DIPLOMA OF PRIMARY CARE DENTISTRY

-RCSI-

PART – 1

CLINICAL SKILLS

PART 1: RESTORATIVE DENTISTRY

PERIODONTICS

B.PERIODONTICS:

∇ Classification of periodontal and peri-implant diseases and conditions:

1. Periodontal health, gingival diseases, and conditions:

- Periodontal and gingival health.
- Gingivitis: dental biofilm induced.
- Gingival diseases: non-dental biofilm induced.

2. Periodontitis:

- Necrotizing periodontal diseases.
- Periodontitis.
- Periodontitis as a manifestation of systemic disease.

3. Other conditions affecting the periodontium:

- Systemic diseases or conditions affecting the periodontal supporting tissues.
- Periodontal abscesses and endodontic-periodontal lesions.
- Mucogingival deformities and conditions.
- Traumatic occlusal forces.
- Tooth and prosthesis-related factors.

4. Peri-implant diseases and conditions:

- Peri-implant health.
- Peri-implant mucositis.
- Peri-implantitis.
- Peri-implant soft and hard tissue deficiencies.

∇ Classification of periodontal disease and conditions:

I. Gingival diseases:

A. Plaque induced:

1. Gingivitis associated with plaque only:

- Without local contributing factors.
- With other local contributing factors.

2. Gingival disease modified by systemic factors:

- Endocrine system: puberty-associated gingivitis, menstrual cycle-associated gingivitis, pregnancy-associated gingivitis, pyogenic granuloma, diabetes mellitus-associated gingivitis.
- Gingivitis associated with blood dyscrasias, e.g. leukaemia-associated gingivitis.

3. Gingivitis modified by medications:
- These would include drug-influenced gingival enlargement and drug-induced gingivitis, e.g. oral contraceptive-associated gingivitis and drug-induced gingival overgrowth due to phenytoin or ciclosporin.

4. Gingival disease modified by malnutrition:
- These would include ascorbic acid-deficiency gingivitis (scurvy) and gingivitis due to protein deficiency.

B. **Non-plaque induced**:
- These include gingival lesions of specific bacterial, viral, or fungal origin (e.g. 1° herpetic gingivostomatitis, % Herpes simplex virus), lesions of genetic origin (e.g. hereditary gingival fibromatosis), gingival manifestations of systemic conditions (mucocutaneous disorders, allergic reactions), traumatic lesions, and foreign body reactions.

II. Chronic periodontitis Localized. Generalized.
III. Aggressive periodontitis Localized. Generalized.
IV. Periodontitis as a manifestation of systemic disease
V. Necrotizing periodontal diseases Necrotizing ulcerative gingivitis (NUG). Necrotizing ulcerative periodontitis (NUP).
VI. Abscesses of the periodontium
VII. Periodontitis associated with endodontic lesions
VIII. Developmental or acquired deformities and conditions.

⇒ The largest change in the reclassification was undoubtedly in the working group relating to the diagnosis of periodontitis.
⇒ The new classification system requires the clinician to not only diagnose periodontitis and whether it is localized or generalized (there is an added distribution category of molar/incisor relationship), but to also comment on the stage and grade of the disease, to reflect on whether the disease is stable, in remission, or active, and finally to list the identified risk factors.
⇒ The distribution of localized or generalized is still based on **30% of sites affected.**

Epidemiology of periodontal disease:

⇒ Various scoring systems such as gingival, plaque, and periodontal indices for measuring periodontal disease have been developed, some for use at a population level (Community Periodontal Index, Community Periodontal Index of Treatment Needs (CPITN)) and some for screening and management of individual patients (e.g. BPE). There is no single ideal index.

⇒ Gingivitis precedes periodontitis but there is no evidence to suggest that periodontitis develops in the absence of gingivitis.

⇒ Although mild to moderate periodontitis is common, severe periodontitis affects a relatively small subset of the population.

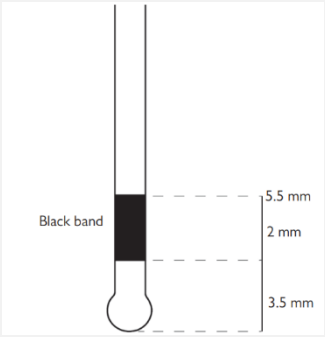
⇒ Certain risk factors such as smoking, poorly controlled diabetes, and colonization by specific bacteria at high levels have been identified.

Basic Periodontal Examination:

∇ The BPE was developed from **the CPITN,** it is a simple screening tool that will be used to determine what further examination is needed and therefore provides a direction of the next phase of Rx.

