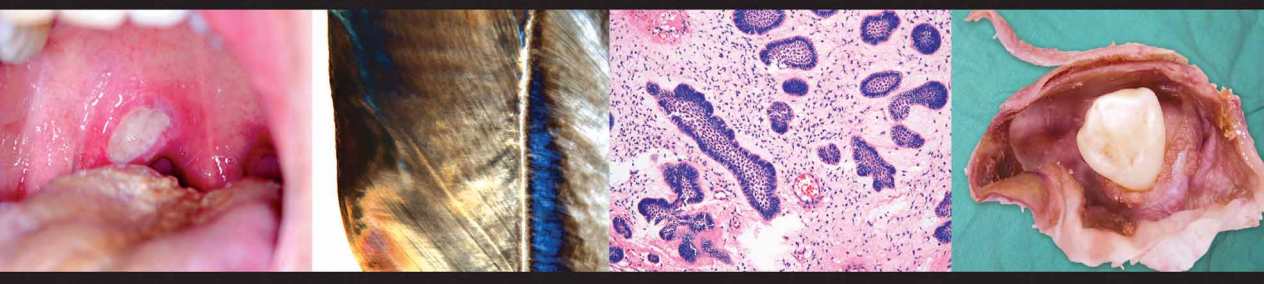
ORAL & MEDICINE PATHOLOGY

A Guide to Diagnosis and Management



Saman Warnakulasuriya
WM Tilakaratne

Foreword
Poul Erik Petersen

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Chapter

3

Periodontal Diseases

Philip M Preshaw

Chapter Outline

Epidemiology of Periodontal Disease
History, Examination and Assessment of Risk Factors
- History
- Examination
- Risk Factor Assessment

Diagnosis, Treatment Planning and Empowering the Patient
Gingivitis
Periodontal Diseases
- Chronic Periodontitis
- Aggressive Periodontitis

Microbiology, Histology and Pathogenesis
- Microbiology
- Histology and Pathogenesis
- Direct Injury by Plaque Bacteria
- Host Derived Inflammatory Mediators
- Pathogenesis and Clinical Signs of Disease

Periodontal—Systemic Interface
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- Coronary Heart Disease
- Pregnancy Outcomes

Treatment of Periodontal Disease
- Plaque Control
- Root Surface Debridement (RSD)
- Adjunctive Treatments
- Risk Factor Management
- Maintenance Care
- Surgical Therapy

Self-assessment Questions

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INTRODUCTION

Periodontal disease is a common, chronic inflammatory disease of the tooth supporting structures. It is one of the most common diseases known to mankind, and its sequelae, mobile teeth and loss of teeth, can have a major impact on quality of life and nutrition. Good oral health is an essential prerequisite for maintaining good general health. The dental team therefore have a major role in contributing to the health and wellbeing of their patients. Given that periodontal diseases are, for the most part, largely preventable and the tissue destruction that ensues is largely irreversible, it remains the responsibility of the dentist to examine patients carefully, to reach an accurate diagnosis, to communicate their findings to the patient, and to provide appropriate therapy. The first step in improving our management of periodontal diseases is to ascertain the prevalence of these conditions in the population.

EPIDEMIOLOGY OF PERIODONTAL DISEASE

Epidemiology describes the study of the distribution of disease in human populations. It is important to distinguish between the incidence of a disease and the prevalence of a disease. Incidence refers to the number of new cases of a disease that develop over a given time period in a population. However, we are unable to identify the exact point in time at which a patient develops periodontitis, and therefore in periodontal epidemiology we do not attempt to measure disease incidence. Instead, we rely on disease prevalence, which refers to the proportion (percentage) of people in a population affected by the disease at a given point in time.

To ascertain the prevalence of periodontal disease, we need to be able to accurately and reproducibly define whether or not a patient has the disease. But, how is periodontitis diagnosed or defined? Many studies in the 1960s and 1970s used a very low threshold for defining the presence of periodontitis, or even failed to distinguish between gingivitis and periodontitis, which resulted in very high reported prevalence rates in the periodontal literature. Data from research conducted at the time suggested that periodontitis inevitably commenced in early adulthood, and that almost the entire population was affected by the time individuals reached their 40s. Tooth loss was thought to increase gradually with age, in a relatively linear fashion. These observations led to a theory of periodontal disease that we might now refer to as the 'Old Theory of Periodontitis' (**Box 3.1**).

