular findings of white gelatinous conjunctival plaques.
- These plaques are movable over the underlying tissue.
- Epi-bulbar blood vessels cane be hyperemic resulting in bloodshot appearance.
- HBID does not affect the nasal, esophageal and anogenital mucosa.

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ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

2) Irritant Contact-Related Keratosis

- Irritant contact stomatitis which is caused by chemical products used in toothpastes, mouthwashes, and dental restoration can result in oral mucosal injury.
- Much of the time the oral mucosa is in contact with these products for a short period of time or saliva dilutes and buffers the irritants reducing the potential for an adverse reaction.
- However, with increased duration, frequency and concentration of the chemical the patient may have a reaction and develop keratosis, ulceration, erythema, vesicles, edema or combination of these.
- Although the clinical presentation of irritant contact stomatitis share similarities with allergic contact stomatitis, patch testing is negative.
- Three contact related lesions that can present as white and keratotic oral lesions which have a unique histology are:

a) Contact to some ingredients in toothpaste:

- In some individuals' certain dentifrices can result in superficial sloughing of the oral mucosa.
- The buccal mucosa and vestibule is usually affected and the appearance is of white strings easily removed with a finger without leaving any ulceration or erythema.

out leaving any ulceration or erythema [21–25]. Ingredients associated with superficial mucosal desquamation are sodium lauryl sulfate (SLS), triclosan and tetrasodium and/or tetrapotassium pyrophosphate [21, 22]. SLS is a common synthetic detergent added to toothpaste for foaming and cleaning. Both triclosan, an antimicrobial agent, and sodium pyrophosphate are added to toothpaste either as a single ingredient or combined in tartar-control toothpaste to prevent plaque development. In addition to these ingredients, flavoring agents are added to mask the bitter taste of pyrophosphate. The exact prevalence is unknown but most likely these reactions are uncommon. Clinicians may be concerned

Fig. 6 **a** Superficial sloughing of the oral mucosa due to the use of triclosan and pyrophosphate containing toothpaste. There is peeling of the superficial keratin without any underlying erythema or erosion. Total resolution of the condition was achieved upon discontinuation of the tartar control toothpaste. **b** Photomicrograph demonstrates

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

2) Irritant Contact-Related Keratosis

b) Amalgam

- Oral contact lesions to amalgam restorations can occur as a keratotic or lichenoid reaction.
- Amalgam reactions are generally considered as Type IV hypersensitivity reactions.
- The lesions usually occur in buccal mucosa or tongue where prolonged contact with amalgam occurs.
- Amalgam contact reactions have clinical overlap with oral lichen planus, but contact reactions are usually single and resolve upon amalgam removal.

Fig. 7 **1a** Oral lichenoid contact reaction to dental amalgam presenting as areas of erythema and white plaques on the left buccal mucosa. Note the large amalgam restorations that directly contacts the affected mucosa. **1b** Oral lichenoid contact reaction to dental amalgam often

c) Cinnamon flavoring agents

- These include cinnamic aldehyde, cinnamic acid and cinnamon oil and can cause contact stomatitis.
- Cinnamon is used in many products such as toothpaste, mouthwashes, gum, candy and soft drinks.
- Most cases of cinnamon contact stomatitis is associated with prolonged contact of the offending agent.
- Clinical presentation can vary.
- The sloughing of mucosa with edema and erythema of gingiva like above is associated with cinnamon contact stomatitis by toothpaste.
- Shaggy hyperkeratotic oral lesions are usually seen with cinnamon contact stomatitis with gum and candy.

magnification ×100). **2a** Oral lichenoid contact reaction of the right buccal mucosa to cinnamon flavored chewing gum. Within 10 days of discontinuing the gum, the lesion completely resolved.

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ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

3) Smokeless Tobacco Keratosis:

- It is the keratotic changes in the oral mucosa where smokeless tobacco product is placed.
- AKA snuff dippers lesion, snuff pouch and spit tobacco keratosis.
- Early lesions tend to have a film white to gray opalescent appearance with a wrinkled surface and minimal mucosal thickening.
- With progression the lesion becomes more keratotic with furrowing and thickening of epithelium.

Fig. 8 **a** Typical clinical presentation of an early smokeless tobacco keratosis demonstrating an area of superficial keratosis with slight wrinkling, lacking any appreciative mucosal thickening. **b** A more advanced lesion demonstrates obvious mucosal thickening and wrinkling of the mucosa with intervening furrows. Flecks of smokeless

B) Potentially malignant lesions:

1) Leukoplakia

- Leukoplakia is defined as a predominantly white lesion that cannot be classified as any other definable lesion.
- White lesions for which a local cause is identified should be classified according to a established cause and not be included among leukoplakia's.
- Leukoplakia is therefore a term to denote a keratotic lesion of exclusion.
- It is common in older males and can be located on lip, buccal mucosa, gingiva, floor of mouth or tongue.

FIGURE 2. Leukoplakia-like lesion of left lateral tongue that on biopsy, showed morsicatio mucosae oris.

FIGURE 5. Dysplastic leukoplakia of the right lateral tongue (note sharp demarcation).

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ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

B) Potentially malignant lesions:

2) Actinic Chelitis:

- It is caused by sun damage to the lower lip.
- It is well documented that long term solar exposure produces irreversible damage to lower lip.
- It presents as dry, mottled and opalescent skin, with elevated gray and white plaques that cannot be removed by rubbing.
- Loss of definition of vermillion border also occurs.
- Invariable amount of ulceration and crusting is present.
- More commonly seen in Caucasian males over the age of 40.

> males over the age of 40, but given the affinity of Australians for the sun, close monitoring of tissue change on the lower lip in younger persons is recommended.