∇ This technique is used to screen for those patients requiring more detailed periodontal examination in the dental practice setting. It examines every tooth in the mouth (except third molars), thus taking into account the site-specific nature of periodontal disease.

∇ A World Health Organization (WHO) periodontal probe is used:



- It has a ball end which is 0.5mm, then a coloured (normally black) band from 3.5 to 5.5mm.
- The mouth is divided into sextants, two buccal and one labial segment per arch.
- All teeth in each segment are explored and the highest score per sextant recorded, usually in a simple six-box chart.
- The probe is 'walked' around the sulcus in each sextant with a light probing force (20–25g) and the highest score is recorded in each sextant.
- For a sextant to qualify for recording, it must contain at least two teeth.

⇒ **When to record the Basic Periodontal Examination:**

1. All new patients should have a BPE recording including children and adults.
2. For patients with BPE code 0, 1, or 2 on a previous BPE recording, the BPE should be followed up at each follow-up examination.
3. For patients with BPE codes 3 and 4, further detailed periodontal charting would be needed and further Rx as indicated in the following sections.
4. BPE should not be used around implants.
5. Six-point pocket charting is recommended instead.

0 = pockets <3.5 and the first black band is completely visible:

- Healthy periodontal tissues.
- No bleeding after gentle probing.
- No calculus and no overhangs on restorations.
- Rx: no need for periodontal Rx.

1 = pockets <3.5 and the first black band is completely visible

- Gingival bleeding after gentle probing.
- No pockets >3
- No calculus.
- No plaque retaining factors (e.g. overhanging restoration).
- Special investigations would not be needed; however, it is important to note that any recession would be unaccounted for.
- Rx: OHIs.

2 = pockets <3.5mm. First black band is visible, but plaque retention factors present (calculus/overhang)

- Plaque and bleeding charts can be recorded as part of special investigations.
- Rx: OHIs plus removal of plaque retentive factors including sub- and supragingival calculus.

3 = coloured area of probe remains partly visible in deepest pocket in sextant l deepest pocket 4 or 5mm

- More detailed periodontal charting required.
- Plaque and bleeding charpockets 6mm.
- More detailed periodontal charting required for the entire dentition.
- Plaque and bleeding charts can be recorded as part of special investigations.
- Radiographs should be considered to assess bone levels and to establish true attachment loss.

- Rx: initial therapy including self-care advice (OHIs and risk factor control), record six-point periodontal pocket chart of involved sextants at a review 3 months post initial therapy. If pocket depths persist despite initial therapy and excellent OH, root surface instrumentation would be required.

4 = coloured area of probe disappears into pocket l one or more teeth in sextant has a pocket >6mm

- More detailed periodontal charting required for the entire dentition.
- Plaque and bleeding charts can be recorded as part of special investigations.
- Special investigations would include radiographs to assess bone levels and to establish true attachment loss.
- Rx: OHIs, six-point periodontal pocket chart, root surface debridement, and referral to a specialist may be indicated.

**** = furcation involvement***

- More detailed periodontal charting required for the entire dentition.
- Plaque and bleeding charts can be recorded as part of special investigations.
- Special investigations would include radiographs.
- Rx: treat according to BPE code. More complex Rx and referral to a specialist may be necessary.

⇒ Patients with a sextant code of 4 or * will require a full probing depth chart, plus recordings of mobility, recession, and furcation involvement, and radiographs. The BPE cannot be used for close monitoring of the progress of Rx. If the black band disappears on probing a pocket, perform a full periodontal examination in that sextant.

⇒ BPE probing is not appropriate for implants sites. The soft tissue connection to implants is not the same as teeth, therefore peri-implant soft tissues are less resistant to probing. In addition, the position of the implant in relation to the bone and soft tissues may present deeper probing depths.

⇒ The BPE cannot be used to monitor the response to periodontal Rx because it cannot provide information on how sites change within a sextant, after Rx. The BPE is a screening tool.

Oral microbiology:

- The mouth is colonized by microorganisms a few hours after birth, mainly by aerobic and facultative anaerobic organisms.
- The eruption of teeth allows the development of a complex ecosystem of microorganisms.
- More than 700 different species can colonize the mouth and >400 species may be found in periodontal pockets.
- Resident oral microflora form multi-species biofilms on oral surfaces. In health there is a balanced relationship between oral microflora and the host which is mutually beneficial.
- The resident microflora are important in preventing colonization by exogenous microbes.
- Some resident oral bacteria can reduce dietary nitrate to nitrite which confers benefits on the host cardiovascular and gastrointestinal systems.
- Microbial composition alters with health and disease.
- The composition of the biofilms varies with the site: biofilms in occlusal fissures are mainly Gram +ve and facultatively anaerobic. They metabolize host and dietary sugars. Biofilms in periodontal pockets have large amounts of obligately anaerobic Gram –ve rods and cocci and are proteolytic in metabolism.