Box 3.1: Theories of periodontitis

The old theory of periodontitis

- Periodontal disease is inevitable following gingivitis
- Periodontal disease is uniformly distributed in the population
- Disease severity is correlated with plaque levels
- There is a linear, progressive loss of attachment over time
- The severity of periodontitis increases with age

The new theory of periodontitis

- Gingivitis and mild periodontitis are common, and gingivitis is present in a majority of most populations
- Approximately 10 to 15 percent of most populations exhibit advanced periodontitis
- Gingivitis precedes periodontitis but not all sites with gingivitis develop periodontitis
- Periodontitis is not a natural consequence of ageing (but older people may present with more loss of attachment as a result of a lifetime of tissue breakdown events)
- In some patients, periodontitis may progress with episodes of disease activity interspersed with periods of quiescence, in a nonlinear manner.

Today, we can recognize the methodological shortcomings of these early studies. Guidelines for assigning a diagnosis for the presence of periodontal disease in epidemiological studies have been developed (see reading list). We now realize that although gingivitis and mild periodontitis (characterized by isolated shallow

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pockets of 4–5 mm) are common, advanced periodontitis (characterized by multiple deep pockets of 6 mm or greater) is much less prevalent than previously thought. Although estimations of the prevalence of advanced periodontitis vary according to the population studied, a consistent finding is that advanced periodontal disease affects approximately 10 to 15 percent of adults in most populations. Though this is less than estimates of disease prevalence that were reported previously, this still represents a very large proportion of most populations, with large numbers of people affected. Furthermore, plaque and calculus are almost ubiquitous, and are found in the vast majority of adults.

In addition to studying the epidemiology of periodontal disease, researchers have investigated the nature of periodontitis progression. Early studies considered that loss of attachment and alveolar bone destruction continued in a linear fashion over time, and that once periodontitis was initiated, its progression was inevitable. More recent longitudinal monitoring studies have revealed that, for the majority of periodontitis patients, most sites are stable at any given time, and only a relatively small proportion demonstrate disease progression. It must be remembered, however, that these findings are based on repeated examination of patients using a periodontal probe. The periodontal probe is a somewhat crude diagnostic tool. The probe is attempting to identify tissue breakdown events at the base of the pocket that are resulting in microscopic alterations in the epithelial and connective tissue attachment to the tooth, and therefore it is unsurprising that we are unable to identify with any degree of reliability which periodontal sites are actively undergoing breakdown when we examine a patient. Nonetheless, we can summarize the findings of more recent epidemiological surveys into what we can call the 'New Theory of Periodontitis', as shown in **Box 3.1**. This theory reveals that severe forms of periodontitis are found in approximately 10 to 15 percent of most populations, representing a very large number of individuals across the world.

HISTORY, EXAMINATION AND ASSESSMENT OF RISK FACTORS

A thorough history and a careful examination are fundamental to making the correct diagnosis. During the history, patients should be allowed to explain the problem from their perspective, and the dentist should ask open questions to encourage the patient to talk freely. The dentist should be nonjudgemental and should be sympathetic to the patient. Many patients consider that periodontal disease is 'all their own fault', but given what we know about the importance of the host inflammatory responses in determining the progression of disease (see below), we know this is simply not true. The dentist should be supportive, while also communicating effectively and clearly with the patient so that the patient will understand their role in managing the condition.

History

It helps, when taking a history, to follow a structure so that important questions are not missed. A suggested scheme is presented in **Table 3.1**. Given that most cases of periodontitis do not cause pain, many patients may not be aware that they have a problem until the condition is very advanced. Therefore, the onus for detection of disease rests firmly with the clinician. Gingival bleeding is one of the most common complaints of patients with periodontal disease, usually when toothbrushing or eating. If the condition is more advanced, and has been present for a number of years, patients may present with mobile teeth, or teeth that have drifted, opening up spaces between teeth. Teeth drift when their periodontal support is compromised to such an extent that the teeth are no longer in equilibrium with forces from occlusion and the adjacent soft tissues.

Many patients are very concerned about bad taste in the mouth and halitosis. Halitosis is a common finding in patients with periodontitis and can result from many causes including:

- Accumulation of plaque and calculus

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▪ Table 3.1
Structured history taking