3) Erosive lichen planus

4) Chronic hyperplastic candidosis

- The malignant potential for erosive lichen planus and v=chronic hyperplastic candidiosis is still questionable, but such lesions should be treated accordingly and monitored carefully.

5) Non healing extraction socket (more than 6 weeks)

- This also raises suspision for possible alveolar carcinoma.

Fig. 7 Non healing extraction socket.

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

C) Malignant Lesions

Oral Cancer

- Dentists play a critical role in the management of oral cancer, g=from detection of premalignant lesions, early detection of oral cancer, management of the oral cancer patients dentition both prior to and post definitive treatment, surveillance of recurrent or new primary tumor in conjunction with the treating specialist, and rehabilitation of missing teeth in conjunction with treating maxillofacial surgeon or prosthodontist.
- The high rate of oral cancer in Australia is due mainly to lip cancer related to solar irradiation, which has been decreasing slowly over the past decade in men, but increasing slightly in young women.
- The incidence of intra-oral cancer has been gradually increasing.
- Survival of oral caner are very poor approximately 50 % overall.

Squamous Cell Carcinoma

- The etiology of oral squamous cell carcinoma is predominantly related to tobacco and alcohol consumption, however other factors may be involved.

Clinical features of oral cancer:

- The clinical features of oral cancer can vary from exophytic mass to a deep chronic ulcer but by this stage these changes are quite obvious upon clinical examination.
- There is no common appearance to oral cancer, but two critical clinical signs that are present in almost all cancers i.e. INDURATION AND FIXATION.

> induration and fixation. Induration is defined as an increase in the tissue density (the tissue becomes hard), and fixation is defined as loss of tissue mobility (the tissue is fixed and does not move). Clearly, a diligent

- The difficulty arises when the lesions are not frankly malignant, one such condition is oral leukoplakia.
- Simply, stated leukoplakia is white patch that cannot be rubbed off, and for which there is no specific diagnosis.
- Clinically the easiest way to assess a white lesion is to designate the lesion as either homogeneous or non-homogeneous.
- This does not offer a diagnosis but allows triage of lesions.
- Homogeneous lesions are those that are consistent in appearance and texture across the entire surface of the lesion. E.g. Frictional Keratosis.
- Non-Homogeneous lesions are those That are uneven in color, composition and texture from one area to another.
- Non Homogeneous lesions should be refreed or biopsied without delay.

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

Clinical features of oral cancer:

without delay. In a recent study, it was shown that non-homogenous leukoplakia accounted for 15–20 per cent of malignant development, whereas only 3 per cent of the homogenous leukoplakia developed carcinomas.[18]

Fig 1. Oral squamous cell carcinoma presenting on the left lateral tongue as an exophytic growth in the centre of a non-homogenous leukoplakia.

Fig 3. Homogenous leukoplakia on the buccal mucosa which is consistent in appearance and texture across the entire surface of the lesion.

Fig 4. Non-homogenous leukoplakia on left lateral tongue which is uneven in colour, composition, and texture from one area to another.

Fig 2. Oral squamous cell carcinoma presenting on the right lateral tongue as a deep chronic ulcer.

Fig. 3 White lesions.

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ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

C) Malignant Lesions

Erythroplakia or Erythro leukoplakia:

- It is a red or mixed red and white patch that cannot be clinically and pathologically be diagnosed as any other condition.
- Erythroplakia is much less common than leukoplakia, but has higher malignant transformation.
- Malignant transformation rates of oral erythroplakia are considered to be highest among all potentially malignant lesions and conditions.

Fig 5. Erythroleukoplakia on the right lateral tongue presenting as a mixed red-white patch.

Fig. 4 Erythematous lesion.

If there is any suspicion of a lesion that may represent oral cavity cancer, the lesion site and size should be documented, a clinical photograph taken if possible, and urgent referral made to an Oral & Maxillofacial Surgeon who is involved in a Head and Neck Oncology MDT.

Malignant lesion other than SCC:

- Pigmented Melanoma

Fig. 8 Pigmented lesion.

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ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

Diagnostic Aids

- Conventional screening practice for oral neoplastic lesions involves visual scrutiny of oral tissues with the naked eye under projected incandescent or halogen illumination.
- Visualization is the principal strategy used to find patients with lesions at risk of oral malignancy.

a) Vital Staining

- OraScreen is one such tool which has been used to identify suspicious lesions, and relies on a Meta chromic vital dye known as toluidine blue.
- Toluidine Blue has been used to satin abnormal tissue blue.

> toluidine blue. Toluidine blue has been used to stain abnormal tissue blue[44–47] and although helpful at identifying some at-risk lesions and frank oral cancers, it struggles to clearly delineate potentially malignant lesions and therefore has significant high levels of both false-negative and false-positive reactions.[48–50]

- Toluidine Blue Staining is useful in helping to delineate oral squamous cell carcinoma, but its reliability in screening potentially malignant lesion is poor.

b) Chemiluminescence

- Vizilite is another new diagnostic aid used to assist clinicians in visualization of oral mucosa abnormalities.

Fig 6. ViziLite visualization kit incorporating the acetic acid mouthwash, the chemiluminescent lightstick capsule, and the disposable retractor.

Fig 7. ViziLite chemiluminescent light after activation of chemicals in the lightstick capsule.

c) Auto Fluorescence

d) Oral Brush Biopsy

d) Histopathology

- Scalpel biopsy and Histopathology is the gold standard in definitive diagnosis of malignant and potentially malignant lesions.