Microorganisms worth noting:

1. Streptococcus mutans group:
   - Several species are recognized within this group, including S. mutans and S. sobrinus. Facultative anaerobe. Synthesizes dextrans, l plaque formation.
   - Colony density rises to >50% in presence of high dietary sucrose. Able to produce acid from most sugars.
   - Most important organisms in the aetiology of caries.

2. S. oralis group:
   - Includes S. sanguinis, S. mitis, and S. oralis.
   - Account for up to 50% of streptococci in plaque.
   - Heavily implicated in 50% of cases of infective endocarditis.
   - These are pioneer species.

3. S. salivarius group:
   - Accounts for about half the streptococci in saliva.
   - Inconsistent producer of dextran.

4. S. intermedius, S. anginosus, S. constellatus (Formerly S. milleri group.):
   - Common isolates from abscesses in the mouth and at distant sites.
   - Believed to contribute to periodontal disease progression.

5. Lactobacillus 2° colonizer in caries:
   - Very acidogenic.
   - **Often found in dentine caries.**

6. Porphyromonas gingivalis:
   - Obligate anaerobe associated with chronic periodontitis and aggressive periodontitis.

7. Aggregatibacter actinomycetemcomitans:
   - Microaerophilic, capnophilic, Gram –ve rod.
   - Particular pathogen in aggressive periodontitis.

8. Tannerella forsythia:
   - Anaerobic, Gram –ve
   - Implicated in periodontal diseases.

9. Prevotella intermedia:
   - Found in chronic periodontitis, LAP, necrotizing periodontal disease, and areas of severe gingival inflammation without attachment loss.

10. P. nigrescens:
   - New, possibly more virulent.

11. Fusobacterium Obligate anaerobes:
   - Originally thought to be principal pathogens in necrotizing periodontal disease. Remain a significant periodontal pathogen.
   - These species are known to be ‘quorum sensing’ organisms that are capable of sensing and influencing their environments via chemical cues.
   - This can elicit a stronger host response.

12. Spirochaetes:
   - Obligate anaerobes implicated in periodontal disease; present in most adult mouths.
   - Borrelia, Treponema, and Leptospira belong to this family.

13. Actinomyces israelii:
   - Filamentous organism; major cause of actinomycosis.
   - A persistent rare infection which occurs predominantly in the mouth and jaws and the female reproductive tract.
   - **Implicated in root caries.**

Host defences:

⇒ The host response to the biofilm is meant to be protective but can also cause local tissue damage.

⇒ Both inflammatory and immunologically mediated pathways can contribute to periodontal damage.

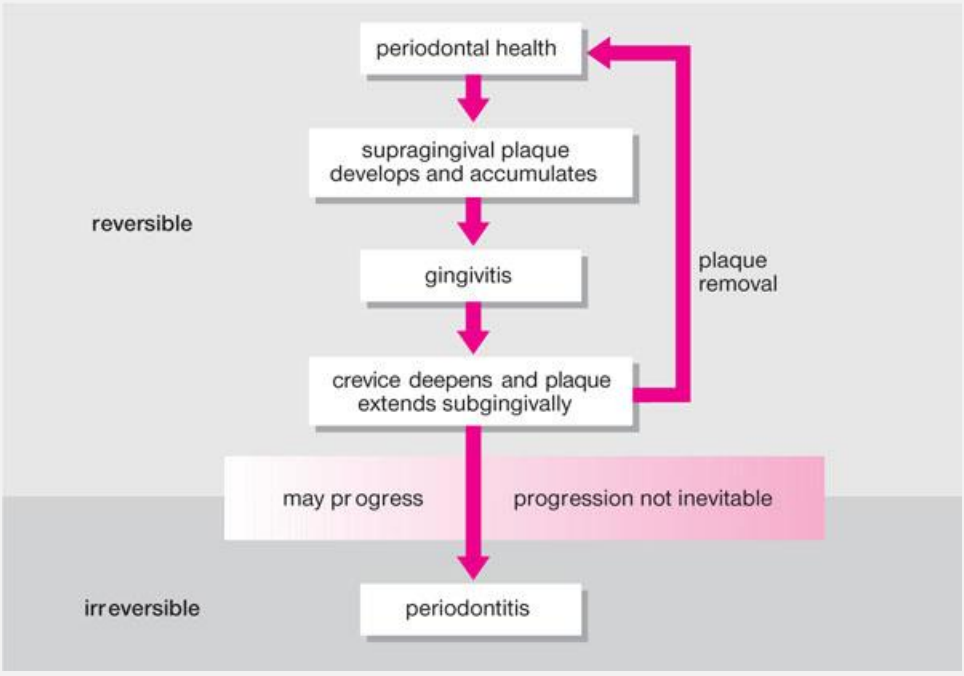
➢ Innate host defences:

- Intact epithelium acts as a physical barrier.
- If junctional epithelium develops into pocket epithelium its protective function is due to its permeable structure.
- Supragingivally, saliva prevents drying of the oral tissues and has antimicrobial effects via salivary IgA, salivary peroxidase, lysozyme, and lactoferrin.
- The inflammatory response is relatively non-specific.
- There is a fluid component in the form of gingival crevicular fluid.
- This washes out non-adherent bacteria from the crevice and contains inflammatory mediators (cytokines, prostaglandins, and matrix metalloproteinases).
- The cellular component includes neutrophils and macrophages.

➢ Specific immune response:

a. Humoral response: involves antibody production.
b. Cell-mediated response: T-helper cells produce cytokines, assist in the differentiation of B cells to plasma cells, and activate neutrophils and macrophages.

⇒ Systemic risk factors can modify this host response.



Plaque biofilm:

- Dental plaque, which is a biofilm, is an adherent mass of diverse micro-organisms in a muco-polysaccharide matrix.
- It cannot be rinsed off but can be removed by brushing.
- Biofilms are made up of symbiotic communities of different microorganisms.
- They develop in a structured way and are spatially and functionally organized.
- The species within communicate with each other.
- They are less susceptible to host defences and antimicrobial agents than planktonic bacteria.
- Resident bacteria can dampen the immune response via communication with mucosal cells. If this balanced coexistence breaks down, disease can occur.

⇒ It forms in stages: Biofilm formation Although it is possible for plaque to collect on irregular surfaces in the mouth, to colonize smooth tooth surfaces it needs the presence of acquired pellicle.

⇒ This is a thin layer of salivary glycoproteins, formed on the tooth surface within minutes of polishing. The pellicle has an ion-regulating function between tooth and saliva and contains immunoglobulins, complement, and lysozyme.

⇒ Bacteria recolonize the tooth surface in a predictable sequence. The pioneer species are attached by weak van der Waals forces (reversible adhesion).

⇒ It leads to a stronger, irreversible attachment.

⇒ Co-adhesion of the new colonizers to the already attached bacteria l diversity.

⇒ Attached organisms multiply and biofilm forms. Bacteria synthesize extracellular matrix.

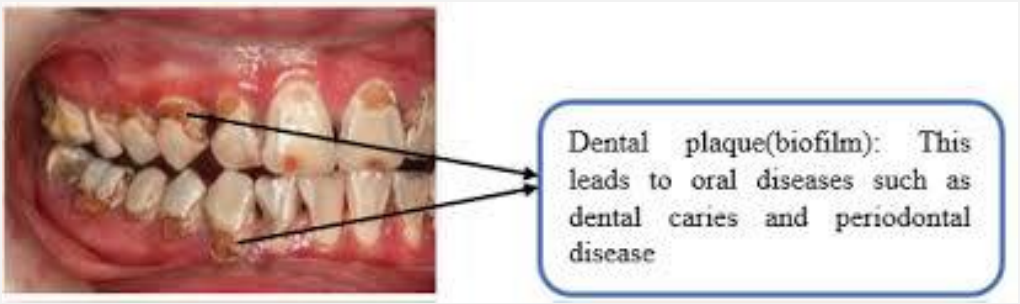
⇒ Detachment of cells from the biofilm allows colonization of new surfaces.

⇒ Cocci predominate in plaque for the first 2 days, following which rods and filamentous organisms become involved.

⇒ This is associated with i numbers of leucocytes at the gingival margin.

⇒ Between 6 and 10 days, if no cleaning has taken place, vibrios and spirochaetes appear in plaque and this is associated with clinical gingivitis.