Presenting complaint	• In the patient's own words. • Patients will typically mention bleeding gums, bad breath, mobile teeth, drifting of teeth, gingival recession. However, pain is not usually reported in gingivitis or periodontitis.
History of presenting complaint	• How long has there been a problem? • Has the patient been aware of this for weeks, months, years?
Previous dental history	• Is the patient a regular dental attender? • Has any periodontal treatment been provided in the past? If so, by whom (e.g. dentist, hygienist, periodontist)? • How long did the treatment appointments last? • Was the treatment a simple 'scale and polish' or was it more thorough root surface debridement? • If teeth have been lost, why was this?
Use of oral hygiene products	• What is the patient using? • Frequency of tooth brushing? • Are interdental cleaning brushes being used?
Family history	• Do any other family members have periodontal problems (this could potentially indicate aggressive periodontitis, which has a familial aggregation)
Social history	• Is the patient employed? What do they do? • Sources of stress in life?
Smoking history	• If a current smoker, how many cigarettes per day, and for how many years have they smoked? • If an ex-smoker, when did they quit? For how many years did they smoke, and how many cigarettes did they used to smoke per day?
Medical history	• Record and update the medical history at each visit. • Are there risk factors for periodontal disease, e.g. diabetes? • Are there medical conditions that impact on dental management or provision of periodontal treatment? • Are there nutritional deficiencies or dietary problems? • Which medications does the patient take, and can these impact on periodontal status or treatment?

- Food debris
- Purulent exudate from a periodontal pocket
- Excessive bacterial growth on the dorsum of the tongue
- Necrotizing ulcerative gingivitis.

If pain is present, it may indicate necrotising ulcerative gingivitis, or an acute periodontal abscess. Gingival recession that has resulted in exposure of the roots may also result in dentine sensitivity. Pain is not otherwise a typical feature of chronic periodontitis.

Examination

The intraoral examination should include several components. An initial assessment of the level of oral hygiene and plaque control is important. The degree of gingival inflammation should be also assessed. All patients should be screened for periodontal disease by probing, and the quickest method is to record the basic periodontal examination (BPE), also known as the Community Periodontal Index of Treatment Needs (CPITN).

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A WHO (World Health Organization) periodontal probe should be used: this has a 0.5 mm diameter ball end and a colored band extending 3.5 to 5.5 mm from the tip. The dentition is divided into sextants, the periodontium around each tooth is probed and the highest score in each sextant is recorded. The score codes assigned are used as a guide to determine the need for periodontal treatment **(Table 3.2)**. If the results of the screening examination indicate the presence of periodontitis (i.e. BPE code 3 or code 4), then a detailed periodontal examination should be undertaken (i.e. full charting). If there is only one tooth in a sextant, no score is recorded for that sextant, and instead, the tooth is assessed together with the teeth in the neighboring sextant.

For those patients requiring a full periodontal examination, this should be performed at 6 sites per tooth, together with measurement of gingival recession (and calculation of loss of attachment by addition to the probing depth measurements) and bleeding on probing. Bleeding on probing results from the trauma caused to the epithelial pocket lining and connective tissue by the probe, but given that many factors can affect whether the tissue bleeds in addition to the degree of inflammation (such as the force of probing, sharpness of the probe tip), presence of bleeding on probing has a low sensitivity for periodontal disease progression. However, absence of bleeding on probing is a good indicator of periodontal stability.

Furcation involvement should be assessed with a curved furcation probe (Nabers probe). Classification is as follows:

- *Class I:* The tissue destruction does not exceed more than 3 mm (or not more than one-third the tooth width) into the furcation
- *Class II:* The tissue destruction is >3 mm (or more than one-third the tooth width) into the furcation but does not completely pass through the furcation
- *Class III:* It has through-and-through involvement, and the lesion extends across the entire width of the furcation.

A tooth with a furcation involvement is a compromised tooth. It has been estimated that the surface area of the root trunk of the first maxillary molar is approximately 30 percent of the total root surface area. In other words, by the time the furcation can be detected, a significant

▪ Table 3.2
The basic periodontal examination (BPE)

*Code**	*Probing*	*Treatment needs*
0	Colored band on the probe is completely visible No calculus detected No bleeding on probing	No need for periodontal treatment
1	Colored band on the probe is completely visible No calculus detected Bleeding on probing	Oral hygiene instruction (OHI)
2	Colored band on the probe is completely visible Calculus detected (supra- or subgingival), or overhanging restoration(s)	*OHI:* Elimination of plaque-retentive areas, scaling, root surface debridement (RSD)
3	Colored band on probe is partly visible, indicating probing depth of >3.5 mm but <5.5 mm	OHI, RSD
4	Colored band on probe completely disappears into pocket, indicating probing depth of >5.5 mm	OHI, RSD. Complex additional treatment may be required. Referral to a periodontal specialist may be necessary.

* The symbol (*) should be added to the score when furcation involvement is present. The score should then be recorded, for example, as 4*, or 3*, etc.