ORAL CANCER AND OTHER WHITE LESIONS OF MOUTH

Diagnostic Aids

d) Histopathology

Diagnosis of lesions of the oral mucosa has traditionally been based on histopathological evaluation of a full thickness incisional scalpel biopsy of the lesion. Scalpel biopsy and histopathological examination of tissue specimens remain the definitive diagnostic investigations used to delineate malignant and potentially malignant lesions. Although this is still currently considered the gold standard, one must be mindful of the limitations of this process. Specialist pathologists differ in their definition of dysplasia and may report such lesions differently. In a study designed to test the

- Ideally the biopsy for oral cancer should be done by Oral and Maxillofacial Surgeon.

The diagnosis of oral cancer is dependent on obtaining a sample of tissue from the lesion, a biopsy. Ideally, the biopsy should be done by an Oral & Maxillofacial Surgeon as the treating surgeon has the opportunity to complete a full head and neck examination, including exact measurements, palpation of lesion thickness and clinical examination of the

OBSTRUCTIVE SLEEP APNOEA AND SLEEP BRUXISM WITH SLEEP MEDICINE

SOURCES: Article on Sleep-disordered breathing–clinical spectrum (ADJ 2024), Article on Myofunctional therapy for obstructive sleep apnoea (ADJ 2024), Relationship between sleep bruxism and obstructive sleep apnoea: A population-based survey (ADJ 2024), The status of dental sleep medicine education in Australia and New Zealand in 2024 (ADA), Precision medicine approaches in obstructive sleep apnoea: The role of dentist–sleep physician partnerships (ADJ 2024), Oral appliance therapy for snoring and obstructive sleep apnoea (ADA 2024), Article on Exploring the links between periodontal diseases and obstructive sleep apnoea (ADA 2024), Obstructive sleep apnoea, sleep bruxism and gastroesophageal reflux–mutually interacting conditions? A literature review (2024 ADA ARTICLE), Sleep-disordered breathing in children seeking orthodontic care—an Australian perspective (ADA 2023)

Sleep Disordered Breathing (SDB):

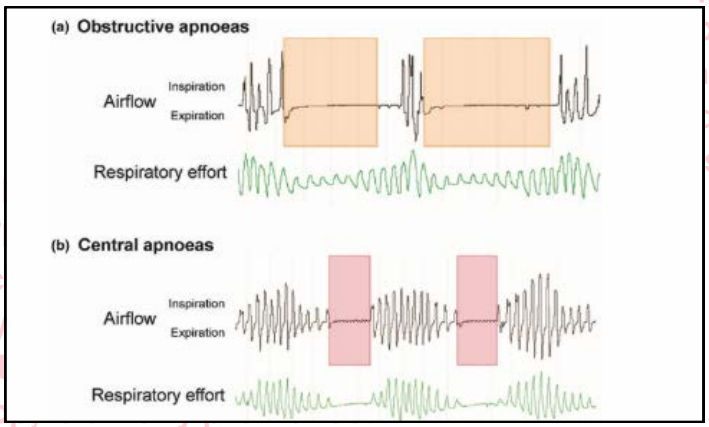
- **Sleep disordered breathing interferes with normal ventilation, oxygenation and sleep quality.**
- **The severity of SDB ranges from:**
  i) habitual snoring
  ii) upper airway resistance and
  iii) more severe obstructive sleep apnoea (OSA).
- **The condition has been associated with obesity and enlarged tonsils, however, there is emerging evidence of associations with various malocclusions.**
- **The International Classification of Sleep Disorders (ICSD) groups sleep-related breathing disorders into four major categories**

1. Obstructive sleep apnoea disorders

- **Obstructive sleep apnoea is a highly prevalent disorder characterized by repetitive collapse of the upper airway during sleep.**

2. Central sleep apnoea disorders:

- **Central Sleep apnoea is characterized by an interruption in ventilatory drive and consequent loss of respiratory effort, causing apnoeic events despite patency of the upper airway.**
- **This is distinct from obstructive sleep apnoea where there is preserved respiratory effort against an obstructed upper airway.**



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3. Sleep-related hypoventilation disorders:

- Hypoventilation is a failure to maintain adequate alveolar ventilation for the effective exchange of oxygen and carbon dioxide.
- A key feature for the diagnosis of sleep-related hypoventilation disorders is the demonstration of elevated arterial CO2, either on arterial blood gas analysis transcutaneous CO2 measurements.
- Hypoventilation is often exacerbated during REM sleep during which there is a physiological loss of muscle tone in the skeletal muscles and an increased reliance on the diaphragm to maintain ventilation.
- Treatment of hypoventilation in sleep will depend upon the cause.
- Obesity hypoventilation syndrome (OHS) may be responsive to CPAP, particularly in the presence of concomitant OSA or may require bilevel ventilation therapy.
- Weight loss, in conjunction with a dietician and/or endocrinologist, is a critical component of OHS therapy, including consideration of bar iatric surgery in selected patients.

4. Sleep-related hypoxemia disorder

- Disordered sleep architecture in children has been shown to affect children's behavior causing more aggression as well as memory and executive dysfunction.
- There is increasing evidence that the morbidity associated with SDB in childhood reflects that which is experienced by adults.
- Several links between SDB and poorer academic performance in the pediatric population has also been reported.
- From a research and clinical viewpoint, ***Sleep bruxism (SB), obstructive sleep apnoea (OSA) and gastroesophageal reflux disease (GERD)*** are sleep-related phenomena that establish a multifaceted network of conditions that are rarely found in isolation.

Risk Factors associated with Sleep disordered breathing in adults:

It can be divided into two categories:
a) soft tissue
b) skeletal or structural

- Within the soft tissue, category obesity is a key risk factor that is independently associated with SDB and OSA.