⇒ **It is generally felt that the move towards a more Gram –ve anaerobe-dense plaque is associated with the progression of gingivitis and periodontal disease.**



Summary points for plaque biofilm:

- Plaque biofilm develops in a structured manner.
- It starts by development and formation of the acquired pellicle.
- Streptococci are the earlier species to develop.
- Formation of a biofilm and the presence of plaque has a direct correlation with gingivitis.
- Fusobacterium nucleatum are quorum sensing species that can initiate an environmental change towards the more pathogenic species.
- Incipient dysbiosis may occur.
- If the host response is such that there is resulting periodontal tissue damage, frank dysbiosis will occur.

Calculus:

⇒ Calculus (tartar) is a calcified deposit found on teeth (and other solid oral structures) and is formed by mineralization of plaque deposits. The mineral content of supragingival calculus derives from saliva, that for subgingival is from gingival crevicular fluid.

1. Supragingival calculus:

- This is most often found opposite the openings of the salivary ducts, opposite the parotid (Stensen's) duct and on the lingual surface of the lower anterior teeth opposite the submandibular/sublingual (Wharton's) duct.
- It is usually creamy-coloured but can become stained a variety of colours.

2. Subgingival calculus:

- This is found (not surprisingly) underneath the gingival margin and is firmly attached to tooth roots. It tends to be brown or black, is extremely tenacious, and is most often found on interproximal and lingual surfaces.
- It may be identified visually, detected by touch using a BPE or CPITN or any periodontal probe, or on radiographs.
- It is associated with subsequent periodontitis in some cases. With gingival recession it can become supragingival.

- **Composition:**

- It consists of up to 80% inorganic salts, mostly crystalline, the major components being calcium and phosphorus.
- The microscopic structure is basically that of a randomly orientated crystal formation.
- There are different morphological types (octacalcium phosphate, hydroxyapatite, whitlocktite, brushite).

- **Formation:**

- Calculus is always preceded by plaque deposition, the plaque serving as an organic matrix for subsequent mineralization.
- Initially, the matrix between organisms becomes calcified with, eventually, the organisms themselves becoming mineralized. Subgingival calculus usually takes many months to form, whereas friable supragingival calculus may form within 2 weeks.

- **Pathological effect:**

- Calculus (particularly subgingival calculus), is associated with periodontal disease.
- This may be because it is invariably covered by a layer of plaque.
- Its principal detrimental effect is probably that it acts as a retention site for plaque and bacterial toxins.
- The presence of calculus makes it difficult to implement adequate OH.
- Anti-calculus dentifrices contain crystal growth inhibitors, e.g. triclosan, zinc citrate, to prevent formation of supragingival calculus.
- They have not been shown to be effective against subgingival deposits.

- **Progression:**

- From gingivitis to periodontitis can occur as there is a shift from 'friendly' commensal bacteria to periodontopathic bacteria and their products and the host response that ensues.
- The way in which plaque does this is complex.
- It involves the oral environment, the pathogenicity of organisms, host defence, and plaque maturity.
- Some individuals may have large amounts of plaque without developing periodontitis, others may have periodontal destruction with relatively small amounts of plaque.
- The shift of microbial species in the gingival sulcus from Gram +ve facultative fermentative organisms to predominantly Gram –ve anaerobic and proteolytic organisms has been strongly associated with periodontal break-down previously and the associated changes in the host response have also been associated.

➢ **Risk factors:**

A. Local factors:

- Those which predispose to plaque accumulation, like tooth position and morphology, calculus, overhangs and appliances, occlusal trauma, and mucogingival state.

B. Systemic factors:

- Those which modify the host response, like smoking, diabetes, obesity, genetic factors, immune status, stress, age, and nutrition.
- Modifiable risk factors such as smoking are important in managing periodontal disease.

Clinical features of gingivitis and periodontitis:

A. Gingivitis:

- The classic triad of redness, swelling, and bleeding on gentle probing are diagnostic and are usually associated with a complaint by the patient that their 'gums bleed on brushing'.
- The 'knife-edge' margins and stippled appearance associated with health disappear and are replaced by a more rounded, shiny appearance.
- Pain is not usually a feature.
- Halitosis may be present.
- Affects gingiva only.
- It is not associated with alveolar bone resorption or apical migration of the junctional epithelium.
- Probing depths >3mm can occur in chronic gingivitis due to an increase in gingival size because of oedema or hyperplasia (false pockets).

⇒ The recent 2017 World Workshop on the Classification of Periodontal Diseases came up with working definitions of health and gingivitis:

Box 5.6 Patients with an intact periodontium

I. Health

- Probing attachment loss: no.
- PPD: ≤3mm.
- BOP: <10%.
- Radiographic bone loss: no.