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degree of periodontal breakdown has already occurred.

Tooth mobility should also be assessed by applying a labiolingual, horizontal force to each tooth, using the handles of dental mirrors. A mobility index can be recorded as:

0 Normal, physiological mobility (<0.3 mm)
1 Horizontal mobility > 0.3 mm to 1.0 mm
2 Horizontal mobility 1.0 to 2.0 mm
3 Horizontal mobility > 2.0 mm and/or vertical mobility.

Occlusal examination should be undertaken when indicated. Many patients with periodontitis present with mobile teeth, and occlusal interferences may contribute to this. It is important to note that trauma from occlusion does not cause periodontitis; however, in the presence of pre-existing periodontitis, trauma from occlusion may exacerbate mobility and lead to an increased rate of alveolar bone loss. Fremitus should be assessed, and the presence of traumatic incisal relationships should be determined. Traumatic incisal relationships are classified by the Akerly classification:

1. Lower incisors in compete overbite, contacting palatal mucosa but distal to the palatal gingival margins of the upper anterior teeth.
2. Lower incisors in complete overbite, contacting palatal gingival margins of the upper anterior teeth (can result in localized trauma and recession at the palatal gingival margins of the maxillary anterior teeth).
3. As for class 2, but there is also shearing trauma of the gingival margins at the labial aspects of the lower anterior teeth as the upper incisors slide past the lower incisors (predisposed by lack of posterior support, and class II division 2 overbite).
4. Palatal attrition, i.e. dental trauma as the lower incisors contact the palatal surfaces of the upper incisors.

Occlusal trauma can be defined as injury to the attachment apparatus of the teeth as a result of occlusal forces, and is classified as primary occlusal trauma or secondary occlusal trauma. Primary occlusal trauma is occlusal trauma that affects periodontally healthy teeth (i.e. those with normal attachment and bone support), and is usually caused by excessive occlusal forces (e.g. as a result of an occlusal interference). Secondary occlusal trauma is that which affects periodontally compromised teeth (which have pre-existing attachment loss and bone loss), and may result from either normal or excessive occlusal forces.

A classic appearance of a periodontally compromised tooth that also is subject to trauma from occlusion (i.e. secondary occlusal trauma) is that of a crater-like defect with extensive bone loss and a very mobile tooth (**Fig. 3.1**). This is distinct from the radiographic appearance of a periodontally healthy tooth subject to a traumatic occlusion (i.e. primary occlusal trauma). In the healthy situation, trauma from occlusion (primary occlusal trauma) may result in widening of the periodontal membrane space and increased mobility, but will not cause periodontitis. If the traumatic interference is removed, then the periodontal membrane space returns to normal and mobility decreases.

Radiographic investigation is an essential part of the assessment of patients with periodontitis. Modern panoramic machines provide high quality images at reduced radiation exposure compared to full mouth periapicals. Radiographic features that can be identified include:

- Pattern of bone loss: horizontal or vertical, localized or generalized, irregular or regular extent of bone loss, usually described as a proportion or percentage of the root affected
- Furcation bone loss

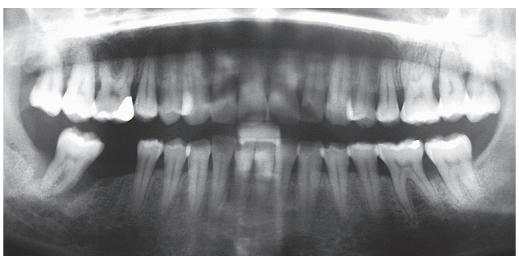

Fig. 3.1: Trauma from occlusion resulting in exacerbation of pre-existing periodontal disease. Radiograph showing generalized horizontal alveolar bone loss affecting between one-third and one-half the root length, being most advanced at molar sites and the maxillary incisors. Extensive vertical defects/crater defect at tooth 47, in traumatic occlusal relationship

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- Variations in root anatomy
- Interproximal calculus
- Widening of the periodontal membrane space
- Overhanging restorations.

A decreased alveolar bone height on a radiograph is a historical record of previous periodontal involvement and provides no information on current disease activity.

Risk Factor Assessment

Assessment of risk factors (or predisposing factors) is one of the most important parts of the assessment of a patient with periodontitis, yet is often overlooked. Risk factors can be broadly classified as systemic risk factors or local risk factors.