Risk factors associated with SDB	
Soft tissue	Skeletal/structural
Adenotonsillar hypertrophy Obesity Metabolic syndrome (lipid abnormalities, insulin resistance, fatty liver disease) Poorly controlled asthma Hypotonia	Midfacial hypoplasia Retrognathia Micrognathia Acutely angled skull base Narrow maxillary arch Nasoseptal obstruction Macroglossia Structural abnormalities of the upper airway e.g. cleft palate, laryngomalacia, choanal stenosis, choanal atresia, vocal cord palsy

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Signs and symptoms of Sleep disordered breathing:

- The variations in the signs and symptoms of SDB make it a condition that is underrecognized and underreported.
- For example, when adults become lethargic due to insufficient sleep, children become hyperactive and consequently there are reports of children with SDB being misdiagnosed with attention deficit disorders.
- Symptoms such as snoring are considered 'normal' by parents but snoring in children is actually a cardinal sign of SDB in children.

Symptoms of SDB (5–18 year olds)
Snoring
Witnessed apnoeas
Frequent arousals
Mouth breathing/dry mouth
Nocturnal sweating
Failure to thrive
Nasal congestion
Hyper-extended neck
Recurrent otitis media/Upper respiratory infections (URI)
Nightmares
Sleep talking
Confusional arousal
Sleepwalking
Daytime sleepiness/persistent naps
Restless sleep
Enuresis
Hyperactivity, inattention
Difficulty waking up in the morning
Drooling
Morning headache
Sleep in knee-chest position

- OSA is one of the most common sleep disorders, carrying significant health risks.
- Sleep bruxism (SB), obstructive sleep apnoea (OSA) and gastroesophageal reflux disease (GERD) are sleep-related phenomena that are gaining increasing attention as they can affect the prognosis of dental treatment and may also have an influence on orofacial pain.
- Sleep bruxism is a masticatory muscle activity (MMA) during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic) activity. It is not a movement disorder or a sleep disorder in otherwise healthy individuals.
- Sleep Bruxism must be distinguished from awake bruxism (AB), which may have different etiology, comorbidities and clinical consequences because of the different spectrum of muscle activities that may feature the various activities in relation to the circadian manifestation.

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OBSTRUCTIVE SLEEP APNOEA AND SLEEP BRUXISM WITH SLEEP MEDICINE

Signs and symptoms of Sleep disordered breathing:

- As per recent consensus papers, bruxism cannot be viewed as the disorder in any case. Even in cases of severe bruxism where there is presence of clinical consequences, bruxism itself should be viewed as a sign of an associated or underlying condition, rather than the actual disorder itself.
- Gastroesophageal reflux disease (GERD) is a chronic gastrointestinal condition characterized by the regurgitation of gastric contents into the esophagus. The pathophysiology of GERD is multifactorial.
- GERD is of great interest in dentistry, as it can cause severe dental erosion resulting from intrinsic chemical tooth wear accompanying the typical battery of medical symptoms (e.g. heartburn, regurgitation, chest pain, chronic cough, laryngitis or asthma).

Obstructive Sleep Apnoea (OSA)

- Obstructive sleep apnoea (OSA) is a disorder characterized by repetitive airway collapse leading to intermittent oxygen desaturations, fragmented sleep, and exaggerated intra-thoracic pressure swings.
- Repetitive episodes of upper airway closure during sleep is seen in patients with OSA.
- OSA is also considered a risk factor for numerous co-morbidities including hypertension, coronary artery disease, atrial fibrillation, congestive heart failure, stroke, diabetes, cognitive dysfunction, insomnia and mood disorders.
- The presence and severity of OSA are broadly defined by the number of times per hour of sleep that the air way completely (Apnoea) or partially (Hypopnoea) collapses, triggering a drop in blood oxygenation levels, termed the apnoea–hypopnoea index (AHI).
- The AHI is most often determined via an overnight sleep study or polysomnogram (PSG) and used clinically to distinguish the presence and severity of OSA and guide therapeutic intervention.
- Dentists trained in dental sleep medicine have a significant role as part of a collaborative multi-disciplinary team led by specialist sleep physicians in the overall management of OSA.

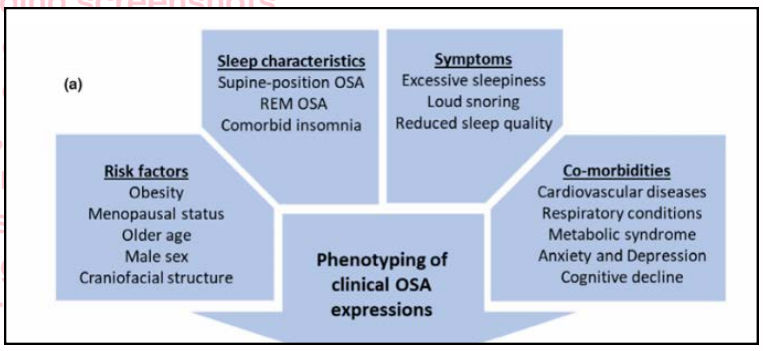
OBSTRUCTIVE SLEEP APNOEA AND SLEEP BRUXISM WITH SLEEP MEDICINE

Dental Sleep Medicine:

- **Dental sleep medicine** is concerned with the study of the oral and maxillofacial causes and consequences of sleep-related problems such as sleep-disordered breathing (snoring or obstructive sleep apnoea), sleep bruxism, orofacial pain and sleep-related complaints, and to some extent gastro-esophageal reflux disease (GERD)and/or insomnia. Obstructive sleep apnoea (OSA) is a key condition within dental sleep medicine.
- **Dental sleep medicine** is a rapidly evolving field, and it is the responsibility of each dentist to ensure that they are well educated and trained in the field so that they can assess new developments and provide evidence-based care for patients.
- However, Dental schools across Australia and New Zealand are still not providing adequate levels of education in the field of dental sleep medicine.
- Compared to dentists considered competent in practicing dental sleep medicine, the current graduating dental students have a significant disparity with regards to the education they receive.
- There is an urgent need for dental schools to increase the amount of time spent teaching dental sleep medicine, and to provide greater depth of knowledge in all areas of dental sleep medicine in order to ensure standards are met for the current needs in practicing dental sleep medicine.