II. Gingivitis

- Probing attachment loss: no.
- PD: <3mm.
- BOP: ≥10%.
- Radiographic bone loss: no.

⇒ They also defined a patient who had a reduced periodontium but not through periodontitis, a patient who had crown lengthening or the distal aspect of a second molar where there had been an impacted third molar removed:

Box 5.7 Patients with a reduced periodontium

I. Health
- Probing attachment loss: yes.
- PPD: ≤3mm.
- BOP: <10%.
- Radiographic bone loss: possible.

II. Gingivitis
- Probing attachment loss: yes.
- PPD: <3mm.
- BOP: ≥10%.
- Radiographic bone loss: possible.

B. Chronic periodontitis:

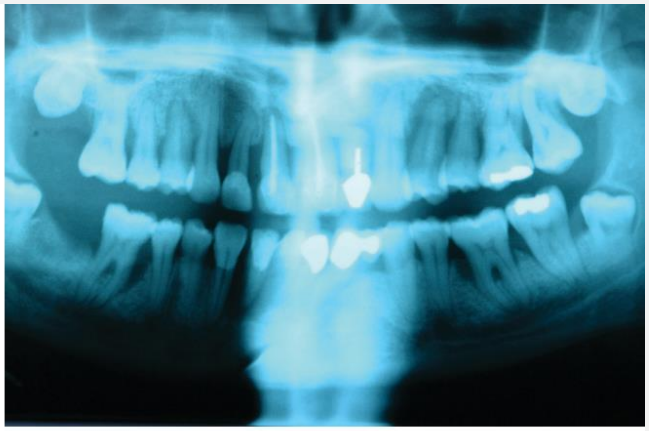


Fig. 5.2 DPT showing the typical appearance of established periodontitis; the patient was a diabetic who smoked.

- Clinical signs may include gingival inflammation and bleeding, pocketing, gingival recession, tooth mobility, tooth migration, discomfort, and halitosis.
- It affects gingiva, PDL, cementum, and alveolar bone.
- At earlier stages there is usually very little in the way of obvious signs or symptoms therefore probing is essential.
- It can be regarded as a progression of the combination of infection and inflammation of gingivitis into the deep tissues of the periodontal membrane.
- All periodontitis develops out of gingivitis but not all gingivitis progresses to periodontitis.
- Some people with poor OH may develop gingivitis but not periodontitis.
- Some people with good OH and little in the way of gingivitis may develop periodontitis.
- The proportion of sites that do progress in a subject or population is not known, and the factors l progression are not well understood.
- Periodontitis is classified as localized when 30% of sites are affected.

⇒ The staging of the disease describes the degree of attachment loss.
⇒ The grade describes the rate of progression of the disease to date.

Radiographic examination:

- Used to support clinical diagnosis in cases with BPE scores of 3, 4, or *, in monitoring the bone levels and in monitoring stability of periodontal health.
- Standardized sequential radiographs allow monitoring of disease.
- Radiographs can be used to aid diagnosis and help determine the prognosis of specific teeth when combined with the clinical examination, patient history, and risk factors.
- All radiographs should be able to view crestal bone levels for initial assessment and also for long-term review to track changes in bone level over time.
- Care should be taken to ensure that each exposure is suitably justified and provides clear benefit to the patient.
- The number and type of radiographs will depend on findings during the clinical examination.

  - Horizontal bitewings provide a good view of interproximal bone and are useful for relatively minor degrees of bone loss (pocketing<5mm) and to detect calculus deposits.

  - Horizontal bitewings are routinely taken in practice to assess caries and thus they can give an indication of the interproximal bone loss and also any plaque retentive factors such as poorly contoured restorations. Subgingival calculus would be detected in such radiographs too.

  - Vertical bitewings may be indicated when there are relatively mild degrees of bone loss (where pocketing is<5mm) Vertical bitewings provide a view of the crestal bone levels in relation to the CEJ in opposing arches. Vertical bitewings are difficult to take in patients with shallow palatal arches &/or shallow floor of mouth, and periapical radiographs may be more suited. In addition, vertical b/ws may not show the full root length hence making it difficult to assess the percentage of bone loss in relation to the root length.

  - Full mouth periapicals (preferably of the long cone parallel technique), have been the radiographs of choice for patients with severe periodontal disease. They can clearly demonstrate root surface deposits, furcation involvement, extensive bone loss, intrabony pocketing, and periodontal–endodontic lesions. Periapical radiographs also allow for assessment of the root morphology.