Systemic risk factors include smoking and diabetes. Smoking is the most important systemic risk factor for periodontitis and a detailed smoking history must always be recorded. Patients must always be informed of the importance of this risk factor and encouraged to quit. Smoking has wide ranging impacts on inflammation and immune responses, and is a significant negative modifier of multiple types of periodontitis. It is important to record in the patient notes that the importance of smoking as a risk factor has been explained to the patient. Similarly, poorly controlled diabetes is a major risk factor for periodontitis. However, patients with well controlled diabetes do not appear to be at increased risk for periodontitis. Local risk factors include multiple anatomic and iatrogenic factors that typically are plaque retentive (e.g. overhanging margins of restorations, orthodontic appliances, etc).

DIAGNOSIS, TREATMENT PLANNING AND EMPOWERING THE PATIENT

A correct diagnosis is essential for correct treatment planning. It is important not to adopt a 'one size fits all' approach to periodontal treatment. The treatment must be individualized to the patient, and that starts with a thorough history to understand the needs of the patient, and requires a correct diagnosis. A diagnosis can be assigned based on the disease criteria established by the American Academy of Periodontology in 1999 (**Table 3.3**). Reaching a correct diagnosis is dependent on

▪ Table 3.3
Classification of periodontal diseases

Gingival diseases	**A. Plaque-induced gingivitis:** This is the most common type of gingival condition, and may be associated with plaque only, or further modified by, for example, systemic drugs (e.g. those that cause drug induced gingival overgrowth), or hormonal variations (e.g. those seen in pregnancy) **B. Nonplaque induced gingivitis:** This includes specific infections (e.g. primary herpetic gingivostomatitis), gingival manifestations of conditions such as lichen planus, and traumatic gingival lesions
Chronic periodontitis	This is the most common form of periodontitis. It is characterized by some/all of the following: • Most prevalent in adults but can occur in children/adolescents • Subgingival calculus commonly found • Associated with a variable microbial pattern • Slow/moderate rate of progression, but may have periods of rapid progression • Can be classified on basis of extent (e.g. localized or generalized) or severity (e.g. amount of attachment loss) • Can be associated with local predisposing factors (e.g. plaque retentive features such as overhangs) • Can be modified by/associated with systemic exposures such as smoking, or conditions such as diabetes

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Aggressive periodontitis	'Aggressive periodontitis' (AP) replaces previously used terms such as 'rapidly progressive periodontitis' or 'juvenile periodontitis'. The primary characteristics of AP are: • Except for the presence of periodontitis, patients are otherwise clinically healthy • Rapid attachment loss and bone destruction (often characterized by very irregular pattern of alveolar bone loss, multiple vertical defects, extensive bone loss) • Familial aggregation (which is why it is important to ask about family members when examining patients with suspected aggressive periodontitis) Secondary characteristics of AP are: • Plaque levels are sometimes less than might be expected given the severity of the periodontal disease (this situation might be typically seen in a very concerned and motivated patient who clearly has a genetic susceptibility and is maintaining good oral hygiene, yet still has advanced disease) • Elevated levels of A*ggregatibacter actinomycetemcomitans* (especially in localized AP) and *Porphyromonas gingivalis* • Phagocyte abnormalities and a hyper-responsive macrophage phenotype, including elevated levels of PGE_2 and IL-1β (though there is no diagnostic test for a hyper-responsive phenotype and this is essentially a research finding only) AP can be further classified as localized (localized to the first molars and incisors, and typically has a circumpubertal onset) or generalized (affecting at least 3 permanent teeth other than first molars and incisors, usually affecting people <30 years, but patients may be older, and with a pronounced episodic nature of attachment loss and alveolar bone loss).
Periodontitis as a manifestation of systemic disease	This includes periodontitis associated with **hematological disorders** (such as acquired neutropenia and leukemias) and periodontitis associated with **genetic disorders** (such as cyclic neutropenia, leukocyte adhesion deficiency syndromes, Papillon-Lefèvre syndrome, Chediak-Higashi syndrome, Ehlers-Danlos syndrome, hypophosphatasia.
Necrotizing periodontal diseases	**A. Necrotizing ulcerative gingivitis (NUG)** Characterized by gingival necrosis, with 'punched-out' papillae, pain, gingival bleeding, halitosis, pseudomembranous sloughing. Predisposing factors include stress, smoking, poor diet, HIV infection. **B. Necrotizing ulcerative periodontitis (NUP)** Characterized by necrosis of the gingiva, PDL and alveolar bone. Most typically seen in patients with significant systemic illness, e.g. HIV, severe malnutrition, immunosuppression.
Abscesses of the periodontium	**A. Gingival abscess,** localized to the marginal gingiva or interdental papilla **B. Periodontal abscess,** localized abscess in the pocket, may result in bone loss **C. Pericoronal abscess,** also known as pericoronitis
Periodontitis associated with endodontic lesions	These are combined periodontal-endodontic lesions, typically referred to as 'perio-endo lesions'. Either the periodontal or endodontic lesion may be the cause or the result of the other, or both lesions may develop independently on the same tooth, until they coalesce into a single lesion.
Developmental or acquired conditions	**A. Localized tooth related factors** (e.g. anatomic factors, iatrogenic factors such as overhangs, root resorption, root fractures) **B. Mucogingival deformities around teeth** (e.g. localized recession defects, lack of keratinized gingiva, high frenal attachment) **C. Mucogingival deformities on edentulous ridges** (e.g. reduced ridge height or width) **D. Occlusal trauma** (primary occlusal trauma or secondary occlusal trauma)