Etiology, Symptoms and Risk Of OSA:

- The aetiology of OSA is complex and is influenced by multiple factors including craniofacial structure, obesity and fat distribution, upper airway collapsibility, pharyngeal muscle tone during sleep, arousal threshold and ventilatory control.
- The typical features used to define the expressions of OSA include age, sex, anatomical characteristics, sleep posture and sleep stage, symptomology, and comorbidities.
- These traits can be broadly grouped into:
  a) risk factors for developing OSA
  b) sleep characteristics of OSA,
  c) symptoms of OSA, and
  d) comorbidities that often have bidirectional relationships with OSA



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OBSTRUCTIVE SLEEP APNOEA AND SLEEP BRUXISM WITH SLEEP MEDICINE

Etiology, Symptoms and Risk Of OSA:

- Two major side effects and predictors of OSA are
  i) loud and persistent snoring accompanied with air 'gasping' (often identified by a bed partner) and
  ii) excessive daytime sleepiness.
- Daytime sleepiness is a hallmark complaint of untreated OSA that is associated with poorer health outcomes and elevated rates of occupational and motor vehicle accidents.
- OSA patients have very high rates of comorbid cardiovascular diseases such as systemic hypertension and coronary artery disease, respiratory diseases such as COPD, and metabolic disorders such as diabetes mellitus and dyslipidaemia.
- A high proportion of OSA patients suffer from comorbid insomnia, and there are high rates of anxiety, depression, and neuro cognitive impairment in OSA patients.
- Signs of OSA includes:

Table 3. Signs of OSA[10]

Signs of OSA
Obesity
Increased neck circumference
Increased waist circumference
Retrognathia
Maxillary constriction
Overjet
Overbite
Tonsillar hypertrophy
Macroglossia
Oropharyngeal narrowing (assessed by Mallampati class)
Soft palate erythema and oedema
Nasal obstruction
Hypertension

- Obesity is the most obvious of phenotypic factors which provides the opportunity for a targeted management approach and weight loss is a critical component of therapy in many patients

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Relationship between OSA, Sleep Bruxism and GERD

- It has been reported that patients with OSA have a significantly higher Sleep Bruxism prevalence than the general population.
- However, the results of different investigations pertaining to the relationship between SB and OSA appear to be inconsistent, highlighting the need for further research to characterize the relationship between SB and respiratory events during sleep.
- As in the case of OSA, some authors hypothesized that tooth wear and GERD symptoms severity are related.
- Some papers suggest the existence of a possible association between SB and GERD, which would lead to a more rapid loss of tooth substance because grinding and/ or clenching on teeth covered by acid accelerates the speed of hard tissue loss.
- The interconnection between the three conditions (i.e. SB, OSA and GERD) is suggested because GERD relates to a worsening in OSA severity.
- However, there is not enough scientific evidence yet to define a clear pattern of epidemiological and temporal relationship, if any, between SB, OSA and GERD.

Relationship between OSA and periodontal disease:

- There is growing evidence supporting obstructive sleep apnoea as an emerging risk factor for periodontal diseases.
- Several studies showed a significant association between sleep duration and sleep quality with the risk of Periodontal Diseases(PD). In particular, sleeping less than 5–6 hours or more than 8–9 hours increase the risk of periodontitis.
- OSA is considered a risk indicator for oral and PDs. The most recent meta-analysis assessing the relationship between OSA and periodontitis showed that the diagnosis of sleep-disordered breathing (mainly OSA) was associated with an increased odd of severe periodontitis.
- This association is independent of tooth loss and other established risk factors for periodontitis including, age, male sex, smoking, diabetes, and obesity.
- It remains unknown whether a “cause effect” relationship exists between OSA and PDs or whether the observed association represents an intersection of prevalence between two common diseases sharing multiple risk factors, consequences, and comorbidities.

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Relationship between OSA and periodontal disease:

- OSA may influence the development and progression of PDs by:

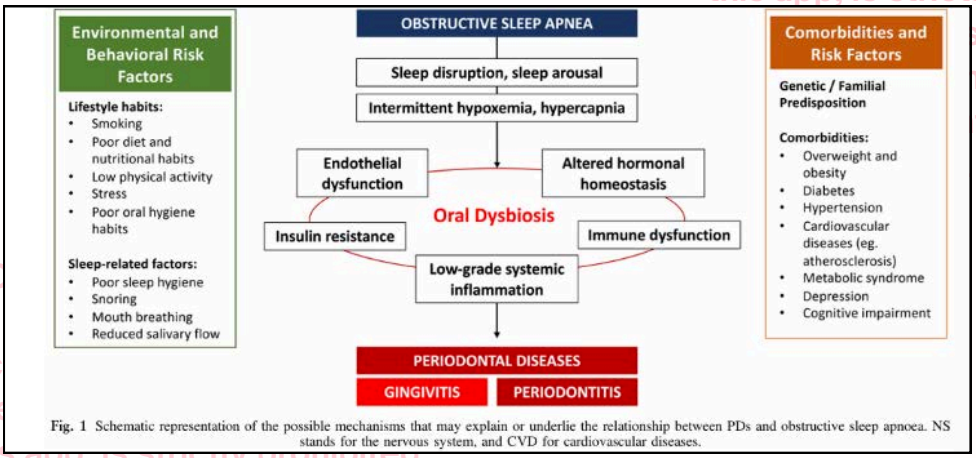

Fig. 1 Schematic representation of the possible mechanisms that may explain or underlie the relationship between PDs and obstructive sleep apnoea. NS stands for the nervous system, and CVD for cardiovascular diseases.