  - Full mouth periapical radiographs also allow for assessment of the extent of bone loss in relation to the root length and thus can provide a more accurate way to classify the grading according to the new periodontal classification.

➢ There is no indication for DPT for routine screening purposes; however, if the patient is not able to tolerate IO radiographs due to reasons such as gagging, then an OPT may be considered to assess bone levels in periodontitis patients.

Diagnosis and monitoring:

- Take a good history.
- Consider any medical or systemic factors and any other risk factors such as smoking.
- Conduct a BPE recording and a more advanced six-point periodontal charting for the greater BPE scores.
- Take appropriate radiographs for monitoring.
- Consider your diagnosis from a periodontal perspective based on the classification system and any other associated diagnosis (periodontal–endodontic lesions, caries, etc.).

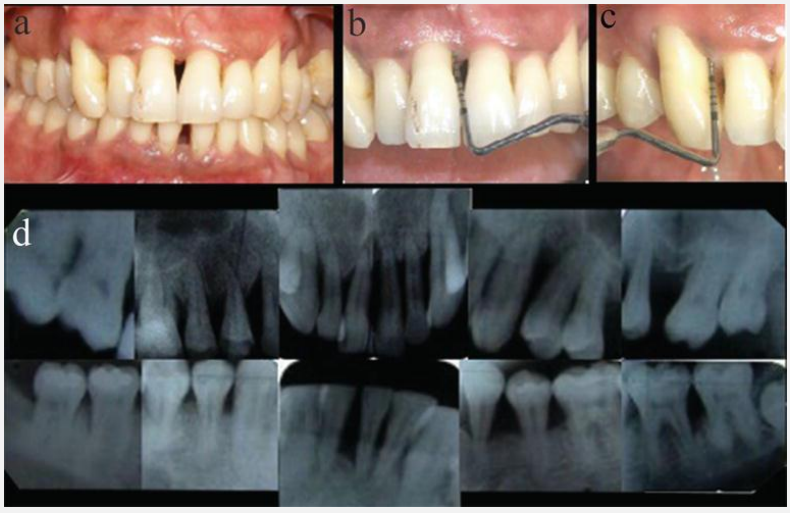
Aggressive periodontitis:

- It is a type of periodontitis in the 1999 classification and replaced disease terminology like early-onset periodontitis in previous classification systems.
- Aggressive periodontitis historically described a group of rare and often severe, rapidly progressive forms of periodontitis.
- Often characterized by an early age of onset and tending to occur in families and with non-contributory medical history.
- The amounts of plaque are out of proportion with the severity of periodontal destruction.
- Often associated with **Aggregatibacter actinomycetemcomitans.**

⇒ Two main forms:

a. Generalized aggressive periodontitis (GAP): previously known as generalized juvenile periodontitis.

b. Localized aggressive periodontitis (LAP): previously known as localized juvenile periodontitis.



Treatment:

- Achievement of adequate supragingival plaque control.
- Subgingival instrumentation to disrupt biofilm but this may not eradicate virulent organisms.
- Non-surgical approach with adjunctive use of systemic antibiotics is the preferred Rx option.
- Amoxicillin/metronidazole combination seems to provide additional benefit to non-surgical management. In the situation where amoxicillin or metronidazole cannot be prescribed, azithromycin has a good evidence base.
- Surgery has a role but there is no consensus regarding the use of systemic antibiotics for this approach.
- Increase evidence that regenerative surgical techniques are a suitable option for defects associated with aggressive periodontitis.
- Regular supportive care is important.

Periodontal abscess:

- This is a localized collection of pus within the tissues adjacent to a periodontal pocket. It occurs either due to the introduction of virulent organisms into an existing pocket or d drainage potential.
- Commonly occurs in furcations.
- Multiple or recurrent abscesses may indicate underlying immunocompromise, like poorly controlled diabetes.
- Clinically there may be swelling, pus from a pocket or sinus, pain, tenderness to percussion, and signs of periodontitis.
- There may be systemic involvement.
- Differential diagnosis Gingival abscess, pericoronal abscess, periapical abscess, combined periodontal/endodontic lesion, or other (cyst/tumour).