having taken a thorough history, a detailed and accurate clinical examination with appropriate radiographic assessment and interpretation, and assessment of risk factors.

Having made a diagnosis, it is then necessary to formulate a treatment plan. The treatment plan should be comprehensive in scope, and should include management of risk

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factors in addition to conventional periodontal therapies. Aspects that can be considered in the treatment plan are indicated in **Table 3.4**.

It is clear that successful periodontal therapy requires a motivated and compliant patient as well as a motivated clinician. Periodontal management should be considered a team effort, and it should be made clear to the patient that they have a role to play in managing their condition, and they will be supported in this by the dental team. Most periodontal conditions are chronic, representing long-term problems that require long-term solutions. Most importantly, behavior change is required of patients (e.g. to improve their plaque

▪ Table 3.4
Aspects to consider when formulating a treatment plan

Plaque control	Plaque control is the essential component of any periodontal therapy. Without plaque control, any form of periodontal treatment will fail. Oral hygiene instruction (OHI) should be provided that is relevant to the patient and is achievable. Don't overload the patient with too much information at once. Demonstrate toothbrush use in the patient's mouth. Explain the rationale for cleaning subgingivally. Reassure the patient that bleeding on brushing is not a reason to stop brushing. Suggested oral hygiene aids include: • Manual brush—use with crevicular technique to ensure bristles penetrate subgingivally • Powered brush—very different technique from manual brush, ensure patient is using correctly • Single tufted brush—excellent in recession defects, around single standing teeth, at areas of crowding • Floss—for interproximal cleaning, usually at sites with no attachment loss • Bottle brushes—essential for interproximal cleaning at sites with recession in periodontitis patients • Mouthrinses—may be used to supplement brushing but must not be used instead of brushing
Root surface debridement (RSD)	The cornerstone of periodontal therapy. The focus of RSD is the disruption of the subgingival biofilm and removal of plaque and calculus. Calculus is plaque retentive but otherwise does not directly contribute to periodontitis. Disruption of the biofilm is essential to reduce the bacterial challenge. This should then result in resolution of inflammation in the periodontal tissues, leading to shrinkage of the tissues, formation of a long junctional epithelium, and a reduction in probing depths. Shallower pockets are easier to maintain and clean than deep pockets, and inflammation is easier to control at shallow sites compared to deep sites. RSD can be undertaken with hand or ultrasonic instruments. Root planing is no longer an appropriate therapy, as it is clear that planing away the root surface does not benefit the patient, and can result in tooth sensitivity. For this reason RSD is the treatment of choice and involves multiple light strokes to disrupt the biofilm.
Risk factor management	Risk factor management complements OHI and RSD, and can take many forms: • Elimination of local risk factors (e.g. replacing overhanging restorations) • Smoking cessation (essential, but difficult to achieve) • Liaison with the medical practitioner to assist in medical management (e.g. of diabetes)
Periodontal surgery	Periodontal surgery may be the primary therapy in specific situations, e.g. connective tissue grafts to cover recession defects or gingivectomy surgery for drug induced gingival overgrowth. Periodontal surgery may also be indicated at specific nonresponding sites after effective nonsurgical management, and the aim of such surgery is to improve access for cleaning (for the clinician and the patient).
Adjunctive treatments	Adjuncts may be local (e.g. placement of local drug delivery systems) or systemic (e.g. systemic antibiotics or host modulating agents)

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control, to quit smoking, to attend long-term for periodontal maintenance care). In order for behavior to change, the patient must know the reason for the change, and therefore needs to be educated about periodontal disease and its management. This involves spending time talking to the patient to ensure they understand what is expected of them. The patient must be empowered to manage their problem, and must be supported by the dentist and hygienist.