- Dentists play a key role in the screening, prevention, and treatment of OSA.
- Dentists must be aware that patients suffering from sleep disorders, and OSA in particular, are more likely to have Periodontal diseases, particularly severe periodontitis.

Screening and Diagnosis for sleep disordered breathing and OSA.

- Dentists have a role in screening patients for sleep-disordered breathing.
- Patients who complain of signs and symptoms associated with OSA, including snoring, choking, gasping, daytime sleepiness, and changes to mood and cognitive processes, should be screened using validated questionnaires.
- There are several screening questionnaires which combine self-report of symptoms associated with OSA and clinical findings such as BMI and neck circumference.
- There are several screening questionnaires which combine self-report of symptoms associated with OSA and clinical findings such as BMI and neck circumference.
- Potential screening tools include the STOP-BANG, Berlin Questionnaire, and the OSA50, often with the Epworth Sleepiness Scale, which is a subjective measure of daytime sleepiness used in conjunction with screening questionnaires to identify patients with symptomatic OSA.
- The pediatric sleep questionnaire (PSQ), can be used in children aged between 2 and 18 years without any severe medical or mental impairments.

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Screening and Diagnosis for sleep disordered breathing and OSA.

- As orthodontists, are in frequent contact with the child and adolescent patients, they are in a central position to detect and direct referrals for SBD.
- The timely identification of at-risk individuals through routine use of the PSQ in orthodontic populations can facilitate early diagnosis and treatment for SDB.
- Implementing the PSQ as a targeted screening tool could be beneficial for pediatric and general dental practitioners who, like orthodontic practitioner, have close interaction with children but the prevalence of SDB is likely lower.
- These screening questionnaires tend to have high sensitivity and low specificity
- Patients who have a positive screening test should be referred for further assessment utilizing PSG to confirm the diagnosis of OSA. Polysomnography (PSG) is the gold standard test to diagnose and assess the severity of SBD in children but PSG is expensive in terms of time, resources and equipment.
- The initiation of treatment for OSA should be based on a diagnosis using objective testing, either with polysomnography (PSG) or a home sleep apnoea test (HSAT).
- Dentists may refer a patient suspected of OSA for a diagnostic PSG to their general medical practitioner, who may then on-refer the patient to a specialist sleep physician.

Clinical Guide: Screening for obstructive sleep apnoea

- Patients with symptoms suggestive of OSA such as snoring and waking gasping, mood and cognitive changes, and excessive sleepiness should be assessed with a validated screening test.
- Commonly used screening tests include the Berlin Questionnaire, STOP-BANG, OSA50 and the Epworth Sleepiness Scale.
- Patients with a positive screening test should be referred to their general medical practitioner or to a specialist sleep physician for diagnostic evaluation.

- Patient Centered treatment For OSA includes:

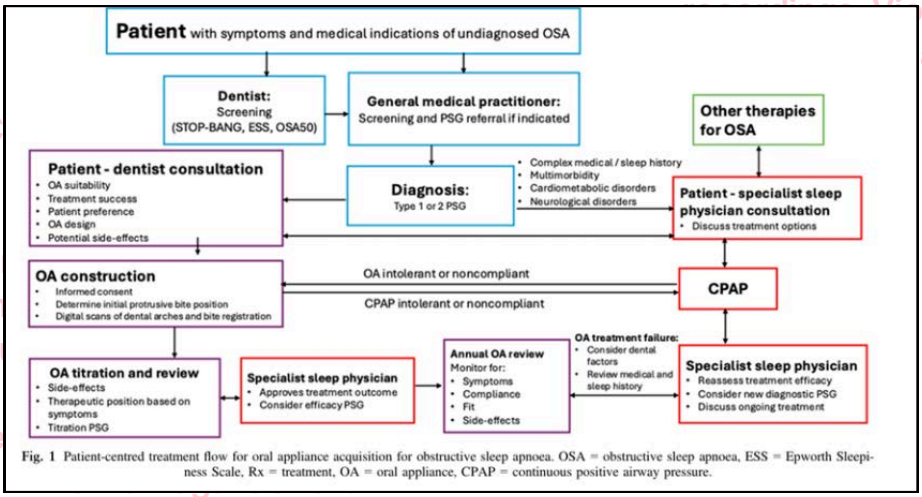

Fig. 1 Patient-centred treatment flow for oral appliance acquisition for obstructive sleep apnoea. OSA = obstructive sleep apnoea, ESS = Epworth Sleepiness Scale, Rx = treatment, OA = oral appliance, CPAP = continuous positive airway pressure.

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OBSTRUCTIVE SLEEP APNOEA AND SLEEP BRUXISM WITH SLEEP MEDICINE

Treatment Options:

- There are multiple treatment options for OSA.
- The choice of therapy is dependent on the wishes of the patient and the realistic consideration of treatment needs versus benefits as discussed with their medical team.