Table 5.1 Features of periapical vs periodontal abscess

Periapical abscess	Periodontal abscess
Non-vital	Usually vital
TTP vertically	Pain on lateral movements
May be mobile	Usually mobile
Loss of lamina dura on X-ray	Loss of alveolar crest on X-ray

Periodontitis associated with endodontic lesions:

- It is essential to sensibility test any heavily restored tooth with periodontal involvement.
- A combined periodontal–endodontic lesion is where both lesions coalesce regardless of whether the origin is primarily periodontal (necrotic pulp due to periodontal involvement) or primarily endodontic (periodontal tissues involved after pulp necrosis).
- Given the relative frequency of both periodontal disease and periapical pathology, it is not surprising that both may occur together, which can result in diagnostic confusion.
- In fact, there is little evidence to support the popular notion that periodontitis leads to pulp necrosis.
- However, there is no doubt that pulp pathology can exacerbate periodontal problems.

⇒ **Treatment of combined lesion:**

1. First, resolve the acute infection and inflammation by drainage (&/or antibiotics), then treat with orthograde RCT (the greater the pulpal component, the better the prognosis).
2. The apparent periodontal lesion will often be seen to resolve to a substantial degree over a period of months, therefore, the decision to carry out surgery should be deferred.
3. Combined apical surgery and periodontal surgery is quite feasible but carries a poorer long-term prognosis.
4. The worst prognosis applies to those teeth where the periapical/pulpal pathology has been due entirely to apical extension of the periodontal pocket.
5. These are often diagnosed after the fact when endodontics completely fails to resolve the lesion.

⇒ **Principles of treatment:**

- Establish diagnosis, based on current classification.
- Record location, extent, and severity and any associated risk factors.
- The overall aim could be summarized as the creation of a healthy periodontium which the patient is both capable of, and willing to, maintain.

- It is often convenient to divide the principles of periodontal therapy into three phases:

1. <u>The initial (cause-related) phase:</u>

- This is where the aim is to control plaque and address modifiable risk factors (smoking cessation counselling, liaise with GMP if poorly controlled diabetes).
- Periodontal disease is an infection due to the presence of plaque biofilm, therefore, disruption of the plaque biofilm and control of plaque is the key to success.
- More complex Rx will always fail in the absence of effective plaque control.
- Includes recording of baseline indices, OHI, scaling and root surface debridement, and elimination of plaque retention factors.
- Response is monitored 8–12 weeks after Rx and a further plan made.
- If successful, can move to supportive phase.
- If residual disease, then move to corrective phase.

2. <u>The corrective phase:</u>

- This is designed principally to restore function and, where relevant, aesthetics.
- Corrective techniques include further non-surgical therapy, periodontal access surgery, regenerative surgery, mucogingival surgery, resective surgery (like gingivectomy), selected use of local and systemic antibiotics where indicated, Rx of furcation lesions, restorative work, endodontics, and occlusal adjustment.

- The aims of this phase are to:
  - ∂ Eliminate pathological periodontal pockets, or to create a tight epithelial attachment where the pocket once existed.
  - ∂ Arrest loss of, and in some cases improve, the alveolar bone support.
  - ∂ Create an oral environment which is relatively simple for the patient to keep plaque free.

3. <u>The supportive phase:</u>

- ∂ This aims to reinforce a patient's motivation so that their OH is adequate to prevent recurrence of disease.
- ∂ This phase is receiving increase attention due to the relative ease with which disease activity can be monitored by probing and chair-side diagnostic assay.

⇒ **Non-surgical treatment:**

- Non-surgical Rx is best done after good OHIs have been provided and techniques demonstrated.
- Non-surgical Rx is normally done under LA at one quadrant per visit.
- Ultrasonics are used to disrupt the biofilm and mechanically remove supra- and subgingival deposits and these can be supplemented with hand instruments.
- There is limited evidence to show the difference between a quadrant approach and full mouth disinfection; however, both offer tangible benefits to patients.

⇒ **Periodontal Surgery:**

- Periodontal surgery is contraindicated in the presence of plaque and poor plaque control by the patient.
- Periodontal surgery is mostly considered after a course of non-surgical debridement.
- Periodontal surgery may be indicated for a variety of reasons and the choice of the surgical procedure is dependent on the desired outcome.
- A very brief overview of the main steps is given.

Furcation involvement:

⇒ Classification:

1. **Class 1**:

- Probe can be inserted<3 mm between the roots.
- Requires scaling and root planning, possibly with furcation plasty.

2. **Class 2**:

- Horizontal probing depth exceeding 3mm but not extending fully through the width of the furcation area.
- GTR together with graft materials and EMD can be used to treat class 2 furcations.

3. **Class 3**:

- Horizontal through-and-through destruction in the furcation area.
- May require tunnel preparation, &/or root resection, &/or extraction.
- GTR less predictable with Class 3 defects.

The End