The best way to empower and motivate a patient is to give them personalized biofeedback about their condition. This involves explaining the periodontal problem as is relevant to the patient, demonstrating problem areas in the mouth, and demonstrating solutions to those problems. Important areas to cover as part of this process include:

- Identifying the patient's long-term goals for their periodontal health, i.e. what do they want to achieve? For many patients, they will have goals such as preventing tooth loss, reducing bleeding or mobility, eliminating halitosis. The treatment plan must aim to achieve these goals, as this is the best way to achieve compliance.
- Set targets, based on the patient's goals as well as the clinician's goals. For example, set targets for plaque reduction, for probing depth reductions (such as: aim to reduce the number of pockets 5 mm or greater by 90%).
- Assess baseline oral hygiene habits (methods, frequency, duration) and then introduce better techniques (revise methods, frequency, duration), making new oral hygiene technique relevant to the clinical situation for that patient.
- Explain about periodontal disease by providing structured information about what has happened so far, the consequences if untreated, what can be expected following treatment, and the importance of plaque control.
- Explain about the importance of modifying behavior, such as quitting smoking and the benefits that can be expected.
- Reassure the patient, provide a supportive environment for them.
- Continually motivate and support the patient, help them to achieve the goals they want to achieve.

All of this takes time, but ultimately is much more likely to result in a successful outcome.

GINGIVITIS

Gingivae have a distinctive appearance that is clearly different from the inflamed gingival tissues seen in gingivitis. In this healthy patient, the gingival tissues are pink and noninflamed. The interdental papillae fill the interdental spaces below the contact point.

Gingivitis is defined as the inflammation of the gingival tissues. The most common form of gingivitis is plaque-induced gingivitis. The gingiva become erythematous, edematous, shiny, and soft or spongy in texture. Sulcus depths increase (false pockets) as a result of tissue swelling from inflammatory edema. The interdental papillae and marginal gingiva are initially involved before inflammation spreads to the attached gingiva. The gingival tissues may bleed on gentle probing. The clinical appearance of gingivitis (**Fig. 3.2**) is quite distinct from that of healthy gingival tissues (**Fig. 3.3**).

The inflammatory response that characterizes gingivitis is confined to the gingival tissues, and does not involve the other periodontal tissues. The condition may persist indefinitely.



Fig. 3.2: Clinical appearance of gingivitis. There is generalized erythema of the marginal gingival tissues as a result of plaque accumulation. The interdental papillae are swollen, and the interdental papilla between 13 and 12 is particularly inflamed

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Fig. 3.3: Clinical appearance of healthy gingiva. There is no gingival edema and no bleeding



Fig. 3.4: Clinical appearance of desquamative gingivitis associated with lichen planus. The inflamed tissues are fiery red in appearance and the inflammation affects the entire width of the keratinized gingiva. Lacy white striations typical of lichen planus can be seen at other anatomical sites

Gingivitis is reversible with effective plaque control and is so common within populations as to be almost considered a physiological condition rather than a disease state. This is a controversial statement, but is supported by the finding that even in clinically healthy tissues, if these are examined histologically, infiltrating inflammatory cells can always be identified (albeit in reduced numbers compared to gingivitis or periodontitis).

Treatment for gingivitis focuses around patient education to improve plaque control, together with necessary instrumentation:

- Instruction in toothbrushing and use of interdental cleaning aids
- Supra- and subgingival instrumentation (including any combination of ultrasonics, hand instruments, prophylaxis, etc.) to remove plaque and calculus and to disrupt the biofilm
- Elimination of plaque retentive factors.

Some other forms of gingivitis are reviewed in **Box 3.2**. These are much less common than plaque-induced gingivitis, but nonetheless are not rare. Among them desquamative gingivitis is a particular presentation of both oral lichen planus (**Fig. 3.4**) and mucous membrane pemphigoid.

PERIODONTAL DISEASES

The most frequently occurring form of periodontal disease is chronic periodontitis. The other important form of periodontitis that dentists must be aware of is aggressive periodontitis.

Chronic Periodontitis

According to the classification proposed by the American Academy of Periodontology in 1999, chronic periodontitis can be defined as 'an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment and bone loss'. The authors went on to say that chronic periodontitis is initiated and sustained by bacterial plaque, but host defence mechanisms play an integral role in its pathogenesis. Previous descriptions of periodontitis in the 1960s and 1970s focused solely on the etiological role of bacteria in their definitions. The fact that definitions of this common disease have changed over time reflects our improving understanding of the pathogenesis of the condition. Perhaps a reasonable definition of chronic periodontitis based on current understanding would be:

Chronic periodontitis is bacterially-induced inflammation of the periodontium.