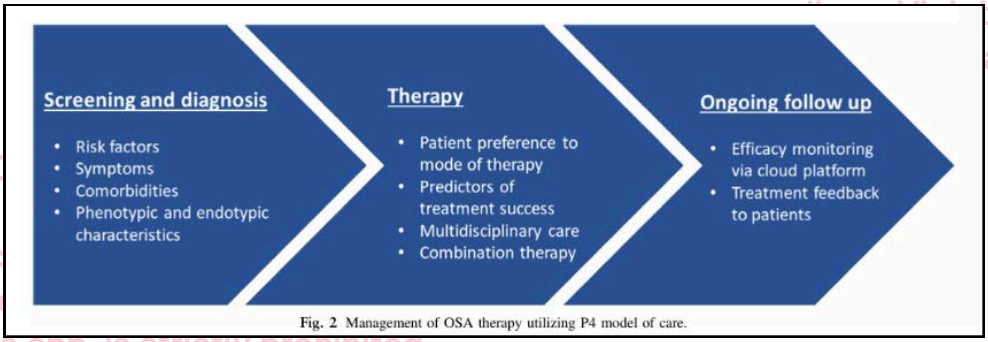

Fig. 2 Management of OSA therapy utilizing P4 model of care.

- Treatment options for Sleep Obstructive apnoea includes:

Table 1. Treatment options for obstructive sleep apnoea

Modality	Indications	Poor responders
CPAP[9]	• Adults with OSA • Excessive sleepiness • Hypertension • Sleep physician recommendation	• Low arousal threshold • Comorbid insomnia
Oral appliance therapy[8, 10, 11]	• Mild–moderate OSA • Snoring • Anatomic upper airway constriction • Severe OSA with high REM AHI, high hypoxic burden or multiple comorbidities when CPAP noncompliant or intolerant	• Obesity class 2 or 3 (BMI ≥35 mg/kg^2) • REM predominant OSA • High hypoxic burden • Insufficient teeth for retention • Central apnoea • Systolic hypertension with moderate–severe OSA
Bilateral hypoglossal nerve stimulation[12, 13]	• BMI≤32 kg/m^2 • Age 21–75 years • No positional OSA (AHI <10) • Obstructive AHI: 20–60 • Combined mixed+ central AHI <10 • No positional OSA (defined as non-supine AHI < 10 + supine AHI ≥ non-supine AHI × 2)	• Concentric collapse of airway during apnoea using DISE (1 in 4 patients) • Mean difference in AHI was 18 at 5 years in 129 patients
Maxillomandibular advancement[14, 15]	• Clinically significant OSA with skeletal discrepancy (retrognathia, midface deficiency) • Failed CPAP • Surgical success defined as >50% reduction and AHI <20 and surgical cure as AHI <5	• Medically complex • Temporomandibular disorders
Positional devices[16, 17]	• Reduce time in supine position	• Low arousal threshold • Higher NREM AHI
Weight loss[18] • Dietary modifications[19] • Surgical[20] • Pharmacological[21]	• Obesity and OSA • Other medical disorders that are impacted by obesity	• 20%↓ in BMI associated with 57% ↓ in AHI • Poor response associated with fat distribution, ethnicity, supine OSA, REM OSA, anatomy, and Type 2 diabetes mellitus • Risk for some relapse of weight loss with time
Oral/nasal expiratory positive airway pressure[22, 23]	• Increase supine NREM AHI	• Increases poor sleep quality (leads to lower sleep efficiency)
Pharmacotherapy for OSA (acetazolamide and atomoxetine-plus-oxybutynin)[24]	• To increase muscle responsiveness • Reduce arousability • Change chemosensitivity	• Intolerance to side effects such as drowsiness and dry mouth
Aerobic exercise, 45 min of walking twice daily,[25, 26] resistance training,[27] and high-intensity training[28]	• Improve sleep quality • Improve health • Reduce rostral fluid shift	• Non-compliant • Health issues prevent compliance
Combination therapies[22, 23, 29–31]	• Insufficient clinical response to one therapy • Increased side effects from one therapy at a therapeutic dose • OA + CPAP = 35–40% reduction in CPAP pressure[29] • OA + positioner device reduces time spent in supine sleep	• Increased cost • Increased complexity of treatment and monitoring

AHI, apnoea-hypopnoea index; BMI, body mass index (kg/m^2); CPAP, continuous positive airway pressure; DISE, drug-induced sleep endoscopy; OA, oral appliance; OSA, obstructive sleep apnoea; RDI, respiratory disturbance index; REM, rapid eye movement.

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Treatment Options:

a) CPAP (Continuous Positive Airway Pressure)

- CPAP remains the gold standard therapy for OSA.
- Continuous positive airway pressure (CPAP) therapy is the usual first-line treatment for OSA and is the most efficacious therapy for modifying parameters of OSA severity.
- There is a dose–response relationship between hours of usage and improvement in daytime symptoms, with CPAP most effective when worn on most nights for the duration of sleep time.
- However, low adherence CPAP treatment is a major issue.
- Therefore, the management of OSA requires multi-disciplinary care that effectively manages both OSA and any associated comorbid conditions in a manner that is well accepted by the patient.

b) Oral Appliance (OA) therapy/ Mandibular Advancement Splint (MAS) Therapy

- Mandibular advancement splint (MAS) therapy is the leading treatment alternative to CPAP which may be considered after intolerance to PAP or on the basis of patient preference.
- Provision of an oral appliance (OA) can improve the health and quality of life of patients who suffer from obstructive sleep apnoea (OSA).
- Oral appliance (OA) therapy involves the provision of a removable appliance worn over the maxillary and mandibular teeth to protrude (advance) the mandible during sleep to treat snoring and obstructive sleep apnoea(OSA).
- These appliances may be referred to as:
  a) mandibular advancement appliance,
  b) a mandibular advancement splint/device or
  c) an oral appliance.
- MAS devices have the advantage of being highly portable, silent and often preferred by users to CPAP treatment.
- The provision of OA therapy should be instituted by a dentist trained and competent in the field of Dental Sleep Medicine (DSM) as part of a multidisciplinary team.
- Once OA therapy has commenced, the providing dentist is responsible for monitoring the adherence and the efficacy of treatment.
- The dentist is also responsible for referring the patient to the appropriate medical practitioner as required and for communicating the patient's progress.
- OA may be constructed after sleep physician-reported polysomnography (PSG) has diagnosed either primary snoring, with mild to moderate OSA, or severe OSA in a patient who is CPAP intolerant or noncompliant or would prefer this type of therapy.

OBSTRUCTIVE SLEEP APNOEA AND SLEEP BRUXISM WITH SLEEP MEDICINE

Treatment Options:

b) Oral Appliance (OA) therapy/ Mandibular Advancement Splint (MAS) Therapy

Clinical Guide: Oral appliance assessment and informed consent

- The provision of OA therapy must be under the supervision of a dentist trained in DSM.
- OA is one of several non-CPAP therapies available for patients who have a specialist sleep physician-reported diagnosis of OSA.
- Patient's preferred treatment is an indication for provision of OA therapy.
- The patient and the dentist must have an interactive discussion covering OA therapy which should include patient eligibility, treatment efficacy, side effects, appliance design, treatment protocol, and the need for ongoing maintenance and monitoring of the appliance.

- **The selection of patient for Oral appliance therapy is also important:**

Clinical Guide: Oral appliance therapy selection criteria
The provision of OA therapy should only be considered if the patient meets the selection criteria:

- An adequately restored dentition with sufficient dental or implant-borne support for appliance retention.
- The health of the periodontium and the TMJs and supporting structures should be stable.
- Imaging should be based on the standard required for dental examination only (x-rays) and prescription of advanced imaging such as CBCT, CT and MRI should be limited for the assessment of oral and maxillofacial pathology.

- **Success of Oral appliance therapy:**

Clinical Guide: Treatment success with oral appliance therapy
Successful OA therapy requires a combination of:

- Regular use for at least 4 h/night.
- Improvement in the quality-of-life outcomes relevant to each patient such as excessive daytime sleepiness, mood and cognitive change.
- Clinically relevant reductions in the AHI and oxygen desaturation and improvements in sleep quality.
- OA efficacy may be altered by advancing age and lifestyle dependent changes which may influence the severity of OSA.
- As patients with OSA are at increased risk for the onset and exacerbation of cardiometabolic, cognitive and other medical disorders, they should be routinely monitored after OA provision so that changes in the effectiveness of OA therapy are detected early and dealt with by further titration of the appliance or referral to their supervising medical practitioner for review.

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OBSTRUCTIVE SLEEP APNOEA AND SLEEP BRUXISM WITH SLEEP MEDICINE

Treatment Options:

b) Oral Appliance (OA) therapy/ Mandibular Advancement Splint (MAS) Therapy

- **The selection of patient for Oral appliance therapy is also important:**

Table 3. Clinical considerations of potential side effects related to oral appliance therapy

Clinical Considerations	Clinical Presentations of Side Effects
Temporomandibular function	• Transient morning jaw soreness • Persistent temporomandibular joint (TMJ) tenderness or pain • Persistent tenderness or pain of the muscles of mastication • New onset TMJ sounds, catching or locking • Morning temporal headache
Intraoral tissues	• Soft tissue and tongue irritation • Gingival irritation • Excessive salivation and drooling • Dry mouth
Occlusion	• Altered occlusal contacts / bite changes • Incisor position changes • Decreased overjet and overbite • Alterations in the position of the mandibular canines and molars • Development of interproximal gaps (food traps)
Teeth and restorations	• Tooth mobility • Tooth fractures related to changes in occlusion • Damage to dental restorations caused by appliance retention
Appliance	• Breakage related to sleep bruxism • Allergies to appliance material • Gagging • Anxiety related to fear of choking or difficulty swallowing

- **Teamwork required For Oral appliance therapy:**

Clinical Guide: Multidisciplinary teamwork for oral appliance therapy

- Dentists must work as part of a collaborative, multidisciplinary medical team which is led by the supervising medical practitioner.
- Dentists have a role in screening symptomatic patients for sleep-disordered breathing and in the provision of OA therapy for snoring and OSA.
- Dentists must not provide diagnostic PSG for patients with the intent to diagnose and prescribe OA therapy without the input of the supervising medical team.
- Dentists must follow-up patients undergoing OA therapy on an annual basis.

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OBSTRUCTIVE SLEEP APNOEA AND SLEEP BRUXISM WITH SLEEP MEDICINE

Treatment Options:

c) Myo-functional therapy:

- Myofunctional Therapy (MT) is one of the newest treatments for OSA, and it consists in combinations of oropharyngeal exercises that is, mouth and throat exercises.
- These combinations typically include daily exercises involving several muscles and areas of the mouth, pharynx and upper respiratory tract, to strengthen/improve the efficacy of Upper Airway muscles and to expand the opening of the upper airway.
- MT can effectively relieve snoring and daytime sleepiness in OSA patients, reduce AHI, and improve nocturnal hypoxia.
- Combined with CPAP, MT can also improve patient compliance with treatment.

d) Surgical treatments for OSA:

- Surgical approaches, while not as effective as CPAP in lowering the AHI, may have a role in selecting patients who are intolerant or non-compliant with CPAP or MAS and who have suitable anatomical characteristic.
- Radio-assisted uvulopalatoplasty (UPPP) combined with tonsillectomy has success rates between 60% and 70%.

e) Other therapies for OSA

- Non-surgical weight loss is well shown to reduce parameters of OSA, such as AHI, and should form part of the overall management of OSA.
- Positional therapy, which consists of strategies to minimize sleep in the supine position, is an effective therapy for patients with supine-predominant OSA.
- Combination therapy may be beneficial for patients who have a partial treatment response to monotherapy or to improve tolerability.

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