This definition confirms the role of bacteria in initiating and perpetuating the condition, but firmly underscores the importance of inflammation in the pathogenesis of the disease. Indeed, the inflammatory response is responsible for the majority of tissue destruction seen in patients with periodontitis. Some of the characteristics of chronic periodontitis are indicated in **Table 3.3** and the clinical appearance

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Box 3.2: Different types of gingivitis

Pregnancy gingivitis

Etiology	Increased levels of circulating estrogen and progesterone during pregnancy exacerbate existing gingivitis. The hormones increase capillary permeability leading to increased erythema and edema.
Clinical features	Severity tends to increase throughout pregnancy. Often a degree of resolution during 3rd trimester and after parturition. A local gingival overgrowth (i.e. pregnancy epulis) may result from chronic irritation or mild trauma to the tissues.
Treatment	Prevention, education, plaque control, instrumentation.

Plasma cell gingivitis

Etiology	Precise details unclear, but is a contact hypersensitivity reaction often caused by dietary components (e.g. cinnamon). Microscopically, the epithelium is atrophic with massive infiltrate of plasma cells in the connective tissues.
Clinical features	Gingival tissues are fiery red, and the lesion involves the entire width of the attached gingiva. The lesion is also very sore.
Treatment	Identify the causative allergen. May require involvement of dermatologist for patch testing. Plaque control, gentle cleaning techniques.

Desquamative gingivitis

Etiology	Not a discreet clinical entity, but is a term used to describe a gingival manifestation common to plasma cell gingivitis and mucocutaneous disorders such as lichen planus and benign mucous membrane pemphigoid.
Clinical features	Fiery red, desquamative lesions affecting the entire width of keratinized gingiva and may be localized or generalized throughout the mouth **(Fig. 3.4)**
Treatment	Liaise with oral medicine specialist. Plaque control essential. Topical steroids may be required, potentially systemic steroids in advanced cases.

Necrotizing ulcerative gingivitis (NUG)

Etiology	A fusiform-spirochetal complex is traditionally associated with NUG. Ulceration of the gingival epithelium occurs with necrosis of the connective tissues. Superficially, deposits of fibrin are intermeshed with dead and dying epithelial cells, neutrophils and bacteria (creating the characterizing pseudomembranous sloughing). Deeper tissues demonstrate a dense infiltrate of neutrophils, characteristic of nonspecific inflammation.
Clinical features	'Punched out' ulcers covered with a yellow-gray pseudomembranous slough. The tips of the interdental papillae are affected first, rapidly followed by the marginal gingiva. NUG is very painful and there is also marked halitosis. A pre-existing gingivitis is usually also present. Fever and regional lymphadenopathy are associated with severe disease.
Risk factors	Pre-existing gingivitis (confirming a poor standard of plaque control). Smoking (major risk factor, favors gram-negative flora, impacts on immune responses). Mental stress (NUG often seen in college students and army personnel). Malnutrition (predisposes to infection and severe NUG, and may extend onto the external face, resulting in the very serious condition noma or cancrum oris). HIV infection (may also predispose to NUG that may progress to necrotizing ulcerative periodontitis)
Treatment	Reduce/stop smoking. Oral hygiene instruction, soft brush, mouthrinses useful if brushing very painful. Metronidazole 200 mg three times per day for 3 to 5 days. *Instrumentation:* Ultrasonics useful when pain starts to subside, careful prophylaxis. *Note:* Incomplete treatment can result in recurrence and eventually, loss of gingival contour, which may create esthetic problems.

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Saman Warnakulasuriya OBE BDS FDSRCS Dip Oral Med PhD DSc is a Professor of Oral Medicine and Experimental Pathology, King's College London, and an Honorary Consultant in Oral Medicine, King's and Guy's Hospitals, London, UK. He has published over 200 scientific papers and lectured extensively on the subject of oral cancer and precancer from the levels of basic science to the management. He is the Director of the World Health Organization (WHO) Collaborating Center for Oral Cancer and Precancer (2005-2015).



WM Tilakaratne BDS MS FDSRCS PhD FRCPath is a Professor of Oral Pathology, University of Peradeniya, Sri Lanka, and a Fellow of the Royal College of Pathologists, UK. He has extensive teaching experience in oral and maxillofacial pathology having lectured in different countries. He has published more than 100 scientific papers in the area of oral pathology, especially in the field of oral cancer and precancer. He was the councilor for Asia of the International Association of Oral Pathologists.



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